Toksikolojide tanı yöntemleri ve yeni antidotlar

Doç. Dr. İlhan KORKMAZ

Sivas Cumhuriyet Üniversitesi

Tanı yöntemleri

- Hikaye
- Fizik muayene bulguları
- Labaratuar değerlendirmesi
    - Plazma
    - İdrar
- Radyolojik incelemeler

TOXİDROM	VİTAL BULGULAR	BİLİNÇ	PUPİL ÇAPI	CİLT	BAĞIRSAK SESLERİ	DİĞER	MUHTEMEL TOKSİNLER
Sempato-mimetik	Ht Taşikardi Hipertermi	Ajitasyon Deliryum	Dilate	Terli	Normal/ Artmış		Amfetamin Kokain
Kolinerjik	Bradikardi KB genelde normal sınırda	Konfüzyon-koma Fasikülasyonlar	Küçülmüş	Terli	Hiperaktif	DUMBELS;	
Antikolinerjik	Ht Taşikardi Hipertermi	Ajitasyon Deliryum Koma Konvülziyon	Dilate	Kuru Sıcak	Değişken	İleus Üriner retansiyon	

TOXİDROM	VİTAL BULGULAR	BİLİNÇ	PUPİL ÇAPI	CİLT	BAĞIRSAK SESLERİ	DİĞER	MUHTEMEL TOKSİNLER
Sedatif hipnotikler	KB N KH N VI N Solunum depresyonu	Somnolans Koma	Küçülmüş veya N	N	N		Barbitürat BZD Etanol
Seratonin sendromu (NMS)	Hipertermi HT Taşikardi	Ajitasyon Konfüzyon Koma	Dilate	Terli	Artmış	Nöromuskuler hipereksitablite	SSRI Lityum MAO inh. Meperidin Dextrometorfan
Opioidler	**Solunum depresyonu Bradikardi Hipotansiyon Hipotermi**	**Depresyon Koma Öfori**	**Pinpoint**	**Normal**	**Normal/azalmış**		

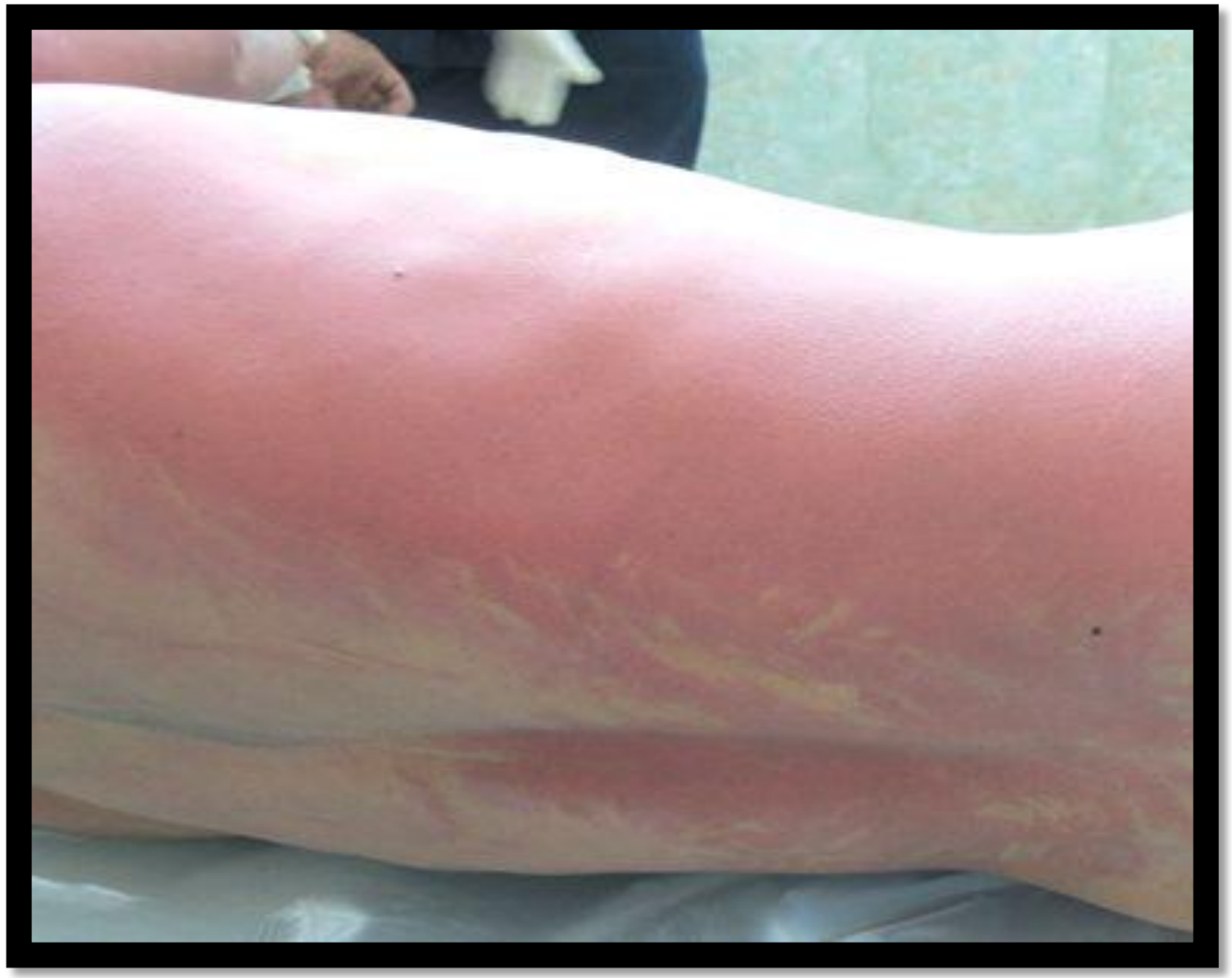
Minör toksidromlar	Semptom ve bulgular	Etken madde
α-1 antagonisti:	SSS depresyonu, taşikardi, miosis	Klorpromazin Ketiapin Klozapin Risperidon
α-2 agonist:	SSS depresyonu, bradikardi, miosis	Klonidin Tetrahydrozolin Tizanidin
Na kanal blokörü:	SSS toksisitesi, QRS genişlemesi	TCA
K kanal blokörü	SSS toksisitesi, uzamış QT	Fenotiazinler TCA Metadon Butirofenon
Klonus/myoklonus	SSS depresyonu, myoklonik kasılmalar	Lityum SSRI

