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Cyanide- A Silent Killer in Sudden and Unexplained Death

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Chapter 1 : Introduction

Cyanide is a highly potent and rapidly acting poison that poses lethal risks in various contexts, making it a significant concern in cases of sudden and unexplained death. Cyanide (CN-) is known as a molecular compound for its interaction with cytochrome c oxidase, a critical enzyme in cellular respiration. When cyanide binds to this enzyme, it effectively halts aerobic metabolism, leading to the rapid development of severe lactic acidosis and profound shock. Cyanide is a naturally occurring substance that smells like almonds. A chemical molecule known as cyanide is made up of a cyanide group, which is made up of a carbon atom triple-bonded to a nitrogen atom, along with additional components like hydrogen or potassium. This toxin interferes with mitochondrial oxygen use and acts quickly. its frequent use as a suicide agent. Because cyanide inhibits cytochrome-c oxidase-dependent cellular respiration, it becomes lethal quickly after exposure. A naturally occurring substance, cyanide smells like almonds. This innocuous substance can be found in various fruits and plants, such as lima beans, cassava plants, apricots, apples, and peaches. Because cyanide is highly reactive and quickly degraded to produce other chemicals, trace levels of it present in organic materials are not a major problem. High temperatures, however, can cause a significantly higher concentration of hydrogen cyanide to be released from contemporary synthetic materials like plastics, papers, fabrics, and equipment. Since modern synthetic polymers containing nitrogen have become more widely used, the risk of cyanide toxicity has increased in frequency and severity. Because the gaseous form of hydrogen cyanide reacts rapidly when inhaled, it poses a particular risk to people (G Doman, et al.,2022).

Cyanide can be distinguished in two form:

1. Inorganic -it contains potassium cyanide (KCN)

2. Organic cyanide – the CN group is joined to a carbon atom in inorganic molecules to form organic cyanides, also known as nitriles

Different forms of cyanide exist, each having unique properties and applications

(A) Solid form – inorganic salts of cyanides, such substance potassium cyanide (KCN) and sodium cyanide, are crystalline solids.

(B) Liquid form – Liquid form, like acetonitrile (CH3CN), is frequently employed as a solvent in both industry and laboratories. As compared to inorganic cyanides, they are less poisonous. Hydrogen cyanide is a liquid cyanide that is volatile and readily evaporates, becoming a gas

(A) Gaseous cyanide – a white gas that smells slightly of almonds.

(B) Cyanides can be found in nature – it can be obtained from plants and seed such as apple seeds, bitter almonds and cherry pits that contain cyanogenic glycosides. These are organic form which are found in plant materials as solid or powders, emit cyanide upon metabolism.

(C) Complex cyanide – ferrocyanides and ferricyanides are examples of complex compounds that can be formed by cyanide and metals

All around the worlds cyanide is readily available and extensively accessible.(S Giantin, frazin , F Brusa, V Montemurro… - Animals, 2024) The compound is not readily seen cyanide poisoning can be categorized according to its severity and mode of occurrence.

1. Acute poisoning

It can also be called short-term cyanide poisoning, it occurs when a lot of cyanide is quickly absorbed, swallowed, or inhaled. The rapid onset of symptoms includes:

a. Breathing problems
b. Absence of consciousness
c. A seizure
d. Heart arrest

If acute poisoning is not treated right away, it can often be fatal.

2. chronic poisoning

It is also called as long-term poisoning, this happens when small amounts of cyanide are exposed over an extended period. Consumption of foods containing cyanide or industrial environments might cause it the following symptoms are

a. weakness
b. the headache
c. discomfort
d. brain problems

3. poisoning by cyanide at work or occupational cyanide poisoning

This includes those who work in mining, chemical manufacturing, or electroplating.

4. poisoning by environmental cyanide or environmental cyanide poisoning

Contaminated water, soil or air can expose people, frequently as a result of industrial spills or inappropriate disposal of material that contains cyanide. when cyanide is consumed. It effects severely spread throughout the body. Quickly target cytochrome oxidase, a vital enzyme found in our cells, mitochondria, once they enter the bloodstream. the enzyme is essential to the electron transport chain, the metabolic process that produces adenosine triphosphate (ATP), the main source of energy for all cellular processes. Because its strong binding and high affinity for the iron atom in cytochrome c oxidase, cyanide significantly inhibits the activity of the enzyme. This obstruction stops cells from using the oxygen that the blood provides by interfering with the last phase of aerobic respiration.(RJ Moses , GI Edo , AN Jikah , OL Emarkpor , JJ Agbo – vegetos,2024) The main ways that cyanide poisoning happens are by inhaling smoke from burning synthetic materials, consuming cyanide salts or eating specific seeds or plants that contain cyanogenic chemicals, it can be brought on by skin absorption or medication use (sodium nitroprusside).

