WWW.IJRPR.COM

International Journal of Research Publication and Reviews

Journal homepage: www.ijrpr.com ISSN 2582-7421

A Review on Ingested Cyanide: Risks, Clinical Presentation, Diagnostic Approach, and Treatment Challenges

Rahul praseed [1*], Dr. Lathika Nayak [2]

[1.]Final Year Pharm D, Srinivas College of Pharmacy, Valachil, Mangalore, Karnataka State, India.
[2.]Assistant professor ,Srinivas college of pharmacy Valachil ,Mangalore Karnataka State, India.
Email:rahulpraseed@gmail.com.

ABSTRACT :

Cyanide poisoning remains one of the most dangerous toxicological emergencies, particularly following ingestion. Due to its rapid cellular action and high lethality, early recognition and management are critical. This review focuses on ingested cyanide poisoning, detailing its sources, mechanisms of toxicity, clinical features, diagnostic strategies, and therapeutic challenges. Cyanide acts by inhibiting cytochrome oxidase in mitochondria, causing histotoxic hypoxia despite adequate oxygenation. The ingested form, often through cyanide salts or cyanogenic compounds, poses diagnostic difficulties because of nonspecific early symptoms. Timely antidote administration and supportive measures significantly improve outcomes. Challenges remain in rapid detection, limited availability of antidotes, and differential diagnosis with other causes of metabolic acidosis. Public health preparedness and clinical awareness are vital for effective management.

Keywords: Cyanide ingestion, hydroxocobalamin, sodium thiosulfate, diagnostic challenges, toxicology, metabolic acidosis

1. INTRODUCTION

Cyanide is an extremely potent cellular toxin that interferes with aerobic metabolism, leading to rapid tissue hypoxia and death. Although inhalation is a common route in industrial exposure, ingestion of cyanide salts or cyanogenic foods represents a major toxicological concern. Cyanide salts such as sodium cyanide and potassium cyanide are highly soluble and can cause death within minutes when ingested. Worldwide, cyanide ingestion occurs in cases of deliberate self-harm, accidental exposure in industries, and consumption of improperly processed cyanogenic foods like cassava. Understanding its clinical course and management challenges is crucial for healthcare providers, especially in resource-limited settings where diagnostic tools and antidotes may not be readily available.[1]

Parker-Cote and colleagues conducted a systematic review of acute cyanide cases over a 48-year time period [9]. They found 84.3% of the cases were from ingested cyanide, compared to 7.8% inhalation. While the cellular mechanism of oral cyanide is not unique, the toxicokinetic and toxicodynamic of oral cyanide is, thus clinical effects are different than those of inhaled cyanide [10]. In addition, oral exposures to cyanide may result in greater absorption when compared to the inhalational route. With oral exposures, patients can have continued absorption of the toxin once it is ingested. In contrast, inhalational exposures are dependent on the patient's respirations and are limited secondary to the development of apnea from cyanide toxicity. [2]Current FDA-approved therapies are not tailored specifically against oral cyanide poisoning. Furthermore, these therapies are not developed for use by first responders or bystanders in the pre-hospital setting, creating a major treatment gap. A poll conducted by the American College of Emergency Physicians found that 90% of physicians report hospitals are not well equipped for mass casualties incidents (MCI), citing access to appropriate medications as a major concern.[3]

2. SOURCES AND RISKS OF INGESTED CYANIDE

Cyanide exposure by ingestion may occur through multiple routes. The most common sources include industrial chemicals, laboratory reagents, and cyanogenic plants. Intentional ingestion is also frequently reported in suicidal cases due to the compound's easy availability in some sectors.[4]

• Industrial and Laboratory Sources :Sodium cyanide and potassium cyanide are used in electroplating, jewelry cleaning, and mining. Improper handling or ingestion of contaminated materials can lead to fatal outcomes.

• Food-related Sources: Certain plants, particularly cassava, bamboo shoots, and bitter almonds, contain cyanogenic glycosides such as linamarin and amygdalin. Poor processing or overconsumption of these foods may lead to chronic or acute toxicity.

