Analjezi ve sedasyon komplikasyonları; ne yapalım?

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Analjezi ve sedasyon komplikasyonları; ne yapalım?

Ağrı: Vücudun belirli bölgelerinden ortaya çıkarak farkına varılan ve sıklıkla dokuların zedelenmesi sonucu oluşan hoşa gitmeyen bir duygudur

Analjezi: Hastanın bilinç düzeyini değiştirmeden ağrının kesilmesi veya hissedilmesinin azaltılması

Sedasyon: Hastanın çevreye ilgisinin ve dış uyaranlara karşı cevaplılığının azaltılmasıdır

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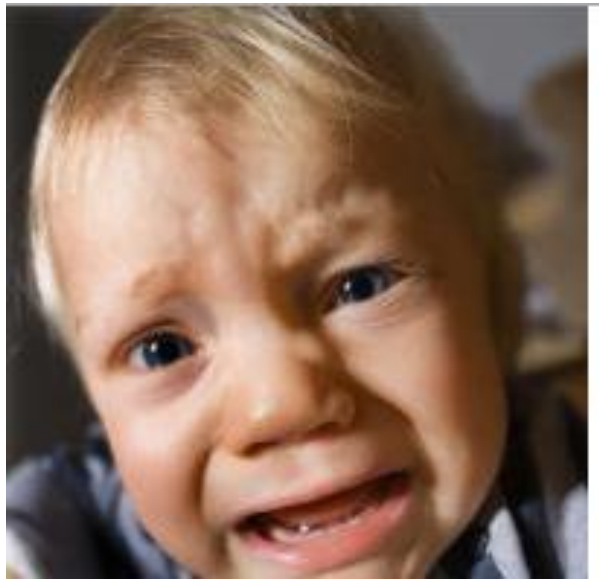
TABLE 37-1 Levels of Sedation and Analgesia

	Responsiveness	Airway	Breathing
Minimal sedation (aka "anxiolysis")	Normal but slowed response to verbal stimulation	Unaffected	Unaffected
Moderate sedation (aka "conscious sedation")	Purposeful response to verbal or physical stimulation	Usually maintained	Usually adequate
Deep sedation	Purposeful response after repeated or painful physical stimulation	May be impaired	May be suppressed

Neden Sedasyon Analjezi ?