Liquid cyanide solution can enter the body through the skin or be eaten. When the body breaks down cyanogenic substances found in some seeds (Appleseeds, Apricot kernels), cassava, and other plants . cyanide is released. The rate of cyanide poisoning development is influenced by the entry point, symptoms usually appear most quickly after inhalation. Solid cyanides, such as sodium cyanide and potassium cyanide, typically appears as a white crystalline or granular powder.

Cyanide is one of the oldest poison, large dose of cyanide prevent cells from using oxygen and eventually these cell die . the heart , respiratory system and central nervous system are most susceptible to cyanide. Cyanide directly stimulates

the chemo- receptors of the carotid and aortic bodies that results in hypernoea the central origin of death is due to respiratory arrest. hydrocyanide acid (HCN), sodium cyanide (Na CN) and potassium cyanide (KCN) or cyanogen are very potent, extremely lethal and most rapidly fatal. poisoning with hydrocyanic acid is acid is always fatal because of the low fatal dose and the rapidity with which it acts.

Cyanide is found in manufacturing and industrial sources such as insecticides, photographic solutions and jewelry cleaners cyanide exist in gas, liquid and solid forms .it can cause human toxicity via multiple routes including inhalation, ingestion. (Sumana Sen, 2023)

SIGNS AND SYMPTOMS

When inhalation as a gas, its action occurs within seconds. Massive doses may produce sudden loss of consciousness and prompt death from respiratory arrest. After ingestion, symptoms appear within minutes, during which the victim may perform certain volume acts, such as corking or throwing away the bottle or walking a little distance.

Gastrointestinal tract

The features of GIT involvement occurs, after the ingestion of cyanides and include a burning taste, throat numbness, salivation, frothing at a mouth, nausea, vomiting and substernal and epigastric.

Central nervous system leads to dizziness, headache, sweating, anxiety, confusion, drowsiness, syncope, opisthotonus, seizures, coma and death.

Respiratory system

Tachypnea and dyspnea develop due to the stimulation of respiratory center and carotid chemo receptors caused by local hypoxia bradypnoea and irregular respiration, pulmonary oedema, cyanosis and respiratory arrest in the later stage. a bitter almond like odor may be detected in the breath.

Cardiovascular system

Hypertension along with reflex bradycardia. This is followed by hypotension, tachycardia, arrhythmias, etc. the venous oxygen tension approaches that of arterial oxygen tension and, therefore the venous blood in the initial stage is bright red.

Lactic acidosis It develops in the later stages as cyanide inhibits mitochondrial cytochrome oxidase, thereby blocking electron transport and preventing oxygen utilization and oxidative metabolism. lactic acidosis occurs as a consequences of anaerobic metabolism. (Raghavendrarao, Venkata morusupalli,2023)

Chapter 2: Literature Review

1 In the year 2020, Jorn Chi-Chung Yu, Asraf Mozayani state that for cyanide poisoning cases, the presence of cyanide at the scene of crime and the source of cyanide should be investigated during the search. In their paper, they discuss the medicolegal aspects of cyanide in forensic factors of cyanide poisoning. They are concluding that after death, when the body starts decomposing, no signs of cyanide poisoning should be confirmed. Even in liver mortis and scent shall not be included solely to include or exclude in case of poison. The observation of Gastric burns shows blood cherry red color, which confirms that the death of a person is due to the acute CN poisoning. The forensic approach for this case is based on lab testing, as not all samples were collected and tested in labs. As each case is unique in its own right.

2 In the year 2022, a case study was discussed on the finding of cyanide by the microdiffusion technique, where the case report was made. In Italy, Rome Hotel, a man was found dead with holding syringe attached to a butterfly needle inserted in his left forearm. The syringe contains cloudy pinkish fluid. When police stars investigating found a broken propofol glass vial plus sealed ones, a NaCl opened plastic vial, and six more sealed and packed with smaller disposable syringes and needles. And open white crystalline powder labelled as

Potassium Cyanide. When a forensic team comes to collect visceral fluids such as bile, blood, and urine after analysis, they detect propofol in blood and bile samples in techniques GC/MS–TOF method to confirmation of propofol in biological fluids.

