Original Research Paper

Volume - 14 | Issue - 07 | July - 2024 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Forensic Medicine

MURDEROUS SUICIDE - A DILEMMA OF FIVE SUSPICIOUS DEATHS (CYANIDE POISONING)

Dr. G. Mary Sowjanya	Assistant Professor, Department Of Forensic Medicine And Toxicology, Kamsrc, Lb, Nagar, Hyderabad
Dr. Raghavendra Rao. M. V*	Professor Of Microbiology, World Academy Of Medical Sciences, Netherlands, Europe *Corresponding Author
Dr. Mahendra Kumar Verma	Senior Resident, Department Of Forensic Medicine And Toxicology, Aiims, Bibinagar, Hyderabad, Ts, India.
Dr. Bashetti Srilatha	Associate Professor, Department Of Biochemistry, Apollo Institute Of Medical Sciences & Research, Hyderabad, Ts, India

ABSTRACT Cyanide is a deadly xenobiotic. Cyanide is one of the earliest, original and primeval poisons. Cyanide prevents cells from using oxygen and eventually these cells die. Death is due to respiratory arrest .Hydrocyanic acid or cyanogen is very potent, extremely lethal and most rapidly fatal. Poisoning with hydrocyanic acid is almost always fatal because of the low fatal dose and the rapidity with which it acts. Cyanide is lethal, inhibits oxidative phosphorylation, a process where oxygen is utilized for the production of essential cellular energy sources in the form of ATP. It does so by binding to the enzyme cytochrome C oxidase and blocks the mitochondrial transport chain. Cyanide poisoning produces cellular hypoxia, depletion of ATP, and leading to metabolic acidosis.

KEYWORDS : Oxidative phosphorylation, Prussic acid, Cytochrome oxidase, Cyanogenic glycosides, Toxic kinetics

INTRODUCTION

Cyanide is a lethal compound, binds to ferric iron in cytochrome oxidase a3, thereby inhibiting oxidative phosphorylation, which leads to the depletion of intracellular adenosine triphosphate (ATP). [1] Cyanide exists in gas, liquid, and solid forms, it can cause human toxicity via multiple routes including inhalation, ingestion, parenteral administration, and dermal or conjunctival contact.[2] Cyanide has been used as a poison in mass homicides and suicides. During World War II, the Nazis used cyanide as an agent of genocide in gas chambers.[3]Cyanide poisoning, whether it be accidental or intentional, remains a major threat to civilians and military personnel worldwide.[4] 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 [5] 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. [6] 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. [7] In 1982, seven people in the USA died after ingesting an over-thecounter medication laced with cyanide. [8] In 2017 terrorists plot to contaminate food supplies with cyanide was foiled and later in that same year, ISIS advised attackers to, Binject food for sale in markets with cyanide poison. [9]Large-scale poisoning of the food supply could lead to mass casualties if we are not adequately prepared to respond to and do not have adequate supplies of the appropriate antidotes. [10]

Cyanide is readily available, easy to use, and highly lethal making it an ideal chemical weapon. [11] Individuals ingesting cyanide may be unaware they are being poisoned and therefore likely to consume larger amounts prior to developing symptoms. [12]Urine cyanide concentrations can also be affected by smoking status. Additionally, due to filtration by the kidneys, urine cyanide levels are much lower than the levels found in the blood [13] Methods to identify cyanide exposure, require a significant amount of time to result, thus delaying antidote delivery. [14] 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 the diagnosis and treatment of cyanide poisoning.[15] Although several forensic studies have demonstrated the significance of cyanide poisoning in fire victims using blood cyanide levels, the association between the cause of cardiac arrest and the concentration of cyanide among fire victims has not been sufficiently investigated [16] The signs and symptoms of oral cyanide are similar to those of inhaled cyanide; however, the timing and severity differ [17] Exposure to cyanide via the inhalation route results in symptoms within seconds of exposure, whereas symptoms following ingestion occur in minutes to hours. [18] Cyanide fits the description of an ideal terrorist weapon as it is, plentiful, readily available, does not require special knowledge for use, is capable of causing mass confusion, panic, and social disruption, and requires large quantities of specific resources to combat. [19]The threat of oral cyanide poisoning via food and water supplies is high. [20]