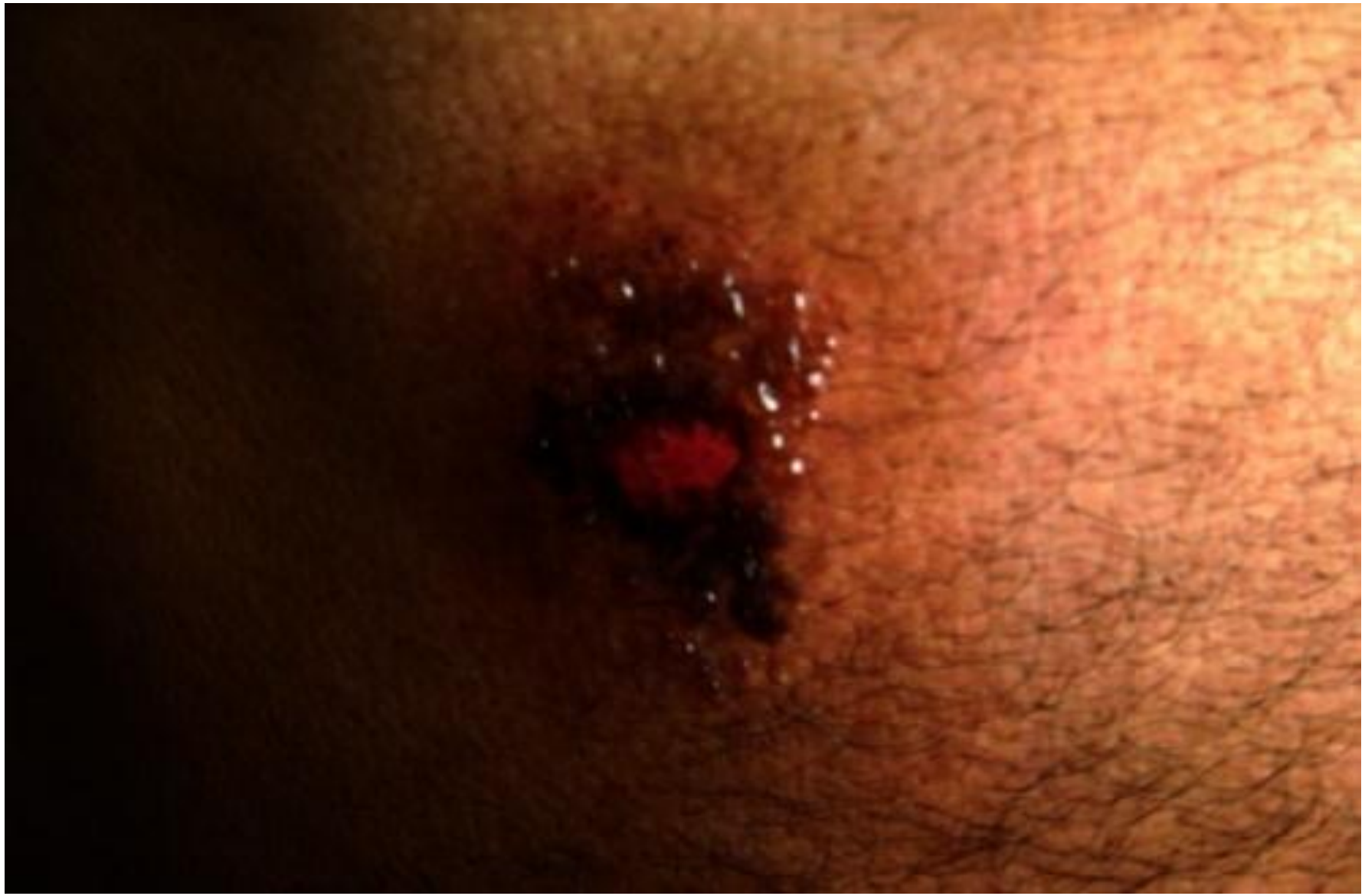
Hikaye

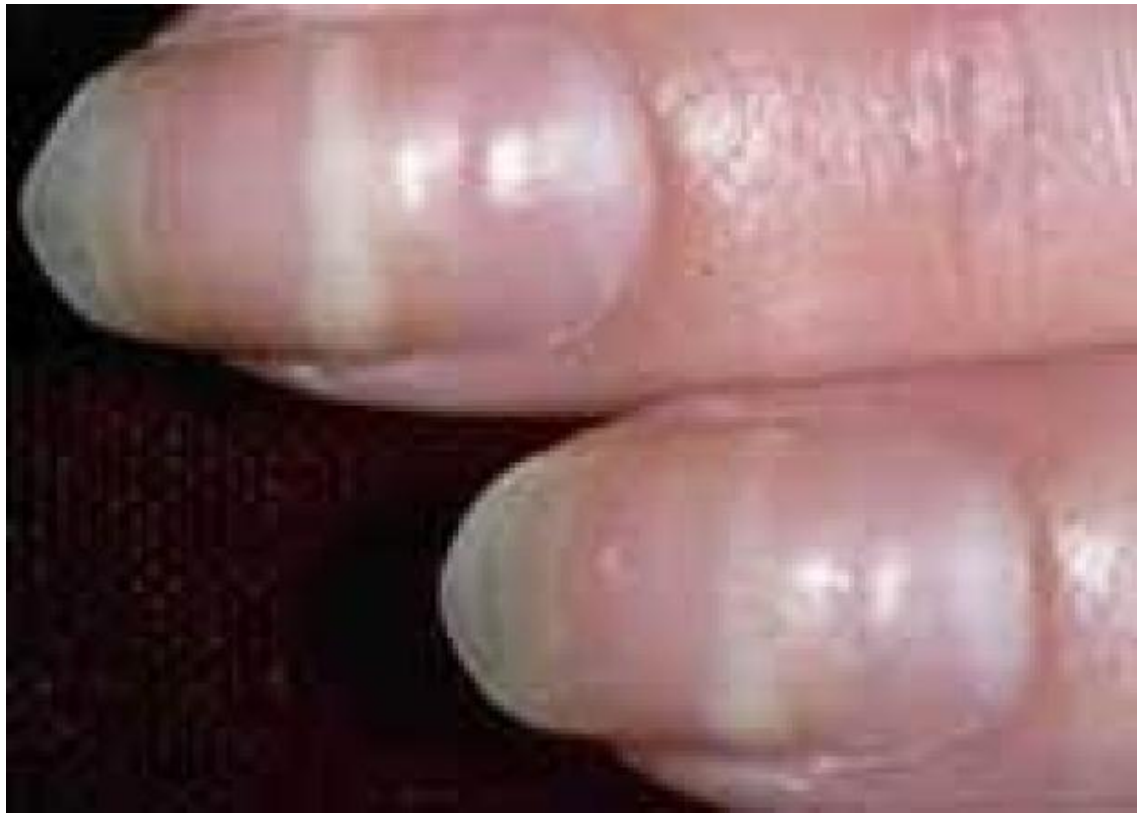
- Rutin kullandığı ilaçlar
- Maruziyet şekli
- Ek hastalıkları
- Mesleği