3 A research has been conducted by Erica Manandar ,2020 Cyanide poisoning due to accidental or deliberate exposure is a serious health hazard. The present FDA-approved cyanide antidotes for cyanide poisoning are effective, but each of them has some major drawbacks. Dimethyl trisulfide (DMTS), a sulfur donor that detoxifies cyanide by reducing it to thiocyanate, is a candidate next-generation cyanide antidote. While a validated analytical procedure to analyze DMTS is not yet available from any matrix, one will be crucial for the approval of DMTS as a therapeutic drug against cyanide poisoning. The limit of detection (LOD) with this technique was 0.06 µM, and the dynamic range was 0.5 – 100 µM. The technique described herein provides room for further research on DMTS as a potential antidote for cyanide poisoning

4 A case series of cyanide poisoning by cemyigit deveci, Mehmet atilgan , a case study was discussed on the cyanide is used in industrial processes for gold extraction ,metal refinery or in organic synthesis and other chemical processes . it can be detected in nature ,in edibles such as apple or apricot seeda and bitter almond , these substance are very well known to be extremely toxic to human it presents a cases of a murder suicide incident. A family of four was found dead in their home with a suicide note left by the father revealed that he has poisoned his family and subsequently committed suicide due to his unemployment and financial problems .

5 It is a case series of Refik Akman,2022this is a case of 30 year 30-year-old man found dead in front of his car with a note on the windshield warning people about the presence of cyanide. the man also had a 3-suicide and a small cup with a red cap that is labeled as cyanide.

6 A case study of Kamisha L. johnson davis, 2020 A case of cyanide poisoning secondary to inhalational cyanide smoke exposure is discussed. In the case of cyanide poisoning here the patient got exposed to domestic fire smoke and developed cyanide poisoning. The patient was managed with amyl nitrite, sodium nitrite and sodium thiosulfate, and hyperbaric oxygen. Sources of exposure, toxicokinetics, treatment, and laboratory evaluation of cyanide have been explained.

7 A research has been conducted by Gulshan cikriksi , yunsur cevik ,2020 Cyanide is a rapidly acting, lethal poison that interfere with mitochondrial oxygen utilization.Clinical presentations are a function of route, duration and dosage of exposure. Serum lactate concentration may be employed for the confirmation of diagnosis as well as to anticipate the severity of poisoning. Supportive treatment, decontamination, and adjunct antidotal therapy comprise the key features of management. Antidotal treatment regimen is recommended based upon the availability of antidotes as well as the specificity of the diagnosis.

8 A research has been conducted by Espinosa (2020 Cyanide) it is an easily accessible chemical in the market utilized in different like manufactured of insecticides , photography and jewelry. Poisoning cases from this compound have a high rate of mortality, particularly those in which the antidotes are not given promptly.

9 The case report of zhipeng cao state that Acetone cyanohydrin (ACH), an organic cyanide, is mainly used in the production of methyl methacrylate (MMA).ACH can decompose spontaneously or enzymatically into acetone and highly toxic hydrogen cyanide (HCN) and be potentially toxic to its contacts. ACH poisoning lacks specific and reliable autopsy findings for diagnosis, and relevant toxicological studies are necessary. Due to the chemical properties of ACH that allow it to easily decompose.

10 A research has been conducted by Gerry R boss Cyanide, a metabolic toxin, is an emerging chemial risk and ingestion is the most frequent method of exposure.The purpose of this review is

to assess the risks of oral cyanide and its distinctive toxicokinetics, and to address the insufficient available rapid diagnostics and treatments for mass casualty incidents.

Chapter 3: Problem Identification

Its fast toxicity, variety of exposure sources, and difficulties in prompt identification and treatment make cyanide poisoning a major global health concern. Early detection of cyanide poisoning is still challenging despite advances in medical toxicology because symptoms can be mistaken for other illnesses, causing cases to be misdiagnosed or delayed.

Cyanide is a fast reacting poison after intake of this a person may have to face severe health problems , and it effects the vital organs of the body may may lead death within 10 minutes .

Chapter 4: Methodology

Various research papers were examined and considered. This project was prepared using an inductive technique. This study provides an exploratory view. The foundation of this study is secondary data that has been gathered and taken into account. The provided secondary data was thoroughly analyzed. For the appropriate analysis, the data is collected by reviewing numerous research and review publications.