Molecular geometry of Hydrogen cyanide (HCN) is depicted in figure 1 HCN – Systemic /Chemical Asphyxiant Prussic acid - 2% aqueous HCN Scheele acid - 4% HCN Gaseous form – Cyanogen Odour – Bitter Almonds; 20%-40% humans cannot detect the odour Normal level – 15 to 30 mcg Boiling point: -26 c Colourless, Transparent, Volatile Decomposed by light

Uses

1) **Industrial :** Electrical plating, Metal processing, Photography, Synthetic rubber production, Plastics manufacture
2) **Agriculture** : Insecticide & Rodenticide
3) **Laboratories** : Various processes
4) **Medical** :

- a)**Laetrile -** Synthetic amygdalin ,Alleged anticancer drug
- b)**Sodium Nitroprusside -** Antihypertensive in Hypertensive Crisis ;i.v.

Mechanism Of Action

- **Inhibition Of Cytochrome-P- Oxidase**

i. Cytochrome oxidase cyanide complex
ii. Electron transport system block
iii. ATP depletion
iv. Histotoxic Anoxia
v. Anaerobic energy production
vi. Metabolic Acidosis

- **Inhibition Of Antioxidant Enzymes**

i. Catalase, GLU-dehydrogenase, GLU-reductase, Superoxide dismutase
ii. Lipid peroxidation - Neurotoxicity

Sources

a) Widely distributed in nature.
b) Cherry laurel leaves, Bitter almonds, Kernels of common cherry, Plum, Apricot, Peach, other stone fruits , ordinary bamboo shoots, oilseeds, Beans , all parts of cassava plant etc.
c) Amygdalin --->Emulsin ----> HCN {hydrolysis} Amygdalin = Glucose + Benzaldehyde + Cyanide
d) Combustion :- Burning of plastic furniture (polyurethane/ polyacerylonitrile), Burning of wool, Cigarette smoking.

Toxicokinetics

- **Absorption**

HCN quicker than salts Liquid HCN -Skin&mucous membranes Gaseous form – Resp.tract Delay – food & alcohol

- **Conversion**

Cyanide salts + HCL ---> HCN [Achlorhydric- Grigori Rasputin]

- **Metabolism**

Humans natural CN Detoxification by Rhodanese enz.in liver, kidneys {1mg of CN/kg/hr}

- **Excretion**

Urine, Breath

Fatality

- Fatal Dose

1. On Ingestion

- HCN 50 to 60mg
- NaCN, KCN – 200 TO 300mg
- Bitter Almonds – 50 to 80 {PrunusAmygdalus}.

2. On Inhalation

- 100 to 200 ppm in air
- 1:50,000 fatal in hrs
- 1:10,000 in few mins
- 1:2000 immediately.

- **Fatal Period**

i. HCN – 2 to 10 mins
ii. KCN/NaCN – 30 mins.

Signs And Symptoms

Cyanide toxicity is considered in a person with sudden CVS collapse in the context of occupational exposure [laboratory/industrial work] or in fire with hemodynamic instability increased lactic acid / coma

Acute Poisoning

- **Inhalation**
- Observed within Seconds
- Sudden loss of consciousness
- Prompt death [respiratory arrest]

- **Ingestion**
- Occurs in Minutes
- Absorption & metabolism
- Voluntary Acts
- Major Systems involvement
- Stimulation of Aortic & Carotid bodies
- Lactic Acidosis – Mitochondrial cytochrome oxidase inhibition, Anaerobic metabolism

Chronic Poisoning

- Mild doses (less than fatal dose) cause Chronic poisoning
- Continued inhalation of Cyanide leads to

(a) Tobacco Amblyopia- Progressive loss of vision. Exclusive in heavy smokers. (b) Lebers Heriditary Optic Atrophy- Congenital deficiency of Rhodanese enzyme, Acute visual failure due to sensitivity of optic nerve to cyanide ,observed in Males.(c) Tropical Ataxic Neuropathy- In Cassava / Tapioca consumers

- Symptoms are peripheral sensory neuropathy, optic atrophy, ataxia, deafness, glossitis, stomatitis, dermatitis
- Linamarin & Lotaustralin are the 2 cyanogens in tuber When inhaled 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 voluntary acts, such as corking, or throwing away the bottle or walking a little distance.

