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An Overview of Cyanide Poisoning in Humans and Animals

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ABSTRACT

Cyanide poisoning poses a significant global health risk, affecting both humans and animals due to its rapid and often fatal effects. Cyanide compounds, such as hydrogen cyanide, potassium cyanide, and cyanogenic glycosides found in certain plants, interfere with cellular respiration by blocking cytochrome oxidase, causing cytotoxic hypoxia and organ failure. Human exposure to cyanide is primarily linked to industrial activities such as mining and electroplating, as well as smoke inhalation during fires. Symptoms of poisoning include respiratory distress, dizziness, and, in severe cases, cardiac arrest and death if left untreated. In animals, particularly livestock, poisoning often results from consuming cyanogenic plants such as sorghum and specific grasses. Different species have varying susceptibility to cyanide, with cattle showing signs such as respiratory distress, seizures, and death after ingesting these plants. Detection methods for cyanide, including spectrophotometry, gas chromatography, and ion-selective electrodes, which analyze cyanide levels in blood, urine, or tissues. Treatment generally involves the administration of antidotes, such as hydroxocobalamin, sodium thiosulfate, and sodium nitrite, which neutralize cyanide in the body. The current study aimed to highlight the importance of stringent regulatory measures on cyanide usage in industrial and agricultural contexts to prevent accidental poisoning. Environmental monitoring and processing of safe food are vital to reduce the incidence of cyanide poisoning in both humans and animals.

Keywords: Animal, Antidote, Cyanide, Human, Law, Poisoning

INTRODUCTION

Cyanide poisoning is a significant global health concern that affects both humans and animals. Cyanide, a highly toxic chemical compound, exists in various forms, including hydrogen cyanide, cyanide salts, and cyanogenic glycosides found in certain plants (Parker-Cote et al., 2018). It is widely recognized for its rapid and potentially lethal effects on living organisms by inhibiting cellular respiration, thus halting the production of adenosine triphosphate (ATP), which is essential for cellular energy (Dorooshi et al., 2020). This disruption hinders cells from utilizing oxygen, resulting in rapid organ failure (Dorooshi et al., 2020). The accumulation of cyanide toxins in the body can cause various organ dysfunctions, including dizziness, seizures, loss of consciousness, and arrhythmia (Woolf, 2022). Without timely medical intervention, this condition may lead to death (Abeyasinghe et al., 2011).

In humans, cyanide poisoning often results from accidental or intentional exposure through industrial processes such as mining, electroplating, and chemical manufacturing (Jadav et al., 2022). Additionally, the combustion of certain materials, such as plastics and tobacco, releases hydrogen cyanide gas, which can be inhaled, leading to acute poisoning (Jadav et al., 2022). In some regions, the consumption of foods containing cyanogenic compounds, such as cassava, improperly processed bitter almonds, and apricot seeds, poses a risk of chronic cyanide toxicity, which may lead to neurological disorders (Akyildiz et al., 2010).

Animals are vulnerable to cyanide poisoning, with livestock, wildlife, and pets at risk (Brasel et al., 2006; Oruc et al., 2006; Kennedy et al., 2021). Accidental ingestion of cyanogenic plants, such as sorghum, cherries, and certain grasses, is a common cause of poisoning in animals, particularly grazing livestock, including cattle (Giantin et al., 2024). Wildlife, especially birds and aquatic species may be exposed to cyanide through industrial effluents, such as those released during gold mining, where cyanide is used to extract gold from ore (Eisler and Wiemeyer, 2004; Brasel et al.,

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2006). In pets, there have been reports of dogs ingesting apricots in households, these fruits containing cyanide and dogs developing poisoning (Houlton et al., 2024). This environmental contamination can lead to mass mortality events, significantly impacting biodiversity and ecosystems (Eisler and Wiemeyer, 2004).

