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New Insights into Drug Toxicity and Antidotes

Dr. Raghavendra Rao M V[1], Dr. Raj Kumar Kudari[2], Dr. Sreevani Namani[3], Dr. Shyamala[4]

1. Professor of Microbiology, and Senior Executive Vice-President, World Academy of Medical Sciences, Netherlands, (Europe)
2. Principal and Professor, St. Mary's College of Pharmacy Chebrolu, Guntur-522212, Andhra Pradesh, India
3. Assistant Professor,Department of Biochemistry, Commonwealth University College of Medicine, Saint Lucia, Caribbean Islands
4. Assistant Professor, Department of Physiology, Commonwealth University College of Medicine, Saint Lucia, Caribbean Islands

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ABSTRACT

In recent years, "tried and true" remedies for patients have fallen like autumn leaves when medical scientists discovered that the treatment was either worthless or worse than the disease. Antidotes are an important aspect of poisoning therapy. Some antidotes play a critical role in saving lives or reducing the severity and duration of toxicity. Antidotes are reversal agents, they are mostly rapid-acting and are used in situations of medication overdose or toxicity. Most often, in the case of an ingested poison, treatment involves administering a slurry of activated charcoal that is finely ground. Toxins stick to the surface and can then be harmlessly excreted. Antidotes mediate their effect either by preventing the absorption of the toxin, by binding and neutralizing the poison, antagonizing its end-organ effect, or by inhibiting the conversion of the toxin to more toxic metabolites. In severe poisoning, the antidote is only an adjunct to supportive treatment, and its use should not distract the physician from delivering adequate attention to airway, breathing, circulation, and decontamination.

Keywords : Antidote, Hemoperfusion techniques, Therapeutic index, N-acetylcysteine (NAC), Atropine, Dig-Fab

INTRODUCTION

Antidote administration may not only reduce free or active toxin levels but also mitigate the toxin's end-organ effects through mechanisms such as competitive inhibition, receptor blockade, or direct antagonism.(1)

In a small proportion (%3C;2%) of toxins, antidotes have been identified. It must be stressed that the expected benefit of the antidote must be weighed against its potential side effects and toxicity.(2)

When antidotes are administered appropriately, they may limit morbidity and mortality, as demonstrated in paracetamol and digitalis overdose.(3)

The International Programme of Chemical Safety broadly defines an antidote as a therapeutic agent that counteracts the toxic actions of a drug/toxin.(4)

Broadly, antidotes have been looked at as agents that “modify the kinetics of the toxic substance or interfere with its effect at receptor sites.”(5)

It is estimated that nearly 45,900 people in India die of snake bite, the highest mortality rate in the world for snake bite. Those who die are from the lower socioeconomic groups, such as farmers, villagers, children, and those with very little publicity value for the media. Polyvalent antisnake venom (ASV) is widely available, manufactured in India, and is the antidote of choice. (6)

ASV is a costly medicine, and chief medical officers are hesitant to leave large stocks in the PHCs for fear of misuse/pilferage by the personnel. Precious little is being done to address the issue of the unavailability of ASV in adequate dosage at the point of first contact for care.[7]

It seems rather sad that at a time when noncommunicable diseases and other lifestyle diseases are getting the attention of health policy makers, this neglected problem is remaining the same. There have been questions in the Lok Sabha from time to time regarding deaths due to snake bites and the availability of ASV.[8]

Scorpion sting is another problem that is widespread in South India, but not so in the northern parts of the country. The antidote for this venom was never widely or easily available in the country, though manufactured in India. Excellent results in the management of these cases with prazosin have decreased the number of deaths.[9]

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Broadly, antidotes have been looked at as agents that "modify the kinetics of the toxic substance or interfere with its effect at receptor sites." (12)

This may be as a result of prevention of absorption, binding, and neutralizing the poison directly, antagonizing its end-organ effect, or inhibition of conversion to more toxic metabolites. (13)

A reduction in the free or active toxin level can be achieved by agents that "bind" to the toxin. This binding can be either specific or nonspecific. (14)

Specific binding occurs in the form of chelating agents for heavy metal poisoning, Digi-Fab for digoxin overdose, hydroxycobalamin for cyanide poisoning, or bioscavenger therapy (human butyryl cholinesterase) for organophosphorus poisoning. (15)

where the antidote enables the formation of inert complexes that are then eliminated from the body. Activated charcoal has been included in the list of nonspecific antidotes because it can decrease the toxin levels by its high adsorption capacity and by interrupting the enterohepatic recirculation of the toxin. A higher charcoal to drug ratio will more effectively inhibit systemic absorption; while 10:1 is ideal, some reports suggest that a 40:1 ratio. (16)

charcoal to drug ratio might be superior. Activated charcoal has been in use for over a century, and while it has been reported to be the most common form of gastrointestinal decontamination in the poisoned patient, its use has declined from 7.7% to 5.9%. (17)