Cyanide, particularly its rapid absorption through inhalation, ingestion, and percutaneous routes, and its strong inhibition of cytochrome c oxidase in the mitochondrial electron transport system, leading to cellular hypoxia. A broad overview should address the analytical strategies utilized to detect cyanide in biological matrices (blood, urine, tissues) and environmental samples (air, water), such as spectrophotometry and gas chromatography-mass spectrometry (GC-MS).

Chapter 5: Result and discussion

After consumption of cyanide poisoning our body gives various symptoms according to their dose and their toxicity level. severe toxicity may lead to deep coma, dilated non-reactive pupils. to make them easier to understand, these signs and symptoms of cyanide toxicity used to identify them were compiled to a table.

Signs and symptoms of cyanide toxicity		
Mild toxicity	**Moderate toxicity**	**Severe toxicity**
1. Nausea 2. Dizziness 3. Drowsiness	1. Loss of consciousness for a short period 2. Convulsion 3. Vomiting 4. Cyanosis	1. deep coma 2. Dilated non-reactive pupils 3. deteriorating cardio-respiratory function

Table No 1(This table shows the signs and symptoms of the toxicity level that affects our body.)

The signs of cyanide poisoning and possible color changes in the skin over time. The development and severity of the signs can be extremely variable based on the dose, route of exposure (inhalation, ingestion, dermal), and the general health status of the individual .

Here are some important considerations-

1.Bitter Almond Odor: There can be a characteristic bitter almond odor on the breath, but about 40% of individuals are unable to smell this odor because it is something that is genetically determined.
2.Cyanosis: Though most commonly linked to oxygen deficiency, cyanosis is not a notable initial presentation of acute cyanide poisoning, as the venous blood is arterialized. Instead, it is more likely to occur with prolonged respiratory arrest or shock.
3.Red Cherry Skin: This traditional sign is not always present and can be obscured by other causes such as inadequate circulation or shock.
4.Time Sensitivity: Cyanide poisoning, particularly in acute form, is an emergency situation. The development of symptoms can be quick and lethal.

Time after exposure	Common symptoms	Potential skin color changes
Seconds to minutes (high dose /inhalation)	Rapid onset : weakness ,dizziness ,headache ,confusion ,nausea, vomiting ,shortness of breath,rapid breathing ,rapid heart rate ,anxiety , loss of consciousness ,cardiac arrest .	Cherry red skin – due to inability of tissues to extract oxygen from the blood , leading to higher oxygen content in venous blood .
Minutes to hours (lower dose /ingestion / dermal)	Slower onset : headache , dizziness , vertigo , confusion ,weakness , nausea , vomiting ,abdominal pain , shortness of breath ,chest pain or tightness,rapid or slow heart rate , anxiety ,muscles tremors ,loss of consciousness.	Initially ,skin may appear normal or flushed . as hypoxia progresses (bluish colour of the skin ,lips and nail beds)may occur due to inadequate oxygen delivery .
Chronic exposure (low levels over time)	Gradual onset : headache , dwowsiness , nausea , vomiting ,altered taste and smell , anxiety ,weakness , potential for neurological issue	Bright red flush – may be early symptom . if the chronic exposure leads to significant hypoxia.

Table no -2 symptoms come in the body and change in skin colour according to time.

CHAPTER 6

CONCLUSION AND SCOPE OF FURTHER WORK

Chapter 6: Conclusion and Scope of Further Work

The goals of research into cyanide poisoning are examine how cyanide suppresses oxidative phosphorylation and impacts cellular metabolism. Create dependable and quick techniques for cyanide detection in biological samples.

this review has demonstrated that although we understand a great deal about how cyanide kills the body by preventing cells from utilizing oxygen and how we can identify it, there are still considerable challenges.

we need to develop better means of researching the effects of cyanide on people. Maybe using more sophisticated laboratory-cultivated tissues or computer simulations. Secondly, we need to know more about the long-term health issues that could be caused by small exposures to cyanide. Thirdly, the creation of faster and simpler diagnostic tests for cyanide poisoning, particularly in urgent cases, is vital. We must also consider more tailored treatments since individuals respond differently.

