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A REVIEW ON INGESTED CYANIDE: RISKS, CLINICAL PRESENTATION, DIAGNOSTICS, AND TREATMENT CHALLENGES

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Abstract
Ingestion is the predominant mode of exposure for cyanide, a metabolic toxin that is becoming a growing chemical hazard. Attacks on US soil as well as global food and water resources have been threatened by terrorist groups. Because of the special toxic kinetics and hazardous dynamics of oral cyanide, high dose exposures, severe symptoms, and a delayed onset of symptoms are typical outcomes. Many suicidal and homicidal deaths are caused by cyanide, one of the poisons with the fastest rate of action. Cyanide can be released by burning some natural materials, such wool and silk. In addition to addressing the dearth of quick diagnostics and effective therapies for mass casualty incidents, this review aims to assess the dangers associated with oral cyanide and its distinct hazardous kinetics. We will also go over the methods used currently to create novel treatments. Papers pertaining to risk, clinical presentation, diagnosis, existing therapy, and novel therapeutic approaches were cited in this study. In a mass casualty incident, first responders require new treatments designed for oral cyanide exposures that are safe, simple to administer, and expedient. In order to find an antidote that is quick to take effect, safe, efficient, and easy to administer, research is now underway.

Keywords: Cyanide, toxic kinetics, toxic dynamics antidote.

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Introduction

Cyanide is one of the most feared chemicals because it is an efficient and lethal poison. The lethal capacity of cyanide has been exploited for mass murder or terrorism in several infamous cases [1]. Cyanide poisoning, whether it be accidental or intentional, remains a major threat to civilians and military personnel worldwide. It is readily available, highly lethal, and easily weaponized. Oral cyanide in particular is the largest threat compared to other routes of exposure, with potassium cyanide (KCN) and sodium cyanide (NaCN) being the most frequently ingested cyanide salt (1). According to a study reporting data from the National Forensic Service headquarters in Seoul, Korea, there were 255 cyanide poisoning deaths reported from 2005 to 2010, the majority from self-harm. Cyanide remains on the list of potential terrorist threats by various US governmental agencies. One of the most well- known incidents of a large-scale oral cyanide poisoning was the Jonestown massacre, which resulted in more than 900 deaths after drinking cyanide-laced Flavor-Aid (2).

Cyanide is a chemical compound that contains a cyano-group, consists of a carbon atom triple bonded to a nitrogen atom, in combination with other elements such as potassium or hydrogen. It is a rapidly acting, lethal poison that interfere with mitochondrial oxygen utilization (3). Cyanide used as a method of execution since ancient times; and with the media news about its repetitive use as a suicide agent last year in Turkey, attention to cyanide poisoning was increased. This paper aims to appraise the evidence base for the clinical management of cyanide toxicity.

Etiology

Cyanide has many natural, industrial and even household sources. Most common cause of cyanide poisoning in Western countries is smoke inhalation. Cyanide is formed during the incomplete combustion of nitrogen-containing materials, such as wool, silk, and melamine which are increasingly being used in homes(4). Therefore, inhalation of toxic fumes in domestic fires can be the cause of cyanide toxicity among fire victims.

Pathophysiology

Cyanide causes rapid toxicity upon exposure, in large

part due to the inhibition of cytochrome-c oxidase-dependent cellular respiration. It avidly binds to the ferric ion (Fe3+) of cytochrome oxidase a3 and inhibits this final enzyme in the mitochondrial cytochrome complex. Cells cannot utilize oxygen because of their poisoned electron transport chain; and as a result, cellular pseudohypoxia and acidosis occur. Also, a small amount of cyanide may bind to the ferrous (Fe2+) iron of hemoglobin, to form cyanohemoglobin, which is unable to transport oxygen, thereby further exacerbating tissue hypoxia (5).

Cyanide is rapidly absorbed through the respiratory tract and mucous membranes, and it can also be absorbed through the gastrointestinal tract and skin. Intravenous and inhaled cyanide exposures produce a more rapid onset of signs and symptoms than oral or transdermal ingestions because they provide fast diffusion and direct distribution to target organs via the bloodstream (6).