Intravenous lipid therapy has been in use in humans for both lipophilic local anesthetics and nonlocal anesthetic agents (β-blockers, calcium channel blockers, and psychotropic drug overdose). This works on the lipid sink principle, where lipophilic drugs, with an octanol to water partitioning coefficient of log p > 2, are trapped in the plasma lipid compartment.(18)

Lipid emulsion therapy has also been proposed to have a direct inotropic effect through an increase in calcium levels in cardiac myocytes. (19)

Enhancing the elimination of toxins with the use of antidotes can be done either through hemoperfusion techniques (charcoal or resin-based) or urinary alkalinization (targeting a pH > 7.5) with intravenous sodium bicarbonate therapy. (20)

POISONS & ANTIDOTES

Four Forms of Poisons

Poisons typically appear in four primary forms:

- **Solids**: These include items like pills or powders.
- **Liquids**: Examples include household cleaners or alcoholic substances.
- **Gases**: Carbon monoxide is a key example.
- **Sprays**: This category includes items like insecticides or aerosols.

These forms are important because they influence how poisons interact with the body and how they should be handled in emergency situations.

Classification of Toxic Agents

Toxins can be grouped into two broad categories:

1. **Toxins with specific treatments available**, which include cases where antidotes or other specific medical interventions can help.

2. **Toxins without known antidotes**, where care is focused on supporting the patient through the symptoms while the body works to recover.

The core principle in managing toxicological emergencies is often referred to as **"Treat the patient, not the toxin."** This emphasizes the importance of providing supportive care, especially since antidotes for many poisons are not available.

What is an Antidote?

An antidote is a substance that neutralizes or diminishes the harmful effects of a poison or drug. The primary ways antidotes work include:

- Preventing the toxin from being absorbed into the body
- Binding to the toxin to make it inactive
- Blocking the toxin's effect on its target receptor
- Helping to convert harmful metabolites into less toxic ones

The safety of a chemical agent is often measured by its **therapeutic index** (TD50/ED50), which compares the toxic dose to the effective dose. Antidotes are seen as substances that increase the lethal dose of a toxin, making it less harmful.

How Do Antidotes Work?

Antidotes generally operate through several mechanisms:

1. **Reducing the active toxin levels**: This can involve specific or nonspecific binding, or enhancing the elimination of the toxin.

2. **Blocking the toxin's target site**: This can include competitive inhibition of enzymes or blocking receptor sites.

3. **Reducing toxic metabolites**: This is done by either binding the toxic metabolites or helping convert them into safer forms.

4. **Counteracting the toxin's harmful effects**: Some antidotes can mitigate the damage caused by toxins, such as reversing respiratory depression or organ damage.

Mechanisms of Antidotal Therapy

Antidotes work by four main strategies to reduce the toxin's effects:

1. **Direct action on the toxin**: This includes binding agents, such as chelators for heavy metals or activated charcoal, which can bind the toxin and help eliminate it.

2. **Interfering with the toxin's binding site**: This can involve blocking enzyme activity or receptor sites, as seen with antidotes like **naloxone** for opioids or **flumazenil** for benzodiazepines.

3. **Reducing the formation of toxic metabolites**: For example, **N-acetylcysteine (NAC)** is used in paracetamol toxicity to prevent liver damage by binding to harmful metabolites.

4. **Counteracting the effects of the toxin**: Medications like **atropine** for organophosphate poisoning or **vitamin K** for warfarin overdose are used to reverse the toxic effects.

Reducing the Active Toxin Level

Antidotes can reduce the active toxin in several ways:

- **Specific binding agents**: These include chelators for heavy metals, **Digi-Fab** for digoxin, and other agents that form inert complexes to facilitate elimination.

- **Nonspecific binding**: **Activated charcoal** is commonly used to adsorb a range of toxins, though its use has declined due to limited evidence and potential risks such as aspiration.

- **Lipid emulsion therapy**: Used in cases of toxicity from lipophilic drugs, this method works by trapping the toxins in fat compartments in the bloodstream.

- **Enhancing elimination**: This can involve techniques like **hemoperfusion** or **urinary alkalinization** to help the body excrete the toxin more effectively.

