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A Fatal Complication of Otitis Externa: Subdural Empyema and Bacterial Meningitis – A Case Report

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Introduction

Otitis externa is a common inflammatory condition of the external auditory canal, typically presenting as a superficial and benign disease. However, in the presence of risk factors such as advanced age, immunosuppression, or diabetes mellitus, the infection may become invasive and extend to adjacent anatomical structures. Despite the widespread use of antibiotics, otologic infections can still lead to intracranial complications. Particularly in cases of delayed treatment, the infection may spread to the mastoid air cells, temporal bone, and even meningeal tissues, resulting in life-threatening complications such as mastoiditis, subdural empyema, and bacterial meningitis. This report presents a rare and severe clinical case where an initial diagnosis of otitis externa progressed to mastoiditis and subsequently to subdural empyema and meningitis due to contiguous spread of the infection.

Case

A 70-year-old male patient was brought to the emergency department by his relatives with complaints of headache, speech disturbance, and impaired cooperation. Vital signs were stable. The patient had a known history of hypertension but no history of immunosuppression, diabetes, or chronic otitis. According to the relatives, he had been diagnosed with otitis externa approximately one week earlier and was prescribed topical antibiotic treatment.

On the physical examination, the right external auditory canal was edematous and hyperemic, with evident serous discharge. Neurologically, the patient was alert but disoriented, exhibiting nuchal rigidity and a positive Kernig's sign.

With a preliminary diagnosis of meningitis, cranial imaging and lumbar puncture were planned. Laboratory findings included leukocytosis (20,33×10⁹/L), elevated procalcitonin (7,33 µg/L), and increased CRP (171,4 mg/L). Cranial CT revealed decreased aeration of the right mastoid air cells (Figure 1) and a 6 mm subdural collection in the right frontoparietal region (Figure 2). Cerebrospinal fluid (CSF) analysis revealed markedly elevated protein concentration (978.78 mg/L), increased albumin levels (372.3 mg/L), and pleocytosis (120 cells/mm³), while glucose levels remained within normal limits. Polymerase chain reaction (PCR) testing via a molecular meningitis panel identified Streptococcus pyogenes as the causative pathogen.

Following the diagnosis, empirical antibiotic therapy with ceftriaxone, vancomycin, and ampicillin were initiated within the first hour. The patient was admitted to the intensive care unit and managed with a multidisciplinary approach.

Discussion

Although otitis externa commonly presents as a localized and self-limited infection, in some cases it may progress with deep tissue invasion and result in serious complications. In this case, the infection extended to the mastoid air cells and meningeal tissues, culminating in subdural empyema and bacterial meningitis. Topical antibiotics are generally preferred in the treatment of otitis externa. However, systemic antibiotic therapy should be considered in the presence of periauricular spread, systemic symptoms (fever, altered mental status, nuchal rigidity), or failure to respond to topical treatment. The patients presenting to the emergency department with otologic complaints accompanied by neurological findings, intracranial complications must be ruled out. Head and neck CT and MRI play a critical role for diagnosis. MRI is particularly useful when identifying subdural collections and meningeal involvement. CSF analysis including evaluation of protein levels, leukocyte count, and glucose concentration provides important diagnostic clues.

In this case, Streptococcus pyogenes, a rare but aggressive etiologic agent of meningitis, was isolated from the CSF. Early diagnosis and initiation of empirical antimicrobial therapy are essential for favorable clinical outcomes.

In emergency medicine, otologic complaints should not be underestimated. Especially in elderly or high-risk patients, advanced diagnostic workups, including imaging and CSF analysis may be lifesaving. This case demonstrates that seemingly benign external ear infections can rapidly evolve into systemic and neurological emergencies.

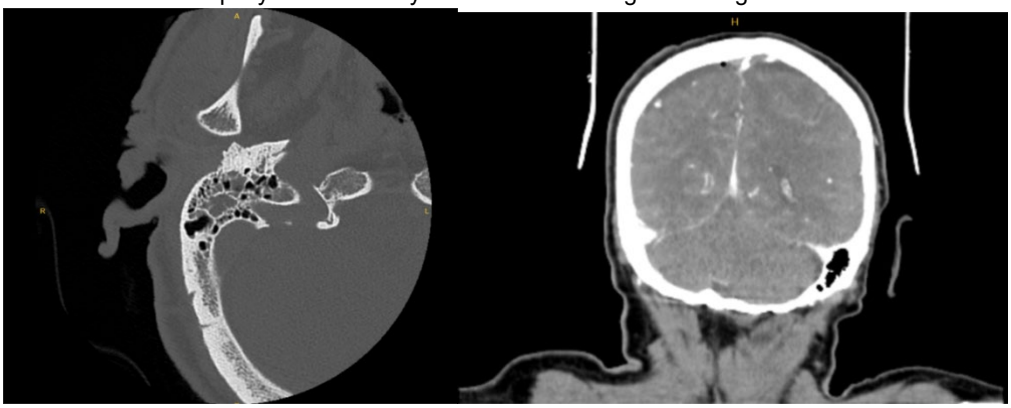

Figure 1: Decreased aeration in the right mastoid air cells

Figure 2: Subdural empyema measuring 6 mm in the right frontoparietal region

Conclusion

Although the combination of mastoiditis, subdural empyema, and meningitis following otitis externa is rare, it poses a high risk of

morbidity and mortality. Therefore, elderly patients presenting with systemic signs such as headache or altered consciousness, clinicians should maintain suspicion. Prompt imaging and CSF analysis are crucial for early diagnosis. Early initiation of antibiotic therapy and a multidisciplinary treatment approach are key factors in improving patient outcomes.

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Rare Intussusception in Pregnancy: Challenges in Diagnosis and Treatment

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Introductıon

Intussusception is a rare pathology characterized by the telescoping of one segment of the intestine into the lumen of a distal segment (1). While it is commonly observed in childhood, it is exceptionally rare in adults and even more so during pregnancy (2). Factors such as hormonal changes, increased intra-abdominal pressure due to uterine growth, and physiological slowing of bowel motility during pregnancy may contribute to the risk of intussusception (3). However, because pregnancy-related changes can result in atypical symptom presentation, diagnosis may be delayed. Therefore, intussusception should always be considered in the differential diagnosis of pregnant patients presenting with abdominal pain and bowel obstruction symptoms (4). This case report details a 28-week pregnant patient who developed small bowel intussusception and her clinical management.

Case Report

A 23-year-old, 28-week pregnant patient presented to an external emergency department with complaints of abdominal pain and was referred for further examination and treatment. The primary complaint was abdominal pain that had persisted for 5 h, initially mild but increasing in severity over time. Additionally, she reported nausea and loss of appetite. Her obstetric history was unremarkable, and she had no prior abdominal surgeries.

On physical examination, the patient was in good general condition, cooperative, and oriented, with stable vital signs. Abdominal examination revealed significant tenderness in the left upper and lower quadrants, although there were no signs of rebound or guarding. The uterus was palpated in accordance with the gestational age, and no signs of preterm labor were detected. The laboratory results were as follows: WBC, 9.29; Hgb, 11.2; and CRP, 0.80, with normal liver and kidney function tests.

Obstetric ultrasonography showed fetal biometry consistent with 28 weeks of gestation, a posteriorly located placenta, and a normal amniotic fluid index. However, an abdominal ultrasound performed at the external center revealed bowel wall thickening and lumen narrowing over a 9 cm segment in the left upper and lower quadrants. Due to suspected intussusception, urgent consultations with obstetrics and general surgery were requested. Advanced imaging was planned to evaluate for mechanical bowel obstruction, but the patient was non-compliant with MRI. Therefore, she was admitted to the general surgery ward for monitoring and treatment.

During her follow-up in the general surgery ward, there was no increase in acute phase reactants, abdominal findings improved, and bowel movements resumed. After 24 h of observation, the patient was discharged without requiring surgical intervention.

Dıscussıon

Intussusception during pregnancy is a rare but high-risk gastrointestinal pathology with significant maternal and fetal morbidity. The incidence of intussusception in adults is low and is often associated with an underlying predisposing factor (e.g., tumors, inflammatory diseases, adhesions, lymphoid hyperplasia) (5). However, most cases occurring during pregnancy are idiopathic, likely due to altered bowel motility and mechanical effects.

The clinical presentation of bowel intussusception in pregnancy is nonspecific and often overlaps with common pregnancy-related symptoms, leading to delays in diagnosis and increased complication risks. Literature reports indicate that intussusception accounts for approximately 6% of mechanical bowel obstructions in pregnancy (6). While ultrasonography is the first-line imaging modality, MRI provides higher diagnostic accuracy and can become the gold standard for diagnosis.

Although conservative treatment is commonly used for pediatric intussusception, it is generally unsuccessful in adults and particularly in pregnant patients. Consequently, surgical intervention is often necessary. In our case, the patient improved during follow-up, with resumed bowel movements and symptom resolution, making it one of the rare cases where surgery was not required. When surgical intervention is necessary, manual reduction of the intussuscepted segment should be attempted first; if unsuccessful, resection and anastomosis should be performed.

Conclusıon

Persistent abdominal pain, nausea, and vomiting suggestive of bowel obstruction in pregnant patients should always raise suspicion for intussusception. Early diagnosis and appropriate surgical management play a crucial role in preserving maternal and fetal health.

MRI is emerging as an increasingly used modality for diagnosing intussusception during pregnancy. Further case reports and large-scale studies are needed to enhance the understanding of this rare but clinically significant condition.

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A Patient with Loss of Eye Movements and Balance: A Rare Case Study of Miller Fisher Syndrome

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Introduction

Miller-Fisser syndrome (MFS) is an immune-mediated inflammatory demyelinating peripheral neuropathy. It is now widely considered to be a variant of Guillain-Barre syndrome (GBS). It is relatively rare in clinical practice and accounts for only 5% of GBS (1).

The typical clinical manifestations of MFS primarily include extraocular muscle paralysis, ataxia and reduced or absent tendon reflexes, collectively referred to as the MFS triad. The presence of anti-GQ1b antibodies is crucial for the diagnosis of MFS.

MFS and GBS are thought to result from an abnormal acute autoimmune response to a previous infection (e.g. Campylobacter jejuni, Cytomegalovirus, Epstein-Barr virus or human immunodeficiency virus). A cross-reaction between peripheral nerve antigens and microbial/viral components through molecular mimicry is thought to drive the inflammatory process of this disease. Approximately two thirds of cases are preceded by symptoms of upper respiratory tract infection or diarrhea and approximately 50% develop after infection (2).

MFS is mainly associated with dysfunction of the third, fourth and sixth cranial nerves. Other risk factors associated with the disease include the use of certain drugs (heroin, suramin, streptokinase and isotretinoin), use of TNF-alpha antagonist therapy, other concurrent autoimmune diseases (systemic lupus, Hodgkin's disease and sarcoidosis), surgery, epidural anesthesia, bone marrow transplantation and vaccinations (3).

The clinical hallmark of MFS is the triple presentation of acute ophthalmoplegia, areflexia and ataxia preceded by a bacterial or viral illness. Physical examination findings include typical findings for GBS including facial paralysis, distal hyporeflexia without signs of upper motor neuron dysfunction, and loss of light and vibration sensation in the distal extremities (4).

Case

A 14-year-old male patient presents to the emergency department with complaints of diplopia starting in the morning. There is no known history of previous illness, no recent history of upper respiratory tract infection or gastroenteritis. The patient had a snow ball in his eye two days ago. There was limitation of outward gaze in the left eye. He described inability to stand and ataxic gait.

There was no pathology in blood tests. Computed tomography of the brain and orbit revealed no pathology. The patient was consulted to ophthalmology. No pathology was found on orbital MRI. Central pathology was primarily considered in the patient. Pediatric neurology was consulted and the patient was hospitalized in the pediatric neurology service. On initial examination, the patient had ataxia of the 6th cranial nerve palsy. Other cranial nerve examinations were normal. Deep tendon reflexes were present in the upper and lower extremities. There was no dysdiadokinesis dysmetria on cerebellar examination.

In the ward follow-up of the patient, deep tendon reflexes could not be obtained in the upper and lower extremities and muscle strength was 4\5. Tremor was detected in the finger nose test. IVIG treatment was started considering Miller Fisher syndrome. In the sixth hour of treatment, the patient was referred to a higher center due to progression of the current condition.

Discussion

Molecular mimicry between the peripheral nerve and microbial/viral antigens is thought to occur through activation of the adaptive immune system. Humoral and cell-mediated lymphocyte mobilization is thought to play an important role. Gangliosides are important markers for autoimmune activity (5).

The clinical hallmark of MFS is a triad of acute ophthalmoplegia, areflexia and ataxia preceded by a bacterial or viral illness. Distal paresthesia with or without weakness is also present. Symptoms peak in an average of four weeks or less. Other symptoms include diplopia or blurred vision, dysarthria, dizziness and limb tingling. Cranial nerve involvement is typical and causes facial, oculomotor or bulbar weakness that may extend to the limbs. Physical examination findings include findings typical of GBS, such as facial paralysis, distal hyporeflexia without signs of upper motor neuron dysfunction, and loss of light and vibration sensation in the distal extremities. Autonomic dysfunction such as hypertension, hypotension or cardiac arrhythmia is seen in advanced untreated GBS/MFS. Interestingly, corneal reflex may be impaired (4)

If there is clinical suspicion for MFS and/or GBS, lumbar puncture with appropriate cerebrospinal fluid (CSF) studies is required to further narrow the differential diagnosis. A hallmark of GBS and MFS is albuminocytologic dissociation, if present, or a combination of normal cell count and elevated protein level in the CSF, which is found in approximately 90% of the disease. However, only half of

patients have albuminocytologic dissociation on initial analysis and a normal protein level does not exclude the diagnosis, especially early in the disease. Approximately 10% of patients with GBS have normal CSF studies. Approximately 15% to 20% have a slightly increased cerebrospinal fluid cell count (5 to 50 cells per microliter). In addition, nerve conduction studies may support the diagnosis and provide prognostic information (6).

The detection rate of anti-GQ1b antibody in MFS patients can reach 70-90%, which is considered an important evidence for the diagnosis of MFS. GQ1b is enriched in the extramedullary portion of human oculomotor, trochlear and abducens nerves. While the positive rate of this antibody is relatively high in patients with ophthalmoplegia, the rate of anti-GQ1b antibody is significantly reduced in patients without ophthalmoplegia (3).

Electrodiagnostic studies for MFS may show reduced or absent sensory responses without slowing of sensory conduction studies. Computed tomography and magnetic resonance imaging scans of the spine may show thickening and thickening of the intrathecal spinal nerve roots and cauda equina, with an increase in some spinal nerve roots. Abnormalities of the posterior columns of the spinal cord and brain oculomotor, abducens and facial nerves have been described in the literature (4).

MFS is mainly treated with adequate supportive care, pain control, respiratory support when needed and immunotherapy. Although used in the past, oral or intravenous (IV) steroids are no longer recommended for the treatment of GBS or MFS because they are ineffective. Corticosteroids can slow recovery from GBS, they are only recommended in case of neuropathic or radicular pain . IV immunoglobulin (IVIG) and plasma exchange are effective treatments for GBS and severe cases of MFS. There is no difference in primary mortality, disability and intubation time outcomes between IVIG and plasmapheresis. Patients with MFS usually do not need immunotherapy, possibly because they have a good prognosis and spontaneous recovery. IVIG should be considered in patients with severe Miller Fisher syndrome who have swallowing and breathing difficulties, despite a lack of supporting evidence. In general, IVIG is preferred over exchange because of convenience, availability and minimal side effects (7).

Prophylactic treatment of deep vein thrombosis (DVT) should be initiated immediately to reduce the risk of pulmonary embolism. Administration of prophylactic doses of subcutaneous heparin or enoxaparin is appropriate. Alternatively, mechanical compression stockings may be used in adult patients unable to walk. If autonomic dysfunction is present, additional supportive therapy may be required. If the patient is moderately to severely bradycardic and at risk of asystole, the patient may require a pacemaker. If dysphagia is present, a nasogastric tube may be required for feeding and nutrition. Bladder catheterization may relieve patients with urinary retention. A bowel regimen will be indicated to help with constipation. Early physical therapy during illness and early rehabilitation when the patient clinically improves are crucial (8).

Inpatient and intensive care unit (ICU) placement is an important consideration for a patient with acute GBS and variant MFS. This is based on the severity of symptoms and, most importantly, respiratory status. Mechanical ventilation is required for 20% to 30% of patients who develop respiratory failure; endotracheal intubation and even tracheostomy may be required. Symptoms of respiratory muscle fatigue include tachycardia, tachypnea, asynchronous chest/abdominal movement and marked use of accessory muscles. As noted, all patients with acute debilitating symptoms are hospitalized for supportive care. Intensive care unit admission and mechanical ventilation is recommended in patients with at least 1 major criterion or 2 minor criteria. Major criteria include hypercapnia partial carbon dioxide greater than 48 mm Hg, hypoxemia partial oxygen less than 56 mm Hg on room air, vital capacity less than 15 mL/kg body weight, and negative inspiratory force less than -30 cm H2O. Minor criteria include inadequate/weak cough, dysphagia and atelectasis as evidenced on chest x-ray (4).

The outcome of MFS is generally good with a case fatality rate of less than 5%. Average recovery times range from 8 to 12 weeks.Hyponatremia in GBS is a predictor of poor outcome with the development of syndrome of inappropriate antidiuretic hormone secretion (SIADH). Approximately 21% to 48% of GBS patients may suffer from hyponatremia.Hyponatremia is an independent predictive factor for mortality, questioned in more recent studies, making respiratory status and complications in the intensive care unit the most important predictors of mortality and morbidity (9).

The most common complication is generalized fatigue, reported in three quarters of patients with GBS and MFS. Recovery tends to be better in patients with MFS (3).

Conclusion

Miller Fisher syndrome is a rare and complex neurological disorder. Recognized by paralysis of the eye muscles, loss of coordination and loss of reflexes, it usually occurs when the immune system attacks the nerves. Early diagnosis and treatment can significantly speed up the recovery process. However, patients need to be carefully monitored and receive supportive care during the treatment process. Although it is a rare disease, with appropriate treatment and physiotherapy, most patients recover.

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Arterial blood gas analysis as a systematic diagnostic approach in the evaluation and treatment of critical illnesses in emergency departments

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Introduction and Purpose: The analysis of arterial blood gases (ABG) is a pivotal component of evaluating and treating critically ill patients in emergency departments. It allows rapid and accurate evaluation of patients' respiratory, circulatory, and acid-base balance disorders. We aim to improve the interpretation of ABG results in emergency departments and to facilitate clinical decision-making processes and more general understanding of acid-base disorders from these interpretations.

Materials and Methods: ABG testing involves the analysis of specific components; The normal blood pH range is between 7.35 and 7.45, which indicates whether the blood is acidic or alkaline. PaO□, which is expressed as a range of 75–100 mmHg, is a measurement of the partial pressure of oxygen in arterial blood. This measurement reflects the oxygen-carrying capacity of the blood. PaCO2, which measures the partial pressure of carbon dioxide in arterial blood, is an essential indicator of the blood's carbon dioxide content. It provides information about the ventilation status. The standard value of bicarbonate (HCO3) is 22-28 mEq/L. Bicarbonate is among the primary buffer systems that regulate the body's acid-base balance. A low HCO3- level is indicative of metabolic acidosis, while a high level is indicative of metabolic alkalosis.

Results and Conclusion: Healthcare professionals can monitor the acid-base balance of patients with AKG analysis, which should be performed following the steps below.

Step 1: The pH of blood gas is used to indicate the presence of clinical acidosis when it falls below 7.4, and alkalosis when it rises above 7.4.

Step 2: If the changes in pH and PaCO2 are in the same direction, that is, if both increase or decrease, it indicates that the event is metabolic.

3. If the direction of change of pH and PaCO2 are in different directions, the event is respiratory; if pH is below 7.4, there is respiratory acidosis, and if pH is above 7.4, there is respiratory alkalosis. Thus, primary acid-base disorders such as metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis are detected.

4. a)- If there is metabolic acidosis, it shows that HCO3- has decreased. Expected PaCO2= 1.5x [HCO3) +8±2 and is compared with the PaCO2 value in ABG. If they are equal, there is isolated metabolic acidosis; if they are increased, there is respiratory acidosis; if they are low, there is respiratory alkalosis.

b)- If there is metabolic alkalosis, it shows that HCO3- has increased; it is calculated with expected PaCO2= 0.9x [HCO3) +16 and compared with the value in ABG. If they are equal, there is isolated metabolic alkalosis; if they are increased, there is respiratory acidosis; if they are low, there is respiratory alkalosis.

5.a)- 5. If respiratory acidosis is present, PaCO2 is increased and the expected HCO3 value is calculated with the formula HCO3=24+(PaCO2- 40) / 10 and compared with the value in ECG. If it is equal, isolated respiratory acidosis, if it is increased, metabolic alkalosis and if it is decreased, metabolic acidosis accompanies.

In respiratory acid-base disorders, if pH [H+]/pCO2 ratio is below 0.3, it is chronic. If it is between 0.3 and 0.8, it is acute over chronic, and if it is above 0.8, it is acute.

6. The anion gap (AG) is usually calculated using the formula [Na +] – ([Cl –] + [HCO 3 –]), where potassium is excluded for simplicity, and the normal AG is 8 ± 2 mmol/L. If the base value is < -2, there is metabolic acidosis, and if > +2, there is metabolic alkalosis.

7. If the pH is 7.4, there is a mixed acid-base disorder. According to the [HCO3-] and PaCO2 values, if [HCO3-] is increased, there is metabolic alkalosis; if it is decreased, there is metabolic acidosis. If PaCO2 is increased, there is respiratory acidosis, if it is decreased, there is respiratory alkalosis.

In conclusion, correct interpretation of blood gas test provides valuable information about the patients' condition. It guides the treatment according to the clinical situation and can accelerate the recovery process of the patients.

Keywords: Acid/base, Arterial blood gas analysis (ABG), Partial pressure PaCO2 (PaCO2), Serum bicarbonate (HCO3-).

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Fahr Syndrome: A Case Diagnosed After Syncope

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Introduction

Fahr syndrome is a rare neurodegenerative disease characterized by symmetric calcifications in the basal ganglia, cerebellar nuclei, and periventricular white matter. It is often associated with hypoparathyroidism, pseudohypoparathyroidism, genetic mutations, or

metabolic disorders. The clinical presentation can vary widely, ranging from seizures, movement disorders, psychiatric symptoms, and cognitive decline to asymptomatic cases.

Patients presenting to the emergency department with syncope, head trauma, or altered consciousness are common. However, these symptoms can sometimes mask underlying metabolic or neurological causes. Here, we present a case of a patient diagnosed with Fahr syndrome following a syncope episode and head trauma.

Case Presentation

A 54-year-old female presented to the emergency department with complaints of syncope and head trauma one hour prior. The patient could not recall the incident, and her relatives reported finding her unresponsive on the ground. Her medical history included hypertension and thyroid disease, but no neurological disorders were noted in her family history.

On physical examination, she was conscious, cooperative, and oriented with a Glasgow Coma Scale (GCS) score of 15. An ecchymosis was observed in the left periorbital region. Pupils were equal and reactive to light, with no lateralizing neurological signs. Motor strength was assessed as 5/5 in all extremities.

Laboratory results showed a blood glucose level of 184 mg/dL and a blood pressure of 80/50 mmHg. Electrolyte levels and other biochemical parameters were within normal ranges. A non-contrast computed tomography (CT) scan of the brain revealed extensive bilateral calcifications in the basal ganglia, cerebellar nuclei, and periventricular white matter.

Given the absence of metabolic disorders such as hypocalcemia or hypoparathyroidism, the patient was diagnosed with idiopathic Fahr syndrome. Intravenous hydration and cardiac monitoring were initiated, and the patient was referred to neurology and neurosurgery outpatient clinics for further evaluation and follow-up.

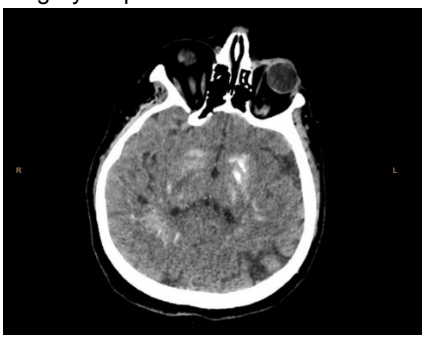

Figure: Widespread hyperdense nodular appearances primarily suggestive of calcifications in the bilateral basal ganglia, dentate nuclei, and bilateral cerebral white matter.

Discussion

Fahr syndrome, can be inherited or idiopathic. It is frequently associated with genetic mutations. Metabolic disorders like hypoparathyroidism, pseudohypoparathyroidism, and hypomagnesemia are common secondary causes. In cases without identifiable metabolic or genetic causes, a diagnosis of idiopathic Fahr syndrome is established.

Clinical manifestations are diverse, with seizures, extrapyramidal symptoms, psychiatric disorders, and cognitive decline being the most common. Extrapyramidal features, including parkinsonism, dystonia, and choreiform movements, may lead to motor dysfunction. Psychiatric symptoms such as depression, anxiety, and psychotic disorders are also prevalent, while cognitive decline resembling dementia significantly impacts quality of life.

In asymptomatic cases, the diagnosis is often incidental. In our case, the patient presented with syncope and head trauma, leading to further investigation and diagnosis through imaging studies.

Conclusion

Fahr syndrome should be considered in patients presenting with syncope and head trauma, particularly in cases of unexplained loss of consciousness. Radiological findings play a crucial role in diagnosis, and long-term neurological and psychiatric follow-up is recommended for these patients.

Further studies are needed to explore the underlying mechanisms and potential therapeutic approaches for Fahr syndrome.

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3650

A rare case: total gastrıc necrosıs—but why?

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Introductıon

Total gastric necrosis is an exceptionally rare clinical entity due to the extensive vascular supply to the stomach. It is frequently associated with high mortality and may result from vascular, mechanical, chemical, or infectious etiologies. In most cases, the underl-

ying cause can be identified preoperatively, or an intraoperative mechanical insult can be demonstrated. A high index of clinical suspicion and thorough physical examination are crucial for early diagnosis. The most common presenting symptoms include nausea, vomiting, and severe abdominal pain.

Case

A 22-year-old female patient presented to the emergency department with complaints of lower abdominal pain, bloating, and difficulty in passing flatus, which had begun one day prior. She reported experiencing similar symptoms during every menstrual cycle and stated that she had started menstruating the previous day. The patient requested to be discharged after receiving analgesic treatment. On physical examination, there was significant tenderness in the lower quadrants without signs of peritoneal irritation (negative rebound and guarding). Her vital signs were within normal limits. Laboratory tests were obtained, and she was monitored in the emergency department. Initial venous blood gas analysis revealed metabolic acidosis with a pH of 7.33, bicarbonate (HCO3) of 18.2 mmol/L, and a markedly elevated lactate level of 10.09 mmol/L. Intravenous fluid resuscitation was initiated. A follow-up examination performed 45 minutes later revealed worsening abdominal pain, raising suspicion of an acute abdomen. Additional laboratory results showed leukocytosis (WBC: 33,400/mm³), neutrophilia (NEU%: 93.6), elevated amylase (343 U/L), lipase (1007 U/L), and CRP (0.57 mg/dL). A contrast-enhanced abdominal computed tomography (CT) scan was performed. CT imaging demonstrated extensive pneumoperitoneum and free intra-abdominal fluid, suggestive of upper gastrointestinal perforation. Additionally, there was an absence of gastric wall enhancement with diffuse intramural gas (suggestive of total gastric necrosis). A filling defect of approximately 5 mm was noted at the origin of the celiac trunk, along with non-enhancing areas in the spleen, consistent with splenic infarction. Given these findings, median arcuate ligament syndrome (MALS) and celiac artery compression syndrome were considered as possible underlying etiologies. The general surgery team was consulted, and the patient underwent emergency total gastrectomy and splenectomy. Postoperatively, she was admitted to the anesthesia intensive care unit for further management.

Dıscussıon

Median arcuate ligament syndrome (MALS) is a rare vascular compression disorder caused by excessive pressure from the median arcuate ligament on the celiac artery and the surrounding celiac plexus. This condition is predominantly observed in young, thin females and presents with postprandial epigastric pain and weight loss. MALS remains a diagnostic challenge and often requires extensive workup. The definitive treatment is surgical release of the median arcuate ligament, with reported success rates of approximately 75%.

Keywords: Total Gastric Necrosis, Median Arcuate Ligament Syndrome, Abdominal Pain, Vascular Compression Syndrome

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4034

Acute Carbon Monoxide Poisoning Following Waterpipe Use: A Case Report

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Introduction

Carbon monoxide (CO) poisoning is a potentially life-threatening clinical condition that arises from exposure to carbon monoxide, a colorless, odorless, and non-irritating gas. CO binds to hemoglobin with an affinity approximately 250 times greater than that of oxygen, forming carboxyhemoglobin (COHb), and thereby significantly impairs tissue oxygenation. The clinical presentation can vary widely, ranging from mild symptoms such as headache and fatigue to severe neurological and cardiovascular complications including seizures, coma, and death.

In recent years, waterpipe smoking has gained popularity among young adults. When performed in poorly ventilated environments, it can result in significant CO exposure. Compared to cigarette smoking, waterpipe use has been shown to produce much higher levels of COHb. This report presents a case of a young adult who developed acute CO poisoning after prolonged waterpipe use and required hyperbaric oxygen therapy.

Case

A 25-year-old male presented to the emergency department after experiencing dizziness, chest pain, and a syncopal episode following prolonged exposure in an enclosed waterpipe café. On initial evaluation, the patient was alert, cooperative, and oriented with a Glasgow Coma Scale (GCS) score of 15. His vital signs were as follows: systolic blood pressure 120 mmHg, heart rate 86 bpm, and oxygen saturation 95% on room air. Physical examination findings were non-specific.

The patient's only known medical history was anxiety disorder. Electrocardiogram (ECG) showed normal sinus rhythm, and cardiac biomarkers (troponin, CK-MB) were within normal limits. Arterial blood gas analysis revealed a COHb level of 30%. High-flow oxygen therapy was initiated via a non-rebreather mask. During clinical observation, the patient's symptoms improved significantly. However,

due to the high COHb level and the history of syncope, the patient was referred to as hyperbaric oxygen therapy via emergency medical services.

Discussion

In Turkey, most CO poisonings are caused by faulty heating systems or natural gas leaks. However, the rising popularity of waterpipe smoking in confined spaces has emerged as a significant source of CO exposure. Waterpipe smoke contains CO not only from the tobacco itself but also from the charcoal used to heat the tobacco, in addition to heavy metals and other toxic compounds. Studies have demonstrated that even a single session of waterpipe smoking can result in significantly higher COHb levels compared to cigarette smoking.

Symptoms of CO poisoning are often non-specific, including headache, dizziness, weakness, and nausea. Severe cases may present with syncope, seizures, coma, or cardiac arrhythmias. Diagnosis is confirmed by clinical history and COHb levels obtained from arterial blood gas analysis. However, it is important to note that COHb levels do not always correlate with the severity of clinical presentation.

The first step in treatment is removal from the exposure source and administration of 100% oxygen. Hyperbaric oxygen therapy (HBOT) is recommended for preventing neurological sequelae, facilitating faster CO elimination, and improving tissue oxygenation. HBOT is especially indicated in cases with syncope, altered mental status, cardiac involvement, or COHb levels exceeding 25%.

Conclusion

This case highlights the clinical relevance of acute CO poisoning following waterpipe use. In young adults presenting with nonspecific symptoms after waterpipe exposure in enclosed spaces, CO poisoning should be considered in the differential diagnosis. Early recognition, prompt administration of oxygen therapy, and referral for hyperbaric oxygen therapy when indicated can significantly reduce the risk of complications. Public awareness regarding the health risks associated with waterpipe use is crucial to prevent similar cases in the future.

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4083

Optic nerve sheath diameter in the treatment of hyponatremia

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Introductıon

Hyponatremia is one of the most common electrolyte disorders (1). According to the European clinical practice guidelines, hyponatremia is defined as a serum sodium (Na^+) concentration below 135 mmol/L. The prevalence of hyponatremia among patients presenting to the emergency department (ED) ranges from 3% to 10%, depending on the environmental and demographic characteristics of the local population (2).

Symptoms associated with hyponatremia can vary from mild, nonspecific complaints such as dizziness, headache, or nausea to severe, life-threatening conditions like cerebral edema (3). Due to the osmotic gradient between the brain and plasma, water shifts from the extracellular to the intracellular compartment, leading to cellular swelling in the brain. This typically occurs when hyponatremia develops rapidly, leaving little time for cerebral adaptation to the hypotonic environment (2). Ultimately, cerebral edema can lead to increased intracranial pressure (ICP).

Various invasive and non-invasive methods for measuring ICP have been employed for years, particularly in the fields of neurology and neurosurgery. While invasive techniques usually involve the placement of a catheter into the cranial cavity, they are associated with potential complications such as infection and bleeding. Non-invasive alternatives include computed tomography (CT), magnetic resonance imaging (MRI), transcranial Doppler ultrasound, and optic nerve sheath diameter (ONSD) measurement (4). Among these, transorbital ultrasonography, a non-invasive method, allows estimation of ICP by measuring the ONSD. The optic nerve sheath (ONS) is surrounded by cerebrospinal fluid (CSF) and the dura mater, and is connected to the intracranial subarachnoid space, making it sensitive to changes in CSF pressure. Bedside ultrasonographic measurement of ONSD is a reliable, non-invasive method for detecting elevated ICP. Studies have shown that ONSD measurements correlate well with direct ICP measurements and exhibit low inter-observer variability (6).

Monitoring serum sodium levels during the correction of hyponatremia is crucial to prevent complications such as central pontine myelinolysis or osmotic demyelination syndrome (7,8). Since laboratory testing for electrolytes takes longer, blood gas analyzers are commonly used in EDs to monitor the correction of hyponatremia. However, repeated needle punctures for sample collection and the high cost of blood gas analysis represent significant drawbacks.

The aim of this study is to investigate the relationship between changes in ONSD and increases in serum Na^+ levels, as well as

associated clinical improvement, in patients presenting to the emergency department with symptomatic hyponatremia and treated with hypertonic saline (3% sodium chloride).

Changes in ONSD have been observed in patients presenting with hyponatremia (9). To the best of our knowledge, there are no detailed studies exploring ONSD changes during the correction of hyponatremia. With this study, we aim to examine whether there is a relationship between serum Na^+ levels and ONSD, to assess how hyponatremia treatment affects ONSD, and to determine whether ONSD could potentially serve as a monitoring tool during the treatment of hyponatremia.

Methods

This study included 40 patients who presented to the emergency department of Ufuk University Dr. Rıdvan Ege Hospital between September 25, 2023, and April 1, 2024, and were found to have hyponatremia (Na <135 mmol/L) upon arrival. Patients under the age of 18, those with a history of cerebrovascular accident , a history of ophthalmologic or cranial surgery, a diagnosis of glaucoma, altered mental status, or those unable to provide informed consent were excluded. Patients meeting the inclusion criteria and who demonstrated an increase in serum sodium levels after hypertonic saline therapy were enrolled in the study.Written informed consent was obtained from all participants prior to inclusion. Institutional Review Board approval was granted for this prospective observational study (Approval No: 12024861-29, dated 20/09/2023). The study was conducted in accordance with the principles outlined in the World Medical Association Declaration of Helsinki**.**

Hyponatremia Treatment

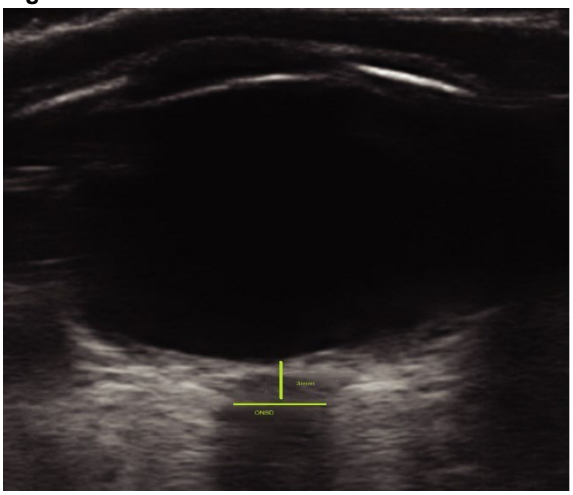
Treatment was administered based on the European Clinical Practice Guidelines (2). According to these guidelines, patients with acute hyponatremia (onset <48 hours) presenting with moderate or severe symptoms were initially treated with 150 mL of hypertonic saline (3% NaCl) over a 20-minute period, followed by reassessment of serum sodium levels. The treatment goal in symptomatic patients was to achieve a prompt increase of 5 mmol/L in serum sodium levels, while ensuring that the total correction did not exceed 8–10 mmol/L within 24 hours (2).

Venous blood gas samples were obtained at presentation and again following hypertonic saline treatment. Sodium levels were recorded. Demographic data, vital signs, initial blood gas parameters, mental status, post-treatment blood gas results, and pre- and post-treatment ONSD measurements were all documented. All blood gas analyses were performed using a calibrated ABL800 FLEX © blood gas analyzer (Radiometer Medical ApS, Copenhagen, Denmark).

Measurement of Optic Nerve Sheath Diameter (ONSD)

All patients were examined in the supine position, and measurements were performed on the right eye. While the patients' right eyes were gently taped shut and covered with a transparent film, they were asked to focus their left eyes on the ceiling. Transorbital ultrasonographic assessments were performed in the transverse plane using a 7–13 MHz linear probe ultrasound system (Mindray, Model DC-T6). Anatomically, the ONSD was measured 3 mm posterior to the papillary segment of the optic nerve. All measurements were conducted by an emergency medicine specialist experienced in ocular ultrasonography.

Figure 1.ONSD Measurement



Statistical Analysis

Statistical analysis was performed using SPSS version 29.0 (SPSS Inc., Chicago, IL, USA). Data were presented as mean ± standard deviation (SD), median, or as number of patients (%), where appropriate. The Kolmogorov-Smirnov test was used to assess the normality of distribution for continuous variables. Depending on the distribution, continuous variables were analyzed using either the independent t-test or the Mann-Whitney U test. Categorical variables were analyzed using the chi-square (χ^2) test or Fisher's exact test, as appropriate. Pearson's correlation test was employed for correlation analysis. Changes in variables over time were assessed using repeated-measures analysis of variance (ANOVA). A p-value of <0.05 was considered statistically significant.

Results

Among the 40 patients included in the study, 24 were female and 16 were male. The mean age of the participants was 67.2 years (range: 52–90 years). The mean serum sodium (Na^+) level at presentation was 115.7 mEq/L, while the post-treatment mean Na^+ level was 135.9 mEq/L. The mean optic nerve sheath diameter (ONSD) at admission was 5.46 mm, which decreased to a mean of

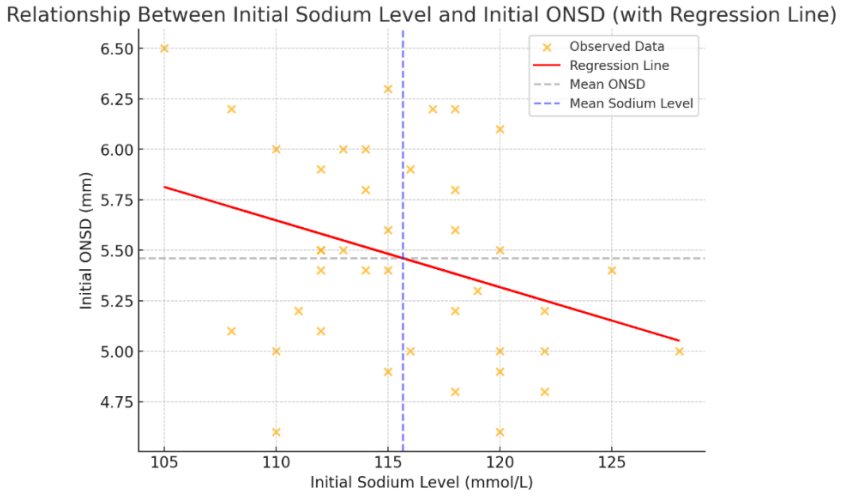
5.12 mm following treatment(Table 1)

Table 1. Descriptive Statistics for Numeric Variables

Variable	Mean	Min	Max
Age	67.2	52	90
Admission Na (mmol/L)	115.7	105	128
Admission ONSD (mm)	5.46	4.6	6.5
Post-treatment Na (mmol/L)	135.9	133	140
Post-treatment ONSD(mm)	5.12	4.2	6

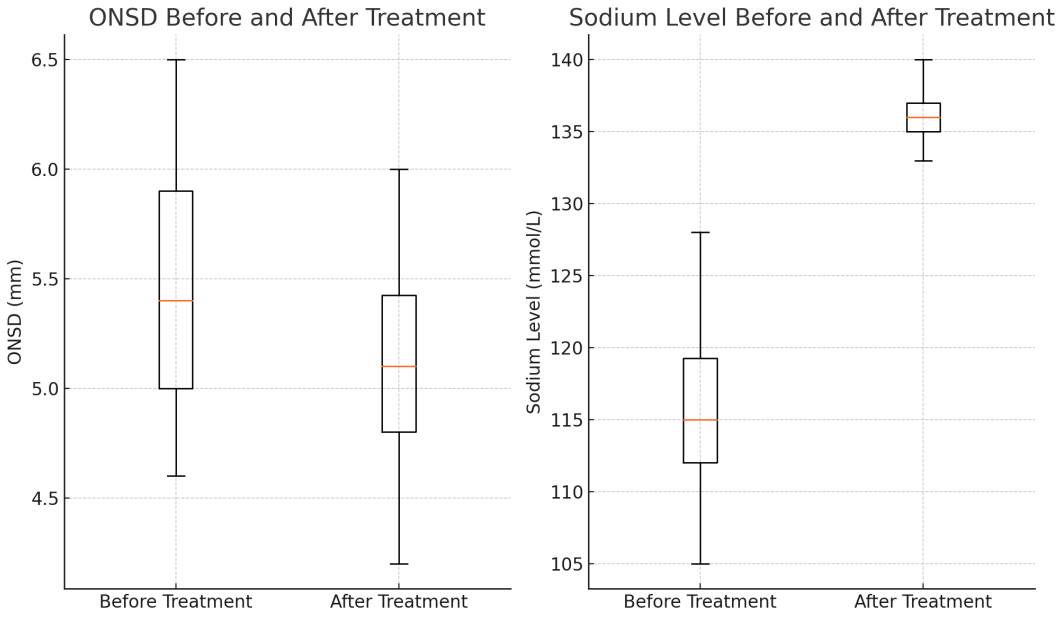
The correlation analysis between serum sodium levels prior to treatment (hyponatremia) and pre-treatment optic nerve sheath diameter (ONSD) revealed a correlation coefficient of -0.32, indicating a low to moderate negative relationship. The p-value was 0.043, which is statistically significant ($p < 0.05$). These findings suggest that as serum sodium levels decrease (i.e., the severity of hyponatremia increases), the ONSD tends to increase. In other words, more severe hyponatremia is associated with greater ONSD dilation (Figure 2).

Figure 2. Scatter plot showing negative correlation between admission serum sodium and pre-treatment ONSD



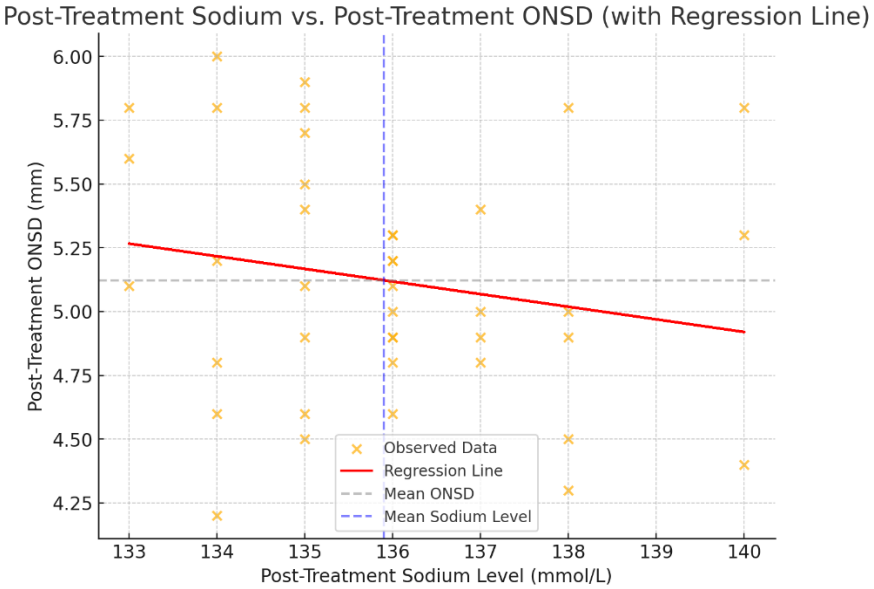
A decrease in ONSD was observed in 38 out of 40 patients following treatment, indicating that the intervention had a measurable effect on ONSD in the vast majority of cases. On average, The mean post-treatment ONSD decreased by 0.34 mm. This finding demonstrates that the correction of hyponatremia led to a statistically significant reduction in ONSD (Figure 3).

Figure 3.Pre/Post-retmatment Sodium and ONSD



Analysis of the relationship between post-treatment sodium levels and post-treatment ONSD revealed a weak negative correlation. This suggests that the change in sodium levels after treatment had no marked effect on ONSD. Moreover, the correlation was not statistically significant(p>0.5)(Figure 4).

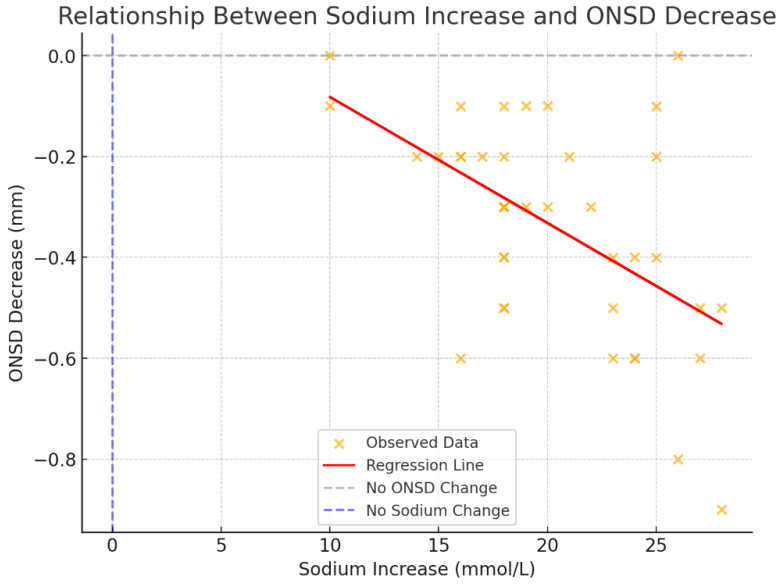
Figure 4.Post-treatment Sodium and Post-treatment ONSD



Correlation Between the Increase in Sodium Levels and the Reduction in ONSD

The correlation analysis demonstrated a correlation coefficient of -0.54, indicating a moderate negative relationship, with a p-value of 0.0003, which is statistically significant ($p < 0.05$). These findings suggest that the increase in serum sodium levels following the treatment of hyponatremia is significantly associated with a reduction in ONSD. In other words, as sodium levels rise with treatment, ONSD tends to decrease in a consistent and clinically meaningful manner (Figure 5).

Figure 5.Relationship Between Sodium Increase and ONSD Decrease



As part of the ROC analysis, we employed the Youden Index method to identify the optimal cut-off value that provides the best balance between sensitivity and specificity. The optimal threshold was determined to be an increase of 14 mmol/L in serum sodium.

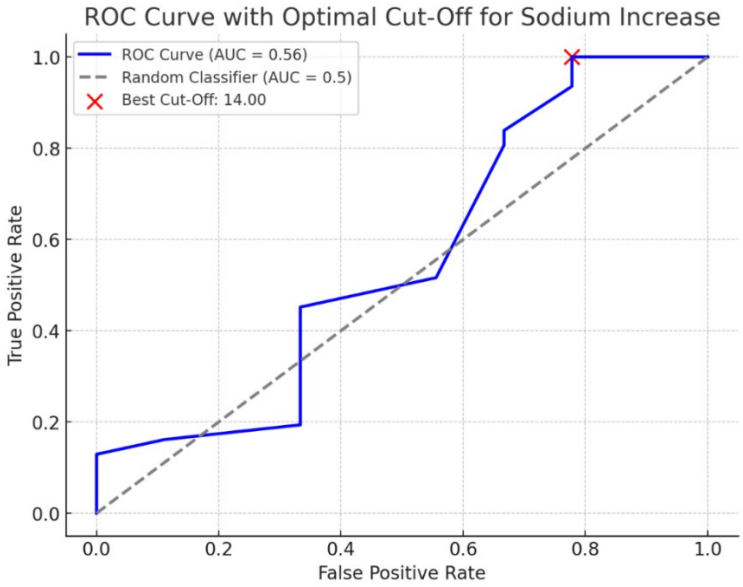
Sensitivity: 1.0 (100%) → This threshold correctly identified all cases with a decrease in ONSD.

Specificity: 0.22 (22%) → The low specificity indicates a high rate of false positives.

The area under the curve (AUC) was calculated as 0.56, suggesting that the increase in sodium levels is a weak predictor of ONSD reduction.

These findings imply that while sodium elevation may influence ONSD reduction to some extent, it is not a strong or definitive predictor. The analysis highlights a measurable, yet limited, predictive value of sodium increase on ONSD decrease (Figure 6).

Figure 6.ROC Analysis



Discussion

In this study, we evaluated the relationship between changes in optic nerve sheath diameter (ONSD) and increases in serum sodium (Na^+) levels before and after hypertonic saline treatment in patients presenting to the emergency department with hyponatremia. Our findings indicate that as the severity of hyponatremia increases, there is a tendency for ONSD to expand, and that ONSD significantly decreases following the correction of hyponatremia.

Hyponatremia is a serious electrolyte imbalance that can lead to cerebral edema. In cases of rapidly developing hyponatremia, brain cells fail to adapt to the hypotonic environment, resulting in intracellular fluid accumulation and increased intracranial pressure (ICP) (10). Previous studies have demonstrated a relationship between elevated ICP and ONSD expansion, suggesting that bedside ultrasonographic measurement of ONSD is a promising non-invasive tool for clinical monitoring (11,12). Our study further supports these findings by showing that correction of hyponatremia leads to a reduction in ONSD, which moderately correlates with the increase in serum sodium levels.

The existing literature supports the reliability of ONSD measurements in monitoring intracranial pressure changes. Prior studies have proposed the use of ONSD as a non-invasive alternative to invasive methods in conditions such as traumatic brain injury, intracranial hemorrhage, and hydrocephalus (6,13). However, studies specifically investigating changes in ONSD during the correction of hyponatremia remain limited. Our study addresses this gap and contributes valuable data to the field.

According to our findings, there is a moderate negative correlation between the increase in serum sodium levels and the decrease in ONSD following treatment ($r = -0.54$, $p < 0.05$). This suggests that treatment of hyponatremia may reduce intracranial pressure, which can be monitored through ONSD measurements. However, our ROC analysis indicated that sodium increase is not a strong predictor of ONSD change (AUC = 0.56), though it may have a limited but meaningful influence. Therefore, while While ONSD measurements are clinically valuable, they should not be solely relied upon for the management and monitoring of hyponatremia and should be supported by additional clinical assessment methods.

Conclusion

Our study demonstrated a significant reduction in ONSD following the treatment of hyponatremia. However, it also revealed that ONSD measurements alone may not be sufficient for the treatment and follow-up of hyponatremia and should be complemented by other clinical evaluation methods.

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Paraneoplastic Polyneuropathy: A Case Report,

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Introduction

Paraneoplastic neurological syndromes (PNS) are rare but clinically significant conditions associated with malignancies. These syndromes represent a group of disorders that occur in the absence of direct tumor invasion, metastasis, or treatment-related complications, and are most often considered to be immune-mediated. Among these, paraneoplastic polyneuropathy is frequently underrecognized, yet it can profoundly affect a patient's quality of life and prognosis.

Lung cancer, particularly small cell lung cancer (SCLC), is the malignancy most commonly associated with paraneoplastic syndromes, increasing the incidence of paraneoplastic polyneuropathy in these patients. Although the exact pathophysiology remains unclear, the presence of antineuronal antibodies—particularly anti-Hu (ANNA-1)—suggests an autoimmune mechanism. These antibodies target neural tissues, leading to widespread dysfunction of the motor, sensory, and autonomic nervous systems

Clinically, paraneoplastic polyneuropathy often presents as a progressive, symmetric, and distally predominant sensory neuropathy. However, motor symptoms and autonomic dysfunction may also be prominent in some cases. Importantly, the neurological symptoms may precede the diagnosis of the underlying malignancy, providing a critical clue for early cancer detection. Nonetheless, diagnosis remains challenging and requires a comprehensive evaluation, including clinical, electrophysiological, and laboratory investigations.

Due to the rarity of paraneoplastic syndromes and their association with polyneuropathy, the number of reported cases in the literature remains limited. This highlights the need for increased awareness to ensure accurate diagnosis and appropriate management.

This case report aims to detail the clinical presentation, diagnostic workup, and management approach of a patient who developed paraneoplastic polyneuropathy secondary to lung cancer.

Case Presentation,A 74-year-old male patient with no known chronic illnesses other than a previously diagnosed pulmonary malignancy presented to the emergency department with complaints of bilateral weakness in the legs and hands persisting for ten days. Medical history revealed that the patient had been treated with intravenous immunoglobulin (IVIG) for paraneoplastic polyneuropathy in February 2024.

On initial evaluation, the patient was conscious, oriented, and cooperative, with stable vital signs. Neurological examination revealed decreased muscle strength of 4/5 in the proximal upper extremities bilaterally, and 4/5 in hand and elbow flexion. In the lower extremities, proximal strength was 3/5, and dorsal foot extension was 1/5 bilaterally. The patient also exhibited paresthesia in the hands, and deep tendon reflexes were globally absent. Laboratory and central imaging studies showed no acute pathological findings.

Given the clinical presentation, the patient was preliminarily diagnosed with paraneoplastic polyneuropathy and was referred to the departments of pulmonology and neurology. He was admitted to the neurology service for further evaluation and treatment.

Discussion

Paraneoplastic polyneuropathy is a rare but clinically important complication of malignancy, with implications for both early diagnosis and later disease progression. This case describes a patient with a known history of lung cancer who presented with a recurrence of neurological symptoms after previously being treated for paraneoplastic polyneuropathy. Neurological evaluation confirmed the persistence and progression of paraneoplastic neuropathy symptoms.

The patient’s neurological findings indicated widespread sensory and motor dysfunction consistent with polyneuropathy. Significant proximal and distal muscle weakness, particularly in the lower extremities, as well as paresthesia and global absence of reflexes, suggested a paraneoplastic process. The absence of acute abnormalities in laboratory and neuroimaging studies supported a neurologic etiology. It is presumed that the paraneoplastic mechanism had been reactivated due to immune dysregulation associated with the underlying malignancy.

The patient's admission to the neurology ward emphasized the importance of a multidisciplinary approach in managing paraneoplas-

tic polyneuropathy. Optimal treatment typically involves control of the primary tumor, immunomodulatory therapies, and symptomatic management. Given that IVIG had previously led to partial symptom stabilization, reconsideration of immunomodulatory therapy was deemed appropriate in this case.

The prognosis of paraneoplastic polyneuropathy is closely linked to the control of the underlying malignancy. Therefore, early recognition of recurrent neurological symptoms, along with appropriate immune-directed therapy and cancer management, requires careful clinical evaluation. This case underscores the chronic and potentially relapsing nature of paraneoplastic polyneuropathy, and highlights the need for long-term neurological monitoring and interdisciplinary care in patients with malignancy.

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4744

Indispensable in emergency cases of dichloromus (dichloromuscoriol) and acute coronary syndrome: kounis syndrome

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Introductıon

Kounis syndrome (KS) is defined as the simultaneous occurrence of acute coronary syndrome including coronary spasm, acute myocardial infarction and stent thrombosis resulting from the activation of mast cells and platelets with allergy, hypersensitivity, anaphylaxis or anaphylactoid conditions and inflammatory cells such as macrophages and T lymphocytes that provide intercellular communication(8). The basic pathophysiology of Kounis syndrome is that cardiac mast cells are located in the intima layer of coronary arteries and atherosclerotic plaques. During an allergic reaction, these cells are activated like mast cells in the skin and lungs and release mast cell mediators. Histamine causes coronary vasoconstriction, increases tissue factor synthesis, and activates platelets. It also facilitates arrhythmia development by affecting the cardiac conduction system (9). Proteases cause plaque erosion and rupture by activating matrix metalloproteinases. In addition, it increases angiotensin-II levels and causes vasoconstriction. Thromboxane and platelet-activating factor both cause vasoconstriction and activate platelets (9,10,11).

Kounis syndrome has three types according to its pathophysiology and the presence of coronary artery disease: In Type I, coronary vasospasm induced by allergic mediators such as histamine, thromboxane, and leukotrienes is present in the absence of atherosclerotic risk factors and coronary artery disease. In type II, patients with atherosclerotic coronary artery disease have ACS as a result of coronary vasospasm, plaque erosion, or rupture triggered by these mediators. In type III, the presence of eosinophils and mast cells in the thrombus material removed in some patients who developed stent thrombosis after drug-eluting stent implantation suggested a hypersensitivity reaction in these patients (10). In Kounis syndrome, allergic symptoms and acute coronary syndrome should be treated together.

Case

A 61-year-old female patient with known diabetes mellitus, essential hypertension, dementia and lumbar disc herniation (LDH) was brought to us by 112 teams after developing chest pain, shortness of breath, nausea and vomiting complaints after having dichloronmuscoril im injection at the Family Health Centre where she went with the complaint of low back pain: 59 beats/min, blood pressure: 100/60 mmHg, oxygen saturation was 87% in room air, body temperature was 36.8 °C, there was no rash, itching complaint, periorbital oedema, uvula oedema and shock findings on physical examination. The ECG of the patient showed ST-segment elevation in

inferior leads (D2, D3, aVFd) and ST collapse in D1, aVL. The patient was treated with 80 mg prednisolone for 1000cc SF, 1 amp decort, 1 amp avil, 1 nebule pulmicort combivent with nebuliser, 1 nebule pulmicort combivent for hypotension, shortness of breath, and low SO2 due to drug allergy in the emergency department. The patient underwent ECHO in the emergency department, and a contraction defect was seen in the medial wall of the inferior wall, including the inferior septum. Coronary angiography was performed urgently, and severe vasospasm was seen in the RCA (right main coronary artery). The patient was discharged after 2 days of follow-up and treatment in the coronary intensive care unit.

Dıscussıon

Diclofenac sodium and thiocolchicoside injection is a common symptomatic treatment preferred for the symptoms of different diseases, such as LDH (lumbar disc herniation) pain, post-traumatic pain, and muscle pain due to upper respiratory tract infections. The mechanism of anaphylaxis plays an important role in understanding Kounis syndrome. Acute coronary syndrome is a rare presentation of anaphylaxis. This phenomenon is called Kounis syndrome or allergic acute coronary syndrome (12). As in anaphylaxis, in Kounis syndrome, an allergic attack causes the release of inflammatory mediators, especially histamine (2,13,15).

In addition to causing peripheral vasodilatation, histamine may cause vasospasm and narrowing of coronary vessels by acting on coronary histamine receptors (4,13,14). This vasospasm leads to the clinical manifestations of Kounis syndrome, which include various ECG changes (including but not limited to ST segment elevations, ST segment depressions, heart block, and other cardiac arrhythmias) (13,14). ST-segment elevations are considered the most common ECG change associated with Kounis syndrome (15). The clinical manifestations of Kounis syndrome are broad and include classic anginal symptoms such as chest pain, dyspnoea, palpitations, and sudden cardiac death (13,14,15); 25% of patients will have a medical history of allergic reaction, and 53% of patients will experience anaphylaxis (15). Kounis syndrome can be difficult to diagnose. One way to help screen for this condition is to perform an ECG and measure troponin levels in patients presenting with anaphylaxis (16). In addition to clinical features, ECG findings, and troponin values, there are other diagnostic tests to consider. It is important to keep Kounis syndrome in the differential diagnosis in patients with symptoms consistent with an acute coronary syndrome or an allergic reaction, as this has implications for management. Important general considerations include avoiding morphine in the management of anginal symptoms, as it may worsen mast cell degranulation and thus worsen vasospasm (8,14). Fentanyl is a better option for pain control when necessary (14). Epinephrine is the cornerstone of anaphylactic shock treatment (6). However, it may worsen vasospasm in the case of Kounis syndrome (22).

If epinephrine is used, providers should choose sulfite-free intramuscular epinephrine (14). When haemodynamic stability is present, antihistamines, steroids, and elimination of allergic damage are indicated in the management of Kounis syndrome (6,14).

The emergency care provider should also provide standard care treatments for acute coronary syndrome. Despite the unknown effects of aspirin on allergic reaction pathways, it should be administered to patients with suspected Kounis syndrome, given its clear benefits in the treatment of acute coronary syndrome (6).

Vasospasm is reduced by the use of vasodilator drugs such as nitroglycerin or calcium channel blockers, which can be given if the patient is haemodynamically stable (14). β-blockers are frequently used in the treatment of acute coronary syndrome; however, in the case of Kounis syndrome, they may worsen vasospasm through an unopposed α-effect and should be avoided (8,14). The use of β-blockers leads to less effective treatment of allergic symptoms (6,14). In these patients, reversal of the β-blocker with glucagon by bypassing the β-adrenergic pathway may be useful, and stimulation of myocardial contractility and heart rate via glucagon is recommended (6).

Conclusıon

Diclofenac sodium and thiocolchicoside injection is a common symptomatic treatment preferred for the symptomatic treatment of many diseases. Emergency physicians need to consider Kounis syndrome in the differential diagnosis of patients with chest pain and shortness of breath who present to emergency departments with allergic reactions due to many drugs used. In these patients, ECG changes, troponin elevations, or anginal symptoms together with allergic, anaphylactic symptoms indicate possible Kounis syndrome. Awareness of Kounis syndrome by physicians in emergency departments will ensure that the management of both acute coronary syndrome and allergic reactions/anaphylaxis will be healthier and more effective, leading to significant reductions in patient mortality and morbidity.

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4932

Pneumomediastinum due to tooth extraction: A case report

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Introduction

Mediastinal emphysema is defined as the presence of free air between mediastinal structures. It is rare and self-limiting. Mediastinal emphysema can be iatrogenic, traumatic, infectious, or spontaneous. Iatrogenic occurs after head and neck surgery, intubation, mechanical ventilation, esophageal perforation, and dental surgery. It can also occur as a result of facial fractures, intraoral trauma, and head and neck infections. The reason for its development after tooth extraction is the use of tools that provide compressed air in dental applications. The use of high-speed air turbine drills during dental procedures is thought to cause subcutaneous and mediastinal emphysema. It is a rare complication following routine tooth extraction.

Case

A 49-year-old male patient was admitted to the emergency department with complaints of right shoulder swelling and chest pain. On physical examination, general condition was good, consciousness was clear, oriented, coherent, blood pressure 140/80 mmHg, pulse 94/min, respiratory rate 22/min, temperature 36.8°C and oxygen saturation 94%. Palpation revealed redness, swelling, increased temperature, and crepitations under the skin around the right shoulder. Auscultation of the lungs revealed bilateral expiratory rhonchi and basal inspiratory rales. Complete blood count showed WBC: 14.58/mm3, CRP: 232 mg/dL, biochemical parameters and blood gas were within normal limits. The patient had a history of bilateral tooth extraction 1.5 months ago. CT of the neck showed diffuse air densities in the soft tissue in the right cervical region (Figure 1).

Contrast-enhanced chest CT showed free air and fluid in the mediastinum consistent with emphysema. A leukocytic cystic abscess approximately 10 cm in diameter (Figure 2) and diffuse mediastinitis were observed in the right paratracheal area (Figure 3).

After consultation with thoracic surgery, the patient was admitted to the thoracic intensive care unit. The patient received symptomatic treatment with support and monitoring for close follow-up. Bed rest, coughing, and avoidance of exertion were recommended. Pain control with analgesics and antibiotherapy was initiated. The patient underwent surgery under general anesthesia. The lung was found to be adherent from the apex, involving the anterior and posterior mediastinum. An abscess focus was seen under the pleura extending from the superior inferior vena cava and inferior azygos vena cava to the inferior pulmonary ligament. The pus in the abscess formation was aspirated and the postmediastinum was irregularized with SF. There were no complications in the early postoperative period. The patient was observed in the thoracic intensive care unit for 3 days. He was transferred to the ward and his general condition improved significantly after the 4th day. The swelling in the right neck decreased and the crepitations on palpation decreased significantly, and the patient was discharged.

Discussion

Pneumomediastinum after trauma was first described by Laennec in 1819 and mediastinal emphysema by Turnbull in 1900. Mediastinal emphysema can be spontaneous, iatrogenic, traumatic, or infectious. Iatrogenically, it can develop after intubation, mechanical ventilation, esophageal perforation, head and neck surgery, and dental surgery. It may also occur as a result of head and neck infections, facial fractures, and intraoral trauma. Rarely, it can be seen as a result of the use of compressed air equipment during tooth extraction (1). It has also been reported during periodontal and endodontic treatments, tooth extractions, and after temporomandibular joint and facial fracture surgery. The roots of the molars (1-2-3) are directly connected to the sublingual and submandibular areas. The sublingual area is connected to the pterygomandibular, parapharyngeal and retropharyngeal areas. From here, air travels to the mediastinum and causes mediastinal emphysema (2). The use of compressed air during tooth extractions introduces a non-sterile mixture of water and oil into the soft tissues. Subcutaneous emphysema occurs when air is forced under the dermis. If a large amount of air passes through, emphysema and even pneumothorax can occur in deep tissues such as the mediastinum and pericar-

dium.

During the procedure, intraoral and intrathoracic pressures increase with the rapid passage of air into the subcutaneous tissues. Frequent vomiting, coughing, crying, and excessive valsalva during dental procedures are risk factors for subcutaneous emphysema. Subcutaneous emphysema and pneumomediastinum may be observed after severe sneezing (3). Free air in the retropharyngeal region may cause hearing loss, dysphonia, dysphagia, chest pain, back pain, and dyspnea. Retrosternal pain and dyspnea are characteristic findings of pneumomediastinum (4).

In mild forms of subcutaneous emphysema, a small amount of air leaks into the local tissue. In subcutaneous emphysema, there is mild swelling, mild discomfort, and crepitation on palpation under the skin of the face and neck. Subcutaneous emphysema often has a good prognosis. Respiration may be suppressed or dysphagia may occur as a result of excessive air volume. In the presence of orbital emphysema, vision loss may occur (5). Pneumomediastinum usually has a good prognosis and is usually self-limiting, but complications such as tracheal compression, pneumothorax, air embolism, pneumopericardium, and mediastinitis may rarely occur. Pneumopericardium may also occur when pressurized air passes through the deep facial area into the pericardium. Examination may reveal deep audible heart sounds and mediastinal crepitation (Hamman's sign) heard during a heartbeat. In severe cases of pneumomediastinum, venous distention, hypotension, hypercarbia, and acidosis may be observed. Air in the mediastinum is diagnostic on radiological studies. Air can be seen as a thin line around the borders of the heart on a PA chest radiograph. Treatment includes bed rest, coughing, and avoidance of exertion. Pain is controlled with analgesics and spontaneous resolution is expected. In most cases, physical examination findings have been observed to improve after the 2nd and 3rd day of supportive care (6). Surgical decompression should not be used routinely in severe emphysema. Endotracheal intubation or even tracheostomy has been required in a few patients with airway obstruction (7). When pneumomediastinum occurs as a result of intrathoracic pathology, avoidance of exertion increases the amount of free air, which increases the risk of cyanosis, collapse, dyspnea, and tissue perfusion disturbances due to large vessel air compression (8)

.In our case, the patient presented to our emergency department 45 days after tooth extraction with complaints of increasing right shoulder swelling, redness, and chest pain. During surgery under general anesthesia, the pus in the abscess formation in the mediastinum was aspirated and the postmediastinum was irrigated with SF. With appropriate antibiotherapy, the patient's symptoms resolved after 4 days. In literature review, one case of pneumomediastinum was reported after tonsillectomy, and four cases of pneumomediastinum and cervical emphysema were reported after tooth extraction. In our case, mediastinal abscess developed due to late presentation, and the abscess was drained surgically, and treatment and follow-up were provided.

Conclusion

Pneumomediastinum should be considered by the clinician in the setting of shortness of breath or chest pain after tooth extraction. Pneumomediastinum is a life-threatening complication that can occur after dental procedures. Dentists should avoid the use of compressed air equipment as much as possible because, although rare, mediastinal emphysema may develop after tooth extraction.

Figure 1. Diffuse air density in cervical soft tissue

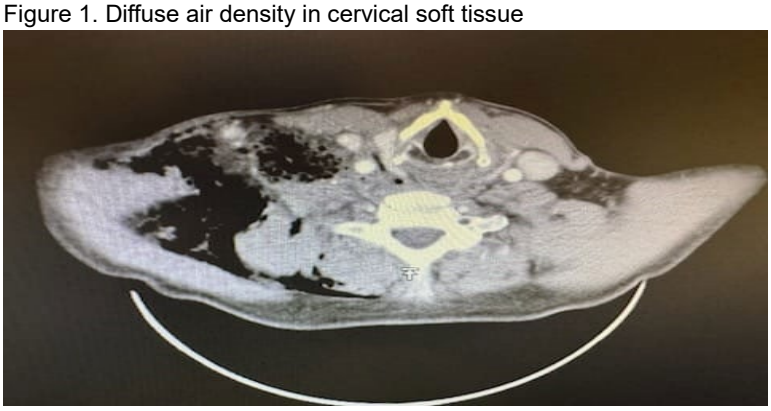


Figure 2. Pneumomediastinum, appearance of a leukemic cystic abscess in the right paratracheal area.

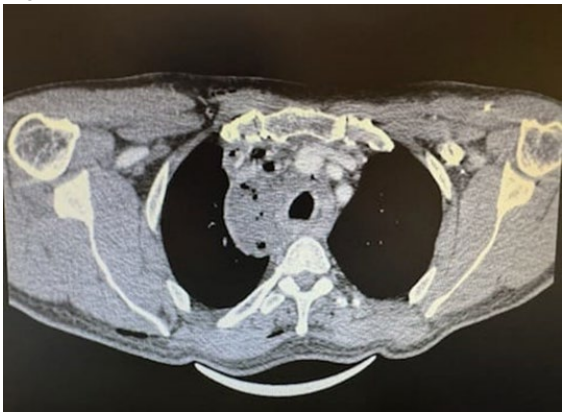
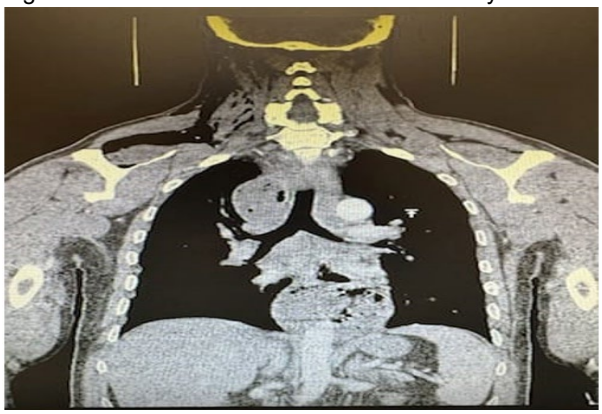


Figure 3. Picture of diffuse mediastinitis and cystic abscess in the right paratracheal area.



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5082

A Rare Case of Mechanical Thrombectomy: Bilateral Renal Infarction

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Introduction

Renal infarction is a rare ischemic event caused by complete or partial occlusion of the main renal artery or its segmental branches, which may ultimately lead to renal ischemia. Renal infarction is most commonly caused by a cardiac embolus or in situ thrombosis (1). Other etiologic causes of renal infarction include aortic thromboembolism, trauma, renal artery dissection, coagulation disorders or other atheroembolic diseases (2).

Cardioembolic disorders are the most common etiology of renal infarction and account for up to 55% of cases. Within this group, 64% to 75% are reported to have atrial fibrillation (3).

Patients present with sudden onset of abdominal or flank pain with symptoms such as nausea, vomiting, hematuria and occasional fever. Physical examination may reveal abdominal or lumbar tenderness(4). Computed tomography and renal doppler ultrasonography are the most commonly used imaging modalities. CT angiography is a highly effective method in terms of visualizing obstructions in the renal artery (5). Treatment usually includes strategies to resolve the embolus and approaches to support renal function.

This case report describes a patient who presented to the emergency department with severe abdominal pain and was diagnosed with renal infarction using computed tomography (CT) angiography.

Case

A 75-year-old woman presented to the emergency department with severe abdominal pain starting around the umbilicus. She had a history of coronary artery disease, atrial fibrillation and hypertension and antibiotic treatment was recently started for cellulitis. The patient was recently started on new generation oral anticoagulant therapy but she never used it.

Vital parameters are stable on physical examination. GCS 15; consciousness is clear, oriented and cooperative. Abdominal examination is comfortable with no signs of defense rebound. No pathology in blood tests and urinalysis. No pathology on standing plain abdominal radiography. When abdominal pain was not relieved with analgesic treatment, abdominal computed tomography angiography was performed. The imaging showed bilateral renal infarction. The patient was processed by interventional radiology. Selective renal angiography was performed and both renal arteries were completely occluded. Thrombectomy was performed in both renal arteries. The patient was hospitalized in the nephrology ward for follow-up. Left nephrectomy was performed because of retroperitoneal hemorrhage on the left side in the patient who had a decrease in hemogram during follow-up.

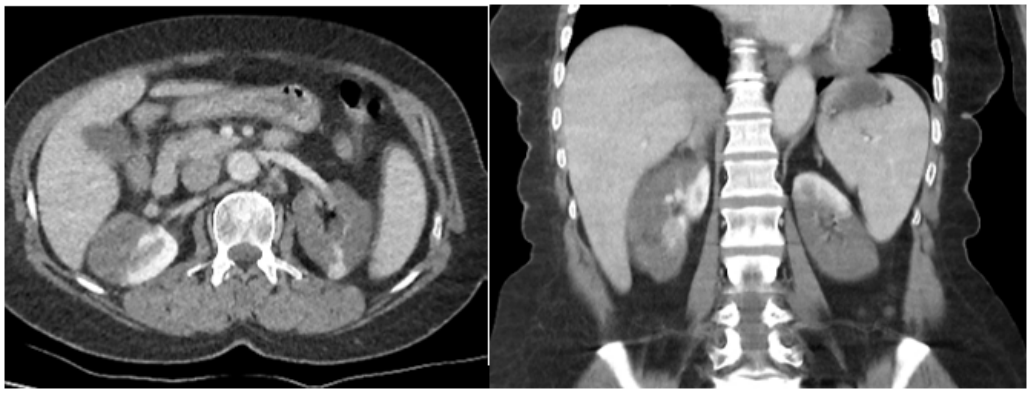

Figure 1 Reduced contrast uptake in both kidneys

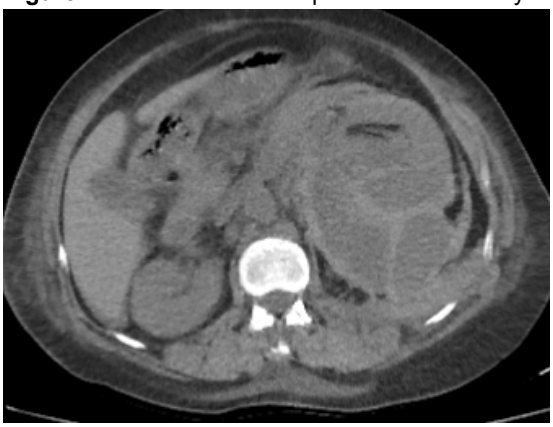

Figure 2 Retroperitoneal hematoma due to hemorrhage in the left renal artery on postoperative day 10

Discussion

Thrombotic mechanisms include endothelial damage that activates thrombus formation in situ. Another ischemic mechanism occurs in atrial fibrillation when emboli from the heart dislodge and eventually occlude the renal artery. All renal segmental arteries are terminal arteries, so a complete or partial reduction in vascular flow due to embolic or thrombotic occlusion leads to renal ischemia and infarction with focal tissue necrosis of the kidney. The associated deterioration in renal function can manifest as elevated creatinine levels and decreased glomerular filtration rates (GFR), leading to acute kidney injury, chronic kidney disease (CKD) and sometimes end-stage renal failure.

Patients with acute renal infarction typically present with sudden onset of abdominal or flank pain with associated symptoms including nausea, vomiting, hematuria and occasional fever. Findings of abdominal or lumbar tenderness may be present on physical examination (4). Skin color changes (e.g., "blue foot" syndrome or livedoreticularis) are noted in about one-third of cases (6).

Differential diagnoses for renal infarction include: aortic aneurysm, aortic dissection, appendicitis, diverticulitis, gastroenteritis, mesenteric ischemia, nephrolithiasis, pyelonephritis, renal cell carcinoma and gynecological disorders. Nephrolithiasis and pyelonephritis are often confused with renal infarction.

Risk factors for general atherosclerotic disease include male gender, significant smoking history, hypertension, hypercholesterolemia, diabetes and advanced age. Atrial fibrillation is a common source of emboli leading to renal infarction (7).

The diagnosis of renal infarction should be considered in patients who suddenly develop abdominal or flank pain with decreased renal function, elevated lactate dehydrogenase (LDH), hematuria or proteinuria and who have no urolithiasis or other diagnosable explanation for their symptoms. The risk is increased if the patient has heart disease, especially atrial fibrillation, or is older than 60 years (2).

Early diagnosis is important for prompt initiation of revascularization therapy that optimizes renal function recovery. Treatment options include intravascular thrombolysis, systemic thrombolysis, anticoagulation and antiplatelet therapy. Clinical presentation, underlying risk factors, time between symptom onset and treatment, and partial or complete arterial occlusion affect the likelihood of kidney recovery.

CT angiography usually shows a marked hypodensity or wedge-shaped perfusion defect in the kidney, which is best understood in the arterial phase (8). CT renal angiography is performed to better evaluate the vasculature, identify specific vessels of interest and determine the degree of obstruction. Renal CT arteriography is the gold standard examination for renal infarction and provides a definitive diagnosis and appropriate treatment decision (9).

The most important step in the treatment of bilateral renal infarction is to dissolve the clots and restore renal blood flow. Anticoagulation therapy is the most common treatment modality and helps prevent embolism (10). In addition, in some cases, endovascular treatment methods can be used to remove the embolus or open the vessels (11). Renal function can be preserved with early diagnosis and treatment, but if left untreated, acute renal failure may develop and dialysis treatment may be required (12).

Prognosis improves with early diagnosis. Duration of warm ischemia, collateral flow and pre-existing renal disease tend to affect prognosis following catheter-directed thrombolysis (13). Prognosis may also be affected by embolic infarction in other organs such as the spleen, intestine, liver and lungs (14). The presence of extra-renal emboli increases the length of hospitalization and overall morbidity and mortality (10).

Conclusion

Suspecting and rapidly diagnosing renal infarction because of its nonspecific clinical manifestations allows for catheter-directed thrombolysis, which is the best recommended treatment and ideally administered within 6 hours of infarction. Rapid diagnosis improves the prognosis of patients.

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Myasthenia gravis secondary to thymoma

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Introduction

Myasthenia Gravis (MG) is an autoimmune disease that causes muscle weakness due to dysfunction at the neuromuscular junction (the connection between nerves and muscles). The disease results in impaired transmission of nerve signals that control muscle contractions, due to autoantibodies developed against acetylcholine receptors. This dysfunction primarily affects voluntary muscles, often leading to weakness in the eye muscles, swallowing, and speech muscles. While Myasthenia Gravis is more commonly seen in young women and older men, it can occur in individuals of all age groups. Additionally, in some cases, pathological changes in the thymus gland, particularly thymoma, may play a role in the development of the disease.

The clinical symptoms of Myasthenia Gravis are characterized by muscle fatigue and weakness, which may improve with rest or become more pronounced later in the day. The disease commonly manifests with symptoms such as ptosis (drooping eyelids), diplopia (double vision), speech difficulties, and dysphagia (difficulty swallowing). Diagnosis is based on clinical findings, electrophysiological tests, and antibody screening, with special consideration given to the role of the thymus gland and other potential etiological factors.

In this study, we present a case of Myasthenia Gravis associated with thymoma. The patient's clinical course, diagnostic approach, and treatment process will be discussed in detail.

Case

A 46-year-old male patient presented to the emergency department with complaints of slurred speech, throat burning, itching, and difficulty swallowing for the past 15 days. The patient has no known chronic medical history and is not on regular medication. On physical examination, Glasgow Coma Score (GCS) was 15, blood pressure was 110/80 mmHg, heart rate was 86 beats per minute, respiratory rate was 18 breaths per minute, axillary temperature was 36.2°C, and blood glucose level was 106 mg/dL. Electrocardiogram (ECG) showed sinus rhythm. Neurological examination revealed the patient was alert, cooperative, and oriented, with slurred but meaningful speech. Pupils were equal and reactive to light (direct and indirect reflexes ++/++), with no nystagmus. Muscle strength in both upper and lower extremities was 5/5 bilaterally. Cerebellar tests were bilateral and coordinated, and deep tendon reflexes (DTR) were normal bilaterally. Oropharyngeal examination was normal, and no palpable masses or lymph nodes were detected.

The patient had previously been evaluated with a suspected cerebrovascular event (CVE) diagnosis, but no acute pathology was found. He was referred from an outside facility for further evaluation. Routine laboratory tests were ordered, and to rule out acute cranial pathologies, a brain CT and diffusion MRI were performed. The brain CT report indicated "no intracranial hemorrhage." Diffusion MRI showed no acute diffusion restriction. Laboratory results revealed no significant abnormalities.

The differential diagnosis included viral upper respiratory tract infections, cranial nerve palsies, oropharyngeal masses, vocal cord paralysis, and Myasthenia Gravis. Consequently, a neck CT and thoracic CT were requested. The thoracic CT report noted "A 39x23 mm soft tissue density nodule in the anterior mediastinum, potentially indicative of lymphoma or thymoma." Thymoma-associated Myasthenia Gravis was suspected. Consultation with neurology and thoracic surgery was obtained for further evaluation and treatment. The patient did not require emergency admission; however, a follow-up in the outpatient clinic was recommended for further diagnostic evaluation and treatment. The Acetylcholine Receptor Antibody (AchR) level was 9.212 (negative <0.25, borderline 0.25–0.4). In the outpatient follow-up, intravenous immunoglobulin (IVIG) therapy was initiated, and a total thymectomy was planned. The patient’s symptoms improved with IVIG treatment, and no active symptoms were observed post-surgery.

Conclusion

Myasthenia Gravis is an autoimmune neuromuscular disorder characterized by muscle weakness and fatigue. The diagnosis of this disease is based on clinical findings, electrophysiological tests, and antibody screening. Pathological changes in the thymus, particularly thymoma, are an important factor in the disease's etiology. In this case, we discussed a patient diagnosed with Myasthenia Gravis associated with thymoma, and the clinical course, diagnostic approach, and treatment strategies have been detailed. The patient initially presented with slurred speech, dysphagia, and oropharyngeal symptoms, and following further investigations, a diagnosis of Myasthenia Gravis associated with thymoma was confirmed. The patient responded well to treatment.

This case highlights the rare but significant association between Myasthenia Gravis and thymoma. Early diagnosis and appropriate treatment can significantly improve patients' quality of life. IVIG therapy and thymectomy are effective management strategies, and the improvement in the patient’s symptoms demonstrates the success of these treatments. In the differential diagnosis of Myasthenia Gravis, early and careful neurological evaluation, along with comprehensive diagnostic testing, plays a crucial role in increasing diagnostic accuracy and guiding treatment decisions.

This case further emphasizes the importance of timely diagnosis and the application of appropriate treatment protocols in controlling the symptoms of Myasthenia Gravis and improving patient outcomes.

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A case of necrotising fasciitis after intramuscular injection

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Introductıon

Necrotising fasciitis (NF) is a serious life-threatening soft tissue infection characterised by rapidly spreading necrosis and infection affecting subcutaneous tissue and fascia (1). Infection may usually occur after trauma, surgical interventions or microtraumas (2). Intramuscular injection is a rare but serious condition that may be the trigger of such infections (3). The rapidly progressive nature of NF makes early diagnosis and treatment mandatory, because delay in the diagnosis of the disease may increase morbidity and mortality rates. In the literature, broad-spectrum antibiotherapy and aggressive surgical debridement are recommended in the treatment of such cases. Although streptococcal toxic shock syndrome (STSS) has similar symptoms with NF, it progresses with hypotension, systemic toxicity and multiple organ failure as a distinctive feature, which may complicate the treatment process (4).

Case

A 67-year-old male patient with known DM presented to the emergency department with left leg pain that started one day ago. He was discharged after IM injection and no abnormal blood test results. One day later, he presented to the emergency department

again with complaints of pain, redness, bruising and swelling extending from the left inguinal region to the ankle after intramuscular injection. On physical examination, it was found that ecchymotic-bullous lesions developed in the affected area and these lesions spread to the perineal region. As a result of biochemistry tests and ultrasonography imaging, necrotising fasciitis was diagnosed. However, the patient's condition deteriorated rapidly before surgical intervention could be performed, and cardiopulmonary arrest developed on the same day during the intensive care unit and the patient died.

Discussion

Necrotising fasciitis is defined as a fatal infection that spreads rapidly and usually develops after trauma (5). The infection causes necrosis in the skin and underlying tissues, which rapidly leads to complications including sepsis, toxic shock and multiorgan failure (6). While most patients have painful, reddened and swollen areas in the early stages of NF, these findings can often be confused with other soft tissue infections. The most critical factor in the diagnosis of NF is the presence of severe pain and swelling and redness disproportionate to the lesions. After the diagnosis is made, broad-spectrum antibiotherapy should be initiated and surgical debridement should be performed (7).

Although streptococcal toxic shock syndrome (STSS) has similar clinical findings to necrotising fasciitis, this syndrome, especially caused by group A beta haemolytic streptococci, leads to a more severe clinical picture. STSS is characterised by sepsis and multiple organ failure, and delayed diagnosis and treatment may significantly increase mortality. Therefore, it is vital to start treatment without differentiating NF and STSS (8). Early diagnosis and rapid surgical interventions increase the chance of survival of patients.

Early debridements, removal of necrotic tissues, antibiotherapy and intensive care support make the treatment of patients more effective. However, the faster the surgical intervention is performed, the more favourable the prognosis of the patients will be. A multidisciplinary approach, early diagnosis and optimisation of the treatment process are of vital importance in the management of such cases (9).

Conclusion

Necrotising fasciitis after intramuscular injection is a rare but potentially fatal complication. Early diagnosis, broad-spectrum antibiotherapy and surgical debridement are important treatment modalities that can improve the prognosis of patients. However, if patients do not respond to treatment, the disease progresses rapidly and complications develop, intensive care support is required. Factors that differentiate it from different clinical pictures such as streptococcal toxic shock syndrome should ensure that the correct treatment is initiated.

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Thromboembolic Complications Associated with the Use of Prothrombin Complex Concentration in Patients Over Sixty Years of Age

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Introduction

Oral anticoagulants (OACs) are drugs commonly used to reduce the risk, treat and prevent the development of systemic venous thromboembolism (1). Patients are at high risk of bleeding despite using this drug group at therapeutic doses. In the presence of life-threatening bleeding, the most effective and safe treatment to reverse the anticoagulant effect is based on many guideline recommendations (2-4). In reversal of the anticoagulant effect, the use of Quitamine, Fresh Frozen Plasma (TDP) and Prothrombin Complex Concentrate (PCC) is preferred (5-7). However, TDP transfusion may cause adverse effects such as acute lung injury, circulatory system overload and allergic/anaphylactic reaction. PCC, on the other hand, can be used as an effective agent in reversing life-threatening hemorrhages due to its factor 2, 7, 9 and 10 content together with protein C and S without causing these effects. There are publications in the literature indicating that this group of factor Xa inhibitors produce the same effect (8).

Reversal of anticoagulation may cause a number of complications, notably the risk of recurrent thromboembolism. Rapid reversal

through PCC poses a risk ranging from 1.4% to 28% (9, 10), while TDP is between 7% and 12%. Especially in terms of complications, use of more than one dose of PCC, use of anticoagulants due to thromboembolic events and use of rivaroxaban have been found to pose a risk (1). However, there are unclear points about these complications. In this study, we aimed to determine the rates of thromboembolic complications in patients aged 60 years and older with life-threatening bleeding due to anticoagulant therapy.

Materials And Methods

Study Design

The study was a retrospective review of thromboembolic events after PCC in patients aged 60 years and older who presented to a tertiary hospital emergency department for bleeding associated with anticoagulant use between October 2023 and September 2024.

Study Population and Variables

All patients aged 60 years and older who presented to the emergency department with an INR (International Normalized Ratio) value >1.5, such as life-threatening intracranial, gastrointestinal, intra-abdominal bleeding after the use of warfarin or new generation oral anticoagulants or who had to undergo any urgent procedural procedure between the specified dates were identified by scanning the Hospital Information Management System (HIMS). Patients under 60 years of age and patients who received Fresh Frozen Plasma before PCC administration were excluded.

The patients were divided into four groups according to the PCC, TDP and vitamin K used to reverse the anticoagulant effect. Group 1 was classified as patients who received PCC alone, group 2 as patients who received PCC and vitamin K together, group 3 as patients who received TDP and group 4 as patients who received TDP and vitamin K together. PCC use in the emergency department is based on weight, INR and life-threatening condition caused by coagulopathy. However, problems in drug supply are a limiting factor for use in appropriate indications.

Pulmonary embolism, ischemic stroke, deep vein thrombosis, myocardial infarction or thrombosis of another site within 72 hours after reversal of anticoagulant effect was defined as "early thrombotic event". Thromboembolic events that may cause readmission during hospitalization or outpatient follow-up within 60 days after reversal of anticoagulant effect were defined as "late thrombotic events". Data on complications were confirmed by laboratory and/or imaging examinations through HIS and patient record files.

Demographic data (age, gender, comorbidities, anticoagulant used and the reason for use) obtained from HIS and emergency department patient registration files, complaint at admission, value of coagulation parameters before and after treatment at admission, treatment and dose administered, emergency department outcome, if there was a condition requiring inpatient follow-up and treatment, how many days the patient was followed up by which clinic, whether thromboembolic event developed and if thromboembolic event developed, whether it occurred in the early or late period were recorded on the study form.

Results

The study data were obtained from a retrospective review of 166 records of 166 patients aged 60 years and older who met the inclusion criteria and presented to the emergency department with life-threatening bleeding due to anticoagulant use. The mean age of the patients was 77.7 ± 10.4 years and 52.4% (n=87) were female. Warfarin was the most commonly used anticoagulant in 70.5% (n=117) and 79.5% were taking it for atrial fibrillation. After oral anticoagulant use, 34.3% (n=57) presented to the emergency department with symptoms and complaints of gastrointestinal bleeding. Demographic characteristics of the patients are given in Table 1.

After bleeding, 71.1% (n=118) received a total of 163 units of TDP and 28.9% received a total of 99 vials of PCC. The median time to normalization of coagulation parameters after treatment was 5 hours (IQR 3.30-7.00) (Table 2). TEC developed in 9% (n=15) after treatment and 4 patients in the PCC group and 11 patients in the TDP treatment protocol group. There was no statistical difference in the rate of complications between the two treatment protocol groups (p=0.84). However, there was a significant difference in response time to life-threatening bleeding and normalization of INR level after treatment (p<0.001). Warfarin was the most commonly used anticoagulant in patients who developed TEC. Unstable angina pectoris and acute myocardial infarction occurred in 2.4%. Characteristics of patients who developed TEC are given in Table 3.

Table 1: Demographic characteristics of the patients.

Characteristics, n=166	Value
Age (years), mean, (± SD*)	77 ± 10.4
60-64 years old, n (%)	16 (%9.6)
65-69 years old, n (%)	25 (%15.1)
70-74 years old, n (%)	21 (%12.7)
75-79 years old, n (%)	24 (%14.5)
80 years and older, n(%)	80 (%48.2)
Gender, n (%)	
Male	87 (%52.4)
Female	79 (%47.6)
Comorbid Diseases, n (%)	
Atrial Fibrillation,	144 (%86.7)
Hypertension	128 (%77.1)
Coronary Artery Disease,	61 (%36.7)
Diabetus mellitus,	57 (%34.3)

Congestive Heart Failure,	39 (%23.5)
Chronic renal failure,	25 (%15.1)
COPD**,	19 (%11.4)
Cancer,	19 (%11.4)
Valve Disease	13 (%7.8)
Anticoagulant Used, n (%)	
Warfarin	117 (%70.5)
Rivaroxaban	20 (%12.0)
Apixaban,	15 (%9.0)
Edoxaban	12 (%7.2)
Dabigatran	2 (%1.2)
Reason for Anticoagulant Use, n (%)	
Atrial Fibrillation,	132 (%79.5)
Prosthetic Cover	14 (%8.4)
Ischemic Stroke,	11 (%6.6)
Pulmonary Embolism,	8 (%4.8)
Deep Vein Thrombosis	1 (%0.6)
Presenting Complaint, n (%)	
Bloody or black stools,	31 (%18.7)
Body Bruising	30 (%18.1)
Shortness of breath	16 (%9.6)
Red Urine	16 (%9.6)
Change of Consciousness,	13 (%7.8)
Abdominal Pain	12 (%7.2)
Bloody Vomiting	10 (%6)
Bleeding from the anus	7 (%4.2)
Fainting	6 (%3.6)
General Condition Disorder,	6 (%3.6)
Dizziness,	5 (%3)
Nosebleeds	4 (%2.4)
Joint Swelling	4 (%2.4)
Bloody sputum	2 (%1.2)
Trauma,	2 (%1.2)
Bleeding Gums	1 (%0.6)
Headache	1 (%0.6)
Bleeding Area, n (%)	
Gastrointestinal System,	57 (%34.3)
There is no bleeding in any body part,	36 (%21.7)
Musculoskeletal System,	32 (%19.3)
Urinary System	17 (%10.2)
Brain,	14 (%8.4)
Orafarinks,	3 (%1.8)
Nasopharynx	3 (%1.8)
Lung	2 (%1.2)
Batin	2 (%1.2)
Emergency Department Outcome, n (%)	
Discharged from the emergency room,	63 (%38)
Emergency to ICU admission,	53 (%31.9)
Admission from Emergency to Clinic,	45 (%27.1)
Ex in the emergency room	5 (%3)
Hospital outcome, n (%)	
Discharged from hospital,	118 (%71.1)
Death in hospital	48 (%28.9)

*SD, standard deviation. **COPD, Chronic Obstructive Pulmonary Disease.

Table 2: Treatment protocols for life-threatening bleeding.

Treatment Protocol	Value
PCC*, n (%)	38 (%22.9)
20 ml,	6 (%3.6)
40 ml,	24 (%14.5)
60 ml,	8 (%4.8)
PCC + vitamin K, n (%)	10 (%6)
20 ml + vitamin K,	1 (%0.6)
40 ml + vitamin K,	9 (%5.4)
TDP**, n (%)	114 (%68.7)
1 unit TDP,	74 (%44.6)
2 units TDP,	33 (%19.9)
3 units TDP,	2 (%1.2)
4 units TDP,	2 (%1.2)
TDP + vitamin K, n (%)	4 (%2.4)
1 unit of TDP + vitamin K,	6 (%3.6)
2 units of TDP + vitamin K,	1 (%0.6)

*PCC, Prothrombin Complex Concentrate. **TDP, Fresh Frozen Plasma.

Discussion

In case of life-threatening bleeding due to oral anticoagulant use, PCC administration may have a better effect than TDP in terms of early treatment response and normalization time of INR value. This allows early intervention in patients requiring surgical or interventional procedures after bleeding, but caution should be exercised in terms of TEC that may occur. In our study, the rate was 9% for both treatment protocols and was lower compared to the literature (1, 11, 12). When analyzed according to the treatment protocol, it was observed that TEC occurred in 4 of the patients who received PCC and 11 of those who received TDP, and there was no statistical significance. The limited number of patients in the study is thought to be the most important reason for this situation.

The rate of warfarin use was higher in patients who developed TEC and we think that this was due to the fact that its use as an anticoagulant is more common and older than factor Xa inhibitors. In some studies in the literature, it has been reported that PCC use causes TEC more frequently in bleeding occurring after factor Xa inhibitors, especially rivaroxaban use (1). In our study, only two of the patients who received PCC were using factor Xa inhibitors. Although this did not seem to be statistically significant, it had a rate of 50% in the group. At the same time, in the presence of life-threatening bleeding, the complication rate increased as the PCC dose increased, while in the TDP group, complications developed after a single dose administration.

The duration of complication development ranged between day 1-60 in patients who underwent TDP and between day 19-60 in the PCC group. These durations were found to be higher compared to other studies (11). However, due to the current clinical status of the patients, it is necessary to re-anticoagulation after discontinuation of anticoagulant treatment and patient-based evaluation in terms of comorbid conditions such as underlying cancer.

This study has several limitations that should be noted. First, due to the retrospective design of the study, the cause and effect relationship cannot be clearly demonstrated. In addition, retrospective data collection within the electronic hospital health record may have led to missing or incomplete data, resulting in data limitations. The doses of treatment administered were at the discretion of each provider and not all patient-specific factors may have been documented.

Despite limitations, the results provide a direction for future studies. Additional studies evaluating PCC for reversal of life-threatening bleeding should consider an in-depth review of TEC. Providers should also assess patient-specific factors that increase the likelihood of developing TEC with the necessity of PCC therapy.

The results of the study may shed light on the conditions that may occur after treatment of life-threatening bleeding in people receiving PCC for anticoagulation reversal. In the last decade, PCC has become the preferred method of anticoagulation reversal in the face of acute life-threatening conditions to avoid anticoagulation and extra fluid overload. However, in emergency situations where PCC is considered, what may happen after the dose is administered is often overlooked. The results of this study suggest that several patient populations are more likely to develop TEC, but in particular, those receiving TDP to reverse the effect of warfarin are more likely to develop TEC even after a single dose. However, patient-based prospective studies are needed to obtain specific results.

Table 3: Patients with thromboembolic complications and their characteristics.

Age/Gender	Application Complaint	Anticoagulant/ Reason for use	Treatment Protocol	TEC	TEC Development time	Outcome
78 / K	Vomiting with blood (GIS)	Edoxaban Prosthetic cover	20 ml PCC	USAP**	Day 60	Day 3 Discharge from Gastroenterology clinic
73 / K	Shortness of breath	Warfarin	40 ml PCC	USAP	Day 23	13th discharge from

		Atrial fibrillation				ICUʏ
85 / K	Abdominal pain (GIS)	Rivaroxaban Atrial fibrillation	60 ml PCC	Pulmonary embolism	Day 19	Exutus on day 21 in the ICU.
90 / E	Bruising on the body (musculoskeletal system)	Warfarin Atrial fibrillation	40 ml PCC + vitamin K	Ischemic stroke	Day 24	Discharged from the emergency room
86 /E	Blood from the rectum (GIS)	Warfarin Atrial fibrillation	1 unit TDP	Ischemic stroke	Day 60	Exutus on day 69 in the ICU.
85 / K	Abdominal pain	Warfarin, Atrial fibrillation	2 units TDP	DVT***	Day 3	He was discharged from the emergency room.
76 / E	Bloody stools (GI)	Warfarin Atrial fibrillation	1 unit of TDP + vitamin K	USAP	Day 26	Day 26 discharge from Gastroenterology clinic.
68 / E	Shortness of breath	Warfarin Atrial fibrillation	1 unit TDP	DVT	1. day	Exutus on day 1 in the ICU.
65 / E	Bruising on the body (musculoskeletal system)	Warfarin Atrial fibrillation	2 units TDP	Artery embolism	Day 5	He was discharged from the emergency room.
79 / E	General condition disorder	Apixaban, Atrial fibrillation	1 unit TDP	Artery embolism	1. day	Exutus on day 2 in the internal medicine clinic.
62 / E	Abdominal pain (musculoskeletal system)	Warfarin, Atrial fibrillation	1 unit of TDP + vitamin K	Myocardial infarction	Day 7	She was discharged from the General Surgery clinic on day 7.
75 / E	Bruising on the body (musculoskeletal system)	Warfarin, Atrial fibrillation	1 unit TDP	Myocardial infarction	Day 27	He was discharged from the emergency room.
62 /E	Confusion (Brain)	Warfarin Prosthetic cover	1 unit TDP	Ischemic stroke	Day 12	Day 14 exutus in the ICU.
82 / K	Bloody urine (urogenital system)	Warfarin, Atrial fibrillation	1 unit TDP	Peripheral vein thrombosis	Day 15	He was discharged from the emergency room.
77 / E	Bloody stools (GI)	Edoxaban, Atrial fibrillation	1 unit TDP	USAP	Day 5	Exutus on day 8 in the ICU.

*TEC, Thromboembolic Complication. **USAP, Unstable Angina Pectoris. ***DVT, Deep Vein Thrombosis. ʏICU, Intensive Care Unit.

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5767

Ruptured Abdominal Aortic Aneurysm: A Case Report from an Emergency Medicine Perspective

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Introduction

An abdominal aortic aneurysm (AAA) is defined as a focal dilation of the aorta measuring more than 1.5 times the normal diameter. While most AAAs are asymptomatic and incidentally discovered, rupture is a life-threatening complication associated with extremely high mortality. It is estimated that a significant proportion of patients with ruptured AAA's die before reaching medical care. The emergency department plays a critical role in the early recognition and management of this vascular emergency.
This case report presents the clinical course of a patient with a prior history of endovascular aneurysm repair (EVAR) who developed syncope following physical exertion and was diagnosed with a ruptured AAA.

Case Presentation

A 79-year-old male presented to the emergency department following a syncopal episode that occurred shortly after climbing a tree. He had a known history of abdominal aortic aneurysm and had undergone EVAR several years prior. Upon arrival, his level of consciousness was preserved; however, he appeared pale, diaphoretic, and lethargic. He also reported pain in the left inguinal region. Vital signs were as follows: blood pressure 71/53 mmHg, heart rate 92 bpm, and oxygen saturation 95% on room air. Despite aggressive intravenous fluid resuscitation, hypotension persisted.
Computed tomography angiography (CTA) revealed patent superior mesenteric artery (SMA) and bilateral renal arteries. A significant stenosis was observed at the origin of the celiac artery, while the inferior mesenteric artery was non-opacified, suggestive of occlusion. A pant-leg type endograft was visualized, extending from the infrarenal aorta to both common iliac arteries. Marked aneurysmal dilatation of the distal abdominal aorta and both iliac arteries were noted, measuring up to 185 mm at its widest point.
At the distal right common iliac artery, contrast extravasation was seen extending from within the endograft lumen into the aneurysm sac, consistent with a suspected Type 1b-3 endoleak. A heterogeneous hematoma was present within the aneurysm sac. Additionally, a large hemoperitoneum was identified in the left abdominal quadrant, measuring approximately 18 × 12 cm. Based on these radiological findings, a diagnosis of ruptured abdominal aortic aneurysm was established. The vascular surgery team was urgently consulted, and the patient was taken for emergency surgical intervention.

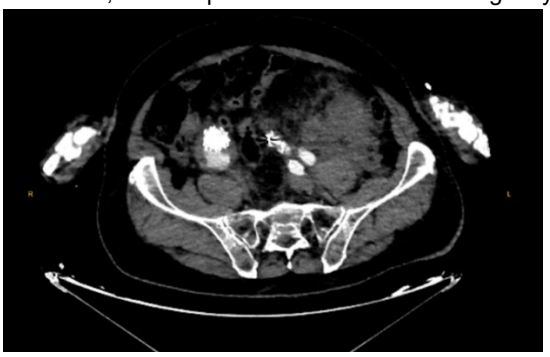

Figure: Contrast extravasation into the aneurysm sac at the right common iliac artery

Discussion

Ruptured AAA is a catastrophic vascular emergency that requires immediate recognition and intervention. Mortality remains exceedingly high; retrospective data suggest that nearly 50% of patients with ruptured AAA die before reaching the hospital, and among those who do, another 50% may not survive surgery or postoperative period.
In this case, the presence of a prior EVAR and subsequent development of an endoleak likely contributed to increased intra-sac pressure, culminating in rupture. Endoleaks occur in up to 20% of EVAR patients and represent one of the most serious complications following the procedure.
From an emergency medicine standpoint, elderly patients presenting with syncope, flank or groin pain, and signs of shock must be evaluated for potential AAA rupture. Bedside ultrasonography is a rapid and useful initial imaging modality, particularly in unstable patients. In hemodynamically stable cases, CTA remains the gold standard for anatomical assessment and surgical planning. Early

diagnosis and expedited vascular surgery consultation are paramount to improving survival in this time-critical condition.

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Adrenal Insufficiency: A Life-Threatening Endocrine Crisis in the Emergency Department – A Case Report

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Introduction

Adrenal insufficiency is a rare but potentially life-threatening endocrine disorder characterized by the inadequate production of cortisol and/or aldosterone from the adrenal cortex. The acute form of this condition, adrenal crisis, carries a high risk of mortality, particularly in undiagnosed patients or in those with known insufficiency who fail to receive adequate steroid replacement during periods of physiological stress. In the emergency department (ED), adrenal crisis should be considered in the differential diagnosis of patients presenting with unexplained hypotension, hypoglycemia, or altered mental status.

This case report presents the emergency evaluation and management of a male patient with a history of diabetes mellitus (DM), hypertension (HT), and coronary artery disease (CAD) who presented with clinical features consistent with adrenal crisis.

Case Presentation

A 65-year-old male presented to the emergency department with complaints of decreased urinary output, persistent vomiting, and altered mental status over the preceding few days. On the initial assessment, the patient was alert, cooperative, and oriented, but appeared hemodynamically unstable. His blood pressure was 79/44 mmHg, heart rate 90 bpm, and oxygen saturation was 93% on 2 L/min nasal oxygen.

Point-of-care capillary glucose testing revealed severe hypoglycemia (25 mg/dL), prompting the initiation of intravenous dextrose infusion. Despite three consecutive boluses of dextrose, the patient's blood glucose levels remained low. Due to sustained hypotension, norepinephrine infusion was initiated, but blood pressure remained low. Physical examination revealed no peripheral edema, and lung auscultation was normal.

Arterial blood gas analysis showed a pH of 7.07, bicarbonate (HCO□□) of 8.5 mmol/L, and PCO□ of 29 mmHg, consistent with uncompensated metabolic acidosis. Laboratory tests revealed potassium 5.4 mmol/L, sodium 136 mmol/L, calcium 9.1 mg/dL, and phosphate 6.7 mg/dL. Inflammatory markers were elevated with white blood cell count of 30.08 x10⁹/L, CRP 29 mg/L, and procalcitonin 1.76 ng/mL. Urinalysis and further investigation identified a urinary tract infection as the likely source of systemic infection.

No significant findings were noted on imaging studies. In light of the metabolic abnormalities, persistent hypoglycemia, and refractory hypotension, a preliminary diagnosis of adrenal insufficiency was made. Empirical glucocorticoid therapy was initiated with intravenous hydrocortisone. Following treatment, the patient's blood pressure improved to 125/61 mmHg, blood glucose stabilized at 251 mg/dL, and blood gas parameters showed marked improvement (pH 7.30, HCO□□ 13.4 mmol/L). He was admitted to a multidisciplinary intensive care unit for further monitoring and management.

Discussion

Adrenal crisis is a medical emergency caused by the acute inability of the body to mount an adequate cortisol response during stress. It typically presents with hypotension, hypoglycemia, hyponatremia, and metabolic acidosis, and can rapidly progress to shock and death if not promptly recognized and treated. Although relatively rare in emergency practice, the consequences of delayed diagnosis can be fatal.

In this case, features such as refractory hypotension, recurrent episodes of hypoglycemia, and uncompensated metabolic acidosis strongly supported the diagnosis of adrenal crisis. Prompt initiation of glucocorticoid therapy, even prior to laboratory confirmation, was life-saving and aligned with current emergency medicine guidelines.

Emergency physicians must maintain suspicion for the patients presenting with unexplained hemodynamic instability, particularly when vasopressor therapy is ineffective. Recognition of the adrenal crisis triad, hypotension, hypoglycemia, and altered mental status, is critical to ensure timely management.

Conclusion

Adrenal insufficiency, particularly when manifesting as an adrenal crisis, represents a critical endocrine emergency that requires rapid recognition and intervention in the emergency department. For the patients presenting with unexplained hypotension, altered mental status, or persistent hypoglycemia, adrenal crisis should be actively considered and not ruled out prior to initiating treatment. In cases of strong clinical suspicion, empirical glucocorticoid replacement therapy should not be delayed pending diagnostic confirmation. Early management is essential to stabilize hemodynamics and prevent potentially fatal outcomes.

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5923

Balancing bleeding and thrombosis risks: severe thrombocytopenia, cerebral microbleeds, and pulmonary embolism following neurosurgery

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Introduction

Pulmonary embolism (PE) is a potentially fatal complication often managed through anticoagulant therapy.[1] However, the coexistence of cerebral microbleeds significantly complicates therapeutic decisions, due to an elevated risk of intracranial hemorrhage.[2] The presence of severe thrombocytopenia further complicates management, making anticoagulation risky due to bleeding potential.[3] This case highlights the complex therapeutic dilemma faced by clinicians managing patients with concurrent hemorrhagic and thrombotic complications, underscoring the importance of careful clinical assessment and multidisciplinary collaboration among emergency and internal medicine specialists also importance of treatment timing.

Case

A 72-year-old female presented to the emergency department with a 2-day history of fever, productive cough with purulent sputum, and rapid deterioration in general condition. Her medical history included type 2 diabetes mellitus and surgical resection of glioblastoma multiforme (GBM), performed three months previously, followed by adjuvant chemotherapy with temozolomide completed ten days prior to admission.

Upon arrival, her vital signs were normal, except for desaturation on pulse oximetry, which was 85%. The patient appeared lethargic, with a Glasgow Coma Scale (GCS) score of 13. Physical examination revealed bilateral pulmonary rales, absence of rhonchi, and bilateral +1 pitting edema in the lower extremities. Laboratory evaluations revealed severe pancytopenia: platelet count of 1000/μL, hemoglobin level of 7.2 g/dL, and leukocyte count of 120/μL. Pulmonary artery angiography computed tomography (CT) demonstrated pulmonary embolism involving bilateral segmental pulmonary arteries, accompanied by diffuse bilateral ground-glass opacities consistent with pneumonia (Figure 1). Cranial imaging showed postoperative changes, multiple cerebral microbleeds predominantly in the left frontal region, and a subdural effusion (Figure 2).

The patient received transfusions of thrombocyte concentrate to manage her severe thrombocytopenia and underwent multidisciplinary consultation involving neurology, hematology, oncology, and intensive

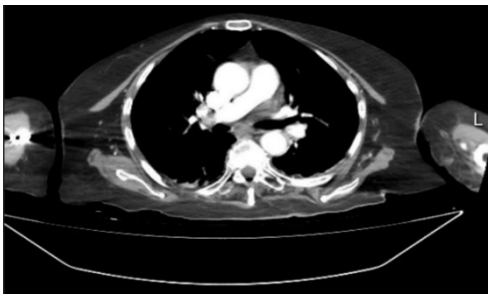

Figure 1 computed tomography pulmonary artery angiography showing pulmonary embolism segments

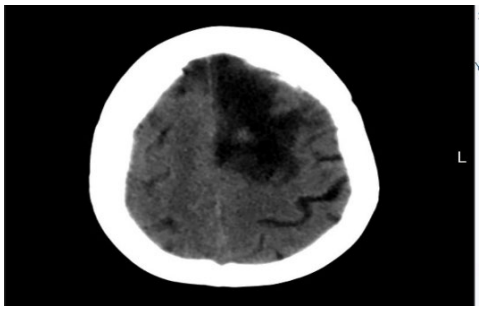

Figure 2 Brain Computed Tomography showing cerebral micro bleeding areas and post operative changes in the left frontal region

care specialists. She was promptly transferred to the intensive care unit (ICU) due to her deteriorating clinical status. Anticoagulant therapy with low molecular weight heparin (LMWH) was initiated cautiously once her platelet count stabilized above 50,000/μL, achieved on the 30th day of hospitalization. After seven days in the ICU, she was transferred to the oncology ward for further care. Following a total hospital stay of approximately 40 days, her clinical condition got better due to inoperable tumor progression, necessitating transfer to palliative care.

Discussion

This case underscores the clinical paradox and complexity inherent in simultaneously managing conditions predisposing patients to both hemorrhagic and thrombotic complications. Severe thrombocytopenia poses a major barrier to initiating anticoagulation therapy, despite the critical need posed by the presence of pulmonary embolism[4-6]. Concurrently, cerebral microbleeds increase the risk of intracranial hemorrhage, further complicating anticoagulation decisions.[2] Starting anticoagulant therapy before complete diagnosis may cause the increase hemorrhagic complications of surgery or increase hemorrhage volume, that is why timing of anticoagulant

therapy timing plays a crucial role in this patient's therapy.[3] Emergency physicians play a crucial role in early recognition, prompt diagnosis, initiating necessary transfusions, and coordinating early multidisciplinary consultations.[1] Management strategies must be carefully balanced, prioritizing meticulous monitoring of hematologic parameters, precise adjustment of anticoagulant dosages, and tailored supportive care to achieve optimal patient outcomes.

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5946

An Emergency Patient with Acute Paraplegia: Leriche Syndrome

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Introduction

Leriche syndrome, commonly referred to as aortoiliac occlusion disease (AIOD), is a product of atherosclerosis affecting the distal abdominal aorta, iliac arteries and femoropopliteal vessels (1).

When symptomatic, AIOD classically presents with the triad of claudication, sexual dysfunction and absence of femoral pulses. Claudication refers to cramping leg pain that may recur with exercise. Leriche syndrome is caused by atherosclerosis (2)

Ten percent of patients with peripheral arterial disease (PAD) may be asymptomatic, so the exact prevalence and incidence of Leriche syndrome is unknown. However, the prevalence of PAD increases with age. The prevalence of PAD was found to be 14% in patients older than 69 years (3).

Acute occlusion of the abdominal aorta (AOAA) is a rare vascular emergency with high morbidity and mortality rates. The typical presentation includes bilateral lower limb ischemia with pulselessness, paresthesia, pallor and intense pain. AOAA associated with flaccid paraplegia due to spinal cord ischemia is extremely rare and poorly documented (4).

In this case, we describe the diagnosis of Leriche syndrome in a patient who presented to the emergency department with sudden onset of weakness and loss of sensation in both legs.

Case

A 68-year-old male patient presented with complaints of loss of strength and pain in both the right and left legs that started half an hour after dinner. On admission to the hospital, the loss of strength in the right leg resolved spontaneously and the loss of strength in the left leg continued. She described leg pain while walking for about three days. On physical examination, there was no pulse in the left leg and the leg was cold and pale. There was no difference in diameter between the legs. His known diseases were diabetes mellitus, atrial fibrillation and prostate cancer. The patient stated that he could not use his anticoagulant given for atrial fibrillation for several days.

Doppler ultrasonography showed a monophasic flow pattern distally from the common femoral artery of the left lower extremity. Computed tomography angiography was performed. Distal occlusion of the aorta from the infrarenal level was detected. There was also a thrombus in the right atrium. The patient was consulted to cardiovascular surgery. Urgent operation was planned and the patient was transferred to intensive care unit.

Discussion

Acute aortic occlusion (AAO) is a rare, life-threatening condition estimated to occur in 2.7-5.0 cases per million per year. It primarily affects older adults with cardiovascular comorbidities. AAO refers to blockage of blood flow through the aorta due to acute thrombosis or embolism, leading to ischemia of downstream tissues. Current data estimate that approximately 20% of these cases are saddle aortic embolism, which has a higher mortality rate (5).

This condition is usually characterized by bilateral acute lower extremity ischemia with rhabdomyolysis and ischemic peripheral nerve damage, leading to limb anesthesia and paralysis. In rare cases, in the absence of medullary compression, spinal cord infarction mimicking cauda equina syndrome may be associated (6).

Risk factors for AAO include coagulopathy, smoking, heart or lung disease, hypertension, diabetes mellitus, history of cancer, previous vascular surgery and chronic kidney disease. AAO caused by arterial embolism typically occurs in the setting of atrial fibrillation,

ventricular thrombus, heart failure, valvular disease and rarely tumor embolism (7).
Coronary artery disease is observed in 10-71% of patients with peripheral arterial disease; therefore, electrocardiography is also recommended to rule out coronary artery disease (4).
Arterial dissection in the iliac arteries may mimic the symptom profile of Leriche syndrome, causing claudication and absence of femoral pulses (8).
Without treatment, the prognosis of Leriche syndrome is poor. However, with modern medical treatment, the results are good. In some cases with slow progression or onset of Leriche syndrome, collaterals may develop as a self-compensating mechanism (9).
Leg ischemia, heart failure, myocardial ischemia/infarction, gangrene and even death are potential complications of Leriche syndrome (10).
This case highlights that in all cases of lower limb paresthesia and paralysis, neurovascular examination, including assessment of pulse and skin changes, is essential to avoid misdiagnosis. Cases presenting with urinary and fecal incontinence can easily be mistaken for a neurological etiology.

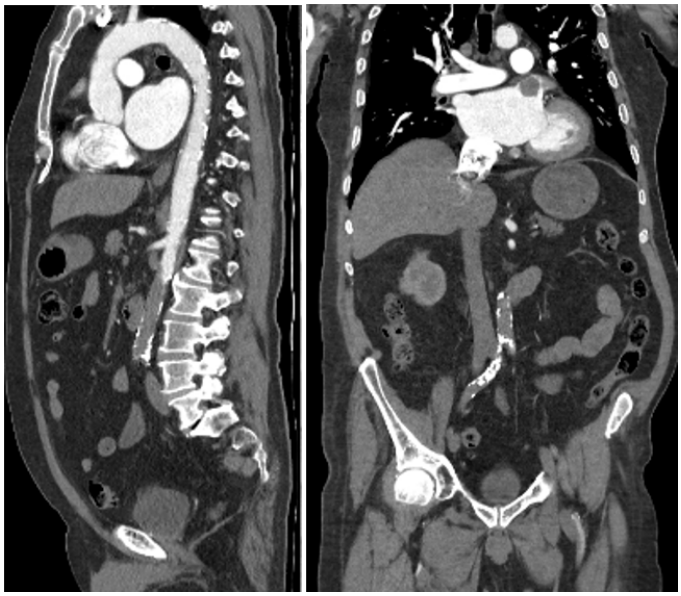

Figure 1 Occlusion at the location indicated by the arrow on coronal and sagittal sections

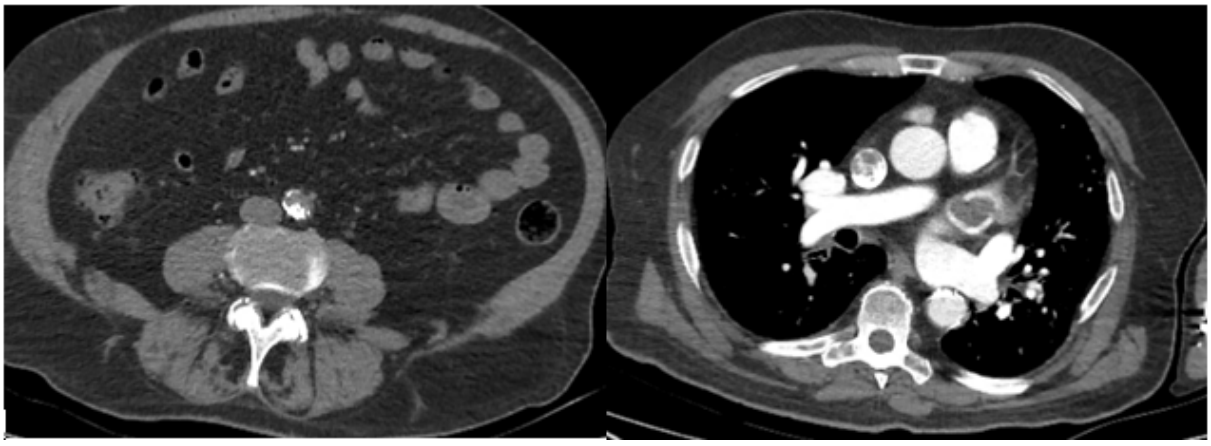

Figure 2 Infrarenal occlusion of the abdominal aorta and thrombus in the right atrium in axial section

The first-line imaging modality in patients with suspected AAO is thoracic, abdominal and pelvic CT angiography. Once diagnosed, the cornerstone of management for patients with AAO is the initiation of anticoagulation, which reduces the risk of ongoing embolism or thrombus progression (secondary thrombus). Other essential components of AAO management include maintenance of normal oxygen saturation, intravenous fluid resuscitation targeting euvolemia, and pain control (7).
A relative decline in open aortic surgery for AAO and increased use of hybrid and fully endovascular surgical techniques have been noted. Direct revascularization should be recommended as the surgical treatment method (11).

Conclusion

Leriche syndrome is a serious complication of peripheral arterial disease that can be successfully managed with early diagnosis and appropriate treatment. Both surgical and endovascular treatment options can improve patients' quality of life and relieve their symptoms. Attention to clinical signs and the use of accurate diagnostic methods are critical in the management of this disease. Leriche

syndrome may present with paraplegia; therefore, in cases with acute paraplegia, infrarenal acute arterial occlusion should be considered in the differential diagnosis and pulses should be checked.

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6118

Evaluatıon Of Tenascın-C Parameter Level In Case Of Acute Ischemıc Stroke

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1T.C. Sağlık Bakanlığı Gediz Devlet Hastanesi Acil Tıp

2T.C. Sağlık Bakanlığı Kayseri Şehir Hastanesi Acil Tıp ABD

3T.C. Sağlık Bakanlığı Kayseri Şehir Hastanesi Tıbbi Biyokimya ABD

4T.C. Sağlık Bakanlığı Kayseri Şehir Hastanesi Radyoloji ABD

1. Introduction

Stroke, according to the definition of the World Health Organization (WHO), has been described as a condition related to focal or global loss of cerebral functions that can start rapidly and last for 24 hours or longer, and may also cause death [1].

Stroke is the second most common cause of death worldwide, ranking third among the causes of disability in the world in terms of disability-adjusted life years (DALY) [2].

85% of all strokes are classified as ischemic strokes. For these patients, the most effective treatment methods that have been proven are intravenous thrombolytic treatment or mechanical removal of the clot. Patients should be diagnosed as soon as possible to apply the thrombolytic treatment protocol with proven effectiveness. Indications and contraindications for this treatment should be evaluated. Even if this whole process is managed as quickly as it should be, many patients may be deprived of this treatment option because the deadline for treatment has passed.

Tenascin-C is a multimodular protein containing four different parts: an N-terminal junction domain, a set of epidermal growth factor-like domains (EGF-L), a set of fibronectin type III-like domains (FNIII), and a C-terminal fibrinogen-like globule (FBG) [3]. Tenascin-C is the prototype and best-characterized member of the tenascin family.

Tenascin-C is produced during the fetal period in a wide variety of developing organs such as the teeth, mammary glands, and nervous system. Expression levels decrease as people get older. Nearly negligible amounts of Tenascin-C were detected in healthy adult tissues. It is produced temporarily with tissue damage, and its production stops after tissue repair is completed [4].

It is known that chronic inflammation is an important factor in atherosclerosis-related complications. However, the factors controlling these immune mechanisms have not been clearly determined. Toll-like receptor-4 (TLR-4) is a key signaling molecule in the body's immune response. It plays an important role in atherosclerosis, thanks to its ability to promote inflammation, proteolysis, and thrombosis. Tenascin-C binds to TLR-4 via its fibrinogen-like part (FBG). Expression of both Tenascin-C and TLR-4 is increased in unstable human atheroma [5].

The inflammatory response to acute cerebral ischemia is an important factor in stroke pathophysiology and outcome. The immune response begins locally in vessels that have low perfusion, occluded vessels, and in the ischemic brain parenchyma. These inflammatory factors spread throughout the organism [6]. Studies examining the effect of Tenascin-C in ischemic stroke cases, which are very closely related to inflammation, are extremely limited.

In the guidance of current publications in the literature, we carried out this study to reveal the importance of Tenascin-C in ischemic

stroke cases. We examined Tenascin-C, a biomarker that predicts the starting time of stroke, which has a major morbidity and mortality rate, to accelerate the diagnosis and to determine the best treatment method for ischemic stroke cases.

2. Methods

The study we presented was conducted prospectively for a period of 5 months between August and December 2020 at Kayseri City Training and Research Hospital Emergency Department. Written informed consent was obtained from all of the participants. Necessary permissions were obtained for the study with the decision of Kayseri City Training and Research Hospital Clinical Research Ethics Committee dated 09.07.2020 and numbered 115. This study has been prepared by the STROBE checklist.

2.1. Patient Group

Patients over the age of 18 who presented to our emergency department on an outpatient basis or by ambulance and whose consent was obtained were included in the study. The following symptoms were evaluated in the patients' application: numbness and/or loss of strength in the face, arm, and/or leg on one side of the body, blurred consciousness, difficulty speaking and/or understanding, difficulty walking, dizziness, loss of balance and coordination, loss of vision on one or both sides, double vision, and severe headache symptoms. 64 patients diagnosed with acute ischemic stroke, supported by clinical findings, laboratory parameters, and radiological examinations, were included in the study.

Patients under the age of 18, those who did not give consent for the study, those who refused treatment, those who had cerebral vascular injury secondary to trauma (traumatic brain injury), and those who had a cerebral hematoma and subarachnoid hemorrhage on their brain CT were excluded from the study. Additionally, patients with sepsis, malignancy, acute renal failure, and severe hepatic failure, which were thought to increase Tenascin-C in previous studies in the literature, were not included in the study.

A control group was formed with 64 people over the age of 18 who had received an informed consent form, had none of the symptoms in the inclusion criteria, and had similar age and gender characteristics as the case group. These individuals had applied to the emergency service for other reasons.

2.2. Collecting Data and Analysing Measurements

Participants' demographic characteristics, disease history, examination findings, start time and duration of symptoms, radiological findings, vital values at admission, and blood tests for differential diagnosis of stroke (including hemogram, blood glucose, kidney function tests, electrolytes, coagulation parameters, and arterial blood gas) were evaluated, as well as serum Tenascin-C levels and clinical outcomes. The data obtained were recorded on the Patient Follow-up Form prepared specifically for the study.

Patients diagnosed with acute ischemic stroke were divided into three subgroups based on stroke intensity, as determined by anamnesis and physical examination findings according to the NIHSS: group 1 (0-6 points, mild-moderate), group 2 (7-15 points, moderate-severe), and group 3 (16-42 points, severe-very severe). Serum Tenascin-C levels were compared to evaluate stroke severity between the groups, and the relationship between the patient's NIHSS scores and serum Tenascin-C levels was also analyzed.

Blood samples were taken from the patients in a seated position and after a 20 min rest following 12h of fasting. The sera were separated after the samples were centrifuged for 10 minutes at 5000 rpm (NF 400 centrifuges, Turkey). They were kept at −80 °C until assays for ACE 2 level was performed by an experienced clinical biochemist. Serum Tenascin-C levels were studied by sandwich enzyme immunoassay method using Human Tenascin-C Enzyme Immunoassay kit (Range: 15,6-2000 pg/mL, USCN Business Co. Ltd, Wuhan, China). Tenascin-C level was analyzed according to the manufacturer's instructions and expressed as pg/mL. The concentrations of the samples were calculated through calibration curves obtained from study standards with known levels. Blood was collected from the control group in the same way, and their Tenascin-C levels were also measured.

2.3 Statistical Analysis

To obtain statistically significant results, the required minimum number of patients in each group was 64 (alpha:0.05, 1-beta: 0.80). The analysis was made with the program GPower 3.1 (University of Dusseldorf/Germany).

In summarizing the data obtained from the study; Descriptive statistics were presented as mean ± standard deviation or median [minimum-maximum] depending on the distribution of numerical variables. Categorical variables were summarized as numbers and percentages.

In a comparison of two independent groups; Independent Samples T-Test was used when numerical variables showed normal distribution, and Mann Whitney U test was used when numerical variables did not show normal distribution.

Kruskall-Wallis H test was used in the comparisons of more than two independent groups and when the numerical variables did not show normal distribution.

Pearson Chi-Square has used in 2 × 2 tables with expected cells 5 and above, and Fisher's Exact Test was used in tables with expected cells below 5 in the comparison of differences between categorical variables according to groups. Fisher Freeman Halton test was used for R × C tables with expected cells below 5.

Spearman's Rho correlation Coefficient was used in cases where the relations between numerical variables did not show normal distribution.

Statistical analyzes were done with "Jamovi project (2020), Jamovi (Version 1.6.13.0) [Computer Software] (Retrieved from https://www.jamovi.org) and JASP (Version 0.14.1.0) (Retrieved from https://jasp-stats.org) programs. In statistical analysis, the level of significance was considered as 0.05 (p-value).

3. Results

The mean age of the participants was 71.5 ± 11.7 years and 69 of them (53.9%) were female. 62 of the patients (96.9%) arrived at the hospital by ambulance (Table 1).

In this study, the median Tenascin-C value in the patient group (110.2 [64.4 – 777.6]) was significantly higher than the control group (98.6 [11.1 – 176.6]) (p=0.002) (Table 2).

In Table 3, it was determined that there was a significant, linear, same-directional, and low-level relationship between the age of the patients and Tenascin-C levels (p=0.316; r=0.011). There was a significant, linear, reverse, and low-level correlation between symptom duration and tenascin-C level (r=-0.371; p=0.013). On the other hand, there was no significant and linear relationship between Systolic blood pressure (BP), Diastolic BP, NIHSS score, and Glasgow Coma Scale (GCS) levels and Tenascin-C value (Table 4; p>0.05 for each).

In Table 4, the differences between Tenascin-C values, which is according to NIHSS, of the patient group (n=64) were statistically significant (table 4; p=0.05). Tenascin-C value of the patients in the 2nd group was significantly higher than the patients in the 1st group (table 4; p=0.007).

4. Discussion

The diagnosis of ischemic stroke, which is a very important cause of death and disability all over the world, can be determined clinically with high accuracy. Although many studies have been conducted on ischemic stroke, it is a disease whose definitive diagnosis cannot be clarified without confirmation by radiological imaging methods. In ischemic stroke patients, where the most appropriate treatment should be given as soon as possible, the ideal time for diagnosis and initiation of treatment has not yet been reached. Although there are many studies on this issue, a diagnostic biomarker could not be obtained [7]. However, according to a review published in 2021, markers of inflammation, atherogenesis and stress response were evaluated as the most promising prognostic biomarkers among the studies analyzed [8]. In this study, the value of Tenascin-C in the diagnosis of ischemic stroke and its relationship with parameters such as infarct volume and stroke onset time were investigated.

It was determined that there was a significant, linear, same-directional, and low-level relationship between the age of the patients and Tenascin-C levels (p=0.316; r=0.011). It is known that atherothrombotic diseases increase in society with advancing age. Depending on this situation, it is known that the incidence of ischemic stroke increases and has a more fatal course [9]. Rothwell et al. In a population-based study, the incidence of ischemic stroke was 35 per 100.000 aged 35-44 years. However, this rate increases to 952 per 100.000 at the age of 75-84 [10]. Altough, when the literature was examined, no significant relationship was found between Tenascin-C levels and age. We believe that the data of our study will be confirmed by larger-scale studies.

It is known that chronic inflammation is a predisposing factor in atherosclerosis-related complications. However, the factors controlling these immune mechanisms have not been fully determined [11]. Toll-like receptor-4 (TLR-4) is a key signaling molecule in the body's immune response. It plays an important role in atherosclerosis, thanks to its ability to promote inflammation, proteolysis, and thrombosis [12]. Tenascin-C binds to TLR-4 via its fibrinogen-like globe (FBG). Expression of both Tenascin-C and TLR-4 is increased in unstable human atheroma [5]. When viewed in the publications examining the relationship between Tenascin-C and other diseases in which chronic inflammation and atherosclerosis processes are prominent in the etiopathogenesis, Tenascin-C levels of the patients were found to be higher, similar to our study. Gao et al. in their study, Tenascin-C level was found to be significantly higher in patients with coronary artery disease compared to those without. It has also been emphasized that Tenascin-C level can be used as a useful marker to evaluate the severity of atherosclerosis [13]. Kenji et al. showed that Tenascin-C protein production was increased in the coronary plaque region in human atherectomy samples from patients with a history of acute coronary syndrome. Tenascin-C was detected most prominently in the human coronary plaque where macrophage and lymphocyte accumulation is present by immunostaining method [14]. Celik et al. found higher Tenascin-C levels in patients with a history of pulmonary thromboembolism (PTE) compared to the control group. It has also been predicted that it may be useful in determining the severity of PTE [15]. In our study, the median Tenascin-C value in the patient group (110.2 [64.4 – 777.6]) was significantly higher than the control group (98.6 [11.1 – 176.6]) (p=0.002). In this context, when the literature was examined, a few publications examining the relationship between Tenascin-C and ischemic stroke was found. Clancy et al. In his retrospective studies conducted with 336 ischemic stroke patients in 2014, similar to our study, the median Tenascin-C value of the patient group was found to be significantly higher than the control group [5]. In their analysis published in 2020, Okada et al. suggested that TNCs play a potentially important role in pathological changes through neuroinflammation and should be considered as a future therapeutic target in stroke patients [16]. Chelluboina et al. investigated the effects of Tenascin-C on brain damage after ischemic stroke in mice and concluded that Tenascin-C induction mediates ischemic pathogenesis and contributes to brain damage [17]. Considering the studies on the subject in the literature, we think that the main reason for the higher Tenascin-C levels in ischemic stroke patients is chronic inflammation and atherosclerosis processes.

There was a significant, linear, reverse, and low-level relationship between symptom duration and Tenascin-C level (r=-0.371; p=0.013). Suzuki et al. A study examining the relationship between vasospasm developing after subarachnoid hemorrhage and Tenascin-C observed that the measured Tenascin-C levels peaked in a short time and then gradually decreased [18]. Suzuki et al. In another study conducted by them, Tenascin-C levels were measured in the cerebrospinal fluid after subarachnoid hemorrhage, and it was observed that it reached the highest levels in the first three days and then followed a decreasing course [19]. Led by these data, our results showed that Tenascin-C levels can be used to predict the starting time of symptoms in ischemic strokes.

The differences between Tenascin-C values of the patient group (n=64) according to the NIHSS were statistically significant (p=0.05). In light of these data, the Tenascin-C value of the patients in the NIHSS Group 2 (7-15 points) was significantly higher than the patients in Group 1 (0-6 points) (p=0.007). Ulker et al. showed in their study that initial NIHSS scores are very useful in determining prognosis in the early period. 90% of patients with NIHSS scores between 0 and 6 had a good or very good recovery after 2 weeks. On the other hand, the recovery rate in patients with an NIHSS score of 16 and above has been calculated as approximately 10% [20]. In the

study by Wouters et al, baseline NIHSS was assessed as a strong predictor of functional outcome 90 days after stroke in acute ischemic stroke patients [21]. In our study, it was determined that Tenascin-C could be effective in predicting the severity of the disease. When we analyze our data, we think that Tenascin-C was not statistically significantly higher in Group 3 (16-42 points) due to the low number of patients in this group. Tenascin-C should be examined on this subject with larger studies in the future.

5. Limitations

The study's weakness is that it was carried out in a single center with a minimum number of participants. In addition, another limitation is that the Tenascin-C parameter was not measured in the days after the application during the follow-up of the participants.

6. Conclusion

Based on the findings of our study, Tenascin-C can be used as a supporting biomarker in determining the duration and severity of the ischemic stroke, and may shorten the time of diagnosis and initiation of treatment in the emergency room, thanks to more comprehensive studies to be conducted in the future.

We declare no competing interests.

There was no funding source for this study.

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TABLES

Table 1. Sociodemographic characteristics of the patient and control groups

Group	
Patient	64 (50)
Control	64 (50)
Age	71.5 ± 11.7
Gender	
Male	59 (46.1)
Female	69 (53.9)
Way of arrival	
By ambulance	62 (96.9)
Outpatient	2 (3.1)

Descriptive statistics were presented as mean ± standard deviation or median [minimum-maximum] depending on the distribution for numerical variables and also they were presented as number (%) for categorical variables.

Table 2. Comparison of some demographic and laboratory results according to patient and control groups

	Patient (n=64)	**Control (n=64)**	**p value**
Age	72.3 ± 11.4	70.6 ± 12	0.407‡
Gender			
Male	30 (46.9)	29 (45.3)	0.859*
Female	34 (53.1)	35 (54.7)	
Tenascin-C value	110.2 [64.4 – 777.6]	98.6 [11.1 – 176.6]	**0.002†**

Descriptive statistics were presented as mean ± standard deviation or median [minimum-maximum] depending on the distribution for numerical variables and also they were presented as number (%) for categorical variables

*. Mann-Whitney U test was used.

†. PearsonChi-Square, Fisher'sExact or FisherFreemanHalton tests were used.

‡. Independent samples T-Test was used.

Table 3. Correlation of tenascin values with demographic and clinical characteristics of the patient group (n=64)

	Tenascin-C value	
	r	**p**
Age	0.316	**0.011**
Symptom duration	-0.371	**0.013**
Systolic BP (mmHg)	-0.004	0.976
Diastolic BP (mmHg)	0.062	0.626
NIHSS score	0.227	0.072
GCS	-0.240	0.056

Spearman's rho correlation coefficient was used.

Abbreviations: BP, Blood Pressure; GCS, Glasgow Coma Scale; NIHSS, National Institutes of Health Stroke Scale

Table 4. Comparison of Tenascin-C values of the patient group (n=64) according to the National Institutes of Health Stroke Scale (NIHSS)

	Tenascin-C value	**p***
NIHSS score		
1. group; NIHSS, 0-6 point, mild-moderate	94.63 [64.39 – 333.42]	**0.005**
2. group; NIHSS, 7-15 point, moderate-severe	163.88 [68.37 – 777.57]	
3. group; NIHSS, 16-42 point, severe-very severe	104.98 [72.35 – 212.44]	

Descriptive statistics were presented as median [minimum-maximum] depending on the distribution of numerical variables

*. Kruskal-Wallis H test was used.

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Emergency use of endotracheal tube as a substitute for chest tube in pneumothorax management: a case report

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Introduction

Traumatic pneumothorax and hemothorax are life-threatening conditions that can lead to high morbidity and mortality if not promptly recognized and treated (Adal et al., 2024). Thoracic trauma accounts for approximately 25% of trauma-related deaths and is especially fatal when uncontrolled air or blood accumulation leads to tension physiology (Shah et al., 2022). In such cases, the standard treatment is tube thoracostomy, which involves evacuating air and blood from the pleural cavity (Shah et al., 2022).

However, in resource-limited environments or in cases of equipment shortages, access to a standard chest tube may not always be possible. Indeed, in economically disadvantaged regions, mountainous terrains, or during mass casualty events and natural disasters, standard chest tubes may not always be readily available (Adal et al., 2024; Odion-Obomhense & Udoka, 2022). Under these circumstances, alternative pleural drainage methods may be required to save lives. The literature has documented the use of wide-lumen catheters, Foley catheters, intravenous lines, and even endotracheal tubes (ETTs) as temporary substitutes for chest tubes (Keskin & Kuran, 2024; Newton et al., 2021; Schober et al., 2025).

This case report discusses a temporary pleural drainage procedure using an ETT in an emergency department where standard chest tubes were unavailable due to resource constraints.

Case

A 59-year-old male patient was brought to the emergency department after falling from a height of approximately six meters. On initial evaluation, he was nearly unconscious, experiencing significant respiratory distress, with an oxygen saturation of 85%.

Physical Examination

A wide laceration was noted on the scalp, and bilateral periorbital ecchymosis (“raccoon eyes”) was observed. Breath sounds were absent on the right hemithorax.

Radiological Findings

A computed tomography (CT) scan revealed a large right-sided pneumothorax and hemothorax, along with mediastinal shift to the left. Partial lung collapse and pulmonary contusion were also present (Figure 1).

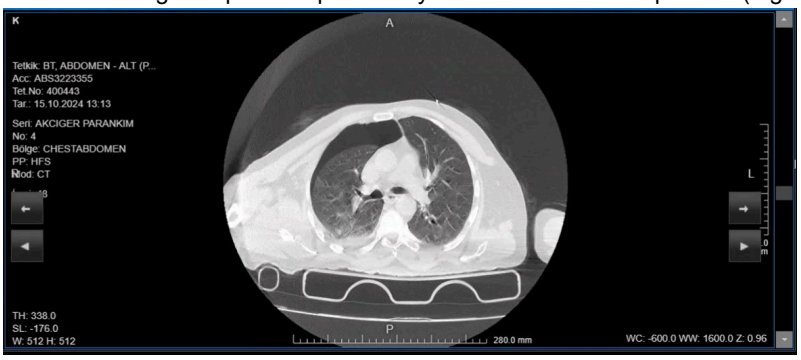

Figure 1: Axial computed tomography (CT) scan demonstrating a significant right-sided pneumothorax and hemothorax. Mediastinal shift to the left is evident, accompanied by partial lung collapse and pulmonary contusion.

In the coronal CT view, a 7.5-mm endotracheal tube was visualized within the pleural cavity. The tube appeared to provide temporary drainage for the accumulated air and blood in the right hemithorax (Figure 2).

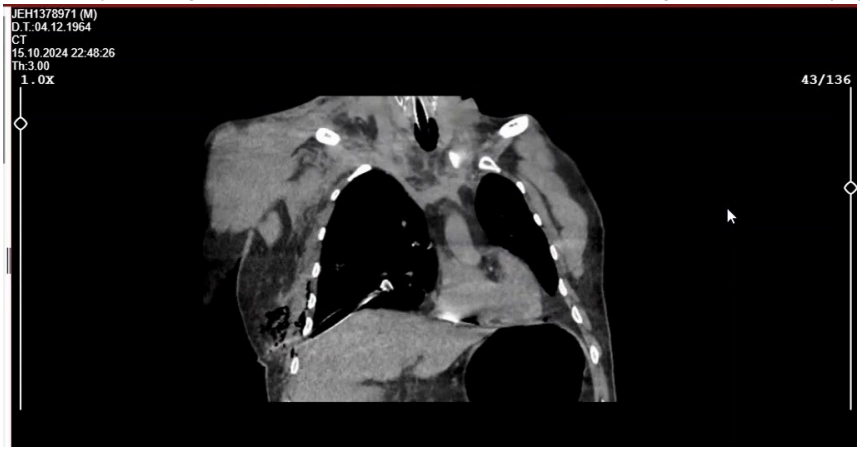

Figure 2: Coronal computed tomography (CT) scan showing the placement of a 7.5-mm endotracheal tube as a temporary chest tube substitute within the right pleural cavity. Increased fluid accumulation (hemothorax) and a large pneumothorax are visible in the

right hemithorax.

Laboratory Results

White blood cell (WBC) count was elevated (23.08 × 10^3/μL), likely indicating a stress response. Arterial blood gas analysis showed metabolic acidosis with a pH of 7.231.

Intervention and Clinical Course

Due to the unavailability of a standard chest tube in the emergency department, a 7.5-mm endotracheal tube—typically used for airway management—was inserted into the right pleural cavity via the thoracostomy opening under sterile conditions. The intervention led to immediate respiratory improvement, with oxygen saturation rising from 85% to 99%. Recognizing the limitations of this approach for long-term drainage, the patient was transferred to a tertiary trauma care center after initial stabilization. Approximately three hours after arrival, the ETT became obstructed by clots and debris. It was subsequently replaced with a standard chest tube at the referral center, and definitive treatment was continued in the appropriate clinical setting (Newton et al., 2021; Odion-Obomhense & Udoka, 2022; Schober et al., 2025).

Ethical Statement

Written informed consent was obtained from the patient's legal guardian for the publication of this case report and its accompanying images.

Discussion

This case highlights the life-saving potential of creative adaptability in emergency medical practice when faced with equipment shortages. The use of an endotracheal tube (ETT) as a chest tube is highly uncommon, with only a limited number of such cases reported in the literature. Nevertheless, in situations where immediate pleural drainage is critical, this approach may be considered as a temporary solution (Schober et al., 2025; Newton et al., 2021).

In a case reported by Newton and colleagues, an ETT was successfully used during prehospital trauma care to prevent re-occlusion of a thoracostomy site (Newton et al., 2021). Similarly, a neonatal tension pneumothorax case was managed with an endotracheal tube when an appropriate-sized chest tube was unavailable, demonstrating positive clinical outcomes (Odion-Obomhense & Udoka, 2022). These cases support the approach used in our report, even though such interventions remain rare and unconventional.

Using a non-standard device like an ETT in place of a chest tube is clearly not ideal and carries inherent risks. The narrow internal lumen of the ETT can quickly become obstructed by clots in cases of traumatic hemopneumothorax, rendering the drainage ineffective. In our case, the ETT became blocked after approximately three hours and had to be replaced with a standard chest tube to restore adequate drainage.

Additionally, if not inserted under sterile conditions, there is a risk of infection. The lack of a one-way valve mechanism in ETTs also limits their ability to fully control air leaks. Therefore, this method should only be considered as a temporary, life-saving intervention, to be replaced with a standard chest tube as soon as it becomes available (Keskin & Kuran, 2024; Shah et al., 2022).

Emergency preparedness is crucial, especially in non-trauma centers and peripheral hospitals. These facilities should always maintain a stock of essential invasive procedure kits, including chest tubes. Emergency physicians should also receive training in alternative thoracic decompression techniques. For instance, current trauma guidelines recommend finger thoracostomy if needle decompression fails, followed by standard chest tube placement when feasible (Shah et al., 2022).

Finally, the development of standardized protocols and scenario-based planning for such exceptional situations may help improve outcomes. Further studies comparing these alternative interventions to conventional chest tube thoracostomy could provide evidence to guide future emergency practice.

Conclusion

Improvised interventions can sometimes be lifesaving in emergency department settings when standard equipment is not available. In this case, temporary pleural drainage using an endotracheal tube provided sufficient stabilization to enable the patient's safe transfer to a tertiary care center.

However, the limitations of this approach were clearly observed. An endotracheal tube cannot fully replace a properly sized chest tube and should only be considered a short-term bridging solution. This case emphasizes the importance of resource availability and trained personnel in trauma management.

It also underlines the need for non-trauma hospitals to maintain essential invasive procedure kits and to develop training programs and protocols on alternative techniques. Such preparedness can be crucial in saving lives during similar critical emergencies (Adal et al., 2024; Shah et al., 2022).

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Acute coronary syndrome induced by methanol intoxication

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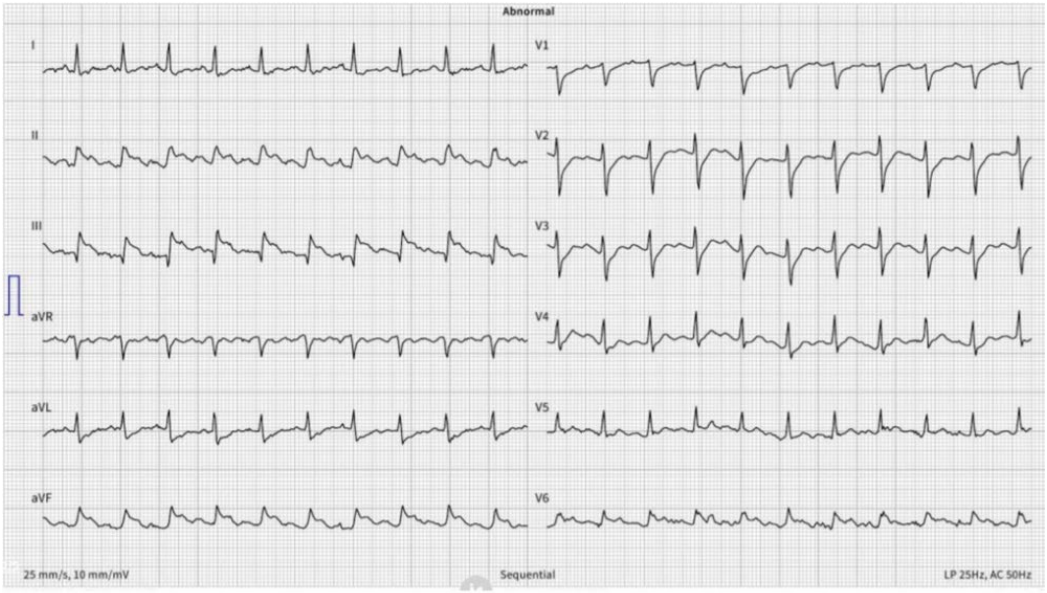
Introduction

Methyl alcohol is a chemical substance commonly used as a raw material in various industrial processes. While methanol itself is not directly toxic, its metabolites can exert harmful effects upon biotransformation in the human body. Methanol intoxication can lead to serious complications including brain injury, visual disturbances (such as diplopia, blurred vision, reduced visual acuity, visual field narrowing, photophobia, and blindness), cardiovascular instability, seizures, metabolic acidosis, and death.

Case

A 73-year-old male presented to the emergency department with complaints of dizziness, shortness of breath, chest tightness, and visual disturbances following the consumption of approximately two glasses of homemade rakı the previous evening. On admission, the patient was alert, oriented, and cooperative. Vital signs were as follows: blood pressure 165/100 mmHg, pulse 90 bpm, temperature 36°C, respiratory rate 22 breaths/min. Auscultation revealed normal heart and breath sounds.

Arterial blood gas analysis revealed a pH of 7.07, PaO□ 91 mmHg, PaCO□ 31 mmHg, HCO□⁻ 9 mmol/L, and oxygen saturation of 86.1%. Laboratory results were as follows: Hb 15 g/dL, WBC 7,000/μL, platelets 201,000/μL, INR 0.96, creatinine 1.56 mg/dL, potassium 5.4 mEq/L, sodium 136 mEq/L, calcium 9 mg/dL, glucose 100 mg/dL, troponin >25,000 ng/L, and blood alcohol level <0 mg/dL.



Electrocardiogram (ECG) showed ST-segment elevation in inferior leads (DII, DIII, aVF) and ST-segment depression in lateral leads (DI, aVL) (Figure 1). The patient was evaluated by cardiology with a preliminary diagnosis of acute coronary syndrome (ACS), and primary percutaneous coronary intervention (PCI) was recommended. During PCI, plaques were identified in the left anterior descending (LAD) and circumflex arteries. The patient was managed medically with aspirin 100 mg, clopidogrel 75 mg, and enoxaparin 0.6 mL, and was referred for outpatient cardiology follow-up

Ophthalmologic consultation was obtained due to visual complaints, but no abnormalities were found. Cranial computed tomography (CT) also revealed no pathology. Given the presence of metabolic acidosis, the nephrology team was consulted, and emergency hemodialysis was initiated. As acidosis persisted post-dialysis, intravenous ethanol therapy was administered. The patient was subsequently admitted to the intensive care unit for further monitoring and treatment.

Conclusion:

Toxic exposures are among the primary presentations in emergency medicine. Regardless of the suspected diagnosis, an ECG should be performed on all patients presenting to the emergency department. In addition to well-known complications, clinicians should be aware of rare and atypical manifestations of intoxication. ECG remains a critical tool in the diagnosis and management of acute coronary syndromes. In this case, STEMI was detected on ECG in the absence of chest pain following methanol intoxication, illustrating how toxic agents may sensitize the myocardium to catecholamine-induced dysrhythmias.

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Traumatic Aort Dissection

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Introduction

High-energy trauma, particularly from motor vehicle accidents, poses significant risks to elderly patients due to age-related physiological changes. These injuries often involve multiple organ systems, including the spine, abdomen, and thoracic region, leading to complex clinical presentations. This case presents a 77-year-old female patient who sustained severe injuries from a high-energy traffic accident, resulting in spinal fractures, abdominal trauma, and ultimately cardiac arrest. It underscores the challenges of managing elderly trauma patients in critical conditions.

Case Report

A 77-year-old female patient was brought to the emergency department following a high-energy traffic accident. The Glasgow Coma Scale (GCS) score was recorded as 14 (E3, M6, V5). Vital signs were stable, and the patient's general condition was evaluated as moderately poor.

On physical examination, widespread abrasions were noted on the scalp and face, along with epistaxis (nasal bleeding). Tenderness was detected upon palpation in both the right and left upper abdominal quadrants. Widespread abrasions were also observed on both lower extremities. No pathological sounds were detected upon auscultation of the lungs. However, there was widespread tenderness on the thoracic wall and in the thoracolumbar spinal region.

Computed tomography (CT) revealed a hematoma near the T10 vertebral fracture and a flap appearance in the aorta. Based on these findings, emergency cardiovascular surgery and neurosurgery consultations were requested.The patient suffered a cardiac arrest during follow-up and was pronounced dead in the emergency department despite resuscitation efforts.

Conclusion

This case emphasizes the critical challenges in managing high-energy trauma in elderly patients. Age-related physiological changes increase vulnerability to severe outcomes, including multi-system injuries and cardiac arrest. Prompt recognition, rapid intervention, and multidisciplinary care are essential for improving survival chances. This case highlights the need for heightened awareness and tailored management strategies when treating elderly trauma patients, underscoring the complexity and urgency of care in this demographic.

KEYWORDS: Aortic Dissection , Traumatic Injury

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The Effect of Door-Lytic and Door-Needle Times on Survival in Acute Ischemic Stroke

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Introductıon

Intravenous (IV) thrombolysis and mechanical thrombectomy, the primary treatment modalities used in acute ischemic stroke, are time-dependent therapies. IV thrombolytic therapy is performed with alteplase, a recombinant tissue plasminogen activator (rtPA) licensed in Turkey in 2006. The US Food and Drug Administration (FDA) approved IV rtPA for stroke treatment within 3 hours of symptom onset in 1996 (1). A meta-analysis published in 2004 suggests that IV thrombolytic therapy should be given within the first 4.5 hours of symptom onset (2). At the same time, mechanical thrombectomy is one of the treatments with proven efficacy and safety when performed within the first 6 hours of acute ischemic stroke (3). In the DAWN and DEFUSE 3 studies, it was shown that the duration of mechanical thrombectomy could be extended up to 16 and 24 hours (4,5). When the literature is examined, it is seen that the duration of reperfusion in acute stroke is a matter of debate and studies on this subject are being conducted continuously. Our aim was to investigate the effect of IV thrombolysis and mechanical thrombectomy treatment times on survival in acute stroke patients admitted to a tertiary hospital with a stroke center.

Material And Methods

Study Design

The study was a retrospective review of patients diagnosed with acute ischemic stroke who presented to the emergency department of a tertiary care hospital between January 1, 2024 and December 31, 2024. Our institution receives more than 140000 emergency patient visits annually and hosts both neurology and emergency medicine residency programs with a comprehensive stroke center. Acute ischemic stroke accounted for 0.7% of ED visits, and approximately 11.8% of ischemic strokes received acute thrombolytic therapy. The current study consisted of consecutive patients presenting over a one-year period with stroke symptoms and receiving thrombolytic therapy.

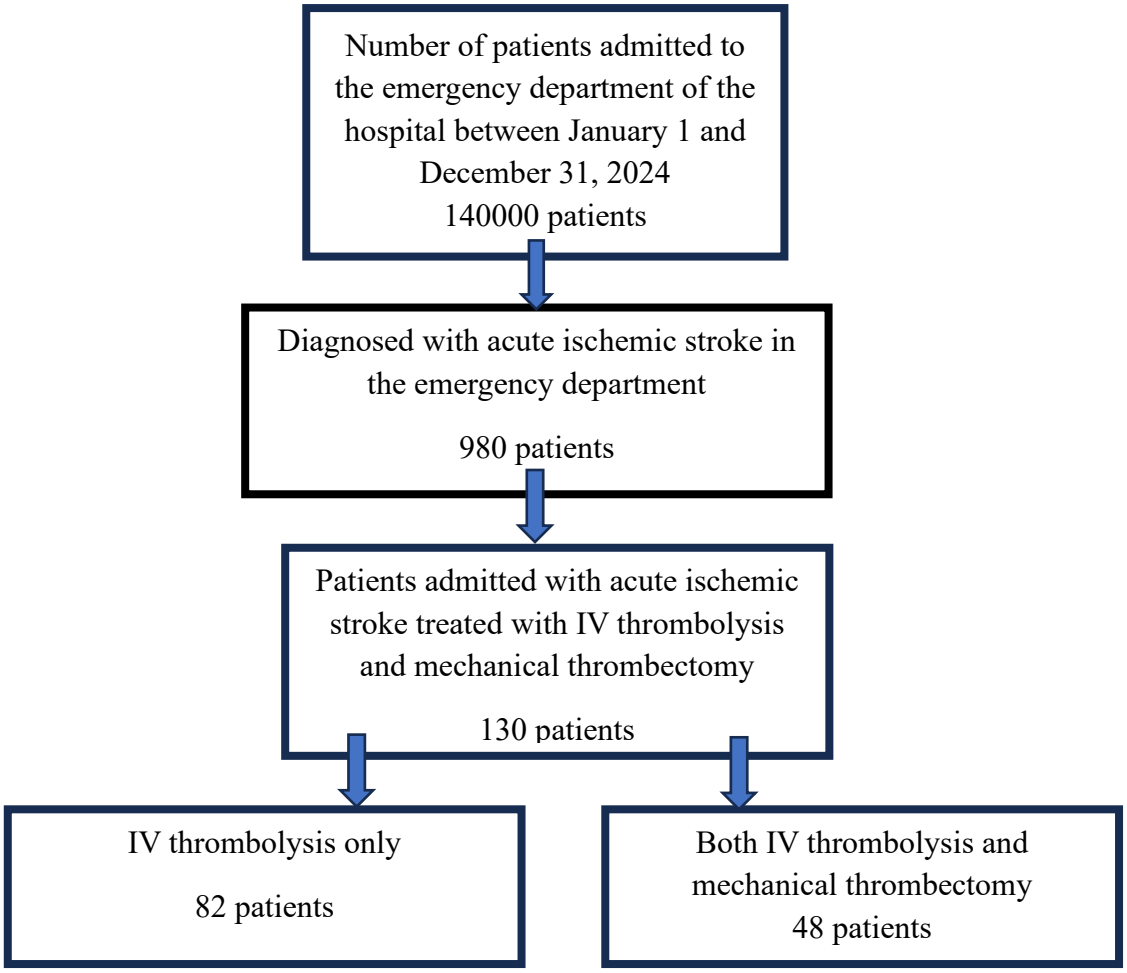

Figure 1. Study Flow Chart

Study Population and Variables

Data on consecutive patients admitted to the emergency department and diagnosed with acute stroke were obtained from emergency department patient files using the computer-based Hospital Information Management System (HIMS) program. Patients who met the eligibility criteria for intravenous thrombolysis with rt-PA according to the local hospital protocol and admitted to the hospital within 4.5 hours of symptom onset were included in the study. Patients who presented to the emergency department more than 4.5 hours after the onset of stroke symptoms, were diagnosed with intracerebral hemorrhage or brain tumor, had incomplete clinical and investigative data, and were younger than 18 years of age were excluded.

In the process starting after admission, the exact time of the first onset of stroke symptoms, the way the patient arrived at the emergency department, the time of arrival at the emergency department, the place of arrival and the location are recorded by the medical staff. time of the first onset of stroke-related symptoms was identified and recorded as the "onset time" by the patients themselves or family members. For people who experienced symptoms while sleeping, symptom onset was determined as the last time they were seen healthy. "Time of arrival in the emergency department" was defined as the time when the patient's registration in the emergency department was completed. "Door-to-lytic time" was defined as the time interval between arrival in the emergency department and initiation of thrombolytic therapy. "Door-needle time" was defined as the time interval between arrival at the emergency department and the start of mechanical thrombectomy treatment. "Symptom-lytic time" was defined as the interval between the onset of symptoms and the start of IV thrombolysis, and "symptom-needle time" was defined as the interval between the onset of symptoms and the start of mechanical thrombectomy. All time measurements were expressed in minutes.

In addition to demographic characteristics, admission symptoms and time of onset, parameters of laboratory and imaging tests, physical examination findings, treatments given in the emergency department and whether complications developed, in-hospital and discharge outcome, whether there was readmission in the 90-day period, admission and discharge National Institutes of Health Stroke Scale (NIHSS) score and modified Rankin Score (mRS) were recorded.

Emergency Service Management

Emergency department acute stroke management is a dynamic process that starts after emergency medical service (EMS) personnel receive a call from the command and control center. Healthcare professionals assess the patient's basic neurological status according to FAST (Face, Arms, Speech and Time) (6) or Los Angeles Motor Scale (LAMS) and report the patient to the stroke center (7). If patients present directly as outpatients, they are taken to the critical patient care area of the emergency department by the

healthcare professional in charge after evaluation in the triage area. After admission, the patient is evaluated by the emergency medicine doctor, neurologist, radiologist and emergency medicine nurse on duty who provide 24/7 service in our institution. The patient, who is taken to the security circle from the moment of admission, reaches the doctor within the first 10 minutes (the first evaluation performed by both the emergency medicine doctor and the neurologist, including the last the patient was well, the suitability of IV thrombolysis and the assessment of stroke severity). Blood is drawn for laboratory analysis and a CT scan of the brain is performed within 25 minutes and interpreted within 45 minutes. If the patient meets the appropriate criteria for thrombolysis, a weight-based calculation is made and administered to the patient. It is aimed to administer thrombolytic therapy in the first 60 minutes after the patient is admitted to the emergency department. At the same time, it is evaluated by interventional radiology in terms of medical thrombectomy and processed if necessary.

Statistical Method

All analyses were performed in Jamovi v.1.6 statistical software (The Jamovi Project (2021) Computer Software, version 1.6. Sydney, Australia). Categorical data are expressed as frequency (n) and percentage. Normally distributed continuous variable data were expressed as mean plus standard deviation (SD) and non-normally distributed data were expressed as median and interquartile range (IQR). Normality of distribution was assessed using the Shapiro-Wilk test. Two-group comparisons were made using Student's t-test or Mann-Whitney U test, depending on whether the data were normally distributed. P values <0.05 were considered statistically significant.

Results

The study included 130 patients who presented to the emergency department with acute ischemic stroke symptoms and received IV thrombolysis or mechanical thrombectomy treatment in 2024. Of these, 50.8% (n=66) were women. 53.1% of the patients were brought to the emergency department by 112. The most common presenting symptom was limb weakness (85.4%). When the patients' medical history was analyzed, hypertension was the most common symptom with 79.2%. When CT findings were analyzed, one or more of the edema findings such as hypodensity in the brain parenchyma, disappearance of gray-white matter, and obliteration of sulci were detected in 77.7% of the patients. When CT angiography findings were analyzed, 49.2% had major vessel occlusion that could be treated with mechanical thrombectomy. MRI findings showed that 49.2% of the cases occurred in the anterior and middle cerebral artery (ACA and MCA) irrigation area. Regarding the etiology, 60% of the patients were atherosclerotic (carotid stenosis) and 36.2% were cardioembolic (atrial fibrillation). The median time between presentation and CT scan was 14.0 (IQR 11.0-22.0) minutes.

Only IV rtPA was administered to 63.1% of the patients, while 36.9% received both IV rtPA and mechanical thrombectomy. As supportive therapy, 63.8% of patients received antihypertensives, 5.4% received anti-edema, and 0.8% received anticoagulants.

When the outcomes of the treated patients in the emergency department were analyzed, 65.4% were hospitalized in the stroke unit and 32.3% in the intensive care unit (ICU). No exitus was observed in the emergency department outcome. 7.0% of the patients were intubated in the emergency department and 4.7% in the interventional procedure room during thrombectomy. Treatment-related complications occurred in 8.5% of patients. The most common complication was cerebral hemorrhage (5.4%). The mean number of days of hospitalization was 12.5± 13.5, intensive care unit 4.2± 9.5, and mechanical ventilator 2.5± 6.6.

When hospital outcome was analyzed, mortality rate was 13.1%. Of the discharged patients, 71.5% were readmitted to the hospital within 90 days and 65.4% were readmitted for routine follow-up. The median NIHSS value at admission was 12.0 (IQR 5.0-18.0), while the median NIHSS value at discharge was 5.0 (IQR 0.7-12.0). The discharge mRS value was 3.0 (IQR 0.0-5.0).

The median symptom-door duration for treated patients was 65.5 minutes (IQR 43.0-99.3). For patients treated with IV rtPA, the median door-to-needle time was 87.5 (IQR 67.0-120.0) minutes and the median symptom-to-needle time was 165 (IQR 135-212) minutes. In patients who underwent mechanical thrombectomy, the median door-needle time was 91.0 (IQR 80.8-130.0) minutes and the median symptom-needle time was 164 (IQR 135-222) minutes. All investigated durations did not statistically affect mortality in hospital outcome (p>0.05).

Table 1. Demographic data and basic characteristics of the patients

Characteristics, n = 130	Value
Gender	
Male, n (%)	64 (49.2)
Female, n (%)	66 (50.8)
Mode of Arrival to the Emergency Room	
112, n (%)	69 (53.1)
Referral from an external center, n (%)	39 (30.0)
Private vehicle, n (%)	22 (16.9)
Application Complaint	
Limb weakness, n (%)	111 (85.4)
Speech disorder, n (%)	97 (74.6)
Facial asymmetry, n (%)	85 (65.4)
Impaired consciousness, n (%)	41 (31.5)

Application Vital Values	
SDB, mean± sd (mmHg)	166± 28.8
DBP, median (IQR) (mmHg)	83.5 (78.5-100.0)
Pulse, median (IQR) (beats per minute)	76.5 (67.0-88.8)
Saturation, median (IQR) (%)	96.0 (94.0-97.0)
Comorbidities	
Hypertension, n (%)	103 (79.3)
Atrial fibrillation, n (%)	46 (35.4)
Diabetes mellitus, n (%)	41 (31.5)
Coronary artery disease, n (%)	39 (30.0)
Cerebrovascular disease, n (%)	23 (17.7)
Malignancy, n (%)	7 (5.4)
Diagnostic Findings	
CT (edema), n (%)	101 (77.7)
CT (dense MCA), n (%)	25 (19.2)
CTA (large vessel occlusion), n (%)	64 (49.2)
MR (ACA, MCA), n (%)	64 (49.2)
MR (PCA), n (%)	14 (10.8)
MR (Lacunar), n (%)	40 (30.8)
Etiology	
Atherosclerotic, n (%)	78 (60.0)
Cardioembolic, n (%)	47 (36.2)
Other (hypotension, anemia), n (%)	5 (3.8)
Admission-CT time, median (IQR) (minutes)	14.0 (11.0-22.0)
Treatment	
IV rtPA, n (%)	82 (63.1)
IV rtPA + mechanical thrombectomy, n (%)	48 (36.9)
Emergency Department Outcome	
Ward hospitalization, n (%)	3 (2.3)
Stroke unit hospitalization, n (%)	85 (65.4)
ICU hospitalization, n (%)	42 (32.3)
Intubation Location	
Emergency department, n (%)	9 (7.0)
Interventional hall, n (%)	6 (4.7)
Stroke unit, n (%)	2 (1.6)
ICU, n (%)	8 (6.2)
Follow-up Time in MV, mean± sd (days)	2.49± 8.61
Length of Stay, mean± sd (days)	12.5± 13.5
ICU Length of ICU Stay, mean± sd (days)	4.21± 9.49
Hospital Outcome, Exitus, n (%)	17 (13.1)
Admission NIHSS, median (IQR)	12.0 (5.0-18.0)
Discharge NIHSS, median (IQR)	5.0 (0.75-12.0)
Discharge mRS, median (IQR)	3.0 (0.0-5.0)
Symptom-Door Time, median (IQR)	65.5 (43.0-99.3)
Door-Litik Duration, median (IQR)	87.5 (67.0-120.0)
Symptom-Lytic Duration, median (IQR)	165.0 (135.0-212.0)
Door-to-Needle Time, median (IQR)	91.0 (80.8-130.0)
Symptom-Injection Duration, median (IQR)	164.0 (135.0-222.0)

IQR: Interquartile Range (25p, 75p), **sd:** standard deviation, **SDB:** Systolic Blood Pressure, **DBP:** Diastolic Blood Pressure, **CT:** Computed tomography, **CTA:** Computed tomography angiography, **MR:** Magnetic Resonance, **ACA:** Anterior cerebral artery, **MCA:** Middle cerebral artery, **PCA:** Posterior cerebral artery, **IV rtPA:** Intravenous recombinant tissue plasminogen activator, **ICU:** Intensive care unit, **MV:** Mechanical ventilator, **NIHSS:** National Institute of Health Stroke Scale, **mRS:** Modified Rankin Scale

Discussion

Acute ischemic stroke is the most common cause of death after coronary heart disease. It is also the leading cause of disability. Stroke patients' prolonged hospitalization, rehabilitation procedures and loss of labor force are a major economic burden for the society. Reducing this economic burden is possible with early recognition of acute stroke symptoms and early access of stroke patients to treatment. In our study, 11.8% of patients admitted with acute stroke could be treated. In another one-year retrospective study conducted in Italy, this rate was found to be 30.1% (8). When both studies are compared, it is thought that awareness about

acute stroke is still insufficient in our country.