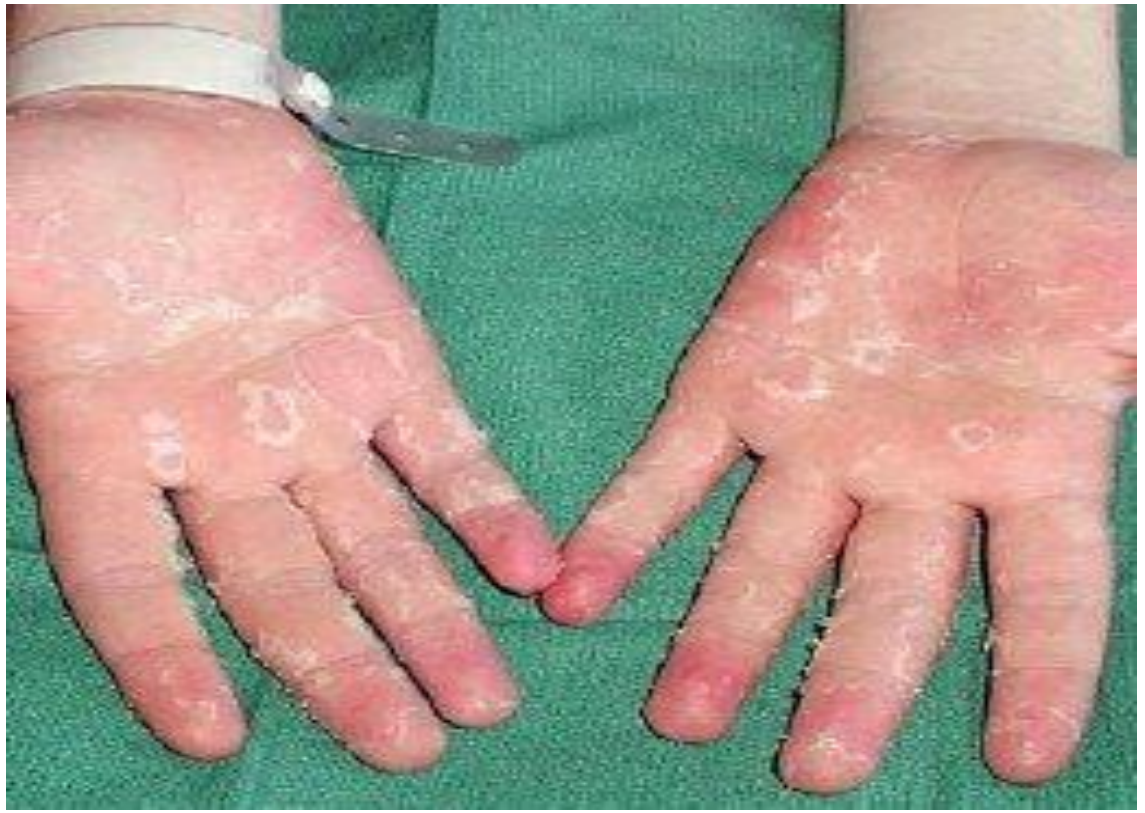
Fizik muayene

Cilt bulguları



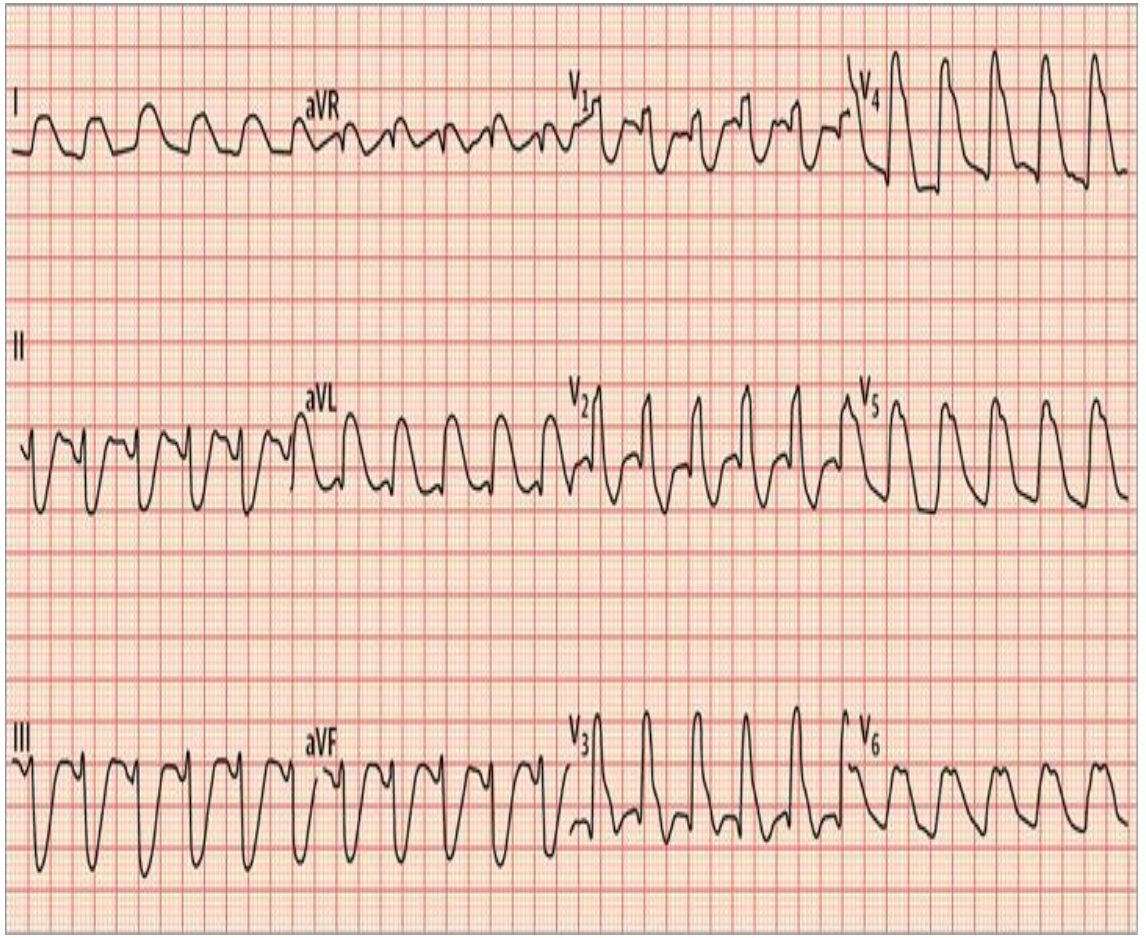




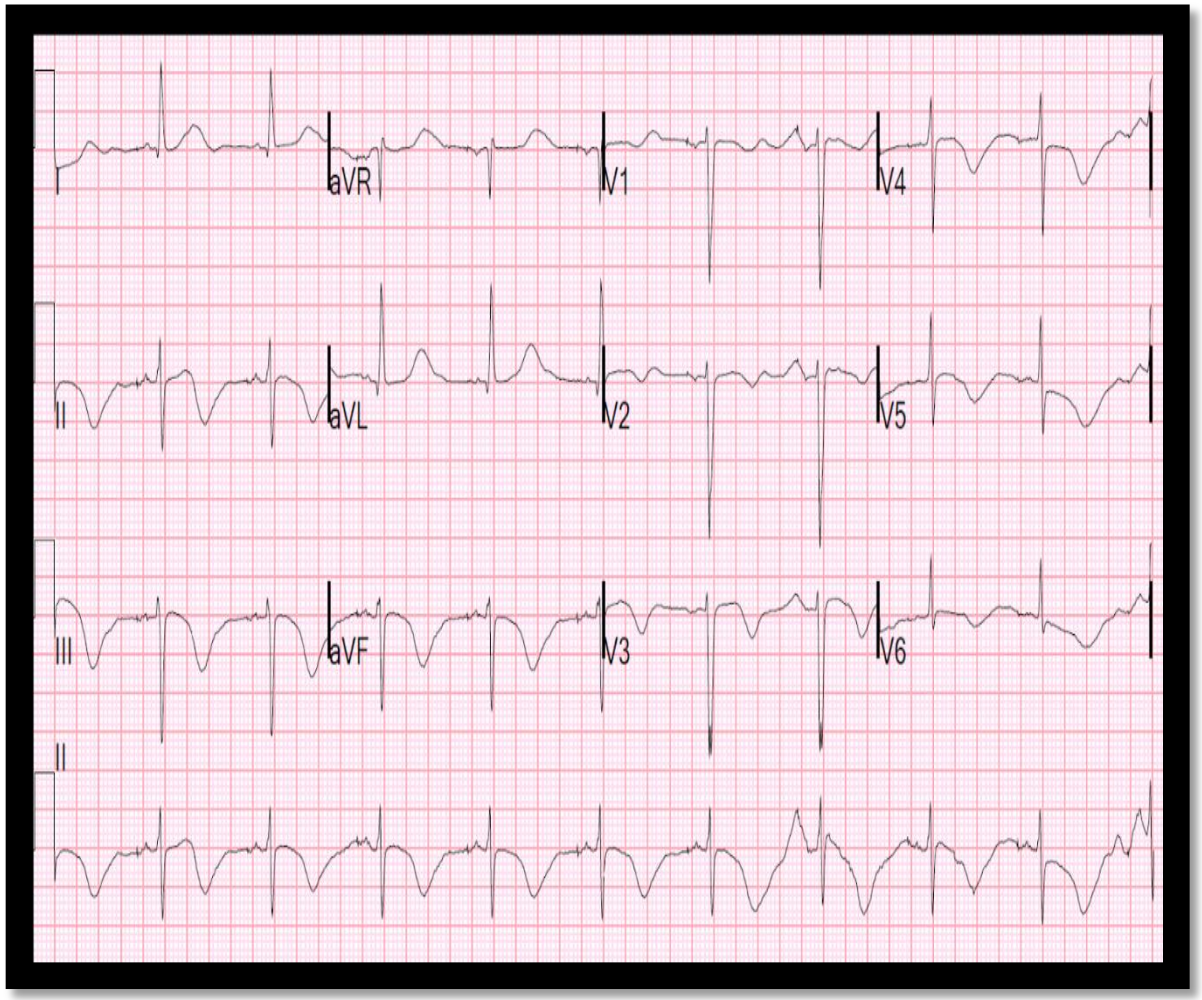


KVS

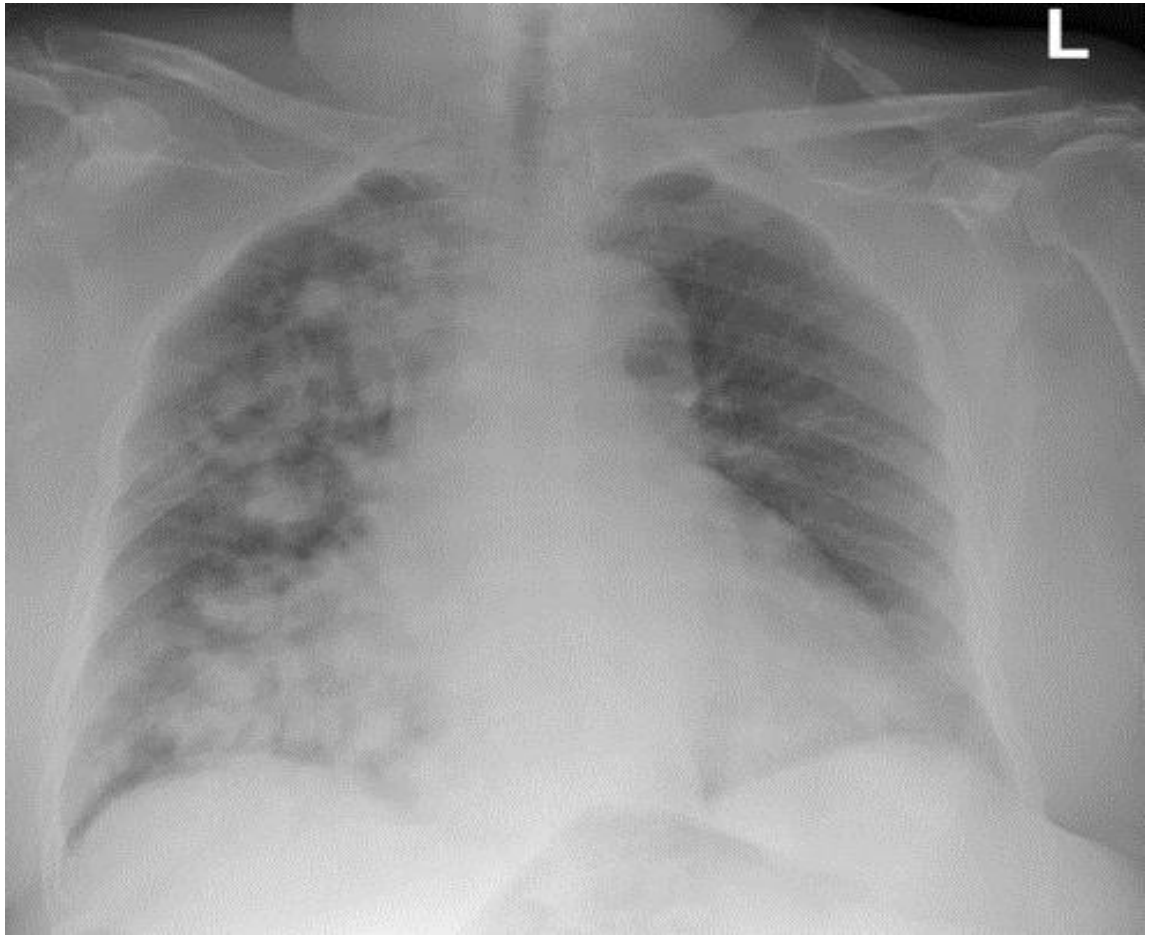
- Amantadin
- β bloker
- Prokainamide
- Klorokin
- Trisiklik antidepresanlar,
- Difenhidramin
- Dizopiramid
- Flekainid
- Maprotilin
- Propoksifen
- Kinidin



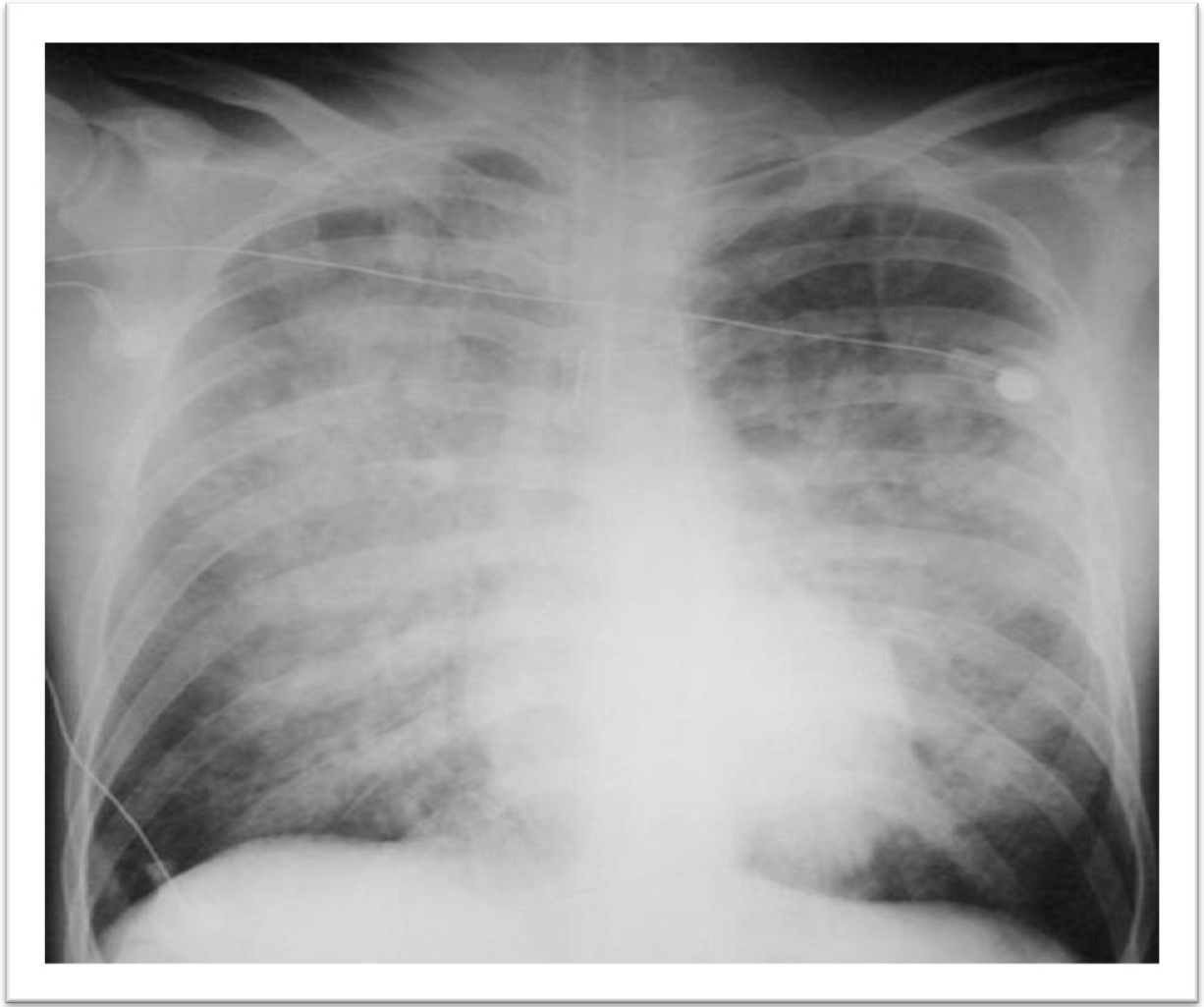
- Sitalopram
- Makrolidler
- Difenhidramin
- Haloperidol
- Levofloksasin
- Fenotiazinler
- TCA
- Metadon
- Butirofenon



- Gaz ve madeni yağlar
- Hidrokarbonlar



- Metal dumanları
- Hidrojen sülfür
- İritan gazlar
- Civa buharı
- Metil bromür ve klorür
- Beta-blokerler
- Fosgen
- Parakuat
- Etilen glikol



Laboratuvar

- Karboksihemoglobin
- Digoksin
- Etanol
- Demir
- Lityum
- Asetaminofen
- Salisilat
- Teofilin
- Arsenik
- Civa
- Kurşun
- Metanol
- Etilen glikol
- Fenitoin
- Karbamazepin
- Paraquat