There are similar clinical signs of cyanide poisoning in both humans and animals which include difficulty breathing, confusion, dizziness, and cardiac arrest (Sabourin et al., 2016; Woolf, 2022). In severe cases, death may occur within minutes to hours if left untreated so, rapid diagnosis and treatment are critical (Giantin et al., 2024). Preventing cyanide poisoning requires stringent regulation of industrial activities, proper food processing, and comprehensive environmental monitoring (Saingam, 2018). The present study aimed to highlight key aspects of cyanide poisoning to enhance understanding of its impact and the necessity for effective prevention and treatment strategies.

Characteristics, properties, and sources of cyanide

Cyanide is a highly toxic substance composed of carbon and nitrogen atoms, commonly found in compounds, such as hydrogen cyanide (HCN), potassium cyanide (KCN), and sodium cyanide (NaCN) (Dorooshi et al., 2020). Cyanide compounds can exist in various physical states, depending on their chemical form and environmental conditions. HCN is a colorless, highly poisonous gas with a faint, bitter almond-like odor, while KCN and NaCN are typically found in solid form, appearing as white crystalline powders or pellets (Parker-Cote et al., 2018). Cyanides are present in a range of sources, including industrial processes, including mining, electroplating, the production of synthetic fibers, pesticides, and fumigation (Jaszczak et al., 2017). Natural sources include plants that produce cyanogenic compounds known as cyanogenic glycosides, such as cassava, stone fruits, and bamboo shoots, which release hydrogen cyanide (HCN) when plant tissue is damaged (Jaszczak et al., 2017). In 2024, reports from certain regions indicated a preference for cultivating Sorghum species due to their favorable growth, particularly in the Northwest of Italy. However, an outbreak of cyanide poisoning was observed in cattle that consumed Sorghum in these areas (Giantin et al., 2024). Eisler and Wiemeyer (2004) discussed the impact of cyanide poisoning on fish, reptiles, and amphibians, resulting from the contamination of rivers due to gold mining activities.

Mechanisms and dosages of cyanide poisoning

Cyanide poisoning exerts its lethal effects primarily by inhibiting cytochrome oxidase in the mitochondrial electron transport chain, thereby obstructing cellular respiration (Dorooshi et al., 2020). The mechanism of this inhibition involves cyanide blocking the activity of an enzyme called cytochrome c oxidase (complex IV), a key enzyme in mitochondrial respiration (Dorooshi et al., 2020). This enzyme is crucial for the production of energy in the form of ATP (adenosine triphosphate) through oxidative phosphorylation (Dorooshi et al., 2020). The inhibition of cytochrome c oxidase prevents cells from utilizing oxygen, leading to cytotoxic hypoxia despite adequate oxygen levels in the blood (Woolf, 2022). The resulting energy crisis forces cells to switch to anaerobic metabolism, which leads to lactic acid accumulation and metabolic acidosis (Yadukul et al., 2014). The depletion of cellular energy subsequently causes widespread cellular damage, organ failure, and potentially death (Dorooshi et al., 2020). The toxicity level of cyanide is influenced by its specific form and route of exposure (Jadav et al., 2022; Woolf, 2022). In humans, previous research has established that the lethal dose fifty (LD_{50}) for KCN ingestion is 140 mg, while the lethal dose hundred (LD_{100}) ranges from 200 to 300 mg (Wolnik et al., 1984; Woolf, 2022). In animals, cyanide poisoning in swine has been documented, with the lethal dose of HCN by inhalation reported to be 2.21 mg/kg or 5893 mg/minute/m³ (Staugler et al., 2018). The specific amounts of cyanide toxins that cause toxicity in other species are shown in Table 1.