In our study, hypertension was found to be the most common disease in the patient's history and muscle weakness was the most common presenting symptom. In another study conducted in the same region, hypertension was found to be present in 70% of acute stroke patients who underwent revascularization treatment and motor deficits were found in 73.8% of the applicants, which was correlated with our study (9).

The time between presentation to the emergency department and CT scanning (door-to-CT time) should be 25 minutes on average. In a study, the door-to-CT time, which was previously 20 minutes on average, was found to be less than 10 minutes with the transportation of the CT device to the emergency department (10).

In our study, stroke-related mortality rate was found to be 13.1%. In a large study including 30947 patients, the 30-day mortality rate of patients admitted to a stroke center was 10.1%, whereas it was 12.5% in a hospital without a stroke center (11). Although the hospital where our study was conducted was also a stroke center, the mortality rate was found to be higher compared to this study. We can mention that diagnosis, treatment and complication management of stroke patients should be investigated in detail.

In the emergency department where we conducted this study, the CT device was located in the emergency department and the median time was 14 minutes. We think that having the CT device in the emergency room may decrease the duration of diagnosis and treatment.

Data from IV rtPA clinical trials show that the therapeutic benefit of rtPA is greatest when given very early in ischemic stroke and decreases during the first 4.5 hours after onset (12, 13). In our study, it was found that door-litic and door-needle times did not affect mortality. We believe that a study on morbidity may be significant. In a study to be planned in a larger population, the effect of door-lytics and door-needle times on mortality and morbidity should be investigated.

Conclusıon

It is thought that door-litic and door-needle times have no effect on mortality. The population should be expanded and studies on morbidity should be conducted in this regard.

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Infected Duplıcatıon Cyst

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Introductıon

Duplication cysts occurring in the intestine are one of the rare pathological structures of the digestive system. These cysts are generally cysts that develop in the intestinal wall, containing fluid or semi-fluid substances and sometimes showing a two-chambered structure. Most duplication cysts are congenital and may be asymptomatic, often remaining undetected in early childhood. However, in some cases, these cysts can become infected and lead to serious complications.

Infected intestinal duplication cysts occur when these cysts become contaminated by bacterial, viral, or fungal infections. Depending on the location of the cyst, infection can lead to symptoms such as abdominal pain, fever, swelling, diarrhea, and sometimes abscess formation. Since an infected cyst can lead to serious complications such as perforation and peritonitis, it is crucial to make a quick and accurate diagnosis.

The infection of intestinal duplication cysts generally develops due to imbalances in the intestinal flora, inflammatory bowel diseases, surgical interventions, or trauma. In the differential diagnosis of such cases, it is essential to determine whether the duplication cyst is infected and distinguish it from other acute abdominal diseases, especially appendicitis, diverticulitis, and bowel perforation.

This case report will discuss the clinical findings, diagnostic process, and treatment course of a patient diagnosed with an infected intestinal duplication cyst.

Case

A 55-year-old male patient presented to the emergency department with complaints of abdominal pain, nausea, and vomiting. Glasgow Coma Score (GCS): 15. Blood pressure: 140/80 mmHg, Pulse: 86 bpm, Respiratory rate: 18 breaths/min, Axillary temperature: 36.2ºC. Fingerstick blood glucose: 146 mg/dL. ECG: Sinus rhythm.

On physical examination, widespread tenderness was noted in the abdomen. There was no defense or rebound tenderness. No organomegaly was detected. No palpable mass. No dullness on percussion.

Medical history: Diabetes, hypertension, thyroidectomy, appendectomy, cholecystectomy. Medications: Levothyroxine, Norvasc, Glucofen.

Laboratory findings: pH: 7.408, pCO2: 42.8, HCO3: 26.4, Lactate: 2.46, Glucose: 90, CRP: >350, WBC: 19, Procalcitonin: Negative, Titers: Negative.

After symptomatic treatment, there was no relief, and as infectious markers remained high, further investigation was planned. Contrast-enhanced abdominal tomography was performed. The CT scan revealed "A tubular structure approximately 120 mm in length with a blind ending at the umbilical-supraumbilical level, related to ileal loops, showing a fluid-filled, distended appearance with surrounding inflamed and dirty-looking fatty tissue (possible infected duplication cyst?)."

The patient’s oral intake was stopped. IV fluid replacement and empirical IV antibiotic therapy were initiated. A general surgery consult was requested, and the patient was admitted to the general surgery intensive care unit. During follow-up, the patient did not require surgery, and after antibiotic therapy, the infectious markers decreased, with normal gas and stool passage. The patient was discharged with complete recovery.

Conclusıon

Intestinal duplication cysts are rare and typically asymptomatic, but they can lead to serious complications if infected. In this case, a 55-year-old male patient presented with abdominal pain and signs of infection, and after further investigations, an infected intestinal duplication cyst was diagnosed. CT findings indicated that the duplication cyst had transformed into an infected structure. The patient was initially treated symptomatically, but further investigations and treatment were planned due to the persistence of symptoms and high infectious markers.

Empirical antibiotic therapy and IV fluid replacement were initiated, and after a general surgery consultation, the patient's condition stabilized. The patient's infectious markers decreased, and bowel function normalized. Consequently, the patient was discharged with no need for surgery.

This case once again highlights that intestinal duplication cysts can become infected, and early diagnosis and appropriate treatment can prevent complications. Furthermore, it emphasizes the importance of a careful approach in the differential diagnosis of acute abdominal conditions.

Keywords: Intestinal duplication cyst, acute abdominal diseases, empirical antibiotic therapy.

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ST Segment Alteration due to cocaine use: A case report

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Introduction

From a public health perspective, drug addiction is becoming increasingly prevalent around the world and in our country. Cocaine acts as a sympathomimetic agent by blocking the reuptake of norepinephrine and dopamine in presynaptic neurons. This can lead to an increase in myocardial oxygen demand, constriction of coronary arteries, and myocardial ischemia or infarction. Cocaine can cause infective endocarditis, dilated cardiomyopathy, fatal arrhythmias, and aortic dissection. Cocaine has been shown to be a potential cause of acute arterial thrombosis in persons without cardiovascular risk factors. In addition to small-diameter coronary and cerebral vessels, thrombosis has been demonstrated in larger-diameter popliteal, femoral, and iliac arteries. Patients presenting to the emergency department with chest pain should be screened for substance abuse.

Case Presentation

A 20-year-old male patient had a history of antiepileptic drug use until the age of 2 years, was followed up for epilepsy until the age of 10 years, and had no epileptic seizures since then. The patient was admitted to our emergency department by his family with complaints of recent chest pain, epigastric pain, and slowing of mental movements. The patient described cramping pain in the epigastric region radiating to the chest, left arm, and back, and stabbing pain. He had no history of exertional dyspnea, angina, shortness of breath, palpitations, nausea, vomiting, abdominal pain, and no known systemic disease, drug allergy, or smoking. His past medical history and family history were unremarkable. On physical examination, he was in good general condition, conscious, coherent and oriented. Temperature was 36.5°C, peak heart rate was 80/min and blood pressure was 120/70 mmHg. On cardiologic examination, heart sounds were rhythmic, S1, S2 were normal, and there were no additional sounds or murmurs. Other systemic findings were normal. Electrocardiographic examination revealed nonspecific ST segment elevations in the V1-V5 leads. Extensive non-specific ST depression was noted in limb leads DII, DIII, and AVF. Complete blood count revealed WBC: 12,000 /mm³, Hgb: 14.7 g/dL, biochemical tests were normal; CK-MB: 0.48 (0-3.6 mcg/L), Troponin-I: <2.50 (0-47 ng/L), CRP: 0.17 mg/L.

The patient was observed and cardiac enzymes were monitored. Echocardiography showed no wall motion abnormality, normal right cavities, no pericardial effusion, normal ascending aorta, increased trabeculation in the apical right ventricle, and normal left ventricular systolic function (ejection fraction: 70%). Pulmonary CT angiography showed normal mediastinal main vessel structures and diameters, both main pulmonary arteries were patent, and there were no findings suggestive of PTE in either main pulmonary artery or its segmental branches. Electrocardiographic evaluation showed non-specific ST elevation in the anterior leads (Figure 1-2) and non-specific ST depression in the inferior leads (Figure 3-4) without cardiac enzyme elevation. The patient was followed in the cardiology unit for three days. Cardiac enzyme levels were not found to be elevated during follow-up. The patient was questioned about substance abuse and was found to have a history of cocaine use prior to hospitalization and a history of substance abuse one year prior. The patient, whose symptoms resolved and whose cardiac enzyme levels were found to be normal, was referred to an outpatient clinic for follow-up.

Discussion

The habit of chewing coca leaves dates back 2000 years. Albert Niemann first isolated cocaine from the leaves of the coca plant in 1860, and it is a highly addictive substance (1). The major toxicities of cocaine use are due to its sympathomimetic effects. Cocaine inhibits the reuptake of presynaptic biological amines such as norepinephrine, dopamine, and serotonin. In cocaine users, the amount of circulating catecholamines can increase 5-fold, which increases myocardial oxygen demand by increasing heart rate, blood pressure, and left ventricular contractility. In addition, cocaine induces myocardial infarction by causing coronary vasoconstriction and increasing platelet aggregation and thrombus formation (2). An increase in catecholamines leads to hypertension by activating the sympathetic nervous system. However, hypotension may also be observed. Cocaine can cause myocardial ischemia and infarction in patients with and without coronary artery disease. It exerts a procoagulant effect by increasing thromboxane production and decreasing antithrombin III and protein C concentrations (3). Another important prothrombotic property of cocaine is that it accelerates the process of atherosclerosis with hypertension, coronary vasospasm and endothelial damage as a result of chronic use. Cocaine has also been implicated in central necrotizing vasculitis and peripheral vasoconstrictive disorders such as Buerger's disease. Spontaneous renal, mesenteric, iliac, femoral, and popliteal artery thrombosis have been reported (4).

It may develop acutely after overdose, or chronic leg ischemia may occur as a result of chronic use (5). With long-term use, an increased risk of endocarditis may be observed as a result of valvular damage due to organisms such as Candida, Klebsiella, and Pseudomonas (6). In chronic use, myocyte necrosis with accumulation of heavy metals such as manganese in cocaine preparations may lead to dilated cardiomyopathy. It can cause supraventricular and ventricular tachycardias by disrupting myocyte contraction and electrical conduction. Cocaine has antiarrhythmic effects by blocking sodium channels. It disrupts cardiac conduction by causing prolongation of the PR interval, QRS complex, and QT interval, and sinus arrest and atrioventricular block may also be observed (3). Hospital visits for chest pain in cocaine users are common, accounting for about 40 percent of all hospital visits. In our case, chest pain after cocaine use, normal cardiac enzymes, and non-specific ST changes were noted. In the literature review, Mir et al. followed three patients who presented with chest pain after substance abuse and found ST segment changes and elevated cardiac enzymes on electrocardiographic studies; however, angiography showed normal coronary artery structure (7). Mckeever et al. reported finding normal coronary arteries on angiography in a 16-year-old patient who was young, had no risk factors for coronary artery disease,

had a history of substance abuse, and was evaluated for suspected myocardial infarction (8). In two adult patients with coronary artery disease, myocardial infarction and sudden cardiac arrest occurred after substance abuse (9).

Conclusion

Although common in adults, chest pain due to myocardial ischemia is rare in adolescents. Therefore, adolescents presenting to the emergency department with chest pain or other symptoms of myocardial infarction should usually be questioned about cocaine use.

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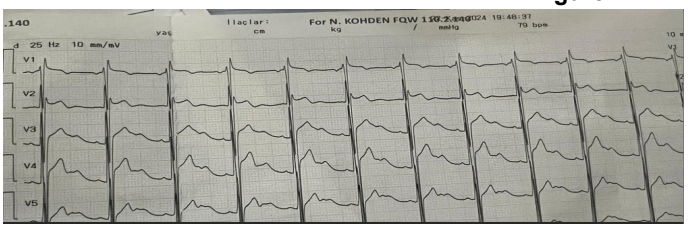
Figure 1-2: V1-V5 ST elevation



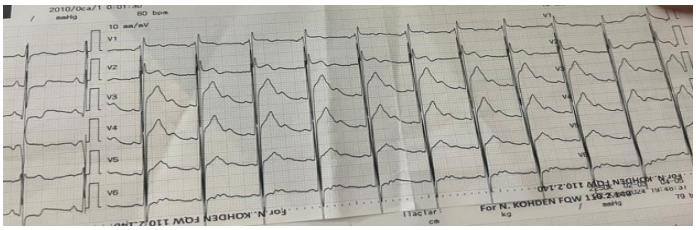
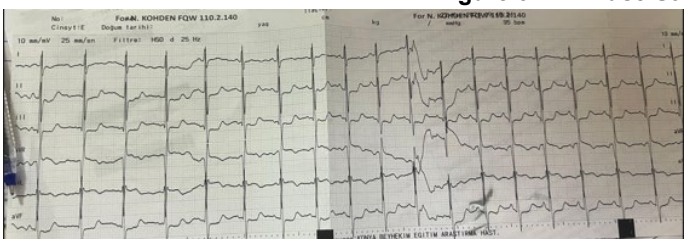
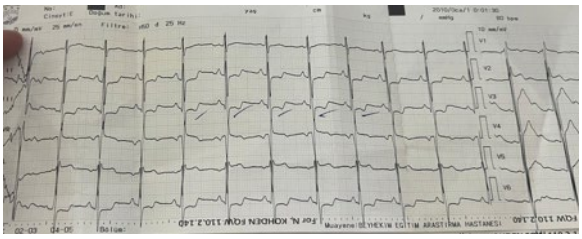


Fıgure 3-4: Diffuse St Depression in D2-D3 and AVF





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Rare but Fatal: Emphysematous Pyelonephritis – A Case Report

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Introductıon

Emphysematous pyelonephritis (EPN) is a rare but life-threatening necrotizing infection of the kidneys characterized by necrosis of the renal parenchyma, suppurative inflammation, and gas formation (1). The most common causative agents include Escherichia coli, Klebsiella pneumoniae, and Proteus species (2). Diabetes Mellitus (DM) is frequently implicated in the etiology. Hyperglycemia, renal ischemia, impaired tissue oxygenation, immune dysfunction, and gas production due to fermentative glucose metabolism by microorganisms are major contributors to pathogenesis (1). Additionally, studies have shown that urinary tract obstruction, advanced age, hypertension, and chronic kidney disease are comorbidities that exacerbate clinical progression and increase the risk of complications (8).

Clinically, patients often present to emergency departments with non-specific abdominal pain, fever, nausea-vomiting, costovertebral angle tenderness, and, in advanced cases, signs of sepsis (3). Due to the non-specificity of symptoms and the frequent presence of

comorbid conditions in typically elderly patients, delays in diagnosis and treatment are common (2). Imaging modalities significantly aid in the diagnostic process, with contrast-enhanced abdominal CT scans considered the gold standard. Radiologic findings supporting diagnosis include gas accumulation in the renal parenchyma, localized abscess formation, and areas of inflammation (3).
Management typically requires a multidisciplinary approach involving intravenous broad-spectrum antibiotic therapy, percutaneous drainage, and nephrectomy in select cases. Although minimally invasive percutaneous interventions have gained popularity in recent years, delayed cases often necessitate more invasive procedures, which are associated with significantly increased mortality (1,3). Therefore, early diagnosis, rapid multidisciplinary coordination, and effective therapeutic interventions are crucial for the management of EPN.
In this case report, we aim to present the clinical assessment and multidisciplinary treatment process of a 55-year-old female patient with comorbidities including hypertension, diabetes mellitus, and hyperlipidemia, who presented with non-specific symptoms and was ultimately diagnosed with emphysematous pyelonephritis following advanced investigations.

Case

A 55-year-old female presented to our emergency department with complaints of reduced oral intake, fever, and nausea. Upon further history taking, it was revealed that she had been previously admitted to a healthcare facility two weeks prior with preliminary diagnoses of pyelonephritis and pneumonia, but had discharged herself voluntarily before completing evaluation and treatment. Her past medical history included hypertension, diabetes mellitus, and hyperlipidemia.
On physical examination, she was conscious, oriented, and cooperative. Respiratory sounds were normal, with no rales or rhonchi. Abdominal examination revealed no guarding or rebound tenderness. Costovertebral angle tenderness was positive. Vital signs were as follows: blood pressure 150/80 mmHg, pulse 113 bpm, SpO□ 96%, and body temperature 36.1°C.
Laboratory findings were as follows: WBC 12.4 x10³/µL, neutrophils 8.7 x10³/µL, GGT 33 U/L, AST 14 U/L, ALT 11 U/L, BUN 11 mg/dL, creatinine 0.98 mg/dL, total bilirubin 0.34 mg/dL, direct bilirubin 0.14 mg/dL, CRP 49 mg/L, and INR 1.06.

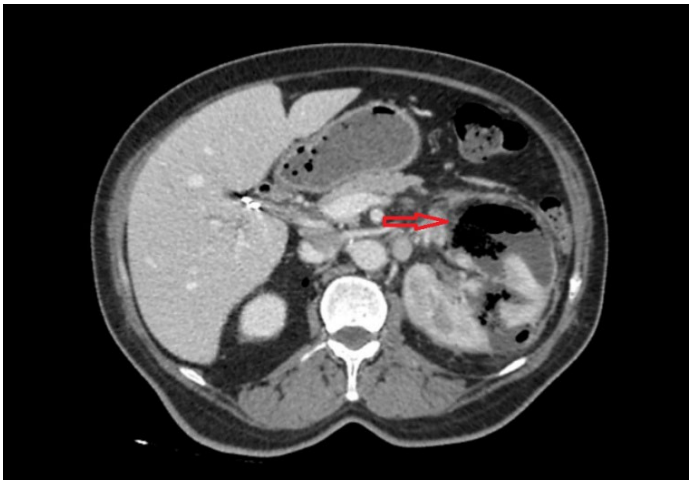

Image 1: Minimal retroperitoneal free air detected between the right adrenal gland and the diapragm.

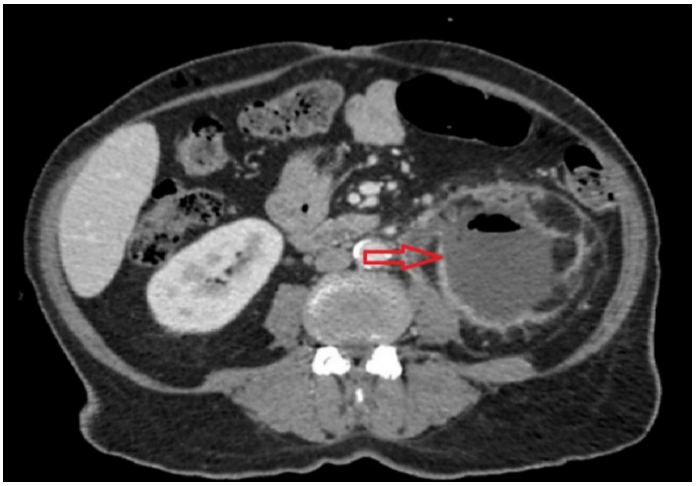

Image 2: A 105x96x133 mm abscess confined by the renal capsule, demonstrating air-fluid levels and extending into the subcapsular space.

Contrast-enhanced abdominal CT revealed a 105x96x133 mm abscess confined to the renal capsule, extending from the anterior parenchyma of the upper pole to involve the entire middle and lower pole parenchyma, with gas-fluid levels and subcapsular extension. Additionally, minimal retroperitoneal free air was observed between the right adrenal gland and diaphragm (Figures 1–2). Suspecting emphysematous pyelonephritis, consultations were requested from Infectious Diseases, Urology, and Nephrology departments.
The Nephrology team recommended intravenous (IV) hydration to monitor renal function. Infectious Diseases suggested initiating IV Amikacin and IV Piperacillin-Tazobactam. The patient was admitted to the urology department for placement of a drainage catheter. Following a three-week course of antibiotic therapy, the patient was discharged in good health.

Dıscussıon

Due to the diagnostic and therapeutic challenges it presents, emphysematous pyelonephritis remains a clinically significant condition. In the case presented, a middle-aged female with risk factors such as diabetes, hypertension, and dyslipidemia experienced delays in diagnosis due to initial incomplete management, resulting in a complicated clinical course. Literature indicates that EPN most commonly affects diabetic patients aged 50–60, with a slightly higher prevalence among females (4), consistent with our case.
From a pathophysiological perspective, diabetes mellitus facilitates gas formation via anaerobic glucose metabolism and promotes rapid infection spread. Impaired renal blood flow and immunosuppression contribute to disease severity (3). Studies report that DM increases mortality risk by 3–4 times, accelerates gas accumulation in infected areas, and increases parenchymal damage (5). Therefore, clinicians should be particularly vigilant when assessing renal infections in diabetic patients.

Our patient's clinical findings aligned with the classic triad described in the literature: flank pain, fever, and costovertebral angle tenderness (6). In elderly populations, however, symptoms may present atypically and less severely (7).

The diagnostic importance of contrast-enhanced abdominal CT is well established. In our case, gas-fluid levels and parenchymal involvement observed on imaging matched typical radiologic findings reported in the literature. Evidence supports that percutaneous drainage is necessary for large abscesses (>6 cm), as antibiotic therapy alone is often insufficient (3). Furthermore, the importance of a multidisciplinary approach in improving treatment outcomes is emphasized. Our case exemplifies this, with the urology department performing percutaneous drainage, infectious diseases initiating broad-spectrum IV antibiotics, and nephrology managing fluid therapy—demonstrating an effective interdisciplinary collaboration as recommended in current guidelines (7).

Conclusıon

In patients presenting with atypical symptoms and comorbidities such as diabetes and hypertension, emphysematous pyelonephritis should be considered in the differential diagnosis of acute kidney infections. Early diagnosis, prompt multidisciplinary management, and appropriate interventions are key to reducing morbidity and mortality.

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Emergency department management following foreign body penetration in the distal third of the right arm

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Abstract: Falls and trauma are common injuries in the pediatric age group. In this case, a simple fall resulted in a foreign body penetration into the distal third of the right forearm. The injury was successfully managed with timely and appropriate surgical intervention. Foreign body injuries can lead to severe complications, and early diagnosis, proper wound care, and a multidisciplinary approach are essential.

Case Presentation: A 7-year-old female child presented to the emergency department following a simple fall, resulting in a foreign body penetration in the distal third of the right forearm. Upon initial examination, the Glasgow Coma Scale (GCS) was 15, and vital signs were stable. There was a wound with an entry point on the distal third of the right forearm, and an exit wound located in the extensor zone 8, measuring 2*2 cm. Due to the patient's pain and agitation, the Allen test could not be performed; however, peripheral pulses were palpable.

Neurological examination was suboptimal due to the patient's age, but no hypoesthesia was noted. The motor examination of the right wrist and fingers was normal. Antibiotics and tetanus prophylaxis were administered before the foreign body was extracted. The patient was then referred to plastic surgery for consultation.

Surgical Intervention: Plastic surgery performed the removal of the foreign body and wound repair. Exploration of the volar aspect of the distal forearm did not reveal any vascular or tendon injury. Penrose drains were placed in both the volar and dorsal aspects to reduce the risk of infection. The skin incisions were appropriately repaired using standard surgical techniques, and the wound was covered with a suitable dressing. Postoperatively, the Allen test was conducted, and vascularization was found to be normal.

Conclusion: This case emphasizes the importance of early intervention, correct diagnosis, and a multidisciplinary approach in the management of foreign body injuries in children. Timely surgical intervention and proper postoperative care have led to a positive outcome in this patient. Regular follow-up showed no complications and a successful recovery. This case demonstrates that, with appropriate surgical techniques and post-operative care, successful outcomes can be achieved in the management of foreign body injuries.

Keywords: Foreign body, pediatric trauma, fall, plastic surgery, emergency department, vascularization, tetanus prophylaxis.

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Idiopathic Isolated Oculomotor Nerve Palsy: A Rare Case Report

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Introduction

The oculomotor nerve (cranial nerve III) innervates four of the six extraocular muscles (superior rectus, medial rectus, inferior rectus, and inferior oblique), the levator palpebrae superioris, and provides parasympathetic fibers to the sphincter pupillae and ciliary muscles, enabling pupillary constriction and accommodation. Dysfunction of this nerve results in ptosis, ophthalmoplegia, diplopia, and sometimes anisocoria.

Common etiologies include microvascular ischemia secondary to diabetes mellitus or hypertension, aneurysms, trauma, neoplasms, or infections. However, in rare cases, no specific cause can be identified despite extensive investigations.

We present a case of isolated oculomotor nerve palsy in which no underlying etiology could be established, highlighting the diagnostic challenges and importance of careful exclusion of life-threatening conditions.

Case Presentation

A 66-year-old woman presented with a five-day history of right upper eyelid drooping and binocular diplopia. Neurological examination revealed right-sided ptosis and limitations in medial, superior, and inferior gaze. Pupillary examination revealed anisocoria, but light reflexes were intact bilaterally.

The patient had a history of type 2 diabetes mellitus, hypertension, and chronic kidney disease. Her medications included metformin, empagliflozin, linagliptin, indapamide, valsartan, and aspirin. She underwent elective coronary angiography two weeks before symptom onset. There was no history of trauma, infection, neurotoxin exposure (e.g., botulinum), or intake of preserved foods.

Cranial MRI with contrast, diffusion-weighted imaging, and CT angiography revealed no pathological findings. Complete blood count, biochemical panels, inflammatory markers, and infection screening were within normal limits.

Evaluations by neurology and neurosurgery teams ruled out surgical or compressive causes. A diagnosis of right complete oculomotor nerve palsy was made, and the patient was admitted to the neurology department for further monitoring and follow-up.

Discussion

The oculomotor nerve can be affected at several anatomical levels, from its nucleus in the midbrain to its course within the orbit. Lesions may present as ptosis, ophthalmoplegia, and pupillary involvement. In patients with diabetes or hypertension, microvascular ischemia is the most common etiology, though it rarely affects the pupil. The presence of pupillary involvement often raises concern for compressive lesions such as posterior communicating artery aneurysms. In this case, although anisocoria was present, imaging ruled out any aneurysmal or mass effect.

Although rare, cases of embolic cranial nerve palsy following coronary angiography have been described. However, no ischemic changes were noted in this patient's diffusion-weighted MRI. Other potential causes such as trauma, infection, neoplasms, toxins, or migraine were excluded based on history and workup.

Given the absence of identifiable etiology despite a comprehensive evaluation, the case was classified as idiopathic isolated oculomotor nerve palsy, a rare but documented clinical entity.

Conclusion

Oculomotor nerve palsy encompasses a wide differential diagnosis, including vascular, compressive, infectious, and inflammatory causes. In rare cases, no specific cause can be determined. In such idiopathic cases, careful evaluation, exclusion of emergent conditions (particularly aneurysms), and close follow-up are essential.

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8756

The importance of inquiring about the last tablet in multiple drug intake: concealed bupropion intake and status epilepticus

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Introductıon

Suicide attempts involving drug overdose are commonly encountered in emergency departments, with repeated medication ingestion potentially leading to severe complications (1). High doses of antidepressants and antipsychotic drugs can have serious adverse effects on the central nervous system (CNS), cardiovascular system, and metabolic functions. Bupropion, a dopamine and norepinephrine reuptake inhibitor, is commonly used to treat major depressive disorder and aid smoking cessation. However, at high doses, it significantly lowers the seizure threshold, with the risk of seizures increasing particularly at doses above 600 mg (2-5). Quetiapine, an atypical antipsychotic used in the treatment of schizophrenia, bipolar disorder, and depression, primarily exerts its effects through dopamine (D2) and serotonin (5-HT2A) receptor antagonism. At high doses, it can cause adverse effects such as hypoten-

sion, cardiotoxicity, altered consciousness, and seizures. The risk of CNS depression and neurological complications is particularly high at doses exceeding 3 grams (6-8). Gabapentin, widely used for epilepsy and neuropathic pain, can cause CNS depression, respiratory failure, and confusion at high doses. When combined with opioids, the risk of respiratory failure significantly increases (9,10). While bupropion-induced seizures are typically generalized tonic-clonic (GTC) in nature, reports of status epilepticus remain extremely rare (8,9). This case presents a patient who developed status epilepticus following a high-dose ingestion of bupropion, quetiapine, and gabapentin with suicidal intent. The case is notable due to the uncommon occurrence of bupropion-induced status epilepticus in the literature.

Case

A 24-year-old male patient was brought to the emergency department following a multiple drug overdose with suicidal intent. His medical history was unremarkable except for a known diagnosis of depression. Upon arrival, the patient stated that he had ingested a large quantity of quetiapine and gabapentin tablets. His initial vital signs were as follows: blood pressure: 120/75 mmHg, heart rate: 88 bpm, respiratory rate: 16 breaths/min, SpO□: 98%, Glasgow Coma Scale (GCS): 15 Since approximately eight hours had elapsed since ingestion, neither activated charcoal nor gastric lavage was administered. The patient was admitted to the Emergency Toxicology Intensive Care Unit for observation with a preliminary diagnosis of multiple drug ingestion and suicide attempt. Four hours after admission, the patient developed generalized tonic-clonic seizures. Despite the administration of diazepam and midazolam, he continued to experience multiple seizures within a five-minute period without regaining consciousness, leading to a diagnosis of status epilepticus. Sedation was initiated, and the patient was intubated. Due to hypotension, inotropic support was started, and sodium bicarbonate therapy was administered. Antipyretic treatment was provided for fever (39°C). While obtaining additional history, the patient's relatives revealed that more medications had been found at home. Upon further investigation, it was discovered that he had ingested a large quantity of bupropion-containing tablets, with empty boxes suggesting a total intake of approximately 4500 mg. The nephrology department recommended dialysis. During follow-up, brain computed tomography (CT) revealed cerebral edema, and the patient was placed on anti-edema and antiepileptic therapy. He was subsequently transferred to a tertiary intensive care unit for continued management.

Dıscussıon

Patients presenting after a drug overdose may initially have normal vital signs, but this should not be reassuring. The pharmacokinetics and pharmacodynamics of the ingested drugs play a crucial role in determining clinical outcomes. Bupropion is particularly notable for its seizure-inducing potential, with the risk significantly increasing at doses above 600 mg (6-8). While seizures due to bupropion overdose are well-documented, most resolve spontaneously, and status epilepticus is rarely reported. However, this case suggests that bupropion toxicity may have more severe neurological consequences than previously thought (9,10). The seizure-inducing effect of bupropion is associated with increased dopaminergic and noradrenergic activity. Additionally, concomitant ingestion of other drugs, such as quetiapine and gabapentin, may have contributed to seizure severity. Quetiapine, known for CNS depressant effects, can also cause QT prolongation, cardiac arrhythmias, and hypotension. At high doses, it may lead to drowsiness and confusion. Gabapentin, although used therapeutically for epilepsy, may paradoxically lower the seizure threshold at high doses (4-7). The combination of these drugs likely exacerbated the patient's condition. There is no specific antidote for bupropion toxicity. Treatment typically includes benzodiazepines and barbiturates for seizure control. Sodium bicarbonate may be beneficial in cases of arrhythmia, and intravenous lipid emulsion therapy can be considered for refractory cases. Status epilepticus management depends on seizure frequency and consciousness level, often requiring intubation and intensive care (4,5,11).

Conclusıon

This case highlights the potential severity of neurological complications from high-dose bupropion ingestion, emphasizing the risk of seizures progressing to status epilepticus. In cases of multiple drug ingestion, particularly involving antidepressants and antipsychotics, clinicians should be aware of the possibility of a more severe clinical course. Additionally, patients may conceal certain medications, leading to an underestimation of the overdose severity. Therefore, repeated questioning and close monitoring are essential in cases of suspected polypharmacy.

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An interesting Case Report of Peripartum Cardiomyopathy, Successful Diagnosis and Treatment

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Introduction

Peripartum cardiomyopathy (PPCM) is a rare cause of cardiomyopathy that occurs during late pregnancy or in the early postpartum period. It is characterized by significant left ventricular dysfunction and heart failure in the peripartum period occurring in the absence of other identifiable causes of heart failure. [1] Echocardiography is crucial for diagnosis, revealing new left ventricular systolic dysfunction, including reduced ejection fraction and fractional shortening. [2] Echocardiography suffices to differentiate it from other causes and usually shows left ventricle dilatation of variable degrees, left ventricle systolic dysfunction, right ventricular and bi-atrial enlargement, mitral and tricuspid regurgitation, pulmonary hypertension, and intracardiac thrombus. [3] Echocardiography criteria to diagnose PPCM includes ejection fraction less than 45%, end-diastolic diameter greater than 2.7 cm/m2 and/or M-mode fractional shortening less than 30%. [3] The available information from multiple studies has shown that PPCM varies geographically. [1] In the United States, the incidence has been reported to be as low as 1 case per 4,000 live births in comparison with the higher incidence in Nigeria of 1 case in every 100 live births. Women older than 25 years with a mean age of 30 years were found more likely to develop PPCM. [1] Other important factors related to the presence of PPCM include hypertensive disorders associated with pregnancy, the presence of anemia, and African descent. [1]

There are approximately 11 hypotheses suggested as a possible causal factor for the development of heart failure.[4]

Viral myocarditis during a peripartum period possibly due to echovirus, coxsackievirus, parvovirus B19, human herpesvirus 6, Epstein bar virus, or human cytomegalovirus. [5]

Unable to clear cardiac antigen autoantibodies due to reduced humoral immunity. [6]

Inadequate response to hemodynamic stress of pregnancy. [6]

Aggressive cardiac cell apoptosis. [7]

Inflammation due to cytokines. [8]

Selenium deficiency due to malnutrition. [9]

Familial predisposition due to a genetic mode of transmission. [10]

Increased prolactin effect on the heart. [11]

Estrogen and progesterone effect. [12]

Myocardial stunning due to adrenergic surge. [12]

Myocardial ischemia. [13]

Some of the most common risk factors for the development of postpartum cardiomyopathy are as follows; Advanced maternal age (more cases reported in both extremes of age), high parity (71% of women diagnosed with PPCM had three or more prior pregnancies), high gravidity, twin pregnancy (more endemic in women with twin pregnancies), use of tocolytic therapy (greater than 4 weeks can cause silent ischemia), african descent (more prevalent in the African population), poverty, hypertension, cocaine abuse. [14]

Clinical characteristics of PPCM resemble those of dilated cardiomyopathy and shared genetic predispositions can be found in approximately 15 % of all cases. [19] Defined biomarkers to distinguish between PPCM and other cardiomyopathies are still largely missing, but careful analyses of the PPCM pathophysiology have enabled the identification of a set of specific diagnostic and prognostic biomarkers. [20]

Symptoms may include; fatigue, feeling of heart racing or skipping beats (palpitations), increased nighttime urination (nocturia), shortness of breath with activity and when lying flat, swelling of the ankles. [15]

Treatment with the “BOARD” scheme (bromocriptine, oral heart failure drugs, anticoagulants, vasorelaxing agents, and diuretics) is recommended for PPCM. Bromocriptine is the new disease-specific therapy for PPCM as it inhibits prolactin secretion and release of its cardiotoxic fragments. [16] Bromocriptine therapy has been found to improve cardiac recovery and reduce the mortality in PPCM patients. [17] The medical management of patients with PPCM is similar to that for other forms of heart failure, and treatment aims to reduce afterload, preload, and to increase contractility. [18] Despite growing literature, mortality due to PPCM remains high (30%–60%) and survivors have a high risk (50%–80%) of developing cardiac failure during future pregnancies. [18]

Case

A 32-year-old patient with a 34-week twin pregnancy with a known history of 3 abortions and 2 cesarean sections started to complain of nausea, headache and shortness of breath following a cesarean section under spinal anesthesia at another medical facility. During the subsequent follow-up, the patient exhibited signs of respiratory failure, she was intubated and referral to our hospital.

The vital signs recorded at the time of the patient's arrival at the emergency department were as follows: Blood pressure 155/95 mmHg, pulse rate 95/min (rhythmic), respiratory rate 16/min, oxygen saturation 89% and body temperature 36.6°C.

On physical examination; Intubated, full outline of unresponsiveness (four score 13), The pupils were isochoric, and the patient was disoriented and uncooperative. Heart sounds were heard in normal rhythm and no murmur was detected. Bilateral widespread rales were heard, and pretibial edema (moderate) was observed in both legs, examination of other systems was evaluated within normal limits.

Bedside ultrasound revealed an ejection fraction of approximately 35%, with left ventricle wall motion characterized as global hypokinetic.

The electrocardiogram (ECG) was assessed as sinus tachycardia. Initial tests revealed lactate of 1.0 mmol/L, partial pressure of oxygen 62 mmHg, partial pressure of carbon dioxide 35 mmHg and base deficit of -1.2, hemoglobin was 9,5 g/dL, hematocrit was 28.1%, with a leukocyte count of 10.2 x 10^3/µL and a platelet count of 206 x 10^3/µL, d-dimer 22910 ng/ml, fibrinojen 282 mg / dl, troponin 1000 ng/L, international normalized ratio (INR) 0.97, c reaktif protein 2 mg/L, creatinine 0.78 mg/dL and brain natriuretic peptide (bnp) 2447 pg/ml.

The patient exhibited signs of peripartum cardiomyopathy, a condition characterized by heart failure that develops shortly after pregnancy. Notably, the patient did not have any other identifiable causes for their heart failure, nor did they have any documented evidence of heart disease prior to the final month of pregnancy. Echocardiography revealed a 35% systolic dysfunction in the left ventricle, and physical examination findings indicated the presence of pulmonary edema as a secondary complication of heart failure. Furthermore, laboratory and radiological findings corroborated the diagnosis of heart failure and pulmonary edema. The patient's treatment was initiated, and the cardiology department was consulted. The patient was subsequently transferred to the intensive care unit for further observation and treatment.

The therapeutic approach was initiated in accordance with the "BOARD" protocol. The patient was given 40 mg intravenous (IV) push of furosemide and a 20 mg/hour IV infusion of furosemide. Dopamine was initiated at a rate of 10 mcg/kg/min to achieve a positive inotropic effect in the patient, who exhibited 40% systolic dysfunction in the left ventricle and global hypokinesia on echocardiography. Furthermore, bromocriptine 2.5 mg twice daily and enoxaparin sodium 0.4 ml once daily were administered as anticoagulant. During the patient's intensive care follow-up, there was a decrease in her pulmonary edema and an improvement in her general condition. She was extubated according to weaning criteria. On the seventh day, the patient was transferred to the cardiology service for ongoing treatment and monitoring.

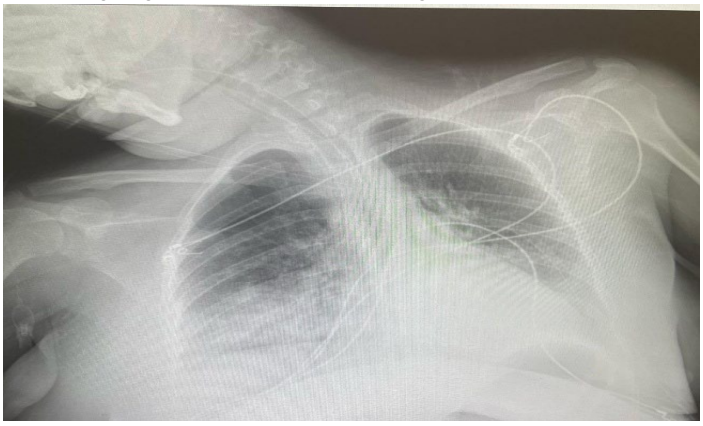

Figure 1

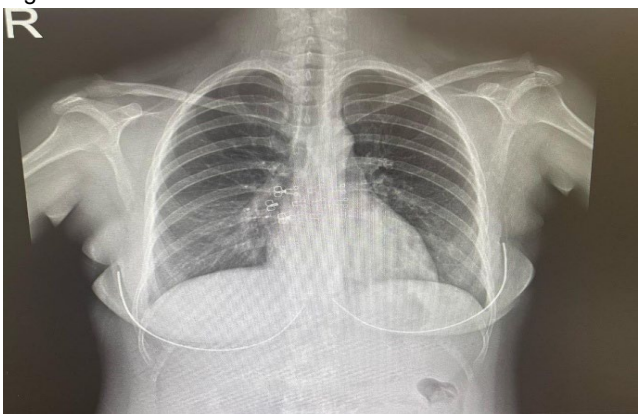

Figure 2

As illustrated in the initial figure, the patient's chest X-ray taken at the time of admission reveals the presence of significant pulmonary edema. In the subsequent figure, after a period of seven days, the treatment has led to a substantial decrease in pulmonary edema.

Discussıon

The etiology behind PPCM is still unclear. Relation with eclampsia and hypertension during pregnancy has been found. Still, the underlying mechanism is unclear. Risk factors for PPCM are african descent, age, pregnancy-related hypertension disorders, multiparity, multiple gestations, obesity, chronic hypertension, and prolonged used of tocolytics. [1] In our case, there was also a twin pregnancy, there is a high number of pregnancies with 3 abortions and 2 cesareans. Symptoms may include; fatigue, feeling of heart racing or skipping beats (palpitations), increased nighttime urination (nocturia), shortness of breath with activity and when lying flat, swelling of the ankles. [15] In the present case, the following symptoms were observed: palpitations, orthopnea, and bilateral moderate pretibial edema.

The most used and widely available biomarkers are BNP and NT-proBNP and although not specific for PPCM normal values can exclude acute heart failure immediately. [21] In our case BNP resulted 2447 pg/ml. BNP elevation confirmed the presentation of peripartum cardiomyopathy in our case.

Initially, case reports were published postulating a potential effect on LV function and recovery of bromocriptine when added to standard of care heart failure treatment (SHFT) in acute PPCM. [22] However, there was some harsh criticism in the face of this new treatment concept considering the impact of bromocriptine on top of SHFT. Two proof-of-concept studies suggested a positive effect of an additive therapy with bromocriptine in PPCM patients. In short, additive bromocriptine treatment resulted in fewer deaths, fewer patients in NYHA functional class III and IV and fewer patients with persistent LVEF <35 %.

The "BOARD" scheme is recommended for the treatment of PPCM. The patient was treated in accordance with the BOARD therapy regimen. During the intensive care follow-up period, the patient exhibited a decrease in pulmonary edema and an improvement in general condition, resulting in extubation according to weaning criteria. On the seventh day, the patient was transferred to the cardiology service for ongoing treatment and monitoring. Subsequent monitoring revealed that the patient did not manifest pulmonary edema, her ejection fraction increased to 55%, and she did not report symptoms of orthopnea, dyspnea, or palpitations.

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Aortic Dissection İn Pellet İnjury

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Introduction

Aortic injury is one of the most dangerous vascular injuries that are of vital importance. It may be non-traumatic due to underlying diseases such as Marfan syndrome, aortic aneurysm, hypertension; It can also occur due to traumatic causes such as gunshot wounds, stabbing or traffic accidents. Although many aortic injuries are visible as a result of examinations, some aortic injuries are only diagnosed intraoperatively and are diagnosed treated during the operation.

Case

The 16-year-old patient, who was brought in as a result of a gunshot wound, was examined from the outside and multiple pellet injuries were observed on the left shoulder and the left side of the neck. When the patient was brought to the emergency room, his blood pressure was 60/30 and her oxygen saturation was around 70. The patient was electively intubated because of increasing respiratory distress. As a result of the examinations, contrast-enhanced computed tomography angiography revealed pneumothorax and hemothorax in the left lung, and aortic dissection at the T4 level, behind the aorta and in front of the T4 vertebra. He was transferred to a higher center and underwent emergency surgery.

Figure 1

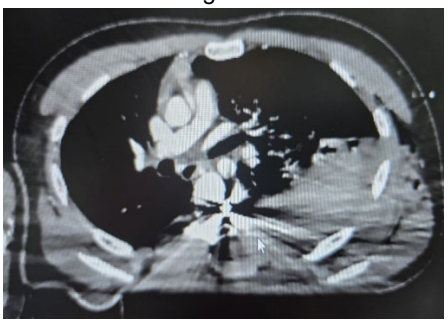


Figure 2

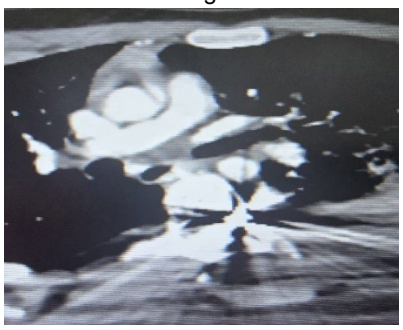
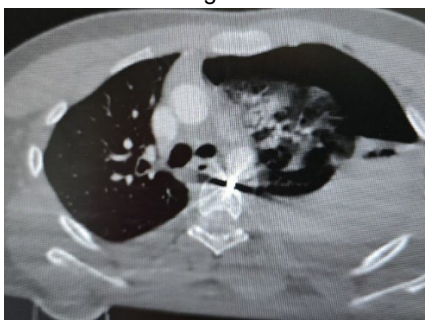


Figure 3



Conclusıon

In multiple gunshot wounds, pellets or bullet fragments cause vital organ injuries. In vital organ injuries, the necessary operative procedures must be planned and performed after the primary intervention has secured the patient's respiratory tract.

Discussion

In gunshot wounds, especially those caused by pellets, the injury areas may be missed. In case of pellet injuries, extensive scanning should be done, the location of the pellet pieces should be found, detailed imaging should be done and intervention should be made, considering that they will cause damage along their path. First of all, the whole body should be scanned with X-ray, the distance between where the pellet is and the area where it enters should be calculated, and screening tests appropriate to this distance should be requested. A single mistake missed may result in the patient losing his life. t should not be forgotten that a pellet entering from the shoulder, neck or upper thorax will cause injuries to the heart, thorax, aorta and abdomen, and scanning should be done in a wide area.

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Pulmonary Embolism with Negative D-dimer: A Case Report

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Introduction

Pulmonary embolism (PE) is a serious clinical condition caused by obstruction of the pulmonary arteries, most commonly due to thrombosis originating from deep veins of the lower extremities. PE accounts for approximately 5–10% of in-hospital deaths and is often a preventable cause of mortality. The diagnostic process typically includes clinical probability assessment, D-dimer testing, and imaging studies. D-dimer, a fibrin degradation product, is frequently used due to its high sensitivity and ability to exclude PE in low-to-intermediate risk patients. However, its specificity is low, and under certain clinical circumstances, false-negative results may

occur.

Case Presentation

A 51-year-old male with a known history of hypertension applied to the emergency department with sudden-onset pain in the left shoulder, back, and flank. He doesn't have symptoms such as; cough, sputum production, chest pain, dyspnea, or hemoptysis. He was a non-smoker and had no prior history of deep vein thrombosis (DVT) or PE. However, he had traveled long distances several times in the past month, and his family history was notable for varicose veins.

On physical examination, the patient was alert, cooperative, and oriented. His blood pressure was 143/99 mmHg, heart rate 92 bpm, body temperature 36.2°C, and oxygen saturation 95% on room air. Auscultation revealed coarse crackles at the base of the left lung. No edema, tenderness, or asymmetry was detected in the lower extremities.

Laboratory tests revealed a white blood cell count of 12.02 ×10^9/L, hemoglobin 15.2 g/dL, and C-reactive protein (CRP) 58.4 mg/L. Troponin and D-dimer levels were within normal limits. Despite the negative D-dimer result, computed tomography pulmonary angiography (CTPA) was performed due to persistent clinical suspicion. CTPA revealed hypodense filling defects in the lateral and posterior basal segments of the left lower lobe pulmonary arteries, consistent with acute PE.

The patient started on low-molecular-weight heparin (LMWH) adjusted for renal function and body weight. Compression socks were applied from ankle to groin. He was admitted to the pulmonology ward for further monitoring and treatment.

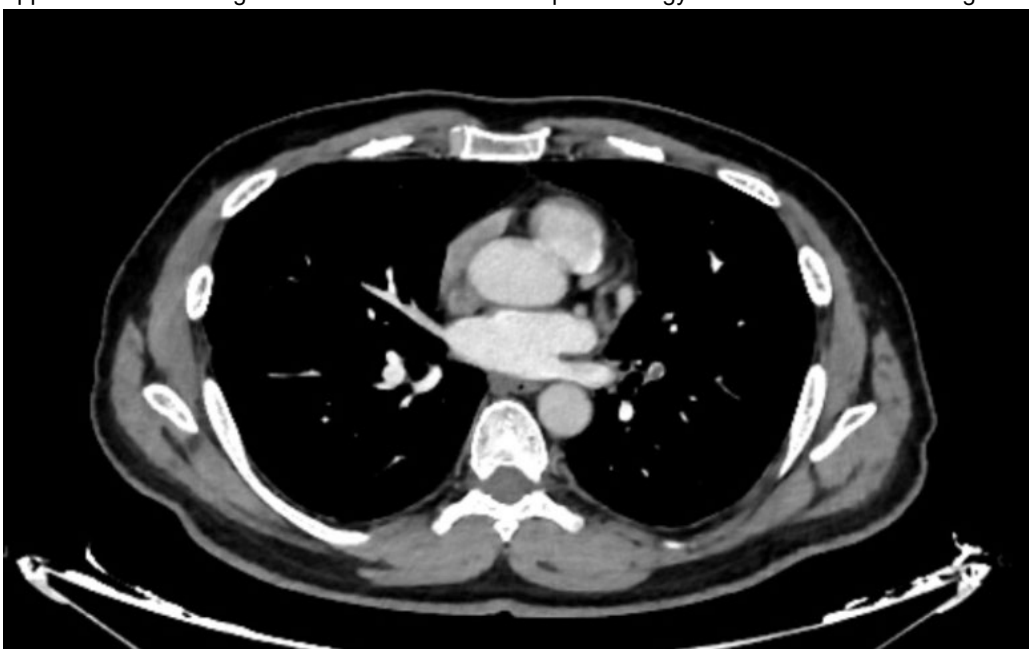

Figure: Hypodense filling defects in the lateral and posterior basal segments of the left lower lobe pulmonary arteries

Discussion

D-dimer is a sensitive marker of fibrinolysis and is elevated in most patients with acute thrombotic events. It is widely used to rule out PE in patients with low or intermediate clinical probability. However, its diagnostic reliability can be compromised in specific scenarios. False-negative D-dimer results have been associated with small, subsegmental emboli, delayed testing, prior anticoagulant therapy, impaired fibrinolytic activity, and assay variability.

In a case reported by Sheikh and Jamhuri, a woman using oral contraceptives presented with PE despite a D-dimer level <200 ng/mL. Similarly, Dhananjaya et al. report'ed that 64.3% of 35 patients with confirmed PE had negative D-dimer results. Such findings underline the potential for misdiagnosis if clinical judgment is overshadowed by test results.

Therefore, in cases of high clinical suspicion, imaging studies such as CTPA should not be withheld solely based on a negative D-dimer result. In this case, early imaging led to timely diagnosis and management, potentially preventing fatal complications.

Conclusion

PE remains a diagnostic challenge in emergency medicine due to its variable presentation. While D-dimer is a valuable tool in the diagnostic algorithm, it should not be used in isolation, especially in high-risk patients. This case highlights the importance of comprehensive clinical assessment and the potential limitations of laboratory testing. Early imaging and prompt treatment are essential to improving outcomes for the patients with suspected PE.

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A syndrome that significantly affects quality of life and causes dysphagia: wallenberg syndrome

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Introductıon

Wallenberg syndrome is a brainstem ischemic stroke affecting the lateral medulla, typically characterized by sensory disturbances, unilateral Horner's syndrome, and cerebellar findings (1). Traditionally, it has been associated with posterior inferior cerebellar artery

(PICA) occlusion; however, studies indicate that the vertebral artery is occluded in 90% of cases(2). Additionally, Wallenberg syndrome has been linked to occlusion of the superior, middle, or inferior lateral medullary arteries (3).

The syndrome manifests with signs of long tract involvement, including contralateral loss of pain and temperature sensation, ipsilateral ataxia, and Horner's syndrome, as well as dysfunction of the fifth, eighth, ninth, and tenth cranial nerves (4-6). There is a significant overlap between Wallenberg syndrome and lateral pontine syndromes; however, dysphagia due to nucleus ambiguus or fascicular involvement is a distinguishing feature of Wallenberg syndrome (5,6). Inferior cerebellar involvement often leads to nausea, vertigo, and ataxia. While most patients with lateral medullary infarction (LMI) experience significant recovery, some may suffer sudden, unexpected death due to respiratory or cardiac arrest, even in the absence of cerebellar edema or basilar artery thrombosis (2). Hiccups are another symptom associated with lateral medullary syndrome, resulting from the sudden contraction of the diaphragm and external (inspiratory) intercostal muscles, followed by glottic closure (7). Dysphagia is a frequently encountered symptom in emergency departments. This case report highlights the importance of considering central pathologies in the differential diagnosis of patients presenting with dysphagia.

Case

An 84-year-old male patient presented to an external emergency department with difficulty swallowing and was subsequently referred to our center. The patient had congenital speech and hearing impairment. Neurological examination revealed that he was oriented, cooperative, and fully conscious. Dysphagia was evident, and the gag reflex was absent. Upon standing, he exhibited imbalance, though no nystagmus was observed. Muscle strength was bilaterally 5/5, and systemic examinations were unremarkable.

Brain imaging revealed no acute pathology on CT. However, diffusion-weighted MRI demonstrated an area in the brainstem consistent with acute infarction (Figure 1). Laboratory tests showed no pathological findings. The patient was then consulted by the Neurology Department and admitted to the Neurology Ward with a preliminary diagnosis of Wallenberg syndrome.

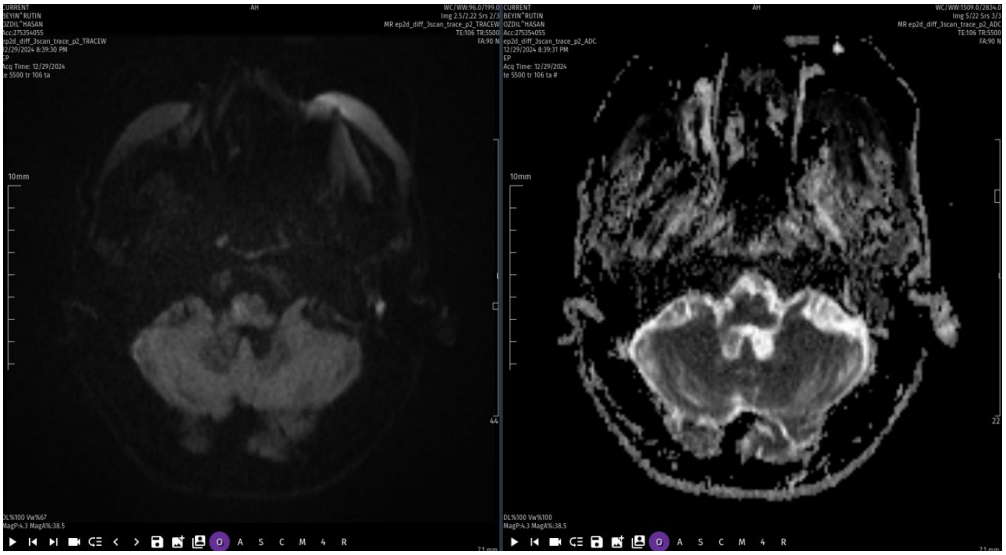

Figure 1. Diffusion MRI Image of the Case

Discussion

Wallenberg syndrome is an ischemic stroke syndrome affecting the lateral medulla, characterized by sensory disturbances, unilateral Horner's syndrome, and cerebellar findings (1). It involves the nuclei and fascicles of the fifth, eighth, ninth, and tenth cranial nerves, leading to symptoms such as contralateral loss of pain and temperature sensation, ipsilateral ataxia, and Horner's syndrome (4-6).

There is significant overlap between Wallenberg syndrome and lateral pontine syndromes. However, the presence of dysphagia due to nucleus ambiguus or fascicular involvement is a key distinguishing feature of Wallenberg syndrome (5,6). Symptoms have a sudden onset in 75% of patients. Among the remaining 25% with a more gradual onset, initial complaints typically include headache, dizziness, confusion, and gait disturbances, with sensory deficits, dysphagia, hoarseness, and hiccups appearing later (8).

Dysphagia is reported in 51–94% of patients with Wallenberg syndrome (9). Our case involved an 84-year-old male, aligning with the literature, which indicates that the syndrome is more common in elderly individuals. Dysphagia is a frequent symptom prompting emergency department visits and is often associated with benign conditions such as upper respiratory tract infections. However, considering central pathologies in the differential diagnosis is crucial.

Wallenberg syndrome primarily affects elderly individuals with vascular risk factors. Like other stroke syndromes, it typically has an acute onset. The most common initial symptoms include vertigo with dizziness, balance impairment with gait instability, hoarseness, and dysphagia, with symptoms progressing over several hours to days. Unlike other stroke syndromes, muscle weakness is generally absent, which may lead to misdiagnosis or underdiagnosis. A thorough neurological examination is essential for accurate diagnosis. While complete Wallenberg syndrome is rare, partial syndromes often provide sufficient diagnostic clues.

Conclusion

Dysphagia is a common reason for emergency department visits, frequently associated with upper respiratory tract infections. However, in elderly patients, central pathologies should always be considered in the differential diagnosis. Dorsolateral medullary ischemic infarction is a complex syndrome often presenting with signs suggestive of central vestibular pathology on audiovestibular evaluation. Our case findings were consistent with the clinical manifestations described in the literature. For accurate diagnosis and treatment, a comprehensive neurological, otological, audiological, and vestibular assessment is recommended.

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The Role of Laboratory Parameters in the Etiology of Epistaxis: A Retrospective Evaluation

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Introduction

Epistaxis, or nasal bleeding, is a common otorhinolaryngological emergency that can affect individuals across all age groups. Although most cases are benign and self-limited, some require emergency medical intervention. Identifying the underlying etiological factors is critical, particularly in recurrent or severe cases. This study investigates the value of routine laboratory tests in clarifying the etiology of epistaxis.

Materials and Methods

This retrospective study was conducted in a tertiary care emergency department between October 1, 2024, and December 31, 2025. Patient records were screened for a diagnosis of epistaxis. Patients aged 18 and older were included, while those with traumatic causes or incomplete records were excluded. Variables such as age, gender, vital signs, and laboratory findings were analyzed.

Descriptive statistics were presented as counts and percentages for categorical variables and as mean±standard deviation or median (minimum–maximum) for continuous variables. Categorical data were compared using the Chi-square test. Parametric and nonparametric continuous variables were evaluated using the T-test and Mann–Whitney U test, respectively. Statistical significance was accepted at $p<0.05$.

Results

A total of 82 patients were initially identified, of which 27 were excluded due to missing laboratory data or incomplete records. Thus, 55 patients formed the study sample. Demographic and clinical characteristics are summarized in Table 1, and laboratory findings in Table 2.

Table 1. Demographic and Clinical Characteristics of Patients

Characteristic	n (%)
Gender (Female)	38 (69.1%)
Gender (Male)	17 (30.9%)
Coumadin Use (Yes)	2 (3.6%)
Coumadin Use (No)	53 (96.4%)
Active Bleeding Present	35 (63.6%)
Active Bleeding Absent	20 (36.4%)

Table 2. Laboratory Parameters and Vital Signs

Parameter	Mean ± SD	Median (Min–Max)
Age		70 (18–95)
Glucose		136 (79–343)
Neutrophil		6.3 (2–12.86)
Lymphocyte		1.35 (0.18–3.97)
Albumin	23.81 ± 3.76	

Platelet		229 (41–660)
INR		1.15 (0.84–6.82)
Hemoglobin		9.9 (7.1–16.4)
CRP		12.5 (0.4–300.6)
Creatinine		1.01 (0.49–5.7)
Urea		71 (22–245)
AST		17 (7–210)
ALT		13 (3–181)
Systolic BP		120 (90–190)
Diastolic BP		75 (40–120)

Discussion

The findings of this study reinforce the association between aging and increased incidence of epistaxis, potentially due to vascular fragility and comorbid conditions like hypertension. The significant differences in ALT and glucose values suggest possible underlying metabolic and hepatic contributions. These parameters may serve as supplementary indicators in selected cases.

This retrospective analysis of 55 patients with epistaxis revealed important associations between clinical presentation and certain laboratory parameters. While further prospective studies are necessary, our data suggest that routine lab work may not always be essential, particularly in patients with known chronic hypertension.

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A Case Report of Spontaneous Pneumomediastinum From an Unusual Cause: Turbulence in the Pilot's Lungs

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Introduction

Spontaneous pneumomediastinum (SPM) is a rare clinical entity thought to occur in approximately 0.002% of the population, resulting in approximately 1 in 30,000 Emergency Department (ED) visits in the United States [1,2] SPM tends to occur during activities that increase intrathoracic pressure, such as forceful coughing or vomiting, intense physical activity, childbirth, or performing the Valsalva maneuver. [3] This condition is usually seen in young males who experience an acute onset of chest pain, dyspnea, odynophagia, and/or voice changes. [4] Physical examination frequently reveals subcutaneous emphysema, hoarse voice, tachycardia, tachypnea, and occasionally a “Hamman’s sign,” which is a crunching sound heard on cardiac auscultation. [5] While patients with SPM typically fare well, the initial investigation should be extensive in order to rule out serious conditions such as esophageal rupture, i.e., Boerhaave syndrome, ruptured viscus, or blunt trauma. [1] Typical imaging modalities include plain film radiographs of the chest, Computed Tomography (CT) scans, esophagrams, and endoscopy. [1] Treatment is usually symptomatic with rest, analgesics, and the use of oxygen if needed. [1] In this article, we wanted to discuss the diagnosis of SPM in a pilot who applied with complaints of dyspnea and cough and had a turbulent flight 2 days ago, as well as the early diagnosis and treatment process.

Case

A 30-year-old male patient applied to the ED with complaints of chest pain, shortness of breath, runny nose and sore throat that had been increasing in the last 24 hours. He states that the pain is constant and especially worsens when taking a deep breath or coughing. He also feels mild pain in his neck and shoulders.The patient states that he has not had any trauma or intense physical activity recently. However, he states that they experienced a sudden loss of altitude due to turbulence on their last flight and that he felt pressure in his chest during this time, and that this pain increased with coughing. He has no history of any lung disease or other health problems before.

The vital signs measured at the time of presentation to the ED were as follows: Blood pressure 125/77 mmHg, pulse rate 114/min (rhythmic), respiratory rate 24/min (superficial and rapid), oxygen saturation 92% (room air) and body temperature 36.8°C.

On physical examination, conscious, cooperative and oriented. Appears slightly restless. Oropharynx hyperemic, mediastinal crackle on auscultation (Hamman sign), neck crepitation on deep palpation. This indicated that air had leaked into tissues where it should not normally be present and raised suspicion for the diagnosis of pneumomediastinum. Heart sounds were heard in normal rhythm

and no murmur was detected. Examination of other systems was evaluated within normal limits.
The electrocardiogram (ECG) was assessed as Sinus tachycardia. Initial tests revealed lactate of 1.8 mmol/L, partial pressure of oxygen 54 mm Hg, partial pressure of carbon dioxide 55 mm Hg and base deficit of -1.2. Hemoglobin was 16.4 g/dL, hematocrit was 46.6%, with a leukocyte count of 12.01 x 10^3/µL predominantly neutrophils at 93.7 % and a platelet count of 258 x 10^3/µL. Laboratory results showed, d-dimer 1400 ng/ml , troponin 4.7 ng/L, International normalized ratio (INR) 0.97, c reaktif protein 2 mg/L and creatinine 0.89 mg/dL.

Figure 1

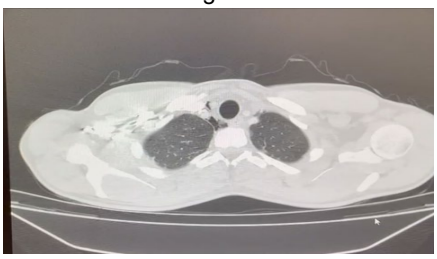


Figure 2

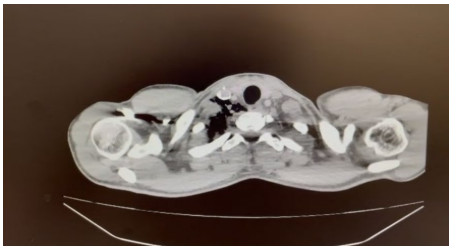
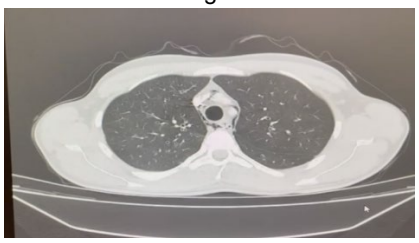


Figure 3



Free air extending to the right side of the neck in Figures 1 and Figures 2 and in the middle regions in Figure 3 confirms the diagnosis of pneumomediastinum.
Here, the cough due to upper respiratory tract infection was thought to be precipitated and the patient, who is also a pilot, was associated with the environmental-high altitude shown among the etiology of pneumomediastinum in the flight 2 days ago, and the diagnosis was first confirmed with the Hamman sign in the physical examination and crepitation in the neck during deep palpation,followed CT Scan with intravenous (IV) Contrast.
In the treatment, ceftriaxone 2 gr IV, salbutamol and budesonid as nebules, 80 mg prednisolone IV and pantoprazole 40 mg IV was given.
The patient was consulted to thoracic surgery and admitted to the thoracic surgery service for follow-up and treatment. During his 2-day stay in the thoracic surgery ward, his complaints regressed. Thereupon, the patient was discharged to be followed up later.

Discussıon

Louis Virgil Hamman's case series,published in 1939, was the first of its kind to characterize SPM. [6] The crunching sound heard during the cardiac cycle of affected patients accordingly bears his name - Hamman's crunch or Hamman's sign - and SPM is also known as Hamman's syndrome. [7] Also in 1939, Charles Clifford Macklin, a pulmonologist, described the pathophysiology of the condition. SPM, he explained, results when a sudden increase in intrathoracic pressure leads to alveolar rupture and consequent air dissection and tracking along the bronchovascular sheaths and into the mediastinum. [5] This mechanism became known as the Macklin effect. [7] In our case, the patient's severe cough and high altitude, this may have led to the Macklin effect and to his SPM.The most common signs and findings in SPM are chest pain, dyspnea and subcutaneous emphysema. [8] The most common symptoms in SPM were dyspne (%85), swelling in neck (%69), chest pain (%69) and cough (%54), and determined subcutaneous emphysema in %85 of them according to the study of Panigrahi and colleagues. [9] Typical physical examination finding is Hamman's sign. [10] Panacek et al. detected the Hamman's sign in %52 of the patients in their SPM series including 17 cases.[11] Hamman's sign was the most significant physical examination finding that led us to this diagnosis.
We established the final diagnosis through CT which is the gold standard test in the literature. [1] In the SPM study of Kim et al. conducted on 64 patients, oxygen inhalation therapy and bedrest were performed to all patients (%100); 57 (%89.1) patients received prophylactic antibiotherapy and 47 (%73.4) patients received analgesic drugs; no mortality and morbidity was detected in the cases.[12] İn the ED for treatment, ceftriaxone 2 gr IV, salbutamol and budesonid as nebules, 80 mg prednisolone IV and pantoprazole 40 mg IV was given. As a result, SPM due to the potential life-threatening risk, a detailed medical history, physical examination and appropriate imaging methods should be used for rapid diagnosis and treatment planning. SPM is a self-limiting clinical condition that responds well to conservative treatment. In our case, pneumomediastinum was diagnosed after physical examination and thoracic CT and treatment was started immediately. The patient's treatment continued in the thoracic surgery department for 2 days and the pneumomediastinum regressed, he was discharged to be called for a follow-up.

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Chorea Following Hyperglycemia

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Introduction

Hemiballismus-hemichorea is a rare movement disorder often associated with lesions in the basal ganglia or subthalamic nucleus. Hyperglycemia due to diabetes can cause choreiform and ballismus-type movements, particularly characterized by changes in the basal ganglia on magnetic resonance imaging (MRI). This condition is referred to as Hyperglycemia-Induced Hemiballismus-Hemichorea(HIHH)(1). It is known that the clinical presentation and radiological findings in HIHH patients can fully or partially reverse when blood glucose levels return to the normal range(2).

This case report presents a patient who developed hemiballismus-hemichorea triggered by hyperglycemia.

Case Report

A 53-year-old female patient presented to the emergency department with involuntary movements in her left upper and lower extremities and choreiform movements on one side of her face. Her medical history included hypertension and type 2 diabetes. The patient also had hypoesthesia on the left side of her face. Her speech rate had slowed, but no dysarthria or aphasia was detected.

At admission, her blood glucose level was measured at 496.6 mg/dL, and her HbA1c was found to be 16%. Other blood tests were within normal limits. Urinalysis revealed glucosuria. A cranial CT scan showed no pathology. Contrast-enhanced brain MRI revealed signal changes at the level of the right basal ganglia, specifically in the right caudate nucleus and putamen, which were considered to be associated with hyperglycemia(Figure 1).

The patient was admitted to the neurology ward. Treatment was initiated to regulate blood glucose and control the involuntary movements. The choreiform movements regressed by approximately 80%. Her blood glucose levels stabilized, and she was discharged.

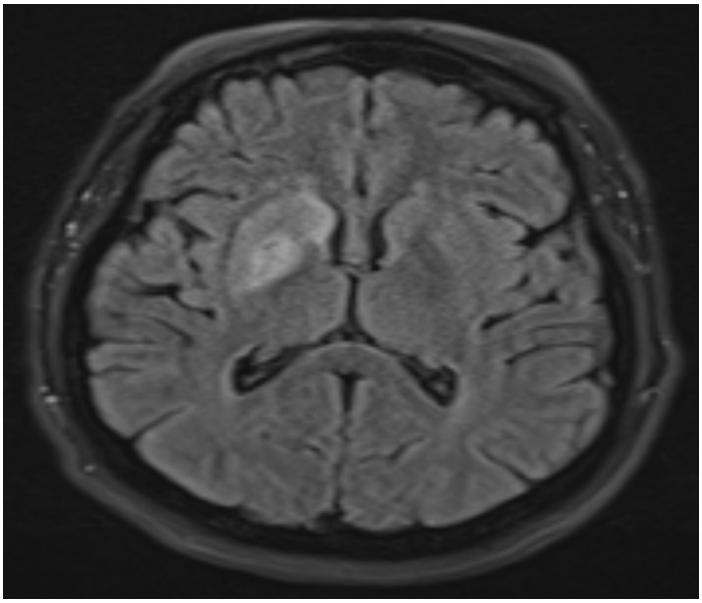

Figure 1. Increased signal in the basal ganglia on the right

Discussion

In the pathophysiology of HIHH, metabolic dysfunction in the basal ganglia due to hyperglycemia is believed to play a role. Studies have reported an association between high blood glucose levels and MRI changes in the basal ganglia (3).

Early glycemic control is considered important for the improvement of clinical symptoms(4). Additionally, in patients diagnosed with HIHH, involuntary movements have been shown to be controlled with the use of neuroleptics and antiepileptic medications(5).

In conclusion, in diabetic patients presenting with sudden-onset movement disorders, the possibility of HIHH should be considered, and appropriate treatment should be initiated.

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A rare case: empagliflozin associated euglycemic diabetic ketoacidosis masked by pneumonia

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Introductıon

Diabetic ketoacidosis (DKA) is an endocrine emergency resulting from insulin deficiency. The American Diabetes Association defines DKA as the coexistence of metabolic acidosis (arterial pH < 7.3 and serum bicarbonate < 18 mmol/L), ketosis (ketonemia or ketonuria), and hyperglycemia (serum glucose > 250 mg/dL) (1). In 1973, Munro et al. first described cases of DKA occurring with normal or mildly elevated serum glucose levels and termed this condition euglycemic diabetic ketoacidosis (EDKA) (2).

Sodium-glucose cotransporter-2 (SGLT-2) inhibitors are a novel class of antihyperglycemic agents used in the treatment of diabetes (3). Their preference is primarily due to significant cardiovascular and renal benefits. However, these agents have also been associated with an increased risk of DKA. Most DKA cases linked to SGLT-2 inhibitor use occur with normal or minimally elevated glucose levels, potentially delaying diagnosis (4). EDKA typically occurs in patients with relatively low blood glucose levels, often associated with starvation, chronic liver disease, pregnancy, infections, and alcohol use. SGLT-2 inhibitors, which increase urinary glucose excretion, are also linked to EDKA (5).

This report presents a case of EDKA associated with SGLT-2 inhibitor use in a patient diagnosed with COVID-19 pneumonia. A review of the current literature on this emerging and rare condition is also provided.

Case

A 50-year-old male patient presented to the emergency department on November 22, 2020, with complaints of nausea, fatigue, cough, sputum production, and shortness of breath. According to the history provided by the patient and his spouse, there were no episodes of syncope, chest pain, vomiting, or diarrhea, but mild abdominal pain was reported. The cough had begun approximately one week prior to admission, and four days before presentation, a SARS-CoV-2 PCR test returned positive. Chest computed tomography (CT) revealed peripheral ground-glass opacities. Based on these findings, the patient was diagnosed with COVID-19 pneumonia, and favipiravir treatment was initiated (dexamethasone and enoxaparin were not administered). The patient's medical history included diabetes mellitus (DM) for one year, hypertension (HT), hyperlipidemia (HL), and depression for the past two years.

The patient had been diagnosed with diabetes mellitus one year prior and reported fasting glucose levels ranging between 130–160 mg/dL and postprandial levels between 170–230 mg/dL. He managed his condition with oral medication and dietary control. His medications included metformin 2x1 g/day, vildagliptin 2x50 mg/day, and empagliflozin 1x10 mg/day. Although diagnosed with hypertension, he was not compliant with prescribed antihypertensive medications; his home blood pressure readings were within a systolic range of 120–140 mmHg. He was taking 10 mg of rosuvastatin for hyperlipidemia. The patient had a history of depression under treatment for two years and a prior suicide attempt. His current psychiatric medications included venlafaxine 150 mg and risperidone 1 mg. The patient also had a history of spinal surgery 20 years ago (due to suspected neuroblastoma) and gastric bleeding requiring surgery 4 years ago.

Upon arrival at the emergency department, his vital signs were as follows: body temperature 36.7°C, blood pressure 155/92 mmHg, pulse 113 bpm, respiratory rate 46 breaths/min, and peripheral oxygen saturation (SpO_2) 82%. The patient was given oxygen at a flow rate of 2 L/min via nasal cannula. Venous blood gas analysis showed: pH 7.21 (reference: 7.35–7.45), PCO_2 25 mmHg (reference: 35–45), PO_2 33 mmHg (reference: 80–100), SO_2 44% (reference: 75–99), lactate 1.3 mmol/L (reference: 0.5–2.2), and bicarbonate (HCO_3^-) 13 mmol/L (reference: 22–26).

Despite oxygen supplementation, arterial oxygen saturation only increased to 88%, so oxygen delivery was escalated to 4 L/min via

face mask. Follow-up arterial blood gas analysis revealed pH 7.24, PO_2 72 mmHg, PCO_2 21 mmHg, HCO_3^- 13 mmol/L, SO_2 93%, and lactate 0.4 mmol/L.

On physical examination, the Glasgow Coma Scale score was 15 and RASS score was 0. Pupils were isochoric, and bilateral direct and consensual light reflexes were present. Lung auscultation revealed coarse breath sounds with equal respiratory effort on both hemithoraces. The patient exhibited abdominal respiration. The abdominal examination was non-tender on palpation. No pretibial edema or signs of soft tissue infection were observed. Laboratory results revealed a blood glucose level of 160 mg/dL, low serum bicarbonate (13 mmol/L), and a high anion gap (22 mEq/L), indicative of metabolic acidosis. Serum lactate level was within normal limits. Except for mild hyponatremia (Na^+ 131 mmol/L), other serum electrolytes were within normal limits. BUN and creatinine levels were also normal. Urinalysis showed ketonuria (80 mg/dL) and glycosuria (2000 mg/dL). The NT-proBNP level was 113 pg/mL (reference: 0–125 pg/mL), and CRP was markedly elevated at 227 mg/L (reference: 0–5 mg/L).

Pulmonary CT angiography, interpreted by the on-call radiologist, showed no filling defects in the main pulmonary artery or its segmental/lobar branches, excluding acute pulmonary embolism. However, peripheral ground-glass and consolidation areas were detected in both lungs, consistent with viral pneumonia, particularly COVID-19. The estimated lung involvement was 35–40%.

The elevated anion gap raised suspicion of euglycemic ketoacidosis potentially caused by infection or SGLT2 inhibitor therapy. To prevent dehydration and correct the acidosis, 1.5 liters of isotonic saline were administered over the first 2 hours. Simultaneously, continuous insulin infusion was initiated at 0.1 IU/kg/hour. Given a potassium level of 4.6 mmol/L, potassium replacement was started at a rate of 20 mmol/hour. To prevent hypoglycemia and promote resolution of ketosis and ketonuria, 5% dextrose infusion at 50 cc/hour was added to the fluid regimen. Empagliflozin therapy was discontinued. Hourly blood glucose and venous blood gas measurements were conducted. Biochemistry panels and urinalysis were scheduled every 4 hours. Improvement in the patient's anion gap metabolic acidosis following IV fluid resuscitation and insulin infusion was consistent with euDKA, most likely attributable to SGLT2 inhibitor use. Following consultation with the endocrinology department, the patient was admitted to the internal medicine intensive care unit.

Discussion

SGLT-2 inhibitors are recommended as second-line agents in the treatment of type 2 diabetes mellitus (T2DM); however, clinicians may also prefer them as first-line therapy (6). Since SGLT-2 inhibitors lower the renal threshold for glucose excretion, the presence of normal or moderately elevated blood glucose levels does not rule out DKA during their use (7). Patients receiving SGLT-2 inhibitor therapy—particularly those who are euglycemic—should be thoroughly evaluated for EDKA (8).

EDKA is considered an acute complication of diabetes and is more commonly observed in patients using SGLT-2 inhibitors. It typically develops in individuals with poorly controlled type 1 diabetes or in type 2 diabetic patients exposed to external stressors such as infection, alcohol consumption, or surgery. This clinical picture is preventable when recognized early and managed appropriately by clinicians (9). In a study by Peters et al., 13 cases of DKA with mild hyperglycemia or normoglycemia were reported in nine individuals treated with canagliflozin, an SGLT-2 inhibitor. Of these, seven had type 1 diabetes, a condition for which SGLT-2 inhibitors are not indicated (10).Although SGLT-2 inhibitors are generally well tolerated in outpatient settings, their use is not recommended in symptomatic T2DM patients with COVID-19 infection due to the risk of anorexia, dehydration, and sudden clinical deterioration. In patients diagnosed with T2DM who contract COVID-19, the use of SGLT-2 inhibitors should be reconsidered, and discontinued in those requiring hospitalization. Patients diagnosed with COVID-19 who are taking SGLT-2 inhibitors should be closely monitored from the onset of infection for euglycemic ketoacidosis, hypovolemia, electrolyte imbalances, and renal function deterioration (11).

In conclusion, while SGLT-2 inhibitors offer significant glycemic and non-glycemic benefits such as weight loss and blood pressure reduction, they also represent an important addition to the antihyperglycemic drug class. Patients presenting to the emergency department with abdominal pain, nausea, vomiting, and respiratory distress while on SGLT-2 inhibitors should be evaluated for EDKA (8, 10). The diagnosis of EDKA in patients with COVID-19 pneumonia receiving SGLT-2 inhibitors is a critical consideration for clinicians (11).

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Violence in Healthcare: The Role and Consequences of Social Media

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Introductıon

Violence in healthcare institutions is defined as "a situation consisting of threatening behavior, verbal threat, economic abuse, physical assault and sexual assault from patients, patients' relatives or any other individual that poses a risk to healthcare workers" (1). Exposure to violence has been increasing in recent years for all professional groups, especially healthcare workers and physicians, and constitutes a serious health problem (2). In our study, we investigated the level of exposure to violence among health care workers working in a tertiary university hospital and how the increase in violence news through social media affected their biopsychosocial lives. The aim of our study is to identify preventable causes and to reveal the measures that can be taken.

Materıal-Methods

The study was conducted between 01.11.2023 - 01.05.2024 in the Emergency Medicine Clinic of XXX University Faculty of Medicine Hospital under the leadership of the research team after obtaining ethics committee approval. The participants who participated in the study were asked the questionnaire questions created by the researchers, which lasted approximately 5 minutes, and the answers were recorded.

Results

A total of 1006 people participated in the study. While 87.8% of the participants stated that they had been subjected to verbal violence, 17.8% stated that they had been subjected to physical violence. While 79.9% of the women thought that the news about violence on social media increased violence in health, the majority of the participants thought that the news was not reflected objectively. Nurses were the occupational group that was exposed to verbal violence the most with 90.2% and gave the most white codes with 36.6%. All professional groups in our study have the opinion that giving code white does not bring a solution to violence incidents. 76.3% of the participants think that X-ray devices and 90.7% think that security guards are ineffective and inadequate in affecting violence.

Dıscussıon

Verbal violence is the most common type of violence in the world (3). In our study, it was found that 87.8% of healthcare workers were exposed to verbal violence. Among healthcare workers, nurses are the group most frequently subjected to violence, while physicians are the group that gives the most white codes (4,5). When the studies in the literature are examined, it is seen that a small proportion of the incidents of violence in healthcare are reported (6) 40.9% of the participants stated that they were negatively affected psychologically, 62.3% stated that their profession was constantly devalued, and 78.4% stated that these news negatively affected their work motivation due to the news on violence in health in social media. The data are consistent with the literature (7). Unverified information on social media has negative effects on people and increases the incidents of violence against healthcare workers.

Conclusıon

Violence in health is one of the world's leading problems and the measures taken in this regard are insufficient. Today, with the effect of social media, people are encouraged to violence with unconfirmed news and information, and the number of patients resorting to violence is increasing. Almost all of those working in the field of health stated that they have been subjected to violence at least once in their working life and that they would like to change their field of work. In order to improve working conditions and to prevent violence, the necessary and deterrent punitive action should be implemented immediately by the competent authorities.

Keywords: Violence in Health, Social Media, Violence News

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1570

Let it be nothing else, this patient is too young for this diagnosis! A case of atypically presenting diverticulitis

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Introductıon

Diverticulitis is a disease that occurs as a result of the inflammation of the diverticula in the digestive system, especially in the large intestine (1). Diverticula are small vesicles formed on the intestinal wall and are mostly asymptomatic. However, diverticulitis develops when they become inflamed and symptoms such as severe abdominal pain, fever, nausea, vomiting and changes in bowel habits may occur (2). Although this disease is mostly seen in the sigmoid colon, it may rarely occur in the small intestine. Although small bowel diverticula are usually asymptomatic, serious complicati, includinging inflammation, haemorrhage, intestinal obstruction and perforation may develop in some patients (3). One of the most important causes of diverticulitis is low fiber diet. Inadequate fiber intake slows down bowel movements and causes hardening of the stool and an increase in the intraluminal pressure (4). With advancing age, the intestinal wall loses its elasticity and the risk of diverticulitis increases. Obesity, sedentary lifestyle and diseases affecting intestinal motility also play a role in the development of diverticulitis (5). Patients usually present with severe abdominal pain felt in the left lower quadrant, nausea, vomiting, constipation or diarrhea. Fever and leucocytosis are also common findings (6). One of the most valuable diagnostic methods is computerized tomography (CT). CT is highly effective in detecting complications including diverticular inflammation, abscess and perforation. Ultrasonography (USG) is an auxiliary method, but its diagnostic sensitivity is low (7). Although colonoscopy is not recommended in the acute period, it can be used to evaluate the general condition of the colon during the healing process. The treatment of diverticulitis varies according to the severity of the disease. In mild cases, antibiotic treatment, a liquid diet, and bowel rest may be sufficient. However, surgical treatment is necessary if complications such as perforation, abscess formation, or intestinal obstruction develop (8). In recent years, laparoscopic surgical methods have become widesprea, andd this technique accelerates the healing process and decreases the complication rates. In cases requiring surgical intervention, procedures such as bowel resection and anastomosis can be performed.

Case

A 36-year-old male patient with no known comorbidities presented to the emergency department with a sudden onset of left lower quadrant pain. Physical examination revealed left lower quadrant tenderness, but no signs of defense or rebound. The patient had no complaints of nausea or vomiting. Bowel functions were normal and gas and fecal discharge was present. Laboratory investigations revealed a white blood cell count (WBC) of 12.000/mm and a C-reactive protein (CRP) level of 11 mgL. Despite IV hydration and symptomatic treatment, the patient's abdominal pain did not improve significantly, and a whole abdominal computed tomography (CT) scan with IV contrast was performed. Imaging revealed heterogeneity and marked fatty appearance in the intra-abdominal fatty tissues adjacent to the colonic anus in the left paracolic region in the left lower quadrant of the abdomen. The findings were evaluated as compatible with diverticulitis. The patient was consulted to general surgery and hospitalized for follow-up and treatment.

Dıscussıon

Diverticulitis is one of the inflammatory bowel diseases usually seen in older age groups. It occurs in association with low fibre diet, intestinal motility disorders and aging. However, it should be kept in mind that diverticulitis may also develop in young adults as in this case.

Because diverticulitis cases in young adults are observed less frequently in the literature, it may lead to delays in the diagnosis. One of the most reliable diagnostic methods is contrast-enhanced computed tomography (5). In this case, symptomatic treatment was initially administered because the patient was young, but the symptoms persisted and the definite diagnosis was made by abdominal CT with IV contrast. Diverticulitis should be considered in the differential diagnosis of young adult patients presenting with left lower quadrant pain.

In the literature, it has been reported that most diverticulitis cases are observed in elderly patients and complication rates are higher in this group (3). Although antibiotic treatment and symptomatic support may be sufficient in mild cases, surgical treatment may be required in the presence of complications such as perforation or obstruction (6). In a study presented by Ünek et al. İt was reported that the mean age of jejunal diverticulitis cases with perforation was 73 years and surgical treatment was required (1). Pehlivanlı et al. Reported a case of diverticulitis causing obstruction in the small intestine and emphasized that this was a rare but serious complication (9). In a case reported by Özdemir et al. İleal diverticulitis with perforation was treated with surgical methods (2). In contrast, in this case, diverticulitis was colonic and developed in a young patient and was successfully managed with conservative methods.

Conclusıon

In conclusion, diverticulitis is a disease that may lead to serious complications if not diagnosed early and treated appropriately. This case is remarkable in terms of showing that diverticulitis is not specific only to elderly patients and can also occur in young adults. Diverticulitis should be considered as a differential diagnosis, especially in young patients presenting with unexplained left lower quadrant pain, and imaging modalities should be used effectively to avoid unnecessary invasive procedures. In this case, because the patient was a young adult and was diagnosed early, a successful treatment was provided with conservative methods and could be managed without the need for surgical intervention. In addition to treatment, dietary modification and lifestyle changes that support intestinal health should be recommended to prevent the development of future complications.

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Axillary artery embolism, a rare condition seen in the emergency department

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Introductıon

Patients with suspected upper extremity embolism typically present with a sudden onset of symptoms [1]. Because atherosclerotic disease affects the upper extremities at a much lower rate than the lower extremities, it is rare for patients to present suddenly with upper extremity ischemia [2,3]. In cases of acute limb ischemia, clinical assessment should proceed rapidly, as the likelihood of recovery is highly dependent on prompt and complete revascularization of the upper limb [1]. Given the frequency with which cardiac etiology is implicated as the source of the embolus, any history of cardiac arrhythmia, coronary artery disease, myocardial infarction, or valvular pathology should be noted. This case is presented to emphasize the importance of considering upper extremity embolism (UEE) in patients presenting to the emergency department with these findings.

Case

A 38-year-old male patient with no known chronic diseases was brought to the emergency room by the emergency medical team (112) with complaints of sudden onset of pain and numbness in his right arm while at work. The patient's arterial blood pressure was 113/76 mmHg, oxygen saturation was 99%, and pulse rate was 105/min. Physical examination revealed no motor loss in the right arm, but there was new-onset discoloration of the fingertips, pain exacerbated by movement, and sensory loss. Based on the patient's history, it was determined that the pain, numbness, and difficulty in movement had begun within the last hour. Doppler ultrasound was ordered due to the absence of pathology in central imaging, lack of distal pulse patency, onset of discoloration, and no relief despite analgesia. The ultrasound revealed occlusion of the right axillary artery. Although the right brachial, radial, and ulnar arteries were patent, no significant flow was observed secondary to proximal occlusion.

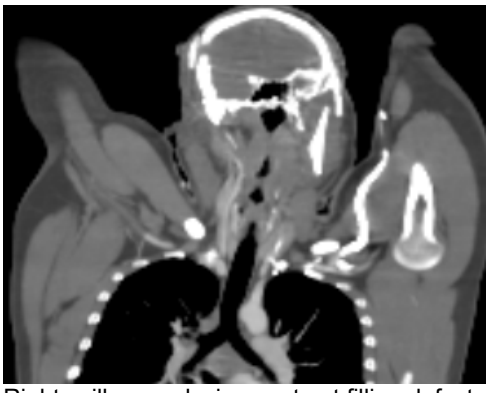
Laboratory results from the patient's blood tests were as follows: CK-MB 2, troponin 6.54, CK 204, WBC 12.11, platelets 245, lactate 3.3, PT 11.6, INR 1.01, and APTT 18.7. CT angiography showed no long-segment contrast material passage at the level of the right brachial artery. Following a thrombectomy procedure, the patient was discharged from the hospital with full patency restored.

Dıscussıon

Sensory deficits alone—or their absence—may suggest the initial stages of ischemia, while the appearance of motor deficits in addition to sensory deficits represents an immediately threatened limb in need of urgent revascularization [4]. Throughout the examination of the affected limb, it should be compared with the unaffected limb. Depending on the duration of symptoms, the patient may exhibit leukocytosis, elevated lactic acid levels, and increased inflammatory markers such as erythrocyte sedimentation rate and CRP. D-dimer has a good negative predictive value in the setting of arterial ischemia, but its significance is uncertain when elevated [5]. Although CT angiography can usually visualize the arterial circulation of the upper extremity, its sensitivity may be reduced for distal arterial branches. This suggests that contrast transmission should be carefully evaluated in the presence of emboli or chronic atherosclerotic plaque.

Conclusıon

As demonstrated in our case, the classic findings in a patient's history that raise suspicion of upper extremity arterial embolism include sudden onset of upper extremity pain, numbness, discoloration (e.g., pallor), coldness in the affected extremity, and possibly reduced motor function or paralysis. Additionally, the patient's ability to pinpoint the exact onset of symptoms should serve as a warning sign for this diagnosis.



Right axillary occlusion contrast filling defect

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A case report of a pediatric patient admitted to the emergency department with eye perforation

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Introduction and Purpose: Perforating eye injuries are one of the most common causes of sudden vision loss in emergency department visits. Perforating eye injuries are most commonly seen in children, traffic accidents, and industrial workers. Workplace-related eye injuries can be disabling and are largely preventable (1). Open globe injuries are full-thickness defects in the eye. Open globe injuries in children constitute 27-48% of all injuries. Prognosis in children is worse compared to closed globe injuries. The type of injury, mechanism, location, and posterior segment status are the factors that most affect the visual prognosis in the injured person (2). In this case, we will share a case of a child who accidentally perforated the eye due to a piece of iron entering the eye.

Case: A 17-year-old male presented to the emergency room with a laceration resulting from a piece of iron entering his eye. He stated that he was injured while working with iron at home. The patient, who had no known comorbidities, was examined and isolated eye trauma was observed (Figure 1). The patient's right light reflex could not be obtained, and his vital signs were as follows; blood pressure: 140/70 mmHg, pulse: 87/min, respiratory rate: 24/min, body temperature: 36.5oC. GCS: 15. Tetanus and antibiotic treatment was administered. In the Computed Tomography (CT) images, it was determined that the right eye globe was disrupted and perforated, and there was a millimetric defective appearance on the skin in the right infraorbital area (Figure 2). The patient was urgently consulted to the ophthalmology department, and after obtaining consent, he was taken to emergency surgery by ophthalmology.

Discussion: Eye perforation is an emergency requiring rapid evaluation and intervention in the emergency department. An open globe injury is a full-thickness traumatic injury to the eye wall (cornea or sclera) that is associated with significant morbidity. The injury may be caused by blunt or penetrating mechanisms and subtypes include penetration, perforation, intraocular foreign body, or globe rupture.

If left untreated, patients may experience endophthalmitis and vision loss. Low visual acuity after injury, globe rupture, presence of intraocular foreign bodies, being elderly, female, child, and delays in surgical intervention are associated with poor prognosis. CT helps to confirm the diagnosis of globe injury (2,3). In a study, it was determined that eye injuries in children were most frequently caused by knives (14.8%), pencils (12.5%), and glass (11.4%), while iron caused 4.5% of injuries. Valsalva maneuver, which would increase intraocular pressure, should be prevented and the eye should be protected from trauma by using a protective shield. Broad-spectrum antibiotics are started against the risk of infection, and intraocular damage is assessed with imaging methods. In addition, the patient is prevented from rubbing the eye and the head is kept in an upright position. It is important to consult the ophthalmology department immediately after the injury and determine the need for surgery to prevent blindness and endophthalmitis (3,4). In this case, an open globe injury occurred as a result of an iron piece penetrating into the right eye. A CT scan was performed

to confirm the diagnosis and detect additional injuries. In accordance with the literature, early treatment was initiated and the ophthalmology department was consulted immediately.

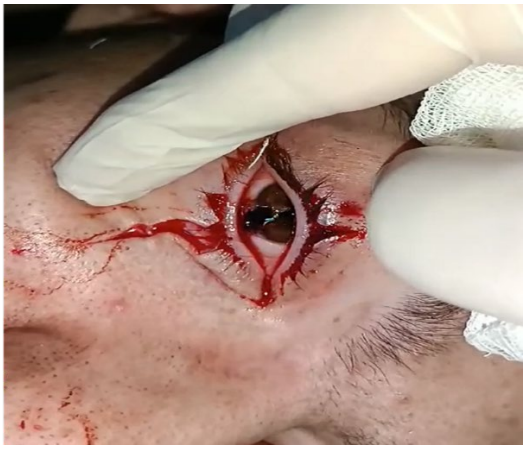

Figure 1. Perforation of right eye, inspection finding

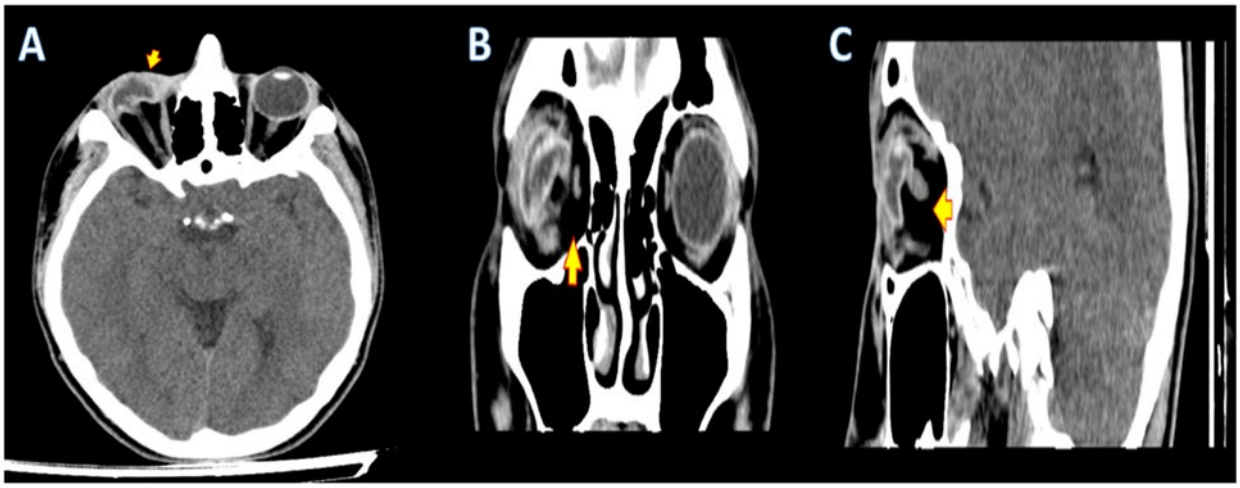

Figure 2. Axial (A), coronal (B) and sagittal (C) orbital CT images reveal right eye globe integrity is disrupted, perforated appearance and millimetric defect on the skin in the right infraorbital area (arrows).

Conclusion: Considering that perforation may occur in eye injuries, we believe that it is vital to obtain the necessary imaging, ensure prompt referral to ophthalmology and related branches, and provide tetanus and antibiotic treatments.

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1612

Dıagnostıc Effıcacy Of Lumbar Puncture In Patıents Presentıng Wıth Suspıcıon Of Menıngıtıs And Encephalıtıs

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Introductıon

Lumbar puncture (LP) is a fundamental diagnostic method used to diagnose central nervous system infections. Meningitis and encephalitis require rapid evaluation in the emergency department. This study aims to evaluate LP results in patients presenting with neurological symptoms in the emergency department.

Materıal And Methods

This retrospective study included 120 patients over 18 years old who presented to the emergency department with altered consciousness, fever, disorientation-cooperation disorders, and other neurological symptoms and underwent LP between 01.06.2024 and 01.12.2024. The LP results of the patients were evaluated, and positive LP rates and diagnoses were determined.

Results

Among the 120 patients over 18 years old who presented with neurological symptoms and underwent LP, bacterial meningitis was diagnosed in 24 patients, and encephalitis in 12 patients.

Examination of cerebrospinal fluid (CSF) findings revealed:

Leukocytosis in 24 patients (20%) (all bacterial meningitis cases)

Increased microprotein levels in 42 patients (35%) (24 meningitis, 18 culture-negative cases)

Decreased glucose levels in 6 patients (5%) (meningitis cases)

Increased glucose levels in 6 patients (5%) (PCR and culture-negative cases)

Discussion

CSF analysis is critical in diagnosing meningitis and encephalitis. In our study, the meningitis diagnosis rate was found to be 20%, while the encephalitis rate was 10%. CSF leukocytosis, protein, and glucose levels assist in diagnosis, but PCR and culture results are required for definitive diagnosis.

Conclusion

LP is a crucial procedure for diagnosing meningitis and encephalitis in the emergency department. CSF analysis parameters should be evaluated together with clinical suspicion. Larger-scale studies are needed to investigate the diagnostic efficacy of CSF parameters.

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A Rare Complication of Upper Respiratory Tract Infection in the Emergency Department: Pneumomediastinum

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Introduction

Spontaneous pneumomediastinum (SPM) is a rare condition caused by free air accumulation in the mediastinum without trauma or iatrogenic etiology. This condition is usually seen in young men typically occurs due to increased intrathoracic pressure from severe coughing, exacarbation of asthma, vomiting, or physical exertion (1,2).

It is mainly caused by alveolar rupture due to increased intraalveolar pressure and air travelling along the bronchovascular sheath into the mediastinum. While SPM is mostly benign and self-limited, it must be differentiated from life-threatening conditions like acute coronary sendrom, pneumothorax, esophageal rupture, mediastinitis (3,4).

This case report aims to discuss a case of spontaneous pneumomediastinum in a previously healthy young woman, focusing on the clinical approach, diagnostic evaluation, and management.

Case

A 19-year-old female patient with no history of chronic illness presented to emergency department (ED) with sore throat, cough, and sharp chest pain. Despite prior emergency visits and treatment for upper respiratory tract infection, her symptoms persisted. Vital signs were within normal limits. On physical examination, oropharyngeal hyperemia was noted, pulmonary auscultation was unremarkable, and no pathological findings were detected in other systemic evaluations. The patient reported a 3–4-day history of symptoms, during which she presented twice to an external ED. Despite medical treatment, her complaints persisted, and her chest pain gradually intensified.

Due to repeated emergency department visits, laboratory tests were performed, revealing a white blood cell (WBC) count of 15,000 and a neutrophil count of 13,000, with no abnormalities noted in other external laboratory results. The patient's electrocardiogram (ECG) was consistent with normal sinus rhythm. Thoracic computed tomography (CT) revealed pneumomediastinum (Figure 1). A consultation with the thoracic surgery department was obtained, and the patient was admitted to the surgical ward. After four days of inpatient follow-up, her symptoms improved, and she was discharged in good condition with a prescription.

Conclussion

Although pneumomediastinum generally has a favorable prognosis, it can be complicated by life-threatening conditions such as pneumothorax, subcutaneous emphysema, mediastinitis, tension pneumomediastinum, and air embolism. Clinicians should consider SPM in patients with persistent chest pain and upper respiratory tract infection symptoms.

Keywords: Pneumomediastinum, chest pain, upper respiratory tract infection

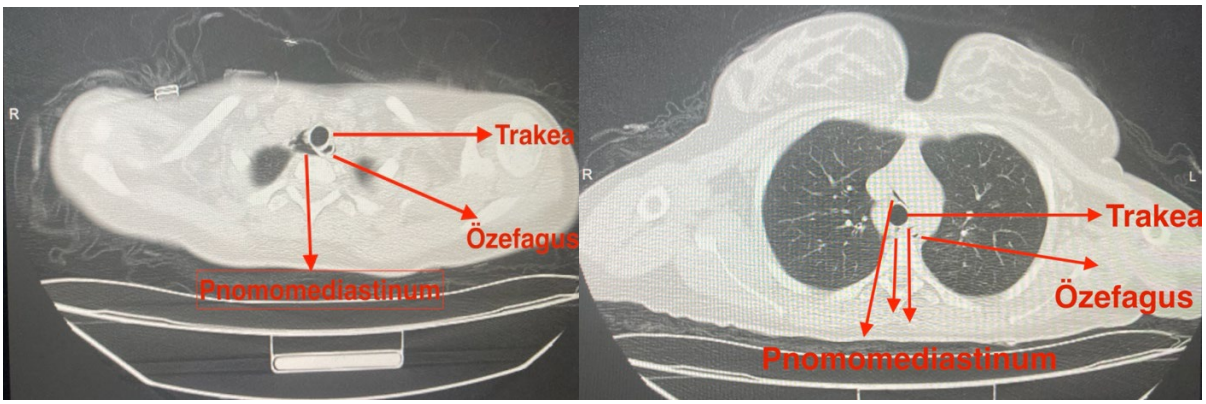

Figure-1: Pnomomediastinum appearance on thorax CT

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Fatal Intraparenchymal and Intraventricular Hemorrhage Due to Methanol Intoxication: A Rare Case Report

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Introductıon

Methanol is a toxic monohydric alcohol commonly found in industrial solvents and counterfeit alcoholic beverages. It is metabolized in the liver to formaldehyde and then to formic acid. Formic acid inhibits cytochrome C oxidase in the mitochondrial respiratory chain, leading to hypoxic tissue damage. Tissues with high energy demands, such as the optic nerves and basal ganglia, are particularly susceptible to its toxic effects.

Symptoms typically emerge after a latent period, usually 6–30 hours post-ingestion, and may include nausea, vomiting, headache, dizziness, photophobia, blurred vision, and progressive altered mental status. Diagnosis is supported by unexplained high anion gap metabolic acidosis and elevated osmolar gap. Since direct methanol measurement is not always feasible, diagnosis often relies on indirect parameters and clinical history.

While early and aggressive treatment can reduce methanol-related mortality to below 20%, delayed presentation or therapy may raise the mortality rate above 50%. This case aims to discuss the clinical, laboratory, and imaging findings of a severe methanol poisoning case and to highlight prognostic indicators.

Case

A 28-year-old male was referred to our emergency department on November 25, 2024, after consuming homemade alcohol and an unknown stimulant, leading to altered consciousness. Initial vital signs were: BP 130/90 mmHg, HR 105 bpm, SpO□ 99%.

He was disoriented and uncooperative, with absent pupillary light reflexes. There was no marked extremity weakness. Glasgow Coma Score (GCS) was 8 (E3M4V1). Respiratory and abdominal examinations were unremarkable. The patient was intubated for airway protection.

Arterial blood gas analysis revealed pH 7.28, HCO□□ 10.9 mmol/L, PCO□ 12.5 mmHg, PO□ 139 mmHg, lactate 4.8 mmol/L. Serum tests showed Na 141 mmol/L, K 4.2 mmol/L, Cl 103 mmol/L, glucose 146 mg/dL, creatinine 0.91 mg/dL, BUN 6.54 mg/dL. Serum osmolality was 391 mOsm/kg; ethanol level was <10 mg/dL. Osmolar gap was 99 mOsm/kg; anion gap was 28 mEq/L. Toxicology screening was negative.

Given the history and high anion/osmolar gaps with negative ethanol levels, methanol poisoning was suspected and treatment was initiated. Emergent hemodialysis was planned after nephrology consultation. Fomepizole was unavailable, so intravenous ethanol was used.

Cranial CT showed bilateral hemorrhagic lesions in the putamen. Neurosurgical consultation was requested. No neurosurgical intervention was indicated. The patient was admitted to the ICU. Despite full supportive care, the patient suffered cardiac arrest and died on November 29, 2024.

Dıscussıon

Methanol toxicity is rare in emergency settings but can be fatal if not promptly diagnosed and treated. The classic triad includes metabolic acidosis, visual disturbances, and CNS depression, although these may not always be synchronous.

High anion gap (>16 mEq/L) and high osmolar gap (>10 mOsm/kg), along with low ethanol levels and lactic acidosis, help distinguish methanol from other toxic alcohols. Bilateral putaminal lesions are a specific neuroimaging marker of methanol poisoning and are linked to severe neurological sequelae and death.

Treatment focuses on inhibiting methanol metabolism (fomepizole or ethanol), removing formic acid (dialysis), and enhancing its metabolism (folate supplementation). While fomepizole is ideal, ethanol is commonly used due to its wider availability in many countries.

Hemodialysis is indicated in cases with pH <7.25, GCS <10, visual symptoms, or serum methanol >50 mg/dL. Folinic acid may help preserve vision by accelerating formic acid metabolism.

Despite timely dialysis and ethanol administration in this case, severe CNS damage led to fatal outcome, emphasizing that even early interventions may not prevent death in advanced stages.

Figure 1

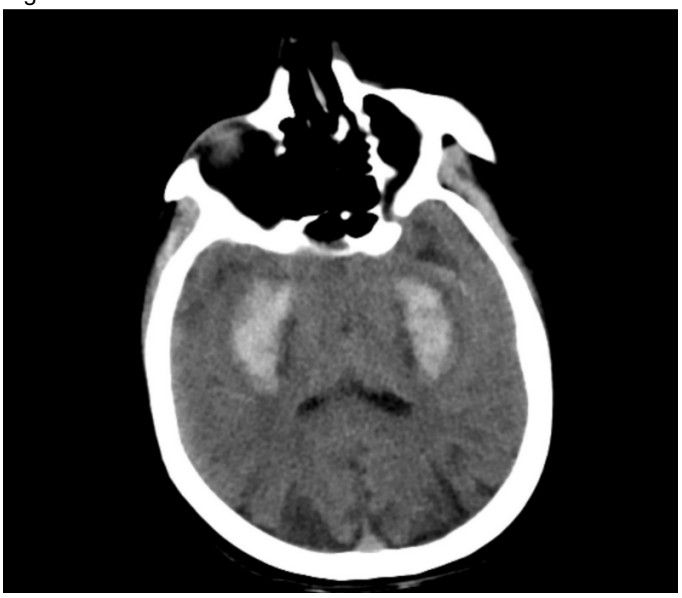
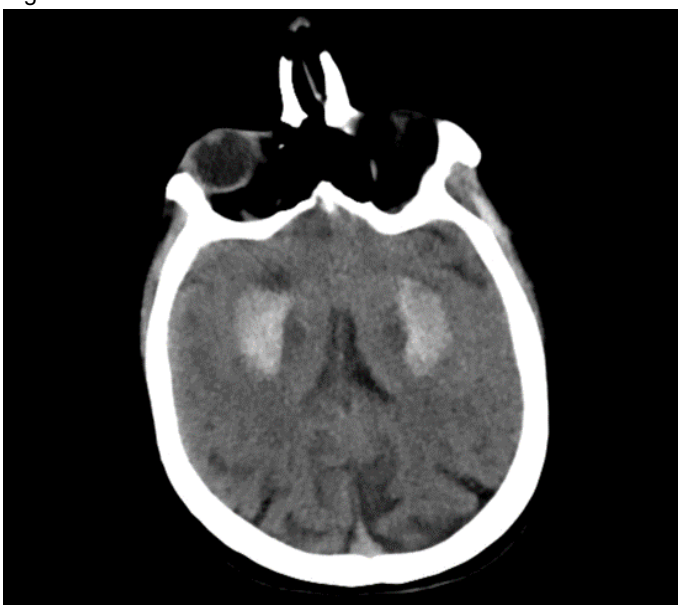


Figure 2



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1942

Fournıer's Gangrene: A Sılent Threat Wıth Fatal Consequences

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Introductıon

Fournier's gangrene is a rare but highly lethal infectious disease characterized by rapidly progressing necrotizing fasciitis in the perineal, genital, and perianal regions (1). It typically has a polymicrobial etiology, involving both aerobic and anaerobic microorganisms (2). The disease can cause both local tissue destruction and systemic complications, posing a life-threatening risk. Fournier's gangrene is most commonly observed in individuals aged 50-60 years and is approximately ten times more frequent in men than in women (3). The most common etiological factors include anorectal, urogenital, or dermatological infections (4). These infections are

often bacterial in origin and progress through the synergistic action of aerobic and anaerobic microorganisms (5). Several predisposing factors contribute to disease development, including diabetes mellitus, alcoholism, obesity, peripheral vascular disease, immunodeficiency, chronic renal failure, and malnutrition (6). Additionally, local factors such as trauma, urogenital surgical procedures, and perianal abscesses can trigger the disease (7). Fournier's gangrene typically begins with perineal pain, swelling, erythema, and tenderness (8). In advanced cases, subcutaneous crepitus, foul-smelling discharge, tissue necrosis, and systemic toxicity may occur. As the infection progresses, sepsis and multiple organ failure can develop, significantly increasing mortality rates (5).

Case

A 49-year-old male patient who presented with redness and pain in the groin area for about a week. According to the patient, the symptoms initially started mildly but worsened over the past two days, spreading particularly to the scrotum. He reported increasing pain, swelling, and tenderness in the scrotal region. The patient's medical history included type 2 diabetes mellitus, diagnosed 10 years ago, and he was on oral antidiabetic therapy. He had no history of hypertension or known cardiovascular disease. Physical examination revealed a markedly edematous and hyperemic scrotum. Tenderness was present on palpation, with a noticeable increase in skin temperature. A necrotic lesion measuring approximately 4x10 cm was observed in the lower scrotal region, with disrupted skin integrity. A foul-smelling, sero-hemorrhagic discharge was noted from the area. Subcutaneous crepitus was detected, suggesting the presence of gas-forming infection. Scrotal ultrasonography revealed significant subcutaneous edema and gas densities. The patient was urgently consulted with the urology department for suspected Fournier's gangrene and was admitted for treatment.

Figure 1: Edematous scrotum

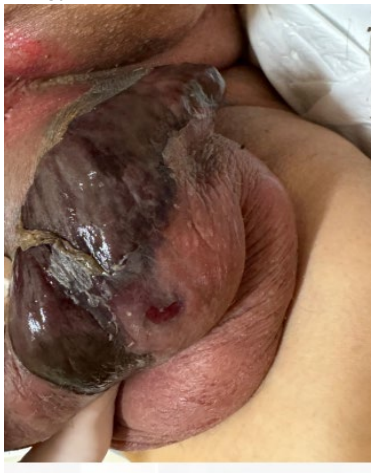

Figure 2: 4x10 cm necrotic area in the scrotum

Discussion

Our case shares similarities with reported cases in the literature in terms of risk factors for Fournier's gangrene and the rapid progression of the disease (2). Initially presenting with groin redness and pain, the rapid spread of symptoms to the scrotum highlights the aggressive nature of the infection (3). As observed in previous cases, early diagnosis and immediate surgical debridement are crucial in managing Fournier's gangrene (4). However, in some cases, delayed diagnosis has been associated with high mortality rates (5). Early detection and intervention in our patient may help prevent severe complications such as sepsis and multiple organ failure (7). Fournier's gangrene is a rapidly progressing infection requiring aggressive surgical debridement and broad-spectrum antibiotic therapy (8).

Conclusion

In emergency departments, particularly in crowded outpatient settings, examining patients with complaints such as genital pain, swelling, erythema, and discharge is often challenging. However, as demonstrated in our case, regardless of conditions, a thorough physical examination is crucial to protect patients from potentially fatal outcomes. The primary purpose of this case report is to emphasize the importance of physical examination and to remind clinicians that conditions like Fournier's gangrene, which can lead to fatal consequences, can be diagnosed solely through clinical examination.

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1973

Serotonin syndrome due to high dose SSRI intake: a case report

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Yüksek doz SSRI alımına bağlı gelişen serotonin sendromu: olgu sunumu

Introduction

Serotonin syndrome (SS), also known as serotonin toxicity, is a life-threatening emergency caused by increased serotonergic activity (via 5-HT receptors) at synapses in the central and peripheral nervous systems. The cause is usually drug-induced (antidepressants, selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), monoamino oxidase inhibitors (MOAIs), etc.). It may occur after use at therapeutic doses, after interactions with other drugs, or after use of high doses for suicidal purposes (1).

Clinical symptoms are classified into three groups: mental, autonomic and neuromuscular, and occur within the first 6-24 hours after exposure (2). Although some cases may be fatal, most recover after discontinuation of the drug (3). This syndrome, whose early diagnosis and treatment is crucial, may not be diagnosed by most physicians due to lack of experience (4). With the case we presented, we wanted to give doctors more information about this syndrome.

Case Presentation

A 27-year-old male patient with a history of hepatitis C and drug addiction was brought to the emergency department (ED) by his family after taking 2700 mg sertraline tablet, 4000 mg fenprobamate tablet, 2500 mg chlorzoxazone tablet, 250 mg dexketoprofen tablet for suicide approximately 6 hours earlier.

On presentation to the ED, consciousness was confused, agitated, disoriented, uncooperative, and the Glasgow Coma Score (GCS) was 14. Vital signs were temperature: 37°C, blood pressure: 160/90mm/Hg, pulse: 120/min, respiratory rate: 25/min, saturation (room air): 96%. Physical examination revealed diaphoresis, flushing, tachypnoea, tachycardia, bilateral mydriatic eyes, no light reflex and diffuse tremor in all extremities. Muscle strength was complete in all four extremities and there was no evidence of lateralisation. Other systemic examinations were normal. Blood gases were pH 7.23, pCO_2:28.7 mmHg, HCO_3:11.8 mmol/L, pO_2:87 mmHg, sO2:96, Lac:1.87 mmol/L, BE(B): -13.9. Glucose, electrolytes, liver and renal function tests, C-reactive protein, complete urinalysis and haemogram were normal, and the blood ethanol test was negative. Toxic substance ingestion was excluded by urine toxicology. Creatine kinase (CK) was found to be minimally elevated at 190 (10-171 U/L).

The ECG showed sinus tachycardia. The patient was monitored in the emergency department. He was hydrated by opening an intravenous line. Activated charcoal was administered by nasogastric catheter (1mg/kg). 114 Toxicology recommendations were obtained. No neurological, endocrine, metabolic or infectious diseases were considered. As a result of her history and current examination findings, she was admitted to the emergency intensive care unit (ICU) for follow-up with a diagnosis of serotonin toxicity. During follow-up in the ICU, involuntary movements such as hyperreflexia and spontaneous myoclonus were noted. Therefore, intravenous diazepam (10 mg/day) was administered. On the 2nd day in ICU, despite hydration, metabolic acidosis (pH 7.27, pCO_2:29.3 mmHg, HCO_3:12.1 mmol/L, Lac:2.5 mmol/L, BE(B): -15) and CK remained elevated, so he was put on dialysis. On the 3rd day, agitation was reduced, CK returned to normal, myoclonic contractions and acidosis improved. The dose of diazepam was reduced and discontinued. The patient, who made a complete recovery during the follow-up, was seen by the psychiatry department and discharged on day 5 with a recommendation for outpatient monitoring.

Discussion

This syndrome is rarely observed following the use of low-dose SSRIs or high-dose and/or concomitant use of serotonergic agents (2,5). It may also occur as a result of concomitant use of drugs that interact pharmacokinetically or pharmacodynamically with serotonergic agents (6). Although early symptoms are usually mild, cases that are not correctly diagnosed can deteriorate rapidly. These patients may be misdiagnosed because of their non-specific symptoms. Although SS shares similarities with other syndromes, it can be easily differentiated using the Hunter criteria (7). The Hunter criteria are characterised by symptoms such as clonus, restlessness, diaphoresis, tremor, hyperreflexia, hypertonia and fever. Most of these symptoms vary according to the severity of the toxicity (8). In our case, agitation, diaphoresis, tremor, hyperreflexia and clonus were present according to Hunter's criteria (9).

No symptom or laboratory finding is diagnostic (10). SS is associated with cognitive and behavioural (confusion, disorientation, agitation, irritability, coma, anxiety, hypomania, lethargy, seizure, insomnia, hallucinations, stupor), autonomic (hyperthermia, excessive sweating, sinus tachycardia, hypertension, tachypnea, mydriasis, tremor, clonus), and psychiatric (psychiatric, psychiatric, psychiatric, psychiatric, psychiatric, psychiatric, psychiatric, psychiatric, psychiatric, psychiatric, psychiatric tachypnoea, mydriasis, non-reactive pupils, skin erythema, hypotension, diarrhoea, abdominal cramps, hypersalivation) and neurological (myoclonus, hyperreflexia, rigidity, tremor, ataxia, bilateral Babinski sign, nystagmus) signs and symptoms (3). In most cases, symptoms are reported within 24 hours of taking a serotonergic agent and are observed within the first 6 hours in approximately 60% of patients (11). Our case also presented with mild to moderate symptoms that developed in the first 6 hours after taking analgesics and myorelaxants together with high-dose SSRIs with suicidal intent (5,9).

To make the diagnosis of SS, a history of serotonergic drug use, clinical findings and other diseases should be excluded. The differential diagnosis of SS includes neuroleptic malignant syndrome (NMS), dystonic reaction, hyperthyroidism, tetanus, malignant hyperthermia and toxicity of some drugs (cocaine, amphetamines, lithium, etc.). Although there are common clinical symptoms between these conditions, there are specific findings for the diagnosis of SS (9). SS is most often misdiagnosed as NMS. The most

important findings that differentiate the two syndromes are the presence of clonus and hyperreflexia in SS, and lead pipe rigidity and high fever in NMS (12). In our case, clonus and hyperreflexia were present, but NMS was excluded because of the absence of rigidity and fever (7). NMS, which is thought to be caused only by dopamine receptor antagonism, is a reaction to antipsychotic drugs. It develops a few days after taking the drug and patients take weeks to recover. However, when we look at the literature, SS starts between 1 day and 1 week after taking interacting drugs together. In most cases, it has been observed that the symptoms resolve within 2-7 days after discontinuation of these drugs (13). In our patient, who had no history of antipsychotic use, symptoms appeared within the first 6 hours and resolved within 3 days (14).

Management of SS depends on the case and the severity of toxicity. Severe cases should be followed in the intensive care unit (10). There is no specific antidote for serotonin toxicity. Non-specific serotonin receptor blockers such as benzodiazepines, propranolol, chlorpromazine, cyproheptadine and methysergide may be used (14). The first stage of treatment includes early diagnosis, removal of the offending drugs and withdrawal of the serotonergic agent (12). In severe cases, intubation, mechanical ventilation and paralysis should be considered. Autonomic instability, hypertension and hyperthermia should be controlled. Antipyretics and dantrolene should not be used. Vital signs are closely monitored and intravenous fluid/electrolyte support is given as necessary (2,3). Benzodiazepines are the mainstay of treatment in most cases because of their non-specific inhibitory effects on serotonergic transmission (9). Our patient, who was diagnosed with AS at an early stage and was treated with sedation and supportive care in the ICU after administration of activated charcoal, gradually recovered within 3 days.

Conclusion

SS is a rare but life-threatening complication of antidepressants. It should be considered in patients with AS who present with altered consciousness, tremor, hyperreflexia and clonus shortly after taking therapeutic or toxic doses of SSRIs. It is most commonly misdiagnosed as NMS. History, physical examination and Hunter criteria are used to differentiate from other conditions. Early diagnosis, drug withdrawal and supportive care are the most effective treatment modalities. SSRIs should be carefully monitored when used alone or in combination with other medications.

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1996

The Effect Of Lar Ratıo On Mortalıty In Posttraumatıc Subdural Hemorrhage

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Subdural hemorrhage is a hemorrhage between the dura mater and the arachnoid membrane, which surround the brain. They can be classified as acute, subacute and chronic. Acute hemorrhages are often traumatic. Early recognition and treatment is important. If not treated appropriately, they have high mortality and morbidity. There are some markers and indices to make the decision for intensive care unit hospitalization and to predict mortality. Posttraumatic subdural hemorrhage is a condition that causes serious mortality and morbidity (1-3). Clinically, it presents with very different symptoms. The most common symptom is headache (4). The most

effective imaging method in the diagnosis is computed tomography (5). Predicting the mortality rate of subdural hemorrhage, which is a serious cause of mortality, is uncertain for physicians.

The aim of our study was to determine the importance of lymphocyte/albumin(s) ratio in mortality prediction.

Materıal Method

The data of adult patients over 18 years of age who were admitted to the emergency department of Malatya Training and Research Hospital with a diagnosis of subdural hemorrhage in the last 1 year were retrospectively analyzed. 30-day mortality and prognosis estimation of blood parameters [hemogram:leukocyte(10⅜uL) (WBC),neutrophil(10/uL) (neu),
lymphocyte(10¾uL) (lym), biochemistry: albuming/dL) (alb), LAR index importance and whether there are inflammatory parameters that can be used in prognosis estimation. In total, 58 patient data were accessed and our study was conducted with the data of 46 patients who met the inclusion and exclusion criteria.

Inclusion criteria:

Patients over 18 years of age with a diagnosis of subdural hemorrhage and hospitalization

Patients admitted to our hospital between 2024 and 2025 and hospitalized with a diagnosis of subdural hemorrhage, whose data were retrospectively obtained from the hospital information system

Exclusion criteria

Patients under eighteen years of age

Patients whose data cannot be fully accessed in the hospital data system

All patients on anticoagulant and coagulant therapy

Patients who were referred to different hospitals from the emergency department or who refused hospitalization voluntarily and had a previous diagnosis of subdural hemorrhage were excluded from the study.

Statistical Analysis

IBM SPSS statistics 27 program was used for statistical analyses while evaluating the findings obtained in the study. The conformity of the parameters to normal distribution was evaluated by shapiro wilk test. Descriptive statistical methods were given as mean, median, standard deviation, percentage (25-75% (Inter Quantile Range-IQR) and frequency. When comparing quantitative data, student t test was used for comparisons of normally distributed parameters between two groups and mann whitney u test was used for comparisons of non-normally distributed parameters between two groups. For quantitative data (blood parameters and inflammatory indices), correlation analysis was performed to determine the relationship with length of intensive care unit stay and length of hospital stay. Our study was planned retrospectively and all patients who met the inclusion and exclusion criteria were includeds

Fındıngs

Initially 58 patients were included in our study. Forty-six patients who met the inclusion criteria were included. Shaporowilk normality test was performed.

21.7% (10) of our patients were female and 78.3% (36) were male. While 82.6%(38) of our patients survived, 17.4%(8) of them died. All of our patients were followed up in intensive care unit.

The mean age of our patients was 77.5 years (72.5-84.2) and blood values were respectively:
WBC(10¾uL) 8.4 (7.1-11.0), neutrophil(10%/uL) 6.2 (4.5-9.6), lymphocyte(10%uL) 1.4 (0.8-
1.9), albuming/dL) 3.55 (3.15-3.85), and are given in Table 1.

Table 1.Median and IQR (Inter Quantile Range) values of blood parameters with nonparametric distribution

	Median	IQR
WBC(10^3/uL)	8,4	7,1-11,0
Neu(10^3/uL)	6,2	4,5-9,6
Lym(10^3/uL)	1,4	0,8-1,9
ALB(g/dL)	3,55	3,15-3,85
LAR	0,46	0,38-0,66

When our patients were grouped into survivors and ex-patients, alb values were lower in ex-patients. Blood parameters of the patients are given in Table 2.

Table 2.Association of survivors and ex-patients with mortality

		n	p valeu
WBC	alive	38	0,9**
	ex	8	
Neu	alive	38	0,79**
	ex	8	
Lym	alive	38	0,62**

	ex	8	
Alb	alive	38	0,036**
	ex	8	
LAR	alive	38	0,839**
	ex	8	

**Mann Whitney U test, yb: intensive care unit

The correlation values for albumin and LAR were (p=0.046,r=-0.296) for albumin and (p=0.255,r=0.171) for LAR and are given in Table 3.

Table 3.Association of blood parameters and LAR score with hospital and intensive care unit length of stay

Parameters	n	r	p Valeu	Parameters	n	r	p Valeu
Alb	HASTKALIS	-0,435	0,003**	Alb	YBKALIS	-0,296	0,046*
LAR		0,176	0,242	LAR		0,171	0,255

*. Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).

Discussion

Subdural hemorrhage is more common in the male gender (1). In our study model, the literature was similar and the male gender was predominant. The fact that the male gender is more active in social life than the female gender in our country may explain this difference in gender.

Senol et al. reported in a study that patients with subdural hemorrhage were compatible with the age group of geriatric patients (4). The mean age of our patient group was 77.5 years which was compatible with the literature. In geriatric patients, rupture of the bridge veins which are stretched secondary to brain atrophy and loss of the properties and flexibility of the veins may explain the predominance of subdural hemorrhage in the geriatric patient age group.

It is a known fact that albumin, which is a negative acute phase reactant, is low in traumatized patients or in patients with poor general condition. In our study, low levels of albumin were found in patients with mortal course.

No study was found between LAR ratio and mortality in subdural hemorrhage. Although the LAR rate was not statistically different in patients with mortal outcome, the LAR rate was higher in patients with mortal outcome, the LAR rate was higher in patients with mortality.

We think that future studies with large patient groups will provide a new scale that can be used in the prediction of mortality with LAR.

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2002

Severe Necrotizing Soft Tissue Infection Following A Human Bite In An Elderly Woman, Complicated By Pulmonary Embolism: A Case Report

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Introduction

Human bites account for a relatively small proportion of bite wounds, yet they carry a disproportionately high risk of infection and severe morbidity (1–3). This heightened risk is attributable to the dense polymicrobial inoculum of human saliva and the tendency for patients to present late, after infection is established (4). Indeed, human bite wounds often appear minor initially, leading to underes-

timation; however, without prompt treatment they can progress to serious complications including deep space infections, septic arthritis, or even systemic infection (2). Necrotizing fasciitis is a rare but life-threatening complication of bite injuries, characterized by rapidly spreading necrosis of subcutaneous tissue and fascia (5). While necrotizing soft tissue infections (NSTIs) are uncommon in otherwise healthy individuals, bite-associated NSTIs have been documented, particularly in patients with risk factors such as diabetes, immunosuppression, or advanced age (6,7). These infections require urgent surgical intervention and carry high mortality if not recognized early (5).

We present the case of a 70-year-old woman with asthma who developed fulminant necrotizing soft tissue infection in her forearm after a seemingly innocuous human bite from her young grandchild. The case is notable for its atypical source (a bite by a child), the aggressive course requiring multiple surgeries, and the occurrence of a major thromboembolic complication (pulmonary embolism) during recovery. Through this case, we aim to highlight the clinical features and management of human bite infections progressing to necrotizing fasciitis, review appropriate antibiotic and surgical strategies, and discuss the need for multidisciplinary care and complication surveillance. This report adds to the growing literature emphasizing that no human bite should be dismissed as trivial, especially in high-risk hosts, and underscores key lessons for emergency and critical care management of such infections.

Case Presentation

Patient Information: A 70-year-old woman with a history of chronic asthma (on inhaled bronchodilators) presented to the emergency department with a severely infected wound on her left forearm. Four days prior, she had sustained a bite injury to that arm from her 3-year-old grandchild during play. Initially, the bite was small and the patient did not seek immediate care, but over the subsequent days she developed progressive swelling and pain in the arm.

Clinical Findings: On examination at presentation, the left forearm was markedly swollen with tense, erythematous skin and areas of dusky discoloration. Widespread hemorrhagic bullae were noted over the bite region, and underlying soft tissue crepitus was suspected. The wound area had patches of black necrosis and foul, purulent discharge, indicating a deep infection (Figure 1). Despite the alarming local findings, the patient was alert with only mild fever (37.8°C) and a blood pressure of 110/70 mmHg. She reported severe pain in the arm but denied any other focal symptoms. Distal pulses in the hand were palpable, and there were no motor or sensory deficits in the limb. Systemic examination was otherwise unremarkable, and there were no signs of end-organ dysfunction at that time.

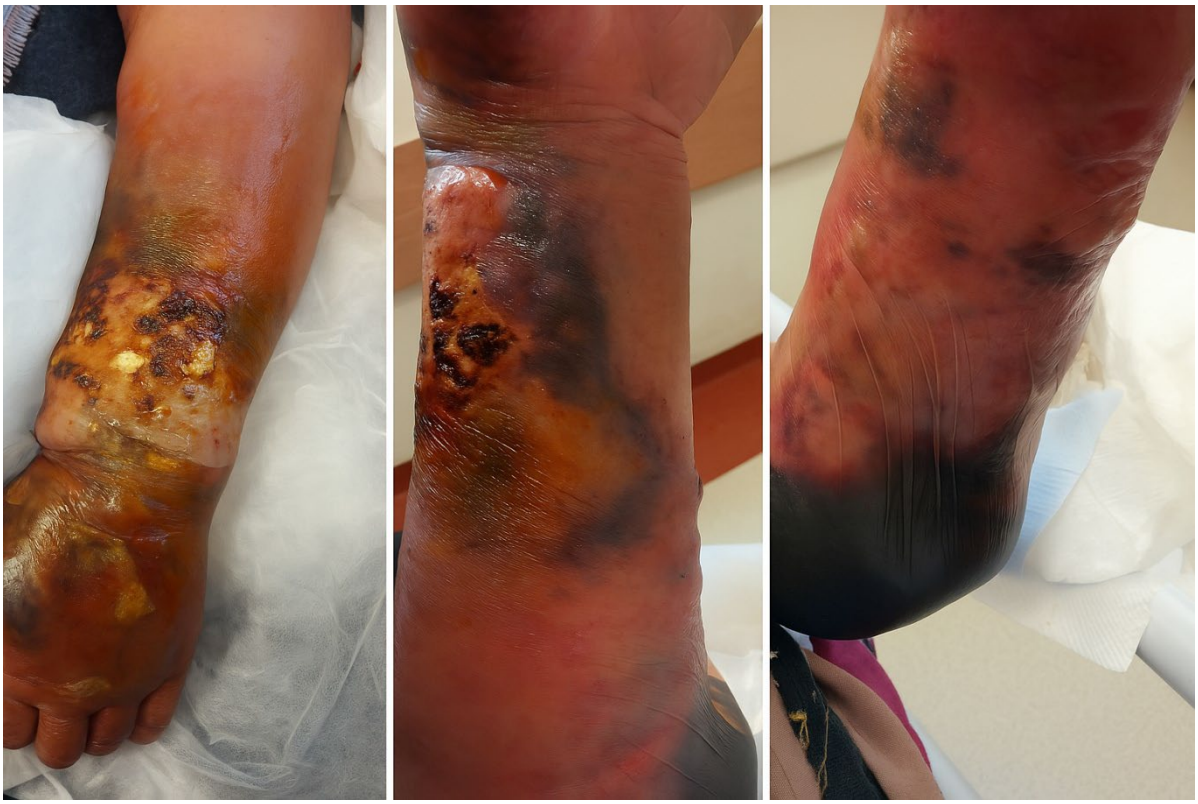

Figure 1. Marked swelling of the left forearm, with skin tension, erythema, and patchy areas of dusky discoloration, widespread hemorrhagic bullae over the bite region, black necrotic patches, and foul-smelling purulent discharge indicative of deep infection.

Initial Investigations: Laboratory results revealed a profound inflammatory response. C-reactive protein was 394 mg/L (normal <5) and procalcitonin 3.36 ng/mL, consistent with severe infection. However, the white blood cell count was 5.0×10^9/L, which is within normal range, possibly reflecting an impaired immune response due to age or early consumption of leukocytes. Notably, the patient had hyponatremia (Na 125 mmol/L) and slightly prolonged coagulation indices (INR 1.47, PTT 39.1 seconds), suggesting systemic inflammatory effects. Platelets were mildly reduced (131×10^9/L). Blood lactate was not initially measured, but high inflammatory markers pointed toward sepsis. Plain radiographs of the forearm showed soft tissue swelling without bony injury. An urgent contrast-

enhanced computed tomography (CT) of the left forearm was obtained to evaluate the extent of infection. The CT revealed diffuse edema and heterogeneous fluid collections in the subcutaneous tissues tracking from the distal forearm toward the elbow (approximately 3–4 cm in length), with gas present in the soft tissues. There was no bone destruction or foreign body. These radiologic findings were consistent with necrotizing fasciitis and abscess formation. Given the clinical and imaging picture, a diagnosis of necrotizing soft tissue infection (likely polymicrobial necrotizing fasciitis) was made.

Emergency Management: The patient was started on broad-spectrum antibiotics immediately upon recognition of a severe soft tissue infection, even before culture results. Intravenous piperacillin-tazobactam (4.5 g every 8 hours) was initiated to cover gram-positive, gram-negative, and anaerobic organisms common in bite wounds. In addition, IV teicoplanin (loading dose 600 mg twice on day 1, then 600 mg daily) was given to ensure coverage of methicillin-resistant Staphylococcus aureus (MRSA) and other gram-positive pathogens. Tetanus toxoid and tetanus immunoglobulin were administered given the nature of the injury. Aggressive fluid resuscitation and analgesia were provided. The surgical team (Plastic and Reconstructive Surgery) was consulted emergently, and the patient was transferred to the operating room on the day of admission for exploratory surgery and source control.

Surgical Intervention: The patient underwent urgent surgical exploration and debridement of the left forearm under regional anesthesia. Intraoperative findings confirmed extensive necrotizing fasciitis, with approximately 10×10 cm of devitalized dorsal forearm skin and underlying necrotic subcutaneous tissue and fascia. Purulent fluid and gas were encountered in the wound. A fasciotomy was performed to relieve compartment pressure, and all necrotic tissue was excised back to healthy, bleeding margins. Tissue samples were sent for culture and histopathology. The wounds were left open with antiseptic dressings, and the patient was transferred to the intensive care unit (ICU) for postoperative monitoring and care.

ICU Course and Further Management: In the ICU, the patient remained on broad antibiotics and supportive care. Teicoplanin and piperacillin-tazobactam were continued as per infectious disease recommendations. She also received albumin infusions for hypoalbuminemia, and vasopressor support was not required as her blood pressure remained stable. Daily wound inspections were performed. Repeat surgical debridements were done on hospital day 3 and day 11 due to persistent areas of necrosis identified at the wound margins, which is common in necrotizing fasciitis to ensure all devitalized tissue is removed. Wound cultures from the initial and subsequent surgeries surprisingly showed no bacterial growth; this was attributed to prior antibiotic administration. Histopathology of the excised tissue was consistent with acute severe soft tissue infection (necrotizing fasciitis).

Throughout this period, the patient's respiratory status and hemodynamics were stable. She required bronchodilator therapy for her asthma and received low-dose systemic corticosteroids (intravenous dexamethasone 8 mg daily) in the ICU for possible relative adrenal insufficiency and to mitigate airway inflammation. Importantly, venous thromboembolism (VTE) prophylaxis with subcutaneous enoxaparin (40 mg twice daily, adjusted dose for her body weight) was instituted early, given her reduced mobility and the prothrombotic state associated with infection. A vascular surgery consult had been obtained on admission because her arm ultrasound showed thrombophlebitis in superficial veins around the infected area; this was managed with limb elevation and the same anticoagulation prophylaxis.

By day 16, the infection was under control and the patient was transferred out of the ICU to the surgical ward. The forearm wound was being managed with daily antiseptic dressings. Laboratory markers of infection steadily improved (CRP down to 50 mg/L by the third week).

Complication – Pulmonary Embolism: On hospital day 23 (7th day on the ward), the patient developed sudden onset of pleuritic chest pain and acute dyspnea. Her oxygen saturation dropped to 65% on room air, improving only to 80% with high-flow oxygen. Tachycardia (126/min) and mild hypotension (BP 100/70 mmHg) were noted, with an otherwise normal sinus rhythm on ECG. Given the acute presentation in a postoperative, infection-recovery setting, pulmonary embolism (PE) was suspected despite ongoing prophylaxis. An urgent CT pulmonary angiogram was performed, which revealed filling defects in branches of the right pulmonary artery to the middle and lower lobes, confirming an acute PE. Bilateral minimal pleural effusions and basal atelectasis were also noted. The patient was transferred back to the ICU for closer monitoring and management of the PE. Full-dose anticoagulation was initiated with enoxaparin (~60 mg subcutaneously twice daily). A cardiology evaluation showed a normal cardiac ultrasound aside from mild right heart dilatation, and no evidence of heart failure. Given her asthma history, a pulmonology consult was also obtained; inhaled budesonide was started for bronchospasm, and supplemental oxygen was administered. Over the next 48 hours, her respiratory status improved on anticoagulation and supportive care.

Further Hospital Course: After stabilization of the PE, the patient spent an additional 5 days in ICU, then returned to the ward. Broad-spectrum antibiotic therapy was revised on infectious disease consultation: due to the prolonged course and concerns about resistant organisms, the regimen was switched to meropenem (1 g every 8 hours) plus linezolid (600 mg every 12 hours), and piperacillin-tazobactam with teicoplanin were discontinued. The wound continued to show healthy granulation tissue after the serial debridements. On hospital day 37, a fourth surgical debridement was performed to remove a small residual area of non-viable tissue, and at that time a decision was made to perform reconstruction of the soft tissue defect. A split-thickness skin graft was harvested from the lateral thigh and applied to the forearm wound, which measured approximately 8×6 cm after all debridements. A negative-pressure wound therapy dressing (VAC) was placed over the graft to promote adherence.

In the fourth week, the patient had a brief episode of hospital-acquired pneumonia evidenced by a new cough with green sputum on day 39. Sputum cultures grew Pseudomonas aeruginosa (susceptible to meropenem). With the addition of an inhaled mucolytic (N-acetylcysteine 1200 mg daily) and continued antibiotics, this was managed without progression to severe pneumonia. A short course of diuretics was given for the pleural effusions. The patient also experienced transient episodes of atrial fibrillation with rapid ventri-

cular response (heart rate ~124 bpm), likely due to infections and stress; cardiology recommended a low-dose beta-blocker (metoprolol 50 mg daily) and low-dose aspirin for rate control and stroke prophylaxis given her age, alongside therapeutic enoxaparin.

Outcome: By hospital day 45, the patient had markedly improved. Her forearm graft was taking well with no signs of residual infection. Inflammatory markers normalized (CRP <10 mg/L) and she was afebrile, breathing comfortably on room air with oxygen saturation ~91%. She had no further chest pain or respiratory difficulty. The left arm was healing; although she had some stiffness of the wrist and fingers, physical therapy was initiated to improve range of motion. The patient was discharged home on day 45 with instructions for wound care and follow-up. At discharge, she was transitioned from enoxaparin injections to oral rivaroxaban for a planned total of 6 months of anticoagulation for the pulmonary embolism. She was also prescribed oral amoxicillin-clavulanate to continue antibiotic coverage for an additional week as a precaution, and given inhalers for her asthma.

Discussion

This case demonstrates the severe consequences a human bite can have in an elderly patient, evolving into necrotizing fasciitis and systemic complications. Human bite wounds are often polymicrobial and notoriously prone to infection; even a small bite can seed a mix of bacteria leading to aggressive soft tissue infection (2). In our patient, the combination of an inoculum of oral flora and a delayed presentation created the perfect setting for a necrotizing soft tissue infection. Similar instances have been reported in the literature. For example, Li et al. describe a 68-year-old man who sustained a seemingly minor forearm bite from a toddler, which within 24 hours progressed to gas gangrene-like necrotizing fasciitis and required an emergency arm amputation (7). That patient had underlying myelodysplastic syndrome (evolving to leukemia), highlighting how immunocompromise can predispose to such fulminant infections. Our patient, while not overtly immunosuppressed, was 70 years old and possibly had an attenuated immune response due to age; notably, she did not mount a high leukocyte count despite extensive infection. Advanced age and comorbid conditions (like asthma requiring steroids) may reduce the ability to contain infection, allowing it to spread unchecked.

Microbiology: Human bite infections typically involve a polymicrobial mix of organisms from the biter's saliva and the victim's skin flora (2). Commonly isolated bacteria include Staphylococcus aureus (including MRSA), various streptococci (especially viridans streptococci), and anaerobes such as Eikenella corrodens, Fusobacterium, Prevotella, and Peptostreptococcus (2). Eikenella corrodens, a gram-negative facultative anaerobe, is particularly associated with human bites (especially clenched-fist "fight bites") and is inherently resistant to certain antibiotics like clindamycin and first-generation cephalosporins (2). In our case, no organism grew on culture, likely due to early empiric antibiotic administration. However, the clinical presentation and course suggest a polymicrobial necrotizing infection. Broad coverage was necessary to address the possible mix of streptococci (which can drive toxin-mediated tissue destruction) and anaerobes from oral flora. It is worth noting that group A Streptococcus is a common cause of monomicrobial necrotizing fasciitis in other settings, but bite-induced cases more often involve multiple organisms (Type I NSTI), or occasionally unusual pathogens (a prior case identified Aeromonas hydrophila in a bite wound, possibly from oral contamination with water). Regardless of the specific pathogen(s), the management principles remain the same.

Management: Early and aggressive management of necrotizing infection is paramount. Guidelines emphasize prompt surgical debridement as the cornerstone of therapy, combined with broad-spectrum antibiotics and intensive supportive care (5,8). Our approach was in line with these recommendations. We initiated empiric antibiotics covering aerobic and anaerobic bacteria as soon as necrotizing fasciitis was suspected. For less severe bite wounds without necrosis, amoxicillin-clavulanate is generally the first-line antibiotic therapy. In the hospital setting, ampicillin-sulbactam is a common IV equivalent. However, in a rapidly spreading infection with systemic signs, broader coverage is warranted. IDSA guidelines for necrotizing fasciitis advise empiric therapy such as vancomycin (or linezolid) plus piperacillin-tazobactam, or a carbapenem, to cover MRSA, gram-negatives, and anaerobes (8). Our patient received teicoplanin (a glycopeptide similar to vancomycin) plus piperacillin-tazobactam, which is an appropriate regimen consistent with these recommendations. This combination likely helped control the infection early, even though definitive culture guidance was lacking. The subsequent switch to meropenem and linezolid was a logical step to prevent resistance and ensure continued broad coverage during her prolonged ICU stay.

In total, four surgical debridements were performed, reflecting the often iterative process of managing NSTIs – repeated exploration is frequently needed until all necrotic tissue is removed and the infection is fully controlled. Aggressive surgical management significantly improves survival in necrotizing fasciitis (9). In our case, early fasciotomy was also performed due to concern for compartment syndrome in the swollen forearm; this likely prevented further ischemic damage. The collaboration of emergency physicians, surgeons (plastic/reconstructive and orthopedic as needed), intensivists, and infectious disease specialists was crucial in this case, illustrating the recommended multidisciplinary approach to complex infections (9). Close cooperation among specialties facilitates timely decisions – for instance, surgical teams can perform life-saving debridements while intensivists manage sepsis and organ support, and infectious disease experts tailor antimicrobial therapy (9).

Complications: Despite optimal care, our patient developed a major complication – a pulmonary embolism. Patients with severe infections and prolonged immobilization are at significant risk for venous thromboembolism. The systemic inflammatory state in sepsis can induce a hypercoagulable condition, sometimes termed sepsis-associated coagulopathy. A recent analysis of millions of sepsis cases reported acute PE in approximately 2.3% of patients with septic shock (10), highlighting that infection itself is a risk factor for thrombotic events. Our patient received standard prophylaxis, yet still developed a PE in the third week of hospitalization. This underscores that even with prophylactic anticoagulation, high-risk patients can sustain thromboses; thus, a high index of suspicion for PE is needed when clinical signs arise. Fortunately, the PE was recognized early and managed with therapeutic anticoagulation, and the patient had no long-term cardiopulmonary sequelae. This case suggests consideration of enhanced VTE prophylaxis

strategies in patients with NSTIs (for example, intermediate-dose anticoagulation in those with multiple risk factors), though such decisions must be balanced against bleeding risk on a case-by-case basis.

Another aspect illustrated by this case is the importance of supportive care and monitoring for other organ systems. Our patient's course was complicated by transient bacteremia (presumed given the high procalcitonin, though blood cultures were negative) and a mild hospital-acquired pneumonia, which were managed with adjustments in antibiotics and respiratory therapy. The use of adjunctive therapies in necrotizing fasciitis (such as intravenous immunoglobulin for streptococcal toxic shock, or hyperbaric oxygen therapy) was considered but deemed unnecessary in this case after improvement with standard care. Throughout her ICU stay, meticulous attention was given to fluid balance, renal function, and nutrition (including albumin supplementation to address hypoalbuminemia common in critical illness).

Outcomes and Follow-Up: Thanks to prompt surgical and medical intervention, the patient survived a condition that can be fatal in up to 20–40% of cases, according to various series (8). Limb function was preserved, albeit with expected scarring and some stiffness requiring rehabilitation. The decision to perform a skin graft at the later stage provided wound closure that likely sped up recovery; earlier closure is usually deferred until infection is clearly resolved. On follow-up at 2 months post-discharge, the patient's grafted forearm had healed well, and she had regained useful function of her hand. She remained on rivaroxaban for anticoagulation and had no recurrence of thromboembolic events. Her case underscores that with aggressive management, even elderly patients with necrotizing fasciitis can have good outcomes, though the journey is often prolonged and fraught with potential setbacks.

Lessons and Recommendations

This case highlights several key principles in managing complex bite wound infections and NSTIs:

High suspicion and early intervention: Clinicians should maintain a low threshold to suspect deep infection in any bite wound that shows progressive swelling, severe pain, or systemic signs. Early diagnosis and surgical consultation can be lifesaving (4). Even before definitive imaging or culture results, if necrotizing fasciitis is suspected, initiate broad-spectrum antibiotics and arrange prompt surgical exploration.

Multidisciplinary management: Engage a team that may include emergency physicians, surgeons (for debridement and reconstruction), infectious disease specialists (to guide antimicrobials), intensivists (for organ support), and others as needed (9). In our case, input from plastic surgery, cardiology, pulmonology, and rehabilitation services all contributed to the patient's recovery.

Empiric antibiotic strategy: Use broad empiric antibiotics for serious bite wounds. A combination covering Staphylococcus/Streptococcus (including MRSA), gram-negatives, and anaerobes is recommended in NSTIs (8). Tailor antibiotics based on cultures if available, and involve infectious disease experts for adjustments. For less severe infections, narrower coverage (e.g. amoxicillin-clavulanate for outpatient therapy) is appropriate, but escalation is required if there are signs of systemic spread or tissue necrosis.

Serial debridement and wound care: Adequate surgical debridement is often not one-and-done. Plan for possible repeat operations to remove all necrotic tissue. Adjunct techniques such as negative-pressure wound therapy can assist in wound healing once the infection is controlled. Do not attempt definitive wound closure (grafting or suturing) until infection is clearly resolving; interim use of wound VAC or dressings is preferred.

Monitor and prevent complications: Patients with severe infections are vulnerable to complications such as organ failure, secondary infections, and thromboembolism. Implement prophylactic measures (e.g. anticoagulation, ulcer prophylaxis) and closely monitor vital signs, lab trends, and clinical status. In our patient, vigilant monitoring enabled early detection of the pulmonary embolism and a pneumonia, which were addressed before leading to further deterioration.

Conclusion

Human bite injuries, even those that initially appear minor, must be approached with caution due to the high risk of infection and potential for devastating outcomes. This case illustrates how a bite from a small child led to a necrotizing soft tissue infection in an elderly woman – a reminder that any breach in the skin by human teeth can inoculate dangerous pathogens. Early recognition and a proactive treatment strategy were critical in averting limb loss or death. The successful outcome for our patient was achieved through prompt surgical debridement, appropriate broad-spectrum antibiotics, and comprehensive supportive care in an ICU setting. Clinicians should be aware that necrotizing fasciitis can complicate bite wounds, particularly in patients with comorbidities or delayed presentation, and that management requires both aggressive local control of infection and supportive measures to prevent systemic complications. Additionally, the occurrence of a pulmonary embolism in this case emphasizes the need for thromboprophylaxis and high vigilance for thrombotic events in patients immobilized or critically ill due to severe infections.

In summary, this case underscores the principle that "no human bite should be underestimated." Early intervention, multidisciplinary coordination, and attention to complications allowed our patient to recover fully. Emergency and critical care providers should remember that behind an innocuous-looking bite may lurk a limb- and life-threatening infection. Timely surgery, correct antibiotic choices, and vigilant critical care can make the difference in outcomes for these rare but perilous cases (6, 9).

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Colchicine Intoxication: An Innocent Drug or a Deadly Poison?

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Introductıon:

Colchicine is a drug used in the chronic treatment of Familial Mediterranean Fever (FMF), as well as in the management of acute attacks of gout arthritis and pseudogout. Although colchicine intoxication is not frequently encountered in emergency departments, it is a condition that requires attention due to its life-threatening symptoms (1). Acute poisoning is rare but can present with a wide range of clinical manifestations, from gastrointestinal and hematological changes to cardiogenic shock (2).

Case:

An 18-year-old female patient presented to our emergency department with severe nausea, vomiting, and diarrhea that had started a few hours before her admission. She had no known chronic diseases except for allergic asthma. Her family history revealed FMF in her mother and sibling and Ankylosing Spondylitis in her father. On physical examination, she exhibited diffuse abdominal tenderness, more pronounced in the upper quadrants. Laboratory results were as follows: WBC: 13,100, NEU%: 85.1, CRP: 21.05 mg/dL, LDH: 4,485 U/L, ALT: 914 U/L, AST: 2,416 U/L, CK: 1,244 U/L, INR: 1.83, and Troponin I: 1,544 ng/L. No additional pathology was identified. Abdominal imaging revealed a heterogeneous liver appearance with reduced density, hepatosteatosis, and pericholecystic fluid accumulation.

Upon admission, the patient denied any drug intake or unusual food consumption. However, during follow-up and upon further questioning, she admitted to having taken 3-4 boxes (approximately 30 tablets) of colchicine three days prior in a suicide attempt. Consultation with the national poison control center (114 Poison Advisory Hotline) was conducted. Due to the risk of cardiogenic shock, the patient was admitted to the intensive care unit (ICU) for close monitoring.

Dıscussıon:

Although colchicine is primarily used to treat acute attacks of gout and pseudogout, it has also been effective in the treatment of Familial Mediterranean Fever (FMF) and Behçet's syndrome for the past 50 years. Studies indicate that doses of <0.5 mg/kg cause minor toxicity with a 100% recovery rate, while doses between 0.5-0.8 mg/kg lead to major toxicity with a 10% mortality rate (3). At doses exceeding 0.8 mg/kg, mortality occurs within 72 hours due to cardiogenic shock. However, prognosis is not always directly proportional to the ingested dose.

Currently, there is no known antidote for colchicine toxicity (2). Patients exposed to high doses of colchicine should be monitored in the ICU with appropriate fluid-electrolyte therapy, hemodynamic monitoring, and respiratory support. Fluid and electrolyte replacement therapy is a priority to reduce gastrointestinal losses (1). Daily complete blood count (CBC) monitoring is essential to detect anemia, leukopenia, and thrombocytopenia, and preventive measures should be taken to mitigate potential hemorrhagic complications (4).

Keywords: Familial Mediterranean Fever, Colchicine, Suicide

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A Rare Complication of Acute Myocardial Injury and Successful Patient Management: Ventricular Septal Defect

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Introduction

Ventricular septal defect (VSD) following myocardial infarction (post-MI VSD) is a rare yet life-threatening complication. With the prompt management of acute coronary syndromes and early revascularization, its incidence has progressively declined. However, despite technological advancements, a substantial reduction in mortality rates has not been achieved. The hemodynamic instability of patients at the time of presentation, along with the fragility of the ischemic myocardial tissue in the intervention area, significantly contributes to the high mortality risk.

Case

A 70-year-old male patient presented to our emergency department with a four-day history of angina. Coronary angiography revealed total occlusion with slow antegrade flow in the left anterior descending (LAD) artery and the first diagonal branch. Transthoracic echocardiography performed in the emergency department identified a post-MI VSD measuring 3 × 4 cm in the apical region. No accompanying valvular pathology was observed and the ejection fraction (EF) was calculated as 35%. Surgical intervention was planned for simultaneous revascularization and VSD repair.

The patient was taken to surgery under intra-aortic balloon pump (IABP) support and cardiopulmonary bypass was initiated. Myocardial protection was achieved using antegrade modified Del Nido cardioplegia. Saphenous vein grafts were used for distal anastomoses of the LAD and first diagonal branch. To explore the apical VSD, a ventriculotomy was performed approximately 2 cm lateral to the LAD. The tissue adjacent to the ventricular septum appeared fragile. A pericardial patch reinforced with pledgets was sutured to the healthy tissue surrounding the septal defect using 4-0 Prolene sutures, closing both sides of the defect in a single layer. The ventriculotomy incision was also closed using 3-0 Prolene sutures, reinforced with pledgets and a pericardial patch. Figure 1 displays photographs related to the surgical repair. The schematic drawing of the surgically repaired VSD is shown in Figure 2. Proximal anastomoses were performed on the ascending aorta. Following deairing, the cross-clamp was removed and the heart resumed spontaneous activity. The patient was weaned off cardiopulmonary bypass under IABP support without the need for inotropic agents.

The patient was transferred to the intensive care unit in stable condition and was extubated six hours postoperatively. IABP support was weaned off on the first postoperative day. On the second postoperative day, the patient was transferred to the ward for further monitoring and was discharged on the seventh day. Echocardiography performed at the 12-month follow-up revealed an ejection fraction (EF) of 40%, with no residual shunting across the interventricular septum.

Figure 1: Surgical Photos of VSD Repair

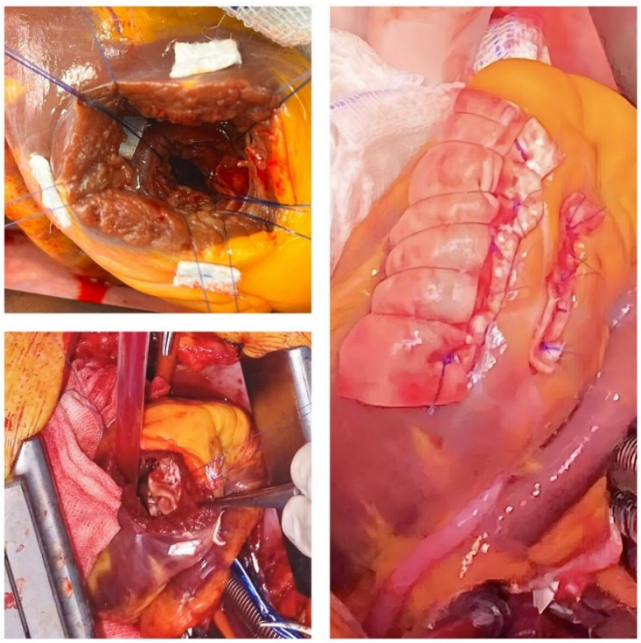
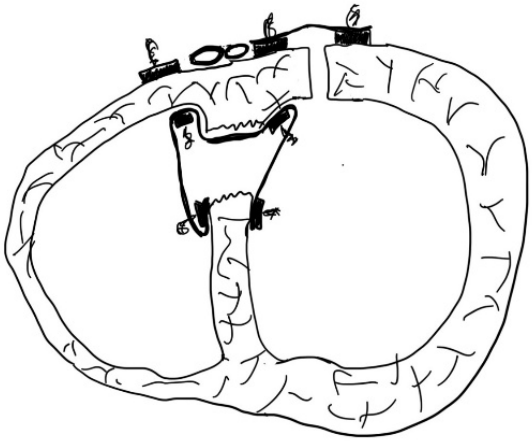


Figure 2: The cross-sectional schematic drawing of the surgically repaired VSD



Discussion

The mortality rate of post-MI VSD is high. In the preoperative period, afterload should be reduced through medical and, when necessary, mechanical support before proceeding with surgery. Various surgical techniques have been described for this condition. One of the challenges in surgery is the fragility of ischemic tissue, especially around the VSD. The presence of a residual shunt after surgery or the formation of a new tear at the patch site during the procedure are significant factors increasing mortality. Modifications in surgical techniques that allow the suture line to be placed on healthy tissue can improve survival outcomes. For this purpose, Komeda and colleagues introduced the "infarct exclusion method", in which they performed ventricular remodeling using a pericardial patch. In the "double-patch technique" described by Balkanay and colleagues, the pericardial patch was sutured to the intact areas of the VSD to prevent the formation of residual shunts. We believe that excluding the revascularized ventricular area with a pericardial patch may negatively affect ventricular function. Therefore, instead of completely excluding the ventricular structures adjacent to the septum, we used the intact tissues to secure the patch. By covering both surfaces of the VSD, we reduced the likelihood of residual shunting. Additionally, this approach provides a more pressure-resistant alternative compared to a single patch. Unlike the previously described techniques, our method does not require the use of biological adhesives to minimize the risk of residual shunting.

Numerous techniques have been described for VSD closure. The technique we applied is also a safe and easily implementable method. Since post-MI VSD is a rare condition, we have had limited opportunities to test this technique in a larger patient population. Further applications in a greater number of patients will provide a better understanding of its outcomes.

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A Rare Cause of Childhood Stroke: Minor Head Trauma

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Introduction

Head trauma is an important cause of mortality and morbidity in pediatric patients over 1 years of age. Falls from the same level and falls from height are the most common causes of head trauma. The majority of these patients who present to the emergency department are consistent with mild head trauma (1). Although cases consistent with severe head trauma are fewer in number, the pathologies leading to mortality and morbidity are more frequently observed in this patient group. Subdural, epidural, subarachnoid, and intracerebral hemorrhages, cerebral contusion and edema, and diffuse axonal injury are among the major trauma-related pathologies observed in these patients (1,2).

Ischemic stroke following mild head trauma is a rare post-traumatic pathology in the pediatric age group. Although the exact cause is unknown, it is thought to be related to vasospasm or thrombosis of the lenticulostriate arteries following trauma. It is usually observed in the basal ganglia region (2,3,4). This case report presents a 3-year-old patient who was admitted to the emergency department following mild head trauma and was subsequently diagnosed with ischemic stroke secondary to trauma.

Case

A 3-year-old girl was admitted to our emergency department (ED) after falling from a seat approximately 25 inches high, 12 hours earlier. She has no known history of chronic disease and presented with malaise and impaired consciousness. The patient fell on the left parietal region and a small hematoma was identified in that area. Her parents reported that she had initially been evaluated at a local ED after the fall, where her head computed tomography (CT) and laboratory results were normal, and she was discharged. However, due to her persistent symptoms, they brought her to our ED.

Her vital signs were stable and she had isolated head trauma, other systemic examination was normal. On admission, she was lethargic, uncooperative, and had a Glasgow Coma Scale (GCS) score of 11. Neurological examination revealed weakness in her left arm and leg, with muscle strength graded at 4/5. Laboratory tests showed hemoglobin at 10.5, glucose at 69, pH at 7.28, and bicarbonate at 12, while other laboratory results were within normal limits. Both head and spinal CT scans were normal. The patient was consulted with a neurosurgeon. The neurosurgeron recommended a diffusion MRI, and the performed diffusion MRI revealed diffusion restriction compatible with acute ischemia in the right lentiform nucleus and the posterior part of the body of the caudate nucleus (Figure-1).

The patient was consulted with pediatric neurology based on imaging findings and was admitted to the pediatric intensive care unit with a preliminary diagnosis of ischemic cerebrovascular event. The patient was also evaluated by pediatric hematology and cardiology, and advanced investigations for the etiology of ischemic stroke were inconclusive. The patient was diagnosed with ischemic stroke secondary to minor head trauma. After 7 days in intensive care and 1 day of pediatric ward follow-up, the patient was discharged. According to the patient's relatives, one week after discharge, the weakness in the left upper and lower extremities gradually improved and returned to normal without any sequelae.

Conclusion

In pediatric patients, head trauma commonly causes intracerebral hemorrhage; however, in rare cases, it can also lead to ischemic stroke. Clinicians should be aware of this rare complication, especially when neurological deficits persist despite normal initial imaging.

Keywords: Minor head trauma, childhood stroke, pediatric head trauma

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Ruptured Pulmonary Hydatid Cyst Presenting as Massive Pleural Effusion in a 10-Year-Old Boy: A Case Report

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Introduction

Hydatid disease is a significant public health problem in many endemic regions, including the Mediterranean, Middle East, and parts of Asia (1). It is caused by the larval stage of Echinococcus granulosus, a tapeworm whose life cycle involves canines as definitive hosts and livestock as intermediate hosts. Humans are accidental intermediate hosts who become infected by ingesting parasite eggs. The most commonly affected organs in human echinococcosis are the liver and lungs (1). Notably, in pediatric patients the lungs are often the predominant site of hydatid cysts, whereas adults more frequently present with hepatic cysts (1). Pulmonary hydatid cysts can grow to considerable size due to the elastic lung tissue and negative intrathoracic pressure, often without early symptoms. Over time, patients may develop cough, chest pain, or dyspnea as the cyst enlarges, but many remain asymptomatic until a complication occurs (2). The most dreaded complication is cyst rupture. When a pulmonary hydatid cyst ruptures, it can spill fluid and scolices into bronchial or pleural spaces, leading to severe clinical manifestations. We present a case of a 10-year-old boy with a massive left pleural effusion as the initial presentation of a ruptured pulmonary hydatid cyst. The case underscores the importance of considering hydatid disease in the differential diagnosis of unexplained pleural effusions in children from endemic areas and highlights the management approach for this potentially life-threatening complication.

Case Presentation

A 10-year-old male from a rural area presented to a district hospital emergency department with acute respiratory distress. His symptoms began a few days prior with cough and left-sided chest pain, progressively worsening to severe dyspnea on the day of admission. There was no history of trauma. The patient's medical and family history were unremarkable, although it was noted that he lived in an area where livestock farming was common.

On examination, the child appeared anxious and tachypneic. Vital signs showed a respiratory rate of 40/min, heart rate of 130/min, blood pressure 105/70 mmHg, and oxygen saturation 91% on room air. He had decreased expansion of the left hemithorax. On auscultation, breath sounds were markedly diminished over the entire left lung field with pronounced dullness to percussion, consistent with a large pleural effusion. The right lung had vesicular breath sounds without rales. There were no signs of urticaria or rash.

Initial laboratory investigations revealed mild leukocytosis (WBC 13,000/μL) with eosinophils in the upper normal range, and an elevated C-reactive protein of 5 mg/dL. Liver and renal function tests were within normal limits. A chest radiograph demonstrated almost complete opacification of the left hemithorax with mediastinal shift to the right, suggestive of a massive left pleural effusion. The right lung was clear. Given the significant respiratory compromise, an urgent ultrasound of the chest was performed to characterize the effusion and guide management. Ultrasound confirmed a large pleural fluid collection on the left side. Notably, within the fluid there appeared to be an ill-defined septated cystic structure in the lower half of the thorax, raising suspicion of an underlying lesion rather than a simple empyema.

A diagnostic and therapeutic thoracentesis was carefully performed, yielding approximately 150 mL of clear, pale yellow fluid. Analysis of the fluid showed an exudative profile (protein 4.8 g/dL, LDH 600 U/L) but was negative for bacteria on Gram stain and culture. Given the atypical nature of the effusion fluid and the ultrasound finding of a cystic structure, a contrast-enhanced computed tomography (CT) scan of the chest was obtained for further evaluation. The CT scan revealed a large cystic lesion (~10 cm in diameter) occupying the left lower lobe, with evidence of partial rupture. The cyst contained irregular internal membranes and air-fluid levels (Figure 1). The left pleural space was filled with fluid, and collapsed lung parenchyma was noted draped around the cyst. No other cysts were seen in the lungs or abdomen. These imaging features were highly suggestive of a hydatid cyst with rupture into the pleural cavity.

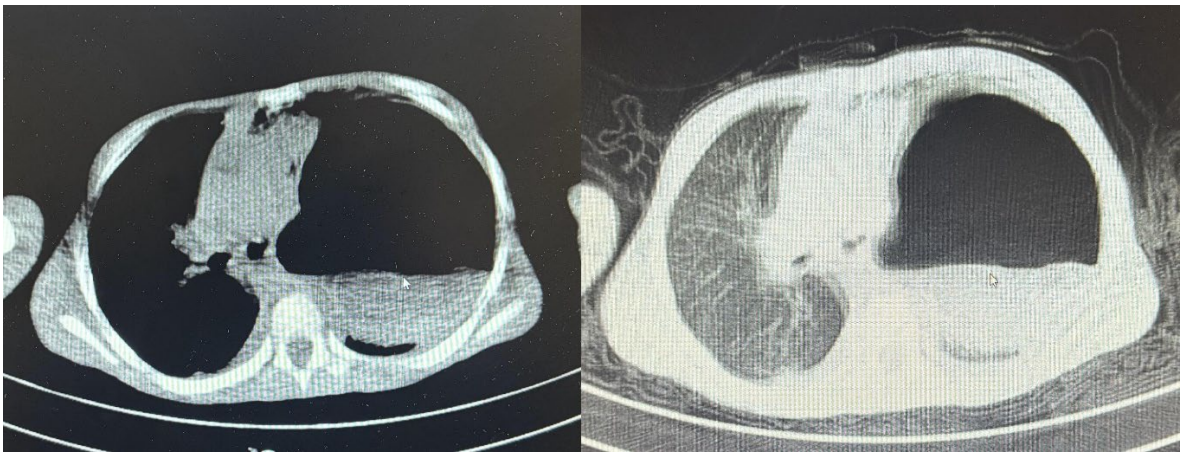

Figure 1. The CT scan; a large cystic lesion (~10 cm in diameter) occupying the left lower lobe, with evidence of partial rüptüre.

The patient was started on high-flow oxygen and intravenous fluids. Considering the likely diagnosis of a complicated hydatid cyst, we initiated albendazole (10 mg/kg) to begin antiparasitic coverage, and the pediatric surgery service was consulted. The child was transferred to a tertiary care center with cardiothoracic surgical capability the same day. Upon arrival, serological testing for hydatid disease (IgG ELISA) was performed and later returned positive, although definitive diagnosis was to be made via surgery.

Within 24 hours of presentation, the patient underwent a left posterolateral thoracotomy. Intraoperatively, about 1 liter of serous pleural fluid was encountered and evacuated. A large hydatid cyst was found protruding from the left lower lobe into the pleural space (Figure 2). The cyst had ruptured, and the free cyst fluid had spilled into the pleural cavity; however, fibrous adhesions localized the spillage. The surgical team took precautions to avoid contamination, including packing the area with sponges soaked in hypertonic saline (a scolicidal agent). The cyst was opened carefully. Thick laminated membranes (endocyst) and numerous daughter cysts were evacuated from the cavity. The bronchial communications were identified and sutured. A capitonnage procedure was performed – the residual cyst cavity in the lung was obliterated by suturing the edges together in layers, to eliminate dead space. Two chest tubes were placed (one apical and one basal) and the thoracotomy was closed in layers.