Blocking the Toxin-Binding Site

Some antidotes work by directly interfering with the toxin's action at the receptor or enzyme level:

- **Enzyme inhibition**: For example, **fomepizole** or **ethanol** can prevent the conversion of methanol and ethylene glycol into toxic metabolites by inhibiting alcohol dehydrogenase.

- **Receptor blockade**: **Naloxone** competes with opioids at opioid receptors, while **flumazenil** does the same at the benzodiazepine receptor.

Reducing Toxic Metabolites

Once toxins are metabolized into harmful substances, antidotes can either bind to these metabolites or convert them into less harmful forms:

- **N-Acetylcysteine (NAC)** restores glutathione in the liver, helping prevent damage from the toxic metabolite of paracetamol.

- **Sodium thiosulfate** can convert cyanide into a less toxic substance, **thiocyanate**, which is easier for the body to eliminate.

Counteracting the Harmful Effects of Toxins

Some antidotes do not directly alter the toxin but mitigate its effects:

- **Atropine** helps reduce the toxic effects of organophosphate poisoning by blocking muscarinic receptors.

- **Pyridoxine** can prevent seizures in cases of isoniazid poisoning.

- **Folinic acid** can reverse the toxic effects of methotrexate.

Timing and Duration of Antidote Administration

The effectiveness of an antidote is often time-sensitive, meaning that earlier administration generally leads to better outcomes. The decision to use an antidote depends on factors such as:

- The type of toxin involved

- The time since exposure

- The antidote's mechanism of action

- The dose and route of exposure

For antidotes that reduce toxin levels, earlier use is typically more beneficial. For antidotes that act on metabolites or toxic effects, the timing may be more flexible.

Duration of Antidotal Therapy

The length of time an antidote should be used depends on the nature of the toxin, how much was ingested, and how quickly symptoms resolve. For toxins with a long half-life, ongoing treatment may be required, including continuous infusions.

Evidence of Antidote Efficacy

Most decisions about antidote use are based on clinical experience, case reports, and pharmacokinetic data rather than on large-scale randomized controlled trials. However, in some cases, particularly for drugs like activated charcoal, limited research has led to questions about its widespread use. Nonetheless, the lack of definitive evidence does not prevent clinicians from using antidotes when they believe the benefits outweigh the risks.

common dangerous substances

Pain relievers, sedatives, antidepressants, cleaning supplies, alcohols, antifreeze, pesticides, certain heavy metals (arsenic, lead), carbon monoxide, and snake/insect venoms, while extremely potent natural/synthetic toxins include Botulinum toxin, Dioxin, and Sarin, with household items like antifreeze being a frequent, sweet-tasting hazard, and plants like poison hemlock and mushrooms (e.g., Death Cap) being highly toxic.

Common sedatives include barbiturates, benzodiazepines, gamma-hydroxybutyrate (GHB), opioids, and sleep-inducing drugs such as zolpidem (Ambien) and eszopiclone (Lunesta). Sedatives are central nervous system depressants and vary widely in their potency. They are usually available as a pill or liquid. Though sedatives are used widely for their medicinal properties, abuse of sedatives can result in dependence and addiction.

Pain relievers

Pain relievers, or analgesics, reduce pain, inflammation, and fever. Common types include Acetaminophen (Tylenol) for pain/fever, and NSAIDs (ibuprofen, naproxen) for pain and inflammation. They are used for headaches, muscle aches, arthritis, and dental pain. Risks include liver damage (overdose), stomach irritation, ulcers, and kidney issues, requiring careful dosage management.

Commonly used sedatives

Commonly administered sedatives to critically ill patients worldwide are propofol, benzodiazepines, ketamine, and barbiturates.

Propofol

It is a fast-acting intravenous sedative-hypnotic agent used to induce/maintain general anaesthesia and provide sedation for procedures or in the ICU known as "milk of amnesia" due to its milky appearance, it acts within 30–60 seconds, typically allowing rapid recovery. It is used for sedation in adults and children over two months, often in intensive care for ventilated patients.

Pesticides

The alcohols, methanol, ethylene glycol, and diethylene glycol, have many features in common, the most important of which is the fact that the compounds themselves are relatively non-toxic but are metabolized, initially by alcohol dehydrogenase, to various toxic intermediates. These compounds are readily available worldwide in commercial products as well as in homemade alcoholic beverages, both of which lead to most of the poisoning cases, from either unintentional or intentional ingestion.