Labaratuarda ipuçları

Anyon gaplı metabolik asidoz

MUDPILES

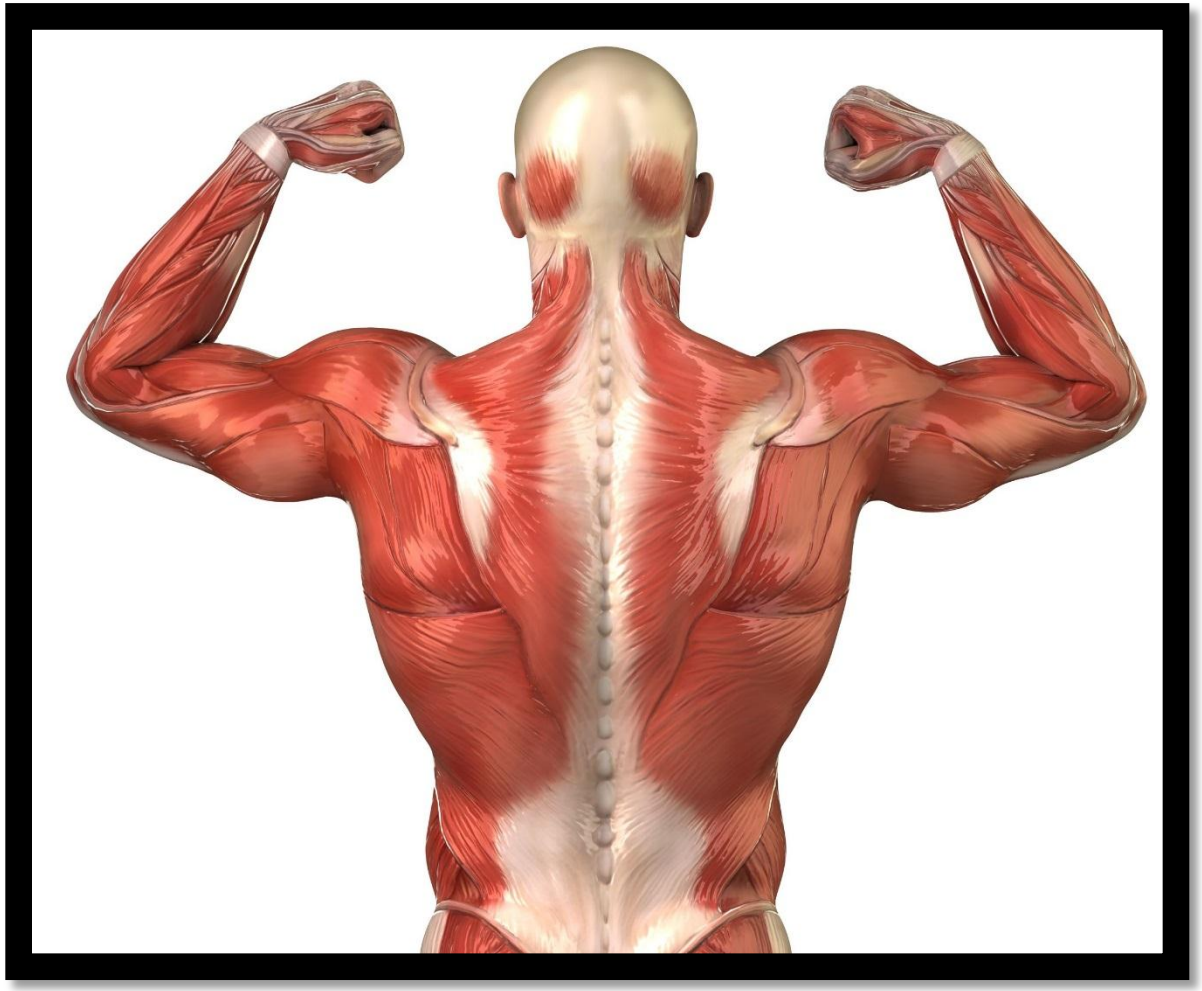
- **M**ethanol, metformin
- **U**remi
- **D**iyabetik ketoasidoz
- **P**ropylene glycol
- **I**soniazid, iron, masif ibuprofen
- **L**aktic asidoz
- **E**thylene glycol
- **S**alisilat

CAT

- **C**ellular asphyxiants (cyanide, carbon monoxide, hydrogen sulfide)
- **A**lkolik ketoasidoz
- **T**ylenol

RABDOMYOLİZ

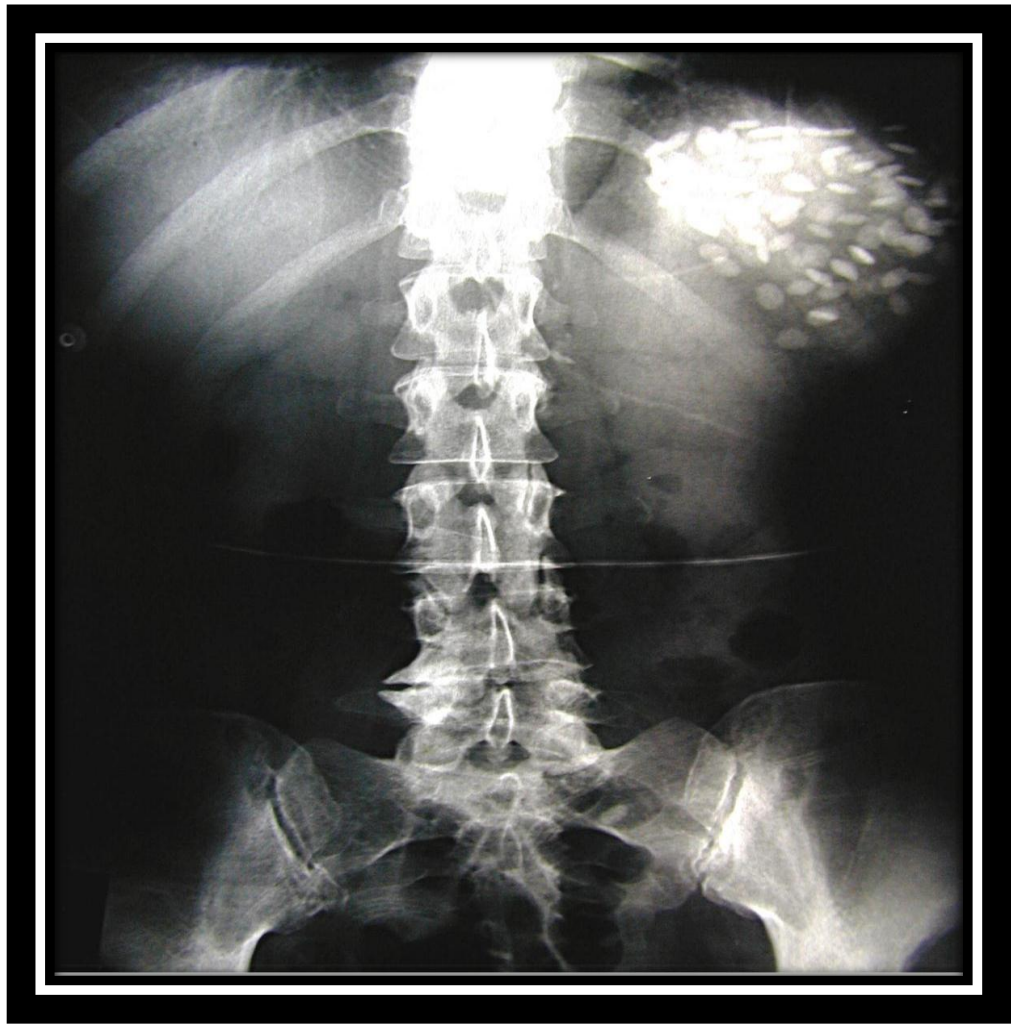
- Nöroleptik malign sendrom
- Serotonin sendrom
- Statinler
- Uzamış immobilizasyona neden olan opioid veya antipsikotik kullanımı
- Konvülziyona neden olan ilaçlar



RADIOOPAK MADDELER

CHIPPED

- **C**hloral hydrate, **c**alcium carbonate
- **H**eavy metals
- **I**ron
- **P**henothiazines
- **P**otassium chloride
- **E**nteric-coated pills
- **D**ental amalgam, drug packets



Yeni antidotlar??

Ann Emerg Med. 2018 Mar;71(3):314-325.e1. doi: 10.1016/j.annemergmed.2017.05.021. Epub 2017 Jun 29.

Expert Consensus Guidelines for Stocking of Antidotes in Hospitals That Provide Emergency Care.

Dart RC[1], Goldfrank LR[2], Erstad BL[3], Huang DT[4], Todd KH[5], Weitz J[6], Bebarta VS[7], Caravati EM[8], Henretig FM[9], Delbridge TR[10], Banner W[11], Schneider SM[12], Anderson VE[13].

Author information

Abstract

We provide recommendations for stocking of antidotes used in emergency departments (EDs). An expert panel representing diverse perspectives (clinical pharmacology, medical toxicology, critical care medicine, hematology/oncology, hospital pharmacy, emergency medicine, emergency medical services, pediatric emergency medicine, pediatric critical care medicine, poison centers, hospital administration, and public health) was formed to create recommendations for antidote stocking. Using a standardized summary of the medical literature, the primary reviewer for each antidote proposed guidelines for antidote stocking to the full panel. The panel used a formal iterative process to reach their recommendation for both the quantity of antidote that should be stocked and the acceptable timeframe for its delivery. The panel recommended consideration of 45 antidotes; 44 were recommended for stocking, of which 23 should be immediately available. In most hospitals, this timeframe requires that the antidote be stocked in a location that allows immediate availability. Another 14 antidotes were recommended for availability within 1 hour of the decision to administer, allowing the antidote to be stocked in the hospital pharmacy if the hospital has a mechanism for prompt delivery of antidotes. The panel recommended that each hospital perform a formal antidote hazard vulnerability assessment to determine its specific need for antidote stocking. Antidote administration is an important part of emergency care. These expert recommendations provide a tool for hospitals that offer emergency care to provide appropriate care of poisoned

Expert Consensus Guidelines for Stocking of Antidotes in Hospitals That Provide Emergency Care 2018- 2009 Farklılıklar

- Glucarpidase
- İdarucizumab
- Levocarnitine
- Lipid emülsiyonu
- Uridine triacetat

Oncologist. 2018 Jan;23(1):52-61. doi: 10.1634/theoncologist.2017-0243. Epub 2017 Oct 27.