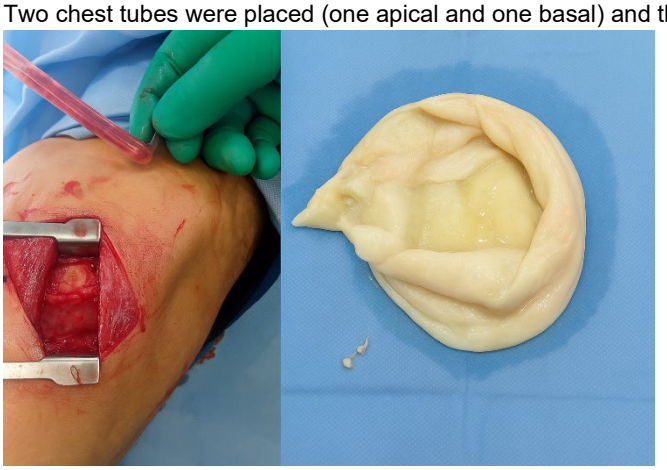

Figure 2. Intraoperative and postoperative imaging of pulmonary hydatid cyst.

The patient was extubated in the recovery room and had an uncomplicated postoperative course. Chest tube drainage was serosanguinous and steadily decreased; both tubes were removed by postoperative day 4. The patient was discharged on postoperative day 7 in good condition. At discharge, a 3-month course of albendazole was prescribed (15 mg/kg/day in two divided doses) to eradicate any residual scolices and minimize recurrence risk. On follow-up at 1 and 3 months, the boy was asymptomatic and chest X-rays showed full expansion of the left lung with no fluid re-accumulation.

Discussion

Hydatid cyst rupture into the pleural space is an infrequent but serious complication of pulmonary hydatidosis. In children, pulmonary hydatid cysts often remain silent until they become large or rupture (2). In one pediatric series, nearly 40% of pulmonary hydatid cases had evidence of cyst rupture at presentation (2), highlighting how common this complication can be in endemic regions. Our patient's initial presentation with isolated massive pleural effusion and respiratory distress is relatively uncommon. Typically, an intact lung cyst might present with cough, chest pain, or hemoptysis, whereas rupture into a bronchus classically produces a sudden cough with salty tasting sputum or even expectoration of cyst fragments (3). Rupture into the pleural cavity, on the other hand, can lead to overflow of cyst fluid and cause pleural effusion, hydropneumothorax, or empyema(3). In this case, the entire hemithorax was opacified due to a combination of the large cyst and pleural fluid—mimicking an empyema or massive parapneumonic effusion on initial imaging.

Pleural involvement in hydatid disease is generally secondary to a pulmonary or hepatic cyst eroding into the pleural space (3). It is a relatively rare occurrence overall, reported in only about 2–7% of all hydatid disease cases(3), but higher rates have been observed among those with lung cysts. For example, in a large clinical series of 412 pulmonary hydatid cases, 76 patients (approximately 18%) developed pleural rupture complications such as effusion or pneumothorax (4). Similarly, our patient's cyst was located peripherally in the lung and ruptured into the pleural cavity, leading to a massive effusion. Once a cyst ruptures, the released fluid and scolices provoke an intense pleural inflammatory reaction. This can manifest as pleural effusion, often initially sterile, but it may become secondarily infected (empyema) if not promptly treated (3). In our case, no bacteria were grown from the pleural fluid, indicating we intervened before infection set in.

Another critical concern with ruptured hydatid cysts is the risk of systemic allergic reactions. The fluid within a hydatid cyst is antigenic; when it spills, patients can develop urticaria, bronchospasm, or even anaphylactic shock (5). Fortunately, our patient did not experience anaphylaxis despite the cyst rupture. It's possible that the rupture was contained by pleural adhesions limiting exposure of antigens to systemic circulation. Nonetheless, clinicians must be vigilant for signs of an allergic reaction in similar scenarios, and epinephrine and steroids should be readily available during any interventions such as aspiration or surgery. Notably, diagnostic aspiration of a hydatid cyst is contraindicated unless absolutely necessary, due to the risk of anaphylaxis and dissemination of the infection (5). In this case, a minor thoracentesis was performed to relieve pressure and confirm the effusion; it was done with caution, and indeed only clear fluid was obtained without collapse of the cyst itself, thus avoiding a severe reaction.

Imaging is paramount in diagnosing a hydatid cyst and its complications. In our patient, chest ultrasound first raised the suspicion of a complex cystic structure rather than a simple effusion. Chest CT then provided a definitive diagnosis by visualizing the cyst with internal septations and membranes. Certain radiologic signs are characteristic of ruptured hydatid cysts. One classic finding is the "water lily sign," which appears when the endocyst membrane detaches and floats in the fluid of the parent cyst cavity, resembling a water lily on radiographs or CT (6). This sign was essentially observed in our patient's CT as undulating membranes within the cystic lesion. Other described CT signs of hydatid cyst rupture include the "air crescent sign" (air between the pericyst and collapsed endocyst) and the "meniscus sign," among others (3). Recognizing these imaging features can help differentiate a ruptured hydatid cyst from other causes of cystic lung lesions or complex pleural collections. In ambiguous cases, serologic tests (such as IgG ELISA for echinococcus) are useful supportive tools, though they may be false-negative in up to 10–20% of pulmonary cases (3). In our patient, the positive serology further supported the diagnosis but was obtained after imaging already pointed to a hydatid cyst.

The definitive treatment for pulmonary hydatid cysts—especially those that are complicated by rupture—is surgical. Surgery both removes the parasite and addresses the complications (such as evacuating pleural fluid and re-expanding the lung) (7). In our case, urgent surgical intervention via thoracotomy allowed complete removal of the cyst and thorough cleansing of the pleural space. The preferred surgical technique for lung hydatid cysts is a parenchyma-sparing approach if possible. This usually involves cystotomy (opening and evacuating the cyst) followed by capitonnage, which is the closure of the residual cavity by a series of purse-string sutures to approximate the cavity walls (3). Capitonnage helps eliminate dead space and reduces postoperative air leaks while preserving lung tissue. We were able to perform cystectomy and capitonnage without the need for anatomical resection in this child. This is important because children have greater potential for lung regeneration and we aim to avoid lobectomy unless absolutely necessary. Literature suggests that lung resection (segmentectomy or lobectomy) should be reserved for cases where the lung tissue is severely destroyed or infected, or when there are multiple cysts in a single lobe that preclude simple closure (3). In our patient, although the cyst was large, the surrounding lung was viable and successfully re-expanded after surgery.

Outcome after surgical management of pulmonary hydatid disease is generally excellent, provided that spillage is controlled and postoperative care is adequate. One consideration is the use of adjunctive antiparasitic medication. Albendazole, a benzimidazole antihelminthic, is recommended as an adjuvant therapy in cases of ruptured cysts or those with risk of spread (3). Postoperative albendazole (dosed at 10–15 mg/kg/day) for at least 3 months has been shown to significantly reduce recurrence rates (4). In our case, we administered albendazole for 3 months after surgery. This regimen is in line with standard practice, as noted in prior studies where postoperative albendazole led to zero recurrences over 6–12 months of follow-up (7). The drug helps to kill any scolices that might have escaped into the bloodstream or pleural cavity, thereby preventing new cyst implantation. Tolerance to albendazole is generally good in children, with liver enzymes monitored during therapy.

This case also underlines the importance of early referral and intervention. Complicated hydatid cysts have higher morbidity and prolonged hospital stays compared to intact cysts (8). In a comparative study, patients with ruptured hydatid cysts had significantly more postoperative complications and longer recovery, underscoring that prompt surgical management before complications escalate is vital (8). Our patient was managed in a timely fashion, which likely contributed to his smooth recovery without residual disability. Had there been delays, the cyst could have evolved into a fulminant empyema or caused permanent damage to the lung.

From a public health perspective, this case is a reminder that hydatid disease remains endemic in certain regions and can present in the emergency setting in various forms. Emergency and primary care physicians in endemic areas (such as our region) should include hydatid cyst in the differential diagnosis of unexplained pleural effusions or atypical empyemas, especially in pediatric or young adult patients. Simple investigations like chest ultrasound can provide clues (such as internal membranes or daughter cysts in the fluid) that point toward echinococcosis. Once suspected, appropriate precautions (avoiding indiscriminate needle aspiration) and early involvement of surgical teams can improve outcomes.

Conclusion

Rupture of a pulmonary hydatid cyst should be recognized as a rare cause of acute pleural effusion and respiratory distress in child-

ren living in endemic areas. In our 10-year-old patient, a massive “effusion” was in fact due to a burst Echinococcus cyst in the left lung. The case highlights the importance of high clinical suspicion and the use of imaging to achieve correct diagnosis. Definitive management requires prompt surgical intervention to remove the cyst and address complications. Lung-preserving surgical techniques, combined with postoperative albendazole therapy, offer an excellent prognosis in most cases. This case reinforces that in any child from a region where hydatid disease is prevalent, an unexplained pleural effusion or hemithorax opacification warrants evaluation for a possible hydatid cyst. Early diagnosis and treatment are crucial to prevent serious outcomes, and interdisciplinary care (emergency, radiology, surgery, infectious disease) is essential for successful management of this complex condition.

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On the Right? On the Left? Or Both Sides of It? Unexpected Fracture Case Report

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Introduction and Purpose: In patients presenting to the emergency department trauma department, upper extremity and specifically elbow joint traumas are a subject that requires utmost attention in terms of anamnesis, physical examination and examination. We evaluated a very rare case of bilateral radial head fracture resulting from a simple fall.

Case:A 36-year-old male patient presented to our emergency room trauma unit shortly after 00:00 on Saturday night with complaints of pain in both elbows after falling from a chair 6 days before his admission.Physical examination revealed normal range of motion in both upper extremities, no restriction in supination and pronation movements of both forearms. Neurovascular examination was normal in both extremities, no edema or swelling in both arms upon palpation, and a mild pain complaint at the level of the raidus head in both arms. The patient was asked for the necessary imaging tests. The x-ray revealed a Mason type-2 fracture in the left raidus head and a Mason type-1 fracture in the right radius head.Oral analgesia was prescribed and he was treated conservatively with bilateral slings for approximately 2 weeks, followed by elbow mobilization and physical therapy.

Discusıon: When the patient was first examined, the mechanism of trauma, the time that had passed, the day and time of emergency room application, and the findings obtained as a result of the physical examination were taken into consideration, there was a suspicion and prejudice that there could be abuse of emergency room services and unnecessary report requests.In cases of such patient applications, loss of motivation and attention is frequently observed in emergency room doctors regarding the necessary anamnesis, physical examination, and evaluation of requested tests.Isolated radial head fractures are not a common type of fracture. Radial head fractures are common, with an estimated incidence of 2.5 to 2.8 per 10,000 inhabitants per year.(1) They comprise approximately 2% of all fractures around the elbow.(2)Bilateral radial head fractures are rare and are usually seen with fractures and dislocations associated with severe trauma.(3) In this case, we wanted to emphasize the importance of carefully examining all requested tests after a good history and physical examination of patients presenting to the emergency department with mild trauma, regardless of external factors.

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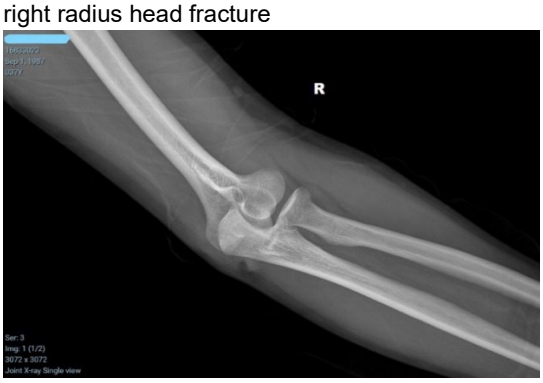
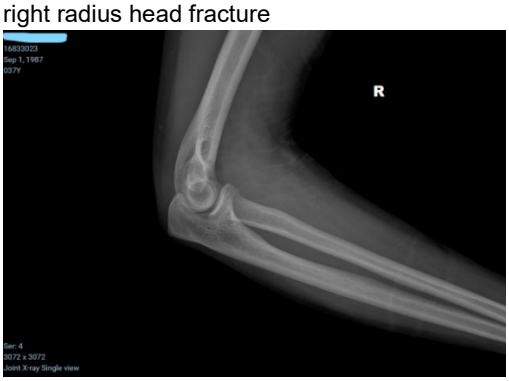
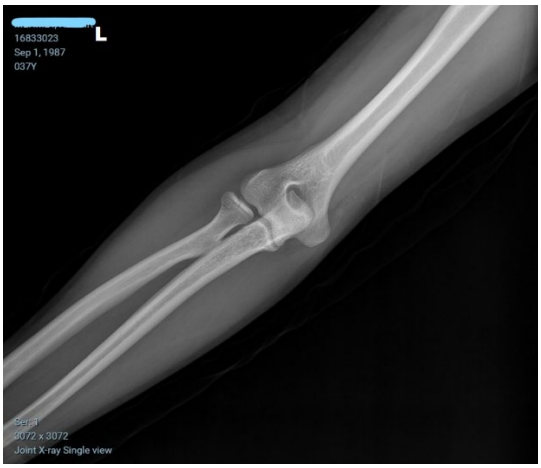
right radius head fracture



right radius head fracture



left radius head fracture



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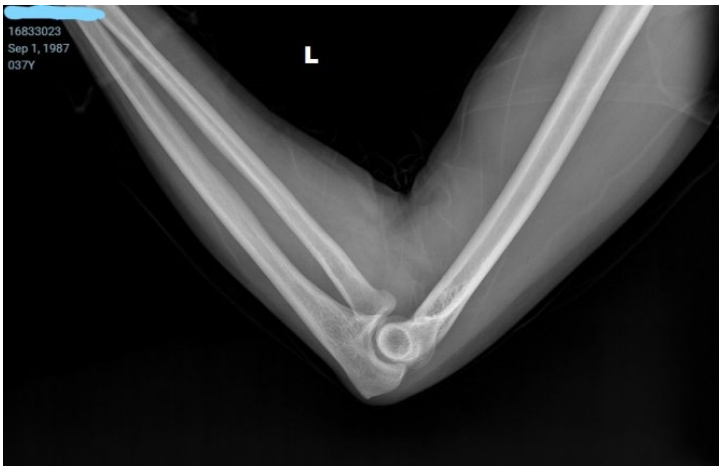

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Relationship between Laboratory Parameters and Balthazar Severity Score in Acute Pancreatitis

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Acute pancreatitis is a disease characterized by the activation of digestive enzymes, which are not normally active in the pancreas under normal conditions, by various etiologic factors, causing digestion and inflammation of pancreatic tissues and surrounding adjacent tissues (1). In 25-30% of patients, the clinical picture may become severe enough to threaten life and mortaliteye rates may 2). In the diagnosis of AP, sudden onset of abdominal pain in the back, elevation of serum lipase and amylase ratios more than 3 times higher than normal values and imaging methods are used (3).

Various scores are used to determine the severity and prognosis of the disease in patients diagnosed with AP. Immature granulocyte percentage (IG%) has started to be used in clinics as a new inflammatory indicator (4).

Material-Methods:

The study was conducted on 250 patients who presented to the Emergency Department of XXX University Faculty of Medicine Hospital with abdominal pain and were diagnosed with "Acute Pancreatitis" between January 01, 2018 and August 01, 2021. The relationship between the blood values of the patients we recorded and the patients grouped according to the Balthazar Severity Score was examined.

Results:

According to the Balthazar Severity Score we calculated, there were 194 (77.6%) patients in the mild group and 56 (22.4%) patients in the moderate group. When we compared these clinical severity groups according to laboratory values, a statistically significant difference was found between the two groups in terms of WBC, neutrophil, lymphocyte, NLO and PLO among the complete blood count parameters (p values 0.001, <0.001, 0.016, <0.001, 0.006, respectively). Immature granulocyte percentage (IG%) was not statistically significant between the two groups, whereas LDH and Lipase/Amilase ratio were statistically significant (p values <0.001 and 0.001, respectively). In terms of CRP, the results can be considered borderline significant (p= 0.051). When the cut-off for NLO was 14.9082, the sensitivity was 46.4%, specificity 85%, PPV 47.2% and NPV 84.6%. When the cut-off for lipase/amylase ratio was 2.2773, 69.6% sensitivity, 57.2% specificity, 26.3% PPV and 86.7% NPV were found.

Discussion:

Abdominal pain is a common presentation to the emergency department worldwide. AP is one of the most important diagnoses to be considered among these presentations due to its high mortality and morbidity rates.

Although biochemical parameters used in the diagnosis of AP are useful in making the diagnosis, they are incomplete in determining the severity and prognosis of the disease (5). However, the Lipase/Amilase ratio in our study was statistically significant in predicting the severity of the disease (p=0.001).

It has been shown that immature granulocyte (IG) cells can be used as an early inflammation marker (6). In our study, IG% value did not yield statistically significant results in differentiating severe cases. Only a statistically significant difference was found between patients admitted to the intensive care unit and patients admitted to the ward and patients discharged.

Conclusıon:

Contrary to the studies conducted to recognize severe AP cases early, IG% was not statistically significant in our study. Lipase/Amilase ratio seems to be one of the parameters that can be used to differentiate severe cases.

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Hair-thread tourniquet syndrome: A case report

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Introduction

Hair-thread tourniquet syndrome (HTTS) is a rare, preventable condition that occurs when a body appendage is tightly wrapped with a hair strand or thread-like material, resulting in circulatory disturbance. It typically affects the genital area, fingers, and toes. Hair tourniquet syndrome, which occurs when hair or hair-like material becomes entangled around the coronal sulcus of the penis, is characterized by progressive circulatory disturbance. This condition can lead to serious complications, including glans necrosis. Other risk factors contributing to the development of the syndrome include circumcision, wearing tight clothing, poor hygiene, and low socioeconomic status. Early diagnosis and treatment of HTTS is vital because delayed diagnosis can result in serious complications such as ischemia, tissue necrosis, and even auto-amputation. However, complete recovery is possible with early diagnosis and appropriate treatment (1-3).

In this case report, we aimed to present a rare case of hair tourniquet syndrome in a child who presented with penile pain, redness, and dysuria complaints.

Case Report

A 2-year-old male patient was brought to the emergency department by his mother with complaints of penile pain, redness, and burning sensation during urination. According to his history, the complaints started approximately 24 hours ago, gradually intensified, and his restlessness increased in the last few hours. The patient had no significant medical history and no recent history of infection or trauma.

On physical examination; the patient was in good general condition, conscious, oriented, and cooperative. His vital signs were measured as follows; body temperature: 36.8°C, pulse: 98/min, respiratory rate: 22/min, blood pressure: 95/60 mmHg, oxygen saturation: 99%. Significant edema, erythema, and circular constriction were detected on the penile shaft. Detailed examination revealed the presence of a single hair strand completely encircling the penile shaft (Figure 1). The patient was diagnosed with hair-thread tourniquet syndrome. Under local anesthesia, the hair strand was carefully cut and removed using a scalpel and clamp. Post-procedure evaluation showed no signs of deep cuts or necrosis in the penile tissue. Doppler ultrasonography performed to assess circulation showed normal blood flow.

The patient was kept under observation in the emergency department for 4 hours after the procedure. During this time, significant improvement in pain and restlessness was observed. The patient, who developed no problems during the follow-up period and had normal urinary output, was discharged with necessary recommendations given to the family.

Discussion

Hair-thread tourniquet syndrome (HTTS) is an important clinical condition that particularly occurs in the newborn and infant period and can lead to serious complications if not diagnosed early (4).

This syndrome is particularly seen in newborns and young children, and the affected anatomical regions vary according to age. In approximately 44.2% of cases reported in the literature, the penis is affected, in 40.2% the toes, and in 8.6% the fingers. When analyzed by age groups, fingers are generally affected up to 1.5 years of age, penis between 4 months and 6 years, and the genital

area between 7-13 years (3).

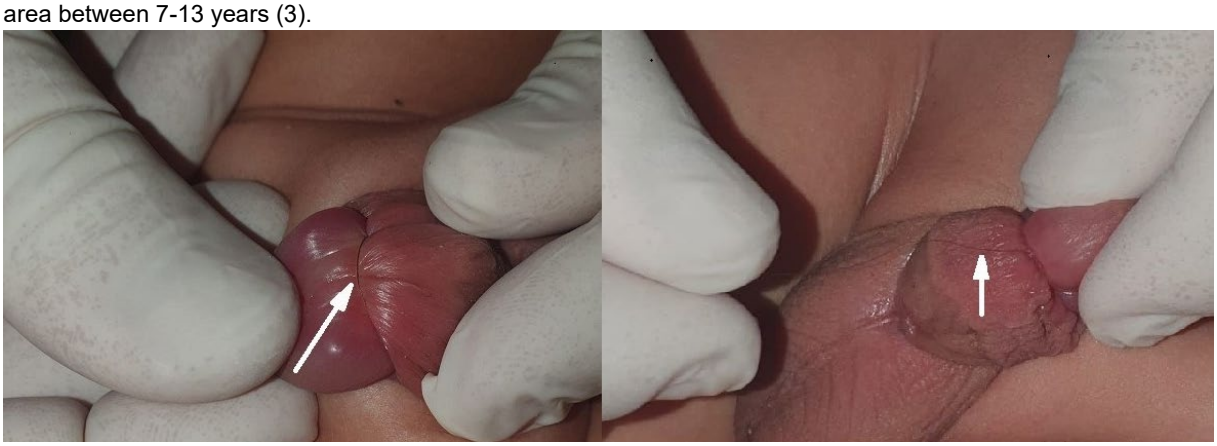

Figure 1. A single strand of hair completely surrounding the shaft of the penis

In our case, consistent with these data in the literature, penile hair tourniquet syndrome was observed in a 2-year-old male child. Risk factors for the syndrome include telogen effluvium in postpartum mothers, poor hygiene conditions, low socioeconomic status, and tight clothing (5). Knowledge of these risk factors and informing families about them is important in taking preventive measures. Careful examination of extremities and the genital area, especially in the newborn period, is vital for early diagnosis. In our case, the family was of foreign nationality, had low income, and poor hygiene conditions.

Treatment approaches vary depending on the affected area and degree of tissue damage. Non-surgical methods include depilatory creams, mineral oil application, and hair/thread dissolving solutions, while advanced cases may require longitudinal incision or surgical removal of the tourniquet material (4). Although the prognosis is generally good with early diagnosis and appropriate treatment, serious complications such as ischemia, tissue necrosis, auto-amputation, and secondary infections can be seen in delayed cases (5). In our case, successful treatment was achieved without any complications due to early diagnosis and appropriate intervention.

HTTS shows that it is not sufficiently recognized by physicians due to its rare occurrence, and therefore delays in diagnosis and treatment may occur. (1) Especially in pediatric patients presenting with unexplained genital pain and restlessness, keeping this syndrome in mind and performing careful physical examination is critically important. Early diagnosis and treatment in HTTS is crucial to prevent complications. If cases are not appropriately treated in a timely manner, serious clinical conditions such as finger, penis, or clitoris amputation may develop (2).

Conclusion

In infants with unexplained restlessness, one should be cautious about HTTS, and it should not be forgotten that areas where this syndrome may occur should be carefully examined.

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2449

Comprehensive Evaluation of Splenium Lesions: A Multitude of Potential Causes

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Karamanoglu Mehmetbey University

Introduction:

Transient splenial lesions of the corpus callosum can occur in many conditions, including cancer, drug use, metabolic and cerebrovascular disorders, and infections.Brain magnetic resonance imaging often reveals signs of cytotoxic edema in the splenium, which typically resolve as clinical symptoms improve.In most cases, the prognosis is favorable (1).

We present a case of 31-year-old-male with a corpus callosum lesion with a complaint of leg pain and numbness which was resolved 2 days after its presentation.

Case report:

A 31-year-old male patient presented with complaints of left leg pain and numbness on the left side of his face. He uses hydroxychloroquine for known Sjögren's disease. There are no other known medical conditions.

On examination:

Vital signs: Blood pressure was 120/60 mmHg, pulse was 80 beats per minute, oxygen saturation was 98%, and temperature was 36.6°C.

Neurological examination: No focal deficits were observed. Cranial nerves were intact, and there were no signs of motor or sensory deficits. The patient demonstrated normal coordination and cerebellar function. Reflexes were normal, and there were no signs of meningeal irritation.

No significant findings were observed in laboratory tests. Acute diffusion restriction was noted in the corpus callosum on diffusion MRI (Figure 1). No acute pathology was detected on brain tomography.

The patient was referred to neurology, and a brain MRI was performed to exclude malignancy. Subsequently, the patient was admitted to the neurology department and consulted with rheumatology to exclude rheumatologic causes. The lesion was attributed to an ischemic etiology. He was discharged on the second day of hospitalization, as he had no further complaints and no acute pathology was detected in follow-up examinations.

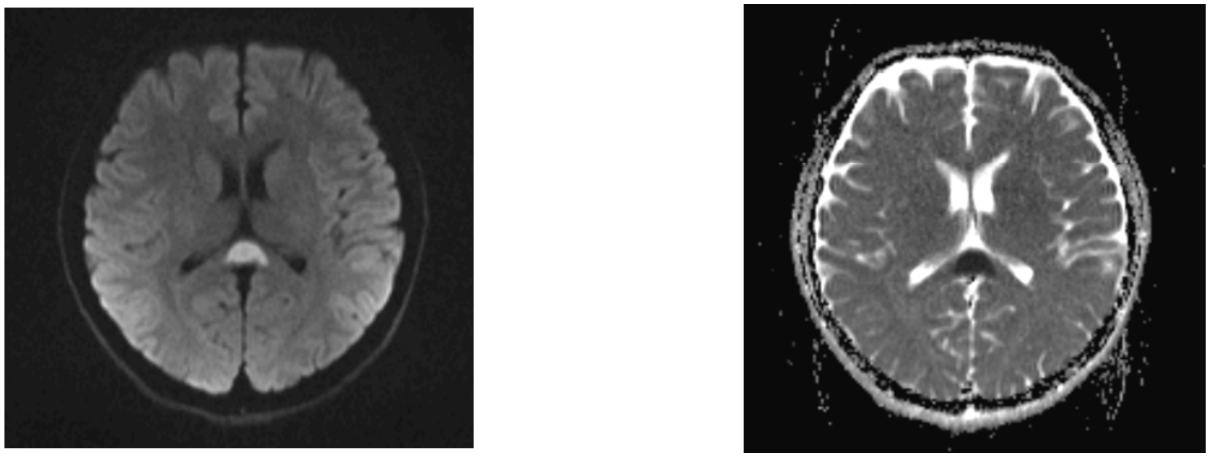

Figure 1:MRI scan

Discussion

The differential diagnosis of splenium lesions includes ischemic infarction, acute disseminated encephalomyelitis, multiple sclerosis, lymphoma, glioblastoma multiforme, posterior reversible encephalopathy syndrome, diffuse axonal damage, hydrocephalus and extrapontine myelinolysis. Since splenial lesions are associated with many etiologies, their clinical manifestations are also dependent on the underlying disease. Fever, headache, seizure, delirium, vomiting, diarrhea, cognitive impairment, impaired consciousness, behavioral changes, dysarthria, somnolence, ataxia, papilledema, visual disturbances, neck stiffness, and motor disorders are the clinical manifestations reported in the literature (2,3,4,5). Our case showed left leg pain and numbness on the left side of his face. Some cases reported after COVID-19 vaccination in the literature which showed excellent prognosis with high dose methylprednisolone (6).

On diffusion-weighted magnetic resonance (MR) images, CLOCCs appear as areas of restricted diffusion. These lesions do not enhance with contrast material, are typically located along the midline, and are relatively symmetrical. The corpus callosum involvement generally presents in one of three patterns: a small round or oval lesion in the center of the splenium, a lesion that is centered in the splenium but extends laterally through the callosal fibers into the surrounding white matter, or a lesion that is posteriorly centered but extends into the anterior corpus callosum (7).

Conclusion:

Splenium lesions can result from a wide range of etiologies, and their clinical manifestations vary depending on the underlying condition. Symptoms can include cognitive impairment, seizures, motor disturbances, and changes in consciousness. Given the variety of potential causes, a comprehensive evaluation is vital for accurate diagnosis and management.

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2478

A Case Of Type A Aortıc Dıssectıon Presentıng Wıth Inferıor Stemı

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Introductıon:

Aortic dissection can present with a wide range of symptoms, affecting multiple systems, including chest pain, hoarseness, flank pain, and limb weakness. The most common symptom in patients with Type A Acute Aortic Dissection is chest pain, which is present in 64–82% of cases at the time of admission. Since treatment strategies differ significantly, the most crucial differential diagnosis is acute coronary syndromes (1,2,3). The use of point-of-care ultrasound (POCUS) in the emergency department can facilitate the early diagnosis of aortic dissection, potentially improving patient mortality and morbidity.

Case:

A 66-year-old male patient presenting with chest pain was brought to our emergency department by EMS (112 teams). The initial electrocardiogram (ECG) revealed ST elevation in the inferior leads, leading to a diagnosis of STEMI, and the patient was administered 300 mg of aspirin. Upon arrival, his blood pressure was 98/53 mmHg, heart rate was 57 bpm, and oxygen saturation was 95%. His medical history included hypertension and diabetes mellitus, along with a family history of cardiac disease.

The ECG obtained in the emergency department showed >2 mm ST-segment elevation in leads D3 and AVF, accompanied by complete AV block, along with reciprocal >2 mm ST-segment depression in leads D1 and AVL (Figure 1).

Point-of-care ultrasound (POCUS) revealed an aortic root dilation of 58 mm in the parasternal long-axis view, with a mobile echogenic structure suggestive of a dissection flap (Figure 2). In the suprasternal notch window, a mobile echogenic structure consistent with a dissection flap was also observed within the aorta (Figure 3).

The patient was prepared for surgery, and a simultaneous consultation with cardiovascular surgery was initiated. To confirm the extent and location of the dissection, CT angiography was planned (Figure 4).

The patient was diagnosed with Type A Aortic Dissection and was taken for emergency surgery by the cardiovascular surgery team.

Dıscussıon:

In Type A Aortic Dissection, the dissection membrane may sometimes extend to the coronary ostium, particularly affecting the right coronary artery, leading to acute myocardial ischemia associated with ischemic ST-T changes on ECG (4). Acute myocardial infarction (AMI) due to the propagation of acute Stanford Type A Aortic Dissection is a rare but devastating event, occurring in approximately 3% of patients with aortic dissection (1). Differentiating patients with coronary malperfusion due to acute Stanford Type A Aortic Dissection from true acute myocardial infarction poses a significant challenge for emergency physicians. Misdiagnosis is particularly common in patients presenting with ST-segment elevation on ECG (5,6). A key diagnostic finding is the presence of an intimal flap separating the true and false lumens.

Transthoracic echocardiography (TTE) is highly valuable in assessing aortic valve dysfunction, pericardial tamponade, and wall motion abnormalities.

Given the high mortality associated with acute aortic dissection, rapid diagnosis and appropriate interventions to facilitate early surgical management are crucial. Considering that delayed diagnosis is a major contributor to mortality, the use of point-of-care ultrasound (POCUS) in the emergency department enables early diagnosis and improves survival outcomes. Greater emphasis should be placed on ultrasound training and POCUS education in emergency medicine residency programs and medical school curricula.

Keywords: Emergency Department, Aortic Dissection, Chest Pain, POCUS

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Figure 1: ST-segment elevation >2 mm in leads D3 and AVF, accompanied by complete AV block, along with reciprocal ST-segment depression >2 mm in leads D1 and AVL.

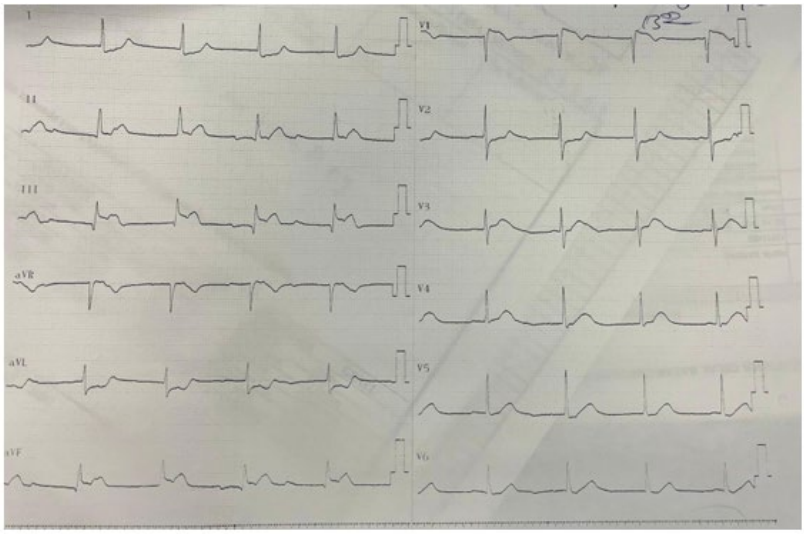


Figure 2: Enlarged aorta and dissection flap appearance in the parasternal long-axis view.

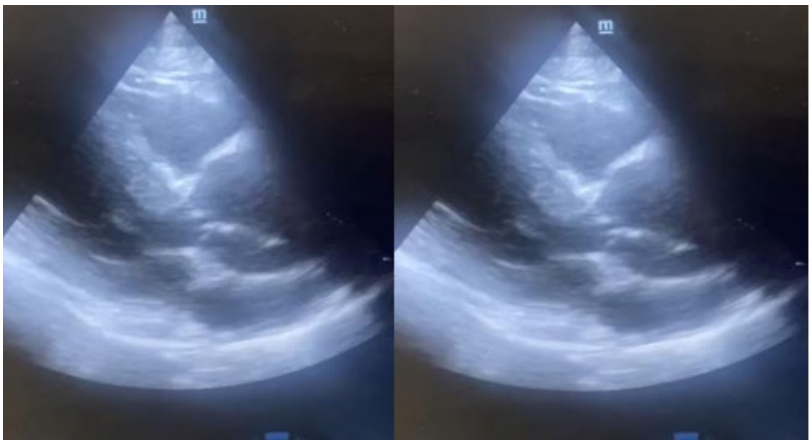


Figure 3: Dissection flap within the aorta in the suprasternal window.

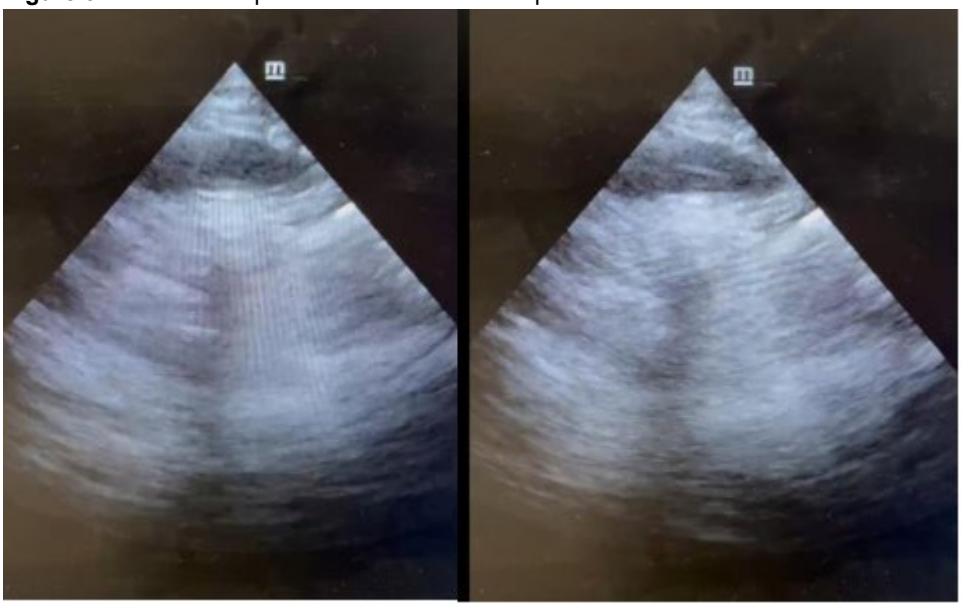
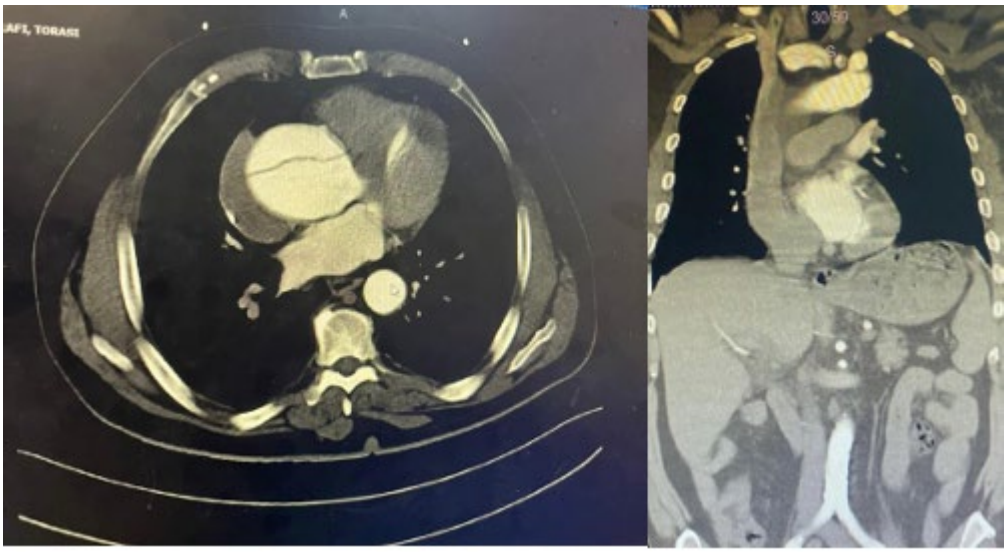


Figure 4: Axial and coronal CT angiography sections of Type A Aortic Dissection.



2795

Neutropenic Enterocolitis at the Frontline: Chemotherapy Agents and Prognostic Indicators in the Emergency Department

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Introduction

Neutropenic enterocolitis (NE), also known as typhlitis, is a potentially life-threatening gastrointestinal condition frequently seen in patients undergoing intensive chemotherapy. It is characterized by inflammation, bowel wall thickening, and risk of necrosis and perforation, primarily due to mucosal injury combined with profound neutropenia (1,2). Although rare, its reported incidence ranges between 0.8% and 2.6% in adult cancer patients (2). The condition may progress rapidly to sepsis, requiring urgent medical attention.

While several chemotherapeutic agents are implicated in the pathogenesis of NE, their direct influence on prognosis remains unclear. Agents such as taxanes, platinum compounds, and antimetabolites are known to damage the gastrointestinal mucosa through mechanisms like oxidative stress and disruption of microtubule function (3,4,5,6). However, it is still uncertain whether patients who develop NE after exposure to these agents experience worse clinical outcomes than those exposed to other drugs. Prior studies have largely focused on incidence and pathophysiology rather than on comparative mortality data, especially in adult populations (7,8).

This study investigates whether specific chemotherapeutic agents are associated with increased mortality or adverse clinical outcomes in adult patients diagnosed with NE in the emergency department.

Methods

Study Design and Setting

A single-center retrospective cohort study was conducted in the emergency department of a tertiary academic hospital that serves as a regional oncology referral center. Adult patients (≥18 years) admitted between January 1, 2020, and January 1, 2024, with a diagnosis of neutropenic enterocolitis were included.

Inclusion Criteria

Patients were eligible if they:

Had a history of chemotherapy within the past 30 days,

Had an absolute neutrophil count (ANC) <1000/mm³,

Presented with gastrointestinal symptoms such as diarrhea, abdominal pain, or fever,

Showed radiological evidence of bowel wall thickening (>4 mm in CT or ultrasound) (9).

Chemotherapy Agents and Outcomes

Chemotherapy agents were categorized as:

Taxanes: Docetaxel, Paclitaxel

Platinum Compounds: Cisplatin, Carboplatin, Oxaliplatin

Antimetabolites: 5-Fluorouracil (5-FU)

Anthracyclines: Doxorubicin
Others: Irinotecan, Cyclophosphamide, Vinca alkaloids, Etoposide
Primary outcomes were in-hospital mortality and occurrence of adverse clinical events (sepsis, ICU admission, or rehospitalization).
Statistical Analysis
Univariate logistic regression was conducted to assess the association between each chemotherapy agent and the primary outcomes. Results were reported as odds ratios (ORs) with 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant. Statistical analysis was performed using SPSS v21.0.

Results

Out of 113 patients with NE, 20 patients (17.7%) died and 29 (25.6%) experienced at least one adverse clinical event. Docetaxel was the most frequently used agent (27.5%), followed by 5-FU (24.8%) and cisplatin (18.3%). Table 1 summarizes the univariate logistic regression findings. No statistically significant association was found between any chemotherapeutic agent and mortality or adverse outcomes.

Table 1. Univariate Analysis of the Association Between Chemotherapy Agents and Mortality or Adverse Outcomes

	n (%)	Death (OR 95% CI)	p (Death)	Adverse Outcome(OR 95% CI)	p (AO)
Docetaxel	30 (27.5)	1.55 (0.55 - 4.35)	0.410	1.26 (0.5 - 3.21)	0.622
5-Fluorouracil (5-FU)	27 (24.8)	1.02 (0.33 - 3.11)	0.979	0.73 (0.26 - 2.05)	0.553
Cisplatin	20 (18.3)	0.77 (0.20 - 2.92)	0.699	0.66 (0.2 - 2.17)	0.493
Paclitaxel	19 (17.4)	0.47 (0.10 - 2.23)	0.342	0.27 (0.06 - 1.27)	0.098
Carboplatin	18 (16.5)	0.51 (0.11 - 2.41)	0.393	0.3 (0.06 - 1.38)	0.121
Doxorubicin	16 (14.7)	0.60 (0.12 - 2.86)	0.517	0.91 (0.27 - 3.07)	0.875
Oxaliplatin	17 (15.6)	1.46 (0.42 - 5.07)	0.550	0.82 (0.25 - 2.77)	0.755
Irinotecan	15 (13.8)	1.13 (0.29 - 4.46)	0.859	1.46 (0.45 - 4.7)	0.527
Cyclophosphamide	12 (11.0)	0.88 (0.18 - 4.36)	0.873	1.44 (0.4 - 5.2)	0.578
Vinca alkaloids	12 (11.0)	0.88 (0.18 - 4.36)	0.873	1.44 (0.4 - 5.2)	0.578
Etoposide	7 (6.4)	0.73 (0.08 - 6.41)	0.775	1.11 (0.2 - 6.07)	0.903

Discussion

Although chemotherapeutic agents are well-recognized contributors to the development of NE, our study found no statistically significant association between specific agents and mortality or adverse outcomes once the condition had been diagnosed. This finding aligns with the notion that while chemotherapy initiates mucosal injury and immunosuppression, downstream complications may be more influenced by the clinical course rather than by the initial drug exposure (7,8,11).

Taxanes such as docetaxel and paclitaxel are known to disrupt microtubules in rapidly dividing cells, including intestinal epithelium, contributing to mucosal breakdown and bacterial translocation (3). Platinum compounds such as cisplatin and oxaliplatin promote oxidative stress and DNA cross-linking, delaying mucosal repair (4). 5-FU and doxorubicin contribute to mucosal injury via inflammation and chromatin torsion mechanisms (5,6).

Nematolahi et al. emphasized that systemic factors such as infection, immune suppression, and timing of supportive therapy play a more substantial role in prognosis than the chemotherapeutic agent itself (7). Similarly, Benedetti et al. reported that early imaging and intervention were more predictive of outcome than treatment history (8). Bertozzi et al. highlighted the complex systemic pathophysiology of NE, suggesting that progression involves more than local mucosal damage (10).

Conclusion

Although chemotherapeutic agents are implicated in the pathogenesis of NE, this study found no evidence that specific agents significantly influence mortality or adverse clinical outcomes once NE is established. Clinical risk stratification in the emergency department should focus on current symptoms, imaging findings, and systemic involvement rather than chemotherapy history alone.

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2843

Clınıcal Cerebrovascular Event But Dıagnosıs Is Aortıc Dıssectıon

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Introductıon

Aortic dissection, defined as the separation of the intimal layer from the medial layer along the long axis of the aorta, is an acute cardiovascular emergency with a high mortality rate, requiring rapid diagnosis and treatment. Its incidence ranges from 5 to 30 cases per million. It most commonly occurs in hypertensive patients over the age of 40. Hypertension is known to be the most significant risk factor for the development of aortic dissection (1,2). In aortic dissection, early diagnosis is crucial due to the poor prognosis. In particular, acute dissections involving the ascending aorta and aortic arch have a mortality rate of 1-3% per hour during the first few days (3). Patients typically present to the emergency department with sudden onset, severe, tearing chest pain. However, there is a broad range of symptoms. Patients may also present with hoarseness (due to recurrent laryngeal nerve paralysis), hemoptysis, neurological deficits, superior vena cava syndrome, or Horner's syndrome.

Case Presentatıon

An 83-year-old male patient with a known history of hypertension presented to our emergency department. He was found in a supine position by a family member half an hour before coming to the hospital. Upon arrival, the patient was greeted in the resuscitation room. His vital signs at presentation were as follows: blood pressure 111/77 mmHg, oxygen saturation 97%, and pulse rate 132 beats per minute. The Glasgow Coma Score was 15, and his capillary blood glucose was 132 mg/dL. On physical examination, the patient exhibited aphasia and left-sided hemiplegia as pathological findings. Arterial blood gas analysis revealed no acute abnormalities, and imaging studies were requested. A brain CT scan showed no acute pathology, leading to the recommendation for a diffusion MRI. However, due to severe cervical lordosis, the patient was unable to tolerate the MRI, and therefore, the diffusion MRI could not be performed. As a result, a brain CT angiography and carotid CT angiography were requested to evaluate the brain's vascular structures. No acute abnormalities were detected on the brain CT angiography; however, the carotid CT angiography revealed a dissection-like appearance in the aortic arch region. Following this, a thoracic aorta CT angiography and abdominal aorta CT angiography were ordered. These imaging studies revealed a dissection extending from the aortic arch to the iliac arteries, with no flow detected in the right brachiocephalic artery. Urgent consultations were requested with cardiovascular surgery and cardiology. An echocardiogram performed by cardiology showed severe aortic insufficiency and a pericardial effusion surrounding the heart. Cardiovascular surgery diagnosed the patient with a Type 1 aortic dissection and planned an emergency operation. Unfortunately, the patient died postoperatively.

Dıscussıon

Early diagnosis and prompt initiation of treatment are essential for patients presenting to the emergency department, as the prognosis is poor (2,3). Hypertension, connective tissue diseases, atherosclerosis, aortic coarctation, bicuspid valve, pregnancy, thoracic aortic aneurysm, previous aortic surgery, inflammatory and infectious diseases, iatrogenic and traumatic factors, genetic factors, and Marfan Syndrome are all recognized risk factors for aortic dissection (4). The defining characteristic of aortic dissections is a tear in the intimal layer, followed by the formation and progression of a subintimal hematoma (5). This dissecting hematoma often fills approximately half of the aorta, and sometimes the entire circumference. It forms a false lumen, which can reduce blood flow to the major arteries branching from the aorta (6). Thoracic aortic dissections are anatomically classified into two separate methods. According to the Stanford classification, Type A dissections involve the ascending aorta, whereas Type B dissections do not. The DeBakey classification evaluates dissections in three types: Type I involves the ascending aorta, aortic arch, and descending aorta; Type II is limited to the ascending aorta. Type III dissections involve the descending aorta distal to the left subclavian artery, with Type IIIa originating distal to the left subclavian artery. As they extend distally and proximally, they are typically located above the diaphragm. On the other hand, Type IIIb extends only distally and may reach below the diaphragm. Ninety percent of dissections occur within the 10 cm segment distal to the aortic valve. The second most common location is immediately distal to the left subclavian artery (7).

Since the symptoms of patients diagnosed with aortic dissection are often non-specific, the most critical factor in the diagnosis is,

undoubtedly, the consideration of aortic dissection as a possibility. In our case, although the clinical examination initially suggested a cerebrovascular event, the diagnosis of aortic dissection reminded us of the broad clinical spectrum of aortic dissection.

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Unexpected Densıty On Ct Imagıng In A Case Of Esophageal Atresıa

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Introductıon

Esophageal atresia (EA) is an anomaly seen in 1 in 4000-5000 live births and has many types; the most common is distal tracheo-esophageal fistula (EA+TEF). It may be accompanied by other system anomalies in 50% of cases. The presence of multiple system anomalies together affects mortality and morbidity (1,2).

Foreign body ingestion is more common in children than in adults worldwide (3). When foreign bodies swallowed in childhood and in adults are compared, it is seen that those in adults generally have sharp edges, while those in childhood are rounder objects. These are frequently coins and batteries in children; bone fragments, meat, and cartilage in adults. It has been observed that foreign body ingestion is more related to food in adults and that the food is often unchewed. It has been determined that the risk increases as the time to hospital admission is delayed following foreign body ingestion (4). It has been determined that foreign bodies are localized in the upper esophageal stricture in 87%, in the middle esophageal stricture in 13%, and very rarely in the lower esophageal stricture (5). Foreign body ingestion is less common in adult patients than in pediatric patients, but since adult patients are caused by psychiatric and neurological reasons and can be difficult to cooperate, swallowed objects are generally sharper, and more serious complications can be seen as diagnosis is delayed (6). Many retrospective studies have shown that the longer the foreign body remains in the esophagus, the higher the morbidity and mortality of esophageal perforations, so early intervention is important in removing foreign bodies from the esophagus (8).

Case Report

A 23-year-old male patient applied to the emergency department after feeling a tightness in his esophagus after eating a biscuit in the morning and then vomiting repeatedly. The patient had a history of surgery due to postnatal esophageal atresia and 6 endoscopic balloon dilatation due to esophageal stenosis. Vital signs at the time of emergency admission were oxygen saturation 98%, blood pressure 120/80 mmHg, pulse 85/min. No pathology was detected in the respiratory examination. A thoracic-abdominal tomography was performed for the patient who was suspected of having a foreign body in the alimentary tract. When the patient's tomography scans were examined, a hyperdense lesion in the esophagus (Figure 1-2) was seen. No significant stenosis was detected in the esophagus. The patient was consulted to the general surgery and gastroenterology clinics due to the foreign body in the esophagus. The patient was scheduled for an endoscopy by the gastroenterology department and food residue was found in the esophagus in the endoscopy performed the next day. The food residue was advanced endoscopically into the stomach. An area compatible with 'Barret's Esophagus' was observed at the lower end of the esophagus and a biopsy was taken. The patient, who had no additional pathology, was discharged with recommendations.

Dıscussıon

It has been observed that esophageal foreign bodies are most often stuck in anatomical narrowing areas. The esophagus has three anatomical narrowing areas. The pharyngoesophageal junction is the first narrowing area and is the most common place where foreign bodies are stuck. The second narrowing point is the point where it crosses the left main bronchus, also known as the middle narrowing. The last narrowing point is the part where the lower end of the esophagus passes the diaphragm, 3 cm above the cardia. The width of the esophagus varies according to whether it is empty or full. While its width is 1-1.5 cm when empty, it can reach 2.5-3 cm with increased pressure. The constrictor muscle activity of the pharynx is extremely strong, objects can be pushed into the esophagus from here, but the muscle activity of the upper part of the esophagus is weak, so foreign bodies are mostly located under the cricopharyngeus muscle. The most common complaints of patients after swallowing a foreign body are dysphagia and odynophagia. Hypersalivation and dyspnea are also common complaints (9). Foreign bodies in the alimentary tract are a common problem encountered all over the world. Approximately 1500 people die each year in the USA due to secondary problems caused by swallowing foreign bodies (10). The causes are local; in countries where fish is consumed extensively, swallowing fish bones is a common problem, or in Muslim societies, swallowing the needle used for turbans is common, depending on the intensity of headscarf use (11-12).

Esophagogastroduodenoscopy is considered the gold standard method for diagnosis of foreign bodies found in the esophagus. Treatment includes removal of the foreign body with a foley catheter, rigid and flexible esophagoscopy, pushing the foreign body into the stomach, removal of the foreign body with magill forceps, intravenous glucagon administration and surgical procedures (10).

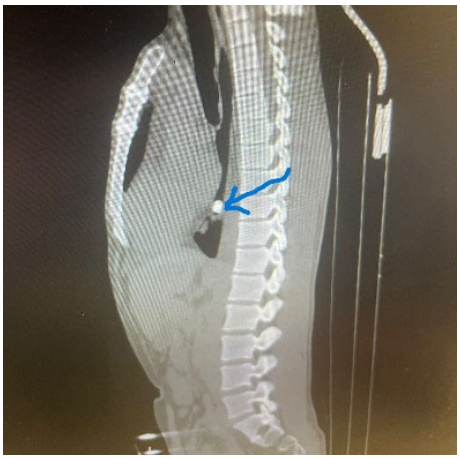

Figure 1. BT-Thorax sagittal section

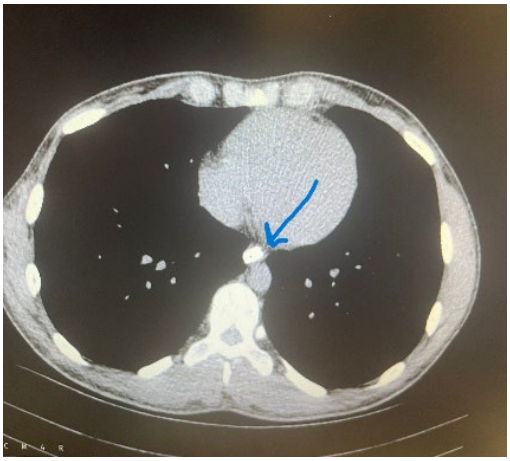

Figure 2. BT-Thorax transverse section

Conclusıon

It should be kept in mind that even soft foods such as biscuits can cause complications such as obstruction in patients with a history of esophageal atresia or a history of previous surgery, and that advanced imaging methods should be applied in patients with suspected foreign bodies in the alimentary tract. We wanted to emphasize the importance of detecting esophageal foreign bodies and the role of endoscopic intervention in the treatment process in patients with esophageal atresia with this case, which we think is also interesting in terms of the hyperdense appearance of a soft food substance in tomography. Since various treatment methods can be applied to patients presenting to the emergency department with a foreign body in the alimentary tract, a multidisciplinary approach and regular follow-up are also important for these patients.

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A Transport Method: "Body Packing": A Case Report

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Introduction

Drug trafficking has been carried out through various methods throughout history, becoming more complex over time. One common method today is "bodypacking," which involves swallowing drugs packaged in airtight packets to be transported through the digestive system. A similar situation is "bodystuffing," where a person swallows drugs, usually in a panic during a police raid (1).

Both methods pose serious medical risks. In bodypacking cases, complications such as packet rupture or bowel obstruction can occur, while "bodystuffing" can lead to rapid toxic effects and even death due to irregular and poor packaging causing drug absorption from the digestive system. This condition, called "body packer syndrome," occurs when drug packets leak or damage the intestinal wall and requires emergency medical intervention (2,3).

In this case report, we will discuss the medical management and complications experienced by a 21-year-old body packer patient. Our purpose in sharing this case is to highlight the points to be considered in the correct diagnosis and treatment of similar patients presenting to the emergency department, and to raise awareness among physicians working in emergency services regarding medical approaches.

Case

A 21-year-old male patient, apprehended while illegally entering the country through the port, was referred to the emergency department for a routine health check-up after detention. Physical examination revealed that he was conscious, oriented, and cooperative. His pupils were isochoric with bilateral positive light reflexes. Breath sounds were normal. Diffuse tenderness was present in the abdomen, but there was no guarding or rebound tenderness. Vital signs were as follows: oxygen saturation 98%, body temperature 36.3°C, blood pressure 140/80 mmHg, heart rate 110 bpm (tachycardic).

An anterior-posterior abdominal X-ray was performed (Image 1). Upon observing hyperdense lesions among gas shadows within the small bowel loops, a non-contrast abdominal CT scan was performed. The CT scan revealed numerous hyperdense foreign bodies, averaging 33x11 mm in size and 18 mm in diameter, extending from the distal duodenum to the rectum (Images 2a and 2b). Laboratory findings were: WBC 6700/μL, Hb 15.1 g/dL, PLT 335,000/μL, CRP 5.69 mg/dL, Na 135 mEq/L, Creatinine 0.93 mg/dL. Urine analysis revealed an amphetamine level of 615 ng/ml.

The patient's condition was evaluated with the general surgery team. As there were no signs of acute abdomen or bowel obstruction, it was decided that the patient would expel the foreign bodies naturally. Laxative and enema treatment were administered to expedite the process. Following treatment in the emergency department, the patient defecated 51 packages of powdered methamphetamine wrapped in condoms.

Due to the patient's stable condition, he was admitted to the general surgery ward for 48 hours of asymptomatic observation. No pathological findings were detected in his vital signs during this period. After observation, the patient was discharged with necessary recommendations.

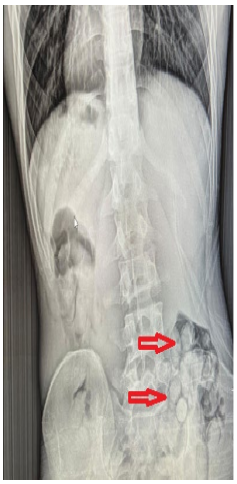

Image 1: Hyperdense lesions seen on abdominal radiographs (AXR) in the upright position.

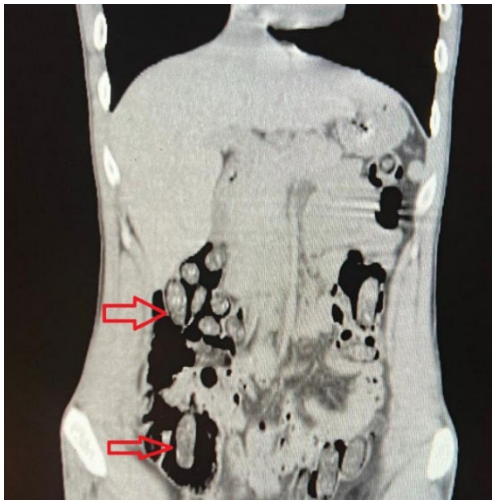

Image 2a: Hyperdense lesions in the form of tubular foreign bodies (axial)

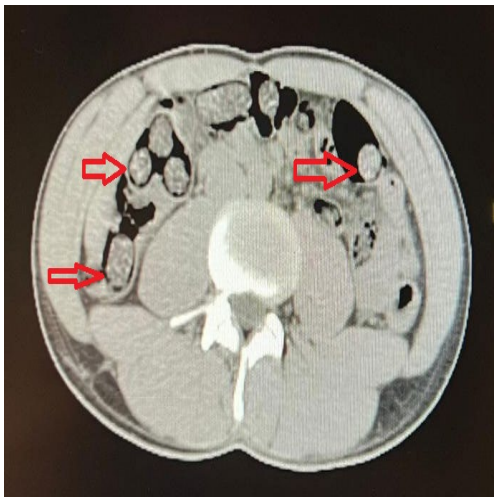

Image 2b: Tubular foreign body-shaped hyperdense lesions (coronal)

Discussion

This case report discusses the management of a 21-year-old male patient who was carrying 51 packages of methamphetamine in his gastrointestinal system. Early and accurate identification of suspected patients is the most critical aspect in body packer cases. While the patient's medical history, physical examination findings, and clinical signs may raise suspicion, imaging techniques are essential for definitive diagnosis.

As in this case, an abdominal X-ray is often the preferred initial assessment tool. According to the literature, the presence of hyperdense lesions within the intestinal loops on radiography, representing the packages, can be a significant diagnostic finding (4). However, radiographic examinations may not always be sufficient. In such cases, non-contrast abdominal CT has a high degree of accuracy in determining the number, size, and location of the packages (5). In the presented case, suspicious findings were detected on plain X-ray, and the number and distribution of the packages were confirmed by CT. The literature states that CT is the gold standard for diagnosis, especially in cases with a risk of complications (6).

Treatment management varies depending on the patient's clinical condition and the number, size, and contents of the packages. Conservative treatment is preferred in patients without significant symptoms or findings suggestive of complications such as bowel obstruction or perforation. According to the literature, the natural expulsion of the packages with the use of laxatives and enemas is

an effective and safe method in such cases (7). In our case, a conservative approach was also applied as no acute abdominal findings or obstruction were detected, and the elimination of packages was supported with laxative therapy. Studies show that the use of oral laxatives shortens the elimination time of packages in the gastrointestinal system and reduces the need for surgical intervention (8).

Surgical intervention is necessary in cases of bowel obstruction, perforation, package leakage, or the development of acute abdomen (9). In such situations, surgical removal of the packages protects the patient from life-threatening complications. However, it should be considered that surgical interventions can increase the risks of anesthesia and postoperative complications. Therefore, the decision for surgery should be made with careful consideration.

The greatest risk in body packer cases is the rupture or leakage of the packages they carry. If the integrity of the packages is compromised, the drug they contain can be rapidly absorbed from the gastrointestinal system, leading to a toxic dose and serious complications. This situation can lead to fatal consequences such as loss of consciousness, seizures, cardiac arrhythmias, and sudden cardiac arrest (1, 9). Studies have shown that early surgical intervention to remove packages with a high risk of leakage reduces mortality and morbidity (10).

In this case, the absence of any package leakage was possible thanks to both the patient's regular clinical monitoring and the effective use of imaging methods. The most important goal in body packer cases is to identify potential complications early and take preventive measures. At this point, a multidisciplinary approach and patient monitoring plan are of critical importance. Effective communication and coordination between general surgery, emergency services, and toxicology units are fundamental factors for the successful management of such cases (11).

Conclusion and Recommendations

Body packer cases require early diagnosis and appropriate treatment management, necessitating a multidisciplinary approach. This case contributes to the literature by demonstrating that conservative treatment can be a safe and effective option. However, the risks should be carefully assessed for each patient, and the necessity of surgical intervention should be determined using an individualized approach.

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Spontaneous Pneumomediastinum

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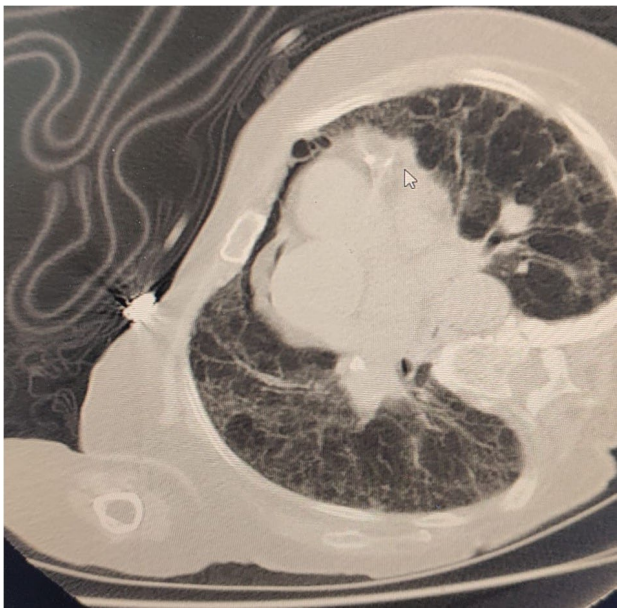
Introductıon

Pneumomediastinum is defined as the presence of free air in the mediastinum. It is a dangerous finding that is often associated with serious complications. Pneumomediastinum is basically divided into two groups: spontaneous pneumomediastinum, which does not have a primary source, and secondary pneumomediastinum, which develops secondary to an underlying pathology. Secondary pneumomediastinum is caused by trauma, intrathoracic infections, or damage to the aerodigestive tract, and similar causes. (1) Since spontaneous pneumomediastinum is less common than pneumomediastinum secondary to trauma, we wanted to draw attention to spontaneous pneumomediastinum through this case.

Case Presentatıon

86-year-old female patient, bedridden, home care patient, was brought to the emergency room via 112 with general deterioration and hypotension. When the patient's history was examined, it was seen that she had Alzheimer's disease and had a stroke. The patient's vital signs showed F: 36.5 °C HR: 140/min BP: 80/50 mmHg SpO2: 92. The patient's relatives did not describe any trauma history.

Physical Examination: Glasgow Coma Score was calculated as 10 (G4,S2,M4). The patient had a decubitus wound in the lumbosacral region. There were no pathological examination findings other than these findings in the patient's physical examination.



Due to hypotension, the patient was given 500 cc normal saline and 1 g paracetamol, and intravenous fluids were given. After fluid replacement, the patient's blood pressure was monitored as normotensive. Hemogram, biochemistry, blood gas, troponin and procalcitonin blood samples were collected from the patient. Sinus rhythm was observed in the patient's electrocardiography. HR: 103 beats/min. Due to the high CRP and procalcitonin levels and general condition deterioration, brain, thorax, abdomen and pelvis CT was performed to find the focus of infection and to search for the etiology causing the general condition deterioration. No signs of acute bleeding were observed in the patient's brain tomography. No significant pathology was detected in the patient's abdomen and pelvis CT.

Thoracic CT report: Air densities were observed in the mediastinum and the appearance was interpreted in favor of pneumomediastinum. Widespread mosaic attenuation pattern and interlobular septal thickening were observed in both lungs (infectious process? pulmonary edema?)

The patient's tomography was interpreted as pneumomediastinum and since our clinic does not have a thoracic surgery department, a referral was planned for the patient. The patient was consulted by the infectious diseases department because of the patient's high CRP and UTI in the urine test. Urine and blood cultures were taken from the patient upon the recommendation of the infectious diseases department. The internal medicine opinion was obtained because the patient was anuric and hypernatremia was seen in the blood tests. The patient, whose referral procedure was completed during the procedures, was transferred to a more advanced center.

Biochemistry:

GLUCOSE:155 MG/DL, UREA:77.1 MG/DL EGFR:55.11 CREATININ:0.94 MG/DL,AST:121 U/L, ALT:76 U/L, ALKALENE PHOSPHATASE: 70 U/L, GGT: 31.6 U/L,, TOTAL BILIRUBIN:0.27 MG/DL, DIRECT BILIRUBIN: 0.07MG/DL, INDIRECT BILIRUBIN:0.2 MG/DL, CRP:194.9 MG/L, SODIUM:154 MMOL/L, POTASSIUM: 3.96 MMOL/L,,

TROPONINE: 38.7, INR:1.3, APTT:21 SEC, PT:12

Full Blood Count

WBC: 10.14 K/uL, HGB: 10.5 G/DL, MCV: 120.9 fL, PLT: 272 K/uL, Procalcitonin: 1.13 ug/lt

Blood Gas

BE(VT):-4.3:, CA++:1.16MMOL/L, COHB:0.5, GLUCOSE:120 MG/DL, HCO3: 18.7 MMOL/L, HCT:32, HHB:7.9, K+:3.33MMOL/L, LACTATE:3.97 MMOL/L, METHB:0.4, NA+:155 MMOL/L, O2CAP:14.9, O2HB:91.2, PCO2:27.7 PH:7.447, PO2:66 MMHG, TCO2:19.5 MMOL/L, THB:10.8 G/DL.

Bacteria in Complete Urine Examination: 635 Leukocytes: 349.

Dıscussıon

Spontaneous pneumomediastinum or spontaneous mediastinal emphysema was first officially described by Louis Hamman in 1939 (1). Although the incidence of spontaneous pneumomediastinum cannot be given clearly, a study by Newcomb, Clarke and colleagues reported an incidence of 1 in 29,670 emergency room visits and a study by Ivan Macia and colleagues reported an incidence of 1 in 44,511 cases (2). The incidence of pneumomediastinum has also been reported to be between 1/32,000 and 1/7,000 in various hospital records (3) (4). Spontaneous pneumomediastinum may present with chest pain, dyspnea and hoarseness, associated with cutaneous emphysema (5). In our case, it was not known whether these symptoms were present or not, since verbal communication could not be established with the patient.

In patients with pneumomediastinum, tracheostomy, tube thoracostomy and subcutaneous catheterization can be applied for treatment, while some patients are followed conservatively. As in patients with subcutaneous catheters, the improvement in respiratory function tests of patients followed conservatively on the 3rd and 7th days compared to the initial values was found to be statistically significant (4). In our patient, the SpO2 value was 92 at the beginning and no respiratory pathology was observed in the blood gas. We think that this is due to the mild severity of the pneumomediastinum and the difficulty the patient has in expressing himself.

Pneumomediastinum is more common in the young population. (6)(7)(8) This is thought to be due to the loose and flexible mediastinal tissues in young individuals. In contrast, these tissues become fibrotic in the elderly, making air migration difficult. Additionally, pneumomediastinum is more common in men (7 out of 10 cases) (6)(8). Contrary to these general data, our patient was 86 years old and a woman, which shows that it should be considered in the differential diagnosis and creates a wealth of literature.

Conclusıon

Spontaneous pneumomediastinum is less prevalent than traumatic pneumomediastinum and poses a diagnostic challenge due to its presentation with nonspecific clinical manifestations. It is a condition that is frequently at risk of high mortality and morbidity. We wanted to emphasize that our patient is not in the age and gender group in which spontaneous pneumomediastinum is commonly seen epidemiologically. Spontaneous pneumomediastinum should be considered in all epidemiologic groups, especially in the hypo-

tensive patient group.

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2996

Relationship between seizure type, EEG findings, and inflammatory markers in epilepsy patients presenting to the emergency department

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Introduction

Epilepsy is a complex neurological disorder characterized primarily by recurrent seizures resulting from abnormal electrical discharges in the brain. These seizures can manifest in diverse forms, influencing patients; physical, cognitive, and emotional well-being. It affects approximately 50 million people worldwide, making it one of the most prevalent neurological conditions (1). Seizures can manifest in various forms, including focal, generalized, and unknown onset seizures, each with distinct electroencephalographic (EEG) patterns that assist in diagnosis and prognosis (2). Recent research has suggested a strong correlation between seizure activity, EEG abnormalities, and neuroinflammatory responses, shedding light on potential biomarkers for epilepsy management and prediction (3). Inflammation is increasingly recognized as a key factor in epileptogenesis, with cytokines such as interleukin-6 (IL-6) and C-reactive protein (CRP) playing critical roles in modulating neuronal excitability and seizure susceptibility (4). Elevated levels of inflammatory markers have been associated with prolonged and more severe seizure episodes, particularly in generalized tonic-clonic seizures (GTCS), which are known to elicit significant systemic inflammatory responses (5). Advancements in EEG technology have facilitated the identification of seizure-related inflammatory biomarkers, which could serve as predictive tools for seizure occurrence and treatment response. For instance, recent studies indicate that blood–brain barrier disruptions, as reflected in serum protein alterations, may serve as potential biomarkers for seizure prediction and disease progression in epilepsy patients (3). Furthermore, longitudinal studies examining EEG dynamics alongside inflammatory marker fluctuations have demonstrated that persistent inflammatory activity is associated with increased seizure recurrence and cognitive decline (6).

This study aims to explore the interplay between different seizure types, EEG patterns, and inflammatory markers to provide deeper insights into the pathophysiology of epilepsy. By examining these relationships, we seek to contribute to the development of more effective diagnostic and therapeutic strategies for epilepsy management.

Materials and Methods

This study is a retrospective analysis of patients diagnosed with epilepsy and its subtypes (ICD codes G40.0-G40.9) who presented to the emergency department of Niğde Ömer Halisdemir University Training and Research Hospital between September 1, 2024, and March 1, 2025. Patients under 18 years of age, those experiencing seizures due to traumatic causes, and those diagnosed with intracranial tumors were excluded from the study. The demographic data of the included patients, such as age, sex, seizure type (partial/tonic-clonic), and electroencephalography (EEG) findings (focal/generalized), were recorded.

Additionally, inflammatory markers obtained from emergency department hemogram and biochemical parameters were analyzed, including the systemic immune-inflammation index (SII); [(Neutrophil×Platelet)/Lymphocyte count], neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), lymphocyte-to-monocyte ratio (LMR), C-reactive protein/albumin ratio (CAR), and leukoglycemic index (LGI); [Leukocyte count×Glucose (mg/dL)]/1000. The relationship between these inflammatory markers, seizure types, and EEG findings was investigated.

Statistical Analysis

Data analysis was performed using SPSS version 27. Descriptive statistics were presented as mean ± standard deviation or median (25th–75th percentiles) for continuous variables, and frequency (percentage) for categorical variables. The normality of data distribution was assessed using visual methods (histograms, stem-and-leaf plots, scatter plots, and box plots) and analytical tests (Kolmogorov-Smirnov test, skewness, and kurtosis). Comparisons between categorical and continuous variables were conducted using the

Mann-Whitney U test for nonparametric distributions. The correlation between LGI and PLR was evaluated using Spearman's correlation analysis.

Results

A total of 102 epilepsy patients were included in the study, comprising 59 males (57.8%) and 43 females (42.2%). The median age of both male and female patients was 31 years (IQR: 21–78; 22–46). Among the patients, 55 (53.9%) experienced tonic-clonic seizures, while 47 (46.1%) had partial seizures. EEG findings revealed generalized discharges in 63 patients (61.8%) and focal abnormalities in 39 patients (38.2%).

The LGI level was found to be significantly higher in patients with tonic-clonic seizures and those with generalized EEG discharges ($p < 0.001$). Conversely, PLR levels were significantly higher in patients with partial seizures ($p = 0.006$) and those with focal EEG findings ($p = 0.019$). Spearman's correlation analysis demonstrated a weak negative correlation between LGI and PLR ($r = -0.252$, $p = 0.011$). No statistically significant relationship was found between other inflammatory markers and seizure types or EEG findings.

	Seizure type			EEG findings		
	Tonic-clonic	Partial	p value	Generalized	Focal	p value
LGI, median (IQR)	0.97(0.78-1.4)	0.57 (0.50-0.73)	**<0.001**	0.92 (0.75-1.36)	0.57 (0.50-0.73)	**<0.001**
PLR, median (IQR)	98.36 (71.13-125.36)	120.67 (95.42-169.64)	**0.006**	99.07 (71.45-131.07)	119.83 (96.82-161.66)	**0.019**

Spearman's correlation analysis: **LGI-PLR: (r = -0.252, p = 0.011)**

Discussion

This study evaluated the relationship between seizure type, EEG findings, and inflammatory markers in patients with epilepsy. In recent years, the association between epilepsy and inflammation has received increasing attention. Soltani et al. (5) suggested that proinflammatory cytokines and the systemic inflammatory response play a significant role in the pathophysiology of epilepsy. In particular, elevated levels of C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) have been shown to be associated with the severity and frequency of seizures in patients with epilepsy.

Although the mechanisms by which seizures influence inflammatory markers have not yet been fully elucidated, several explanations have been proposed in the literature. Bronisz et al. (7) reported that disruption of the blood-brain barrier in patients with epilepsy may affect the levels of inflammatory markers and increase seizure frequency. They also noted that patients with uncontrolled generalized motor tonic-clonic seizures exhibited significantly elevated inflammatory markers, and that EEG findings may reflect this inflammatory process (8). In our study, significantly higher LGI levels in patients with tonic-clonic seizures support the hypothesis that tonic-clonic seizures may induce a more widespread and intense inflammatory response.

The finding of elevated PLR in patients with partial seizures and those with focal findings on EEG suggests that more localized but pronounced inflammatory processes may be prominent in certain forms of epilepsy. This finding was also emphasized in the study by Andrawes et al. (8).

Previous studies have shown that LGI is associated with hyperglycemia and increased white blood cell counts, which may be linked to systemic inflammation and seizure activity (6). In our study, the weak negative correlation between LGI and PLR suggests that inflammatory markers may exhibit variable effects depending on the seizure type.

Limitations

The current study has a retrospective design and does not provide a clear time frame regarding how inflammatory markers change in the preictal, ictal, or postictal periods. In the patient records and discharge summaries available through the hospital automation system, patients were categorized only as having partial or tonic-clonic seizures, and no data were available on absence seizures or other seizure types. Additionally, potential confounding factors such as the use of antiepileptic drugs were not examined in detail. Prospective and long-term follow-up studies may provide a better understanding of the role of inflammation in epilepsy.

Conclusion

This study demonstrated that seizure type and EEG findings are associated with certain inflammatory markers in patients with epilepsy. In particular, the association between tonic-clonic seizures and higher LGI levels suggests that this seizure type may elicit a more pronounced systemic inflammatory response. On the other hand, the observation that PLR is elevated in patients with focal seizures and those with focal EEG findings indicates that inflammation may be involved through different mechanisms in specific epilepsy subtypes.

Future prospective studies should further evaluate whether inflammatory markers can serve as potential biomarkers in the diagnosis or management of epilepsy. Biomarker-based personalized epilepsy management may hold the potential to improve patient prognosis.

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3069

Severe acute pancreatitis due to hypertriglyceridemia: a case report.

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Introductıon And Purpose

Hypertriglyceridemia (HTG) is a significant cause of acute pancreatitis (AP). Early clinical diagnosis of HTG-induced pancreatitis is important to ensure appropriate treatment and prevent subsequent attacks (1, 2). The risk of acute pancreatitis increases gradually as serum triglyceride levels rise above 500 mg/dL (5.6 mmol/L), and increases significantly at levels above 1000 mg/dL (11.3 mmol/L) (3-7). The risk of developing acute pancreatitis is approximately 5% in those with serum triglycerides >1000 mg/dL (11.3 mmol/L) and between 10% and 20% in those with triglycerides >2000 mg/dL (22.6 mmol/L) (8). In a prospective study of 116,500 individuals with triglyceride levels between 443 mg/dL and 885 mg/dL, the incidence rate of acute pancreatitis was noted to be 0.12% (9). The risk of acute pancreatitis also increases with the number of previous acute pancreatitis attacks. In another large retrospective cohort study of 7,119,195 patients, of whom 4158 (0.058%) had one or more acute pancreatitis attacks in the previous year, the incidence rate of acute pancreatitis was <1%, but it increased significantly with each prior acute pancreatitis attack and even at lower triglyceride levels (9).

The severity of acute pancreatitis in patients with hypertriglyceridemia (HTG) depends on both the inflammatory response caused by the pancreatitis itself and the damage caused by lipotoxicity resulting from triglyceride hydrolysis. In most cases, HTG is transient and returns to near-normal levels within two to three days depending on the etiology and optimal management (10). However, severe HTG and high lipase levels (more than 3 times the upper limit of normal) are associated with very high levels of free fatty acids (FFAs), and systemic inflammation resulting from acute pancreatitis can be further complicated by direct activation of toll-like receptor (TLR) 2 and TLR4 by FFAs and direct lipotoxicity (11, 12). There are no clear biomarkers to determine the effects of lipotoxicity independent of acute pancreatitis with normal triglyceride levels, but several studies have observed a decrease in serum calcium levels in more severe cases (12, 13).

In this case report, we will discuss severe acute pancreatitis attacks that developed due to high triglyceride levels in a young male patient.

Case Report

A thirty-four-year-old male patient presented to the emergency department of Konya Beyhekim Training and Research Hospital with complaints of sudden onset of severe abdominal pain and nausea. The patient had a history of hypertriglyceridemia and was using Lipofen SR 250 mg 1x1. He had no other known illnesses. Detailed anamnesis revealed that he had experienced intermittent mild epigastric pain in the last 2-3 months, but it was not this severe and responded to symptomatic treatments.

On physical examination, there was belt-like tenderness in the abdomen and severe epigastric tenderness in attacks. No defense or rebound was detected on abdominal examination. Other system examinations were unremarkable. Laboratory tests revealed; serum Creatinine: 2.3 mg/dL (outside the reference range, high), urea: 22.8 mg/dL (outside the reference range, high), amylase: 614 U/L (outside the reference range, high), lipase: 2401 U/L (outside the reference range, high), triglyceride level (initial measurement): 1229 mg/dL, calcium 10.1 mg/dL, HbA1c = 8.2%, and cardiac markers were within normal limits. Abdominal computed tomography (CT) was performed for further evaluation in the patient with suspected acute pancreatitis. The CT scan revealed significant edema in the head of the pancreas and stranding in the peripancreatic fatty tissue (findings consistent with inflammation) (Figure 1). No stones or dilatation were observed in the biliary tract on tomography. Bedside ultrasound (FAST) did not show any stones, wall thickening, or hydrops in the gallbladder.

The patient was admitted to the second-level emergency intensive care unit with diagnoses of acute pancreatitis and acute kidney injury (AKI). The treatment protocol was arranged as follows: nil per os (NPO), intravenous (IV) fluid replacement, parenteral analgesics (for pain control) (gradual sedoanalgesia with Parol, Buscopan, Fentanyl, Contramal, Aldolan), close laboratory monitoring. In addition to the relevant treatments, triglyceride-lowering approaches were added with an internal medicine consultation recommendation; continuous intravenous GIK solution (solution containing Glucose, regular Insulin, and potassium) infusion was initiated.

However, despite these treatments, there was no significant regression in the patient's clinical symptoms (severe abdominal pain and vomiting). The need for analgesics continued. Follow-up laboratory values showed no significant decrease in triglyceride levels,

with the triglyceride level seen as 1251 mg/dL, and hypocalcemia and borderline hyponatremia (Na=133) developed.

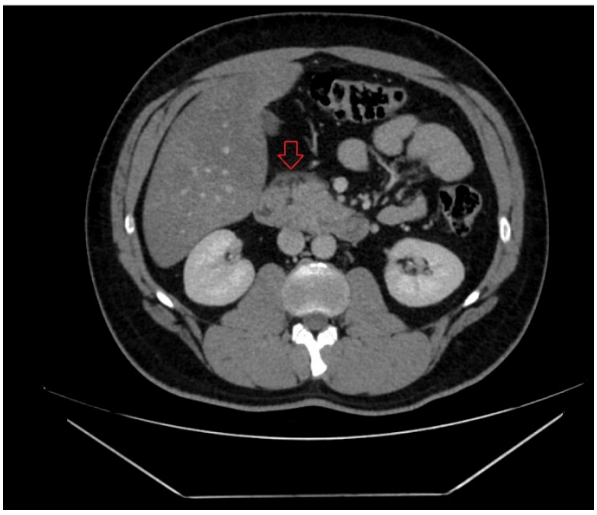

Figure 1. Abdominal computed tomography (CT) image showing stranding and inflammatory findings in the fatty planes at the head of the pancreas (red arrow).

Due to unresponsiveness to medical treatment and insulin infusion, ongoing symptoms, and persistent high triglyceride levels, the patient was transferred to a third-level tertiary center where more advanced treatment modalities (plasmapheresis) could be applied. The patient was transferred to the Anesthesiology and Reanimation Intensive Care Unit of a university hospital in Konya. On admission to external health facility (university), the serum triglyceride level was measured as 1800 mg/dL. It was observed that the serum creatinine level returned to the normal range with adequate ongoing IV fluid replacement. The patient was consulted with the gastroenterology department. Due to the diagnosis of severe AP on the basis of hypertriglyceridemia and unresponsiveness to medical treatment, an urgent plasmapheresis decision was made. The patient underwent plasmapheresis placing a right femoral central venous catheter. The post-procedure control triglyceride level decreased to 650 mg/dL. A significant improvement was observed in the patient's clinical condition after plasmapheresis, abdominal pain decreased, nausea-vomiting regressed, and the need for analgesics decreased.

In addition to plasmapheresis treatment, the patient's treatment was continued as follows: IV fluid replacement (maintenance), IV calcium replacement was performed considering the potential risk of hypocalcemia. Isotonic (SF) infusion and intravenous saline treatments continued. Medical treatment was initiated for long-term triglyceride control: Fenofibrate SR (Lipofen SR) 200 mg/day, omega-3 fatty acids (fish oil capsule) 3x2 capsules/day, analgesic treatment was continued as needed. The patient, who was on nil per os (NPO) for a total of four days, started oral feeding on the 4th day.

After a total of 4 days of monitoring in the intensive care unit, the patient, who remained clinically and biochemically stable, was transferred to the gastroenterology service. Upon tolerating oral intake in the ward, a low-fat diet was initiated. Medical treatments (Fenofibrate, Omega-3) were continued. The pre-discharge control triglyceride level was found to be 650 mg/dL. The patient, whose general condition was good and complaints had regressed, was discharged with dietary recommendations, medication adjustments, and a planned outpatient clinic follow-up.

Discussion

This case report presents the clinical presentation and management of severe hypertriglyceridemia-related acute pancreatitis (HTG-AP) that developed in a young male patient. Acute pancreatitis, regardless of its etiology, is a potentially life-threatening condition. In the presented case, the patient's high amylase (614 U/L) and lipase (2401 U/L) values at admission, along with peripancreatic inflammation findings detected on CT, supported the diagnosis of acute pancreatitis. The very high triglyceride levels measured during follow-up, such as 1251 mg/dL and 1800 mg/dL, indicated that the etiology was most likely HTG (1). It is known that in severe HTG, lipemic serum can interfere with laboratory measurements, potentially suppressing amylase and lipase levels. The markedly elevated enzyme levels in this case suggest that this interference may not always occur or may be partial.

The high creatinine (2.3 mg/dL) and urea (22.8 mg/dL) values detected in our patient at admission suggest acute kidney injury (AKI) secondary to intravascular volume loss and/or systemic inflammatory response associated with acute pancreatitis. Indeed, the rapid return of renal function tests to normal with aggressive fluid resuscitation supports this situation. Early and adequate fluid replacement is vital in preventing complications in acute pancreatitis.

One of the main goals in the treatment of HTG-AP is to rapidly reduce serum TG levels (14). One of the frequently used methods for this purpose is intravenous insulin infusion (14, 15). Insulin accelerates the breakdown of chylomicrons and VLDL by increasing the activity of the lipoprotein lipase (LPL) enzyme and promotes the uptake of FFAs by peripheral tissues (16, 17). Glucose-insulin-potassium infusion was also administered to our patient at the initial center, but the expected significant decrease in TG levels was not achieved. This indicates that insulin therapy alone may not be sufficient in some patients, or that the severity of the underlying

HTG may be resistant to this treatment.

In cases of severe HTG-AP that are unresponsive to medical treatment or present with very high TG levels (>1000-2000 mg/dL), therapeutic plasma exchange (plasmapheresis) or lipid apheresis is an effective treatment option (18). Plasmapheresis is the process of removing plasma from the body to eliminate triglyceride-rich lipoproteins and possibly pro-inflammatory cytokines, and replacing it with fresh frozen plasma or albumin solution (18). This method can achieve a rapid and significant reduction in TG levels. In the presented case, the TG level of 1800 mg/dL was reduced to 650 mg/dL after a single session of plasmapheresis (using albumin solutions), and a significant improvement in the patient's clinical symptoms was observed in parallel with this decrease. This result supports the effectiveness of plasmapheresis in selected HTG-AP cases (19). The decision for plasmapheresis is usually considered in patients with TG > 1000 mg/dL who show signs of organ failure or do not respond to traditional medical treatment (19, 20).

Following plasmapheresis, fenofibrate and omega-3 fatty acids were added to the patient's treatment. Fibrates (such as fenofibrate) are peroxisome proliferator-activated receptor-alpha (PPARα) agonists that lower TG levels by increasing LPL activity and reducing hepatic VLDL production (21). Omega-3 fatty acids similarly exhibit a TG-lowering effect by reducing hepatic VLDL synthesis and secretion (22). These drugs are used after acute treatment to ensure long-term TG control and prevent pancreatitis recurrence.

The patient's HbA1c value of 8.2% in this case suggested that pancreatic function had been impaired for some time and that secondary diabetes due to pancreatitis might be present. In particular, the history of intermittent, non-severe pain radiating to the epigastric region suggests that the pancreatitis spanned a 2-3 month period and progressively increased its severity. The development of diabetes after pancreatitis is a known condition in the literature (23).

This case also highlights the importance of a multidisciplinary approach in the management of HTG-AP. Coordination between the emergency department, intensive care unit, internal medicine, gastroenterology, and apheresis unit played a critical role in the successful execution of the patient's diagnosis and treatment.

One limitation of this case presentation is that the underlying cause of the patient's HTG (whether it was primary familial dyslipidemia or a secondary cause) was not investigated/is unknown in detail. It is important to make this distinction and determine the long-term management strategy during post-discharge outpatient follow-ups.

Conclusıon

Hypertriglyceridemia is a significant cause of acute pancreatitis, although it rarely leads to serious complications. It should be considered, particularly when serum triglyceride levels are above 1000 mg/dL. Early diagnosis, aggressive fluid replacement, and pain control are the cornerstones of treatment. Intravenous insulin infusion can be initiated to rapidly lower serum triglyceride levels. However, in severe cases that do not respond to conservative treatment and insulin infusion, or in those presenting with very high triglyceride levels, plasmapheresis is an effective and safe treatment method that can rapidly reduce triglyceride levels and lead to clinical improvement. This case once again demonstrates the role of plasmapheresis in the management of severe HTG-AP and the importance of a multidisciplinary approach. After the acute attack is controlled, investigation of the underlying cause, appropriate dietary adjustments, and long-term lipid-lowering medical treatment (fibrates, omega-3, etc.) are necessary to prevent recurrences.

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3087

Case Report: Fournier Gangrene

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Introductıon

Fournier's gangrene is a rare and severe form of necrotizing fasciitis affecting the external genitalia, perineum, or perianal regions. It predominantly occurs in males, with risk factors including diabetes, HIV, alcoholism, and other conditions that compromise the immune system. The disease has a high mortality rate of 20-30%. Due to the increasing age and diabetes prevalence in the population, there is a growing need for heightened clinical awareness, particularly regarding early diagnosis and treatment (1).

We present a case of 50-year-old male with Fournier gangrene possibly originated from transspheric fistula and abscess.

Case Report

50-year-old patient presented to the emergency department with swelling and pain in scrotal area. Patient had a history of transsphincteric grade 3 perianal fistula, recurrent urinary tract infections and inferior myocardial infarction.

In physical examination patient's vital signs were stable with a blood pressure of 131/80, heart rate of 81 and SpO2 of 96 in room air. His fever was 37.2 and his neurological exam was insignificant with a GCS score of 15. Upon inspection of testes major swelling and hyperemia in right testis was noted. Furthermore, there was crepitation upon palpation. Additionally, a necrotic area was noted on the right testis (figure 1). The Phren sign was positive.

Figure 1

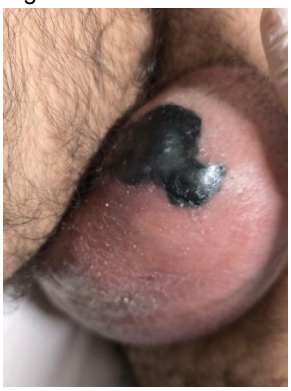
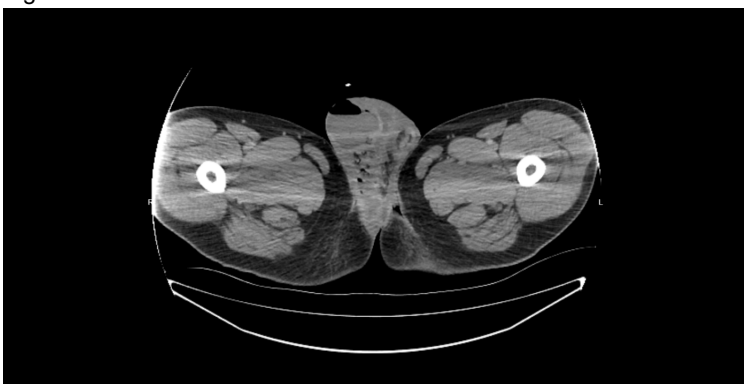


Figure 2



His FBC, biochemistry panel, urine culture and blood gas were taken:
WBC was 18.11 K/uL, NEU count was 15.85 K/uL, LYMPH count was 1.15 K/uL, HGB was 12.8 g/dl
Glucose was 88mg/dl, Creatinine 1,15mg/dl, ALT: 32 U/L AST:38 U/L CRP:330,9 mg/L
Ph:7.421, Pco2:35,3 mmHg, HCO3 22,4 mmol/L Lactate 2.93 mmol/L.
No growth in the urine culture.

HIV screening was negative.

Subsequently, an ultrasound and CT scan were performed. A fluid loculation with high internal density, possibly an abscess, was detected extending widely between the right scrotal fascial layers, reaching a depth of approximately 4-5 cm, containing multiple echogenicities likely corresponding to air bubbles. (Figure 2 and 3)

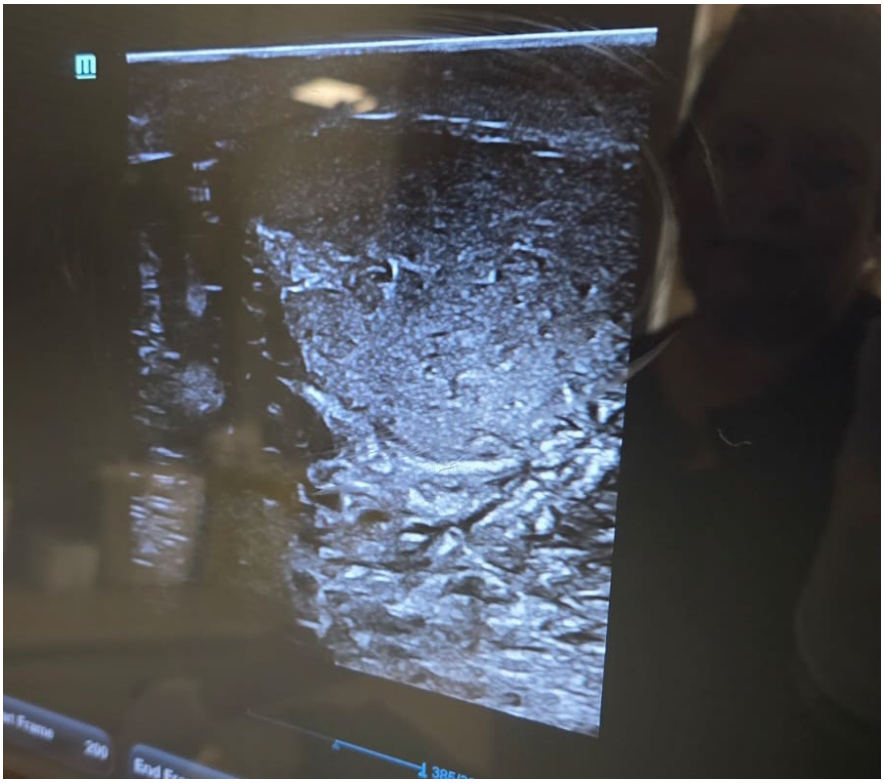


Figure 3: USG scan

Broad-spectrum antibiotics were initiated, and General Surgery and Urology were consulted. Urgent surgery was recommended by Urology due to a diagnosis of Fournier's gangrene. The patient was transferred to a more advanced facility for surgery.

Dıscussıon:

Most common symptoms of Fournier Gangrene are perineal pain, erythema, cellulitis, fever, abscess and crepitus (2). Our case represents a textbook presentation of Fournier including all key symptoms upon presentation. Especially, patients with diabetes mellitus, hypertension, alcoholism and advanced age (typically 53-55 years old) are at significant risk (3).

In cases of clinical suspicion, emergency computed tomography can be very beneficial for early diagnosis. Asymmetric fascial thickening, subcutaneous emphysema, fluid collections and abscess formation are common in FG. Even though subcutaneous emphysema is a hallmark feature for FG, it is not seen in all cases. Although ultrasound can be used for diagnosis, its specificity is lower than CT scan (4). Our case exhibited nearly all features of Fournier Gangrene in both USG and CT scans.

Early treatment of Fournier Gangrene in the emergency room consists of fluid resuscitation and the administration of broad-spectrum antibiotics. In FG patients, gram-positive and gram-negative bacteria with anaerobic bacteria are commonly isolated. Recently, the community-acquired methicillin-resistant Staphylococcus aureus (CA-MRSA) became an important and dangerous cause of FG. Additionally, fungal infections have been detected in some cases (5).

Early debridement surgery is necessary to reduce mortality. After surgery reconstruction methods such as skin grafting and flap reconstruction may be used. Negative pressure therapy, hyperbaric oxygen therapy, tissue adhesives and local application of honey and bactericidal agents have also been reported in the literature as adjuvant therapy methods. However, their efficacy has not been proven (6). Fournier Gangrene has a high mortality rate, reaching 18% which has not changed over the past 25 years (7).

Conclusion:

Fournier’s gangrene has a high mortality rate that has remained unchanged for the past 25 years despite advancements in medicine. Therefore, maintaining a high level of clinical suspicion is crucial for timely diagnosis and intervention. Early detection and prompt treatment are essential to improving patient outcomes. We present this case because it serves as an excellent example of Fournier’s gangrene, demonstrating both the characteristic clinical features and radiological findings.

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3125

A Rare And Potentıally Fatal Condıtıon Easıly Mıssed In The Emergency Department: Ludwıg's Angına

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Introductıon

Emergency department visits with soft tissue infections and related complaints are quite frequent. While the management of these patients in the emergency department is often simple drainage and antibiotic therapy, some rare soft tissue infections can be fatal (1). One of the most important of these is Ludwig's Angina (LA). LA is a rapidly progressive soft tissue infection that causes airway obstruction, which can lead to death. LA is usually caused by periodontal infections due to oral streptococci or aerobic-anaerobic oral mucosal flora bacteria (2).

Case

A 32-year-old male patient presented to the emergency department with complaints of right cheek pain and sublingual swelling. He reported experiencing swelling and pain in his jaw and right cheek for approximately 5-6 days. He had been started on oral flucloxacillin (500mg) tablets by his dentist with a suspected dental abscess. On the 2nd day of antibiotic treatment, the patient's complaints did not improve; instead, the swelling worsened. Upon arrival at the emergency department, the swelling in his right cheek and submandibular area had spread toward his neck, and the base of his tongue was edematous (Figure 1). In addition to the pain, he reported difficulty swallowing and shortness of breath. The patient had difficulty speaking. Physical examination revealed limited mouth opening, and the oropharynx could not be clearly evaluated. The vital signs were as follows: BP: 130/75 mmHg, SpO2: 98%, Pulse: 95 bpm, Temperature: 39.5°C. Lung sounds were normal. Blood tests were performed, and intravenous treatment was initiated in the emergency department with 1g paracetamol, 60 mg prednisolone, 45 mg pheniramine, 2g ceftriaxone, and 500 cc normal saline.The laboratory results were as follows: CRP: 237 mg/dL, WBC: 15 x 10^3/µL, NE: 13 x 10^3/µL, Procalcitonin: 0.31 µg/L, Hgb: 11.1 g/dL, Plt: 178,000/µL. Neck ultrasonography revealed an increased and edematous right submandibular gland with hypoechoic areas in the posteromedial gland, suggestive of an infectious process, and follow-up was recommended for possible abscess. There was also thickening and heterogeneity in the muscle plane superior to the right mandible, thickening in the skin and subcutaneous tissues, and mild edema in the left submandibular areas. Reactive lymph nodes in the right cervical chain, the largest measuring 18x8 mm, were suggestive of infection. A neck CT scan was requested for better visualization of the involvement (Figure 2). The neck CT scan showed increased soft tissue thickness narrowing the air column on the right at the oropharynx level, a lymph node measuring approximately 15x7 mm at the right submandibular gland level, and edematous thickening in the fatty planes between the lymph nodes at this level, consistent with infection.

The patient was consulted with an Ear, Nose, and Throat (ENT) specialist with a preliminary diagnosis of Ludwig’s angina. Subsequently, an infectious diseases specialist was consulted for treatment planning. The patient was admitted to the ENT service.

During the follow-up in the service, treatment with ampicillin + sulbactam, metronidazole, and decadron was initiated, and it was learned that alpha-hemolytic streptococcus grew in the culture taken. On the 10th day of IV antibiotic therapy, the patient's complaints subsided, and he was discharged without any additional complaints.

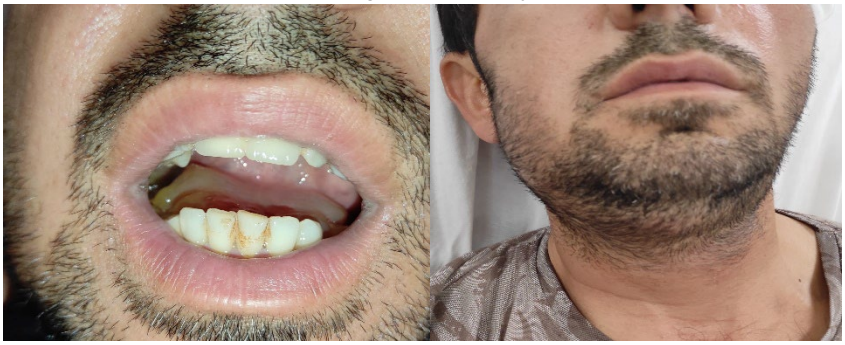

Figure 1: Right cheek and neck swelling, sublingual edema.

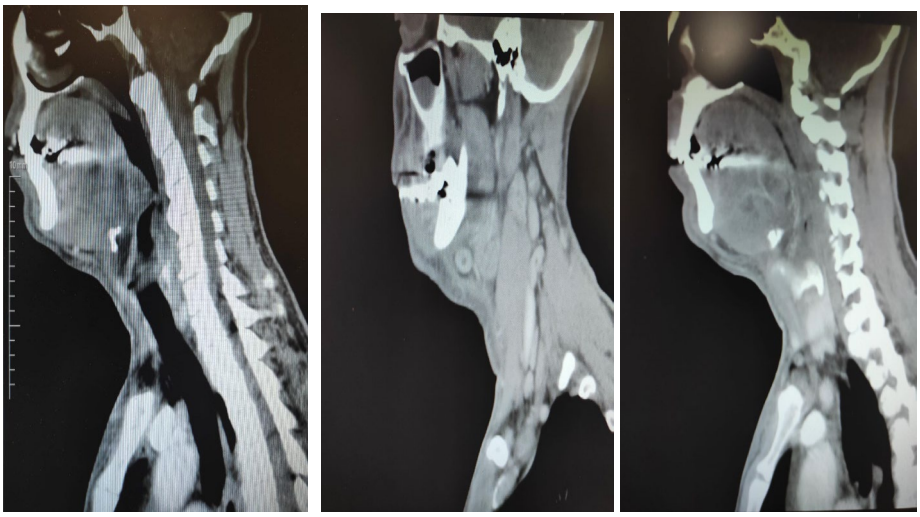

Figure 2: Increased soft tissue thickness narrowing the air column at the oropharyngeal level, a lymph node measuring approximately 15x7 mm at the right submandibular gland level, and edematous appearance in the fatty planes between the lymph nodes at this level.

Dıscussıon

LA is a rapidly progressive and potentially fatal form of cellulitis that occurs in the sublingual, submental, and submandibular regions. It generally originates from recently extracted or infected teeth (3,4). The most serious complication of LA is asphyxia due to significant edema in the soft tissues of the neck (5). Other complications include the spread of infection to the mediastinum, carotid sheath, skull base, and meninges, which increases mortality from 20% to 50% (4). Emergency department visits with complaints of toothache and swelling in the area after tooth extraction are common. It is also a widespread practice to prescribe analgesics and antibiotics without detailed examination and imagingduring these visits. However, emergency department visits for rapid and fatal LA are similar.

In our case, there was also swelling thought to originate from an infected tooth. The patient had difficulty breathing. The presence of fever and difficulty swallowing, in addition to the swelling in the neck, alerted the examining physician. Advanced laboratory tests and imaging were requested, considering the preliminary diagnoses of abscess and cellulitis in the neck region, and treatment was initiated by making an early diagnosis of LA.

Conclusıon

Every soft tissue infection seen in the face and neck region can be a potential LA. Especially in patients presenting to the emergency department with these complaints, the diagnosis of LA should be kept in mind. Because early diagnosis and early treatment of LA, which can have a serious fatal course especially due to airway obstruction, significantly reduces the mortality of LA. Making the diagnosis of LA at the emergency department presentation is very valuable in this sense.

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Did you eat salad too?

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Introduction and Purpose

Anticholinergic syndrome is a clinical condition that can arise from 600 different substances. The involvement of muscarinic and nicotinic receptors can lead to various central and peripheral responses in the patient. We described one of the three similar cases that came to our emergency department within two days.

Case

A 60-year-old female patient was brought to the emergency department with complaints of nonsensical speech and movements, inappropriate laughter, and altered consciousness, which began one hour ago. Vital signs: Blood pressure (BP): 154/108 mmHg, blood sugar (BS): 163 mg/dl, pulse: 121 beats/min, temperature: 37.5°C, SpO2: 98%, electrocardiogram (ECG): sinus tachycardia. Her medical history includes Diabetes Mellitus (DM) and Hypertension (HT). She is taking oral antidiabetic and antihypertensive

medications.

On examination, the patient was partially cooperative and disoriented. Light reflex (LR) was bilaterally negative, and pupils were bilaterally 4 mm and mydriatic. There was no neck stiffness. Her skin was dry, and there was redness on her face. Imaging and laboratory tests did not reveal any pathology that could explain the current clinical picture.

The patient's history revealed that she had eaten a full plate of salad containing arugula and watercress two hours before the onset of symptoms. Despite treatment, the patient's agitation continued. Following the administration of 2 mg of physostigmine, the patient's level of consciousness returned to normal.

Discussion and Conclusion

Atropa Belladonna (deadly nightshade) has a strong anticholinergic effect. It can cause toxicity within 1 to 4 hours after ingestion. Central effects can lead to clinical conditions and symptoms such as fever, memory loss, agitation, hallucinations, delirium, and coma. Peripheral effects include mydriasis and increased intraocular pressure in the eyes, dryness in mucous membranes and sweat glands, skin redness, and retention in the gastrointestinal and urinary systems.

Treatment generally involves observation, monitoring, and supportive care. For moderate to severe anticholinergic syndrome, pharmacological sedation is strongly recommended to achieve adequate control. Physostigmine may be used in patients with severe agitation and delirium due to pure anticholinergic toxicity who do not benefit from benzodiazepines.

The occurrence of a large number of cases within a short period should be regarded as a public health issue. Although reporting is mandatory, activating the surveillance system means addressing the problem at its source.

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3241

The unexpected culprit of abdominal pain: a case of mesenteric panniculitis

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Introductıon

Mesenteric panniculitis (MP) is a rare, benign inflammatory disorder characterized by inflammation, fat necrosis, and fibrosis of mesenteric fat tissue (1). First described by Jura in 1924, it has also been referred to as sclerosing mesenteritis and mesenteric lipodystrophy (2). Although MP primarily affects the mesentery of the small intestine, it can, in rare instances, involve the mesocolon, omentum, and retroperitoneal region (3). The precise etiology remains unclear; however, MP has been associated with various factors, including prior abdominal surgery, trauma, mesenteric ischemia, pancreatitis, autoimmune diseases, and malignancies (4). Some studies suggest a potential link between MP and underlying malignancies (5).

MP is often asymptomatic and incidentally detected during imaging performed for unrelated reasons (6). When symptomatic, the most common presenting complaint is chronic or acute abdominal pain (7). Other nonspecific symptoms include loss of appetite, weight loss, nausea, vomiting, diarrhea, constipation, and fever. In some cases, patients may present with a palpable abdominal mass (8).

Physical examination findings are variable. Some patients exhibit abdominal tenderness, while others may present with guarding or rebound tenderness (9). Laboratory findings are typically unremarkable, though some cases may show leukocytosis and elevated C-reactive protein (CRP) levels (5). Imaging plays a key role in diagnosis. While ultrasonography (USG) may reveal increased echogenicity and heterogeneity in mesenteric fat tissue, computed tomography (CT) remains the gold standard for diagnosis (6). CT findings commonly include increased mesenteric fat density due to inflammation, involvement of adjacent vascular structures, and the characteristic "mesenteric swirl sign." Magnetic resonance imaging (MRI) can provide additional diagnostic clarity, particularly in ruling out malignancies (7).

The management of MP is guided by symptom severity. Asymptomatic cases generally do not require treatment, with spontaneous resolution reported in some instances (9). Mild symptomatic cases may be managed with nonsteroidal anti-inflammatory drugs (NSAIDs) or low-dose corticosteroids (4).

Case Presentatıon

A 49-year-old female patient with no known chronic illnesses presented to the emergency department with sudden-onset abdominal pain, nausea, and vomiting. On physical examination, her vital signs were stable, and mild abdominal tenderness was noted without guarding or rebound tenderness. Laboratory tests revealed a mild elevation in inflammatory markers. As the patient did not respond to symptomatic treatment, contrast-enhanced abdominal CT was performed for further evaluation. The imaging findings were consistent with mesenteric panniculitis. The patient was subsequently evaluated by the general surgery department and admitted for further assessment and management.

Dıscussıon

Mesenteric panniculitis is a rare inflammatory disease that is frequently asymptomatic but may present with acute or chronic abdominal pain, nausea, vomiting, and alterations in bowel habits (1). Although the precise etiology is unknown, MP has been associated with prior abdominal surgery, trauma, infections, autoimmune diseases, and malignancies (2). However, in this case, the patient had no history of chronic disease or previous surgery, suggesting that MP may occur without a clear predisposing factor.

The patient's presenting symptoms were consistent with those reported in the literature. Abdominal pain is the most frequently documented symptom, often leading patients to seek medical attention (3,4). In some cases, severe pain and systemic symptoms can mimic an acute abdomen. However, in this case, the patient's symptoms were relatively mild, with no guarding or rebound tenderness observed. This highlights the diverse clinical presentation of MP, which can range from mild discomfort to severe abdominal pain.

Imaging is central to the diagnosis of MP. In this case, due to the patient's lack of response to symptomatic treatment, contrast-enhanced CT was performed, revealing findings characteristic of mesenteric panniculitis. The literature supports the use of CT as the gold standard for diagnosing MP, with key imaging findings including increased mesenteric fat density, involvement of adjacent vascular structures, and the mesenteric swirl sign (6).

Conclusıon

Mesenteric panniculitis is a rare condition with a broad clinical spectrum and nonspecific symptoms. As in other reported cases, diagnosis is primarily established through imaging, and management is largely symptomatic. However, given the potential association with underlying malignancies or inflammatory conditions, each case should be carefully evaluated to exclude other possible etiologies.

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Rectal Foreign Body: A Case Report

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Introduction: Foreign bodies located in the rectum have been seen increasingly in recent years and constitute one of the important emergency problems due to the complications they cause. They can be placed in the rectum as a result of sexual assault, self-treatment of anorectal disease, accident, rarely swallowing and most frequently sexual satisfaction. They are most commonly seen in men aged 30-40. Although rare, they can also be seen in older ages. Patients usually present with rectal and lower abdominal pain. Unreliable anamnesis, palpation of foreign bodies during rectal examination and foreign body opacity on plain abdominal radiography are important for diagnosis. They can cause complications such as rectal bleeding, perforation, laceration and infection. However, the mortality rate is quite low.

Case: An 80-year-old male patient presented to the emergency department with complaints of tenesmus and lower abdominal pain. The patient provided conflicting information in the detailed anamnesis. There was no abdominal defense or rebound on physical examination. Complete blood count, sedimentation, C-reactive protein level and blood biochemical tests were normal. A bottle-shaped foreign body was seen at the pelvic level on plain radiography (Figure 1). Tetanus prophylaxis was administered. There was no perforation observed on abdominal computed tomography (CT) (Figures 2,3). The foreign body was removed. The patient was followed up in the general surgery department for 3 days and was discharged when no perforation was detected on follow-up imaging. The patient presented with complaints of bloody diarrhea 20 days after discharge. After perforation was excluded on imaging methods, the patient was started on dual antibiotic therapy.

Conclusion: Rectal foreign bodies are a difficult process that begins with diagnosis in the emergency department, continues with treatment, and then includes patient follow-up. One of the most common problems encountered in treatment is the delay in hospital admissions due to patients' embarrassment and hesitation to seek medical help. Most patients come after failing to remove the foreign body and do not give a realistic history. It is important for the diagnosis to include an rectal examination to abdominal examination. Plain abdominal radiographs may be important in determining the size and location of the rectal foreign body. Contrast-enhanced abdominal tomography is an important diagnostic tool for the detection of early perforation and other pathologies that may accompany it, such as intra-abdominal bleeding and damage to adjacent organs and structures. Rectosigmoidoscopy has an impor-

tant place for diagnosis and treatment purposes In the treatment of rectal foreign bodies, surgical or non-surgical methods may be preferred depending on the characteristics, location, complexity of the rectal foreign body. After nonsurgical removal of rectal foreign bodies, it is important to monitor patients for at least 24 hours and then perform follow-up rectosigmoidoscopy and/or imaging to see possible complications such as bleeding and perforation. Psychiatric consultation may be necessary for the behavioral etiology and psychotherapy of rectal foreign body cases.

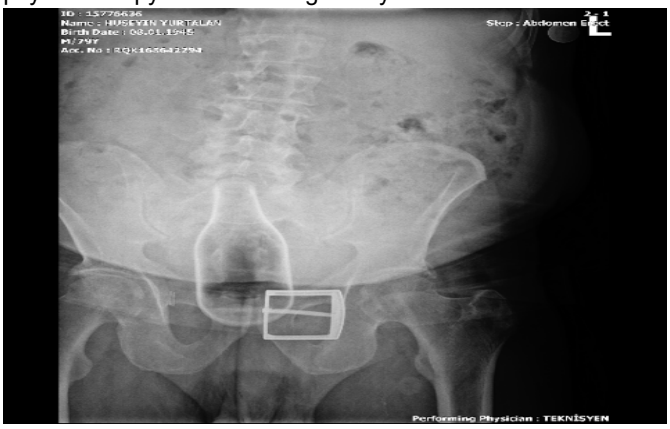

Figure 1. Bottle in the pelvic region on plain radiography

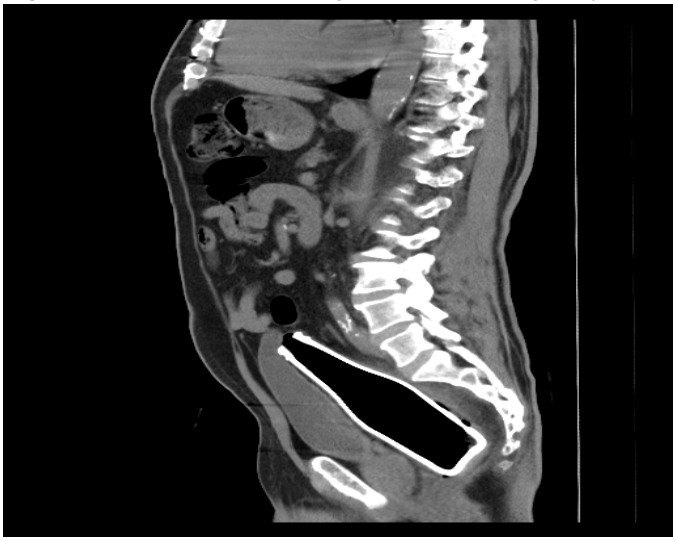

Figure 2. Bottle in the rectosigmoid region in sagittal CT

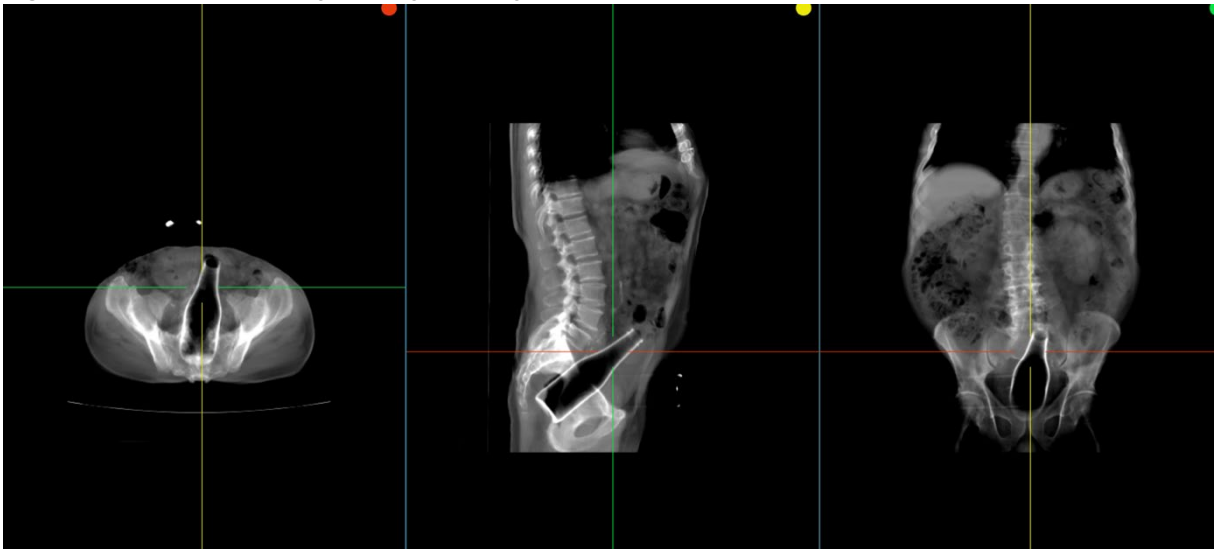

Figure 3. The image of the entire bottle was obtained after minimum intensity projection (MinIP) on axial, sagittal and coronal CT reformats.

Keywords: Foreign body, rectum, CT

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3290

Cold agglutinin disease: ice-cold effects, warm solutions

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Introductıon

Cold Agglutinin Disease (CAD) is a rare but clinically significant autoimmune hemolytic anemia. The disease is characterized by agglutination and hemolysis triggered by IgM antibodies binding to erythrocyte surface antigens at low temperatures (1). CAD can be classified as either primary (idiopathic) or secondary. In secondary cases, Mycoplasma infection, Epstein-Barr virus (EBV), and lymphoproliferative diseases are important triggering factors (2). This report discusses the diagnosis, clinical course, and treatment of CAD in a 25-year-old male patient, emphasizing the importance of a multidisciplinary approach.

Case

A 25-year-old male patient was admitted to the emergency department with complaints of high fever, cough, and shortness of breath persisting for approximately two weeks. His medical history revealed recent exposure to extreme cold and cyanosis in his hands and feet. On physical examination, his temperature was 38.8°C, heart rate was 104 bpm, respiratory rate was 90 breaths per minute, and blood pressure was 99/67 mmHg. Pulmonary auscultation revealed coarse rales in the left middle and lower lobes, and CT imaging showed peribronchial reticular and nodular infiltrative areas in both lower lung lobes, indicating atypical pneumonia (3).

A blood sample taken at room temperature showed artificially low erythrocyte count (0.35 × 10^6/mm^3) and hematocrit value (4.2%) due to agglutination detected by an automated hematology device. Mean corpuscular volume (MCV) was elevated (121.7 fL), while platelet count was within normal limits. However, after warming the sample to 37°C in a water bath, the erythrocyte count increased to 1.3 × 10^6/mm^3, and the hematocrit level rose to 16.4%. The direct Coombs test was positive, and immunofixation electrophoresis detected a monoclonal IgM kappa band, supporting the diagnosis of CAD (4, 5).

Dıscussıon

In this case, the patient's clinical history and physical examination findings strongly suggested infection-associated CAD. The infiltrative lesions observed on CT imaging indicate that Mycoplasma or viral agents may have damaged lung tissue, triggering an immune response (6). Laboratory findings emphasize the importance of sample handling techniques. The artificial decrease in erythrocyte count observed in samples taken at room temperature could lead to misdiagnosis and incorrect treatment planning, making proper sample handling essential (7).

In the pathophysiology of CAD, IgM antibodies activated in cold environments bind to the "I" antigen on the erythrocyte membrane, leading to complement system activation and hemolysis. This results not only in classic symptoms such as anemia and jaundice but also in peripheral vascular signs like acral distress and livedo reticularis (8). Diagnostic tools such as direct antiglobulin testing and thermal amplitude analysis play a critical role in confirming the disease. Additionally, laboratory teams must be aware of potential inconsistencies in test results and ensure samples are warmed to 37°C for accurate reassessment (9).

Treatment strategies focus on preventing the patient's exposure to cold, controlling infections, and employing plasmapheresis in severe hemolysis cases. Supportive treatments and, if necessary, immunosuppressive therapies aim to halt disease progression. This case highlights the critical importance of a multidisciplinary approach in the diagnosis and treatment of CAD, emphasizing the need for close collaboration between clinical and laboratory teams (10).

Conclusıon

This case report details the diagnosis, clinical course, and treatment of infection-associated Cold Agglutinin Disease in a 25-year-old male patient. Careful interpretation of clinical history, CT imaging, and laboratory tests is essential for the accurate diagnosis and effective treatment of rare conditions like CAD. Proper sample collection techniques and a multidisciplinary approach play a crucial role in preventing potential complications. Early diagnosis and appropriate treatment strategies are vital for improving patient quality of life.

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3404

An interesting cause of abdominal pain in the emergency room

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Introduction

Abdominal pain is a common cause of emergency department admissions. It is challenging and high of readmissions. Careful evaluation and systematic approach towards the cause prevents overlooking potentially serious conditions (1).

Many blood tests and imaging methods, including direct radiography, ultrasound and tomography, are used in diagnosis.

In this case report, we aimed to present an interesting case of abdominal pain who presented to the emergency department with complaints of abdominal pain and constipation for 2 days.

Case report

A 76-year-old woman presented to our emergency department with complaints of abdominal pain and constipation for 2 days. In her anamnesis, she stated that she had frequent constipation complaints before. Her medical history included asthma, hypertension (HT), anxiety, total abdominal hysterectomy + bilateral salpingooopherectomy (TAH+BSO), and appendectomy. On physical examination, the patient's general condition was good, oriented, cooperative and vital signs were fever: 36.5°C, pulse rate: 103/min, blood pressure: 110/70mmHg, respiratory rate: 16/min. The abdomen was distended, there was diffuse tenderness and bowel sounds were hypoactive (2-4/min). Blood tests revealed white blood cell count (WBC) 13,800 /mm3, hemoglobin count (Hgb) 6.9 g/dL, lactate 2.5 mmol/L. She was otherwise normal. Standing plain abdominal radiography (SPAR) revealed approximately 10-12 similarly shaped hyperdense foreign bodies (long dimension measured 15 mm and short dimension measured 5 mm) in the left lower quadrant and left upper quadrant (Figure 1). Subsequently, abdominal computed tomography (CT) performed for differential diagnosis showed nodular foreign bodies with dense calcific-metallic character with different locations and contents, one in the stomach and the others in the colon, measuring 15 mm at the most prominent location, with a density of 2500-3000 HU (Figure 2). General surgery and internal medicine consultations were requested. General surgery did not consider emergency surgery and recommended outpatient clinic. The patient left the hospital voluntarily without waiting for internal medicine consultation.

The next day, the patient's complaints increased and vomiting and black stools were added to her complaints. The patient's general condition was poor and rectal examination revealed melena. In blood tests, leukocytosis, anemia and lactate level deteriorated further (Hgb: 4.9 g/dL, lactate: 5.8 mmol/L). The patient was consulted to the gastroenterology department considering gastrointestinal system (GIS) bleeding. The patient was hospitalized in the intensive care unit with a prediagnosis of GI bleeding for further investigations and treatment. Endoscopy performed 3 days later revealed no nodular foreign body with calcific-metallic appearance, but a large submucosal mass on the posterior wall of the gastric antrum-corpus junction, approximately 6 cm in size, with 2 white exudate ulcers of 6-7 mm in size. This vision was consistent with gastrointestinal stromal tumor (GIST). The patient showed clinical improvement in the follow-up and was discharged with a recommendation for general surgery outpatient clinic control.

Discussion

Abdominal pain is a common condition that accounts for 5-10% of emergency department admissions. However, 20% to 30% of patients with abdominal pain leave the hospital without a definitive diagnosis (2).

Laboratory and imaging methods are used in the diagnosis and differential diagnosis of diseases underlying abdominal pain (3-6). In our case, we ordered blood tests for abdominal pain. Blood tests revealed leukocytosis, anemia and elevated lactate levels. SPAR and CT scans were then ordered as imaging modalities. SPAR revealed approximately 10-12 similarly shaped hyperdense foreign bodies in the left lower and left upper quadrants. Subsequently, an abdominal CT scan confirmed the presence of foreign bodies in the stomach and colon and excluded other possible causes of abdominal pain.

Afterwards, the patient developed melena and gastroenterology department was consulted considering GIS bleeding. Endoscopy and colonoscopy were performed by the gastroenterology department 3 days later. No nodular foreign bodies with calcific-metallic appearance were detected in endoscopy and a large submucosal lesion compatible with GIST with 2 white exudate ulcers was observed on the posterior wall of the gastric antrum - corpus junction. When the patient's previous examinations were examined, en-

doscopy and colonoscopy performed 5 months ago due to the patient's anemia showed no metallic appearance and SPAR performed 6 months ago showed no hyperdense image.

Figure 1. Hyperdense foreign bodies on standing plain abdominal radiography

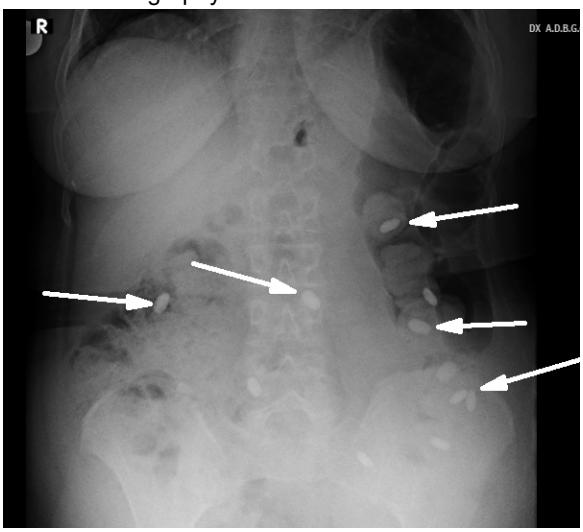
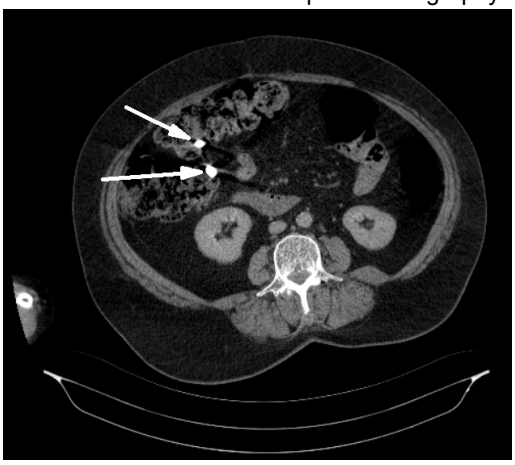


Figure 2. Nodular foreign bodies with dense calcific-metallic character on abdominal computed tomography



We considered atypical eating disorder as the cause of the foreign bodies present in the patient. Atypical eating disorders are eating behaviour disorders that lead to medical, social and psychological problems and affect quality of life negatively (7,8). Epidemiologic risk factors for atypical eating disorders include cultural characteristics, gender, age, mental disorders in the family, family lifestyle, socio-economic class, personality role, previous psychological disorders, genetics, sexual orientation and occupation (9). Atypical eating disorder are based on a type of self-injurious behaviour involving the ingestion of non-nutritive objects. The result is usually excretion of the ingested substances through feces, but rarely requires surgical intervention (10). Our patient was an elderly woman with anxiety disorder and psychiatric drug use. We thought that the patient had atypical eating disorder and the high-density calcific-metallic nodular lesions on imaging to be excreted through feces.

Conclusion

It should be kept in mind that patients presenting with abdominal pain may have atypical eating disorders underlying the high-density calcific-metallic nodular lesions seen in the stomach and intestines in imaging methods.

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3420

Determining Brain CT Indications in Pediatric Patients Presenting to the Emergency Department with Minor Head Trauma Using PECARN Score and Emergency Physician Judgment via an Artificial Intelligence Algorithm

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Introduction and Purpose: Pediatric traumatic brain injury (TBI) is one of the most common causes of acquired disability and death. In addition, traumatic brain injury is a very heterogeneous condition and the risk of long-term disability after survival is high in children with head trauma. Fast and correct use and prompt intervention will reduce bad results. In addition to classical diagnostic methods, modern diagnostic methods will also contribute to this situation.Our aim in our research is to create an artificial intelligence (AI) cleaner with the pediatric expanding ChatGPT-4o program in emergency services with minor head trauma and to capture brain CT scan indications with PECARN score and emergency medicine expert assistance. This is envisaged to assist in decision making for minor head trauma and follow-up brain CT scans, as well as the PECARN head trauma clinical scoring system and clinics, as well as the major trauma language models (LLM) stored with AI systems.

Materials and Methods: Our retrospective study included patients aged 18 and under who had minor head trauma and had computed tomography images taken at Ankara Bilkent City Hospital Emergency Medicine Clinic between March 2019 and March 2024. Our study included 1169 patients whose incident history, physical examination, age, gender and tomography records were taken at the hospital. We formulated the data for the study, calculated the PECARN score for the patient and also interviewed the emergency medicine specialist for the patient. The obtained data were labeled using ChatGPT-4o.

Results and Conclusion: When ChatGPT-4o recommendations were compared according to CT findings, a statistically significant difference was found between the groups, and this difference stems from the difference between the decisions it makes for findings that may have clinical significance and those that do not (Table 1). When compared with the comparison imaging results, it was seen that ChatGPT was more successful in terms of results according to expert opinions and PECARN guideline recommendations (Table 2). In addition, the compatibility between ChatGPT-4o recommendations and expert opinions was lower than that with PECARN (Table 3). A general overview of CT scan request distributions by PECARN guidelines, expert opinion, and ChatGPT-4o recommendations is presented in Table 4.

Number of Patients Requesting CT According to PECARN Guidelines and ChatGPT-4o Recommendations

			PECARN Guide		total patient	
		BT not recommend	BT recommend	follow it		
	BT not recommend	331(35.1)	6(7.1)	16 (11.3	335	
ChatGPT-4o	BT recommend	141 (15.0)	62 (72.9)	59 (41.5)	262	
	follow it	470 (49.9)	17 (20.0)	67 (47.2)	554	
		942 (100.0)	85 (100.0)	142 (100.0)	1169	
	kappa value:0,112 (p<0,001)					

Table 1

Number of Patients Requesting CT According to PECARN Guidelines and Expert Opinion

		PECARN Guidelines			
		BT not required	BT is required	Follow-up	total
	BT not required	764	11	44	819
	BT is required	89	73	64	225
expert opinions	Follow-up	89	1	34	124
	total	941	85	142	1169
	kappa value	0.377(p<0.001)			

Table 2

Number of Patients Requesting CT According to ChatGPT-4o and Expert Opinion

			expert opinions		
		BT not required	BT is required	Follow-up	total
	BT not required	309	29	24	353
ChatGPT-4o	BT is required	109	125	28	262
	Follow-up	410	71	72	553
	total	819	225	124	1168
	kappa value:**0.173 (p<0,001)**				

Table 3

CT Request Evaluation from Patients Aaccording to PECARN Guidelines, Expert Opinion, and ChatGPT-4o Recommendations

Parameters (n=1169	**n(%)**
PECARN Guide	
CT is not recommended	942 (80.6)
CT is recommended	85 (7.3)
Follow-up (Requested if clinical observation result is necessary)	142 (12.1)
Expert Opinion	
I don't want	819 (70.1)
ı want	225 (19.3)
Follow-up (Requested if clinical observation result is necessary)	124 (10,6)
CHATGPT-4o	
BT Not Recommended	53 (30.2)
BT Recommended	262 (22.4)
Follow-up	554 (47.4)

Table 4

3448

Bile duct obstruction: Mirizzi syndrome

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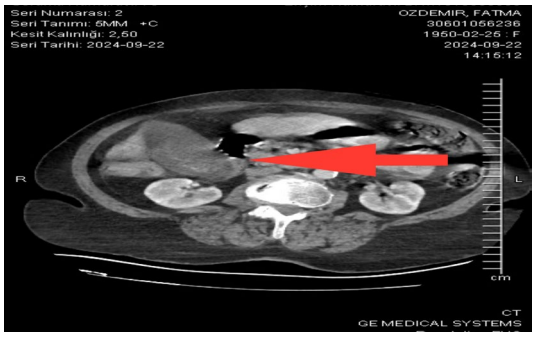
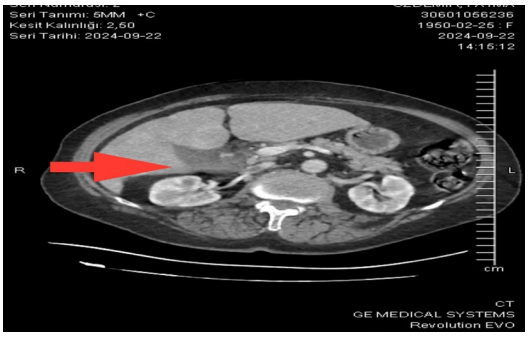
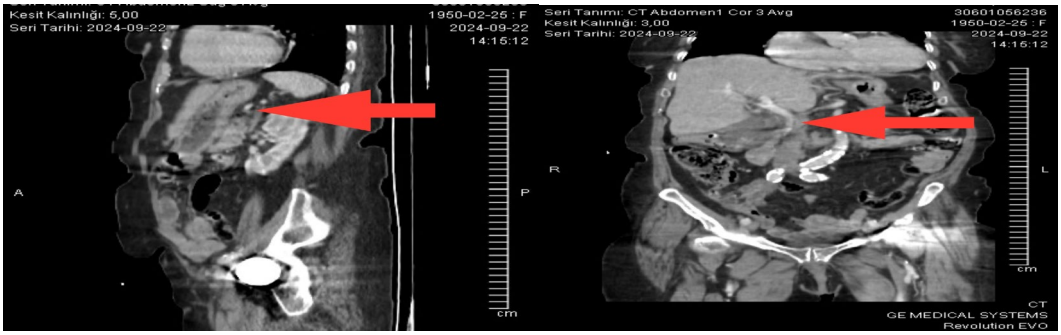
Introduction and Purpose : Mirizzi syndrome is an obstruction of the common bile duct caused by external compression of the common bile duct by a large stone sitting in the Hartman pouch of the gallbladder. Obstructive jaundice (obstructive icterus) develops in these patients. Obstructive jaundice is usually accompanied by fever and right upper quadrant pain. Inflammation also develops here. Chronic inflammation can lead to necrosis of the wall of the main hepatic duct and choledochal wall and eventually to fistula (cholecystohepatic and cholecystocholedochal fistula).

Materials and Methods : A 75-year-old woman patient. She presented to the emergency department with right upper quadrant pain. On examination, the patient had deficiency and rebound in the right upper quadrant. Other quadrant examinations were normal. There was gas stool discharge. The patient's medical history: hypertension, coronary artery disease.Vital signs fever: 36.7 C saturation:%96 blood pressure: 123/76 mm/hg. Blood sugar : 109 mg/dl. In the tests performed, wbc:10.7 10^3/dl, total bilirubin:1.36 mg/dl direct bilirubin:0.76 mg/dl ast:36 U/L alt:53 U/L crp:6 mg/L

Computed tomography was performed in the emergency department.







Findings and conclusion: Imaging showed a collection area in the gallbladder bed and centralization of the intrahepatic bile ducts. . Mirizzi syndrome was considered and the patient was consulted with general surgery. Percutaneous gallstone drainage was performed in the follow-up of the patient and this procedure was repeated in the following periods, but complete recovery was not achieved in the follow-up of the patient. Cholecystectomy was planned.

Conclusion: The most important point in the diagnosis of Mirizzi syndrome is to suspect this syndrome. The absolute solution of the disease is surgery. Complications in delayed diagnosis and treatment will significantly decrease the success of the treatment and reduce the comfort zone of the patient. Therefore, it should be ensured that these patients receive surgical treatment immediately.

3674

Globus Pallıdus Infarctıon Secondary To Carbon Monoxıde Intoxıcatıon

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Introductıon

Carbon monoxide (CO) is an odorless gas produced during the incomplete combustion of organic materials. The most common causes of CO poisoning include malfunctioning or inadequately ventilated gas heating appliances, stoves, and fires (1). CO enters the human body through the inhalation of flue gases and has an affinity for hemoglobin approximately 240 times greater than that of oxygen. This property allows CO to rapidly bind with hemoglobin, forming carboxyhemoglobin (COHb), which prevents oxygen transport to tissues. As a result, tissue hypoxia develops, and organs with high oxygen demand are particularly affected (2).

In CO toxicity, not only does the oxygen-carrying capacity of blood decrease, but disruption of the cellular respiratory chain also plays a significant role. Therefore, even if carboxyhemoglobin (COHb) concentration decreases, permanent tissue damage in the myocardium and brain may still occur (3). Seizures and other early neurological complications observed in severe CO poisoning are associated with significant toxicity, and aggressive hyperbaric oxygen therapy (HBOT) is recommended for these patients (4).

Radiological findings in CO poisoning can be variable (4). Current clinical guidelines recommend neurological imaging in patients with altered consciousness or neurological deficits instead of routine screening. Globus pallidus necrosis (GPN) is one of the characteristic neuroimaging findings of CO poisoning (5).

Case Report

A 60-year-old female patient was brought to the emergency department by EMS (112) due to suspected smoke inhalation following a stove malfunction at home. It was reported that the patient remained in the environment for over 24 hours after exposure and that her husband had succumbed to carbon monoxide (CO) intoxication.

On physical examination, the patient was conscious, cooperative, and oriented; however, lower extremity muscle weakness and aphasia (inability to speak) were noted. Her vital signs upon admission were as follows: oxygen saturation: 85%, blood pressure: 120/80 mmHg, heart rate: 140 bpm, temperature: 36.6°C, with bilateral coarse crackles on auscultation. Electrocardiography (ECG) revealed atrial fibrillation with a rapid ventricular response, and arterial blood gas analysis showed a negative carboxyhemoglobin (COHb) level.

The patient was placed in a secured environment and initiated on 100% oxygen therapy. Intravenous diltiazem was administered for rate control, which was successfully achieved. Due to the presence of neurological deficits following intoxication, brain computed tomography (CT) and diffusion-weighted magnetic resonance imaging (MRI) were performed. MRI revealed bilateral globus pallidus involvement. The neurology department was consulted for further evaluation and management. The findings were considered con-

sistent with carbon monoxide intoxication.

Consultation with the hyperbaric and toxicology advisory center concluded that hyperbaric oxygen therapy (HBOT) was not indicated for this patient. Meanwhile, the patient continued to receive 100% oxygen therapy in the emergency department. After approximately 16 hours of oxygen therapy, the patient's speech improved, and motor deficits regressed. She was discharged with a recommendation for neurology outpatient follow-up.

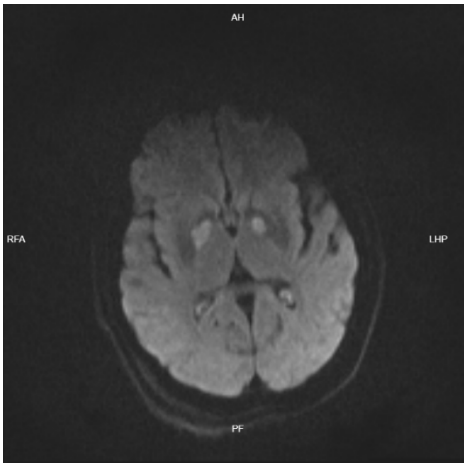

Figure 1: Bilateral globus pallidus involvement is observed in the patient's MRI

Dıscussıon

Deaths due to CO poisoning are accidental and typically occur in residential or occupational settings. Fatal CO poisoning is more common during the colder months of the year, particularly when faulty heating devices are used, and ventilation systems are closed (6).

Carbon monoxide (CO) poisoning is one of the most critical medical emergencies, leading to life-threatening conditions, including cardiovascular and neurological sequelae. Acute CO poisoning can result in myocardial ischemia, ventricular arrhythmias, syncope, seizures, and coma (4).

Carbon monoxide inhibits the mitochondrial electron transport system and activates polymorphonuclear leukocytes, leading to brain lipid peroxidation. This process may explain the delayed effects of CO poisoning, such as delayed encephalopathy. In patients exposed to carbon monoxide, acute brain injury is primarily caused by hypoxia. Neurons typically require large amounts of oxygen and glucose, making them highly susceptible to the effects of ischemia and hypoxia in the central nervous system. Acute carbon monoxide poisoning primarily leads to widespread hypoxic and ischemic encephalopathy, particularly affecting the gray matter (7).

Observing structural changes in the gray matter nuclei of patients with acute carbon monoxide poisoning using diffusion-weighted MRI is valuable for assessing the extent of deep gray matter damage, quantifying the severity of injury, and investigating its characteristics and relationship with memory and cognitive impairment (8).

CO binds to hemoglobin with a much higher affinity than oxygen, leading to hypoxemic hypoxia. Therefore, standard treatment primarily involves high-flow oxygen therapy, including hyperbaric oxygen therapy (HBOT), which accelerates the elimination of CO. Delayed neurological sequelae may develop after a lucid interval of 3 days to 4 weeks; however, the role of HBOT in these cases remains controversial. Thus, further studies are needed on this subject (9).

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3706

Traumatic Aortic Transection Following Motor Vehicle Collisions: A Four-Patient Case Series

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Introductıon:

Traumatic aortic transection (TAT) is a rare but life-threatening consequence of high-energy blunt trauma, most commonly from motor vehicle collisions (MVCs). The injury classically involves the aortic isthmus and often coexists with other thoracoabdominal injuries, leading to diagnostic challenges. Mortality rates are high without timely diagnosis and intervention. Emergency physicians (EPs) are often the first to evaluate these patients, and early suspicion, imaging, and stabilization are critical. This case series presents four male patients with TAT secondary to MVCs, highlighting their ED presentations, imaging findings, and initial management strategies. All patients survived and underwent successful definitive treatment.

Materıals And Methods:

We retrospectively reviewed four male patients, aged 26, 32, and 38 (two patients), with a mean age of 33.5 ± 5.7 years, who presented to a tertiary ED between January 2019 and January 2024. All patients sustained TAT secondary to MVCs and were diagnosed by contrast-enhanced thoracoabdominal computed tomography angiography (CTA). Clinical data, imaging, interventions, and outcomes were evaluated.

Results

All patients were hemodynamically stable at presentation and had a Glasgow Coma Score of 15. One patient was a pedestrian struck by a vehicle; the others were in-vehicle occupants. Aortic injury was located at the isthmus in all cases. Associated injuries included pneumothorax, hemothorax, hepatic and splenic lacerations, renal trauma, and long bone or vertebral fractures. CTA confirmed aortic transection in all patients. Blood pressure control and resuscitation were promptly initiated. All patients were admitted to the intensive care unit and underwent thoracic endovascular aortic repair. The median hospital stay was 13 days (IQR 7–21). No mortality occurred. This case series underscores the importance of maintaining a high index of suspicion for TAT in patients presenting after high-velocity MVCs, even in the absence of shock. CTA should be performed early in the evaluation of suspected cases. Emergency physicians are essential in initiating hemodynamic control, imaging, and surgical coordination, all of which contribute to improved survival in TAT.

Dıscussıon

Traumatic aortic transection, though rare, is a critical injury with high early mortality if undiagnosed. The mechanism typically involves rapid deceleration resulting in shearing forces at the aortic isthmus, the most common site of injury. In our series, all patients were young adult males involved in high-energy MVCs, consistent with existing literature. While some patients present with hemodynamic instability, others—like Cases 2 and 3—may be deceptively stable, underscoring the importance of maintaining a high index of suspicion in blunt chest trauma. A widened mediastinum on chest X-ray may offer a clue but is neither sensitive nor specific. CTA remains the gold standard for diagnosis and should be promptly performed in high-risk mechanisms or suspicious clinical presentations. Initial ED management focuses on permissive hypotension, pain control, and rapid surgical consultation. Early TEVAR, now preferred over open repair, offers excellent outcomes with lower morbidity and mortality.

Conclusıon

This case series emphasizes the vital role of emergency physicians in recognizing and initiating management of traumatic aortic transection. Despite variable presentations, all patients benefited from early CTA and timely vascular intervention. High clinical suspicion, especially in the context of high-energy blunt trauma, is essential for improving survival in TAT. As access to endovascular techniques increases, coordinated emergency care can lead to excellent outcomes in these life-threatening injuries.

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3714

Features Of Thrombosıs And Restenosıs Of Stents In Patıents After Coronavırus Infectıon: A Systematıc Revıew Of Clınıcal Cases.

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Introduction

One of the most serious complications of coronary stenting is restenosis and in-stent thrombosis, the prevalence of which can reach 20-25%. This is a major problem for health systems worldwide as it leads to increased mortality from myocardial infarction. One of the important factors influencing the formation of restenosis and thrombosis in patients with coronary heart disease (CHD) is the presence of systemic inflammation, including infectious inflammation, which can damage the endothelium, plaques and trigger hypercoagulation mechanisms , creating a prothrombotic effect.

Purpose of the work:

To study the impact of COVID -19 on the formation of thrombosis and restenosis coronary artery stents in patients with coronary artery disease

Modern concepts of stent thrombosis

In- stent restenosis is a decrease in vessel lumen after percutaneous coronary intervention, angiographically defined as a decrease in stent patency by 50% or more [KuntzR 1993; MehranR 1999; AlfonsoF 2014]. According to the classification of the Academic Research Consortium (2006), stent thrombosis is divided into acute (up to 24 hours after stenting), subacute (more than 24 hours to 30 days), late (more than 30 days to 1 year) and very late (more than 1 year after stenting) [Food and 2006, C utlip D]. According to the clinical course, the following types are distinguished:

1) definite (angiographically confirmed) stent thrombosis , when symptoms are consistent with acute coronary syndrome;

2) probable stent thrombosis , when sudden cardiac death or myocardial infarction is observed in the stented vessel basin within 30 days after stenting without angiographic confirmation;

3) possible stent thrombosis , when unexplained death occurs a month or more after stent implantation [Food and 2006, C utlip D].

Risk factors for coronary stent thrombosis

related to the patient himself: for early thrombosis, these are diabetes, previous ST- elevation MI , smoking, thrombocytosis, and anemia (OR <5 for all of the listed risk factors); for late and very late thrombosis, the listed risk factors can be supplemented by end-stage chronic kidney disease, low ejection fraction, African-American descent, and advanced age (OR <5 for all of the listed risk factors); OR =5-10 for malignant neoplasms, peripheral arterial disease; OR > 10 for violations of dual antiplatelet therapy and genetic polymorphism;

Coronary vessel injury related to: location in the left anterior descending coronary artery, small vessels, ulcerated or aneurysmal lesion, prolonged lesion or multiple stent implantations , lesion at the bifurcation of the artery, baseline TIMI flow grade 0–1, CABG with saphenous vein graft (OR < 5 for all listed risk factors); presence of in- stent restenosis with drug-eluting stent, geographic miss, bifurcation or type C lesion (OR = 5–10 for all listed risk factors); post-procedural TIMI flow < 3 (OR > 10).

stent -related : strut thickness and stent base area , incomplete endothelialization , inflammatory reactions (OR<5 for all listed risk factors); small diameter stent (OR 5-10).

Related to the stenting procedure : no administration of thienopyridines (clopidogrel); no use of heparin before the procedure; injury to multiple vessels; use of multiple stents ; use of glycoprotein IIb / IIIa inhibitors; overlapping technique (implantation of stents in an overlapping manner); residual stenosis when using drug-eluting stents (OR<5 for all listed risk factors); use of low doses of bivalirudin (OR 5-10); insufficient stent size or its incorrect positioning; vessel dissection ; residual stenosis or incomplete expansion of the vessel or stent (OR>10) [Gori T 2019].

The role of coronavirus infection in the development of thrombosis and restenosis stents

Studies by Chinese scientists conducted at the beginning of the COVID-19 pandemic demonstrated high rates of acute inflammatory myocardial injury, reaching 17-19.7% of all patients with COVID-19.

It should be taken into account that acute myocardial injury in COVID-19 is not always accompanied by ischemic manifestations, but in all cases an increase in the level of cardiac markers, such as troponin , was noted, the level of which had a direct correlation with the severity of coronavirus infection [Shi S 2020, Zhou F 2020, Huang C 2020].

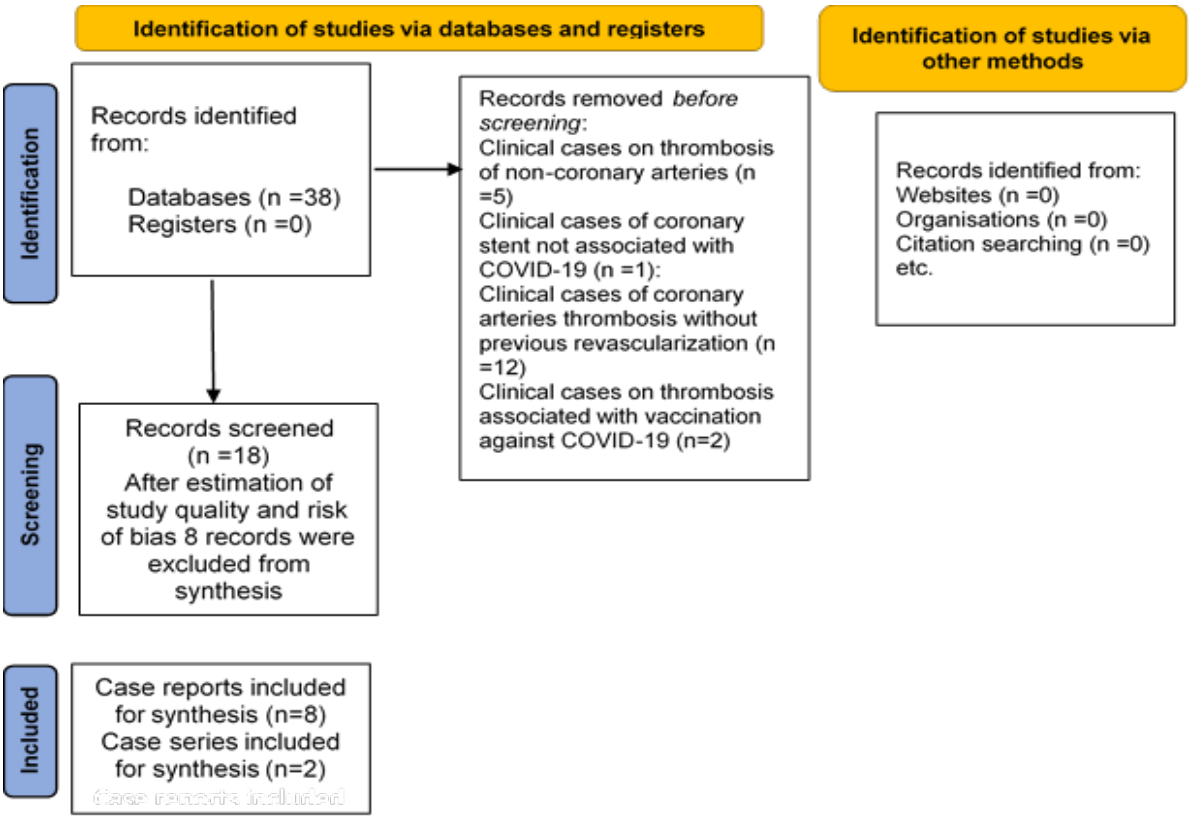
Thus, in a study by Wang D (2020), it was shown that among people with COVID -19 treated in intensive care units, the proportion of patients with elevated troponin levels reached 25% [Wang D].

Materials and methods of research:

PubMed and Scopus databases for relevant publications containing case reports and case series of restenosis. stent and stent thrombosis associated with COVID-19, including case reports published between 2020 and present. Thirty-eight full-text publications were screened and manually checked for further analysis.

The protocol was developed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analysis Protocols (PRISMA-P) [72 Page , M. 2021] and registered in PROSPERO (ID: CRD42024506976).

The study was carried out with the financial support of the Science Committee of the Ministry of Education and Science of the Republic of Kazakhstan (grant No. AP 19677465).



Characteristics of included cases

Reference	Age and sex	Data about COVID-19	Chest X-Ray	SpO2	Previous revasularisation	Current revascularisation	Coronary angiography	EhoCG (EF)	ECG	Risk factors	Outcome
Hinterseer M, 2020[56]	65-year-old male	Positive RT-PCR	Bilateral pulmonary infiltrates	78%	Stenting of marginal artery and LAD four years ago; in-stent thrombosis 2 years ago	Thrombus aspiration, stenting	Thrombus in in stent thrombosis in LAD	EF of 35%	ST-segment elevation in aVR, complete RBBB	DM, HTN, hyperlipi-demia	Died
Kunal S, 2021[57]	40-year-old male	Positive RT-PCR	Bilateral pulmonary consolidations	95%	Stenting in LAD two years ago due to STEMI	Balloon angioplasty and thrombus aspiration	Non-occlusive thrombus in the LAD stent	EF of 35%	ST- segment elevation in V1-V6	N/A	Survived
Zaher N, 2020[59]	51-year-old man	Positive RT-PCR	Pulmonary vascular congestion	91%	Previous myocardial infarction with DES placed in the LAD.	LCX angioplasty with overlapping DES. Acute rethrombosis minutes later. Balloon angioplasty	100% occlusion of LCX with diffuse disease in the LAD	Dilated left ventricle with low EF	ST elevations in III, aVF, V5, and V6	CAD, HTN, DM	Died
Hauguel-Moreau M, 2022[62]	65-year-old man	Positive RT-PCR	COVID-19 interstitial pneumonia	N/A	10 years before 2 DES were implanted PDA due to STEMI. 2 years ago a DES was implanted in mid-LAD.	Two DES implanted in mid-LAD and PDA	Acute dual thrombotic occlusion in the LAD and PAD DES	EF of 25%	Anterior and inferior ST-segment elevation with Q waves	Psoriatic arthritis, HNT, CAD	Survived
Liebenberg J, 2022[64]	71-year-old man	Positive RT-PCR	No findings	88%	Forty-six hours before two DES stents were placed to LCX and RCA	Balloon inflations and thrombus aspiration. Intracoronary injection of metalyse	Subacute stent thrombosis of both the LCx and RCA stents	EF of 40–45%	Posterior ST-elevation MI	HNT	Died
Elkholy, K, 2021[65]	48-year-old male	Positive RT-PCR	Bilateral interstitial lung infiltrates	N/A	3 days before thrombectomy and placement of a DES was performed to LAD	Angioplasty and thrombectomy of the lesion in the mid-RCA	100% occlusion of the stent, and novel 100% occlusion in the RCA	EF of 40–50%	ST elevation in I, III, aVL, aVF, and V2-V5; AV block	DM, HNT, hyperlipi-demia, and smoking	Died
Ayan M, 2020[66]	64-year-old black man	Positive RT-PCR	Bilateral lung infiltrates, ARDS	83%	100% thrombotic occlusion of the second obtuse marginal artery 3 days ago, PCI with DES	Balloon angioplasty and new Resolute Onyx stent	Complete thrombotic occlusion of the second obtuse marginal artery	EF of 45–50%	ST segment depressions in lateral leads.	Chronic hepatitis C, HNT, and tuberculosis	Survived
Sidhu N, 2022[67]	56-year-old man	Positive RT-PCR	No findings	Norm	Implantation of 2 everolimus-eluting stents to LAD 8 days ago.	DAPT and dabigatran 110 mg after five days of enoxaparin	Dual stent thrombosis of LAD (30% and 75%)	EF of 45%.	Lateral wall ST elevation MI	Dislipide-mia	Survived

Abbreviations: CAD, coronary arteries disease; CKD, chronic kidney disease; DES, drug-eluting stent; DM, diabetes mellitus; EF, ejection fraction; HTN, hypertension; LAD, left anterior descending artery; LCX, left circumflex artery; LVD, left ventricle dysfunction; MI, myocardial infarction; N/A, not available; PAD, peripheral artery disease; PDA, posterior descending artery; RCA, right coronary artery; RT-PCR, reverse transcription polymerase chain reaction.

Characteristics of included case series

Reference	Age and Sex	Data about COVID-19	Chest X-Ray	SpO2	Previous Revasularisation	Current Revascularisation	Coronary Angiography	EhoCG (EF)	ECG	Risk Factors	Outcome
Prieto-Lobato A, 2020[71]	49-year-old man	IgG was positive for COVID-19	Bilateral lung infiltrates	90%	Balloon angioplasty and stenting of LCX with 2 overlapped stents 30 min ago	Intracoronary tirofiban and proximal overexpansion of stent	Acute LCX stent thrombosis	EF of 45%	Lateral ST-elevation MI	DM, LVD	Survived
	71-year-old man	Positive RT-PCR	Bilateral lung infiltrates	96%	Inferior STEMI seven years ago treated with RCA DES	Thrombectomy, tirofiban, and 2 DES restored flow	Very late RCA stent thrombosis	EF of 55%	ST- elevation in the precordial leads	CKD	Survived
	85-year old man	Positive IgM serological testing	No findings	95%	PCI with LAD artery DES implantation 15 years ago	Balloon angioplasty, thrombectomy, and tirofiban	Very late LAD artery stent thrombosis, and neoatherosclerosis	EF of 30%	Anterior ST-elevation with Q waves	LVD, age	Survived
	86-year-old man	Positive RT-PCR	No findings	95%	LAD artery DES due to MI two years ago	A new DES was implanted to LAD	LAD stent thrombosis	EF of 45%	Anterior ST-elevation	DM, CKD, PAD, age	Survived
Batenova G, 2023[33]	65-year-old man	Positive RT-PCR	Bilateral pneumonia	97%	DES stenting RCA five months ago	Balloon angioplasty and DES implantation	RCAin- stent restenosis	EF of 25%	ST-depression in precordial leads	HNT, DM	Survived
	69-year-old man	Positive RT-PCR	No findings	95%	DES stenting to RCA 7 years ago	Balloon angioplasty and DES implantation	RCA in-stent restenosis	EF of 35%	ST-depression in precordial leads	HNT	Survived
	66-year-old woman	Positive RT-PCR	No findings	87%	DES stenting to LAD 8 years ago	Balloon angioplasty and DES implantation	LAD in-stent thrombosis	EF of 55%	ST- elevation in V5-V6	HNT, DM	Survived
	80-year-old man	Positive RT-PCR	Bilateral pneumonia	78%	DES stenting to LAD 4 years ago	Balloon angioplasty and DES implantation	LAD in-stent thrombosis	EF of 26%	ST- elevation in aVL,V3-V6	HNT, stroke, DM, AF	Died
	66-year-old man	Positive RT-PCR	No findings	92%	DES stenting to LCX 8 years ago	Balloon angioplasty and DES implantation	LCX in-stent restenosis	EF of 34%	ST- elevation and Q V1-V3	HNT, DM, CKD	Survived
	59-year-old man	Positive RT-PCR	No findings	95%	DES stenting to LCX 8 years ago	Balloon angioplasty and DES implantation	LAD in-stent thrombosis	EF of 41%	ST-elevation V1-V6	HNT	Survived
	71 -year-old woman	Positive RT-PCR	Bilateral pneumonia	95%	DES stenting to LAD 11 months ago	Balloon angioplasty and DES implantation	LAD in-stent restenosis	EF of 44%	A new LBBB	HNT, stroke, CKD	Survived
	46-year-old man	Positive RT-PCR	Bilateral pneumonia	97%	DES stenting to LAD 5 years ago	Balloon angioplasty and DES implantation	LAD in-stent restenosis	EF of 52%	ST- depression in V5-V6	HNT	Survived
	70 -year-old woman	Positive RT-PCR	No findings	98%	Stent implantation to LCX 14 years ago	Balloon angioplasty and DES implantation	LCX in-stent restenosis	EF of 56%	Lateral ST-elevation	HNT, stroke, hypothyreosis	Survived
	65-year-old man	Positive RT-PCR	No findings	97%	CABG in LAD and RCA 13 years ago	DES implantation to LAD	Thrombosis of LAD and RCA	EF of 48%	Lateral ST-elevation	HNT, DM	Survived

Abbreviations: CKD, chronic kidney disease; DES, drug-eluting stent; DM, diabetes mellitus; EF, ejection fraction; HTN, hypertension; LAD, left anterior descending artery; LCX, left circumflex artery; LVD, left ventricle dysfunction; MI, myocardial infarction; N/A, not available; RCA, right coronary artery; RT-PCR, reverse transcription polymerase chain reaction.

Conclusion

Our analysis of literary sources indicates an increased incidence of restenosis and stent thrombosis in patients with coronary heart disease against the background of coronavirus infection.

Considering that this infection continues to infect residents of all countries of the world, special attention should be paid to people suffering from ischemic heart disease who have undergone revascularization . Since this category of patients has an increased risk of complications, in case of COVID -19 infection they should be provided with access to the fastest possible examination of the cardiovascular system (ECG, echocardiography , laboratory tests), coronary angiography and confirmation of the presence of coronavirus infection (PCR test, study of immunoglobulins G and M).

It is necessary to monitor the adherence of such patients to antiplatelet therapy and inform them about the risks associated with CVI. To more accurately determine the relationship between CVI and stent restenosis and thrombosis, it is necessary to continue clinical studies on larger groups of patients using thematic registries and databases.

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An Analysis of Consultations Requested from the Green Zone in the Emergency Department

Consultations in the Green Zone

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Introductıon

Emergency departments (EDs) are critical healthcare units that operate 24/7, providing immediate care to patients with urgent health needs. These units frequently deal with life-threatening conditions such as myocardial infarction, respiratory arrest, multiple trauma, severe bleeding, burns, and traffic accidents. Any disruption at any stage of this system can adversely affect the efficiency of the entire service chain. In recent years, the role of emergency departments has gained increasing importance in many countries due to economic factors, clinical necessities, and transformations in healthcare systems. Accordingly, various strategies have been adopted to develop cost-effective health policies aimed at ensuring the efficient use of emergency medical services (1,2).

The functioning of EDs in hospitals is regulated according to specific guidelines and instructions. In this framework, each patient admitted to the emergency department is assigned a triage code by the physician responsible for the triage process, based on standardized triage scales. These codes are color-coded as green, yellow, and red—ranging from the lowest to the highest level of urgency. Patients categorized under the green triage code are generally in stable condition, present as outpatients, and have mild complaints that do not require immediate intervention. Such patients can typically be evaluated and treated in an ambulatory setting (3).

In Turkey, emergency departments serve a wide patient population and constitute a critical component of the healthcare system. According to 2020 data, the total population of the Republic of Turkey was reported as 84,680,273, while the number of outpatient clinic visits was 136,905,290. Emergency department visits totaled 129,588,470, and consultations in the green zone of emergency services accounted for 22,092,239 of these. These figures emphasize the central role of emergency departments in healthcare delivery in Turkey and the substantial patient load they bear. Notably, EDs are not only utilized for life-threatening emergencies but are also frequently preferred by a significant number of patients requiring outpatient care (4).

Healthcare services are inherently complex, functionally interdependent, and require multidisciplinary collaboration. Rising global healthcare costs have further highlighted the importance of efficient service delivery, compelling healthcare providers to focus on quality management, operational efficiency, and economic sustainability. One of the most prominent reflections of the need for multidisciplinary work in EDs is the consultation process. A consultation is initiated when the physician primarily responsible for a patient's care requests input from a specialist in another field, based on the patient's clinical condition. Once the consultation is requested, the process formally begins. The consultant physician evaluates the patient, provides recommendations within their area of expertise, may request additional tests, perform procedures, or suggest discharge plans. The efficiency of this process depends on various factors such as the type of hospital, the volume of ED visits, inpatient capacity, and staff shortages, all of which may directly affect consultation management (1,3,5).

This study aims to examine consultations conducted in the green zone of the emergency department, with the goal of evaluating this frequently utilized area of EDs from the perspective of consultation practices. By analyzing consultation requests for green zone patients, we aim to shed light on current consultation patterns and practices within emergency departments.

Methods

This retrospective, observational study was conducted to examine consultation requests among adult patients who presented to the green zone of the emergency department of a tertiary care hospital during the first three weeks of January 2024. The analysis included cases in which a consultation was requested, resulting in a total of 922 patients being included in the study. The variables collected included: Demographic data (age, gender), Time of presentation (categorized as working hours: 08:00–15:59, after-hours: 16:00–23:59, and nighttime: 00:00–07:59), Diagnosis code, Consultation department requested, Time (in hours) from presentation to consultation request, Whether a repeat consultation was requested, and Final patient disposition.

The data were analyzed using the Jamovi Statistical Software and the Python programming language. Descriptive statistics were presented based on the distribution of variables: for continuous variables, mean ± standard deviation (SD) or median with interquartile range (IQR: 25th–75th percentile) were reported; for categorical variables, frequencies (n) and percentages (%) were calculated. The normality of distribution for continuous variables was assessed using the Kolmogorov-Smirnov test. For normally distributed variables, the **Student's t-test** was used, whereas the Mann-Whitney U test was applied for non-normally distributed variables. Differences in consultation requests between weekdays and weekends were also analyzed using the Mann-Whitney U test. The age variable was compared by gender using the Mann-Whitney U test. A p-value of <0.05 was considered statistically significant. All personal identifiers were removed, and only anonymized data were used to ensure the protection of patient confidentiality.

Results

During the first three weeks of January, a total of **38,337** patients were admitted to the green zone of the emergency department. Among these, 2.4% (n = 922) required consultations, resulting in 1,079 total consultation requests. Of the patients for whom consultations were requested, 65.1% were male (n = 600) and 34.9% were female (n = 322).

When analyzing the age distribution, the overall median age was 41.0 years (Interquartile Range [IQR]: 25.0–59.75). The median age of male patients was 38.5 years (IQR: 24.0–55.0), while the median age of female patients was 46.0 years (IQR: 27.25–68.0). The median age of female patients was significantly higher than that of males, and this difference was statistically significant (Mann-Whitney U test, $p < 0.05$, 95% CI: 4.0–13.5).

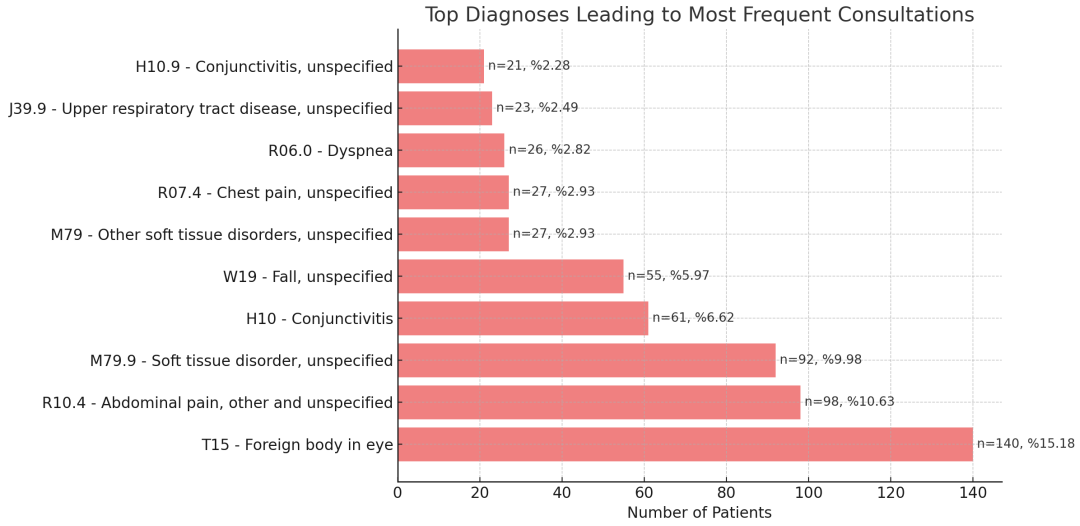
Daily consultation request volumes showed a mean of 51.38 ± 9.69 consultations per day. Regarding the distribution of consultation requests by time of day: 46.42% were made during after-hours (16:00–23:59), 45.12% during working hours (08:00–15:59), and 8.46% during night hours (00:00–07:59).

The mean number of consultation requests per patient was also compared between weekdays and weekends. The average consultation request per **patient** was 1.17 on weekdays and 1.16 on weekends. **The** Mann-Whitney U test revealed **no** statistically significant difference between these two groups (p = 0.2442, 95% CI: -0.07 to 0.09).

The number of consultation requests per patient ranged from 1 to 5. 87.85% (n = 810) of patients received a single consultation request, 8.79% **received** two, 2.06% **received** three, 1.08% **received** four, and only 2 patients (0.22%) **received** five consultation requests.

An analysis was performed on the distribution of patients by diagnostic codes associated with the consultation requests. The most common diagnosis was foreign body in the eye (T15), accounting for 15.18% (n = 140) of consultation requests. This was followed by abdominal pain (R10.4) at 10.63% (n = 98), and unspecified soft tissue disorders (M79.9) at 9.98% (n = 92). Conjunctivitis (H10) accounted for 6.62% (n = 61), and unspecified fall (W19) accounted for **5**.97% (n = 55) of consultation requests. The most common diagnoses leading to consultations are presented in Figure 1.

Figure 1. Most Common Diagnoses Leading to Consultation Requests.



The study also examined the rate of repeat consultation requests (re-consultations) according to the final clinical outcomes of the patients. Among patients who were admitted to the hospital ward, 14.35% (n = 32) required a re-consultation. This rate was **20.93%** (n = 9) among those admitted to the intensive care unit (ICU). In contrast, the re-consultation rate was 4.21% (n = 26) among patients who were discharged and 3.45% (n = 1) among those who refused treatment. The highest re-consultation rate was observed among referred patients, with 50.00% (n = 1)**.** Overall, the re-consultation rate in the emergency department was 7.48%**,** and it was most commonly observed in patients who required hospitalization in the ward or ICU.