Pesticides kill or control forms of animal and plant life. Herbicides destroy or control weeds and other unwanted vegetation.

Heavy metals

Heavy metals, such as arsenic, cadmium, mercury, and lead, are highly poisonous. Heavy metal poisoning is treated with chelating agents, which bind to metal ions to enhance their elimination, often administered via IV or orally. Common antidotes include DMSA

(Succimer) for lead/mercury, DMPS for arsenic/mercury, CaEDTA for lead, and Deferoxamine for iron. These must be administered under medical supervision.

Snake and Insect venom

Snake and insect venoms are complex mixtures of enzymes, peptides, and toxins that immobilize prey, provide defence, and aid digestion.

Snake venom, mainly from Elapidae and Viperidae families, contains neurotoxins, hemotoxins, and cytotoxins, while insect venoms often cause intense pain and localized tissue damage. Both can cause severe systemic reactions that require rapid treatment with antivenom.

Opioids

Opioid overdose is the most common cause of drug-related deaths worldwide. Naloxone is an effective treatment when instituted early. The emergence of highly potent novel synthetic opioids such as nitazenes and fentanyl analogues has led to a systematic reappraisal of the evidence on management of opioid toxicity with naloxone and development of evidence-based clinical guidelines to reduce opioid-related deaths.

Antidote to Opioids--Naloxone

Naloxone is a non-selective, competitive, μ receptor antagonist with greater receptor affinity than exogenous opioids. Naloxone is highly lipophilic and therefore rapidly distributes throughout the central nervous system with a time of onset to action of seconds to minutes after intravenous administration. The bioavailability of naloxone after oral and rectal administration is 1-2% and 15%, respectively; thus, naloxone must be administered parenterally via intravenous (IV), endotracheal, intramuscular (IM), intraosseous (IO), subcutaneous (SC), and/or intranasal (IN) routes. Naloxone has a rapid onset of action, reaching maximal serum concentration within 2 minutes after IV administration, 10 minutes after IM administration, and 15–30 minutes after IN administration. Naloxone distributes to the central nervous system and equilibrates with the plasma within minutes. Naloxone is extensively metabolized in the liver to inactive metabolites with a serum elimination half-life of 30–90 minutes, which is shorter than the half-life of many opioids, requiring careful monitoring and repeat dosing when necessary. If other routes such as endotracheal, intramuscular, or intraosseous are used, then the onset of action may be 5-10 minutes, or even longer if the patient is hypotensive. This is clinically important in situations such as cardiac arrest and may alter the duration of resuscitation efforts. Formulations available in the UK and EU include naloxone 0.4 mg/mL and 2mg/2mL for intravenous injection and naloxone 1.26 mg/0.1 mL and 1.8 mg/0.1mL for intranasal administration. In the United States, the FDA-approved naloxone formulations include naloxone nasal sprays (2, 3, 4, and 8 mg), prefilled naloxone injection devices for intramuscular (IM) or subcutaneous (SC) use, and generic naloxone for IV, IM or SC use. The 3 mg and 4 mg intranasal atomised spray formulated in 0.1 mL were approved in 2023 for over-the-counter, non-prescription use in response to the need to reduce opioid-related deaths. The Evzio auto-injector has been withdrawn, but the Zimhi auto-injector is a prefilled syringe containing 5 mg/0.5 mL naloxone for IM injection into the anterolateral aspect of the thigh, through clothing if necessary. (35)

Barbiturates

Barbiturates have been used historically to treat insomnia and psychiatric disorders, provide anaesthesia, and manage alcohol withdrawal, elevated intracranial pressure, and seizures. Once extremely popular for a broad spectrum of indications in the late 20th century, the use of these drugs has declined mainly in favour of agents with more favourable safety profiles. However, barbiturates are still prescribed or obtained illicitly, and their misuse, whether intentional or not, can lead to grave harm or death.(36)

Antidote

Current recommendations for enhanced elimination are based on the pharmacokinetic and toxicokinetic properties of each barbiturate drug, clinical trials, case reports, and expert opinion. Adequately controlled trials supporting these strategies are lacking, however. Thus, enhanced elimination is only recommended for barbiturate toxicity with coma, need for respiratory support, or need for cardiovascular support (ie, when clinical benefits are likely to outweigh the risks of treatment).(37)

Conclusion

Antidotes are agents that neutralize toxins by preventing absorption, binding/neutralizing the poison, or inhibiting its conversion into harmful metabolites. They work via mechanisms like receptor blockade, chelating agents, or direct antagonism to reduce

toxicity. While only a few dozen common antidotes exist for hundreds of toxins, they are critical for life-saving interventions in emergencies. The highest mortality rate in the world for snakebite. Successful outcomes in a toxicological emergency not only require appropriate management of airway, breathing, and circulation but also the knowledge and application of appropriate antidote therapy. The latter may result in reducing the intensity of the poisoning and improving outcomes.