Clinical Presentation

The clinical presentation of cyanide poisoning varies with the physicochemical form of cyanide, the dose, route of entry, co-toxicants (carbon monoxide, etc.), and delay since exposure. Because of being more sensitive to hypoxia, central nervous system and cardiovascular system dysfunction are most prominent. Nonspecific signs of cyanide poisoning are nausea, vomiting, headache, dizziness, confusion, coma, seizures, dilated pupils, and abnormal vital signs. Cyanide toxicity is expected to have two characteristic symptoms theoretically; those are cherry-red skin and the odor of bitter almonds from the victims' breath (7).

Survivors of severe cyanide toxicity may develop neurologic sequelae including Parkinsonism, dystonia, and dyskinesia, reflective of the involvement of the structures within the basal ganglia. Neuroimaging may demonstrate radiologic changes several weeks after the exposure. The effects of acute cyanide poisoning on brain structure and function are unpredictable. The resolution of symptoms is variable, and treatment is supportive.

Chemical Analysis

Methods currently available for the isolation of cyanide from biological materials are the microdiffusion methods and distillation methods. These methods use a modification of the colorimetric procedures developed by Aldridge and Epstein (8) for the determination of the isolated cyanide. One disadvantage of the Aldridge method is that benzidine, a reagent used in the colorimetric procedure, is carcinogenic. The pyridine-pyrazolone reagent used in the Epstein method is unstable, and pyridine is noxious and unpleasant in routine work. Two fluorometric procedures, one by Morgan et al and the other by Groff et al, have been used to measure cyanide isolated from biological fluids. A gas chromatographic procedure for the determination of cyanide in biological specimens based on its conversion to cyanogen chloride has also been reported (9).

Carbon monoxide poisoning: a more complex disease than previously recognized:

Automobile exhausts

Petrol is a complex mixture. Its combustion is of major environmental concern due to the production of large amounts of, not only carbon dioxide and carbon monoxide but also of

nitric oxide and organic volatile compounds. Chronic exposure to automobile exhausts is considered a major concern regarding its effects on the respiratory system. The lone, recognized, toxicant remains carbon monoxide, while the potential for other compounds to cause injury to humans remains ignored.

Symptoms and Signs of Cyanide Ingestion

The signs and symptoms of oral cyanide are similar to those of inhaled cyanide; however, the timing and severity differ. Exposure to cyanide via the inhalation route results in symptoms within seconds of exposure, whereas symptoms following ingestion occur in

minutes to hours. Relatively few or no symptoms can occur following consumption of small amounts of cyanide. These low-dose exposures frequently cause headaches, dizziness, mild confusion, abdominal cramping, nausea, and vomiting. Large-dose exposures eventually lead to dyspnea, respiratory depression, apnea, hypotension, arrhythmias, coma, and seizure. These large-dose effects can result in irreversible injury and death within minutes of the onset of symptoms (10).

- During or immediately after exposure to small doses of cyanide, the following signs and symptoms may develop:

1. Chest pain.
2. Chest tightness.
3. Confusion.
4. Dizziness.
5. Eye pain.
6. Eye tearing.
7. Excitement.
8. Difficulty breathing.

Management

The treatment of patients poisoned with cyanide includes supportive care and adjunctive antidotal therapy.

General Treatment

The Treatment of cyanide poisoning includes:

Table 1 Comparison of the signs and symptoms induced by carbon monoxide and cyanide poisonings

Signs and symptoms	CO poisoning	CN poisoning
Headache	+	+
GI disturbances	++	+

Dizziness	++	+
Confusion	++	+
Loss of consciousness	+ (transitory)	+ (sustained)
Seizures	+	+
Coma	+	+
Hypotension	++	++
Cardiac arrhythmias	++	++
Myocardial ischemia	++	++
Lactic acidosis	++ (does not correlate with arterial blood gases or carboxyhemoglobin levels)	++ (correlates with blood gases and cyanide blood gases or carboxyhemoglobin levels)

While the list is similar for both asphyxiants, the analysis of the conditions of occurrence, the delay in onset, the magnitude, and the frequency of signs and symptoms suggest that the toxidromes are significantly different.

1. Supportive treatment adapted to the severity of the poisoning and aimed at reversing organ failure, including coma, seizure, shock, lactic acidosis, and acute respiratory failure.
2. Decontamination depends on the supposed route of absorption.
3. Specific therapy which deals with the administration of antidotes to cyanide.