Figure 2. Distribution of Patient Outcomes by Mean Number of Consultations.

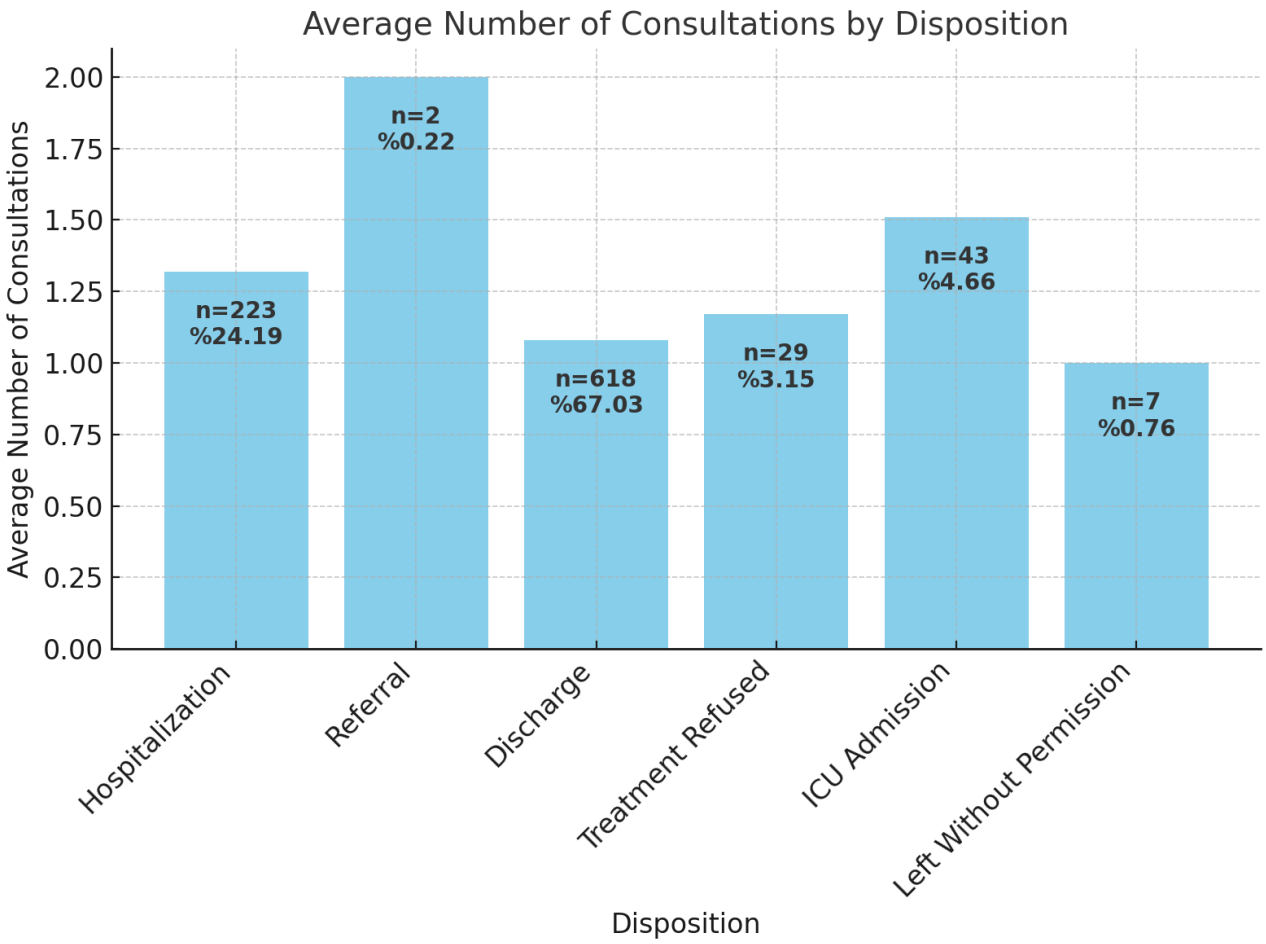
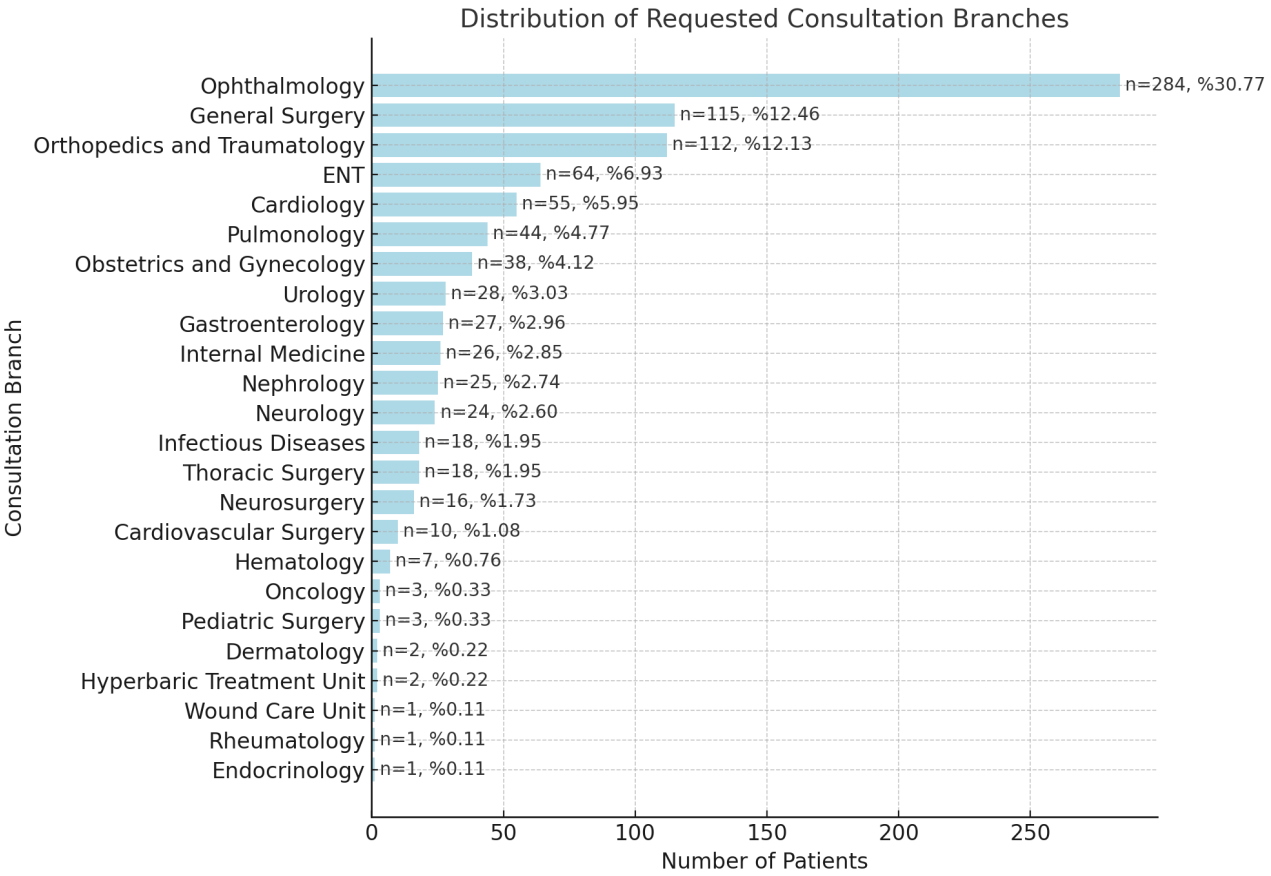


Figure 3. Distribution of Initial Consultation Specialties.



The study also calculated the average number of consultation requests based on patient outcomes. For discharged patients, the mean number of consultations was 1.09; for ward-admitted patients, it was 1.33; and for those requiring ICU admission, it was 1.51. Patients who refused treatment had an average of 1.17 consultations, while those who were referred had the highest mean with 2.00 consultations.

When analyzing the distribution of final outcomes, discharge was the most frequent outcome (67.03%, n = 618), followed by ward admission (24.19%, n = 223) and ICU admission (4.66%, n = 43). Less frequent outcomes included treatment refusal (3.15%, n = 29) and referral to another center (0.22%, n = 2). The distribution of patient outcomes according to the average number of consultations is presented in Figure 2.

The distribution of the initial consultation specialties was also analyzed in the study. It was observed that the majority of consultation requests were directed to the Ophthalmology department (30.77%, n = 284). This was followed by General Surgery (12.46%, n = 115), Orthopedics and Traumatology (12.13%, n = 112), and Otorhinolaryngology (ENT) (6.93%, n = 64).

The distribution of consultation specialties is presented in Figure 3.

The distribution of initial consultation requests by time of day was analyzed based on the top three most frequently consulted specialties. For Ophthalmology, 52.46% of consultations were requested during working hours, 45.07% during after-hours, and 2.46% during the night shift. In General Surgery, 40.00% of consultations occurred during working hours, 45.22% during after-hours, and 14.78% during the night shift. For Orthopedics and Traumatology, 39.29% of consultations were requested during working hours, 58.04% during after-hours, and 2.68% during night hours.

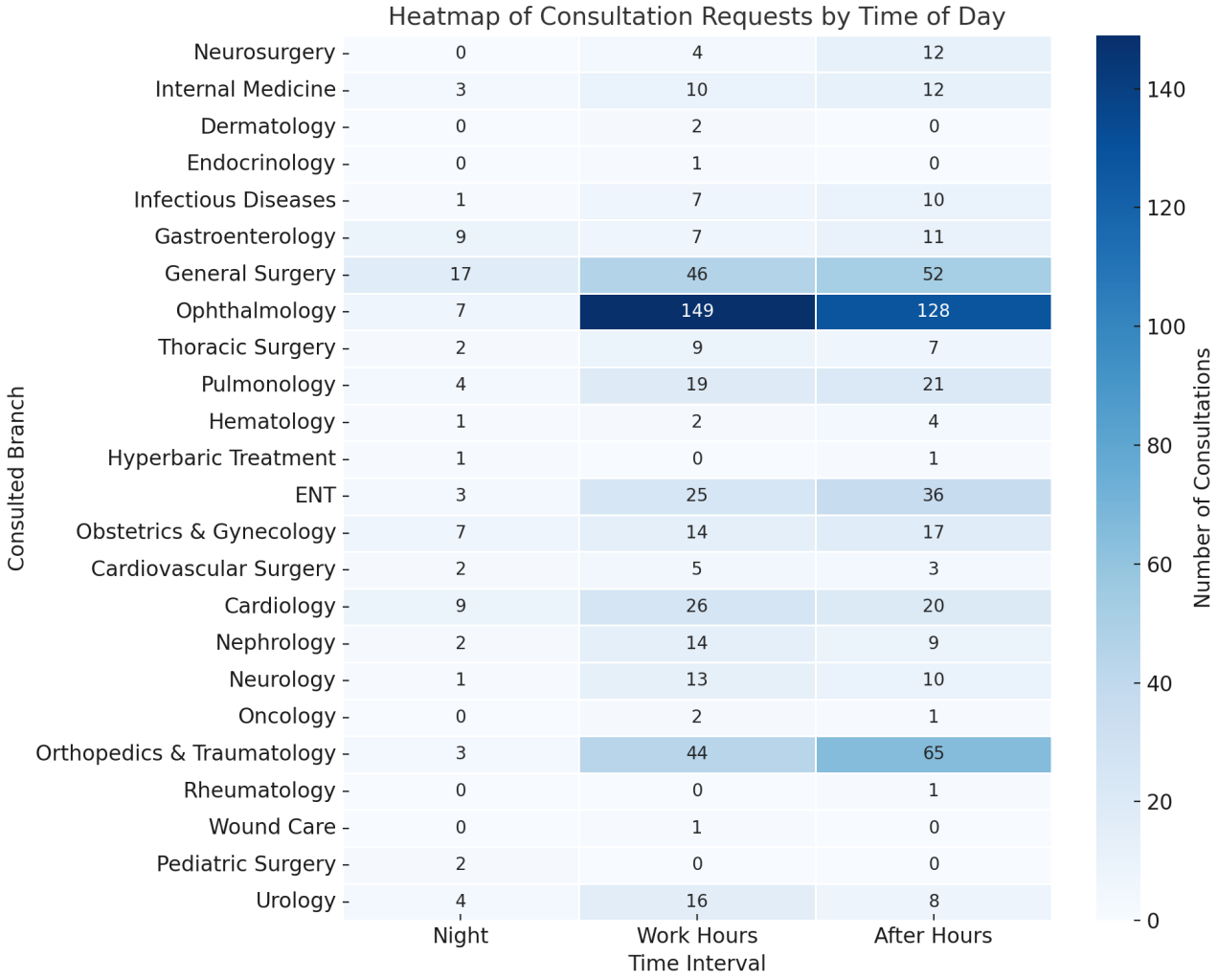
The least frequently consulted specialties were identified as the Wound Care Unit, Rheumatology, and Endocrinology. All consultations for the Wound Care Unit (100%) were requested during working hours, with no consultations recorded during after-hours or night shifts. All Rheumatology consultations (100%) occurred during after-hours, while Endocrinology consultations were exclusively (100%) requested during working hours, with no requests during after-hours or night shifts.

When visualized as a heat map showing consultation frequencies by specialty and time of day, it was observed that Orthopedics and Traumatology had a higher proportion of after-hours consultations, whereas Ophthalmology and General Surgery received more consultation requests during regular working hours. Across all specialties, consultation requests during the night shift remained notably low. The heat map is presented in Figure 4.

The study also examined the mean age and gender distribution of patients based on the specialty to which consultations were requested. Results indicated that while certain specialties had older patient populations, others were more frequently consulted for

younger individuals. The highest mean age was observed in patients referred to Dermatology, with an average of 68.00 ± 25.46 years. This was followed by Infectious Diseases (65.61 ± 17.67 years) and Internal Medicine (53.92 ± 17.58 years), indicating that these specialties were more often consulted for elderly patients. In contrast, the youngest patient group was observed in the Endocrinology consultations, with a mean age of 25.00 years; however, due to limited data, standard deviation could not be calculated. In terms of gender distribution by specialty: Neurosurgery consultations were predominantly female, with 87.50% (n = 14) women and 12.50% (n = 2) men. In Internal Medicine, 32.00% (n = 8) of patients were female and 68.00% (n = 17) were male. Dermatology had an equal gender distribution, with 50% (n = 1) male and 50% (n = 1) female. Among Infectious Diseases consultations, 38.89% (n = 7) were female and 61.11% (n = 11) were male. The sole patient consulted for Endocrinology was male (100%, n = 1).

Figure 4. Heat Map of Consultation Requests by Specialty and Time of Day.



The study also analyzed mean age and gender distribution by clinical outcomes. Certain outcomes were associated with higher mean patient ages. The highest mean age was observed among patients admitted to the intensive care unit (ICU): 63.19 ± 16.70 years. Patients admitted to the general ward had a mean age of 53.02 ± 20.71 years, while those who were discharged were younger, with a mean age of 37.90 ± 20.51 years. Patients who refused treatment had a mean age of 42.69 ± 22.18 years, and referred patients had a mean age of 43.50 ± 28.99 years.

These findings suggest that patients requiring ward or ICU admission tended to be older, whereas those who were discharged were generally younger. Gender distribution by outcome revealed the following: Among ICU-admitted patients, 69.77% (n = 30) were female, and 30.23% (n = 13) were male. In ward admissions, 54.71% (n = 122) were female, and 45.29% (n = 101) were male. Among discharged patients, the female proportion was 69.42% (n = 429), and males constituted 30.58% (n = 189). Among those who refused treatment, the distribution was more balanced: 44.83% (n = 13) female and 55.17% (n = 16) male. All patients referred to other centers were female (100%, n = 2). These results indicate that female patients constituted a higher proportion among those admitted to the ward or ICU, and also among those discharged.

In terms of outcomes by consultation specialty: Among patients consulted by Internal Medicine, 72.00% were admitted to the ward, 20.00% were discharged, and 8.00% were admitted to the ICU. In Infectious Diseases, 38.89% were admitted to the ward, 44.44%

were discharged, and 16.67% required ICU care. Of those consulted by Neurosurgery, 93.75% were discharged, and 6.25% were admitted to the ward. Certain specialties showed dominant outcome patterns: All patients consulted by Dermatology were discharged (100%), while the sole Endocrinology patient refused treatment (100%). No ICU or ward admissions were observed in Rheumatology, Endocrinology, or Wound Care Unit consultations. In contrast, higher rates of ward and ICU admissions were recorded in Internal Medicine and Infectious Diseases consultations. When the outcome distributions by specialty were visualized using a stacked bar chart, it was observed that specialties such as Internal Medicine and Infectious Diseases had higher hospitalization and ICU admission rates, whereas Dermatology and Neurosurgery were associated more frequently with discharges.

The study also examined discharge rates by initial consultation specialty. Results indicated that while some specialties had high discharge rates, others were more frequently associated with hospital or ICU admissions. Specialties with the highest discharge rates included: Dermatology (100.0%), Neurosurgery (93.75%), and Ophthalmology (81.72%). In these specialties, the vast majority of patients were discharged, with minimal need for hospital ward or ICU admission. In contrast, lower discharge rates were observed in specialties such as: Internal Medicine (20.00%), Infectious Diseases (44.44%), and General Surgery (54.35%).

A substantial proportion of patients in these groups required inpatient or intensive care.

Notably, among patients referred to Internal Medicine, the hospitalization rate was 72.00%, and the ICU admission rate was 8.00%. When discharge rates by specialty were visualized using a horizontal bar chart, it became evident that Dermatology, Neurosurgery, and Ophthalmology were predominantly associated with patient discharge, while Internal Medicine and Infectious Diseases showed higher rates of ward or ICU admissions.

The study also analyzed the distribution of hospital ward admissions according to the initial consultation specialty. Results indicated that consultation requests from certain specialties were more frequently associated with inpatient admissions. The specialties with the highest rates of ward admission were: General Surgery: 28.7% (n = 64), Pulmonology: 9.42% (n = 21), and Internal Medicine: 8.07% (n = 18). Approximately one-third of the patients consulted by General Surgery were admitted to the hospital ward, and Pulmonology and Internal Medicine also showed relatively high admission rates. Conversely, some specialties had very low rates of ward admission, including: Cardiovascular Surgery: 0.9% (n = 2), Pediatric Surgery: 0.45% (n = 1), and Neurosurgery: 0.45% (n = 1).

The study also examined the distribution of intensive care unit (ICU) admissions according to the initial consultation specialty. Some specialties showed a notably higher association with ICU admissions. The specialties with the highest ICU admission rates were: Cardiology: 46.51% (n = 20), Nephrology: 13.95% (n = 6), and Gastroenterology: 9.30% (n = 4). On the other hand, specialties with the lowest ICU admission rates included: Thoracic Surgery: 2.33% (n = 1), Hematology: 2.33% (n = 1), and Cardiovascular Surgery: 2.33% (n = 1).

Additionally, the study investigated the timing of the initial consultation requests in relation to the patient's arrival time in the emergency department. The findings showed that the majority of consultations were requested within the first few hours of presentation. 16.92% of consultation requests were made within the first hour (n = 156), 9.44% in the second hour (n = 87), and 12.26% in the third hour (n = 113).

Consultation requests made 10 and 20 hours after admission were extremely rare, with only 0.11% (n = 1) for each time point. These findings suggest that while consultation requests are predominantly made early in the patient's stay, in rare cases they may also be initiated much later.

Discussion

Patients presenting with non-urgent and mild complaints frequently prefer emergency departments and are generally classified under the green zone following triage evaluation. However, the admission of patients who do not require emergency intervention significantly contributes to overcrowding in emergency departments and hinders the effective delivery of healthcare services. This overcrowding has several adverse consequences: it leads to prolonged waiting times, delays in the management of truly urgent cases—thereby compromising patient safety, increases patient dissatisfaction, elevates healthcare costs, reduces the quality of services, and results in critical safety issues. Additionally, the excessive workload negatively affects staff performance and increases the risk of burnout among emergency healthcare professionals (6–8).

Consultation is a critical component of patient management in the emergency department. In this process, a physician from another specialty is invited to contribute to the diagnosis and treatment of the patient. The consulting physician evaluates the patient, records their recommendations in the medical record, and may initiate interventions relevant to their specialty. This multidisciplinary approach enables more effective patient care and is considered an essential and ongoing aspect of emergency medicine practice. Previous research has shown that the consultation process is one of the major factors contributing to increased patient waiting times. Scientific investigations into emergency department consultations date back to the mid-20th century and have continued to evolve since then (9–16). However, our literature review revealed no studies focusing specifically on consultations within the green zone of emergency departments. This highlights the potential novel contribution of our study to the literature.

A study evaluating emergency department challenges from the perspective of healthcare workers also identified problems in the consultation process. In the literature, the frequency of consultations in emergency departments is reported to vary between 20% and 60%, while studies conducted in Turkey indicate this rate ranges between 19.6% and 39.1% (5, 17, 18). In our study, the consultation request rate in the green zone was found to be 2.4% (n = 922). This lower rate is attributable to the focus on green zone patients, who generally present with less severe conditions.

Additionally, we found that 87.85% of patients received only one consultation, and the re-consultation rate was 7.48%, most com-

monly observed among patients admitted to the hospital ward (14.35%) or ICU (20.93%). These findings suggest that the consultation process may be more complex and prolonged in certain patient groups.

In a study by Kılıçaslan et al., 47.24% of patients admitted to the emergency department were triaged as non-urgent (green zone). Of these patients, 42.9% presented between 08:00–16:59, 42.6% between 16:00–23:59, and 14.5% between 00:00–07:59. The overall consultation rate was reported as 19.66%, with the most frequently consulted specialties being cardiology, orthopedics, and internal medicine (19).

Another study conducted over a two-week period reported 318 consultation requests for 228 patients, indicating that 28.8% of emergency department patients required at least one consultation. Distribution by time intervals showed: 40 consultations between 00:00–05:59, 71 between 06:00–11:59, 107 between 12:00–17:59, and 100 between 18:00–23:59. Among these patients, 55.3% were discharged, 27.9% were admitted, and 9.1% were referred to other facilities (20).

Different studies have reported variation in the most frequently consulted specialties. One study identified orthopedics, general surgery, and neurology as the top specialties, while another found internal medicine, cardiology, and orthopedics to be the most commonly consulted (21, 22). Admission and referral rates among consulted patients also vary. In one study by Aydın et al., 12.2% of patients were admitted, and 4.5% were referred. Another study by Köse et al. reported an admission rate of 1.4% and referral rate of 0.3% (21, 22).

In a study conducted at a foundation university in Ankara, consultation was requested for 2,155 out of 16,383 patients. Among them, 1,774 received one consultation, 234 received two, 84 received three, 28 received four, and 12 received five consultations (23).

Another study reported 342 consultation requests for 276 patients, with internal medicine being the most frequently consulted specialty (72%). Among non-urgent cases, internal medicine (31%), neurology (13.8%), and obstetrics and gynecology (10.3%) were the most frequently consulted specialties. Furthermore, 80.1% of these patients were admitted, while 15.9% were discharged (24).

In our study, the most frequently consulted specialties were ophthalmology (30.77%), general surgery (12.46%), and orthopedics and traumatology (12.13%). Similar to other studies, orthopedics and general surgery consistently rank among the most commonly consulted departments. However, the high frequency of ophthalmology consultations in our study likely reflects the unique profile of green zone patients.

Regarding the time of consultation, 46.42% were requested during after-hours (16:00–23:59), 45.12% during regular hours (08:00–15:59), and 8.46% during night hours (00:00–07:59). The lower consultation rate during the night aligns with previously reported findings.

Analysis of patient outcomes revealed that most cases resulted in discharge (67.03%), followed by ward admission (24.19%) and ICU admission (4.66%). These rates are largely consistent with the literature. However, the relatively high rate of ward admissions in our study suggests that some patients triaged to the green zone may still require significant diagnostic or therapeutic intervention.

Conclusıon

It is well recognized that the consultation process in emergency departments significantly impacts patient flow, time management, healthcare staff workload, and hospital admission and referral processes. However, the lack of sufficient research specifically addressing the consultation dynamics of green zone patients highlights the need for further investigation in this area. Our study contributes a novel perspective to the literature by examining the consultation patterns and outcomes among green zone patients in the emergency department setting.

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A rare anaphylaxıs ın the emergency department: anaphylaxıs ın elderly patıents and sesame

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Introductıon

Sesame (Sesamum indicum) belongs to the Pedaliaceae family and has been used as both food and medicine since ancient times. The earliest records of its use date back to 2450 BC [1]. Sesame allergy is less common in Western countries but more prevalent in the Middle East. Although the exact mechanisms are not well understood, sesame allergy can be life-threatening. Reports of allergic reactions to sesame began appearing in the 1950s and have increased significantly over the past three decades. A recent study found that the prevalence of sesame allergy among young adults (ages 17-18) in Israel was 0.09% [2]. In Lebanon, the prevalence of sesame sensitivity, defined by positive IgE and skin prick tests, was 3.9% in newborns, 2.65% in children, and 1.9% in adults, with anaphylaxis as the only clinical presentation in these cases [3]. Recent studies have also identified sesame allergy and anaphylaxis as growing concerns in Saudi Arabia, Iran, Kuwait, and Turkey [4-7]. Interestingly, sesame allergy has not been reported in the world's top 10 sesame-producing nations, including the largest producers, India and China [8]. Like many food allergies, sesame allergy typically develops in infancy or childhood. However, a recent study indicates that 25% of cases arise in adulthood.

Case

A 75-year-old male patient with no known comorbidities presented to the emergency department. His medical history revealed that he contacted the 112 Ambulance Service after experiencing sudden illness, shortness of breath, erythema, pruritus, and urticaria after consuming cake. Upon initial assessment, his arterial blood pressure was 66/44 mmHg, pulse rate was 116 beats per minute, fingertip oxygen saturation was 94%, and body temperature was 36.1°C. The patient exhibited bronchospasm upon arrival, but no uvular edema was observed. An electrocardiogram showed sinus tachycardia. The patient was diagnosed with anaphylaxis and received 0.5 mg of intramuscular adrenaline. Fluid resuscitation was initiated, followed by intravenous administration of pheniramine and dexamethasone. Despite partial improvement, symptoms persisted, necessitating hospital admission for further monitoring and treatment. The patient was subsequently transferred to the toxicology critical care unit for continued care.

During the patient's intensive care stay, a detailed medical history revealed no prior sesame allergy in childhood or adolescence. His first allergic reaction occurred at age 60 after consuming halva. Later, he experienced similar reactions after eating sesame bagels and tahini sauce, leading him to avoid sesame-containing foods. However, the patient had never undergone outpatient evaluation for sesame allergy. He had a history of three anaphylactic episodes following sesame ingestion, each requiring intramuscular adrenaline. After this latest reaction, the patient's relatives discovered that the cake he consumed contained tahini, confirming the allergen. This case highlights the patient's awareness of his allergy. Despite treatment for facial erythema and hand edema, the patient remained under observation in the intensive care unit for three days. After stabilization and symptom resolution, he was discharged with a prescription and advised to follow up at the allergy and immunology outpatient clinic.

Discussion

Although global data indicate an increasing prevalence and severity of sesame allergy, it remains relatively uncommon in emergency settings. Lack of awareness regarding food ingredients may contribute to underdiagnosis. In this case, the patient's awareness of his allergy facilitated identification. Scientific studies indicate that sesame contains both protein and lipid allergens, yet the precise pathophysiology of sesame allergy remains unclear. Unlike other major food allergens such as peanuts, milk, and eggs, research on sesame allergy is still in its early stages. Anaphylaxis is defined as a rapid-onset, escalating reaction that poses a life-threatening risk. Common triggers include food, medications, and insect bites. Patients presenting with circulatory disturbances, bronchospasm, or laryngeal involvement (e.g., stridor, voice changes, odynophagia) should be observed in a hospital setting for at least 24 hours. Diagnosis primarily relies on clinical criteria. In some cases, laboratory tests (such as elevated histamine and total tryptase levels) may support the diagnosis, but they lack specificity for anaphylaxis. In our case, hypotension and bronchospasm were key indicators of anaphylaxis [4-7].

A review of the literature reveals that sesame-related allergies and anaphylaxis are more prevalent in the Middle East [2-7]. While regulations exist for many common food allergens, some countries lack specific labeling requirements for sesame, leading to unintentional exposure. While food allergies typically manifest in infancy or childhood, our case demonstrates that symptoms can also emerge later in life, with initial signs appearing at age 60 [9]. This case contributes significantly to the literature by highlighting the need for considering food allergies in elderly patients presenting with first-time anaphylaxis.

Conclusion

Patients may present to the emergency department with hypersensitivity symptoms such as urticaria, pruritus, and erythema, or with severe anaphylaxis. Regardless of prior allergy history, patients with anaphylaxis must receive immediate intramuscular adrenaline and be admitted for monitoring.

While food-related allergies and anaphylaxis typically develop in childhood, they can also appear later in life, as demonstrated in our case. Research suggests that our country has an elevated risk of sesame allergy compared to many other nations. Proper food labeling of sesame-containing products is essential. Additionally, food allergies should be considered in elderly patients presenting with anaphylaxis in the emergency department.

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Valvular Regurgitation in Acute Ischemic Stroke: A TOAST Analysis of Cardioembolic Subtypes

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Introduction

Acute ischemic stroke (AIS) remains a leading cause of morbidity and mortality worldwide, accounting for approximately 62% of all cerebrovascular events (1). The etiology of AIS is heterogeneous, necessitating systematic classification to guide diagnosis, treatment, and prognosis. The Trial of ORG 10172 in Acute Stroke Treatment (TOAST) classification system categorizes ischemic strokes into five subtypes: large artery atherosclerosis, cardioembolic, small vessel occlusion, stroke of other determined etiology, and stroke of undetermined etiology (2). Among these, cardioembolic strokes are particularly critical, constituting 20–30% of ischemic strokes, and are often associated with severe disability and recurrence (3). Atrial fibrillation (AF) is the most common cause of cardioembolic stroke, but emerging evidence suggests that structural cardiac pathologies, such as valvular disorders, may also contribute to thromboembolic risk (4).

Mitral regurgitation (MR) and tricuspid regurgitation (TR) are valvular abnormalities characterized by backward blood flow into the left and right atria, respectively. MR affects 2–3% of the general population, with prevalence increasing with age (5), while TR is present in up to 85% of adults, though severe cases are less common (6). While these conditions are traditionally viewed as hemodynamic disorders, their association with ischemic stroke remains understudied. MR and TR may predispose to thrombus formation due to atrial enlargement, stasis, or endothelial dysfunction (7). However, current guidelines do not classify MR or TR as independent risk factors for stroke, and their role in cardioembolic stroke pathogenesis remains debated (8). This gap in knowledge underscores the need for studies evaluating the interplay between valvular regurgitation, AF, and stroke mechanisms.

The present study aims to investigate the prevalence of MR and TR in AIS subtypes, particularly cardioembolic strokes, and their association with AF. By analyzing echocardiographic, electrocardiographic, and clinical data, this work seeks to clarify whether valvular regurgitation independently contributes to thromboembolic risk or acts synergistically with AF. Given the high prevalence of MR and TR in aging populations, understanding their role in stroke etiology could refine risk stratification and therapeutic strategies.

Materıals And Methods

Study Design and Methodology

This thesis study was conducted at Kırşehir Ahi Evran University Training and Research Hospital after obtaining approval from the Non-Interventional Research Ethics Committee of Kırşehir Ahi Evran University Faculty of Medicine (decision number: 2022-02/19, date: January 25, 2022).

The data for this thesis study were obtained by retrospectively analyzing the diagnostic and therapeutic processes during the hospitalization of patients admitted to the Emergency Medicine Clinic of Kırşehir Ahi Evran University Training and Research Hospital between January 1, 2019, and December 31, 2021, who were diagnosed with acute ischemic stroke after excluding intracerebral hemorrhage via computed tomography (CT) and subsequently admitted to the neurology inpatient ward or intensive care unit. This period included the COVID-19 pandemic, which affected the entire world.

A total of 403 acute ischemic stroke patients were enrolled in the study, meeting the predefined inclusion criteria of being aged over 18 years and having a confirmed diagnosis of acute ischemic stroke, while exclusion criteria comprised individuals under 18 years of age, those diagnosed with acute hemorrhagic stroke or transient ischemic attack (TIA), patients with incomplete medical data, those who did not undergo CT imaging, and pregnant individuals.

Data Collection

Due to the retrospective nature of our study, informed consent was not obtained from the included patients. Patients meeting the inclusion criteria were enrolled in the study group. Patient data were accessed through medical records, hospital archives, and the automation system. Sociodemographic characteristics such as age, gender, and length of hospitalization were recorded.

Established ischemic stroke risk factors—hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease, congestive heart failure, alcohol and tobacco use, and history of prior ischemic stroke—were evaluated and recorded. Neurological examinations at emergency department admission and clinical follow-up examinations in the neurology ward or intensive care unit were documented. Electrocardiography (ECG) was performed at admission, and rhythm Holter monitor results recorded during hospitalization in the neurology ward or intensive care unit were analyzed for atrial fibrillation. Echocardiographic findings during hospitalization, including ejection fraction, valvular pathologies, and cardiac pathologies potentially causing cardioembolic stroke, were also evaluated and recorded.

At least one of the following imaging modalities performed at admission was reviewed: computed tomography (CT), computed tomography angiography (CTA), or diffusion-weighted imaging (DWI). The presence of large vessel atherosclerosis and the vascular territory of the infarct area were noted. Stroke subtypes were classified and recorded using the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) and Oxfordshire classifications.

Since our hospital is the only tertiary center in Kırşehir province, nearly all follow-ups of patients were conducted by our neurology clinic. Subsequent clinical outcomes and mortality data were obtained from the hospital automation system and patients' e-Nabız (national digital health platform) records.

Statistical Analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences 23.0 (SPSS Inc; Chicago, IL, USA). Normality assumptions for continuous variables were tested using the Kolmogorov-Smirnov and Shapiro-Wilk tests. The homogeneity of variances was assessed via Levene's test. Descriptive statistics for variables are presented as mean ± standard deviation, median (min-max), and frequencies (n (%)).

Continuous variables were analyzed using ANOVA based on variable type and assumption fulfillment. Post-hoc comparisons of significant differences identified by ANOVA were performed using the Duncan multiple comparison test. Groups without significant differences in Duncan's test were labeled with the same letter. Categorical data were analyzed using the Chi-square test or Fisher-Freeman-Halton exact test, depending on category counts and expected values. A p-value <0.05 was considered statistically significant.

Results

The study group comprised 403 patients. Analysis of demographic characteristics revealed an age range of 21–100 years, with a mean age of 72.35±12.43. Of the patients, 50.4% (n=203) were female and 49.6% (n=200) were male (Table 1).

Table 1: Age and gender characteristics of acute ischemic stroke patients.

Parameter	Patients
Age (years)	72.4 ± 12.4
Gender	
Female	203 (50.4%)
Male	200 (49.6%)

The values are expressed as n (%) or mean ± SD.

All patients had available electrocardiograms. Atrial fibrillation was significantly more prevalent in the cardioembolic stroke group (88.6%, n=109; p=0.000). Echocardiographic findings were evaluated for all patients. Reduced ejection fraction (EF <40%) was most frequently observed in the cardioembolic stroke group (26.3%, n=30; p=0.000). Mitral regurgitation (MR) (19.3%, n=22) and tricuspid regurgitation (TR) (16.7%, n=19) were also significantly more common in the cardioembolic stroke group (p=0.000). Left ventricular hypertrophy (LVH) was significantly higher in the atherothrombotic stroke group (21.1%, n=43; p=0.000) (Table 2).

Table 2. Diagnostic cardiological test analysis of acute ischemic stroke subtypes according to the TOAST classification.

	TOAST Classification					
Parameter	Atherothrombotic n (%)	Cardioembolic n (%)	Lacunar n (%)	Other Etiologies n (%)	Undetermined n (%)	**p-value**
Age (mean)	72.2±12.4a	74.7±13.2a	68.3±12a	51.8±19b	72.8±11a	**0.000**
Female (n, %)	100 (49.0%)	64 (56.1%)	26 (52.0%)	2 (33.3%)	11 (37.9%)	0.378
ECG Findings						
SR	185 (94.9%)	14 (11.4%)	48 (96.0%)	5 (83.3%)	22 (75.9%)	**0.000**
AF	10 (5.1%)	109 (88.6%)	2 (4.0%)	1 (16.7%)	7 (24.1%)	
Ejection Fraction						
≥50%	186 (91.2%)	73 (64.0%)	49 (98.0%)	6 (100.0%)	24 (82.8%)	**0.000**
41–49%	10 (4.9%)	11 (9.6%)	0 (0.0%)	0 (0.0%)	1 (3.4%)	
<40%	8 (3.9%)	30 (26.3%)	1 (2.0%)	0 (0.0%)	4 (13.8%)	
Echocardiography						
MR	10 (4.9%)	22 (19.3%)	0 (0.0%)	0 (0.0%)	3 (10.3%)	**0.000**
TR	8 (3.9%)	19 (16.7%)	1 (2.0%)	1 (16.7%)	2 (6.9%)	
LVH	43 (21.1%)	13 (11.4%)	4 (8.0%)	0 (0.0%)	1 (3.4%)	
Normal	142 (69.6%)	50 (43.9%)	45 (90.0%)	5 (83.3%)	21 (72.4%)	

Values are expressed as n(%) or mean ± SD. TOAST: Trial of ORG 10172 in Acute Stroke Treatment; SR: Sinus Rhythm; AF: Atrial Fibrillation; MR: Mitral Regurgitation; TR: Tricuspid Regurgitation; LVH: Left Ventricular Hypertrophy. *ANOVA; &: Chi-square test; €: Fisher-Freeman-Halton exact test.

Discussion

Our study revealed that MR and TR were significantly more prevalent in the cardioembolic stroke group (19.3% and 16.7%, respectively) compared to other TOAST subtypes. Notably, AF coexisted in 57.1% of MR cases (20/35) and 77.4% of TR cases (24/31), suggesting that valvular regurgitation may exacerbate thromboembolic risk in the presence of arrhythmia. These findings align with prior studies indicating that atrial enlargement secondary to MR or TR promotes blood stasis, a critical factor in thrombogenesis (9). For instance, left atrial dilatation in MR patients increases the risk of left atrial appendage thrombus formation,

even in the absence of AF (10). Similarly, TR-associated right atrial enlargement may contribute to paradoxical embolism through a patent foramen ovale, though this mechanism was not directly assessed in our cohort (11).

The high prevalence of AF in MR and TR patients (88.6% in cardioembolic strokes) highlights a potential bidirectional relationship. AF may worsen valvular regurgitation due to loss of atrial contraction and ventricular rate irregularity, while volume overload from MR/TR could precipitate atrial remodeling and AF (12). This vicious cycle creates a prothrombotic milieu, amplifying stroke risk. Our data support this hypothesis, as 18/20 MR patients and 19/24 TR patients with AF experienced cardioembolic strokes. These observations suggest that MR and TR, when combined with AF, may serve as markers of heightened embolic risk, warranting aggressive anticoagulation in eligible patients.

Contrary to conventional views, our findings challenge the notion that MR and TR are merely bystanders in stroke pathogenesis. Although current guidelines do not recommend anticoagulation for isolated MR or TR (13), the strong AF comorbidity in our cohort implies that valvular pathologies may identify a subgroup of patients who benefit from early rhythm control or thromboprophylaxis. For example, the 2023 European Society of Cardiology guidelines emphasize AF management in valvular heart disease but do not address regurgitation-specific risks (14). Our study underscores the need to integrate valvular assessment into stroke risk scores, particularly for patients with borderline CHA□DS□-VASc scores.

The association between left ventricular hypertrophy (LVH) and atherothrombotic strokes (21.1%, p=0.000) further illustrates the multifactorial nature of stroke etiology. LVH, often secondary to hypertension, correlates with small vessel disease and microthrombosis (15). However, its absence in cardioembolic strokes reinforces the distinct mechanisms underlying TOAST subtypes.

Study Strengths and Limitations

This study has several limitations. First, the retrospective design precludes causal inferences. Second, the single-center cohort may limit generalizability, though our hospital's role as the sole tertiary center in Kırşehir mitigates referral bias. Third, the lack of long-term follow-up data hinders the assessment of stroke recurrence or anticoagulation outcomes. Future prospective studies should address these gaps while exploring interventions tailored to MR/TR-associated strokes.

Conclusion

This study highlights the significant prevalence of mitral and tricuspid regurgitation in cardioembolic strokes, particularly in conjunction with atrial fibrillation. While MR and TR are not traditionally recognized as independent stroke risk factors, their frequent coexistence with AF suggests a synergistic role in thrombus formation. Clinicians should consider echocardiographic screening for valvular pathologies in stroke patients, especially those with cryptogenic or cardioembolic etiologies. These findings advocate for updated risk stratification models that incorporate valvular abnormalities and AF interplay, potentially guiding personalized anticoagulation strategies. Further research is needed to elucidate whether MR and TR independently contribute to embolic risk or merely reflect atrial cardiopathy. Addressing these questions could transform secondary stroke prevention, reducing the global burden of cerebrovascular disease.

Acknowledgement

This presentation is based on research findings from Dr. Ömer Jaradat's dissertation.

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From Rat Poison to Phosphine Gas: A Case of Zinc Phosphide Poisoning and Clinical Management

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Introduction

Zinc phosphide poisoning is a rare but highly lethal toxicological emergency encountered in the emergency department. Zinc phosphide is an inorganic rodenticide that reacts with gastric acid to release phosphine gas, a potent cellular toxin (1). Phosphine gas inhibits mitochondrial oxidative phosphorylation and cellular respiration, leading to acute energy failure and widespread generation of reactive oxygen species (ROS) (1). This mechanism can result in multi-organ damage, and zinc phosphide ingestion is particularly associated with myocardial depression, acute pulmonary edema, severe metabolic acidosis, and acute renal failure (2,3). Given the high risk of these life-threatening complications, early recognition and aggressive supportive treatment are critical to improving patient outcomes (4). We present the case of a 42-year-old male with intentional zinc phosphide (rat poison) ingestion, detailing his clinical course, management, and a discussion of this intoxication in light of the literature.

Case Presentation

A 42-year-old man presented to the emergency department approximately one hour after ingesting a rodenticide containing zinc phosphide in a suicide attempt. He was conscious, alert, and oriented upon arrival. His primary complaints were nausea, vomiting, and dizziness. On examination, his vital signs were within normal limits. Cardiovascular and respiratory examinations were unremarkable, with no signs of hemodynamic instability or respiratory distress. The abdominal examination revealed mild diffuse tenderness without rebound or guarding, and no other abnormalities were noted. He confirmed that the substance ingested was a commercially available rat poison containing zinc phosphide, which he had brought with him to the hospital (Figure 1).

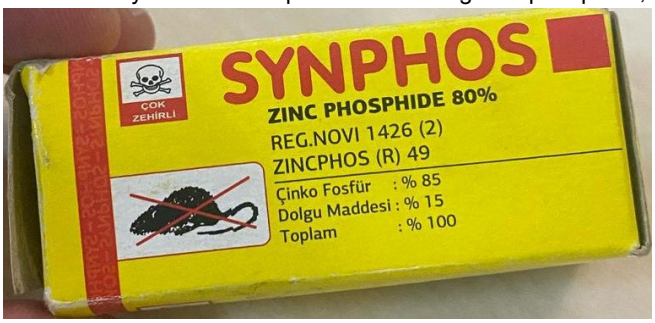

Figure 1: The zinc phosphide-containing rodenticide product ingested by the patient. The packaging (in Turkish) indicates a high concentration (80%) of zinc phosphide (active ingredient, "Çinko Fosfür"), marked with a skull-and-crossbones icon and the warning "Çok zehirli" (very poisonous). Such baits release phosphine gas upon contact with stomach acid, leading to the toxic effects described in the text.

Initial laboratory investigations revealed a mild metabolic acidosis on arterial blood gas analysis (pH 7.30, HCO□□ 20.6 mmol/L). An electrocardiogram (ECG) showed sinus rhythm with non-specific ST-T wave changes, but no acute ischemia or arrhythmias. In the emergency department, the patient was promptly treated with activated charcoal for gastrointestinal decontamination. Intravenous fluids were started to maintain adequate perfusion and to address potential dehydration or shock. Due to the significant risk of delayed systemic toxicity and serious complications, the decision was made to admit the patient to the intensive care unit (ICU) for close monitoring and supportive care.

In the ICU, the patient's hemodynamic status, respiratory function, and renal function were continuously monitored. Serial measurements of electrolytes and acid–base status were performed, and supportive treatments were adjusted as needed. The healthcare team remained vigilant for signs of deterioration such as hypotension, arrhythmias, respiratory failure, or organ dysfunction. Fortunately, the patient's clinical course remained stable. He did not develop any severe complications such as cardiovascular collapse, pulmonary edema, or renal failure during his ICU stay. Over the next 48–72 hours, the patient's symptoms improved with supportive care. By the third hospital day, his condition had markedly improved, and he was transferred

from the ICU to a general medical ward for continued observation. He was eventually discharged in good health after a period of further monitoring, with appropriate psychiatric follow-up arranged for the suicide attempt.

Discussion

Zinc phosphide poisoning is infrequently reported in the literature, but it carries a high mortality risk and is a condition of great clinical importance. Ingested zinc phosphide reacts with water and gastric hydrochloric acid to produce phosphine (PH_3) gas, which is the primary toxic agent responsible for systemic effects (1). Phosphine gas is rapidly absorbed and distributes via the bloodstream, where it causes profound cellular injury. At the mitochondrial level, phosphine inhibits cytochrome c oxidase and disrupts oxidative phosphorylation, leading to a failure of ATP production (1,2). The resulting cellular hypoxia and energy crisis are compounded by the generation of ROS and free radicals, causing lipid peroxidation and oxidative damage in tissues (1). This pathophysiology underlies the multi-organ toxicity observed in phosphide poisonings. Clinically, acute zinc phosphide/phosphine toxicity can affect nearly every major organ system: patients can develop myocardial depression and circulatory collapse, acute lung injury with pulmonary edema, refractory metabolic acidosis, hepatic injury, coagulopathy, and acute kidney injury (2,3). These complications often progress rapidly and have been associated with a high case fatality rate in phosphide poisoning incidents.

Outcomes in zinc phosphide poisoning are closely related to the ingested dose and the promptness of medical intervention. In reported cases, patients may initially present with mild gastrointestinal symptoms (nausea, vomiting, epigastric pain) or even remain asymptomatic in the very early period, only to deteriorate hours later as phosphine gas is released and absorbed (1). Rapid onset of severe manifestations is a hallmark of this poisoning. For instance, cases have documented the development of profound hypoglycemia and severe metabolic acidosis following zinc phosphide ingestion, reflecting the extent of mitochondrial dysfunction and systemic shock (4). Notably, even patients who look stable or only mildly ill on presentation can undergo sudden clinical decompensation within a short time frame (5). Life-threatening arrhythmias, hypotension, and organ failure can emerge within hours after ingestion (5). Therefore, a high index of suspicion and thorough early evaluation are mandatory, even if initial signs are benign. Close observation is warranted for at least 24–48 hours in a monitored setting, given the potential for abrupt deterioration.

Management of zinc phosphide poisoning is largely supportive, as no specific antidote exists for phosphine gas toxicity (2). The cornerstone of treatment is aggressive supportive care and prevention of further phosphine generation/absorption. Early gastrointestinal decontamination is recommended if the patient presents soon after ingestion – typically with gastric lavage and administration of activated charcoal (if not contraindicated) to bind any remaining toxin. In our case, the patient received activated charcoal within about an hour of ingestion, which likely helped limit the amount of phosphine gas produced from the rodenticide in the stomach. Supportive care in an ICU setting is critical due to the risk of multi-organ failure. This includes intravenous fluid resuscitation to manage hypotension, vasopressor support if the patient develops shock, and supplemental oxygen or mechanical ventilation if respiratory failure or severe pulmonary edema occurs. Electrolyte imbalances and metabolic acidosis should be corrected with appropriate fluids, bicarbonate, and other measures as needed. There is no universally accepted antidotal therapy, but various adjunct treatments have been explored in phosphide poisoning cases (such as magnesium sulfate for arrhythmias or high-dose insulin euglycemia therapy for cardiogenic shock in aluminum phosphide poisoning by analogy), although their benefits remain inconclusive (2,6,7). Recent literature has proposed structured management algorithms for phosphide poisoning, which aim to guide clinicians through stepwise supportive interventions and have shown promise in improving survival rates (6). Key aspects of these protocols include vigilant monitoring of cardiac rhythms and hemodynamics, early use of invasive supportive measures when warranted, and proactive management of complications. For example, the development of acute lung injury or pulmonary edema should prompt consideration of early endotracheal intubation and mechanical ventilation before full respiratory decompensation ensues (3). Similarly, any signs of myocardial injury or arrhythmia would warrant early intensive cardiac care. In cases that did progress to significant cardiac toxicity or respiratory failure, studies have found that early aggressive interventions (such as ventilation support, circulatory support, and other ICU therapies) are associated with better outcomes and lower mortality (8).

In the case presented, the patient benefitted from early medical intervention and intensive monitoring, which likely prevented the evolution of severe phosphine-related complications. The ingestion was identified promptly, and gastric decontamination was performed before the toxin could cause irreversible damage. The patient's care in the ICU, with continuous observation and supportive treatment, meant that any emerging issue could be managed immediately. Indeed, our patient did not develop the hypotension, arrhythmias, or organ failures that are often seen in severe cases of zinc phosphide poisoning, and he recovered uneventfully. This favorable outcome is consistent with reports in the literature that emphasize early and aggressive supportive care can significantly improve survival even in serious phosphide poisonings (8). By contrast, delays in treatment or larger ingestions are frequently associated with fulminant toxicity and poor prognosis.

Conclusion

Although zinc phosphide poisoning is uncommon, it poses a significant mortality risk and requires a high level of clinical suspicion and preparedness in the emergency setting. Early diagnosis, prompt gastrointestinal decontamination (such as activated charcoal administration), and close monitoring in an ICU are fundamental measures that can alter the course of the poisoning in the patient's favor (1,2). In this case, the combination of rapid intervention and intensive supportive care averted the development of life-threatening complications and resulted in a successful recovery. This case underscores the importance of clinician awareness

and a proactive, multidisciplinary approach in managing phosphide poisonings. Emergency physicians and critical care providers should be educated about the unique dangers of metal phosphide rodenticides so that appropriate measures can be instituted without delay. Moreover, the development and adherence to standardized clinical management protocols for phosphide poisoning are critical to ensure optimal and timely treatment for these patients (7). By following evidence-based guidelines and maintaining vigilance for sudden deterioration, healthcare teams can substantially improve outcomes in patients who ingest zinc phosphide or similar highly toxic substances.

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Evaluatıon Of Inıtıal And Secondary Trıage Procedures In Patıents Admıtted To The Emergency Department

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Introductıon

In recent years, emergency departments (EDs) in our country have been increasingly utilized not only by patients with life-threatening conditions but also by those whose medical needs do not require urgent intervention. This inappropriate use of ED resources presents significant challenges in patient management and resource allocation. To address this issue, triage systems are implemented by emergency healthcare providers to distinguish between emergent and non-emergent patients and to prioritize care and treatment accordingly.

Triage is a critical component in ensuring the efficient operation of emergency services, allowing timely and effective intervention for those in genuine need. The accurate application of triage significantly affects patient outcomes, including treatment initiation time, survival rates, waiting time, and overall discharge processes. Conversely, incorrect triage decisions not only jeopardize patient safety but also result in suboptimal utilization of limited healthcare resources.

Given these considerations, the precise determination of a patient's triage category at the time of initial assessment is of utmost importance. A well-executed triage process ensures that patients receive care in accordance with the severity of their condition, supporting both clinical effectiveness and the sustainability of emergency care systems.

The aim of this study is to evaluate the effectiveness and appropriateness of the triage system used in the Adult Emergency Department of Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital. Specifically, the study compares the initial triage codes assigned at patient admission with the subsequent triage code modifications during follow-up to assess the necessity of the emergency visit. Additionally, the knowledge and attitudes of emergency department personnel regarding triage practices are analyzed to evaluate the adequacy and effectiveness of the current triage system in place.

Materıal Method

This study is a descriptive and cross-sectional research conducted prospectively. The study covers patients who were admitted to the emergency department during March 2025. The study was carried out with a total of 4854 patients who presented to the Adult Emergency Department of Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital and whose data were recorded in the Hospital Information Management System (HIMS).

The patients' initial triage codes at admission were compared with the modified codes recorded during follow-up to evaluate the necessity of their emergency visits. Data were obtained through the HIMS, and basic statistical methods were used in the analysis.

Results

A total of 4854 patients were included in the study. Of these patients, 55,91% were female and 45,09% were male. The mean age was 40,7 ± 18,2 years.

When the initial triage codes were compared with the codes assigned during follow-up, it was found that 4841 patients were upgraded from green to yellow, 11 patients progressed from yellow to red, and 2 patients were downgraded from yellow to green. These findings suggest that the initial triage classifications often did not reflect the patients' actual clinical conditions, indicating a high need for reassessment and potential improvement in triage accuracy.

Graphic. 1. Distribution of Triage Code Changes

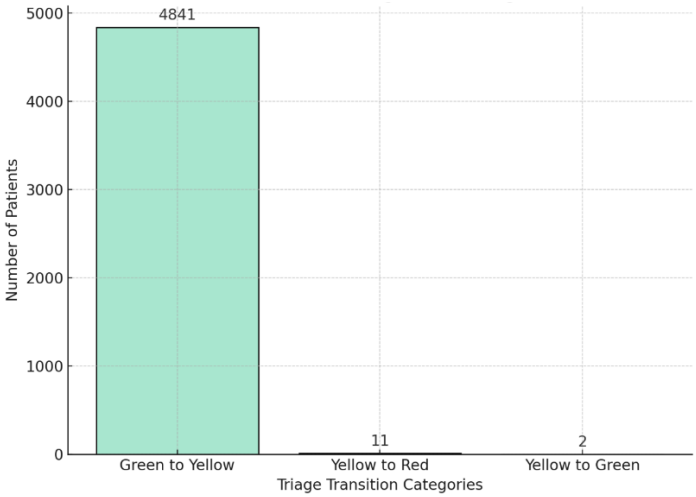

Table 1. Diagnostic Distribution of Patients Whose Triage Level Escalated from Yellow to Red

Fatigue/Malaise with Pain	3
Pain with Nausea/Vomiting	2
Diabetes Mellitus (DM)	2
Pneumonia	1
Myalgia	1
Dyspnea	1
Vertigo	1

Table 2. Diagnostic Distribution of Patients Whose Triage Level Escalated from Yellow to Green

Soft tissue injury	1
Acute Upper Respiratory Tract Infection (AURTI)	1

Table 3. Diagnostic Distribution of Patients Whose Triage Level Escalated from Green to Yellow

Given the total number of 4841 patients in this section, there is a wide variety of diagnoses. Therefore, only those diagnoses observed in more than 20 patients will be presented in the table, while the remaining diagnoses will be grouped under 'Others'.

Acute Upper Respiratory Tract Infection (AURTI)	1243
Soft tissue injury	1055
Myalgia	383
Non-specific pain	281
Abdominal and pelvic pain / abdominal pain	288
Nausea/Vomiting	219
Vertigo	196
Gastroenteritis and colitis	187

Headache	125
Renal colic	90
Anxiety	77
Acute cystitis / cystitis	75
Hypertension	73
Urinary tract disorder / infection	68
Fatigue/Malaise	67
Allergy	62
Fall	55
Assault-related trauma	55
Conjunctivitis	49
Low back pain	45
Constipation	38
Gastritis	28
Cholelithiasis	25
Other(pneumonia, Immunization, Dysmenorrhea, Hyperglycemia, Anemia, Migraine, general condition disorders, etc.)	57

Out of the 11 patients whose triage category was upgraded from yellow to red, 6 (54,54%) were admitted to the observation unit, with a mean observation time of 167.5 minutes. Of the 4.841 patients whose triage category was upgraded from green to yellow, 1.953 (40.34%) were admitted to the observation unit, with a mean observation time of 100.93 minutes. Among the 4,841 patients whose triage category was upgraded from green to yellow, 7 (0,14%) were admitted to different units and departments within the hospital, with a mean observation time of 188.28 minutes. The distribution of these units and departments is presented in Table 4 below.

Table 4. Departmental Distribution of Hospitalized Patients with Triage Upgrade from Green to Yellow

Coronary ICU	2
Obstetrics and Gynecology Department	2
Level III Adult ICU	2
Internal Medicine Department	1

Discussion

The findings of this study highlight a significant inconsistency between initial and subsequent triage categorizations among patients presenting to the emergency department. A considerable number of patients (n=4841) were initially classified as non-urgent (green), but were later upgraded to urgent (yellow) triage categories. This pattern suggests potential limitations in the initial triage evaluation process and reflects the challenges faced by triage personnel under time pressure and high patient loads. Furthermore, in the emergency department where this study was conducted, general practitioners are primarily responsible for the management of patients in the green zone, while emergency medicine specialists oversee the care in the yellow and red zones.

The clinical features observed in the 11 patients whose triage category was upgraded from yellow to red illustrate the complexity of emergency medical care. Symptoms such as generalized pain, dyspnea, and chronic disease complications may initially appear mild but can rapidly progress to life-threatening conditions. This underlines the critical importance of early recognition and accurate triage.

Among these patients, six required admission to the observation unit with an average stay of nearly three hours. This not only reflects the clinical severity of their conditions but also highlights the increased resource burden resulting from delayed identification. Improving the initial triage process is therefore essential to ensure timely interventions and optimize the use of emergency department resources.

Additionally, the admission of a subset of patients (n=7) from the green-to-yellow group to inpatient services such as Coronary ICU and Internal Medicine indicates that even patients initially triaged as non-urgent may require high-level medical care. This is consistent with previous findings that early triage decisions have a direct impact on treatment prioritization, patient flow, and clinical outcomes.

The results underscore the importance of continuous training and evaluation for triage staff, along with system-level improvements in triage protocols. Enhancing triage performance not only supports clinical decision-making but also improves patient safety, optimizes emergency service efficiency, and conserves healthcare resources.

Conclusıon

This study demonstrates a high rate of triage code modification in patients presenting to the emergency department, particularly among those initially assessed as non-urgent. The substantial number of patients who required reassessment and eventual escalation in triage level reveals a gap between perceived and actual clinical severity at the time of presentation.The findings suggest that triage accuracy remains a critical factor in emergency department operations. Inaccurate triage can lead to delays in care, inappropriate resource allocation, and, in severe cases, adverse patient outcomes. Therefore, there is a clear need for enhanced triage protocols, periodic performance evaluations, and targeted education programs for emergency department personnel.

It is necessary to revise and clarify the triage categories recommended by the current Ministry of Health directive on the implementation procedures and principles of emergency services in inpatient healthcare facilities in our country, by establishing more clearly defined standards. In particular, the green and yellow triage categories need to be clearly and distinctly defined.

Improving the initial triage process contributes not only to better patient outcomes but also to the more effective and sustainable use of emergency medical services, particularly in high-volume centers. Investing in triage system quality improvement will have long-term benefits for both healthcare providers and patients.

In summary, strengthening the triage process is a key component in delivering high-quality, safe, and equitable emergency medical care.

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3902

Isolated Pulmonary Laceration: A Case Report

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Introduction: Trauma-related deaths are the third leading cause of death in all age groups, after cardiovascular disease and cancer. The primary causes of trauma are motor vehicle accidents, falls, firearms, sharp and penetrating instruments, and burns. Two-thirds of motor vehicle accidents involve thoracic trauma. Chest trauma is divided into blunt and penetrating chest trauma. Blunt trauma accounts for a significant portion of thoracic trauma. The mortality rate for patients with thoracic injuries and related complications from blunt trauma is 15-25%. Lung lacerations are more common in penetrating chest trauma but can also be seen in blunt thoracic trauma. Computed tomography (CT) is the gold standard imaging modality and is more sensitive than chest radiography for detecting lung, pleural, and bone abnormalities in patients with chest trauma.

Case: An 18-year-old male patient was brought to the emergency department after a motor vehicle accident. The patient's general condition was moderate, conscious, systemic blood pressure was 110/70 mmHg, pulse was 105/min, and respiration was hyperpneic. Physical examination revealed widespread tenderness in the left posterior thoracic wall. No significant feature was detected in his medical history. Routine biochemical tests were within normal limits. Contrast-enhanced thoracic and abdominal CT scans were performed for the patient with multiple trauma. Thoracic CT scan showed hemorrhagic fluid-air levels in all basal segments of the left lower lung and in the paravertebral areas of the right lower lung and multiple laceration areas with widespread ground-glass areas consistent with contusion (Figures 1A-1B,2). No associated rib or vertebral fractures were observed. Abdominal CT scan showed no

traumatic organ damage, free air, or fluid. The patient was consulted to thoracic surgery and admitted to the intensive care unit for follow-up and treatment.

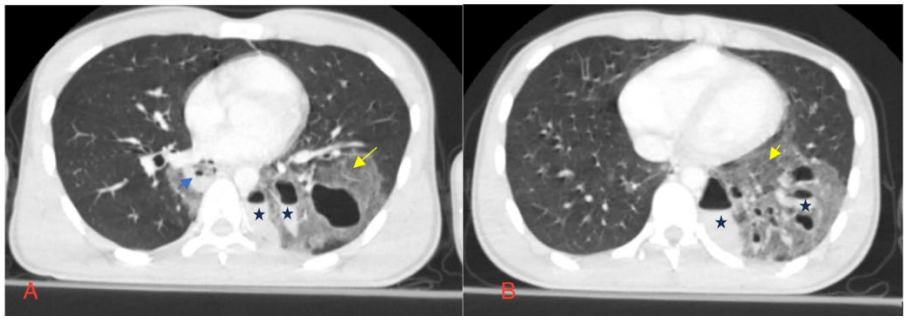

Figure 1A. Thorax CT axial sections; laceration cavity in the lower lobe of the right lung (blue arrow) **Figures 1A-1B.** Laceration cavities causing multiple air-fluid leveling in the lower lobe of the left lung (asterisks), with contusional ground-glass areas around them (yellow arrow).

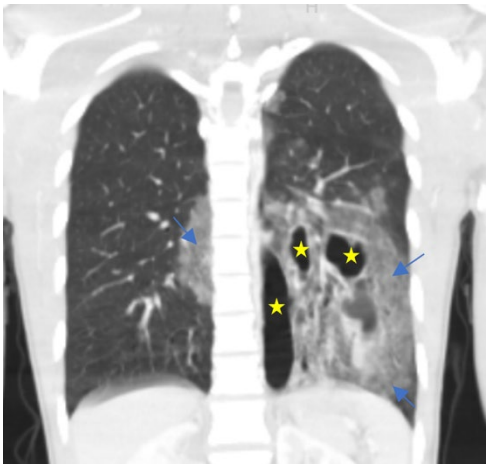

Figure 2: Air densities secondary to bronchial damage in the laceration areas (asterisks) and widespread contusional ground glass appearance in the parenchyma on coronal CT sections (arrows)

Conclusion: Lung lacerations usually occur as a result of severe blunt chest trauma, penetrating injuries, and rib fractures. As a result, disruption of the alveolar walls and rupture of the pulmonary parenchyma occur. Approximately 50% of pulmonary lacerations may not be noticed on the initial chest radiograph because they are often hidden by the consolidation associated with pulmonary contusion. On thoracic CT imaging, an image resembling a cavitary mass with air-fluid leveling in the parenchyma occurs due to the combined damage to vascular structures and bronchial structures in pulmonary lacerations. The patient's trauma history is an important clue in the differential diagnosis. Accompanying parenchymal contusion areas are frequently observed. Rib fractures and pneumothorax accompany most patients. These findings were not observed in our case. Therefore, it should be kept in mind that isolated contusions and lacerations can rarely be seen in thoracic trauma.

Keywords: lung laceration, thorax CT, trauma

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3979

A critical patient, two critical diagnoses

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Introductıon

Thoracic aortic mural thrombus is a rare condition that can lead to systemic arterial thromboembolismb (1). Systemic arterial thromboembolism is a serious and potentially life-threatening event that may cause ischemia due to vascular occlusion in various organs

and systems. Therefore, early diagnosis and treatment are crucial for a favorable prognosis (2).
Gastrointestinal system perforation is another life-threatening condition that results from a full-thickness injury to the digestive tract. Early detection and prompt intervention significantly improve patient outcomes (3).

Case

A 71-year-old female patient presented to an external medical center with abdominal pain. During follow-up, her condition deteriorated, leading to intubation, and she was transferred to our facility for further evaluation and treatment.
Her medical history included diabetes mellitus, hypertension, umbilical hernia surgery, and anemia. On admission, her vital signs were: BP 100/60 mmHg, HR 97 bpm, SpO□ 99%, and blood glucose 287 mg/dL. Physical examination revealed melena on rectal examination, with no other abnormal findings. Laboratory results showed: WBC: 49.6/mm³, HGB: 6 g/dL, blood glucose: 261 mg/dL, creatinine: 1.89 mg/dL, BUN: 34 mg/dL, AST: 18 U/L, ALT: 7 U/L, potassium: 6.5 mmol/L, sodium: 131 mmol/L, pH: 7.22, PCO□: 35 mmHg, HCO□: 14, lactate: 9.6, Troponin: 17.8 ng/L (<14)
CTA revealed a mural thrombus causing 80–90% occlusion in the aortic arch and descending thoracic aorta, along with gastrointestinal perforation (Figure 1). The patient was evaluated by multiple specialties. Cardiovascular surgery recommended close monitoring, while general surgery planned an operation. The patient was admitted to the intensive care unit. Despite surgical intervention, she unfortunately passed away on the third day of hospitalization.

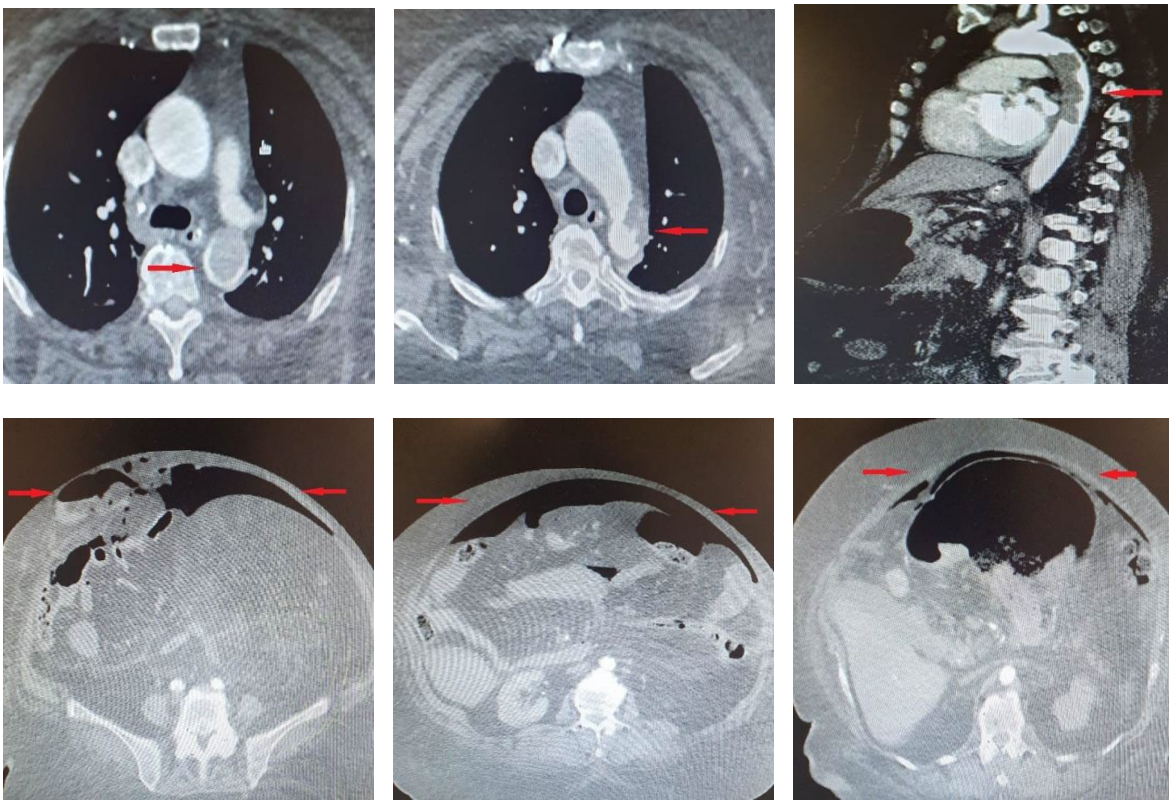

Figure 1: CT Images of the Patient

Dıscussıon

Abdominal pain is one of the most common reasons for emergency department visits, accounting for 5–10% of cases. As seen in our patient, abdominal pain can be an early indicator of life-threatening conditions with high morbidity and mortality.
Thoracic aortic thrombus (TAT) is a rare condition that can cause systemic arterial thromboembolism (1). Systemic arterial thromboembolism can result in severe and potentially fatal complications, including cerebral infarction, myocardial infarction, visceral ischemia, and limb ischemia. Early diagnosis and treatment are essential for improving outcomes. Although TAT may be an incidental finding, peripheral embolic events are common, making early detection challenging (2).
In patients with TAT, the most common initial symptoms include limb pain, ischemic rest pain, skin discoloration, gangrene, and necrosis. Abdominal pain is the second most frequent symptom, while neurological deficits, such as sudden vision loss, dysarthria, aphasia, paraplegia, or paresthesia, are less common (4).
Gastrointestinal (GI) perforation, which can involve the stomach, duodenum, small intestine, or colon, results from full-thickness damage to the GI wall, leading to the leakage of intraluminal contents into the peritoneal or retroperitoneal space. The clinical presentation varies based on location. **Esophageal perforations** present with acute chest pain, odynophagia, and vomiting. **Gastroduodenal perforations** manifest as sudden, severe abdominal pain. C**olonic perforations** often have a more insidious course, potentially leading to secondary bacterial peritonitis or localized abscess formation.
Most cases are associated with high mortality and morbidity, necessitating urgent surgical intervention (5,6).

Conclusıon

Gastrointestinal perforation and TAT are both life-threatening conditions with high mortality rates, yet their coexistence is rare. Patients with these conditions may present to the emergency department with only abdominal pain. This case emphasizes the importance of considering multiple critical diagnoses in critically ill patients presenting with abdominal pain in the emergency department.

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A Rare Case Of Perıneal Laceratıon Followıng Blunt Trauma: Dıagnostıc And Therapeutıc Approach

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Introductıon

Perineal injuries resulting from blunt trauma are a relatively rare condition (1). The perineum is a diamond-shaped area located in the inferior part of the pelvis (2). Since the perineal region contains muscle groups that assist in defecation and micturition, it is of great functional importance. Lacerations in the perineal area are particularly challenging due to the risk of contamination and defecation. Injuries involving the urethra, anus, rectum, genital extensions, and pelvic nerves can result in permanent urinary, gastrointestinal, and sexual dysfunctions (3). In this report, we present a case of a patient who presented with a perineal laceration following blunt trauma.

Case Presentatıon

A 24-year-old male patient was brought to the emergency department after a minibus rollover accident. Upon arrival, his general condition was good and GCS was 15. His vital signs were within normal limits and stable. On physical examination, the abdomen was soft with no guarding or rebound tenderness. There was no thoracic tenderness or spinal tenderness along the vertebral line. There was no pelvic tenderness. Rectal examination revealed bleeding in the rectal area. The patient also had pain and abrasions on various extremities. Upon detailed inspection of the rectal area in the knee-elbow position, broad-bordered abrasions were observed in the perianal region, along with two lacerations approximately 1 cm in size at the 4 and 6 o'clock positions. After initial evaluation and treatment, the general surgery department was consulted and the patient was admitted for observation. During follow-up, serous discharge was noted from the wound site. In the following days, a rectosigmoidoscopy was performed, advancing up to 50 cm proximally, but no pathology was identified in the rectum or the examined segments. On retroflexion, the anal canal appeared normal.

Laboratory Findings : Biochemistry: Glucose: 119 mg/dL, Urea: 25.7 mg/dL, eGFR: 94.12, Creatinine: 1.1 mg/dL, AST: 27 U/L, ALT: 40 U/L, Alkaline Phosphatase: 87 U/L, GGT: 52.2 U/L, Albumin: 43.8 g/L, Amylase: 72 U/L, Calcium: 9.65 mg/dL, Total Bilirubin: 0.41 mg/dL, Direct Bilirubin: 0.08 mg/dL, Indirect Bilirubin: 0.33 mg/dL, CK: 190 U/L, CK-MB: 29.9 U/L, CRP: 6.5 mg/L, Sodium: 138 mmol/L, Potassium: 4.34 mmol/L, Chloride: 102.1 mmol/L, INR: 1.11 Complete Blood Count: WBC: 12.99 K/µL, Neutrophils: 8.29 K/µL, Lymphocytes: 3.85 K/µL, Hemoglobin: 17.6 g/dL, MCV: 88.1 fL, Platelets: 401 K/µL

Dıscussıon

Although perineal injuries, particularly those caused by blunt trauma, are rare, they pose significant challenges in terms of evaluation and management. Anatomically, the perineum contains complex structures responsible for defecation, urination, and sexual function; hence, injuries to this area carry a high risk of functional loss (4,5). As in our case, in lesions presenting with limited perianal skin lacerations and abrasions, thorough evaluation is crucial to rule out deeper injuries to soft tissue, sphincters, urethra, or rectum (6).

Rectal bleeding may indicate deeper tissue damage. Therefore, endoscopic examination is important in such cases to assess sphincter integrity and rule out mucosal injuries. In our patient, rectosigmoidoscopy confirmed the preservation of rectal mucosa and anal canal integrity. This approach is supported by the literature, as excluding internal organ injuries is recommended to reduce

complications (7).

Studies have shown that low-grade perineal injuries can often be successfully managed conservatively without surgical intervention (8). Similarly, in our case, no deep tissue injury requiring surgery was identified, and a conservative follow-up approach was chosen. The serous discharge from the wound was considered part of the superficial healing process and did not necessitate invasive intervention.

One of the major risks in perineal trauma is wound contamination and infection. Therefore, early wound cleansing, appropriate antibiotic prophylaxis, and regular wound care are simple yet essential steps in preventing complications (9). Moreover, it is crucial to rule out accompanying injuries such as pelvic fractures or rectal-bladder disruptions, which may be associated with high-energy trauma (10). In our case, the absence of pelvic tenderness and the lack of significant internal organ injuries on imaging guided our follow-up process.

Conclusıon

In conclusion, perineal injuries following high-energy trauma, such as traffic accidents, may result in significant morbidity. Clinical awareness, early diagnosis, and appropriate treatment can enhance long-term survival and functional recovery. A multidisciplinary approach and careful monitoring are essential for preventing complications and achieving optimal treatment outcomes.

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Diyabetik Ketoasidoza Bağlı Gelişen Akut Solunum Sıkıntısı Sendromu: Olgu Sunumu

Introduction

Diabetic ketoacidosis (DKA) is a potentially life-threatening complication that occurs mostly in patients with type 1 diabetes mellitus (DM) or, less commonly, type 2 DM. Respiratory failure that develops during DKA is associated with increased morbidity and mortality (1). Acute respiratory distress syndrome (ARDS) is a diffuse inflammatory lung injury that develops suddenly from many direct or indirect causes. According to the literature, ARDS is a rare complication associated with DKA (2). Therefore, we present a case who was admitted to the intensive care unit (ICU) for DKA and developed ARDS during treatment.

Case Presentation

A 65-year-old male patient with a history of type 2 DM on insulin treatment was admitted to the emergency department (ED) with complaints of altered consciousness, shortness of breath, and weakness. On admission to the ED, his temperature was 36.9°C, pulse rate was 100/min, blood pressure was 140/80 mmHg, fingertip SO_2 in room air was 90%, respiratory rate (RR) was 20/min, and Glasgow Coma Score (GCS) was 14. The patient was tachypneic and apathetic. Other physical examination findings were normal. Biochemistry revealed a glucose level of 390 mg/dl and a potassium level of 5.03 mmol/L. Blood gas results were pH:7.11, $PaCO_2$:23 mmHg, HCO_3:7.3 mmol/L, PaO_2:95 mmHg, SO_2:95, lactate:1.45 mmol/L, and BE(B):-20. Complete urinalysis revealed glucose 4+, ketones 3+, leukocytes negative. The calculated anion gap was 23.70 mEq/L. Electrocardiography (ECG), posteroanterior chest radiograph (PAAG) (Figure 1), and other laboratory tests were normal, and the patient was admitted to the ICU with a diagnosis of DKA. In the ICU, hydration with 0.9% NaCl (3 L/24 hours), insulin infusion (0.1 IU/kg/hour), potassium chloride replacement (40 mL/24 hours), and empiric antibiotic treatment (ceftriaxone 2 g/day) were administered. Fluid and insulin infusion was continued until the ketonuria improved, then gradually tapered and discontinued. Glycemic control was achieved with treatment, and subcutaneous insulin was switched. During the patient's follow-up, he developed sudden respiratory distress on day 4. His vital

signs at the time of respiratory distress were as follows: blood pressure: 110/80 mmHg, pulse: 130/min, HR: 28/min, SO_2: 80%, and temperature: 36.8. The patient's blood glucose was measured as 232 mg/dl. GCS: 12, arterial O_2 pressure/inspiratory O_2 fraction (PaO_2/FiO_2): 200 was calculated. Diffuse rales were noted on auscultation. Blood gases were pH:7.50, $PaCO_2$:31.5 mmHg, PaO_2:55 mmHg, HCO_3:24 mmol/L, SO_2:81%, lactate:2 mmol/L, and BE(B):-2. PAAG and chest computed tomography showed bilateral diffuse infiltration (Figure 2,3). Bedside echocardiography showed normal valve, wall motion and ejection fraction, and non-invasive continuous positive airway pressure (CPAP) support was initiated with a diagnosis of ARDS. While conservative fluid resuscitation, antibiotics (meropenem 6gr/day and linezolid 1200 mg/day) and regular subcutaneous insulin treatment were administered, the patient, whose saturation and mean arterial pressure decreased on day 6, was intubated and continued to be followed with vasopressor support. The patient was monitored under deep sedation, continuous forced ventilation (CMV) mode, plateau pressure ≤30 cmH_2O, low tidal volume (5-6 mL/kg), high positive end-expiratory pressure (PEEP) (10-15 cm/H_2O), and intermittent prone position. Respiratory rate and FiO_2 levels were titrated according to blood gas results. However, with arterial blood gas monitoring, SO_2 levels remained low with gradually increasing $PaCO_2$ levels. The patient with elevated renal function tests (urea: 221 mg/dL, creatinine: 5.2 mg/dL), no urine output, and worsening acidosis (pH: 7.0) was dialyzed on day 8. Blood, urine, and tracheal aspirate cultures were negative for growth. Sudden cardiac arrest occurred during follow-up on day 9, and the patient did not respond to cardiopulmonary resuscitation and was pronounced dead.

Discussion

Although often seen in type 1 diabetics, in recent years it has been observed that 27% of patients hospitalised for DKA are type 2 diabetics (3). According to case reports in the literature, the development of ARDS due to DKA is most commonly observed in young people and children (4,5). ARDS, characterised by sudden onset, progressive hypoxaemia and bilateral pulmonary infiltrates, is a very rare complication of DKA. The mechanism of development of ARDS in DKA is thought to be multifactorial (6). ARDS involves an inflammatory process in the alveolar-capillary membrane leading to an accumulation of protein-rich fluid in the alveolar space. Many causes of acute direct and indirect lung injury have been identified as potential triggers of ARDS (4). The most common causes of ARDS are pneumonia, sepsis, aspiration, toxic inhalation, drowning, burns and trauma. Our case was an adult patient with type 2 DM who developed ARDS during DKA treatment and in whom other possible precipitating factors were excluded.

Although ARDS due to DKA is rare, it has a high mortality rate (5). Although the mechanism of ARDS is unclear, it is known that diabetic patients have increased capillary permeability (6). Two case reports of patients with DKA have suggested that severe acidosis may cause ARDS by increasing capillary membrane permeability and altering alveolar surfactant metabolism (4,6). However, although many patients with DKA are acidotic, ARDS develops in only some of them (5). In our case, although the metabolic acidosis at presentation with AS was mild, the fact that ARDS developed suggests that different mechanisms may cause this condition (6). Fluid resuscitation is the most important part of DKA management (7). It has been reported that volume resuscitation during treatment can also lead to the development of ARDS (3). Excessive fluid volume leaks into the alveoli with the effect of low oncotic pressure and may worsen oxygenation by increasing pulmonary wedge pressure. Therefore, it is essential to ensure optimal fluid balance (8). In our case, fluid resuscitation during treatment may have contributed to the development of ARDS (7).

Conclusion

We presented a case of an adult who died after developing ARDS as a result of DKA treatment. This presentation highlighted the importance of early recognition and appropriate management of ARDS, which is a rare complication of DKA. Vital signs and clinical findings should be closely monitored in these patients.

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4120

Mortality due to pneumonia: A retrospective chart review of patients diagnosed with pneumonia

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Introduction

Community-acquired pneumonia (CAP) is a major cause of morbidity and mortality worldwide and is particularly severe in the elderly, those with comorbidities and those with compromised immune systems. CAP places a significant burden on healthcare systems in terms of emergency department visits and hospitalizations. Therefore, early and accurate determination of disease severity is critical for planning appropriate treatment strategies and efficient use of resources (1).

In clinical practice, several scoring systems have been developed to assess the prognosis of patients with CAP and to predict the risk of mortality. These scores help to determine the need for hospitalization, predict the need for intensive care, and plan overall clinical management strategies. The CURB-65 score (confusion, urea level, respiratory rate, blood pressure, and age ≥65 years) is widely used because of its ease of use and predictive power (2). The Pneumonia Severity Index (PSI), which provides a more detailed assessment, offers high accuracy in predicting mortality by incorporating a large number of clinical and laboratory parameters (3). The qSOFA (Quick Sequential Organ Failure Assessment) score, which has become particularly prominent in the diagnosis of sepsis in recent years and has been used in patients with CAP, provides a rapid risk assessment using three parameters such as low blood pressure, high respiratory rate and change in mental status (4).

The aim of this study was to compare the efficacy of qSOFA, CURB-65 and PSI scores in predicting in-hospital mortality in patients diagnosed with CAP. The data obtained may provide important clues as to which score is more useful in clinical decision making and may help to optimize patient management.

Materials and Methods

This retrospective study was conducted in the Emergency Department (ED) of a regional academic hospital. The medical records of patients older than 65 years admitted to the ED for CAP between January 1, 2024, and December 31, 2024, were retrospectively analyzed. Patients with missing data, those younger than 65 years, and those with a Glasgow Coma Scale score of 3 on admission to the ED were excluded from the study. In addition to patient demographics, the following information was collected: PSI, CURB-65, and qSOFA score. Data were analyzed using SPSS version 22.0 (SPSS Inc, Chicago, IL, USA).

Results

A total of 248 patients diagnosed with community-acquired pneumonia with a mean age of 70.42 ± 16.30 years were included in the study. Of these, 137 (55.2%) were male and 111 (44.8%) were female. Mortality was observed in 47 patients (18.9%), while 201 patients (81.1%) survived.

When high-risk scores were compared between survivors and non-survivors, all three scoring systems-qSOFA, CURB-65, and PSI-showed statistically significant associations with in-hospital mortality ($p < 0.001$ for all; Table 1). Among deceased patients, the proportion classified as high-risk was 89.4% for qSOFA, 89.4% for CURB-65, and 95.7% for PSI. In contrast, the proportion of high-risk cases among survivors was significantly lower for all scores: 19.9% (qSOFA), 21.9% (CURB-65), and 33.3% (PSI). Detailed data regarding score distributions and statistical outcomes are presented in Table 1.

Logistic regression analysis further supported the predictive value of each scoring system (Table 2). High-risk qSOFA was associated with the highest odds of in-hospital mortality (Wald = 18.32, 95% CI: 3.61-31.69, $p < 0.001$). CURB-65 (Wald = 7.09, 95% CI: 1.51-15.25, $p = 0.008$) and PSI (Wald = 8.46, 95% CI: 2.14-50.56, $p = 0.004$) also showed statistically significant associations. These results indicate that all three scoring systems are effective in predicting mortality in hospitalized patients with community-acquired pneumonia, with qSOFA showing the strongest individual predictive power in this cohort. Detailed data regarding score distributions and statistical outcomes are presented in Table 2.

Discussion

In this study, we evaluated the effectiveness of three commonly used severity scoring systems-qSOFA, CURB-65, and PSI in predicting in-hospital mortality among patients diagnosed with CAP. Our results show that all three scoring systems were significantly associated with mortality and successfully discriminated between survivors and non-survivors.

Among the scoring systems evaluated, qSOFA showed the strongest association with mortality in logistic regression analysis, with a wide confidence interval and the highest Wald value. This finding is consistent with the growing evidence supporting the use of qSOFA as a rapid bedside tool, particularly in settings where laboratory data may be limited. Although originally developed to identify sepsis outside the ICU, the simplicity and high predictive power of qSOFA in our cohort suggest that it may also serve as an effective tool in the initial risk stratification of patients with CAP.

CURB-65, a well-established score developed specifically for CAP, also showed a significant association with mortality. It remains a practical tool, particularly in emergency and internal medicine settings, given its ease of use and inclusion of key physiologic variables. Our results support its continued use in clinical decision making, particularly in determining the need for hospitalization and potential ICU admission.

PSI, although more comprehensive and requiring detailed clinical and laboratory data, showed high sensitivity in identifying patients at risk of death. Nearly all patients who died (95.7%) were classified as high risk by PSI, underscoring its utility in identifying patients with poor prognosis. However, its relative complexity may limit its routine use in time-sensitive clinical settings.

Our findings are consistent with previous studies showing that higher scores on qSOFA, CURB-65, and PSI are associated with

increased mortality in CAP patients (3,5,6). However, the strength of qSOFA in this study is particularly noteworthy and warrants further investigation, especially given its simplicity compared to PSI.

Limitations

This study has several limitations. First, it was conducted in a single center, which may limit the generalizability of the findings. Second, although all scoring systems were evaluated at admission, dynamic changes in clinical status were not assessed over time. Lastly, we did not compare these scores with more recently developed or machine-learning-based prediction models.

Conclusion

All three scoring systems—qSOFA, CURB-65, and PSI—were effective in predicting in-hospital mortality among CAP patients. Among them, qSOFA showed the highest predictive value in our cohort. Considering its simplicity and ease of use, qSOFA may be a valuable tool for early mortality risk assessment in emergency settings. Nevertheless, each scoring system has its unique strengths and limitations, and their use should be tailored to the clinical context and available resources.

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Table 1. The comparion of qSOFA, CURB-65 and PSI between patients who died and survived.

	Survival (n=201)	Dead (n=47)	p value
qSOFA, high risk	40 (19.9)	42 (89.4)	<0.000
CURB-65, high risk	44 (21.9)	42 (89.4)	<0.000
PSI, high risk	67 (33.3)	45 (95.7)	<0.000

Table 2. The logistic regression analysis resuls

	Wald	%95 Confidence interval	P value
qSOFA, high risk	18.32	3.61 – 31.69	<0.000
CURB-65, high risk	7.09	1.51 – 15.25	0.008
PSI, high risk	8.46	2.14 – 50.56	0.004

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PSİKOZ SONRASI SCROTAL STRANGÜLASYON

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Introductıon

Penile strangulation by a foreign object is a rare condition [1]. It requires urgent intervention and treatment, as it may lead to vascular injury or even necrosis [2]. This is a surgical emergency, and delayed intervention can result in penile necrosis, gangrene, and amputation. Therefore, time plays a critical role in the management of these patients [3].

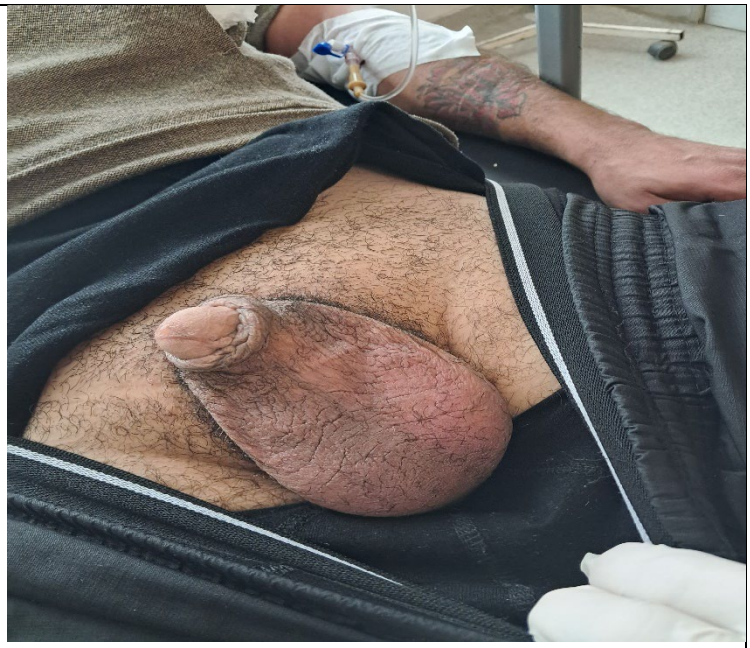
Due to the rarity of such presentations, there is no standardized protocol for the management of these cases, and clinicians often need to think outside the box. Here, we present a case of a 40-year-old male inmate with penile and scrotal strangulation.

Case Report

A 40-year-old male prisoner was brought in with complaints of back pain. The patient was unresponsive to verbal communication.

He was conscious, oriented, and cooperative, with a Glasgow Coma Scale score of 15. The abdomen was soft, with no guarding or rebound tenderness. No palpable mass was detected in the inguinal region. There were no neuromotor deficits. Distal pulses were bilaterally equal and palpable.

On inspection, a string was observed wrapped around the base of the penis and scrotum. The penis and scrotum were edematous and cyanotic. The patient's vital signs were stable, and there were no abnormalities in his blood tests.



Upon discovery, the string was removed.

A Doppler ultrasound was performed.

Doppler Ultrasound: Both testes and epididymides were in normal positions, with normal sizes and morphology. Vascularization was normal in the RDUS (Real-time Doppler Ultrasound) evaluation. No pathological amount of free fluid was detected between the layers of the tunica vaginalis.

The patient was consulted with a urology specialist, who determined that emergency surgical intervention was not necessary.

The patient was also referred to psychiatry. Since he was nonverbal during the psychiatric assessment, he was placed under observation due to the risk of suicide.

One hour after the string was removed, the penis and scrotum returned to their normal color and size. Following a 24-hour observation period, the patient was admitted to the psychiatric unit.

Discussion

Prolonged application of foreign objects to the penis can result in persistent venous congestion, obstructing lymphatic drainage and venous return. As the swelling and edema progress, intrapenile tissue pressure increases, which can cause skin erosion and ischemic necrosis of penile tissues. If not treated in a timely manner, irreversible damage may occur, and in some cases, auto-amputation of the penis has been reported [4,5].

Bhat et al. developed a simple classification system for the trauma caused by penile strangulation [6]:

Grade I: Distal penile edema without skin ulceration or urethral injury.

Grade II: Skin injury and corpus spongiosum constriction without evidence of urethral injury; distal penile edema with decreased sensation.

Grade III: Injury to the skin and urethra without urethral fistula; loss of distal penile sensation.

Grade IV: Complete division of the corpus spongiosum, urethral fistula, and corpus cavernosum constriction with loss of distal penile sensation.

Grade V: Gangrene, necrosis, or complete amputation of the distal penis.

There is no standardized protocol for the removal of a constricting penoscrotal object. The method of removal depends on the degree of trauma, duration of entrapment, type of material used, and equipment availability.

In our case, the strangulation was relieved by simply untying the knot made with the string. No signs of ischemia were found on ultrasound imaging.

As we were unable to communicate verbally with the patient, it remains unclear whether he applied the string himself or if someone else did it, as well as the purpose of the act.

Conclusıon

Penile strangulation with a constrictive device is a urological emergency with potentially serious clinical consequences. Clinicians should use the least traumatic technique to remove the constricting device from the genital area as soon as possible after entrapment.

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4262

Acute Heart Failure and Serotonin Syndrome Induced by Venlafaxine Overdose: A Lethal Case Report

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Introduction

Venlafaxine was the first agent to be marketed in the serotonin neuropeptinephrine reuptake inhibitor class (1). It is a more effective antidepressant than SSRIs in controlling symptoms of major depression and in treatment-resistant depression (2). Venlafaxine inhibits reuptake at serotonergic and noradrenergic receptors similar to tricyclic antidepressants, but does not affect histaminergic, muscarinic or adrenergic receptor mechanisms (3). However, in a study analyzing overdoses of antidepressants, venlafaxine-related overdoses were found to have a higher risk than other serotonergic drugs (4). This case report describes a fatal case of cardiotoxicity due to venlafaxine overdose.

Case

A 42-year-old woman with major depression was admitted to the emergency department 6 hours after taking a total of 6.3 g of venlafaxine extended-release tablets from her own medication with suicidal intent. On admission, the patient's consciousness was confused and GCS was 12 (3-5-4). The patient's pupils were bilaterally mydriatic.

The patient's ECG was consistent with sinus tachycardia of 125 bpm and the patient's blood pressure was 180/100 mmHg. The patient's body temperature was 37.8 C. One hour after admission, the patient had a generalized tonic clonic seizure responsive to diazepam. Routine blood tests were taken. There was no abnormal value in the blood tests at the time of admission except creatinine 1.02 mg/dL. The patient was clinically diagnosed with serotonin syndrome and supportive treatment was started. The patient was admitted to the emergency intensive care unit for monitorized follow-up. Within 8 hours, the patient had 3 more seizures characterized as JTK and these seizures were terminated with 1 mg diazepam. The patient was evaluated by a neurologist for recurrent seizures and changes in the state of consciousness. Diffusion MRI and EEG tests were performed. No pathology was found on brain imaging and EEG. The neurologist recommended levetiracetam for the patient's seizures. Because of hypertension and tachycardia, the patient was evaluated by a cardiologist with suspicion of venlafaxine-induced cardiotoxicity. Echocardiography was performed and the patient's EF: 65 and no pathology was found. Cardiology recommended amlodipine for the hypertensive patient. Supportive therapy and fluid resuscitation were continued.

At the 24th hour of admission, the patient's state of consciousness was GCS:15 (4-6-5) agitated. Blood tests taken at the 24th hour revealed WBC 21.4 thousand/uL and creatinine 0.98 mg/dL. The patient's ECG was compatible with 160 bpm sinus tachycardia and the patient's blood pressure was 170/80 mmHg. The patient had flushing. Diarrhea started at the 24th hour of follow-up. At the 40th hour, dyspnea developed and echocardiography was repeated by a cardiologist because of pulmonary edema findings. Echocardiography performed 40 hours after admission revealed EF:35 with global hypokinesia. Diuretic treatment was started for the patient with pulmonary edema due to acute heart failure. Creatinine was found to be 1.67 mg/dL and Hs Troponin 258.0 ng/dL in blood samples taken at 40th hour. The patient's state of consciousness regressed and the GCS was 2-4-2 at the 48th hour of admission and the patient was intubated. Anuria and hypotension developed at the 72nd hour of admission. Treatment with inotropic agents was started. At the 72nd hour of admission, creatinine was 2.42 mg/dL, AST 184 U/L, CPK 6420 U/L, Hs Troponin 296.0 ng/dL. The patient died in the 90th hour of admission despite supportive treatment and life support in intensive care unit.

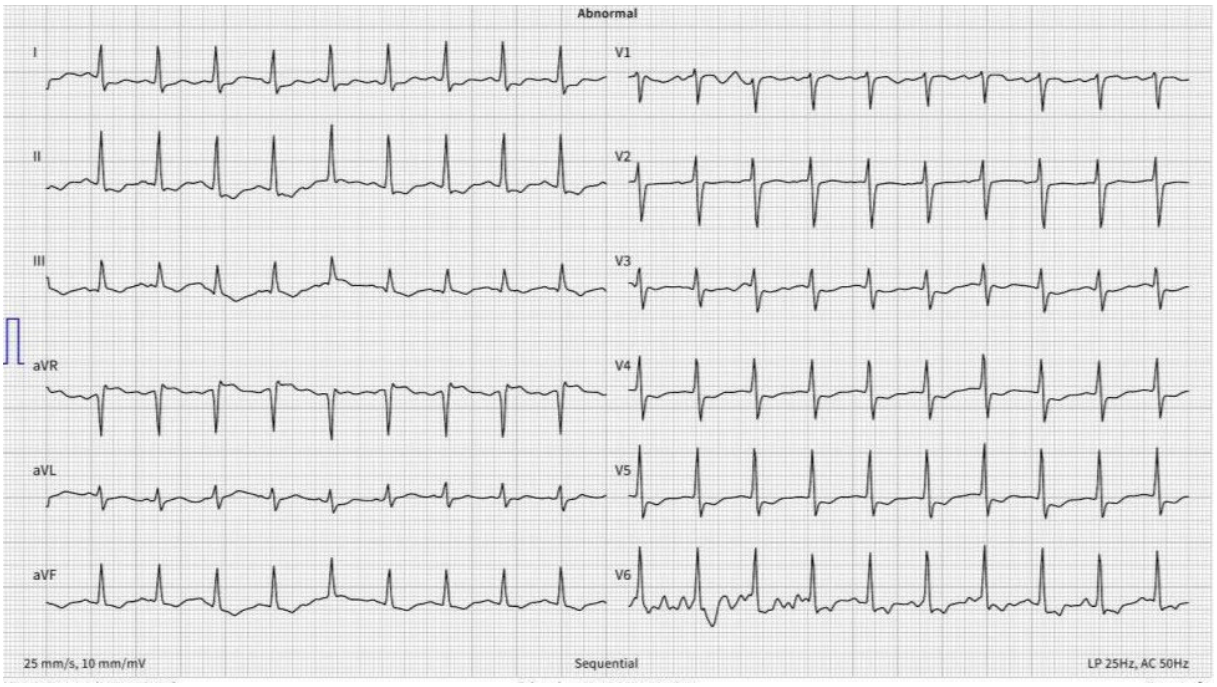

Figure 1. Electrocardiographic Findings at Admission in a Patient with Venlafaxine Overdose

Discussion

Venlafaxine is well absorbed and metabolized in the liver by cytochrome p450 enzymes. Different effects may be observed in patients due to genetic polymorphism. Following administration of extended-release forms of venlafaxine, peak plasma concentrations of venlafaxine and O-desmethylvenlafaxine (ODV) are reached within 5.5 and 9 hours, respectively. Venlafaxine and its metabolites are mainly eliminated from the kidneys (5).

Since venlafaxine is a potent reuptake inhibitor like SSRI group medications, it has the potential to cause serotonin syndrome (6). Serotonin syndrome is a life-threatening syndrome that occurs as a result of overactivation of peripheral and central postsynaptic 5HT-1A and 5HT-2A receptors due to serotonergic agent use. Altered mental status, neuromuscular hyperactivity and autonomic hyperactivity are observed in serotonin syndrome. Although serotonin syndrome is a diagnosis of exclusion, there is no gold standard test for the diagnosis. The diagnosis is made clinically by a medical toxicologist. Quick diagnosis and supportive treatment may prevent mortality related to serotonin syndrome (7-9).

Cardiotoxicity due to venlafaxine ingestion has not been extensively reported. Even after overdose, its effect on cardiac conduction is limited. The limited effect on cardiac conduction is explained by the fact that venlafaxine does not affect alpha adrenergic and histaminic receptors (10). However, cases of venlafaxine-induced heart failure with preserved cardiac conduction function have been reported. The mechanism underlying this clinical condition is thought to be catecholamine-induced myocardial damage associated with inhibition of norepinephrine reuptake (11, 12).

Venlafaxine overdose can lead to severe toxicity, primarily manifesting as serotonin syndrome and, in rare cases, delayed cardiotoxicity. While serotonin syndrome is a well-documented complication of venlafaxine toxicity, the development of acute heart failure with preserved initial cardiac function remains an underrecognized and potentially fatal consequence. This case highlights the importance of continuous cardiac monitoring in patients with venlafaxine overdose, even when initial cardiac function appears normal. The pathophysiological mechanism is likely related to catecholamine-induced myocardial injury due to norepinephrine reuptake inhibition. Early recognition, aggressive supportive care, and a multidisciplinary approach involving emergency physicians, cardiologists, and intensivists are essential for optimizing patient outcomes. Given the potential for delayed-onset cardiac complications, clinicians should maintain a high index of suspicion and ensure prolonged hemodynamic and cardiac function surveillance in such cases. This case underscores the need for further research to better understand the mechanisms underlying venlafaxine-induced cardiotoxicity and to develop targeted management strategies.

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Copper Sulfate (Bordeaux Mıxture) Exposure And Myocardıal Infarctıon

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Introductıon

Pesticides are widely used in agriculture to protect crops, yet they can have significant adverse effects on human health. The accumulation of heavy metals in the body, increased oxidative stress, and their potential link to cardiovascular diseases are growing concerns. (1) Copper sulfate, a commonly used fungicide, exemplifies these risks.

Copper Sulfate and Its Effects on Human Health

Copper sulfate is a chemical compound widely used to prevent plant diseases. The copper ions it contains can trigger oxidative stress, leading to cellular damage. These toxic effects may have detrimental consequences for the cardiovascular system.

Oxidative Stress and Cardiovascular Damage

Copper sulfate exposure can increase free radical production, leading to oxidative stress and vascular damage. (2) This oxidative stress accelerates atherosclerosis, potentially resulting in vascular blockages and increasing the risk of myocardial infarction.

Electrolyte Imbalance and Blood Pressure

Copper sulfate exposure can disrupt electrolyte balance, potentially leading to hypertension a significant risk factor for heart attacks.

Controversial Effects and Cardiac Sensitivity

There are varying opinions regarding the impact of pesticide exposure on human health. While studies suggest that long-term exposure to high doses has toxic effects, evidence regarding the cardiovascular risks of short-term exposure remains inconsistent. This case provides a valuable opportunity to examine the relationship between copper sulfate exposure and myocardial infarction.

Case Report

A 62-year-old male patient presented to an external emergency department with complaints of chest pain radiating to his back, accompanied by sweating and chills. He reported that his symptoms had persisted for six hours following pesticide exposure. The patient had no known comorbid conditions.

On examination, he was alert, oriented, and cooperative, with stable vital signs. His ECG showed a normal sinus rhythm. Laboratory results revealed:

Lactate: 3.4
Troponin: 253
CK-MB: 48

Due to suspicion of myocardial infarction, a cardiology consultation was requested. An echocardiogram revealed anteroseptal myocardial damage, and coronary angiography was planned. The patient was subsequently hospitalized, and an emergency angiography was performed. A stent was placed in the left anterior descending artery (LAD). After monitoring in the coronary intensive care unit, the patient was discharged.

Dıscussıon

1. Effect of Pesticide Exposure on Cardiovascular Risk

The impact of pesticide exposure on cardiovascular health has gained increasing attention in recent years. (3) Studies suggest that individuals exposed to pesticides may have a higher incidence of cardiovascular diseases due to the toxic effects of these chemicals. While most research focuses on long-term exposure, this case highlights that even short-term exposure can lead to serious cardiovascular events. Therefore, the acute effects of pesticides should not be overlooked.

2. Clinical Challenges

Identifying the cardiovascular effects of pesticide exposure can be challenging in clinical practice. In patients presenting with myocardial infarction, pesticide exposure may be an overlooked factor, particularly when more common cardiovascular risk factors are absent. (4) Healthcare professionals should maintain a high index of suspicion, especially when evaluating patients who work in agricultural settings.

3. Toxicological Profile of Copper Sulfate and Risk Management

To minimize the toxic effects of pesticides, stricter regulations on substances like copper sulfate and the establishment of exposure limits are necessary. (5,6) Additionally, proper training and the use of personal protective equipment should be emphasized for individuals working with these chemicals. Clinically, the rapid identification of acute pesticide-related toxic effects can facilitate early intervention and improve patient outcomes.

Conclusıon

Pesticides, including copper sulfate (Bordeaux mixture), pose potential cardiovascular risks that should not be underestimated. Chemical exposure can trigger oxidative stress and electrolyte imbalances, leading to severe cardiovascular events such as myocardial infarction. This case underscores the need for increased awareness of these risks, improved clinical assessment protocols, and enhanced safety measures for individuals working with pesticides.

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Diffuse Axonal Injury in the Corpus Callasum That Should Not Be Forgotten in Severe Post-Traumatic Headache; Case Report

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Diffuse axonal injury (DAI) is a type of traumatic brain injury (TBI) that occurs as a result of blunt trauma to the brain. In the United States, traumatic brain injury is the leading cause of death and disability among children and young adults. Diffuse axonal injury (DAI) primarily affects the white matter tracts in the brain. Clinically, patients with DAI may present a spectrum of neurological dysfunction. This can range from clinically insignificant to comatose. Patients are classified according to the Glasgow coma scale from mild to severe. DAI should be kept in mind, especially if patients classified as mild as GKS 12-15 do not have neurological deficits. In this case, we planned to present the development of DAI in the corpus callasum of a young patient who presented to the emergency department due to recurrent headache after blunt trauma.

Case Report

22-year-old female patient 5 days ago FALLING FROM OWN LEVEL Blunt Trauma After Emergency Application Present. Initial Admission Examination NECK stiffness negatıve. kernıg negatıve. neurologıcal examınatıon natural. fever 36 ta:110/60 gcs:15 ır +/+,5 after applying to the emergency room with headache intermittently throughout the day and no bleeding or subacute ischemia image in repeated ct scans, the patient was examined on the 5th day after the examination. C.CT AND DIFF. MR imaging suggested acute ischemia image/diffuse axonal damage at the level of corpus callosum splenium. The patient's 5-day examination General Condition Good, Conscious, Oriented-Cooperative, Pupils Isochoric, Ir +/+. No pathology was observed in Cranial Nerve examination. four extremities were mobile, muscle strength was full. Vitals were stable. Neurology and brain surgery were consulted. Since 5 days had passed and the patient was stable, he was discharged as an outpatient clinic control.

Dıscussıon

In the case we presented, although the persistent headache, especially despite the low-energy trauma, initially alerted emergency medicine clinicians in terms of bleeding, it is useful to examine patients in whom no bleeding was detected in examination and computed tomography in terms of DAI in further examination. We think that it will be important in terms of contributing to the literature, as it is a case that was rarely detected before and was diagnosed more often in postmortem cases, and that our case had high GCS, did not cause any neurological deficits and presented with only headache.

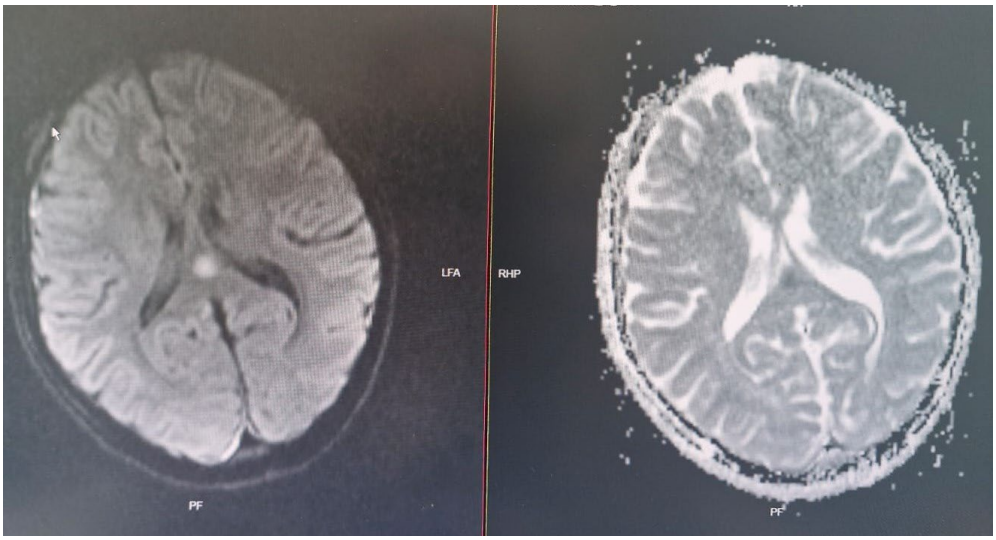

Figure 1. MRI image of the patient

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4436

The Silent Attack: Acute Hepatitis Triggered by Epstein-Barr Virus – A Rare Case Report

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Introductıon

Epstein-Barr Virus (EBV) is a ubiquitous herpesvirus, primarily known for causing infectious mononucleosis, particularly in adolescents and young adults. While the classic triad of fever, pharyngitis, and lymphadenopathy is well-documented, EBV can rarely present with atypical and potentially serious complications such as acute hepatitis.

In this case, we report a 20-year-old male with no significant travel or alcohol history, presenting with progressive abdominal pain, elevated inflammatory markers, and hepatomegaly. Imaging revealed periportal edema, gallbladder wall thickening, ascites, and pleural effusions, prompting evaluation for acute hepatitis of viral etiology.

The purpose of this case is to highlight the diagnostic challenges and clinical course of EBV-induced acute hepatitis, emphasizing the importance of considering EBV in patients with systemic symptoms and abnormal liver imaging, even in the absence of classic mononucleosis features.

Materials And Methods

This case report is based on the clinical evaluation, laboratory investigations, and imaging studies of a 20-year-old male patient admitted to the emergency department with complaints of progressive abdominal pain, dysuria, and a history of recent antibiotic use. The diagnostic process included:

Comprehensive physical examination

Laboratory tests: complete blood count, liver and renal function tests, inflammatory markers (CRP, procalcitonin), coagulation profile, urinalysis, and serologic screening (including ELISA for EBV and other viral markers)

Radiologic imaging: abdominal ultrasound and contrast-enhanced computed tomography (CT)

Clinical follow-up to assess symptom progression, treatment response, and ruling out alternative diagnoses

All findings were evaluated in accordance with the patient's medical history, medication use, and risk factors. The final diagnosis of EBV-related acute hepatitis was made by exclusion and supported by clinical presentation, laboratory abnormalities, and radiological features.

Dıscussıon

Epstein-Barr virus (EBV) is a widespread human herpesvirus that commonly causes infectious mononucleosis, especially in adoles-

cents and young adults. While most EBV infections are self-limited and benign, atypical and severe presentations involving hepatic, renal, and pulmonary systems have been increasingly recognized in clinical practice.

In the presented case, the patient lacked the hallmark features of mononucleosis, such as pharyngitis, lymphadenopathy, and fatigue. Instead, he developed acute hepatitis with systemic inflammatory response, renal dysfunction, ascites, and bilateral pleural effusions—findings that are rare in primary EBV infection but documented in a limited number of case reports. The absence of classical signs complicated the diagnostic process, emphasizing the importance of a broad differential diagnosis in patients with systemic symptoms and liver dysfunction.

Liver involvement in EBV infection is relatively common, with mild elevations in aminotransferases seen in up to 80% of cases. However, clinically significant hepatitis, especially with hepatic enlargement and ascites, is rare. In our patient, imaging revealed hepatomegaly, periportal edema, and gallbladder wall thickening, consistent with inflammatory hepatic insult. Elevated CRP and procalcitonin levels initially suggested bacterial infection, leading to empiric antibiotic use, which was later discontinued when bacterial etiology was excluded.

Renal dysfunction in EBV infection is usually secondary to systemic inflammation, hypoperfusion, or immune-mediated mechanisms. Similarly, pleural effusions may reflect capillary leak, hypoalbuminemia, or inflammation-induced serositis. The presence of these findings in a young, otherwise healthy individual underscores the potential of EBV to cause multi-organ involvement even in immunocompetent patients.

This case also highlights a common clinical pitfall—empiric antibiotic treatment in patients with viral syndromes and non-specific systemic inflammation. Early recognition of viral etiologies and cautious interpretation of inflammatory markers can prevent unnecessary antimicrobial use and associated complications.

Overall, this case contributes to the growing body of literature describing EBV-related hepatitis and systemic involvement. Clinicians should remain vigilant for atypical EBV presentations and consider the virus in the differential diagnosis of acute hepatitis, particularly when standard bacterial and viral panels return negative.

Results And Conclusion

The patient was diagnosed with Epstein-Barr virus (EBV)-related acute hepatitis based on clinical presentation, elevated inflammatory markers, hepatomegaly, periportal edema, and exclusion of other viral or toxic causes. Despite the absence of classic mononucleosis symptoms such as pharyngitis or lymphadenopathy, EBV was identified as the likely etiology.

The patient responded well to supportive treatment, including hydration, antipyretics, and close monitoring. Antibiotics were discontinued after ruling out bacterial causes. His liver function and inflammatory markers gradually improved, and he was discharged with outpatient follow-up.

This case highlights the importance of considering EBV as a differential diagnosis in young patients presenting with unexplained hepatitis and systemic inflammation, even in the absence of classic signs. Early recognition can prevent unnecessary antibiotic use and ensure appropriate supportive care. Clinicians should be aware of such atypical presentations to improve diagnostic accuracy and patient outcomes.

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Management of Spontaneous Ureteral Perforation: A Case Report and Literature Review

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Introduction

Ureteral stones are one of the most seen causes of spontaneous ureteral perforations. However, there are some less frequent conditions that cause obstruction which can result in ureteral perforation. Such as; tumors, pregnancy, enlarged lymph glands, ruptured renal cysts, congenital anomalies, renal transplants, retroperitoneal fibrosis, and scarring resulting from radiation therapy.(1)

We present a case of a 61-year-old female with spontaneous ureteral perforation possibly originated from nephrolithiasis.

Case Report

A 61-year-old woman presented to the emergency room with a new onset of abdominal pain. Her medical history was insignificant apart from hypertension. She denied vomiting, painful urination or other associated symptoms except nausea.

On physical examination, there was tenderness in the left lower quadrant. However, the rebound was negative. A Biochemistry panel, FBC and urine sample were taken.

In Biochemistry Panel: GLUCOSE: 109 MG/DL UREA: 37 MG/DL EGFR:60,95 CREATININE: 1,00 MG/DL , CRP: 2,9 MG/L, SODIUM: 137 MMOL/L, POTASSIUM : 4,40 MMOL/L, CHLORINE: 105,4 MMOL/L,

In Full Blood Count: WBC:12,04 K/uL , NEU# :10.05 K/uL , LYMPH# : 1.35 K/uL , , HGB: 14G/DL

In the urine sample: Protein was negative, nitrite: negative, Glucose: negative, PH:5.5 Leu: 3+, Erythrocyte: 92 Leukocyte: 63.

After that Abdominal CT scan with contrast was performed (Figure 1).

In the scan the left ureter and collecting system were dilated. A 4 mm calculus was observed in the proximal ureter. Parapelvic and cortical cysts were present in both kidneys. There was significant contamination in the perirenal area of the left kidney. Contamination suspected to be due to calixyal rupturing and a new CT scan was done in order to see contrast's excretion from the urinary tract (Figure 2).

There was contrast extravasation at the lower pole of the left kidney. Surrounding fat planes appeared contaminated, with band-like fluid densities observed indicating calyceal rupture. Urology was consulted and a double-J stent was placed in the patient. The procedure was completed without complications. A follow-up was recommended by urology in one month.

Figure 1: CT Scan with contrast

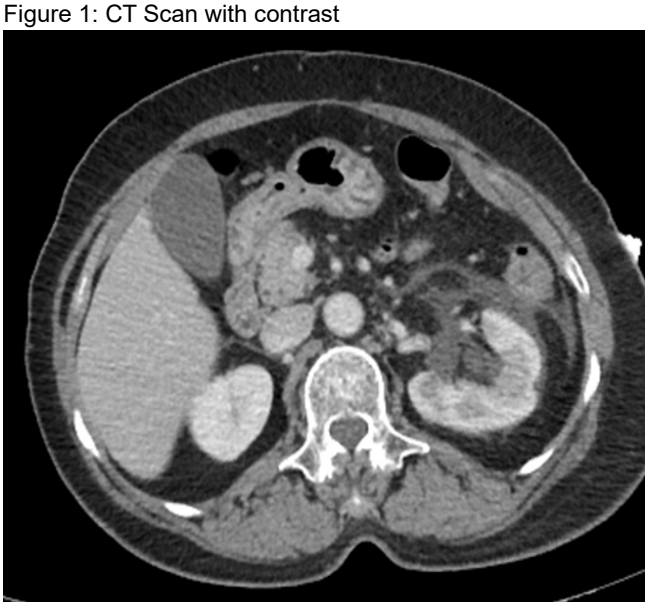
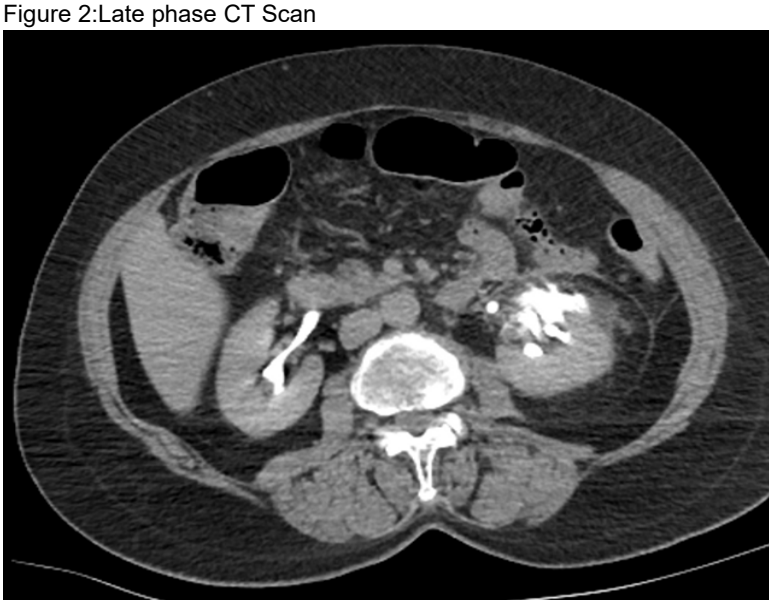


Figure 2:Late phase CT Scan



Discussion

The most common causes of spontaneous ureteral perforation include urinary conditions such as ureteral calculi, strictures, and tumors.(2) Akpinar et al. analyzed 91 documented cases of spontaneous ureteral rupture and found that in 72% of them, the underlying cause was stone disease.(3)

Renal colic has the same symptoms as spontaneous perforation of the ureter. In a study about spontaneous ureteral perforation cases, sudden onset of flank pain was observed in all patients, while nausea and vomiting were present in 80%. Both renal colic and spontaneous ureteral perforation can present with abnormalities in laboratory tests, just as complete blood count may show leukocytosis, urinalysis can reveal red blood cells and leukocytes.(4)We want to emphasize that, especially in emergency settings, ureteral perforation should be considered, as it can present exactly like renal colic and can be overlooked but may lead to serious morbidity and/or mortality.

USG is the first imaging method for renal colic. However, a late-phase contrast-enhanced CT scan (5–20 minutes after contrast medium administration) can identify urinoma and urinary system leaks. Furthermore, the differential diagnosis includes diverticulitis, cholecystitis, and appendicitis, which can be diagnosed with a CT scan. (5). In our case, a late-phase contrast-enhanced CT scan was performed, as it is an effective method for detecting urinoma and urinary system leaks. If there is any clinical suspicion, a CT scan should be performed, as it is essential for diagnosing various pathologies, including perforation, as was the case in our patient.

Spontaneous ureteral rupture is considered a rare condition. Due to this, there is no established protocol for managing this condition.(6) In 3 out of the 4 cases, spontaneous healing of the rupture was observed within 7 days or less, as confirmed by CT scan following conservative medical treatment.(3) Starvodimos reports on 5 patients with spontaneous ureteral rupture who were treated with Double J ureteral stents, resulting in resolution.(7) Choi et al. managed a case of upper ureter rupture using a pigtail percutaneous nephrostomy catheter, and 7 days later, antegrade pyelography showed no contrast extravasation at the rupture site.(8) Even though there are various approaches to this condition, the most common treatment approach in the literature was Double J ureteral stent(9)

In cases of fever or hemodynamic changes, the possibility of urinoma infection should be kept in mind, and antimicrobial therapy along with drainage may be considered. Also, elevated creatinine can be seen due to the absorption of urinoma (9).

Conclusion

Spontaneous ureteral rupture should always be considered in the differential diagnosis of patients presenting with complex symptoms following renal colic. In our case, although initial urine analysis did not immediately suggest perforation, the clinical presentation was unclear. This emphasizes the importance of considering further diagnostic evaluation in cases of suspected complex renal colic.

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In the shadow of a heavy body: neurological reflections of sacral trauma

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Introductıon

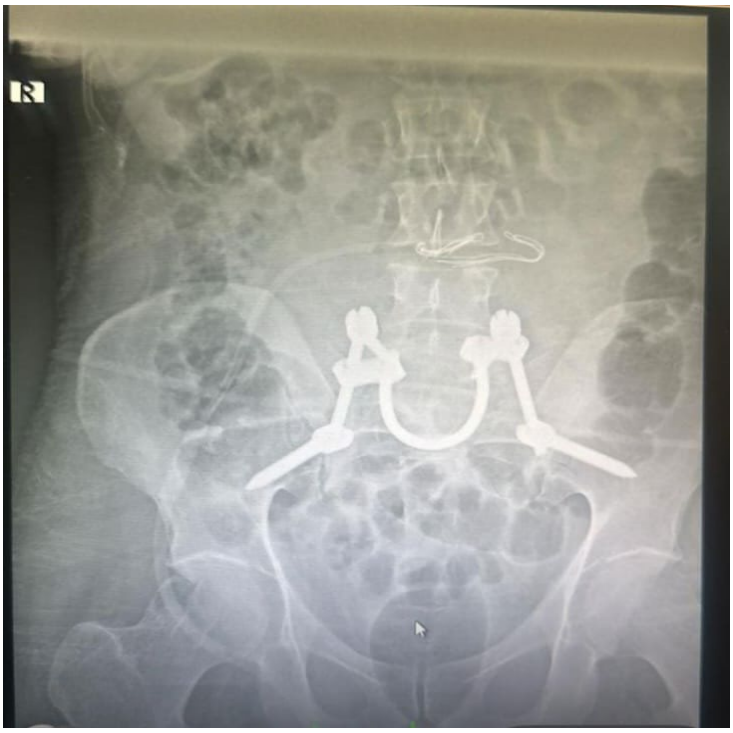
Sacral fractures are among the serious spinal injuries that develop as a result of high-energy traumas. The sacrum is a bone located at the lowest part of the spine, provides axial load transfer and is in close anatomical relationship with the pelvic ring. Sacral fractures usually occur as a result of major traumas such as traffic accidents, falls from heights, work accidents and heavy object impacts. These fractures can cause mechanical instability and also lead to neurological deficits due to their close relationship with nerve structures. One of the most common classifications of fractures occurring in the sacrum is the Denis classification. According to this classification, sacral fractures are divided into three different zones. Zone 1 fractures are located in the lateral parts of the sacrum and are generally considered stable. Zone 2 fractures are at the level of the sacroiliac joint and can affect pelvic stability. Zone 3 fractures involve the sacral foramina and the midline of the sacral canal. These fractures can cause serious neurological deficits by damaging the nerve roots and usually require surgical intervention (1). One of the most important complications of sacral fractures is neurological damage. The sacrum is a region that contains nerve structures including the sacral plexus, and fractures in this region can lead to serious problems such as loss of strength, loss of sensation, and urinary and fecal incontinence in the lower extremities. In particular, damage to the S2-S4 nerve roots may impair sphincter control and patients may develop perineal hypoesthesia. In sacral fractures that compress nerve roots, patients typically present with symptoms such as loss of sensation in the perineal region, difficulty urinating or, conversely, urinary incontinence, loss of rectal tone, and loss of strength in the lower extremities (2). Patients usually complain of severe pain in the lumbosacral region. This pain may increase with palpation and becomes more pronounced, especially when direct pressure is applied to the sacrum. Muscle strength tests should be performed to assess whether there is neurological damage in the lower extremities. Hip flexion, knee extension, foot dorsiflexion and plantar flexion can be tested to detect asymmetries or significant strength losses in muscle strength. While patellar reflexes are usually preserved, the Achilles reflex may weaken or disappear, especially when the S1 root is affected. However, sphincter tone should definitely be evaluated in patients with suspected cauda equina syndrome. Findings such as decreased sphincter tone and urinary or fecal incontinence during rectal examination indicate a condition requiring urgent surgical intervention (3). Imaging methods are of great importance for definitive diagnosis in sacral fractures. Although direct radiographs are often used as the first step, the majority of sacral fractures can only be clearly evaluated with computed tomography (CT). CT allows detailed examination of the fracture line and also reveals accompanying pelvic ring injuries. Magnetic resonance imaging (MRI) is used to evaluate whether the nerve roots are affected and should be preferred especially in patients with neurological deficits (4). The treatment approach is determined by considering the type of fracture, the presence of neurological deficits, and the general condition of the patient. Stable sacral fractures can be managed with conservative treatment. Absolute bed rest, analgesic treatment, physical therapy, and rehabilitation are recommended for such patients. However, surgical treatment becomes inevitable in unstable fractures with neurological deficits. The main purpose of surgical treatment is to remove the bone fragments that compress the spinal canal and to restore the mechanical stability of the sacrum. Decompression and spinopelvic fixation are among the most common surgical approaches that support neurological recovery. Spinopelvic stabilization using pedicle screws and rod systems both supports the union of the fractured segments and allows early mobilization of the patient (5). Patients should be closely monitored in the postoperative period. The recovery process may vary in patients with neurological deficits. The time and degree to which the pressure on the nerve roots decreases is one of the most important factors determining the patient's chances of recovery. The rehabilitation process should be started early and patients should be

directed to a physical therapy program. A multidisciplinary approach should be applied to bladder and bowel management, especially in patients who develop sphincter dysfunction. It has been reported in the literature that patients who undergo early surgical intervention have better functional results in the long term (6). A multidisciplinary approach should be adopted in the diagnosis and treatment process of sacral fractures, and orthopedics, neurosurgery, physical therapy and rehabilitation teams should work together. Considering the risk of permanent neurological deficits in such patients, appropriate treatment should be planned as soon as possible and early intervention should be performed (7).

Case Report

A 36-year-old male patient was admitted to the emergency department after being trapped in a high-energy trauma after a heavy object fell on his back at work. The patient's symptoms included loss of sensation in the scrotal region, urinary incontinence, fecal discharge, and severe pain in the lumbar and sacral regions. No signs of trauma or hematoma were observed in the scrotal region during physical examination. When motor and sensory examinations were evaluated in the lower extremities, mild loss of strength was detected bilaterally. When direct radiographs and lumbosacral CT scans taken in the emergency department were examined, fractures were detected at the S1 and S2 levels and it was seen that the fracture fragments created significant compression towards the posterior part of the spinal canal. The patient was provided with emergency stabilization, further evaluation by the spinal surgery and neurology departments was recommended, and an operation was planned (Figure 1).

Figure 1. X-ray after the surgery



Discussion

Sacral fractures often develop due to high-energy trauma and in many cases are accompanied by neurological complications (4). Fractures occurring in the posterior part of the sacrum can cause serious motor and sensory disorders by compressing the nerve roots (6). In this case, the fracture occurring at the S1 and S2 levels caused urinary and fecal incontinence in the patient by creating posterior compression on the spinal canal. It has been reported in the literature that neurological deficits due to sacral fractures frequently develop in similar cases (2). In a study conducted by Angthong et al., it was stated that complex lumbosacral fracture-dislocations are highly associated with neurological deficits and that surgical stabilization supports neurological recovery (2). A detailed neurological examination is necessary in patients after trauma. Neurological complications of sacral fractures include cauda equina syndrome, sacral plexopathy and sphincter dysfunction (3). Particularly, loss of sphincter function is an important indicator requiring surgical intervention and can affect the long-term prognosis of the patient (5). In our case, the S1-S2 fracture, which caused significant posterior compression into the spinal canal, resulted in loss of sensation in the scrotal region and urinary/fecal incontinence. Similar cases in the literature show that neurological recovery is possible after surgical stabilization (4). However, the degree of neurological damage and the presence of secondary damage to the nerve tissue are the main factors determining the recovery process (4). Therefore, early detection of neurological deficits in traumatic spinal injuries, especially in patients with sacral fractures, is of great importance. In cases where the spinal canal is compressed posteriorly, emergency decompressive surgery should be considered to prevent neurological deficits (6). Finally, good knowledge of the motor sensory areas that the damaged nerve structure will affect in sacral region fractures helps to direct the neurological examination correctly. In this context, evaluation of the anatomical distribution of the sacral nerve roots is critical in understanding the neurological complications associated with sacral fractu-

res (5).

Conclusıon

This case report includes the evaluation of a patient who had a sacral fracture and developed a neurological deficit after a work accident. Neurological evaluation is of great importance in the diagnosis and management of sacral fractures. In particular, in patients who develop urinary and fecal incontinence, the sacral nerve roots should be evaluated and early surgical intervention should be planned. Similar cases in the literature show that neurological recovery is possible with early diagnosis and appropriate surgical intervention.

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The Predictive Value of the Hemoglobin-Albumin-Lymphocyte-Platelet (HALP) Score in the Prognosis of Crimean-Congo Hemorrhagic Fever

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Introduction and purpose

Crimean-Congo Hemorrhagic Fever (CCHF) is a zoonotic viral infection with a high mortality rate, representing a significant public health concern, particularly in endemic regions. The identification of early prognostic biomarkers is of great importance in facilitating clinical management and reducing mortality. In this study, the predictive power of a combined scoring system—based on hemoglobin, albumin, lymphocyte, and platelet values—was evaluated in forecasting the prognosis of patients with CCHF.

Material and Methods

This retrospective study was conducted in the emergency department of Erzurum City Hospital. A total of 79 patients diagnosed with CCHF, aged over 18 years and without missing data, were included in the study. Hemoglobin, albumin, lymphocyte, and platelet values at the time of admission were obtained from the electronic medical record system. The Hemoglobin-Albumin-Lymphocyte-Platelet (HALP) score was calculated individually for each patient using these parameters.

Results:

A total of 79 patients diagnosed with Crimean-Congo Hemorrhagic Fever (CCHF) were included in the study. The mean age of the patients was 50.89±16.70 years, and 64.6% (n=51) were male. Among the included patients, 76 were discharged, while 3 patients died. When comparing these two groups, the lymphocyte count was found to be significantly lower in the non-survivor group **(p = 0.021).** However, no statistically significant differences were observed in terms of hemoglobin, albumin, platelet count, or HALP score ($p > 0.05$). The clinical and laboratory characteristics of the patients are presented in Table 1.

Table 1

Variable	Total (mean±SD)	Survivors (mean±SD)	Non-survivors (mean±SD)	P value
Age	50.89 ± 16.70	50.55 ± 16.70	59.33 ± 17.62	0.480
Gender, male	51 (64.6%)	48 (63.2%)	3 (100%)	0.488
Albumin	37.48 ± 4.68	37.66 ± 4.58	32.83 ± 6.01	0.297
HGB	13.35 ± 2.31	13.35 ± 2.25	13.28 ± 4.18	0.980
LYMPH#	0.95 ± 0.63	0.95 ± 0.64	0.77 ± 0.04	**0.021**
PLT	119.60 ± 90.27	121.61 ± 91.11	68.78 ± 49.83	0.198
HALP Score	5.74 ± 5.71	5.64 ± 5.63	8.19 ± 8.67	0.663
WBC	4.34 ± 3.64	4.37 ± 3.70	3.59 ± 1.37	0.445
ALT	105.09 ± 119.00	105.01 ± 120.26	107.00 ± 99.86	0.976
AST	160.80 ± 228.16	155.09 ± 222.34	305.33 ± 379.32	0.564
Creatinine	1.01 ± 0.76	0.95 ± 0.61	2.57 ± 2.24	0.336

HGB: Hemoglobulin, Lymph: Lymphocyte, Plt: Platelet, WBC: White Blood Cell, ALT:Alanin aminotransferaz, AST: Aspartat aminotransferaz

Conclusion

In this study, the potential predictive value of the HALP score and its individual components (hemoglobin, albumin, lymphocyte, and platelet) in estimating in-hospital mortality among patients diagnosed with Crimean-Congo Hemorrhagic Fever (CCHF) was investigated. According to our findings, the lymphocyte count was significantly lower in the non-survivor group compared to the survivors ($p = 0.021$). This finding is consistent with previous studies in the literature suggesting that lymphocytopenia in viral infections may be associated with disease severity and poor prognosis.

Limitations of our study include the small number of fatal cases and its retrospective design. Nevertheless, it is one of the few studies examining the role of the HALP score and basic hematological parameters in the prognosis of CCHF, highlighting its significance.

Keywords: CCHF, HALP SCORE, PROGNOSİS

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Chronic Methemoglobinemia: A Rare Case with Dramatic Response to Methylene Blue Following Informed Refusal and Recurrent ICU Indication

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Introduction

Methemoglobinemia is a rare hematologic disorder characterized by the oxidation of hemoglobin's iron from the ferrous (Fe^{2+}) to the ferric (Fe^{3+}) state due to oxidative agents, impairing its oxygen -carrying capacity. Under normal physiological conditions, methemoglobin levels in the blood remain between 1–2% (1). Oxidative stress increases the oxidation of hemoglobin, leading to elevated methemoglobin formation. Methemoglobin is unable to bind and transport oxygen effectively, resulting in impaired oxygen delivery to tissues. Clinically, methemoglobinemia presents with decreased oxygen saturation and central cyanosis that does not respond to oxygen therapy. The diagnosis is confirmed by arterial blood gas analysis, and treatment involves the use of reducing agents such as methylene blue (2).

Case Report

A 46-year-old female patient presented to the emergency department with complaints of cyanosis of the lips and fingertips. She had traveled from Konya to Istanbul to visit family. The patient reported a history of intermittent cyanosis of the lips and fingers since childhood, which had also been present prior to the trip. Upon the recommendation of her relatives, she initially presented to a secondary care emergency department. Physical examination revealed no pathological findings in either lung, with equal bilateral respiratory movements. Respiratory rate was 15 breaths/min, blood pressure 135/65 mmHg, heart rate 90 bpm, and peripheral oxygen saturation (SpO_2) was 84%. The patient was neither tachypneic nor dyspneic. She denied any recent medication use, but reported frequent consumption of beetroot in Konya, raising the possibility of a dietary etiology.

Referral to a tertiary care center for further evaluation and treatment was requested via the national referral system. However, the patient declined the referral, stating that her symptoms were not new and had occurred intermittently since childhood. After signing an informed refusal form, she left the hospital voluntarily.

Two days later, the patient presented to a tertiary emergency department as an outpatient with similar complaints. Blood gas analysis revealed results nearly identical to the previous visit. Due to the indication for intensive care, intravenous methylene blue therapy was initiated in the emergency department.

Venous blood gas analysis revealed the following values: pH 7.394, pCO_2 40.7 mmHg, pO_2 37.6 mmHg, HCO_3^- 23.9 mmol/L, base excess (BE) 0.1 mmol/L, Na^+ 137.0 mmol/L, K^+ 4.5 mmol/L, Ca^{2+} 1.06 mmol/L, glucose 136 mg/dL, lactate 1.8 mmol/L, and methemoglobin (MetHb) level of 38%, confirming the diagnosis of methemoglobinemia.

Follow-up blood gas analysis performed two hours after treatment showed stable values: pH 7.394, pCO_2 40.7 mmHg, pO_2 37.6 mmHg, HCO_3^- 23.9 mmol/L, BE 0.1 mmol/L, Na^+ 137.0 mmol/L, K^+ 4.5 mmol/L, Ca^{2+} 1.06 mmol/L, glucose 136 mg/dL, lactate 1.8 mmol/L, and a significantly decreased MetHb level of 3%.

Discussion

In this case, the patient was found to have chronic methemoglobinemia resulting from a clinical condition to which she had grown accustomed. Chronic methemoglobinemia is typically congenital in origin and may be triggered by specific stressors or environmental factors. The condition often resolves spontaneously. We believe that the patient's refusal to be transferred from the initial facility was primarily due to her previous experiences with similar episodes that had resolved without treatment. Furthermore, the fact that her admission was prompted by her relatives rather than her own concern over the clinical symptoms supports this assumption.

In this case, no clear trigger for the methemoglobinemia episode was identified; however, it was noted that the patient consumed large quantities of beetroot in her hometown, which may have played a contributing role. No medication-related cause was found.

In a review published by Hord et al. in 2009, the biological transformations of dietary nitrates and their potential health effects were comprehensively discussed. It was emphasized that excessive consumption of high-nitrate foods (e.g., beets) could increase the risk of methemoglobinemia under certain conditions [3].

The treatment of methemoglobinemia varies depending on the patient's clinical status and the level of methemoglobin. In mild cases, withdrawal of oxidative agents and supportive care may be sufficient. However, in cases where methemoglobin levels exceed 20% or severe symptoms are present, specific treatment becomes necessary [4].

Feature	**Acute Methemoglobinemia**	**Chronic (Genetic) Methemoglobinemia**	**Present Case**
Age	35 years	10 years	46 years
Onset	Sudden, post-medication	Congenital	Intermittent since childhood
Symptoms	Sudden cyanosis, dyspnea	Mild cyanosis, asymptomatic	Cyanosis of lips and fingers, mild dyspnea
Vital Signs	Sat 85%, tachycardia	Sat 85%, normotensive	Sat 84%, BP 135/70 mmHg, HR 90 bpm, afebrile
Clinical Course	Rapid deterioration	Stable, asymptomatic	Stable, no tachypnea or dyspnea
Diagnosis	Co-oximetry, medication history	Genetic testing, co-oximetry	Clinical + co-oximetry
Treatment	Methylene blue	Antioxidant therapy (vitamin C, riboflavin)	Full recovery with methylene blue

Methylene blue activates the NADPH-dependent methemoglobin reductase pathway, which reduces methemoglobin back to hemoglobin. It is typically administered intravenously at a dose of 1–2 mg/kg, and its effect is usually observed within a few hours. This treatment is particularly effective in acquired methemoglobinemia [5]. However, in patients with G6PD deficiency, methylene blue is contraindicated due to the risk of hemolytic anemia [6,7].

High-flow oxygen therapy may be administered to alleviate symptoms of hypoxia. This supportive treatment helps improve tissue oxygenation impaired by methemoglobinemia [8].

In cases of severe hypoxia or anemia due to methemoglobinemia, blood transfusion may be necessary. This approach is particularly considered for patients who are unresponsive to methylene blue or in whom it is contraindicated [9].

In acquired methemoglobinemia, identifying and removing the offending oxidative agent is crucial. For instance, in drug-induced cases, discontinuation of the responsible medication often leads to symptom resolution [10].

Conclusion

This case illustrates that the diagnosis of chronic methemoglobinemia may be delayed due to stable vital signs and the presence of longstanding symptoms. Prompt recognition and administration of methylene blue led to rapid symptom resolution in this patient, emphasizing the importance of a multidisciplinary approach in such rare presentations.

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The Frequency of Hyponatremia in Intensive Care Unıt and Its Relationship with Mortality

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Introduction:

Intensive care units (ICUs) are environments where life-threatening conditions are managed, and critical interventions are performed. Therefore, the early detection and effective management of electrolyte imbalances in ICU patients can have a direct impact on overall prognosis. One of the most common electrolyte imbalances, hyponatremia, is defined as a serum sodium level below 135 mEq/L. This condition increases the risk of complications, particularly in critically ill patients, by impairing cardiovascular, neurological, and renal functions (1).

Recent studies have increasingly underscored the clinical significance of electrolyte imbalances. In particular, hyponatremia—a condition marked by low serum sodium levels—has emerged as an important prognostic indicator across a range of patient populations. A comprehensive meta-analysis demonstrated that individuals with hyponatremia might experience a 2.5- to 3.5-fold increased risk of mortality compared to those with normal sodium levels (2). This finding highlights the critical need for early detection and prompt management of hyponatremia, as it may significantly impact overall patient outcomes and guide future therapeutic strategies.

The frequent observation of hyponatremia in ICU patients is not merely a reflection of acute conditions but is also considered an indicator of their overall clinical status. Literature reviews have reported a positive correlation between hyponatremia and mortality, indicating that this condition prolongs ICU stay and delays recovery (3). However, most existing studies are single-center or based on limited patient populations, and comprehensive data on the global impact of hyponatremia remain scarce.

This large-scale retrospective study aims to demonstrate the significance of hyponatremia as a biomarker in determining the prognosis of ICU patients. The findings of this study are expected to provide guidance in monitoring electrolyte imbalances, developing early diagnosis and intervention strategies, and contributing to both clinical practice and future research.

Materials and Methods:

This retrospective cohort study was designed to examine the data of patients over 18 years of age who were admitted to ICUs in international centers between January 1, 2024, and January 1, 2025. The study was conducted using data obtained from hospitals' electronic health records, with the necessary ethical approvals.

Patient Population and Criteria:

Patients included in the study were those admitted to the ICU who were 18 years or older and had sufficient medical records. Exclusion criteria included an ICU stay of less than 24 hours, missing laboratory data, absence of serum sodium measurement at admission, and the presence of other critical electrolyte imbalances.

Data Collection Process:

Patient demographic information (age, gender), clinical characteristics, comorbidities (such as hypertension, diabetes, cardiovascular and renal diseases), admission laboratory results (particularly serum sodium levels), ICU length of stay, and mortality data were collected using standardized data collection forms. Hyponatremia was defined as a serum sodium level below 135 mEq/L at the time of admission.

Statistical Analysis Methods:

After assessing data distribution with the Shapiro-Wilk test, continuous variables were presented as mean ± standard deviation, and categorical variables as percentages and frequencies. Comparisons between hyponatremia and normonatremia groups were performed using t-tests for continuous data or Mann-Whitney U tests for non-parametric data, while chi-square tests were used for categorical data.

Initially, univariate analysis examined the effects of variables such as age, cardiovascular disease, renal failure, and hyponatremia on mortality, calculating odds ratios (OR) and 95% confidence intervals (CI) for each variable. These variables were then included in a multivariate logistic regression model to identify independent predictors of mortality.

A receiver operating characteristic (ROC) curve analysis was conducted to evaluate the predictive performance of serum sodium levels for mortality, determining the optimal threshold, sensitivity, and specificity parameters. Statistical analyses were performed using a reliable statistical software package such as SPSS (version X), with a p-value <0.05 considered statistically significant.

Results:

A total of 500 patients were included in the study. The average patient age was 64.8 ± 13.7 years, with 58% male and 42% female. The most common comorbidities were hypertension (45%), diabetes (30%), and cardiovascular diseases (25%). According to the defined criteria, hyponatremia was present in 25% of the patients. The observed mortality rate was 40% in the hyponatremia group, compared to 18% in the normonatremia group ($p<0.01$) (Table 1).

Univariate analysis revealed significant associations between mortality and variables such as age, cardiovascular disease, renal failure, and hyponatremia ($p<0.05$). Specifically, hyponatremia demonstrated a strong association with mortality in univariate analysis (OR=2.8, 95% CI: 1.9–4.1, $p<0.001$).

In multivariate logistic regression analysis, age, cardiovascular disease, renal failure, and hyponatremia were included in the model.

Even after controlling for other factors, hyponatremia remained an independent predictor of mortality (OR=2.3, 95% CI: 1.5–3.5, p<0.001). This finding supports the notion that hyponatremia independently increases mortality risk in ICU patients (Table 2).

ROC curve analysis assessed the predictive performance of serum sodium levels for mortality. The obtained area under the curve (AUC) was 0.74 (95% CI: 0.69–0.79). A threshold value of 132 mEq/L provided the best discrimination for mortality prediction, with a sensitivity of 78% and specificity of 65%. This result suggests that hyponatremia could be a crucial biomarker for assessing ICU patients' prognosis.

Furthermore, ICU stay was significantly prolonged in patients with hyponatremia, with an average stay of 9.5 days compared to 6.2 days in the normonatremia group (p<0.01). These findings indicate that hyponatremia not only increases mortality risk but also extends ICU length of stay.

In our cohort, patients with hyponatremia demonstrated markedly worse outcomes compared to those with normal sodium levels. Specifically, in-ICU mortality was significantly higher among the hyponatremia group, with 40.0% of patients succumbing compared to only 18.0% in the normonatremia cohort, corresponding to an odds ratio of 2.50 (95% CI: 1.40–4.40, p=0.001). Additionally, the median length of hospital stay was prolonged in hyponatremic patients (9.5 days [IQR, 6–17]) versus 6.2 days [IQR, 5–12] in their normonatremic counterparts (p=0.001).

Moreover, the requirement for mechanical ventilation was significantly greater in the hyponatremia group, affecting 20.0% of patients compared to 10.7% in the normonatremia group, with an associated odds ratio of 2.07 (95% CI: 1.18–3.62, p=0.001). The duration of mechanical ventilation was also longer among hyponatremic patients, with a median duration of 4 days [IQR, 2–9] compared to 2 days [IQR, 1–6] (p=0.05). Although the need for renal replacement therapy was higher in the hyponatremia group (12.0% vs. 4.8%, OR=2.72, 95% CI: 1.31–5.64), this difference did not reach statistical significance (p=0.14) (Table 4).

Table 1. Demographic, Clinical, and Laboratory Characteristics of the Study Population

Variable	Total (n=500)	Hyponatremia Group (n=125)	Normonatremia Group (n=375)	p-value
Age (mean ± SD)	68.5 ± 17.2	67 ± 16	69 ± 18	0.517
Gender (Male, %)	259 (53%)	81 (66%)	198 (50%)	**0.014***
Comorbidities				
Diabetes, n (%)	89 (17%)	25 (19%)	56 (16%)	0.536
Congestive heart failure, n (%)	50 (10%)	6 (5%)	51 (13%)	0.064
Coronary artery disease, n (%)	75(15%)	19 (15%)	55 (15%)	1.000
Cirrhosis, n (%)	5 (1%)	1 (1%)	4 (1%)	1.000
Active neoplasia, n (%)	30 (6%)	6 (7%)	24 (6%)	1.000
Current smoker, n (%)	40 (8%)	12 (10%)	29 (7%)	0.485
Chronic obstructive pulmonary disease (COPD), n (%)	48 (9%)	15 (12%)	34 (8%)	0.386
Respiratory insufficiency, n (%)	5 (1%)	1 (1%)	3 (1%)	0.538
Medications Used				
ACE inhibitors, n (%)	52 (11%)	16 (14%)	41 (9%)	0.214
ARBs, n (%)	50 (10%)	11 (9%)	37 (10%)	0.832
Loop diuretics, n (%)	30 (6%)	11 (10%)	13 (4%)	0.065
Thiazide diuretics, n (%)	40 (8%)	4 (3%)	37 (10%)	0.060
Vital Signs				
Body temperature (°C, mean ± SD)	38.6 ± 0.9	38.9 ± 0.9	38.4 ± 0.9	**<0.001***
Body temperature >38.5°C, n (%)	135 (50%)	55 (65%)	80 (47%)	0.010*
Systolic blood pressure (mmHg, mean ± SD)	148 ± 20	148 ± 18	149 ± 22	0.709
Diastolic blood pressure (mmHg, mean ± SD)	85 ± 11	83 ± 9	86 ± 12	0.110
Respiratory rate (breaths/min, mean ± SD)	29 ± 7	30 ± 7	28 ± 7	**0.034***
Clinical Outcomes				
Need for mechanical ventilation, n (%)	49 (9%)	21 (17%)	16 (6%)	**0.003***

Mortality, n (%)	60 (12%)	21 (18%)	36 (9%)	**0.042***

Table 2. Univariate and Multivariate Logistic Regression Analysis for Predicting Mortality

Variable	Univariate OR (95% CI)	p-value	Multivariate OR (95% CI)	p-value
Age (per 1-year increase)	1.03 (1.01–1.06)	0.008*	1.02 (1.00–1.05)	0.045*
Male gender	1.80 (1.10–2.95)	0.020*	1.50 (0.90–2.50)	0.120
Hyponatremia (<135 mmol/L)	2.50 (1.40–4.40)	0.002*	2.10 (1.10–3.90)	0.025*
Respiratory rate (per 1 increase)	1.05 (1.01–1.09)	0.015*	1.04 (1.00–1.08)	0.048*
Mechanical ventilation	5.20 (2.60–10.40)	<0.001*	4.50 (2.10–9.60)	<0.001*

Table 3. Distribution of Etiologies in Hyponatremia Patients

Etiology	Number of Patients (n)	Percentage (%)
SIADH (Inappropriate ADH secretion) due to:		
• Pneumonia	24	19.2
• Medications (e.g., SSRIs, carbamazepine)	8	6.4
• Subarachnoid hemorrhage	10	8.0
• Elective post-operative status	6	4.8
• Emergency post-operative status	10	8.0
• Severe sepsis	7	5.6
Trauma	25	20
Heart failure	10	8.0
Renal failure	10	8.0
Thiazide diuretic use	6	4.8
Liver cirrhosis	6	4.8
Hypothyroidism	2	1.6
Hypocortisolism	1	0.8
Total	125	100

Table 4. Comparison of Outcomes

Outcome measure	Hyponatremia Group	Normonatremia Group	OR	Statistical Test	p-Value
In-ICU mortality (number of patients/total)	50 (40.0%)	67.5(18.0%)	2.50 (1.40–4.40)	Mantel-Haenszel test	0.001
Length of hospital stay (median [IQR] in days)	9.5[6–17]	6.2[5–12]		Wilcoxon rank-sum test	0.001
Need for mechanical ventilation (number of patients/total)	25 (20.0%)	40 (10.7%)	2.07 (1.18–3.62)	Pearson chi-square test	0.001
Duration of mechanical ventilation (median [IQR] in days)	4 [2–9]	2 [1–6]		Wilcoxon rank-sum test	0.05
Need for renal replacement ther-	15 (12.0%)	18 (4.8%)	2.72 (1.31–5.64))	Pearson chi-square test	0.14

apy (RRT) (number of patients/total)					

Discussion:

Our study revealed a statistically significant relationship between the frequency of hyponatremia at ICU admission and mortality based on univariate and multivariate analyses. The fact that hyponatremia remained an independent predictor of mortality even after adjusting for critical factors such as age, cardiovascular disease, and renal failure suggests that this electrolyte imbalance directly affects patient prognosis rather than merely reflecting disease severity.

These findings align with previously reported studies on the hyponatremia-mortality relationship (4-5). Hyponatremia is thought to be associated with pathophysiological mechanisms such as neurohormonal activation, increased inflammatory responses, and impaired cardiovascular stability. This suggests that systemic imbalances in ICU patients are not limited to electrolyte disturbances but can have a broader impact on the overall disease course.

The 132 mEq/L threshold value identified by ROC curve analysis could be used in clinical practice for risk stratification and early intervention strategies. The high sensitivity and specificity of this threshold indicate that serum sodium levels can be a reliable biomarker for mortality prediction in ICU patients.

However, the retrospective nature of the study introduces certain limitations. Factors such as data incompleteness, measurement errors, and variations in patient management protocols across centers must be considered when interpreting the results. Additionally, the effects of diuretic use, fluid balance management, and other potential confounders were not fully controlled. Therefore, future prospective and randomized controlled studies are needed to further explore the mechanisms underlying the increased mortality associated with hyponatremia.

In conclusion, our findings suggest that close monitoring of electrolyte imbalances and early intervention, particularly in correcting hyponatremia, may play a potential role in reducing mortality risk in ICU settings. This study highlights the clinical importance of hyponatremia in ICUs and serves as a foundational step in developing hyponatremia management strategies for future research and clinical applications.

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Isolated Truncal Ataxia as the Sole Presentation of Suspected Vertebral Artery Dissection: A Diagnostic Challenge in the ED

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Introductıon: Truncal ataxia, a sign of midline cerebellar or brainstem dysfunction, poses a diagnostic challenge in emergency settings due to its broad differential diagnosis and potential to mimic benign conditions such as intoxication or peripheral vertigo. Posterior circulation strokes and vertebral artery dissections are particularly elusive, often evading initial imaging. This case illustrates a rare presentation of suspected vertebral artery dissection manifesting as isolated truncal ataxia, with unremarkable early neuroimaging, underscoring the need for clinical vigilance and serial imaging in the ED.

Case: A 64-year-old woman with no known comorbidities presented to the emergency department at 4 a.m. with acute-onset inability to maintain upright posture and pronounced nausea. She denied headache, limb weakness, vertigo, or visual disturbances. Her family reported flu-like symptoms for three days prior but no recent trauma or substance use.

On arrival, her vital signs were stable. Neurological examination revealed isolated truncal ataxia—manifested by inability to sit or stand unsupported. Cranial nerve examination was normal, with full extraocular movements, no facial asymmetry, and symmetrical limb strength. Reflexes were brisk and plantar responses were flexor bilaterally. Cerebellar limb testing (finger-nose and heel-shin) was unremarkable, further confirming isolated axial involvement.

Initial laboratory workup was unremarkable, with normal glucose, electrolytes, renal and hepatic panels. Cardiac biomarkers and ECG were within normal limits. Emergent non-contrast brain CT and diffusion-weighted MRI (DWI) revealed no acute pathology. Due to persistent ataxia, intravenous fluids, dimenhydrinate, and piracetam were administered, with partial symptomatic improvement observed over the following hours.

Given the absence of focal deficits or imaging findings, the patient was initially monitored conservatively. However, truncal ataxia persisted, prompting a second DWI-MRI and CT angiography of the head and neck approximately eight hours after the first scan. The control diffusion MRI again showed no infarct, but CT angiography revealed a double lumen image in the left vertebral artery, suggestive of fenestration or dissection.

Based on these findings, the neurology team admitted the patient for further monitoring and management. Antiplatelet therapy was initiated. On follow-up, the patient's postural stability improved, though gait ataxia persisted mildly.

Dıscussıon: This case highlights several critical teaching points for emergency physicians. First, truncal ataxia—while often attributed to benign causes such as vestibular dysfunction or drug intoxication—can be the sole manifestation of posterior circulation compromise, particularly cerebellar or brainstem ischemia.

In this patient, early imaging failed to detect pathology. This diagnostic latency is well-documented: posterior fossa strokes are frequently missed on initial CT scans due to beam-hardening artifacts and limited resolution in the infratentorial compartment. Even MRI with DWI can yield false negatives in the hyperacute phase, particularly within the first 6 hours, as demonstrated in recent studies.

Vertebral artery dissection (VAD), while rare, is an increasingly recognized etiology of posterior circulation ischemia in adults, especially in the absence of trauma. VAD may present with subtle signs including isolated ataxia, diplopia, or headache, or may be entirely asymptomatic until ischemic events ensue. In this case, the dissection was suspected only after a second imaging round and correlated clinically with isolated truncal ataxia—highlighting the essential role of repeat imaging in evolving neurologic presentations.

From a pathophysiologic standpoint, truncal ataxia arises from dysfunction of the cerebellar vermis and fastigial nuclei, which are primarily supplied by the posterior inferior cerebellar artery (PICA), a branch of the vertebral artery. A dissection in this territory, even without infarction, may cause transient ischemia or local hemodynamic disturbance sufficient to impair midline cerebellar function.

The case also exemplifies the limitations of relying solely on neuroimaging for diagnosis. A high index of suspicion—prompted by subtle clinical signs such as inability to sit upright or falling backward—is paramount. The patient's preserved limb coordination and absence of vertigo further argue against peripheral causes and strengthen the case for central involvement.

In conclusion, this report underscores the importance of recognizing isolated truncal ataxia as a potential harbinger of vertebral artery dissection and posterior circulation ischemia, even when initial imaging is inconclusive. Emergency physicians should maintain diagnostic vigilance in such presentations and pursue serial imaging when clinical symptoms persist. Incorporating truncal ataxia as a red flag in emergency stroke protocols may enhance early detection of posterior fossa pathology and improve patient outcomes.

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A retrospective study, analysis of Syrian patients under temporary protection presenting to the emergency department

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Introduction

The immigration problem, because of war and economic difficulties, stands out as a multidimensional social and economic issue worldwide. Following the Syrian civil war, millions of people were forced to leave their country and migrate to different regions, especially neighboring countries. As of 2024, Turkey is hosting the most significant number of Syrians worldwide. People from Syria who live in Turkey are under the "Temporary Protection" status, and these individuals can benefit from free healthcare services. In this context, the number of Syrians under temporary protection has been recorded as 2,901,478, and 114,599 reside in the province of Izmir [1].

These people have problems with basic shelter, nutrition, hygiene, and access to health services. In terms of health services, it is known that emergency services are the units that these people use most intensively [2-4]. This is due mainly to the lack of access to primary health services [5-7]. This situation has led to a significant increase in emergency service application rates. In addition, it creates additional costs for the health system and causes difficulties in operation. This study aims to fill the gap in knowledge in this

area by examining the diagnoses, demographic data, and outcomes of Syrian patients who applied to the Emergency Department of İzmir Tepecik Education and Research Hospital.

Materials and methods

This study was conducted retrospectively in a tertiary ED. The hospital where the study was performed has an average of 15000-18000 patients admitted to the ED monthly. Ethical approval was received from a local ethics committee (Decision number: 2024/07-09 Date: 19/08/2024). The tenets of the Declaration of Helsinki performed the study.

Population Study

Patient information was obtained through the Hospital Information Management System (HIMS). The study enrolled all patients who visited the ED between October 01, 2023, and April 01, 2024.

Patient selection

Inclusion criteria

In our hospital, Syrian patients with temporary protection status' social security are selected as "Syrian Patients." Therefore, patients whose social security coverage was selected as "Syrian Patients" in their emergency department applications and registered were included in the study.

Exclusion criteria

Patients who re-applied with similar complaints within 48 hours were excluded from the study.

Data collection and standards

Data on patients admitted to the hospital were collected retrospectively through the HIMS. The collected information was grouped as follows:

Patients' age, gender, application day and time information, application methods, and reasons.

Diagnoses received in the emergency department [grouped according to ICD-10 (International Classification of Diseases) diagnosis codes].

Length of stay in the emergency department

Consultation requirements

Emergency department outcome information (discharge, hospitalization, referral, or death)

If hospitalization occurred, in-hospital outcome information (discharge, referral, or death)

Statistical analysis:

Statistical analyses were performed using the JAMOVI v. 2.3.28 software package (The Jamovi Project, Sydney, Australia). The Kolmogorov-Smirnov test was used to check the normality of data distribution. Categorical data were presented as frequency and percentages; numerical data were presented as mean and standard deviation if normally distributed, and median and interquartile range (IQR) values otherwise.

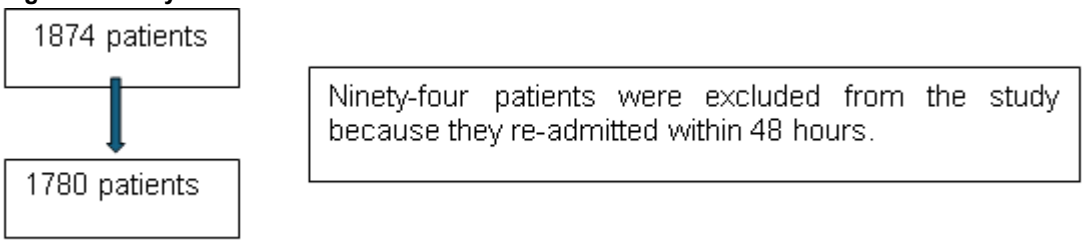
Pearson's chi-square test will be used to test whether the differences between pairwise dependent groups are significant. Fisher's exact test will be used when a table has a cell with an expected frequency of less than 5.

Mann-Whitney U test was used to compare two groups regarding non-normally distributed data. All analyses will be performed at a 95% confidence level, and p-value <0.05 will be considered statistically significant.

Results

A total of 1874 patients were enrolled in the study that matched the inclusion criteria. Subsequently, 94 patients who met the exclusion criteria were excluded from the study **(Figure 1).**

Figure 1: Study Flow Chart



1780 patients were included in the study. 53.1% (n=946) of the patients were male and the median age was 31 (IQR:24-42) years. 57.1% (n=1017) of the patients applied to the emergency department between 16:00 and 08:00, 70% (n=1246) on weekdays, and 97.7% (n=1739) as self-presentation **(Table 1).**

The most common presenting complaint was symptoms of upper respiratory tract disease. (n=480, 27%) **(Table 2).**

There were 1263 (71%) patients who were followed in the emergency department for less than 1 hour **(Table 3).**

We observed that patients arriving by ambulance and patients for whom consultation was requested stayed in the emergency department for a statistically significant longer time (p<0.001).

No consultation was requested in 1562 patients (87.8%) applying to the emergency department. The rate of consultation requests from patients arriving by ambulance was statistically significantly higher than from self-presentation (p<0.001).

The most frequently requested consultation was ophthalmology (n=67, 30.7%). Of all patients for whom consultation was requested, 52 (23.9%) were hospitalized. This rate was 32.5% (n=49) for consultations requested from branches other than ophthalmology.

1700 patients (95.5%) were discharged from the emergency department, while 52 patients (2.9%) were admitted to hospital. **(Table 4).**

Table 1: Analysis of emergency department application times and types

	Number(n)	Percentage (%)
Application time		
08:00-16:00	763	42.9%
16:00-08:00	1017	57.1%
Application day		
Mid-week	1246	70%
Weekend	534	30%
How to apply		
Self-presentation	1739	97.7%
Ambulance-arrival	41	2.3%

Table 2: Reasons for application of patients

Reason for Application	Number(n)	Percentage (%)
Symptoms of upper respiratory tract disease	480	27.0%
Trauma	299	16.8%
Gastroenterological symptoms	250	14.0%
Musculoskeletal symptoms	250	14.0%
Symptoms associated with the urinary system	111	6.2%
Eye-related symptoms	90	5.1%
Neurological symptoms	86	4.8%
Cardiological symptoms	86	4.8%
Dermatological symptoms	82	4.6%
Other symptoms	46	2.7%
Total	**1780**	**100%**

Table 3: Length of stay of patients in the emergency department

Length of stay	Number (n)	Percentage (%)
Less than an hour	1263	71%
One to four hours	486	27.3%
More than four hours	31	1.7%
Total	**1780**	**100%**

Patient diagnoses were recorded in accordance with the ICD-10 diagnosis coding system. The most common diagnosis group was diseases of the musculoskeletal system and connective tissue (28.7%), entered with the code "M" **(Table 5).**

Discussion

In our study, the most common presenting complaints of Syrian patients were upper respiratory tract disease, trauma, and gastroenterological symptoms. We observed similar findings in many studies [4,8,9].

In a study, the most common reason for admission was found to be gunshot wounds [10]. This difference may be because the province where the study was conducted is on the Syrian border, the camps are not used effectively, and the war has decreased in intensity over the years.

However, compared to the findings of Gulacti et al. (2017) [4], the rate of trauma cases was higher in our study. This difference may be because the war was intense at the time Gulacti et al. conducted the study, and people were mostly living in isolated camps.

Also, the fact that Syrian people today live in settlements rather than in isolated camps may have increased their exposure to minor/major traumas.

Table 4: Outcomes of patients in the emergency department and during hospitalization

Patient outcomes	Number (n)	Percentage (%)
Discharged from the emergency department	1700	95.6%
Hospitalization	52	2.9%
Discharged from hospital	46	
Transfer to another hospital	3	
Death in hospital	3	
Leaving the emergency department with a treatment refusal signature	24	1.3%
Transfer from the emergency department to another hospital	4	0.2%
Total	**1780**	**100%**

Table 5: Patients' diagnosis code groups

Patients' diagnosis code groups	Number(n)	Percentage (%)
M: Diseases of the musculoskeletal system and connective tissue	510	28,7%
J: Diseases of the respiratory system	454	25.5%
R: Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified	399	22.4%
N: Diseases of the genitourinary system	115	6.5%
S and T: Injury, poisoning and certain other consequences of external causes	92	5.2%
L: Diseases of the skin and subcutaneous tissue	59	3.3%
K: Diseases of the digestive system	57	3.2%
H: Diseases of the eye and adnexa	28	1.6%
I: Diseases of the circulatory system	17	0.9%
Y: External causes of morbidity and mortality	17	0.9%
Other diagnosis	32	1.8%
Total	**1780**	**100%**

Some studies emphasize the economic burden of refugees on health systems [4,8,11]. In 2022, total health expenditures in Turkey were recorded at 606 billion 835 million TL [12]. The impact of health services for Syrian patients on these total expenditures is significant. This situation reveals the need for more international cooperation regarding the financing and accessibility of health services. In addition, the use of primary health services by Syrian patients can effectively reduce costs.

In a study conducted in a tertiary hospital like ours (earlier than the arrival of Syrian people in our country), the average length of stay of patients in the emergency department was observed to be two hours. Again, it was found that 12.5% of patients were hospitalized [13]. We found that the average length of stay of Syrian patients in the emergency department was shorter, and they were hospitalized at a much lower rate.

As in our study, studies show that the rate of consultation requests is significantly higher in patients who apply by ambulance and that the length of stay in the emergency department increases when consultation is requested [14]. This finding may indicate that Syrian patients use ambulances for more serious reasons.

In the study by Donmez et al., the hospitalization rate of patients who requested consultation was 27.9% [15]. Our study is also

consistent with the literature in this respect. The consultation threshold was used appropriately, resulting in a high hospitalization rate in that population.

In our study, the rate of discharge of Syrian patients after applying to the emergency department was 95.6%. Gulacti et al. (2017) [4] also found high early discharge rates in their study. This indicates that Syrian patients prefer hospital emergency departments to primary healthcare services institutions.

In another study done between 2012 and 2016 years, the hospitalization rate of Syrian patients was determined as 46.8% [10]. This difference may be because, today, Syrian patients use emergency services with non-urgent complaints rather than serious complaints. This is another indicator that patients do not use primary healthcare services effectively.

Conclusion

This study evaluates the reasons for Syrian patients' emergency room visits, after-application processes, and their impact on the healthcare system. Our findings show that Syrian patients often visit the emergency room with non-urgent complaints and have high discharge rates. This situation further increases the existing pressure on the functioning of emergency rooms.

Redirecting Syrian patients to primary health services, increasing the accessibility of these services, and educating patients on this issue is important in reducing the burden on emergency services and ensuring more effective use of health services. After these are done, in case of applications with non-urgent complaints, solutions such as not covering the service fee, billing the service recipient, etc., may be applicable. In addition, policies that will facilitate the integration of Syrian patients into health services, as well as international cooperation and support, will make significant contributions to the sustainability of the health system. This study is consistent with similar findings in the literature. The study is intended to be a guide for the future of emergency health services provided not only to Syrian patients but to all patients.

As a result, primary health care services should be improved when planning health services, and patients under temporary protection status should be encouraged in primary health care units.

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Acute Lower Extremity Ischemia at an Early Age: A Rare Case Report

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Introduction

Peripheral arterial disease (PAD) is a chronic and progressive vascular disease that usually affects the arteries of the lower extremi-

ties and occurs as a result of systemic atherosclerosis. The disease causes arterial lumen narrowing or occlusion, preventing adequate blood flow to the tissues. Its clinical spectrum ranges from asymptomatic forms to critical limb ischemia (CLI), such as intermittent claudication (claudication), rest pain, and progressive ischemic ulcers or gangrene [1].

The prevalence of PAD increases with age, reaching 20% in individuals over the age of 70 [2,3]. However, PAD cases seen in the young age group are rare and are mostly associated with important risk factors such as smoking, morbid obesity, diabetes, hyperlipidemia, and familial predisposition. PAD seen in the young age group often causes a delay in diagnosis because vascular pathologies are not among the first causes considered in this age group [4].

Acute extremity ischemia is a clinical condition in which arterial circulation is suddenly disrupted, requiring urgent intervention and racing against time. This condition is often caused by embolism, thrombosis, or traumatic arterial injuries. Rapid clinical evaluation, use of accurate imaging methods, and a multidisciplinary approach are of great importance in the diagnosis and management of acute ischemic conditions [1,3,5]. In this presentation, a case of acute lower extremity ischemia in a 35-year-old male patient with no known atherosclerotic disease but with significant risk factors is presented, emphasizing that PAH can develop at a young age and the diagnosis and treatment process of this clinical condition is discussed.

Case

A 35-year-old male patient presented to the emergency department with a complaint of sudden onset and severe pain in the left lower extremity. It was learned that the patient had been experiencing pain in his left leg while walking for 5–6 months, but his symptoms subsided with a short rest. It was stated that the pain started to occur during rest in the last few days.