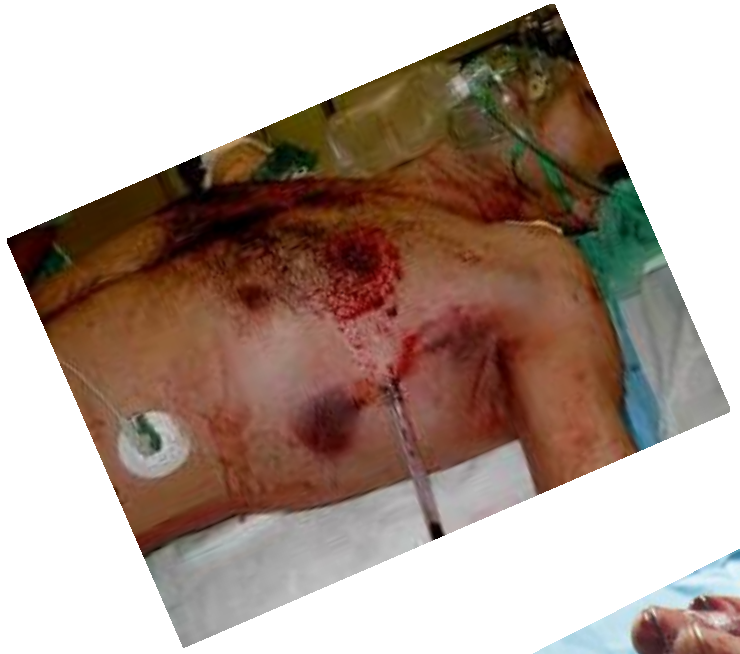


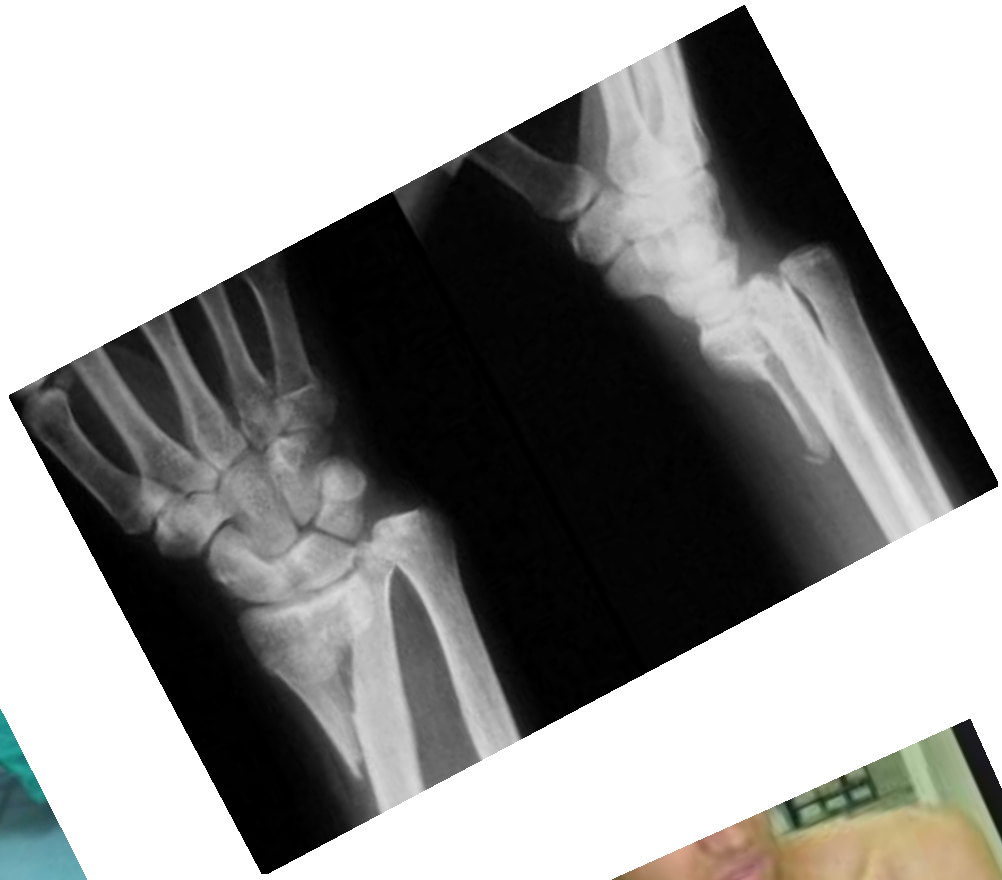
Ağrının giderilmesi

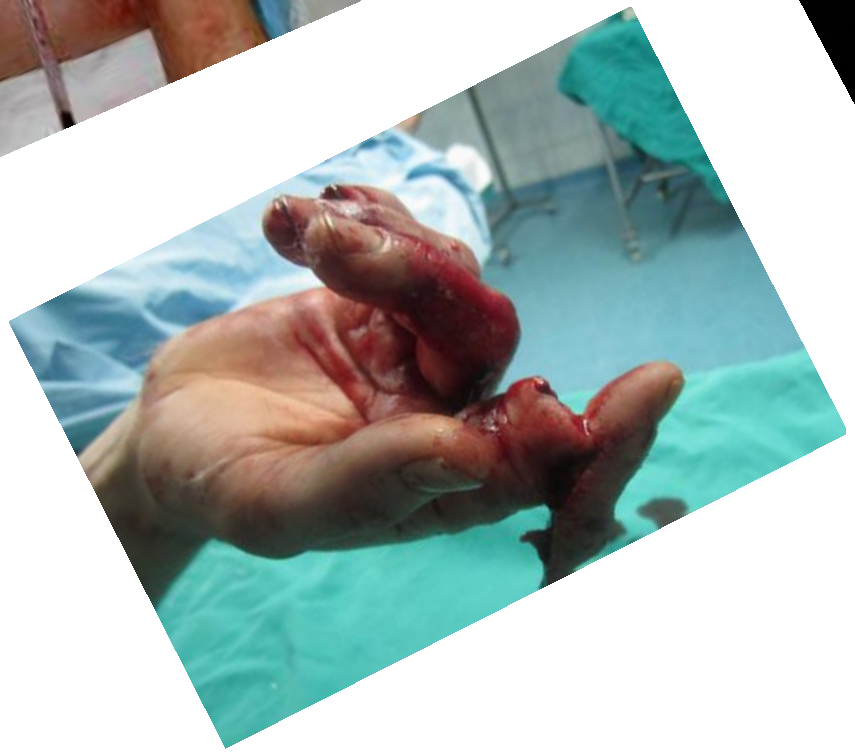
Hastanın korku ve endişelerinin giderilmesi

Hasta ve hekim memnuniyeti

Neden Sedasyon Analjezi ?







Neden uygulamaktan çekiniyoruz?

Aşırı sedasyon korkusu...

Komplikasyonlarla başa çıkamama...

Kullanılan ilaçlar hakkında ve komplikasyonları hakkında bilgi sahibi olmama

Fizik muayene bulgularını değiştirir mi?

Neden uygulamaktan çekiniyoruz?

Hemodinamik ve solunumsal bozukluk

Hastanın tedaviyi kabul etmemesi

Hekimin havayolu yönetimi,

ileri kardiyak yaşam desteği bilgisi

Neden uygulamaktan çekiniyoruz?

Hastanın monitorizasyonu için gerekli malzemelerin olmaması

Kullanılacak olan ilaçlara karşı alerji


Genel anesteziye gerek yoktu, lokal yaptık. Sadece dilini uyuşturduk.
Çığlık atıp bağırma diye.
Erdil Yaşaroğlu © komikaze.net


şimdi anestezi uzmanı geliyor. seni uyutucak ve hemen ameliyata başlıycaz tamammı?

Analjezi ve sedasyon komplikasyonları; ne yapalım?

Sedoanaljeziye bağlı mortalite ve morbidite oranı düşüktür

Acil servislerde komplikasyon oranı **% 2,3-7,7**

Allerjik reaksiyon

Solunum depresyonu

Kardiyo pulmoner arrest nadiren görülebilir

Sedasyon ve Analjezide Önemli Noktalar

Uygun koşullar	Eğitilmiş personel	Hasta değerlendirilmesi
Eğitilmiş personel...	Uygulanan ajanı tanıma	SAMPLE...
Hasta değerlendirilmesi...	Bilinç ve klinik gidiş takibi...	Endikasyon...
Onaylanmış izin...	Komplikasyon ve yan etki...	Fasting time...
Yardımcı alet ve destek...	İleri havayolu ve KPR deneyimi	Vitaller, havayolu (?) ,
Monitörizasyon...	Destek personel...	Bilinç ....
Dokümantasyon...		FM zorunlu...
Taburculuk tavsiyeleri...		

Sedoanaljezi-açlık süresi

Genel olarak yakın zamanda oral alımda aspirasyon riski **derin sedasyonda** artar

Yakın zamanda oral alım yüzünden sedasyonun yapılması ya da ertelenmesi kararı verilirken bu risk işlemin aciliyeti ile dengelenmelidir

İşlemden önce oral alımı ***üç saatten fazla*** sürede olan hastalar herhangi bir sedasyon seviyesinde düşük aspirasyon riski taşırlar

ASA Sınıflaması ve Sedasyon Riski

(American Society of Anesthesiologists-Amerikan Anestezistler Derneği)

Sınıflama	Sedasyon Riski
ASA 1 **(Normal sağlıklı insan)**	Minimal
ASA 2 **(Hafif sistemik hastalık, normal hayat)**	Düşük
ASA 3 (Orta-ciddi derecede sistemik hastalık, bazı fonksiyon sınırlaması mevcut)	Orta
ASA 4 **(Hayatı tehdit eden ciddi sistemik hastalık mevcut)**	Yüksek
ASA 5 **(Ölmek üzere olan hasta)**	Aşırı yüksek

Miller MA, Levy P, Patel MM. Procedural sedation and analgesia in the emergency department: what are the risks? Emerg Med Clin North Am. 2005 May;23(2):551-72.

Analjezi ve sedasyon komplikasyonları; ne yapalım?

Monitörizasyon

İnteraktif

- Bilincin görsel ve verbal takibi
- Bulantı, kusma

Mekanik

- Kan basıncı
- Nabız sayısı
- Oksijen satürasyonu
- Solunum sayısı
- ET_{CO2}

Analjezi ve sedasyon komplikasyonları; ne yapalım?

Malzemeler

Yüksek akımlı oksijen kaynağı

Aspiratör ve geniş sondalar

Damar yolu gereçleri

Havayolu gereçleri

Monitor gereçleri (EKG, Pulse oksimetre, TA)

Resüsitasyon ilaçları

Geri döndürücü ilaçlar

Yeterli ekip

Analjezi ve sedasyon komplikasyonları; ne yapalım?