Oxygen is the foundation of antidotal treatment, and several antidotes are used for the treatment of cyanide poisoning (11). Oxygen-based antidotes may seem illogical, given what is known about the pathophysiology of the intoxication. However, both experimentally and clinically, oxygen therapy is an effective treatment. Normobaric oxygen therapy should be undertaken as soon as possible. Hyperbaric oxygen therapy has also been proposed, but its role remains controversial.

Antidotes

Cyanide is a rapidly lethal toxin and antidotal treatment must take place immediately to be effective. However, patients may benefit from receiving antidotes after some delay. In a case series antidote was administered to 14 consecutive patients beginning a median 2.1 hours (15 min – 5 ½ hour) after cyanide ingestion or inhalation, and 10 patients survived. This confirms that excellent supportive care can gain additional time to treat with antidotes.

Antidotal treatment of cyanide poisoning involves three strategies which are the binding of cyanide, induction of methemoglobinemia, and use of sulfur donors.

Direct cyanide binding: Hydroxocobalamin and dicobalt edetate are in this group.

Hydroxocobalamin

Hydroxocobalamin has been employed as a cyanide antidote since the early 1970s11. It is a vitamin B12 precursor and with its cobalt component in its structure binds to intracellular cyanide with a greater affinity than cytochrome oxidase and forms cyanocobalamin; a stable molecule that is excreted in the urine.

Hydroxocobalamin is available as Cyanokıt® (2.5 g/vial, 2 vials). The recommended initial dose is 5g intravenously over 15 minutes. Depending on the severity of poisoning it may be repeated for a total of 10g. Its pediatric dose is 70mg/kg11. Existed data suggest that hydroxocobalamin is lacking in clinically significant adverse effects; rare adverse effects included dyspnea, facial edema, and urticaria18. It may cause a reddish discoloration of the skin, plasma, urine, and mucous membranes17. Intravenous infusion of hydroxocobalamin has confounding effects on therapeutic measures such as total hemoglobin, carboxyhemoglobin, methemoglobin, and oxyhemoglobin, which makes the assessment of smoke inhalation victims difficult (12).

In general, hydroxocobalamin as first-line antidotal therapy is effective and safe in acute cyanide poisoning.

2. Dicobalt edentate

Dicobalt edetate is a cobalt compound, whose efficacy is based upon the fact that cyanide combines with cobalt to form a relatively non-toxic complex. It is found as Kelocyanor %1,5® (300mg/20ml) ampules at the National Poison Counselling Center. The recommended adult dose is 300 mg over 1 minute, followed immediately by 50 ml of 50% dextrose.

Adverse effects reported have included hypertension, tachycardia, nausea, retrosternal pain, sweating, palpebral, facial, and laryngeal edema, vomiting, urticaria, and/or a feeling of impending doom; which appear particularly when administered in the absence of intoxication'

3. Nitrites

Nitrite-based cyanide antidotes oxidize hemoglobin to methemoglobin which provides an attractive alternative binding site for cyanide, in direct completion with the site on the cytochrome, and it formed a less toxic compound (13).Amyl nitrite was the first cyanide antidote, since 1888. Its use is attractive for first aid because it's inhaled from a crushed capsule, so easy to use; however it only

produces about 7% methomoglobin which is insufficient to bind a lethal dose of cyanide. It should be administered one capsule at a time and held in front of the patient's mouth for 15 seconds,

Followed by rest for 15 seconds, until intravenous access is obtained and sodium nitrite infusion is started. Sodium nitrite administered intravenously with adjustment to maintain methemoglobin levels ≤ 40%2.

4. 4-Dimethylaminophenol

4-Dimethylaminophenol is a potent methomoglobin inducer which is used as a choice of antidote in German and Austria with a recommended intravenous dose of 3, 25 mg/ kg. Adverse effects include reticulocytosis, nephrotoxicity, and hemolysis (14).

5. Sulfur donors: Sodium thiosulfate is in this group.

Sodium thiosulfate applies a sulfur molecule to rhodanese and allows formation of thiocyanate and regeneration of the native enzyme. Thicyanate excreted renally. Its recommended adult dose is 12.5 g by slow intravenous route and pediatric dose is 7 g/m2. Adverse effects include local skin and muscle pain at infusion site, nausea, vomiting, headache and disorientation. Its slow onset of action is a disadvantage for its use as the sole medication in antidotal therapy (15).