It was learned that the patient, who had no known chronic disease in his medical history, smoked approximately one pack of cigarettes per day and had been a smoker for over 15 years. In his physical examination, the patient's body mass index (BMI) was calculated as 43.3. The left leg was cold and pale, and the femoral, popliteal and distal pulses were not palpable. The ankle-brachial index (ABI) was measured as 0.5 for the left lower extremity. Laboratory tests performed in the emergency department revealed a blood sugar level of 350 mg/dL. Other parameters were normal. There was no known diabetes diagnosis.

Imaging Findings

Arterial Doppler ultrasonography showed monophasic flow in the left common femoral and superficial femoral arteries. Echogenic thrombus materials were observed in the lumens of the left popliteal artery, tibialis anterior, tibialis posterior and dorsalis pedis arteries, and flow could not be detected in these vessels with color Doppler.

In the computed tomography (CT) angiography taken to support the diagnosis, it was determined that the flow in the left superficial femoral artery was not observed from the distal and that there was an occlusion.

Clinical Evaluation and Intervention

According to these findings, acute arterial embolism and/or thrombosis was considered in the patient. The patient, who was evaluated by the cardiovascular surgeon, was taken to the operating room for an emergency interventional procedure. Vascular patency was achieved by intervening in the area of occlusion with endovascular intervention. The patient was monitored in the postoperative period and antithrombotic treatment was started.

Discussion

PAH is usually seen in elderly individuals, but it can also be seen in young patients in some cases. These patients often have serious underlying risk factors [2]. In the presented case, smoking, morbid obesity, and probably uncontrolled hyperglycemia caused the disease to occur at an early age.ABI measurement is very important in peripheral circulation assessment with its bedside applicability and non-invasive structure [6]. In this patient, the ABI value also supported severe ischemia. Arterial Doppler and CT angiography were guiding both in confirming the diagnosis and in the intervention plan.

In the presented case, the long-standing history of claudication indicates the presence of chronic peripheral artery disease that had previously developed but not been diagnosed. However, the increase in the severity of the patient's pain shortly before the emergency admission, the onset of rest pain, and the emergence of sudden circulatory disorder findings indicate that an acute thrombotic or embolic event was added to this underlying chronic picture.

Therefore, the case should be evaluated as acute-on-chronic extremity ischemia. This condition, especially when seen at a young age, makes diagnosis difficult and can often lead to delayed interventions. It is of great importance for physicians in the emergency department to approach this diagnosis without excluding it, especially in young individuals with significant risk factors (smoking, obesity, latent diabetes, etc.).

Conclusion

Although PAH is rare at a young age, there should be no delay in diagnosis in individuals with risk factors. This case shows that serious vascular occlusion is possible at an early age and that careful evaluation in the emergency department and rapid diagnosis and treatment are of vital importance. Early-noticed PAH is a condition that can prevent limb loss with timely intervention. In particular, conditions accompanied by acute arterial occlusion due to underlying chronic vascular diseases should be defined as “acute ischemic events on a chronic basis” and should be intervened rapidly. Recognition of such conditions allows for the timely initiation of limb-saving interventions in the emergency department.

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4940

Evaluatıon Of Trauma Severıty In Electrıc Bıcycle Injurıes

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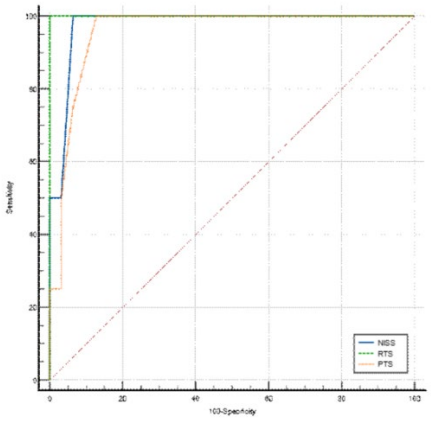
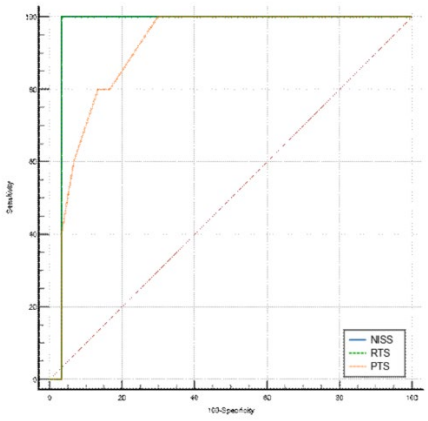
Introduction and Purpose:In our study, we aimed to determine the demographic data of the patients who visited the emergency department with electric bicycle (EB) injuries and evaluated the severity of trauma by using anatomical and physiological trauma scoring systems[New Injury Severity Score(NISS),Revised Trauma Score(RTS) and Pediatric Trauma score(PTS)].
Materials and Methods:This prospective, descriptive, and single-center study was conducted on 106 patients with EB injuries who visited the Kayseri City Hospital emergency department between September 15, 2023,and December 15, 2023.Written consent was obtained from the patient and their relatives.ROC(receiver operator characteristic) analyses were performed on NISS,RTS and PTS scores.
Results and Conclusion:The median age of the patients was 21.5(15-39.5) years, and 69.8%(n=74) were male.While 12.8%(n=13) of the patients were admitted to the ICU,6.6%(n=7) were found to have died.A statistically significant difference was detected between NISS,RTS, and PTS scores and ICU admission(p values=<0.001, <0.001, and 0.002, respectively).A statistically significant difference was found between NISS, RTS, and PTS scores and mortality(p values =<0.001,<0.001, and 0.002, respectively).The NISS score(AUC= 0.982) had the highest discriminatory ability in predicting ICU admission(p < 0.001).When the NISS score was greater than 13, its sensitivity in predicting ICU admission was 100%,specificity was 97.9%, positive predictive value was 86.7%, negative predictive value was 100%, positive likelihood ratio was 46.5, and negative likelihood ratio was 0.0.The RTS score(AUC= 0.996)demonstrated the highest discriminatory ability in predicting mortality(p< 0.001).When the RTS score was ≤10, its sensitivity in predicting mortality was 100%, specificity was 93.9%, positive predictive value was 53.8%,negative predictive value was 100%,positive likelihood ratio was 16.5,and negative likelihood ratio was 0.0.In our study,NISS,RTS and PTS scores all had very high discriminatory ability in predicting ICU admission and mortality in patients presenting with EB injuries.Its sensitivity and negative predictive value were 100%, particularly in predicting ICU admission when the NISS score was greater than 13 and in predicting mortality when the RTS score was equal to or less than 10.
Discussion:In our study, NISS,RTS and PTS scores all had very high predictive ability in predicting ICU admission and deaths associated with the development of EB.In particular, when the NISS score was over 13, the value of richness and negative vision in predicting admission to the intensive care unit was 100%, and when the RTS score was 10 and below, death was found to be 100%.

Comparison of ROC curves of scores for mortality



Comparison of ROC curves of scores for ICU admission



Distribution of injury sites in patients presenting with electric bicycle injuries		
Injury area	n	%
Head	54	50,9
Neck	3	2,8
Anterior thorax	10	9,4
Posterior thorax	8	7,5
Abdomen	11	10,4
Shoulder	24	22,6
Arm	6	5,7
Forearm	28	26,4
Wrist	10	9,4
Hand	20	18,9
Pelvis	12	11,3
Thigh	10	9,4
Knee	37	34,9
Leg	8	7,5
Ankle	14	13,2
Foot	10	9,4
Vertebra	4	3,8

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Imaging Techniques Preferred in Patients Presenting to the Emergency Department with Renal Colic: Which Technique for Which Patient

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Introduction and Purpose: Objective: Renal colic is one of the frequently encountered acute conditions in the emergency department. In most cases, pain is alleviated with simple analgesics, but in some instances, even the strongest painkillers may not be effective. Although patients sometimes present with additional complaints such as macroscopic hematuria, decreased urine output, fever, nausea, and vomiting, in other cases, the only symptom may be flank pain. The aim of this study is to determine when and which imaging techniques should be used in these patients.

Materials and Methods: Method: A total of 132 patients who presented to the emergency department with renal colic between January 2025 and February 2025 were retrospectively evaluated. The patients' presenting complaints, laboratory techniques, imaging methods, and treatment procedures were recorded.

Results and Conclusion: Results: All 132 patients presented with flank pain, 6 of whom had macroscopic hematuria, 101 had microscopic hematuria, 12 had microscopic hematuria with fever, 8 had fever along with nausea and vomiting, and 5 patients had isolated flank pain. A total of 64 patients had no imaging performed, all of whom had a history of prior stone passage. 41 patients had a radiograph (KUB), 27 had an ultrasound (USG), and 23 underwent spiral CT. It was observed that 5 patients were pregnant, and all of them underwent ultrasound.Conclusion: Asking whether patients with renal colic have a history of prior stone passage and referring them to urology after pain relief helps protect patients from unnecessary imaging and radiation. Additionally, for stones that cannot be visualized on KUB and do not cause microscopic hematuria, we believe that performing an ultrasound (USG) or, if necessary, a spiral CT scan would be beneficial to clarify the cause of pain.

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Basilar Artery Thrombosis: Management of a Challenging Cerebrovascular Crisis

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Introductıon

Ischemic stroke is a significant clinical condition within cerebrovascular diseases, resulting in neuronal damage due to impaired cerebral oxygen and glucose supply. **Basilar artery occlusion (BAO) represents a severe and life-threatening stroke subtype**, as it compromises the perfusion of the brainstem, a region responsible for essential motor, sensory, and autonomic functions [1].

Given its high morbidity and mortality, **timely diagnosis and appropriate intervention are critical** for optimizing patient outcomes. However, its relatively low incidence and variable clinical presentation pose diagnostic and therapeutic challenges. This case report presents a patient diagnosed with **basilar artery thrombosis**, highlighting the diagnostic approach, therapeutic strategies, and challenges encountered in the management of this cerebrovascular emergency.

Case

A 56-year-old female patient presented to the emergency department with complaints of dizziness, lethargy, weakness, and vomiting. On physical examination, no significant motor weakness or sensory deficits were detected in the extremities; however, the patient exhibited difficulty walking. She had a known history of hypertension, and her initial blood pressure was 240/140 mmHg. As part of the initial management, a nicardipine infusion was initiated to achieve blood pressure control.

A non-contrast brain CT scan revealed no evidence of intracranial hemorrhage. Diffusion-weighted MRI demonstrated acute diffusion restriction at the region of brainstem (figure 1), and CT angiography of the brain identified a thrombus in the basilar artery. However, due to the time of presentation, intravenous thrombolytic therapy was contraindicated. The patient was promptly referred for endovascular intervention, but mechanical thrombectomy could not be successfully performed due to chronic arterial stenosis.

During the procedure, heparin was administered, and post-procedural management included 100 mg of aspirin (ASA), 75 mg of clopidogrel, and 60 mg of enoxaparin twice daily. The patient was subsequently admitted to the neurology stroke unit for further medical management and monitoring. After 11 days of medical treatment, the patient remained asymptomatic and was discharged with a recommendation for outpatient follow-up.

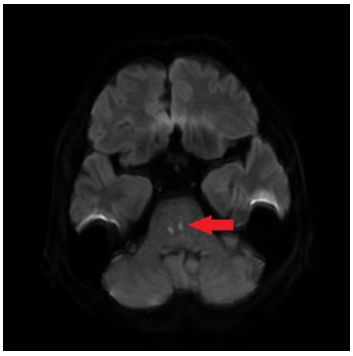

Figure 2: DWI- MRI demonstrated diffusion restriction (red arrow)

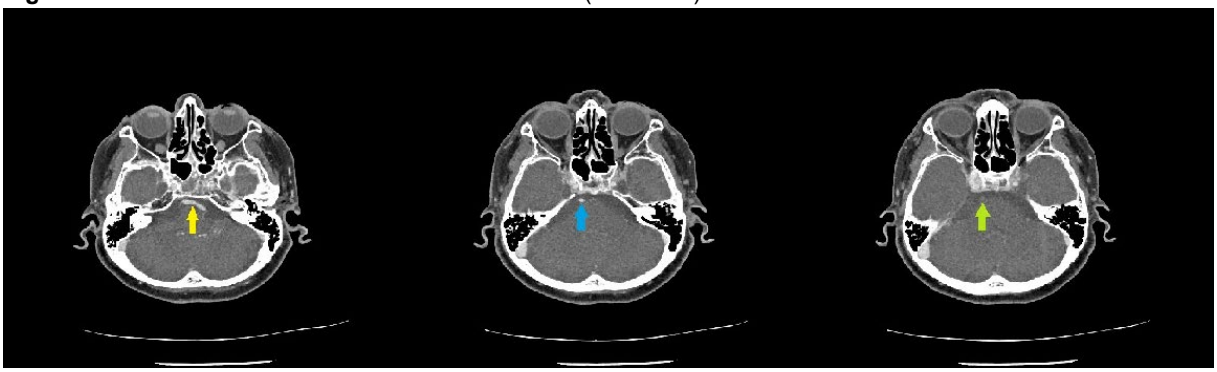

Figure 2: Contrast-enhanced imaging demonstrating persistent basilar artery flow (yellow and blue arrows) and the occluded basilar artery segment following thrombus formation (green).

Discussion

Basilar artery occlusion is a medical emergency requiring immediate intervention, as early diagnosis and treatment can be lifesaving. The most critical factors determining the course of management include the timely administration of thrombolytic therapy and endovascular intervention. In this case, intravenous thrombolysis was contraindicated due to the time of presentation, and mechanical thrombectomy was attempted. However, thrombectomy was unsuccessful due to chronic arterial stenosis. Despite this, the administration of heparin during the procedure and subsequent initiation of aspirin (ASA), clopidogrel, and enoxaparin had a beneficial impact on the patient's clinical course.

A key consideration in the management of basilar artery occlusion is the rapid assessment of eligibility for thrombolytic therapy, as prompt decision-making can significantly influence outcomes [2]. Numerous studies in the literature have evaluated the efficacy of thrombolysis and thrombectomy in basilar artery occlusion, demonstrating that treatment success varies depending on factors such as the patient's overall condition, the degree of arterial stenosis, and thrombus characteristics [4,5].

Time is a critical determinant in the management of basilar artery occlusion, and treatment should be initiated as early as possible [3]. In some cases, patients may be ineligible for thrombolytic therapy, necessitating alternative treatment strategies. As demonstrated in this case, structural abnormalities such as chronic arterial stenosis can lead to thrombectomy failure [5]. However, this does not indicate the end of therapeutic options. Optimized medical management remains crucial, allowing for the development of individ-

ualized treatment plans that can improve survival and clinical outcomes.

Conclusion

Basilar artery occlusion is a critical cerebrovascular condition that requires early diagnosis and intervention. Thrombolysis, thrombectomy, and anticoagulation are potential life-saving strategies; however, individualized patient evaluation is essential. A multidisciplinary approach and timely intervention play a pivotal role in improving survival and overall prognosis.

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Potentially Fatal Teleradiology Error in Tomography Reporting – A Case Presentation of Pulmonary Embolism

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Introductıon

In the present day, technological advancements and easy access to technological tools in healthcare facilities, along with the increasing integration of the internet and technology, have ushered in a higher quality of healthcare services. These developments have introduced various new applications into our lives (1).

Telemedicine refers to the provision of healthcare services through digital technology when the physician and the patient are physically distant from each other (2). The term "tele" has Latin origins, meaning distant, far away, or at a certain distance (3). The World Health Organization (WHO) defines telemedicine as the remote use of information and communication technologies for diagnosis and treatment (4). Telemedicine can be utilized in various fields such as teleradiology, teleconsultation, telesurgery, telegeriatrics, telehomecare, teledermatology, telepsychiatry, telepathology, and telerehabilitation, among many others (5,6).

Imaging techniques are an indispensable tool in modern medicine. One of the significant challenges in this field is the increasing number of diagnostic examinations. Consequently, radiologists find it difficult to allocate sufficient time for analyses. In our country, the number of radiologists is proportionally decreasing over time.

This decline persists despite the rise in annual imaging procedures. Given the current conditions, it can be asserted that teleradiology is the most effective method to cope with this problem. Tele-radiology enables radiologists to provide services without being physically present where the patient is, contributing to the development and continuity of healthcare services (7).

Pulmonary embolism (PE) is a frequently encountered and high-mortality condition in emergency clinics. The mortality rate is notably high, around 12%, not only in ambulatory patients but also in hospitalized individuals. Diagnosing PE may not always be straightforward. Challenges in obtaining computed tomography angiography (CTA), acknowledged as the gold standard for diagnosis, due to renal dysfunction and pregnancy, stand out as major difficulties in the diagnostic process (8).

One of these challenges is obtaining the report of the CT scan performed for emergency department (ED) physicians. The reporting process, at times, can take hours, leaving emergency physicians to manage cases without the radiology report. Consequently, emergency medicine physicians are able to accurately recognize findings indicative of PE in CTA images with high precision. Additionally, albeit rare, mismatches between clinically anticipated CT reports and erroneously reported results can occur, adding to the complexity of the situation (8).

Here, our aim is to present a case where pulmonary embolism was detected in CT angiography images, despite being reported as normal in the teleradiology report.

Case Presentatıon

A 70-year-old female patient presented to an external center with complaints of dyspnea. After evaluation there, she was referred to our ED for further imaging needs. Upon arrival, the patient had a Glasgow Coma Scale score of 15, and her neurological examination was unremarkable. She had no tachypnea, and there was no desaturation in room air. Vital signs were within normal limits, and she had no fever. The electrocardiogram (ECG) showed a normal sinus rhythm with no arrhythmias, ST-T changes, S1Q3T3 pattern, or bundle branch block. D-dimer was elevated at 2.77 (<0.55). Other tests (complete blood count, biochemistry, blood gas, and cardiac markers) were within normal ranges. Contrast-enhanced CT pulmonary angiography (CTA) revealed a filling defect consistent with emboli in the distal branch of the right main pulmonary artery (Image). Surprisingly, the teleradiology report provided via telemedicine reported the CT image as normal. The patient was admitted to the Chest Diseases inpatient service for further follow-up and treatment.

Dıscussıon

The Turkish Medical Association's website highlights some potential negative consequences of the "service procurement" method actively applied in the field of radiodiagnostics in Turkey (9). Under the heading "Effects on the Number and Process of Reporting," it is stated: "Another duty of radiologists is to dedicate the necessary time after appropriate imaging is performed to detect pathology in the images and contribute to the diagnostic process by creating an appropriate differential diagnosis list. However, in many hospitals in different cities, hundreds of films taken in a short period are pooled together, and distribution is made from this pool. In this

system where payment is made based on the number of examinations, the situation of reporting hundreds of examinations in a short time arises. The number of reports written in a short time is well above the reporting reading standards recommended by international guidelines. This situation can lead to the oversight of existing pathology" (9).
Furthermore, in this article, under the heading "On the Nature of Teleradiology," it is mentioned: "In societal emergencies and disaster situations such as the COVID-19 pandemic and earthquakes, remote reporting can be an option. However, performing routine healthcare services and radiological imaging through these companies disrupts the communication that is crucial between clinicians and radiologists. This communication gap can lead to missed diagnoses and unnecessary examinations, resulting in increased radiation exposure. Additionally, diagnostic monitors in hospital radiology units are screens specifically designed for this purpose from a technical standpoint and differ significantly from commercial monitors in the market. It is questionable whether the technical infrastructure used in the home environment for remote reading of reports utilizes this specialized equipment. This could be a factor affecting the diagnostic processes" (9).

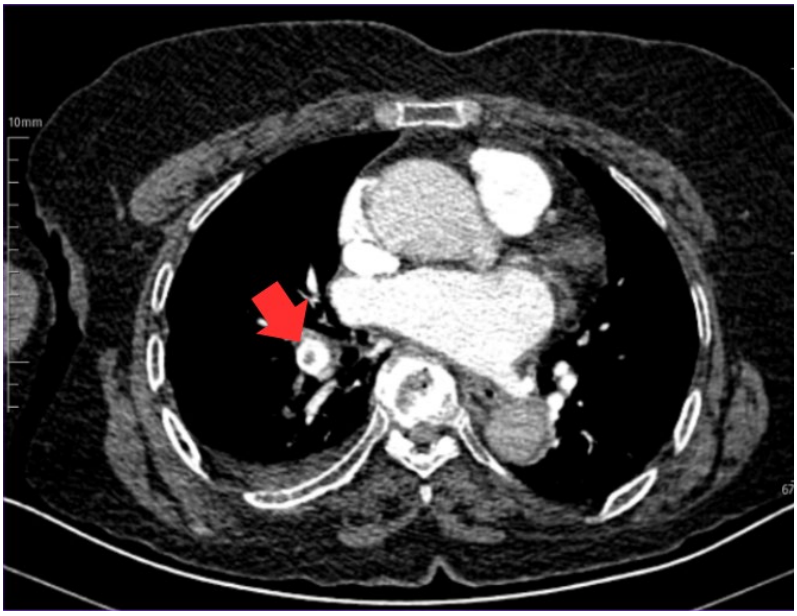

Image. Red arrow - Filling defect (thrombus) observed in the distal left pulmonary artery in the acquired CT angiography (CTA).

In the same article, under the heading "Effects on Qualified Reporting," it is stated: "Rapid reporting of a large number of patients in a short time leads to omissions in diagnoses and superficial descriptions of pathologies that require detailed characterization. This situation can result in the emergence of reports lacking in quality, leading to erroneous clinical management. Frequently, it necessitates obtaining a second opinion from in-house radiologists and/or repeating the same examination a second time due to suboptimal imaging, resulting in an increase in radiation dose. Additionally, the distance and communication gap between the radiologist and the report author prevent the second checks that the radiologist should perform after writing the report. This, in turn, can lead to erroneous reporting, causing serious problems in clinical workflow" (9).
In 2021, the Turkish Society of Radiology prepared a "TeleRadiology Guide" (10). Telemedicine is defined in this guide as applications where healthcare services are remotely conducted through information technologies independently of location. TeleRadiology, as a subset of Telemedicine, is described as applications that allow the transfer, storage, processing, evaluation, and reporting of radiological images and related information from the location where they are obtained to a different location digitally. However, legal responsibility regarding tele-radiology reports is not discussed in this guide.
Clinical manifestations of PE are nonspecific. The most common symptom is chest pain, often abrupt and pleuritic in nature, more commonly seen in distal pulmonary artery embolisms. Central pulmonary artery embolisms, on the other hand, may cause pressure and pain in the anterior chest, warranting differential diagnosis from acute coronary syndrome and aortic dissection. Shortness of breath is the second most frequent complaint, and a sudden increase in pre-existing dyspnea along with worsening oxygenation in individuals with known cardiopulmonary disease should raise suspicion of PE. In cases of large emboli, severe shortness of breath may be accompanied by a fear of death. Arterial blood gas (ABG) analysis may reveal hypocapnia, respiratory alkalosis, and hypoxia, although ABG can be normal in 40% of cases. In our case, the blood gas values were within normal ranges. Chest X-ray may be required to differentiate from other conditions, but in our case, a chest X-ray was not performed. ECG findings may include signs of right ventricular strain, such as T-wave inversion in leads V1-V4, S1Q3T3 pattern, and sinus tachycardia. In our case, the ECG was normal.
Most cases are brought to the emergency clinic after experiencing syncope. In the majority of these cases, when they are brought to the emergency clinic, blood pressure is normal or systolic blood pressure is not less than 90 mmHg. In our case, the patient presented to the outpatient clinic and had normal vital signs with no evidence of hypotension.

D-dimer, a fibrin degradation product, is often the initial test sought for many patients suspected of PE. In our case, D-dimer was requested, and the result came back elevated.

In the diagnosis of PE, CTA is now widely accepted as the gold standard. The resolution of new-generation multidetector CTA devices is excellent, capable of revealing even small thrombi that were difficult to visualize with earlier devices. Findings such as filling defects or signs of vascular narrowing can directly or indirectly indicate the presence of a thrombus. A negative CTA is crucial evidence for excluding the diagnosis of PE. In our case, a CTA was requested, and the evaluation was performed by emergency physicians before the radiology report was available. As a result of this assessment, a thrombus was detected in the distal right main pulmonary artery, and the patient was referred to the Pulmonary Diseases department. The patient was admitted to the Pulmonary Diseases Service for diagnosis and treatment. The teleradiology report received through the system, however, was reported as normal.

The error in the teleradiology report is not an uncommon situation for emergency physicians. Although the experience of emergency physicians often leads to resolving such situations without harm to patients and without clinical delays, this circumstance opens up discussions about the accuracy and legal responsibility levels of teleradiology reports.

During our literature and internet data review for this case presentation, we were unable to find any information, document, or source regarding the legal status of teleradiology reports.

Conclusıon

We believe that advanced clinical studies are needed to determine the accuracy of teleradiology reports. This way, accuracy percentages of teleradiology reports can be revealed, and a transition to a more patient-centric teleradiology reporting system can be facilitated. Additionally, there is a need for legal and administrative studies that clearly outline the legal aspects of teleradiology reports.

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Sılent Danger: The Hıdden Rısks Of Hypocalcemıa In Elderly Patıents Undergoıng Colonoscopy Preparatıon

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Karamanoglu Mehmetbey University

Introductıon

Hypocalcemia is a potentially life-threatening condition that can present with a wide spectrum of clinical manifestations, ranging from mild paresthesia to severe tetany and cardiac arrhythmias. Hypocalcemia is defined as a serum calcium level below the normal range(1). Common causes include hypoparathyroidism, vitamin D deficiency, chronic kidney disease, and certain medications(2). Symptoms can include neuromuscular irritability, paresthesia, muscle cramps, tetany, and even seizures(3). Timely diagnosis and correction are crucial to prevent complications We report a case of a 78-year-old woman presenting with symptomatic hypocalcemia due to 3 day restriction to undergo colonoscopy. This case highlights the importance of prompt recognition and appropriate management of hypocalcemia in elderly patients.

Case Report

The 78-year-old female patient presented to our service with complaints of muscle spasms in the fingers for approximately 2-3 hours, with all fingers in a flexed position and unable to extend. She had no known comorbidities. According to the history obtained, it was noted that a colonoscopy was scheduled for the afternoon of the same day, and that the patient had been on a liquid diet for the past 3 days due to this. Blood samples were taken.In Biochemistry, Glucose: 75 mg/dL Urea:53 mg/dL Cre: 1.32 mg/dL AST: 25 U/L , ALT : 20 U/L Albumin: 3,5 g/L Calcium: 6.5 mg/dL (normal range 8.5-10.5 mg/dL) Total Bilirubin: 1.0 mg/dL Direct Bilirubin: 0.2 mg/dL Indirect Bilirubin: 0.8 mg/dL CK (Creatine Kinase): 120 U/L CK-MB (Creatine Kinase-MB): 15 U/L CRP (C-Reactive Protein): 13 mg/L Sodium: 138 mmol/L Potassium: 4.2 mmol/L Chlorine: 103 mmol/L Troponin: 0.01 ng/L In Full Blood Count: WBC : 6.0 k/µL Neutrophil Count: 3.0 k/µL Hgb: 12 g/dL MCV: 85 fL Plt: 250 k/µL. Calcium replacement therapy was initiated. The patient's clinical

symptoms started to improve.

Discussion

Hypocalcemia is a common yet often underrecognized electrolyte disturbance that can manifest in a variety of clinical forms. While the causes of hypocalcemia are diverse, this case illustrates a particular scenario in which the restriction of dietary calcium intake, as part of a liquid diet before colonoscopy, precipitated a significant decline in serum calcium levels.

In this patient, the presentation of muscle spasms in the fingers, characterized by a flexed position of all fingers that could not be extended, is suggestive of neuromuscular irritability, which is one of the hallmark signs of hypocalcemia. The pathophysiology of hypocalcemia involves increased neuromuscular excitability due to reduced calcium concentrations, which leads to symptoms such as tetany, paresthesia, and muscle cramps (4,5). This patient's symptoms were likely triggered by the prolonged restriction of calcium intake during her preparation for colonoscopy, highlighting the importance of ensuring adequate nutritional intake, particularly calcium, in elderly patients.

The patient's serum calcium level of 6.5 mg/dL was significantly below the normal reference range of 8.5-10.5 mg/dL, confirming the diagnosis of hypocalcemia. Other potential causes of hypocalcemia include hypoparathyroidism, vitamin D deficiency, and chronic kidney disease, none of which were identified in this patient's history or laboratory workup. In elderly patients, the risk of developing hypocalcemia is increased due to factors such as reduced intestinal calcium absorption, impaired renal function, and the use of medications that may interfere with calcium metabolism (6). However, in this case, the hypocalcemia was likely primarily due to the acute dietary restriction.

The initial management of hypocalcemia involves the prompt administration of calcium supplements, either orally or intravenously, depending on the severity of the symptoms and serum calcium levels. This patient was started on calcium replacement therapy, and her symptoms began to resolve shortly thereafter. It is essential to monitor serum calcium levels closely during treatment to prevent both hypocalcemia and hypercalcemia, as inappropriate correction can lead to complications (7).

This case emphasizes the need for careful pre-procedural management in elderly patients, especially those undergoing procedures like colonoscopy that require dietary restrictions. Adequate hydration and nutrition, including appropriate calcium intake, should be prioritized to prevent electrolyte disturbances, particularly in vulnerable populations such as the elderly.

Conclusion

Hypocalcemia can occur due to a variety of causes, including dietary restrictions. In this case, a 78-year-old woman developed symptomatic hypocalcemia after a 3-day liquid diet in preparation for colonoscopy. Prompt recognition and appropriate management, including calcium replacement therapy, were crucial to her recovery. This case underscores the importance of monitoring and managing electrolyte levels in elderly patients, particularly during periods of altered nutritional intake.

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A Rare Case Report: Isolated Nervus Oculomotor Paralysis

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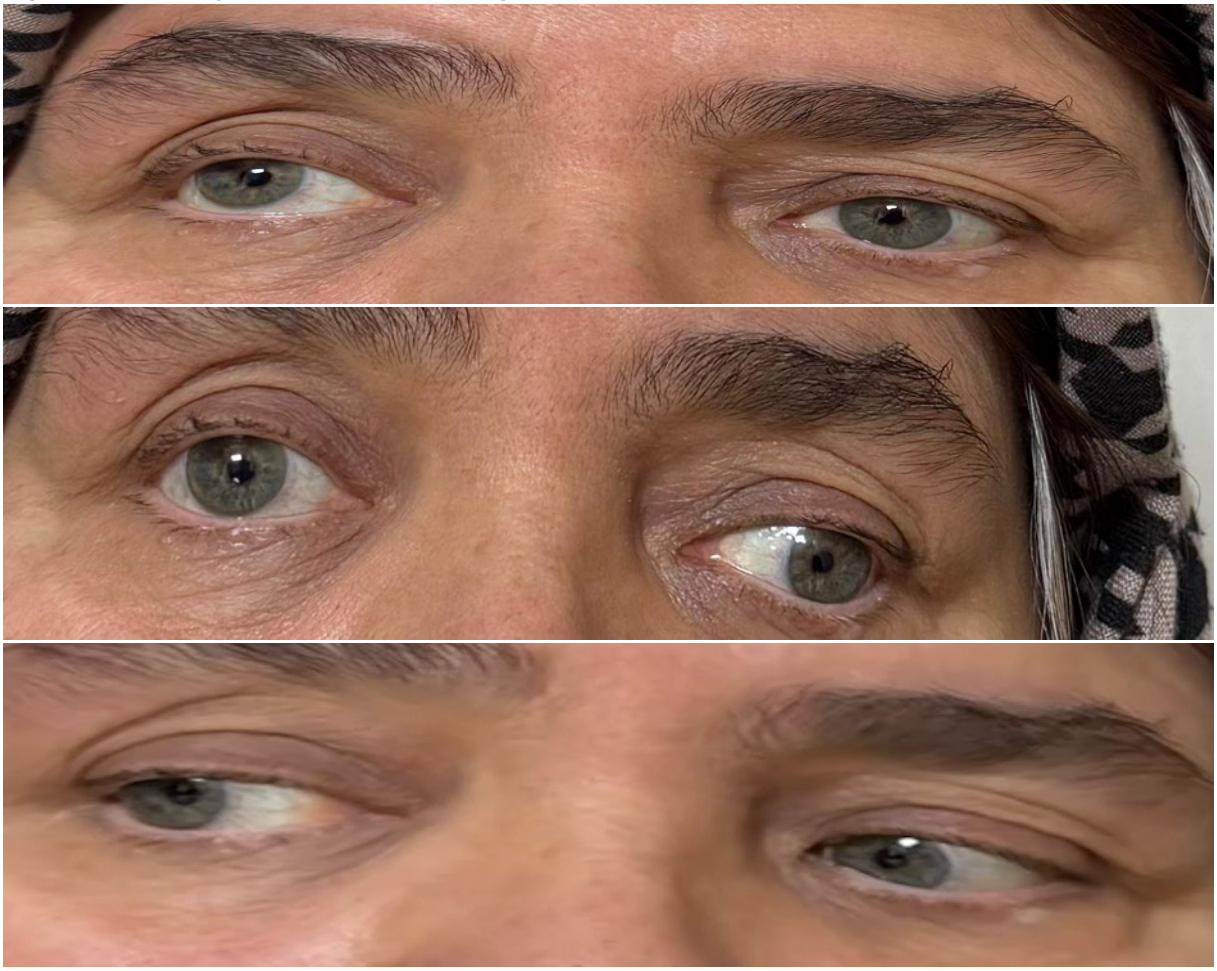
Introduction

The oculomotor nerve (3rd cranial nerve), the trochlear nerve (4th cranial nerve), and the abducens nerve (6th cranial nerve) are named as the motor nerves of the ocular structure and provide innervation of the eye muscles (1). The most common group in the paralysis of these nerves is the idiopathic group, which constitutes 25% of cases. In addition, tumors, ischemic diseases, vascular malformations, traumas, migraine, radiation therapy, chemotherapy, and vascular risk factors can cause cranial nerve paralysis (2). Isolated 3rd cranial nerve paralysis is generally a cranial nerve injury that occurs around the age of 60, with vascular risk factors (diabetes mellitus, hyperlipidemia, hypertension, coronary artery disease, etc.) in its etiology, resolves spontaneously, and no other diagnosis is made with further tests (3, 4). The aim of this study is to emphasize that isolated third cranial nerve paralysis, which is rarely seen in patients presenting with sudden-onset headache, diplopia, and loss of balance, should be kept in mind.

Case Presentation

A 62-year-old female patient presented to the emergency department with complaints of sudden-onset dizziness, headache, and diplopia. The patient, who had no history of trauma, stated that these complaints were new and sudden in onset. The patient, whose headache was subjected to pain scoring, scored 9 out of 10. When the patient's medical history was questioned, she stated that she had diagnoses of hypertension (HT) and Type 2 Diabetes Mellitus (DM) for 15 years. The patient had been receiving antihypertensive and insulin with oral antidiabetic treatment for 15 years and 10 years, respectively. The patient had a family history of Type 2 DM in first-degree relatives. In the patient's physical examination, no lateralizing findings or loss of strength were detected in the neurological examination. In our examination, pathological findings included outward and downward deviation in the right eye, and limitation of inward, upward, and downward gaze (Figure 1).

Figure 1: a: Primary Gaze b: Left Gaze c: Right Gaze



In the laboratory parameters taken from the patient, the blood glucose level was 320 mg/dl. No other pathological parameters were detected. With the preliminary diagnoses of cerebrovascular disease, the patient was planned for brain tomography (CT) and diffusion magnetic resonance (MR) imaging. As a result of the imaging, no acute pathology was detected. When the patient's past medical data were examined, periods of impaired blood glucose regulation were detected. Isolated 3rd cranial nerve paralysis was considered after the examination in the patient with a long-standing history of DM. The patient was consulted to the Ophthalmology and Neurology departments. The patient was admitted to the Neurology Service for blood glucose regulation and monitoring of the current condition.

Conclusion

Isolated oculomotor nerve paralysis (ONP) is a clinical condition that usually develops due to microvascular ischemia caused by conditions such as diabetes, hypertension, aneurysms, and trauma (5). In our case, the long history of diabetes mellitus (DM) and hypertension (HT) and the impaired blood glucose regulation suggest that ONP may be of microvascular ischemic origin. Diabetes is an important factor in the etiology of isolated oculomotor nerve paralysis. In patients presenting with various neurological symptoms such as dizziness, headache, and diplopia, diabetes should be questioned, and patients with a diagnosis of diabetes presenting to the emergency department should undergo a detailed neurological and eye examination (6, 7). Cranial nerve palsies due to diabetes are usually due to ischemic damage to the vasa nervorum, and spontaneous recovery is expected in most cases. Pupillary involvement is observed in compressive lesions such as aneurysms, while the pupil is generally preserved in paralyses of microvascular origin. The preservation of pupillary reflexes in our patient is an important finding supporting diabetic microangiopathy (8).

Early diagnosis and follow-up are of great importance in such cases. Blood glucose regulation and control of vascular risk factors are the basic components of treatment. A multidisciplinary approach plays a critical role in the management of patients.

Key Words: Oculomotor, Paralysis, Diabetes Mellitus

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Beyond Eye Pain: Diagnosis of Bilateral Posterior Scleritis in the Emergency Department

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Objective: Scleritis is a rare but serious chronic inflammation of the sclera, often associated with autoimmune systemic diseases. In patients presenting with eye pain, delayed diagnosis may lead to permanent vision loss. Therefore, scleritis should be considered in the evaluation of patients with eye pain in the emergency department. In this case, we aimed to emphasize the importance of early recognition of scleritis in the emergency department.

Figure 1. Swelling, pain, tearing, and redness starting in the left eye and later spreading to both eyes and eyelids.

Figure 2. Thickening and increased contrast enhancement in the posterior segments of the bilateral sclera, with mild haziness in the retrobulbar fat tissue.

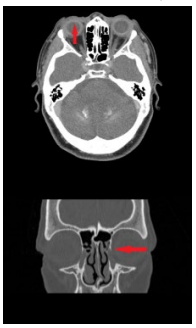


Figure 3

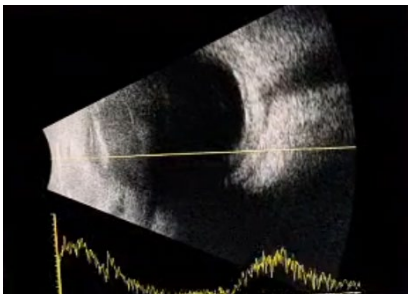


Figure 4

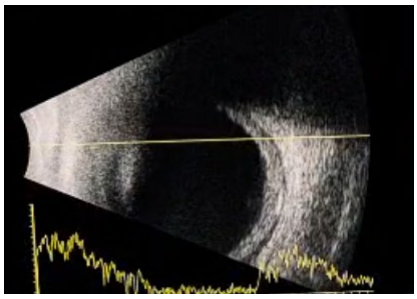
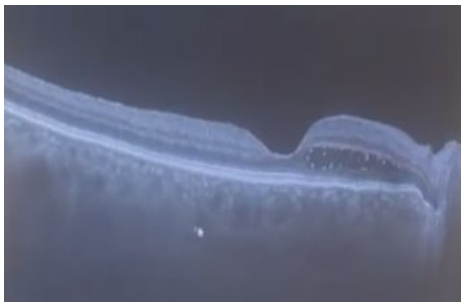


Figure 5



Case: A 54-year-old female patient presented to the emergency department with eye pain persisting for about three weeks. Her medical history included diabetes mellitus (DM), but she was not using medication. On physical examination, swelling, pain, tearing, and redness spreading from the left eye to both eyes and eyelids were observed (Figure 1). The pain was more severe in the left eye. Orbital CT imaging revealed thickening and contrast enhancement in the posterior segments of the bilateral sclera, along with mild infiltration in the retrobulbar fat tissue (Figure 2). The findings suggested scleritis, and the patient was referred to ophthalmology.

In the ophthalmologic evaluation, eye movements were free but painful bilaterally. Color vision assessment was limited due to poor patient cooperation. On biomicroscopy, both eyelids were red and edematous; conjunctiva was hyperemic, cornea was clear, anterior chambers were calm, and pupils were central and regular. Posterior synechia was present in the left eye. On ocular ultrasonography, bilateral scleritis was suspected (Figures 3 and 4 - T sign). Fundus examination of the right eye was normal. OCT of the right eye showed papillomacular subretinal fluid (SRF); choroidal fold and papillitis were seen in the left (Figure 5). With a diagnosis of bilateral scleritis, the patient was admitted to the ophthalmology ward for further investigation and treatment for immune system diseases such as rheumatoid arthritis, systemic lupus erythematosus, and granulomatosis with polyangiitis.

Conclusion: This case highlights the critical role of early diagnosis and appropriate treatment of scleritis in the emergency department in preventing vision loss. As scleritis may be linked to systemic diseases, comprehensive evaluation is needed. Immunosuppressive agents and corticosteroids may be effective in treatment; however, regular follow-up is crucial to prevent possible complications.

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Datura Stromonium Poisoning: A Report On Six Cases

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Introductıon

Throughout history, plants have drawn attention for both their medicinal and toxicological aspects, securing an important place in human history. In cases of poisoning, plants containing tropane alkaloids have posed a significant toxicological threat. In this context, the plant Datura stramonium, also known as "thorn apple" or "devil's trumpet," is among the herbal sources that can cause poisoning (1). Datura stramonium contains potent toxins known as tropane alkaloids, primarily scopolamine, atropine, and hyoscyamine, which can have serious effects on the nervous system (2). These effects can manifest particularly as anticholinergic symptoms, delusions, and hallucinations.

Datura stramonium is a common plant that can grow as a weed and may be consumed unknowingly. The leaves, seeds, and flowers of the plant contain the main toxic agents. Tropane alkaloids typically affect the central nervous system and the peripheral nervous system. The onset of symptoms usually occurs 30 minutes to 2 hours after consumption (3). In the clinical picture of poisoning, peripheral anticholinergic effects such as dry mouth, tachycardia, blurred vision, and urinary retention, along with central nervous system effects like agitation, delirium, and hallucinations, are prominent (4).

Used for medicinal purposes since ancient times, Datura stramonium frequently leads to poisoning cases due to misuse. Its use among young individuals, particularly for its hallucinogenic effects, can result in serious consequences, placing this group at specific risk for poisoning (5). Poisonings typically occur as a result of random consumption, misidentification of the plant, or psychiatric misuse. In recent years, the growing interest in such plants, especially among the youth, has increased the frequency of these cases worldwide (6).

This case series includes six patients who experienced hallucinations and anticholinergic symptoms as a result of poisoning with Datura stramonium, reflecting central nervous system effects. The main aim of our study is to analyze this case series to highlight the clinical symptoms and treatment processes associated with Datura stramonium-induced poisonings. Additionally, we aim to contribute to the existing literature and raise awareness regarding the management of such poisonings.

Case Report

A family of six presented to our emergency department with symptoms that began two hours after consuming a dish made from a wild plant known locally as "siren/kırçan" (a type of wild herb). The symptoms varied among family members. Among the plants collected from the family, a specimen of Datura stramonium was identified. A consultation with the poison control center indicated that there was insufficient data on oral intakes, and it was noted that symptoms could include nausea, vomiting, abdominal pain, dermatitis, aspiration pneumonia, and visual hallucinations. The use of activated charcoal and gastric lavage was not recommended. Symptomatic treatment was advised, and monitoring for at least 24 hours in a service or intensive care unit was suggested based on the patient's clinical condition. Below, the symptoms, examination findings, laboratory results, treatments administered, and clinical outcomes for each case are detailed individually.

Case 1

An eighty-six-year-old female patient with a history of hypertension and diabetes presented with complaints of visual hallucinations, euphoria, agitation, and subsequent deterioration of consciousness. Upon emergency admission, her consciousness was lethargic, with vital signs showing a pulse of 135 bpm, temperature of 37,8°C, respiratory rate of 28 breaths per minute, and blood pressure of 170/110 mmHg; physical examination revealed normal breath sounds, a soft abdomen, and no defense or rebound tenderness. An

electrocardiogram showed a sinus rhythm of 140 bpm. The results of her blood tests are summarized in Table 1. No acute pathology was found on chest X-ray or brain CT. Due to her altered level of consciousness, activated charcoal and gastric lavage were not performed. The patient received 1 mg of IV physostigmine. She was admitted to our emergency department for monitoring and treatment. Approximately two hours after admission, her consciousness improved, and by the sixth hour, her complaints had diminished, with no complications developing. The patient was discharged in good health 24 hours after admission.

Case 2

A seventy-seven-year-old female patient with a history of hypertension presented to our emergency department with complaints of visual hallucinations, euphoria, nausea, and vomiting. Upon emergency admission, her consciousness was clear, partially cooperative, with vital signs showing a pulse of 66 bpm, temperature of 36,2°C, respiratory rate of 16 breaths per minute, and blood pressure of 110/70 mmHg; physical examination revealed normal breath sounds, a soft abdomen, and no defense or rebound tenderness, with a normal neurological examination. An electrocardiogram showed a sinus rhythm of 66 bpm. The results of her blood tests are summarized in Table 1. No acute pathology was found on chest X-ray or brain CT. The patient was admitted to our emergency department for monitoring and treatment. Approximately four hours after admission, her complaints had diminished, with no complications developing. The patient was discharged in good health 24 hours after admission.

Case 3

A fifty-seven-year-old female patient with no known chronic illness presented to our emergency department with complaints of euphoria, nausea, and vomiting. Upon emergency admission, her consciousness was clear, partially cooperative, with vital signs showing a pulse of 77 bpm, temperature of 35,8°C, respiratory rate of 18 breaths per minute, and blood pressure of 110/70 mmHg; physical examination revealed normal breath sounds, a soft abdomen, and no defense or rebound tenderness, with a normal neurological examination. An electrocardiogram showed a sinus rhythm of 81 bpm. The results of her blood tests are summarized in Table 1. No acute pathology was found on chest X-ray or brain CT. The patient received gastric lavage and activated charcoal. She was admitted to our emergency department for monitoring and treatment. Approximately four hours after admission, her complaints had diminished, with no complications developing. The patient was discharged in good health 24 hours after admission.

Case 4

A fifty-year-old male patient with no known chronic illness presented to our emergency department with complaints of euphoria, agitation, nausea, and vomiting. Upon emergency admission, his consciousness was clear, partially cooperative, with vital signs showing a pulse of 92 bpm, temperature of 36,8°C, respiratory rate of 18 breaths per minute, and blood pressure of 130/80 mmHg; physical examination revealed normal breath sounds, a soft abdomen, and no defense or rebound tenderness, with a normal neurological examination. An electrocardiogram showed a sinus rhythm of 90 bpm. The results of his blood tests are summarized in Table 1. No acute pathology was found on chest X-ray or brain CT. The patient received gastric lavage, activated charcoal, and 10 mg of IV haloperidol. Approximately five hours after admission, his complaints had diminished, with no complications developing. Thirteen hours after admission, he left the hospital against medical advice, accepting all risks including death.

Case 5

A fifty-nine-year-old female patient with a history of hypertension and diabetes presented to our emergency department with complaints of nausea, vomiting, and euphoria. Upon emergency admission, her consciousness was clear, partially cooperative, with vital signs showing a pulse of 96 bpm, temperature of 36°C, respiratory rate of 20 breaths per minute, and blood pressure of 120/80 mmHg; physical examination revealed normal breath sounds, a soft abdomen, and no defense or rebound tenderness, with a normal neurological examination. An electrocardiogram showed a sinus rhythm of 88 bpm. The results of her blood tests are summarized in Table 1. No acute pathology was found on chest X-ray or brain CT. The patient received gastric lavage and activated charcoal. She was admitted to our emergency department for monitoring and treatment. Approximately four hours after admission, her complaints had diminished, with no complications developing. Thirteen hours after admission, she left the hospital against medical advice, accepting all risks including death.

Case 6

A sixty-two-year-old female patient with no known chronic illness presented to our emergency department with complaints of nausea, vomiting, and euphoria. Upon emergency admission, her consciousness was clear, partially cooperative, with vital signs showing a pulse of 72 bpm, temperature of 35,9°C, respiratory rate of 16 breaths per minute, and blood pressure of 110/60 mmHg; physical examination revealed normal breath sounds, a soft abdomen, and no defense or rebound tenderness, with a normal neurological examination. An electrocardiogram showed a sinus rhythm of 88 bpm. The results of her blood tests are summarized in Table 1. No acute pathology was found on chest X-ray or brain CT. The patient received gastric lavage and activated charcoal. She was admitted to our emergency department for monitoring and treatment. Approximately four hours after admission, her complaints had diminished, with no complications developing. The patient was discharged in good health 24 hours after admission.

Discussion

Poisoning incidents involving plants such as Datura stramonium are often observed, particularly in rural areas, due to the misidentification and consumption of these plants. The perception that these poisonings are "natural" leads to them being considered harmless or less harmful, which results in underestimating the risk and downplaying the toxic effects (7).

The symptoms of the six patients included in the case series specifically overlap with Datura stramonium poisoning. Classic symptoms of anticholinergic syndrome, including agitation, confusion, hallucinations, tachycardia, mydriasis, and dry skin, were observed to varying degrees in these cases (8). In Case 1, the patient was confused and agitated, exhibiting tachycardia and visual hallucina-

tions. In other cases, non-specific symptoms such as nausea, vomiting, and euphoria were more prominent. This variety suggests that the effects of Datura stramonium may be related to the dose taken, the individual's age, underlying medical conditions, and individual differences in toxin metabolism (9).

The lack of awareness regarding the toxicity of herbal products sold without prescriptions or collected from nature facilitates the occurrence of such cases (10). Additionally, the involvement of elderly individuals in most of these cases indicates that the elderly population may be more sensitive to anticholinergic toxins (11). The decreased metabolic clearance with age may explain this situation.

Table 1.

	WBC	NEU	HBG	PLT	BUN	KRE	ALT	AST	PH	HCO3	CO_2	CRP	PKES
Case 1	8,27	5,57	11	357	24	0,75	7	13	7,39	24	40	6	-
Case 2	7,1	4,5	11,2	380	19	0,88	7	15	7,38	24	41	3	-
Case 3	8,1	4,5	12,8	282	14	0,60	8	18	7,40	25	43	3	9624
Case 4	8,2	6,3	12,3	339	17	1,35	22	14	7,36	28	57	17	8607
Case 5	10,6	8,9	14,4	185	14	0,56	13	17	7,35	26	56	8	8748
Case 6	11,4	9,4	12,6	310	16	0,87	8	26	7,37	23	43	11	9650

Treatment for Datura stramonium poisoning should largely be symptomatic and supportive. In the cases reviewed, gastric lavage and activated charcoal were administered; however, the literature indicates that these approaches may be effective shortly after toxin ingestion (usually within 1 hour) and may provide limited benefit if a longer time has passed (12). Furthermore, although there is limited data on the efficacy of activated charcoal, its use is suggested in patients with a low risk of complications (13).

Another important aspect of managing poisoning is controlling anticholinergic syndrome. The literature recommends the use of acetylcholinesterase inhibitors, such as physostigmine, in cases with severe central nervous system symptoms. In our case series, we used physostigmine in one patient. The risks and contraindications for the use of physostigmine should be considered, and routine use should be avoided for each patient (14).

When our cases were examined, it was observed that sedation was effective in controlling agitation symptoms. The use of agents such as antipsychotic medications like haloperidol appears appropriate for symptomatic treatment of anxiety, agitation, and hallucinations. However, caution should be exercised when using antipsychotic medications to control anticholinergic conditions, as these agents can also trigger extrapyramidal symptoms and lead to other involuntary movement disorders (15).

Datura stramonium poisonings pose serious dangers to public health, in addition to individual misconsumption habits. It is essential to raise awareness among the public in rural areas about recognizing toxic plant species and avoiding them (16). In this context, it is important for local governments and health authorities to organize educational activities.

Individuals living in rural areas may often delay recognizing poisoning symptoms and seeking necessary medical assistance. Therefore, programs that promote the recognition of basic symptoms and encourage immediate consultation with healthcare institutions in relevant situations are needed. Additionally, healthcare workers should receive training on the management of such poisonings and be familiar with guideline practices.

This case series overlaps with other case series in the literature. For example, a study on Datura stramonium toxicity in elderly individuals similarly reported prominent hallucinations, tachycardia, and agitation (17). Furthermore, another case series conducted in India emphasized that traditional herbal remedies accounted for a significant portion of consumption errors involving these toxins and that the severity of toxicity varied from person to person (18).

These findings in the literature support the observations in our study and demonstrate that Datura stramonium produces similar effects at both individual and population levels.

Although Datura stramonium poisonings can lead to serious clinical outcomes symptomatically, appropriate and timely treatment can largely result in complete recovery. In this case series, it was observed that typical anticholinergic symptoms resulting from the accidental consumption of this plant were effectively managed. It is critical for healthcare workers to receive training regarding these toxic plants. Broader case series and studies can provide meaningful contributions to standardizing the management of such poisonings and strengthening public health policies.

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Evaluation of the relationship between blood lipase level and Computed Tomography Severity Index in patients presenting to the emergency department with abdominal pain

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Introduction

Acute pancreatitis (AP) is an inflammatory condition affecting the pancreas, typically caused by gallstones or excessive alcohol consumption. Most AP cases present a mild course, with patients often experiencing rapid improvement after receiving fluid resuscitation, pain control, and early initiation of oral feeding.

Nonetheless, around 20-30% of cases can be severe, with a mortality rate up to 15% among those affected. Therefore, early recognition of these patients is critical, and treatment should be initiated promptly and more intensively.

There are many studies in the literature that examine the lipase levels and CTSI scores of patients with pancreatitis and the relationships between high lipase and CTSI scores in patients with pancreatitis alone. However, we could not encounter a study that evaluates the presence of pancreatitis and nonpancreatic pathologies on computerized tomography (CT) scanning together in patients with elevated lipase levels.

In our study, we aimed to evaluate the lipase levels and CTSI scores of patients who presented to the emergency department (ED) with abdominal pain and were diagnosed with pancreatitis on CT scanning. We also aimed to examine the patients who, according to the other pathologies, had elevated lipase levels but no finding of pancreatitis on CT interpretations.

Material and methods

This study was conducted retrospectively in a tertiary ED. The hospital where the study was performed has an average of 15000-18000 patients admitted to the ED monthly. Ethical approval was received from a local ethics committee (Decision number: 2024/03-10 Date: 03.04.2024). The study was performed using the tenets of the Declaration of Helsinki.

Study population

Patient information was obtained through the Hospital Information Management System (HIMS). All the patients who applied to the ED between June 1, 2023, and January 1, 2024, were enrolled in the study.

Patient selection

Inclusion criteria

According to the International Classification of Diseases Codes 10th Revision, the patients who were coded as preliminary diagnosis, that is, R10.0, R10.1, R10.2, R10.3, R10.4, and who had ordered serum lipase levels requested as laboratory tests and who underwent abdominal CT imaging were included in the study.

Exclusion criteria

The patients whose CT images were too poor for interpretation (artifacts, etc.), those who had no tomography images in the HIMS, and those who had normal lipase levels and no findings of pancreatitis on CT interpretations were excluded from the study.

Comorbid diseases or medications may affect lipase levels. Thus, we also excluded those patients with comorbid diseases and medications from the study.

Data collection and standards

The patients' sociodemographic and clinic features (age, gender, and laboratory results) were obtained from the HIMS files.

All CT images (contrast-enhanced or non-contrast) were separately interpreted again by a radiology specialist as a gold standard. Besides pancreatitis diagnostic criteria (presence of one or more of the following findings on CT: increased pancreatic size, inflammatory changes in pancreatic tissue and peripancreatic fatty tissue, and fluid collection with irregular borders), other pathologies on the CT imaging have also been interpreted by the same radiologist.

The levels of lipase were measured using a Beckman Coulter laboratory kit, and the levels between 3 and 67 U/L were accepted as normal.

Patients' CTSI scores were calculated by summating the Balthazar score and the degree of pancreatic necrosis score. The Balthazar score was assessed from A, B, C, D, and E based on the severity of pancreatitis, with corresponding scores of 0, 1, 2, 3, and 4, respectively. Pancreatic necrosis was scored as 0 for no necrosis, 2 for less than 30% necrosis, 4 for 30-50% necrosis, and 6 for more than 50% necrosis. The overall CTSI score was categorized as mild (0-3), moderate (4-6), and severe (7-10) **(Table 1).**

Table 1: Parameters and calculation of CTSI score

Grade	Grading of pancreatitis	Score
A	Normal pancreas	0
B	Pancreatic enlargement	1
C	Pancreatic inflammation and/or peripancreatic fat	2
D	Single peripancreatic fluid collection	3
E	Two or more fluid collections and/or retroperitoneal air	4
Necrosis Percentage		**Score**
0		0
<30		2
30-50		4
>50		6
Result (Multiply by grading of pancreatitis score and necrosis percentage score)		
0-3	Mild acute pancreatitis	
4-6	Moderate acute pancreatitis	
7-10	Severe acute pancreatitis	

Statistical analysis:

Statistical analyses were performed using Jamovi Ver. 2.3.28 software package (The Jamovi Project, Sydney, Australia). The Kolmogorov-Smirnov test was used to check the normality of data distribution. Categorical data were presented as frequency and percentages, numerical data as mean and standard deviation if normally distributed, and median and interquartile range (IQR) values otherwise. Mann-Whitney U test was used to compare two groups regarding non-normally distributed data. Statistical significance was taken as $p<0.05$.

Results

Three thousand three hundred eight patients were enrolled in the study that matched the inclusion criteria. Three hundred forty-four patients who met these criteria were included in the study, while 2964 patients were excluded **(Figure 1).**

344 patients were included in the study. 53.2% (n=183) of the patients were male and the mean age was 57 (19-92) years. 333 (%97) of the patients had elevated lipase. 231 (69.3%) of the patients with elevated lipase, there was no evidence of pancreatitis on their CT findings. Only 3% (n=11) of the patients, although the lipase levels were in the normal range, were evidence of pancreatitis on their CT images (**Table 2**).

Table 2: Relationship between lipase level and pancreatitis

	Elevated Lipase Levels		
Pancreatitis on CT	Yes	No	**Total**
Yes	102	11	113
No	231	2946	3177
Total	**333**	**2957**	**3290**

Lipase level was found to be a highly sensitive and specific laboratory parameter for the diagnosis of pancreatitis [Sensitivity: 90.27% (83.25% - 95.04%; 95% CI); Specificity: 92.73% (91.77% - 93.61%; 95% CI); Positive Likelihood Ratio: 12.41 (10.81 – 14.25; 95% CI); Negative Likelihood Ratio: 0.1 (0.06 – 0.18; 95% CI); Prevalence: 3.43% (2.84% - 4.12%; 95% CI); Positive Predictive Value: 30.63% (27.78% - 33.64%; 95% CI); Negative Predictive Value: 99.63% (99.35% - 99.79%; 95% CI); Accuracy: 92.64% (91.7% - 93.51%; 95% CI)].

For patients with pancreatitis on CT, mean lipase levels were 2378 U/L (min: 7 U/L max: 29593 U/L). The mean lipase level of patients with no pancreatitis on CT interpretations was 285 U/L. We found that the blood lipase levels of patients with pancreatitis on

CT imaging were statistically significantly higher than those without (p<0.01).
Six patients diagnosed with pancreatitis had necrosis on their CT scans (4 had lower than 30%, one had between 30% and 50%, and one had over 50%). We found mean lipase level of patients with necrosis was 392.3 U/L. The 107 patients diagnosed with pancreatitis had no necrosis on CT images. The mean lipase level of these patients was 2500.6 U/L. Lipase levels were significantly lower in patients with pancreatitis with necrosis on CT scans than those without (p<0.01).
According to the CTSI score, 81 patients were defined as mild pancreatitis (0-3 points). Their mean lipase level was 2061.2 U/L. Also, 30 patients were defined as having moderate pancreatitis (4-6 points), and their mean lipase level was 3426.4 U/L. There was no statistically significant difference between these CTSI score groups according to the mean lipase levels (p>0.05).
According to the CTSI score, only two patients were defined as having severe pancreatitis (7-10 points). The mean lipase level of these two patients was 89 U/L. When we compared the mean lipase level of severe pancreatitis patients, with mild and moderate pancreatitis patients' mean lipase levels, we determined that the low lipase levels were statistically significant in patients with severe pancreatitis (p<0.01).
We observed that lipase levels tend to decrease as the severity of pancreatitis increases. However, we found a significant opposite relationship between the severity of pancreatitis and lipase levels **(Table 3).**

Figure 1: Study Flow Chart

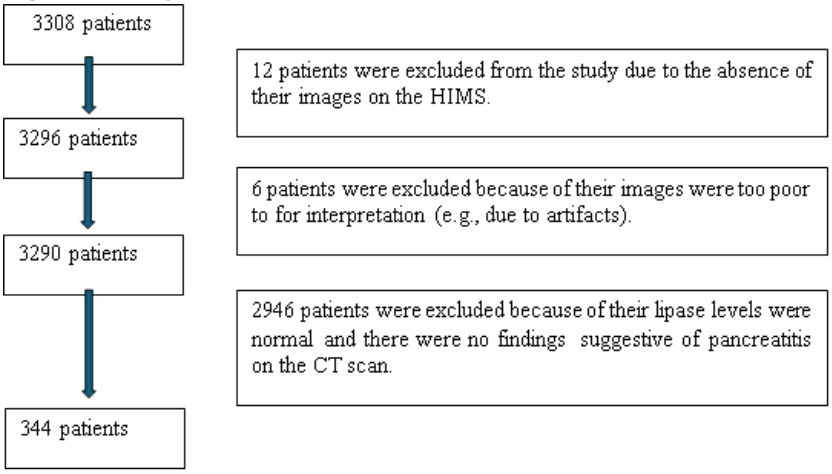

Table 3. Distribution of patients according to CTSI score

CTSI Score	Patients (n)	Mean Lipase Level (U/L)
Mild Pancreatitis (0-3)	81	2061.2
Moderate Pancreatitis (4-6)	30	3426.4
Severe Pancreatitis (7-10)	2	89

No additional pathology was detected in 33.6% of patients (n=38) with pancreatitis on CT images. The most common accompanying finding with elevated lipase and pancreatitis was gallstones (29.2%; n=33) **(Table 4)**.
Of the 52 patients (22.5%) with no pancreatitis on CT scans but elevated lipase levels, no accompanying pathological findings on CT interpretations are absent. In 35 patients (15.2%) with no pancreatitis on CT scans but elevated lipase levels, the most common accompanying finding was gallstones on CT interpretations **(Table 5).**

Discussion

This study investigates the relationship between lipase levels in patients diagnosed with pancreatitis and the disease severity determined by CTSI scores. It also investigates other pathologies in patients with high lipase levels but not diagnosed with pancreatitis. The findings are consistent with those reported data in the current literature.

Lipase Levels and Sensitivity in Diagnosing Pancreatitis

Our results demonstrate that lipase levels exhibit high sensitivity and specificity for pancreatitis. As shown in previous studies by Banks et al. (2013) (1), Koizumi et al. (2006) (2), and Gomez et al. (2012) (3), lipase is a sensitive biomarker for acute pancreatitis, with a significant proportion of patients presenting with elevated lipase levels.
However, our study also confirms that elevated lipase alone is not definitive for diagnosis, a finding that is consistent with prior research (4). In our cohort study, 97% of patients had elevated lipase levels, yet 69.3% did not show evidence of pancreatitis on CT scans. This result aligns with the conclusions of Banks et al. (2013) (1). Additional imaging findings are required to confirm pancreatitis.
Furthermore, in 3% of all patients in our study, there was evidence of pancreatitis on CT despite normal lipase levels. This finding further supports the idea that imaging is crucial for an accurate diagnosis. In addition to this finding, studies by Muniraj et al. (2015)

(5), Manjuck et al. (2005) (6), and Hameed et al. (2015) (4), which emphasize that many etiologies other than pancreatitis cause

Table 4. Accompanying pathologies observed in patients with pancreatitis (Elevated lipase or not)

Name of Pathology	Number of Patients	Percent (%)
No pathology	38	33.6
Gallstones	33	29.2
Liver cirrhosis	18	15.9
Choledocholithiasis	9	8
Mesenteric panniculitis	6	5.3
Cholecystitis	5	4.4
Pancreatic malignancy	3	2.7
Chronic renal failure (renal atrophy)	1	0.9
TOTAL	**113**	**100**
Pancreatitis with Elevated Lipase Levels		
Name of Pathology	**Number of Patients**	**Percent (%)**
No pathology	31	30.4
Gallstones	31	30.4
Liver cirrhosis	17	16.7
Choledocholithiasis	9	8.8
Mesenteric panniculitis	6	5.9
Cholecystitis	5	4.9
Pancreatic malignancy	2	2
Chronic renal failure (renal atrophy)	1	11
TOTAL	**102**	**100**
Pancreatitis without Elevated Lipase Levels		
Name of Pathology	**Number of Patients**	**Percent (%)**
No pathology	7	63.6
Gallstones	2	18.2
Liver cirrhosis	1	9.1
Pancreatic malignancy	1	9.1
TOTAL	**11**	**100**

Table 5. Accompanying pathologies observed in patients with elevated lipase levels with no pancreatitis

Name of Pathology	Number (n)	Percentage (%)
No pathology	52	22.5
Gallstones	35	15.2
Liver Cirrhosis	29	12.6
Ureteral stone	22	9.5
Pancreatic malignancy	19	8.2
Mesenteric panniculitis	18	7.8
Cholecystitis	12	5.2
Extra-pancreatic malignancy	11	4.8
Ileus	11	4.8
Choledocholithiasis	9	3.9
Chronic renal failure (Renal atrophy)	5	2.2
Acute appendicitis	4	1.7
Perforation	2	0.9
Hernia	1	0.4
Mesenteric ischemia	1	0.4
TOTAL	**231**	**100**

elevated lipase, also support the need for CT.

Relationship between CTSI Score and Lipase Levels

We found that lipase levels did not correlate with the severity of pancreatitis as assessed by the CTSI scores. While the patients with

mild and moderate pancreatitis CTSI scores had higher mean lipase levels, only two patients with severe pancreatitis CTSI scores had significantly lower lipase levels.

Kalokhe et al. (2022) (7) and Swaroop et al. (2004) (8) emphasized no relationship between lipase levels and pancreatic necrosis. The lower lipase levels in patients with necrosis may be due to extensive tissue destruction, which reduces the enzyme's production. We did not find any study in the literature clearly emphasizing an inverse relationship between lipase levels and pancreatic necrosis. The inverse relationship we found in our limited number of patients must be confirmed with a more extensive case series.

Moreover, no statistically significant difference was observed in lipase levels between mild and moderate CTSI score groups, supporting findings that lipase levels alone are unreliable indicators of disease severity (7).

CT Imaging and Differential Diagnosis

Gallstones were the most commonly observed finding, in both patients with evidence of pancreatitis on CT and those with elevated lipase levels but no evidence of pancreatitis. This finding highlights the importance and necessity of the use of CT in distinguishing the probable causes of elevated lipase levels, such as gallstones and pancreatitis. This necessity also corroborates the reported observations of Banks et al. (2013) (1).

The literature has highlighted the importance and necessity of using CT to distinguish the probable causes of elevated lipase levels. These studies have also noted that imaging modalities like CT are essential in evaluating the severity and complications of pancreatitis, especially when laboratory findings are ambiguous (4,5,9).

Study Limitations

The number of patients was limited because our study had a single-center and retrospective design, and the number of patients with necrosis on imaging was small. However, a more comprehensive study with a larger patient population is required.

Conclusion

Lipase is still a valuable, sensitive, and specific biomarker for diagnosing pancreatitis. However, the combination of clinical evaluation, laboratory results, and advanced imaging techniques remains the cornerstone of accurate pancreatitis diagnosis and management. CT imaging and CTSI scoring should be used with lipase levels to provide a more comprehensive assessment of pancreatitis severity. Further research is necessary to elucidate better the complex relationship between lipase levels and the progression of the disease.

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5702

Cardiopulmonary Arrest İn Contrast-Enhanced Tomography: Typical İmaging Findings With 2 Cases

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Introduction: Cardiopulmonary arrest (CPA) is most commonly defined as loss of circulation requiring resuscitation with chest compressions, defibrillation, or both. In-hospital CPA mortality remains high despite improved survival in recent years. Early recognition of CPA with prompt initiation of cardio-pulmonary resuscitation (CPR) to restore circulation leads to better outcomes. Computed tomography (CT) is used in unstable patients to evaluate possible causes such as aortic aneurysm rupture, aortic dissection, pulmo-

nary thromboembolism, and in trauma patients organ lacerations. While the majority of CPA is detected by clinical signs or monitoring, in some cases it can develop during CT imaging. Herein, we report CPA's CT findings through two cases.

Case: Patient 1; 74-year-old female with hypertension was brought to the emergency room with shortness of breath and confusion. CT scan performed to rule out aortic dissection. After the CPA findings were seen, the scan was terminated and CPR was started on the patient. After CPR, spontaneous circulation was achieved and the patient was admitted to the intensive care unit. Patient 2; 67-year-old male patient with no additional illness was admitted to the emergency room with shortness of breath. Pulmonary CT angiography was performed with suspicion of pulmonary embolism, and CPA was observed. The patient was quickly removed from the CT table and CPR was started. The patient did not respond to CPR and died.

High-density contrast material pooling was observed in the right atrium, inferior vena cava, hepatic veins, and liver parenchyma on CT of patient 1(Figure 1). On patient 2's CT, the contrast material coming through the venous vascular access is observed with high density in the superior vena cava (SVC), right atrium, inferior vena cava (IVC), azygos vein, and hepatic veins, but not in the right ventricle, pulmonary cardiovascular systems, and left heart centers (Figure 2).

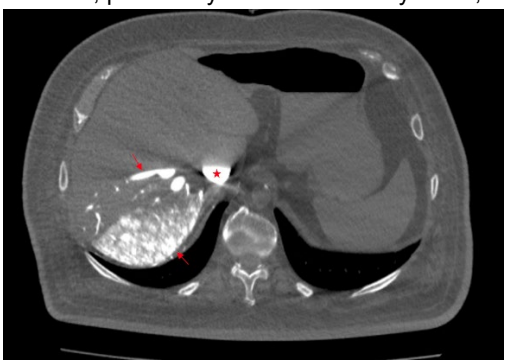

Figure 1. Typical appearance of contrast material shows venous pooling in IVC (asterisk) and hepatic veins (arrows) in a CPA patient.

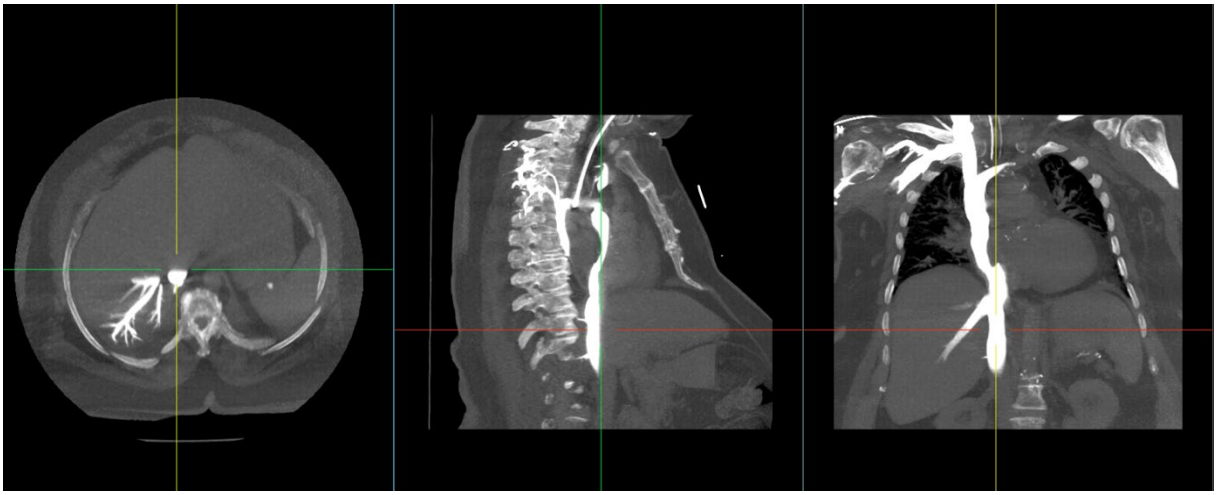

Figure 2. Axial sagittal and coronal reformatted CT sections (left to right, respectively). Dependent venous pooling and layering of contrast material within SVC, right atrium, IVC, and other dependent venous structures.

Conclusion: Although cardiac arrest during CT scanning is rare, knowing the imaging findings is important to stop the scan and start resuscitation quickly if necessary. In CPA, the contrast material given intravenously does not progress to the left heart chambers and pulmonary arterial system, but refluxes from the right heart chambers to the IVC and hepatic veins, and accumulates in the venous system. As a result, a hyperdense appearance is observed on CT scan due to contrast material reflux in the IVC, azygos vein, hepatic veins and renal veins (1-3). Contrast material reflux into the liver parenchyma is evident in posterior segments. Contrast material-blood leveling is seen in the distal parts of the IVC (4,5). Knowing the CT findings essentially prevents wasting time on differential diagnosis and facilitates rapid intervention. To prevent possible late resuscitation, risky patients should be monitored during the scan and the scan should be performed with the accompaniment of resuscitation personnel and equipment.

Keywords: Cardiopulmonary arrest, CT, venous contrast pooling

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5716

Pleural Effusion; Single Centre Experience

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Introduction and Objective: Pleural effusion is defined as an abnormal accumulation of fluid between the parietal and visceral pleura. The nature of the fluid varies depending on the underlying etiology. Transudative effusion develops in heart failure and hypoalbuminemic conditions such as nephrotic syndrome and liver failure. Exudative effusion develops in conditions such as infection, malignancy, and connective tissue disease. Parapneumonic effusion is the most common type of pleural effusion in children. This study aims to present the single center experience of patients followed up for pleural effusion in our hospital.

Materials and Methods: A retrospective analysis was performed on the data of 53 pediatric patients under the age of 18 years who were followed up for pleural effusion at the Department of Pediatrics, Necmettin Erbakan University Faculty of Medicine Hospital between 2019 and 2024. The study evaluated age, gender, etiology of pleural effusion, fluid characteristics, and treatment modalities. The SPSS 22 (IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp) package programme was used in the study.

Table 1: The relationship between the day of hospitalisation and age at diagnosis, effusion depth, fluid LDH level and serum CRP level

		Age at diagnosis (month)	Depth of effusion (mm)	Fluid LDH level (U/L)	Serum CRP level (mg/L)	Day of admission
Age at diagnosis (month)	r	1,000	,067	-,089	,028	-,209
	p	.	,634	,570	,842	,134
	n	53	53	43	53	53
Depth of effusion (mm)	r	,067	1,000	,255	,219	,337*
	p	,634	.	,099	,115	,013
	n	53	53	43	53	53
Fluid LDH level (U/L)	r	-,089	,255	1,000	,488**	-,024
	p	,570	,099	.	,001	,877
	n	43	43	43	43	43
Serum CRP level (mg/L)	r	,028	,219	,488**	1,000	,174
	p	,842	,115	,001	.	,214
	n	53	53	43	53	53
Day of admission	r	-,209	,337*	-,024	,174	1,000
	p	,134	,013	,877	,214	.
	n	53	53	43	53	53

* p<0.05, ** p<0,01

Results and Conclusion: Of the patients included in the study, 50.9% were male. The mean age at diagnosis was 132.42 (12-214) months. Radiological examination revealed a unilateral effusion in 67.9% of patients with a median effusion depth of 30 mm (14-47.5). Thoracentesis was performed in 81.1% of patients, while in 10 patients (18.9%) thoracentesis was not performed due to minimal effusion. It was observed that 81.4% of the thoracentesis fluid was exudative. The median CRP level was 88.1 mg/L (17.2-154) and the median fluid LDH level was 572 U/L (228-1139). A statistically significant positive correlation was found between these two levels (r:0.488, p:0.001). When analyzing the etiology of the pleural effusion, pneumonia was found in 64.2% of patients, tuberculosis in 22.6%, and other causes such as malignancy and chronic kidney disease in 13.2%. A total of 27 patients (50.9%) underwent tube thoracostomy, and 48.1% of these patients received fibrinolytic therapy. The mean hospital stay was 16 days (12-21.5). Patients with an effusion depth greater than 1 cm had a longer hospital stay (p=0.02). Determining the etiology of pleural effusion is important for treatment and follow-up. Our study found that the most common cause of pleural effusion was pneumonia, which is consistent with the existing literature. However, it is also important to consider rare conditions such as childhood malignancy.

Keywords: pleural effusion, pneumonia, tuberculosis, thoracentesis

5760

Right ICA Dissection Following Extension

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Introduction

Internal carotid artery (ICA) dissection accounts for 2% of strokes and 10-25% of strokes in young individuals[1]. ICA dissections mostly develop spontaneously. The main risk factors include connective tissue diseases, trauma, and cervical spine manipulation[2]. Medical treatment, mechanical thrombectomy, and stent placement are preferred endovascular treatment methods[3].

In this case, we present a case of cerebral infarction caused by ICA dissection in a patient whose neck was placed in an extension position for the excision of a lesion on the lip.

Case Report

A 43-year-old female patient visited the emergency department with complaints of numbness in her left arm and leg two days after undergoing a 10-minute local anesthesia procedure in which her neck was placed in an extension position for the excision of a lesion on her lip. She had no known a medical disease. Her Glasgow Coma Score was 15, and her vital signs were stable. Hypoesthesia was detected in the left upper and lower extremities, while sensory and motor functions in the right upper and lower extremities were completely normal.

Diffusion MRI revealed two areas of diffusion restriction at the level of the centrum semiovale and the right periventricular white matter (Figure 1). Brain and neck CT angiography (CTA) revealed dissection and thrombosis in the right ICA (Figure 2).

Routine laboratory tests were normal. Tests for vasculitis showed no specific findings. Echocardiography revealed an ejection fraction (EF) of 60%, with no vegetations or thrombi. Tumor marker tests and thoracoabdominal CT performed for malignancy screening showed no abnormal findings.

Discussion

Carotid artery dissection can occur spontaneously or due to trauma. Movements such as cervical vertebral hyperextension, sudden head rotation, or lateral flexion can cause tension in the internal carotid artery and lead to damage in the intimal layer[4]. In this case, it is thought that hyperextension of the neck led to tearing of the vessel wall, resulting in thrombosis.

It is important for physicians to avoid head and neck maneuvers that may cause carotid artery dissection and for emergency physicians to be aware of such complications.

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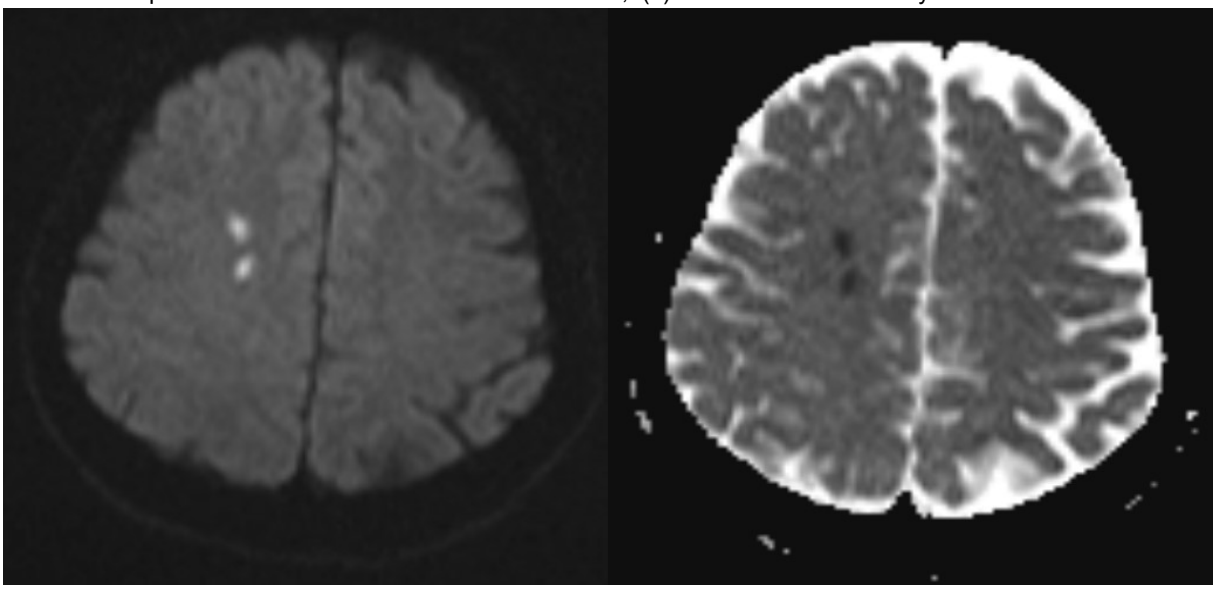

Figure 1. Two areas of diffusion restriction at the level of the centrum semiovale and the right periventricular white matter.

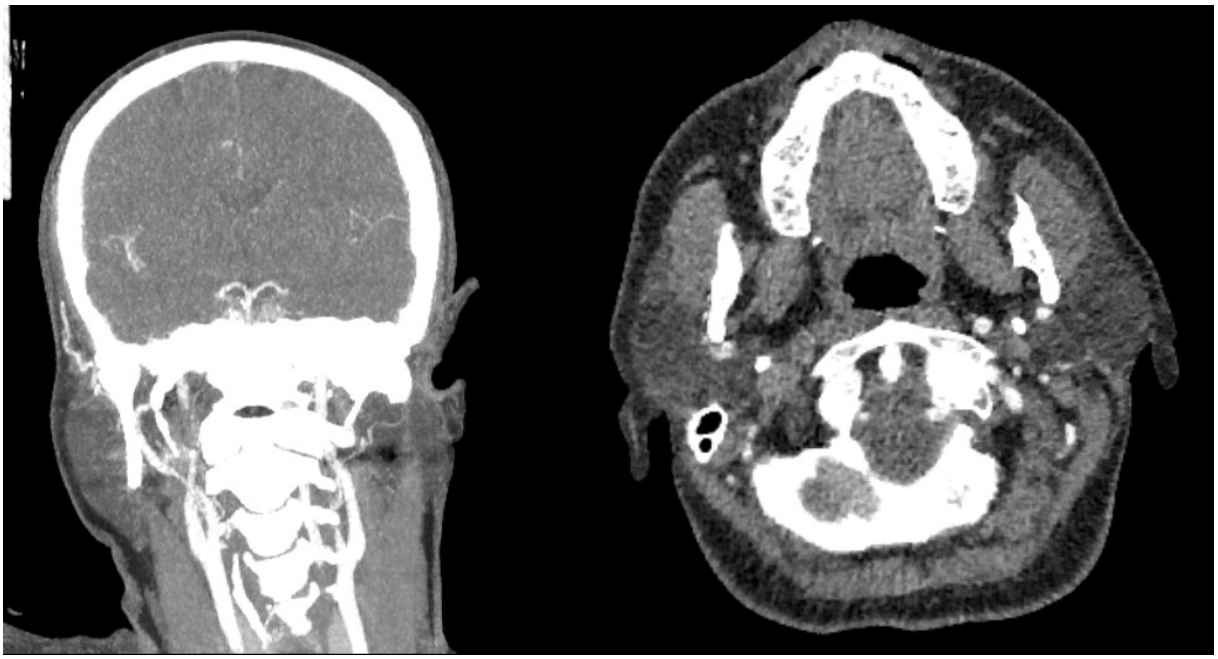

Figure 2. Thrombus in the right ICA.

It was decided that stent placement would not be necessary unless the patient had clinical deterioration and that dual antiplatelet therapy would be administered for 6 months

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Utilization of an Artificial Intelligence Chatbot in the Descriptive Statistics of an Emergency Medicine Study Descriptive Analysis of Emergency Department Data Using ChatGPT

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Introductıon

In scientific research, methodology and data analysis remain central and continually debated issues. Data analysis refers to the process of extracting meaningful results by processing data using appropriate statistical techniques. This process essentially begins with the classification of data obtained through research, followed by the application of suitable statistical methods. In statistical analysis, two fundamental approaches are employed: **descriptive statistics** and **inferential statistics** (1).

Descriptive statistics typically constitute the first step of data analysis, enabling researchers to gain a preliminary understanding of their data. It is a subfield of statistics that involves collecting, organizing, summarizing, and presenting data in a purely descriptive manner without relying on probabilistic processes. The aim here is not to generalize findings but to understand and identify patterns or issues in the dataset. In every research study, descriptive statistics are essential to explore, diagnose, and interpret the data (2). The purpose of descriptive statistics is to reveal the characteristics or features of a given population and to present them in a concise and meaningful format. This can be done through quantitative summaries or visual representations such as tables, counts, ranks, or graphs (3).

ChatGPT (Chat Generative Pre-trained Transformer), developed by OpenAI and released on November 30, 2022, is an artificial intelligence model based on a large language model. Initially launched as a free research preview, version 3.5 is currently available to all users free of charge, while the more advanced version 4.0 is offered through a subscription model. Trained on an extensive text corpus and continuously fine-tuned via user inputs, ChatGPT is capable of generating both text and images across a wide range of domains. Its applicability in the medical field is expanding, with new use cases being explored every day (4–6).

Although traditional statistical software packages have been used in medical research for years, the high cost of commercial tools has led to a growing interest in open-source alternatives. One such option is **Jamovi**, a free and open-source statistical package built on the R programming language. Alongside these, the proliferation of AI-powered tools—frequently introduced in new iterations—raises new questions about the potential and limitations of such technologies in medical statistics.

Large language model-based AI systems like ChatGPT mark the beginning of a new era in medical statistics. Whereas traditional statistical analysis often requires a certain level of programming knowledge, ChatGPT offers the possibility of performing statistical tasks using only natural language commands. For instance, a user might input, “Analyze the distribution of the age variable in my dataset,” and the model would return the appropriate statistics. Additionally, while working with large datasets is often time-consuming, ChatGPT can generate statistical summaries quickly, thereby saving researchers valuable time. It can instantly compile gender distributions, mean ages, and visit time categories into tables or charts.

Thanks to its advanced AI-powered data visualization capabilities, users can generate heatmaps, box plots, histograms, or word

clouds without requiring technical expertise. For example, a command such as “Create a heatmap based on patient visit hours” can prompt the model to process the data and return an appropriate visual. With its flexibility and user-friendly interface, ChatGPT also allows for quick data cleaning and recategorization of variables extracted from Hospital Information Management Systems (HIMS). For example, time-of-visit variables can be easily recoded into working hours, prime time, and night hours. Moreover, ChatGPT provides a low-cost and accessible alternative to commercial statistical software, requiring no installation or licensing. It can be used from any internet-connected device, streamlining the data analysis process in medical research.

AI models like ChatGPT have the potential to significantly accelerate, democratize, and simplify descriptive statistical workflows in medical research. By making statistical analyses more accessible, such technologies can help optimize time management—especially in fast-paced clinical fields like emergency medicine.

In this study, we aim to evaluate and illustrate the performance of the ChatGPT artificial intelligence model in conducting descriptive statistical analyses in medical research, supported by practical examples.

Materıals And Methods

This study was conducted with a retrospective design and involved the analysis of data from 53,308 adult patients who presented to the green zone of the emergency department of a tertiary care hospital in January 2025. Data were obtained through the Hospital Information Management System (HIMS) and recorded into an electronic spreadsheet. Following data cleaning, the analysis was carried out across seven key variables.

The dataset included the following variables for each patient: gender, age, day of the month, time of admission, case type, and diagnosis code. The gender variable was binary categorical (female, male), and frequencies and percentages were calculated. Age was treated as a discrete numerical variable, and its distribution was examined using a histogram. The Shapiro-Wilk test was applied to assess normality. For variables not normally distributed, the median and interquartile range (25th–75th percentiles) were reported, while for normally distributed variables, the mean and standard deviation were presented.

The time of admission was originally recorded in hour format and was categorized into multiple time segments. In the standard classification, time was divided into three groups: 08:00–15:59 (working hours), 16:00–23:59 (after hours), and 00:00–07:59 (night hours).

In the alternative classification, time was recategorized as: Working Hours (08:00–15:59), After Hours (16:00–19:59), Prime Time (20:00–23:59), and Night Hours (00:00–07:59).

The case type variable was reclassified by merging similar categories under five main groups: Emergency case, forensic case, occupational accident, traffic accident, exceptional condition.

Diagnosis codes were based on the ICD classification system. The most frequent diagnoses were grouped and analyzed accordingly.

Data analysis was conducted using ChatGPT Plus, the advanced GPT-4-based model developed by OpenAI. Responses generated by the AI model were processed using the Python programming language and relevant statistical libraries (e.g., pandas, scipy, matplotlib, seaborn).

Within the scope of descriptive statistics, gender distribution was calculated as frequency (n, %) and visualized using a pie chart. A histogram was used to evaluate the distribution and normality of the age variable. For time-based analyses, daily patient admissions were analyzed by gender, case type, and age group, and heatmaps were created to display time-of-admission patterns. In the diagnosis analysis, the top 20 and next 20 most frequent diagnosis codes were identified and visualized with horizontal bar charts. Additionally, diagnoses related to cardiac complaints were filtered and displayed via heatmaps across time categories.

A significance level of $p < 0.05$ was considered in all statistical analyses. The SciPy library in Python was used for normality testing.

This study was conducted retrospectively, and individual patient identities were protected. The data were anonymized and used solely for statistical purposes. Since the study involved only secondary, anonymized data and no patient intervention, ethical committee approval was not required. The primary aim of the study was to evaluate the potential use of artificial intelligence in descriptive medical statistics.

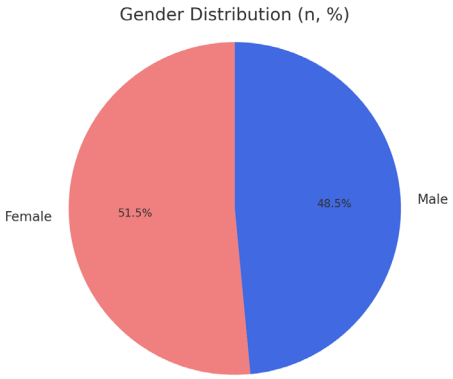
Results

Following the upload of the dataset into the artificial intelligence model, the first analysis request involved generating a **frequency distribution table for the gender variable**. The output is presented in **Table 1**.

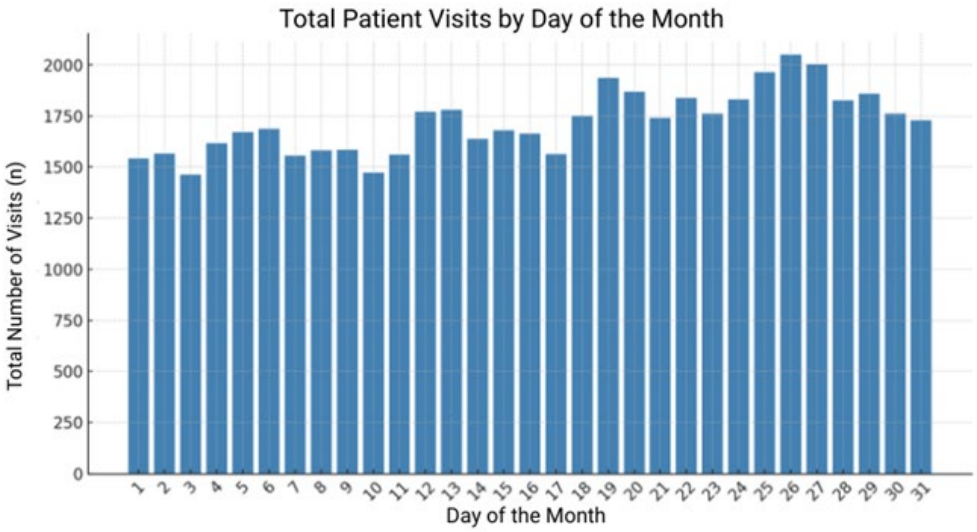
Table 1. Frequency distributions of genders.

Gender	**Frequency n (%)**
Female	27.438 (51.47)
Male	25.870 (48.53)

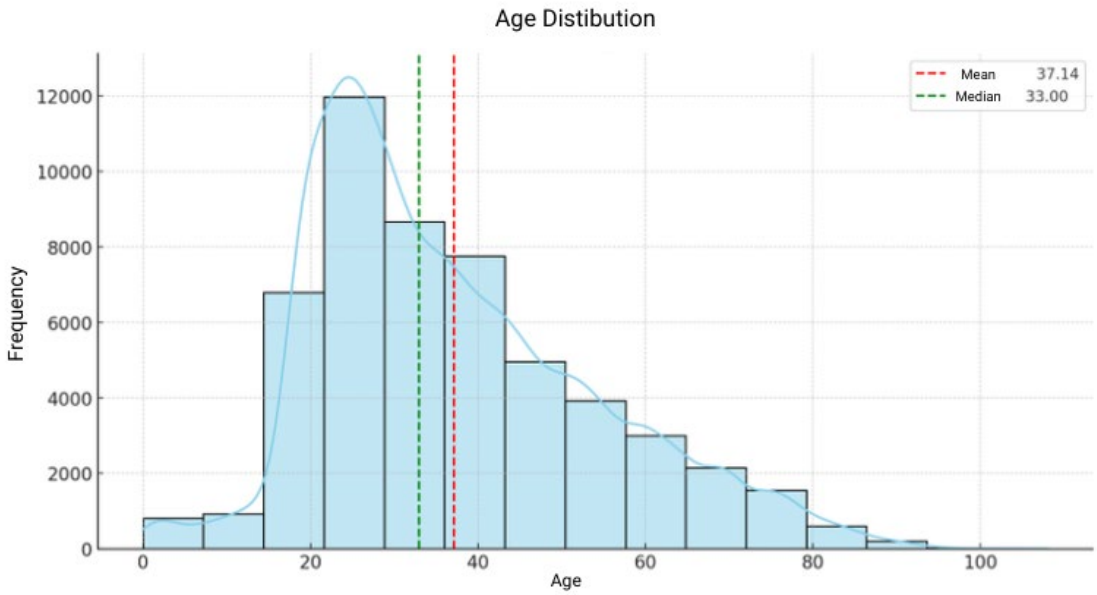
Subsequently, the distribution was requested to be visualized as a pie chart.



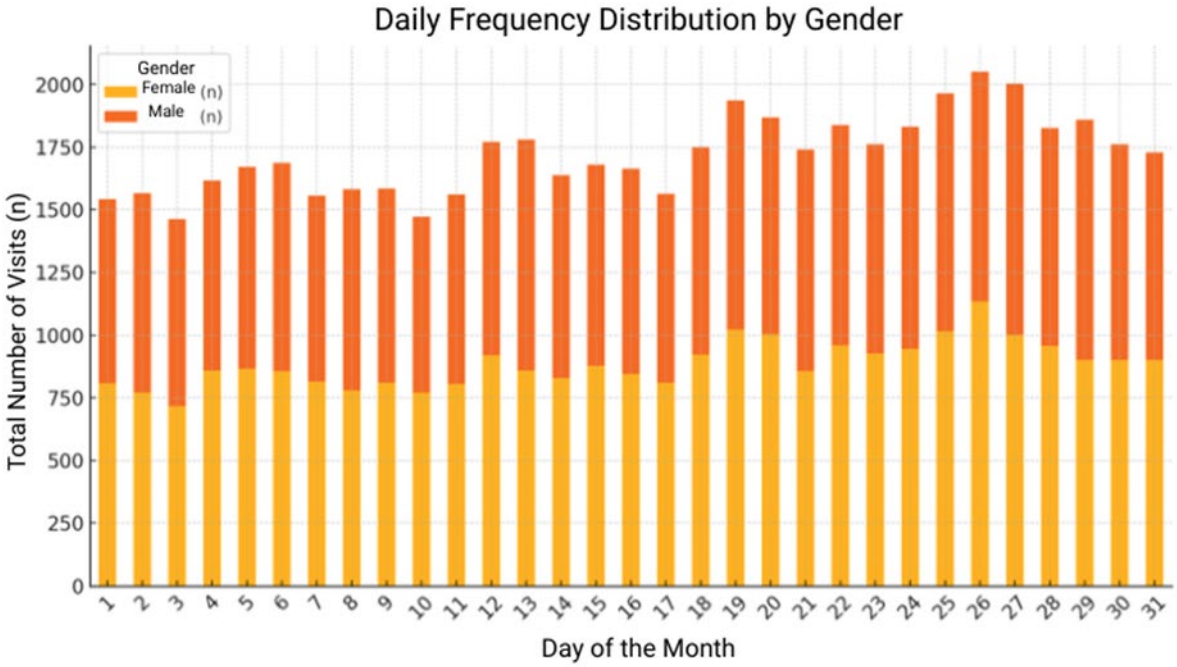
Next, the **daily number of patient visits throughout the month** was requested to be visualized using a **bar chart**.



Next, a **daily distribution table of patient visits by gender** was requested (**Table 2**).



Subsequently, a **bar chart** illustrating the **daily number of patient visits by gender** was requested.



Subsequently, a bar chart illustrating the daily number of patient visits by gender was requested.
Next, the model was asked to analyze the daily average number of patient visits. In the output, it was stated that, according to the Shapiro-Wilk test, the data followed a normal distribution ($p > 0.05$), and therefore, it would be appropriate to report the results using the mean and standard deviation: 1719.61 ± 153.76. Subsequently, a histogram of the age variable was requested.
The model was then asked to perform a normality analysis for the age variable and to determine appropriate descriptive measures accordingly. In its response, it stated that, based on the Shapiro-Wilk test, the assumption of normal distribution was not met ($p > 0.05$), and therefore, instead of using the mean, it would be more appropriate to report the median and interquartile range (IQR: 25th–75th percentiles). The output values were as follows: Median: 33.0 (IQR: 24.0–48.0).
Next, the model was instructed to reclassify the age variable into the following categories: 0–2 years, 2–6 years, 6–17 years, 18–64 years, and 65 years and older, and to generate a corresponding frequency table.

Table 2. Daily distribution of patient visits by gender**.**

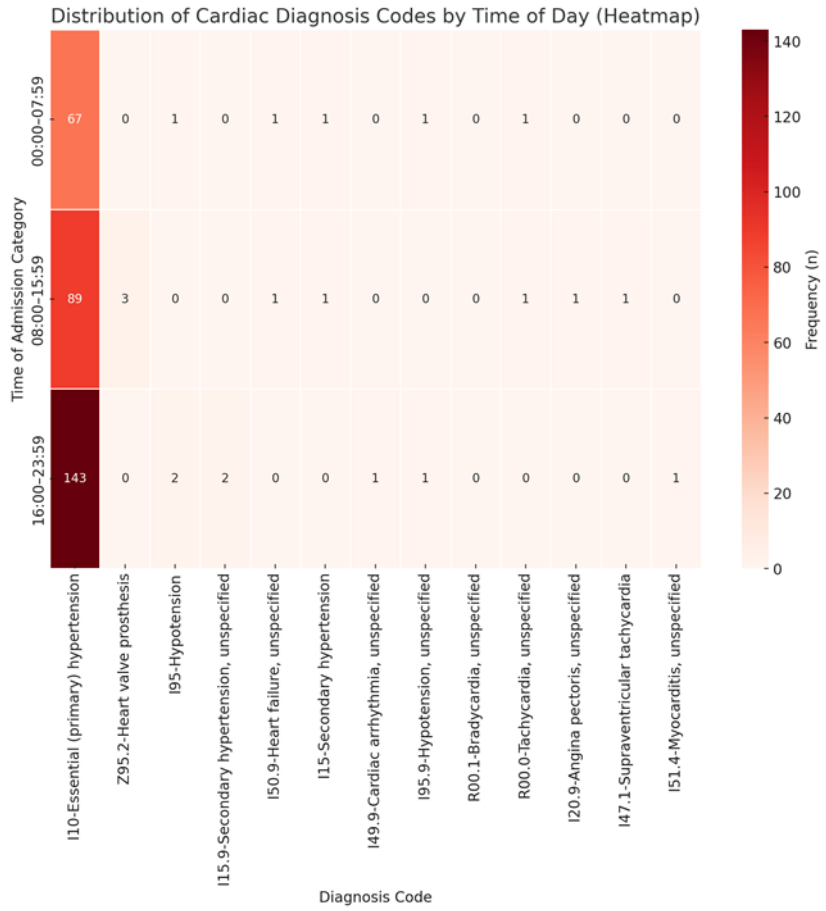
Day of the month	**Patient visits n (%)**	**Female n (%)**	**Male n (%)**
1	1.541 (2.89)	807 (52.37)	734 (47.63)
2	1.566 (2.94)	770 (49.17)	796 (50.83)
3	1.463 (2.74)	717 (49.01)	746 (50.99)
4	1.617 (3.03)	858 (53.06)	759 (46.94)
5	1.670 (3.13)	866 (51.86)	804 (48.14)
6	1.686 (3.16)	856 (50.77)	830 (49.23)
7	1.556 (2.92)	815 (52.38)	741 (47.62)
8	1.582 (2.97)	781 (49.37)	801 (50.63)
9	1.585 (2.97)	811 (51.17)	774 (48.83)
10	1.472 (2.76)	771 (52.38)	701 (47.62)
11	1.560 (2.93)	805 (51.6)	755 (48.4)
12	1.770 (3.32)	919 (51.92)	851 (48.08)
13	1.780 (3.34)	860 (48.31)	920 (51.69)
14	1.637 (3.07)	828 (50.58)	809 (49.42)

15	1.679 (3.15)	877 (52.23)	802 (47.77)
16	1.664 (3.12)	844 (50.72)	820 (49.28)
17	1.563 (2.93)	810 (51.82)	753 (48.18)
18	1.749 (3.63)	922 (52.72)	827 (47.28)
19	1.936 (3.63)	1.022 (52.79)	914 (47.21)
20	1.869 (3.51)	1.003 (53.67)	866 (46.33)
21	1.741 (3.27)	857 (49.22)	884 (50.78)
22	1.837 (3.45)	959 (52.2)	878 (47.8)
23	1.762 (3.31)	926 (52.55)	836 (47.45)
24	1.830 (3.43)	945 (51.64)	885 (48.36)
25	1.964 (3.68)	1.016 (51.73)	948 (48.27)
26	2.051 (3.85)	1.133 (55.24)	918 (44.76)
27	2.003 (3.76)	1.001 (49.98)	1.002 (50.02)
28	1.826 (3.43)	957 (52.41)	869 (47.59)
29	1.860 (3.49)	900 (48.39)	960 (51.61)
30	1.761 (3.3)	902 (51.22)	859 (48.78)
31	1.728 (3.24)	900 (52.08)	828 (47.92)
TOTAL	53.308 (100)	27.438 (51.47)	25.870 (48.53)

Table 3. Distribution of age groups.

Age Group	**Frequency n (%)**
0-2 age	234 (0.44)
2-6 age	396 (0.74)
6-17 age	1488 (2.79)
18-64 age	46.314 (86.88)
65+ age	4.876 (9.15)

Subsequently, the values in the admission time column of the dataset were requested to be categorized into three groups: 08:00–15:59, 16:00–23:59, and 00:00–07:59. Based on these categories, the model was then instructed to generate a bar chart showing the daily number of patient visits for each time group.

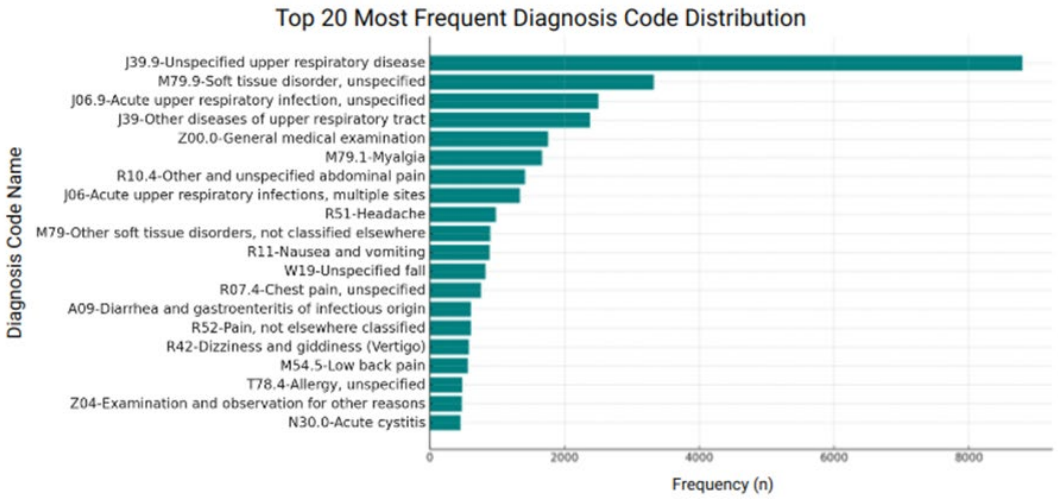


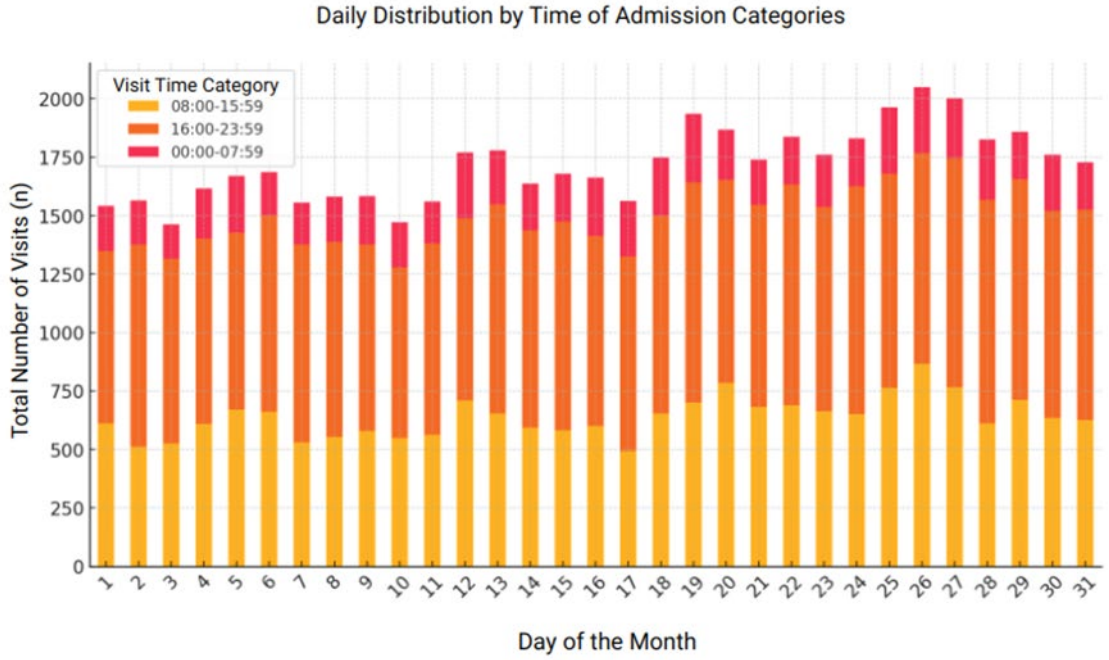
Next, a distribution chart for the Diagnosis Codes variable was requested. As output, a chart displaying the "Top 20 Most Frequent Diagnosis Codes" was provided.

When a line chart was initially requested for the most frequent diagnosis codes related to cardiac complaints, the model instead recommended using a heatmap to visualize the distribution of cardiac diagnosis codes across time intervals based on color intensity. Finally, the admission time variable was requested to be recategorized into the following new time intervals: Working hours (08:00–16:59), After hours (16:00–19:59), Prime time (20:00–23:59), and Night hours (00:00–07:59). Based on this new classification, a heatmap illustrating the daily number of patient visits according to these time categories was generated.

Discussion

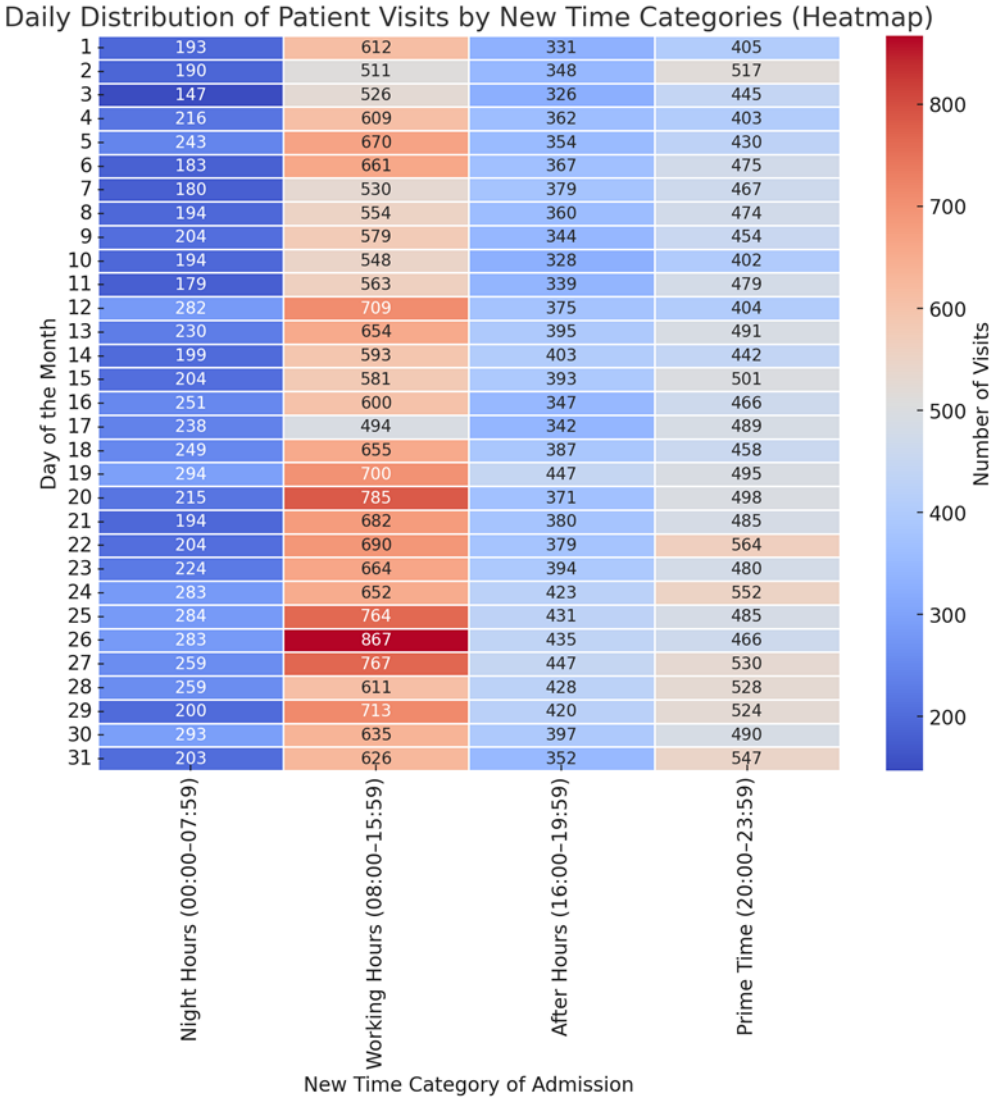
Ronald Fisher, one of the most renowned and influential figures in the field of statistics, emphasized in his seminal work On the Mathematical Foundations of Theoretical Statistics that the primary goal of statistical methods is to simplify data. According to Fisher, a dataset should be summarized using a minimal number of measures that sufficiently represent the whole. This perspective reflects the fundamental principle of descriptive statistics, which helps researchers understand the structure of datasets and provides various tools for data visualization (7, 8).





In medical research, data from hundreds or even thousands of observational units—such as patients, animals, cells, or other biological samples—may be collected, yet researchers typically focus on a limited set of parameters. Data exploration and summarization constitute a core stage in the statistical analysis process, aiming to accurately describe and summarize the data, thereby highlighting its essential features. Descriptive statistical methods offer a general overview of the analyzed sample by summarizing parameters and visualizing them. The fundamental properties of large datasets can be communicated in a simplified manner using a few statistical measures or visual tools. During this process, researchers identify the appropriate parametric model that best represents the data distribution, thus laying the groundwork for subsequent hypothesis testing and analytical methods. Additionally, the primary goals of descriptive statistics include analyzing the research data, determining the distribution, identifying possible errors and outliers, and evaluating missing data and potential biases. Overall, presenting the data in a clear and comprehensible manner demonstrates that the researcher has collected, analyzed, and interpreted the data accurately and objectively (8).

The first step in the data analysis process is to evaluate whether the assumptions underlying hypothesis testing are met. If the dataset fails to meet these assumptions, the validity of the statistical analyses may be compromised, leading to inaccurate findings and misinterpretation. Therefore, the suitability of the data distribution should be carefully examined prior to analysis (9).



Most robust statistical tests rely on the assumption of normal distribution. For this reason, before conducting any statistical analysis, the distributional properties of the data must be thoroughly assessed (9). In our study, when we prompted the AI model to evaluate the normality of our numerical variables, it responded appropriately by generating histograms and applying the Shapiro-Wilk test to assess distributional assumptions. As a result, the model reported median and interquartile ranges for non-normally distributed variables, and mean and standard deviation for those that followed a normal distribution.

In our study, the AI model successfully analyzed a dataset of over 53,000 entries within a short period, accurately executing the requested prompts and presenting the results in the form of tables and charts. The analysis process was carried out smoothly on a standard hospital computer running a Linux-based operating system, without encountering any freezes, crashes, or errors. Moreover, the browser-based nature of the AI model eliminates the need for installing additional software, enhancing user accessibility.

One notable advantage of ChatGPT is its ability to interpret instructions and propose alternative solutions, demonstrating its potential for use in medical statistics. Its strength in data visualization, in particular, can assist researchers and authors in presenting their findings more effectively and comprehensibly. Visualization techniques suggested by the model—such as bubble charts, word clouds, Pareto charts, heatmaps, and box plots—may significantly enhance the presentation quality of future research.

The descriptive statistical analyses and visualizations presented in this study serve merely as examples. Researchers can utilize AI-powered systems like ChatGPT to perform more detailed analyses and generate numerous tables and figures across various data subsets. However, the method also has certain limitations and potential drawbacks that must be considered. While AI models like ChatGPT facilitate the statistical analysis process, they operate within defined boundaries. The model's capabilities are mostly limited to basic descriptive statistics, and its reliability for advanced statistical modeling—such as regression analysis, hypothesis testing, or ANOVA—remains uncertain. Therefore, relying solely on AI for studies requiring inferential statistics may be misleading.

Another important concern is the possibility of errors generated by AI. ChatGPT may occasionally produce incorrect or misleading outputs. For example, it might apply an inappropriate normality test or misinterpret the results. Such mistakes can directly compromise the accuracy of the analysis and lead to erroneous scientific conclusions. Researchers should always manually verify and

cross-check the results obtained through AI.

Moreover, data privacy concerns must not be overlooked when using AI-based tools. Since large language models operate on cloud-based systems, there is potential risk in processing sensitive patient information. Relying on AI-driven analysis without ensuring data security could raise ethical issues and lead to violations of patient confidentiality. It is therefore essential to use anonymized data and to comply fully with legal regulations concerning data sharing and privacy.

Lastly, the accessibility of such systems is not equal for all researchers. Advanced versions of ChatGPT (such as GPT-4) require a paid subscription, which may raise questions about cost-effectiveness compared to open-source statistical software like R or Python. Furthermore, using AI-based analysis tools may still require a basic level of technical knowledge. Without fundamental statistical literacy, users may misinterpret the outputs generated by the model, leading to incomplete or incorrect decisions.

Conclusıon

While AI-powered tools such as ChatGPT offer fast and practical solutions in medical statistics, their limitations and potential risks should not be overlooked. Although the analyses generated by artificial intelligence can save time for researchers, they should be considered solely as supportive tools, and all results must be independently verified using conventional statistical methods.

Given the limitations related to data security, error potential, and statistical modeling, it is essential to maintain a critical perspective to ensure scientific rigor. On the other hand, the emerging concept of "prompt engineering", a likely profession of the future, is something that we, as physicians and researchers, should not remain distant from. To make the most of the potential offered by AI technologies in medical data analysis, we must approach these systems with both awareness and critical thinking.

As the digital transformation continues to accelerate in clinical and academic domains, physicians and researchers should not only observe but actively participate in shaping this new era. When used appropriately, AI-assisted statistical tools can enhance research productivity and contribute meaningfully to the advancement of medical science. However, it is imperative to operate with a clear understanding of their capabilities and boundaries, as this is essential for maintaining scientific accuracy and integrity.

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The Role Of Socıal Medıa In Medıcal Educatıon And Traınıng

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Introductıon:

It is obvious that social media is with us at every moment of our lives in this information and information age. Although social media is generally thought to be used for entertainment, it is possible to access different content in every field of education on many platforms (1,2). SM, whose use is increasing in every sector today, offers great convenience and opportunities to users in the health sector (3).

Materıal-Methods:

The study was conducted on medical faculty students at XXX university and the contribution of SM to medical education was investigated. The participants were asked a questionnaire created by the researchers, which lated approximately 5 minutes.

Results:

In the study with 541 participants, only 9 (1.7%) of the participants stated that they did not use social media. The study was completed with 532 (98.3%) participants who reported using social media.

63.3% of the students stated that they had received online education before their university education. While 31.1% of the participants were not satisfied with online education, 29.9% of them were undecided about their satisfaction with online education. While 48.4% of the students preferred a system that combines classical and online education, 45% did not prefer such a hybrid system.

70.3% of the participants thought that SM has an impact on medical education. Students stated that they felt the need to research interventional procedures (69.9%) and physical examination (56.6%) the second most on SM.

318 of the students (60.6%) stated that they needed to confirm the information from the source books related to that subject.

Discussion:

Today, with the effect of the digital transformation in the world, there is almost no area where social media is not used. When the literature is examined, it can be said that social media is becoming an indispensable part of life day by day (4,5). In order to add a different dimension to education with the help of technology in the coming years, it will be among the main goals of educational institutions to increase the experiences and skills of instructors related to online education and to ensure that students can participate in their education comfortably and disciplined.

Conclusion:

With the impact of developing technology, social media has become an important part of information acquisition and medical education. It should be aimed to make the use of SM a part of medical education by eliminating the barriers due to information pollution in SM, increasing the experience and skills of trainers related to online education.

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Fascicular Ventricular Tachycardia Confused with Supraventricular Tachycardia: A Case Report

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Introduction

Idiopathic ventricular tachycardia (VT) is defined as episodes of VT in the absence of structural heart disease as evidenced by electrocardiography (ECG), echocardiography (ECHO) and coronary angiography. Idiopathic VTs are classified as outflow tract tachycardias and left ventricular fascicular tachycardias (LVFTs) (1).

Emergency department (ED) physicians may have difficulty recognising and treating VT and its subtypes. This is because supraventricular tachycardia (SVT) with conduction abnormalities can result in large complex tachycardias. In addition, fascicular VT, a subgroup of idiopathic VT, can be confused with typical VT or SVT. The diagnosis of fascicular VT is important in terms of treatment and prognosis (2). Here, we aim to present a case of fascicular VT that was initially thought to be SVT and was treated, but with no response.

Case Presentation

A 27-year-old male patient presented to A&E with palpitations. He stated that he had undergone ablation with a diagnosis of SVT, was not taking any medication and had no co-morbidities. On presentation to the emergency department, blood pressure was 130/70 mm/Hg, pulse rate 166/min, temperature 36°C and saturation 96% (room air). The physical examination was normal. The ECG showed a heart rate of 166/min, QRS segment (140 ms), extreme axis deviation and right bundle branch block (RBBB) (Figure 1). Vagal manoeuvres, adenosine (6,12,18 mg intravenous (IV)), metoprolol (5 mg IV) and amiodarone (300 mg IV) were administered according to the narrow QRS tachycardia algorithm. There was no decrease in heart rate after these treatments. ECHO performed in the emergency department was normal. Laboratory results were normal. As atrioventricular (AV) dissociation and capture beats were also present on the ECG, fascicular VT was considered. Diltiazem 25 mg (IV push) was administered in the emergency department. After obtaining consent from the patient whose heart rate did not return to normal, electrical cardioversion was performed with 50, 100, 150 joules under sedation. After all these treatments in the emergency department, the patient whose heart rate could not be controlled was admitted to the coronary intensive care unit and verapamil (5 mg intravenously) was administered. Normal sinus rhythm was achieved after verapamil. After a period of follow-up in the coronary ICU, verapamil and propafenone HCL were prescribed and the patient was discharged (Figure 2).

Discussion

Idiopathic LVFT is divided into three subgroups according to ECG findings: 1) left posterior fascicular VT with left axis deviation and RBBB, 2) left anterior fascicular VT with right axis deviation and RBBB, 3) upper septal fascicular VT with normal axis and narrow QRS (1). Cardiomyopathy has been described in 6% of cases and is usually reversible after successful ablation. While most occur at rest, exercise, stress and catecholamine infusion may be precipitating factors (3). The most common type is fascicular VT associated with RBBB, as seen in our case. It is often seen in young (15-40 years) men without structural heart disease (4).

Fascicular VT is a difficult arrhythmia to recognise as it can mimic both SVT and VT. On the 12-lead ECG, capture/fusion beats and AV dissociation may be seen, suggesting a diagnosis of VT rather than SVT (5). In contrast to VT, in fascicular VT the RS interval is usually <80 ms and the QRS duration varies between 100 and 140 ms. ECG findings may vary depending on the origin of the tachycardia (3,6). Fascicular VT may be confused with SVT, which can lead to difficulties in diagnosis and delays in treatment (7).

Fascicular VT, which has a re-entry mechanism in Purkinje fibres, is also known as verapamil-sensitive VT (2). In the treatment of fascicular VT, vagal manoeuvres, adenosine and propanolol are generally ineffective. Amiodarone has been shown to reduce tachycardia but not to restore sinus rhythm (3). Radiofrequency ablation has been reported to be successful in selected patients (6). Many case reports have reported that verapamil is the most appropriate treatment, but should not be used in the setting of AS (2,3,7). In our case, other antiarrhythmic treatments were ineffective and sinus rhythm was achieved with verapamil treatment.

Figure 1

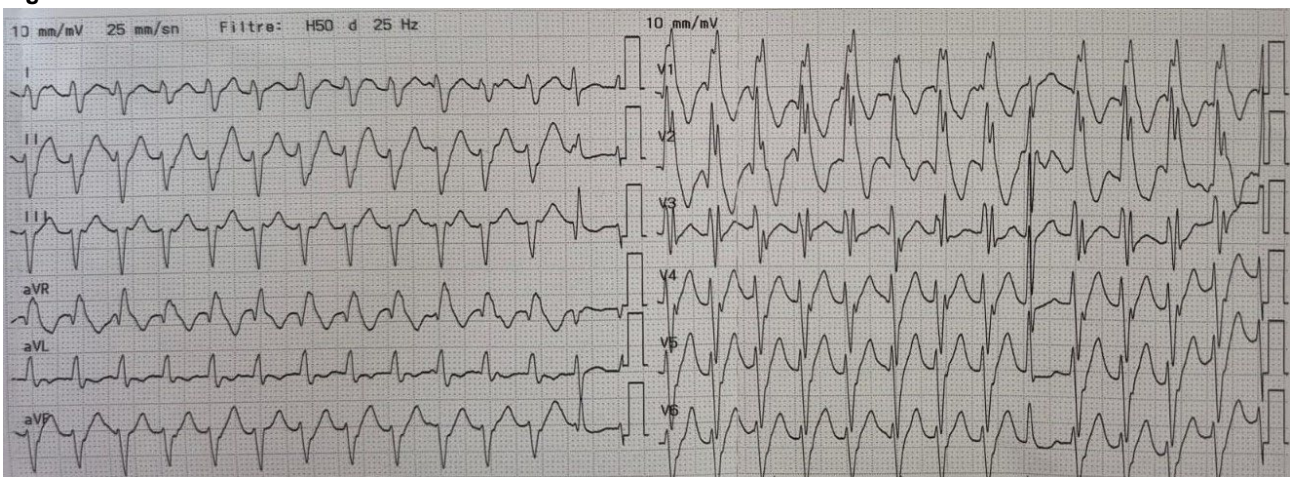
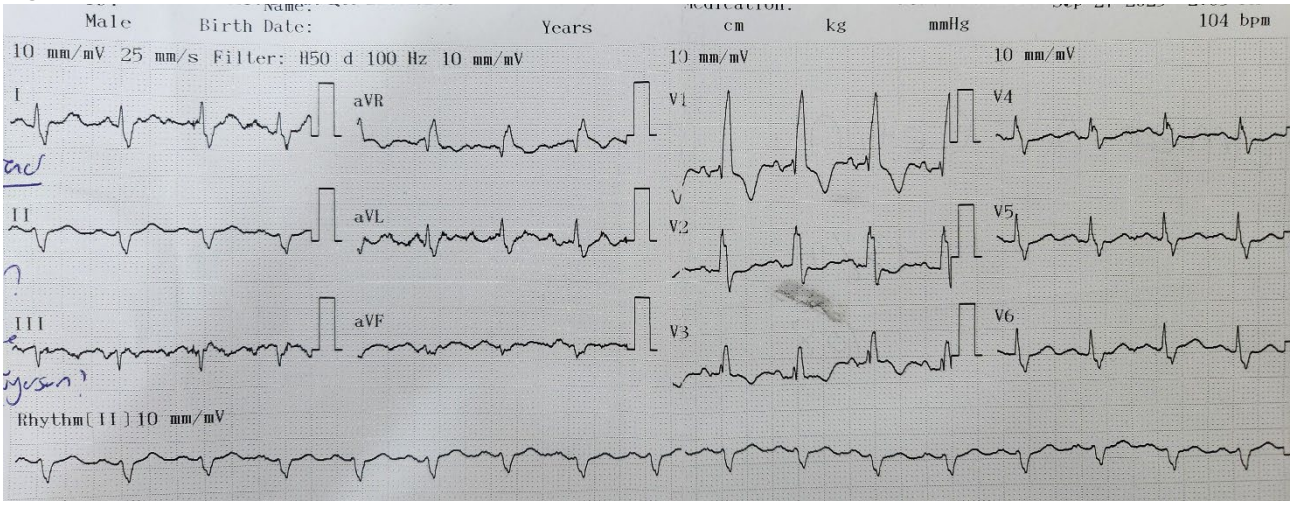


Figure 2



Conclusion

Fascicular VT is an idiopathic arrhythmia that is often confused with SVT and does not respond to standard treatment. In the setting of ED, fascicular VT should be considered in the presence of refractory tachycardia accompanied by RBBB and left axis deviation on the ECG

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6117

Adenosine and Ventricular Tachycardia in Surgically Corrected Congenital Heart Disease: A Potential Risk?

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Karamanoglu Mehmetbey University

Introduction:

Supraventricular tachycardia is defined as atrial rates higher than 100 beats per minute originating from tissues at the His bundle and above. Traditionally, all types of tachycardias, except for ventricular tachycardia and atrial fibrillation (AF), are classified as SVT (1). The overall incidence has been found to be 1.03 per 1,000 patient-years. Major congenital heart diseases and cardiomyopathies are associated with a higher incidence and increased mortality rates (2). Vagal maneuvers and adenosine play a crucial role in both the treatment and diagnosis of SVT, particularly in cases where the ECG findings are inconclusive (1).

On the other hand, transposition of the great arteries (TGA) is a congenital cardiac malformation characterized by atrioventricular concordance and ventriculoarterial discordance. The incidence of TGA is estimated to be 1 in 3,500–5,000 live births, with a higher prevalence in males (3). Currently, the arterial switch operation is the preferred treatment, while the Senning and Mustard procedures were formerly used for correction (4). In patients who have undergone Mustard or Senning repairs, atrial re-entrant tachycardias are common, and the use of antiarrhythmic medications is limited due to proarrhythmic risks (5).

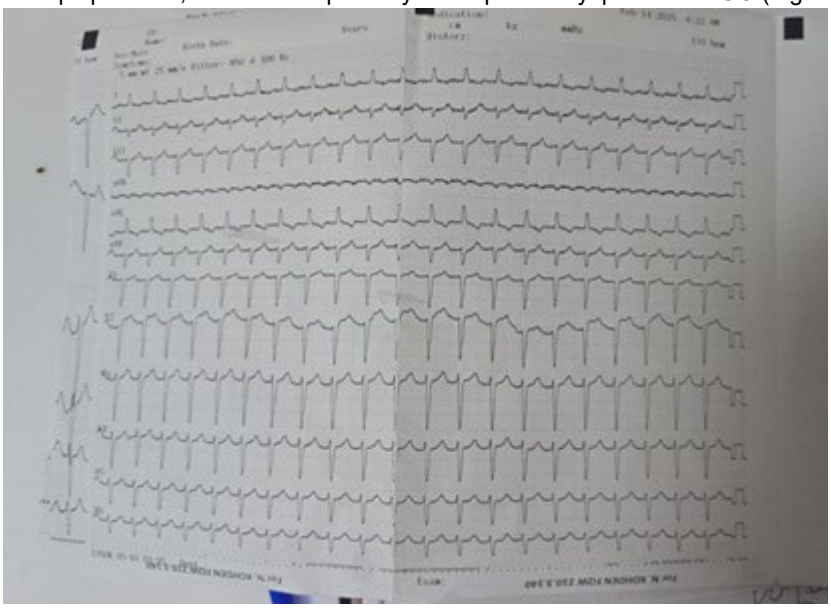
Although adenosine has been reported to induce ventricular arrhythmias in some patients, particularly in those with syndromes such as Wolff-Parkinson-White (WPW), its risks and associations with congenital heart diseases remain poorly understood (6,7).

In this case report, we describe a 28-year-old patient with a history of transposition of the great arteries correction surgery who developed monomorphic ventricular tachycardia following adenosine administration.

Case Report:

A 28-year-old male patient presented to the emergency room with a complaint of palpitation. Due to transposition of the great arteries, ventricular septal defect, and pulmonary atresia, he had corrective surgery at the age of nine. The patient has a history of pulmonary embolism – once five years ago and most recently one week ago, which was diagnosed with segmental emboli and resulted in his admission and discharge two days before his presentation. He was prescribed warfarin 5 mg once daily, enoxaparin 6000U twice daily, and amiodarone 200 mg twice daily. However, the patient had not been using amiodarone consistently. He did not have any known rhythm disorder and was regularly followed by the cardiology department.

On physical examination, the patient was distressed. He was alert and cooperative, with a Glasgow Coma Score of 15. His heart rate was 131, blood pressure was 126/84, respiratory rate was 18 per minute, and his oxygen saturation was 90% on room air. Apart from palpitations, he did not report any chest pain or dyspnea. An ECG (Figure 1) was then performed.

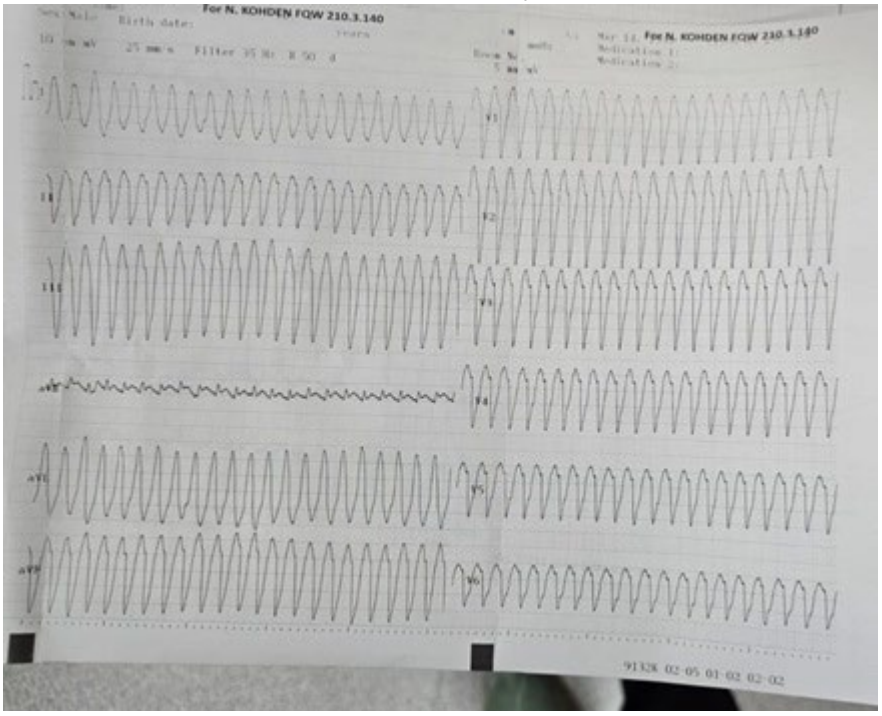


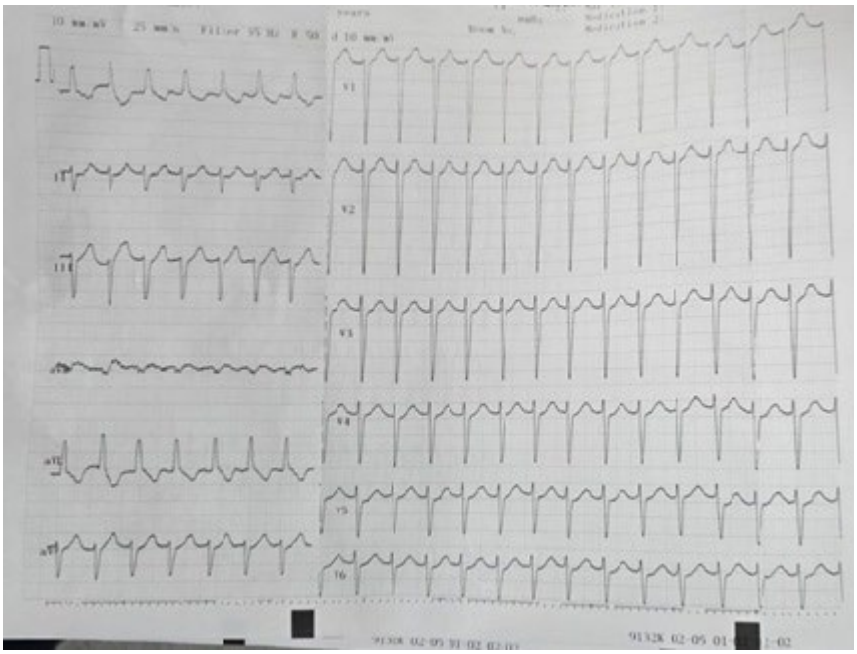
His ECG was evaluated as supraventricular tachycardia, and the patient's vitals were monitored, including a defibrillator setup. Vagal maneuver and carotid massage started without any clinical response. Subsequently, six mg of adenosine was administered intravenously. Following that, his heart rate increased, and his heart rhythm progressed to monomorphic ventricular tachycardia (Figure 2).

A quick ABC assessment was applied, and the patient was found to be stable with a pulse, remaining conscious with a GCS score of 15. His vital signs remained stable, with a blood pressure of 115/72. A 300 mg amiodarone infusion was started, and three minutes after the onset of ventricular tachycardia, it resolved. (Figure 3).

His FBC and biochemistry panel were within reference ranges and showed no significant findings. His troponin I level was negative (<10). In his venous blood gas analysis, the pH was 7.354, pCO□ was 44.3 mmHg, HCO□ was 24.7 mmol/L, and his lactate level was 1.52 mmol/L. His INR was 1.60, APTT was 28.9 seconds and PT was 16.1 seconds. After the patient stabilized, he was admitted to the cardiology department for monitoring, and his amiodarone therapy was continued as maintenance. During his admission,

he did not show any VF or VT rhythms, and his troponin levels remained stable. Also, enoxaparin was administered because the INR was not at the required level. Due to his arrhythmia, the patient was transferred to another hospital for ablation treatment.





Discussion:

In the surgical correction of the transposition of the great arteries, the Atrial Switch Procedure (Mustard or Senning operation) was the gold standard for years. As a result of this procedure, the morphological right ventricle supports systemic circulation. RV failure and tricuspid regurgitation are common in these patients. Sinus node dysfunction and atrial arrhythmias are frequent. Associated defects, such as abnormalities of the tricuspid valve, ventricular septal defect, and pulmonary stenosis, are present in most patients (8). Our patient also had a ventricular septal defect and pulmonary atresia. Due to scarring and altered anatomy, atrial re-entrant tachycardias are common in these patients. However, the exact mechanisms behind this phenomenon remain unknown (5).

In cases of narrow QRS SVT, vagal maneuvers are the first-line treatment, stimulating the vagus nerve, which causes the release of acetylcholine and slows the heart rate (9). After that, adenosine, an endogenous purine nucleoside, is the preferred first-line drug for treating narrow complex SVT, even when a clear ECG-based diagnosis is unavailable (1). It prolongs AV conduction, leading to a transient AV block, which results in the termination of tachycardia (10). Adenosine has some absolute contraindications. It is not

recommended for patients with hypersensitivity to adenosine, heart block, or clinically active bronchospasm (11). In SVT cases involving an accessory pathway, such as in Wolff-Parkinson-White (WPW) syndrome, extreme caution is required. Blocking the AV node may result in enhanced conduction from the atria to the ventricles via the accessory pathway because adenosine does not affect these pathways. In such cases, adenosine use can trigger severe tachycardia and even cardiac arrest (12). Ibutilide and procainamide are preferred alternatives to adenosine in AVRT patients (1).

In our case, the reason for the progression to ventricular tachycardia after adenosine remains unclear. It is known that scar tissue and congenital heart diseases following surgery can contribute to reentrant tachycardias (13). However, there is limited data on the acute management of tachycardias in congenital heart disease patients. Differences in surgical approaches and the complex mechanisms involved in reentrant tachycardias make this phenomenon even more complicated (8,13).

We suggest that further investigations and studies should focus on this issue to establish a standardized approach for managing these patients. Due to the altered conduction pathways and scarring from prior surgeries, these patients may exhibit unpredictable responses to standard SVT treatments, potentially leading to life-threatening arrhythmias. Like WPW syndrome, a tailored risk assessment should be considered before administering AV nodal blocking agents. Future studies should evaluate whether alternative antiarrhythmic strategies or electrophysiological mapping techniques could help prevent adverse outcomes in this high-risk population.

Conclusion:

Managing SVT in patients with surgically corrected congenital heart disease is challenging due to altered anatomy and arrhythmogenic structure. Our case highlights the risk of unexpected ventricular tachycardia following adenosine administration, underscoring the need for caution. Like WPW syndrome, alternative treatments like procainamide or ibutilide may be safer in these patients. Further research is needed to establish clear guidelines for acute arrhythmia management in this high-risk group.

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Subarachnoid Hemorrhage Presenting with Acute ST-Segment Elevation: A Neurocardiological Case Report

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Introduction

Subarachnoid hemorrhage (SAH), a life-threatening neurological condition, is commonly due to cerebral aneurysm rupture. It accounts for approximately 5% of all strokes but contributes disproportionately to stroke-related deaths and long-term disability [1]. Prompt diagnosis and intervention are crucial in determining outcomes. Although the hallmark symptoms of SAH include sudden-onset severe headache, vomiting, and decreased consciousness, it may also present with systemic manifestations, including elect-

rocardiographic (ECG) abnormalities and elevated cardiac enzymes, thereby mimicking acute coronary syndromes (ACS), complicating the initial diagnostic process [2,3]. Several case reports and clinical studies have documented ECG abnormalities in patients with SAH, including ST-segment elevation, QT prolongation, and T-wave inversion. According to current literature, neurogenic stunned myocardium, caused by excessive sympathetic activation and a spike in catecholamines, is responsible for these misleading cardiac manifestations [4,5].

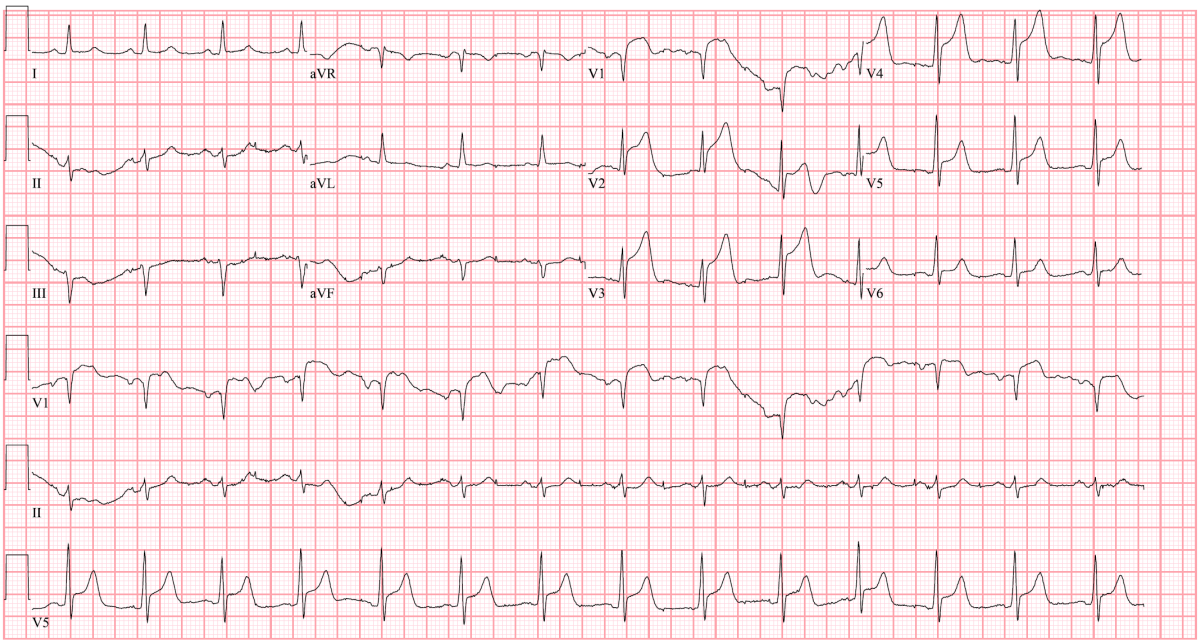
This case report examines a complex clinical scenario involving a 60-year-old male who initially presented with anterior ST-segment elevations consistent with acute anterior MI, underwent percutaneous coronary intervention (PCI). Subsequently, neurological symptoms emerged, revealing underlying SAH. This case highlights the critical neurocardiac interactions and emphasizes the importance of recognizing SAH in differential diagnoses of acute cardiac events.

Case Presentation

A 60-year-old male with hypertension and coronary artery disease presented to the emergency department with sudden-onset syncope. His chronic medications included low-dose aspirin (Coraspirin) and clopidogrel (Plavix). On examination, he was conscious and oriented. Vital signs on admission were notable for a blood pressure of 160/90 mmHg, pulse rate of 110 beats per minute, and oxygen saturation of 96% on room air. An initial ECG showed significant anterior ST-segment elevation, consistent with acute anterior ST-elevation myocardial infarction (STEMI) **(Figure 1).** Laboratory evaluation revealed significantly elevated high-sensitivity troponin levels (10,000 ng/L; reference: 0–14) and D-dimer levels (3,007 µg/L; reference: 0–500). Based on these findings, immediate admission to the cardiology department was arranged for emergent coronary angiography. The Angiography showed stenoses secondary to previous myocardial infarction. Percutaneous coronary intervention (PCI) was performed successfully, and the patient was subsequently admitted to the intensive care unit for post-procedural monitoring.

Within hours of the procedure, the patient developed a severe, sudden-onset headache accompanied by nausea and vomiting. Neurological examination revealed progressive confusion. A non-contrast cranial computed tomography (CT) was performed and indicated a subarachnoid hemorrhage characterized by increased density in the basal cisterns, due to cerebral aneurysmal rupture, and the patient was consulted to neurosurgery **(Figure 2a).** Hydrocephalus was detected during follow-up imaging, prompting external ventricular drainage (EVD) **(Figure 2b).** In subsequent imaging after EVD, the development of acute subdural hematoma has been documented as a complication that is believed to be associated with heparinized angiography. Emergency evacuation of hematoma and decompressive craniectomy was performed afterwards. **(Figure 2c).** Despite these interventions, postoperative intensive care management was complicated by hemodynamic instability requiring vasopressor support, and the patient's condition deteriorated progressively, leading to his death from hemodynamic and neurological decline.

Figure 1. ECG showing ST-segment elevation in leads V1–V5, initially interpreted as anterior STEMI.



Discussion

This case illustrates the complex pathophysiological interaction between acute neurological events and cardiac manifestations, often referred to as the brain-heart axis. SAH-induced ECG alterations, including ST elevation, QT prolongation, and T-wave inversion, are common yet challenging to distinguish from ACS [2,3,8]. These abnormalities are related to the abrupt release of catecholamines, which results in myocardial dysfunction and potential arrhythmias [4,5,9]. In SAH cases, these changes are typically diffuse and fluctuate significantly with neurological status, unlike the localized and consistent patterns seen in myocardial infarction [3,5]. Neurogenic stunned myocardium from intense catecholamine release is the accepted pathophysiological mechanism behind SAH-induced ECG changes [4,9].

Awareness of SAH is critical, particularly in atypical presentations or altered mental status, to prevent diagnostic delays [1,9]. Antithrombotic therapy, though standard in managing ACS, can exacerbate intracranial hemorrhage, leading to fatal complications as observed in this case. Thus, it is critical to maintain a high index of suspicion for SAH in any patient with unexplained neurological signs, especially when the cardiac picture is ambiguous or evolving. In our case, invasive cardiac procedures based on initial ECG findings were complicated by the subsequent discovery of SAH. The emergence of complications like hydrocephalus and subdural hematomas considerably exacerbates the prognosis and necessitates immediate multidisciplinary intervention [1,7]. Management challenges involve balancing cardiac and neurological needs.

Conclusion

SAH can present with ECG abnormalities indistinguishable from AMI. Clinicians should consider the possibility of SAH in patients presenting with cardiac symptoms and exhibit atypical neurological findings. Prompt recognition and multidisciplinary management are essential to improving clinical outcomes.

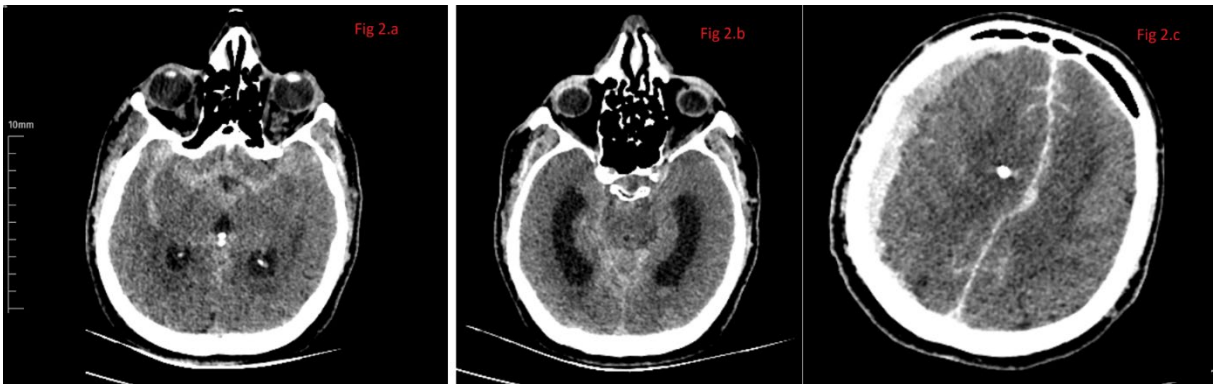


Figure 2. CT scan showing subarachnoid hemorrhage, hydrocephalus, and subdural hematoma post-intervention. (a) Cranial CT scan showing subarachnoid hemorrhage in the basal cisterns; (b) Ventricular enlargement indicating hydrocephalus requiring EVD; (c) Post-EVD imaging showing acute subdural hematoma.

Keywords

Subarachnoid hemorrhage, electrocardiographic anomalies, ST-segment elevation, myocardial infarction, neurocardiology

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6150

A Rare Postoperative Complication: Hematoma and Abscess of the Falciform Ligament

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Introduction

Falsiform ligament (FL) pathologic lesions were first described in 1909 [1]. Cyst, tumor, inflammation and necrosis due to omphalitis in infants, abscess that may occur after biliary obstruction and infection in adults, hematoma that develops after antiaggregant drug use or operation are among the pathologies that can be seen in the FL. Clinically, it may present with symptoms and signs such as right upper quadrant pain, mass, fever, obstructive jaundice and portal pyemia. FL hematoma or abscess is a rare clinical entity and

has only been reported in a few cases in the literature [2,3]. In this article, we aimed to present our case of postoperative FL hematoma and abscess with imaging findings since it is unique in the literature to our knowledge.

Case

A 58-year-old male patient presented to the emergency department of our hospital with epigastric pain. He had no history of drug use and had undergone umbilical hernia surgery three days before. Complete blood count and biochemical laboratory tests were normal. Physical examination revealed tenderness in the epigastric region. There was an operation-related scar in the umbilical region. Ultrasonography was performed after a palpable mass was detected in the epigastric region on deep palpation. Ultrasonography (Siemens, Acuson Antares, Germany) showed a heterogeneous hyperechogenous mass lesion with a liver-related appearance extending from the epigastric region to the superior umbilicus. The patient was then evaluated with non-contrast abdominal computed tomography (CT). The examination was performed with a 64 multidetector CT scanner (Toshiba, Aquilon, Otawara, Japan) in axial, coronal and sagittal planes. The lesion was observed as an inhomogeneous mass starting from the anterior abdominal wall at the level of the umbilicus and extending along the ligamentum teres into the FL between both lobes of the liver (Figure 1). A fine needle puncture of the mass in the epigastric region yielded a material compatible with hematoma. The patient was followed up for regression of FL hematoma. The patient was discharged without any complication.

Approximately two months later, the patient underwent laparoscopic cholecystectomy at an outside center with a diagnosis of cholelithiasis. A few days after the second operation, he was admitted to our center again with the complaints of a painful mass in the epigastric region and purulent discharge from the trocar entry site. Contrast-enhanced abdominal CT scan revealed a dense fluid collection with air values at the site of a previous falsiform hematoma (Figure 2). Clinical examination revealed purulent discharge from the trocar entry site and a diagnosis of FL abscess was made. Percutaneous drainage and antibiotherapy were applied. The patient was discharged without any complication during follow-up.

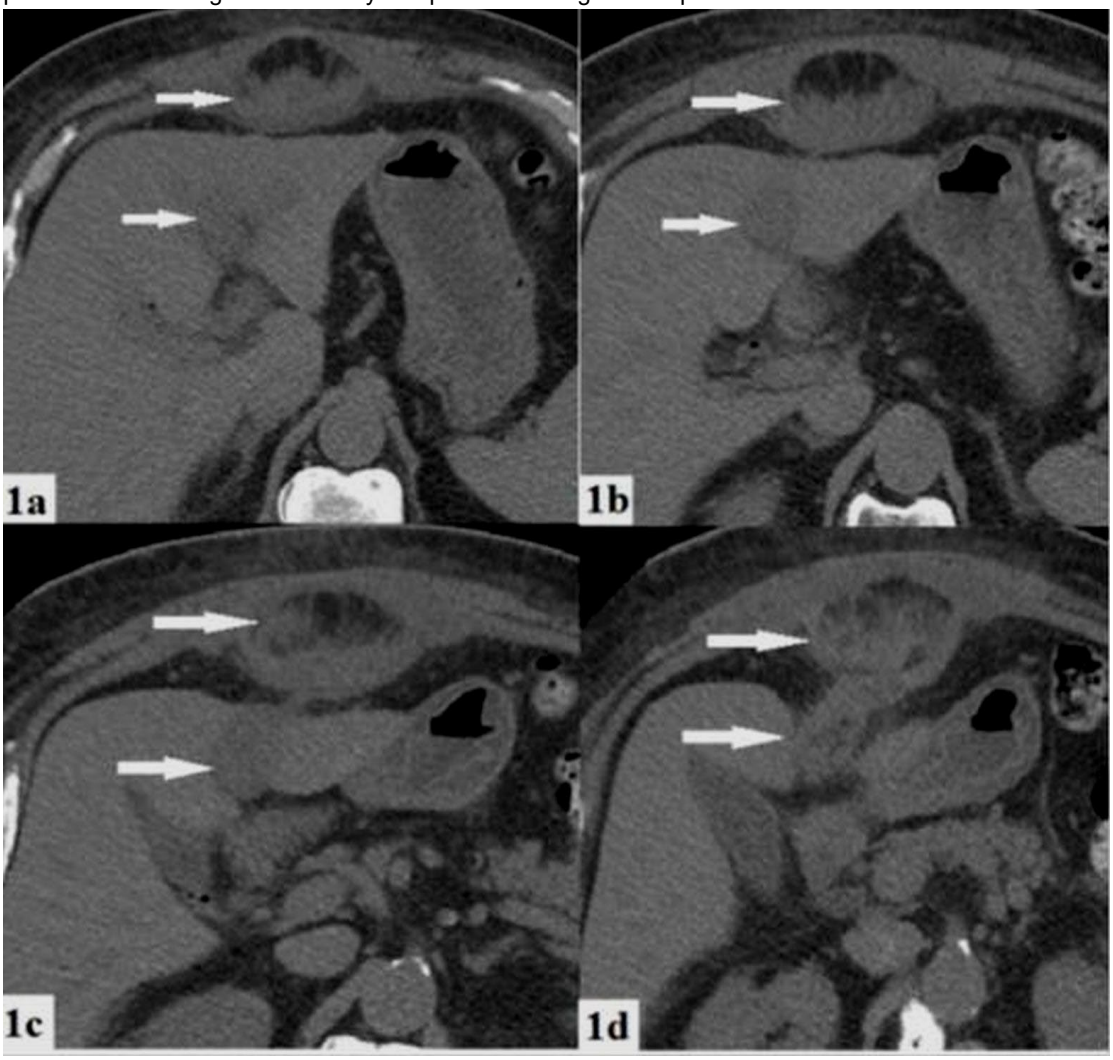

Figure 1: The falciform ligament hematoma is shown with arrows on a non-contrast abdominal CT scan

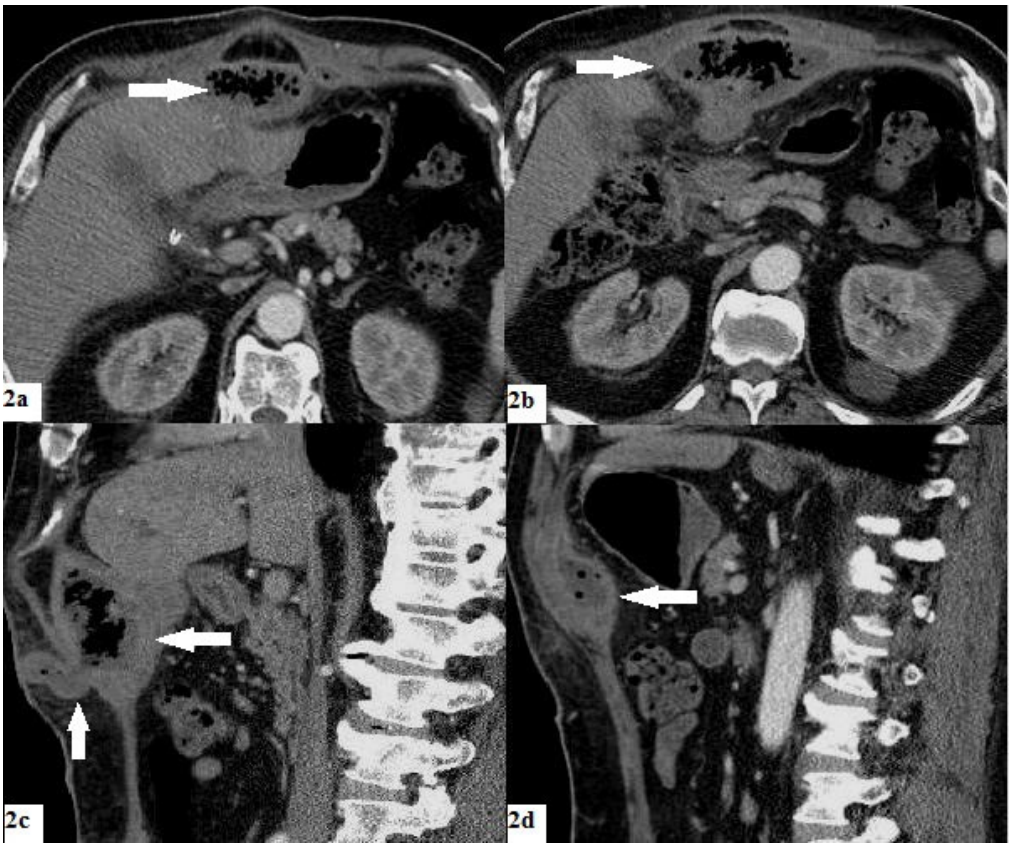

Figure 2: Contrast-enhanced abdominal CT scan showing a falciform ligament abscess with arrows.

Discussion

The FL is a ligament that suspends the liver to the anterior abdominal wall. It superficially divides the liver into two main lobes, right and left. It contains the round ligament (ligamantum teres hepatis) formed by the remnant of the left umbilical vein from the umbilicus to the left portal vein branch, paraumbilical veins and adipose tissue. The paraumbilical veins originate from the umbilical portion of the left portal vein within the FL and their ends are directed towards the umbilicus and periumbilical veins. In the literature, FL pathologies include cyst, tumor, abscess and hematoma [1-4]. Abscessation of FL hematoma is not mentioned. Our case is the first and only case in the literature in this regard.

FL hematoma is a rare entity reported in only a few cases in the literature [5,6]. The etiology is related with anticoagulant drug use or previous surgeries. Rarely, spontaneous hemorrhage may develop within the FL [7,8]. In the patient we presented, there was no drug use. He was operated with a diagnosis of incarcerated umbilical hernia. We think that the maneuvers performed during this operation caused incision of the paraumbilical veins passing through the round ligament and led to the development of FL hematoma.

FL abscess has been reported only a few times in the literature and has been shown to develop due to ventriculoperitoneal shunt infection, cholecystitis, pancreatitis and umbilicus infection in children (1-3,9). In our case, FL abscess was observed as a complication after laparoscopic cholecystectomy at an external center. The contamination of a previously developed FL hematoma during the second operation may be the cause of FL abscess development. In these patients, we think that trocar insertion away from the FL may prevent contamination of the hematoma.

The diagnosis of FL hematoma or abscess is usually based on physical examination findings and radiologic imaging modalities. Ultrasonography should be the first method of investigation. CT provides more detailed information about the size and location of the lesion. In patients with hematoma, non-contrast CT is performed first, but contrast-enhanced CT will be more useful for the differential diagnosis of abscess and mass. In our case, the diagnosis was made by physical examination, ultrasonography and CT.

Treatment requires ligament excision depending on the size of the hematoma or follow-up for regression of the hematoma. Abscess treatment includes percutaneous or laparoscopic drainage, antibiotherapy and ligament excision if necessary.

In conclusion, FL hematoma or abscess is a rare clinical entity that may present with an epigastric mass and acute abdominal symptoms. The diagnosis can be easily made with radiologic examinations, especially multidetector CT. It is a condition that should be kept in mind, especially in patients presenting to the emergency department with complaints of upper abdominal pain and swelling.

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Percheron artery infarction: a case report on differential diagnosis with carbon monoxide and methanol poisoning

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Introductıon

Percheron artery infarction is a rare type of ischemic stroke that occurs due to the occlusion of a single artery originating from the P1 segment of the posterior cerebral artery, which supplies both paramedian thalami (1). It is characterized by bilateral thalamic infarction and, in some cases, involvement of the rostral mesencephalon. Clinically, it can present with symptoms such as sudden changes in consciousness, confusion, vertical gaze palsy, and memory impairment (2). However, since these symptoms are nonspecific, metabolic, toxic, infectious, and neurodegenerative causes should also be considered in the differential diagnosis.

In particular, carbon monoxide and methanol poisoning can lead to clinical and radiological findings similar to those of Percheron artery infarction due to their involvement of deep gray matter structures (3,4). Carbon monoxide poisoning is associated with bilateral lesions in the globus pallidus and thalamus and can cause neurological symptoms (5). Methanol poisoning is characterized by bilateral involvement of the putamen and thalamus, leading to symptoms such as optic neuritis, altered consciousness, nausea, vomiting, and abdominal pain (6).

In this case report, we discuss a 67-year-old female patient diagnosed with Percheron artery infarction and emphasize the importance of differentiating carbon monoxide and methanol poisoning in the diagnostic process.

Case Presentatıon

A 67-year-old female patient was brought to the emergency department by the 112 ambulance team due to a loss of consciousness. According to her husband, the patient was healthy and fully conscious when she woke up at around 4:00 AM to pray, after which she went back to sleep. However, at approximately 10:00 AM, she could not be awakened. Additional information from family members revealed that the patient did not consume alcohol or tobacco, lived in a house with a central heating system, and had a medical history of diabetes and hypertension, for which she was taking her medications regularly.

Upon initial evaluation in the emergency department, the patient's general condition was poor, and her Glasgow Coma Score (GCS) was recorded as 8. Her vital signs were as follows: blood pressure, 141/78 mmHg; pulse, 82 bpm; temperature, 37.4°C; oxygen saturation, 96%; and blood glucose level, 138 mg/dL. Neurological examination revealed that the patient was in a stuporous state, with no orientation or cooperation. Her pupillary light reflexes were normal bilaterally, with isocoric pupils. There were no signs of meningeal irritation, and her facial muscles were symmetric. Although a full assessment of muscle strength was not possible, she was observed to move all four extremities in response to painful stimuli. Laboratory tests and neuroimaging were performed. Arterial blood gas analysis revealed a pH of 7.35, pCO□ of 48.8 mmHg, pO□ of 81 mmHg, lactate of 1.6 mmol/L, HCO□ of 23.8 mmol/L, and COHb of 1%.

A brain CT scan showed hypodense areas in the bilateral thalamic regions (Figure 1). Diffusion-weighted MRI revealed hyperintense signals in the bilateral thalami, with diffusion restriction on the ADC map, findings consistent with acute ischemia (Figure 2). After consultation with the neurology and interventional radiology departments, no large vessel occlusion was identified, and endovascular treatment was not recommended. The patient was admitted to the intensive care unit for further monitoring and treatment.

Dıscussıon

Percheron artery infarction is a rare type of stroke caused by the occlusion of a single thalamoperforating artery originating from the P1 segment of the posterior cerebral artery, which supplies both paramedian thalami (1). This type of stroke is characterized by bilateral thalamic infarction and, in some cases, involvement of the rostral mesencephalon. Clinically, it can present with symptoms such as sudden changes in consciousness, confusion, vertical gaze palsy, and memory disturbances (2). However, since these symptoms are nonspecific, diagnosing the condition can be challenging, and a broad range of differential diagnoses should be considered. In particular, toxic and metabolic encephalopathies can mimic the clinical presentation of Percheron artery infarction.

Bilateral thalamic involvement is not limited to vascular causes; it can also be associated with toxic, metabolic, infectious, and neurodegenerative conditions. Carbon monoxide (CO) and methanol poisoning can produce clinical and radiological findings similar to those of Percheron artery infarction. CO poisoning leads to hypoxia-related damage, particularly affecting the globus pallidus, thala-

mus, and white matter, and can present with symptoms such as headache, dizziness, altered consciousness, and neurological deficits (3). In cases of suspected CO poisoning, carboxyhemoglobin (COHb) levels should be assessed via arterial blood gas analysis, and the patient's history of potential exposure should be carefully evaluated. In this case, CO poisoning was ruled out due to normal COHb levels.

Figure 1 BT images of the patient

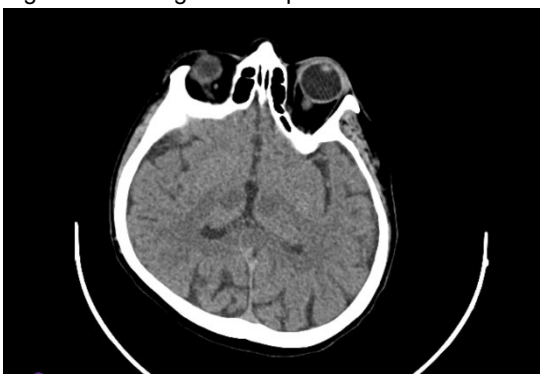
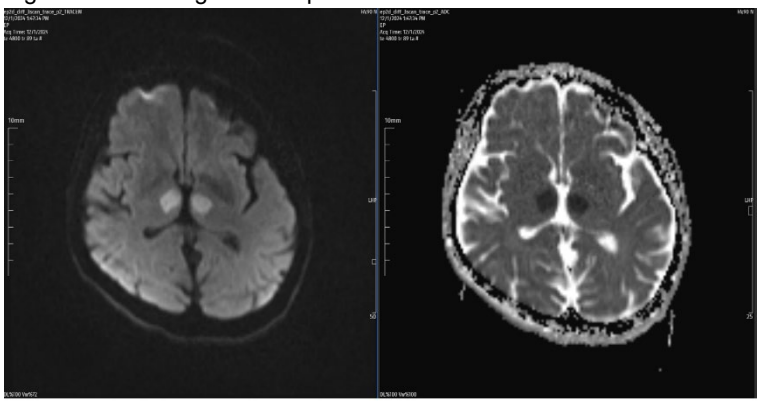


Figure 2: MRI images of the patient



Methanol poisoning, on the other hand, typically results in bilateral putamen and thalamic involvement and is associated with symptoms such as optic neuritis, altered consciousness, nausea, vomiting, and abdominal pain (4). The toxic effects of formic acid, a metabolite of methanol, can severely impact the optic nerves, leading to vision disturbances (5). In this case, methanol toxicity was excluded based on arterial blood gas analysis and ophthalmological examination findings.

Other potential causes of bilateral thalamic involvement include hypoglycemia, Wernicke's encephalopathy, herpes encephalitis, and prion diseases (6). Hypoxic-ischemic encephalopathy, often following cardiac arrest, can also affect gray matter structures and should be considered in the differential diagnosis. Therefore, a thorough evaluation of the clinical history and radiological findings is essential.

Diffusion-weighted MRI is one of the most valuable diagnostic tools for Percheron artery infarction. Imaging findings that demonstrate diffusion restriction and align with vascular distributions are particularly useful in differentiating this condition from other causes. In this case, the ischemic nature of the imaging findings, consistent with vascular structures, led to the prioritization of vascular causes, while toxic and metabolic encephalopathies were excluded. The most crucial factor in the differential diagnosis is the combined assessment of the clinical history and imaging findings.

Percheron artery infarction presents a diagnostic challenge due to its nonspecific and rare clinical manifestations. In this case, after excluding toxic causes, radiological findings confirmed the diagnosis of Percheron artery infarction. This case highlights the importance of ruling out toxic encephalopathies and underscores the need to consider this condition in the differential diagnosis. A multidisciplinary approach and advanced imaging techniques are critical for an accurate diagnosis.

Conclusıon

As demonstrated in this case, Percheron artery infarction can present to emergency departments with altered consciousness. In patients with altered consciousness and bilateral thalamic involvement on diffusion-weighted MRI, Percheron artery infarction should always be considered in the differential diagnosis. Clinical history, arterial blood gas analysis, and ophthalmological evaluation should be assessed collectively to exclude toxic-metabolic encephalopathies such as carbon monoxide and methanol poisoning. Early diagnosis and appropriate management are crucial for improving prognosis.

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Effects Of Dark Time And Chronobiological Time On Traffic Accidents Admitted To The Emergency Department

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Introduction and Purpose: Human biology and behavior have adapted exquisitely over time to favor optimal readiness during activity and renewal and repair during rest. These alternating time states and related processes are regulated by a genetically inherited endogenous circadian rhythm, or biological clocks. The model derived from rodent experiments assumes that the circadian system

is governed by a single master biological clock, the suprachiasmatic nucleus of the hypothalamus, which contains a relatively small number of clock genes that coordinate the dependent peripheral clocks of cells, tissues, and organs. However, this model may be more complex, as several studies suggest the existence of additional autonomous brain oscillators, including those in the cerebral hemispheres, that exhibit circadian periods equal to and different from the 24-hour period under different experimental conditions. Accidents can occur at any time of day and may be more common during the day in some environments than during other work shifts. Furthermore, the etiology of accidents includes not only worker-centered determinants such as circadian time, which refers to cognitive and physical performance rhythms, or factors such as sleep propensity and recent sleep history related to fatigue, but also the nature of the job tasks and how they are performed. It also includes the ecology of the workplace in terms of hazards, whether alone or in combination with others, which can be programmed in time over a 24-hour period, all of which are interconnected.

Simon Folkard coined the phrase “dark time” in 1997 to draw attention to the fact that, for people who are usually active during the day, the risk of a driving accident is greater at night than during the day. The 24-hour temporal pattern in driving accidents involves, at least in part, the circadian rhythms of fatigue and sleep propensity, cognitive and physical performance, and behavior, which are controlled by endogenous clocks.