Taburculuk kriterleri

En az yarım saat süreyle stabil vital bulgular

Respiratuar distres bulgularının olmaması

Bulantı, kusma veya sersemlik hissinin olmaması veya çok az olması

Alert, oriente olması ve komutlara uyması

Oral alım olması

Sorumlu bir yakının olması

Post anestezik derlenme skoru (Modifiye Aldrete skorlaması)

BİLİNÇ DURUMU	**Tamamen uyanık ve oryante (isim, yer,zaman)**	**2**
	Sesli uyarana yanıt mevcut	**1**
	Yanıt yok	**0**
AKTİVİTE	**Tüm ekstremitelerini istemli olarak ve emirlere uygun hareket ettiriyor**	**2**
	Sadece iki ekstremiteyi hareket ettiriyor	**1**
	Hareket yok	**0**
SOLUNUM	**Derin soluyabiliyor ve öksürebiliyor**	**2**
	Dispne, kısıtlı solunum veya takipne	**1**
	Apneik veya mekanik ventilasyon desteğinde	**0**
DOLAŞIM	**Kan basıncının preanestezik ölçümün ± %20'si seviyesinde olanlar**	**2**
	Kan basıncının preanestezik ölçümün ± %20-49'u seviyesinde olanlar	**1**
	Kan basıncının preanestezik ölçümün ± %50'si seviyesinde olanlar	**0**
OKSİJEN SATURASYONU	**Oda havasında SpO_2 > % 92**	**2**
	SpO_2 > %90 düzeyinde tutmak için O_2 desteğine ihtiyaç duyanlar	**1**
	O_2 desteğine rağmen SpO_2< %90 olması	**0**
TOPLAM SKOR	**İdeal olarak hasta toplam skoru 10 olduğu zaman taburcu edilmeli (minimum 9)**	

(Aldrete JA. The post-anesthesia recovery score revisited.J Clin Anesth 1995;7:89–91.)

İlaçlar

Lokal Anestezi

Ketamin Komplikasyonları

Fentanyl Komplikasyonları

Benzodiazepin Komplikasyonları

Etomidate Komplikasyonları

Lokal Anesteziklere Bağlı Toksisite

Bupivakain, mepivakain ve lidokain intravasküler uygulanması

Refrakter nöbetler
Kardiyovasküler kollaps

% 20 İntravenöz lipid uygulanması 1.5 ml/kg bolus

İntravenöz Lipid Emülsiyonu

2015 AHA

Nörotoksisite belirtileri görülen veya lokal anestezik toksisitesine bağlı kardiyak

arrest olan hastalarda, standart resüsitatif bakım ile eşzamanlı olarak

İntravenöz Lipid Emülsiyonu uygulanabilir

Ketamin

0.5-1.0 mg/kg iv; 2-4 mg/kg im

Disosiyatif analjezi, sedasyon, amnezi

Etkinin başlangıcı 1-2 dk

Etki süresi 30-60 dk

Ketamin

Santral Sinir Sistemi

Etkilerini opiat benzeri ve NMDA reseptörleri üzerinden gösterir

Yan etkilerinde Naloxan kullanılabilir

Ketamin

Ketamin kullanımı sonucu istenmeyen psikolojik reaksiyonlar ortaya çıkabilir

- Canlı rüyalar
- İlizyonlar

Bu yan etkiler yaşla ↓, dozla ↑, erkeklerde ↓ değişebilir

Yan etkileri için benzodiazepinler (midazolam, lorazepam, diazepam) kullanılabilir

Ketamin Kardiyovasküler Sistem

Ketamin kan basıncını, nabızı, kardiyak outputu artırır

Benzodiazepinler, inhale anestezikler ve propofol ketaminin bu etkilerini azaltır

Hemodinamik değişiklikler ketamin dozuyla ilişkili değildir

Ketamin

Ketamin için en çok üzerinde durulan konu ICP artışıdır.

Doğal olarak TBY hastalarında kullanılması noktasında çeşitli tereddütler mevcuttur

Prospektif bilgisi ve FDA halen uyarıyor

Literatür incelendiğinde günümüze kadar olan çalışmalarda farklı sonuçlar elde edilmiştir

Table 1. Cerebral Hemodynamic Effects of Ketamine in Human

Reference	Quality score	Size/study group	Dose, ketamine	Hemodynamics ↑ increase, ↓ decrease, ↔ no change	Study setting/ intracranial compliance	Concomitant medication	Ventilation
Mayberg et al. (11) 1995	NA	20 Ket	1 mg/kg bolus, racemic	↔ MAP, CPP, $AVDO_2$ ↓ VMCA, ICP	Neurosurgical patients, mildly raised ICP	Isoflurane anesthesia with N_2O	Controlled, normoventilation
Strebel et al. (12) 1995	3	6/6/6/6 Ket/ C/Mid/ Esm	2 mg/kg bolus, racemic	↑ VMCA and ↑ MAP ↓ VMCA and ↔ MAP ↑ VMCA and ↓ MAP	Neurosurgical patients, no cerebral compromise	Isoflurane anesthesia + 0.1 kg/mg midazolam, or 5 mg esmolol boli	Controlled, normoventilation
Kolenda et al. (16) 1996	5	16/17 Ket/ Fen	65 mg/kg/d racemic, cont. infusion	↑ MAP and CPP, compared to fentanyl	Head-injured patients, ICU/ increased ICP	6.5 mg/kg/d midazolam, cont. infusion	Controlled, normoventilation
Albanese et al. (17) 1997	NA	8 Ket	1, 3, 5 mg/kg racemic, bolus	↔ MAP, CPP, $SvjO_2$, ↔ VCMA/ ↓ ICP	Head-injured patients, ICU/ increased ICP	3 mg/kg/h propofol, cont. infusion	Controlled, normoventilation
Bourgoin et al. (18) 2003	5	12/13 Ket/ Suf	4.92 ± 1.5 mg/ kg/h racemic, cont. infusion	↔ ICP and CPP, ↑ HR, compared to sufentanil	Head-injured patients, ICU/ increased ICP	98.4 ± 30 mg/kg/h midazolam, cont. infusion	Controlled, normoventilation
Sakai et al. (26) 2000	3	7/7/8 Awa/ Pro/ProKet	2 mg/kg/h racemic, cont. infusion	↔ MAP, HR, VMCA no cerebral compromise ↔ VMCA, $paCO_2$, compared to propofol alone	Surgical patients, propofol, cont. infusion	6 mg/kg/h normoventilation	Controlled or hypo- or hyper- ventilation
Nagase et al. (27) 2001	3	15/15 Ket/C	1 mg/kg racemic, bolus	↔ VMCA, compared to isoflurane alone	Surgical patients, no cerebral compromise	Isoflurane anesthesia without N_2O	Controlled, hypoventilation
Engelhard et al. (28) 2001	3	12/12 ProKet/ Sevo	2.5 mg/kg/h S+, cont. infusion	↔ autoregulatory index, compared to sevoflurane anesthesia	Surgical patients, no cerebral compromise	1.5–2.5 μg/kg/ml propofol, targeted plasma concentration	Controlled, normoventilation
Vollenweider et al. (29) 1997	NA	10/10/10 S+/R−/C	Each isomer: 15 mg bolus + 0.84–1.2 mg/kg/h, cont. infusion	S+: ↑ rCMRGlu anterior cingulate, frontal, parietal, left sensorimotor cortices, thalamus R−: ↓ rCMRGlu temporomedial cortex, left insula	Volunteers, no cerebral compromise	None	Spontaneous
Holcomb et al. (30) 2001	NA	13/10 Ket/C	0.3 mg/kg bolus, racemic	↑ rCBF in anterior cingulate, medial, inferior, frontal cortices ↓ rCBF (relative) in cerebellum	Volunteers, no cerebral compromise	None	Spontaneous
Langsjö et al. (8) 2003	NA	9 Ket	30, 100, 300 ng/ ml racemic, targeted plasma concentration	Global ↑ rCBF, highest in anterior cingulate, thalamus, putamen, frontal cortices, ↔ $rCMRO_2$, ↑ rCBV frontal cortex	Volunteers, no cerebral compromise	None	Spontaneous

Anaesthesia
Journal of the Association of Anaesthetists of Great Britain and Ireland
Anaesthesia, 2009, 64, pages 532–539 doi:10.1111/j.1365-2044.2008.05835.x

REVIEW ARTICLE

Anaesthesia in haemodynamically compromised emergency patients: does ketamine represent the best choice of induction agent?