The major organs/systems involved are the GIT, CNS, respiratory and cardiovascular systems.

Gastrointestinal Tract:

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

Central Nervous System:

The involvement of CNS leads to dizziness, headache, sweating, anxiety, confusion, drowsiness, syncope, opisthotonus, seizures, coma and death.

Respiratory System:

Initially, tachypnoea and dyspnoea develop due to the stimulation of respiratory centre and carotid chemoreceptors caused by local hypoxia. Bradypnoea, hyperpnoea and irregular respiration (characteristically, a short inspiration and prolonged expiratory phase), pulmonary oedema, cyanosis and respiratory arrest in the later stage. A bitter-almond like odour may be detected in the breath.

Cardiovascular System:

Initially, 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 stages is bright red. This may be easily demonstrable by examining the fundus for retinal arteries and veins.

Lactic Acidosis: Lactic acidosis 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 consequence of anaerobic metabolism.

Diagnosis

a)History, Physical examination, Characteristic odour of bitter almonds. (b)Lee-Jones Test 5ml gastric aspirate + few crystals of ferrous sulphate + 5drops of 20%NaOH Boil & Cool Add 10 drops of 10% HCL Greenish blue precipitate indicates CYANIDE [Due to Prussian blue formation] [purple colour seen in salicylates.] (c) Serum Cyanide levels Blood cyanide levels > 0.2 mcg/ml toxic Lethal cases have >1 mcg/ml

Management

[1] Stabilisation

Assisted ventilation, 100% oxygen, IV access, metabolic acidosis treatment, vasopressors for hypotension

[2] Decontamination Cutaneous exposure

Ingestion -stomach wash

[3] Anti Dotal Therapy Cyanide Antidotes :-

(a) Met hemoglobin generators : Amyl nitrite ,sodium nitrite, Methylene blue, Phenones 4-Di Methyl Amino Phenol, p-amino octanoyl phenone, p-amino propio phenone. (b) Sulfur Donors - Sodium Thiosulfate. (c) Direct binding agents - Dicobalt edetate [kelocyanor], Hydroxo cobalamin [vit B12, Cyanokit], Cyanohydrin forming drugs like Aldehydes, Dihydroxy acetone, Glyoxal.

Method Of Administration

- **Lilly Cyanide Kit/Lilly Antidote Kit:**

Constitutes 2 Met haemoglobin generators & 1 sulfur donor.

(1) 0.2 ml Amyl nitrite ampoule broken handkerchief soaked in it & pt made to inhale for 15-30s.
(2) Sodium nitrite 10ml of 3% given very slow i.v. for 5 minutes. Do not remove needle.
(3) Sodium thiosulfate slow i.v. infusion of 50 ml of 25% sodium thiosulfate in 1000ml of 5% glucose solution [5ml/min infusion rate]
(4) If symptoms persist/re appear repeat both Sodium nitrite & sodium thiosulfate at half the initial dose at the end of 1 hr.

4] Exchange Tranfusion

MethHb 50%

[5] Supportive Therapy

Observation of 24 – 48 hrs Lactic acidosis – NaHCO3 i.v. Anticonvulsants Cardiopulmonary resuscitation

Postmortem Appearances

The skin presents a livid or violet appearance. Postmortem staining is often bright red due to formation of cyan-methaemoglobin, and also due to the fact that the tissues cannot take up oxygen of the blood, leaving it bright red even in the veins (cyanide being lethal in small quantities and, therefore, the total amount of the poison in the body may not be sufficient for generalized discoloration). The fingers may be clenched, fingernails blue and there is usually froth at the mouth and

nostrils. The eyes may be bright, glistening and prominent with dilated pupils Jaws are usually firmly closed. Rigor mortis sets in early and lasts longer.