Accidents and accidental injuries are important public health problems that require multidisciplinary research approaches and solutions, especially as technologies that require attention become increasingly integrated into work and daily life. The variation in the number and risk of accidents over a 24-hour period depends on many factors, including the nature and intensity of daytime duties and other shifts, the characteristics of working arrangements, and the training and age of workers, so that in some environments accidents and injuries may be more common during the day than at night. We introduced the term dark time to warn that people who are usually active during the day tend to be at greatest risk of driving and work-related accidents at night, when fatigue and sleepiness increase and cognitive and physical abilities are impaired. The risk of a car or truck driving accident actually shows two peaks, a large peak between 02:00–03:00 at night and a smaller peak between 14:00–15:00. Fatigue and drowsiness play a significant role in night driving accidents, as previously reported, and has been repeatedly confirmed for nighttime industrial and disaster accidents.

Afyonkarahisar is a regional hub in Turkey. Since it serves as an intersection in Turkey, millions of vehicles pass through Afyonkarahisar every year and hundreds of accidents occur. In our study, patients between the ages of 18-100 who applied to the Afyonkarahisar Health Sciences University Health Application and Research Center Adult Emergency Department due to traffic accidents inside or outside the vehicle between 01.01.2021-01.03.2025 will be included in the study. Studies on chronobiology are important studies that will provide both efficiency and control in health. Considering that patients who apply to the emergency department due to traffic accidents exhibit chronobiological changes, making changes appropriate to this situation can reduce the emergency department load and increase the quality of the service provided. Our goal in our study is to determine the chronobiological cycle of our emergency department and the increase or decrease in traffic accidents applied in bad times according to years, seasons, night and day, make the necessary preparations and increase the quality of the service provided.

Materials and Methods: Our study will determine which season and hour there are more accidents and we aimed to find information about the situations to be careful about. We included 2868 patients in our study between 01.02.2021-30.12.2024. 1962 (68.4%) of them are male and 906 (31.6%) are female. In our study, it was found that the most frequent accidents occurred in the summer months with 36.20% between 2021-2024 (figure 1). It was also found that the most frequent accidents occurred on Friday (figure 2). It has been determined that accidents are most common between 16:00-20:00 after work (figure 3).

Figure 1: Number of accidents between seasons by year

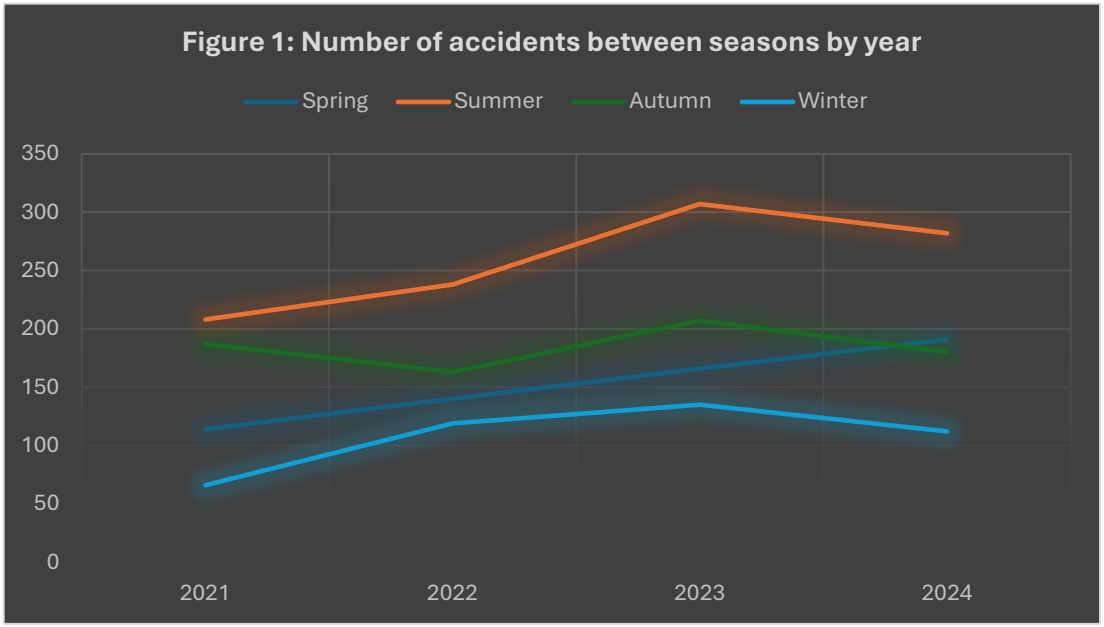


Figure 2: Number of accidents between days by year

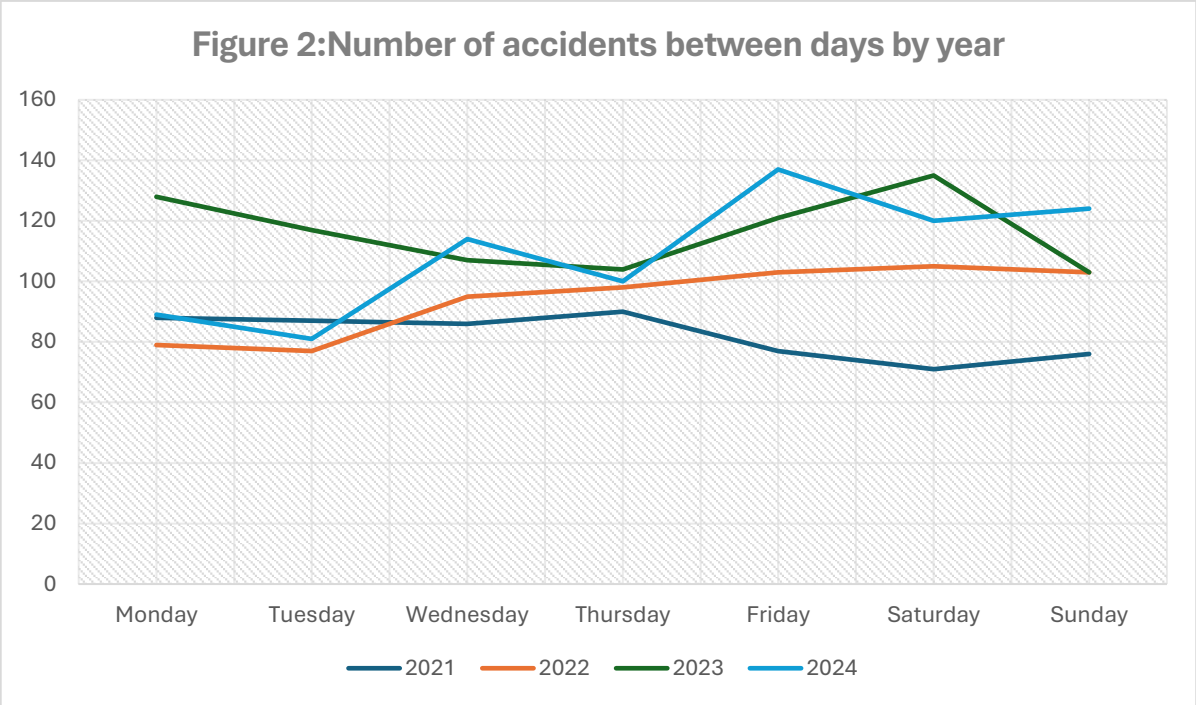
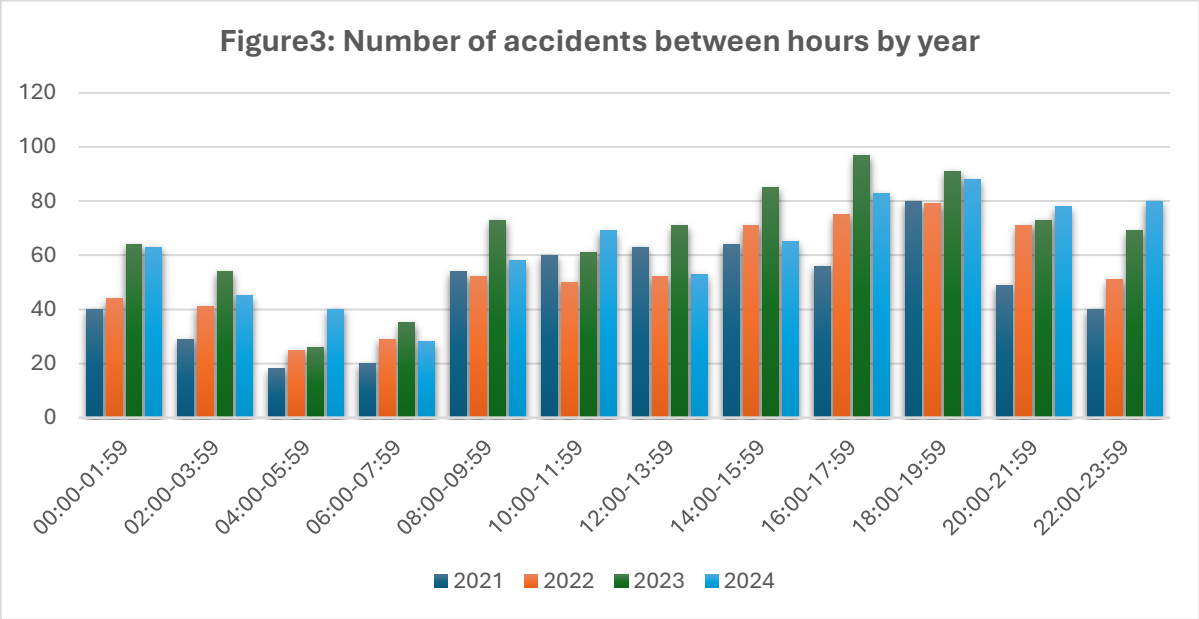


Figure 3: Number of accidents between hours by year



Results and Conclusion: In our study, we aimed to find solutions to the questions of how precautions can be taken and how such accidents can be prevented in the light of this information. Especially on Fridays and after work, inspections should be increased and drivers should be more careful. Due to summer vacation and more frequent use of highways, it is necessary to be more careful in the summer in order to prevent accidents.

Keywords: black time , chronobiological time , traffic accidents

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Fatal Cyfluthrin Intoxication Following Accidental Topical Exposure in a 67-Year-Old Woman: A Case Report

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Introductıon: Pyrethroids such as Cyfluthrin act by modifying the gating kinetics of voltage-gated sodium channels, leading to neurotoxicity in insects. These agents are considered relatively safe for humans due to rapid metabolic inactivation and low dermal absorption. However, in rare instances, especially in vulnerable populations, severe systemic toxicity can occur. To date, literature on human fatalities from Cyfluthrin exposure remains limited, particularly in cases involving dermal routes. Here, we present a fatal case of presumed Cyfluthrin poisoning in an elderly woman with no significant comorbidities, providing insight into the progression and systemic manifestations of pyrethroid toxicity.

Case: A 67-year-old woman, a shepherd by profession, was brought to the emergency department after collapsing in a remote pasture area. Earlier that morning, she had applied a Cyfluthrin-based insecticide spray to goats for flea control. Within hours, she developed acute retrosternal chest pain radiating to her back and shoulders, followed by profuse vomiting and a transient loss of consciousness. Upon arrival at the emergency department, she was hypotensive (BP: 65/45 mmHg), bradycardic (HR: 65 bpm), and hypoxic (SpO□: 88% on room air). Physical examination revealed no external injuries or signs of trauma.

Initial laboratory investigations were notable for extreme hyperglycemia (glucose: 420 mg/dL), severe metabolic acidosis (pH: 6.95, bicarbonate: 14 mmol/L), and elevated cardiac and hepatic markers (Troponin T: 62 ng/L, AST: 2900 U/L, ALT: 2200 U/L, LDH: >2700 U/L). Renal function tests showed acute kidney injury (Creatinine: 2.86 mg/dL; Urea: 148 mg/dL). Coagulation profile was deranged with INR: 1.51 and fibrinogen: 244 mg/dL. D-Dimer was significantly elevated at 6400 ng/mL.

A transthoracic echocardiogram revealed a 2–3 cm circumferential pericardial effusion without signs of tamponade. Chest radiograph and non-contrast thoracoabdominal CT were unremarkable except for mild bilateral pleural effusions and pericardial fluid. There were no radiological signs of ischemic heart disease or pulmonary embolism. Toxicology screening was not immediately available, but Cyfluthrin exposure was strongly suspected based on history.

The patient was transferred to the intensive care unit (ICU) with a working diagnosis of toxic-induced lactic acidosis, ischemic hepatitis, and multiorgan dysfunction. She was treated with high-dose intravenous sodium bicarbonate, N-acetylcysteine infusion, broad-spectrum antibiotics, and aggressive fluid resuscitation. Continuous hemodynamic and renal monitoring were initiated. Despite normalization of blood glucose and partial improvement in acid-base status, her clinical condition progressively deteriorated.

By ICU day 2, the patient developed disseminated intravascular coagulation (DIC), marked by thrombocytopenia (platelets: 55 $\times 10^3/\mu L$), elevated D-dimer (>21,000 ng/mL), and worsening liver function tests. She remained oliguric despite volume expansion, and hemodialysis could not be initiated due to hemodynamic instability. Hematology, gastroenterology, and cardiology consultations supported the diagnosis of systemic inflammatory response syndrome (SIRS) secondary to toxic exposure and advised continuation of supportive care.

On day 8 of hospitalization, the patient experienced sudden cardiac arrest. Cardiopulmonary resuscitation was initiated promptly, and multiple doses of intravenous adrenaline were administered. Despite 45 minutes of advanced life support, return of spontaneous circulation was not achieved, and the patient was pronounced dead.

Dıscussıon: This case demonstrates a rare but fatal outcome following presumed Cyfluthrin intoxication via dermal exposure. Although Cyfluthrin is considered low-risk in humans, its toxic potential in elderly or dehydrated individuals should not be underestimated. The rapid onset of symptoms and severe systemic findings—particularly lactic acidosis, hepatic transaminase surge, coagulopathy, and myocardial involvement—suggest direct cytotoxic and mitochondrial mechanisms. Previous studies have linked pyrethroid toxicity with oxidative stress, sodium channel dysfunction, and calcium dysregulation, all of which may contribute to the observed clinical manifestations.

Delayed recognition and lack of specific antidotal therapy for Cyfluthrin limit treatment options to supportive care. In this case, early decontamination was attempted, but the systemic toxicity had likely progressed. The history of fluid restriction, NSAID use, and possible dehydration could have exacerbated absorption and impaired clearance, contributing to the fulminant clinical course.

To our knowledge, this represents one of the few documented cases of fatal Cyfluthrin intoxication following topical application. While human case reports are rare, the growing use of pyrethroids in agriculture and veterinary practice necessitates heightened clinical vigilance and public education regarding safe use.

In conclusion, cyfluthrin, though typically regarded as a safe pyrethroid, can cause life-threatening toxicity through dermal absorption in specific scenarios. This case underlines the importance of early identification, decontamination, and aggressive supportive therapy in suspected insecticide poisoning. Further studies and toxicological surveillance are essential to better understand the risk factors and pathophysiological mechanisms of pyrethroid-related fatalities.

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6491

Unexpected diagnosis in adulthood: diabetic ketoacidosis as the first symptom of diabetes

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Introductıon

Diabetic Ketoacidosis (DKA) is one of the most serious acute complications of diabetes and is characterized by hyperglycemia, ketosis, and metabolic acidosis, usually because of insulin deficiency or insufficiency. Although DKA is particularly common in individuals with type 1 diabetes, in some cases it can also occur in patients with type 2 diabetes. This clinical picture, which requires urgent diagnosis and treatment, may lead to mortality and morbidity in the absence of rapid intervention [1]. The basic mechanism in the development of DKA is an absolute or relative insulin deficiency. Insulin deficiency leads to hyperglycemia by increasing the process of gluconeogenesis and glycogenolysis in the liver. At the same time, the absence of insulin increased the mobilization of free fatty acids from adipose tissue, leading to the production of ketone bodies (acetoacetate, beta-hydroxybutyrate). As a result, metabolic acidosis occurs with a decrease in the blood pH [2]. DKA may be a common initial presentation in patients presenting with newly diagnosed diabetes . A significant proportion of patients with diabetes, especially in childhood and adolescence, can be recognized directly by the development of ketoacidosis rather than by hyperglycemic symptoms [3]. The main clinical manifestations of DKA are polyuria, polydipsia, weight loss, nausea, vomiting, abdominal pain, weakness, dehydration, hypotension, and signs of metabolic acidosis such as Kussmaul respiration. Patients may also present with neurological symptoms such as confusion or coma [4]. The main goals for treating DKA are to restore the fluid-electrolyte balance, correct hyperglycemia, reverse ketosis, and treat the underlying cause. As a first step, dehydration should be corrected by intravenous fluid therapy. Usually, 0.9% NaCl solution is preferred and administered at a rate of 10-20 mL/kg in the first hour. When glucose levels drop, hypoglycemia should be prevented by adding 5-10% dextrose [6]. Insulin treatment is initiated as a low-dose intravenous insulin infusion (0.1 U/kg/hour). If the serum potassium level falls below 3.3 mEq/L, potassium replacement should be performed before starting insulin therapy. Metabolic parameters should be monitored hourly during treatment [7]. Education of newly diagnosed diabetic patients is critical to prevent long-term complications. Patients and their relatives should be given detailed information about insulin use, nutrition, hypoglycemia, and hyperglycemia management. In addition, support should be provided for regular blood glucose monitoring and lifestyle changes [8]. Newly diagnosed diabetes and diabetic ketoacidosis are clinical conditions that require urgent intervention. Early diagnosis and appropriate treatment are vital in reducing mortality and morbidity. A multidisciplinary approach should be adopted in the treatment process of patients, and long-term follow-up should be planned.

Case Presentatıon

A 27-year-old woman with no known comorbidities presented to the emergency department with complaints of anorexia, excessive water drinking, dry lips, difficulty in walking and weakness for the last two days. On physical examination, her pulse rate was 111 beats/min, blood pressure was 139/58 mm Hg, and oxygen saturation was 98%. The blood glucose level was found to be 400 mg/dL. Arterial blood gas analysis revealed pH: 6.88, bicarbonate: 5.1 mmol/L and potassium: 4.3 mmol/L. Complete urinalysis revealed 3+ ketone positivity. The patient was diagnosed with diabetic ketoacidosis in the emergency department, and appropriate fluid and insulin treatment was rapidly initiated. The endocrinology department was consulted, and the patient was hospitalized for further investigation and treatment.

Dıscussıon

Diabetic ketoacidosis (DKA) is a serious metabolic complication that usually occurs at the time of the initial diagnosis of type 1 diabetes mellitus (T1DM) or in existing diabetic patients with triggering factors such as infection, stress, or non-compliance with insulin

therapy. In this case, it is noteworthy that a 27-year-old young adult with no known comorbidities presented with DKA, although he had not been diagnosed with diabetes. Although it has been reported in the literature that DKA usually occurs in patients with newly diagnosed type 1 diabetes mellitus in childhood and adolescence, it should be taken into consideration that DKA may also be encountered in the adult age group at the time of diagnosis [1]. In the literature, it has been reported that DKA is mostly seen in childhood and pediatric patients typically present with symptoms such as polydipsia, polyuria and weight loss [4]. However, our case is different in terms of newly diagnosed diabetes mellitus presenting with DKA in adulthood. In a case report published at Bezmialem University, it was emphasized that DKA associated with newly diagnosed diabetes mellitus is more frequently seen in young adults and this may be an atypical picture [5]. At this point, our case differs from other pediatric cases in the literature. Undiagnosed diabetes mellitus or discontinuation of insulin therapy usually plays a role in the development of DKA. In our case, the patient was newly diagnosed and presented with marked hyperglycemia (blood glucose 400 mg/dL). In a previously published study, it was reported that the mean blood glucose level in newly diagnosed patients ranged between 350-600 mg/dL and these values may be higher in severe DKA [2].

Conclusıon

In conclusion, the presented case demonstrates that newly diagnosed diabetic patients in adulthood may also present with DKA and develop severe metabolic acidosis requiring emergency intervention. Therefore, it is important to consider DKA as a differential diagnosis in adult patients without a diagnosis of diabetes in emergency departments

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Bithalamic Infarction: A Report of Two Cases

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Introduction

Bithalamic infarction is a rare but clinically important stroke syndrome. It is most often associated with occlusion of the arteria Percheron and presents with symptoms such as altered consciousness, oculomotor disturbances, loss of cognitive function or memory, and pseudobulbar syndrome. The paramedian region of the thalamus is supplied bilaterally by the arteria Percheron and occlusion of this vessel can cause bithalamic infarcts. Although the exact incidence is unknown, it is estimated to account for 0.6-2% of ischemic strokes. In this case report, two cases of bithalamic infarcts with two different clinical presentations will be discussed.

Case 1

A 69-year-old woman presented to the emergency department with complaints of diplopia and headache that started one day ago. She was diagnosed with hypertension, diabetes mellitus, atrial fibrillation and rheumatoid arthritis and had no history of influenza, fever or trauma. On physical examination, he was conscious, oriented-cooped, and had no neurological lateralizing deficit. Pupillary light reflex was present bilaterally and nuchal rigidity was not observed. Laboratory findings included CRP: 10 mg/L, glucose: 156 mg/dL, creatinine: 0.62 mg/dL, potassium: 4.40 mmol/L, sodium: 139 mmol/L, lactate: 1.3 mmol/L, pH: 7.40. Vital signs, blood pressure was 150/70 mmHg, fever was 36.6°C, pulse rate was 96 beats/min and saturation was 98% on room air.

Diffusion MR imaging revealed bithalamic infarction reflected on FLAIR sequence. The patient was consulted to neurology and hospitalized for follow-up and treatment was started. Visual functions improved without sequelae and the patient was discharged after treatment.

Case 2

A 61-year-old woman was admitted after a sudden loss of consciousness the night before. The patient who was diagnosed with hypertension, hyperlipidemia and diabetes mellitus was evaluated as nonoriented noncooperative GCS:4 and intubated. According to the information obtained from his relatives, it was learned that he had no history of flu infection, fever, nausea, vomiting, travel or

trauma in the last three weeks. No information was obtained about seizures before loss of consciousness.
Physical examination revealed no nuchal rigidity. Pupils were middilated and light reflex was weak in both eyes. Babinski reflex was positive on both sides. Vital signs were stable. Laboratory tests revealed WBC: 8.000/mm³, Hb: 11 g/dL, PLT: 399.000/mm³, glucose: 250 mg/dL, creatinine: 0.8 mg/dL, sodium: 134 mmol/L, potassium: 4.5 mmol/L, pH: 7.40, lactate: 4.4 mmol/L and CRP: 17.6 mg/L.
MR examination showed findings consistent with bithalamic infarction on diffusion-weighted MR and FLAIR sequence. The patient was hospitalized in the intensive care unit and follow-up and treatment was started. He was extubated during intensive care unit follow-up. There was no change in the state of consciousness and right hemiparasia was observed during follow-up. He was fed with a nasogastric catheter. After a long intensive care unit follow-up, the patient was transferred to the internal medicine service for sugar regulation with his current condition.

Figure 1: Image of bithalamic infarct on MR ADC and TRACE sequence

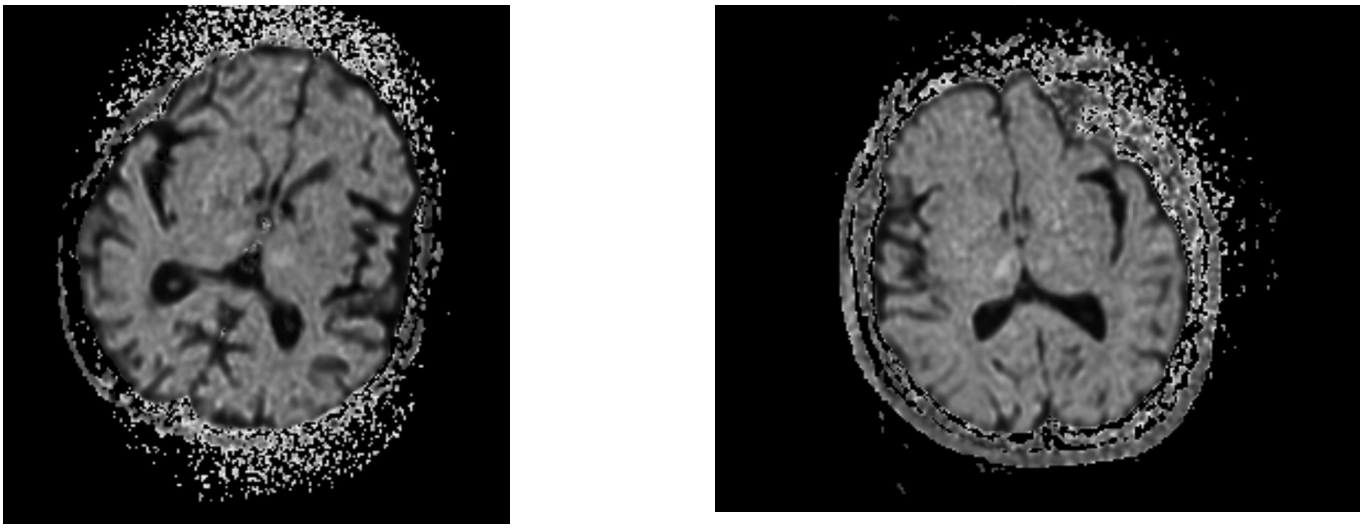
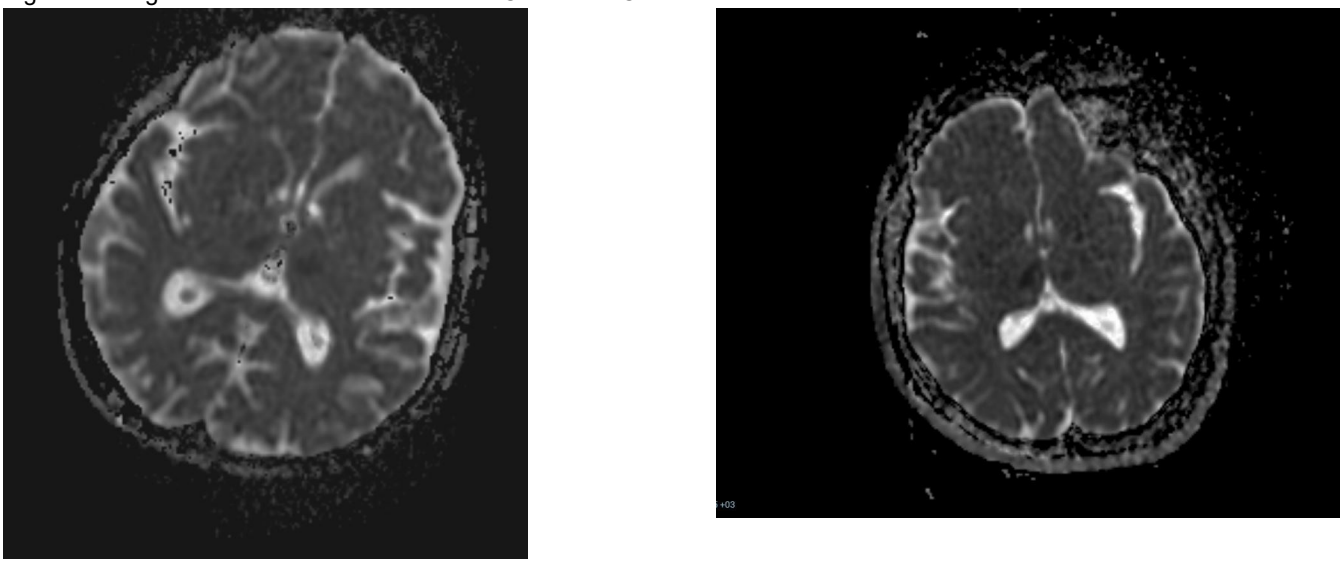


Figure 2: Image of bithalamic infarct on MR ADC and TRACE



Discussion

The thalamus is an important region of the brain that processes and transmits sensory information to cortical areas. It also plays a role in many critical processes such as cognitive functions, consciousness, motor control and autonomic regulation. When a thalamic infarct develops, decreased level of consciousness, confusion, disruption in sleep-wake cycle, memory problems, oculomotor disorders, dysphasias and extrapyramidal findings may occur. It is also associated with bilateral thalamic lesions, pseudobulbar syndrome and apraxia.(2,6,5)
Bithalamic infarcts are mostly associated with occlusion of the arteria Percheron. Arteria Percheron is a single artery supplying the paramedian thalamus and rostral mesencephalon (1,2).
In a clinical spectrum analysis, it has been shown that the majority of bithalamic infarcts are associated with vascular risk factors

including atrial fibrillation, hypertension and diabetes (3).
Imaging findings of arteria Percheron infarcts have been examined in detail in previous studies and the importance of early diagnosis with diffusion MRI has been emphasized (3,6,4).
The first of these cases represented a case presenting with relatively mild symptoms and was referred for early diagnosis, whereas the second case presented with a more severe clinical picture and loss of consciousness. Percheron artery occlusion has a wide clinical spectrum and should be diagnosed with careful neurologic examination and appropriate imaging modalities.
Treatment may vary depending on the underlying cause. In acute ischemic strokes, intravenous thrombolytic therapy or mechanical thrombectomy should be considered in appropriate patients. However, since arteria Percheron occlusion is usually of small vessel disease or cardioembolic origin, antithrombotic therapy and rehabilitation of stroke sequelae are important.

Conclusion

Bithalamic infarction is a rare but serious neurologic sequelae. As seen in the cases we discussed, bithalamic infarcts may present with different clinical presentations. Early diagnosis and treatment are critical for the prognosis of patients. Recognition of Arteria Percheron occlusion is important for clinical approach and appropriate treatment strategies

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Fırst Seızure In The Emergency Department And Contrast-Enhanced Cranıal Mrı

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Introductıon

Determining the underlying causes in a patient experiencing a seizure for the first time is critically important, especially in the elderly population and in patients with a history of malignancy. Seizures are clinical conditions that result from abnormal electrical activity in the brain parenchyma and are usually secondary to systemic, structural, infectious, metabolic, or neoplastic causes. In elderly patients presenting with seizures, cerebral metastases should be considered one of the most important structural causes, particularly in those with a history of malignancy. Renal cell carcinoma (RCC) is a malignancy that can metastasize to distant organs such as the lungs, liver, bones, and brain. Although brain metastases due to RCC are rare, these metastases often present as hemorrhagic lesions and may be the first sign of neurological symptoms. Therefore, seizure presentation in a patient with a history of RCC requires a detailed evaluation for metastatic disease. This case discusses the evaluation, differential diagnosis, and the role of contrast-enhanced cranial MRI in the emergency department of a 68-year-old patient with a history of RCC who experienced her first seizure. The presence of a malignancy history in a first-time seizure patient broadens the differential diagnosis and prioritizes the likelihood of metastatic disease. Brain metastases due to RCC usually present as hemorrhagic lesions with surrounding edema and mass effect. Such metastases may be the first indication of neurological symptoms and generally reflect advanced disease at diagnosis. Hence, careful evaluation of neurological symptoms in patients with a history of malignancy is crucial for early diagnosis and treatment. Moreover, advanced imaging techniques play a critical role in confirming the diagnosis and assessing the structure, location, and characteristics of the lesion. In particular, contrast-enhanced cranial MRI has high sensitivity in differentiating hemorrhagic lesions and identifying causes such as metastasis, primary tumors, or infarcts. This case emphasizes the importance of a multidisciplinary approach in evaluating neurological symptoms in patients with a malignancy history and highlights the role of advanced imaging techniques in the clinical decision-making process.

Case Report

A 68-year-old female patient presented to the emergency department with sudden loss of consciousness and generalized convulsions. According to her relatives, the seizure lasted approximately 1–2 min, followed by a postictal confusion period of approximately 15 min. Her medical history included renal cell carcinoma (RCC), diagnosed and treated with nephrectomy 1 year ago. She also had a history of hypertension and diabetes mellitus and was on regular medication. There was no known history of neurological disease or seizures before this event. On physical examination, vital signs were stable, and no focal findings were noted on neurological examination. Laboratory tests including electrolytes, blood glucose, and renal function tests were within normal limits. Because of her malignancy history, imaging studies were planned to exclude structural pathology.

Figure 1: Brain CT: A subcortical-cortical hypodense area in the right frontotemporal region with hyperdense areas consistent with hemorrhage was observed. (Acute hemorrhagic infarction?)

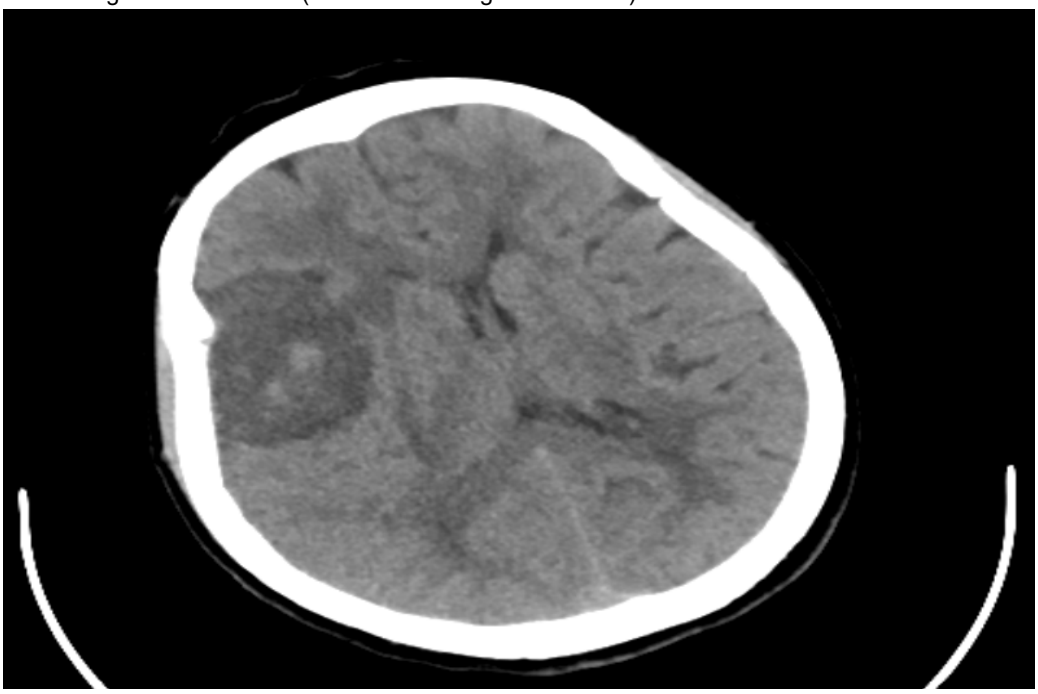
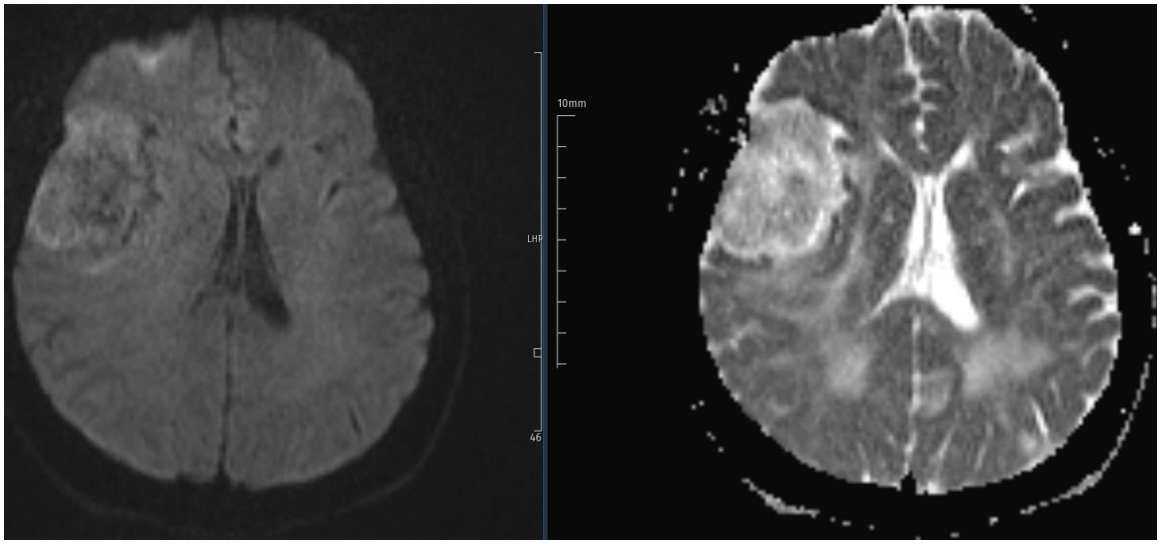


Figure 2: Diffusion MRI: A hemorrhagic lesion approximately 5x4 cm in size with heterogeneous contrast enhancement was detected in the right frontotemporal region. The absence of significant vasogenic edema in the surrounding parenchyma prompted a recommendation for further evaluation for hemorrhagic infarct versus mass.



To clarify the diagnosis, a contrast-enhanced cranial MRI was performed.

Dıscussıon

Renal cell carcinoma (RCC) is a malignant tumor with high metastatic potential, commonly spreading to organs such as the lungs, bones, liver, and brain.

Although brain metastases from RCC are rare, they typically occur in advanced stages of the disease and significantly worsen the prognosis. Brain metastases in RCC patients may present with acute neurological symptoms or seizures. In this case, the patient presented with a seizure in the context of a history of RCC.

Imaging modalities are crucial for the diagnosis of brain metastases. While CT provides a rapid assessment, it may be limited in differentiating between hemorrhage and mass. In this case, CT revealed a hyperdense area in the right frontal lobe but could not distinguish between a mass and hemorrhage. Diffusion-weighted MRI demonstrated hemorrhagic features and mass effect but was insufficient for a definitive diagnosis.

Contrast-enhanced MRI provided detailed information on anatomical features and contrast enhancement patterns, proving decisive in diagnosing RCC-related brain metastasis.

Figure 3: Contrast-enhanced cranial MRI: A lobulated mass lesion located cortically-subcortically at the level of the Sylvian fissure in the right frontotemporal region, measuring 46x41x45 mm (APxTRxCC), showing hypointensity on T1-weighted images, marked hyperintensity with hypointense foci on T2-weighted images, and intense heterogeneous contrast enhancement after gadolinium injection. Perilesional edema was noted. Dural enhancement was observed at this level. The right lateral ventricle was compressed. A 5 mm midline shift to the left was present. Additionally, in the medial cortex of the right temporal lobe at the hippocampus and parahippocampal level, an expansile, non-enhancing nodular lesion approximately 23x15 mm in size was observed, appearing hyperintense on T2-weighted and FLAIR sequences. In a patient with RCC history, metastasis was considered most likely, with ischemia as a less probable differential.

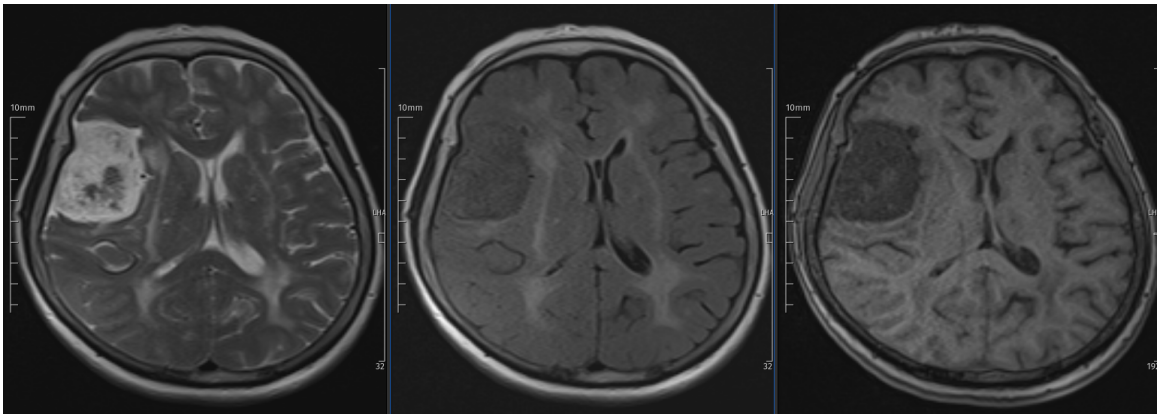
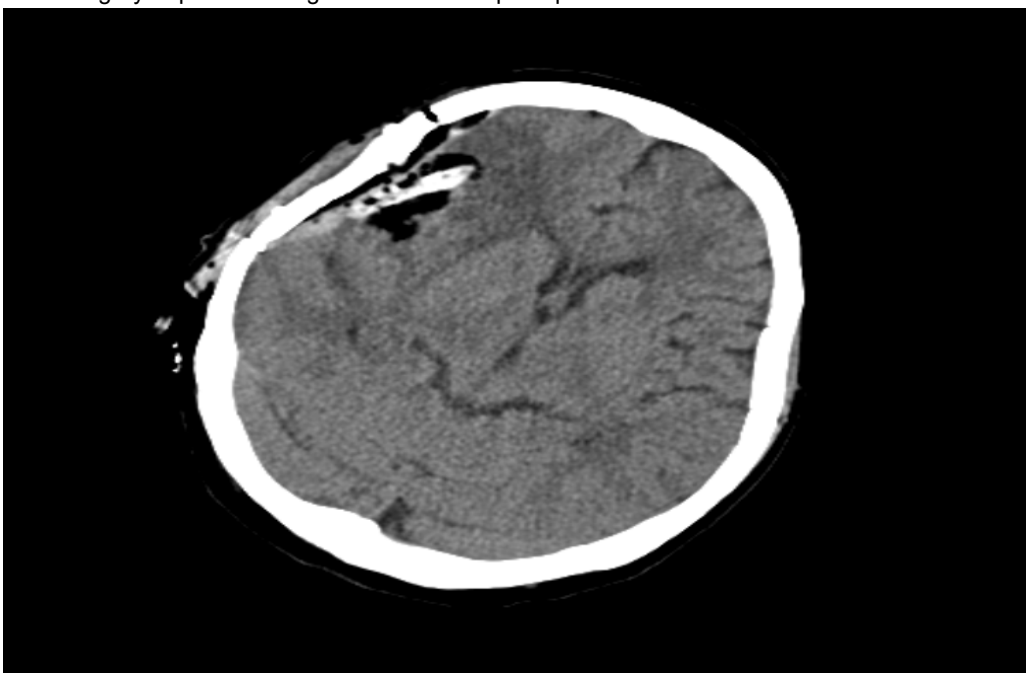


Figure 4: These findings were consistent with brain metastasis due to RCC. The patient was evaluated and operated on by the neurosurgery department. Figure 4 shows the postoperative brain CT.



Surgical treatment is an important option for brain metastases from RCC, especially in cases of solitary lesions and in patients with a good general condition. In this case, the lesion in the right frontal lobe was totally resected, and histopathological examination confirmed RCC metastasis. Surgical treatment alleviates mass effect and improves symptoms and quality of life.

The prognosis of RCC patients with brain metastases is generally poor; however, a multidisciplinary approach can improve both survival and quality of life.

Conclusıon

In conclusion, although brain metastases from RCC are rare, they can cause severe clinical manifestations. This case underscores the importance of a careful evaluation of neurological symptoms in RCC patients. Contrast-enhanced magnetic resonance imaging

plays a critical role in revealing both the anatomical and physiological characteristics of metastatic lesions. This technique offers a significant advantage in distinguishing hemorrhagic components from tumor characteristics, guiding appropriate clinical management strategies.

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6679

Self-Mutilation (Self-Harm)

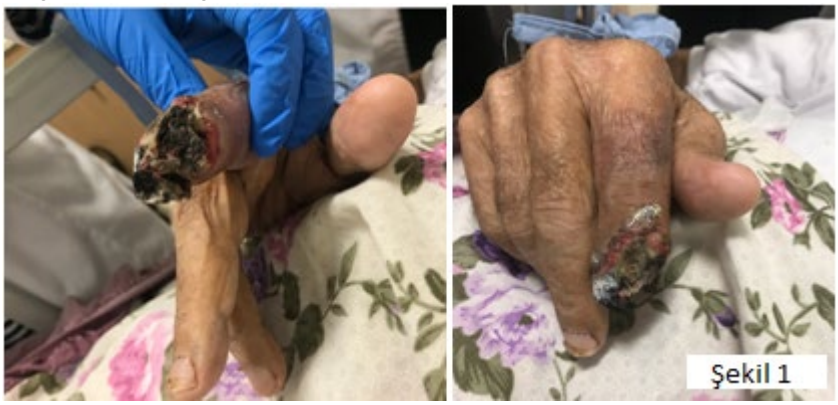
Sezen Argın
Adana 5 Ocak Devlet Hastanesi

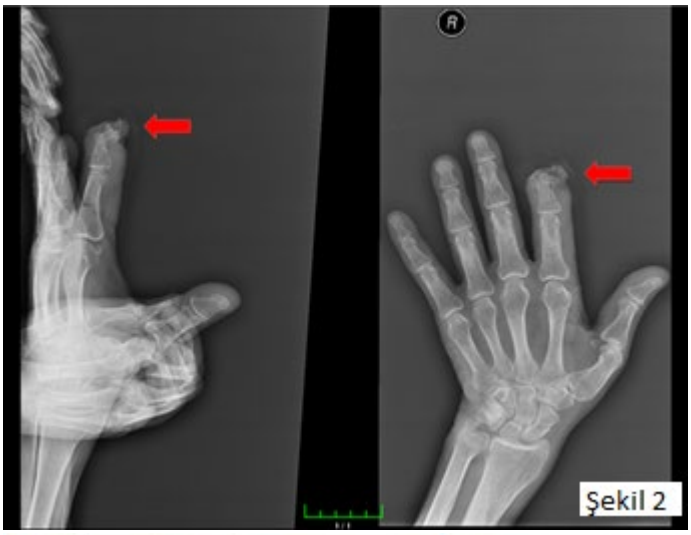
Introductıon

In psychiatry, self-mutilation (self-harm, self-injury) is defined as a behavior that causes serious harm to body structures intentionally and consciously without the intention of committing suicide (1) (2). Cultural environments such as chaining oneself in religious ceremonies, stabbing oneself with a skewer, social environments where groups stabbing themselves at concerts are affected can trigger self-mutation, and it can also be frequently seen in mentally retarded individuals, people with borderline and other personality disorders, and eating disorders (3) (4). The prevalence of self-mutilation varies between 700-750 per 100,000 people (4). It can be examined in three groups. The most severe form is major self-mutilation and can be seen in those with mental retardation, substance addiction, schizophrenia, and bipolar disorder. Skinning, eye removal, limb or genital organ amputation may be observed. In stereotypical self-mutilation, actions such as hair pulling, self-biting, head banging can be seen. It is often associated with mental retardation. It can be seen in schizophrenia, autism, Tourette Syndrome. The superficial-moderate type is the best and most common form. There is usually no disability. It may manifest itself as hair pulling, reddening of the skin. It is usually associated with social and cultural events and the patient aims to escape from negative emotional states by harming himself/herself with actions such as cutting, burning, and chaining. It can be seen in those with eating disorders, trichotillomania, and post-traumatic stress disorder (3) (4) (2).

Case

An 87-year-old male patient, immobile due to Alzheimer's, was brought to us by his relatives with the complaint of an increasing wound on his finger. When the anamnesis was examined, it was learned that the patient had bitten his own finger 15 days ago and the wound had a foul odor and was getting worse. The patient's general condition was good but his cooperation was limited due to Alzheimer's. On examination, the right hand 2nd finger was amputated from the middle of the median phalanx, had a necrotic appearance and a foul odor (Figures 1 and 2). There were no pathological results other than high CRP in the patient's examinations. The patient was consulted with the Plastic Surgery Department after direct radiography was taken. The patient was transferred to the Plastic Surgery Department for cleaning the wound and applying a local flap instead of amputation (Figure 3). The patient was discharged with healing after wound closure with antibiotics and his follow-up radiograph 1 week later is shown in Figure 4.





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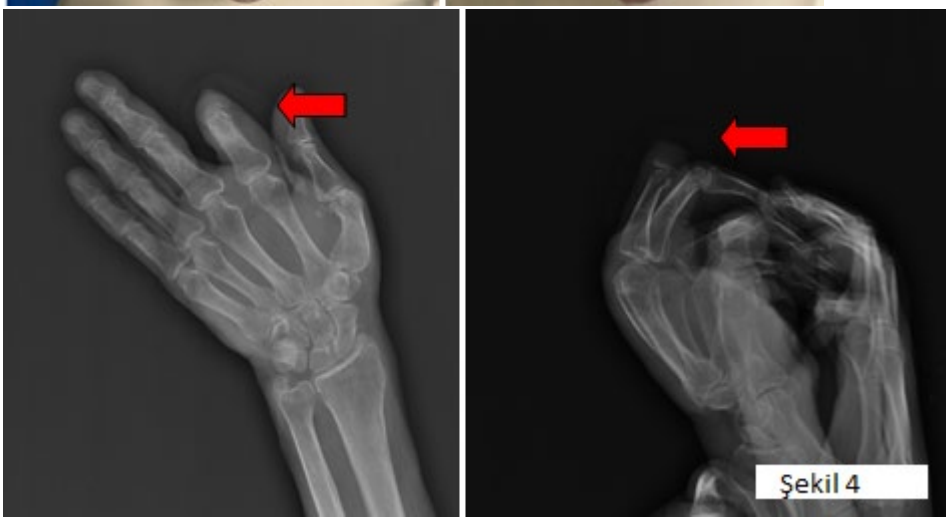

Şekil 4

Discussion

Since self-mutilation is associated with many psychiatric diseases, patients should be directed to the psychiatry department upon discharge. In patients presenting with mental retardation and bites, attention should be paid to the oral mucosal flora and antibiotics should be started. Since these cases may also involve abuse, in case of suspicion, the cases may require legal action.

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6708

Systemic Botulinum Intoxication After Gastric Botulinum Toxin Injection: A Case Report

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Introductıon

The synaptic effects of botulinum toxin have contributed to the understanding of neurophysiological mechanisms since the mid-20th century and have expanded its therapeutic applications (1,2,6). Botulinum neurotoxin-induced neuromuscular transmission disorders are rarely encountered in clinical practice but require accurate diagnosis and effective management (1). Produced by Clostridium botulinum, this toxin inhibits acetylcholine release by targeting synaptic vesicle proteins, resulting in flaccid paralysis (2). Serotypes A, B, and E are the most commonly implicated in human toxicity (2,6). In addition to its therapeutic use in neurological and gastrointestinal conditions, botulinum toxin has gained popularity in aesthetic and weight management applications.

In the fight against obesity, botulinum toxin is endoscopically injected into the gastric smooth muscle to delay gastric emptying and suppress appetite (3,5,10). However, this off-label application carries a risk of systemic toxin diffusion and iatrogenic botulism, necessitating caution (3,4,5,10). Such complications may present in previously healthy individuals with neurological symptoms including dysarthria, dysphagia, respiratory depression, ophthalmoplegia, and limb weakness.

This report presents the diagnostic process, intensive care follow-up, EMG findings, and treatment strategy of a 27-year-old female patient diagnosed with iatrogenic botulism after gastric botox injection.

Case

A 27-year-old female patient with no known chronic illnesses or medication history presented to the emergency department with progressive fatigue, blurred and double vision, diffuse muscle pain, dyspnea, and urinary incontinence that had developed over three days. The patient reported that symptoms began four days after undergoing gastric botox injection and worsened progressively with episodes of weakness in the upper extremities and transient muscle spasms.

Vital signs were stable. Ophthalmologic examination revealed decreased visual acuity with bilateral positive pupillary light reflexes. Laboratory tests including electrolytes, liver and thyroid function tests, and arterial blood gas analysis were within normal limits. No signs of systemic inflammation or metabolic disturbances were noted. Routine cranial CT and MRI scans showed no pathology.

Based on the recent gastric botox procedure and progressive neurological symptoms, iatrogenic botulism was suspected. The National Poison Information Center (114) was contacted, and Heptavalent Botulinum Antitoxin (HBAT) was recommended. However, due to limited availability of the antitoxin and the patient's stable clinical status, consultation was obtained from the Neurology Department at Istanbul Kartal Dr. Lütfi Kırdar City Hospital. The neurology specialist concluded that antitoxin therapy was not immediately required and recommended symptomatic and supportive care. Accordingly, the patient was started on pyridostigmine (Mestinon®) at a dose of 60 mg four times daily (total 240 mg/day). Our hospital's neurology team also evaluated the patient and agreed on initiating pyridostigmine and close monitoring.

During ICU monitoring, the patient experienced desaturation to 88% during sleep, which improved to 99% with verbal stimulation. Therefore, mechanical ventilation was not required, and oxygen therapy was initiated via nasal cannula at 3 L/min. Stability was achieved without invasive respiratory support. Electromyography (EMG) revealed typical findings of presynaptic neuromuscular junction dysfunction, including decreased CMAP amplitude and high-frequency facilitation.

On the third day of follow-up, full strength (5/5) was observed in the upper extremities, and the lower extremities remained stable. Due to ongoing dysphagia, oral intake was limited to soft R2 diet. Ophthalmology consultation was obtained for persistent visual complaints. Orbital MRI and neuroimaging studies revealed no abnormalities. The patient was advanced to an R3 diet on day four with continued improvement.

By the seventh day, nearly all symptoms including respiratory, motor, and autonomic manifestations had resolved, except for mild persistent blurred distance vision. The patient had initially been prescribed pyridostigmine at 60 mg three times daily (180 mg/day), which was later increased to 60 mg four times daily (240 mg/day) in response to clinical improvement. She was discharged with a prescription for pyridostigmine 3x60 mg and referred for outpatient neurology follow-up.

Dıscussıon

Botulinum toxin inhibits acetylcholine release by targeting SNARE complex proteins (synaptobrevin, SNAP-25, syntaxin), thereby blocking neuromuscular transmission (7,8). Systemic spread of the toxin can result in cranial nerve palsies, peripheral muscle weakness, and autonomic dysfunction. Iatrogenic botulism can arise from dosing errors, injection techniques, or individual susceptibility (3,6).

Diagnosis relies on detailed history, symptom progression, and clinical examination, supported by electrophysiological testing. Serological tests are controversial but EMG findings are often diagnostic (1,3,6,9,10). This case highlights the rarity of iatrogenic botulism following gastric injection and underscores the importance of early recognition, multidisciplinary management, and individualized treatment strategies to prevent complications.

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6801

A rare thoracic trauma: Two cases of flail chest

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Key words: flail chest, thoracic trauma, emergency department

Introduction and Purpose:

Flail chest is a serious medical condition that occurs when a segment of the rib cage separates from the rest of the thoracic cage due to multiple rib fractures. The purpose of this case report is to examine the emergency department presentation and subsequent management of patients diagnosed with flail chest.

Case 1: A 59-year-old male patient was referred to us with a diagnosis of flail chest due to a motor vehicle accident. Intubation and a thoracic tube were applied to the left hemithorax. Computed tomography (CT) images revealed multiple rib fractures in the left hemithorax, pneumothorax, subcutaneous emphysema, hemithorax and lung contusion. (Image 1) He was admitted to the intensive care unit. A rib plate was applied by the thoracic surgeon. After treatment, the patient was discharged.

Case 2: A 22-year-old male patient was brought to our emergency department by 112 teams after a motor vehicle accident and was electively intubated due to confusion and desaturation. CT scans revealed multiple rib fractures in the right hemithorax, widespread contusion areas, subcutaneous emphysema, minimal hemithorax and pneumothorax. (Image 2) The patient underwent a right tube thoracostomy. A rib plate was applied by the thoracic surgeon.

Image1 Computed thorax tomography image of the first case

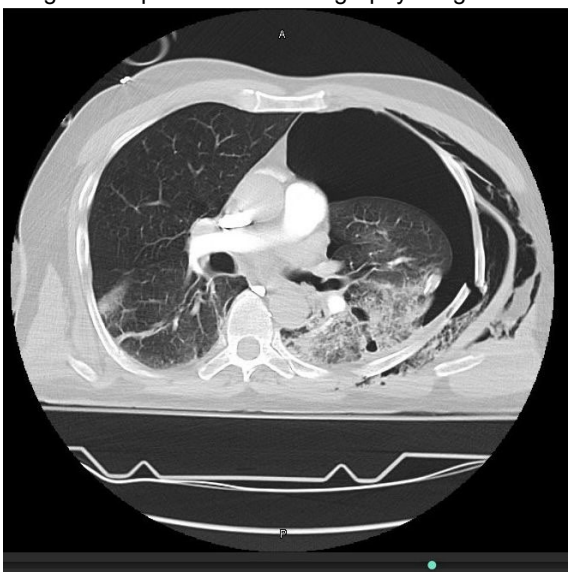
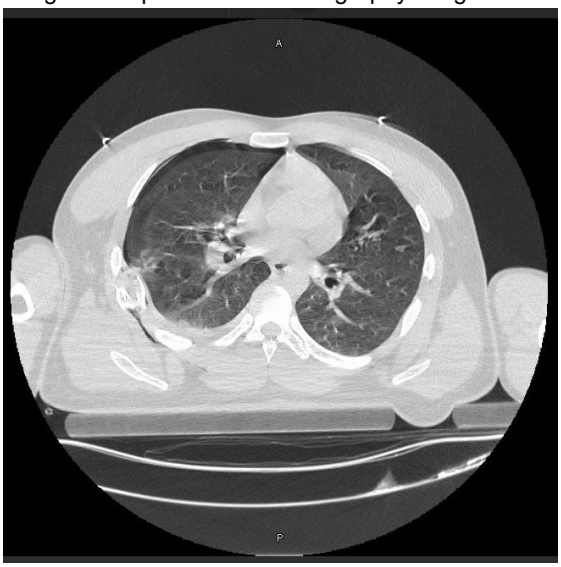


Image 2Computed thorax tomography image of the second case



Discussion-conclusion:

Flail chest has a high mortality rate and is usually caused by blunt trauma such as vehicle accidents or falls. Symptoms include severe pain, dyspnea, and visible deformity of the chest wall. It is usually diagnosed by physical examination and imaging techniques such as X-rays or CT scans. However, in a case in shock and with shallow breathing, paradoxical movement may not be

evident. During examination of the chest wall, asymmetric movement of this independent area and palpation of crepitation and friction due to friction of the fracture ends are helpful in diagnosis. In addition; paradoxical breathing, dyspnea and respiratory distress, painful breathing, crepitation and subcutaneous emphysema, tachypnea, tachycardia findings support the diagnosis.

The most effective treatment method known is intermittent positive pressure mechanical ventilation. However, the indication for mechanical ventilation is not only the presence of paradoxical movements. If the patient is tachypneic, if PaO2 <60 mmHg and PaCO2 >45 mmHg in the blood gas, mechanical ventilation should be applied. Analgesia and respiratory support are important in treatment. Narcotics, epidural analgesia, local anesthetics and intercostal nerve block can be used for analgesia.

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Delayed Presentation of a Retained Central Venous Guidewire Mimicking a Clavicular Abscess: A Case Report from the ED

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ıntroductıon: Central venous catheterization (CVC) via the Seldinger technique is a foundational skill in emergency medicine, providing rapid vascular access in critically ill patients. Despite its routine application, this procedure is not exempt from preventable complications, especially under high-stress conditions. Among the most insidious is the inadvertent retention of the guidewire—a so-called "never event" that paradoxically continues to occur even in well-resourced healthcare systems. This case illustrates how such an error, committed silently during a previous emergency operation, presented a year later not as a cardiovascular complication, but as a localized soft tissue infection. The discovery was made not in the operating room nor during routine follow-up, but in the emergency department—underscoring the role of emergency physicians not only as acute care providers but also as detectives of latent system failures.

We present this case to highlight the phenomenon of procedural blindness, wherein the apparent completion of a familiar task creates cognitive closure, masking critical steps like guidewire removal. This report aims to reframe the retained guidewire not merely as a technical error, but as a system-level sentinel event requiring attention from emergency medicine leadership.

Case: A 40-year-old male presented to our emergency department with a seven-day history of swelling, redness, and purulent drainage under the right clavicle. He denied fever, chills, or trauma. His past medical history was notable for a massive upper gastrointestinal bleed one year earlier, requiring emergency exploratory laparotomy. During that hospitalization, a right subclavian CVC had been inserted intraoperatively. The surgical course had been uneventful, and he was discharged after stabilization.

On physical examination, he was afebrile and hemodynamically stable. A 3x3 cm erythematous, fluctuant lesion with mild purulent discharge was noted in the right supraclavicular region. No systemic signs of infection were observed. Routine point-of-care ultrasound raised suspicion for a linear hyperechoic structure, prompting a posteroanterior chest radiograph. The radiograph revealed a retained guidewire extending down the right subclavian vein into the thoracic inlet.

Laboratory values, including white cell count and CRP, were mildly elevated. Blood cultures were negative. Vascular surgery and interventional radiology were consulted. Given the absence of systemic involvement and stable positioning of the wire, elective surgical retrieval was scheduled. The guidewire, measuring 45 cm, was successfully removed under fluoroscopic guidance. The patient was discharged with a short course of oral antibiotics.

Dıscussıon: This case exemplifies how latent iatrogenic errors, originating from high-pressure procedural settings, may surface months or even years later in unrelated clinical contexts. Retained guidewires typically result from breakdowns in procedural safety steps such as:

Skipping verbal confirmation of guidewire removal

Absence of procedural checklists

No post-procedural imaging

Overreliance on experience in the absence of standardized oversight

These vulnerabilities are amplified in emergency or operative settings, where urgency, noise, and cognitive overload can compromise attention to detail. In this case, the guidewire was likely left in situ during an emergent intraoperative line placement—an environment where confirmatory imaging is often deferred.

The role of the emergency physician in this case is particularly notable. Presented with a seemingly routine abscess, the clinician's high index of suspicion and decision to obtain imaging led to the diagnosis of a rare and dangerous complication—transforming the ED from a site of acute care to one of system-level error detection. This underscores the expanded cognitive responsibility of emergency physicians, who often encounter complications originating from outside their department but requiring their diagnostic vigilance.

The concept of procedural blindness, where familiarity with a procedure breeds cognitive complacency, has not been sufficiently addressed in emergency medicine literature. Even experienced operators may unintentionally skip essential steps when environmental stressors distort perception and judgment. Cognitive forcing strategies, such as mandatory guidewire counts or visual confirmation prior to catheter flushing, may serve as low-cost interventions.

Furthermore, this case invites a discussion of "safety debt"—delayed manifestations of small procedural oversights that accumulate risk over time. Post-procedural chest radiographs should not merely be used to confirm catheter tip placement or rule out pneumothorax, but also to explicitly verify guidewire removal, particularly in emergency insertions.

In conclusion, in emergency medicine, time-critical decisions must be balanced with procedural safety. This case of delayed presentation of a retained guidewire—discovered only because of an alert emergency physician—illustrates the dangerous intersection between procedural routine and systemic vulnerability. A renewed emphasis on checklists, procedural time-outs, and imaging verification is essential. Emergency departments must institutionalize these safeguards, not only to protect patients but also to shield providers from the fallout of entirely preventable errors.

This case serves as a reminder that the emergency physician is not merely a responder to crises, but a frontline gatekeeper for identifying—and interrupting—systemic errors long after they occur.

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A Case of Lithium Intoxication

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İntroduction: Lithium is a cornerstone in the treatment of bipolar disorder, known for stabilizing mood and preventing manic or depressive episodes(1). However, its narrow therapeutic index makes careful monitoring essential, as small changes in blood levels can lead to toxicity(2). Lithium works by influencing neurotransmitter activity and ion transport across cell membranes, though its exact mechanisms remain unclear(2). While effective, lithium has a range of potential side effects, including gastrointestinal disturbances (nausea, vomiting), weight gain, and renal impairment like nephrogenic diabetes insipidus(3). More severe effects, such as tremors, cognitive impairment, and motor coordination problems, can occur, particularly with higher blood levels(4). Therapeutic lithium levels typically range from 0.6 to 1.2 mEq/L, with toxicity risks emerging above 1.5 mEq/L. Mild toxicity (1.5–2.0 mEq/L) can cause nausea and tremors, moderate toxicity (2.0–2.5 mEq/L) leads to confusion and ataxia, while severe toxicity (>2.5 mEq/L) can result in seizures, renal failure, or even death (5,6).

Case: A 49-year-old male patient with a known history of bipolar affective disorder and diabetes mellitus (DM) presented to our emergency department. The patient is unemployed and lives alone with his mother. He has a history of four previous psychiatric hospitalizations due to aggressive behavior, auditory hallucinations, and harm to others.

Medications: Sodium valproate 1500 mg/day, Lithium carbonate 1200 mg/day and Aripiprazole 10 mg/day. It was noted that the patient had been using lithium irregularly.

The patient initially presented with dizziness and a feeling of faintness lasting for two days. On admission: Glasgow Coma Scale (GCS): 15, General condition: Stable, oriented, and cooperative. No abnormalities detected on neurological examination. Laboratory findings: Creatinine: 1.44 mg/dL, Urea: 69 mg/dL. No other significant abnormalities were found.

Brain CT and MRI were performed to rule out central nervous system abnormalities. The patient was treated with adequate hydration, and as his symptoms improved, he was discharged with recommendations for neurology and psychiatry outpatient follow-up.

Second Presentation (Four Days Later): The patient was brought to the emergency department again after experiencing syncope in a supermarket, along with incontinence, inability to walk, altered mental status, and whole-body tremors. On examination:Conscious, cooperative, but unable to provide meaningful responses to questions. No focal neurological deficits. Tremors observed throughout the body (rhythmic and variable, not suggestive of an organic movement disorder). No fever or neck stiffness was detected. Vital signs:Blood pressure: 152/70 mmHg, Heart rate: 99 bpm,Blood glucose: 88 mg/dL .ECG: Normal sinus rhythm, no abnormalities. Laboratory findings:Creatinine: 1.76 mg/dL (increased from baseline),Urea: 113 mg/dL (elevated). Brain CT and MRI showed no evidence of a central neurological event.

Neurology, psychiatry, and internal medicine consultations were requested. As lithium levels could not be measured in our facility, the patient's blood samples were sent to an external laboratory, which reported a lithium level of 3.9 mEq/L. Based on this level, The Poison Control Center was consulted, and close monitoring of mental status and urine output was advised. Internal medicine recommended hemodialysis, and the patient underwent 4 hours of hemodialysis with 1000 cc ultrafiltration (UF). After dialysis, the lithium level decreased to 2.24 mEq/L. However, due to ICU bed unavailability, the patient had to be monitored in the emergency department for several days, with daily laboratory tests performed. Two days after admission, the patient's general condition worsened, GCS declined. He was entubated. Internal medicine was consulted again. Three additional hours of hemodialysis were recommended. Following hemodialysis, the patient was transferred to a center where lithium levels could be monitored under ICU(intansive care unit) observation.

Discussion: Lithium toxicity is a serious condition, particularly in patients with renal impairment and poor adherence(5). In this case, irregular lithium use, dehydration, and chronic kidney disease likely led to lithium accumulation. The patient initially presented with mild symptoms, which temporarily improved, but later deteriorated with altered mental status, tremors, and syncope. Renal dysfunction impaired lithium clearance, worsening toxicity(3). Despite hemodialysis, the patient can suffer cardiac arrest, highlighting the neurotoxic and systemic complications of lithium overdose (6). This case underscores the importance of regular lithium and renal function monitoring, medication adherence, and early intervention in lithium toxicity. Hemodialysis remains the key treatment for severe cases, and multidisciplinary management is crucial for patient safety(7). Strict follow-up and patient education are essential to prevent life-threatening complications (1). In conclusion, it is a general rule that organic causes must be ruled out before making a psychiatric diagnosis for any patient. When evaluating psychiatric patients, the side effects of the medications they use, as well as the effects of their inappropriate use, should be taken into consideration.

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Hypertriglyceridemia-Induced Pancreatitis In The Emergency Department: A Retrospective Case Series

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Introduction: Hypertriglyceridemia-induced pancreatitis (HTG-IP) represents a distinct and increasingly encountered etiology of acute pancreatitis (AP), particularly in the context of rising metabolic disorders such as diabetes mellitus, obesity, and familial hyperlipidemia. Unlike other common causes of AP, HTG-IP is often associated with more severe clinical presentations and may lead to worse outcomes if not promptly recognized and managed. In emergency department (ED) settings, early identification of HTG-IP is critical, as targeted interventions can significantly impact disease progression and prognosis.

Despite its growing clinical relevance, data on the demographic and clinical profiles of HTG-IP patients presenting to the ED remain limited. Furthermore, the relationship between initial severity indicators and patient outcomes has not been fully elucidated.

This study aims to characterize the demographic features and clinical presentations of patients diagnosed with HTG-IP in the ED and to evaluate disease severity and short-term outcomes to contribute to the timely and effective management of this patient population.

Material Method: This retrospective cohort study was conducted at the Emergency Department (ED) of Muğla Training and Research Hospital between July 1, 2019, and January 1, 2023. Adult patients who presented with lipemic serum—suggestive of hypertriglyceridemia—were identified through electronic medical records and included in the analysis.

For each patient, demographic data (age, sex), laboratory triglyceride levels, and acute pancreatitis severity scores, including Ranson, Imrie, and Harmless Acute Pancreatitis Score (HAPS), were recorded. The diagnosis of hypertriglyceridemia-induced pancreatitis (HTG-IP) was based on clinical criteria supported by laboratory findings and imaging, where available.

The primary outcome was in-hospital mortality. Secondary outcomes included admission to the intensive care unit (ICU) and hospital length of stay. All collected data were analyzed to evaluate potential associations between severity scores and clinical outcomes.

Results: During the study period, out of 419,505 emergency department (ED) visits, 10 patients were diagnosed with hypertriglyceridemia-induced pancreatitis (HTG-IP), corresponding to a prevalence of 2.38 per 100,000 ED visits. The cohort included five male and five female patients, with a mean age of 52.7 ± 18.4 years. The mean serum triglyceride level at admission was 1183.3 ± 1105.6 mg/dL.

No in-hospital deaths occurred. One patient (10%) required admission to the intensive care unit (ICU), and the mean hospital length of stay was 8.7 days. Eight patients (80%) had mild acute pancreatitis (AP), whereas two (20%) presented with severe disease. Recurrent HTG-IP episodes were observed in two patients.

Severity scoring revealed a median Ranson score of 1.0 (IQR: 1.0), a median Imrie score of 1.5 (IQR: 1.75), and a median Harmless Acute Pancreatitis Score (HAPS) of 0.5 (IQR: 1.0).

Discussion: This study highlights the low prevalence yet high clinical relevance of HTG-IP among ED presentations. Although rare,

HTG-IP is associated with a potentially more severe course compared to other etiologies of AP. The absence of mortality and the limited need for ICU admission in this small cohort suggest that early identification and management may positively influence outcomes.

The observed recurrence in two patients underscores the importance of long-term lipid control and follow-up in preventing future episodes. Severity scores were generally low, which aligns with the predominance of mild AP in this cohort, yet the variability in triglyceride levels reflects the heterogeneity of disease expression.

The pathophysiology of HTG-IP is believed to involve the excessive breakdown of triglycerides into free fatty acids, which induces pancreatic endothelial injury, inflammation, and ischemia. Despite its low incidence, the potential for systemic complications warrants prompt recognition and targeted intervention.

Conclusion: Hypertriglyceridemia-induced pancreatitis, while uncommon, remains a clinically significant subtype of acute pancreatitis with the potential for severe complications. Early diagnosis, supportive care, and triglyceride-lowering therapy are essential to optimize outcomes. The findings of this study support the need for increased awareness of HTG-IP in the ED setting and emphasize the importance of metabolic control to prevent recurrence.

Further prospective studies are necessary to better define risk stratification models and to establish standardized treatment protocols, including the role of plasmapheresis, intravenous insulin, and novel lipid-lowering therapies.

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Unexpected Cardiopulmonary Collapse in an Ambulatory Patient: A Case of Resuscitation-Responsive Saddle Pulmonary Embolism

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Introductıon: Pulmonary embolism (PE) is a major cause of cardiovascular morbidity and mortality worldwide. Saddle PE, defined as a large thrombus lodged at the bifurcation of the main pulmonary artery, represents one of the most severe forms of PE, associated with a high risk of sudden cardiac arrest and death [1]. Though it comprises only 3–6% of all PE cases, its presentation may be insidious, and patients may initially appear hemodynamically stable, as seen in up to 45% of saddle PE cases [2,3]. This can delay diagnosis and treatment.

In the ED, the challenge lies in maintaining a high index of suspicion, particularly in elderly patients presenting with unexplained dyspnea, tachypnea, or hypoxemia in the absence of clear pulmonary or cardiac pathology. The administration of thrombolytic therapy during cardiac arrest secondary to massive PE remains debated but is increasingly supported by guideline recommendations in select cases [4].

We present the case of an elderly male who presented with subtle symptoms, deteriorated rapidly, and was successfully resuscitated after thrombolysis for confirmed saddle PE. The case emphasizes the importance of timely diagnosis, rapid imaging, and ED-based thrombolytic resuscitation.

Case: A 79-year-old male presented to our tertiary care emergency department with worsening shortness of breath over three days. The symptom had persisted for more than two weeks but had notably worsened recently. He denied chest pain, hemoptysis, fever, or syncope. His medical history included hypertension and gallstones. He reported a long smoking history (55 pack-years) and a sedentary lifestyle.

Notably, he had visited outpatient clinics four times in the preceding month for similar symptoms, where he had received symptomatic treatment without definitive diagnosis.

On presentation, his vital signs were: blood pressure 180/83 mmHg, heart rate 72 bpm, respiratory rate 30/min, SpO□ 83% on room air, and temperature 36°C. He appeared agitated, with increased work of breathing and abdominal accessory muscle use. Physical

exam revealed diffuse bilateral rhonchi on lung auscultation. There was no unilateral leg swelling or calf tenderness. Peripheral pulses were equal bilaterally. Homans' sign was negative.

Arterial blood gas analysis showed PaO□ of 49 mmHg on room air and respiratory alkalosis. Point-of-care echocardiography showed no pericardial effusion or gross right heart dilation. Due to ongoing hypoxemia and clinical suspicion of PE, emergent CTPA was performed, revealing a large saddle embolus occupying the main pulmonary artery bifurcation and extending into both main branches.

Unfractionated heparin (6000 IU IV bolus) was initiated, followed by continuous infusion. Approximately 30 minutes later, the patient suddenly developed bradycardia followed by pulseless electrical activity. Immediate CPR was initiated. During resuscitation, the patient received 1 mg atropine and 5 mg epinephrine in total. Alteplase was administered with a 10 mg intravenous bolus, followed by a 40 mg infusion over 15 minutes.

During CPR, the rhythm evolved into ventricular fibrillation, which was successfully defibrillated at 120 joules. ROSC was achieved after 15 minutes of CPR.

Post-resuscitation, he remained hypotensive and hypoxic (SpO□ 69%), requiring vasopressor support (norepinephrine infusion), intubation, and mechanical ventilation. Sodium bicarbonate (7 ampoules IV push followed by continuous infusion) was administered for severe acidosis. A brief trial of nitroglycerin infusion was discontinued due to hypotension. He was transferred to the intensive care unit under the care of pulmonary and critical care services.

Dıscussıon: This case highlights several pivotal aspects relevant to emergency medicine. First, saddle PE can initially masquerade as a relatively benign respiratory condition, particularly in older adults with chronic comorbidities and preserved blood pressure. The absence of classic findings such as chest pain or lower extremity edema can lead to diagnostic delay. Up to one-third of patients with PE present without chest pain, and nearly 60% have no clinical signs of deep vein thrombosis [3].

Second, the rapid deterioration observed underscores the fulminant nature of central pulmonary embolism. Large thrombi obstructing central pulmonary circulation may cause acute right ventricular overload, ischemia, and electromechanical dissociation. Early imaging, particularly CTPA, remains the gold standard in diagnosis, but bedside echocardiography showing right ventricular dilation or dysfunction can serve as a surrogate in unstable patients [1].

Third, this case illustrates the feasibility and potential benefit of administering thrombolytics during CPR in massive PE. Current guidelines from the ESC and AHA support thrombolysis in patients with cardiac arrest due to PE if no contraindications exist [3,4]. In our case, thrombolysis was administered promptly following radiological confirmation, which likely contributed to successful ROSC.

Although concerns about bleeding complications persist, meta-analyses suggest that the risk of major hemorrhage from thrombolysis during CPR is outweighed by potential survival benefits in appropriately selected cases [4]. Our patient experienced no immediate hemorrhagic complications following alteplase.

From an emergency medicine perspective, this case reinforces the need for timely suspicion of PE in elderly patients with persistent, unexplained dyspnea—especially in the absence of fever or radiographic consolidation. The ability to mobilize imaging, anticoagulation, airway management, and resuscitation protocols swiftly is essential in improving survival.

In conclusion, **s**addle PE poses a unique diagnostic and therapeutic challenge in the ED. This case underscores the importance of recognizing atypical presentations, maintaining a low threshold for imaging in high-risk patients, and being prepared to initiate thrombolytic therapy during cardiac arrest. In selected patients, even those presenting initially with stable vital signs, early intervention may reverse an otherwise fatal trajectory. Emergency physicians play a critical role in the timely recognition and life-saving management of massive PE.

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Evaluation of Clinical Parameters Associated with Interventional Treatment and Amputation in Patients Diagnosed with Peripheral Arterial Disease: A Retrospective Study Based on Emergency Department Admissions

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Introduction

Peripheral arterial disease (PAD) is a chronic vascular condition characterized by impaired arterial circulation, especially in the lower extremities. It develops as a result of atherosclerotic narrowing or occlusion of peripheral arteries. This condition may lead to ischemia, functional impairment, non-healing ulcers, and ultimately serious complications such as limb amputation [1]. It is estimated that

over 200 million people worldwide are affected by PAD, with prevalence increasing significantly with age—approaching 20% in individuals over the age of 70 [2,3].

PAD is often diagnosed at advanced stages, particularly when patients present with critical limb ischemia (CLI). This delays treatment success and increases the likelihood of amputation [4]. In emergency department admissions, patients often present with acute symptoms such as severe pain, skin discoloration, and absence of peripheral pulses, requiring rapid and accurate clinical evaluation to guide treatment decisions [5].

Treatment approaches vary depending on disease stage, symptom severity, vascular anatomy, and the patient's general clinical condition. Medical therapy, interventional procedures (angioplasty, stenting), and surgical revascularization are the most commonly employed methods. However, in some advanced cases with irreversible tissue damage or necrosis, amputation becomes unavoidable. The decision for amputation is based not only on anatomical ischemia but also on tissue viability, preservation of sensory-motor function, and capillary perfusion [4,6,7].

In this context, evaluating the relationship between clinical parameters and decisions regarding interventional treatment and amputation is crucial for effective patient management and optimal utilization of healthcare resources. This study aims to compare clinical parameters in patients with PAD presenting to the emergency department to determine which factors influence treatment decisions.

Materials and Methods

This retrospective descriptive study was conducted using data from patients diagnosed with peripheral arterial disease (PAD) who presented to the Emergency Department of Recep Tayyip Erdoğan University Training and Research Hospital between January 2023 and December 2024. The study was carried out in accordance with the principles of the Declaration of Helsinki. A total of 90 patients were included.

Data Collection and Variables

Demographic data (age, gender), clinical findings (capillary refill, sensory loss, motor function status), PAD staging (Stage I: Asymptomatic, Stage IIa: Claudication, Stage IIb: Rest pain, Stage III: Ischemia/gangrene), simplified PAD grouping (Stages I–IIa vs. IIb–III), Doppler flow status, and acute/chronic presentation were recorded. Treatment modality (medical vs. interventional) and amputation status (performed vs. not performed) were defined as outcome variables. Data were retrospectively collected from the hospital information system and consultation records.

Statistical Analysis

The normality of continuous variables was assessed using the Shapiro–Wilk test. The Mann– Whitney U test was used for non-normally distributed numerical variables. Categorical variables were compared using the Chi-square test or Fisher's Exact Test where appropriate. Variables found significant in univariate analysis were included in the multivariate logistic regression model. Independent risk factors for amputation were presented with odds ratios (OR) and 95% confidence intervals (CI). A significance level of 0.05 was considered statistically significant. Analyses were performed using the Jamovi statistical package (version 2.3) based on the R platform.

Results

The study included 90 patients diagnosed with PAD in a tertiary emergency department (43 women, 47 men). The median age of all participants was 80 years (IQR: 72.0–86.0). Patients undergoing interventional treatment were significantly older (83.0 vs. 78.0, $p = 0.022$). Nearly all patients in Stage IIb and III underwent intervention ($p < 0.001$). Findings such as absent capillary refill (59.3%), widespread sensory loss, and deep paralysis were significantly more common in the intervention group (Table 1).

Patients who underwent amputation were significantly older than those who did not (86.0 vs. 79.0, $p < 0.001$). All patients in Stage III underwent amputation ($p < 0.001$). Deep sensory loss, motor paralysis, and absence of venous Doppler flow were significantly associated with amputation (Table 2).

According to the logistic regression model, each one-year increase in age was associated with a 9% higher likelihood of amputation (OR: 1.09; $p = 0.0293$). Although female patients had a 3.47 times higher risk of amputation compared to males, this was not statistically significant ($p = 0.0652$). Patients classified as "chronic-on-acute" had 10.3 times higher amputation risk compared to those with acute presentation ($p = 0.0061$). Similarly, chronically presenting patients had a 4.7 times increased risk compared to acute patients ($p = 0.0308$) (Table 3).

Discussion

This study evaluated the association between clinical parameters and treatment decisions (intervention and amputation) in patients with PAD presenting to the emergency department. Our findings revealed that age, disease stage, capillary refill status, and the presence of sensory or motor deficits significantly influenced these decisions. The fact that all Stage III patients underwent amputation highlights the PAD stage as a direct predictor of treatment outcome. This is consistent with the literature, where patients presenting at advanced stages with tissue necrosis or gangrene are more likely to undergo amputation rather than revascularization [8–10].

Absent capillary refill reflects severe perfusion deficit and was strongly associated with both intervention and amputation. Similarly, extensive sensory loss and motor dysfunction are indicators of the depth and duration of ischemia.

Reported amputation rates in PAD vary between 10% and 30%; our study's amputation rate (22%) aligns with this range [11–13]. Notably, patients who underwent amputation were largely in advanced clinical stages where limb salvage was no longer feasible, underscoring the importance of early diagnosis and referral.

Table 1. Comparison of Clinical Parameters by Interventional Treatment Status

Interventional procedure		**No**	**Yes**	**Total**	**P value**
Gender	Male	20 (64.5)	27 (45.8)	47 (52.2)	0.141
	Female	11 (35.5)	32 (54.2)	43 (47.8)	
Age	Median (IQR)	78.0 (64.0 - 82.5)	83.0 (74.5 - 88.5)	80.0 (72.0 - 86.0)	0.022
Stage 1	Stage I	2 (6.5)	0 (0.0)	2 (2.2)	<0.001
	Stage IIa	23 (74.2)	0 (0.0)	23 (25.6)	
	Stage IIb	5 (16.1)	39 (66.1)	44 (48.9)	
	Stage III	1 (3.2)	20 (33.9)	21 (23.3)	
Stage 2	Stage I-IIa	25 (80.6)	0 (0.0)	25 (27.8)	<0.001
	Stage IIb-Stage III	6 (19.4)	59 (100.0)	65 (72.2)	
Capillary refill	Good	9 (29.0)	1 (1.7)	10 (11.1)	<0.001
	Slow	20 (64.5)	23 (39.0)	43 (47.8)	
	None	2 (6.5)	35 (59.3)	37 (41.1)	
Sensory loss	None	10 (32.3)	0 (0.0)	10 (11.1)	<0.001
	Minimal	17 (54.8)	10 (16.9)	27 (30.0)	
	Extensive	3 (9.7)	31 (52.5)	34 (37.8)	
	Deep anesthesia	1 (3.2)	18 (30.5)	19 (21.1)	
Muscle dysfunction	None	21 (67.7)	8 (13.6)	29 (32.2)	<0.001
	Mild-Moderate	9 (29.0)	26 (44.1)	35 (38.9)	
	Deep paralysis	1 (3.2)	25 (42.4)	26 (28.9)	
Venous Doppler flow	None	16 (51.6)	59 (100.0)	75 (83.3)	<0.001
	Yes	15 (48.4)	0 (0.0)	15 (16.7)	
Arterial Doppler flow	Yes	2 (6.5)	0 (0.0)	2 (2.2)	<0.001
	Poor	13 (41.9)	0 (0.0)	13 (14.4)	
	None	16 (51.6)	59 (100.0)	75 (83.3)	
Acute/Chronic	Acute	4 (12.9)	41 (69.5)	45 (50.0)	<0.001
	Chronic/Acute	3 (9.7)	10 (16.9)	13 (14.4)	
	Chronic	24 (77.4)	8 (13.6)	32 (35.6)	

In our logistic regression analysis, age was found to be a statistically significant predictor of amputation (OR: 1.09, p = 0.0293). Each additional year of age increased the risk by approximately 9%. Although amputation risk was higher in women (OR: 3.47), the result did not reach statistical significance (p = 0.0652), suggesting the need for further studies with larger samples [14].

When comparing acute versus chronic presentations, the chronic-on-acute group had a significantly higher amputation risk (OR: 10.32, p = 0.0061), followed by the purely chronic group (OR: 4.72, p = 0.0308). This emphasizes the progressive nature of PAD and the critical importance of timely intervention [15].

The strengths of this study include its real-world clinical data and its focus on emergency decision-making. However, limitations include its single-center design, limited sample size, and retrospective nature.

Conclusion

This study demonstrates that interventional treatment and amputation decisions in patients presenting with PAD are strongly associated with clinical parameters such as advanced age, disease stage, absence of capillary refill, and severe sensory-motor deficits.

Patients undergoing intervention were significantly older and more likely to be in advanced stages. Similarly, nearly all amputated patients were in Stage III, showing severe ischemic findings such as loss of capillary perfusion and deep motor/sensory deficits. Logistic regression analysis confirmed these clinical parameters as strong and significant predictors of amputation.

Table 2. Comparison of Clinical Parameters by Amputation Status

Amputation		No	Yes	Total	P value
Gender	Male	42 (60.0)	5 (25.0)	47 (52.2)	0.012
	Female	28 (40.0)	15 (75.0)	43 (47.8)	
Age	Median (IQR)	79.0 (68.2 - 84.0)	86.0 (83.2 - 91.0)	80.0 (72.0 - 86.0)	<0.001
Stage 1	Stage I	2 (2.9)	0 (0.0)	2 (2.2)	<0.001
	Stage IIa	23 (32.9)	0 (0.0)	23 (25.6)	
	Stage IIb	44 (62.9)	0 (0.0)	44 (48.9)	
	Stage III	1 (1.4)	20 (100.0)	21 (23.3)	
Stage 2	Stage I-IIa	25 (35.7)	0 (0.0)	25 (27.8)	0.004
	Stage IIb-Stage III	45 (64.3)	20 (100.0)	65 (72.2)	
Capillary refill	Good	10 (14.3)	0 (0.0)	10 (11.1)	<0.001
	Slow	43 (61.4)	0 (0.0)	43 (47.8)	
	None	17 (24.3)	20 (100.0)	37 (41.1)	
Sensory loss	None	10 (14.3)	0 (0.0)	10 (11.1)	<0.001
	Minimal	27 (38.6)	0 (0.0)	27 (30.0)	
	Extensive	31 (44.3)	3 (15.0)	34 (37.8)	
	Deep anesthesia	2 (2.9)	17 (85.0)	19 (21.1)	
Muscle dysfunction	None	29 (41.4)	0 (0.0)	29 (32.2)	<0.001
	Mild-Moderate	35 (50.0)	0 (0.0)	35 (38.9)	
	Deep paralysis	6 (8.6)	20 (100.0)	26 (28.9)	
Venous Doppler flow	None	55 (78.6)	20 (100.0)	75 (83.3)	0.054
	Yes	15 (21.4)	0 (0.0)	15 (16.7)	
Arterial Doppler flow	Yes	2 (2.9)	0 (0.0)	2 (2.2)	0.076
	Poor	13 (18.6)	0 (0.0)	13 (14.4)	
	None	55 (78.6)	20 (100.0)	75 (83.3)	
Acute/Chronic	Acute	40 (57.1)	5 (25.0)	45 (50.0)	0.004
	Chronic/Acute	6 (8.6)	7 (35.0)	13 (14.4)	
	Chronic	24 (34.3)	8 (40.0)	32 (35.6)	

Table 3. Logistic Regression Analysis of Amputation Risk Factors

Variable	SE	OR (95% GA)	p-value
Age	0.040	1.09 (1.009 – 1.181)	0.0293
Gender	0.675	3.47 (0.925 – 13.03)	0.0652
Chronic/Acute – Acute	0.851	10.32 (1.949 – 54.67)	0.0061
Chronic – Acute	0.719	4.72 (1.154 – 19.29)	0.0308

These findings provide valuable guidance for emergency physicians making time-sensitive decisions. In cases where patients present with advanced PAD indicators (e.g., absent capillary refill, deep sensory loss, motor dysfunction, advanced stage, and older age), immediate consideration of amputation rather than revascularization may be warranted.

Our study highlights the importance of early clinical assessment and appropriate risk stratification in optimizing individualized treatment strategies for PAD. Further multi-center and prospective studies are warranted to support the findings and contribute to national clinical guidelines.

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A rare case ın the emergency department: superıor vena cava syndrome

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Introductıon

Superior Vena Cava Syndrome (SVCS) is a clinical condition resulting from the obstruction of superior vena cava circulation, impeding blood drainage from the head, neck, arms, and upper thorax into the right atrium. Numerous symptoms manifest in VCSS due to tumor-induced pressure. Cyanosis, dyspnea, edema and congestion in the head, neck, and extremities are the main symptoms observed in patients [1,2]. Intrathoracic malignancies account for 60%-85% of VCSS patients. The superior vena cava was blocked in 60% of these cases before diagnosis. The occurrence of venous blockage can be caused by a primary tumor in the superior vena cava, external compression by mediastinal lymph nodes, or direct penetration by the tumor. VCSS is frequently associated with non-small cell lung cancer (nearly 50%), small cell lung cancer (22%), and lymphomas (12%) [3-6]. Common reasons for visiting the ED are the anticipated primary symptoms of VCSS (cyanosis, dyspnea, etc.). The purpose of this case presentation is to examine VCSS for patients who come to the Emergency Department (ED) for these reasons.

Case

The blue code team brought a 62-year-old male patient with no known chronic illness to the chest diseases clinic after he fell ill while waiting in line. At first, the patient's blood pressure was 113/76, oxygen saturation was 91%, pulse was 112, and during the physical examination, redness, edema, jugular vein distention, and difficulty breathing were observed. The patient's history indicates that she has been experiencing a persistent cough and facial redness for the past week. The thoracic CT scan showed a mass

that was consistent with the superior mediastinum and a 2 cm effusion in the right pulmonary hemithorax. The patient's blood taken in the ED did not show any significant pathology. Small cell lung carcinoma was diagnosed by the patient through a fine needle aspiration biopsy performed in the ward where she was admitted. The patient passed away on the 13th day of hospitalization after experiencing a cardiac arrest following a syncope while attempting to get out of bed during ward follow-up, at the stage when radiotherapy treatment was planned.

Dıscussıon

The management of VCSS, which is considered an oncological emergency, involves both cancer treatment and alleviation of obstruction symptoms. The signs and symptoms of VCSS can include edema of the face or neck, arm swelling, engorged veins in the anterior chest wall, chest pain, shortness of breath, dizziness, syncope, orthopnea, and the presence of facial plethora. The average life expectancy for patients with VCSS is six months. Studies have shown that when symptoms are alleviated with chemotherapy and radiotherapy, the treatment is more effective [7]. In our case, a patient who primarily did not have an oncology diagnosis and presented with complaints similar to those in the literature was presented. The similar to the literature, the patient who died during the treatment planning phase highlights the shortness of the survey in these patients. Considering that these patients can suddenly deteriorate in the AS as well, it is important to be more vigilant in their follow-up and to remain alert for potential complications.

Conclusıon

In our scenario, VCSS can be presented to ASs in either a stable or unstable manner. Vital signs that indicate upper respiratory tract infection symptoms or allergy-related face erythema and dyspnea may be present in VCSS. In the absence of a confirmed cancer diagnosis, VCSS should be included in the differential diagnosis of these individuals, and jugular venous distension should be meticulously assessed during the physical examination. The presence of erythema and edema in the upper extremities should be considered as a cautionary tale for this diagnosis.

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Snakebite in Pregnancy: Compartment Syndrome and Emergency Fasciotomy

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Introductıon:

With the effect of tropical climate in Turkey, poisoning cases such as snake bites and scorpion stings are frequently encountered. These envenomations may cause serious toxic effects on healthy children and young adults and may lead to high morbidity and mortality (1). In cases of poisoning, local symptoms such as pain, edema, redness, bullae formation and skin necrosis may occur in the bitten or stung area. In addition, systemic symptoms such as nausea, vomiting, sweating, hypotension, anaphylaxis, paresthesia, delirium and coma may also be observed (2,3,4).

Case:

A 31-year-old 15-week pregnant patient was brought to our emergency department by ambulance from an external center due to snake bite. When the patient arrived, she was conscious, GCS was 15 and vital signs were stable. There was a hyperemic bite lesion on the dorsal surface of the proximal 5th finger of the right hand (Figure 1), and there was diffuse edema, hyperemia and tenderness in the distal part of the hand, wrist and forearm. On arrival, sensory motor examination was normal, distal pulses were patent, capillary refill time and other system examinations were normal. Abdominal ultrasonography showed positive fetal heart movements. Tetanus prophylaxis, iv antibiotherapy and analgesia were administered without abnormalities in electrocardiography and laboratory tests. Elevation and ice were applied to the right upper extremity. During follow-up, edema and hyperemia spread to the proximal forearm and arm. The patient was asked to receive snake antivenom and the procedure and risks were explained. However, the patient did not accept the treatment due to her pregnancy status and possible risks. At the 18th hour of the incident, hypoesthesia, pallor, impaired capillary filling and subsequent limitation in motor movements developed along with severe pain in the right upper extremity (Figure 2). The patient was consulted with orthopedics with a diagnosis of compartment syndrome and was taken into emergency operation for fasciotomy. Right upper extremity fasciotomy was performed under axillary nerve block

(Figure 3). On the 10th day of follow-up, edema regressed and motor and sensory examinations were completely normal. The fasciotomy was closed and the patient was discharged with recovery.

Dıscussıon:

Snake venom is especially known for its hemotoxic effects. The most effective treatment method for coagulation disorders and compartment syndrome related to snake venom is antivenom administration (5,6). When snake venom directly contacts the tissue, it immediately initiates tissue damage and triggers the ischemic process. In this process, local symptoms such as edema, redness and pain may develop in the bitten area. Complications such as bullae, abscesses and ecchymosis can also occur. Findings such as absence of pulse, coldness, severe pain, paresthesia, delayed capillary filling and pain on passive stretching may be indicators of compartment syndrome and these patients should be evaluated for emergency fasciotomy (7). In our case, antivenom treatment could not be administered because of the patient's pregnancy status. However, the patient developed signs of compartment syndrome during follow-up and emergency fasciotomy was performed and the patient was discharged on the 10th day with complete recovery.

KEYWORDS: Snakebite, Pregnancy, Emergency fasciotomy

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Figure 1:

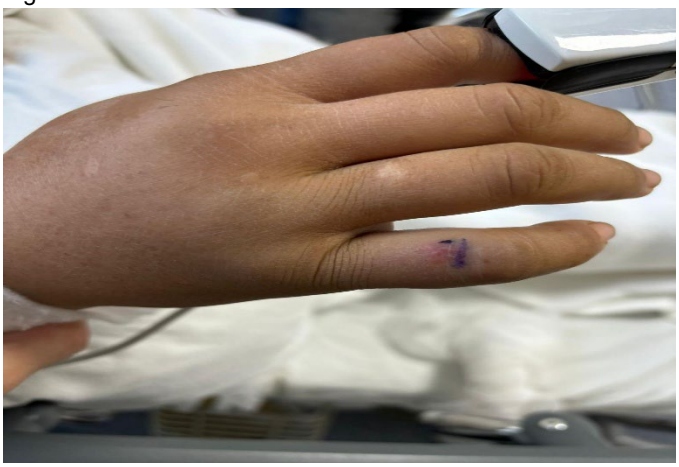

Figure 2:

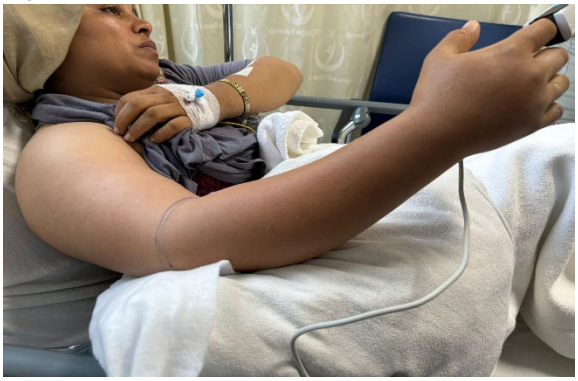

Figure 3:

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Euglycemic Diabetic Ketoacidosis Induced by Dapagliflozin: A Case Report

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Introductıon:

Sodium-glucose cotransporter-2 (SGLT-2) inhibitors are increasingly used in the management of type 2 diabetes mellitus (T2DM) due to their glycemic control and cardiorenal protective effects. However, these agents carry a rare but serious risk of euglycemic diabetic ketoacidosis (eDKA)—a life-threatening condition characterized by high anion gap metabolic acidosis in the absence of marked hyperglycemia. Because its clinical presentation is often subtle, eDKA can be overlooked in emergency department (ED), delaying diagnosis and treatment.

Case:

A 62-year-old male presented to the emergency department with complaints of nausea, vomiting, and dizziness that began two hours after receiving a punch to the right side of his head. His medical history was significant for T2DM, hypertension, coronary artery disease (status post-percutaneous coronary intervention in 2019), chronic kidney disease, peripheral arterial disease, and a prior hemorrhagic stroke. His prescribed medications included dapagliflozin (10 mg/day), antihypertensives (amlodipine, bisoprolol, perindopril), antiplatelet agents (cilostazol, clopidogrel), lipid-lowering therapy (atorvastatin), and neuropathic pain modulators (pregabalin, gabapentin).

On presentation, the patient was hypertensive with a blood pressure of 200/98 mmHg, a heart rate of 90 bpm, and an oxygen saturation of 98% on room air. He was alert, oriented, and cooperative. Neurological examination revealed no cranial nerve deficits or lateralizing motor weakness, and muscle strength was preserved bilaterally. Cardiovascular, respiratory, and abdominal examinations were unremarkable. Given the hypertensive urgency, oral amlodipine (10 mg) was administered, followed by intravenous nicardipine infusion. Supportive treatment with intravenous analgesics (dexketoprofen) and antiemetics (dimenhydrinate) was initiated.

Laboratory investigations revealed metabolic acidosis with a pH of 7.30, pCO□ of 43.6 mmHg, bicarbonate of 18.2 mmol/L, and an anion gap of 11.6 mEq/L. Serum glucose was mildly elevated at 247 mg/dL, and urinalysis was positive for ketones. Renal function tests showed a creatinine level of 1.15 mg/dL and urea of 57.7 mg/dL. Serum electrolytes, including sodium (140 mmol/L) and potassium (3.53 mmol/L), were within normal limits.

Given the presence of high anion gap metabolic acidosis with ketonemia in the absence of significant hyperglycemia, eDKA was suspected. Endocrinology consultation was obtained, and dapagliflozin was immediately discontinued. The patient was initiated on intravenous dextrose therapy (500 mL of 5% dextrose with 10 U crystalline insulin at a continuous infusion rate of 100 mL/h) to correct ketoacidosis while preventing hypoglycemia.

Non-contrast brain computed tomography (CT) performed at admission and repeated at six hours revealed no acute hemorrhagic changes or evidence of traumatic brain injury. Chronic ischemic changes were observed in the periventricular white matter, consistent with his history of cerebrovascular disease. Diffusion-weighted magnetic resonance imaging (MRI) confirmed the absence of acute infarction or diffusion-restricted lesions.

Serial blood gas analyses demonstrated progressive improvement in metabolic parameters, with a pH of 7.34, pCO□ of 39 mmHg, bicarbonate of 21.4 mmol/L, and a glucose level of 218 mg/dL. His nausea and dizziness gradually resolved with ongoing therapy. The patient remained hemodynamically stable and was discharged with resolved metabolic acidosis, strict instructions to discontinue dapagliflozin, and scheduled endocrinology follow-up for alternative diabetes management.

Discussion:

eDKA remains an underrecognized but potentially life-threatening adverse effect of SGLT-2 inhibitors. The absence of significant hyperglycemia often results in diagnostic delays, particularly in emergency settings where DKA is typically suspected only in cases of marked hyperglycemia.

This case highlights the potential for eDKA to develop even in the absence of clear precipitating factors, reinforcing the importance of clinical suspicion. The patient's use of dapagliflozin, in conjunction with his ketonemia and low bicarbonate levels, strongly implicated SGLT-2 inhibitor-induced ketoacidosis as the etiology of his metabolic disturbance. While the precise trigger remains uncertain, mild trauma and transient nausea may have contributed to transient insulin deficiency and enhanced ketogenesis.

Management of eDKA requires discontinuation of the offending agent, aggressive fluid resuscitation, insulin therapy, and carbohydrate supplementation. Unlike typical DKA, where insulin is titrated based on glucose levels, eDKA necessitates dextrose coadministration to prevent hypoglycemia while resolving ketosis. Serial monitoring of acid-base status and electrolyte levels is crucial for ensuring a safe and effective recovery.

Conclusion

This case highlights the critical role of emergency physicians in recognizing and managing eDKA, a life-threatening but often overlooked complication of SGLT-2 inhibitors. Early identification and timely intervention are paramount to preventing adverse outcomes. In the ED, patients presenting with nausea, vomiting, or altered mental status and unexplained anion gap metabolic acidosis should be evaluated for eDKA, particularly if they are on SGLT-2 inhibitors. As the use of these agents expands, increased awareness among emergency clinicians will be essential in ensuring prompt and effective treatment.

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Review Of Emergency Medicine And Primary Healthcare Services During Flood-Induced Disasters: The Example Of Hurricane Daniel In Libya

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Introduction

According to World Bank data, the population of Libya in 2021 was reported to be 6,812,341, with an annual population growth rate of 1.1%, and a life expectancy at birth of 72 years. According to the WHO's 2015 report, 22% of Libya's population lives in rural areas, and the life expectancy at birth is 75 years. The disease burden (in 2012) was composed of 77.8% non-communicable diseases, 9.8% communicable diseases, and 12.3% injuries. The healthcare workforce is reported to be 19 doctors, 68 nurses, and midwives per 10,000 people in 2009 (1,2).

The Daniel hurricane, which began in Greece on September 5-6, affected Libya on September 10.Strong winds, heavy rainfall, and dam collapses affected the cities of Benghazi, Bayda, Marj, Susa, and Derna, causing a disaster of immense destruction and calamity (3). According to the latest assessments, it has been reported that 250,000 people were affected, 4,300 people died, 8,500 people are still missing, 42,000 people were displaced, more than 11,000 buildings were damaged, and 126 healthcare facilities were harmed.4. In our study, it is aimed to examine the emergency and public health services that should be provided in flood-related disasters using the example of the Libya disaster.

Material Method

In our study, the open-source EM-DAT disaster database was used, and emergency and basic health services in flood disasters were examined using experiences from Turkey obtained from open sources. The EM-DAT database was established in 1988 through the collaboration of the Centre for Research on the Epidemiology of Disasters (CRED) and the WHO. Currently, the organization, which is part of Health and Society attached to the University of Louvain (UCLouvain), is supported by the United States Agency for International Development (USAID).This database includes cases with 10 or more deaths, 100 or more affected individuals, declaration of a state of emergency, and calls for international assistance. Data from before 2000 has been excluded from the study due to the possibility of bias. No sample calculation was performed, and the data from 2000 to September 2023 were included in the study.

In the study, the number of disasters occurring worldwide, types of disasters, the distribution of flood disasters by regions, losses caused by flood disasters, and flood disasters by years and months were taken as variables. The data obtained from the research were transferred to a computer environment and analyzed using the IBM SPSS 23.0 (IBM SPSS Statistics, Version 23.0 Armonk,

NY: IBM Corp.) program.

In descriptive analyses, frequency data are presented as numbers and percentages, while continuous numerical data are presented as mean ± standard deviation, and ordinal variables are presented using median, 1st quartile, and 3rd quartile.

Since open-source data was used, ethical committee approval was not obtained.

Results

The EM-DAT database contains more than 26,000 records included from the 1900s to the present day. Since 2000, 36.7% of the disasters that have occurred worldwide have been classified as technological, while 63.3% have been classified as natural disasters (Table 1). The proportion of disasters caused by floods among all disasters is 27.5% (3,954).It has been reported that in disasters related to floods, approximately 1,756,605 people were affected, 329,709 people were injured, 128,092 people died, 3,617,172 people became homeless, and approximately 853,474,688,000 USD in damages occurred.

Tablo 1 Natural Disasters Occurring in the World

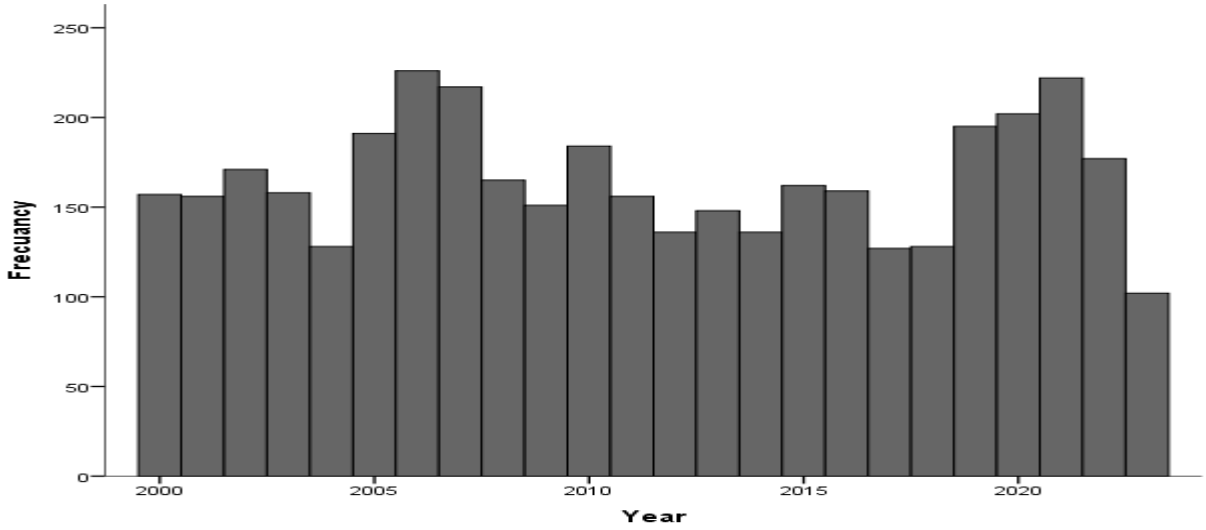
Disaster Type	n (%)	Disaster Subtype	n (%)
Technological Disasters	5658 (36.7)	Transportation Accidents	887 (15.7)
		Industrial Accidents	904 (16)
		Others	3867 (68.3)
Natural Disasters	9750 (63.3)	Biological	920 (9.4)
		Climatological	690 (7.1)
		Extraterrestrial	1 (0.01)
		Geophysics	784(8)
		Hydrological	4395 (45.1)
		Meteorological	2960 (30.4)

The numbers of people affected by flood disasters worldwide by region are given in Table 2. The disaster in which the most people were affected by a flood event occurred in China in 2003, affecting 150,000,000 people. Between 2020 and 2023, the median number of people affected per flood disaster was 10,800, with the 1st and 3rd Quartile values being 1,500 and 59,449, respectively. A minimum of 1 and a maximum of 6,054 people have died per incident related to these disasters.

Tablo 2 Flood Disaster Incidence Rates by Region

Regions	n	%
Africa	933	23.6
America	856	21.6
Asya	1595	40.3
Europe	469	11.9
Oceania	101	2.6

It has been observed that the most flood-related disaster in the world occurred in 2006, and between 2020 and 2023, flood disasters were most frequently reported in the 6th, 7th, and 8th months (Figure 1).



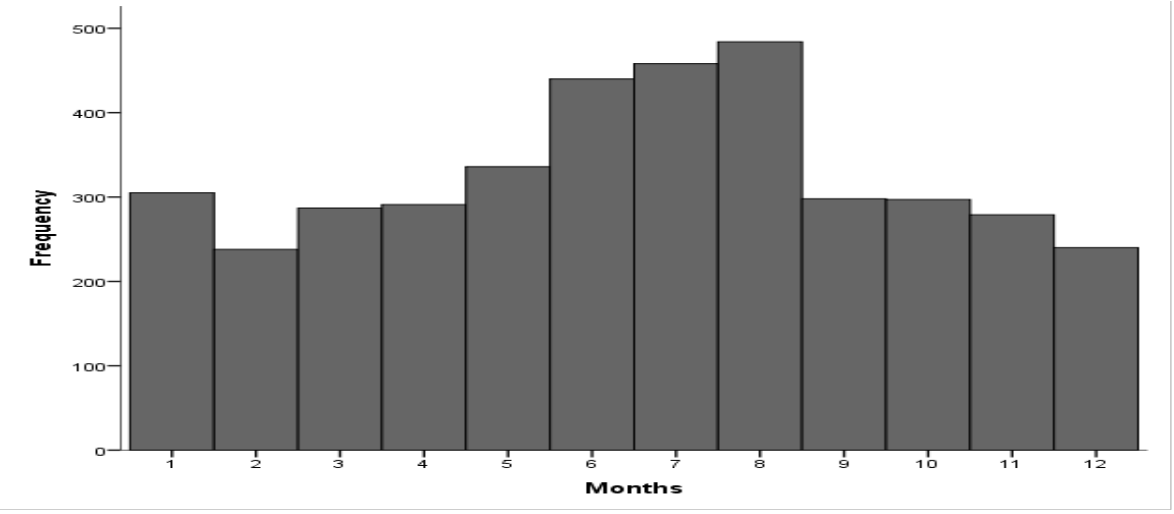


Figure 1 The Frequency of Flood Disasters by Year and Month

In Libya, 97.1% (67) of the disasters published in the EM-DAT database are of technological disaster type, while 2.9% (2) are natural disasters.In Libya, two flood disasters were reported before the recent catastrophe, the first of which occurred in 2013, affecting 2,000 people and resulting in 16 deaths, while the second occurred in 2019, affecting 20,000 people, injuring 30, and causing 4 deaths.

Discussion

In our study, as mentioned in the literature, it has been observed that hydrological disasters and meteorological disasters related to weather conditions have increased recently (6). In our study, numerical data on the magnitude of the impacts of disasters and data on the consequences of flood disasters have been presented.Considering that disasters are preventable, the importance of a modern and integrated disaster management system is better understood (7).

Flooding is a disaster dependent on geographical features and seasonal transitions.Therefore, the regions and temporal characteristics of the flood disaster identified in our study are important for predicting and taking precautions against the areas and times where the disaster may recur.One of the largest flood disasters in recent years occurred in Libya.Floods are classified based on their formation speed as slow, fast, and sudden; and in terms of their formation location as coastal, urban, dam, and river floods.The flood that occurred in the city of Derna, Libya, emerged as a sudden water overflow affecting areas close to the coastline after heavy rainfall and a dam burst (8).

Floodwaters are also carrying sand, causing homes and gardens to be filled with sand, buildings to collapse, and vehicles to be swept away, making it difficult to reach people and living beings.Search and rescue operations are important in this regard and should be services that start early.Our country has also supported Libya with trained and experienced search and rescue personnel and equipment (9).

The ongoing turmoil in Libya has also weakened the healthcare system.Considering that the flood disaster is the largest flood that has occurred in Libya according to EM-DAT data, it is foreseeable that the capacity to cope with the incident would be inadequate.The Ministry of Health of the Republic of Turkey announced that, as an initial step, they provided support to those affected by the flood disaster in Libya with a team of 159 healthcare personnel, field hospitals, medical equipment, and other supplies (10,11). Vaccination (Recommended vaccines: Measles-Mumps-Rubella (MMR), Diphtheria, Tetanus, Pertussis, Chickenpox, Polio, and annual flu vaccine) (12) was carried out by travel health teams before the trip, and the journey took 2 days by ship departing from the port of Izmir.Within Libya, the city of Derna was reached by road.

In disasters, basic health services and emergency health services are services that should be provided starting from the zero hour of the disaster.

The most common early health impact of a flood disaster and the leading cause of death is drowning.The most common early health impact of a flood disaster and the leading cause of death is drowning.Other significant causes are electric shock, injuries related to being swept away by floods or dragged by floating objects, and deaths associated with these.Bites from aquatic creatures and individuals contaminated with chemical, biological, or radioactive substances, psychological conditions, and suicide attempts are also other issues that may be encountered.Emergency medical services primarily combat the effects in this area.At the same time, they must also be prepared for acute situations that may arise in the field teams.The teams working in this field can be divided into pre-hospital (field medical teams, injured or patient transfer teams) and hospital teams.Those working in field and transfer teams should be prepared and experienced in hypothermia, hypoglycemia, arrhythmias, and advanced life support.At the same time, they should not forget that field personnel may encounter smoke and dust exposure, which can be confused with airway disease exacerbations, heat/cold strokes, and traumas.Those working in hospital emergency medical teams should be careful with triage, as saving more patients with fewer resources is one of the fundamental principles of disaster medicine.They should be prepared and have reviewed their medical equipment and supplies in relation to the situations and patients mentioned above.When evaluating patients, triage

and examination cards, as well as the recording of information related to the procedures performed, are points that should not be overlooked in terms of preventing repeated treatments and follow-up.In resuscitated patients, body temperature should be targeted in the range of 32-35 °C. It should not be forgotten that in the subacute period, there will be an increase in cutaneous infections, aspiration pneumonia, viral infections, and gastroenteritis conditions (13-16). The equipment and personnel sent by the Ministry of Health have provided services in this context regarding emergency healthcare services (10).

With the flood disaster, areas of focus for public health and basic health services include the provision of sufficient and clean water in a short period, difficulties in accessing safe food, the establishment of safe shelter areas for displaced persons, an increase in infectious diseases (especially cholera, cryptosporidiosis, nonspecific gastroenteritis, polio, rotavirus, typhoid-paratyphoid outbreaks), and nutrition problems, primarily malnutrition.Therefore, public health services and community information efforts are extremely important from the moment of the disaster.In these areas, floodwaters should not be used, contact with these waters should be avoided, food contamination should be prevented, attention should be paid to consuming packaged water, efforts should be initiated early for chlorination and the provision of clean water sources, water analyses should be conducted promptly, and a surveillance system should be implemented.After the incident, it is important to focus on combating pests and rodents, as well as houseflies and mosquitoes, which may increase in the area.An important matter is the proper storage and burial of corpses, as well as waste collection and management services (17,18). Routine public health services, such as childhood vaccinations, monitoring of non-communicable diseases, and child-adolescent-reproductive health services, should be reinstated as short- to medium-term goals (19).

Conclusıon

The Libya flood disaster is an important catastrophe that causes serious destruction and demonstrates the extent to which dam-related flood events can reach.Without a doubt, the most important step in the management phase of the flood disaster is to ensure that urban planning is conducted away from riverbeds and dam/reservoir drainage areas.In disaster management, emergency medical services and primary healthcare services are important healthcare services that need to be carried out simultaneously.Although the disasters experienced have painful consequences, the organization of health services in disasters is of vital importance, and each incident should be evaluated as a lesson to prevent the repetition of mistakes.The Libya flood disaster, being a dam-related flood event and considering the serious consequences that emerged, should be addressed in detail.

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Immature Granulocyte Levels and Clinical Severity in Acute Coronary Syndrome: A Preliminary Study

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Introduction

ST-segment elevation myocardial infarction (STEMI) remains a major cause of morbidity and mortality worldwide(1, 2), despite considerable advances in reperfusion strategies and modern pharmacological therapies . Early risk stratification is critical in managing patients with acute coronary syndromes (ACS), especially in the emergency setting. Traditionally, prognostic tools have included troponin levels, N-terminal pro-brain natriuretic peptide (NT-proBNP), and scoring systems such as the GRACE and TIMI scores (3, 4). However, increasing evidence supports the central role of inflammation in the pathophysiology of myocardial infarction (MI), particularly in STEMI, where excessive inflammatory responses occur due to ischemic injury (5).

The immature granulocyte (IG) count, easily measurable via automated complete blood count (CBC) analyzers, reflects the presence of early myeloid precursors in peripheral blood and serves as a surrogate marker of systemic inflammation(6, 7). Although the diagnostic and prognostic utility of inflammatory markers such as neutrophil-to-lymphocyte ratio (NLR) and C-reactive protein (CRP) have been previously reported, recent studies have explored the potential of IG levels as a predictor of disease severity and short-term mortality in various critical conditions including STEMI (6, 8, 9).

In a study by Korkut et al. (6), elevated IG levels measured at emergency department admission were significantly associated with in-hospital mortality among patients with STEMI. The authors reported that an IG cut-off value of 0.65 predicted mortality with 72.2% sensitivity and 77.8% specificity (AUC: 0.740, 95% CI: 0.635–0.846, $p<0.001$)[1]. Furthermore, IG was found to be an independent predictor of mortality, alongside age, ejection fraction, anemia, and Killip class.

Despite its practicality and low cost, the IG count remains underutilized in routine clinical risk assessment for ACS patients. Given its potential role in reflecting the inflammatory burden and early prognostic implications, further investigation into its clinical relevance is warranted. The current study aims to evaluate the relationship between IG levels and clinical severity parameters in patients diagnosed with acute coronary syndrome, with a focus on their potential prognostic significance.

Methods

This retrospective, single-center observational study was conducted in the Emergency Department of Ondokuz Mayis University Faculty of Medicine between January 1, 2022, and January 1, 2023.

Patients presenting to the emergency department with a diagnosis of acute coronary syndrome (ACS) during the study period were screened for eligibility. Inclusion criteria consisted of being 18 years of age or older, having a diagnosis of ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI) based on clinical and electrocardiographic findings, and having a complete set of laboratory and clinical data available at the time of presentation. Specifically, the availability of immature granulocyte (IG) count, high-sensitivity troponin I (hs-TnI), liver enzyme levels (AST, ALT), and echocardiographic left ventricular ejection fraction (LVEF) was required for inclusion in the study.

Patients were excluded if they were under 18 years of age, had an active systemic infection unrelated to ACS, or if their clinical or laboratory data were incomplete.

Demographic data, comorbidities (such as hypertension, diabetes mellitus, history of coronary artery disease, chronic heart failure, smoking status, and family history), vital signs, and relevant laboratory parameters were retrieved from electronic health records. IG and IG% values were automatically reported as part of the complete blood count using a standardized hematology analyzer. Electrocardiogram findings at admission were used to classify patients as STEMI or NSTEMI, and Killip class was also recorded as an indicator of clinical severity.

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 21.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as medians due to non-normal distribution, which was tested using the Kolmogorov–Smirnov test. Categorical variables were expressed as frequencies and percentages. Pearson's chi-square was employed for categorical variables where appropriate. Receiver operating characteristic (ROC) curve analysis was used to evaluate the diagnostic performance of IG and IG% in identifying high-risk clinical presentations, with STEMI serving as a surrogate marker of increased clinical severity. A p-value of less than 0.05 was considered statistically significant.

Results

A total of 83 patients diagnosed with acute coronary syndrome (ACS) were included in the final analysis. The median age was 68 years (range 38–91), and 61.4% of the patients were male. ST-elevation myocardial infarction (STEMI) was present in 30.1% (n=25), while 69.9% (n=58) were diagnosed with non-ST-elevation myocardial infarction (NSTEMI). Age, hs-troponin, IG, IG%, AST, ALT, and ejection fraction (EF) were presented as medians with minimum and maximum values.

IG levels ranged from 0 to 1.2 (median 0.0), and IG% ranged from 0.1% to 4.5% (median 0.4%). The median hs-troponin level was 1565 ng/L, and the median EF was 50% (Table 1).

Table 1. Baseline Clinical and Laboratory Characteristics of the Study Population

	Median (min-max)
Age	68 (38 - 91)
HsTroponin	1565 (2 - 25000)
IG	0 (0 - 1,2)
IG%	0,4 (0,1 - 4,5)
AST	31 (10 - 586)
ALT	21,2 (5 - 143)
EF	50 (30 - 69)

Hypertension was the most prevalent comorbidity (80.7%), followed by diabetes mellitus (45.8%) and coronary artery disease (45.8%). The prevalence of chronic heart failure was 36.1%, and 26.5% of patients reported a positive family history of cardiovascular disease (Table 2).

Table 2. Distribution of Demographic Features and Comorbid Conditions

Gender		
Male	51	61.4
Female	32	38.6
ACS		
NSTEMI	58	69.9
STEMI	25	30.1
HT		
Yes	67	80.7
No	16	30.1
DM		
Yes	38	45.8
No	45	54.2
Dyslipidemia		
Yes	13	15.7
No	70	84.3
CAD		
Yes	38	45.8
No	45	54.2
Smoking		
Yes	41	49.4
No	42	50.6
Family History		
Yes	22	26.5
No	61	73.5
CHF		
Yes	30	36.1
No	53	63.9

A statistically significant association was found between Killip classification and MI type (p = 0.008). Killip class II and IV were more frequently observed among STEMI patients, whereas class III was exclusive to the NSTEMI group. This finding suggests a higher clinical severity in STEMI presentations (Table 3).

Receiver operating characteristic (ROC) analysis showed that IG had a modest but statistically significant diagnostic performance in predicting STEMI. The area under the curve (AUC) was 0.665 (95% CI: 0.536–0.794, p = 0.018). At a cut-off value of 0.35, IG demonstrated a sensitivity of 76.0% and specificity of 56.1% (Table 4) (Figure 1).

Discussion

In recent years, it has become increasingly clear that inflammatory mechanisms play a key role in the pathophysiology of acute

coronary syndromes (ACS), especially in ST-elevation myocardial infarction (STEMI). The intense inflammatory response triggered by ischemic injury in STEMI has been shown to impact infarct size, healing dynamics, and overall clinical outcomes (5, 10). Given this, there is growing interest in identifying inflammatory biomarkers that may aid in early risk stratification in patients with ACS.

Immature granulocytes (IGs), which represent early-stage myeloid precursors, tend to appear in peripheral circulation in response to systemic inflammatory stimuli. IGs can be quantified automatically through standard complete blood count testing, making them a convenient and cost-effective biomarker (6, 7). Several previous studies have investigated the prognostic significance of IG levels in critically ill patients, including those with sepsis (8), intracerebral hemorrhage (9), and various intensive care conditions (7).

A recent study by Korkut et al. demonstrated that higher IG levels on admission were significantly associated with in-hospital mortality among STEMI patients. They identified a cut-off value of 0.65 for IG, which predicted mortality with an area under the curve (AUC) of 0.740, achieving sensitivity and specificity rates of 72.2% and 77.8%, respectively (6). Although our study could not perform a mortality-based analysis due to the limited number of fatal cases, IG levels were found to be significantly elevated in the STEMI group when compared to NSTEMI patients. This observation suggests a potential link between IG elevation and increased clinical severity in ACS.

Table 3. Association Between Killip Classification and ACS Type

		ACS			
		NSTEMI	STEMI	Total	p value*
Killip Classification	I	29 (80.6)	7 (19.4)	36	0.008
	II	19 (54.3)	16 (45.7)	35	
	III	9 (100.0)	0 (0.0)	9	
	IV	1 (33.3)	2 (66.7)	3	

*Pearson Chi Square test

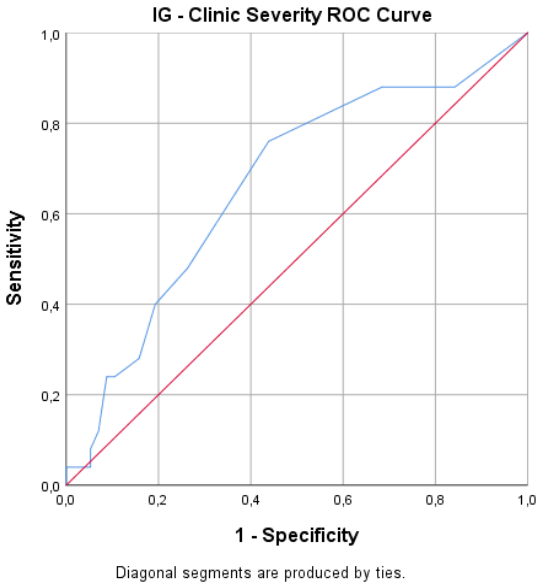

Figure 1. Diagnostic Performance of IG for Identifying STEMI (ROC Analysis)

Table 4. Diagnostic Performance of IG for Identifying STEMI (ROC Analysis)

AUC (95% CI)	0.665 (0.536-0.794)
p	**0.018**
Cut off value	0.35
Sensitivity:	76.0 %
Specificity:	56.1 %

Moreover, our ROC analysis confirmed that IG levels provided a modest yet statistically significant ability to distinguish STEMI presentations (AUC = 0.665; p = 0.018). While these findings alone may not establish IG as an independent prognostic factor, they are consistent with the idea that higher IG levels reflect a more intense systemic inflammatory burden, which often correlates with worse clinical status in ACS.

Inflammatory markers such as the neutrophil-to-lymphocyte ratio (NLR) and C-reactive protein (CRP) have been widely studied in relation to ACS outcomes (3, 4). IG, however, may offer unique advantages due to its real-time availability, minimal cost, and automatic reporting from routine hematological panels. The relevance of systemic inflammation in cardiovascular disease progression has also been highlighted by Mozos et al., reinforcing the importance of incorporating inflammatory markers into clinical decision-making frameworks (5).

Additional studies have validated the role of IG as a marker of physiological stress. For instance, Lima et al. showed its utility in detecting infection in ICU patients (7), while Sinaga et al. found a significant association between elevated IG levels and 30-day mortality in septic patients (8). These results, taken together with our own, support the hypothesis that IG could serve as a general indicator of disease severity across various acute pathologies.

However, our study is not without limitations. Being retrospective and conducted at a single center, the generalizability of our findings is limited. Furthermore, the relatively small sample size and low incidence of adverse events, particularly mortality, restricted our ability to explore the prognostic utility of IG in depth. Although patients with known systemic infections were excluded, undiagnosed or subclinical inflammatory conditions might have influenced IG levels in some cases.

This investigation should be viewed as a preliminary study aimed at evaluating the possible role of IG in the clinical assessment of ACS patients. Due to the limited sample size and lack of mortality data, definitive conclusions regarding its prognostic value could not be drawn. Nevertheless, the significant association between IG levels and STEMI presentations provides a rationale for future prospective research. Upcoming studies with larger sample sizes and more advanced analytical approaches are warranted to determine whether IG can serve as a reliable biomarker for both short- and long-term outcomes in ACS.

Conclusion

In this preliminary study, immature granulocyte (IG) levels were found to be significantly higher in patients presenting with ST-elevation myocardial infarction (STEMI) compared to those with non-ST elevation myocardial infarction (NSTEMI). Although mortality analysis could not be performed due to the limited number of fatal cases, the association between elevated IG and STEMI suggests that IG may reflect increased clinical severity in acute coronary syndromes (ACS).

Given its rapid availability, low cost, and potential link to systemic inflammation, IG represents a promising candidate for further investigation as a clinical marker in ACS. Future prospective studies with larger cohorts are needed to evaluate its prognostic value, particularly in relation to short- and long-term mortality outcomes.

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Transfusion-Related Acute Lung Injury (TRALI) and Concurrent Acute Coronary Syndrome in the Emergency Department: A Multidisciplinary Case Report

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Introductıon

Blood transfusion is a vital and frequently employed intervention in emergency medicine. While it serves as a critical therapeutic measure in patients with severe anemia or acute hemorrhage, it is not devoid of potential complications. Among these, Transfusion-

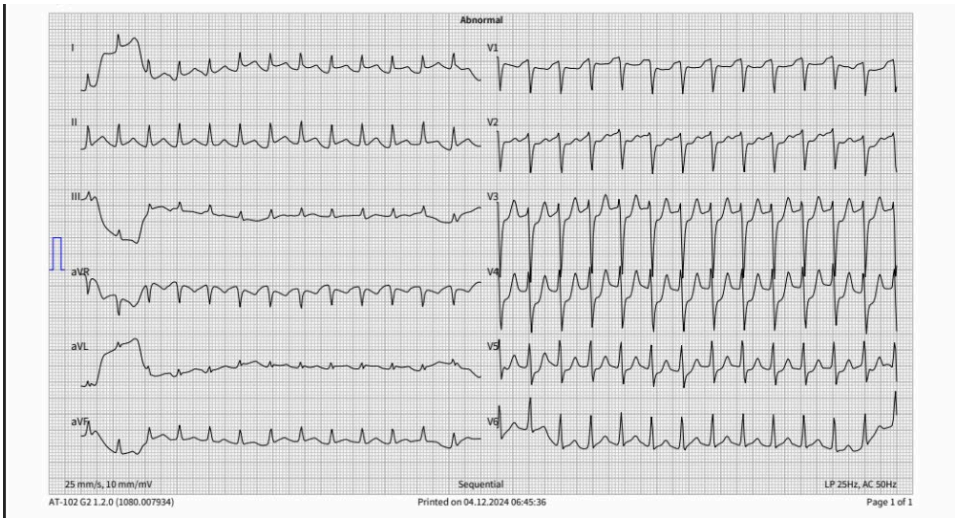
Related Acute Lung Injury (TRALI) represents one of the most severe and life-threatening adverse reactions. It is a clinical syndrome characterized by the sudden onset of non-cardiogenic pulmonary edema within six hours of transfusion and is associated with high morbidity and mortality rates (1,2). The clinical presentation typically involves acute hypoxemia and bilateral pulmonary infiltrates without evidence of circulatory overload or pre-existing cardiac dysfunction. Despite advancements in transfusion safety protocols, TRALI remains underdiagnosed due to its overlapping symptoms with ARDS, pneumonia, and cardiogenic pulmonary edema (3,4). This case report illustrates a complex scenario where TRALI developed simultaneously with Acute Coronary Syndrome (ACS), complicating the diagnostic approach and necessitating prompt, multidisciplinary intervention. It highlights the importance of early recognition, accurate diagnosis, and integrated management strategies in optimizing patient outcomes (5).

Case

A 66-year-old female patient with no known chronic medical conditions presented to the emergency department with a four-month history of persistent abdominal pain. Over the past three months, she also reported significant weight loss of approximately 15 kilograms and progressive anorexia. Upon presentation, she was alert, oriented, and cooperative, with a Glasgow Coma Score of 15. Her initial vital signs revealed tachycardia (heart rate: 110 bpm), normotensive blood pressure (120/73 mmHg), and normal oxygen saturation on room air. On physical examination, abdominal tenderness was present, but no defence or rebound tenderness was observed.

Initial laboratory analysis demonstrated a hemoglobin level of 6.5 g/dL, consistent with severe microcytic anemia. Abdominal computed tomography (CT) imaging showed a thick-walled hypodense lesion in the left adnexal region suggestive of a possible ovarian mass, heterogeneity of the uterine cervix, several small pelvic lymph nodes, and a moderate pleural effusion in the right hemithorax measuring up to 3 cm. The patient was consulted with gynecology and urology departments. Surgical intervention was not considered.

The patient was transfused with two units of erythrocyte suspension. Approximately 30 minutes following completion of the second transfusion, she developed acute respiratory symptoms including palpitations, tachypnea, and a significant drop in oxygen saturation. She also exhibited an altered level of consciousness. An electrocardiogram revealed atrial fibrillation with rapid ventricular response. Intravenous metoprolol was administered to the patient and sinus rhythm was successfully achieved. However, clinical deterioration continued with bilateral coarse pulmonary crackles, reduced Glasgow Coma Score (11), and blood gas analysis confirming both hypoxemia and hypercapnia. Noninvasive ventilation was started, but this was not tolerated adequately and endotracheal intubation and mechanical ventilation were required.

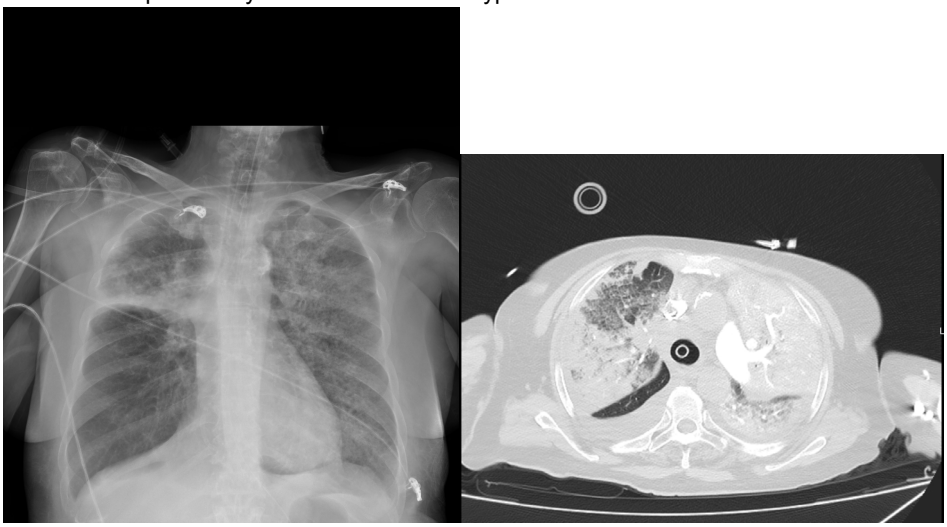


Post-intubation thoracic CT scan demonstrated bilateral pleural effusions, total collapse of the left upper lobe, diffuse ground-glass opacities in both lower lobes, and nodular infiltrates in the right upper lobe. These radiographic findings, in conjunction with the clinical context and timing of symptom onset, were deemed highly suggestive of TRALI. The patient was promptly transferred to the intensive care unit (ICU), where she was sedated with continuous propofol infusion. Subsequent laboratory investigations revealed a dramatic rise in serum troponin levels, increasing from 2200 ng/L to 50000 ng/L over several hours. Given the marked troponin elevation and hemodynamic instability, urgent cardiology consultation was sought. Coronary angiography demonstrated complete occlusion of the right coronary artery, and a successful percutaneous coronary intervention (PCI) was performed. The patient continued to be monitored in the intensive care unit for multidisciplinary approach and treatment.

Discussion

TRALI represents a serious and potentially fatal complication of blood transfusion, characterized by the acute onset of respiratory distress, non-cardiogenic pulmonary edema, and bilateral chest infiltrates. It is now recognized as one of the leading causes of transfusion-related mortality, surpassing hemolytic reactions in some surveillance reports (1,9). Pathophysiologically, TRALI is understood to involve a two-hit mechanism: a predisposing clinical condition that primes the pulmonary endothelium, followed by exposure to transfused mediators such as antibodies or bioactive lipids that trigger an inflammatory cascade (10).The resulting capillary

leak leads to pulmonary edema and severe hypoxemia.



In the presented case, the diagnosis of TRALI was established based on the temporal relationship between transfusion and symptom onset, the absence of cardiac dysfunction, and characteristic imaging findings. Notably, the simultaneous occurrence of ACS posed significant diagnostic and therapeutic challenges. The elevation in cardiac biomarkers and coronary artery occlusion further complicated clinical management, necessitating urgent revascularization in addition to respiratory support (5,8).

Management of TRALI is primarily supportive, focusing on oxygenation, hemodynamic stability, and cessation of further transfusions. While pharmacologic interventions such as corticosteroids and diuretics have been proposed, evidence supporting their efficacy remains limited and inconclusive (11). Early identification and intervention are crucial to improve patient prognosis. The presence of comorbid conditions, particularly cardiovascular compromise, may exacerbate the clinical severity of TRALI and should prompt a collaborative treatment strategy involving emergency physicians, intensivists, pulmonologists, and cardiologists (3,12).

This case underscores the importance of heightened clinical suspicion for TRALI in patients presenting with acute respiratory deterioration post-transfusion, especially in the emergency department setting. It also reinforces the need for rapid differentiation from other causes of pulmonary compromise, including cardiogenic pulmonary edema, aspiration, and sepsis-associated ARDS. Multidisciplinary management and adherence to evidence-based protocols should always be important for the clinician to improve outcomes in such complex clinical presentations.

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7244

A rare complication of abdominal pain after upper gastrointestinal endoscopy: iatrogenic gastric perforation

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Introductıon

Gastric perforation is an emergency surgical condition resulting from a breach in the gastric wall, leading to the leakage of gastric contents into the abdominal cavity, which can cause serious complications. This condition creates a clinical picture known as “acute abdomen,” which is life-threatening and requires immediate medical intervention. Gastric perforation typically presents with acute peritonitis and free air in the abdominal cavity. Life-threatening complications such as intra-abdominal infection,peritonitis,sepsis, and multi-organ failure may develop.Therefore, these patients require prompt diagnosis and treatment.

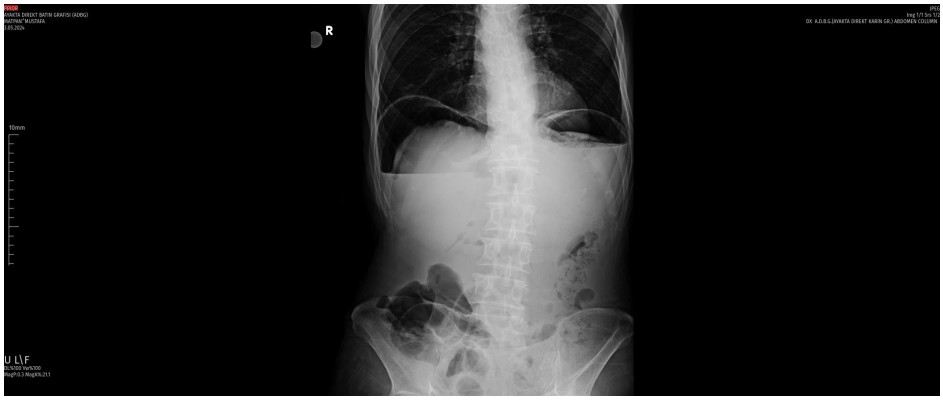
The most common cause of gastric perforation is peptic ulcer disease. Other causes include excessive alcohol consumption, NSAID use, trauma, malignancies, and, rarely, certain infections. Iatrogenic gastric perforation, though rare, is also a significant cause, usually occurring because of upper gastrointestinal endoscopy.

Upper gastrointestinal (GI) endoscopy is a diagnostic procedure used to evaluate diseases of the esophagus,stomach, and proximal duodenum. Endoscopy is primarily used for diagnostic purposes.

However, with technological advancements, its use for therapeutic purposes has also become more widespread(1). Upper GI endoscopy is generally considered a safe procedure; however, all endoscopic procedures are invasive and carry a risk of iatrogenic perforation. Although rare, perforation during upper GI endoscopy is a serious complication(2,3). While the risk of perforation is low, procedural difficulties, anatomical locations of perforations, and the experience of the endoscopist can increase the likelihood of this complication(3). Perforations most commonly occur in the esophagus but can also be seen in the stomach and duodenum(1). Iatrogenic endoscopic perforations may require emergency surgery and can occasionally be fatal. Early diagnosis and treatment can prevent mortality and morbidity. Early detection is crucial because the leakage of contaminated food or fluids into the thoracic or abdominal cavity can lead to fatal infections. In cases of acute iatrogenic perforation diagnosed early, endoscopic clips can be used to close the defect, allowing for rapid healing. However, late diagnosis can lead to progressive inflammation around the area, making endoscopic closure more challenging(3).

Case Report

A 63-year-old male patient presented to the emergency department with worsening abdominal pain that started 3 h after endoscopy. On physical examination of the abdomen, diffuse tenderness, guarding, rebound, and a board-like rigidity were detected on palpation; bowel sounds were decreased on auscultation. Laboratory tests showed elevated WBC (11,000) and CRP(22.18), but no other abnormalities. X-ray showed a double-contour air image under the diaphragm, and abdominal CT with contrast revealed the following: “Free fluid was observed in all quadrants of the abdomen. Free air findings were present in the abdomen. The appearance was interpreted as being consistent with the perforation. The edematous appearance of the distal gastric wall was noted. The perforation site may be located here. Clinical correlation is recommended.” The patient was diagnosed with “Acute Abdomen and Gastric



Perforation” and underwent emergency surgery by the General Surgery department. The patient, who was followed up in the General Surgery intensive care unit, was discharged with recovery on the 7th postoperative day.

Figure 1: Abdominal X-ray: A double-contour air image was observed under the diaphragm.

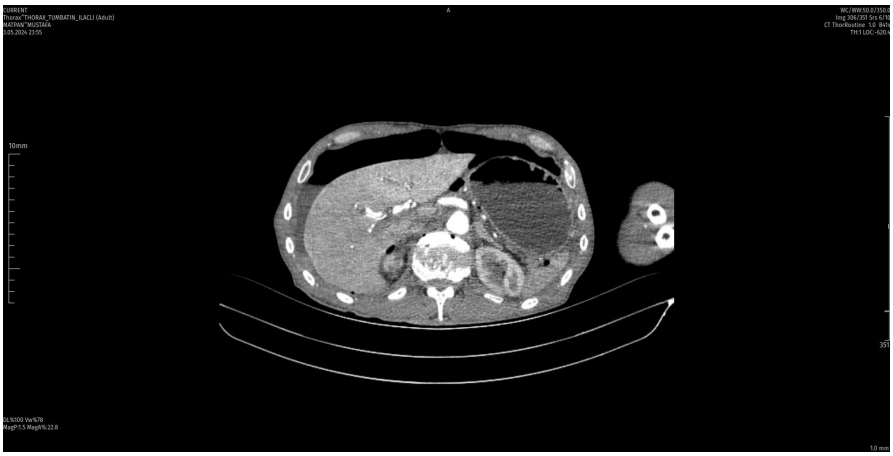


Figure 2: In abdominal CT with contrast, free fluid and free air are observed in all quadrants of the abdomen. The image also highlights an edematous apperance of gastric wall distal to the stomach.

Dıscussıon

Gastrointestinal endoscopy plays a crucial role in the diagnosis and treatment of many GI disorders. It is a fundamental procedure for obtaining biopsy samples and assessing digestive system diseases, with its applications continually expanding(1,3). Although rare, complications such as perforation, which can lead to morbidity and mortality, have been reported in the literature(1-3,5).

GI perforations can occur due to peptic ulcer disease, trauma, iatrogenic causes, foreign bodies, appendicitis, inflammation, tumors, and other conditions. Peptic ulcer disease is the most common cause of gastric perforation(4). Although rare, iatrogenic GI perforation can result from endoscopic interventions(1-3,5). The most frequent iatrogenic cause is upper GI endoscopy(3,5). Iatrogenic perforations are more common in patients with pre-existing gastric pathology(6). Early diagnosis and treatment are crucial for saving lives(3). Patients should be closely monitored for potential complications after endoscopic procedures.

After upper GI endoscopy, patients should be monitored for bleeding and perforation-related complications. Those scheduled for discharge should be adequately informed about possible complications, both for themselves and their caregivers(7).

Conclusıon

This case presentation involves the evaluation of a patient who presented to the emergency department with a gastric perforation following an endoscopic procedure. Abdominal examination and advanced imaging play a critical role in the early diagnosis and treatment of gastric perforation. In patients presenting with anacute abdomen, early surgical intervention is necessary. Early diagnosis, appropriate surgical intervention, and postoperative care are crucial for minimizing complications and mortality.

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7258

Diplopia: A Rare Side Effect of Hyponatremia: A Case Report

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Introduction

Hyponatremia, defined as a serum sodium concentration of less than 135 mEq/L, is the most common electrolyte disorder encountered in patients presenting to the emergency department (1). In daily clinical practice, hyponatremia, which we frequently encounter, can occur due to a decrease in sodium intake from the diet, diseases that cause excessive water retention in the body, or conditions that cause excessive sodium loss from the body. Depending on the rate and severity of the decrease in serum sodium concentra-

tion, patients with hyponatremia may be asymptomatic or symptomatic with various complaints such as nausea-vomiting, headache, weakness, lethargy, muscle cramps, dysarthria, seizures, loss of consciousness, and coma (2). Treatment of hyponatremia varies depending on the underlying cause and the severity of the hyponatremia.

Idiopathic intracranial hypertension (IIH) is a syndrome characterized by increased intracranial pressure of unknown cause. Although the underlying pathophysiological mechanisms of the increase in intracranial pressure in idiopathic IIH are not fully understood, it has been reported that it may develop due to an excessive increase in the production of cerebrospinal fluid (CSF), a decrease in CSF absorption, or increased cerebral venous pressure (3). IIH diagnosis is made by measuring CSF pressure with lumbar puncture, an invasive procedure. In cases where lumbar puncture cannot be performed, the diagnosis is based on the patient's complaints, eye examination findings, and findings on imaging methods.

Upon reviewing current literature, we observed cases of transient blindness due to postoperative acute hyponatremia (4), vision impairment due to ophthalmoplegia following acute hyponatremia in a patient with nasopharyngeal cancer (5), and neuromyelitis optica associated with acute hyponatremia (6). However, to our knowledge, there are no reported cases of diplopia resulting from intracranial hypertension in a hyponatremic patient. The purpose of presenting this case is to emphasize that hyponatremia, a common electrolyte disorder, is a potential risk factor for intracranial hypertension.

Case

A 51-year-old male patient presented to the emergency department with complaints of throbbing headache, dizziness, and double vision for the past 4-5 days. His medical history revealed diagnoses of Diabetes Mellitus and Hypertension 3 months prior, for which he was started on an antihypertensive agent (ACE inhibitor) and an antidiabetic agent (Metformin). Further history revealed that the patient had been adhering to a salt- and water-restricted diet since his hypertension diagnosis. His vital signs upon arrival to the emergency department were: Temperature: 36.5° C, Heart rate: 80 bpm, Blood pressure: 125/75 mmHg, Respiratory rate: 16 breaths/minute, and Oxygen saturation: 97% on room air. The patient was cooperative and oriented, with no motor deficits and a muscle strength of 5/5 in all four extremities. Deep tendon reflexes and plantar responses were normoactive. When asked to walk, he exhibited an ataxic gait. He walked normally when one eye was covered. The remainder of the patient's systemic examination was normal.

During the eye examination of a patient consulted for double vision due to eye diseases, the light reflex was bilaterally ++/++, eye movements were free in all directions bilaterally, visual acuity was normal in both eyes (70%/80%, +2.00/+2.00), intraocular pressure was within normal limits in both eyes (15/17 mmHg), and fundus examination revealed blurred optic disc margins and peripapillary hemorrhage in both eyes, along with papilledema and horizontal diplopia.

The ophthalmologist recommended ruling out increased intracranial pressure (ICP), and we wanted to perform a Lumbar Puncture (LP). However, the patient refused LP in the emergency department, so a cranial Computed Tomography (CT) scan of the brain was performed to detect increased intracranial pressure. The cranial CT scan showed no findings suggestive of increased ICP (İmage 1). To better visualize increased intracranial pressure in this case with a normal cranial CT, non-contrast brain T2-weighted Magnetic Resonance (MR) imaging was performed. Axial and sagittal sections of the non-contrast T2-MR revealed findings suggestive of intracranial hypertension (increased bilateral optic nerve tortuosity and perioptic CSF space, flattening of the posterior aspects of the bilateral globes, partial empty sella) (İmages 2-3-4). The patient's laboratory data are provided in detail in Table 1.

After receiving two doses of 3% hypertonic saline solution within 24 hours in the emergency department, the hyponatremic patient was admitted to the nephrology service. The nephrology service permitted a lumbar puncture (LP). The LP revealed a cerebrospinal fluid (CSF) pressure of 42 cmH2O (normal range: 9-16 cmH2O). During the patient's stay in nephrology, they received three additional doses of 3% hypertonic saline solution along with diuretic therapy. On the 5th day of treatment, the patient's plasma sodium concentration returned to normal reference levels, and their diplopia resolved. Detailed investigation revealed that the patient's hyponatremia was due to decreased dietary sodium intake. The patient, with resolution of their symptoms, was discharged with recommendations for outpatient follow-up.

İmage 1: CT image within normal limits.

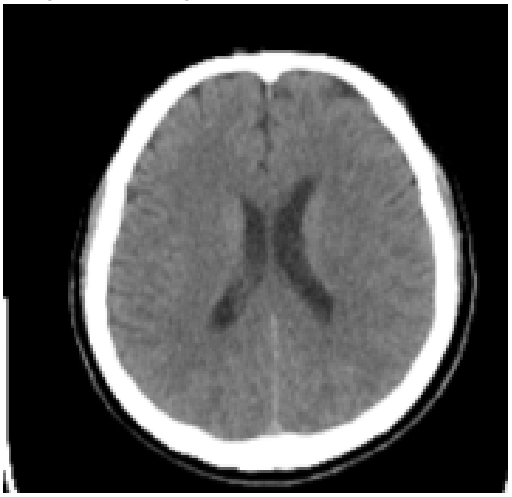

İmage 2: Bilateral optic nerve tortuosity and increased perioptic CSF space.

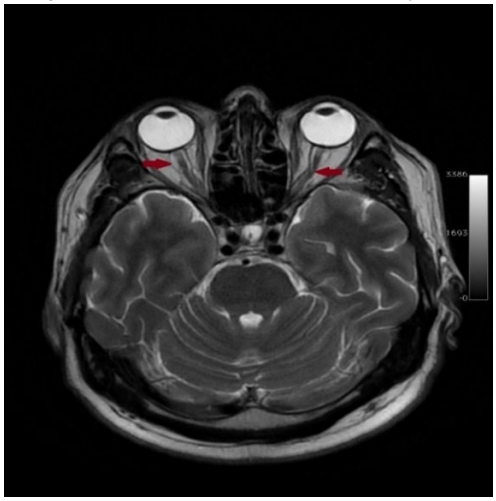

Image 3: Bilateral flattening of the posterior aspect of the eyeballs.

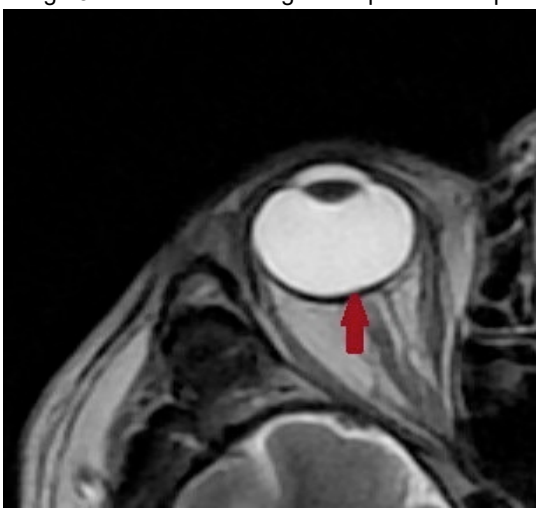

İmage 4: partial empty sella

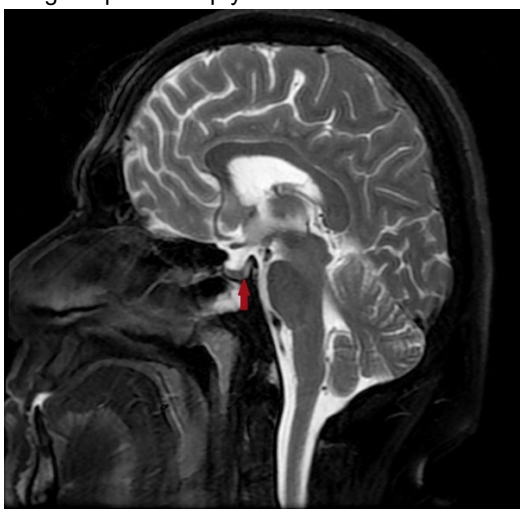

Table 1.Patient's laboratory results upon admission and on the 5th day

Tests	Results at the Time of Application	Results from Day 5	Reference Ranges
WBC (10^3/uL)	13.5	12,02	4.49-12.68
Hemoglobin (g/dL)	15.5	14,3	13.5-16.9
Plateletes Count (10^3/uL)	428	403	173-390
Sodium (mEq/L)	116	137	136-145
Potassium (mEq/L)	3.9	4,01	3.5-5.1
Chlorine (mEq/L)	81	99	96-107
Calsium (mg/dL)	9.5	9,61	8.6-10.0
BUN (mg/dL)	13	12	6-20
Kreatinin (mg/dL)	0.76	0,82	0.7-1.2
GGT (U/L)	45	37	10-71
ALT (U/L)	60	17	0-41
AST (U/L)	30	37	0-40
Amylase (U/L)	46	48	28-100
Lipase (U/L)	28	30	13-60
CPK (U/L)	129	131	39-308
LDH (U/L)	237	223	135-214
Glucose (mg/dL)	174	142	70-110
Laktat (mmol/L)	2.0	1.5	0.4-1.4
Baz exces (mmol/L)	0.8	1.2	2-3
Total Bilirubin (mg/dL)	0.45	0,22	0-1.2
Direct Bilirubin (mg/dL)	0.18	0,11	0-0.3
CTn-I (ng/L)	7.79	5.67	0-47.3
Protrombin time (second)	11.9	11.4	10-14
Activated Partial Thromboplastin Time (seconds)	32.6	31.5	25.1-36.5
International normalized ratio (INR)	1.03	1.08	0.85-1.15
Arter Blood Gas			
pH	7.42	7.40	7.35-7.45
pO_2 (mmHg)	96	96	83-108
pCO_2 (mmHg)	39	38	32-48
HCO_3 (mmol/L)	25	24	22-26

Discussion

Diplopia can occur due to dysfunction of the extraocular eye muscles or conditions that cause increased intracranial pressure (ICP), such as intracranial mass lesions (brain tumor and hematoma), cerebral edema, obstructive hydrocephalus, multiple sclerosis, neuro-Behçet's disease, pseudotumor cerebri, venous sinus thrombosis, increased cerebrospinal fluid (CSF) production, and decreased

CSF absorption. The most important characteristic of diplopia caused by increased intracranial pressure is that the diplopia present with both eyes open disappears when one eye is closed (7). Our patient also stated that diplopia was present when walking with both eyes open but disappeared when walking with one eye closed. Therefore, we suspected that the cause of the diplopia was a central neurological reason (intracranial hypertension) and, to confirm this suspicion, we wanted to measure CSF pressure by performing a lumbar puncture (LP) in the emergency department. However, because the patient did not consent to LP, we could not measure CSF pressure and instead used non-invasive diagnostic methods (fundoscopic examination findings, cranial computed tomography (CT), and cranial magnetic resonance imaging (MRI)) to demonstrate the increase in intracranial pressure.

In our patient's eye examination, findings suggestive of increased intracranial pressure were detected, such as papilledema, peripapillary hemorrhage, blurring of the optic disc margin, and horizontal diplopia. To further strengthen our diagnosis, we performed a cranial CT scan. However, the CT scan did not reveal any findings suggestive of increased intracranial pressure (İmage1). Subsequently, a non-contrast T2-weighted cranial MRI was performed. The T2-weighted cranial MRI revealed findings suggestive of increased intracranial pressure, such as bilateral optic nerve tortuosity and increased perioptic CSF space, flattening of the posterior aspect of the bilateral bulbus oculi, and a partial empty sella (İmages 2-4).

Patients with increased intracranial pressure most frequently present to the hospital with headaches, and less frequently with nausea-vomiting, dizziness, tinnitus, visual complaints, altered consciousness, and weakness in the extremities. A characteristic feature of headaches caused by increased intracranial pressure is that the severity of the headache changes with position, and photophobia accompanies the pain (8). Our patient presented to the emergency department with headache and diplopia, but the headache was not accompanied by photophobia. Although headaches due to increased intracranial pressure may subside after analgesic administration, it has been reported that the pain persists for a long time in some cases (9). Our patient's headache regressed after paracetamol treatment but did not completely disappear.

Although many neurological disorders secondary to hyponatremia have been reported in the literature, cases of visual disturbances are quite rare. We believe that the patient's double vision and headache developed due to increased intracranial pressure, and that hyponatremia is responsible for this increase in intracranial pressure. We can say that intracranial hypertension in hyponatremia develops due to the osmolarity difference between CSF and blood. Indeed, after our patient's hyponatremia was corrected, the intracranial pressure returned to normal and the horizontal diplopia disappeared.

A study of 65 patients with sodium imbalance revealed an association between sodium disorders and increased optic disc diameter, suggesting that optic disc diameter measurement may be useful in predicting hyponatremia diagnosis and prognosis (10).

Conclusion

It should be remembered that patients with hyponatremia presenting to the emergency department with visual complaints should be evaluated for intracranial pressure. Hyponatremia should be considered in the differential diagnosis of intracranial hypertension etiology.

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Leriche Syndrome in the Emergency Department: A Four-Patient Case Series

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Introductıon:

Leriche syndrome, or aortoiliac occlusive disease, is a rare but serious manifestation of chronic atherosclerotic occlusion of the distal abdominal aorta and/or iliac arteries. It typically presents with a classic triad of claudication, absent femoral pulses, and impotence in male patients. However, acute presentations resembling vascular catastrophes, including paraplegia and severe limb ischemia, have also been reported. Early recognition and rapid vascular consultation are crucial, especially in emergency department (ED) settings,

where diagnostic delays may result in irreversible ischemic damage or death. This case series highlights four patients—three males and one female—diagnosed with Leriche syndrome, with varied acute and subacute presentations.

MATERIALS AND METHODS:

We retrospectively reviewed four patients diagnosed with Leriche syndrome between January 2019 and January 2024 in a tertiary ED. The cohort consisted of three men (ages 81, 61, 80) and one woman (age 72). Clinical presentation, physical examination, imaging, treatment strategy, and disposition were extracted from the medical records.

RESULTS

Case Series Presentation

Case 1: An 81-year-old male presented to the ED with sudden-onset thoracolumbar pain and a syncopal episode earlier that afternoon. He developed acute paraplegia shortly afterward. On examination, he was alert, oriented, and hemodynamically stable. Motor and sensory function were absent in both lower extremities. Femoral, popliteal, and pedal pulses were nonpalpable bilaterally, and both limbs were cold and pale. Emergency computed tomography angiography (CTA) showed no dissection flap in the thoracic aorta but revealed a thrombus causing total occlusion of the abdominal aorta distal to the renal arteries. Given the clinical picture of acute Leriche syndrome with spinal cord ischemia, urgent surgical intervention was recommended. After discussing the high perioperative risks with the patient and family, vascular surgery was consulted and the patient was admitted to the cardiovascular surgery intensive care unit (CVS-ICU). He underwent emergent revascularization with an aortobifemoral bypass.

Case 2: A 61-year-old male was referred from an outside hospital after sudden-onset severe abdominal pain in the morning. CTA performed at the referring center revealed total occlusion of the infrarenal abdominal aorta. His medical history included coronary artery bypass grafting (CABG) performed four years earlier, at which time he was advised of the need for future abdominal aortic revascularization. He had longstanding bilateral claudication with walking limitation at 30 meters. In the ED, he was alert and hypertensive (BP 165/100 mmHg). His abdomen was distended and diffusely tender. Bilateral femoral pulses were absent, and both lower limbs were cool with nonpalpable distal pulses. Despite no neurological deficit, the severity of ischemia prompted an urgent vascular consultation. He was admitted to the CVS-ICU for emergent operative management and underwent successful open aortic thrombectomy.

Case 3: An 80-year-old male was evaluated in the ED due to low back pain and bilateral foot pain. He had a known history of hypertension and a sedentary lifestyle. On arrival, he was hemodynamically stable, oriented, and cooperative. Femoral and distal lower extremity pulses were present bilaterally, though diminished. CTA performed during ED observation revealed a mega aorta with diffuse mural thrombus along the entire thoracoabdominal aortic trajectory. There was no evidence of dissection flap, rupture, or extravasation. The vascular surgery team concluded that this was a chronic degenerative process with no indication for emergency intervention. Medical optimization was recommended, including strict blood pressure control, antiplatelet therapy, and outpatient follow-up for aortic aneurysm surveillance. Given poor oral intake and generalized weakness, the internal medicine service was consulted, and the patient was admitted for supportive care and further evaluation.

Case 4: A 72-year-old female with poorly controlled diabetes and hypertension presented to the ED with progressively worsening bilateral lower extremity pain and rest pain over the past 48 hours. She reported difficulty ambulating and numbness in her toes. On physical examination, her legs were mottled and cool. Femoral pulses were weak, and distal pulses were absent bilaterally. Laboratory tests revealed elevated lactate and mild acute kidney injury. CTA of the aorta and lower extremities demonstrated chronic total occlusion of the distal abdominal aorta extending to the common iliac arteries, consistent with Leriche syndrome with acute-on-chronic ischemia. After consultation with vascular surgery, the patient was admitted for close hemodynamic monitoring and underwent staged revascularization using hybrid endovascular techniques. Limb viability was preserved.

Discussion

Leriche syndrome is a rare but critical vascular condition that may present acutely in the ED. While traditionally described as a chronic occlusive disease in middle-aged men, its clinical spectrum includes acute ischemia, spinal cord involvement, and multiorgan compromise, particularly in elderly patients. The first two cases in our series involved complete occlusion of the abdominal aorta, resulting in acute ischemia requiring emergency surgery. The third patient reflected a more indolent, nonoperative presentation of a mega aorta with diffuse thrombus, managed conservatively. The final case highlights acute-on-chronic limb ischemia in a woman—an underrepresented demographic for Leriche syndrome—requiring staged intervention.

Timely diagnosis depends on a high index of suspicion in patients presenting with bilateral lower extremity ischemia, absent femoral pulses, or acute paraplegia. CTA remains the gold standard for anatomical assessment. Emergency physicians play a crucial role in early stabilization, pain management, hemodynamic optimization, and rapid vascular consultation. Delay in diagnosis or intervention can lead to permanent neurological deficits, limb loss, or death.

Conclusion

This case series underscores the heterogeneity of Leriche syndrome presentations in the ED. Emergency physicians should be vigilant for this diagnosis in patients with sudden lower extremity symptoms, spinal cord findings, or unexplained vascular collapse. Early CTA imaging and multidisciplinary coordination are vital to optimize outcomes. While surgical revascularization remains the cornerstone of treatment in acute presentations, medical therapy and close surveillance are appropriate in selected chronic cases. Recognizing the full clinical spectrum of this rare syndrome can significantly improve patient prognosis and functional recovery.

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7339

Ischemic Infarction In The Young Patient Group

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Introduction:
Ischemic infarction is a cerebrovascular event that occurs due to a reduction in blood flow to the brain. Although it is more commonly seen in older age groups, it can rarely occur in young patients and may result in significant morbidity.In the general population, ischemic strokes are primarily associated with atherosclerotic processes, whereas in young patients, the etiology encompasses a broader spectrum and requires a detailed evaluation.The most common causes of ischemic infarction in young patients include cardioembolic events (e.g., patent foramen ovale, arrhythmia), arterial dissections, thrombophilic conditions, and rare genetic disorders. Infections, migraine-related vascular events, and substance use may also play a role in the etiology. Clinically, young patients often present with sudden-onset hemiparesis, dysarthria, altered consciousness, and visual disturbances. Diagnosis is established through brain imaging modalities (CT, MRI) and additional tests to determine the underlying cause. Acute-phase treatment includes thrombolytic agents and mechanical thrombectomy, while long-term management focuses on risk factor control and secondary prevention strategies.

Case Report:
A 23-year-old female patient presented to our emergency department with a complaint of persistent headache for three days. She described it as the most severe headache she had ever experienced vital signs:
•Blood pressure: 120/90 mmHg
•Heart rate: 89 bpm
•SpO□: 97%
•Temperature: 36.5°
On physical examination, bilateral horizontal nystagmus was noted. The rest of the neurological examination was unremarkable. Diffusion-weighted imaging (DWI) and susceptibility-weighted imaging (SWI) were performed.
Radiology report:A subcentimetric diffusion restriction was observed in the periventricular area of the right frontal region.The patient was admitted to the neurology service for further evaluation and treatment. Appropriate therapy was initiated.

Conclusion: Although ischemic infarction is rare in young patients, its etiology is diverse and requires a comprehensive evaluation. While early diagnosis and appropriate treatment generally lead to a better prognosis, long-term rehabilitation and risk factor management are crucial. Therefore, young patients who experience a stroke should be assessed with a multidisciplinary approach.

KEYWORDS: emergency department, ischemic infarction, stroke

7397

Cement Material in the Vena Cava, Right Atrium, and Pulmonary Artery: A Rare Case Presentation From an Emergency Medicine Perspective

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Introductıon
Vertebral compression fractures (VCF) are common in osteoporotic patients. (1) Percutaneous kyphoplasty (PKP) is a minimally invasive technique for treating painful VCF, involving the percutaneous injection of Poly Methyl Meth Acrylate (PMMA) bone cement into the vertebral body (VB). (2,3) This case report presents a patient in whom PMMA cement migrated via the perivertebral venous plexus to the inferior vena cava (IVC) and right atrium (RA), causing pulmonary cement embolism (PCE), as detected by ultrasonography and computed tomography (CT).

Case
A 72-year-old female with a history of PKP for an osteoporotic vertebral fracture at L2 four months ago presented with intermittent palpitations, blood pressure irregularities and fatigue. She denied chest pain, shortness of breath, hemoptysis or fever. Her medical history included hypertension, percutaneous coronary angiography and atrial fibrillation (AF). The patient was on multiple medications including clopidogrel and acetylsalicylic acid.
On physical examination, the patient's heart rate was 110 beats per minute, respiratory rate was 16 breaths per minute, oxygen

saturation was 99% on room air and blood pressure was 143/89 mmHg. Respiratory examination revealed diminished breath sounds at both lung bases. Bilateral grade 1 pretibial edema was noted. Other system examinations were normal.

The electrocardiogram showed a heart rate of 117 beats per minute and was interpreted as rapid ventricular response AF.

Point-of-care ultrasound (POCUS) revealed an ejection fraction of 60%, normal right-sided structures, no evidence of a D-shaped left ventricle, mitral regurgitation grade 2, tricuspid regurgitation grade 2-3 and a pulmonary artery pressure of 50 mmHg. A hyperechoic, long, thin structure extending from the IVC to the RA was observed. (Figure 1)

Laboratory results showed normal troponin-I levels, normal blood gas analysis, normal prothrombin time, normal creatinine, hemoglobin of 9,2 g/dl and D-dimer of 8600 ng/ml.

A posteroanterior chest X-ray revealed radio-opacity in the right hilar region (Figure 2).

The patient underwent pulmonary angiography CT and thoracoabdominal aorta CT. Imaging revealed metallic cement in the L2 VB, extending into the paravertebral region. A tubular dense structure was seen extending from the left paravertebral venous tract to the IVC and RA, terminating within the RA. Similar dense structures were observed in the right main pulmonary artery, interlobar artery, and most segmental branches, suggesting PCE. (Figures 3,4)

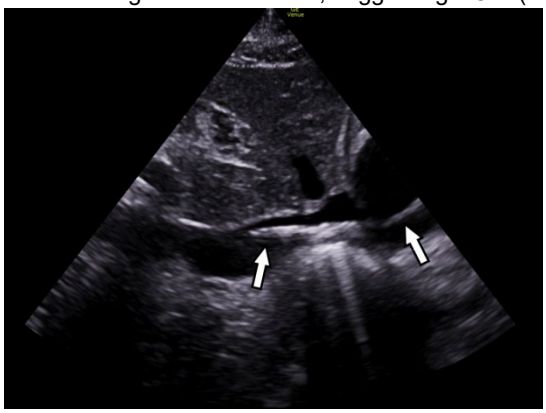

Figure 1: A hyperechoic, thin, elongated structure (arrows) extending from the inferior vena cava to the right atrium, as observed on transthoracic echocardiography.

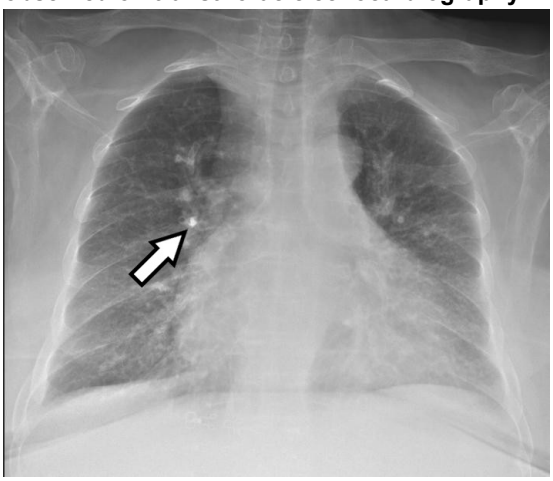

Figure 2: A radiopaque area (arrow) observed in the right hilar region on the posteroanterior chest X-ray.