Consensus Guideline for Use of Glucarpidase in Patients with High-Dose Methotrexate Induced Acute Kidney Injury and Delayed Methotrexate Clearance.

Ramsey LB[1,2], Balis FM[3], O'Brien MM[4], Schmiegelow K[5], Pauley JL[6], Bleyer A[7], Widemann BC[8], Askenazi D[9], Bergeron S[10], Shirali A[11], Schwartz S[12], Vinks AA[13,2], Heldrup J[14].

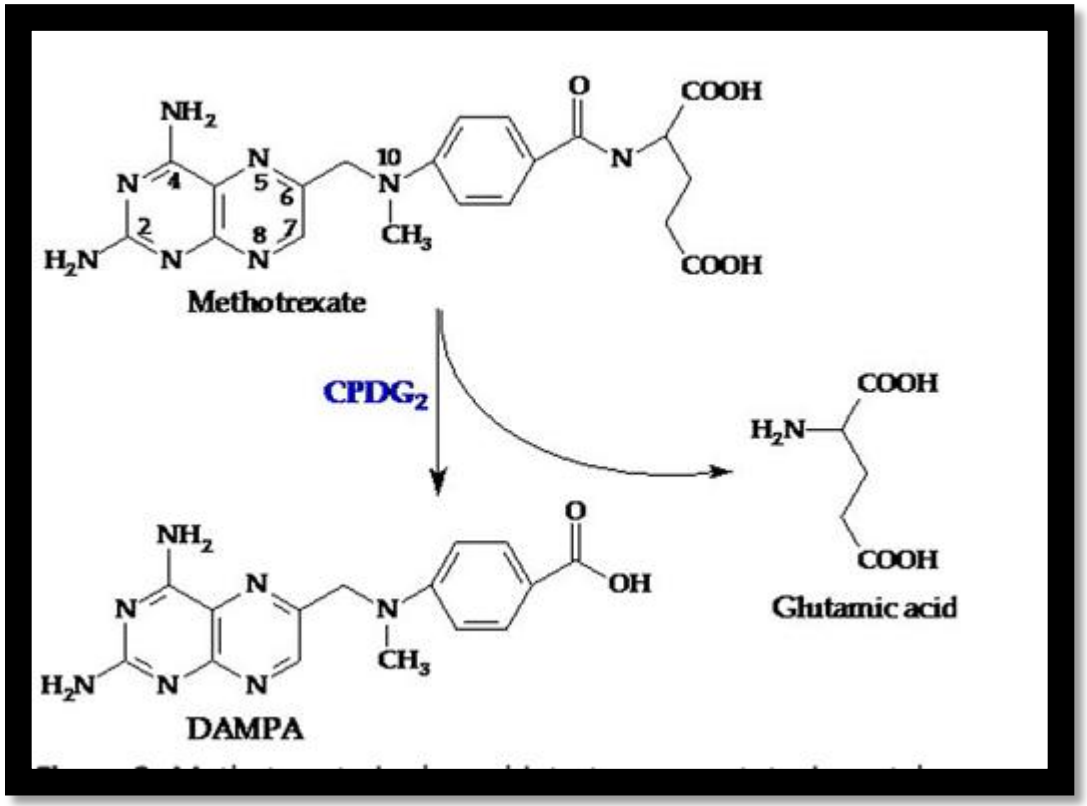
Author information

Abstract

Acute kidney injury due to high-dose methotrexate (HDMTX) is a serious, life-threatening toxicity that can occur in pediatric and adult patients. Glucarpidase is a treatment approved by the Food and Drug Administration for high methotrexate concentrations in the context of kidney dysfunction, but the guidelines for when to use it are unclear. An expert panel was convened to provide specific, expert consensus guidelines for the use of glucarpidase in patients who develop HDMTX-induced nephrotoxicity and delayed methotrexate excretion. The guideline provides recommendations to identify the population of patients who would benefit from glucarpidase rescue by more precisely defining the absolute methotrexate concentrations associated with risk for severe or life-threatening toxicity at several time points after the start of an HDMTX infusion. For an HDMTX infusion ≤24 hours, if the 36-hour concentration is above 30 μM, 42-hour concentration is above 10 μM, or 48-hour concentration is above 5 μM and the serum creatinine is significantly elevated relative to the baseline measurement (indicative of HDMTX-induced acute kidney injury), glucarpidase may be indicated. After a 36- to 42-hour HDMTX infusion, glucarpidase may be indicated when the 48-hour methotrexate concentration is above 5 μM. Administration of glucarpidase should optimally occur within 48-60 hours from the start of the HDMTX infusion, because life-threatening toxicities may not be preventable beyond this time point.

Glucarpidase

- 2012
- 48-60/saat
- 50 ünite/kg
- Bulantı/kusma
- Parestezi
- Flushing
- Başağrısı



See 1 citation found by title matching your search:

Neth J Med. 2018 Jan;76(1):36-39.

Glucarpidase treatment for methotrexate intoxication: a case report and review of the literature.

Boelens AD[1], Mathôt RAA, Vlaar APJ, Bouman CSC.

Author information

Open/close author information list

Abstract

High-dose methotrexate (MTX) induced acute kidney injury can lead to sustained high systemic MTX levels and severe toxicity. A 39-year-old man with lymphoblastic T-cell lymphoma was admitted to our intensive care unit with elevated serum creatinine and prolonged high serum MTX levels. Standard supportive care was complemented by the addition of a relatively novel agent, glucarpidase, which rapidly lowered the extracellular levels of MTX. Several case series support this effect of glucarpidase, but no randomised controlled trial has been performed to show this leads to better outcome. Furthermore, glucarpidase might negatively affect leucovorin rescue therapy. Lastly, glucarpidase carries a significant financial burden. Based on the current evidence we cannot recommend glucarpidase until further research elucidates its role in the treatment of MTX toxicity. There is no randomised clinical evidence to support its use in severe cases and theoretical evidence suggests that after prolonged exposure to high MTX levels glucarpidase administration is unable to reverse high intracellular MTX. We recommend that new randomised

https://www.ncbi.nlm.nih.gov/pubmed/27175894

Am J Ther. 2018 May/Jun;25(3):e333-e338. doi: 10.1097/MJT.0000000000000460.

Idarucizumab for Reversing Dabigatran-Induced Anticoagulation: A Systematic Review.

Thibault N[1,2], Morrill AM[2], Willett KC[2].

Author information

Abstract

BACKGROUND: The approval of the oral direct thrombin inhibitor, dabigatran etexilate, gave patients an alternative to oral anticoagulation with warfarin. Like all anticoagulants, the primary adverse event (AE) associated with dabigatran is bleeding. Until the FDA approval of idarucizumab, there had been no reversal agent for dabigatran-induced anticoagulation in patients with life-threatening or uncontrollable bleeding, or those requiring emergent procedures.

AREAS OF UNCERTAINTY: The primary purpose of this review is to summarize the safety and efficacy of idarucizumab, a monoclonal antibody fragment, and its use as a reversal agent for dabigatran.

DATA SOURCES: A literature search was conducted through MEDLINE (1946 to November week 1 2015) and Embase (1980-2015 week 46) using the search term idarucizumab. Clinicaltrials.gov was consulted for a comprehensive list of ongoing and completed studies. Additional studies were identified through bibliographical citations. Clinical trials in animals and humans published in English evaluating the safety and efficacy of idarucizumab for reversal of anticoagulant treatment with dabigatran were included for review.

RESULTS: Idarucizumab has been shown to significantly reverse the anticoagulant effects of dabigatran in both healthy volunteers and patients requiring a reversal agent because of either overt bleeding or an emergency surgery or invasive procedure. The most common AEs were headache, nasopharyngitis, back pain, skin irritation, hypokalemia, delirium, constipation, pyrexia, and pneumonia. Deaths reported in idarucizumab studies were attributed to either the index event or a preexisting comorbidity. Most adverse effects were minor, but 21 serious AEs have been reported in the published data including thrombotic events.