Table 1. Dosage of cyanide poisoning in different species

Species	Types of cyanide	Dosage	Clinical signs/ Disorder	References
Human	KCN	140-300 mg/person (oral)	Cardiac arrest, seizures, cyanosis, and cherry-red skin coloration	Woolf (2022)
Swine	HCN	2.21 mg/kg or 5893 mg/minute/m³ (inhalation)	Asphyxia, cardiorespiratory arrest, and death	Staugler et al. (2018)
Mice	KCN	9.9-11.8 mg/kg (oral)	Difficulty breathing, convulsions, tremors, seizures, and death	Sabourin et al. (2016)
Fish	NaCN	5-20 µg/l (bathe)	Impact on swimming disrupted respiration, and death	Ramzy (2014)
Coyote	NaCN	2.1-8.3 mg/kg (oral)	Respiratory failure and death	Sterner (1979)
Dog	HCN	2-2.5 mg/kg (oral)	Vomiting, lethargy, ataxia, and unresponsiveness	Nagy et al. (2023); Houlton et al. (2024)
Cat	HCN	2-2.5 mg/kg (oral)	Hypersalivation, vomiting, and diarrhea	Caloni et al. (2013); Nagy et al. (2023)
Cattle	HCN	2 mg/kg (oral)	Hyperventilation, diarrhea, convulsions, shock, and death	Gensa (2019); Kennedy et al. (2021)
Avian	NaCN	4-21 mg/kg (oral)	Lethargy and death	Wiemeyer et al. (1986); Brasel et al. (2006)

KCN: Potassium cyanide, HCN: Hydrogen cyanide, NaCN: Sodium cyanide

Sample collection and methods for detecting cyanide poisoning

Due to the non-specific clinical manifestations observed in animals or humans exposed to cyanide, laboratory testing is necessary to confirm the presence of the toxin (Oruc et al., 2006). Cyanide toxicity can be detected through various analytical techniques, depending on the sample type and method employed. Samples should be collected carefully to avoid contamination with hazardous chemicals. Blood samples are most frequently utilized and should be collected in tubes containing anticoagulants such as heparin or ethylenediaminetetraacetic acid (Kennedy et al., 2021). In some cases, urine samples may also be used for analysis of cyanide (McGorum and Anderson, 2002). Internal organs, including stomach contents, liver, and kidneys, should be tested to confirm poisoning (Jadav et al., 2022). Clothing suspected of toxin contamination should also be examined (Jadav et al., 2022). Methods for cyanide detection include the picrate paper test, which is a qualitative method that utilizes picric acid to observe a color change (Oruc et al., 2006). The Prussian Blue Test is another qualitative analysis method for detecting cyanide concentration in a solution, involving the reaction of ferric compounds to form a colored complex (Oruc et al., 2006). Other techniques that have been developed and used include spectrophotometry, which assesses cyanide concentration by measuring the color change in a solution reacting with cyanide (Nnoli et al., 2013); gas chromatography (GC), which separates and quantifies cyanide using a GC instrument (McGorum and Anderson, 2002); high-performance liquid chromatography (HPLC), which employs liquid-phase separation to determine cyanide levels (Tobarran et al., 2022); and ion-selective electrode (ISE) techniques, which detect cyanide ions in samples via a specialized electrode (Sankaran et al., 2020). Liquid Chromatography-Tandem Mass Spectrometry (LC-MS/MS) is a highly accurate quantitative method for detecting cyanide, capable of identifying trace amounts and simultaneously analyzing multiple compounds (Giantin et al., 2024). Liquid Chromatography-Fluorimetry (LC-Fluorimetry) combines liquid chromatography with fluorimetric detection to separate and measure cyanide and is sensitive enough to detect even trace amounts of cyanide (Kennedy et al., 2021).

Cases of accidental exposure to or misuse of cyanide in different species

Cyanide poisoning has been documented in both humans and animals across numerous countries, with incidents in animals often resulting from accidental exposure. For example, a study by Giantin et al. (2024) linked cattle fatalities to the consumption of sorghum. The affected cattle exhibited clinical signs such as severe respiratory distress, recumbency, and seizures. Necropsy findings revealed typical indicators of cyanide poisoning, including bright red blood, an almond-like odor, and emphysema in the lungs. Further analysis of sorghum samples confirmed cyanide levels exceeding the safety threshold (Giantin et al., 2024). Similarly, in 2002, a case of cyanogenic poisoning in horses was attributed to the ingestion of white clover, with the animals displaying signs of ataxia and neuropathy (McGorum and Anderson, 2002). Another case reported in Turkey in 2006 involved canine deaths due to cyanide poisoning (Oruc et al., 2006). The affected dogs exhibited symptoms such as dyspnea, excitability, and convulsions before death. Postmortem examinations confirmed the diagnosis, with pathological findings showing severe inflammation and hemorrhage in the blood vessels, lungs, and heart (Oruc et al., 2006). Additionally, there have been reports of hawk fatalities, with carcasses found near a water source adjacent to a gold mine in Nevada (Franson, 2017). Postmortem examinations revealed cyanide accumulation in the blood, liver, and brain, which is suspected to have originated from contamination linked to gold mining activities (Franson, 2017).