A pulmonology consultation was obtained and anticoagulation therapy along with a cardiology consultation was recommended. A cardiology consultation was also obtained and the patient was hospitalized for treatment planning.

Dıscussıon

PKP is a minimally invasive method used to treat VCF in osteoporotic patients. (2) PKP uses PMMA, a bone cement used in orthopedic procedures since the 1960s. (2) Complications after PKP can include hypotension, PCE, acute respiratory distress syndrome, spinal cord compression and infection. Cement migration to the IVC and PCE is a potential risk, occurring in 3.5% to 23% in various studies. (3,4)

What is interesting in our case is the migration of the cement to the IVC and RA via the paravertebral venous plexus, and the PCE occurring with mild symptoms. This may be due to the patient's use of anticoagulants for chronic conditions. Based on the patient's medical history and examination findings in the emergency department, POCUS and CT imaging may shed light on rare complications that could have a fatal outcome.

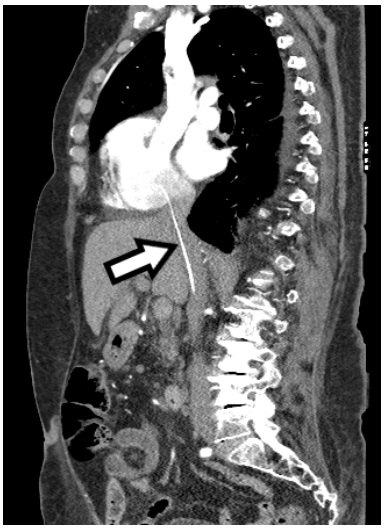

Figure 3: A tubular dense structure (arrow) extending from the left paravertebral venous tract to the inferior vena cava and right atrium on CTPA imaging.

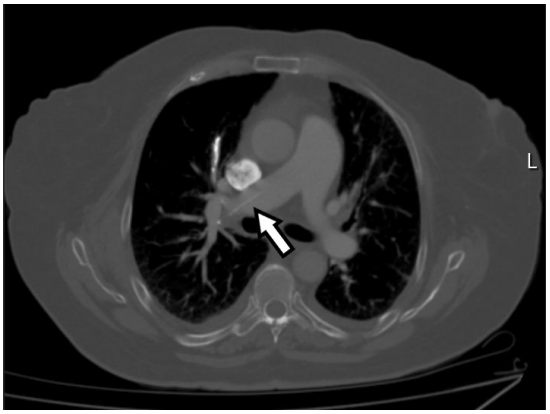

Figure 4: A tubular dense structure (arrow) in the right main pulmonary artery on CTPA imaging.

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7437

A Rare and Easily Overlooked Diagnosis in a Pediatric Trauma Case: Traumatic Asphyxia

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Introductıon:

Traumatic asphyxia is a clinical syndrome characterized by cervicofacial cyanosis, edema, petechiae, subconjunctival hemorrhage, and neurological symptoms following sudden, severe, compressive blunt thoracoabdominal trauma (1). This syndrome is also referred to as acute thoracic compression syndrome, ecchymotic mask, or Perthes syndrome (2). It was first described in 1837 by Oliver d'Angers based on autopsy findings, and its exact incidence remains unknown (3).

The clinical findings associated with traumatic asphyxia can involve the skin, eyes, brain, gastrointestinal system, airway, and chest. The hallmark of the syndrome, cervicofacial cyanosis, is typically accompanied by petechiae, which are generally observed in the head, neck, and upper chest. Morbidity and mortality are related to concurrent cardiovascular, neurological, and pulmonary injuries and their severity (4).

Blunt trauma is particularly common in forensic cases and is a primary cause of crushing injuries to the upper chest. Following trau-

ma, diffuse petechiae may appear in the face and neck regions, giving the patient's face a livid appearance. This condition is terminologically known as an "ecchymotic mask." Although patients with an ecchymotic mask may present with a visually alarming appearance, their clinical condition is often not as severe as it seems (5).

This article presents a case of traumatic asphyxia developing as a result of severe thoracoabdominal trauma.

Case Report:

A previously healthy 9-year-old male patient was brought to the emergency department (ED) after falling from the trailer of a tractor. The patient landed face-down, striking his chest against the ground. Upon arrival at the ED, his vital signs were within normal limits. He was conscious, cooperative, and oriented, with a Glasgow Coma Scale (GCS) score of 15. Numerous petechiae were observed on the scalp, face, neck, and upper chest wall. Bilateral eyelid edema, bulbar conjunctival hemorrhage, and edema in both eyes were noted. No petechiae, purpura, or ecchymosis were found on other parts of the body (Image).

The airway was patent, and there were no signs of respiratory distress. Laboratory test results were within normal limits. Physical examination of other systems was unremarkable. Radiographs of the extremities, along with computed tomography (CT) scans of the cranium, chest, and abdomen performed due to the high-energy trauma, were also reported as normal.

During observation in the ED, the patient's vital signs and clinical condition remained stable. Ophthalmology consultation ruled out any acute pathology. The patient was monitored in the ED for 24 hours and discharged with recommendations after no worsening of symptoms was observed.

Dıscussıon:

The most common cause of traumatic asphyxia in children is motor vehicle accidents (e.g., traffic collisions). Traumatic asphyxia can also occur in situations involving being crushed under heavy machinery or furniture and/or being trapped between two objects (6). Non-traumatic conditions such as epileptic seizures, severe vomiting, whooping cough, and asthma exacerbations may result in similar clinical presentations (7). In these cases, the underlying mechanism is not trauma-related but rather asphyxia induced by the disease, leading to comparable clinical outcomes.

In adults, the frequency of traumatic asphyxia is approximately 1 case per 18,500 accidents. It is exceedingly rare in children, and its actual incidence remains unknown. The true frequency in pediatric cases is uncertain, likely due to underrecognition or underreporting by pediatricians (5).

The treatment process for traumatic asphyxia depends on the severity and duration of compression. While heavy loads may be tolerated for short durations, prolonged exposure to moderate loads can result in fatal outcomes (7). The characteristic features of traumatic asphyxia include petechiae, cranio-cervical cyanosis, subconjunctival hemorrhage, and facial edema involving the upper body, neck, and face. Petechiae usually appear 2-3 hours after the incident and are more prominent in the conjunctiva and oral mucosa. This results in a livid appearance of the patient's face (8). This clinical condition, known as an "ecchymotic mask," was consistent with the diagnosis in our case.

The underlying mechanism of these clinical findings is the increased blood flow from the right atrium to valveless brachiocephalic and jugular veins due to positive pressure generated in the mediastinum following blunt thoracic and upper abdominal trauma. The sudden rise in venous pressure results in petechiae. Petechiae are absent in the lower body because venous valves in the lower extremities control the increased venous pressure. Additionally, if the inferior vena cava is compressed or obstructed, elevated airway pressure may further protect the lower body (5).

Depending on the severity of the trauma, rib fractures, pulmonary contusions, pneumothorax, hemothorax, solid organ lacerations, and neurological sequelae may occur. A study conducted in adults recommended cranial, abdominal, and pelvic CT scans, along with Doppler ultrasonography of the neck and upper extremity vessels and echocardiography, to identify these injuries. However, there are no specific recommendations for children (8). Considering the anatomical and physiological differences between children and adults, performing all these tests in pediatric patients may be unnecessary. Diagnostic workups should be planned based on the severity of the trauma, the patient's GCS, and pediatric trauma score (5).

The prognosis of traumatic asphyxia is generally favorable with effective and timely treatment. Prolonged thoracic compression can lead to cerebral anoxia and neurological sequelae. Therefore, in patients with blunt thoracoabdominal trauma, a detailed trauma history should be obtained, and appropriate diagnostic and therapeutic plans should be implemented (5).

Treatment is generally conservative and recovery is closely related to associated injuries. In uncomplicated cases, the head should be elevated to 30 degrees, and oxygen therapy should be administered to reduce intracranial pressure. In cases with complicated organ injuries, advanced life support measures should be applied according to the patient's clinical condition, and cervical spine immobilization should be ensured (9). In our case, the patient was monitored in the emergency department for 24 hours and discharged with outpatient follow-up recommendations after no worsening of symptoms was observed.

Conclusıon:

In conclusion, the diagnosis of traumatic asphyxia should be considered in pediatric and adult patients with blunt thoracoabdominal trauma. Despite its catastrophic appearance, it is typically a benign condition.

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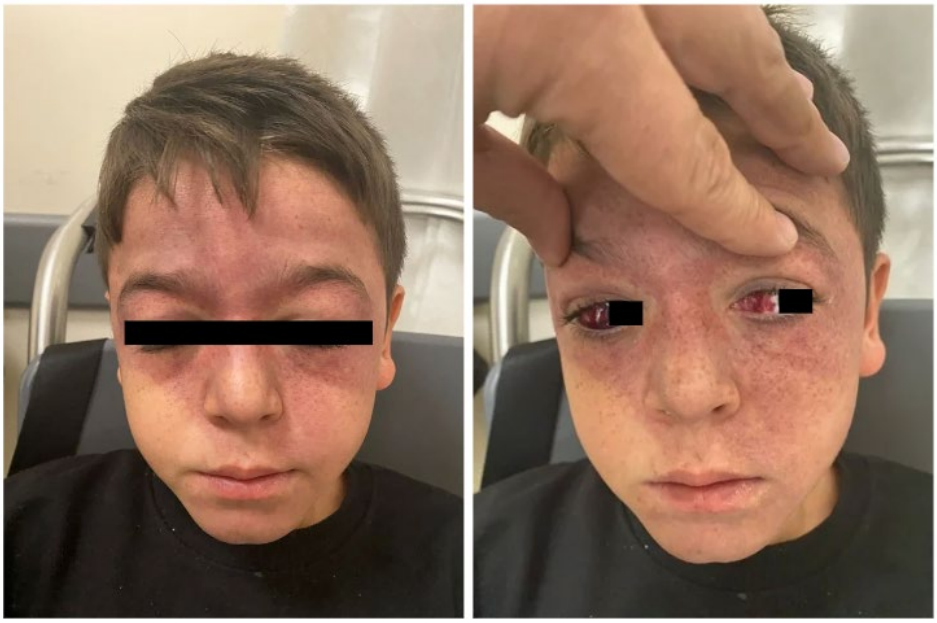
Image. Petechiaes on the face and bilateral eyelid edema, bulbar conjunctival hemorrhage.



7453

Old Enemy At New Address: From Lıver Cyst Hydatıd To Pulmonary Localızatıon

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Introductıon

Hydatid disease is a zoonotic parasitic disease caused by Echinococcus granulosus. It is more common in people who have close contact with animals in endemic areas and is most frequently located in the liver (65%) and lung (25%). The liver is the most commonly affected organ because it is the first barrier through the portal vein; however, in some cases, the parasite can progress to the lungs and cause pulmonary hydatid cyst formation. The cyst usually grows slowly and patients may remain asymptomatic for a long time (1). The most common symptoms in pulmonary hydatid cysts are cough, dyspnea, chest pain and hemoptysis. The enlarged cyst may open into the bronchi and develop a complicated pulmonary hydatid cyst characterized by air-fluid levels. Ruptured or infected cysts may cause severe inflammation and suppurative complications (2). Imaging methods and serological tests are of great importance in the diagnosis of hydatid cyst. Medical (albendazole) and surgical (cystectomy, capitonnage) methods are preferred in treatment according to the patient's clinic (3). Especially in patients with a history of hepatic hydatid cyst, if there is new chest pain or respiratory complaints, pulmonary hydatid cyst should be included in the differential diagnosis.

Case Report

A 33-year-old female patient applied to the emergency department with a complaint of stabbing pain under her left breast. The patient stated that the pain had been worsening for a few days and was occasionally accompanied by dyspnea. She had a known history of asthma and had undergone surgery for a hepatic hydatid cyst approximately three years ago. It was learned that the patient did not complain of cough or sputum production, but occasionally felt mild chest tightness. On physical examination, the patient's general condition was good and he was conscious. Body temperature was 37.8°C, pulse 95/min, blood pressure 120/80 mmHg and respiratory rate 22/min. Auscultation revealed a significant decrease in breath sounds in the lower zone of the left lung. Laboratory tests revealed high infection markers. C-reactive protein level was 201 mg/L and white blood cell count was 17,000/mm³. First, the

patient underwent a chest X-ray and a large cystic lesion was detected in the lower lobe of the left lung. Thoracic computed tomography was performed to clarify the diagnosis and a lesion measuring approximately 4x6 cm in size and with a wall thickness of 4 mm and an air-fluid level were detected in the superior lower lobe of the left lung. The findings suggested a complicated pulmonary hydatid cyst opening into the bronchi. The patient was consulted by thoracic surgery and surgery was planned by thoracic surgery.

Figure-1: Axial section of hydatid cyst in the left lower lobe of the AC

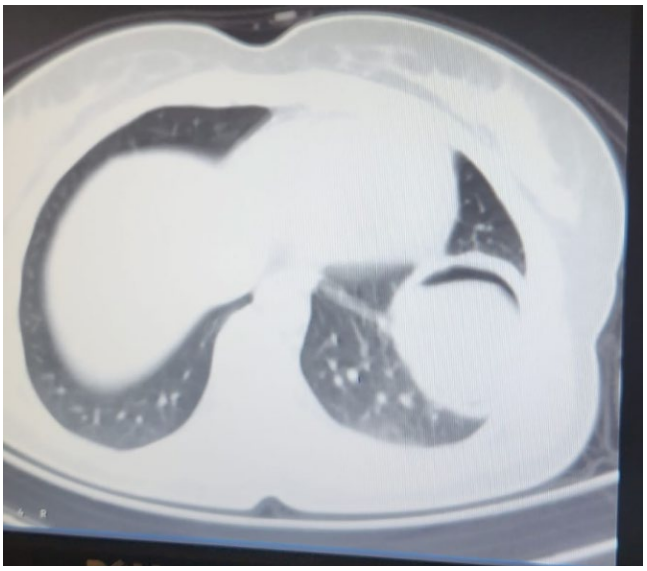
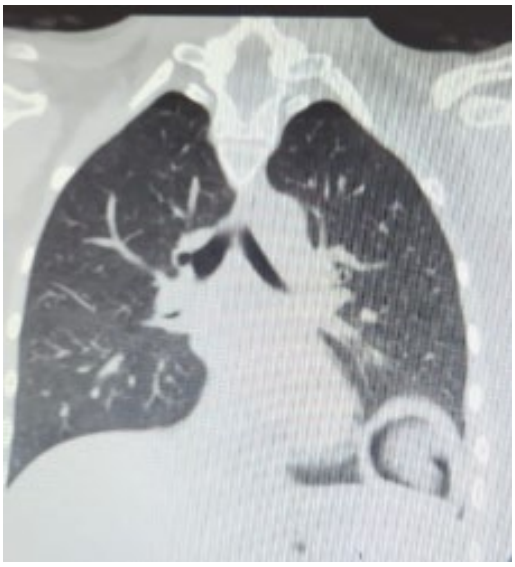


Figure-2: Coronal section of hydatid cyst in the left lower lobe of the AC



Discussion

Hydatid cyst is one of the most common parasitic infections, especially in the liver and lungs. In patients with a history of hepatic hydatid cyst disease, pulmonary hydatid cyst disease should definitely be considered in the differential diagnosis in newly developing respiratory system symptoms (4). This case has similar characteristics to complicated pulmonary hydatid cyst cases described in the literature. Recurrent pulmonary hydatid cyst development is not uncommon in patients who have previously undergone liver hydatid cyst surgery. In our case, the patient's history of liver hydatid cyst suggests that the pulmonary cyst developed as a second focus (5). Most pulmonary hydatid cysts are asymptomatic and are usually detected incidentally during radiological examinations. However, as in our patient, when the cyst opens into the bronchi, it can become complicated with air-fluid level and infection can be added. In this case, patients may present with chest pain, dyspnea, cough and fever (6). Unlike previously reported cases, our case had bronchial dilatation and infection. Similar cases have been described in the literature, and localization in the lower lobes of the lung and bronchial fistulization have been reported to be among the most common complications (7). Treatment options include medical and surgical methods. Surgery was preferred in our case because the cyst opened into the bronchi, became infected, and caused symptomatic deterioration in the respiratory system. Medical treatment (albendazole) is usually recommended in addition to surgery and is helpful in preventing recurrence (8).

Conclusion

In conclusion, pulmonary hydatid cysts should be considered when chest pain and respiratory symptoms develop in patients with a history of liver hydatid cysts. Prognosis is very good with early diagnosis and appropriate treatment.

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Progressive Danger with Increasing Life Span: A Case of Geriatric Suicidal Attempt

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Introduction

The World Health Organization (WHO) predicts that by 2030, one in six people will be 60 or older. By 2050, there will be 1.4 billion people over 60, up from 1 billion in 2020. In 2050, about 2.1 billion people are expected to be 60 or older. According to the 2011 Census, India has 104 million geriatric population (aged 60+) or 8.6% of the population. According to WHO, the annual global suicide rate of 10.5 per 100,000 people accounts for approximately 800,000 suicide deaths each year. Suicide among older people generally receives less attention compared to suicide among younger age groups. In 2017, the suicide rate was 16.17 per 100,000 people aged 50 to 69 years, compared to 27.45 per 100,000 people aged over 70 years. In 2019, the suicide rate for people aged 50 to 69 was 14.25 per 100,000, compared to 24.53 per 100,000 for people aged 70 and over. In recent decades, improvements in health care, longer life expectancies and changes in social patterns have led to a significant increase in the elderly population. With this demographic shift, challenges have emerged, such as rising suicide rates among older adults.

Health is defined by the World Health Organization (WHO) as follows: Health is a state of complete physical, mental and social well-being, not merely the absence of disease or infirmity. This definition indicates that health is a multidimensional construct and includes various factors that affect the overall quality of life of the individual. This case was written to draw attention to mental and social well-being in the World Health Organization's definition of 'health'.

Physical health is a contributing factor in most suicidal behaviors in late life [1] . Vascular disease can predispose to late-onset depression, which often has antecedents in middle age, such as hypertension, smoking, diabetes and metabolic syndrome. A case-control of suicides over 50 years of age found that cerebrovascular risk factor scores were significantly higher in suicide cases [2] .

Older people are more likely to have access to firearms in rural areas. It has been discovered that a higher percentage of suicide attempters in elderly patients come from rural areas than urban areas [3].

Case

A 68-year-old man with known hypertension, atrial fibrillation, heart failure, plegic legs after a traffic accident 33 years ago, who was hospitalized and operated for subdural hematoma and right renal hematoma after a traffic accident 1 year ago, Our patient, who was discharged 3 days ago from the chest diseases service where he had been hospitalized after intensive care unit due to pneumonia and respiratory failure, was on rivoroxaban, atorvastatin, metoprolol, amlodipine, fursosemide and proton pump inhibitors. There was no suicide attempt in his history. She had no diagnosed mental illness. It was learned that the patient did not receive any psychiatric support and resided in a town approximately 100 km away from the city center.

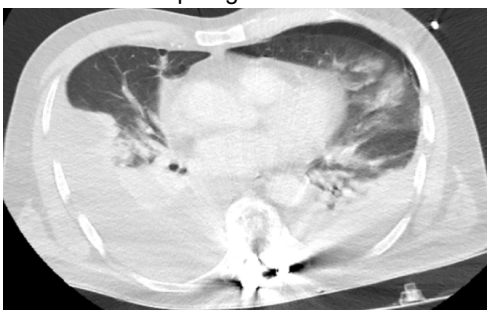
The patient was brought to our clinic after a suicidal gunshot wound. On physical examination, he was and agitated. Two bullet entry holes were observed on the left anterior chest wall, one at the xiphoid level and the other at the level of the posterior costal arch. Two bullet exit holes were observed in the left posterior posterior costal arch and lumbar midclavicular line.

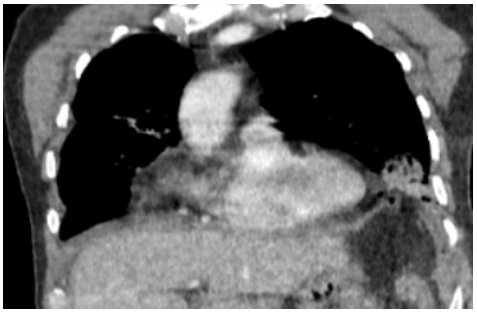
Vital values blood pressure: 99/65 mmHg pulse: 98 beats/min saturation was 95% with supplemental oxygen, temperature: 36.3 degrees Celsius. The patient was disoriented.

Laboratory findings: creatinine: 0.79 mg/dl urea: 69 hemoglobin: 8.1 g/dl, hct: 26.3%, plt: 378 u/L, apt: 19.7 s, pt: 14.1, inr: 1.20, ph: 7.28, base deficit: -6.3, lactate: 1.7 mmol/L ethanol: less than 0.1 promil

The patient was disoriented with a GCS of 14. He was electively intubated. Tetanus and antibiotic prophylaxis and hydration were performed. Erythrocyte suspension preparation was done. Bedside Focused Assessment with Sonography for Trauma (FAST)" evaluation was performed. Free fluid in the hepatorenal and perisplenic area was evaluated in favor of bleeding. The patient was consulted to thoracic surgery, general surgery, cardiovascular surgery and urology units. A left pneumothorax was detected and a chest tube was inserted. After the patient was evaluated in the emergency department, interned to the relevant surgical branches, his treatment in the emergency department was completed and tomography imaging was performed, the patient was immediately taken to the operating room for surgery. The pathologies on the imaging scans are summarized in the figures below.

Figure 1. 10cm pleural fluid in the right hemithorax, 6cm pleural fluid in the left hemithorax and atelectatic changes in the adjacent parenchyma, approximately 25mm pneumothorax appearance in the left hemithorax, air density and heterogeneity in the subcutaneous fat planes in the left anterior, air density under the skin in the muscle planes of the anterior abdominal wall at the left upper quadrant level, heterogeneity in the fat planes partially adjacent to the anterior abdominal wall and linear daniste increases, irregularities in the diaphragmatic face on the left.



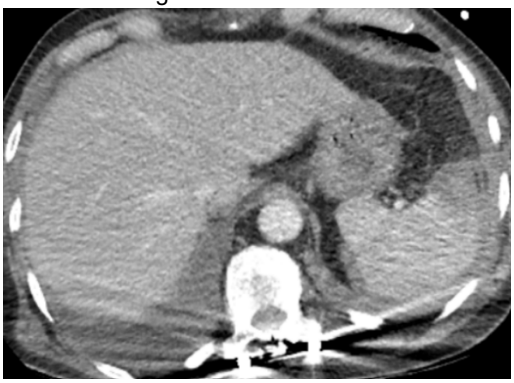


Discussion

Each year on 10 September, attention is drawn to the issues, stigma is reduced and awareness is raised among organizations, government and the general population, while sending a clear message that suicide prevention is possible. The theme of World

Suicide Prevention Day for the three years from 2021 to 2023 is "creating hope through action". The core message of this theme is to instill optimism and positivity by emphasizing other options besides taking one's own life.

Figure 2. Heterogeneous hypodense area of 6x4cm in the subcapsular area in the anterolateral mid-section of the splenic parenchyma (subcapsular hematoma and parenchymal contusion?). Air densities and free fluid are seen in the abdomen.
Splenectomy was performed, diaphragmatic damage was repaired and the patient was followed up in the intensive care unit, and then he was interned to the ward and discharged in the following days. Necessary referrals for psychiatric support were made before discharge.



Conclusion

In conclusion, due to the aforementioned distinguishing variables, the risk of suicide is high in the geriatric population aged 60 years and older. An important cause for public concern is the need for focused preventive measures and treatments. It is essential that health care providers and family members identify warning signs in older adults and provide support and resources to prevent suicide attempts. Significant mobilization of health professionals, policy makers and the community is critical to prevent suicide in older adults. Reducing negative perceptions associated with mental health problems, improving the availability of mental health care, effectively managing resources and providing support are necessary steps to help older adults maintain their autonomy and social relationships. By implementing a comprehensive and interdisciplinary plan, we can help reduce the suicide rate in the geriatric population and promote healthy aging.

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A Critical Cause of Respiratory Distress in a Dialysis Patient: Emergency Department Management of a Lung Abscess

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Introduction

Lung abscess is a necrotizing infection of the lung parenchyma that leads to a pus-filled cavity, often visible on imaging as an air-fluid level (1,2). It typically arises as a complication of aspiration pneumonia or severe pneumonia that undergoes liquefactive necrosis, though a variety of pathogens (bacteria, fungi, even mycobacteria) can be responsible (1,2). Before the antibiotic era, lung abscess carried a mortality as high as 75%, but even today it remains a serious illness with mortality rates of ~10–20% in many series (2). Prompt antibiotic therapy has dramatically improved outcomes – specialized centers report mortality as low as ~4% when aggressive treatment is instituted early (3). Patients with compromised immune systems are at particular risk: those with end-stage renal disease (ESRD) on dialysis have well-documented immune dysfunction in both innate and adaptive immunity (4, 5). Uremia and the dialysis process lead to impaired neutrophil and lymphocyte function, chronic inflammation, and malnutrition, all of which increase susceptibility to severe infections (4, 5). Consequently, infections in dialysis patients tend to progress rapidly and are prone to complications. Delays in diagnosing and treating infections such as lung abscess in this population can therefore be especially devastating, leading to higher morbidity and mortality (3). Early recognition and management are critical in improving outcomes.

In this presentation, we report the case of a young immunosuppressed patient on hemodialysis who developed a large lung abs-

cess. We highlight her clinical presentation, imaging findings, pathophysiology and risk factors, microbiological considerations, emergency department (ED) management, definitive treatment, and clinical course. The case underlines the pivotal role of ED physicians in promptly identifying such life-threatening infections and coordinating multidisciplinary care to achieve a successful outcome.

Case Presentation

Patient: A 20-year-old woman with a history of chronic renal failure on hemodialysis for the past two years presented to the emergency department with a 5-day history of high fever, severe cough, and progressive shortness of breath. She noted generalized weakness and could barely perform her usual activities due to breathlessness. Her cough was productive of thick, yellowish sputum, but she denied hemoptysis. She did not report any recent loss of consciousness, vomiting, or choking episodes. There was no history of diabetes, HIV infection, or chronic lung disease. Her only known medical issue was end-stage kidney disease requiring thrice-weekly dialysis via an arteriovenous fistula; she had no indwelling dialysis catheter.

Initial Vital Signs and Exam: On arrival, the patient appeared ill and in moderate respiratory distress. Her temperature was 38.5°C, blood pressure 95/60 mmHg, heart rate 118 beats/min (sinus tachycardia), respiratory rate 24 breaths/min, and oxygen saturation 91% on room air. She was alert and oriented. Lung examination revealed decreased breath sounds and prominent crackles (rales) over the right upper chest. There were no obvious wheezes. Percussion of the right upper lung field was slightly dull. No egophony or significant pleural friction rub was noted. The left lung and other system examinations (cardiac, abdominal, neurological) were unremarkable. The patient did not have peripheral edema or jugular venous distension. Her arteriovenous fistula site was clean with no signs of infection.

Laboratory Findings: Initial laboratory tests showed a marked leukocytosis (WBC 15,170/μL) with neutrophil predominance, indicating an inflammatory response. C-reactive protein (CRP) was elevated at 173.7 mg/L, consistent with severe infection. Procalcitonin was not immediately available, but erythrocyte sedimentation rate (ESR) was elevated (value not specified). Electrolytes revealed no significant abnormalities aside from those expected in a dialysis patient (her blood urea nitrogen and creatinine were elevated due to ESRD). Arterial blood gas on room air showed a mild hypoxemia (PaO□ in the mid-60s mmHg) with no significant hypercapnia, and a slightly elevated lactate consistent with early sepsis. Two sets of blood cultures and a sputum sample were obtained in the ED prior to initiating antibiotics.

Imaging: Given the patient's fever, cough, and focal exam findings, a chest radiograph was obtained immediately. The chest X-ray suggested a right upper lobe opacity with possible cavitation, prompting further imaging. A contrast-enhanced chest computed tomography (CT) scan was performed, which revealed a large cavitary lesion (~8 × 6 cm) in the right upper lobe with a visible air-fluid level, surrounded by areas of consolidation (Figure 1). No frank pleural effusion or empyema was evident on CT. These imaging findings were diagnostic of a lung abscess in the right upper lobe. The left lung was clear.

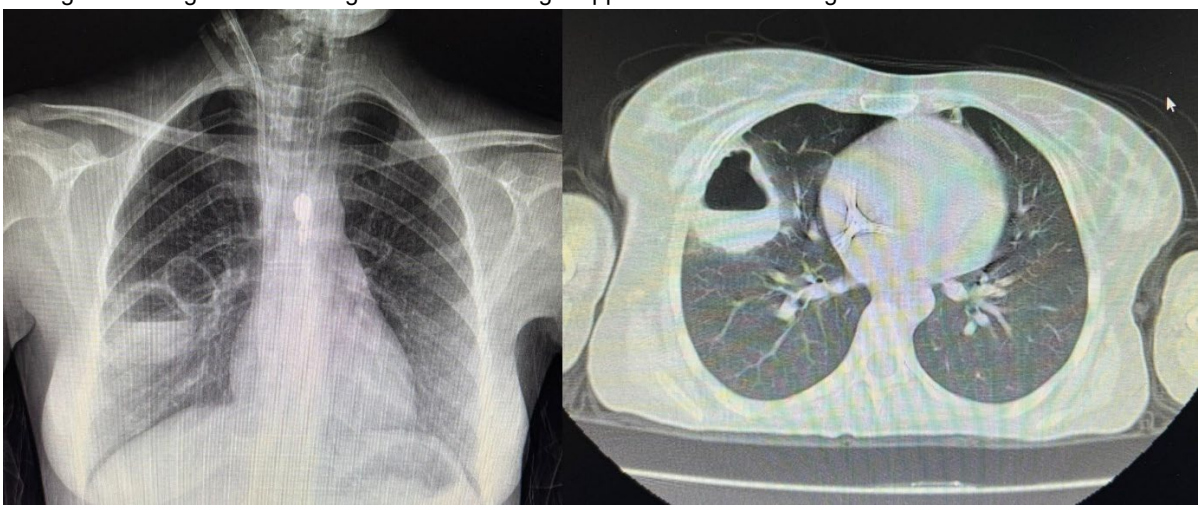

Figure 1: Chest radiograph and axial CT images from the patient demonstrate a large cavity in the right lung containing fluid and air, consistent with a lung abscess.

The thick wall of the cavity and internal air-fluid interface (seen on CT) are hallmark features. Importantly, the CT helped distinguish this intrapulmonary abscess from a loculated pleural empyema or other masses by showing the cavity within lung parenchyma and the absence of significant pleural collection (1). The imaging also confirmed there were no additional abscesses or significant bronchial obstruction.

Pathophysiology and Risk Factors

A lung abscess results from focal infection of lung tissue that undergoes necrosis, creating a walled-off cavity filled with pus. The **primary mechanism** in most cases is aspiration of oropharyngeal contents into the lung (1). Aspirated bacteria (often anaerobes from the oral cavity) infect dependent portions of the lung and can lead to a necrotizing pneumonia and abscess formation. Over time, communication with airways can lead to a bronchopulmonary fistula and the classic air-fluid level on imaging as the pus partially drains into the bronchial tree (1). In our patient, the abscess was in the right upper lobe, which is slightly atypical for aspiration (aspiration abscesses classically involve dependent segments of the right upper lobe when supine, or right lower lobe superior seg-

ment when upright). This raised the consideration of other contributing factors to abscess formation in her case, such as an unusually aggressive organism or impaired host defenses.

Predisposing risk factors for lung abscess include any condition that increases aspiration risk or reduces pulmonary clearance of infections:

Altered consciousness and aspiration: Alcohol intoxication, illicit drug use, general anesthesia, seizures, or stroke can lead to aspiration of oral secretions or gastric contents into the lungs, seeding an infection (1,3). Poor cough reflex or dysphagia (for example, in neurodegenerative diseases) also falls into this category. In one large series, 78.6% of lung abscess patients had a history of loss of consciousness (often due to alcohol abuse) preceding the infection.

Poor oral hygiene or dental disease: Periodontal infection or dental abscesses provide a rich source of anaerobic bacteria that can be aspirated. In the same series, over 80% of patients had significant dental disease (3).

Immunosuppression: Patients with compromised immunity are less able to contain a nascent infection, allowing it to progress to abscess. This includes people with HIV/AIDS, those on chronic corticosteroids or chemotherapy, transplant recipients, uncontrolled diabetics, and patients with ESRD on dialysis (4, 5). ESRD patients have defects in neutrophil phagocytosis, T-cell dysfunction, and hypogammaglobulinemia, along with a chronic inflammatory state (5). These factors make them vulnerable to unusual and severe infections. Notably, **renal failure requiring dialysis has been identified as an independent predictor of higher mortality in patients with severe lung abscess** – in one ICU study, patients on renal replacement therapy had over three times the odds of death compared to others (6). Our patient's only major risk factor was her immunosuppressed status from ESRD; she did not have a history of alcohol use or recent anesthesia, suggesting that her impaired immunity allowed a routine infection to advance unchecked.

Chronic lung diseases or obstruction: Conditions like chronic obstructive pulmonary disease (COPD) or bronchiectasis can impair normal clearance of secretions. Bronchial obstruction by a tumor or foreign body can lead to post-obstructive pneumonia that may cavitate.

Bacteremia with septic emboli: Another pathophysiological route is hematogenous seeding of the lungs. Staphylococcus aureus bacteremia (from IV drug use or infected catheters, for example) can produce multiple lung abscesses via septic emboli to the pulmonary circulation. Infective endocarditis of the tricuspid valve similarly can shower the lungs with bacteria, causing abscesses. These mechanisms are considered **secondary lung abscesses** (as opposed to primary from aspiration) (1). Our patient had an arteriovenous fistula for dialysis and no central catheter, making staphylococcal sepsis less likely, but this remains an important consideration in dialysis patients generally, as catheter-related bloodstream infections are common in that population.

In summary, the pathogenesis in this case appears to be a **primary lung abscess** developing from an acute pulmonary infection that progressed due to the patient's immunosuppressed state. The patient's chronic kidney disease and dialysis-dependent status created a perfect storm: impaired immune defenses (4, 5) allowed a lung infection to necrose and form an abscess, whereas a healthier individual might have cleared a similar pneumonia before cavitation occurred.

Microbiology

Lung abscesses are often polymicrobial infections, especially those resulting from aspiration (1,7). Anaerobic bacteria from the oral flora are classically the predominant pathogens in community-acquired lung abscess. Common anaerobes include **Bacteroides, Prevotella, Fusobacterium, and anaerobic streptococci** (7). These organisms are frequently implicated when a patient has risk factors like poor oral hygiene and aspiration. A hallmark of anaerobic lung abscess is **foul-smelling sputum**; indeed, about two-thirds of patients will produce putrid, offensive sputum when anaerobes are involved (3,7). In our patient's case, the history of productive cough raises the question of whether her sputum was foul-smelling – unfortunately this detail was not documented, but its presence would strongly point toward an anaerobic infection (7).

Aerobic bacteria can also cause lung abscesses. **Streptococcus pneumoniae** (particularly certain serotypes) and **Staphylococcus aureus** (especially post-influenza or in IV drug users) are known to cause necrotizing pneumonias that can evolve into abscesses. S. aureus is a common culprit in lung abscess secondary to septic emboli. **Klebsiella pneumoniae** is a notable cause of aggressive necrotizing lung infections, classically in diabetics or alcoholic patients, leading to abscess formation (often with thick, currant-jelly sputum). In the modern ICU setting, Gram-negative organisms have emerged as significant pathogens: one recent multicenter study of ICU patients found Enterobacteriaceae (such as Klebsiella and E. coli) in 31% of lung abscess cases, S. aureus in 22%, and Pseudomonas aeruginosa in 19% (6). **Pseudomonas** and other nosocomial Gram-negatives are especially relevant in immunocompromised hosts and hospital-acquired cases. Our patient likely acquired her infection in the community, but her immunosuppression puts these less typical organisms on the differential. In fact, immunocompromised patients (e.g. with HIV or on immunosuppressants) can develop lung abscesses from opportunistic organisms that rarely cause abscess in healthy people. For instance, **Nocardia** (an aerobic actinomycete) is notorious for causing cavitary lung abscesses in patients on chronic steroids or with advanced HIV; **Aspergillus** or other fungi can occasionally form fungus balls or abscess-like lesions in immunosuppressed lungs. **Mycobacterium tuberculosis** can produce cavitary lesions in the upper lobes that mimic lung abscess, although technically these are termed tuberculous cavities rather than pyogenic abscesses. In our patient, sputum was tested for acid-fast bacilli and Pneumocystis (given her immunocompromise), and these were negative. Ultimately, no organism was immediately isolated from sputum cultures – this is not uncommon, as anaerobes are difficult to grow from expectorated sputum due to contamination with oral flora and oxygen sensitivity (2). Blood cultures in the ED remained negative, likely because we initiated antibiotics early. Thus, the causative organism in this case was presumed based on clinical context. Given the combination of an upper lobe abscess and a patient with suppressed

immunity but no classic aspiration risk factors, a reasonable inference is that a mix of anaerobes and facultative bacteria from the upper airway were involved, potentially including viridans streptococci and anaerobes – essentially an **aspiration-related polymicrobial abscess** facilitated by her immune impairment.

Emergency Department Management

In the emergency department, rapid recognition and supportive management were crucial. The patient was placed on supplemental oxygen by nasal cannula to address her hypoxemia, improving her O□ saturation into the high 90s. IV access was secured, and empiric broad-spectrum antibiotics were started immediately after cultures were drawn. An urgent consultation with the on-call pulmonologist (chest specialist) was obtained as soon as the chest CT confirmed a lung abscess. The **empiric antibiotic regimen** chosen was **IV ceftriaxone 2 g daily plus IV moxifloxacin 400 mg daily**, a combination that provides broad coverage of both aerobic and anaerobic organisms. Ceftriaxone (a third-generation cephalosporin) was aimed at common Gram-positive and Gram-negative aerobes (e.g. Streptococci, Enterobacteriaceae), while moxifloxacin (a fluoroquinolone) has activity against anaerobes (as well as atypical organisms) and achieves good lung penetration. This regimen was selected in accordance with the pulmonology recommendation, tailored to cover the likely polymicrobial nature of the abscess in a community-onset but immunocompromised setting. Broad-spectrum antibiotic therapy is the cornerstone of lung abscess management, as recommended by current literature (1). ED physicians must initiate such therapy without delay because any postponement in treatment can allow the infection to progress or complications (like empyema, sepsis, or hemorrhage) to develop (3). In classic teaching, either **clindamycin** or a **beta-lactam/beta-lactamase inhibitor (e.g. ampicillin-sulbactam)** is first-line for aspiration lung abscess, due to excellent anaerobic coverage (8, 9). Our choice of ceftriaxone plus moxifloxacin was an alternative broad approach; literature suggests that fluoroquinolones with anaerobic activity can be effective options in lung abscess, especially when combined with a beta-lactam to cover a wide spectrum (2). It is important to note that in hospital-acquired cases or those with risk factors for resistant organisms, empiric coverage might need to be escalated (for example, using **piperacillin-tazobactam** or a **carbapenem** to cover Pseudomonas and other resistant Gram-negatives). In this ED case, however, the team appropriately targeted the likely community-acquired pathogens and her treatment was initiated within an hour of the diagnosis – a rapid response that likely prevented further clinical deterioration.

Supportive care in the ED included IV fluids for sepsis (a 1–2 liter normal saline bolus was given due to her relative hypotension and tachycardia, with careful monitoring because overhydration is a concern in dialysis patients), antipyretics for comfort, and analgesics to relieve pleuritic pain from the inflamed lung. The patient was closely monitored for any signs of respiratory failure or shock. Her respiratory status remained stable on low-flow oxygen, and blood pressure improved modestly with fluids (BP ~100/65 mmHg). She did not require intubation or vasopressors. Given the large size of the abscess, the ED and pulmonology team also considered whether any invasive intervention might be needed. At presentation, there were no signs of abscess rupture or massive hemoptysis, and the decision was to manage conservatively with antibiotics first. A plan was made to admit the patient to the hospital for further care under the pulmonary service, with early involvement of infectious disease specialists and thoracic surgery if needed. The ED team's prompt actions – **early imaging, immediate broad-spectrum antibiotics, culture procurement, oxygen and fluid support, and specialist consultation** – exemplify the critical role of emergency physicians in initiating care for complex infections like lung abscess. By expediting the diagnosis and treatment, the ED set the stage for the patient's recovery before irreversible complications could occur.

Treatment and Clinical Course

After admission, the patient remained on IV antibiotic therapy with ceftriaxone and moxifloxacin as the mainstay. Over the first 48–72 hours, she showed significant clinical improvement: her fevers subsided by the third hospital day, and her cough and work of breathing improved. She was able to wean off supplemental oxygen as her oxygenation normalized. Serial examinations noted that her crackles diminished over time. Inflammatory markers trended down (by day 5, WBC count had decreased to ~9,000/μL and CRP was markedly lower, reflecting treatment response).

No organisms grew from the sputum culture (likely due to prior antibiotic exposure and the difficulty of culturing anaerobes), and blood cultures remained negative. Despite the lack of a specific isolated pathogen, the broad empiric regimen was continued given the patient's clear clinical response. After one week of IV antibiotics, a repeat chest X-ray showed some improvement in the right upper lobe opacity. By the end of the second week of treatment, the patient's condition had improved to near-baseline – she was afebrile, breathing comfortably on room air, and eating well. A follow-up chest CT was obtained at around two weeks to assess the abscess cavity. The CT demonstrated that the cavitary lesion had significantly decreased in size (the cavity diameter reduced from ~8 cm to approximately 4 cm) and the surrounding consolidation had largely cleared. There was no evidence of new fluid collection or empyema. This radiologic improvement corroborated the clinical impression that the infection was responding.

Given her progress, the medical team transitioned the patient to oral antibiotic therapy to complete a prolonged course. Typically, a total of **4–6 weeks of antibiotic therapy** is recommended for lung abscess to ensure complete resolution (2). In this case, after 14 days of IV antibiotics, she was switched to oral moxifloxacin (which has excellent bioavailability and penetration) to continue therapy for an additional four weeks as an outpatient. High-dose **amoxicillin-clavulanate** was considered as an oral step-down option as well, to maintain anaerobic coverage, but since quinolone therapy had been effective and the patient tolerated it, moxifloxacin was continued orally. The patient was discharged home with instructions to finish the antibiotic course, with close follow-up arranged. She was educated on the importance of medication adherence and instructed to watch for any recurrence of fever, cough, or respiratory distress. Arrangements were made for her to follow up in the pulmonary clinic and also to continue her thrice-weekly dialysis sessions (coordinating to avoid antibiotic-dialysis timing issues).

Over the next weeks in follow-up, the patient did well. She reported gradually increasing energy and no return of her respiratory symptoms. A repeat chest X-ray one month after discharge showed only a residual scar in the right upper lobe with no fluid level. By the end of the full antibiotic course, the lung abscess had effectively resolved. The multidisciplinary management – involving emergency medicine, radiology, pulmonology, infectious disease, and the patient's dialysis team – resulted in a cure without the need for surgical intervention or percutaneous drainage. This favorable outcome in a young dialysis patient underscores how effective early and appropriate therapy can be, even in an immunocompromised host. Of note, had the patient not improved sufficiently on antibiotics, the next steps would have included consideration of **image-guided percutaneous drainage** of the abscess or even surgical resection (lobectomy) if necessary, as about 10–20% of lung abscess cases ultimately require invasive drainage or surgery when medical therapy fails (1,8). Fortunately, in this case, the timely antibiotic treatment obviated the need for any invasive procedures.

Conclusion

This case highlights the critical importance of early recognition and aggressive management of lung abscess in immunosuppressed patients. A young woman on dialysis developed a life-threatening lung abscess, yet prompt action in the emergency department – including **high clinical suspicion, swift imaging, and immediate initiation of broad-spectrum antibiotics** – led to a successful outcome. Emergency physicians serve a pivotal role as the first point of contact for such patients; their decisions can dramatically influence the prognosis. In an immunosuppressed or dialysis patient presenting with fever, cough, and respiratory distress, one must maintain a broad differential and a high index of suspicion for severe infections like lung abscess. Delayed or inadequate treatment of a lung abscess can result in complications such as empyema, bronchopleural fistula, sepsis, or hemorrhage, and can significantly increase mortality (3). Conversely, as demonstrated here, early intervention can lead to clinical cure and prevent the need for surgical management.

Multidisciplinary collaboration is often necessary to achieve the best outcomes. In this case, the emergency physician, radiologist, pulmonologist, and infectious disease specialist each contributed to rapid diagnosis and tailored therapy. The patient's dialysis status added complexity (in terms of immune status and antibiotic dosing considerations), illustrating the need for coordination with nephrology as well. By expediting consults and treatment, the ED set in motion a chain of care that minimized complications. Recent studies emphasize that even among critically ill patients, those who receive timely appropriate antibiotics and supportive care have markedly better survival. Emergency providers should be especially vigilant for lung abscess in high-risk groups – for example, an ESRD patient with "pneumonia" not responding to standard treatment – and advocate for definitive imaging (such as CT) to confirm the diagnosis.

In conclusion, **early recognition and management of lung abscess is lifesaving**, particularly in immunosuppressed populations like dialysis patients. A proactive approach in the emergency setting, coupled with broad-spectrum antimicrobial therapy and attentive supportive care, can eradicate the infection and improve patient outcomes. This case reinforces the adage that in emergency medicine, maintaining a high clinical suspicion for serious etiologies of common symptoms (fever, cough, dyspnea) is essential. By promptly identifying a lung abscess and initiating proper therapy, we can significantly reduce morbidity and mortality, even in vulnerable patients (3,6). Ongoing awareness, education, and a multidisciplinary approach are key to managing such complex infections successfully.

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Cerebral Fat Embolism Syndrome-Case Report

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Introductıon

Fat Embolism Syndrome (FES) is a rare complication, typically presenting with respiratory insufficiency following orthopedic trauma.

The classic presentation includes a triad of respiratory distress, petechial rash, and neurological changes (1). When neurological symptoms dominate, the condition is referred to as Cerebral Fat Embolism Syndrome (CFES) (2). Early recognition is crucial, especially in orthopedic trauma patients showing sudden neurological decline. Diagnosing CFES is challenging due to the lack of universal criteria; instead, it relies on clinical assessment and the patient's medical history (3).

Case Report

A 21-year-old male was brought to the Emergency Medicine department complaining of back and leg pain after a traffic accident. There was no history of drug use other than psychiatric medication. Upon arrival at the emergency department, his vital signs were stable, and his Glasgow Coma Scale score was 15. He was conscious, oriented and cooperative, and pupils were isochoric with bilateral positive light reflexes. Abdominal and respiratory examinations were normal. No obvious pathology was observed in the cranial nerve examination. Multiple bone fractures were present in the right lower extremity and muscle strength was not clearly assessed; no obvious muscle strength loss was observed in the other extremities. The patient's imaging showed right femur, fibula and tibial shaft fractures (fig 1), lung contusion, and C6 corpus fracture. Laboratory results were normal, there was no decrease in hemoglobin count. Toxicology results were normal.

The patient, who did not need urgent surgery from the thoracic and neurosurgery departments, had a long leg splint applied to his right lower extremity and was followed up with the operation plan. During the follow-up, the patient's vital signs remained stable. However, a few hours after admission, he suddenly developed confusion and agitation followed by rapid decline in level of consciousness. Thereupon, an emergency bedside ultrasound was performed. Upon the detection of hyperechogenic structures on the echocardiogram (fig 2), CT and diffusion MRI were performed, revealing multiple infarct areas on the diffusion MRI (fig 3) and no hemorrhage on CT. With supportive treatment, intensive care follow-up was planned with a preliminary diagnosis of fat embolism.

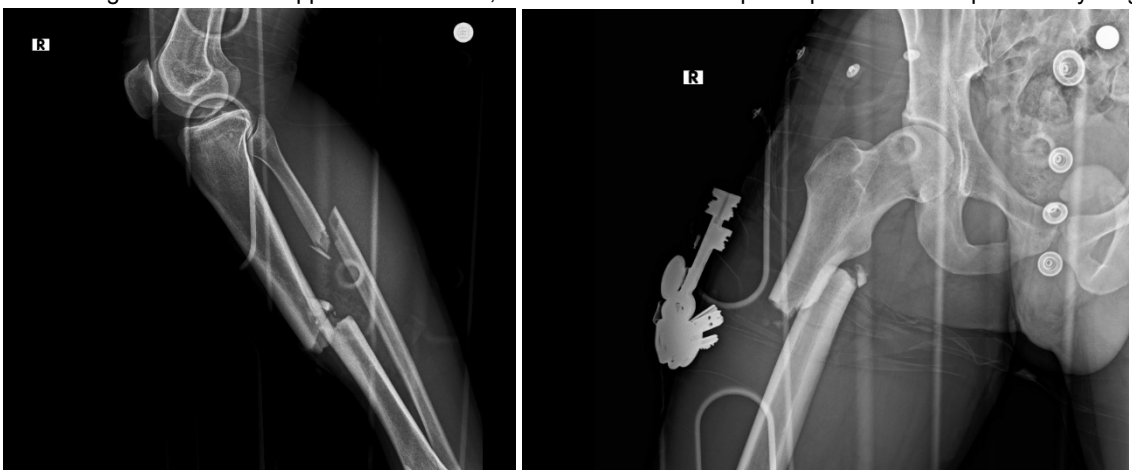

Figure 1. Femur, fibula and tibial shaft fractures

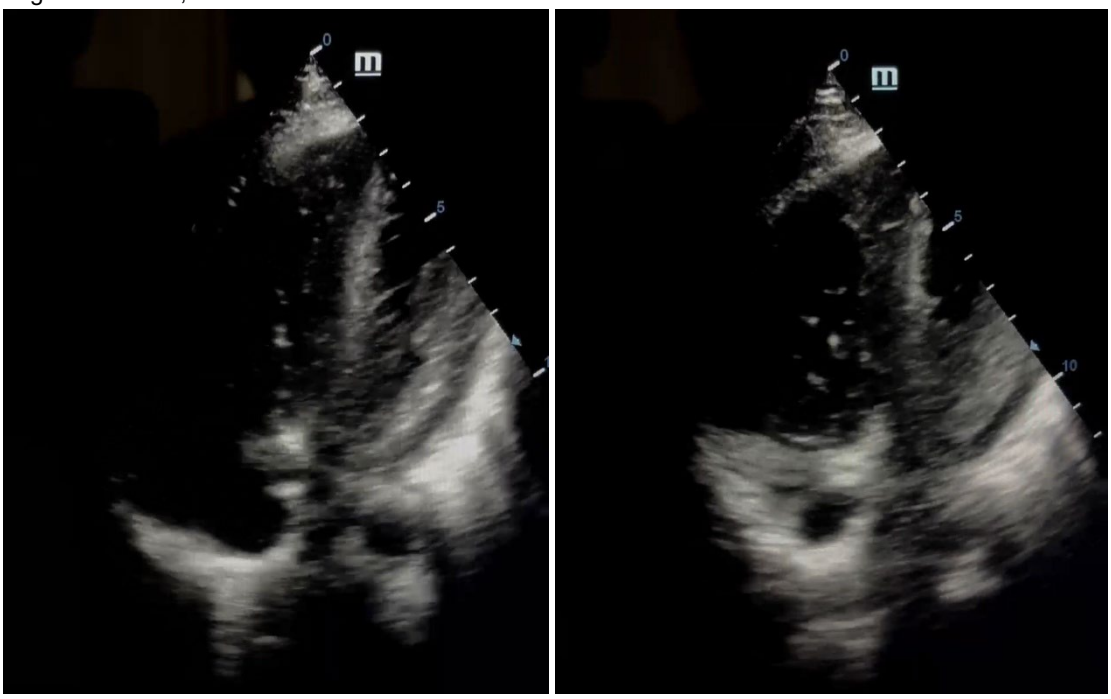

Figure 2. Hyperechogenic structures at Echo

Discussion

Fat Embolism Syndrome (FES) is an uncommon disorder primarily characterized by respiratory insufficiency, that occurs as a rare complication of orthopedic trauma. (1) Typically occurring within 1-2 days following trauma involving long-bone fractures or orthopedic surgery, it presents with a petechial rash, progressive respiratory insufficiency, and declining mental status.(4) However, only a small subset of patients, comprising 0.9% to 2.2%, develops Fat Embolism Syndrome (FES), which is classically characterized by a triad of respiratory symptoms, petechial rash, and alterations in mental status. (3) The term “Cerebral Fat Embolism Syndrome” (CFES) is used to describe cases where neurological symptoms are the predominant features. (2) Cerebral FES (CFES) is a rare variant of FES characterized by isolated neurologic symptoms including ischemic and hemorrhagic strokes, seizures, convulsive and non-convulsive status epilepticus, autonomic impairment, acute encephalopathy, and coma.(1,3) Also, literature reviews and case reports have shown that CFES is common in men with bilateral femur fractures in their early 30s.(5)

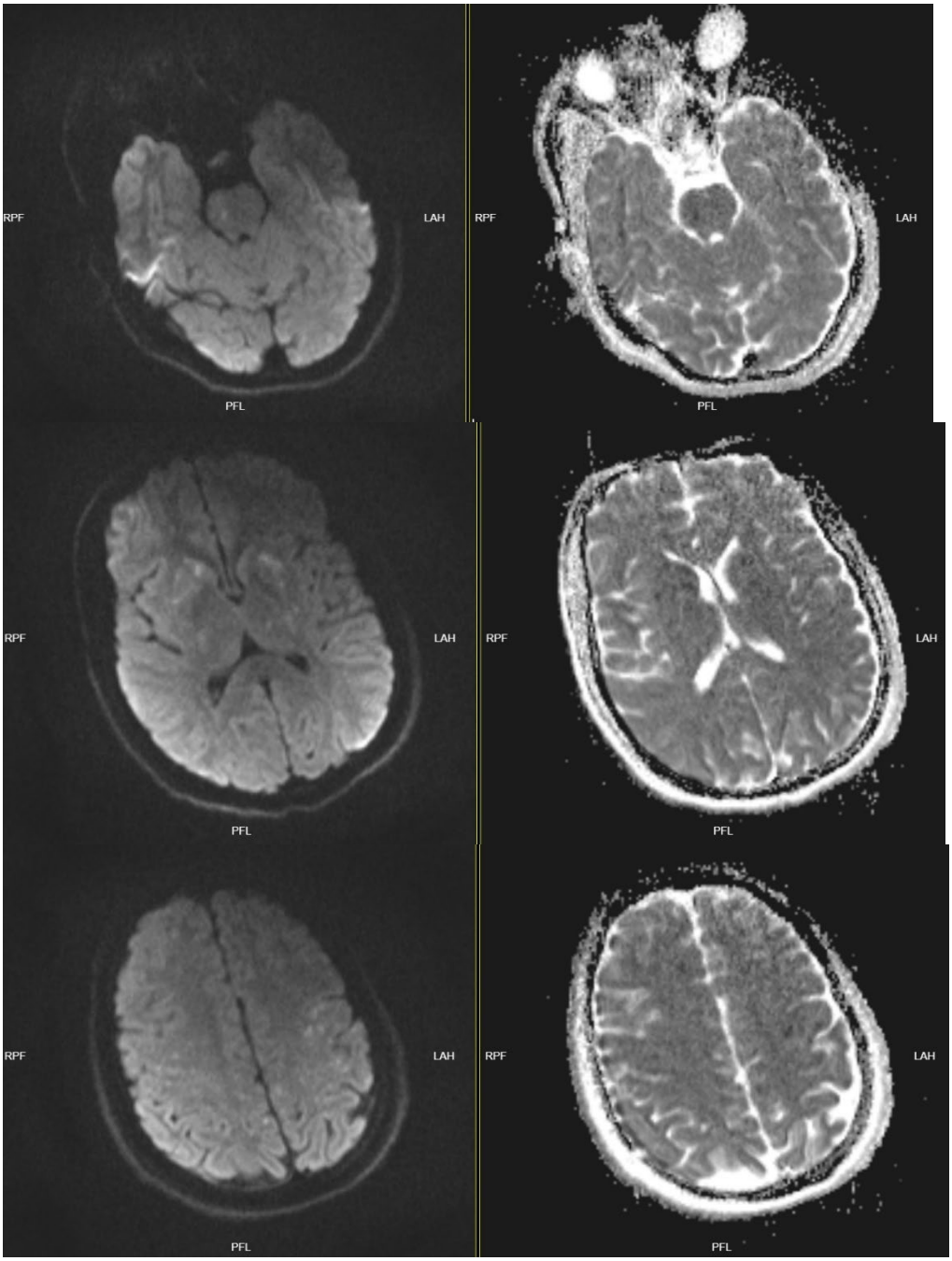

Figure 3. Multiple infarct areas on MRI

This diagnosis should be considered in trauma patients with long bone fractures who were found to have confusion or focal neurologic deficits. CFE should also be considered in patients with a history of remote trauma -such as in this case- who present with new-onset neurologic symptoms (5).

The hypothesized mechanism is that fat globules enter arterial circulation either through an intracardiac shunt such as patent foramen ovale, or, more commonly, microglobules may filter directly through lung capillaries to reach arterial circulation (3).
Magnetic Resonance Imaging (MRI) utilizing either diffusion-weighted imaging or susceptibility-weighted imaging sequences offers the greatest sensitivity for confirming the diagnosis (6,7). While there are no pathognomonic imaging findings, the 'starfield pattern' observed on MRI is the most prevalent and recognized manifestation of Cerebral Fat Embolism Syndrome (CFES) (8).
Also, in the study by Maghrebi et al., transthoracic echocardiography showed the "snowstorm" appearance of numerous hyperechoic particles in the inferior vena cava (9).
There are no definitive or specific treatments for FES; therefore, management is entirely supportive (6).
Considering the multifactorial etiology of fat embolism syndrome, although it cannot be prevented in all patients, it is argued that early operative fixation of long bone fractures reduces the incidence of FES (10,11).
In our case, we discussed a patient who was admitted to the hospital after a traffic accident who developed a change in consciousness during follow-up. The diagnosis of Cerebral Fat Embolism Syndrome (CFES) is challenging due to the non-specific nature of the symptoms and the frequent normality of computed tomography scans. High suspicion should be maintained in patients with orthopedic injuries who experience sudden neurological decline. There are no universal diagnostic criteria for CFES, making the diagnosis dependent on the patient's medical history and clinical manifestations (3).

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Pres In The Emergency Servıce (Posterıor Reversıble Encephalopathy Syndrome)

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Introductıon: Posterior reversible encephalopathy syndrome (PRES) is an acute-onset neurological disorder characterized by a variety of neurological symptoms, including headache, visual disturbances, disturbances of consciousness, confusion, seizures, and focal neurological deficits1. Posterior reversible encephalopathy syndrome (PRES) is a neurological disorder characterized by a spectrum of neurological signs and symptoms and prominent neuroimaging findings reflecting vasogenic edema. Computed tomography (CT) imaging of the white matter of patients reveals edema, especially in the posterior system. The only advantage of MRI is its ability to detect small, focal abnormalities beyond the resolution limits of CT.
Case: A 46-year-old male had chronic kidney disease and was on dialysis 3 days a week. It started with complaints of headache, dizziness, nausea, and vomiting the day before, and he was brought to the emergency room the day after unconscious. At the time of first admission to the emergency room, GKS: 5 (M3, V1, E1), blood pressure: 220/110 mmHg, pulse: 110 beats/min, spo2: 98, blood sugar: 125 mg/dL. Intubation was performed electively. Nicardipine was administered to the patient IV. Laboratory findings were as follows; Glucose: 260 mg/dL, Urea: 111mg/dL, Creatinine: 11.3mg/dL, Potassium: 3.4mmol/L, AST: 9 U/L, ALT: 8U/L, Hemoglobin: 10.5g/dL, Platelet: 115000 microg/L. No hemorrhage or mass was detected in brain tomography. Low-density hypodense areas were observed in both cerebellar hemispheres in brain tomography (Figure 1). No diffusion restriction suggesting acute ischemia was observed in diffusion MRI. High signal areas with ADC response were observed in occipital and cerebellar regions in diffusion MRI (Figure 2). Considering the clinical findings and imaging findings in the patient, PRES was considered in our case. The patient, who received dialysis and IV nicardipine in the intensive care unit, was extubated two days later. Clinical improvement was achieved in the normotensive patient who received nicardipine for another seven days. He was discharged with full clinical improvement after being followed up for approximately one month.
Conclusıon: This case is important in showing that PRES, which develops on the basis of renal failure accompanied by hypertensive crisis, is completely reversible with early diagnosis and appropriate treatment. PRES must be considered in the

differential diagnosis in emergency services and intensive care practice.

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A Comparative Analysis Of Triage Codes Of Patients Transported To Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital Via 112 Emergency Ambulance

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Introductıon

Emergency medical services (EMS) consist of a continuum that begins in the pre-hospital setting and extends through to in-hospital emergency care. Within this continuum, the two principal components are the 112 pre-hospital ambulance services and the hospital emergency departments (EDs). One of the significant challenges affecting the efficiency and sustainability of EMS is the inappropriate utilization of ambulance teams and EDs by patients with non-urgent medical conditions (Yıldırım et al., 2020). As the demand for emergency care continues to grow, healthcare systems are compelled to implement strategies that optimize resource allocation and response efficiency.

Triage has emerged as a critical method in addressing this challenge. According to the Regulation on Emergency Health Services in Türkiye, triage is defined as "a rapid selection and coding process conducted at the scene and at all healthcare facilities where mass casualties are present, aimed at identifying those requiring urgent treatment and transfer" (Ministry of Health of Türkiye, 2023).

Pre-hospital triage focuses on making the most efficient use of limited resources by rapidly assessing patients, identifying medical priorities, and referring them to appropriate care centers, sometimes after administering basic interventions. In contrast, triage in the ED setting is a structured classification system that determines the priority of medical evaluation and treatment based on the urgency of each patient's condition (Başpınar et al., 2022).

Therefore, the aim of this study is to evaluate the necessity of transferring patients to the emergency department by 112 Emergency Medical Services, to assess the knowledge and attitudes of 112 EMS personnel regarding triage, and to determine the appropriateness of emergency department and ambulance utilization in order to enhance the effective use of emergency healthcare services both in the pre-hospital and in-hospital phases.

Materıal Method

This study was designed as a descriptive and cross-sectional study and was conducted prospectively. The study population consisted of patients who were admitted to the emergency department via 112 Emergency Medical Services and had a corresponding case record documented in the ASOS (Emergency Health Automation System). The study covers patients who were admitted to the emergency department during March 2025. A total of 1,026 patients who met the inclusion criteria were evaluated.

Patient data were obtained from the 112 EMS Case Report Forms and the Hospital Information Management System (HIMS) of Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital. These data sources were used to assess the appropriateness of emergency department referrals, analyze triage-related decisions, and evaluate EMS personnel's documentation practices.

Results

A total of 1,026 patients who were admitted to the emergency department via 112 Emergency Medical Services were included in the study. Of these patients, 54,10% were male and 45,90% were female. The mean age was 55,1 ± 23,9 years.

According to the 112 Emergency Medical Services, out of 1,026 patients, 612 were categorized as green code, 379 as yellow code, 28 as red code, 6 as social indication, and 1 as black code. It was observed that the patient categorized as black code was evaluated as red code at Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital.

Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital accepts all patients arriving by 112 Emergency Medical Services ambulance into the red zone. As a result, 1,019 patients are classified as red code, and it was observed that 7 patients were categorized as black code. Among these 7 black code patients, the triage codes in 112 Emergency Medical Services were as follows: 4 were classified as red code, 2 as yellow code, and 1 as green code.

According to the 112 Emergency Medical Services diagnosis codes and the initial diagnosis codes from Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital, 6 out of 1,026 patients were diagnosed with cardiac arrest.

The tables below present the following: In Table 1, the initial diagnoses of patients in the red zone with more than 20 patients at Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital are listed, with the remaining diagnoses categorized as 'Other'. In Table 2, the final diagnoses of patients in the red zone with more than 20 patients are listed, with the remaining diagnoses categorized as 'Other'. In Table 3, the diagnoses of patients with more than 20 cases in 112 Emergency Medical Services are listed, with the

remaining diagnoses categorized as 'Other'.

Table 1. Initial diagnoses of patients brought by ambulance to the red zone at Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital	
Dyspnea	80
Chest Pain	70
Soft tissue injury	63
Fall	40
Nausea and Vomiting	38
Vertigo	36
Abdominal Pain	35
In-vehicle Trauma	33
Primary Hypertension	30
Acute Upper Respiratory Tract Infection (AURTI)	30
Anxiety	30
Motorcycle Accident	25
Cerebrovascular Disease (CVD)	24
Syncope	20
Other (Fever, Chronic Obstructive Pulmonary Disease (COPD), pneumonia, anaphylaxis, alcohol and drug use, gastrointestinal diseases, cardiovascular diseases, general condition disorders, etc.)	472

Table 2. Final diagnoses of patients brought by ambulance to the red zone at Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital	
Soft tissue injury	100
Dyspnea	90
Chest Pain	74
Nausea and Vomiting	41
Fall	39
Acute Upper Respiratory Tract Infection (AURTI)	37
Abdominal Pain	31
Myalgia	31
Anxiety	30
In-vehicle Trauma	29
Vertigo	29
Cerebrovascular Disease (CVD)	25
Primary Hypertension	21
Other (Fever, Chronic Obstructive Pulmonary Disease (COPD), pneumonia, gastrointestinal diseases, cardiovascular diseases, general condition disorders, etc.)	449

In Table 1, the initial diagnoses in the hospital's red zone consist of a total of 88 subgroups, while the final diagnoses in Table 2 consist of 87 subgroups. Since the subdiagnoses of 112 Emergency Medical Services total 136, the number of 'Other' diagnoses is higher.

Dıscussıon

This study provides a comprehensive analysis of triage categorizations and diagnostic consistency for patients transported to Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital via 112 Emergency Medical Services. One of the most striking findings was the high proportion of patients who were classified as green or yellow code in the pre-hospital setting, yet were admitted directly to the red zone upon arrival due to institutional policy. This systematic overtriage may lead to inappropriate allocation of emergency department resources. However, it also reduces ambulance occupancy time and provides the opportunity for the prompt intervention of actual emergency patients.

The frequent diagnoses among red zone admissions—such as dyspnea, chest pain, and soft tissue injuries—reflect common chief complaints seen in emergency departments. A notable observation was that the most common final diagnosis was soft tissue injury, whereas dyspnea and chest pain were more dominant in the initial assessments. This shift may suggest overtriage or challenges in initial assessment accuracy, possibly due to limited pre-hospital diagnostic tools.

The number of diagnosis subgroups in the 112 EMS records (136) was higher compared to hospital records, which may reflect the broader diagnostic coding practices in pre-hospital settings. Additionally, the variation observed between the initial and final diagnoses supports the need for continuous education of EMS personnel in triage and diagnostic reasoning.

Table 3. Diagnoses of patients taken to the red zone by ambulance at Tekirdağ Dr. İsmail Fehmi Cumalıoğlu City Hospital according to 112 Emergency Medical Services.	
Chest Pain	82
Dyspnea	81
Fall	72
Nausea and Vomiting	48
Cerebrovascular Disease (CVD)	36
Unspecified mood [affective] disorder	34
Motorcycle Accident	31
Abdominal Pain	31
Syncope	25
Vertigo	22
Hypotension	21
Other (Hypertension, Fever, Chronic Obstructive Pulmonary Disease (COPD), pneumonia, anaphylaxis, alcohol and drug use, gastrointestinal diseases, cardiovascular diseases, general condition disorders, epilepsy , In-vehicle Trauma etc.)	543

Table 4. Patient's Final Status	
Outpatient Discharge	668
Orthopedics Department	27
Coronary ICU	26
Internal Medicine Department	20
Level III Adult ICU	19
Referral	14
General Surgery Department	12
EX	11
Neurology Department	11
Pulmonology Department	11
Surgical ICU	11
Level II Neurology ICU	9
Obstetrics and Gynecology Department	9
Psychiatry Department	7
Neurosurgery Department	5
Level II Adult ICU	5
Palliative Care Department	4
Infectious Diseases Department	3
Refusal of Treatment	3
Pediatric Surgery Department	2
Pediatrics Department	2
Cardiology Department	2
Nephrology Department	2
Level III Pediatric ICU	1
Hematology Department	1
Geriatrics Department	1
Cardiovascular Surgery Department	1
Medical Oncology Department	1
Prisoner Service	1

Another remarkable finding is the presence of 7 patients evaluated as black code in the hospital, 6 of whom had been categorized with different triage levels in the pre-hospital setting. This discrepancy underlines the importance of dynamic reassessment and the critical nature of continuous monitoring during patient transfer.

Furthermore, a significant portion of these patients were ultimately diagnosed with non-urgent conditions such as soft tissue injuries, anxiety, vertigo, or acute upper respiratory tract infections. This pattern suggests considerable inappropriate use of both ambulance services and emergency department resources. Literature indicates a rising trend in the utilization of EMS for non-urgent cases, which contributes to overcrowding in emergency departments and reduces the overall efficiency in handling critically ill patients

(Booker et al., 2014; O'Cathain et al., 2010).

The mismatch between initial and final diagnoses also underscores the challenges of rapid clinical assessment in the field and the necessity of reassessment at the hospital. Nonetheless, the use of emergency services for non-emergent complaints may reflect broader issues such as limited access to primary care, lack of public awareness about EMS use, or sociocultural tendencies to overrely on emergency systems for convenience (Lowthian et al., 2011).

Despite its contributions, the study is limited to a single center and lacks a long-term follow-up of patient outcomes. Future multicenter studies involving qualitative assessment of triage decision-making processes could provide broader insights into improving emergency care efficiency.

Ultimately, this study highlights the need for improved public education on the appropriate use of emergency services, stricter triage adherence, and more effective triage mechanisms (etc. to increase on-site intervention) at both the pre-hospital and hospital levels.

Conclusıon

The findings of this study emphasize several critical challenges in the organization and utilization of emergency healthcare services. The observed discrepancies between EMS and hospital triage codes, combined with the frequent admission of non-urgent cases to the red zone, point toward systemic overtriage and inefficiencies in patient prioritization. However, it also reduces ambulance occupancy time and provides the opportunity for the prompt intervention of actual emergency patients.

Establishing a more widespread use of on-site intervention systems and promoting interinstitutional communication may contribute to more accurate resource use, improved patient safety, and better emergency service planning. In this context, regular audits and evidence-based policy adjustments will be vital to optimizing the emergency care system in Tekirdağ, Türkiye.

Additionally, the variation between initial and final diagnoses emphasizes the necessity of ongoing training for EMS personnel, as well as the need to harmonize triage standards between the pre-hospital and in-hospital environments.

Moreover, the diverse final statuses of patients, while patients with specialized care needs were effectively transferred to the appropriate departments, the volume of non-urgent admissions emphasizes the necessity of improving pre-hospital triage to reduce unnecessary emergency department visits and optimize resource allocation.

As a result, improving pre-hospital triage systems, enhancing public education on the appropriate use of ambulances and emergency services, and optimizing hospital admission processes are crucial steps in reducing unnecessary emergency department visits and improving resource allocation. By implementing more efficient triage systems and ensuring timely interventions, the healthcare system can better allocate resources for truly critical cases, ultimately leading to improved patient outcomes and system sustainability.

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Silent killer and the visual impairment they cause: a rare face of carbon monoxide exposure

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Introductıon

Carbon monoxide (CO) poisoning occurs when CO, an invisible, odorless, and flavorless gas produced by the incomplete combus-

tion of carbonaceous fuels, is inhaled. Carbon monoxide (CO) form carboxyhemoglobin (COHb) by exhibiting an affinity for hemoglobin that is roughly 200-250 times greater than that of oxygen. This condition inhibits the dissociation of oxygen from hemoglobin, resulting in hypoxia. Simultaneously, carbon monoxide binds to oxygen-transporting proteins such as cytochrome oxidase and myoglobin, resulting in metabolic disturbances at the cellular level. Carbon monoxide poisoning is a prevalent form of harmful gas exposure globally, primarily linked to stoves, barbecues, generators, and vehicle exhaust emissions in confined areas. The intensity of symptoms is determined by the duration of exposure and the ambient carbon monoxide concentration. Mild exposure results in nonspecific symptoms such as headache, dizziness, and nausea, whereas severe exposure can lead to confusion, loss of consciousness, coma, and death (1-3). Neurological symptoms often emerge following the acute phase of carbon monoxide intoxication. Neuroimaging results, including cerebral edema, necrosis in the basal ganglia, and cortical infarcts, were directly correlated with the exposure period. While a direct association between carboxyhemoglobin levels and clinical manifestations is absent, COHb levels >20% are typically associated with symptoms. Late neurological signs include Parkinson's disease, cognitive problems, psychosis, and sensory deficits. Nonetheless, vision impairment resulting from carbon monoxide exposure is a rare problem (4-6). We aim to report this case, which was brought to our Emergency Department (ED) because of blindness, to underscore the significance of obtaining a comprehensive history and performing laboratory investigations for carbon monoxide exposure in patients presenting with visual problems.

Case

A 49-year-old male patient was referred to us from another center with no medical history. It was learned that he was found in a state of unconsciousness but could be awakened by the strong smell of soot. An extinguished fire was found in the scene, and evidence suggested that the patient had been exposed to carbon monoxide gas for an extended period. In the patient's initial examination, GCS: 14, Temperature: 36.7 oC, Pulse:76/min TA:133/77 mm Hg. Conscious, oriented, and cooperative, but he was slightly drowsy. When the patient presented to our ED, the GCS was 15, and the other vital signs were normal. ECG: NSR, and there was no chest pain. It was learned that patients experienced difficulty seeing because of their complaints. The initial central imaging performed at the First Center revealed an ischemic infarct area in the left occipital lobe. This condition was evaluated by neurology as a hypoxic-ischemic event due to prolonged CO gas exposure. In the brain MRI performed in our ED, infarct areas were detected in the bilateral occipital, cerebellar, and PCA watershed areas. The lesions were evaluated by neurology as hypoxic-ischemic events due to CO gas exposure. The patient was referred to a cardiology specialist due to troponin levels above the reference range. His ECG results were normal, and acute coronary syndrome was not considered. It was concluded that the elevation of troponin was secondary to hypoxia. The patient, whose initial COHb level was 25%, had a normal COHb level in the control blood gas analysis in our ED. Following hyperbaric oxygen therapy, the patient was monitored in the toxicology intensive care unit, and HBOT was administered. After 5 sessions of HBO treatment, the patient's follow-up showed a significant reduction in the number of visual disturbance complaints. On the 10th day after discharge, an evaluation by the ophthalmology department revealed a significant improvement in the patient's visual function, and the patient was called for a follow-up outpatient clinic appointment 1 month later.

Dıscussıon

Visual impairment due to carbon monoxide poisoning is a rare outcome in the literature. Limited examples have been documented in the literature (4). This situation also requires consideration of methanol poisoning and caution in this regard. The best way to clarify this situation is to carefully review the data ontained from the scene and for the physician to focus on these details in the medical history. Moreover, laboratory tests are undoubtedly important tools, especially in cases where evidence is insufficient.

Following carbon monoxide exposure, bilateral occipital brain infarction occurred, causing significant visual impairment in the patient. Visual impairment due to carbon monoxide exposure can occur in various ways. Carbon monoxide can directly affect the optic nerve, causing neuropathy, retinal ischemia, or ischemic damage to the occipital brain. Optic neuropathy, central scotoma, visual field deficits, and cortical visual abnormalities associated with CO exposure have been described in the literature. The high oxygen consumption of the occipital brain may make it more vulnerable to carbon monoxide-induced hypoxia (5-7).

In some previously documented cases, neurological outcomes in patients who did not receive hyperbaric oxygen therapy (HBO) have been shown to be irreversible. In our patient, immediate initiation of HBO therapy resulted in significant improvement in visual function. This further confirms that HBO is an extraordinarily effective therapeutic intervention for CO poisoning associated with visual impairment. In carbon monoxide poisoning, hyperbaric oxygen therapy rapidly reduces the half-life of carboxyhemoglobin, facilitating the elimination of carbon monoxide from the body and increasing oxygen delivery to the tissues. Furthermore, it inhibits the formation of free radicals, thereby preventing oxidative stress and aiding in the recovery from hypoxic damage (4, 5, 7, 8). In our case, the patient's COHb level returned to normal, but vision loss persisted. Consequently, HBO therapy was promptly initiated, resulting in a significant improvement in visual function (9). Our patient was transferred to our Emergency Department from another facility and arrived at our hospital after an estimated 8- to 10-hour drive. The HbCO level was within normal limits in our Emergency Department. This is important because it demonstrates the potential advantages of HBO therapy for individuals who have missed the early stages of severe neurological disorders and life-threatening illnesses.

Conclusıon

Carbon monoxide poisoning is a severe toxic disease affecting multiple systems. Neurological complaints are among the most common problems. Visual impairment is a rare condition after carbon monoxide poisoning, with limited cases documented in the literature. HBO therapy, applied 8-10 hours after exposurre and repeated, was advantageous in significantly restoring visual function in our patient. The literature suggests that visual effects may be permanent in patients who do not received HBO therapy. This case

highlights the potential importance of HBO therapy for those with visual problems resulting from CO poisoning.

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A Rare but Serious Complication of Therapeutic Botulinum Toxin: A Case of Iatrogenic Botulism

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Introduction

Iatrogenic botulism is a rare adverse reaction that can occur after the administration of botulinum toxin injections for medical or cosmetic purposes, particularly in cases of overdose or improper application. Botulinum toxin causes muscle relaxation and temporary paralysis by blocking the release of acetylcholine at the neuromuscular junction. Because of these effects, it is widely used in the treatment of neurological disorders, muscle dystonia, chronic migraine, and for aesthetic purposes. However, incorrect dosage, inappropriate injection sites, or systemic spread of the toxin may result in severe side effects such as dysphagia, dysarthria, ptosis, and respiratory muscle weakness. Here we present a patient who developed iatrogenic botulism after therapeutic botulinum toxin injections.

Case Presentation

A 49-year-old female patient presented to the emergency department with nausea, vomiting, shortness of breath, dysphagia, hypersalivation, and hoarseness. Her medical history revealed that she had been receiving botulinum toxin type A (BoNT-A) injections into the neck, masseter muscle, base of tongue, and tip of tongue every other day for one week for the treatment of acute dystonia, with the last injection administered 12 hours prior to presentation. On admission, her vital signs were within normal limits. Physical examination revealed intermittent blurred vision. Ocular and lid movements were normal. There was no uveal edema. Increased lacrimation and accumulation of saliva in the larynx were observed. The patient had dysphonia and dysarthria. Pharyngeal reflex was absent. Bilateral lung sounds were normal on auscultation, but use of accessory respiratory muscles was noted. Intravenous hydration and antiemetic therapy were initiated. The poison control center was consulted and administration of BoNT-A antitoxin was recommended. The patient was admitted to a tertiary intensive care unit for close monitoring. Supportive care, including hydration and symptomatic management, was provided in addition to antitoxin treatment. On the fifth day of ICU admission, the patient was discharged on medical advice with full recovery.

Discussion

Although rare, iatrogenic botulism is a potentially life-threatening complication of botulinum toxin therapy. The increasing use of botulinum toxin type A (BoNT-A) for both therapeutic and cosmetic purposes has heightened awareness of the importance of appropriate dosage, injection technique, and patient selection. This case highlights the clinical manifestations of iatrogenic botulism, which may mimic other neurological conditions and therefore requires a high index of suspicion for timely diagnosis.

The patient's symptoms-including dysphagia, dysarthria, blurred vision, hypersalivation, and respiratory distress-are consistent with a systemic effect of botulinum toxin. These manifestations result from widespread inhibition of acetylcholine release, not only at the targeted neuromuscular junctions, but also in unintended muscle groups and autonomic pathways (1). The absence of fever, normal inflammatory markers, and lack of structural abnormalities supported a toxin-mediated cause rather than an infectious or structural etiology.

Diagnosis of iatrogenic botulism remains largely clinical, relying on a detailed patient history and neurological examination, as laboratory confirmation (e.g., mouse bioassay or serum toxin detection) may not always be available or timely (2). In this case, the temporal relationship between toxin administration and symptom onset, as well as the distribution of symptoms, were key to making the diagnosis.

Treatment is primarily supportive, including airway management, nutritional support, and symptom-specific interventions. In severe cases, as seen here, administration of botulinum antitoxin is indicated to neutralize circulating toxin and prevent further progression of symptoms. Although antitoxin does not reverse existing neuromuscular blockade, early administration can significantly reduce morbidity and hasten recovery (3). The patient's favorable outcome in this case reflects prompt recognition, appropriate supportive

therapy, and timely use of antitoxin.

This case highlights the need for strict adherence to dosing guidelines and injection protocols, especially when multiple anatomical regions are involved. In addition, healthcare providers must be properly trained to recognize early signs of systemic botulinum toxicity. As the use of botulinum toxin becomes more widespread, particularly in non-hospital settings, public health strategies should include practitioner education and clear reporting mechanisms for adverse effects (4).

Conclusion

In this case, the symptoms that developed after multiple injections for the treatment of acute dystonia were consistent with iatrogenic botulism, and the patient was successfully treated with antitoxin therapy and supportive care. This case highlights the importance of a thorough history and physical examination for the clinical diagnosis of iatrogenic botulism. Early diagnosis and appropriate supportive care are essential to prevent complications. When botulinum toxin is administered, factors such as dosage, injection sites, and individual risk factors must be carefully considered, and healthcare professionals should receive adequate training to minimize the risk of misuse.

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Idiopathic Pulmonary Vein Thrombosis Treated With Rivaroxaban

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Introductıon

Pulmonary vein thrombosis (PVT) is a thrombotic event that occurs in the pulmonary veins. It is usually observed in patients with risk factors such as immobilization, surgical interventions, cardiovascular diseases and diabetes. In elderly individuals, a sedentary lifestyle significantly increases the risk of developing thrombosis. Since PVT often presents with nonspecific symptoms, accurate diagnosis can often be difficult. However, early recognition of the disease can ensure that the treatment process is successful. In this article, we describe the diagnosis and management of pulmonary vein thrombosis in an elderly and immobile patient.

Case

A 96-year-old woman presented with a history of diabetes mellitus (DM), immobilization and percutaneous endoscopic gastrostomy (PEGfeeding after a right hip fracture. It was stated that the patient was receiving prolonged oxygen therapy (USOT). The case started with complaints that developed suddenly at night; the patient had shortness of breath, bruising, shivering, impaired consciousness and high fever. On examination in the emergency department, the patient's general condition, blood pressure arterial 89/54 mmHg, oxygen saturation 94% (oxygen support was provided with 4 lt/min nasal cannula), pulse 56 beats/min, respiratory rate 20/min and tachypnea were observed. A decrease in respiratory sounds was observed and an increase in the diameter of the right foot was observed. Blood gas analysis showed pH: 7.49, PCO2: 48.3, PO2: 49, SO2: 84.9 and HCO3: 35.3, indicating hypoxemia and metabolic compensation. Further investigations confirmed the diagnosis of pulmonary vein thrombosis (PVT).

Echocardiography showed normal left ventricular function, but no clear conclusion about the presence of thrombus could not be obtained due to imaging failure. However, after a definitive diagnosis of PVT was made, the patient was interned to the 3rd level intensive care unit after consultations with pulmonology, cardiology, cardiovascular surgery and anesthesia. In the intensive care unit, heparin infusion was started and the patient was followed up there. After clinical stabilization was achieved, the patient's treatment process continued in the ward. Rivaroxaban was started and the patient was discharged when his condition stabilized.

Dıscussıon

Pulmonary vein thrombosis (PVT) is a rare and serious thrombotic condition that usually develops in patients requiring immobilization and bed rest. In elderly individuals, factors such as diabetes mellitus, cardiac diseases and surgical interventions constitute important risk factors for the development of PVT. This case demonstrates that elderly patients are at high risk for thrombus formation and the treatment of this condition requires a multifaceted approach (1).

The clinical manifestations of PVT are often nonspecific and may be confused with other cardiovascular and pulmonary diseases. Findings such as shortness of breath, bruising and fever may be confused with pulmonary embolism, cardiac failure and infection. Therefore, in the diagnosis of pulmonary vein thrombosis, clinical suspicions as well as advanced investigations including blood gas tests, pulmonary imaging and laboratory analyzes are of great importance (2).

However, the patient's course of treatment was successfully managed with anticoagulation therapy (heparin infusion followed by

rivaroxaban). A multidisciplinary approach and the collaboration of cardiology, pulmonology and intensive care specialists stabilized the patient's condition. The treatment process in elderly patients usually carries a high risk and a very careful evaluation should be performed before initiating treatment (3). Anticoagulation is primarily applied in the treatment of pulmonary vein thrombosis. Treatment is usually started with heparin infusion and then continued with oral anticoagulants such as rivaroxaban (4). In the case of larger and more dangerous clots, thrombolytic therapy may be preferred, which provides rapid clot dissolution and can be effective within a few hours to a few days (5). However, in some cases, especially in the presence of large clots or in the absence of response to other treatments, surgical embolectomy may be necessary. This procedure aims to physically remove clots from the pulmonary artery (6). Percutaneous interventional methods can be used in patients with a high risk of bleeding or in patients who cannot receive thrombolytic therapy; with this method, the clot can be removed mechanically with the help of a catheter. In addition, vena cava filters may be preferred in patients with lower extremity deep vein thrombosis who cannot receive anticoagulant therapy or who are at risk of recurrent pulmonary embolism. These filters placed in the inferior vena cava reduce the risk of embolism by preventing clots that may break off from the legs from reaching the lungs (4).

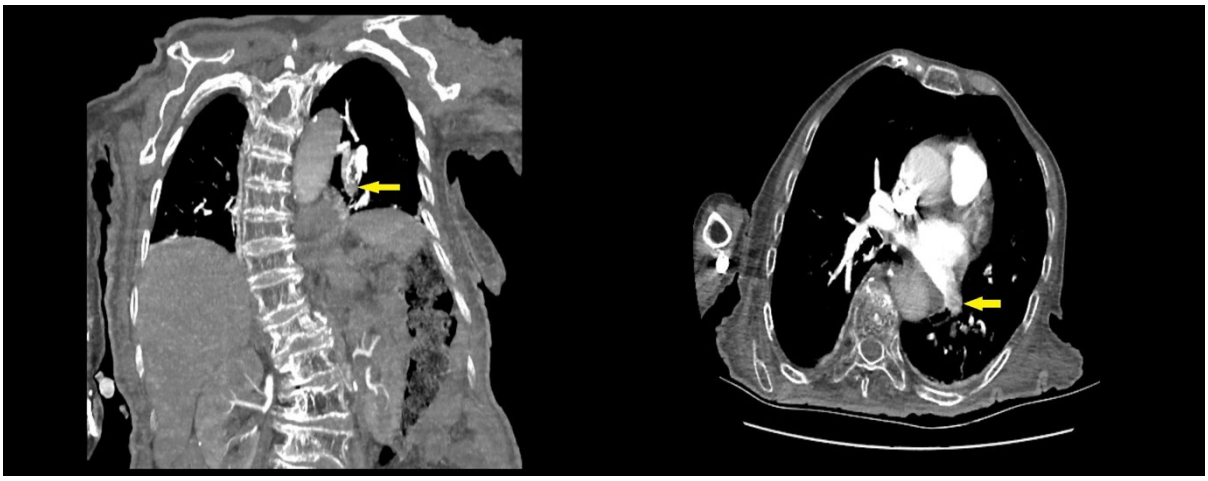

Figure 1. Thrombus material in the pulmonary vein (yellow arrow)

conclusıon

Early recognition, prevention of complications and treatment management of pulmonary vein thrombosis in elderly and immobilized patients is vital. Anticoagulation, oxygen therapy and a multidisciplinary approach are critical. Prognosis can be improved with early diagnosis and appropriate treatment.

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Isolated cerebral fat embolism after long bone fracture: A case report.

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Introduction

Fat embolism syndrome (FES) is a clinical phenomenon characterized by the spread of fat emboli in the systemic circulation. The spread of fat emboli disrupts the capillary vascular bed, affecting the microcirculation and causing a systemic inflammatory response syndrome (1).

Fat embolism syndrome occurs after long bone fractures and total hip arthroplasty, and is less commonly associated with burns and soft tissue injuries (2). The most common clinical findings are respiratory system findings such as hypoxemia and dyspnea, while neurological disorders such as impaired consciousness are the second most common findings (3).

Rarely, some cases present with predominantly neurological findings, and in this case, isolated cerebral fat embolism syndrome is diagnosed (4). In a study examining cases of cerebral fat embolism after traumatic bone fracture, 268 case reports were examined, and the relationship between acute central involvement and patent foramen ovale (PFO) was not clearly understood (5). In a case report in 2022 in which cerebral fat embolism was seen after multiple traumas, patent foramen ovale was seen in the patient, and elective closure was deemed appropriate after discharge (6). In a case report we reviewed, it was emphasized that the presence of PFO as an embolization route is a situation that clinicians should consider in the case of cerebral fat embolism (7). Neuroimaging is a critical tool in the evaluation of cerebral fat embolism, and five distinct brain MRI patterns have been reported in the acute, subacute, and late stages: diffuse cytotoxic edema, referred to as the “Star Field” pattern, diffuse cytotoxic edema in the white matter, vasogenic edema lesions that may develop, petechial hemorrhages in the white matter, and chronic sequelae (8)

In our article, we presented our approach to a 21-year-old patient who had bilateral displaced femoral shaft fractures after a traffic accident and experienced acute neurological deterioration approximately 24 hours later. In our case, the presence of PFO was shown in further examinations, and although there are similar studies in the literature, sufficient research has not been conducted. During the clinical course and treatment, intravenous albumin treatment was applied to correct hypovolemia and increase the binding of fatty acids, and benefit was shown.

Case

A 21-year-old female patient was admitted to an external center due to a vehicle traffic accident. The patient, whose imaging findings at the external center showed bilateral femoral shaft fractures and whose central imaging findings were normal, was referred to us within the first 24 hours of her observation due to acute changes in consciousness and seizures before surgery. A 21-year-old female patient was admitted to an external center due to a vehicle traffic accident. The patient, whose imaging findings showed bilateral femoral shaft fractures and whose central imaging findings were normal, was admitted to the hospital for surgery by orthopedics and traumatology. She was referred to us within the first 24 hours of her observation due to acute changes in consciousness and seizures. At the time of presentation to us, the patient with Glasgow Coma Scale 7, positive direct and indirect light reflexes, no evidence of meningeal irritation, and decerebrate posture was electively intubated with sedation (intravenous ketamine 50 mg and paralyzing agent (intravenous rocuronium 25 mg). The patient did not have deep venous thrombosis, and no pathology was detected in bedside focused traumatic ultrasonography. There were no right ventricular overload findings in the transthoracic echocardiography. There were petechiae, mostly in the neck, and widespread in the upper extremities. The patient, who had no medical history, underwent contrast-enhanced brain magnetic resonance imaging with preliminary diagnoses of diffuse axonal damage and cerebral fat embolism. The patient, who had scattered multiple punctate infarct areas in the brain magnetic resonance imaging and no pulmonary embolism in the thoracic tomography, was accepted as having isolated cerebral fat embolism and was referred to emergency intensive care. was taken into care.

During the treatment process, the patient was monitored under sedation and in addition to supportive treatment, subcutaneous enoxaparin 4000 units twice daily for 8 days and intravenous albumin 100 mg once daily for 3 days were started. The patient who had a witnessed tonic-clonic seizure was started on levetiracetam 500 mg 2*1. In the follow-up, the patient had fever and high acute phase reactants, and official echocardiography and blood culture were studied. Ampicillin sulbactam was given prophylactically. The patient had gram-positive clustered cocci and yeast growth in his culture, and PFO was detected in his transesophageal echocardiography. Vancomycin 1 gram 2*1 and caspofungin 70 mg 1*1 were started for antibiotics. During the intensive care follow-up period, he was immobilized with an external fixator by orthopedics. In our follow-ups, the patient who was clinically stable had his sedative agents revised and reduced. At the end of the fifth day, the patient's Glasgow coma score showed eye movements of 4, motor movements of 5, and he was intubated. The patient, who was stated to have an operation planned by orthopedics and traumatology, was transferred to the anesthesiology and reanimation department on the 8th day of his follow-up, consciously. The patient, who was followed by us for approximately 8 days, was transferred to the anesthesiology intensive care unit for pre-op preparation and follow-up, consciously. The patient, who was followed by anesthesiology for approximately 25 days, was transferred to the ward by orthopedics and underwent bilateral femur surgery in two different sessions. The patient was discharged 7 days after the operation without any neurological or orthopedic sequelae.

Dıscussıon

Fat embolism syndrome is a rare syndrome that occurs within 24-48 days after trauma and is clinically characterized by confusion, respiratory distress, and petechial rash. Its pathophysiology includes the obstructive effect of fat globules entering the circulation after the fracture. History, clinical suspicion, and imaging are at the forefront in diagnosis. In our case, the patient who was followed up with bilateral femur fractures after trauma had symptoms of impaired consciousness and respiratory distress. The patent foramen ovale found in the patient who was intubated during follow-up supported the theory and diagnosis of fat embolism syndrome.

The patient, who was monitored in intensive care, was given low-molecular-weight heparin and albumin during the treatment. The patient's general condition improved during the follow-ups, and neurological and respiratory symptoms improved. The patient, whose GCS increased, was transferred to the relevant departments for follow-up and operation purposes. The patient, who was extubated on the 27th day after intubation, was discharged with recovery on the 36th day after the operations were completed.

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8152

Allergic reaction to pigeon meat consumption: a rare case

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Introductıon

Food allergies are immune-mediated reactions that can occur through different immunological mechanisms and may present with systemic symptoms ranging from mild skin reactions to anaphylaxis. Although poultry meat allergy is rare, cases involving chicken and turkey meat are more frequently reported. Pigeon meat allergy, however, is documented in only a few cases in the literature (1,2).

In this case report, we present a patient with no prior history of food allergy who experienced an anaphylactic reaction after consuming pigeon meat for the first time.

Case

Our patient was a 34-year-old male. He reported consuming pigeon meat for the first time during lunch and developed symptoms of widespread itching, facial redness, lip swelling, wheezing, and shortness of breath within approximately 30 minutes. The patient had no known chronic diseases, was not taking any medications, and had no history of atopic conditions, such as asthma, atopic dermatitis, or allergic rhinitis. He stated that he had previously consumed chicken, turkey, and other poultry without any adverse reactions.

Upon arrival, his vital signs were as follows: blood pressure (BP), 100/60 mmHg; pulse rate (PR), 110/min, respiratory rate, 22/min, and oxygen saturation, 96%. Physical examination revealed widespread erythematous rash on his face and trunk. Auscultation revealed bilateral wheezing and prolonged expiration. Laboratory findings were unremarkable except for elevated serum total IgE (350 IU/mL) and eosinophil counts (450/mm³).

The patient was treated with one dose of intramuscular adrenaline (0.3 mg, 1:1000). Antihistamine (IV pheniramine) and corticosteroid (IV methylprednisolone) therapy was initiated, and oxygen support was provided. He was monitored until his symptoms resolved. The patient was advised to avoid pigeon meat and pigeon-derived products. He was referred to the allergy and immunology outpatient clinic for further evaluation and testing regarding poultry meat consumption.

Dıscussıon

The patient's anaphylactic reaction to pigeon meat consumption suggests IgE-mediated food allergy. The absence of cross-reactivity with chicken and turkey meat indicates a sensitization mechanism specific to pigeon muscle proteins (3).

Allergic reactions to poultry meat are generally associated with IgE-mediated sensitization to proteins such as serum albumins, muscle troponins, and parvalbumins (4).

Bird-egg syndrome: This condition is linked to cross-reactivity between chicken meat and egg proteins (5).

IgE sensitization specific to pigeon muscle proteins: This mechanism has been poorly studied in the literature, and it has been suggested that sensitization to unique epitopes may play a role in meat allergies (6).

Conclusıon

This case highlights a rare case of pigeon meat allergy that has been documented in the literature. The lack of cross-reactivity with chicken and turkey meat suggests the development of an immune response specific to pigeon meat. Since pigeon meat is rarely consumed, such allergic reactions are challenging to identify.

Specific immunoglobulin E (IgE) and provocation tests play critical roles in diagnosing rare allergies. In cases of allergy and atopy, clinicians should consider rare meat allergies and educate patients about emergency treatment and preventive strategies.

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Altered Mental Status In A Gerıatrıc Patıent: Delırıum Or Encephalıtıs?

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Introductıon

Meningitis is an inflammatory disease of the meninges and cerebrospinal fluid (CSF). It can be caused by infectious agents (bacteria, viruses, fungi, parasites) or non-infectious factors. There is no specific history, physical examination finding, or scoring system for diagnosis. Clinical suspicion and experience are crucial. CSF evaluation via lumbar puncture is the gold standard. Most viral meningitis cases are idiopathic, but polymerase chain reaction (PCR) has improved the identification of rare causes. Human herpesvirus 6 (HHV-6) is usually latent and widespread worldwide. Most people get infected in childhood, but it is generally asymptomatic.

Case

A 73-year-old female patient presented to the emergency department with complaints of altered mental status, disorientation, urinary incontinence, reduced oral intake, and inability to recognize relatives for the past two days. One week before symptom onset, she had been hospitalized in intensive care following an angiography. She had previously been diagnosed with delirium and received treatment at another hospital. On examination, she was confused, with limited orientation and cooperation. Her vital signs were stable. Glasgow Coma Scale score was 11. Neck stiffness was questionable, and muscle strength assessment was limited, but spontaneous movement was observed in all four extremities. Laboratory results: GFR: 34, Creatinine: 1.51 mg/Dl, Urea: 183.8 mg/dL, Uric acid: 12.53 mg/dL, CRP: 0.61 mg/dL, WBC: 7.1 x 10^3/µL, Neutrophils: 56.1%, ABG: pH: 7.45, pO□: 55.4 mmHg, pCO□: 33.7 mmHg, sO□: 88%, lactate: 1.92 mmol/L. Cranial imaging showed no acute pathology. The patient was monitored in the emergency department, and cranial MRI revealed diffuse subcortical-periventricular diffusion restriction in both cerebral hemispheres. With a preliminary diagnosis of encephalopathy, lumbar puncture (LP) was planned. The CSF PCR panel detected HHV-6 positivity. After neurology and infectious disease consultations, the patient was started on Ganciclovir (2x2.5 mg/kg IV) and Levetiracetam (2x500 mg IV). She showed dramatic clinical improvement and was admitted to the infectious diseases department.

Dıscussıon

Reactivation of HHV-6, whether primary or latent, can rarely lead to meningitis or meningoencephalitis with a poor prognosis. Few cases of HHV-6 encephalitis have been reported, most of which had fatal outcomes. Due to the widespread nature of the virus and the frequency of childhood infections, HHV-6 infections and reactivations are rare in adults, particularly with central nervous system involvement. Reported cases have shown clinical improvement with intravenous ganciclovir or valganciclovir treatment.

KEYWORDS: Human Herpesvirus 6, Lumbar Puncture, Meningitis

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Elevated CK and Kidney Failure: An Overlooked Detail in Medication Use?

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Introductıon:

Creatine Kinase (CK) was first defined as a biochemical marker in 1966. (1) Elevated CK levels are a sensitive and reliable indicator of muscle damage, and this increase is directly proportional to the extent of muscle damage and the severity of the disease. (3) CK levels can rise due to direct muscle damage in trauma or ischemia in peripheral artery diseases. In addition, heart diseases, some central nervous system disorders, sepsis, thyroid disorders, and certain pharmacological agents can also cause an elevation in CK levels. (2,3) Muscle breakdown can lead to a wide spectrum of problems, ranging from asymptomatic elevated muscle enzymes to

life-threatening electrolyte imbalances, acute kidney failure, multiple organ failure, and even death. Therefore, early diagnosis and treatment are of great importance. (3,4)

Case:

A 47-year-old male patient presented to the emergency department with abdominal pain that had been ongoing for two days. On systemic examination, widespread abdominal pain was noted, but no signs of guarding or rebound tenderness were observed. The patient's medical history includes a recent diagnosis of diabetes. For the past 10 days, the patient has been taking Diamicron MR 30 mg twice daily. Laboratory results showed a GFR of 54, creatinine of 1.54 mg/dL, urea of 37 mg/dL, uric acid of 5.4 mg/dL, CK of 2797 U/L, AST of 77 U/L, LDH of 306 U/L, WBC of 9.47 x 10^3/µL, pH of 7.33 and lactate of 1.64 mmol/L. The patient's ECG showed sinus rhythm. There is no recent history of exercise, trauma, or additional medication use. Given the elevated CK levels, the patient was started on fluid resuscitation in the emergency department. Following a decrease in CK levels, the patient was advised to increase fluid intake, avoid strenuous physical activity, and potentially undergo a medication change. He was also recommended to follow up with the endocrinology department, where he is being monitored, and was discharged.

Dıscussıon:

Trauma, exercise, muscle diseases, heart diseases, some central nervous system disorders, sepsis, thyroid disorders, and certain pharmacological agents can also cause elevated CK levels. (2,3) Diamicron MR is an oral antidiabetic drug from the sulfonylurea class that lowers blood sugar levels. Diamicron MR is used in adults for the treatment of insulin-independent (type 2) diabetes when diet, physical exercise, and weight loss alone are insufficient to control blood sugar. It is used in patients with mild to moderate renal insufficiency, but only with careful monitoring, similar to its use in patients with normal kidney function. (5)

Considering this information, in elderly patients with borderline kidney function and reduced oral intake who are using Diamicron, the patients' CK levels should be carefully monitored. It should be emphasized that these patients need to pay attention to their fluid intake and avoid strenuous physical activities.

KEYWORDS: Creatine Kinase, Oral Antidiabetics, Sulfonylurea Class Antidiabetics

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Acute Maras Powder Intoxıcatıon – Case Report

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Introductıon

Smokeless tobacco has become more prevalent worldwide and is known by different names in various cultures(1). A type of smokeless tobacco known worldwide as Aztec tobacco and in Turkey as "Maras Powder (Otu)" is generally made by mixing the dried leaves of the Nicotiana Rustica plant with the ashes of trees such as oak, although its production methods may vary (2). About a teaspoon of the powder is used either alone or wrapped in cigarette paper and placed on the upper or lower labial mucosa, where it is held for 5 to 10 minutes. The frequency of use varies depending on the level of addiction and the individual (3). In regular cigarettes, the amount of nicotine absorbed per cigarette ranges from approximately 0.05 to 2.5 mg, whereas the amount of nicotine absorbed from Maras Otu ranges from 7 to 9 mg. This indicates that it delivers nearly 10 times more nicotine compared to cigarettes (4).

Use of Maras Powder has been observed to be more common among married men with low educational and income levels. Additionally, its use is more prevalent among individuals over the age of 46 who have previously smoked. These data suggest that younger users may turn to Maras Powder over time as an alternative to cigarettes to enhance satisfaction (5). It is also known that Maras Powder is preferred as a means of quitting smoking (6). Due to its high nicotine content, Maras Powder is a substance that can cause severe toxicity and must be promptly recognized and treated in cases of acute intoxication (7).

Objective: In our case report, we aim to describe the clinical presentation of confusion and syncope resulting from Maras Powder use.

Case Report

A 48-year-old male patient was brought to the emergency department with complaints of syncope and confusion. Apart from a history of coronary angiography three years ago, he had no other medical history and was using a beta-blocker and acetylsalicylic acid (100 mg). Upon admission, his general condition was moderate, he was confused, and his Glasgow Coma Scale (GCS) score was assessed as 12. During the intervention, he exhibited agitation and purposeless movements.

On physical examination, there was no nuchal rigidity, and Kernig and Brudzinski tests were negative. His body temperature was 36.6°C, heart rate was 78 bpm, respiratory rate was 15 breaths per minute, and blood pressure was 105/60 mmHg. His electrocardiogram (ECG) showed a sinus rhythm. A brain computed tomography (CT) scan revealed no signs of acute bleeding, and diffusion

MRI showed no findings suggestive of acute ischemic stroke.

Image 1: Nicotiana Rustica (Wikipedia contributors. Nicotiana rustica (Aztec tobacco, wild tobacco) [Internet]. [cited 2025 Feb 2].

Laboratory tests showed a pH of 7.24, PCO□ of 45 mmHg, HCO□ of 19.5 mmol/L, and lactate of 7.70 mmol/L. Aspartate aminotransferase (AST), alanine aminotransferase (ALT), creatinine, blood urea nitrogen (BUN), and troponin-I levels were within normal limits. His glucose level was 100mg/dl. No findings suggestive of anemia were detected.

A history obtained from the patient's relatives revealed that he had been using Maras Powder regularly for the past two weeks to quit smoking. The patient was started on intravenous 0.9% NaCl (1000 mL), 5 mg midazolam, and nasal oxygen at 2 L/min. He was monitored in the emergency department and continued receiving supportive treatment.

Three hours after admission, arterial blood gas analysis showed pH: 7.37, PCO□: 42 mmHg, HCO□: 23 mmol/L, and lactate: 0.78 mmol/L. As his blood gas values returned to normal, his confusion improved, and his GCS score increased to 15. After 24 hours of observation, he was discharged from the emergency department.

Discussıon:

Nicotine intoxication has become more common in recent years, particularly with the increasing use of e-cigarettes, which often include oral nicotine pouches. It exhibits a bimodal distribution, with accidental ingestion being more frequent in children under the age of 10, while intentional use for suicide is more common in adults (8). Similarly, in our country, cases of Maras Powder intoxication among pediatric patients presenting to the emergency department, particularly in Kahramanmaraş and its surrounding areas, have been reported as accidental consumption (7). In our case, however, intoxication resulted from the unintentional overuse of Maras Powder due to a lack of awareness regarding its high nicotine content.

Acute nicotine intoxication presents with a biphasic pattern due to its ability to both stimulate and inhibit cholinergic receptors. Initially, it may manifest with symptoms such as excessive salivation, nausea, vomiting, diarrhea, and sweating. Additionally, vasoconstriction can lead to pallor and increased blood pressure. Tachycardia and, in some cases, cardiac arrhythmias (such as atrial fibrillation) may also occur (9). After a certain period, nicotine-induced desensitization of acetylcholine receptors can result in confusion, somnolence, muscle weakness, and, in severe toxicity, respiratory depression and cardiac arrest (10). The literature also reports delirium associated with oral nicotine gum use and chest pain due to nicotine-induced coronary vasospasm (11). In cases of Maras Powder intoxication observed in the pediatric population in our country, symptoms such as vomiting, somnolence, metabolic acidosis, and convulsions have been reported (7). In our patient, the presence of hypotension and confusion suggested that these symptoms were primarily due to the acetylcholine-related effects of Maras Powder.

Nicotine's half-life varies depending on factors such as gender and genetic influences. It ranges from approximately 90–150 minutes in non-smokers and 100–200 minutes in smokers (12). Consequently, symptoms tend to resolve quickly, with most patients achieving full recovery within 12 hours (9). In our case, symptom improvement and normalization of blood gas levels were observed within

3 hours, and the patient was discharged in a fully recovered state at the 24-hour mark.

Image 2: Maras Powder (Maras Avucumda. Maraş Otu [Internet]. [cited 2025 Feb 2])

Cotinine, a metabolite of nicotine, is considered the most sensitive and specific biomarker for assessing nicotine exposure. It can be measured in blood, saliva, or urine and has a longer half-life than nicotine (13). However, studies in the literature have shown that cotinine levels do not always correlate with clinical presentation. This discrepancy may be due to the varied causes of nicotine intoxication or liver damage in severe cases affecting nicotine metabolism (10). Additionally, cotinine levels can increase with chronic exposure, meaning that elevated levels do not necessarily indicate acute intoxication. Therefore, routine cotinine measurement in acute poisonings remains debatable (9). There is no specific antidote for Maras Powder/nicotine toxicity. Treatment is primarily symptomatic and supportive. The first priority is to secure the airway and provide respiratory support. Atropine is used to manage parasympathetic symptoms such as excessive salivation, wheezing, and bradycardia. In cases of severe poisoning, endotracheal intubation may be required for airway protection and ventilation support. Seizures should be treated with benzodiazepines. Hypotension is initially managed with fluid boluses and 0.9% NaCl infusion; if unresponsive to volume resuscitation, a vasopressor such as norepinephrine should be administered. Cardiac arrhythmias should be treated according to standard advanced cardiac life support (ACLS) protocols. Nicotine elimination is accelerated in acidic urine, but due to the risks outweighing the benefits, this method is not recommended (9,14). Oral rinsing with water may be advised upon initial presentation. In pediatric patients or cases involving suicidal ingestion of Maras Powder, gastric lavage and activated charcoal administration within the first hours may be beneficial (15). However, in adult intoxication cases, nicotine absorption occurs primarily through the oral mucosa, making these interventions therapeutically ineffective. In our case, due to the patient's agitation upon arrival, intravenous administration of 5 mg midazolam was required.

This case report is, to our knowledge, the only documented case of a smokeless tobacco product or Maras Powder intoxication in the adult population in the literature. Given the broad clinical spectrum of Maras Powder-related intoxication, it should be considered with a high index of suspicion in regions where this product or similar products are commonly used, and patient history should be carefully assessed. Further research is needed to determine the exact amount of Maras Powder consumption that leads to intoxication, its effects on nicotine and metabolite levels, and the rate at which these changes occur. Additionally, studies should investigate the potential impact of other components in Maras Powder, such as wood ash, on nicotine metabolism.

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Portal Vein Thrombosis in a Patient Presenting with Abdominal Pain: A Case Report

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Introduction

Abdominal pain is one of the most common reasons for visits to the emergency department, encompassing a broad range of underlying etiologies. Therefore, the evaluation of abdominal pain requires a meticulous clinical approach and diagnostic process. One of the rare but potentially serious causes of acute abdominal pain is portal vein thrombosis (PVT), a condition characterized by the formation of thrombus within the portal vein. It typically results from disturbances in portal venous blood flow due to mechanical obstruction, inflammation, or a hypercoagulable state.

The clinical spectrum of PVT ranges from asymptomatic presentations to severe abdominal pain, bowel ischemia, or portal hypertension. Early diagnosis and treatment of acute PVT are crucial to prevent intestinal necrosis and other life-threatening complications. The etiology of PVT is diverse, involving inherited or acquired thrombophilias (e.g., Factor V Leiden mutation, protein C or S deficiency), intra-abdominal infections (e.g., appendicitis, diverticulitis), malignancies, and prior surgical interventions.

With the widespread use of imaging modalities and advancements in diagnostic technologies, earlier and more accurate detection of PVT has become possible. However, in patients presenting with abdominal pain, thorough evaluation of the underlying cause and a multidisciplinary approach remain essential. In this report, we present the case of a patient admitted to the emergency department with abdominal pain who was subsequently diagnosed with portal vein thrombosis, and we discuss the clinical features, diagnostic process, and treatment strategies involved.

Case Presentation

A 32-year-old male patient with no known chronic illnesses presented to our emergency department with complaints of persistent abdominal pain lasting for one week. On admission, he was alert, oriented, and cooperative, with stable vital signs. Physical examination revealed normal breath sounds, a soft and non-tender abdomen, and no signs of guarding or rebound tenderness. Electrocardiography demonstrated a sinus rhythm at 77 beats per minute. Laboratory results did not reveal any acute pathological findings.

Abdominal ultrasonography showed mildly heterogeneous liver parenchymal echotexture and a main portal vein diameter of 14 mm. No Doppler flow was visualized within the portal vein, and a thrombus obstructing the lumen was suspected. Absence of flow in the portal vein and its branches was noted, consistent with the diagnosis of portal vein thrombosis. The patient was subsequently referred to gastroenterology, cardiovascular surgery, and general surgery departments for further evaluation. He was admitted to the gastroenterology service for follow-up and treatment.

Discussion

Portal vein thrombosis (PVT) is an uncommon but clinically significant condition characterized by thrombus formation within the portal venous system. In this case, PVT was diagnosed in a young adult with no known comorbidities who presented to the emergency department with abdominal pain. This case highlights that due to its rarity in differential diagnosis, PVT can often be overlooked. However, a multidisciplinary approach and advanced imaging techniques play a vital role in early diagnosis and treatment planning.

Although most cases of abdominal pain in the emergency setting are caused by benign conditions, the possibility of underlying serious and potentially life-threatening causes must always be considered—especially when the pain is unexplained or persistent. In this case, the absence of pathological findings on abdominal examination and normal laboratory tests made it difficult to initially suspect PVT. However, ultrasonography and Doppler studies revealed hallmark findings, such as enlargement of the portal vein, absence of flow, and the presence of an intraluminal thrombus. Ultrasonographic assessment, due to its non-invasive nature and

widespread availability, remains the first-line imaging modality for early detection of PVT in emergency settings.

The etiology of PVT is often multifactorial, commonly associated with genetic or acquired thrombophilic disorders, malignancy, portal hypertension, intra-abdominal inflammation, or prior surgical history. However, as in the present case, the absence of known risk factors may complicate the diagnostic process. In such situations, further evaluation including genetic testing and thrombophilia screening is necessary to determine the underlying cause and assess long-term prognosis. In young patients without known comorbidities, unexplained PVT should prompt investigation for hypercoagulable states.

This case also underscores the importance of a multidisciplinary approach. Following the diagnosis of PVT, the patient was evaluated collaboratively by specialists in gastroenterology, cardiovascular surgery, and general surgery, and was admitted for appropriate treatment. This coordinated care was crucial in preventing complications and improving the patient's clinical outcome. Anticoagulation therapy constitutes the cornerstone of treatment, aiming to resolve the thrombus and restore portal venous flow. In selected cases with complications, surgical or interventional procedures may also be necessary.

In conclusion, portal vein thrombosis is a rare yet critical diagnosis that should be considered in the differential workup of patients presenting with abdominal pain. Early diagnosis and prompt treatment are essential for preventing complications and improving long-term quality of life. Emergency physicians should maintain a high level of suspicion for PVT, especially in patients with unexplained abdominal pain and unremarkable physical or laboratory findings, and advanced imaging should be employed when appropriate. This case demonstrates the value of early recognition and multidisciplinary management in improving outcomes for patients with PVT.

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Sudden Development Of Intraparenchymal Hemorrhage In A Middle- Aged Patıent With A Normal Neurologıcal Examınatıon

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Introductıon

Intracerebral hemorrhage (ICH) is a subtype of stroke characterized by hematoma formation within the brain parenchyma, with or without extension into the ventricles. Non-traumatic ICH accounts for 10–15% of all stroke cases and is associated with high morbidity and mortality.

Risk factors for ICH include chronic hypertension, amyloid angiopathy, anticoagulant use, and vascular malformations. Brain injury is typically classified as primary (direct damage from the hematoma) or secondary (resulting from complications such as increased ICP).

Management of ICH ranges from medical treatment to surgical hematoma evacuation, with ongoing research into less invasive therapies. Non-traumatic ICH can be classified as either primary or secondary. Primary hemorrhages (85% of ICH cases) are primarily related to chronic hypertension or amyloid angiopathy. Secondary hemorrhages are associated with bleeding disorders (iatrogenic, congenital, or acquired), vascular malformations, neoplasms, hemorrhagic transformation of ischemic stroke, and drug use.

Primary or spontaneous ICH accounts for over 85% of hemorrhagic strokes. Diagnosis is often made by exclusion, supported by a history of chronic hypertension, advanced age, and the location of the hematoma. In patients with chronic arterial hypertension, lipohyalinosis and degenerative changes in penetrating arterioles can lead to Charcot-Bouchard aneurysms. More than 60% of primary hemorrhages are related to hypertension, commonly occurring in the posterior fossa, pons, basal ganglia, and thalamus.

Lobar hemorrhages in elderly patients are typically due to cerebral amyloid angiopathy, a degenerative disease characterized by

amyloid deposition in vessel walls and a possible link to apolipoprotein E alleles.

ICH can also result from structural abnormalities such as vascular anomalies or malignancies (secondary ICH). Vascular lesions include AVMs, cavernous angiomas, cerebral aneurysms, and arteriovenous fistulas, which often affect younger, otherwise healthy individuals. Hematomas can also develop due to hemorrhagic transformation of recent ischemic infarcts or neoplastic lesions. Both congenital and acquired bleeding disorders are significant contributors to ICH. The increasing use of anticoagulant and antiplatelet therapies has also led to a rise in ICH cases.

Studies have identified various risk factors for ICH, classified as modifiable and non-modifiable. Non-modifiable factors include non-white ethnicity, advanced age, familial apolipoprotein syndromes, and male sex. Radiological features of cerebral amyloid angiopathy increase the risk of lobar and recurrent ICH. Poorly controlled or untreated hypertension doubles the risk of ICH in elderly individuals. Modifiable risk factors include substance abuse (alcohol, nicotine, cocaine).

Globally, the incidence of both ischemic and hemorrhagic stroke was estimated at approximately 33 million in 2010. Hemorrhagic stroke accounted for one-third of all strokes but caused more than half of all stroke-related deaths. While the global incidence of stroke is approximately 20 per 100,000 people annually, ICH rates are twice as high in low- and middle-income regions compared to high-income countries, largely due to a lack of preventive education and limited access to healthcare. Stroke remains the fourth leading cause of death in the U.S., with ICH accounting for 20% of all cerebrovascular events. ICH is more frequently diagnosed in individuals over 55 years of age and is more common in African and Asian populations. In Japan, the ICH incidence reaches 55 per 100,000, likely due to high alcohol consumption and prevalent hypertension.

Case Report

A 50-year-old male patient with no known medical history presented ambulatory to the emergency department's green zone with complaints of headache and dizziness. The patient also reported experiencing a presyncope episode approximately 30 minutes prior, accompanied by involuntary body contractions resembling a seizure. This episode lasted for approximately 5–10 minutes and did not result in loss of consciousness. Vital signs revealed markedly elevated blood pressure (192/110 mmHg). Physical examination showed no pathological findings. Pupils were isochoric, consciousness was intact, GCS was 15, and muscle strength was normal in all four extremities. Urgent brain imaging was requested. A cranial CT scan revealed a large intraparenchymal hemorrhage in the right parietal lobe of the cerebral hemisphere. The patient was diagnosed with hypertensive intraparenchymal hemorrhage and was promptly referred to neurology and admitted to the stroke intensive care unit.

Dıscussıon

This case describes a 50-year-old male with no known medical history. One of the most striking aspects of this case is that the patient had fully preserved consciousness and a completely normal neurological examination. He presented in a stable condition, ambulatory, and reported only non-specific mild headache and dizziness. At first glance, these symptoms might have been considered indicative of a tension-type headache or a mild systemic issue. However, the most critical detail was the seizure-like event that occurred before the presentation.

If this seizure-like episode had been overlooked or not reported by the patient, the evaluation would have been based solely on headache symptoms, potentially missing a life-threatening hemorrhagic stroke. This could have led to irreversible consequences for the patient and a critical oversight for the medical team.

This case underscores that a neurological examination alone is insufficient and that a detailed patient history is essential. Patients may not always report clear or specific symptoms, and in neurological diseases, seemingly minor complaints may mask serious conditions. Therefore, thorough history-taking is crucial, and even subtle clinical clues should be carefully evaluated.

Conclusıon

Hemorrhagic stroke carries a high risk of morbidity and mortality. In patients with preserved consciousness and a normal neurological examination, it is crucial to identify underlying causes. Advanced diagnostic evaluations should not be overlooked to ensure accurate diagnosis and appropriate treatment.

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A Hidden Brake Lever in the Leg: Unexpected Foreign Body

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Introductıon

Penetrating injuries and suspected foreign bodies in the soft tissue are common reasons for emergency room visits.In addition to its high incidence, retained foreign bodies are responsible for 14% of medical lawsuits and 5% of legal settlements. (1) The search for embedded soft tissue foreign bodies relies heavily on radiological imaging. Detection of these objects is critical because foreign bodies can cause serious infection, abscess, and/or tissue loss.(2) In this case, we present a patient who had a bicycle brake lever without his knowledge and it was removed from his thigh in emergency settings.

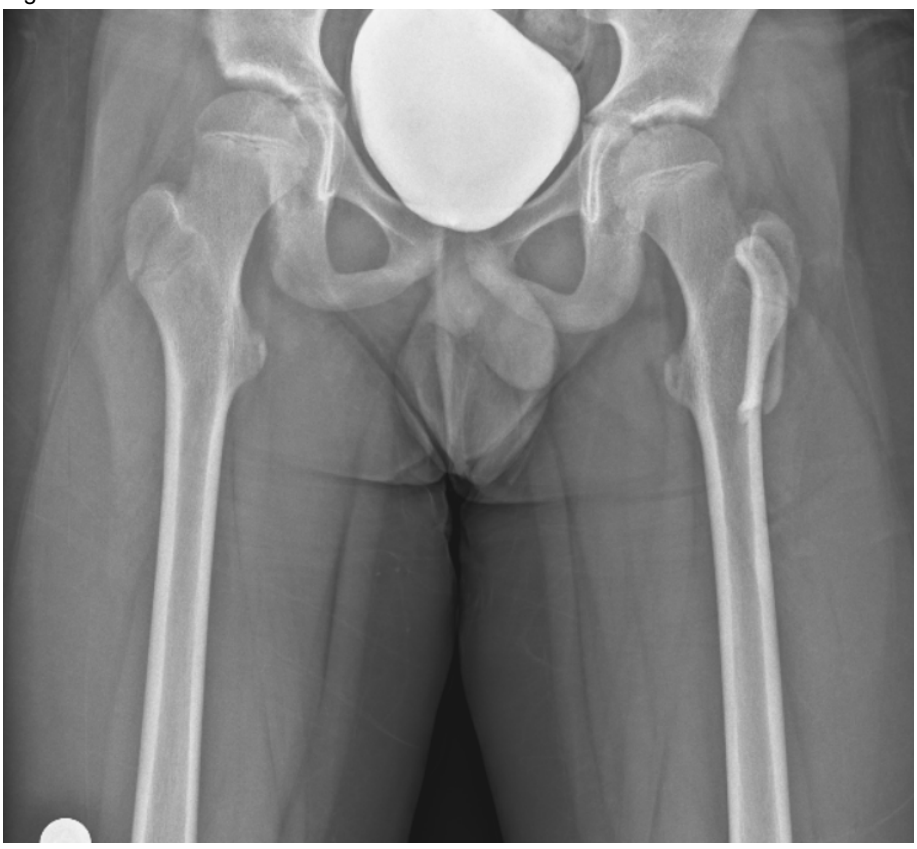
Case Report

An 11-year-old male patient was brought in to ED after falling while riding a bicycle. The patient, who had no head trauma, came with only a description of pain in the leg.

The patient had a 1 cm incision on the anteromedial area of the left thigh and did not describe any foreign body involvement. It was assumed that only a simple incision had occurred during the trauma.

Physical Examination: Inspection revealed a 1 cm wide incision on the anteromedial area of the left thigh. The patient had clear peripheral pulses and no neurovascular deficit was observed and the musculoskeletal examination was evaluated as normal. X-rays were taken for the patient who described tenderness on the midline of the femur on palpation but no foreign body was palpable. A radiopaque foreign body was seen at the level of the greater trochanter on the X-ray. (Fig.1)

Figure 1



CT angiography was performed to evaluate for vascular-nerve injury.

CT Angiography Report: Hyperdense appearance compatible with foreign body was observed on the anterior neighborhood of muscle planes located approximately 3 cm deep under the skin at the level of the femoral head in the anterior left thigh. Millimetric air densities accompany this section. No findings in favor of hematoma were detected in this section. (Fig2)

The patient was consulted with an orthopedic specialist. The foreign body was removed under local anesthesia in the emergency department in collaboration with the orthopedic specialist.

After the foreign body was removed, a control radiograph was taken of the patient and no foreign body was seen. (Fig3)

The patient was discharged with the recommendation of an orthopedic outpatient clinic check-up.

Dıscussıon

Foreign bodies should be removed during the primary wound management phase whenever possible. Metallic fragments can be

detected and localized by conventional radiography, computed tomography (CT), and ultrasonography (3)(4). Foreign bodies can be of various shapes and sizes and usually consist of wood, glass, and metal splinters (5).

Figure 2

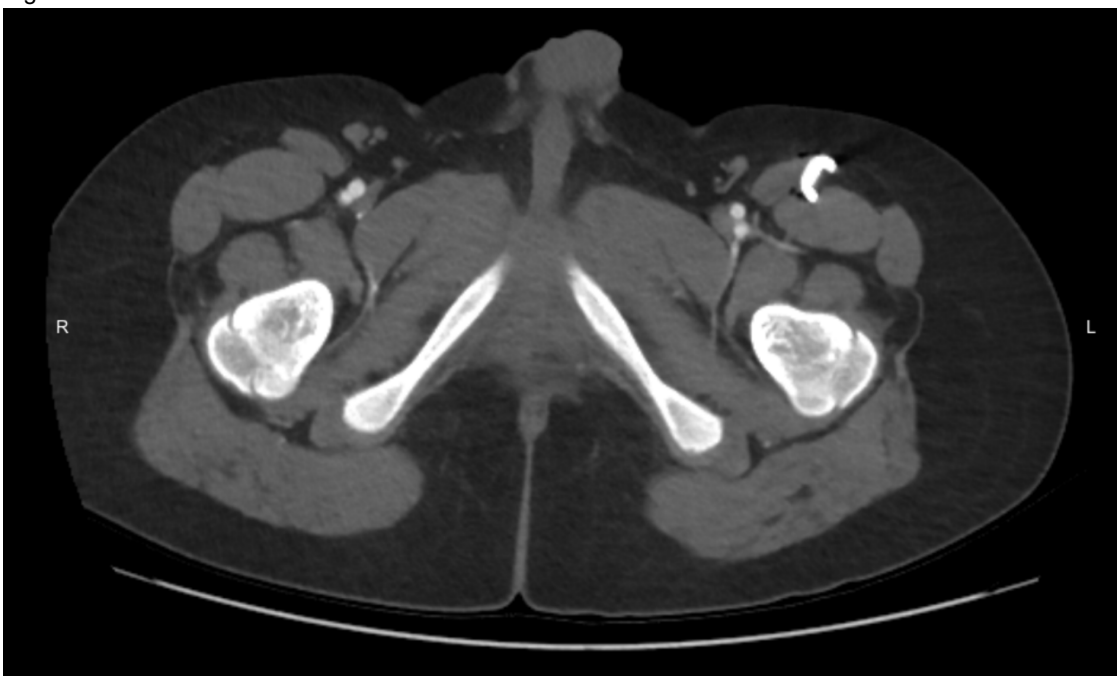
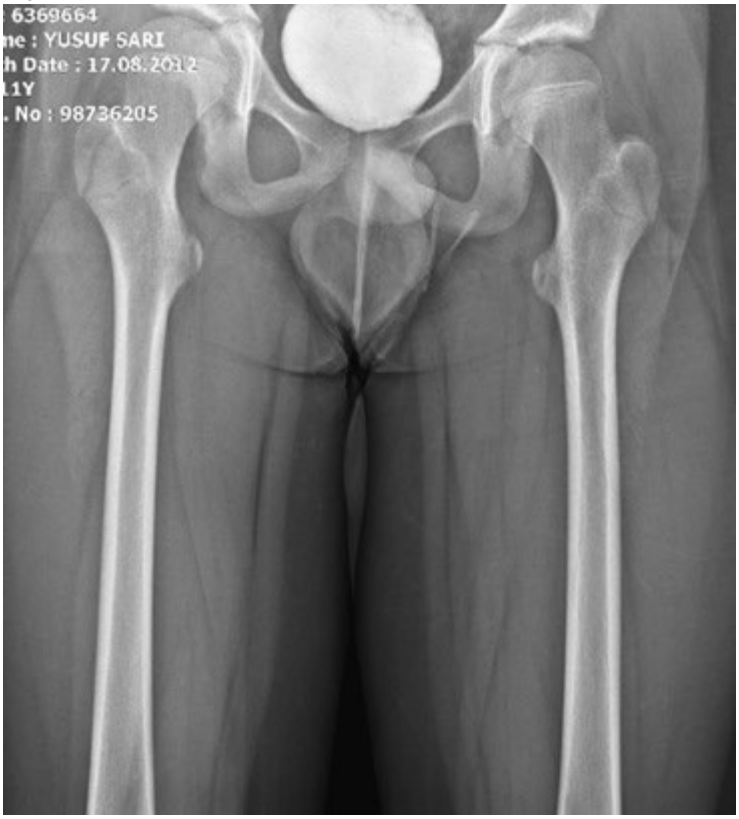


Figure 3



Foreign bodies are one of the most common reasons for early or late emergency room visits (6). Foreign bodies can be found in soft tissue accidentally or iatrogenically. (7) Ultrasonography may be preferred for imaging non-opaque materials. In our case, the opacity of the foreign body helped us reach a conclusion by taking X-rays (8). However, in recent years, it has been observed that ultrasonography, which has become widespread in diagnosis, has replaced radiography. (8),(9) In addition, magnets have also been

used in the literature for metallic foreign bodies. Magnets can be used not only for the removal of foreign metallic objects but also for detection in the emergency room in the presence of a suspicious foreign object. (10) When using magnets, the risk of vascular and nerve injuries should be kept in mind and they should be generally preferred for superficial injuries.

Conclusıon

Although emergency room patients can often provide guidance regarding their condition, the patient in this case was unable to comprehend what had occurred due to the immediate impact of the accident. The individual was visibly astonished when the foreign body was extracted. This case underscores the crucial role of physical examination and imaging, highlighting their significance alongside patient history in accurate diagnosis and management.

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8451

A case of hemothorax due to aortic injury following blunt trauma

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Introduction

Blunt traumatic aortic injury (BTAI) is a rare but fatal complication of thoracic trauma. Although it occurs in approximately 1% of trauma cases, it is the second most common cause of death (1-4). A large proportion of patients with BTAI (up to 80%) die without receiving adequate treatment (5). BTAI is most commonly caused by sudden deceleration, and motor vehicle accidents are the predominant identified mechanism (2, 3, 5-7). The age distribution of BTAI is wide, with a mean age of 35 to 45 years, and there is a male predominance (1-3, 7).

A four-degree classification system is published for traumatic aortic injuries (8). (Table 1)

With the development of thoracic endovascular aortic repair (TEVAR) and the advancement of devices over the last decade, endovascular treatment has replaced open surgery and has resulted in lower mortality rates (9–12). The 2017 European Society for Vascular Surgery (ESVS) guidelines for the management of descending thoracic aortic diseases recommend TEVAR as the first-line treatment for BTAI in patients with favorable anatomy, except for minimal aortic injuries presenting with intimal tears that can be managed conservatively (6).

Guidelines also recommend intervention within 24 hours if high-risk factors such as a large mediastinal hematoma, left-sided hemothorax, aortic coarctation, pseudoaneurysms, abnormal outer wall contours, systolic blood pressure <90 mmHg, or traumatic brain injury are present, otherwise delayed TEVAR is recommended (13).

Case Report

A 25-year-old male driver applied in emergency service for a traffic accident. The four-wheeled car lost control while turning at an intersection, resulting in a frontal impact.

The patient has no known additional disease or history of medication use. There is no alcohol or drug use.

General condition is good. GCS is 15. Neurological examination is normal.

On physical examination, there are severe abrasions on the anterior thorax. There is a left femur deformity. There are superficial cuts and abrasions on the forehead and various parts of the head.

Vital signs TA 90/60, SpO2 92, pulse 115.

Laboratory findings: Hb 14.2, ALT 125, AST 162, other routine tests are normal.

In the imaging tests performed, Brain CT is normal. Thorax CT shows aneurysm and rupture at the level of the aortic arch, accompanied by left hemothorax and hemomediastinum. (Figure 1-2) Abdominal CT shows laceration in the right kidney.

The patient was admitted to cardiovascular intensive care for TEVAR.

Figure 1

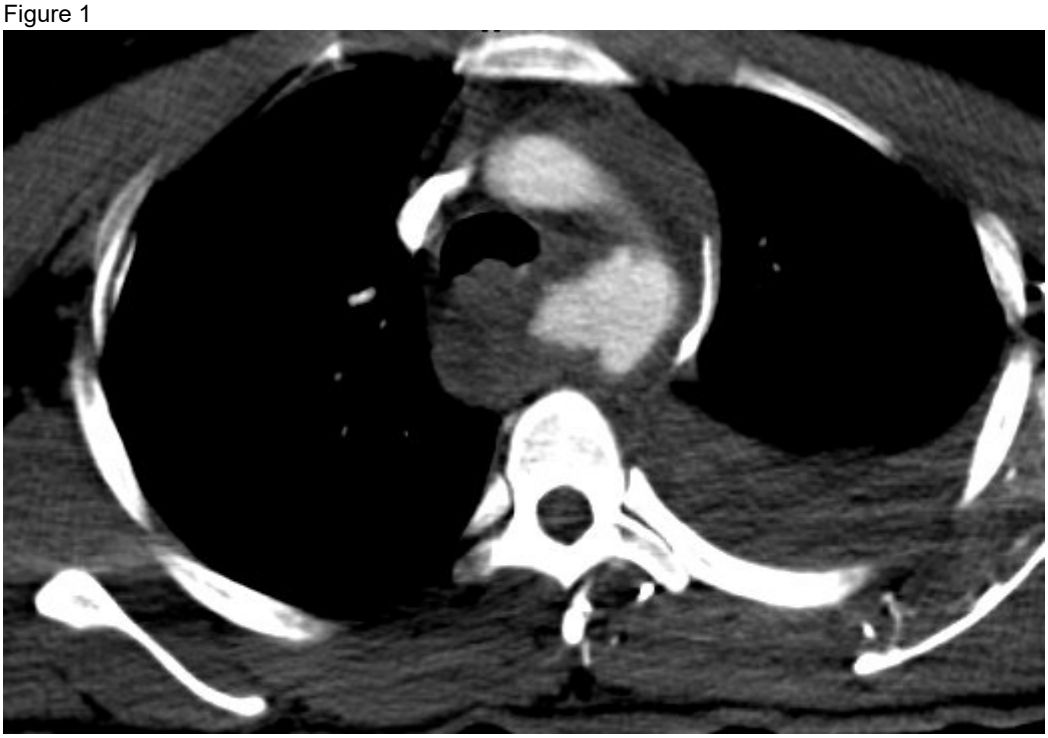

Figure 2

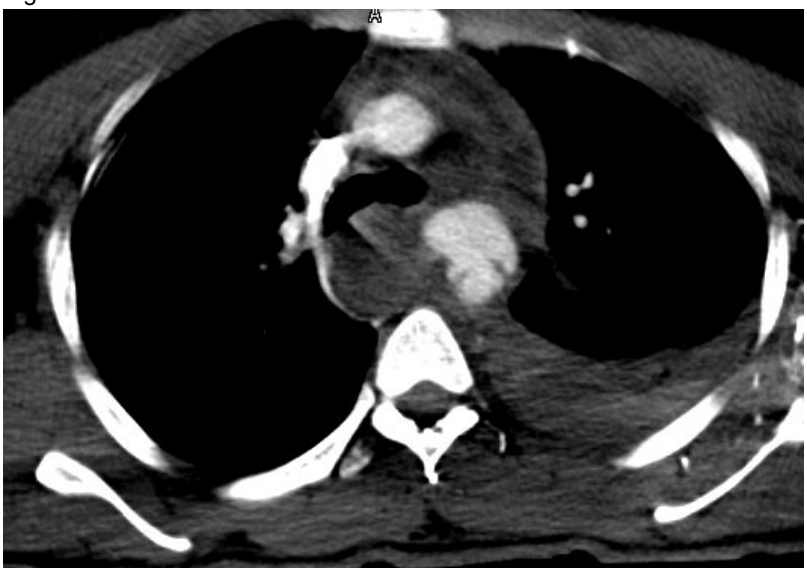

Table 1	
1. Degree	Intimal Injury
2. Degree	Intramural Hematoma
3. Degree	Pseudoaneurysm
4. Degree	Rupture

Conclusion

Hemothorax usually occurs due to lung injuries. In cases of blunt trauma with hemothorax without rib fractures, aortic injury should be considered.

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8526

A cerebrovascular event mimic in the emergency room: vitamin B12 deficiency

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[1]S.B.Ü Erzurum Şehir Hastanesi Acil Tıp Ana Bilim Dalı

1.Introduction and Purpose

Vitamin B12 is a water-soluble vitamin that is necessary for cell division and proliferation, as a coenzyme in important reactions in the body, and is involved in DNA production. Vitamin B12 works together with folic acid in many processes including DNA synthesis, erythrocyte production and myelin sheath synthesis (1). The most common finding in vitamin B12 deficiency is anemia, and fatigue, palpitations and shortness of breath may be observed in patients following anemia. In vitamin B12 deficiency, neurologic symptoms may also be observed in addition to anemia symptoms (2). Neuropathy is one of the most common complications. Paresthesia and numbness primarily start in the feet and legs, accompanied by loss of reflexes, superficial sensory deficit and loss of vibration sense. Later, similar sensory changes start in the hands and weakness in the distal leg muscles is added (3). In this case, we aimed to emphasize the neurologic manifestations of vitamin B12 deficiency.

2.Materials and Methods

A 22-year-old male patient was admitted to the emergency department with complaints of numbness in the right face and right arm and lisping in speech. The patient had no history of any disease and his vital signs were normal and stable. Neurologic examination revealed no motor or sensory deficit and no nuchal rigidity. Minimal ecchymotic areas were observed on the anterior chest wall and dorsal surface of the right arm. Neurologic imaging of the patient revealed no intracranial pathology on brain tomography, and diffusion MR imaging showed no restricted diffusion in the cerebral and cerebellar hemispheres on DAG ADC mapping. Laboratory values of the patient were as follows: WBC: 4.12 10^9/L, HGB: 9.0 g/dl, MCV: 101.0 Fl, PLT: 18 10^9/L and pancytopenia was present. In addition, LDH value was 750 U/L and indirect dominance of bilirubin was increased. A peripheral smear was sent and the patient was consulted to the hematology unit. Hypersegmented neutrophils (figure-1) and macrocytes were observed in the peripheral smear and the patient was interned to the internal service with the diagnosis of megaloblastic anemia.

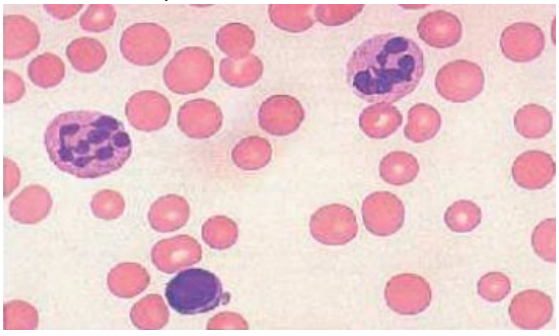

Figure 1: hypersegmented neutrophils seen in peripheral smear

3.Results and Conclusion

Vitamin B12 deficiency may present with neurologic, hematologic, gastrointestinal or psychiatric symptoms. Neurologic findings may

include peripheral neuropathy, myelopathy (subacute combined degeneration), optic neuropathy, encephalopathy, eye movement disorders and extrapyramidal syndrome (4). This may lead to evaluation of this patient group as cerebrovascular events and incorrect treatment regimens in crowded emergency departments. In patients in whom pancytopenia is detected as a result of hemogram performed for any reason, it is extremely important that a peripheral smear is performed after a careful anamnesis and physical examination and further investigations are planned and performed according to the results.

Keywords: Neuropathy, Pancytopenia, Vitamin B12 deficiency

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8685

The deadly consequence of uncontrolled diabetes: emphysematous pyelonephritis

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Introductıon

Emphysematous pyelonephritis (EPN) is a rare, necrotizing, and suppurative kidney infection characterized by gas formation within the intrarenal or perirenal regions, with a high mortality rate. Diabetes mellitus, immunosuppression, obstructive uropathy, and a history of urinary tract infection are risk factors for emphysematous pyelonephritis. In the pathogenesis of emphysematous pyelonephritis, four main factors may play a role, including gas-producing bacteria, high tissue glucose levels, impaired tissue perfusion, and an inadequate immune response. Particularly in diabetic patients, high blood glucose levels create a more suitable environment for gas formation. The fact that anaerobic bacteria contribute to gas production is significant in the selection of anaerobic antibiotic therapy. Among the gas-producing microorganisms, Escherichia coli is the most commonly identified pathogen.The symptoms of emphysematous pyelonephritis are non-specific, with flank pain, nausea, and fever being the most common manifestations. However, emphysematous pyelonephritis may present insidiously and progress to sepsis and septic shock. Although there are no specific distinguishing features in the physical examination compared with other urinary tract infections, costovertebral angle tenderness is a common finding, and in severe cases, crepitus may be detected due to the spread of gas into the subcutaneous tissue.Emphysematous pyelonephritis is diagnosed through imaging. Computed tomography is the most valuable diagnostic tool for emphysematous pyelonephritis. It is undeniably the best imaging modality for detecting renal gas and its spread to surrounding tissues.Treatment options for emphysematous pyelonephritis include medical therapy, drainage procedures such as ureteral stenting or percutaneous nephrostomy, and surgical treatment with nephrectomy. Antibiotic selection should target common bacteria such as Escherichia coli, Klebsiella pneumoniae, and Proteus mirabilis. Patients with kidney stones, renal colic, and flank pain frequently visit the emergency departments. With this case report, we aim to highlight that in patients presenting with these symptoms, especially those diagnosed with diabetes mellitus, emphysematous pyelonephritis should be considered as a potential diagnosis.

Case

A 56-year-old female patient presented to the emergency department with complaints of right flank pain and nausea persisting for approximately 10 days. During this period, she had previously visited the emergency department with the same complaints. At that time, no specific findings were detected in her physical examination or laboratory tests, and she was diagnosed with renal colic and discharged.The patient's medical history revealed a diagnosis of type 2 diabetes mellitus. On physical examination, tenderness was noted in the right costovertebral angle. Her vital signs were as follows: body temperature of 36.8°C, blood pressure of 135/72 mmHg, and pulse rate of 85 beats per minute. Laboratory tests showed a white blood cell count of 11.92/mm³, creatinine level of 0.65 mg/dL, C-reactive protein level of 71 mg/L, and blood glucose level of 595 mg/dL. Urine microscopy revealed abundant leukocytes and erythrocytes.Computed tomography (CT) imaging demonstrated focal caliectatic dilations at the upper, middle, and lower poles of the right kidney, with air images observed within the calyces. Additionally, a hazy appearance was noted in the perirenal fat tissues surrounding the right kidney. The patient was diagnosed with emphysematous pyelonephritis and was consulted with the urology department for further evaluation and treatment. The patient was subsequently hospitalized, and antibiotic therapy was initiated.

Dıscussıon

Emphysematous pyelonephritis is a life-threatening condition if not diagnosed and treated early. The treatment of emphysematous pyelonephritis depends on the extent of kidney involvement and whether renal function is affected, with both medical and surgical approaches available. According to the Huang and Tseng classification, emphysematous pyelonephritis can be categorized into four classes:

Class 1: Gas is present within the kidney but has no external extension.
Class 2: Gas extends into the perinephric region.
Class 3A: Renal and perirenal abscess formation is present.

Class 3B: The infection extends into the pararenal space.
Class 4: Bilateral kidney involvement and renal dysfunction are present.

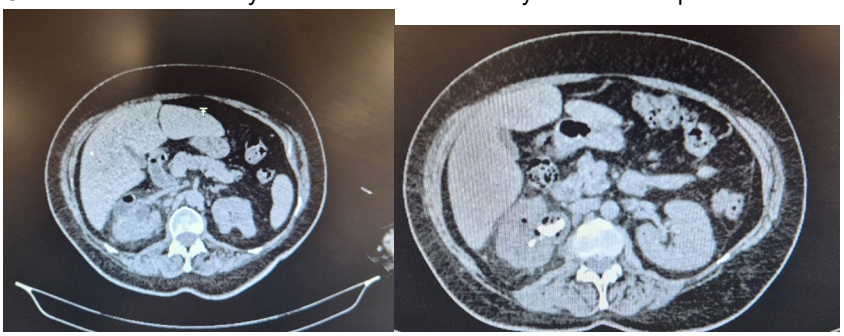

Figure 1: Air images in the right kidney on non-contrast abdominal CT.

The general approach to treatment includes broad-spectrum intravenous antibiotics, glycemic control, fluid and electrolyte balance management, and supportive treatment for hypertension and shock if necessary. Interventional procedures such as percutaneous drainage may be required, and in severe cases, nephrectomy may be necessary.A study conducted by Joris et al. reported a 70% success rate with medical treatment, while interventional treatment had a 30% success rate. In Class 1 and Class 2 cases, intravenous antibiotic therapy is generally effective, whereas in more advanced stages, percutaneous drainage may be necessary. In severe cases, nephrectomy is an option. For Class 3 or 4 cases, where gas formation extends beyond the renal capsule, abscess formation is present, or there is bilateral involvement, treatment decisions should be based on risk factors such as thrombocytopenia, acute renal dysfunction, shock, and altered mental status. If a patient presents with two or more risk factors, nephrectomy is preferred over conservative management. In our case, air images were observed within the calyces of the right kidney, but renal function remained preserved. The patient's general condition was moderate to good, and his vital signs were stable. Therefore, conservative management with antibiotic therapy was chosen. Emphysematous pyelonephritis is a life-threatening condition with a mortality rate ranging from 40% to 90%. The most critical factors influencing mortality are early diagnosis and treatment. Therefore, even in cases where stone-related follow-up seems sufficient and poses minimal risk, imaging should not be avoided in patients with predisposing factors, particularly diabetic patients and female patients, to ensure early detection and appropriate intervention.

Conclusıon

This case emphasizes the importance of the early diagnosis and treatment of emphysematous pyelonephritis. Particularly in patients with a diagnosis of diabetes mellitus, this condition may initially be misinterpreted as renal colic and overlooked. However, it should not be disregarded. With early diagnosis, even medical treatment can be effective, whereas delayed recognition may lead to the need for nephrectomy. Emphysematous pyelonephritis can result in acute kidney failure, sepsis, and septic shock, ultimately leading to death.

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8739

Toxic Hepatitis Following Oral Ingestion of Ferula communis (Giant Fennel): A Case Series

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Erzurum şehir hastanesi acil tıp kliniği

Introduction

Toxic hepatitis is an inflammatory liver injury resulting from exposure to various chemical agents, drugs, herbal products, or toxins. In this report, we present cases of toxic hepatitis associated with ingestion of Ferula communis (Figure 1), commonly known as giant fennel. This plant, native to the Mediterranean region, is traditionally consumed for its presumed medicinal and aphrodisiac properties. However, some species contain hepatotoxic compounds. Patients typically present with symptoms such as fatigue, anorexia,

nausea, vomiting, and jaundice. In this case series, we aim to discuss the clinical spectrum of Ferula communis poisoning in three patients who presented with nausea, vomiting, and headache following ingestion of the plant, and subsequently developed hepatotoxicity.

Case Series

Three members of the same family consumed a plant they identified as giant fennel (Ferula communis), which they had foraged one day prior to presentation. All three presented to the emergency department with complaints of nausea, vomiting, and headache.

Case 1: A 44-year-old male with no known comorbidities. Vital signs on admission: BP 120/74 mmHg, HR 90 bpm, SpO_2 99%, RR 17/min, Temp 36.3°C. Laboratory findings: ALT 113 IU/L, AST 126 IU/L, GGT 123 IU/L, ALP 139 IU/L, CK 216 U/L, LDH 307 U/L, PT 13 s, INR 0.98.

Case 2: A 67-year-old male with a known history of hypertension. Vital signs: BP 170/114 mmHg, HR 110 bpm, SpO_2 93%, RR 27/min, Temp 36.1°C. Laboratory tests showed ALT 441 IU/L, AST 607 IU/L, GGT 19 IU/L, ALP 126 IU/L, Total Bilirubin 1.3 mg/dL, Direct Bilirubin 0.4 mg/dL, CK 95 U/L, LDH 636 U/L, PT 19.5 s, INR 1.51.

Case 3: A 48-year-old female with no known comorbidities. Vital signs: BP 108/67 mmHg, HR 95 bpm, SpO_2 99%, RR 26/min, Temp 36.7°C. Laboratory results: ALT 495 IU/L, AST 438 IU/L, GGT 61 IU/L, ALP 83 IU/L, Total Bilirubin 1.0 mg/dL, Direct Bilirubin 0.5 mg/dL, CK 319 U/L, LDH 750 U/L, PT 17.9 s, INR 1.38.

Table 1: Clinical and Laboratory Data of the Cases

Parameter	Case 1 - 44 male	Case 2 - 67 male (HTN)	Case 3 - 48 female
Blood Pressure (mmHg)	120/74	170/114	108/67
Heart rate (bpm)	90	110	95
SpO□ (%)	99	93	99
Respiratory rate (/min)	17	27	26
Body Temperature (°C)	36.3	36.1	36.7
ALT (IU/ml)	113 (0–50)	441 (0–50)	495 (0–50)
AST (IU/ml)	126 (0–50)	607 (0–50)	438 (0–50)
GGT (IU/ml)	123 (12–64)	19 (12–64)	61 (12–64)
ALP (IU/ml)	139 (40–150)	126 (40–150)	83 (40–150)
Total Bilirubin (mg/dl)	0.7 (0.2–1.2)	1.3 (0.2–1.2)	1.0 (0.2–1.2)
Direct Bilirubin (mg/dl)	0.2 (0–0.5)	0.4 (0–0.5)	0.5 (0–0.5)
CK (U/l)	216 (46–71)	95 (46–71)	319 (46–71)
LDH (U/l)	307 (120–246)	636 (120–246)	750 (120–246)
Prothrombin time (sec)	13.0 (10.9–15.0)	19.5 (10.9–15.0)	17.9 (10.9–15.0)
INR	0.98 (0.80–1.30)	1.51 (0.80–1.30)	1.38 (0.80–1.30)

Figure 1: Ferula communis.

Abdominal ultrasound showed no abnormalities in any of the patients. The national poison control center was consulted. Elevated liver function tests in all three cases were evaluated as consistent with a toxidrome. All three patients were admitted to the emergen-

cy intensive care unit (ICU) for monitoring and supportive treatment. Intravenous hydration was initiated. During approximately three days of follow-up, clinical symptoms gradually resolved. The patients were alert, oriented, and cooperative with stable vital signs and normalized liver function tests by discharge.

Discussion

Ferula communis (giant fennel) is a plant traditionally believed to possess therapeutic properties, although some chemotypes contain toxic compounds. Reports of its toxicity are limited in the literature. In our case series, three patients presented with gastrointestinal and neurological symptoms after ingestion of the plant, and laboratory testing revealed significant hepatic enzyme elevation. One case showed elevated INR suggesting significant hepatotoxicity, while the other two showed increased levels of AST, ALT, and LDH. Although the exact mechanism of toxicity remains unclear, coumarin and furanocoumarin components are suspected to be responsible for hepatotoxic effects. Previously reported cases have documented severe complications such as hemolysis and coagulopathy.

This case series highlights the potential hepatotoxicity of Ferula communis and underscores the risks associated with the unregulated use of herbal products. Early recognition and supportive management of suspected poisonings are crucial to prevent serious outcomes.

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Acute mesenteric ischemia

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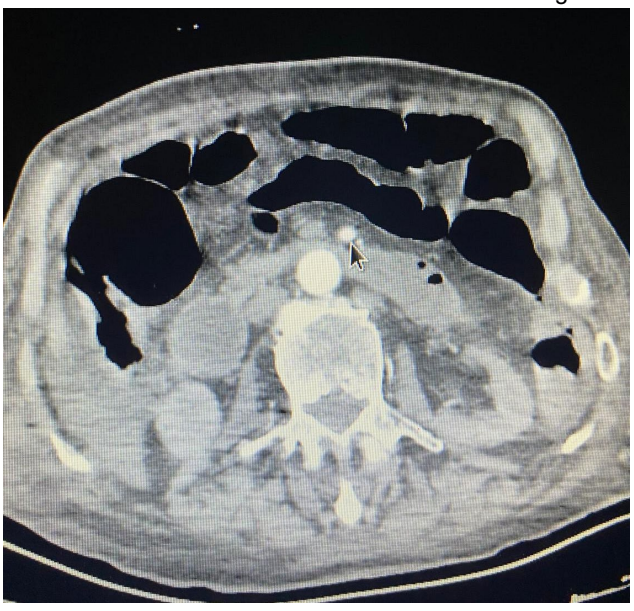
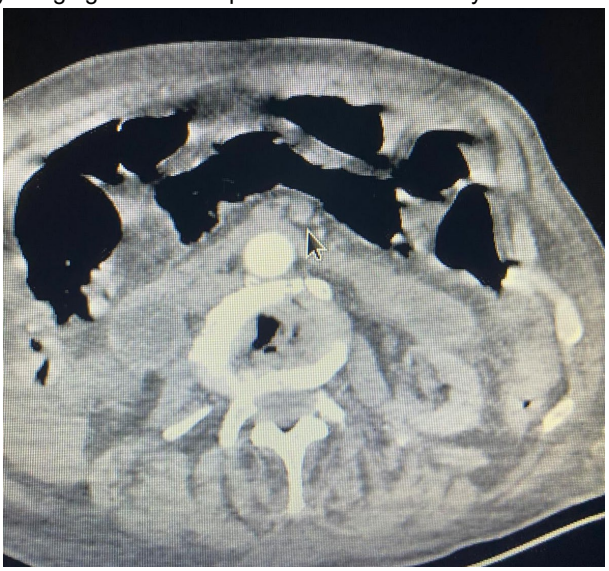
Introduction: Although acute mesenteric ischemia (AMI) is rarely encountered in our emergency departments,it is a diagnostic possibility that should always be kept in mind in the differential diagnosis of abdominal pain.Because its mortality rate can reach 60-70%. Here,we present a 92 year old male patient who presented to our hospital with epigastric and diffuse abdominal pain.

Purpose: Our aim in this study is to draw attention to acute mesenteric ischemia,which is a rare but high mortality case in patients presenting with abdominal pain without any examination findings (defence rebound).

Case: A 92 year old man was admitted to the emergency department with complaints of epigastric pain for five days and sudden new onset of diffuse abdominal pain.He had a history of congestife heart failure and hypertension.Blood pressure was 130/85,saturation:98%,fingerstick blood glucose:120,ECG:salvo waves were present.On abdominal examination,there was diffuse tenderness but no defense and rebound.The fact that lactate was 6 in the laboratory values of our patient alerted us in terms of vascular ischemia.Contrast enhanced abdominal angio tomography imaging revealed superior mesenteric artery embolism.





Discussion: As is well known,AMI is a highly mortal condition resulting in ischemia,cellular damage and necrosis due to sudden interruption of small intestinal blood supply.Although the first thing that comes to mind is the cardioembolic process in patients with atrial fibrillation (AF),this definition of AMI is not limited to this.Until proven otherwise,severe abdominal pain should be considered as AMI regardless of physical axamination findings,because newly developing acute mesenteric ischemia may cause pain but may not give any axamination findings.In our case ,although there were no sings of defense and rebound on abdominal examination ,acute mesenteric ischemia with high mortality was diagnosed.

Keywords:Acute mesenteric ischmeis(AMI)

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Behind The Curtain: The Dark Side Of Embolic Obstruction

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Introduction

One of the most important causes of retinal artery occlusions is a thrombus that develops due to atherosclerosis. These thrombi associated with atheroma constitute approximately 80% of central retinal artery occlusions, and the risk of occurrence increases in individuals with diseases such as hypertension or diabetes (1). One of the important factors that cause occlusion is the embolism. Embolisms vary according to their origin. Carotid embolisms are usually cholesterol, fibrinoplatelet, or calcific. Cardiac embolisms may be calcific or may originate from vegetations coming from heart valves in endocarditis or from wall thrombus formed in atrial fibrillation. Aortic diseases, including dissection, are also considered as sources of embolism. Inflammatory diseases may also play a role in retinal artery occlusions. Temporal arteritis (giant cell arteritis), polyarteritis nodosa, systemic lupus erythematosus (SLE), granulomatosis with polyangiitis, Takayasu arteritis and pancreatitis are included in this group. Some thrombophilic disorders are also associated with retinal artery occlusion. Diseases such as antiphospholipid antibody syndrome, protein S deficiency, protein C deficiency, leukemia, and lymphoma are among these disorders. Infectious agents can also cause occlusion in the retinal vessels. Examples of these infections are toxoplasmosis, mucormycosis, and syphilis. Pharmacological factors include oral contraceptive drug use and substances such as cocaine. Ocular factors such as severe elevation of intraocular pressure, trauma, or optic nerve drusen can also cause retinal artery occlusions. Rare causes include retinal migraine (vasospasm). This condition usually occurs in younger individuals when all other causes have been excluded (2). Retinal damage becomes irreversible over time. Although the period during which treatment may be effective has not been precisely determined, it is thought to be approximately 90-100 minutes. Some sources extended this period to 105 min. However, it is generally recommended that a treatment attempt be made within 24 to 48 hours of the onset of obstruction (3).

Central retinal artery occlusion is a condition that develops because of blockage of the central retinal artery just after it exits the optic nerve. This blockage causes sudden and unilateral painless vision loss. Although the disease is rare, it is estimated to occur in approximately 1 in 100,000 people per year. It mostly occurs in individuals over the age of 60 years and is usually associated with heart valve disease in younger patients. The incidence is slightly higher in men than in women. The most obvious symptom of central retinal artery occlusion is unilateral and painless vision loss that develops within a few seconds. In the vast majority of cases, vision decreased to the level of counting fingers only. In more severe cases, it is thought that the ophthalmic artery may also be affected. Some patients may report a history of temporary vision loss (amaurosis fugax) before the event occurs. Such temporary attacks occur before the occlusion in approximately 10% of patients. In rare cases, vision loss may occur in both eyes, but it is usually not of equal severity. During examination, the retina takes on a pale appearance, while afferent pupillary defect and thinning of the vessels are noticeable. In cases where the blood column shows segmentation, a 'cattle truck' appearance can be observed in the arteries. A distinct cherry-red spot is seen in the macula because the central region is fed by the intact choroid due to retinal edema that develops after occlusion. This is one of the characteristic findings of central retinal artery occlusion. During systemic evaluation, auscultation should be performed for the presence of a murmur in the carotid arteries, heart sounds should be listened to, the radial pulse should be checked for signs of atrial fibrillation, and blood pressure should be measured. Detection of underlying systemic diseases is of great importance in determining possible embolic sources (1). Other diseases that may cause a similar clinical picture to central retinal artery occlusion should also be considered in the differential diagnosis. Retinal detachment, vitreous hemorrhage, retinal vein occlusion, acute glaucoma, and acute optic neuritis are among these diseases. A detailed eye examination and systemic evaluation are necessary to make the correct diagnosis (4).

Case

A 56-year-old male patient noticed significant vision loss in the central visual field of his left eye when he woke up in the morning. When he applied to the emergency department, severe vision loss was detected in his left eye. Laboratory examinations included hemogram, biochemistry, CRP, sedimentation, and cardiac enzyme tests. Brain and carotid CT angiography, PA chest X-ray, and ECG were requested for imaging. The patient had hypertension and coronary artery disease in his medical history. Coronary angiography had been performed before, but no stent had been placed. The systemic examinations were evaluated within normal limits. The medications he was using were Beloc 50 mg, Coraspin 100 mg, Ator 10 mg, and Norvasc 5 mg. Vital signs, laboratory results,

and brain-carotid CT angiography examination were reported as normal. A normal sinus rhythm was observed on the ECG. In the emergency department, intravenous mannitol was administered to reduce the intraocular pressure, and ocular massage was performed with repeated sudden interruptions of 10-15 seconds. His condition was quickly reported to the ophthalmology and underwater and hyperbaric medicine consultant physicians. Hyperbaric oxygen treatment was planned, and he was admitted to the ophthalmology ward. In the eye examination, a distinct cherry red spot was detected in the center of the left macula in the posterior segment examination. In the optical coherence tomography (OCT) examination, edema was detected in all layers of the left eye retina. Fundus fluorescein angiography showed signs of central retinal artery occlusion. Paracentesis was applied from the anterior chamber to reduce the intraocular pressure. In the cardiology evaluation, the sinus rhythm was observed as 55/min in the ECG. The ejection fraction was measured between 60% and 65% in the echocardiography and cardiac functions were evaluated as normal. Although the patient had dyspnea, heart failure was not considered, but coronary artery disease could not be excluded. With the diagnosis of central retinal artery occlusion, the patient received a total of 12 sessions of hyperbaric oxygen therapy over 12 days (double sessions on the first two days, with a two-day break on the weekend). At the end of the treatment process, the patient stated that the "cloudy image" in his left eye did not disappear completely, but it decreased by approximately 30%.

Figure-1: OCT (Optical Coherence Tomography) image of the patient

Discussion

The diagnosis of central retinal artery occlusion is usually made by clinical evaluation, and the examinations performed are mainly aimed at excluding underlying diseases. One of the most important causes of this occlusion is giant cell arteritis. When diagnosed early, vision loss can be reversed with appropriate treatment, and the other eye can be prevented from being affected. Therefore, if there is clinical suspicion, fluorescein angiography and optical coherence tomography are among the important examination methods that support the diagnosis. Especially in patients over the age of 60 years, ESR and CRP values should be measured urgently to exclude giant cell arteritis. In addition, coagulation tests and complete blood, and vasculitis screening tests should be performed to detect coagulation disorders. In the long term, the patient's cardiovascular risk factors should be evaluated and the fasting blood sugar and lipid profile should be examined. Central retinal artery occlusion is an extremely urgent clinical condition in which irreversible retinal damage begins within minutes. According to animal experiments, the time to irreversible damage to retinal cells is approximately 90-100 minutes, and therefore an aggressive treatment protocol should be applied within the first 24 h following acute vision loss. One of the main goals of treatment is to rapidly reduce the intraocular pressure, increase perfusion, and ensure the progression of the occlusive embolism (5). For this purpose, first, ocular massage is applied for 10-15 seconds, repeated with sudden interruptions, to mechanically support blood flow. In addition, lowering the intraocularular pressure with anterior chamber paracentesis, systemic acetazolamide treatment, and intravenous mannitol administration are among the most commonly used methods (6). If the obstructs are considered due to giant cell arteritis, high-dose intravenous corticosteroid treatment should be started early. It is recommended to administer 100% or 95% oxygen from the surface to ensure oxygenation of the inner layers of the retina by diffusion from the choroid. If possible, hyperbaric oxygen treatment started early may be beneficial in terms of preserving visual function (6-8). When using systemic antifibrinolytic agents, caution should be exercised in terms of systemic side effects. It has been shown that interventional methods such as selective intra-arterial fibrinolysis with urokinase and tissue plasminogen activators do not provide superiority over traditional treatments. Other treatment approaches aimed at regulating retinal circulation include aspirin use, calcium channel blockers, retrobulbar and systemic vasodilator drugs, intravenous bolus administration of high-dose methylprednisolone, isovolemic hemodilution, vitreoperfusion, and local hypothermia. In addition, excitotoxin blockade, antioxidant therapy, and free radical scavenging agents are also considered among the treatment options to protect the retina from toxic metabolic damage (9,10). Complications that may occur in the later stages of the disease should also be managed carefully. Panretinal photocoagulation, intravitreal injection, and antiglaucoma treatment can be applied in patients with rubeosis iridis, optic disc, and retinal neovascularization. Intraocular surgical interventions may be inevitable in some cases. In addition, the diagnosis and treatment of systemic diseases underlying central retinal artery occlusion should not be neglected. In this process, a multidisciplinary approach should be adopted between ophthalmologists and cardiologists, hematologists and rheumatologists. Rapid recognition of central retinal artery occlusion in the emergency department and prompt intervention are critical for preventing vision loss. Timely treatment can directly affect the patient’s quality of life by minimizing irreversible retinal damage. Therefore, when faced with this disease, systematic evaluation and the creation of a rapid treatment plan are one of the cornerstones of preventing permanent vision loss.

Conclusion

In conclusion, central retinal artery occlusion is a condition that requires urgent intervention and may be associated with serious systemic diseases. Despite early diagnosis and aggressive treatment, the prognosis is generally poor and permanent vision loss is common. However, these patients should be carefully evaluated not only for eye diseases but also for cardiovascular diseases and coagulation disorders. Early detection and management of systemic risk factors are critical not only to prevent vision loss but also to improve the life expectancy and quality of life of patients.

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An Endoscopic Nightmare: Esophageal Rupture and Subcutaneous Emphysema

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Introduction

Endoscopic procedures are minimally invasive procedures widely used in the diagnosis and treatment of gastrointestinal diseases. However, significant complications can occur during these procedures, albeit rarely. Esophageal rupture is one of the most serious complications after endoscopy and can lead to serious morbidity and mortality (1). Esophageal ruptures usually occur during endoscopic procedures and patients frequently present with symptoms including severe abdominal pain, hypotension, tachycardia and subcutaneous emphysema. Any traumatic procedure that may occur during endoscopy may lead to the development of rupture (2). Subcutaneous emphysema occurs as a result of leakage of gas from the esophageal wall and spread to the subcutaneous tissue and this may be an important sign for early diagnosis of esophageal rupture. This case focuses on the management of a case of esophageal rupture and subcutaneous emphysema after endoscopy.

Case

A 41-year-old woman, scheduled for endoscopy with a prediagnosis of gastric ulcer, presented to our hospital with severe abdominal pain, low blood pressure (60/30 mmHg), tachycardia (115 beats/minute) and tachypnea after the endoscopic procedure. No complications were observed during the endoscopic procedure. However, the patient's condition deteriorated rapidly after the procedure. Physical examination revealed subcutaneous emphysema in the neck and the patient's general condition deteriorated severely. Computed tomography (CT) scan with oral administration of contrast material revealed esophageal rupture and subcutaneous emphysema in the neck. A multidisciplinary approach was adopted, thoracic surgery, general surgery and gastroenterology consultations were obtained and the treatment process was initiated. Hemodynamic status was stabilized and the patient was referred to an external center for surgical intervention and follow-up. Since the patient was unstable at the first emergency admission and the clinic was risky, surgery was planned in an external center and the ruptured area was repaired primary. The patient with stable hemodynamics was transferred to the thoracic surgery service after intensive care unit follow-up and was discharged without complications.

Discussion

Esophageal rupture is a rare complication after endoscopic procedures that requires urgent surgical intervention. Esophageal perforation usually presents with severe pain, respiratory distress, hypotension, bleeding and subcutaneous emphysema. Subcutaneous emphysema occurs as a result of the spread of gas to the subcutaneous tissue and this finding is an important adjuvant factor in the diagnosis of esophageal rupture (3). Advanced imaging techniques such as CT and endoscopy are frequently

used in the diagnosis of esophageal rupture. CT is very useful in determining the severity of the disease and early detection of complications. Early surgical intervention can improve the patient's prognosis. Surgical intervention is usually necessary in the treatment of esophageal rupture. However, it is critical to stabilize the patient before surgical intervention. A multidisciplinary approach provides effective management in the treatment process (4). Collaboration of thoracic surgery, general surgery and gastroenterology specialists may help to achieve favorable results in the treatment process of the patient. This case once again emphasizes the importance of a multidisciplinary approach in the treatment of esophageal rupture.

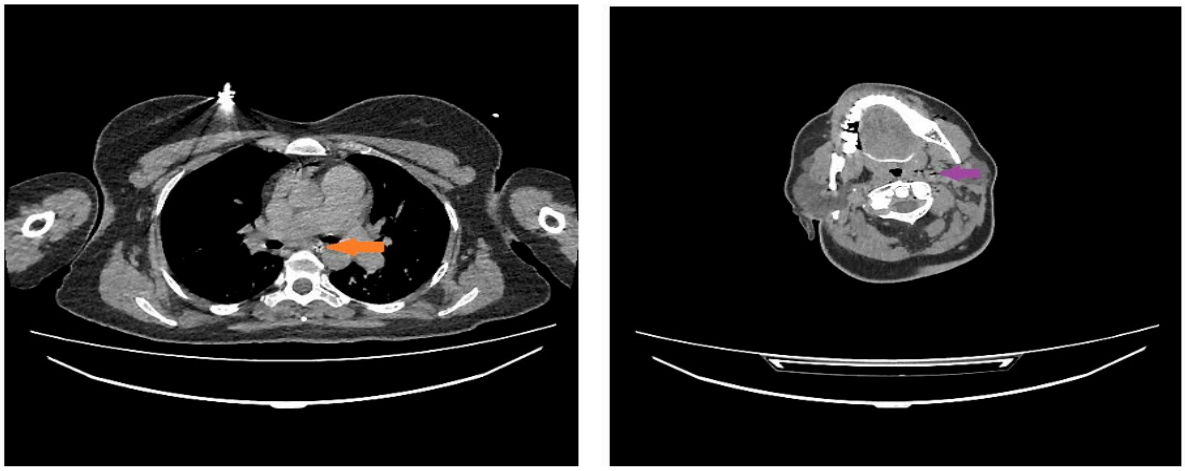

Figure 1. Esophageal contrast agent leakage (orange arrow) and subcutaneous emphysema (purple arrow)

Conclusion

Early diagnosis, surgical intervention and multidisciplinary approach improve the prognosis of patients and increase the effectiveness of the treatment process. This case demonstrates that esophageal rupture is a rare but critical complication after endoscopy and with appropriate treatment, survival rates can be increased.