C. Morris,[1] A. Perris,[2] J. Klein[1] and P. Mahoney[3]

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3 Defence Professor Anaesthesia, Department of Military Anaesthesia and Critical Care, Royal Centre for Defence Medicine, Birmingham Research Park, Edgbaston, Birmingham, UK

Summary
In rapid sequence induction of anaesthesia in the emergency setting in shocked or hypotensive patients (e.g. ruptured abdominal aortic aneurysm, polytrauma or septic shock), prior resuscitation is often suboptimal and comorbidities (particularly cardiovascular) may be extensive. The induction agents with the most favourable pharmacological prop
appear to be ketamine and etomidate. However, etom
countries and impairs steroidogenesis. Ketamine has b
presence of brain injury, but we argue in this review t
cranial pressure or cerebral blood flow are in fact atten
subsequent anaesthesia and the greater general haemo
Ketamine represents a very rational choice for rapid s
compromised patients.

Published in final edited form as:
CNS Neurosci Ther. 2013 June ; 19(6): 390–395. doi:10.1111/cns.12077.

The Emerging Use of Ketamine for Anesthesia and Sedation in Traumatic Brain Injuries

Lee C. Chang[1], Sally R. Raty[1], Jaime Ortiz[1], Neil S. Bailard[1], and Sanjay J. Mathew[2,3]
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SUMMARY

Background—Traditionally, the use of ketamine for patients with traumatic brain injuries is contraindicated due to the concern of increasing intracranial pressure (ICP). These concerns, however, originated from early studies and case reports that were inadequately controlled and designed. Recently, the concern of using ketamine in these patients has been challenged by a number of published studies demonstrating that the use of ketamine

Aims—The purpose of this article was to review the current literatur

, pp 37–39.

J Neurosurg Pediatrics 4:40–46, 2009

ss of ketamine in decreasing intracranial pressure with intracranial hypertension

e

M.D.,[1,3] Yoav Guilburd, M.D.,[3] Ada Tamir, Ph.D.,[3]
ILBURD, M.D.[2,3]

Care and [2]Paediatric Neurosurgery, Meyer Children's Hospital, Rambam Medical ort Faculty of Medicine, Technion, Israel Institute of Technology, Haifa, Israel

ne sedation is often needed in patients with intracranial hypertension. All widely used sedative

J Anesth (2014) 28:821–827
40-014-1845-3

ARTICLE

does not increase intracranial pressure compa ids: meta-analysis of randomized controlled tri

bing Ding · Yao Tong ·
Xiang Zhao · Hao Ren ·

ary 2014 / Accepted: 2 May 2014 / Published online: 24 May 2014
y of Anesthesiologists 2014

Ketamine is traditionally avoided in sedation
patients with risk of intracranial hyper-
er, results from many clinical trials con-
cern. We critically analyzed the published
ects of ketamine on intracranial pressure
r cerebral hemodynamics to determine
e was safe for patients with hemodynamic
rain injuries.
ystematically searched the online databases

administration as the main outc
leads to the same ICP levels as o
confidence interval (95 % CI),
There were no significant diff
pressure values between the t
95 % CI, −2.24, 4.22; $P = 0.55$)
was also comparable with opioi
cerebral perfusion pressure (M
−7.95, 5.8; $P = 0.76$).
Conclusions The results of this

MEDICAL INTELLIGENCE

Revising a Dogma: Ketamine fo Neurological Injury?

Sabine Himmelseher, MD*, and Marcel E. Durie
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Anesthesiology, University of Virginia Health System, Charlo

We evaluated reports of randomized clinical trials in the perioperative and intensive care setting concerning ketamine's effects on the brain in patients with, or at risk for, neurological injury. We also reviewed other studies in humans on the drug's effects on the brain, and reports that examined ketamine in experimental brain injury. In the clinical setting, level II evidence indicates that ketamine does not increase intracranial pressure when used under conditions of controlled ventilation, coadministration of a γ-aminobutyric acid (GABA) receptor agonist, and without nitrous oxide. Ketamine may thus safely be used in neurologically impaired patients. Compared with other anesthetics or sedatives, level II and III evidence indicates

neurocritical care society Neurocrit Care (2014) 21:163–173
DOI 10.1007/s12028-013-9950-y

REVIEW ARTICLE

The Ketamine Effect on ICP in Traumatic Brain Injury

F. A. Zeiler · J. Teitelbaum · M. West · L. M. Gillman

Published online: 11 February 2014

Abstract Our goal was to perform a systematic review of the literature on the use of ketamine in traumatic brain injury (TBI) and its effects on intracranial pressure (ICP). All articles from MEDLINE, BIOSIS, EMBASE, Global Health, HealthStar, Scopus, Cochrane Library, the International Clinical Trials Registry Platform (inception to November 2013), reference lists of relevant articles, and gray literature were searched. Two reviewers independently adverse effects were recorded. Two reviewers independently extracted data including population characteristics and treatment characteristics. The strength of evidence was adjudicated using both the Oxford and GRADE methodology. Our search strategy produced a total 371 citations. Seven articles, six manuscripts and one meeting proceeding, were considered for the review with all utilizing ketamine, while documenting ICP in severe TBI patients.

Fentanil

Morfine göre 75-125 kat daha potenttir.

Yağda çözünürlüğü fazladır

Kan beyin bariyerini çok hızlı geçer.

Etki çok kısa sürede başlar.

İntrinsik anksiyolitik ya da amnestik özellikleri yoktur

Histamin serbestleşmesi yapmaz

Fentanil Komplikasyonları

Nadiren bulantı, kusma, hipotansiyon ve diğer kardiyovasküler değişiklikler.

Hızlı verilen dozlarda (5-15 mcg/kg) nadir görülen bir yan etki, göğüs duvarı rijiditesidir.