Cyanide intoxication in humans is frequently associated with accidental exposure, homicide, and suicide. In 2022, an accidental death from cyanide inhalation in an industrial setting was reported in India (Jadav et al., 2022). The incident occurred in an electroplating chamber, and autopsy findings along with toxicological analyses confirmed cyanide poisoning as the cause of death (Jadav et al., 2022). Another notable case occurred in 2012, where an individual who consumed beer at a bar was later found died due to cyanide poisoning (Nnoli et al., 2013). Initially, the death was attributed to myocardial infarction, but further investigation revealed cyanide as the underlying factor. Nnoli et al. (2013) conducted a comprehensive examination, detecting cyanide in the victim's stomach contents, blood, bile fluid, and a mouth swab, thereby confirming cyanide poisoning as the cause of death. This case highlights the risk of cyanide contamination in food or beverages, whether deliberate or accidental, which can lead to fatal consequences (Nnoli et al., 2013). Additionally, in 2011, a suspected homicide involving cyanide injection was reported in Sri Lanka. The victim exhibited signs of rapid breathing before death, and subsequent analysis of blood samples and the contents of a syringe confirmed the presence of cyanide (Abeyasinghe et al., 2011). Further details are shown in Table 2.

Guidelines for the treatment of cyanide toxicity in humans and animals

In cases of cyanide exposure in humans or animals, the initial step involves determining the likely route of toxin entry into the body. Decontamination should be carried out by thoroughly washing the body and removing any clothing that may have been contaminated with cyanide (Jadav et al., 2022). Monitoring and stabilizing vital signs should be prioritized, with immediate management of life-threatening conditions (Reade et al., 2012). Supportive care is crucial in

managing clinical signs such as respiratory distress, seizures, or unconsciousness, and may include hyperbaric oxygen therapy along with comprehensive intensive care measures such as ventilation, circulatory support, and renal replacement therapy (Reade et al., 2012). Hydroxocobalamin has been reported as an effective antidote, binding cyanide ions to form cyanocobalamin, a less toxic compound that is subsequently excreted in the urine (Kiernan et al., 2020). Additionally, sodium nitrite is used to induce the formation of methemoglobin by oxidizing Fe^{2+} in hemoglobin to Fe^{3+} (Johnson-Davis, 2020). Methemoglobin cannot efficiently bind oxygen, but it has a high affinity for cyanide. It acts as a cyanide binder, preventing cyanide from inhibiting the activity of the cytochrome oxidase enzyme, which facilitates the removal of cyanide from the mitochondria (Johnson-Davis, 2020). However, caution is advised, as excessive use can lead to methemoglobinemia, impairing oxygen transport (Johnson-Davis, 2020). Sodium thiosulfate is also used to convert cyanide into thiocyanate through the action of rhodanese, an enzyme composed of various amino acids and antioxidants, in the liver (Avais et al., 2018). Thiocyanate is a less toxic compound that is excreted by the kidneys (Avais et al., 2018). Sodium thiosulfate is often administered in combination with hydroxocobalamin and sodium nitrite)Tobarran et al., 2022, Table 3).