REFERENCES

1. Binila Chacko, John V Peter, Antidotes in Poisoning, Indian Journal of Critical Care Medicine,Volume 23 | Issue Suppl 4 | Year 2019
2. Karami M, Estachri MRAE. Principles of toxicotherapy: general and specific therapy. Sch Acad J Pharm 2015;4(3):153–156.
3. Wang RY, Kazzi ZN. Editorial: antidotes and rescue therapies. Curr Pharm Biotechnol 2012;13(10):1914–1916. DOI: 10.2174/138920112802273164.
4. Jacobsen D. The relative efficacy of antidotes. J Toxicol Clin Toxicol 2008;33(6):705–708. DOI: 10.3109/15563659509010633.
5. De Garbino JP, Haines JA, Jacobsen D, Meredith T. Evaluation of antidotes: activities of the International Programme on Chemical Safety. J Toxicol Clin Toxicol 2009;35(4):333–343. DOI: 10.3109/15563659709043364.
6. Mohapatra B, Warrell DA, Suraweera W, Bhatia P, Dhingra N, Jotkar RM, et al. Million Death Study Collaborators. Snakebite mortality in India: A nationally representative mortality survey. PLoS Negl Trop Dis. 2011;5:e1018. doi: 10.1371/journal.pntd.0001018. [DOI] [PMC free article] [PubMed] [Google Scholar
7. Mazoomdaar J. Snake bites and poisoned stats. [Last accessed on 2013 Nov 30]. Available from: http://archive.tehelka.com/story_main54.asp?filename=Ne131012SNAKE.asp
8. Lok Sabha questions. Fifteenth Lok Sabha. Question nos. 3762 and 4568. [Last accessed on 2013 Dec 1]. Available from: http://164.100.47.132/LssNew/psearch/qsearch15.aspx .
9. Bhawaskar HS, Bawaskar PH. Scorpion sting: Update. J Assoc Physicians India. 2012;60:46–55. [PubMed] [Google Scholar]
10. Wang RY, Kazzi ZN. Editorial: antidotes and rescue therapies. Curr Pharm Biotechnol 2012;13(10):1914–1916. DOI: 10.2174/138920112802273164.
11. Karami M, Estachri MRAE. Principles of toxicotherapy: general and specific therapy. Sch Acad J Pharm 2015;4(3):153–156.
12. Wang RY, Kazzi ZN. Editorial: antidotes and rescue therapies. Curr Pharm Biotechnol 2012;13(10):1914–1916. DOI: 10.2174/138920112802273164.
13. Jacobsen D. The relative efficacy of antidotes. J Toxicol Clin Toxicol 2008;33(6):705–708. DOI: 10.3109/15563659509010633
14. Pichamuthu K, Jerobin J, Nair A, John G, Kamalesh J, Thomas K, et al. Bioscavenger therapy for organophosphate poisoning - an open-labeled pilot randomized trial comparing fresh frozen plasma or albumin with saline in acute organophosphate poisoning in humans. Clin Toxicol (Phila) 2010;48(8):813–819. DOI: 10.3109/15563650.2010.518970.
15. Peter V, Moran JL, Graham PL. Advances in the management of organophosphate poisoning. Expert Opin Pharmacother 2007;8(10):1451–1464. DOI: 10.1517/14656566.8.10.1451.
16. Pillay VV. Current views on antidotal therapy in managing cases of poisoning and overdose. J Assoc Physicians India 2008;56:881–892.
17. Chyka PA, Seger D, Krenzelok EP, Vale JA, American Academy of Clinical Toxicology, European Association of Poisons Centres and Clinical Toxicologists. Position paper: single-dose activated charcoal. Clin Toxicol (Phila) 2005;43(2):61–87. DOI: 10.1081/CLT-51867.
18. Ozcan MS, Weinberg G. Intravenous lipid emulsion for the treatment of drug toxicity. J Intensive Care Med 2012;29(2):59–70. DOI:
19.1177/0885066612445978. 17. Rothschild L, Bern S, Oswald S, Weinberg G. Intravenous lipid emulsion in clinical toxicology. Scand J Trauma Resusc Emerg Med 2010;18:51. DOI: 10.1186/1757-7241-18-51.
20. Gil H-W, Kim S-J, Yang J-O, Lee E-Y, Hong S-Y. Clinical outcome of hemoperfusion in poisoned patients. Blood Purif 2010;30(2):84–88. DOI: 10.1159/000318585
21. Valerie Gerriets; Jackie Anderson; Preeti Patel; Thomas M. Nappe.,Acetaminophen,NIH,2024
22. Bunchorntavakul C, Reddy KR. Acetaminophen-related hepatotoxicity. Clin Liver Dis. 2013 Nov;17(4):587-607, viii. [PubMed]
23.Ghanem CI, Pérez MJ, Manautou JE, Mottino AD. Acetaminophen from liver to brain: New insights into drug pharmacological action and toxicity. Pharmacol Res. 2016 Jul;109:119-31. [PMC free article] [PubMed)
24. Chandrasekharan NV, Dai H, Roos KL, Evanson NK, Tomsik J, Elton TS, Simmons DL. COX-3, a cyclooxygenase-1 variant inhibited by acetaminophen and other analgesic/antipyretic drugs: cloning, structure, and expression. Proc Natl Acad Sci U S A. 2002 Oct 15;99(21):13926-31. [PMC free article] [PubMed]
25. Alempijevic T, Zec S, Milosavljevic T. Drug-induced liver injury: Do we know everything? World J Hepatol. 2017 Apr 08;9(10):491-502. [PMC free article] [PubMed]
26.Janssen J, Singh-Saluja S. How much did you take? Reviewing acetaminophen toxicity. Can Fam Physician. 2015 Apr;61(4):347-9. [PMC free article] [PubMed]
27.Imani F, Motavaf M, Safari S, Alavian SM. The therapeutic use of analgesics in patients with liver cirrhosis: a literature review and evidence-based recommendations. Hepat Mon. 2014 Oct;14(10):e23539. [PMC free article] [PubMed]