Naloksan ile düzelmeyebilir, asiste ventilasyon gerekebilir

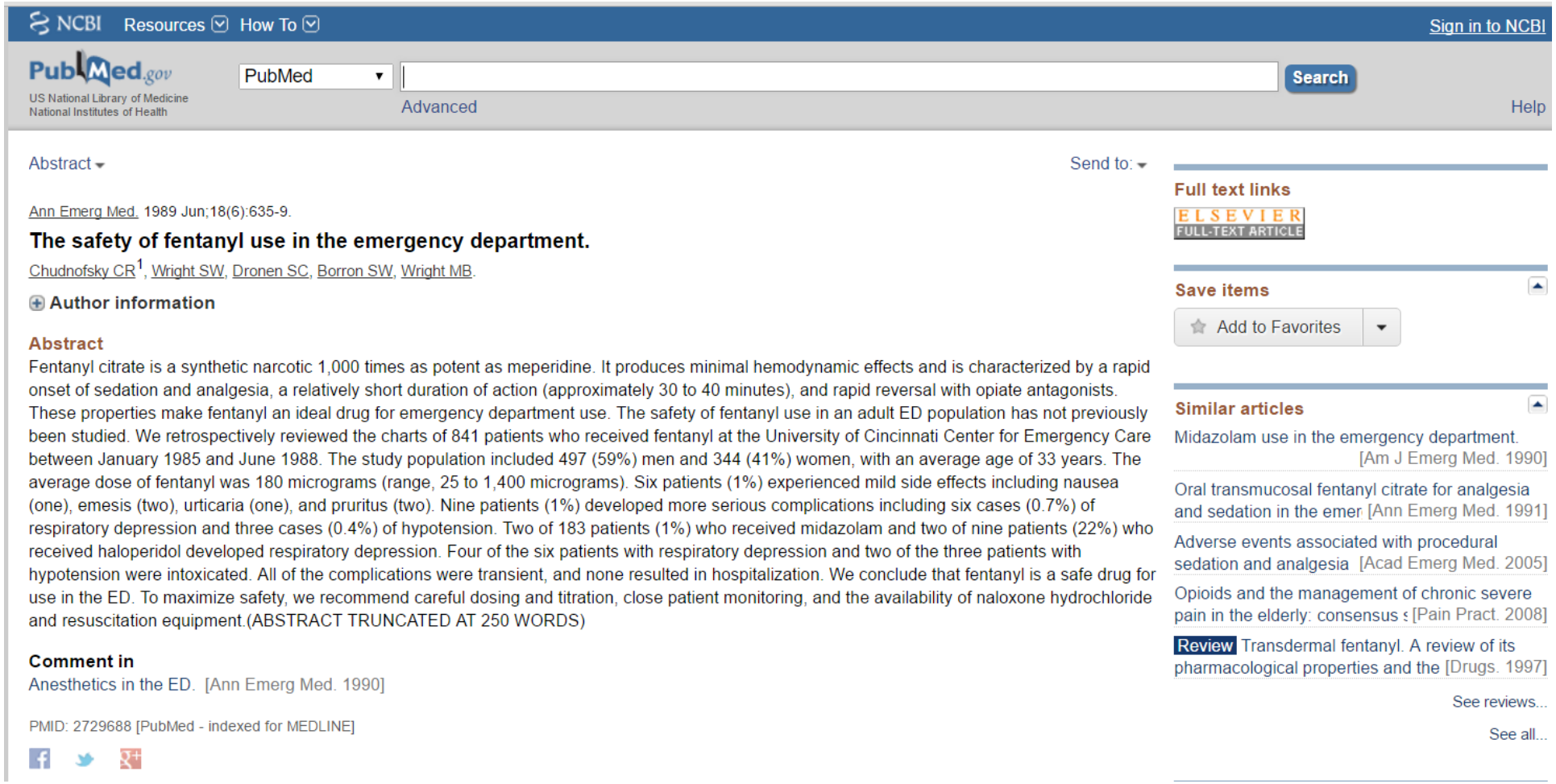
Bazen de farmakolojik paralizi (süksinil kolin) gerekli olabilir.

Fentanil Komplikasyonları

Solunum depresyonu ve göğüs duvarı rijitesi hızlı ve yüksek dozlarda görülür

Nadiren hipotansiyon yapabilir

Nöbet 50-100 mcg/kg gibi yüksek dozlarda



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Ann Emerg Med. 1989 Jun;18(6):635-9.

The safety of fentanyl use in the emergency department.

Chudnofsky CR[1], Wright SW, Dronen SC, Borron SW, Wright MB.

Author information

Abstract

Fentanyl citrate is a synthetic narcotic 1,000 times as potent as meperidine. It produces minimal hemodynamic effects and is characterized by a rapid onset of sedation and analgesia, a relatively short duration of action (approximately 30 to 40 minutes), and rapid reversal with opiate antagonists. These properties make fentanyl an ideal drug for emergency department use. The safety of fentanyl use in an adult ED population has not previously been studied. We retrospectively reviewed the charts of 841 patients who received fentanyl at the University of Cincinnati Center for Emergency Care between January 1985 and June 1988. The study population included 497 (59%) men and 344 (41%) women, with an average age of 33 years. The average dose of fentanyl was 180 micrograms (range, 25 to 1,400 micrograms). Six patients (1%) experienced mild side effects including nausea (one), emesis (two), urticaria (one), and pruritus (two). Nine patients (1%) developed more serious complications including six cases (0.7%) of respiratory depression and three cases (0.4%) of hypotension. Two of 183 patients (1%) who received midazolam and two of nine patients (22%) who received haloperidol developed respiratory depression. Four of the six patients with respiratory depression and two of the three patients with hypotension were intoxicated. All of the complications were transient, and none resulted in hospitalization. We conclude that fentanyl is a safe drug for use in the ED. To maximize safety, we recommend careful dosing and titration, close patient monitoring, and the availability of naloxone hydrochloride and resuscitation equipment.(ABSTRACT TRUNCATED AT 250 WORDS)

Comment in

Anesthetics in the ED. [Ann Emerg Med. 1990]

PMID: 2729688 [PubMed - indexed for MEDLINE]

Full text links

ELSEVIER FULL-TEXT ARTICLE

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Adverse events associated with procedural sedation and analgesia [Acad Emerg Med. 2005]

Opioids and the management of chronic severe pain in the elderly: consensus s [Pain Pract. 2008]

Review Transdermal fentanyl. A review of its pharmacological properties and the [Drugs. 1997]

See reviews...