Table 2. The reported cases of cyanide poisoning in different species

Source of cyanide	Method for detection	Samples	Species	References
Sorghum	LC-MS/MS	Sorghum	Cattle	Giantin et al. (2024)
Laurel	LC-Fluorimetry	Blood	Cattle	Kennedy et al. (2021)
Apricot (*Prunus armeniaca*)	The owner is the informant	Gastric lavage	Dog	Houlton et al. (2024)
No reports were identified in the article	Picrate paper and Prussian blue	Contents in the stomach and intestines	Dogs	Oruc et al. (2006)
White clover (*Trifolium repens*)	Gas chromatography	Blood and urine	Horses	McGorum and Anderson (2002)
The water source adjacent to the gold mine	Spectrophotometry	Heart blood, liver, and brain	Bird	Franson (2017)
Beverages or food	Spectrophotometry	Stomach contents, blood, bile fluid, and mouth swab	Human	Nnoli et al. (2013)
Syringe	Benzidine-copper acetate	Blood and content in the syringe	Human	Abeyasinghe et al. (2011)
Electroplating chambers	Toxicology laboratory (Method not indicated)	Blood, internal organs, and clothes	Human	Jadav et al. (2022)

Table 3. The effective agents in the treatment of cyanide toxicity in humans and animals

Agent	Species	Dosage	References
Dobetin® (cyanocobalamin, thiamine hydrochloride)	Cattle	60 ml (The solution contains cyanocobalamin at 1 mg/ml and thiamine hydrochloride at 100 mg/ml)	Giantin et al. (2024)
Sodium thiosulfate	Cattle	250-500 mg/kg	Gensa (2019)
Hydroxocobalamin	Dog	150 mg/kg	Houlton et al. (2024)
Hydroxocobalamin and sodium thiosulfate	Human	5 g and 12.5 g	Tobarran et al. (2022), Meillier and Heller (2015)
Sodium nitrite and sodium thiosulfate	Rabbit	20 mg/kg and 600 mg/kg	Avais et al. (2018)

Legislation related to cyanide in animals and humans

Due to the presence of cyanide in various plant species, several countries have established regulations to limit permissible levels of cyanide in animal feed. Compliance with these regulations varies by region. For instance, in Asian countries, such as Thailand, the Animal Feed Act (2015) stipulates penalties for exceeding allowable cyanide limits, including imprisonment for up to one year, a fine of up to 2,700 USD, or both (Office of the Council of State, 2015). Japan enacted the Pet Food Safety Law in 2008, which imposes penalties for contamination with harmful agents (Sugiura et al., 2009). Violations can result in imprisonment, fines, or other penalties, depending on the court's discretion (Sugiura et al., 2009). In European countries such as the United Kingdom, regulations governing cyanide levels in food include the Animal Feed Regulations 2015 and the Feed Hygiene Regulation No. 183/2005 (Cheli et al., 2013). Violations of

these regulations may result in substantial fines amounting to several thousand pounds or imprisonment, depending on the court's discretion (Cheli et al., 2013).

In industries where cyanide chemicals are used, various countries have established regulations to control the storage and use of these substances to prevent misuse. In Thailand, the Chemical Substance Control Act of 2012 stipulates penalties for violations, including imprisonment for up to 5 years or fines exceeding 13,500 USD, depending on the nature of the infraction (Saingam, 2018). In South Korea, the Chemical Substance Control Act of 1987 mandates penalties for breaches, which may include imprisonment for up to 5 years or more, and fines potentially exceeding several million dollars (Yoon et al., 2014). In the United States, the Toxic Substances Control Act (TSCA) of 1976 regulates the production and use of hazardous chemicals. Violations of the TSCA may result in imprisonment for up to 5 years or more, and fines reaching up to $50,000 per day, depending on the court's discretion (Ozymy and Ozymy, 2023).

CONCLUSION

Cyanide poisoning remains a significant concern in both medical and veterinary fields due to its rapid onset and high lethality. The widespread presence of cyanide in industrial processes, and natural environments, and its potential use in toxicological crimes underscores the need for stringent safety measures, timely detection, and effective treatment protocols. Understanding the mechanisms, sources, and treatment options for cyanide poisoning is essential for reducing its impact on both human and animal populations. Regulatory enforcement and public awareness are crucial for mitigating the risks associated with cyanide exposure. Future studies should focus on developing methods for the rapid and accurate detection of cyanide in biological and environmental samples, as well as enhancing novel antidotes to minimize side effects.

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All authors have checked for plagiarism, fabrication and/or falsification, dual publication and/or submission, and redundancy.

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