In line with the above information, recommendations regarding antidote selection in cyanide poisoning are summarized in Table.

Table 2. Recommendation for antidotal treatment strategies in case of cyanide poisoning for adults

Situation	Recommended antidote regime
In case of probable cyanide intoxication	
• If hydroxocobalamin is available	Hydroxocobalamin (Cyanokit®) 5 g IV over 15 min and Sodium thiosulfate 12.5 g IV over 30 min
• If hydroxocobalamin is not available and if there is no contraindication to nitrites	Amyl nitrite was inhaled for 15 sec, followed by 15-sec rest until sodium nitrite infusion and Sodium nitrite 300 mg IV over 3-5 min and Sodium thiosulfate 12.5 g IV over 30 min
• If hydroxocobalamin is not available and if there is a contraindication to nitrites	Sodium thiosulfate 12.5 g IV over 30 min
• If hydroxocobalamin and	4-Dimethylaminophenol Dicobalt edetate (Kelocyanor %1,5®) 300 mg over 1 min

Types of cyanide antidotes

These are four types of cyanide antidotes methemoglobin inducers (including amyl nitrite, and 4-dimethylaminophenol), sodium thiosulfate, dicobalt EDTA, and hydroxocobalamin. Methemoglobin inducers seem to act primarily by transforming the ferrous iron of hemoglobin into ferric iron. The methemoglobin formed may then compete with cytochrome oxidase for binding of the cyanide ion. To be effective, some investigators have suggested that these agents should induce a methemoglobinemia on the order of 2040%, though experimental data suggested that mechanisms other than methemoglobin induction may be as important in the antidotal action of these agents. Given their side effects, it has been suggested that their use be abandoned, particularly in the setting of cyanide intoxication by smoke inhalation.2 Sodium nitrite is a component of the cyanide antidote kit used in the US.

Sodium thiosulfate is the natural substrate of rhodanese, or sulfur transferase. In the presence of thiosulfate, this enzyme transforms cyanide into the less toxic thiocyanate, which may be eliminated in the urine. Typical dosing of sodium thiosulfate is 12.5 g, administered iv, over 10 min. This is an effective but slow treatment and, thus, cannot be used as a first line cyanide antidote. Sodium thiosulfate may be administered with impunity in suspected intoxication, in association with another antidote. Thiosulfate must not be mixed in the same vial with the hydroxocobalamin antidote. Due to the anti-thyroid activity of thiocyanates, a thyroid function panel should be obtained within several weeks following recovery of the patient.

- **Antidote Development**

Novel antidotes aimed at treating all routes of cyanide exposure, as well as alternate methods of antidote administration including oral, nebulized, sublingual, intramuscular, and subcutaneous are currently being investigated. In a mass casualty scenario of oral cyanide poisoning, it is likely there would be various degrees of toxicity. Triaging victims into various categories of exposure and administering antidotes to treat victims based on the degree of toxicity should occur.

Several agents such as sulfur-transferases, cobinamide, and dimethyl trisulfide (DMTS) are currently under development. Sodium nitrite and sodium thiosulfate administered intramuscularly at low doses can be effective, but the current FDA-approved formulation is not packaged to be used this way. Furthermore, neither of these have been tested against oral cyanide.

Multiple federal agencies in the USA are seeking antidotes for chemical agent threats, including cyanide. Since chemical agents cannot be administered to humans, antidotes must be tested for efficacy in animal models and moved forward for FDA approval through the Animal Rule. Under the Animal Rule, antidotes must be tested in a validated animal model with a similar

pathophysiology and mechanism of toxicity as to that seen in humans.

Several species are used to test cyanide. Mouse models have frequently been used as the first step in evaluating novel cyanide antidotes. Rabbits have also been used and are more amenable than mice for scaling of doses for human administration and are more amenable to hemodynamic monitoring. Rabbit models of oral cyanide with oral antidotes have been reported and have demonstrated oral antidotes can be effective if given early in the clinical course. The swine model has been shown to mirror the toxicodynamics of oral cyanide exposures in humans as evidenced by the development of hypotension, apnea, and hyperlactatemia.