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Acil serviste kullanımında komplikasyon oranı oldukça azdır. Bu çalışmada, fentanil kullanılan 841 acil servis hastasının sadece 6'sında solunum depresyonu, 3 hastada ise hipotansiyon geliştiği bildirilmiştir

CHEST Selected Reports

Fentanyl-Induced Chest Wall Rigidity

Başak Çoruh, MD; Mark R. Tonelli, MD; and David R. Park, MD

Fentanyl and other opiates used in procedural sedation and analgesia are associated with several well-known complications. We report the case of a man who developed the uncommon complication of chest wall rigidity and ineffective spontaneous ventilation following the administration of fentanyl during an elective bronchoscopy. His ventilation was assisted and the condition was reversed with naloxone. Although this complication is better described in pediatric patients and with anesthetic doses, chest wall rigidity can occur with analgesic doses of fentanyl and related compounds. Management includes ventilatory support and

bronchoscopy was performed for airway anesthesia and inspection. The patient received a total of 150 µg IV fentanyl and 4 mg IV midazolam in divided doses during this 20-min portion of the procedure. Airway examination revealed right vocal cord paralysis, right arytenoid edema, and extrinsic compression of the right lateral tracheal wall. The diagnostic bronchoscope was then withdrawn to insert the endobronchial ultrasound scope. Because the patient was awake at this time, he received an additional 1 mg of midazolam and 100 µg of IV fentanyl. Two minutes after medication administration, the patient was noted to have clenched hands and jaw. His chest wall became rigid, and chest wall movement ceased. The patient developed hypertension to 208/134 mm Hg and oxygen desaturation decrease, with the lowest observed oxygen saturation being 81%. The patient lost consciousness and bag-valve-mask ventilation was initiated to assist patient breaths. Naloxone, 0.2-mg IV, was administered with rapid resolution of rigid-

Fentanyl Chest Wall Rigidity Syndrome—A Case Report

Robert L. Vaughn D.D.S.*
C. Richard Bennett D.D.S. Ph.D.**

INTRODUCTION

Fentanyl (Sublimaze), a phenylpiperidine derivative, is a potent narcotic with rapid onset and short duration. An analgesic agent with these features is suitable for the production of conscious-sedation, neuroleptanalgesia, neuroleptanesthesia, and anesthesia. A side effect, namely rigidity of the chest, jaw, and abdominal muscles, has been noted with fentanyl and fentanyl-containing compounds. This was first described by Hamilton and Cullen.[5]

Chest wall rigidity is a serious complication that must be diagnosed and treated immediately. Respiratory and cardiovascular complications may develop from hypoxemia and hypercarbia with resulting morbidity and mortality. Anyone administering fentanyl must be thoroughly trained in airway management and the use of narcotic antagonists as well as neuromuscular blocking agents.

Case Report

At approximately 2:25 PM it was decided to administer one more aloquot of 0.025 mg. of fentanyl. Blood pressure was 118/80 and pulse was 96, respirations were 16. At 2:26 PM (one minute later) the patient suddenly:

1) became rigid (tonic spasm)
2) ceased breathing
3) the jaw muscles were clenched and the chest wall became very rigid and noncompliant to external pressure.

The patient's eyes remained open and were apparently focusing and following movements. Attempts at artificial ventilation with a manual ventilator (Ambu bag) were unsuccessful. Thirty seconds after the onset of the rigid chest wall syndrome 0.4 mg. of Narcan were administered via the patent intravenous infusion. At approximately 2:27 PM spontaneous respirations resumed and the spasm of the jaw muscles ceased. A few seconds later the patient

Anesth Prog 37:46–48 1990

Ineffective Ventilation During Conscious Sedation Due to Chest Wall Rigidity After Intravenous Midazolam and Fentanyl

William E. Ackerman, MD, James C. Phero, DMD, and Gregg T. Theodore, MD
Department of Anesthesia, University of Cincinnati College of Medicine, Cincinnati, Ohio

Chest wall rigidity has been reported after the administration of high-dose intravenous fentanyl. This case report supports the observation that low-dose intravenous fentanyl may also cause

CASE REPORT

Emergency anesthesia consultation was requested after an obstetrician administered 1 mg (1 ml) of midazolam

CLINICAL INVESTIGATIONS

Anesthesiology
78:629–634, 1993

J. B. Lippincott Company, Philadelphia

Fentanyl-induced Rigidity and Unconsciousness in Human Volunteers

Incidence, Duration, and Plasma Concentrations

James B. Streisand, M.D., Peter L. Bailey, M.D.,* Leon LeMaire, M.D.,† Michael A. Ashburn, M.D.,‡ Stephen D. Tarver, M.D.,§ John Varvel, M.D.,¶ Theodore H. Stanley, M.D.***

Background: **Muscle rigidity frequently accompanies induction of anesthesia with opioids. The authors sought to determine whether unconsciousness and amnesia occur when humans develop rigidity and apnea after intravenous fentanyl (without other concomitant anesthetics).**

Methods: **The incidence and duration of rigidity and level of consciousness were evaluated and associated plasma concentrations of fentanyl were measured in 12 healthy adult**

and adequate oxygenation (Sp_{O_2} > 90%). Wh curred, it started 3 ± 0.9 (range 1–4) min after t fentanyl concentration and lasted for 11.5 ± 5. min. Rigidity started at a plasma fentanyl co 21.5 ± 4.4 (range 16–28) ng/ml and ended at 6 5.2–8.7) ng/ml. Baseline HR was less in the sub sequently developed rigidity (56.7 ± 7.8 *vs.*

Opioid Antagonisti

Naloxone

- Saf μ opioid resp. Antagonisti
- Morfin, Meperidine, Fentanil
- Başlangıç ½-1 amp (0.2-0.4 mg IV)
- 5 ampul'e kadar (2 mg) çıkılabilir
- 15-30 dk etki başlar
- Diagnostik amaçlı

Fentanil

Obez hastalarda girişimsel sedasyon için Fentanil kullanıldığında dikkatli olunmalıdır

Lipofilik olduğu için, obez hastalardaki **yarı ömrü uzundur**

İlk doz verilirken total vücut ağırlığı üzerinden hesaplamak gerekir ama ek doz verilecekse ideal vücut ağırlığına göre hesaplanmalıdır

Benzodiazepin komplikasyonları

Diazepam

Doz: **Erişkin: 2-10 mg**,

Çocuk: 0,05-0,2 mg/kg

Etki başlangıcı hızlıdır ve 2-4 saat devam eder.

Karaciğerde metabolize olur; ***kc yetm ve sirozlu*** hastalarda kullanılmamalıdır

Yarı ömürü uzundur (36-90 saat)

Solunum depresyonu

Hipotansiyon

Opioidlerle kullanıldıklarında doz azaltılmalıdır.

Benzodiazepin komplikasyonları

Midazolam

5 mg/5ml, 15 mg/3 mlamp ,

Sedasyon, Amnezi, Anksiyoliz, İskelet kası gevşemesi

Dozu: ***0.02-0.04 mg/kg iv***

Yetişkinlerde 1-3 mg' lık dozlar

Benzodiazepin komplikasyonları

Midozalam:

Yüksek lipid çözünürlüğü
Hızlı etki başlangıcı 2 dk
Kısa etki süresi 60-90 dk

Kısa süreli sedasyonlarda tercih

Yan etkiler: **Solunum depresyonu, apne, hipotansiyon, karaciğer yetmezliğinde dikkat…**

Benzodiazepin Antagonisti

Flumazenil

Klinik dozlarda saf kompetetif benzodiazepine antagonistidir

Reversibl bağlanır

IV uygulamayı takiben 5-8 dk'da beyinde max konsantrasyona ulaşır

Etki 1-2 dakikada başlar

Yarılanma ömrü 1 saatten azdır

Benzodiazepin Antagonisti

I.V. yetişkinde 0,2 mg 30 saniyede verilir

IV olarak 0.2 mg bolus dozu takiben hasta cevabı izlenir

Gerekli olgularda 0,3-0,5 mg tekrarlanan dozlar titre edilerek 1 dk aralıklarla verilir