CONCLUSIONS: Given the increased use of direct oral anticoagulants, such as dabigatran, a need for specific reversal agents exists. Idarucizumab has been shown to be safe and effective in the reversal of dabigatran-induced anticoagulation in patients requiring emergent or urgent surgery or in patients with severe bleeding.

NEWS

Andexanet Alfa, First Reversal Agent for Factor Xa Inhibitors, Finally Gains FDA Approval

The agent is the second antidote approved for the NOACs, joining idarucizumab, dabigatran's reversal agent.

By Todd Neale | May 04, 2018

ORIGINAL ARTICLE

Full Study Report of Andexanet Alfa for Bleeding Associated with Factor Xa Inhibitors

S.J. Connolly, M. Crowther, J.W. Eikelboom, C.M. Gibson, J.T. Curnutte, J.H. Lawrence, P. Yue, M.D. Bronson, G. Lu, P.B. Conley, P. Verhamme, J. Schmidt, S. Middeldorp, A.T. Cohen, J. Beyer-Westendorf, P. Albaladejo, J. Lopez-Sendon, A.M. Demchuk, D.J. Pallin, M. Concha, S. Goodman, J. Leeds, S. Souza, D.M. Siegal, E. Zotova, B. Meeks, S. Ahmad, J. Nakamya, and T.J. Milling, Jr., for the ANNEXA-4 Investigators*

ABSTRACT

BACKGROUND

Andexanet alfa is a modified recombinant inactive form of human factor Xa developed for reversal of factor Xa inhibitors.

METHODS

We evaluated 352 patients who had acute major bleeding within 18 hours after administration of a factor Xa inhibitor. The patients received a bolus of andexanet, followed by a 2-hour infusion. The coprimary outcomes were the percent change in anti–factor Xa activity after andexanet treatment and the percentage of patients with excellent or good hemostatic efficacy at 12 hours after the end of the infusion, with hemostatic efficacy adjudicated on the basis of prespecified criteria. Efficacy was assessed in the subgroup of patients with confirmed major bleeding and baseline anti–factor Xa activity of at least 75 ng per milliliter (or ≥0.25 IU per milliliter for those receiving enoxaparin).

RESULTS

Patients had a mean age of 77 years, and most had substantial cardiovascular disease. Bleeding was predominantly intracranial (in 227 patients [64%]) or gastrointestinal (in 90 patients [26%]). In patients who had received apixaban, the median anti–factor Xa activity decreased from 149.7 ng per milliliter at baseline to 11.1 ng per milliliter after the andexanet bolus (92% reduction; 95% confidence interval [CI], 91 to 93); in patients who had received rivaroxaban, the median value decreased from 211.8 ng per milliliter to 14.2 ng per milliliter (92% reduction; 95% CI, 88 to 94). Excellent or good hemostasis occurred in 204 of 249 patients (82%) who could be evaluated. Within 30 days, death occurred in 49 patients (14%) and a thrombotic event in 34 (10%). Reduction in anti–factor Xa activity was not predictive of hemostatic efficacy overall but was modestly predictive in patients with intracranial hemorrhage.

CONCLUSIONS

In patients with acute major bleeding associated with the use of a factor Xa inhibitor, treatment with andexanet markedly reduced anti–factor Xa activity, and 82% of patients had excellent or good hemostatic efficacy at 12 hours, as adjudicated according to prespecified criteria. (Funded by Portola Pharmaceuticals; ANNEXA-4 ClinicalTrials.gov number, NCT02329327.)

The authors' full names, academic degrees, and affiliations are listed in the Appendix. Address reprint requests to Dr. Connolly at the Population Health Research Institute, Hamilton Health Sciences, 237 Barton St. E, Hamilton, ON L8L 2X2, Canada, or at connostu@phri.ca.

*A complete list of the ANNEXA-4 investigators is provided in the Supplementary Appendix, available at NEJM.org.

This article was published on February 7, 2019, at NEJM.org.

DOI: 10.1056/NEJMoa1814051

Clinical Case Report

Medicine

OPEN

Sudden valproate-induced hyperammonemia managed with L-carnitine in a medically healthy bipolar patient

Essential review of the literature and case report

Carlo Ignazio Cattaneo, MD[a,*], Francesca Ressico, MD[a], Roberta Valsesia, MD[b], Pierluigi D'Innella, MD[b], Matteo Ballabio, MA[a], Michele Fornaro, MD, PhD[c]

Abstract

Rationale: Valproic Acid is a commonly used psychiatric drug primarily used as a mood stabilizer. Mild hyperammonemia is a Valproic Acid common adverse effect. This report presents an example of treated hyperammonemia on Valproic acid therapy managed with L-carnitine administration in BD patients characterized by sudden vulnerability.

Patient concerns: We report the case of a 29-year-old man suffering from bipolar disorder (BD) and substance use disorder who exhibited sudden altered mental status upon admittance to the inpatient unit. The patient was started on Valproic acid with no improvement.

Diagnoses: The patient had remarkably high ammonia levels (594 μg/dL) without hepatic insufficiency, likely due to his valproate treatment.

Interventions: The patient was administered lactulose, intravenous hydration, and i.v. levocarnitine supplementation 4.5 g/day.

Outcomes: The administration leads to reduction of ammonia levels to 99 μg/dL within 12 hours upon initiation of carnitine therapy and progressive restore of his mental status within 24 hours.

Lessons: Resolution of hyperammonemia caused by Valproic acid therapy may be enhanced with the administration of L-carnitine. An interesting aspect of this case was how rapidly the patient responded to the carnitine therapy.

Abbreviations: AED = antiepiletic drug, AEs = adverse effects, AW = alcohol withdrawal, BD = bipolar disorder, BPD = borderline personality disorder, CBZ = carbamazepine, ECG = electrocardiography, EEG = electroencephalogram, i.m = intramuscular, i.v = intravenous, MR = mental retardation, o.s = oral administration, PB = phenytoin, PHT = phenobarbital, SUD = substance use disorder, TBI = traumatic brain injury, VHE = valproate-induced hyperammonemic encephalopathy, VPA = valproic acid.

Keywords: bipolar disorder, carnitine, hepatic dysfunction, hyperammonemia, neurotoxicity, valproic acid

1. Introduction

Valproic acid (VPA) is a broad-spectrum antiepileptic drug (AED) that inhibits degradation, and promotes postsynaptic transmission of gamma-aminobutyric acid (GABA).[1] VPA is widely used for the treatment of epilepsy, migraine, and a variety of psychiatric symptoms, including bipolar disorder (BD), borderline personality disorder (BPD), and alcohol withdrawal (AW). VPA has been used effectively to reduce agitation and aggression in both acute and postacute traumatic brain injury (TBI) patients,[2,3] as well as a variety of other neuropsychiatric

Published OnlineFirst July 11, 2016; DOI: 10.1158/1078-0432.CCR-16-0638

CCR Drug Updates

Clinical Cancer Research

FDA Approval: Uridine Triacetate for the Treatment of Patients Following Fluorouracil or Capecitabine Overdose or Exhibiting Early-Onset Severe Toxicities Following Administration of These Drugs

Gwynn Ison, Julia A. Beaver, W. David McGuinn Jr, Todd R. Palmby, Jeannette Dinin, Rosane Charlab, Anshu Marathe, Runyan Jin, Qi Liu, Xiao Hong Chen, Xavier Ysern, Olen Stephens, Ge Bai, Yaning Wang, Sarah E. Dorff, Joyce Cheng, Shenghui Tang, Rajeshwari Sridhara, William Pierce, Amy E. McKee, Amna Ibrahim, Geoffrey Kim, and Richard Pazdur

Abstract

On December 11, 2015, the FDA approved uridine triacetate (VISTOGARD; Wellstat Therapeutics Corporation) for the emergency treatment of adult and pediatric patients following a fluorouracil or capecitabine overdose regardless of the presence of symptoms, and of those who exhibit early-onset, severe, or life-threatening toxicity affecting the cardiac or central nervous system, and/or early onset, unusually severe adverse reactions (e.g., gastrointestinal toxicity and/or neutropenia) within 96 hours following the end of fluorouracil or capecitabine administration not been established. The approval is based on data from two single-arm, open-label, expanded-access trials in 135 patients receiving uridine triacetate (10 g or 6.2 g/m^2 orally every 6 hours for 20 doses) for fluorouracil or capecitabine overdose, or who exhibited severe or life-threatening toxicities within 96 hours following the end of fluorouracil or capecitabine administration. Ninety-six percent of patients met the major efficacy outcome measure, which was survival at 30 days or survival until the resumption of chemotherapy, if prior to 30 days. The most

Toxicol Appl Pharmacol. 2018 Aug 15;353:67-73. doi: 10.1016/j.taap.2018.06.012. Epub 2018 Jun 13.