• Intentional or Accidental Poisoning: Cyanide is occasionally used for suicide or homicide because of its rapid action. Accidental ingestion can also occur in laboratories or during transportation of cyanide compounds.[6]

3. TOXICOKINETICS AND MECHANISM OF TOXICITY

After ingestion, cyanide salts rapidly dissolve in gastric acid, releasing hydrogen cyanide (HCN), which is absorbed through the gastrointestinal mucosa. Once absorbed, cyanide binds to ferric ions (Fe3+) in cytochrome c oxidase (Complex IV) of the mitochondrial electron transport chain. This binding halts oxidative phosphorylation, blocking ATP synthesis, and leading to cellular hypoxia despite adequate oxygen levels in blood. Anaerobic metabolism predominates, causing lactic acid accumulation and severe metabolic acidosis.

Detoxification occurs mainly in the liver through the enzyme rhodanese, which converts cyanide to thiocyanate using sulfur donors like thiosulfate. Thiocyanate is less toxic and excreted renally. However, during acute poisoning, the detoxification capacity is overwhelmed, leading to rapid systemic toxicity.[7]

4. CLINICAL PRESENTATION

The clinical spectrum of ingested cyanide poisoning depends on the dose, form, and time to treatment. Early symptoms may appear within minutes and progress rapidly to cardiovascular and neurological collapse.

• Mild to Moderate Exposure:Headache, anxiety, confusion, nausea, vomiting, and dizziness.

• Severe Exposure: Seizures, hypotension, bradycardia, respiratory depression, and loss of consciousness.

• Terminal Stage: Cardiac arrest, metabolic acidosis, and coma. Characteristic findings include a bitter almond odor, bright red venous blood, and high venous oxygen saturation due to impaired oxygen extraction.[8,9]

5. DIAGNOSTIC APPROACH

Diagnosis of cyanide ingestion is primarily clinical because laboratory confirmation often takes time. A history of exposure, rapid onset of central nervous system symptoms, and severe lactic acidosis are suggestive findings.

Laboratory tests may show metabolic acidosis (low pH, elevated lactate >8 mmol/L), elevated venous oxygen saturation, and low anion gap. Cyanide concentration in blood above 0.5 mg/L confirms poisoning, although this may not be immediately available. Carboxyhemoglobin and methemoglobin levels help rule out concurrent exposures such as carbon monoxide.[10]

Table 1. Summary of Diagnostic Features in Ingested Cyanide Poisoning

Parameter	Findings
Blood lactate	>8 mmol/L (severe metabolic acidosis)
Venous blood color	Bright red due to oxygen saturation
Plasma cyanide concentration	>0.5 mg/L (toxic level)

6. TREATMENT AND MANAGEMENT

Management of cyanide ingestion focuses on three priorities: rapid decontamination, supportive care, and antidote therapy.

• Decontamination:Activated charcoal may be used if the patient presents within one hour of ingestion. Gastric lavage should be performed cautiously. Skin decontamination is essential if exposure is suspected.

• Supportive Care: Immediate administration of 100% oxygen, airway management, correction of acidosis with sodium bicarbonate, and seizure control using benzodiazepines are key steps.

• Antidote Therapy: Early administration of specific antidotes greatly improves survival.[12,13]

Table 2. Common Antidotes Used in Cyanide Poisoning

Antidote	Mechanism of Action	Remarks
Hydroxocobalamin	Binds cyanide to form cyanocobalamin (vitamin B12)	Preferred; rapid onset, safe in all ages
Sodium nitrite	Induces methemoglobinemia, binds cyanide to methemoglobin	Use cautiously in hypotension
Sodium thiosulfate	Donates sulfur for conversion to thiocyanate	Used adjunctively with nitrite or hydroxocobalamin
Dicobalt edetate	Chelates cyanide directly	Effective but may cause severe side effects

7. TREATMENT CHALLENGES

Despite available therapies, several challenges complicate the management of ingested cyanide poisoning. Delayed diagnosis, limited availability of antidotes, and the rapid progression of toxicity are major issues. Rural or low-resource settings may lack laboratory facilities or antidote kits. Co-ingestion with other toxic agents and variable absorption of cyanide compounds further complicate management.

8. PREVENTION AND PUBLIC HEALTH ASPECTS

Public health strategies should focus on restricting access to industrial cyanide compounds and promoting safe food processing techniques. Proper detoxification of cassava and public education on potential risks are critical in endemic regions. Hospitals and industries handling cyanide should maintain antidote kits and provide regular staff training for emergency response.[14]

9. CONCLUSION

Ingested cyanide poisoning is a medical emergency requiring rapid recognition and intervention. The high fatality rate reflects both the potency of the toxin and the challenges of timely treatment. While antidotes such as hydroxocobalamin and sodium thiosulfate are effective, delays in diagnosis and limited access hinder outcomes. Enhancing diagnostic capabilities, ensuring antidote availability, and increasing clinical awareness remain essential strategies to improve survival.