- **Current status of antidote therapy**

Hydroxocobalamin has been used for over 40 years in Europe to treat acute cyanide poisoning and was approved for use in France in 1996. The only cyanide antidote available in the US (the threecomponent Cyanide Antidote kit containing sodium thiosulfate, amyl nitrite, and sodium nitrite) has a safety profile that is not compatible with prehospital use in subjects with suspected cyanide poisoning. As previously described, the sodium nitrite component of the kit can cause severe hypotension, resulting in shock and death, and administration must be monitored in a hospital setting. The nitrite components also induce methemoglobinemia, which can cause a potentially fatal reduction in the oxygen-carrying capacity of the blood. Due to the limitations of current antidote options, hydroxocobalamin is under study for possible approval as a cyanide antidote in the US.

A controversy remairecovered ns regarding the usefulness of cyanide antidotes. A number of outstanding textbooks on toxicology recommend the use of specific antidotes when facing cyanide poisoning. In contrast, in the medical literature, there are a number of reports of significant cyanide poisonings were victims completely with supportive therapy alone.

This controversy outlines the problem in assessing antidote efficacy. The efficiency of an antidote has been assessed under experimental conditions using animals poisoned with cyanide. In practice, first responders at the site of a chemical incident provide basic, as well as advanced, life-support. As stated above, advanced life-support is often needed in human cyanide poisonings. Thus, from a clinical viewpoint, the major endpoint when assessing the usefulness of antidotes is to determine whether they decrease and even obviate the need for incremental supportive therapy. Decreasing the need for incremental supportive therapy should be considered a target goal in the setting of a chemical incident. However, this point has not been addressed.

- **Environmental Assessment**

Cyanide is present in many industrial and municipal wastewaters. The most important source of cyanide pollution is from the effluents of electroplating processes, metallurgy, steel processing, and petroleum industries.

The estimate for the U.S. production of hydrogen cyanide was approximately 700 million pounds in 1976, and industrial production had increased annually in the past decade(16). Both HCN and CN in industrial effluent, as well as natural wastes, are toxic to aquatic life. HCN has been determined to be the most toxic form. The present limit for cyanides in waterways has been primarily based in acute toxicity studies where lethality is the end point. Some

consideration should be directed toward low-level long-term studies on cyanide intoxication in mammals by the oral and inhalation routes(17).

Cyanide is one of the 13 priority pollutants in raw waste and secondary effluent samples where the concentration ranges from 0.004 mg/1 to 0.2 mg/ . Under Federal Drinking Water Standards for cyanide, the recommended maximum concentration is 0.01 mg/ I; 0.2 mg/ I is considered the level of rejection. Therefore, waste solutions containing cyanides must be disposed of in such a way as to meet desirable standards. Continuous automatic monitoring equipment for measuring cyanide in air and water should be encouraged, and these devices should include automatic alarm systems to warn when predetermined levels are exceeded (18).

Conclusion

Cyanide is a deadly xenobiotic. Ingestion can lead to a high body burden of cyanide, severe symptoms, and unique toxicodynamics. 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. The concern of a terrorist attack using cyanide, as well as the gradual awareness of cyanide poisoning in fire victims, has resulted in a renewed interest in both the diagnosis and treatment of cyanide poisoning. The formerly academic presentation of cyanide poisoning must be replaced by more useful knowledge, allowing emergency physicians and rescue workers to strongly suspect cyanide poisoning at the scene. Preliminary data suggests that a cyanide toxidrome can be defined considering signs and symptoms induced by cyanide and carbon monoxide, respectively. A number of experimentally efficient antidotes to cyanide exist whose clinical use has been hampered due to serious side effects. Cyanide poisoning is a rapidly lethal, serious poisoning. Clinical features are

dependent on the route, duration and amount of exposure. Serum lactate levels can be used for confirmation of the diagnosis and predict the severity of poisoning. Supportive care, decontamination and adjunctive antidotal therapy are the main element of management. Recommendation for antidotal treatment strategies in case of cyanide poisoning depends on the availability of antidotes and accuracy of the diagnosis.

Author contributions

All authors are contributed equally.

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