Prompt treatment with uridine triacetate improves survival and reduces toxicity due to fluorouracil and capecitabine overdose or dihydropyrimidine dehydrogenase deficiency.

Garcia RAG[1], Saydoff JA[2], Bamat MK[3], von Borstel RW[4].

Abstract

Uridine triacetate has been shown to be an effective antidote against mortality and toxicity caused by either overdoses or exaggerated susceptibility to the widely used anticancer agents 5-fluorouracil (5-FU) and capecitabine. However, a direct assessment of efficacy based on when emergency treatment was initiated was not clinically feasible. In this study we used mouse models of 5-FU overdose and of dihydropyrimidine dehydrogenase (DPD) deficiency to compare the efficacy of uridine triacetate in reducing toxicity and mortality when treatment was initiated at time points from 4 to 144 h after administration of 5-FU. **We found that uridine triacetate was effective both in the 5-FU overdose and DPD deficiency models. Starting treatment within 24 h was most effective at reducing toxicity and mortality in both models, while treatment starting more than 96 to 120 h after 5-FU was far less effective.** Uridine triacetate also reduced mortality in the DPD deficiency model when mice were treated with the 5-FU prodrug capecitabine. The results of this study are supportive of clinical observations and practice, indicating that efficacy declined progressively with later and later treatment initiation. Prompt treatment with uridine triacetate, within 24 h, conferred the greatest protection against 5-FU overexposure.

In Vitro and In Vivo Assessment of EDTA-Modified Silica Nano-spheres with Supreme Capacity of Iron Capture as a Novel Antidote Agent

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[a] Pharmaceutical Sciences Research Center, Shiraz University of Medical Sciences, P.O. Box 71345-1583, Shiraz, Iran
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500 mg/kg ferröz sülfat verildikten sonra NH2-MSN ve EDTA-MSN 10mg/kg oral olarak veriliyor.
24 saat içerisindeki Fe düzeyi takip ediliyor.

In vivo experiments illustrated that both nanoparticles could efficiently be administrated as an antidote agent against iron overdose, but EDTA-MSN nanoparticles were superior to NH2- MSN nanoparticles.

ARTICLE

Detoxification of Organophosphate Poisoning Using Nanoparticle Bioscavengers

Zhiqing Pang,[†,‡] Che-Ming J. Hu,[†] Ronnie H. Fang,[†] Brian T. Luk,[†] Weiwei Gao,[†] Fei Wang,[†,‡] Erdembileg Chuluun,[†] Pavimol Angsantikul,[†] Soracha Thamphiwatana,[†] Weiyue Lu,[‡] Xinguo Jiang,[‡] and Liangfang Zhang*[,†]

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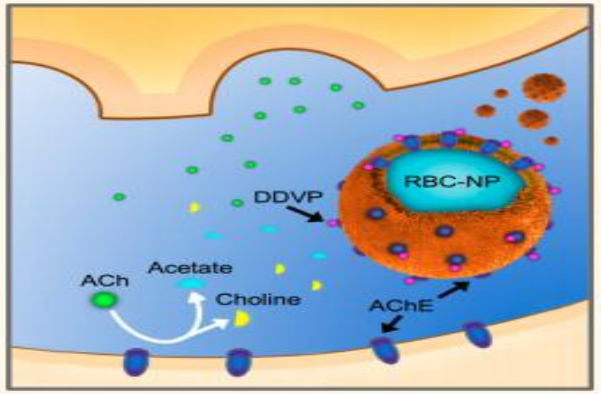
ABSTRACT Organophosphate poisoning is highly lethal as organophosphates, which are commonly found in insecticides and nerve agents, cause irreversible phosphorylation and inactivation of acetylcholinesterase (AChE), leading to neuromuscular disorders *via* accumulation of acetylcholine in the body. Direct interception of organophosphates in the systemic circulation thus provides a desirable strategy in treatment of the condition. Inspired by the presence of AChE on red blood cell (RBC) membranes, we explored a biomimetic nanoparticle consisting of a polymeric core surrounded by RBC membranes to serve as an anti-organophosphate agent. Through *in vitro* studies, we demonstrated that the biomimetic nanoparticles retain the enzymatic activity of membrane-bound AChE and are able to bind to a model organophosphate, dichlorvos, precluding its inhibitory effect on other enzymatic substrates. In a mouse model of organophosphate poisoning, the nanoparticles were shown to improve the AChE activity in the blood and markedly improved the survival of dichlorvos-challenged mice.



KEYWORDS: nanomedicine · biomimetic nanoparticle · biodetoxification · organophosphate · acetylcholinesterase

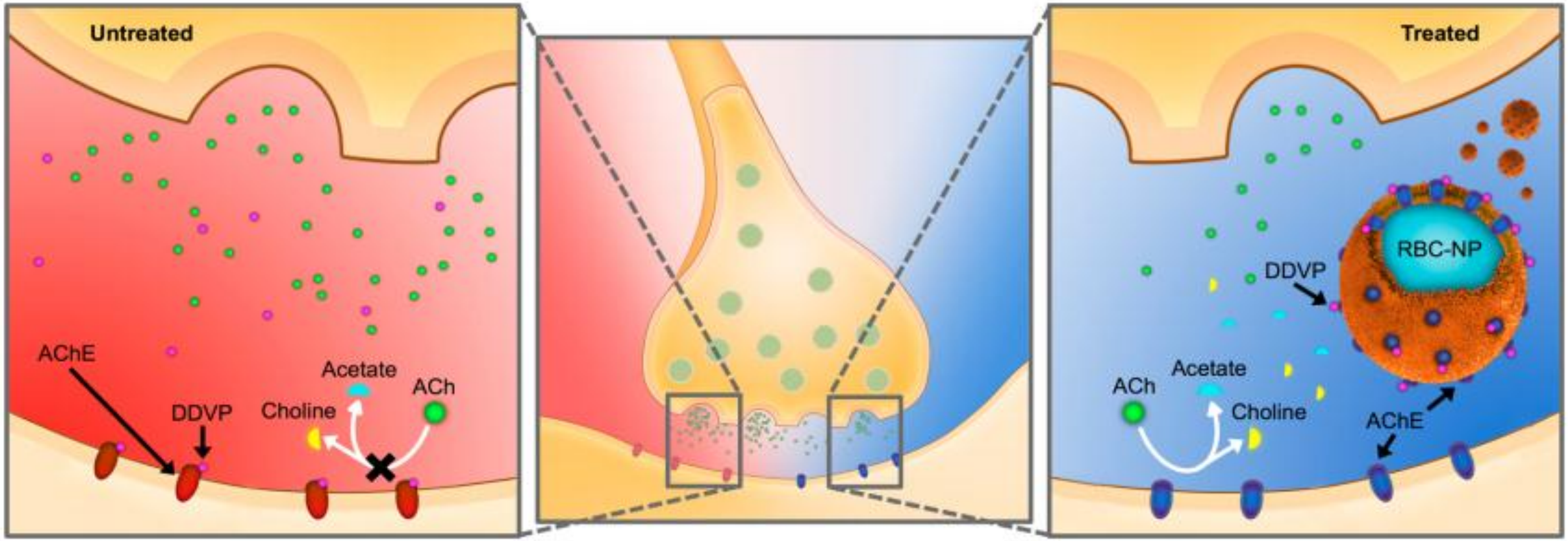


Figure 1. Schematic of RBC-NPs as anti-OP bioscavengers for treating OP poisoning. An idealized depiction of a neuronal synapse (center) under two opposing conditions. With no treatment (left), dichlorvos (DDVP), a model OP, irreversibly binds acetylcholinesterase (AChE), preventing the breakdown of acetylcholine (ACh) into choline and acetate. When RBC-NPs are introduced (right), they scavenge free DDVP molecules in circulation, preserving the ability of endogenous AChE at the synapse to perform the function of breaking down ACh.

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