Toplam 3 mg üstüne çıkılmaması önerilir

Etomidate

Preperat adı: Hypnomidate amp 10 ml, 2 mg/ml, 0.1-0.3 mg/kg iv

Sedatif hipnotik

Etkinin başlangıcı 1 dk, etki süresi 5 -10 dk

Yan etkiler

- Geçici solunum depresyonu
- Miyoklonik kasılmalar
- Bulantı, kusma

Etomidate Yan Etki

Enjeksiyon yerinde ağrı;

- İntravenöz yol alanındaki ağrı için geniş damar yolu açılması
- Salin İnfüzyonu
- Lokal analjezik kullanılabilir

Myoklonus; Opioid analjezik ve benzodiazepinlerin ön ilaç olarak kullanılması bu yan etkiyi azaltabiliyor

Ancak ortaya çıktığında nöbet aktivitesi ile ayırt edilemediği durumlarda gereksiz tetkiklere ve hastanın uzun süre acil serviste zaman geçirmesine neden olabilir

Etomidate

Adrenokortikal hormon süpresyonu; 11-B-Hidroksilaz enzim inhibisyonu sonucu ortaya çıkıyor. Açık bir doz bağımlı adrenokortikoid hormon sentezi süpresyonu yapıyor

Tek doz 5 saat süreyle inhibisyona neden oluyor

Kullanım sonrası 12 saatte bile süpresyon etkisinin gösterildiği çalışmalar var

Etomidate

Karaciğer tarafından hızla hidroliz ediliyor ve inaktif metaboliti oluşuyor

Bu nedenle hepatik kan akımı etki süresini belirler

Etkisiz metabolit idrar ile atılır

Analjezik özelliği yoktur

EEG aktivitesini ve serebral kan akımını deprese eder (barbütüratlara benzer)

Ortalama arteryel basınca etki etmediğinden serebral perfüzyon basıncını düşürmeden intrakraniyal basıncı azaltır

Etomidate

Etomidate histamin salınımını etkilemeyen intravenöz anestezik olduğundan reaktif hava yolu olan hastalarda güvenlidir

Hepatik ve renal yetmezlikte doz ayarlaması önerilir

Kalp hızı, kan basıncı ve kardiyak output korunur

Respiratuar depresan etkileri tiyopental ve propofolden daha azdır

Pulmoner fonksiyonları azalmış hastalarda babütiratlara güvenli bir alternatifdir

Etomidat

Ancak septik hastaları da içeren bu hasta grubunda adrenal yetmezliğe neden olmasının mortalite ile birliktelik göstermesi kaygısı her zaman olmuştur

Etomidat ister tek doz ister infüzyon sonrası kesinlikle kortizol düzeyini düşürdüğü ve adrenal sistemi baskılıladığı bilinmektedir

Crit Care Med. 2012 Nov;40(11):2945-53. doi: 10.1097/CCM.0b013e31825fec26.

Etomidate is associated with mortality and adrenal insufficiency in sepsis: a meta-analysis*.

Chan CM[1], Mitchell AL, Shorr AF.

⊕ Author information

Abstract

OBJECTIVE: To evaluate the effects of single-dose etomidate on the adrenal axis and mortality in patients with severe sepsis and septic shock.

DESIGN: A systematic review of randomized controlled trials and observational studies with meta-analysis.

SETTING: Literature search of EMBASE, Medline, Cochrane Database, and Evidence-Based Medical Reviews.

SUBJECTS: Sepsis patients who received etomidate for rapid sequence intubation.

INTERVENTIONS: None.

MEASUREMENTS AND MAIN RESULTS: We conducted a systematic review of randomized controlled trials and observational studies with meta-analysis assessing the effects of etomidate on adrenal insufficiency and all-cause mortality published between January 1950 and February 2012. We only examined studies including septic patients. All-cause mortality served as our primary end point, whereas the prevalence of adrenal insufficiency was our secondary end point. Adrenal insufficiency was determined using a cosyntropin stimulation test in all studies. We used a random effects model for analysis; heterogeneity was assessed with the I statistic. Publication bias was evaluated with Begg's test. Five studies were identified that assessed mortality in those who received etomidate. A total of 865 subjects were included. Subjects who received etomidate were more likely to die (pooled relative risk 1.20; 95% confidence interval 1.02-1.42; Q statistic, 4.20; I2 statistic, 4.9%). Seven studies addressed the development of adrenal suppression associated with the administration of etomidate; 1,303 subjects were included. Etomidate administration increased the likelihood of developing adrenal insufficiency (pooled relative risk 1.33; 95% confidence interval 1.22-1.46; Q statistic, 10.7; I2 statistic, 43.9%).

CONCLUSIONS: Administration of etomidate for rapid sequence intubation is associated with higher rates of adrenal insufficiency and mortality in patients with sepsis.

Systematic Review Snapshot

TAKE-HOME MESSAGE

Currently, single-dose etomidate has not been shown to cause increased mortality in septic patients requiring intubation; however, sufficiently powered randomized trials are required before definitive conclusions can be drawn.

METHODS

DATA SOURCES

EMBASE, MEDLINE, Cochrane Database, and Evidence-Based Medical Reviews were searched from 1950 to February 2012 without language restrictions. Bibliographies of included articles were hand searched and unpublished data were sought through databases such as ClinicalTrials.gov.

STUDY SELECTION

Prospective comparative studies, either randomized or observational, evaluating etomidate in septic patients were included for the primary outcome of mortality. For the secondary outcome of adrenal insufficiency, retrospective comparative studies were also included. Studies in abstract form, pediatric studies, and studies lacking a control group were excluded. Disagreements were resolved by discussion.

In Patients With Severe Sepsis, Does a Single Dose of Etomidate to Facilitate Intubation Increase Mortality?

EBEM Commentators
Benton R. Hunter, MD
Jonathan Kirschner, MD
Department of Emergency Medicine
Indiana University School of Medicine
Indianapolis, IN

Results

Outcome	RR (95% CI)	No. of Studies	No. of Patients	Heterogeneity, I^2, %
Mortality*	1.20 (1.02–1.42)	5	865	4.9
28-day mortality	1.28 (1.06–1.54)	3	637	46
Adrenal insufficiency	1.33 (1.22–1.46)	7	1,303	43.9

RR, Pooled relative risk; *CI*, confidence interval.
*Either inhospital or at 28 days.

One hundred six articles were identified in the search; 58 were reviewed in full text. Ten total studies were included, 5 of which met criteria for the mortality endpoint and 7 for the endpoint of adrenal insufficiency

systematic review supports the notion that even a single dose of etomidate for intubation causes adrenal suppression.[3-5] The significance of this suppression is unknown. The more important question is whether a

Etomidat'ın entübasyon gerektiren sepsis hastalarında mortaliteyi arttırdığı gösterilememiştir. Buna karşın kesin bir yoruma varmak için daha güçlü randomize çalışmalara ihtiyaç vardır

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Relative adrenal insufficiency in critically ill patient after rapid sequence intubation: KETASED ancillary study☆

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ARTICLE INFO

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ABSTRACT

Purpose: Relative adrenal insufficiency (RAI) has been reported as a predictor of mortality in septic patient; however, its effects on mortality and outcomes for critically ill patients remain debatable. The objective of this study was to assess the effect of RAI on prognostic outcomes in patients after out-of-hospital rapid sequence intubation (RSI) and factors associated with the onset of RAI.