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Isolated Cerebral Fat Embolism Following a Femoral Shaft Fracture: A Detailed Case Report

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Introduction

Fat embolism syndrome is a clinical condition that typically arises within 12–72 hours after traumatic injury, especially fractures of long bones. Its pathophysiology involves the release of fat globules from the bone marrow into the venous system, which may travel to the lungs or bypass the pulmonary circulation to affect the central nervous system. The syndrome's hallmark triad includes respiratory distress, petechial rash, and neurological symptoms, yet not all cases present with this classic constellation. Isolated cerebral fat embolism, devoid of pulmonary or dermatologic involvement, presents a diagnostic dilemma due to its rarity and nonspecific symptoms such as confusion, seizures, or coma. Recent advances in neuroimaging, particularly the use of diffusion-weighted MRI, have enhanced the ability to detect microembolic lesions characteristic of this condition. Herein, we report a case of isolated CFE diagnosed through MRI in a young male patient with a femoral shaft fracture, who exhibited rapid neurological decline.

Case Report

A 27-year-old male with no known comorbidities was brought to the emergency department by emergency medical services following a high-energy motor vehicle accident. The patient had been struck as a pedestrian and was found conscious at the scene. Initial assessment revealed stable vital signs: heart rate of 68 bpm, blood pressure of 130/70 mmHg, and peripheral oxygen saturation (SpO□) of 99% on room air. Glasgow Coma Scale (GCS) was 15. Physical examination showed no signs of cranial trauma or external bleeding. Systemic and neurological evaluations were unremarkable. Musculoskeletal assessment revealed an isolated left fem-

oral shaft fracture, confirmed radiographically. A long leg splint was applied for stabilization, and the patient was admitted for further observation and orthopedic consultation.

Approximately two hours after admission, the patient developed sudden-onset confusion followed by a generalized tonic-clonic seizure. The seizure was managed with 2 mg of intravenous midazolam and oxygen supplementation via face mask at 4 L/min. Postictally, the patient remained somnolent. Arterial blood gas analysis revealed mild respiratory acidosis (pH 7.32, pCO□ 47 mmHg, pO□ 91 mmHg, SpO□ 95%). A differential diagnosis including trauma-related cerebral thromboembolism, fat embolism, and hypoxia-induced seizure was considered. Emergency non-contrast computed tomography (CT) of the brain and thorax yielded no pathological findings. Due to persistent altered mental status, a diffusion-weighted cranial MRI was performed.

DW-MRI demonstrated multiple hyperintense foci in the bilateral basal ganglia, occipital lobes, and parietal cortices, consistent with a 'starfield' pattern typically observed in cerebral fat embolism. No hemorrhagic transformation or mass effect was noted. Neurological examination revealed absent verbal, auditory, and visual responsiveness. Pupillary light reflexes were preserved bilaterally, while corneal and gag reflexes were absent. The patient exhibited aphasia and right-sided decerebrate posturing to noxious stimuli. Based on clinical and radiological findings, a diagnosis of isolated cerebral fat embolism was established.

Following neurology consultation, the patient was initiated on a treatment regimen including high-dose corticosteroids (prednisolone), osmotic diuretics (mannitol), antiepileptic therapy (phenytoin), and prophylactic anticoagulation with low molecular weight heparin. The patient was transferred to the intensive care unit (ICU) for close neurological monitoring and supportive care.

Discussion

Isolated cerebral fat embolism is a diagnostic challenge due to the absence of respiratory or dermatological features commonly associated with FES. It is hypothesized that fat globules enter the systemic circulation either via a right-to-left intracardiac shunt, such as a patent foramen ovale, or through intrapulmonary arteriovenous malformations. These emboli then lodge within the cerebral microvasculature, causing endothelial damage and local ischemia. Clinical manifestations range from mild cognitive dysfunction to coma and seizures, as seen in the current case.

Imaging plays a crucial role in confirming the diagnosis. The 'starfield' pattern on DW-MRI, consisting of multiple punctate hyperintensities scattered throughout the white matter and deep gray structures, is considered pathognomonic. Although CT imaging is often the first-line modality in acute trauma settings, it lacks sensitivity for detecting microembolic cerebral lesions. The time course, clinical deterioration, and MRI findings in this patient strongly supported the diagnosis of isolated CFE.

There is no specific therapy for CFE; management is largely supportive. Corticosteroids may reduce cerebral edema and inflammatory responses, while mannitol helps manage intracranial pressure. Antiepileptic drugs are used for seizure control. Prophylactic anticoagulation is recommended in immobilized trauma patients to prevent venous thromboembolism. Prognosis varies depending on the extent of neurological involvement, but early recognition and ICU-level supportive care may significantly improve outcomes.

Conclusion

Isolated cerebral fat embolism should be considered in trauma patients with long bone fractures who develop sudden neurological deterioration in the absence of cranial injury. Early use of diffusion-weighted MRI can facilitate prompt diagnosis. Multidisciplinary management involving intensive care, neurology, and trauma surgery teams is essential. Future research should focus on improving risk stratification and exploring potential targeted therapies.

Keywords: Fat embolism syndrome, cerebral fat embolism, long bone fracture, diffusion-weighted MRI, neurological deterioration

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Bibliometric analysis of articles in emergency medicine for 2022-2024, Research Trends and Global Collaboration

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Introduction

Emergency medicine is a field of health that is undergoing rapid development and transformation. Consequently, a thorough analysis of extant research in this field is imperative to ascertain prevailing trends and future studies. Bibliometric analyses are utilised to elucidate the quantitative and qualitative characteristics of the extant literature within the pertinent field, thereby illuminating its developmental trajectory and the requisite research areas. Bibliometrics is the analysis of the characteristics of academic publication types, including author, subject, publication information, and citation sources. In recent years, bibliometric analysis studies have

become increasingly prominent, with software such as SciMAT, Gephi, Biblioshiny, Leximancer and VOSviewer being utilised in this field. The existence of scientific databases such as Scopus, Web of Science and Pubmed in the field of medicine, in conjunction with the accessibility of the studies contained therein, has resulted in the application of bibliometrics in the field of health (1).

In a bibliometric analysis study in the field of emergency medicine, the journals in which research in this field was published were classified from Q1 to Q4 (2). The analysis revealed that the keywords "resuscitation" and "trauma" predominated in Q1 and Q2 journals, respectively, while "covid-19" and "pediatrics" emerged as prominent themes in Q3 and Q4 journals. Furthermore, an analysis of the most frequently cited subjects revealed that cardiac arrest, disaster, burns and trauma were the most prevalent. The findings of this study indicated that research in emergency medicine exhibits variation in accordance with the journal in which it is published. In a further bibliometric analysis of Trauma and Emergency Surgery in the field of emergency medicine up to 2023, it was established that the most prolific publishing institution was the University of California, the most publishing country was the USA, and the most prolific journal was the Journal of Neurotrauma and Brain Injury. It has been documented that the majority of the studies under review pertain to the consequences of paediatric traumas, neurological diseases and sports injuries (3). A study was conducted in which the WoS database was used to evaluate studies in the field of emergency medicine in Turkey (4). The analysis revealed a total of 4,048 scientific studies published in four different journals, of which 3,681 were articles. The most cited study was identified as 'User's guide to correlation coefficients', and the majority of the studies were conducted in Turkey, Iran and India.

These studies facilitate comprehension of the evolution of research trends and foci in emergency medicine across different geographical regions. In this context, a bibliometric analysis covering the last three years is of great importance to identify current trends in the existing literature, research gaps and future research needs.

Materials and Methods

In the context of bibliometric analysis, the initial step entails the selection of a database. The present study utilised the Scopus database as its primary research tool. Bibliometric indicators such as citation counts, h-index and co-citation networks are utilised by Scopus to assist academics in estimating the impact of scientific articles and authors (5). The identification and analysis of research trends and patterns of collaboration across fields, institutions and nations can be facilitated. In addition, rigorous data validation techniques are employed by Scopus to ensure the accuracy and completeness of bibliographic information, thus making it a reliable source for bibliometric analysis (6). The information is then filtered from the database.

Search Strategy, Inclusion and Exclusion Criteria

A comprehensive search of the extant literature on "emergency medicine" was conducted in Scopus, yielding a total of 96436 data points. When the publication period of the literature was constrained to 2022-2024, 15,763 studies were identified. Subsequently, 5410 data items were incorporated into the study, with the restriction imposed that they should be categorised as 'medicine', 'article', 'English', 'journal' and 'open access'. Consequently, letters to the editor, theses, conference presentations, books and publications retracted by authors were excluded from the study.

The articles related to emergency medicine in the Scopus database were analysed with the R application. The application under discussion features software designed for bibliometric analysis that does not require any coding, with an interface that runs on the internet (7). The software program VOSviewer was utilised for the purpose of constructing keyword networks (8). Consequently, a comprehensive bibliometric analysis was performed on the relevant articles in the field of "emergency medicine" listed in the Scopus database, utilising quantitative techniques such as Biblioshiny, VOSviewer, Bibliometrix, and R package.

Results

In the analysis of the data, a detailed descriptive analysis of the publications, the status of the institutions/organizations, the status of the countries, the status of the journals, and the cooperation of the countries were evaluated first (Figure 1).

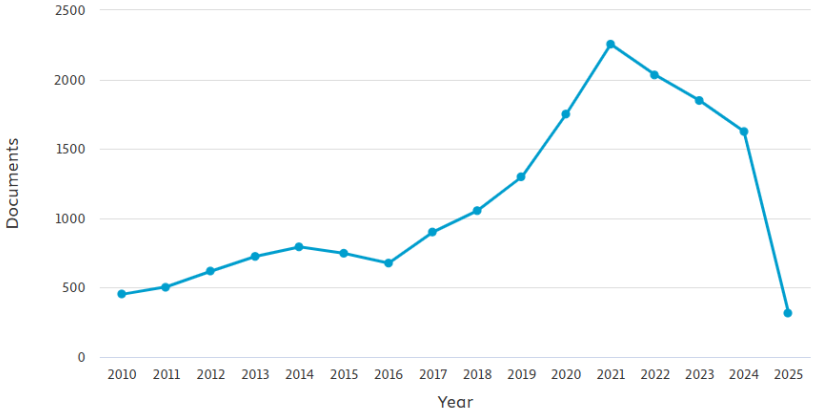
Figure 1: Main information

Figure 1 provides a bibliometric analysis summary of academic publications related to "emergency medicine" during a specific time period (2022-2024). Timespan: This indicates that the period under analysis is 2022-2024. Despite its brevity, this 3-year period is of consequence in terms of evaluating the trend following the pandemic caused by the novel coronavirus (SARS-CoV-2). The fact that data was obtained from 1409 different sources demonstrates that information was collected from a wide range of sources. A total of

5,410 documents demonstrate that it is a highly comprehensive data set. The findings of the study indicated that there had been a year-on-year decline of -13.27% in the growth rate of academic publications. This finding suggests that there has been a decline in publications in this field following the pandemic. The mean age of the documents (2.09 years) indicates that the data is fairly recent. The mean number of citations per document (4.66) indicates that each document has received more than four citations, thus suggesting that the academic impact of the documents under scrutiny is quite strong. The total number of references (162,585) clearly indicates that an extensive literature review was conducted in the studies. The total number of authors is 35,045. This finding suggests that there is a relatively substantial number of researchers in this field. A total of 106 authors work individually, indicating that collaborative academic production is the predominant mode. The mean of 7.99 authors per publication indicates a high level of collaboration. It is evident that 18.85% of the documents were produced through international cooperation. It is evident that global connections are robust; nevertheless, there is a possibility for their enhancement to a greater extent.

Figure 2 illustrates the annual publication rate of articles in the domain of "Emergency Medicine" from 2010 to 2025. A decline in the number of articles was observed after 2022. A decline in the number of articles was observed from 2042 to 1536 in 2024. The year 2021 has been identified as the one with the highest number of articles. This phenomenon may be indicative of a more general upward trend in scientific publications that has been observed in the aftermath of the global pandemic caused by the severe acute respiratory syndrome (SARS-CoV-2) virus. The rationale behind the incorporation of this extensive range within the scope of our study is to elucidate the advancements that have transpired in this domain over the years, with a particular emphasis on the repercussions of the pandemic caused by the severe acute respiratory syndrome (SARS-CoV-2). It is noteworthy that there has been a steady increase in the production of scientific articles, unaffected by the pandemic.

Figure 2: number of articles published by year



The ranking of the journals with the highest number of articles published on "emergency medicine", along with the number of articles published in each source, is presented in Table 1. It is evident that BMJ Open (453 articles) is the publication with the highest number of articles published. This finding indicates a substantial congruence between the research topics and the scope of this journal, thereby underscoring its pioneering contributions within the field. AEM Education and Training is in second place, but there is a significant gap with the first. It is evident that the Western Journal of Emergency Medicine has made a substantial contribution to the field.

Table 1:The most published journals for emergency medicine

Sources	Articles
BMJ Open	453
AEM Education and Training	143
Western Journal of Emergency Medicine	113
BMJ Case Reports	91
JACEP Open	85
BMC Emergency Medicine	82
Journal of Clinical Medicine	69
International Journal of Environmental Research and Public Health	68
BMC Health Services Research	59
International Journal of Emergency Medicine	59

The authors who published the most articles in the field of "Emergency medicine" and the organisations or affiliations of the authors who contributed to the studies are shown in Table 2. It is evident that Zhang Y, JR, Gottlieb M and Wang Y are highly active and contributing authors in their field, making a substantial contribution to the existing literature. The University of Toronto is at the vanguard of this field, as evidenced by its 314 institutions. This demonstrates its leadership and academic influence in this domain. The preponderance of institutions from the United States and the United Kingdom within the top 10 reflects the predominant influence of

these nations in the field of research under discussion.

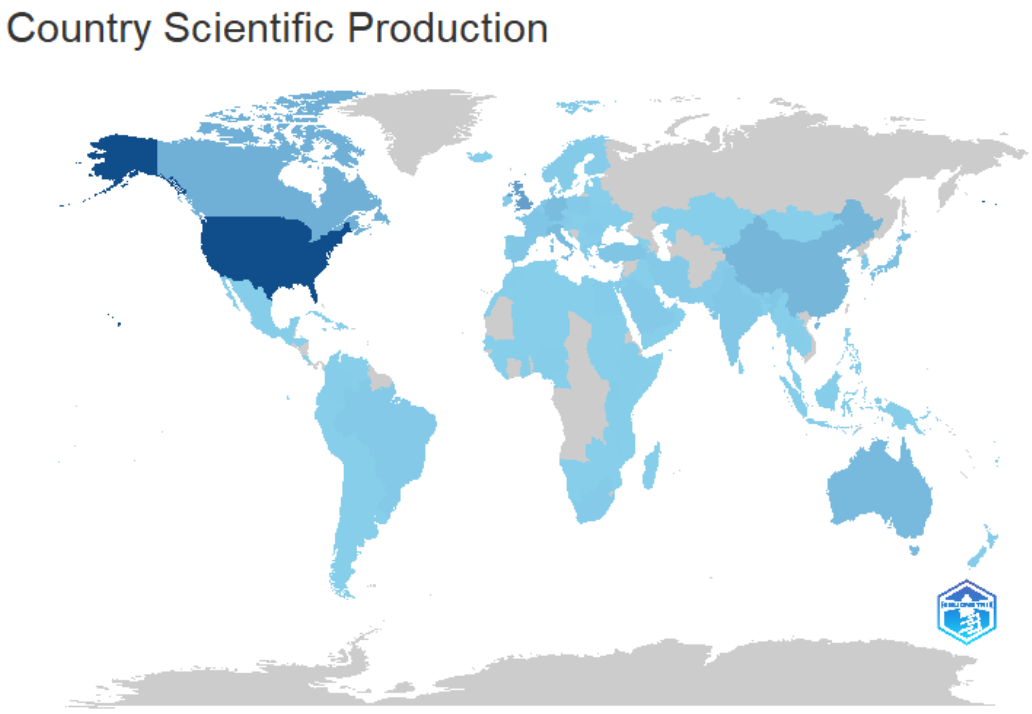
Table 2: Publication status of the authors and Relevant affiliations that have contributed to research

Author	Articles	Affiliation	Articles
Zhang Y	27	University of Toronto	314
JR	24	Harvard Medical School	294
Gottlieb M	23	University of Birmingham	293
Wang Y	23	University of California	283
Liu Y	19	Monash University	208
Chen Y	17	University of Ottawa	192
Li X	17	Mcmaster University	191
Zhang X	17	Mayo Clinic	166
Li Y	16	University of Pennsylvania	165
Wang H	16	University of Calgary	164

Figure 3 shows the countries with the highest number of publications in the field of “Emergency medicine”. The map shows the countries that have published the most articles, the countries that have published the fewest articles and the countries that have published no articles.

Figure 3: Country scientific production

Country	Articles
USA	1452
United Kingdom	451
Canada	295
China	279
Australia	236
Germany	184
Italy	165
Turkey	161
Japan	151
Switzerland	89
India	88
France	87
Spain	82



The USA has published 1452 articles, thus establishing itself as the leader in this field. This is indicative of the research capacity, funding and academic intensity of the US in this field. The leadership of the United States can be attributed to a number of factors, including its extensive academic network, substantial research funding, and its leading position in the medical field. Turkey (161 articles) is positioned eighth. This finding indicates that Turkey has a higher level of productivity in this field than many European and American countries. This table reveals the international nature of the research field and the intense academic effort from different regions.

The paper that is most productive is the one which has received the highest cumulative number of citations. The ten most productive papers are presented in Table 3.

With 1568 citations, the article titled “2023 Alzheimer's disease facts and figures” published in Alzheimer's and Dementia in March 2023 ranks first. With 361 citations, the article “SCAI SHOCK Stage Classification Expert Consensus Update: A Review and Incorporation of Validation Studies” ranks second. With 290 citations, the article titled “Global Initiative for Asthma Strategy 2021: Executive Summary and Rationale for Key Changes” by Reddel et al. (2022) published in the American Journal of Respiratory and Critical Care Medicine ranks third.

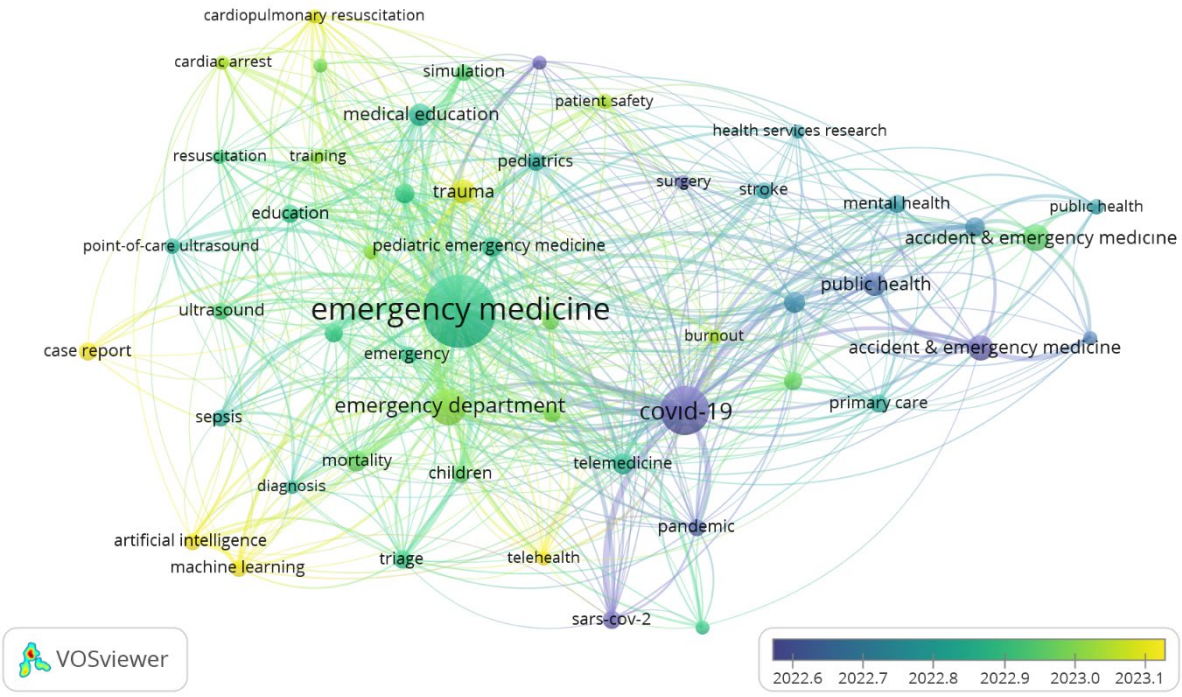
With VOSviewer software, “Overlay visualization” keywords were colored differently according to the year of publication and the time intervals in which they appeared in the literature were determined. In our study, the average (yellow) publication year for the re-prominent terms is 2023. The visual obtained as a result of the organized analysis is given in Figure 4.

.

Table 3: Most Global Cited Documents

Paper	DOI	Total Citations
Na, 2023, Alzheımer's Dementıa	10.1002/alz.13016	1568
Naıdu Ss, 2022, J Am Coll Cardıol	10.1016/j.jacc.2022.01.018	361
Reddel Hk, 2022, Am J Respır Crıt Care Med	10.1164/rccm.202109-2205PP	290
Shanafelt Td, 2022, Mayo Clın Proc	10.1016/j.mayocp.2022.09.002	287
Kaplon H, 2022, Mabs	10.1080/19420862.2021.2014296	274
Rossaınt R, 2023, Crıt Care	10.1186/s13054-023-04327-7	270
Suvereın Mm, 2023, New Engl J Med	10.1056/NEJMoa2204511	262
Hodkınson A, 2022, Bmj	10.1136/bmj-2022-070442	259
Pıerce Rp, 2023, J Telemed Telecare	10.1177/1357633X20963893	189
Shapıro Nı, 2023, New Engl J Med	10.1056/NEJMoa2212663	179

Figure 4: Keyword timeline



Months 6-7 of 2022 in Figure 4 (blue shades): In this period, keywords such as covid-19, sars-cov-2, health policy come to the fore due to the pandemic effect. Research Orientation in this Period: The impact on emergency departments and care of patients with trauma, stroke, surgery in the covid 19 pandemic.

Late 2022 and early 2023 (shades of green): Research appears to cover a wide range of topics, including the main topics of emergency medicine. This period is characterized by studies on medical education, ultrasound, sepsis, diagnosis and mortality of diseases.

First months of 2023 (Yellow tones): A period in which new technologies and studies on artificial intelligence intensify. While studies on the use of concepts affecting the world such as artifical intelligence, machine learning and telehealth in emergency medicine come to the fore. Cardiopulmonary resuscitation and trauma-related studies seem to intensify again.

The word cloud representing the important medical researches in the field of “Emergency medicine” in the last 3 years is given in Figure 5.

We can categorize the word cloud in Figure 5 into different categories according to their content.

Demographics: Human (4758), Female (3831), Male (3568): The words in the top 5 of the most frequently occurring words in these studies show that the studies generally discriminate on the basis of gender. The word human indicates that the studies are about

people. Adult (3276), Aged (1617): These words indicate a focus on age groups and reveal that studies are generally conducted in the adult age group.

Figure 5: Word Cloud

Research Types and Methods: Atricle (4156), Major clinical study (1694), Controlled study (1590): Indicates that the studies conducted were large-scale clinical trials and prospective randomized trials.
Medical and Emergency Services: Emergency medicine (1769), Emergency ward (1705), Hospitalization (869), Hospital emergency service (810): Indicates that studies on emergency health services and inpatient treatment planning are ongoing.

Discussion

A bibliometric analysis of research in the field of "Emergency Medicine" between 2022 and 2024 reveals significant findings and trends in this domain. Despite encountering a decline in growth rate of -13.27% due to the lifting of the impact of the pandemic, the fact that 5410 quality articles were obtained in a 3-year period demonstrates the efficacy and prestige of the studies in this field. It is evident that articles published in 2024 exhibit a notable dearth of citations. This is likely attributable to the temporal requirement for new studies to gain a sufficient place in the literature. However, the average citation rate of 4.66 is noteworthy, especially when considered alongside the average publication age of 2.09. This suggests that the field is generating significant interest, which is a positive indicator for future research and development.

The leadership of esteemed journals such as BMJ Open and AEM Education and Training in this field demonstrates their pivotal role in publishing high-quality research. In addition, it is evident that prolific authors such as Zhang Y, JR, Gottlieb M and Wang Y. have been instrumental in establishing the field's foundation. It is evident that institutions such as the University of Toronto, Harvard Medical School and the University of Birmingham occupy a central position in the realm of academic innovation and collaboration. Whilst the present study demonstrates the significance of international collaboration (18.85%), there is an opportunity to enhance these collaborations to even higher levels in order to facilitate research of a higher quality that incorporates diverse perspectives.

The majority of studies in this field are of the prospective, randomised, controlled, clinical trial type, and are conducted on human subjects rather than experimental studies on animals. The extant studies have focused on age groups (elderly and middle-aged) and have also differentiated between male and female participants. The evaluation of outcomes frequently entailed the measurement of survival, mortality and treatment success.

A close examination of the literature reveals that the research agenda can be subject to change, even over brief periods of time, as evidenced by both keyword analysis and thematic focuses. In 2022, the focus was on the impact of the pandemic on emergency departments. In recent years, however, studies on main topics such as trauma, sepsis, technology integration in emergency departments and the impact of artificial intelligence have continued. For instance, the fields of artificial intelligence and machine learning are evolving as innovative technologies that are fundamentally altering the delivery of emergency healthcare services. This demonstrates that the field of emergency medicine is undergoing a transformation into a multidisciplinary research domain, underpinned by technological advancements aimed at enhancing clinical diagnosis and treatment modalities.

Conclusions

This bibliometric analysis reveals the vibrant and multidisciplinary nature of research in emergency medicine over the last three years. The findings reveal the impact of the pandemic in the number of publications and the variation in thematic focus over the years. The present study has underscored the pioneering status of BMJ Open and the significant contributions of authors such as Zhang Y to the field. Furthermore, institutions such as the University of Toronto are distinguished as pivotal hubs of innovation and

collaboration within this domain.

The decline in the citation rates of papers published in recent years is a natural consequence of bibliometric measurement and should not be taken as a sign of poor quality. It is anticipated that the number of citations of these publications will increase over time, and that they will be incorporated into a broader academic discourse.

The transition from clinical and experimental research to a research agenda encompassing artificial intelligence (AI) and public health perspectives demonstrates the field's responsiveness to changing health needs. In the future, the integration of artificial intelligence (AI) technologies into emergency services, the increasing prevalence of multidisciplinary studies, and the prioritisation of global equity are likely to contribute to enhanced outcomes for research in emergency medicine.

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Pancreatic Pseudocyst with Oral Intake Disorder Due to Gastric Compression: Case Report

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Introduction

Pancreatic pseudocyst is a localized collection of fluid rich in amylase and other pancreatic enzymes, surrounded by fibrous tissue not covered by epithelium (1). Pseudocysts are a common clinical entity complicating the course of chronic pancreatitis in 30% to 40% of patients (2). The etiology of pseudocysts is very similar to the causes of pancreatitis (3). The most common causes are acute or chronic pancreatitis, gallbladder stones, alcohol use and trauma (4). It usually presents with complaints including abdominal pain, nausea, vomiting, weight loss, anorexia and sometimes jaundice (5). Enlargement of pseudocysts may lead to more prominent clinical findings by compressing the surrounding tissues. Especially large pseudocysts may cause obstruction or vascular damage by compressing the stomach, intestines or vessels. Pancreatic pseudocysts are diagnosed by a combination of clinical symptoms and imaging techniques. Imaging techniques are used to assess the size and shape of the pseudocyst and its relationship to other organs. In the laboratory, amylase and lipase levels are usually elevated, but may be within reference ranges (6). Transabdominal Ultrasonography (TUS), Computed Tomography (CT), Magnetic Resonance Pancreatography (MRCP), Endoscopic Ultrasonography (EUS) are used in imaging. Treatment is drainage of the pseudocyst. The main indication for drainage is the presence of complications such as infection, bleeding, gastric outlet obstruction due to compression of the stomach or duodenum and biliary obstruction (7).

Case

A 75-year-old man presented to the emergency department with nausea and vomiting after feeding for 3-4 days. He had a history of dementia, diabetes, hypertension, hyperlipidemia, coronary artery disease, heart failure, chronic kidney disease, surrenal adenoma, benign prostatic hyperplasia and biliary pancreatitis. On physical examination, vital parameters were stable, consciousness was clear, oriented and cooperative. The abdomen was distended, there was tenderness in the upper quadrants with palpation, and no defense or rebound was detected. Total bilirubin: 0.46mg/dl, direct bilirubin: 0.16mg/dl, ALT: 54U/L, AST: 55U/L, GGT: 261U/L, ALP: 532IU/L, GGT: 261IU/L, amylase: 184U/L, WBC: 10.38(**x10^9/L**), CRP: 139mg/L. A nasogastric catheter was inserted and it was seen that bile fluid was coming. Abdominal CT was performed to clarify the differential diagnosis due to elevated acute phase reactants and bile content coming from the nasogastric catheter. Loculated fluid was seen adjacent to the stomach, extending to the perisplenic area surrounding the large and small curvature. It was evaluated as pancreatic pseudocyst with a size of 11 cm above the large curvature and 7 cm below the small curvature. Absence of free fluid in the duct suggested that the fluid was in the retroperitoneal space. Gastroenterology and interventional radiology consultation was requested. Percutaneous cyst drainage was performed by interventional radiology. She was hospitalized in the gastroenterology ward after the procedure.

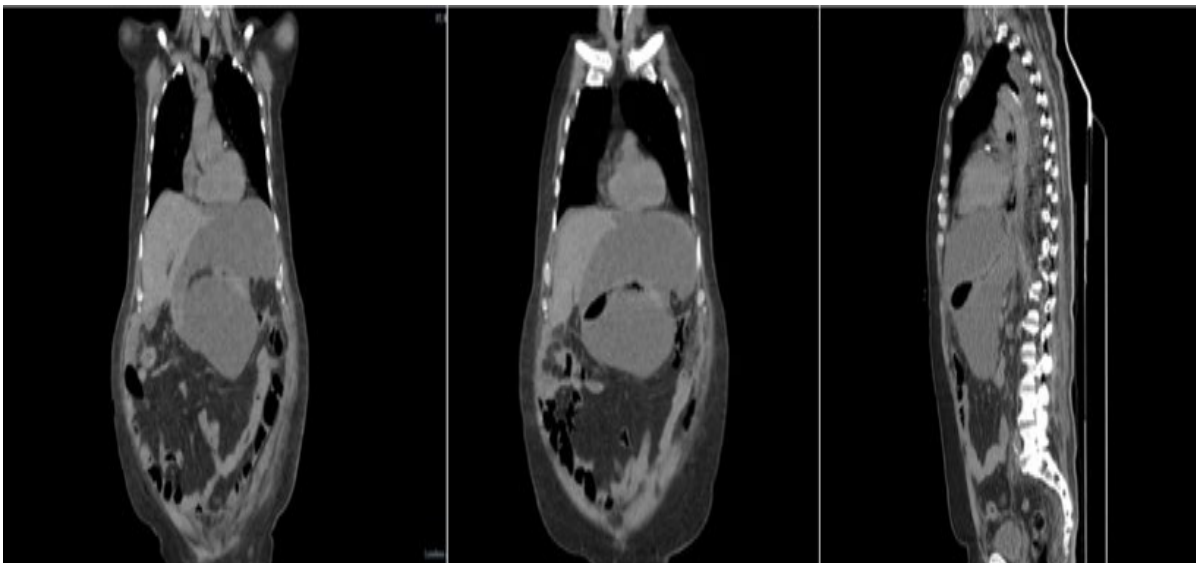

Figure 1. Pancreatic pseudocyst compressing the stomach in CT coronal and sagittal sections

Discussion

Pancreatic pseudocysts are a very common complication of pancreatitis. However, a few cases of giant pancreatic pseudocysts with a maximum diameter of 10 cm or more have been reported in the literature (8). Due to the widespread use of advanced diagnostic and therapeutic methods, large pancreatic pseudocysts are rarely seen today. In the past, it was only possible to detect cysts large enough to cause morphologic abnormalities in adjacent visceral organs using methods such as clinical examination, barium examinations, laparotomy and angiography (9). Today, transabdominal ultrasonography (TUS) is the first imaging method commonly used in the diagnosis of pseudocysts. They are usually seen as round or oval smooth contoured lesions with hypo- or anechoic wall flare. There are signs of acute and/or chronic pancreatitis in the pancreatic parenchyma. Its sensitivity is lower than CT. Abdominal CT can diagnose pseudocyst with 90-100% accuracy. Although MRI is a more sensitive method, it does not provide extra information in the diagnosis. However, MRCP and EUS are very valuable in demonstrating the connection of the cyst with the duct (7). The treatment approach varies depending on the size of the cyst, symptoms, complications and general health status of the patient. Asymptomatic cysts are approached conservatively as long as they are asymptomatic and there is no increase in size. Cysts smaller than 4cm in diameter often resorb spontaneously and rarely cause complications. In general, however, larger cysts are more likely to become symptomatic or cause complications. Without early diagnosis and treatment, giant pseudocysts can have many complications. These complications include massive hemorrhage into the pseudocyst, sepsis with splenic infarction, splenic vein thrombosis, gastrointestinal bleeding and peritonitis with cyst rupture, obstructive jaundice with obstruction of the common bile duct by a large cyst in the head of the pancreas, portal hypertension with cyst compression of the splenic vein or portal vein (10,11,12). Since patients with abdominal pain, nausea, vomiting and impaired oral intake frequently present to the emergency department in the acute period, the role of the emergency department is important in early diagnosis and prevention of complications. Especially in patients with a history of pancreatitis as in our patient, pancreatic pseudocysts should be considered in the differential diagnosis in the absence of elevated pancreatic enzymes but persistent symptoms.

Conclusion

When patients with a known history of pancreatitis present with signs of gastrointestinal obstruction and impaired oral intake, abdominal tomography without delay will be a useful approach to make a rapid diagnosis and initiate treatment before complications develop. In addition, determination of cyst size by tomography is helpful in determining the treatment procedure.

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Evaluatıon Of Rısk Factors Determınıng Early Mortalıty In Patıents Wıth The Upper/Proxımal End Of The Femur

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Introduction

Proximal femur fractures are defined as fractures of the bony structure that includes the femoral head, neck, and the lesser trochanter up to five cm distal. Proximal femur fractures are particularly common in the geriatric population, and the mortality rate within 1 year has been reported as 5% to 20% (1–3). In the FRACTURK study conducted in Turkey, the lifetime probability of hip fracture after the age of 50 was reported as 3.5% in men and 14.6% in women (4).

Our study aimed to determine early-term in-hospital mortality rates and risk factors affecting mortality in patients diagnosed with upper-end femur fractures in the emergency department.

Materials and Methods

Our study was conducted prospectively on patients admitted to the emergency department trauma unit between 01.11.2022 and 31.05.2023, whose proximal end femur fracture was detected by radiological examinations and whose diagnosis was confirmed by the orthopedic specialist. The performance of age, systolic and diastolic blood pressure, pulse, respiratory rate, shock index, and hospital stay in predicting mortality was evaluated by Receiver Operating Characteristic (ROC) Curve analysis. AUC value (the area under the ROC curve), sensitivity, specificity, and 95% confidence interval (CI) values were given as descriptive statistics. Multiple binary logistic regression analysis was also used to determine risk factors affecting 28-day mortality. Statistically significant value was accepted as $p<0.05$.

Results

112 patients with upper/proximal end femur fractures were included in our study. The average age of the patients was 79.6±12.3 years, and 59.8% (n=67) were women. 86.6% of the patients had at least one comorbidity, and the three most common comorbidities were hypertension at 60.7%, diabetes at 27.7%, and coronary artery disease at 19.6%. The median length of hospital stay was seven days. 65.2% of the patients were admitted to the intensive care unit (ICU). The 28-day mortality of the patients is 14.3% (n=16), in-hospital mortality is 11.6% (n=13), out-of-hospital mortality is 2.7% (n=3) in the first 28 days, and 29-90 days mortality is 1%. It was .8 (n=2). 84.8% of the patients had at least one chronic drug use, and 24.1% had anticoagulant/antiplatelet drug use. While 50% of the patients had an intertrochanteric fracture, 49.1% had a femoral neck fracture. Only one patient had a subtrochanteric fracture. Univariate analysis was performed to identify risk factors for 28-day mortality. Univariate analysis of age, systolic and diastolic blood pressure, heart rate, respiratory rate, shock index, length of hospital stay, and ICU admission variables were significant. In these variables' multiple binary logistic regression analysis, heart rate, and hospitalization duration were statistically significant (p values 0.016 and 0.023, respectively). As the heart rate increased by one unit in the hospital, the risk of mortality increased by 1.24 times, while as the length of stay increased by one day, the risk of mortality increased by 1.65 times. When we examined the ROC analyses in predicting mortality, the shock index had the highest AUC value (AUC= 0.988 p=<0.001). When the shock index was >0.76, its sensitivity in predicting mortality was 100%, and its specificity was 90.6%. When we evaluated heart rate performance in predicting mortality, the AUC value was 0.975 and was statistically significant ($p<0.001$). When the heart rate was >99 beats/min, sensitivity was 100%, and specificity was 84.37%.

Discussion

Risk factors for hip fractures include advanced age (>75 years), lack of basic daily living activities, cognitive impairment, malnutrition, high ASA (American Society of Anesthesiologists) score, time of operation and presence of anemia before the operation, as well as factors such as vitamin D deficiency, muscle strength and hyperparathyroidism, which are important risk factors for 1-year mortality after hip fracture (5).

Proximal femur fractures are a serious complication and a major health problem, especially in elderly patients. With the increase in life expectancy in the general population, the number of trauma patients, especially in the elderly, is also increasing. Studies have estimated that femur fractures will double worldwide from 1990 to 2025 and will double again by 2050, with an interval of 7.3 to 21.3 million fractures worldwide. Proximal femur fractures, in particular, have high morbidity and mortality, as well as very high direct costs due to long hospital stays and rehabilitation. Detailed epidemiological assessment provides a valuable resource for stakeholders in the health system to determine risk factors affecting morbidity and mortality, determine therapeutic procedures and prevention strategies, and predict future developments (6).

Conclusion

In our study, elevated heart rate and prolonged hospital stay were risk factors for mortality. The shock index had the highest ability to predict mortality. Our study is a guiding study for emergency department physicians who make the initial evaluation of patients with upper-end femur fractures. Additionally, our study is a pioneering study conducted in the emergency department to determine the types of proximal femur fractures.

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A Hematologıcal And Hepatologıcal Approach: A Rare And Unexpected Of A Pancytopenıa Case Wıth Portal Veın Thrombosıs

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Introductıon

Portal vein thrombosis (PVT) refers to the partial or complete occlusion of any segment of the portal vein. The most common underlying mechanisms contributing to PVT include hypercoagulopathy in patients with liver cirrhosis, malignancy, acquired prothrombotic disorders, or inflammatory conditions. The estimated overall prevalence of PVT is low (approximately 1%); however, studies indicate that patients with cirrhosis or underlying malignancy are at increased risk, with some studies reporting PVT prevalence between 10% and 26% in this population (1,2). Nevertheless, in cases of chronic, gradual thrombus development or limited thrombus extension, PVT may remain asymptomatic.The pathophysiology of PVT is related to the disruption of Virchow's triad, where increased venous stasis, endothelial injury, and hypercoagulable states predispose patients to thrombus formation. PVT can also be categorized based on its etiology as acute or chronic and as cirrhotic or non-cirrhotic (1).Various local or systemic etiologies can contribute to the development of PVT, often involving multiple factors. The clinical presentation of PVT differs depending on whether it is acute or chronic and on the extent of collateral circulation development. Common symptoms of acute PVT include abdominal pain, diarrhea, rectal bleeding, abdominal distension, nausea, vomiting, anorexia, fever, lactic acidosis, sepsis, and intestinal ischemia with obstruction. In contrast, chronic PVT may be entirely asymptomatic or manifest with splenomegaly, pancytopenia, varices, and rarely, ascites (3).

Case Presentatıon

A 42-year-old male of foreign nationality was referred to our emergency department for anemia. The patient had no known medical history or prior medical follow-up. He initially presented to another emergency department with a complaint of scrotal swelling that had persisted for 3 days. Ultrasonographic imaging performed at the external center revealed physiological levels of scrotal fluid and edema of the scrotal skin and spermatic cord. His complete blood count showed hemoglobin at 5.5 g/dL, prompting referral for further investigation and treatment of anemia.Upon admission, the patient's vital signs were within normal limits. Physical examination revealed pretibial edema (+2) in both lower extremities, widespread ascites, scrotal edema, and basal crackles in both lungs. The complete blood count results were as follows: hemoglobin (HGB): 5.5 g/dL, platelet count (PLT): 108 × 10^3/μL, white blood cell count (WBC): 3.84 × 10^3/μL, mean corpuscular volume (MCV): 102 fL, and mean corpuscular hemoglobin (MCH): 30 pg. Coagulation tests showed INR: 1.49, PT: 16.7 s, APTT: 22.8 s, and D-dimer: 9.29 μg/mL. Biochemical parameters were within normal ranges except for an albumin level of 19 g/L. Contrast-enhanced abdominal CT revealed a normal-sized liver, splenomegaly, and ascites. Given these findings, the patient was hospitalized for further evaluation and treatment.During hospitalization, a detailed ultrasound examination of the portal system was performed, revealing the following: "The main portal vein measured 16 mm. Findings consistent with chronic thrombosis with cavernous transformation were observed in the right and left portal vein branches." Consequently, the patient was referred for gastroenterological management.

Dıscussıon

The presented case involves a 42-year-old male patient. One of the challenges of this case was the patient's refugee status, which limited communication. Even with secure communication, the absence of any medical history further complicated the case. While

obtaining a thorough anamnesis is crucial, its feasibility in the emergency department is debatable. Another important consideration is the holistic assessment of the physical examination findings. In this case, the initial presenting symptom of testicular swelling led to scrotal ultrasonography, which had limited diagnostic value. Laboratory findings revealed pancytopenia, initially suggesting a purely hematological condition. However, a broader multidisciplinary approach was essential, making this case a valuable example.
Although portal vein thrombosis may seem rare, its prevalence among cirrhotic patients ranges from 4.4% to 15% and accounts for approximately 5% to 10% of all portal hypertension cases (4). While PVT can be asymptomatic, it has been associated with complications such as pancytopenia, as documented in the literature (3). Although rare, pancytopenia has also been observed in pediatric patients with PVT (5). These findings suggest that pancytopenia should be carefully considered in patients with portal vein thrombosis.

Conclusıon

Anemia is a common finding in emergency departments and can have numerous etiological causes. The diagnosis and treatment should not focus solely on anemia but require a comprehensive evaluation of all clinical and laboratory findings. Pancytopenia, defined as a reduction in all three blood cell lines, may present with symptoms related to anemia, leukopenia, or thrombocytopenia; however, it can also be asymptomatic. It is not a disease itself but a manifestation of an underlying condition affecting the bone marrow or peripheral blood cells (6). Thus, evaluating pancytopenia should include not only hematological but also systemic considerations, as illustrated in this case. Although extensive diagnostic testing may seem resource-intensive, the primary concern should always be the patient's well-being.

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9212

Concurrent ischemic stroke and st-elevation myocardial infarction: a lethal confluence in stroke-heart syndrome – case report and clinical implications

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Introductıon

Stroke is a clinical syndrome caused by the interruption of blood flow to a specific region of the brain. It is caused by 85% vascular occlusion and 15% vascular rupture (hemorrhage, direct cellular trauma, mass effect, increased intracranial pressure, and/or release of biochemical toxins) (1). Cardiac complications are frequent in the initial few days following ischemic stroke, and patients develop signs of myocardial injury, cardiac dysfunction, and arrhythmias, which are frequently overlapping (2). This has been termed SHS (3). We present a case of concurrent AIS and STEMI.

Case

A case of an 83-year-old female with diabetes, hypertension, and a prior ischemic stroke was taken to the district emergency room following syncope. The patient with a Glasgow Coma Scale score of 6 was intubated and transferred to a tertiary emergency center. Blood pressure on arrival was 170/100 mmHg and pulse 70/min. The ECG showed ST-segment elevation in inferior leads, ST depression in lateral leads, and T-wave inversion in V4-6 leads. Computed tomography of the brain revealed no hemorrhage, but diffusion-weighted MRI (DWI) showed extensive acute diffusion restriction in the left cerebral hemisphere. This was a diagnostic of left MCA infarction with myocardial infarction. Laboratory results were creatinine 1.69 mg/dL, high-sensitivity troponin 3583 pg/mL, and CK-MB 121 ng/mL. Coronary angiography later revealed LAD occlusion. The patient was monitored in the intensive care unit but passed away on the seventh day.

Dıscussıon

AIS and its cardiac complications, together constituting SHS, highlight the intricate bidirectional interplay between cerebral and cardiovascular pathophysiology. SHS occurs in the form of myocardial injury, arrhythmias, or cardiac dysfunction days to weeks following stroke and carries a mortality of as high as 20% (1,2). The pathophysiology of SHS is multifaceted, with roles for autonomic imbalance, neurohormonal activation, and systemic inflammation (3). One primary mechanism is the hyperactivation of the sympathetic nervous system via ischemic damage to brain regions regulating autonomic function, such as the insular cortex (4). This results in a catecholamine surge with consequent myocardial stunning, microvascular dysfunction, and arrhythmogenic

substrates (5). The elevated serum troponin level, as in the case of our patient (3583 pg/mL), is a marker for SHS-related myocardial injury, though the failure to distinguish between primary acute coronary syndrome and neurogenic injury remains a challenge (6).

In this case, the concurrent STEMI and AIS highlighted the severity of SHS. Coronary angiography confirmed LAD occlusion, either because of pre-existing atherosclerotic disease worsened by stroke-induced sympathetic stress or de novo plaque rupture due to systemic inflammation (7). The overlap in timing of these events is in accordance with the fact that cardiovascular complications are most common in the first month following stroke, peaking in the second week (8). This timing coincides with maximal inflammatory cytokine release and autonomic instability, both of which render the heart vulnerable (9).

Early SHS diagnosis is crucial. The 2019 AHA/ASA guidelines highlight dual evaluation for cerebral and coronary ischemia in patients who have overlapping symptoms (10). Therapeutically, there is difficulty because anticoagulants or antiplatelets for secondary stroke prevention may increase the risk of bleeding with coronary interventions. Our patient's fatal result , despite revascularization, underscores the prognostic severity of SHS, particularly in elderly patients with comorbidities like diabetes and hypertension that exacerbate both cerebrovascular and cardiovascular risk (2).

Due to the high incidence and mortality of SHS, AIS patients must be subjected to aggressive cardiac monitoring and examination for stroke-heart syndrome to prevent adverse outcomes.

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9441

Atrial Fibrillation Complicated by Spleen Infarction Due to Suboptimal Anticoagulation With Aspirin

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Introduction

Atrial fibrillation is a significant risk factor for thromboembolic events, particularly in elderly populations. Several clinical scoring systems have been developed to guide prophylactic management, with the CHA□DS□-VASc score being one of the most widely used. The CHA□DS□-VASc score takes into account various factors such as Congestive heart failure, Hypertension, Age ≥75 years, Diabetes mellitus, Stroke, Vascular disease, Age 65–74 years, and Sex category. Each of these risk factors contributes to the patient's overall risk score, which helps guide anticoagulation therapy decisions. A higher CHA□DS□-VASc score indicates a greater risk of thromboembolic events, influencing treatment strategies and patient management. It is endorsed by both the European Society of Cardiology (ESC) and the American College of Cardiology/American Heart Association (ACC/AHA) for guiding anticoagulation decisions (1,2).

The role of aspirin in the prevention of thromboembolic events in atrial fibrillation remains clinically limited and is primarily reserved for patients at low risk, defined as CHA□DS□-VASc ≤ 1 (3). While early trials, such as SPAF-1, provided the initial rationale for aspirin use in prophylaxis (4), recent studies have demonstrated its limited efficacy. In a recent metanalysis, aspirin has been associated with only a non-significant one-fifth relative reduction in atrial fibrillation-related stroke risk when compared to placebo, highlighting its diminished role in contemporary stroke prevention strategies (5).

The spleen receives a substantial blood supply, accounting for approximately 5% of the total cardiac output (6). Its inherent vascular architecture also predisposes it to thromboembolic events, similar to other highly perfused organs such as the kidney and brain. Embolic events resulting in splenic infarction can arise from a wide range of aetiologies, including decreased perfusion, hypercoagulable states, and infectious causes such as infective endocarditis or sepsis (7). Additionally, malignant conditions, such as pancreatic tumours invading the splenic artery, may also contribute to embolism (7).

This case report presents an elderly patient with a history of atrial fibrillation and multiple comorbidities who developed an acute

thromboembolic event, resulting in partial splenic infarction. Notably, the patient was under rate control with the non-dihydropyridine calcium channel blocker diltiazem, which is also classified as an antiarrhythmic agent for atrial fibrillation, and was receiving antithrombotic therapy with aspirin. An external cardiology evaluation conducted two months prior to the event had maintained this management approach. This case underscores the limitations of aspirin as monotherapy for the prevention of thromboembolic events in patients with atrial fibrillation, highlighting the need for more effective anticoagulation strategies in high-risk individuals.

Case

An 81-year-old male presented to the emergency department with a three-day history of left-sided abdominal pain. The patient reported that the pain differed from his previous episodes of abdominal discomfort in both intensity and localisation. He had a history of chronic constipation, which typically caused milder pain and was effectively managed with conservative, medical and minimally invasive interventions, including dietary regulation, high enemas, motility-regulating treatments, and lactulose. Of note, the severity of the pain led to a marked reduction in the patient's oral intake of both food and fluids. There were no additional gastrointestinal symptoms such as diarrhoea, acute constipation, or vomiting. The patient also denied any urinary symptoms, including dysuria, haematuria, or a history of nephrolithiasis.

His medical history included hypertension, type 2 diabetes mellitus, atrial fibrillation, benign prostatic hyperplasia, and chronic constipation. Furthermore, he underwent hip arthroplasty approximately 7 years ago and cholecystectomy 30 years prior. His regular medications consisted of diltiazem, amlodipine, metformin, dutasteride, and acetylsalicylic acid.

On admission, physical examination revealed an alert patient (Glasgow Coma Scale score: 15/15) who was haemodynamically stable. His vital signs were as follows:

Blood pressure: 200/103
Heart rate: 91
Temperature: 36,1
Oxygen saturation: 98
Respiratory Rate: 21

Cardiac auscultation revealed normal heart sounds, and pulmonary examination was unremarkable. Abdominal and lower extremity examinations demonstrated a soft, non-tender abdomen and calves. Notably, even with repeated deep palpation, no discomfort suggestive of guarding or rebound tenderness was elicited. This absence of peritoneal signs, despite patient's own expression significant pain, raised concern for ischemia-related aetiologies, prompting us for early imaging for further evaluation.

Laboratory results demonstrated a normal leukocyte count of 8.48 x 10^9/L, haemoglobin level of 12.86 g/dL, and platelet count of 211 x 10^9/L. The patient's biochemistry panel, including kidney and liver function tests, was unremarkable. Blood glucose levels were well-controlled. Amylase and lipase were within normal limits, effectively excluding the possibility of spontaneous pancreatitis Although the patient did not report typical infectious symptoms, such as fever or malaise, his C-reactive protein (CRP) level was elevated at 59.9 mg/dL. Considering the possibility of an atypical cardiac aetiology, an electrocardiogram (ECG) and troponin-I levels were also obtained. The ECG revealed findings consistent with atrial fibrillation, while troponin-I levels were within the normal range, effectively ruling out acute myocardial injury. Urinalysis was also normal. As an initial imaging modality, an abdominal erect X-ray showed no significant pathologies. To exclude lower lobe parenchymal and pleural pathologies that could contribute to abdominal pain, a posteroanterior chest X-ray was performed, which revealed no significant findings.

The patient's initial management included the administration of the antihypertensive amlodipine 10 mg orally and intravenous hydration with 0.9% saline. Given the non-specific laboratory findings, unremarkable initial imaging results, and the disproportionate pain experienced by the patient in relation to physical examination findings, a contrast-enhanced abdominal CT scan was ordered for further evaluation. During the wait for the imaging results, the patient's hydration was continued. Upon receipt of the initial CT report, a diagnosis of splenic infarction was confirmed.

Early consultation with the on-call surgical and cardiology teams was conducted, and the anaesthesia team subsequently accepted the patient for admission to the general intensive care unit (ICU). Anticoagulation, antihypertensive, and antiarrhythmic therapies were promptly initiated. Bedside echocardiography revealed biatrial dilatation, predominantly affecting the left atrium, with a 60% ejection fraction. Enoxaparin sodium was administered for anticoagulation, while blood pressure control was achieved through the use of furosemide and glyceryl trinitrate. Diltiazem was continued as a rate-controlling agent, and pantoprazole was prescribed as a proton pump inhibitor. Further detailed radiological evaluation identified peripheral fat stranding in both kidneys, along with bilateral renal cysts. Additionally, a contralateral renal calculus was noted, indicating concurrent nephrolithiasis, which was considered an incidental finding. Given the potential concern for pyelonephritis, antimicrobial therapy with ceftriaxone was initiated following a consultation with the urology team.

A significant finding on subsequent detailed radiological reassessment was the tortuous course of the splenic artery, accompanied by calcifications at the arterial level. Furthermore, calcified atheromatous plaques were identified in the coronary arteries. These vascular abnormalities may have contributed to the embolic event, further emphasising their potential role as risk factors for splenic infarction in patients with atrial fibrillation.

On the fifth day of hospitalisation, the patient was transferred to the general ward from ICU. Initially, during the emergency examination, the patient did not report any chronic orthopaedic or rheumatological conditions. However, during follow-up, he developed cellulitis on his right ankle and swelling in his toe, prompting the addition of colchicine into his discharge medication list by the internal medicine team. After an additional five days of inpatient management, the patient was discharged with a revised long-term treatment

plan. His anticoagulation therapy was transitioned to rivaroxaban 20 mg daily. Torsemide was introduced to optimise blood pressure control, complementing his pre-existing antihypertensive regimen. Acetylsalicylic acid was discontinued.

Discussion

The patient presented to our emergency department with progressively worsening left-sided abdominal pain, with splenic infarction historically considered a rare aetiology for such a presentation (7, 8). Prior to the advent of modern diagnostic techniques, previous studies reported that up to 90% of splenic infarction diagnoses were made post-mortem (9). However, with the increased availability of CT imaging and a lowered threshold for its use in acute settings, splenic infarction is now recognised in a broader range of clinical presentations and pathogenic processes (7). Despite these advancements, no standardised clinical guideline or consensus exists regarding the diagnostic pathway for splenic infarction (10). This lack of a clear diagnostic approach not only complicates detection and treatment but also highlights the need for improved strategies to prevent and reduce risks associated with this condition.

Atrial fibrillation is a significant comorbidity and a notable risk factor in elderly patients with concurrent underlying conditions. It may contribute to splenic infarction, which is primarily associated with thromboembolic events (9), though other causes should also be considered (11). In our case, the patient had a history of atrial fibrillation and was found to have splenic artery deformation, along with calcific plaques in both the splenic and coronary arteries. The aetiology of splenic infarction is typically multifactorial, with contributing factors rather than a singular event being the primary cause (7). Therefore, in this case, the thromboembolic infarction of the spleen was likely aggravated by the patient's comorbidities, including atrial fibrillation and vascular abnormalities.

Aspirin has been utilised as a long-term therapeutic agent for centuries. Initially employed for its antipyretic and anti-inflammatory properties due to its inhibitory effect on prostaglandin synthesis, its role expanded in the 20th century following the discovery of its antiplatelet effects (12). This discovery led to aspirin's widespread adoption in the prevention and management of cardiovascular and cerebrovascular diseases. Historically, it has even been referred to as a 'wonder drug' in early medical literature, highlighting its perceived therapeutic significance (13). However, as with all pharmacological agents, alternative antiplatelet therapies have been developed to address the limitations of aspirin, once regarded as a 'miracle drug,' in clinical practice.

In patients with atrial fibrillation, the initiation of anticoagulation therapy is typically guided by an individualised assessment of thromboembolic risk using the CHA□DS□-VASc scoring system. Concurrently, the risk of major bleeding complications should be evaluated utilising validated tools such as the HAS-BLED score, a widely established instrument for evaluating haemorrhagic risk in this population (14). This score incorporates several key clinical factors: Hypertension, Abnormal renal/liver function, Stroke history, Bleeding tendency or predisposition, Labile international normalized ratio (INR), Elderly status, and the concurrent use of Drugs or alcohol. The HAS-BLED score provides a comprehensive risk assessment, guiding clinicians in balancing the benefits of anticoagulation therapy with the potential for bleeding. A higher HAS-BLED score is indicative of an increased bleeding risk, prompting closer monitoring and, in some cases, reconsideration of anticoagulation therapy.

Patients deemed eligible for anticoagulation are typically initiated on either direct oral anticoagulants (DOACs) or warfarin therapy (14). Of particular relevance, aspirin is no longer recommended as a first-line agent for thromboembolic risk reduction in atrial fibrillation. When compared to warfarin, aspirin demonstrates reduced efficacy due to the unique haemodynamic characteristics of the left atrial appendage, which, despite being classified as an arterial structure, exhibits venous-like flow patterns that promote fibrin-rich thrombus formation, rendering aspirin less effective (3). In a Japanese trial, aspirin (150–200 mg/day) was associated with worse outcomes, showing a primary outcome rate of 3.1% per year compared to 2.4% per year in the control group with no therapy in atrial fibrillation patients (15). In our case, the use of aspirin likely failed to prevent thrombus formation, potentially allowing migration of atrial-originated emboli, which led to partial tissue necrosis in the spleen. While calcified plaques in the splenic and coronary arteries may have contributed to the embolic event, they are unlikely to have been the primary cause, given their complex and controversial role in cardiovascular pathology. Although calcium scoring is positively correlated with cardiovascular disease, the stabilised nature of plaques may confer some protective effects, as evidenced by the inverse correlation observed between plaque density and cardiovascular risk (16). Furthermore, isolated splenic infarction attributable solely to splenic arteriosclerosis has been infrequently reported in the literature, with most cases involving additional contributory factors (7). This makes it highly unlikely that splenic calcification alone accounted for the embolic event observed in our case.

The newer generation of anticoagulants has shown significant potential to replace warfarin in the management of thromboembolic events (14). These agents offer improved safety profiles and exhibit more predictable pharmacokinetics, reducing the need for routine monitoring (17,18). These advancements position newer anticoagulants as promising alternatives for long-term anticoagulation therapy (3). However, in this context, aspirin no longer remains a strong contender. Some meta-analyses suggest that aspirin may no longer offer broader cardiovascular benefits and, in some cases, might even cause harm, particularly when evaluating its overall net risk-benefit ratio in terms of cardiovascular protection (19).

Conclusion

This case highlights the limitations of aspirin monotherapy in preventing thromboembolic events in patients with atrial fibrillation, emphasising that it should not be considered a primary prophylactic strategy, particularly in high-risk patients. Current evidence indicates that aspirin offers minimal, if any, protective benefit in reducing stroke risk in atrial fibrillation and may be associated with an unfavourable risk-benefit profile, given the growing body of evidence highlighting its insufficiency and potential contribution to embolic complications. This underscores the necessity of guideline-directed anticoagulation therapy, particularly in patients with elevated thromboembolic risk, to ensure optimal stroke prevention and patient outcomes.

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Evaluatıon Of Gdf-15 Bıomarker And Heavy Metal Levels In Acute Ischemıc Stroke Cases

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Introductıon And Objectıve:

Ischemic stroke is a major public health concern due to its high mortality and morbidity, necessitating rapid diagnosis and treatment. Currently, no universally accepted biochemical marker exists for stroke diagnosis. Growth differentiation factor-15 (GDF-15), also known as macrophage inhibitory cytokine-1, is a cytokine from the TGF-β superfamily. GDF-15 levels rise in pathological conditions and are linked to hypoxia, inflammation, oxidative stress, and oncogene activation. Previous studies suggest that serum GDF-15 levels may predict atherosclerotic events and are involved in tissue repair after cardiac injury. Additionally, imbalances in heavy metal concentrations may disrupt physiological homeostasis and contribute to stroke pathogenesis. This study aimed to evaluate the association between GDF-15 and heavy metal levels in acute ischemic stroke patients and assess the potential of GDF-15 as a prognostic biomarker.

Materıals And Methods:

The study enrolled 128 participants: 64 acute ischemic stroke patients and 64 healthy controls. Blood concentrations of 10 heavy metals were measured using inductively coupled plasma mass spectrometry. Serum GDF-15 levels were determined via ELISA (Elabscience, USA).

Results And Conclusıon:

There were no significant differences between groups regarding age, sex, or comorbidities. GDF-15 levels were significantly elevated in stroke patients ($p<0.001$). Lead (Pb) and mercury (Hg) concentrations were also higher in the stroke group. A positive correlation was found between NIHSS scores, GDF-15, and Pb levels. GDF-15 levels varied by age, sex, and stroke subtype. These findings indicate that GDF-15 is elevated in acute ischemic stroke and correlates with stroke severity and age. Certain heavy metals may play a role in stroke development. GDF-15 could serve as a valuable biomarker for evaluating stroke prognosis.

Keywords:

Acute Ischemic Stroke, GDF-15, Heavy Metals, NIHSS Score, Biomarker

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From Vision Loss To Death: The Dark Side Of Methanol Intoxication

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Introduction

Methanol is an alcohol obtained by distilling wood and is widely used as an industrial solvent due to its organic solvent properties (1). It is a colourless, toxic liquid at room temperature. Methanol poisoning primarily occurs through oral consumption of illegally produced alcoholic beverages, but can also occur accidentally or intentionally (2). Although methanol itself is not very toxic, it is metabolized in the body by alcohol dehydrogenase to toxic metabolites such as formaldehyde and formic acid, resulting in high anion gap metabolic acidosis. Formic acid also inhibits cytochrome c oxidase activity in mitochondria, causing cellular hypoxia and damage to retinal ganglion cells (3,4).

Symptoms of methanol poisoning usually appear within 12-24 hours after ingestion due to the slow metabolism of methanol to its toxic metabolites. Early symptoms include vision disturbances, abdominal pain, dizziness, nausea, vomiting, and headaches. If left untreated, it can lead to coma, blindness, gastrointestinal bleeding, putaminal hemorrhage and death in later stages. Some visual disturbances may improve, but permanent vision loss may also occur.

Case Report

Our patient was admitted to the emergency room with blurred vision approximately 36 hours after alcohol consumption. Based on history and clinical presentation, methanol poisoning was suspected. The patient's Glasgow Coma Scale (GCS) score was E4M6V5, blood pressure was 110/70 mmHg, pulse was 85 bpm, and temperature was 36.5°C. Laboratory findings showed that venous blood gas values were pH: 7.10, PCO2: 24 mmHg, PO2: 52 mmHg, HCO3: 12 mmol/L, lactate: 1.8 mmol/L, and base deficit -20.5; This indicated a high anion gap metabolic acidosis. Biochemical parameters were normal and the ethanol level was <10 mg/dl.

There was no pathology in central imaging. The patient was referred to related departments for consultation. During the insertion of the dialysis catheter in the emergency room, respiratory arrest developed. He was intubated and placed on mechanical ventilation. After respiratory arrest, his GCS score decreased to E1M1V1, his metabolic acidosis worsened and he became hypotensive, requiring admission to intensive care. Medical treatment included bicarbonate replacement, ethanol therapy via nasogastric tube at a loading dose of 1.8 cc/kg, and maintenance therapy at 0.45 cc/kg/hour due to the patient's history of chronic alcohol use. Ethanol levels were monitored and kept at therapeutic levels. Vasopressor doses were adjusted based on noninvasive blood pressure monitoring and folic acid was administered at a dose of 1 mg/kg.

Blood pressure regulation continued to be difficult despite vasopressor support. Hemodialysis was initiated but discontinued one hour later due to persistent hypotension despite high-dose inotropic agent support. The patient remained hypotensive and was pronounced dead 12 hours after admission to the intensive care unit.

Discussion

Methanol generally goes through a latent period ranging from 1 to 72 hours before showing its toxic effects. The onset of symptoms varies depending on the route of methanol intake. Methanol metabolism begins with its oxidation to formaldehyde by alcohol dehydrogenase, followed by oxidation to formic acid by aldehyde dehydrogenase. Although methanol itself is not toxic, its metabolites (particularly formic acid) cause permanent neurological damage. The toxic dose of methanol ranges from 20 to 250 ml. High mortality rates in methanol poisoning cases are often caused by delayed hospitalization and delayed diagnosis. Another difficulty is the lack of methanol level analysis in many hospitals in Türkiye, which leads to further delays in diagnosis. Symptoms usually appear within 12-24 hours and usually include central nervous system, gastrointestinal system and visual disturbances.

Since our hospital does not have the capacity to measure blood methanol levels, the patient's history is very important in detecting methanol poisoning if the patient is conscious. Treatment involves correcting metabolic acidosis, administering an antidote to prevent the formation of toxic metabolites, and using hemodialysis to remove methanol and its toxic byproducts from the blood (5). Prognosis is significantly affected by the patient's pH at the time of admission (6,7). Severe acidosis, coma, hypotension, and bradycardia are late findings and indicate a poor prognosis (7,8). Patients who present with preserved consciousness and hyperventilation tend to have better outcomes. Clinical outcomes are more closely related to the severity of acidosis than to methanol concentration. Concomitant ethanol intake may delay the onset of symptoms. The general therapeutic approach for methanol poisoning includes gastric irrigation, ethanol administration, fomepizole, hemodialysis, folate supplementation, and thiamine administration. Ethanol is the classic first-line treatment for methanol poisoning and can be administered orally, intravenously, or via nasogastric tube (9). Ethanol has 10 times greater affinity for alcohol dehydrogenase than methanol and prevents the formation of toxic metabolites. Addi-

tionally, frequent blood sugar monitoring is necessary to prevent hypoglycemia in these patients.

Conclusion

If left untreated, methanol toxicity can be fatal. Methanol poisoning continues to be an important cause of death in our country due to its easy availability and use in counterfeit alcoholic beverages. Physicians should suspect methanol poisoning in patients with altered consciousness and unexplained high anion gap metabolic acidosis, even if there is no clear history. Rapid and effective treatment significantly reduces mortality and morbidity.

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Hidden Threats of Motor Vehicle Accidents: First Rib Fractures

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Introductıon:

First rib fractures are relatively rare compared to other rib fractures due to the rib's well-protected anatomical position in the upper thoracic cavity. These fractures are typically associated with high-energy trauma and may accompany multisystem injuries, including subclavian artery and aortic damage. Given their association with significant morbidity and mortality, early diagnosis and proper management of first rib fractures are essential. In this case report, we present an isolated traumatic first rib fracture without any major thoracic organ injury, aiming to emphasize its clinical importance.

Case Presentatıon:

A 56-year-old male was brought to our Emergency Department with ambulance following a motorcycle accident. Upon arrival, his vital signs were stable, and he was conscious, oriented, and cooperative. The patient reported severe pain in the left shoulder region. Physical examination revealed a 3x2 cm area of dermabrasion on the forehead and restricted range of motion in the left shoulder joint. Emergency imaging identified bilateral pulmonary contusions and non-displaced anterior and posterior fractures of the left first rib, without any other pathological findings. The patient was evaluated by the thoracic surgery team, admitted for observation, and subsequently discharged from the emergency department.

Dıscussıon:

The anatomical location of the first rib provides substantial protection and stability, making its fracture a critical indicator of high-impact trauma. Most first rib fractures are linked to motor vehicle accidents and are often accompanied by intrathoracic injuries such as pulmonary contusion, pneumothorax, or hemothorax. Although rare, first rib fractures may signal the presence of severe thoracic and extrathoracic injuries. Due to the overlapping structures in this region, these fractures are often difficult to detect on plain radiographs. Therefore, computed tomography (CT) remains the most sensitive and specific imaging modality for identifying first rib fractures. Emergency physicians must recognize the potential severity of first rib fractures. A multidisciplinary approach, including early diagnosis, appropriate management, and close monitoring—especially in an intensive care setting when necessary—can significantly reduce the risk of complications and mortality.

KEYWORDS: First Rib Fracture, Isolated İnjury, Motorcycle Accident

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Deep Neck Infection: A Case Report

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Introduction

Deep neck infections (DNIs) are defined as bacterial infections of the soft tissues located within the fascial compartments of the neck. Due to their potential for life-threatening complications, they require urgent medical intervention. DNIs commonly develop secondary to upper respiratory tract infections (URTIs), dental infections, trauma, or post-operative complications following neck surgery. The etiology of these infections is often complex and typically polymicrobial, involving a combination of aerobic and anaerobic bacteria.

The anatomy of the neck, containing multiple vital structures within a compact area, predisposes to rapid spread of infection. Fascial planes and spaces in the neck allow for progression of infection into deeper tissues and even into the mediastinum. DNIs can manifest in various clinical entities such as retropharyngeal abscess, parapharyngeal abscess, prevertebral abscess, and Ludwig's angina.

Clinical presentation of DNI typically includes neck swelling, pain, fever, dysphagia, and trismus. However, the symptoms are often nonspecific and may overlap with other more common causes of neck pain, making early diagnosis and treatment challenging. Hence, a high index of clinical suspicion and appropriate imaging modalities—such as ultrasound, contrast-enhanced computed tomography (CT), or magnetic resonance imaging (MRI)—are critical for accurate diagnosis.

This report presents the case of a 51-year-old male who developed a deep neck infection three days after experiencing symptoms of an upper respiratory tract infection.

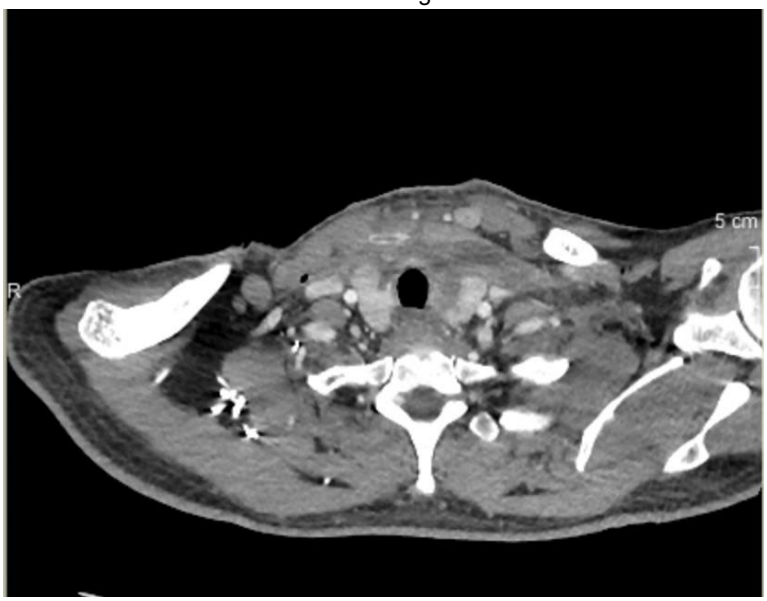
Case Presentation

A 51-year-old male patient with no known chronic illnesses presented to our emergency department with complaints of neck swelling and dysphagia. The patient had received treatment for an upper respiratory tract infection three days prior. On examination, he was conscious, oriented, and cooperative, with stable vital signs. Physical examination revealed mild restriction of mouth opening, fullness of the posterior right lateral oropharyngeal wall, diffuse tenderness and erythema in the neck, no fluctuance, limited right-sided neck movements, and the presence of torticollis.

Laboratory findings showed leukocytosis with a white blood cell count of 26.32 × 10^3/μL, neutrophils at 24.23 × 10^3/μL, hemoglobin at 15.1 g/dL, and a markedly elevated C-reactive protein (CRP) level of 266.88 mg/L. No other significant pathological findings were noted.

Contrast-enhanced CT of the neck and thorax demonstrated a deep neck infection characterized by soft tissue edema and air densities extending from level 2 through levels 3, 4, and 6 on the right side of the neck, adjacent to the hypopharynx. The lesion extended posteriorly to the submandibular gland and prevertebral space, with involvement of the carotid neurovascular bundle at level 2, and reached medially and anteriorly to the right thyroid lobe. The infected area measured approximately 4 × 2 cm in its largest axial dimension and extended over a segment of roughly 9 cm. No well-defined abscess formation was observed. No thrombus or wall thickening was seen in the right jugular vein.

The patient was diagnosed with a deep neck infection and consulted with the otorhinolaryngology (ENT) department. He was admitted to the ENT service for further management and treatment.



Discussion

Deep neck infections are serious infections with high potential for morbidity and mortality due to their location within the complex

anatomical fascial compartments of the neck. The polymicrobial nature of DNIs typically involves both aerobic and anaerobic bacteria. Common predisposing factors include URTIs, dental infections, trauma, and surgical procedures involving the neck.

Early diagnosis is crucial for effective management. However, clinical signs and symptoms such as neck pain, swelling, fever, and dysphagia are often non-specific and may initially be mild. As the infection progresses, these symptoms become more pronounced. Therefore, a high clinical index of suspicion, thorough physical examination, and prompt laboratory and imaging assessments are essential.

Inflammatory markers such as leukocytosis and elevated CRP are common laboratory findings that support the presence of infection. Imaging techniques—particularly contrast-enhanced CT and MRI—are indispensable in evaluating the extent of infection and involvement of critical anatomical structures. These modalities also help determine the need for surgical intervention.

First-line treatment of DNIs involves empirical administration of broad-spectrum intravenous antibiotics. Antibiotic therapy plays a key role in controlling the infection and preventing complications. However, if abscess formation occurs or if the infection progresses, surgical drainage may become necessary. A multidisciplinary approach involving emergency medicine, infectious diseases, radiology, and otorhinolaryngology specialists is essential for optimal management.

The prognosis of DNIs largely depends on early recognition and appropriate treatment. Given the potential for rapid progression and life-threatening complications, clinical awareness must be heightened. In patients presenting with neck swelling and pain following a recent upper respiratory infection, the possibility of DNI should always be considered. Prompt diagnosis and timely intervention can significantly reduce morbidity and mortality.

In conclusion, the management of deep neck infections requires early diagnosis, rapid initiation of treatment, and a multidisciplinary approach. Enhancing clinical awareness and utilizing advanced diagnostic techniques are critical to preventing complications and improving patient outcomes. As demonstrated in this case, with early recognition and proper management, DNIs can be effectively treated.

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Retained foreign body not detected by imaging methods

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Introduction

Retained foreign body accounts for 7% to 15% of emergency department visits, and it has been found that 38% of these foreign bodies are overlooked during the initial evaluation (1). In the United States, 37% of malpractice lawsuits related to emergency departments have been linked to foreign bodies (2). Materials such as wood, acrylic, and some plastics have densities similar to the surrounding soft tissues, making them difficult to visualize, and it has been reported that only 15% of wooden foreign bodies are

detected in plain radiographs (3).

In this case presentation, we aimed to present a case of a retained foreign body in the gluteal region of an 11-year-old male child that was overlooked in imaging methods.

Case report

An 11-year-old male child was brought to the emergency department by his family after reporting that a splinter had entered his left inner hip area while sliding on a slanted board they had placed to play. The patient's general condition was good, with clear consciousness, cooperation, and orientation. His vital signs were as follows: blood pressure 130/70 mmHg, pulse 76/min, respiratory rate 18/min, body temperature 36.5°C, and SaO2 99%. A lesion potentially corresponding to the point of entry was identified at the site of pain, as indicated by the patient, in the left inner hip region (Figure 1). However, no foreign body or hardness was felt upon examination. A superficial ultrasound (US) examination showed no pathology at the lesion site or in the surrounding area. As the patient's severe pain persisted, a pelvic computed tomography (CT) scan was performed. The pelvic CT scan revealed air densities in the region of the skin lesion (Figure 2), but no other pathology was found. Later, as the patient's complaints continued, the area was re-examined along the tract of air densities starting from the skin lesion. During this examination, a palpable hardness was found under the skin on the upper outer side of the right hip. A superficial US examination was requested at the site of the palpable hardness, but no pathology was identified again. Despite the lack of pathology detected in imaging methods, the palpable hardness and the patient's severe pain prompted a small incision to be made at the site of the hardness (Figure 3). Upon finding a foreign body, the child was consulted to the pediatric surgery department. A foreign body measuring approximately 35x0.8 cm was removed during the procedure performed by the pediatric surgery team (Figure 4). The patient was followed and subsequently discharged.

Figure 1. In the initial examination, the area where the patient reported pain, which was considered as the possible entry site, was identified.

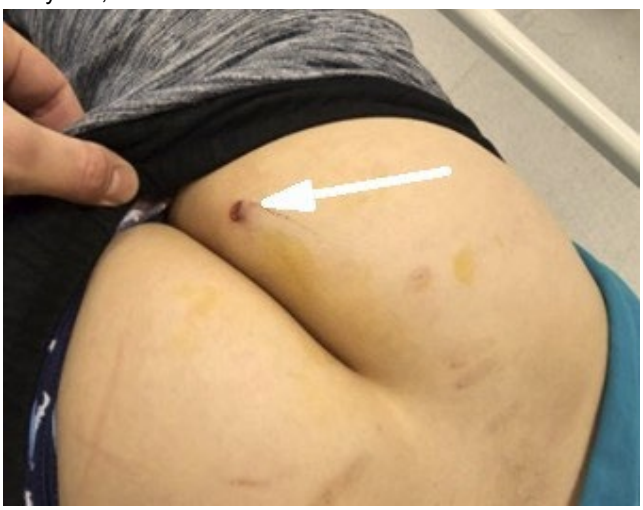


Figure 2. The pelvic CT scan revealed air densities in the region of the skin lesion.

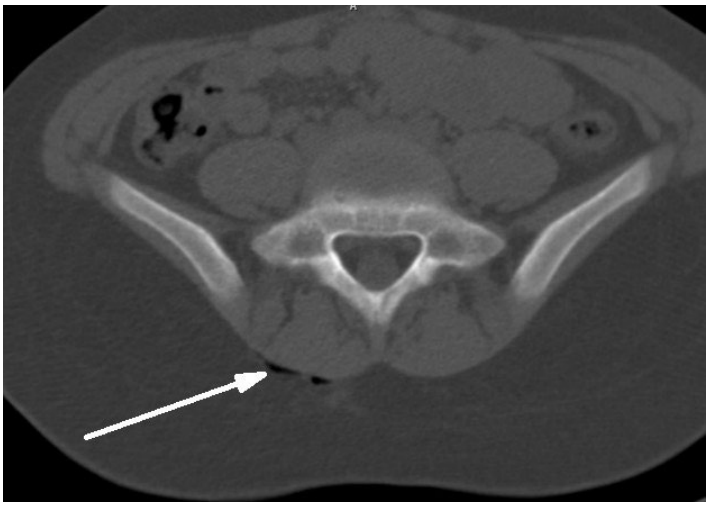


Figure 3. The incision made over the area with hardness during the examination.

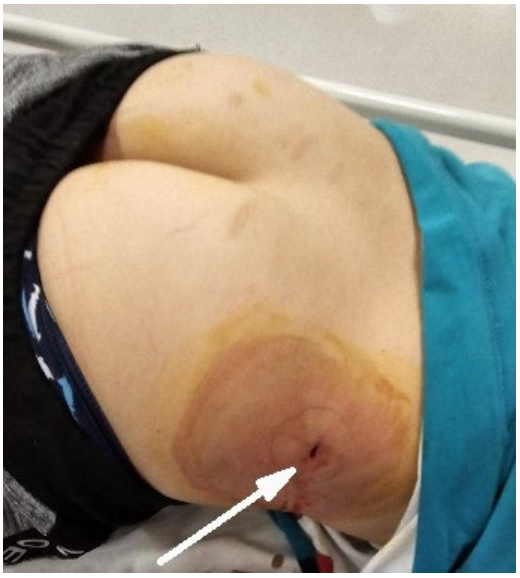
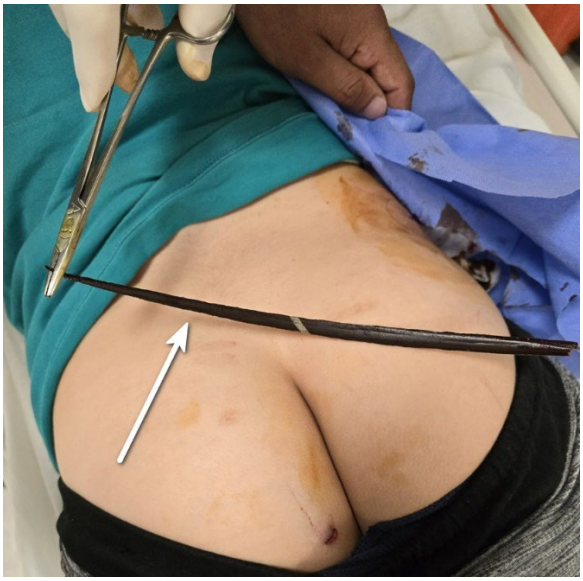


Figure 4. Removed foreign body.



Discussion

In patients presenting with suspicion of a foreign body, a detailed history and physical examination are crucial during the initial assessment. Metals, glass, and wood are the most commonly encountered foreign bodies (4). Metal objects are easily detected in plain radiographs. However, glass and wood are difficult to visualize on radiographs, and glass alone accounts for 50% of foreign bodies that are overlooked despite physical examination and radiography (5). In our case, the foreign body was a piece of wood, which was not detected through imaging methods at the initial stage but was diagnosed later based on suspicion following a careful physical examination.

In cases of injury within the first 24 hours, the entry site can usually be easily seen, and intervention is more straightforward, making the ideal time for diagnosis and treatment within the first 24 hours (6). Delayed treatment can lead to complications such as infection, delayed wound healing, and loss of function (7). Therefore, early diagnosis is essential; failure to make a diagnosis may lead to malpractice claims and compensation lawsuits (5). In our case, the patient presented to the hospital 2 hours after exposure to the foreign body, and the intervention was performed.

Bedside US is an easily accessible, radiation-free, inexpensive, and safe imaging method commonly used in emergency department practice. Since most wooden foreign body insertions result from low-energy trauma, they are typically superficial. US is one of the best imaging techniques for diagnosing superficially located foreign bodies (8,9). However, in our case, the foreign body could not be detected using US.

For detecting deeper foreign bodies CT may be used. CT has been shown to be the best imaging method for plastic, glass, and stone foreign bodies (10). In our case, following the failure to identify the foreign body on USG, a CT scan was performed based on the patient's history and physical examination findings. However, the initial interpretation of the CT scan was normal, with the exception of air densities. Afterward, a foreign body was detected during incision at the site of the palpable hardness, and the foreign body was removed.

Conclusion

In cases of retained foreign body, although imaging methods such as US and CT are important for diagnosis, it should be remembered that in rare cases where imaging methods are not helpful, a thorough history and physical examination remain the most effective approach.

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A Case Of Hyperlıpıdemıa Developıng Pulmonary Embolısm Durıng Plasmapheresıs

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Introductıon

Venous thromboembolism (VTE), clinically manifesting as deep vein thrombosis (DVT) or pulmonary embolism (PE), is the third most common acute cardiovascular syndrome worldwide after myocardial infarction and stroke (1). Epidemiological studies report annual incidence rates for PE ranging from 39 to 115 per 100,000 population (2). While major risk factors include lower extremity fractures, atrial fibrillation, heart failure and major trauma, moderate risk factors comprise blood product transfusions, intravenous catheters, hormone replacement therapies, infections, obesity and hypercholesterolemia (3).

In cases of resistant hypercholesterolemia, plasmapheresis may be employed as a therapeutic modality (4). However, reports regarding the risk of thrombosis during plasmapheresis are limited. İn this article, we present a case of pulmonary embolism that developed during plasmapheresis in a patient with hyperlipidemia.

Keywords: Pulmonary embolism, Hypercholesterolemia, Plasmapheresis

Case Report

A 56-year-old female patient was referred to the emergency department by the cardiology department after pulmonary embolism was detected on coronary computed tomography angiography (CTA). The patient was asymptomatic at the time of admission. One month earlier, she had been hospitalized in the endocrinology department due to hyperlipidemia. During hospitalization, therapeutic plasmapheresis was planned using 10 units of fresh frozen plasma. According to the patient's history, a brief episode of syncope occurred during the sixth unit of the plasmapheresis session, leading to the termination of the procedure.

Subsequently, a coronary CTA was performed by the cardiology team. Twenty days later, the patient returned to the outpatient clinic for the evaluation of the CTA results, which revealed a pulmonary embolism involving the left main pulmonary artery (Figure 1). The patient was then referred to the emergency department by the cardiology physician.

The patient who applied to the emergency department has no active complaints. Her past medical history included diabetes mellitus, hypertension, and hypercholesterolemia. She was on regular medications including Galvus Met, Jardiance, Ecopirin, Tansifa Plus, and Lipanthyl. Vital signs were stable; blood pressure 133/85 mmHg, heart rate 80 bpm, oxygen saturation 95% on room air, and respiratory rate 16 breaths/min. Electrocardiogram (ECG) showed normal sinus rhythm.

Laboratory results revealed blood glucose of 306 mg/dL, pH 7.37, pO□ 39.7 mmHg, pCO□ 43.7 mmHg, bicarbonate 24 mmol/L, and lactate 2.3 mmol/L with normal troponin and other parameters.

A bilateral lower extremity venous doppler ultrasound was performed, revealing no evidence of DVT. A repeat pulmonary CTA was requested, which showed; "A filling defect consistent with an embolism allowing flow is noted in the left main pulmonary artery and its branch leading to the lower lobe. The main pulmonary artery diameter is increased." (Figure 2). Compared to the previous imaging, signs of recanalization were evident. Echocardiographic evaluation revealed normal right heart function. The patient was subsequently admitted to the chest diseases service for further management and follow-up.

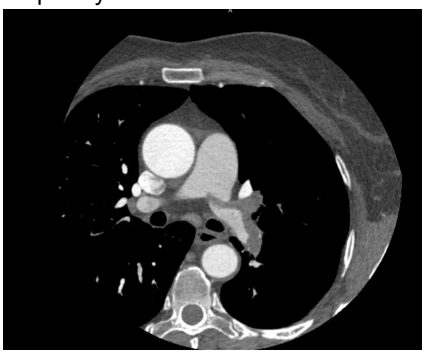

Figure 1: Pulmonary CTA following plasmapheresis

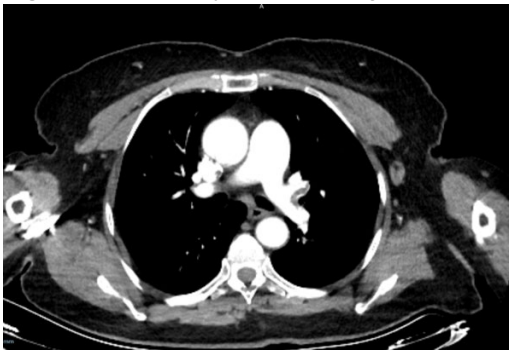

Figure 2: Pulmonary CTA at emergency admission

Dıscussıon

Hyperlipidemia may lead to acute thrombotic events by promoting atherosclerosis and hypercoagulability. Conditions such as hyperlipidemia and diabetes mellitus are known to increase susceptibility to venous thromboembolism. These risk factors can independently precipitate embolic events; however, blood product transfusions may further enhance this risk. Although there is limited evidence in the literature supporting this association, clinicians should be vigilant for new symptoms in patients receiving blood product transfusions.

In patients undergoing plasmapheresis for hyperlipidemia, syncope may be an initial manifestation of pulmonary embolism. Therefore, it is important to pursue further diagnostic evaluation when necessary, particularly if symptoms worsen or new clinical findings emerge.

Conclusıon

In patients undergoing blood product transfusion, any new symptoms should be carefully evaluated. In patients with hyperlipidemia, the development of syncope during or after plasmapheresis may suggest pulmonary embolism. A thorough anamnesis and appropriate additional investigations are critical for timely diagnosis and management.

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Diffuse Subendocardial Ischemia Secondary to Disulfiram-Ethanol Reaction: A Case Report

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Introduction

Disulfiram has been extensively used in clinical practice as an aversive pharmacologic agent for alcohol dependence. Its principal mechanism is irreversible inhibition of aldehyde dehydrogenase, causing acetaldehyde accumulation when alcohol is consumed. This biochemical interaction provokes an aversive reaction known as the disulfiram-ethanol reaction (DER), typically characterized by flushing, nausea, vomiting, hypotension, and tachycardia (1, 2).

Although considered safe under appropriate monitoring, disulfiram may occasionally induce severe adverse events. Reported serious complications include hepatotoxicity, peripheral neuropathy, psychosis, encephalopathy, and cardiovascular complications such as arrhythmias, myocardial ischemia, and in rare cases, myocardial infarction (3-6). Cardiovascular manifestations primarily result from vasodilation, hypotension, and autonomic dysregulation due to the DER (5, 6). Moreover, these hemodynamic changes can lead to transient myocardial ischemia even without underlying coronary artery disease (4, 7).

In this report, we present a unique case of a patient who exhibited diffuse subendocardial ischemia, elevated cardiac biomarkers, and widespread ST-segment depression secondary to DER. This case emphasizes an uncommon but critical cardiac complication associated with the disulfiram-ethanol interaction.

Case Presentation

A 47-year-old male, with no known history of cardiovascular or chronic diseases, presented to the emergency department following the ingestion of a full box of disulfiram tablets (approximately 20 tablets, 250 mg each) together with substantial alcohol consumption at midnight. He was first assessed in a peripheral hospital, where gastric lavage was performed. Due to borderline elevation of troponin levels, he was transferred to our center with an initial suspicion of non-ST elevation myocardial infarction (NSTEMI).

On admission, he was somnolent with a Glasgow Coma Scale (GCS) of 14. Vital signs were: blood pressure 110/70 mmHg, heart rate 76/min, respiratory rate 26/min, temperature 36.4 °C. Physical examination revealed no abnormal cardiopulmonary findings, abdominal tenderness, or focal neurologic deficits. Laboratory findings included elevated serum troponin I (0.143 ng/mL, reference <0.04 ng/mL) and serum ethanol level of 124 mg/dL. Other biochemical parameters, including renal, hepatic, and electrolyte panels, were within normal limits. Blood gas analysis revealed pH 7.43, PCO□ 39.2 mmHg, and HCO□□ 25 mmol/L. ECG demonstrated diffuse ST-segment depression (Figure 1). Transthoracic echocardiography showed preserved left ventricular ejection fraction (65%) and concentric left ventricular hypertrophy, without regional wall motion abnormalities.

Discussion

Disulfiram-ethanol reaction (DER) typically manifests as unpleasant systemic reactions resulting from acetaldehyde accumulation. Although cardiovascular events during DER are uncommon, several reports indicate potential cardiac involvement, including arrhythmias, myocardial ischemia, and hemodynamic instability (1, 4-6). The cardiovascular effects are predominantly attributed to sympathetic stimulation, peripheral vasodilation, and hypotension resulting in myocardial oxygen supply-demand mismatch (4, 5, 7).

In our patient, widespread ST-segment depression and troponin elevation, in the absence of significant coronary artery obstruction or left ventricular dysfunction, strongly suggest transient subendocardial ischemia secondary to DER. This pathophysiology is consistent with other case reports and studies, where disulfiram-alcohol interaction caused cardiac ischemic presentations without underlying coronary artery disease (4, 7, 8). Fuller et al. previously emphasized the importance of careful patient selection for disulfiram therapy due to possible cardiovascular risks in predisposed individuals (4). Furthermore, Chick et al. described rare cardiovascular collapses secondary to DER, reinforcing the significance of careful monitoring and patient education regarding potential reactions with alcohol ingestion (5). Skinner et al.'s meta-analysis underlines that supervised disulfiram therapy improves abstinence rates but emphasizes vigilance due to possible severe reactions from non-adherence and unsupervised alcohol intake (1). Similarly, Carroll et al. and Petrakis et al. reported cardiac and systemic complications in patients inadequately supervised during disulfiram treatment, highlighting critical safety issues surrounding disulfiram's clinical application (8, 9).

Our case uniquely illustrates transient subendocardial ischemia explicitly linked to DER, enriching clinical understanding and emphasizing the potential cardiac hazards inherent in disulfiram-alcohol co-ingestion scenarios. It also underscores the necessity of multidisciplinary management—cardiology, psychiatry, and toxicology—to optimize patient outcomes in DER-related clinical presen-

tations.

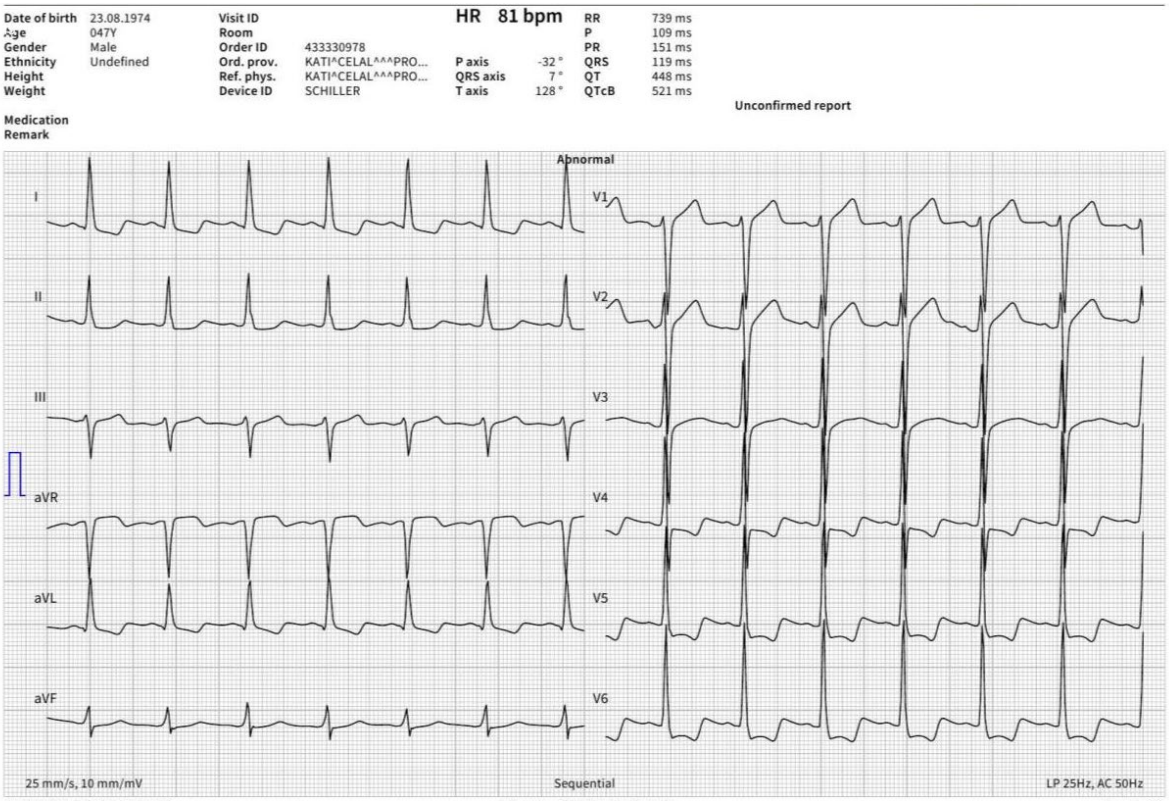

Figure 1. Electrocardiogram (ECG) demonstrating diffuse subendocardial ischemia following disulfiram-ethanol interaction

Cardiology consultation attributed the ECG abnormalities and troponin elevation to secondary non-coronary etiologies, recommending serial cardiac enzyme and ECG follow-up. The National Toxicology Consultation Center (114) advised supportive treatment, including monitoring of glucose, liver, and kidney function. During hospitalization, psychiatric evaluation revealed intermittent alcohol use spanning over two decades and recent psychological stressors contributing to increased alcohol consumption. The patient admitted impulsively ingesting disulfiram tablets without suicidal intention. Psychiatric diagnosis of adjustment disorder with depressive mood was made, and treatment with sertraline 50 mg daily was initiated. The patient's clinical status progressively improved within 48 hours. Serial cardiac enzymes remained stable, ST-segment depression resolved spontaneously, and consciousness fully normalized. He was discharged on the second day with a diagnosis of hypertension and recommendations for outpatient cardiology and psychiatric follow-up.

Conclusion

Clinicians must be aware of potential cardiac complications, including transient subendocardial ischemia, associated with disulfiram-ethanol interaction. Early recognition, prompt supportive treatment, and multidisciplinary collaboration can mitigate severe outcomes, reducing unnecessary invasive procedures. Additionally, rigorous patient education, adherence monitoring, and psychiatric follow-up remain pivotal components in safely prescribing disulfiram.

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The Diagnostic and Prognostic Value of BUN/Albumin and BUN/Creatinine Ratios in Patients with Gastrointestinal Bleeding Presenting to the Emergency Department

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Introduction

Acute gastrointestinal bleeding (GIB) is a frequent and serious condition in emergency departments (EDs), associated with significant mortality if not promptly managed. GIB is classified into upper gastrointestinal bleeding (UGIB) and lower gastrointestinal bleeding (LGIB). UGIB typically presents with hematemesis, melena, and shock, whereas LGIB often manifests as hematochezia [1-3]. Reported mortality rates for UGIB range from 4.5% to 10%, while LGIB has a lower mortality rate of 1.9% to 2.3% [4-6]. Rapid diagnosis and intervention are essential to prevent hemodynamic instability and organ failure [2-4].

Blood urea nitrogen (BUN) is a byproduct of protein metabolism and reflects both renal function and nutritional status. Albumin serves as an indirect marker of nutrition, oxidative balance, and inflammation [7-10]. Combining these parameters results in the BUN/Albumin (B/A) ratio, an accessible and non-invasive indicator of physiological reserve. Prior studies have demonstrated the prognostic utility of the B/A ratio in conditions such as sepsis, pneumonia, COPD, and heart failure [9,10].

Similarly, the BUN/Creatinine (B/Cr) ratio is often used to differentiate between UGIB and LGIB and to predict outcomes [7,8,11,12]. However, the relationship between these ratios and in-hospital mortality, active bleeding, and GIB type in ED patients remains underexplored.

This study aimed to assess the associations of the B/A and B/Cr ratios with in-hospital mortality, active bleeding, and GIB types in patients presenting with gastrointestinal bleeding.

Material and Methods

This retrospective observational study was conducted in a tertiary ED. Ethical approval was obtained (2024-BÇEK/204), and the study adhered to the STARD guidelines [13].

Between June 1, 2019, and June 30, 2024, adult patients presenting with GIB who underwent endoscopy and/or colonoscopy within 24 hours were reviewed. Inclusion criteria were patients aged ≥18 years, with blood biochemistry results within 30 minutes of admission and complete medical records. Exclusion criteria included pregnancy, chronic kidney disease, gastrointestinal malignancy, transfer from other centers, and incomplete data.

Demographics, comorbidities, vital signs, laboratory results, and endoscopy/colonoscopy findings were recorded. GIB type (UGIB or LGIB) and the presence of active bleeding were determined endoscopically. B/A and B/Cr ratios were calculated from initial blood tests. In-hospital mortality (death during hospitalization) and active bleeding (visible hemorrhage during endoscopy/colonoscopy) were defined as poor prognostic outcomes.

All data obtained during the study and recorded in the study form were analyzed using IBM SPSS 20.0 statistical software (Chicago, IL, USA). The normality of the distribution for discrete and continuous numerical variables was assessed using the Kolmogorov-Smirnov test. Descriptive statistics were presented as the median (IQR25-75) for continuous variables and as counts (percentages) for categorical variables. Categorical variables were analyzed using the Chi-square test, while continuous variables were evaluated using the Mann-Whitney U test.

Receiver operating characteristic (ROC) curves were generated to determine the threshold values of B/A and B/Cr ratios for predicting in-hospital mortality, active bleeding, and distinguishing upper/lower GIB, and the area under the curve (AUC) was calculated [14]. Results with a p-value of <0.05 were considered statistically significant.

Results

Out of 1126 reviewed patients, 864 met the inclusion criteria. Of these, 66.2% were male, and the median age was 64 years (IQR 49–74).

The median B/A ratio was 7.4, and the median B/Cr ratio was 24.7. Based on endoscopic findings, 84.5% were diagnosed with UGIB and 15.5% with LGIB. Active bleeding was observed in 16.3% of patients. In-hospital mortality occurred in 7.1%, and 20% of patients required ICU admission.

Patients who died or had active bleeding had significantly higher B/A and B/Cr ratios ($p<0.001$ for all comparisons). UGIB patients also had higher ratios than those with LGIB.

ROC analysis revealed that the B/A ratio had strong prognostic value for mortality prediction (AUC: 0.822, 95% CI: 0.771–0.873, $p<0.001$), with a cutoff of 10.15 yielding 72.1% sensitivity and 80.33% specificity. The B/Cr ratio had a lower AUC of 0.694 for mortality prediction.

For active bleeding prediction, the B/A ratio showed an AUC of 0.720 and the B/Cr ratio 0.690. In differentiating UGIB from LGIB, the B/Cr ratio showed slightly better performance (AUC: 0.612).

Discussion

This study demonstrates that the B/A and B/Cr ratios are valuable markers for predicting poor outcomes in GIB patients. The B/A ratio was superior in predicting mortality and active bleeding, while the B/Cr ratio showed better utility in distinguishing UGIB from

LGIB.

Dehydration, malnutrition, and renal dysfunction are critical factors worsening GIB outcomes. Blood loss induces hypovolemia, enhancing BUN reabsorption, while albumin levels decrease due to inflammation and hemorrhage. The B/A ratio combines these effects, serving as a non-invasive composite marker [15,16].

Previous studies linked the B/A ratio with adverse outcomes in sepsis, pneumonia, COPD, and cardiac diseases. A study among elderly GIB patients identified a B/A cutoff of 15.1 for mortality prediction [16]. Our study, including a broader age range, determined a lower but highly predictive cutoff (10.15), reinforcing the B/A ratio's applicability across different age groups.

Both ratios also demonstrated significant value for active bleeding prediction. The increase in BUN, due to digestion and absorption of blood in the GI tract, alongside hypoalbuminemia, likely explains these findings.

In terms of GIB type differentiation, B/Cr ratio showed a slight advantage. Prior studies have suggested higher B/Cr ratios in UGIB compared to LGIB [11,12,19]. In our cohort, the B/Cr ratio cutoff of 20.09 showed reasonable sensitivity (68.22%) and specificity (53.73%) for UGIB identification.

Identifying high-risk patients early using simple laboratory markers like the B/A and B/Cr ratios can optimize resource allocation, guide early resuscitation efforts, and improve patient outcomes.

Conclusion

The B/A and B/Cr ratios are practical, reliable markers in the early risk assessment of GIB patients. The B/A ratio demonstrated superior accuracy for predicting mortality and active bleeding, whereas the B/Cr ratio showed better performance for UGIB differentiation. Incorporating these ratios into clinical decision-making may enhance patient stratification and management in emergency settings.

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Table 1. Demographic and Clinical Characteristics of the Patients

Gender, n (%)	
Male	572 (66.2)
Age, median (IQR[1] 25-75)	64 (49-74)
Comorbidities, n (%)	
Hypertension	450 (52.1)
Diabetes mellitus	193 (22.3)
COPD[2]	66 (7.6)
CAD[3] /CHF[4]	310 (35.9)
Liver Failure	43 (5)
Malignancy	46 (5.3)
ASA[5] / Antiplatelet Use	255 (29.5)
Anticoagulant Use	115 (13.3)
Laboratory, median (IQR 25-75)	
Hemoglobin (g/dL)	9.4 (7.6-12.1)
Hematocrit (%)	29.7 (24.1-36.8)
BUN[6] (mg/dL)	26.2 (18.2-37.4)
Creatinine (mg/dL)	1 (0.8-1.2)
Total protein (mg/dL)	6.2 (5.6-6.6)
Albumin (g/dL)	3.5 (3.2-3.9)
B/A[7] ratio	7.4 (5.1-11.5)
B/Cr[8] ratio	24.7 (17.7-33.7)
Endoscopy/Colonoscopy Findings, n (%)	
Active bleeding	141 (16.3)
UGIB[9]	730 (84.5)
LGIB[10]	134 (15.5)
Intensive Care Unit Admission	173 (20)
In-Hospital Mortality	61 (7.1)

IQR[1]: Inter Quartile Range, COPD[2]: Chronic Obstructive Pulmonary Disease, CAD[3]: Coronary Artery Disease, CHF[4]: Chronic Heart Failure, ASA[5]: Acetylsalicylic Acid, BUN[6]: Blood urea nitrogen, B/A[7]: Blood urea nitrogen-to-albumin, B/Cr[8]: Blood urea nitrogen-to-creatinine, UGIB[9]: Upper gastrointestinal bleeding, LGIB[10]: Lower gastrointestinal bleeding

Table 2. Comparison of B/A and B/Cr Ratios by Mortality, Active Bleeding, and GIB Types

	Non-survivors, n=61	**Survivors, n=803**	**P value**
B/A[1] ratio	7.1 (4.9-10.7)	15.3 (10.8-22.5)	<0.001
B/Cr[2] ratio	24.1 (17.2-33.1)	31.7 (26.1-40-7)	<0.001
BUN[3] (mg/dL)	25.2 (17.7-35.5)	41.1 (27.5-57.9)	<0.001
Albumin (g/dL)	3.6 (3.2-3.9)	2.6 (2.4-2.8)	<0.001
Creatinin (mg/dL)	0.9 (0.8-1.2)	1.4 (0.9-1.7)	<0.001
	Without Active Bleeding, n=723	**With Active Bleeding, n=141**	**P value**
B/A ratio	7.1 (4.7-10.4)	11.5 (7-18.2)	<0.001
B/Cr ratio	23.4 (17.1-32.1)	31 (25.1-44.1)	<0.001
BUN (mg/dL)	24.7 (17.2-35)	35.5 (25.2-50.4)	<0.001
Albumin (g/dL)	3.6 (3.3-3.9)	2.9 (2.6-3.5)	<0.001
Creatinin (mg/dL)	1 (0.9-1.2)	1.1 (0.9-1.4)	0.052
	LGIB[4], n=134	**UGIB[5], n=730**	**P value**
B/A ratio	6.6 (4.6-9.5)	7.7 (5.3-11.8)	0.003
B/Cr ratio	19.4 (15.2-28.9)	25.6 (18.5-34.5)	<0.001
BUN (mg/dL)	21.9 (15.8-30.9)	27.1 (18.7-38)	<0.001
Albumin (g/dL)	3.5 (3.1-3.8)	3.5 (3.2-3.9)	0.382
Creatinin (mg/dL)	0.9 (0.8-1.2)	1 (0.9-1.2)	0.272

B/A[1]: Blood urea nitrogen-to-albumin, B/Cr[2]: Blood urea nitrogen-to-creatinine, BUN[3]: Blood urea nitrogen, LGIB[4]: Lower gastrointestinal bleeding, UGIB[5]: Upper gastrointestinal bleeding

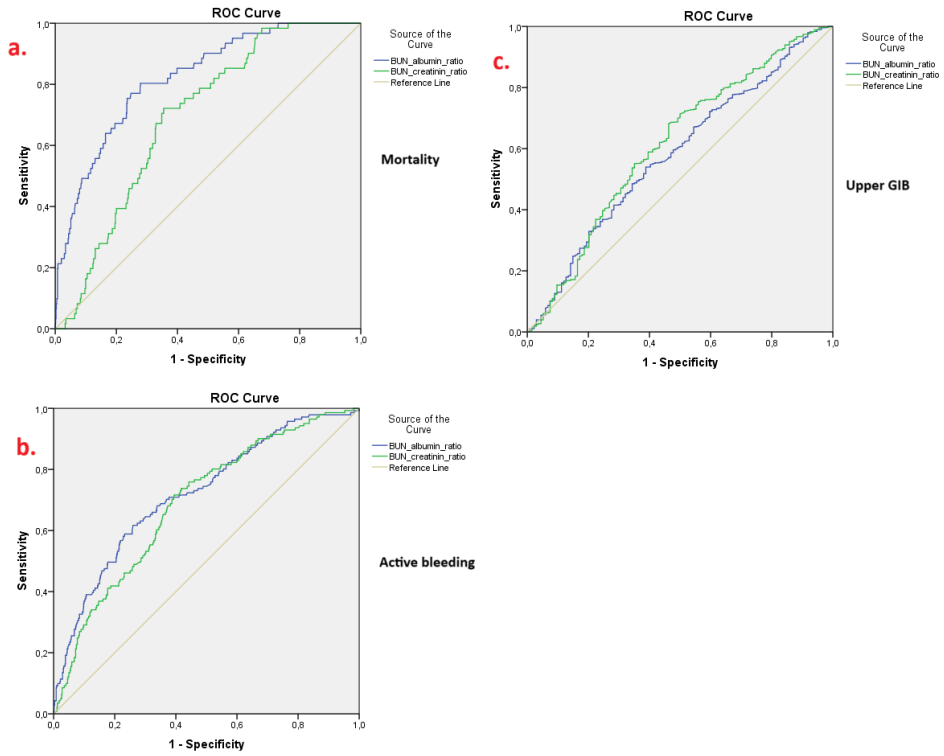

Figure 1. a. ROC analysis to determine B/A and B/Cr ratios threshold between non-survivor and survivor groups b. ROC analysis to determine B/A and B/Cr ratios threshold between those with and without active bleeding c. ROC analysis to determine B/A and B/Cr ratios threshold between upper and lower GIB

Table 3. The prognostic values for B/A and B/Cr ratio levels to prediction of mortality, active bleeding and UGIB.

Variables	AUC[1] (95%CI)	Cut-Off Value	Sensitivity (95%CI)	Specificity (95%CI)	PLR[2] (95%CI)	NLR[3] (95%CI)	Accuracy (95%CI)
For mortality							
B/A[4] ratio	0.822 (0.771 - 0.873)	*10.15	72,1 (68,86 - 75,18)	80,33 (68,16 - 89,4)	3,67 (2,21 - 6,11)	0,35 (0,3 - 0,41)	72,69 (69,58 - 75,63)
B/Cr[5] ratio	0.694 (0.640 - 0.748)	*28.53	64,51 (61,09 - 67,82)	72,13 (59,17 - 82,85)	2,31 (1,54 - 3,47)	0,49 (0,41 - 0,59)	65,05 (61,76 - 68,23)
For active bleeding							
B/A ratio	0.720 (0.673 - 0.767)	*10.05	74 (70,64 - 77,16)	61,7 (53,15 - 69,76)	1,93 (1,56 - 2,39)	0,42 (0,35 - 0,5)	71,99 (68,87 - 74,96)
B/Cr ratio	0.690 (0.644 - 0.736)	*26.48	60,58 (56,91 - 64,16)	71,63 (63,43 - 78,9)	2,14 (1,64 - 2,8)	0,55 (0,48 - 0,63)	2,38 (59,06 - 65,63)
For UGIB[6]							
B/A ratio	0.580 (0.527 - 0.633)	*7.27	53,97 (50,28 - 57,63)	61,19 (52,4 - 69,48)	1,39 (1,11 - 1,74)	0,75 (0,64 - 0,88)	55,09 (51,71 - 58,45)
B/Cr ratio	0.612 (0.558 - 0.667)	*20.09	68,22 (64,7 - 71,59)	53,73 (44,92 - 62,38)	1,47 (1,22 - 1,78)	0,59 (0,49 - 0,71)	65,97 (62,7 - 69,13)

* The best cut-off value calculated according to the Youden index. AUC[1]: Area under curve, PLR[2]: Positive likelihood ratio, NLR[3]: Negative likelihood ratio, B/A[4]: Blood urea nitrogen-to-albumin, B/Cr[5]: Blood urea nitrogen-to-creatinine, UGIB[6]: Upper gastrointestinal bleeding

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Right Adrenal Hemorrhage Masquerading as Nephrolithiasis: A Case of Atypical Presentation and Diagnostic Challenges

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Introductıon

Adrenal hemorrhage is a rare but potentially life-threatening condition that may be caused by either traumatic or non-traumatic (spontaneous) etiology. Traumatic adrenal hemorrhage most frequently occurs after high-impact trauma, such as motor vehicle accidents or falls, and is typically associated with multi-organ injury (1). Conversely, spontaneous adrenal hemorrhage (SAH) is less frequent, accounting for less than 0.3–1.8% of adrenal disease in clinical series, and is usually related to predisposing factors such as anticoagulant therapy, sepsis, coagulopathies, or pre-existing adrenal neoplasms (2,3). SAH may be accompanied by nonspecific symptoms such as flank or abdominal pain, hypotension, fever, or lethargy; the diagnosis is challenging (4).

The clinical significance of adrenal hemorrhage is its potential to induce adrenal insufficiency, hemodynamic instability, or hemorrhagic shock if left undiagnosed (5). Even though a computed tomography scan is the diagnostic gold standard, the absence of classic risk factors or overt trauma typically leads to diagnostic delays (6). This is a case report of a 42-year-old man with no known medical history presenting with colicky abdominal pain initially suspected to be due to nephrolithiasis but later discovered to be secondary to right adrenal hemorrhage. The atypical presentation and absence of typical risk factors emphasize the diagnostic pitfalls and the importance of having an adrenal hemorrhage as part of the differential diagnosis of flank pain.

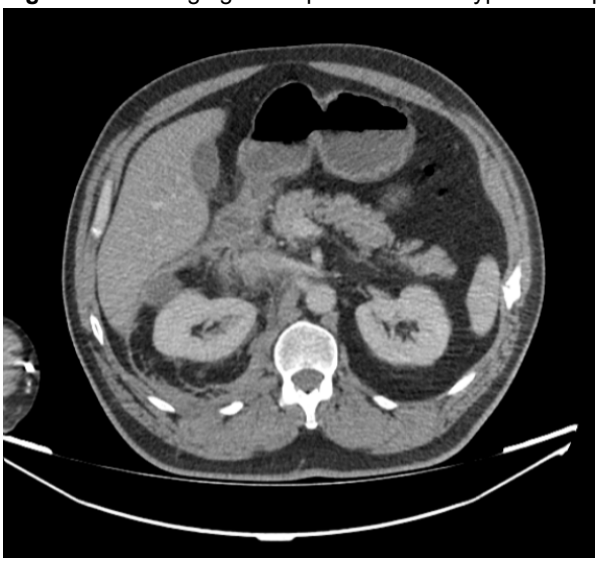
Case

A 42-year-old male with no past medical history presented to the emergency department with abdominal and back pain. He had visited another emergency department two days prior with the same complaint. Due to his colicky pain, nephrolithiasis was suspected, and he was discharged after analgesic treatment and a follow-up with an outpatient urologist.

The patient did not have a history of alcohol consumption, intake of medication, or trauma. His Glasgow Coma Scale (GCS) score was 15. His vital signs were as follows: blood pressure 110/70 mmHg, oxygen saturation 98%, heart rate 91 beats per minute, and body temperature 36.6°C. On examination, there was no guarding and rebound tenderness; however, he had colicky pain in the right upper and lower quadrants extending to the flank area. The right costovertebral angle was positive for costovertebral angle tenderness and no sign of gastrointestinal bleeding on digital rectal examination.

Laboratory findings were remarkable, with 13.99 × 10^3/μL white blood cell count, hemoglobin 11.5 g/dL, and elevated C-reactive protein (CRP) 20.4 mg/L. Renal and liver function tests were within normal limits, and urinalysis was negative for any significant finding. After the stoppage of oral intake and the start of analgesic symptomatic management, a non-contrast abdominal computed tomography (CT) scan was performed, demonstrating densities of perinephric fat surrounding the right kidney compatible with hemorrhage (Figure 1).

Figure 1. CT imaging of the patient shows hyperdense perinephric fat stranding consistent with right adrenal hemorrhage



The patient, whose hemoglobin was stable and never decreased, was diagnosed with right adrenal hemorrhage. After the urology consultation, he was sent to the urology service for analgesia and observation. His status did not alter much, and with adequate pain control, he was discharged on day six of his stay with an outpatient follow-up suggestion of urology.

Discussion

Adrenal hemorrhage, either traumatic or spontaneous, is a clinically significant condition requiring immediate recognition. The traumatic variety is most often encountered in the context of polytrauma. It is more common in the right adrenal gland due to its anatomical compression on the spine by blunt trauma (1). Spontaneous hemorrhage of the adrenal gland is less well documented and typically presents with predisposing conditions. Anticoagulant therapy, particularly with heparin or warfarin, causes 50–60% of SAH, while sepsis (for example, Waterhouse-Friderichsen syndrome), adrenal tumors, or stress-induced adrenal hyperemia are other etiologies (7,8).

This case is noteworthy because there were no typical risk factors. The patient was not on anticoagulants, suffered from trauma, or experienced recent infections, and laboratory evaluation ruled out coagulopathy. His presentation with isolated colicky pain and hemodynamic stability is distinct from SAH presentations where hypotension, fever, or laboratory evidence of adrenal insufficiency are typical (9). The initial misdiagnosis as nephrolithiasis also demonstrates the similarity of symptomatology between renal colic and adrenal disease. Colicky pain, in turn classically secondary to ureteral obstruction, can mimic the referred patterns of retroperitoneal hemorrhage and thus present a challenging diagnosis (10). Imaging played a central role in this case. Abdominal non-contrast CT revealed hyperdense perinephric fat stranding secondary to hemorrhage, confined to the right adrenal region. CT remains the imaging of choice because it can delineate hemorrhage and exclude differential pathologies (11). Of note, in this case, the absence of contrast enhancement avoided confounding factors, as extravasation of contrast can mimic acute bleeding (12). The patient's stable hemoglobin levels during hospitalization also distinguish this case. Most adrenal hemorrhages, particularly larger hemorrhages, are accompanied by a reduction in hemoglobin secondary to continued bleeding (13). Small hemorrhages confined to the adrenal gland or perinephric fat may not significantly impact systemic hemodynamics, as in this case. This agrees with the evidence that small adrenal hemorrhages can resolve spontaneously with conservative management, and larger hemorrhages require intervention (14). This case contributes to the literature because it presents an atypical presentation of SAH without typical risk factors or laboratory derangements. Simon et al. (2020) retrospectively reviewed 45 cases of SAH and reported that 78% had identifiable risk factors, predominantly anticoagulation (15). Our patient's benign course and lack of hemoglobin decline challenge the usual association of SAH with active bleeding or hemodynamic instability, demonstrating variability in disease severity. Adrenal hemorrhage management is based on hemodynamic stability and adrenal function. While our patient merely required monitoring and analgesia, patients with adrenal insufficiency or expanding hematomas will need steroid replacement or surgery (16). Follow-up over the long term to exclude underlying neoplasms is also necessary, although the absence of mass lesions on CT, in this case, reduced concern for malignancy (17).

This case raises the diagnostic dilemma of adrenal hemorrhage when the patient lacks typical risk factors. Initially diagnosed with nephrolithiasis through shared symptoms, hemorrhage was confirmed by non-contrast CT. Stable hemoglobin and lack of adrenal insufficiency mirror its inconstant presentation, with a focus on small hemorrhages in idiopathic flank pain. It supports clinical awareness, prompt imaging, and investigating biomarkers for uncommon cases.

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The Role of Peripheral Perfusion Index in Predicting Biphasic Reactions in Anaphylaxis Patients

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Introduction

Anaphylaxis is a sudden, potentially fatal hypersensitivity reaction that affects multiple organ systems. Common triggers include food, insect stings, and medications [1,2]. Although most patients respond well to early epinephrine administration, a subset experiences symptom recurrence or refractory anaphylaxis—persistent symptoms despite appropriate treatment [3,4]. Additionally, biphasic reactions, where symptoms recur after complete resolution, pose management challenges and necessitate prolonged emergency department (ED) observation, increasing healthcare costs and patient dissatisfaction [4-7].

The Peripheral Perfusion Index (PPI), derived from pulse oximetry, reflects tissue perfusion by measuring the ratio of pulsatile to non-pulsatile blood flow. A value under 1.4 is considered indicative of impaired peripheral perfusion [8-10]. Given that anaphylaxis involves vasodilation, vascular permeability increases, and myocardial depression [1-3], PPI could potentially serve as a non-invasive marker of hemodynamic instability. However, evidence supporting this is scarce. This study aimed to evaluate PPI as a predictor of biphasic reactions in anaphylaxis patients.

Material and Methods

This prospective observational study was conducted between September 2023 and September 2024 at Ankara Atatürk Sanatoryum Training and Research Hospital. Ethical approval was granted (2012-KAEK-15/2765). The study adhered to STROBE guidelines [11].

The study included patients aged 18 years and older who were diagnosed with anaphylaxis in the emergency department between September 1, 2023, and September 1, 2024, and who agreed to participate in the study. The diagnosis of anaphylaxis was based on the clinical criteria established by the National Institute of Allergy and Infectious Diseases and the Food Allergy and Anaphylaxis Network (NIAID/FAAN) [2]. Pregnant women, those who did not consent to participate in the study, and patients who received epinephrine before arriving at the hospital via ambulance were excluded from the study.

Clinical data included demographics, vital signs, triggers, treatments, and outcomes. PPI was measured with a Masimo Radical 7 device at baseline (0 min, before epinephrine), and at 10, 20, and 30 minutes post-treatment, as well as after full symptom resolution. Refractory anaphylaxis was defined as requiring three doses of intramuscular epinephrine and/or an infusion [4]. Patients were observed for at least 6 hours in the ED and monitored for biphasic reactions or return visits within 48 hours.

All data obtained during the study and recorded on the study form were analyzed using IBM SPSS 20.0 (Chicago, IL, USA) statistical program. The distribution of discrete and continuous numerical variables was evaluated using the Shapiro-Wilk test, histogram, and Q-Q plot graphs. Descriptive statistics were presented as mean±standard deviation (SD) or median (minimum - maximum) for discrete and continuous numerical variables, and categorical variables were presented as frequency counts and percentages. Categorical variables were evaluated by chi-squared and continuous variables by t-test or Mann-Whitney U test. The diagnostic accuracy of PPI for predicting biphasic reactions was evaluated using the Receiver Operating Characteristic (ROC) curve analysis [12]. Statistical significance was considered at $p<0.05$.

Results

A total of 104 anaphylaxis patients were included (52.9% female, median age 45 years). Allergies were previously known in 49%, and 9.6% had a prior history of anaphylaxis. Analgesics were the most common triggers (36.5%).

The median baseline PPI was 2.20, rising to 4.20 after symptom resolution. Biphasic reactions occurred in 10.6% of cases, refractory anaphylaxis in 2.9%, and Kounis syndrome in 2.9%. No patients returned with recurrent symptoms during 48-hour follow-up.

Patients who developed biphasic reactions had significantly lower PPI values at all measured time points ($p<0.05$). ROC analysis identified a PPI cutoff of ≤2.17 for predicting biphasic reactions, with a sensitivity of 57% and specificity of 91% (AUC = 0.753).

Additionally, PPI initially decreased at 10 minutes after epinephrine administration, followed by increases at 20 and 30 minutes, reflecting systemic improvement.

Discussion

Our study found a biphasic reaction rate of 10.6%, aligning with existing literature [4-7,13]. Patients with lower PPI values were more likely to develop biphasic reactions, suggesting that PPI could serve as a prognostic tool.

Anaphylaxis-related hypoperfusion results from systemic vasodilation and capillary leak, culminating in distributive shock and decreased cardiac output [1-3,14]. Rapid recognition and treatment are essential to prevent organ dysfunction [14-17]. PPI, being a non-invasive, continuous, and easily obtainable measure, offers valuable hemodynamic insights in acute settings.

Previous research supports PPI's association with critical illness outcomes, including shock, sepsis, and even scorpion envenomation severity [8-10,18]. Consistent with these findings, our study demonstrated that lower PPI values at baseline and early follow-up correlated with biphasic reaction development.

However, while the specificity of PPI was high (91%), its sensitivity was relatively low (57%). Thus, although low PPI should raise

suspicion for biphasic reactions, normal PPI values do not exclude risk. PPI trends, rather than absolute values, may offer better clinical utility, emphasizing the importance of serial measurements over time.

Interestingly, PPI values dipped at the 10-minute mark post-epinephrine, likely reflecting transient exacerbation of hypoperfusion due to initial vasoconstrictive effects. Subsequent increases likely represent therapeutic improvement, illustrating how PPI dynamically mirrors hemodynamic changes during treatment.

Given individual variability in baseline PPI values, fixed cutoffs may be less reliable. Tracking changes within individual patients may better assess evolving clinical status and response to treatment.

Thus, while PPI alone should not dictate clinical decisions, it remains a valuable adjunct for monitoring treatment response and detecting early signs of deterioration in anaphylaxis patients.

Conclusion

Low PPI values may indicate a higher risk of biphasic reactions in anaphylaxis patients. Although not sufficiently sensitive to serve as a standalone predictor, PPI offers a rapid, non-invasive, and cost-effective tool for monitoring hemodynamic changes and treatment responses. Integrating PPI monitoring into anaphylaxis management protocols may help identify high-risk patients who require closer observation.

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Table 1. Demographic and clinical characteristics of the patients

Demographic and clinical characteristics, n(%)	**All patients (n=104)**
Gender, n(%)	
Female	55 (52.9%)
Age median, (IQR[1], 25-75)	45 (35-55)
Time to onset of symptoms (minutes), (IQR, 25-75)	30 (20-45)
Vital signs, (IQR, 25-75)	
Pulse	93 (84-105.75)
Saturation	96 (92.25-98)
Systolic BP[2], 0th minute	90 (80-110)
Systolic BP, 10th minute	110 (96-130)
Systolic BP, 20th minute	115 (106-130)
Systolic BP, 30th minute	120 (110-130)
PPI[3] value, 0th minute	2.20 (1.52-3.67)
PPI value, 10th minute	1.90 (1.10-3.10)
PPI value, 20th minute	3.00 (1.91-4.60)
PPI value, 30th minute	3.51 (2.49-5.46)
PPI value, when symptoms regress	4.20 (3.10-6.35)
History of allergy	51 (49%)
History of anaphylaxis	10 (9.6%)
Possible allergen, n(%)	
Analgesics	38 (36.5%)
Antibiotics	35 (33.7%)
Unknown	8 (7.7%)
Proton pump inhibitors	7 (6.7%)
Multidrug	5 (4.8%)
Bee stings	4 (3.8%)
Food	3 (2.9%)
Contrast agent	2 (1.9%)
Hair dye	1 (1%)
Pastille	1 (1%)
Clinical findings, n(%)	
Skin/mucosal tissue involvement	103 (99%)
Respiratory compromise	40 (38.5%)
Reduced BP	66 (63.5%)
Gastrointestinal symptoms	50 (48.1%)
Syncope	6 (5.8%)
Treatment administered, n(%)	
1 dose of epinephrine	81 (77.9%)
2 doses of epinephrine	22 (21.2%)
3 doses of epinephrine	1 (1%)
Epinephrine infusion	2 (1.9%)
Clinical outcome, n(%)	
Refractory anaphylaxis	3 (2.9%)
Biphasic anaphylaxis	11 (10.6%)
Koinus syndrome	3 (2.9%)

IQR[1]: Inter Quartile Range, BP[2]: Blood pressure, PPI[3]: Peripheral perfusion index

Table 2. Comparison of patients with and without biphasic reaction

	Patients with biphasic reaction (n=11)	**Patients without biphasic reaction (n=93)**	**p**
Age median, (IQR[1], 25-75)	52 (40-61)	45 (34-53)	0.206
Time to onset of symptoms (minutes), (IQR, 25-75)	30 (15-60)	30 (20-45)	0.742
Vital signs, (IQR, 25-75)			

Pulse	90 (82-104)	93 (84-108)	0.394
Saturation	96 (91-99)	96 (92.5-97.5)	0.492
Systolic BP², 0th minute	80 (80-90)	90 (80-110)	0.065
Systolic BP, 10th minute	90 (89-113)	110 (98.5-130)	0.023
Systolic BP, 20th minute	100 (90-116)	116 (108-130)	0.006
Systolic BP, 30th minute	110 (104-120)	120 (110-130)	0.012
PPI³ value, 0th minute	1.60 (1.10-1.90)	2.30 (1.66-3.78)	0.006
PPI value, 10th minute	1.10 (0.70-1.90)	1.95 (1.10-3.25)	0.013
PPI value, 20th minute	1.60 (1.20-2.30)	3.40 (2.10-5.06)	0.001
PPI value, 30th minute	2.20 (1.70-3.60)	3.70 (2.60-5.75)	0.012
Possible allergen, n(%)			
Analgesics	4 (36.4%)	34 (36.6%)	
Antibiotics	5 (45.5%)	30 (32.3%)	
Unknown	0 (0%)	8 (8.6%)	
Proton pump inhibitors	1 (9.1%)	6 (6.5%)	0.699
Multidrug	0 (0%)	5 (5.4%)	
Bee stings	0 (0%)	4 (4.3%)	
Food	0 (0%)	3 (3.2%)	
Contrast agent	1 (9.1%)	1 (1.1%)	
Hair dye	0 (0%)	1 (1.1%)	
Pastille	0 (0%)	1 (1.1%)	
Clinical findings, n(%)			
Skin/mucosal tissue involvement	11 (100%)	92 (98.9%)	0.894
Respiratory compromise	3 (27.3%)	37 (39.8%)	0.524
Reduced BP	10 (90.9%)	56 (60.2%)	0.053
Gastrointestinal symptoms	4 (36.4%)	46 (49.5%)	0.529
Syncope	1 (9.1%)	5 (5.4%)	0.498
Treatment administered, n(%)			
1 dose of epinephrine	3 (27.3%)	78 (83.9%)	
2 doses of epinephrine	8 (72.7%)	14 (15.1%)	0.001
3 doses of epinephrine	0 (0%)	1 (1.1%)	

IQR¹: Inter Quartile Range, BP²: Blood pressure, PPI³: Peripheral perfusion index

Table 3. Diagnostic features of peripheral perfusion index measurement to determine to predict biphasic reaction (PPI cut-off value is ≤2.17).

	Result	95% CI
Sensitivity	56.99%	46.31%-67.22%
Specificity	90.91%	58.72%-99.77%
Positive Likelihood Ratio	6.27	0.96-40.97
Negative Likelihood Ratio	0.47	0.35-0.63
Prevalence	89.42%	81.86%-94.6%
Positive Predictive Value	98.15%	89.02%-99.71%
Negative Predictive Value	20%	15.63%-25.22%
Accuracy	60.58%	50.51%-70.02%

Table 4. PPI values according to time in patients with biphasic reaction

Patient number	0th minute	10th minute	20th minute	30th minute	When symptoms regress	When biphasic reaction develops
1	0,91	0,65	1,30	1,70	1,90	0,71
2	1,75	1,10	1,20	2,75	3,10	1,30
3	1,20	0,60	0,70	2,10	3,70	1,20
4	1,40	1,10	2,60	3,70	4,90	1,30
5	1,10	0,70	1,40	1,52	1,59	0,90
6	1,90	1,60	1,80	2,10	3,10	1,70
7	1,10	0,90	1,20	1,60	1,90	0,90
8	1,90	1,10	1,60	2,20	2,90	1,70
9	2,83	2,26	4,56	5,70	6,10	2,10

10	1,60	2,80	2,30	3,60	4,20	1,80
11	2,10	1,90	2,20	3,20	3,10	1,90

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Can the Wells Score Predict Severity in Confirmed Pulmonary Embolism? A Retrospective Study Based on PESI and sPESI Classification

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Introduction

Pulmonary embolism (PE) is a potentially life-threatening cardiovascular emergency caused by the obstruction of pulmonary arteries, typically due to thromboembolism originating from deep veins of the lower extremities (1). PE presents with a wide spectrum of clinical manifestations, ranging from mild symptoms to hemodynamic collapse, making early risk stratification essential for appropriate management and prognostic evaluation (2).

Several clinical tools have been developed to assess both the probability of PE and its associated mortality risk. Among these, the Wells score is one of the most widely used clinical decision rules for estimating the pre-test probability of PE in suspected cases (3). It incorporates variables such as signs of deep vein thrombosis (DVT), recent surgery or immobilization, tachycardia, hemoptysis, active cancer, and clinical judgment, and stratifies patients into low, intermediate, or high probability categories (3, 4).

In contrast, the Pulmonary Embolism Severity Index (PESI) and its simplified version (sPESI) were specifically developed to predict short-term mortality in patients with confirmed PE (5). PESI incorporates age, comorbid conditions, vital signs, and laboratory findings to classify patients into five risk categories, which have been validated as predictors of 30-day mortality (5, 6). sPESI, derived from PESI, retains prognostic accuracy while simplifying clinical application by using fewer variables (6).

Although the Wells score is primarily designed to estimate the likelihood of PE, its potential association with PE severity or mortality risk as defined by PESI or sPESI has not been thoroughly studied. Investigating the relationship between these tools may offer valuable insights into the clinical utility of the Wells score beyond diagnostic probability alone. Therefore, this study aimed to explore the association between Wells scores and PE severity as stratified by PESI and sPESI, and to evaluate the ability of Wells scoring to predict higher-risk PE in confirmed cases.

Methods

Study Design and Population

This retrospective observational study was conducted in the Emergency Department of Ondokuz Mayis University Faculty of Medicine between January 1, 2022, and January 1, 2023. Adult patients (aged ≥18 years) who were diagnosed with acute pulmonary embolism (PE) via computed tomography pulmonary angiography (CTPA) were included. The diagnosis of PE was confirmed by the presence of a thrombotic filling defect in the pulmonary arteries on CTPA. Patients with incomplete clinical data or non-thrombotic embolic etiologies (e.g., fat, air, or tumor embolism) were excluded from the study.

Data Collection

Patient data were obtained from the hospital's electronic medical records. Collected variables included demographic characteristics, presenting symptoms, initial vital signs, comorbid conditions, laboratory test results, and radiologic findings. For each case, the Wells score, Pulmonary Embolism Severity Index (PESI), and simplified PESI (sPESI) were calculated using clinical and laboratory parameters recorded at the time of admission.

Patients were categorized into three PE severity groups based on the original PESI classification:
Low risk (PESI Class I–II),
Intermediate risk (Class III),
High risk (Class IV–V).

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 21.0. Descriptive statistics were presented as medians for non-normally distributed continuous variables. The Kruskal-Wallis test was applied to compare Wells scores among the three PESI-based risk groups. Where a significant difference was observed, post hoc pairwise comparisons were conducted using the Dunn test with Bonferroni correction. The relationship between Wells and PESI scores was examined using Spearman rank correlation analysis. In addition, Receiver Operating Characteristic (ROC) curve analysis was employed to assess the discriminative ability of the Wells score in identifying high-risk PE patients. Area under the curve (AUC), sensitivity, specificity, and optimal cut-off values were reported. A p-value of less than 0.05 was considered statistically significant.

Results

The Wells scores of patients were compared across three pulmonary embolism severity groups defined by PESI score: low risk (Class I–II), intermediate risk (Class III), and high risk (Class IV–V). Median Wells scores were 1.0 (0.0–4.0), 2.0 (0.0–5.0), and 3.0 (0.0–5.0) for the low-, intermediate-, and high-risk groups, respectively. The Kruskal-Wallis test indicated a statistically significant difference between groups ($p = 0.001$).

Post hoc analysis using the Dunn test revealed that the Wells score was significantly higher in the high-risk group compared to the low-risk group. However, no statistically significant difference was observed between the intermediate group and either of the other groups.

These findings suggest that as PE severity increases according to PESI classification, the Wells score also tends to increase, indicating a potential relationship between clinical probability and disease severity (Table 1).

Table 1. Comparison of Wells Scores According to PESI-Based Pulmonary Embolism Risk Groups

	Low Risk	Intermediate Risk	High Risk	Test Statistic	p*
Wells Score	1.0 (0.0-4.0)[a]	2.0 (0.0-5.0)[a,b]	3.0 (0.0-5.0)[b]	14.601	**0.001**

[a-b]:There is no statistical difference between groups with the same letter (Dunn test), *Kruskal Wallis test

Table 2 presents the results of Spearman correlation analysis between Wells and PESI scores. A statistically significant moderate positive correlation was found between the two scores (r = 0.519, p < 0.001).

This correlation implies that patients with higher Wells scores, which reflect a greater pre-test probability of pulmonary embolism, also tend to have higher PESI scores, which reflect greater clinical severity and mortality risk. This finding supports the hypothesis that the Wells score may carry prognostic value beyond diagnostic estimation alone.

Table 2. Correlation Between Wells Score and PESI Score

		Wells Score	PESI Score
Wells Score	r	1,000	
	p		
PESI Score	r	0,519	1,000
	p	**<0,001***	

*Spearman Correlation

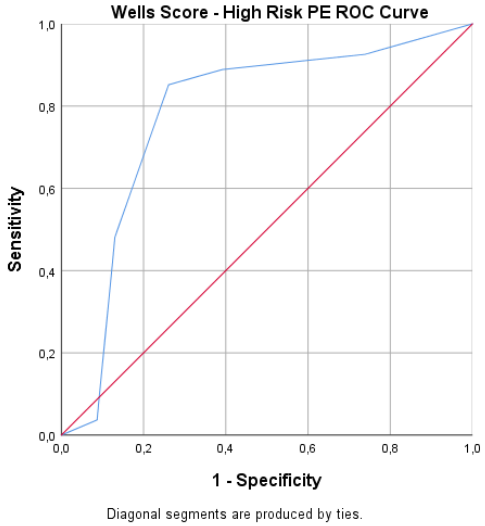

Figure 1. Receiver Operating Characteristic (ROC) Curve for Wells Score in Identifying High-Risk Pulmonary Embolism

Table 3 summarizes the results of the ROC curve analysis evaluating the performance of the Wells score in predicting high-risk pulmonary embolism as defined by PESI Class IV–V. The area under the ROC curve (AUC) was calculated as 0.780 (95% CI: 0.638–0.922), indicating good discriminatory ability. A Wells score cut-off value of ≤2.5 was found to yield a sensitivity of 85.2% and a specificity of 73.9% for predicting high-risk PE. These findings suggest that the Wells score, although originally developed for estimating PE probability, may also provide useful prognostic information in identifying patients at greater clinical risk (Figure 1).

Discussion

Pulmonary embolism (PE) is a significant contributor to morbidity and mortality worldwide and represents a diagnostic and prognostic challenge due to its variable clinical presentation and potential for rapid deterioration (1). Clinical decision tools such as the Wells score have been widely implemented to estimate the pre-test probability of PE, thereby guiding diagnostic imaging and initial management (3). Separately, scoring systems such as the Pulmonary Embolism Severity Index (PESI) and its simplified version (sPESI)

have been developed and validated as reliable tools for risk stratification and mortality prediction in confirmed PE cases (5, 6).

In the present study, a statistically significant relationship was identified between the Wells score and PE severity as classified by the PESI. Patients categorized as high-risk by PESI tended to have higher Wells scores, and a moderate positive correlation between these two scores was observed. These findings suggest that the Wells score, while not originally developed for prognostic purposes, may provide some degree of predictive insight into PE severity and short-term clinical outcomes. While some studies have suggested that the Wells score may serve as a predictor of poor clinical outcomes in patients with pulmonary embolism, other studies have found no significant prognostic utility, indicating that the score may be limited to diagnostic use rather than risk stratification (7, 8). Although the Wells criteria do not incorporate hemodynamic status or comorbidities—as PESI does—they include several variables indicative of disease burden, such as tachycardia, history of malignancy, and clinical signs of DVT, which may indirectly reflect the severity of embolic burden (3).

Table 3. Diagnostic Performance of the Wells Score in Predicting High-Risk Pulmonary Embolism

	Wells Score
Cut Off	≤2.5
AUC (%95CI)	0.780 (0.638-0.922)
Sensitivity	85.2 %
Specificity	73.9 %
p value	**0.001**

The ROC curve analysis in this study also supports the potential prognostic value of the Wells score. An area under the curve (AUC) of 0.780 was found for predicting high-risk PE based on PESI classification, which demonstrates fair to good discriminative capacity. This diagnostic performance, particularly with a cut-off of ≤2.5 yielding both high sensitivity and specificity, reinforces the hypothesis that Wells scoring may have clinical utility beyond diagnostic triage. It is also noteworthy that while PESI and sPESI have consistently shown high prognostic performance, they require a confirmed diagnosis of PE and access to full clinical data for calculation (5, 6). In contrast, the Wells score is designed for use at the bedside during the initial assessment, potentially offering earlier insights into both diagnostic and prognostic considerations. If future research validates this finding, the Wells score could be integrated into early-stage risk stratification algorithms, potentially improving triage and disposition decisions in emergency settings.

Our findings complement existing literature and highlight a clinically relevant hypothesis: that diagnostic and prognostic tools are not mutually exclusive, and that early clinical judgment (as embedded in the Wells score) may inherently capture elements of disease severity. While this study does not propose replacing PESI or sPESI with the Wells score for prognostication, it supports the idea that high Wells scores should raise not only diagnostic suspicion but also clinical vigilance for complications and deterioration.

Limitations

This study has several limitations. First, the sample size was relatively small (n=50), which may limit the statistical power and generalizability of the findings. The study's retrospective design also introduces the possibility of selection and information bias, as data were collected from patient records rather than through prospective observation. Additionally, while the Wells score was calculated using standard criteria, interobserver variability in its application was not assessed. Importantly, this study focused solely on the correlation between scoring systems and did not include clinical outcomes such as mortality, ICU admission, or thrombolysis rates, which would provide further insight into the prognostic utility of the Wells score.

Conclusion

In conclusion, this study suggests that the Wells score, while traditionally used for estimating the likelihood of pulmonary embolism, may also reflect the clinical severity of PE as determined by PESI and sPESI classifications. A statistically significant association between Wells and PESI scores was identified, and ROC analysis demonstrated good discriminatory ability for identifying high-risk patients. These findings support the hypothesis that the Wells score may offer additional prognostic value in emergency department settings, particularly during the early stages of evaluation when comprehensive scoring tools such as PESI may not yet be feasible. Further large-scale prospective studies are warranted to validate these observations and to explore the integration of diagnostic and prognostic scoring tools in the comprehensive management of PE.

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Investigating the Therapeutic Potential of Facet Injections for Lower Back Pain

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Introduction

Worldwide, debilitating lower back pain is the leading cause of disability, with a reported prevalence of 7.3%, the majority of cases being found in middle-to-low income countries (1). Although there are few epidemiological studies covering the overall emergency admissions in Turkey, a regional study showed that lower back pain accounts for approximately 6.5% of emergency department visits among patients aged 18–64. Moreover, in patients aged 45–64, lower back pain was the most common complaint (2).
The mere radiological demonstration of facet osteoarthritis is not sufficient to determine the cause of a patient's lower back pain; in fact, these findings can be incidentally observed in patients without lower back pain (1,3). Despite numerous clinical and radiological methods historically described for identifying facet-mediated pain, their sensitivity and specificity remain low (3).

Facet Joint as a Cause of Lower Back Pain

The facet joint is a synovial joint located between the superior and inferior articular processes of adjacent vertebrae in the spine. It is lined with hyaline cartilage, enclosed by a fibrous capsule, and lubricated by synovial fluid. Its orientation varies by spinal region: cervical joints are angled obliquely to allow rotation, while lumbar joints are aligned sagittally to restrict rotation and support flexion/extension. These joints limit excessive spinal movement, distribute mechanical loads, and contribute to stability (4).
Key stabilizing ligaments include the ligamentum flavum, the interspinous and supraspinous ligaments, and the joint capsule, which prevents hypermobility. The facet joint is innervated by the medial branch of the dorsal ramus from the spinal nerves, receiving dual innervation from adjacent levels (e.g., the L4–L5 joint is supplied by the L3 and L4 nerves). This innervation transmits pain signals and provides proprioceptive feedback (4).
Among the many causes of lower back pain are the intervertebral discs, facet joints, ligaments, sacroiliac joints, myofascial pain, and spinal nerves (3–6). While discogenic pain can be readily distinguished by clinical examination and imaging findings, provocation tests and the absence of midline pain are informative in differentiating sacroiliac and facet joint pain. Increased uptake on SPECT (single-photon emission computerized tomography) of the facet joints is regarded as the only reliable imaging method for facet joint–mediated pain; however, this imaging technique is expensive, involves considerable radiation exposure, and is not recommended as a first-line diagnostic tool (3,6).

Method

With the patient in the supine position on a radiolucent table, the joints identified via clinical examination and MRI are targeted using an anteroposterior (AP) imaging view. A 22-gauge, 90-mm spinal needle is advanced to the lateral upper quadrant of the facet joint so that the needle makes subcapsular contact with the superior articular process. A syringe is prepared by mixing 2.5 cc of betamethasone solution (Cales, Koçak Pharmaceuticals, Turkey) with 7.5 cc of bupivacaine solution (Marcaine 0.5%, Eczacıbaşı Pharmaceuticals, Turkey). This mixture is then injected—0.2–3 cc is administered into the capsule, after which the needle is slightly retracted to the extracapsular space and an additional 1 cc is applied. The estimated injection time per joint is 1–5 minutes.
This method is an amalgamation of two techniques: intra-articular injection and median nerve block, aimed at addressing multiple sources of pain generation within the facet joint. The goal is not to pinpoint the exact source of the pain but to relieve it, since further radiofrequency ablation is not planned for the selected patient group. The modified Oswestry Disability Index (ODI) scale was completed by all patients before the injection and one month afterward. In addition to the standard test, a body diagram was used to record the location of pain, the patients' history of lumbar surgery, medication usage, and the number of emergency department or outpatient clinic visits.
The assumption of normal distribution for quantitative variables was verified using the Shapiro–Wilk test. Descriptive statistics for normally distributed quantitative variables were presented as mean ± standard deviation. For categorical variables, frequencies were shown (%). For independent group comparisons, an independent samples t-test was applied to normally distributed variables. The relationship between groups and categorical variables was examined using the chi-square test. Repeated measures were analyzed with repeated-samples ANOVA. A p-value of <0.05 was considered statistically significant.

Patient Selection

The criteria for patient selection were as follows:
The presence of pain radiating from the lower back to the hips and further down;
The patient's ability to indicate with a finger the point of maximum pain in the lower back, which also triggers the pain;
MRI findings demonstrating facet degeneration and synovial dehydration;

Superimposition of the degenerative facet seen on MRI with the area indicated by the patient.
After achieving pain control, all patients were advised to perform back muscle strengthening exercises.

Results

This study included 23 patients. The ODI scores before the facet joint injection were a mean of 63.22 ± 16.25 (range 32–98), and one-month post-injection the mean was 16.00 ± 15.29 (range 0–54), with the change being statistically significant ($p < 0.001$). (Fig. 1) When comparing medication usage before and after the procedure, the frequency of analgesic use due to lower back pain changed from 3–4 times per week to no use within one month, and this difference was statistically significant ($p < 0.001$). (Fig. 2) Although the frequency of outpatient clinic visits for lower back pain varied between 1 and 3 per month, the number of visits in the one-month period following the procedure approached zero ($p < 0.001$). (Fig. 3) Pain intensity, described by the patients as severe to very severe prior to the injection, was evaluated as mild to very mild one-month post-injection, with this difference also being statistically significant ($p < 0.001$). (Fig. 4) The ODI scores of patients with a history of surgery (73.43 ± 12.84) were higher than those of patients without a history of surgery (58.75 ± 15.84) ($p < 0.44$). Approximately 79.3% of the patients stated that if their symptoms recurred, they would be willing to undergo another facet joint injection.

Discussion

A study conducted by the World Health Organization (WHO) demonstrated that needle insertion into the painful area may play a role in pain control, albeit with a low to very low level of evidence. In addition, theories such as the gate-control theory of pain, endogenous opioid release, and local physiological changes in blood flow have been suggested as contributing factors (7).

In another WHO study on low back pain, it was indicated—with moderate certainty evidence—that exercise is therapeutic in terms of pain control and functional improvement in both adults and older adults (8).

In our study, appropriate patient selection led to the immediate resolution of pain in individuals presenting with lower back pain, with this effect lasting for at least one month. This not only enhanced the patients' quality of life but also enabled them to initiate early-stage exercise programs, thereby contributing to the overall therapeutic effect. The injection technique, through the dispersion of the medication into surrounding tissues, targets multiple anatomical structures that could contribute to facetogenic pain, thus exhibiting a therapeutic benefit.

Meta-analyses have indicated, with level III evidence, that facet joint injections can have long-term efficacy (9). The long-term outcomes of the current study are being monitored and will be reported in future research.

Significant pain control was achieved in patients presenting with severe lower back pain. A notable reduction in ODI scores was observed, and the procedure substantially improved quality of life. Consequently, both the use of analgesic medications and the frequency of outpatient clinic visits due to lower back pain significantly decreased. Among patients with lower back pain, those with a history of lumbar surgery exhibited higher disability scores compared to those without such a history; however, pain control was achieved in this subgroup as well. No side effects related to the injection or the administered medications were observed in any of the patients.

The strength of our study lies in the fact that the described procedure is performed in a very short time, employs simple MRI indication parameters, involves minimal radiation exposure, and prevents the prolonged use of prescription analgesics—thereby reducing recurrent hospital visits. This, in turn, decreases loss of workforce and reduces the burden on the healthcare system.

Conclusion

Lower back pain is one of the most frequently debilitating symptoms worldwide. In patients without a pathology requiring surgery, or in those who continue to experience lower back pain despite improvement in neurogenic symptoms after surgery, facet joint injection not only enhances the patient's quality of life but also reduces the burden on the healthcare system. In this context, with appropriate patient selection, facet joint injections can be considered a therapeutic option. This study serves as a stepping stone for future research.

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Figures

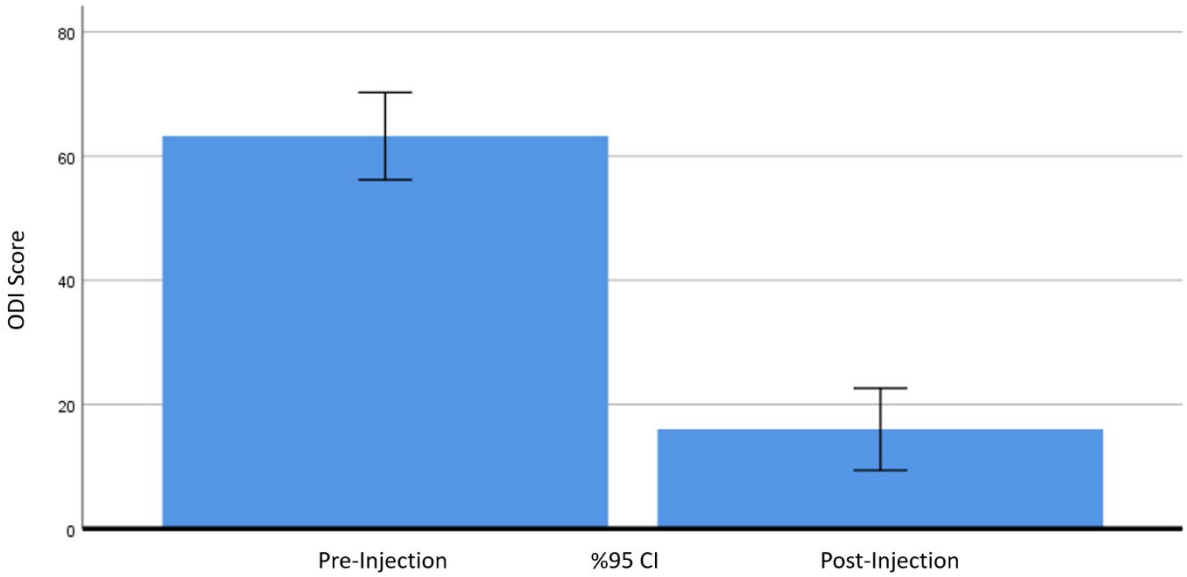

Fig.1: ODI score markedly decreased one month after injection.

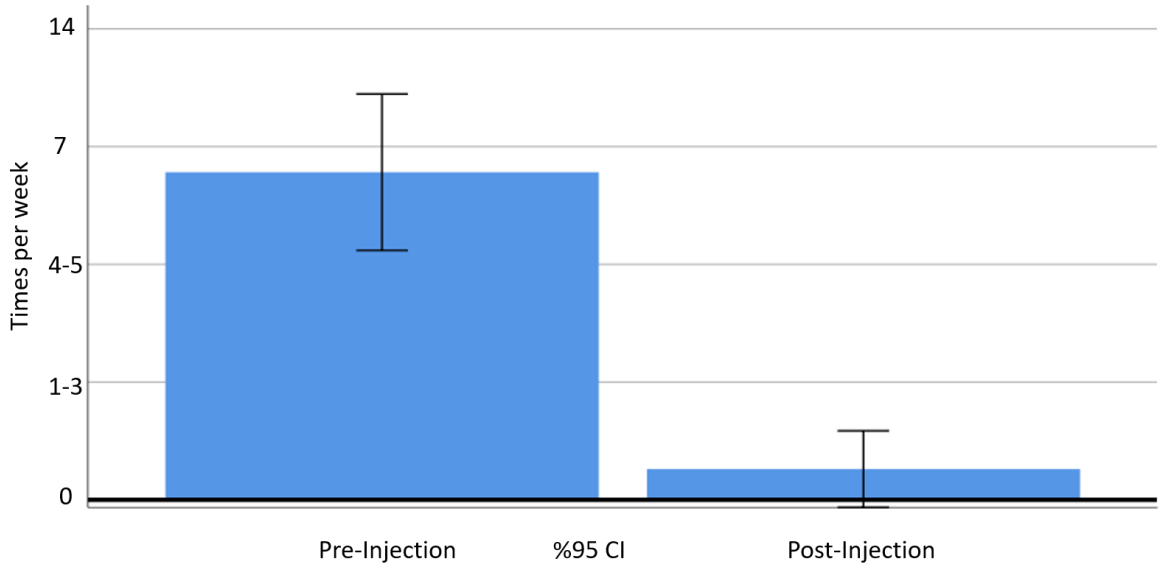

Fig.2: Number of drugs used per week for lower back pain markedly decreased one month after injection.

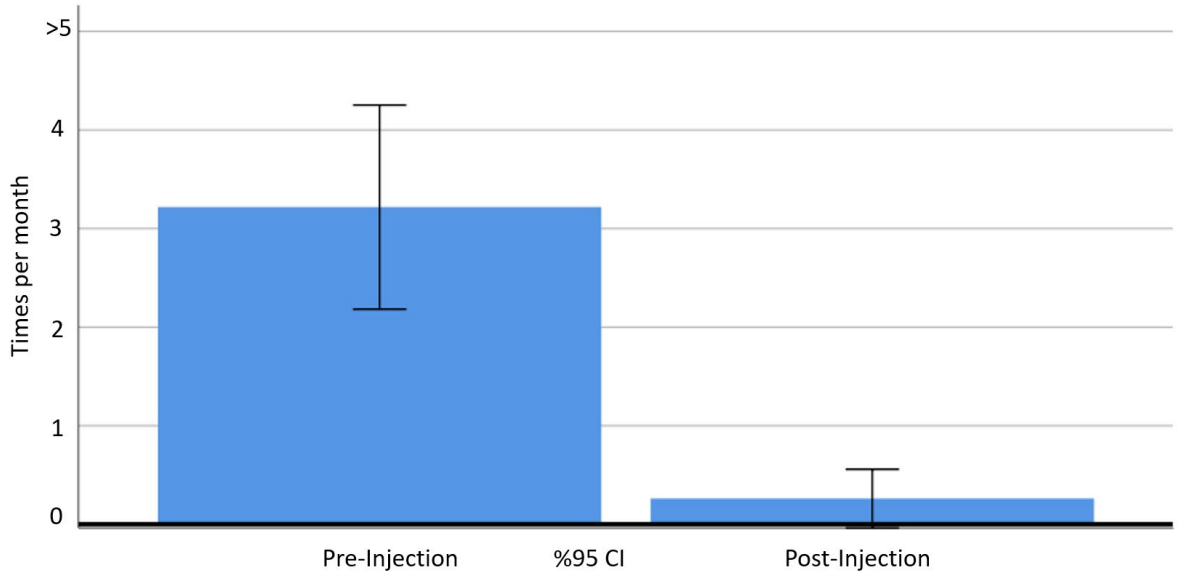

Fig.3: Outpatient/emergency service visits per month markedly decreased one month after injection.

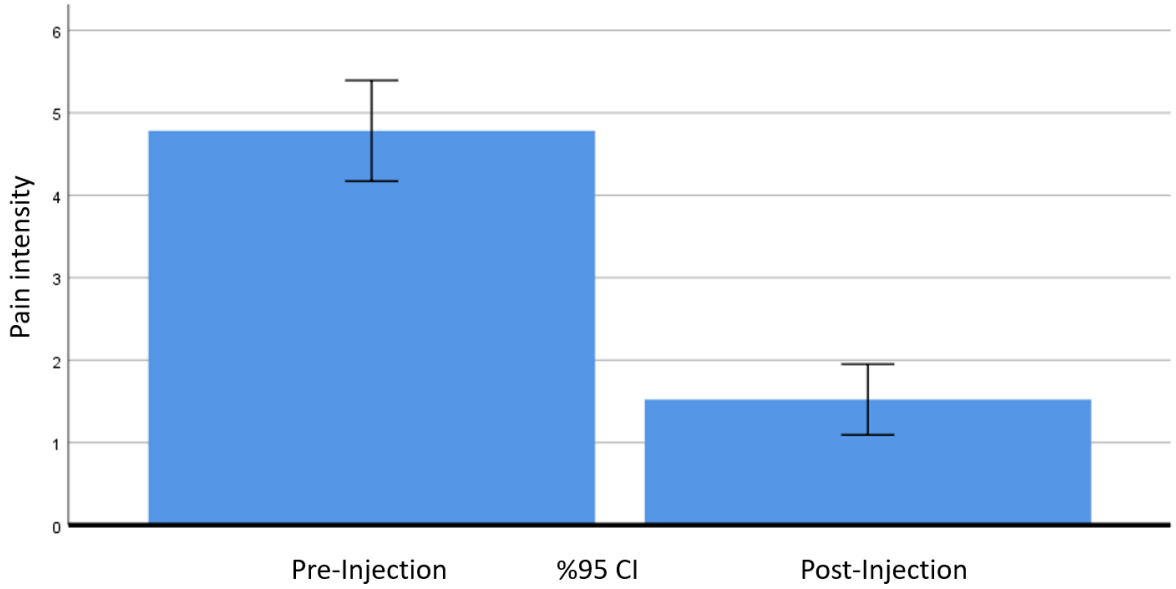

Fig.4: Pain intensity markedly decreased from severe/very severe to mild/very mild one month after injection.

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One CT, One Chance: Rapid Neurological Collapse from a Spontaneous Acute-on-Chronic Subdural Hematoma in an Elderly Patient on Clopidogrel

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Introductıon: Spontaneous acute-on-chronic subdural hematoma (SDH) in elderly patients without a history of trauma remains a rare yet life-threatening entity. In the emergency department (ED), the absence of trauma and the presence of non-specific symptoms often delay recognition. This case report presents an uncommon clinical course in which an elderly woman on clopidogrel, initially neurologically intact, suffered rapid deterioration and was diagnosed with a surgically significant acute-on-chronic SDH on her first cranial CT—emphasizing the role of early suspicion and decisive imaging in emergency medicine.

Case: An 87-year-old female presented to the emergency department with dizziness, nausea, vomiting, and generalized weakness over the past three days. On the morning of admission, she experienced a transient episode of slurred speech lasting approximately

six hours, followed by apparent improvement. Her family reported increasing confusion and somnolence over the hour preceding arrival.

Her past medical history included hypertension, atrial fibrillation, congestive heart failure, prior ischemic stroke (one year ago), and left hip arthroplasty (six months ago). She was taking clopidogrel 75 mg daily, escitalopram 5 mg, betahistine 24 mg twice daily, and perindopril 5 mg. There was no reported history of trauma or head injury.

On arrival, vital signs were: blood pressure 132/72 mmHg, heart rate 72 bpm (irregular), respiratory rate 16/min, temperature 37.1°C, and oxygen saturation 96% on room air. The patient was initially alert and oriented, with a GCS of 15. Cranial nerve exam was unremarkable, motor strength was 5/5 in all extremities, reflexes were symmetric, and no ataxia or meningeal signs were present. Initial labs revealed potassium 2.59 mEq/L, calcium 7.94 mg/dL, INR 2.4, and elevated CRP (156 mg/L). ECG showed atrial fibrillation with controlled rate.

While being monitored in the ED, the patient's mental status deteriorated rapidly within an hour. GCS dropped to 9 (E3M4V2). She developed dysarthria and intermittent dystonic posturing of the left arm. Blood pressure acutely rose to 200/100 mmHg. A single emergent non-contrast cranial CT revealed a right-sided crescentic, mixed-density subdural collection consistent with acute-on-chronic SDH, causing marked mass effect, effacement of the right lateral ventricle, and a midline shift to the left. Hyperdense layering in the frontal region suggested subacute hemorrhagic components. Notably, this diagnosis was established on the first CT, performed only after acute neurological decline.

Neurosurgery was urgently consulted. ED management included head elevation, intravenous mannitol, dexamethasone (4 mg every 6 hours), levetiracetam (500 mg twice daily), ceftriaxone 2 g for prophylaxis, and blood product preparation (2 units erythrocyte suspension, 2 units fresh frozen plasma). The patient was swiftly transferred for emergent burr-hole evacuation. Postoperative recovery in the intensive care unit was uneventful, with improving consciousness and imaging-confirmed hematoma reduction.

Dıscussıon: This case highlights a rare but critical emergency medicine scenario: a spontaneous, non-traumatic acute-on-chronic subdural hematoma in an elderly patient on clopidogrel, diagnosed on the first CT scan obtained following rapid clinical deterioration. Subdural hematomas typically present insidiously in the elderly, and clinical suspicion may be low in the absence of trauma. However, antiplatelet therapy and cerebral atrophy create a physiologic environment prone to spontaneous hemorrhage, even from minor unnoticed events or without any precipitating factors. In this case, the hematoma likely developed chronically and re-bled acutely, leading to sudden decompensation.

What makes this case distinctive is the absence of trauma, the subtle prodromal symptoms, and the fact that diagnosis was made on a single CT, timed precisely after deterioration. Many such patients are misdiagnosed with metabolic encephalopathy or transient ischemic events, especially when early imaging is unremarkable or delayed. This patient's outcome hinged on the emergency physician's clinical vigilance and swift action.

Additionally, the patient's use of clopidogrel and elevated INR significantly increased the risk of bleeding. The combination of atrial fibrillation, a history of cerebrovascular disease, and recent surgery further complicated her risk profile.

Emergency physicians must maintain a high index of suspicion in elderly patients with fluctuating neurological symptoms, even in the absence of trauma. The decision to obtain neuroimaging should not be deferred, especially in patients on antiplatelet agents. This case serves as a reminder that in emergency care, timing is diagnosis, and one CT can change everything.

In conclusion, this case illustrates a dramatic neurological collapse due to a spontaneous acute-on-chronic subdural hematoma in an elderly woman on clopidogrel, diagnosed on a single CT following a rapid clinical change. In the absence of trauma, such presentations can easily be missed. Emergency physicians must remain alert to subtle red flags and act decisively. Early imaging, appropriate neuroprotective measures, and timely surgical intervention were critical in altering the trajectory of this patient's outcome. This case reinforces the need for aggressive evaluation of neurologic changes in elderly, anticoagulated patients—where one opportunity for diagnosis may be all that exists.

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A Rare Case: Brain Abscess Presenting to the Emergency Department with Drowsiness

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Introduction:

Brain abscess is a rare diagnosis in patients presenting to the emergency department with nonspecific symptoms such as headache, altered consciousness, and drowsiness. It is a local bacterial infection of the brain parenchyma surrounded by a central purulent infection, potentially requiring urgent neurosurgical intervention. Neurological imaging is crucial for diagnosis. As in our case, contrast-enhanced MRI studies, which can be confused with acute ischemic infarction, facilitate definitive diagnosis.

Case Report:

A 65-year-old male presented with a 3-day history of headache and excessive but easily arousable sleepiness. His medical history included coronary artery disease, hypertension, and hyperlipidemia. He had no history of antibiotic use in the last 2 months, no diagnosed dental infection, or dental intervention. He reported a recent upper respiratory tract infection that resolved without medication about a month prior.

On emergency admission, his vital signs were normal except for elevated blood pressure. His ECG showed no signs of acute ischemia. Neurological examination revealed suspicious neck stiffness and mild right hemiparesis. Blood tests showed elevated inflammatory markers (WBC 21,250 mg/dL, CRP 19.60 mg/dL, ESR 46). Brain CT and MRI diffusion imaging were performed to rule out intracranial ischemic and hemorrhagic pathologies.

MRI DWI showed a lesion with restricted diffusion, corresponding to findings on FLAIR and CT, with no signal change on SWI, extending from the medial right temporal lobe to the posterior lateral ventricle. Contrast-enhanced cranial MRI was performed due to the difficulty in differentiating the space-occupying lesion as a mass or abscess. He received analgesic treatment and 2 grams of ceftriaxone IV in the emergency department. His blood pressure was reduced to 165/80 and stabilized. Neurology, infectious disease, and neurosurgery consultations were obtained. He was scheduled for hospitalization for abscess drainage, further examination, and treatment by neurosurgery.

Discussion:

This case highlights a rare presentation of brain abscess and a challenging clinical scenario. Brain abscess should be considered in patients presenting with atypical symptoms like drowsiness. Contrast-enhanced MRI is essential for differentiating brain abscess from other pathologies and for definitive diagnosis.

Figure-1:A hypodense area is observed in the area where the brain abscess is located.

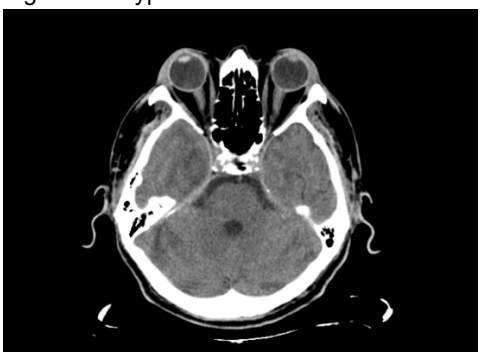

Figure-2: In the area where the brain abscess is located, diffusion binding is observed

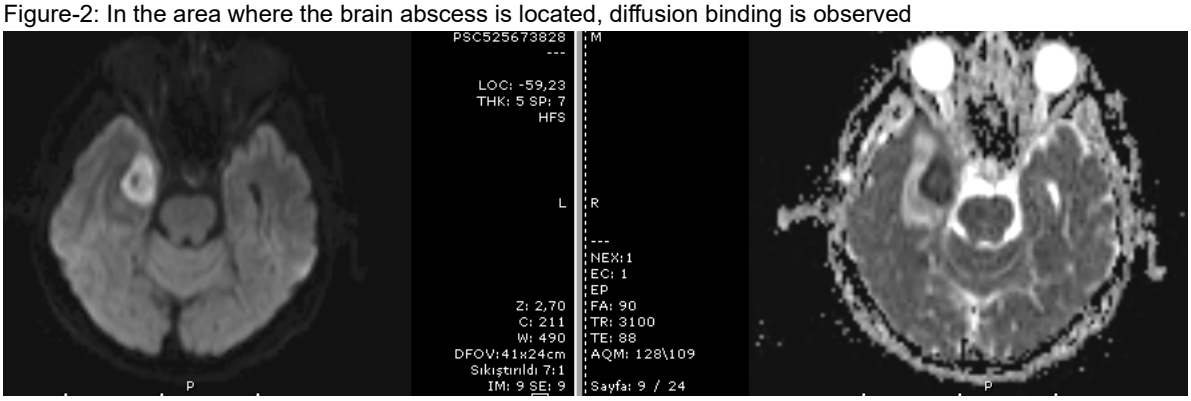

Figure-3: Peripheral contrast enhancement is observed in the area where the brain abscess is located.