Chapter 7: References

1. George, D. J. (2017). *Poisons: An Introduction for Forensic Investigators*. CRC Press.
2. **Medicolegal and forensic factors in cyanide poisoning Jorn Chi-Chung Yu**, AshrafMozayani https://onlinelibrary.wiley.com/doi/10.1002/9781118628966.ch20
3. M. Behymer, Huaping Mo, Naoaki Fujii, Vallabh Suresh, Adriano Chan, Jangweon Lee, Anjali K. Nath, Kusumika Saha, Sari B. Mahon, Matthew Brenner, Calum A. MacRae, Randall Peterson, Gerry R. Boss, Gregory T. Knipp, Vincent Jo Davisson, Identification of Platinum(II) Sulfide Complexes Suitable as Intramuscular Cyanide Countermeasures, Chemical Research in Toxicology, 10.1021/acs.chemrestox.2c00157, **35**,11,(19831996),(2022).doi/abs/10.1002/9781118628966.ch14
4. **Manandhar, Erica, "Analysis of Novel Cyanide Antidote Dimethyl Trisulfide for Pharmacokinetic Studies, and Sulfur Mustard Metabolites for Identification of Biomarker of Inhaled Dose" (2017). Theses and Dissertations. 2166. https://openprairie.sdstate.edu/etd/2166**
5. (Edem, G. D., Ekanem, A. U., Uwah, E., & Ettetor, O. G. A. (2025). Relationship between cyanide poisoning in cassava effluent and epididymis of Albino Wistar rats. *Drug Discovery*, *19*, e1dd2013.
6. Duc, N. D., Sinh, N. P., & Anh, L. N. H. (2024). Clinical Case Report of Acute Heart Injury and Acute Rhabdomyolysis Due to Cyanide Poisoning. *Open Journal of Emergency Medicine*, *12*(2), 29-32.
7. Khan, K., Abdullayev, R., Jillella, G. K., Nair, V. G., Bousily, M., Kar, S., & Gajewicz-Skretna, A. (2025). Decoding cyanide toxicity: Integrating Quantitative Structure-Toxicity Relationships (QSTR) with species sensitivity distributions and q-RASTR modeling. *Ecotoxicology and Environmental Safety*, *291*, 117824.
8. Doman, G., Aoun, J., Truscinski, J., Truscinski, M., & Aouthmany, S. (2022). Cyanide Poisoning. *Journal of Education & Teaching in Emergency Medicine*, *7*(3), S1
9. Mao, Q., Zhao, X., Kiriyama, A., Negi, S., Fukuda, Y., Yoshioka, H., ... & Kitagishi, H. (2023). A synthetic porphyrin as an effective dual antidote against carbon monoxide and cyanide poisoning. *Proceedings of the National Academy of Sciences*, *120*(9), e2209924120
10. Reade, M. C., Davies, S. R., Morley, P. T., Dennett, J., Jacobs, I. C., & Australian Resuscitation Council. (2012). Management of cyanide poisoning. *Emergency Medicine Australasia*, *24*(3), 225-238.
11. Hamel, J. (2011). A review of acute cyanide poisoning with a treatment update. *Critical care nurse*, *31*(1), 72-82.
12. Vick, J. A., & Froehlich, H. (1991). Treatment of cyanide poisoning. *Military medicine*, *156*(7), 330-339

13. .Borron, S. W., & Baud, F. J. (1996). Acute cyanide poisoning: clinical spectrum, diagnosis, and treatment. *Arhiv za higijenu rada i toksikologiju*, *47*(3), 307-321.

14. Reade, M. C., Davies, S. R., Morley, P. T., Dennett, J., Jacobs, I. C., & Australian Resuscitation Council. (2012). Management of cyanide poisoning. *Emergency Medicine Australasia*, *24*(3), 225-238.

15. Bhattacharya, R., & Flora, S. J. (2009). Cyanide toxicity and its treatment. In *Handbook of toxicology of chemical warfare agents* (pp. 255-270). Academic Press.

16. Egekeze, J. O., & Oehme, F. W. (1980). Cyanides and their toxicity: a literature review. *Veterinary Quarterly*, *2*(2), 104-114.

17. Aouthmany, S., Aoun, J., Truscinski, J., & Doman, G. (2022). Cyanide Poisoning Simulation Case. *Journal of Education and Teaching in Emergency Medicine*, *7*(3).

18. Baskin, S. I., Kelly, J. B., Maliner, B. I., Rockwood, G. A., & Zoltani, C. K. (2008). Cyanide poisoning. *Medical aspects of chemical warfare*, *11*, 372-410.

19. Auzepy, P., Veissieres, J.F., and DeParis, M. (1974). Anaphylactic shock due to hydroxocobalamin [French]. Nouv. Presse. Med. 3:152.

20. Hall, A. H., Saiers, J., & Baud, F. (2009). Which cyanide antidote?. *Critical reviews in toxicology*, *39*(7), 541-552.

21. Gracia, R., & Shepherd, G. (2004). Cyanide poisoning and its treatment. *Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy*, *24*(10), 13