Patients and methods: A prespecified ancillary study of KETASED, a randomized prospective multicenter trial, was conducted. Three hundred ten patients who underwent RSI in an out-of-hospital setting had baseline cortisol and adrenocorticotropic hormone response test measurements within 24 hours of intensive care unit admission and were included.

Results: The mean (SD) age was 55 (19) years, with a mean (SD) Sequential Organ Failure Assessment score of 9 (4). Two hundred forty-seven (69%) patients presented with RAI. Baseline characteristics were similar between patients with and without RAI, except for the use of etomidate as a sedative agent (63% of patients with RAI vs 21%, $P < .001$), and history of chronic kidney disease. There was no difference in terms of 28-day mortality between the 2 groups (21% vs 19%, $P = .65$) and in terms of other 28-day prognosis end points.

Conclusion: In critically ill patients who require RSI, RAI is common and is not associated with worsened outcomes in our cohort.

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Chest. 2015 Feb;147(2):335-46. doi: 10.1378/chest.14-1012.

Single-dose etomidate does not increase mortality in patients with sepsis: a systematic review and meta-analysis of randomized controlled trials and observational studies.

Gu WJ, Wang F, Tang L, Liu JC.

Abstract

BACKGROUND: The effect of single-dose etomidate on mortality in patients with sepsis remains controversial. We systematically reviewed the literature to investigate whether a single dose of etomidate for rapid sequence intubation increased mortality in patients with sepsis.

METHODS: PubMed, Embase, and CENTRAL (Cochrane Central Register of Controlled Trials) were searched for randomized controlled trials (RCTs) and observational studies regarding the effect of single-dose etomidate on mortality in adults with sepsis. The primary outcome was all-cause mortality. The Mantel-Haenszel method with random-effects modeling was used to calculate pooled relative risks (RRs) and 95% CIs.

RESULTS: Eighteen studies (two RCTs and 16 observational studies) in 5,552 patients were included. Pooled analysis suggested that single-dose etomidate was not associated with increased mortality in patients with sepsis in both the RCTs (RR, 1.20; 95% CI, 0.84-1.72; P = .31; I(2) = 0%) and the observational studies (RR, 1.05; 95% CI, 0.97-1.13; P = .23; I(2) = 25%). When only adjusted RRs were pooled in five observational studies, RR for mortality was 1.05 (95% CI, 0.79-1.39; P = .748; I(2) = 71.3%). These findings also were consistent across all subgroup analyses for observational studies. Single-dose etomidate increased the risk of adrenal insufficiency in patients with sepsis (eight studies; RR, 1.42; 95% CI, 1.22-1.64; P < .00001).

CONCLUSIONS: Current evidence indicates that single-dose etomidate does not increase mortality in patients with sepsis. However, this finding largely relies on data from observational studies and is potentially subject to selection bias; hence, high-quality and adequately powered RCTs are warranted.

PMID: 25255427 [PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms, Substances

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Crit Care Med. 2012 Jan;40(1):29-35. doi: 10.1097/CCM.0b013e31822d7938.

Corticosteroid after etomidate in critically ill patients: a randomized controlled trial.

Payen JF[1], Dupuis C, Trouve-Buisson T, Vinclair M, Broux C, Bouzat P, Genty C, Monneret D, Faure P, Chabre O, Bosson JL.

Author information

Abstract

OBJECTIVE: To investigate the effects of moderate-dose hydrocortisone on hemodynamic status in critically ill patients throughout the period of etomidate-related adrenal insufficiency.

DESIGN: : Randomized, controlled, double-blind trial (NCT00862381).

SETTING: University hospital emergency department and three intensive care units.

INTERVENTIONS: After single-dose etomidate (H0) for facilitating endotracheal intubation, patients without septic shock were randomly allocated at H6 to receive a 42-hr continuous infusion of either hydrocortisone at 200 mg/day (HC group; n = 49) or saline serum (control group; n = 50).

MEASUREMENTS AND MAIN RESULTS: After completion of a corticotrophin stimulation test, serum cortisol and 11β-deoxycortisol concentrations were subsequently assayed at H6, H12, H24, and H48. Forty-eight patients were analyzed in the HC group and 49 patients in the control group. Before treatment, the diagnostic criteria for etomidate-related adrenal insufficiency were fulfilled in 41 of 45 (91%) and 38 of 45 (84%) patients in the HC and control groups, respectively. The proportion of patients with a cardiovascular Sequential Organ Failure Assessment score of 3 or 4 declined comparably over time in both HC and control groups: 65% vs. 67% at H6, 65% vs. 69% at H12, 44% vs. 54% at H24, and 34% vs. 45% at H48, respectively. Required doses of norepinephrine decreased at a significantly higher rate in the HC group compared with the control group in patients treated with norepinephrine at H6. No intergroup differences were found regarding the duration of mechanical ventilation, intensive care unit length of stay, or 28-day mortality.

CONCLUSION: These findings suggest that critically ill patients without septic shock do not benefit from moderate-dose hydrocortisone administered to overcome etomidate-related adrenal insufficiency.

Çalışmada etomidat kullanılan hastalar iki gruba randomize edilmiş ve bir gruba entübasyon sonrası altıncı saatte 200mg/gün olacak şekilde Hidrokortizon 42 saat süreyle infüzyon halinde verilmiştir

Replasman yapılan grupla yapılmayan grup arasında adrenal yetmezlik gelişmesi, SOFA skorları, hastane kalış süresi, mekanik ventilasyon süresi, yoğun bakım kalış süresi, vazopresör gereksinimleri karılaştırılmıştır tüm gruplar arasında bir fark bulunamamıştır

Bu nedenle bugün etomidat kullanımı durumunda steroid replasmanı önerilmemektedir.

Etomidate

*Hemodinamik olarak **stabil olmayan** septik ya da kritik hastalarda eğer ketamin için bir kontrendikasyon ya da şüphe varsa, Etomidate kullanılabilir*

Mortaliteyi arttırdığı açıkça gösterilememiştir

*ANCAK Sepsis hastaları ve kritik hastalar **hemodinamik olarak stabil iseler***
Etomidate tercih etmek için bir sebep görünmemektedir

Etomidate *adrenal yetmezlik yaptığı bilinmektedir*

Analjezi ve sedasyon komplikasyonları; ne yapalım?

Komplikasyonlara hazırlıklı bulunalım

En iyi bildiğiniz ajanı kullanmalı

Taburculuk kriterleri dikkat edelim

Sabrınız için teşekkürler