28. Mirrakhimov AE, Voore P, Halytskyy O, Khan M, Ali AM. Propofol infusion syndrome in adults: a clinical update. Crit Care Res Pract. 2015;2015:260385. [PMC free article] [PubMed]
29. Euasobhon P, Dej-Arkom S, Siriussawakul A, Muangman S, Sriraj W, Pattanittum P, Lumbiganon P. Lidocaine for reducing propofol-induced pain on induction of anaesthesia in adults. Cochrane Database Syst Rev. 2016 Feb 18;2(2):CD007874. [PMC free article] [PubMed]
30.Honore PM, Spapen HD. Propofol infusion syndrome: early blood purification to the rescue? Crit Care. 2016 Jul 04;20(1):197. [PMC free article] [PubMed]
31.Levin PD, Levin V, Weissman C, Sprung CL, Rund D. Therapeutic plasma exchange as treatment for propofol infusion syndrome. J Clin Apher. 2015 Oct;30(5):311-3. [PubMed]
32. Venkatraman Rajkumar; Vincent R. Lee; Vikas Gupta, National Library of Medicine, NIH, National Center for Biotechnology,2023
33. Kim JJ, Kim YS, Kumar V. Heavy metal toxicity: An update of chelating therapeutic strategies. J Trace Elem Med Biol. 2019 Jul;54:226-231. [PubMed]
34. Raghavendra Rao, Adarsh Meher Nisanth2, Mary Sowjanya3 , Dilip Mathai4, Raghunandan Reddy 5, Jithendra Kumar Naik 6, P. V. Krishna 7,Mahendra Kumar Verma8, **SSRG International Journal of Medical Science!!!**
IJMS-V9I3P102, Volume 9, Issue 3 2022.
35. **Aidan King, MBBS, MRCP1, Ruben HK Thanacoody, MD, FRCP, FRCP (Edin), FEAPCCT1,Opioid overdose: evidence-based management guidelines and new antidote development,European Society of Medicine, Vol 13 No 10 (2025): Vol.13, Issue 10, October 2025**
36. Roberts DM, Buckley NA. Enhanced elimination in acute barbiturate poisoning - a systematic review. Clin Toxicol (Phila). 2011 Jan;49(1):2-12. [PubMed]
37. Mactier R, Laliberté M, Mardini J, Ghannoum M, Lavergne V, Gosselin S, Hoffman RS, Nolin TD., EXTRIP Workgroup. Extracorporeal treatment for barbiturate poisoning: recommendations from the EXTRIP Workgroup. Am J Kidney Dis. 2014 Sep;64(3):347-58. [PubMed]

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