Cyanide is a deadly xenobiotic. Ingestion can lead to a high body burden of cyanide, severe symptoms, and unique toxicodynamic. Many more deaths occur as a result of ingested cyanide compared to other routes of exposure. While many of these deaths are a result of self-harm, cyanide remains a high-risk chemical threat agent . It is readily available, easy to use, and highly lethal making it an ideal chemical weapon . The development of new therapies with clinically relevant animal models specific to oral cyanide should focus on addressing the unique toxicodynamic profile of this route of administration. The development of easily administered and highly effective antidotes for oral cyanide that can be used in a mass casualty setting is important.

REFERENCES

1. Hall AH, Rumack BH. Clinical toxicology of cyanide. Ann Emerg Med. 1986;15(9):1067–74.
2. Baud FJ. Cyanide: Critical care management of acute poisoning. Clin Toxicol. 2007;45(7):737–48.
3. Nelson L, Howland M, Lewin N, Goldfrank L. Goldfrank's Toxicologic Emergencies. 11th ed. McGraw-Hill; 2019.
4. Cummings TF. The treatment of cyanide poisoning. Occup Med. 2004;54(2):82–5.
5. Borron SW, Baud FJ, Barriot P, Imbert M, Bismuth C. Prospective study of hydroxocobalamin for acute cyanide poisoning in humans. Ann Emerg Med. 2007;49(6):794–801.
6. U.S. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Cyanide. 2016.
7. Baskin SI, Brewer TG. Cyanide poisoning. In: Medical Aspects of Chemical Warfare. Borden Institute; 2008. p. 271–86.
8. Reade MC, Davies SR, Morley PT, Dennett J, Jacobs IC. Review article: management of cyanide poisoning. Emerg Med Australas. 2012;24(3):225–38.
9. Vennesland B, Conn EE, Knowles CJ, Westley J, Wissing F. Cyanide in Biology. Academic Press; 1981.
10. Isom GE, Way JL. Effects of oxygen on cyanide intoxication. Toxicol Appl Pharmacol. 1984;74(1):57–62.
11. Gracia R, Shepherd G. Cyanide poisoning and its treatment. Pharmacotherapy. 2004;24(10):1358–65.
12. Borron SW, Stonerook M, Reid F. Efficacy of hydroxocobalamin for acute cyanide poisoning in animal models. Clin Toxicol. 2006;44(S1):5–15.
13. Xu J, Pan Y, Li Y, Zhang S. Cassava cyanide poisoning and its prevention in Africa. Food Chem Toxicol. 2014;69:228–34.
14. Johnson RP. Cyanide toxicity. J Emerg Med. 1989;7(4):387–92.
15. Chandra H, Gupta K. Clinical profile of cyanide ingestion: an Indian hospital experience. Indian J Crit Care Med. 2012;16(3):164–8.
16. Gill JR, Hayes JA. Cyanide poisoning by ingestion of potassium cyanide: autopsy findings. Forensic Sci Int. 1991;52(1):85–9.
17. Marrs TC. The role of thiocyanate in cyanide detoxification. J Appl Toxicol. 1988;8(6):389–95.
18. Sidell FR, Takafuji ET, Franz DR. Medical Aspects of Chemical Warfare. Washington, DC: Borden Institute; 2008.
19. Karch SB. Pathology of Drug Abuse. 5th ed. CRC Press; 2015.
20. Okafor PN, Maduagwu EN. Cyanide contamination of foods and the potential risk to public health. Afr J Biotechnol. 2005;4(13):1462–4.
21. Mégarbane B, Delahaye A, Goldgran-Toledano D, Baud FJ. Antidotal treatment of cyanide poisoning. J Chin Med Assoc. 2003;66(4):193–203.
22. Bigley AN, Xu C, Henderson TJ, Harvey SP. Enzymatic detoxification of cyanide: mechanisms and applications. Chem Biol Interact. 2019;307:44–54.
23. Dunn MA, Sidell FR. Fire-related cyanide poisoning: prehospital and hospital management. Ann Emerg Med. 1989;18(12):1446–53.
24. Barceloux DG. Cyanide. J Toxicol Clin Toxicol. 2008;46(2):125–39.
25. Baskin SI, Hayes AW. Mechanisms of cyanide toxicity and treatment perspectives. Adv Exp Med Biol. 2016;952:365–74.