Internally

The odour of hydrocyanic acid may be noticed on opening the body, but it is liable to fade quickly. The cranial cavity should be opened first, as the odor is usually well-marked in the brain tissue. Blood-stained froth may be found in the trachea and bronchi. Pulmonary oedema is evident. The mucosa of the stomach and intestines is often congested.

In case of cyanides, lips and mouth may be corroded and the mucous membranes of the stomach and duodenum may be bright red to brown in color due to the effect of potassium carbonate (present as an impurity in the potassium cyanide) and the probable formation of cyanhaemochromogen from the effects of the cyanide on hemoglobin in the presence of an alkali. Intestines : congested

Toxicological Analysis

Blood + Fluoride [with liquid paraffin layer cover] should be sent Brain, lungs in addition to other viscera Lungs sent intact in a nylon bag to prevent evapouration Spleen best specimen [more RBC High concentration of HCN] well stoppered bottles are used Avoid Embalming before Autopsy [as formalin destroys CN] HCN retards decomposition & not produced due to putrefaction. [<1/10 of that in true poisoning]

Medicolegal Importance

- Suicide

HCN & its salts suicidal pills/rings/hollow teeth[Terrorists-LTTE]

- **Accidental**

By the inhalation of fumes of acid – Fumigation, chemists etc Bitter almonds ingestion Cattle poisoning by Kadvi juar / linseed plant ingestion Building fire [0.3mg%HCN in blood]

- **Homicidal**

Rare except for mass homicides-ZYKLON B [nazis in 2nd world war], chemical terrorism, warfare Judicial execution / judicial gassing [USA]

- **Miscellaneous**

i. Cherry laurel water / aqua laurocerasi [leaves] –used in asthma, cough, dyspepsia. NERO used to poison wells of enemies with these
ii. Sodium nitroprusside
iii. LAETRILE/ Vit B7

Case History

- The Deceased [D1] a 31yrs old male ,stock market share holder.
- Went to see the Dindi Project with his Wife [D2] 27yrs old female, son [D3] 2.5yrs old male, aunt [D4] 42yrs female & Kin [D5] 16yrs old female informing his Uncle in the morning.
- Deceased persons [D1] Uncle filed a missing complaint when the family didn't return & found their mobiles were switched off at night.
- On investigation in the next morning the Police found the Deceased [D1] & his son [D3] dead in the car and the remaining persons [D2,D4,D5] dead half a km away in the bushes.
- Deceased were brought for autopsies to Osmania general hospital , Hyderabad, Telangana (shown in figure 19 and 20).
- Subjected to autopsy after receiving requisitions along with inquests from police.

Salient Autopsy Findings

- **External Examination Common Findings :-**

Face Congested Conjuctivae of both eyes Congested Lips Cyanosed Nails Cyanosed

Additional Findings:-

- D1 : Fine white froth from mouth
- D2 : Regurgitated food particles at both nostrils, Dried serous discharge from right angle of mouth
- D4 : Thick mucoid discharge from both nostrils and mouth
- D5 : Strong Garlicky smell, irritating eyes emitting from all body cavities

Postmortem Changes

Rigor mortis present throughout the body of all the deceased. Postmortem lividity seen over back of the body of all deceased

Injuries

- D2 : Multiple abrasions of 0.5 cmx0.75cm size over an area of 15 x 10cm over right flank of front of lower part of chest and upper part of abdomen ; Injuries are obliquely placed ; Brownish in color with occassional loss of skin cuticle exposing yellowish base.
- D5 : Multiple abrasions of 0.25cm x0.25cm size over an area of 7cm x 4cm on front of upper part of right shoulder. Multiple abrasions of 0.23cm x 0.25cm size over an area of 5cm x 4cm over outer aspect of upper part of right arm. Injuries are obliquely placed ; Brownish in colour with occassional loss of skin cuticle exposing yellowish base

Internal Examination

- Common Findings All viscera Congested Lungs :-

Congested, edematous; on cut section white fine froth seen in all lobes of both lungs. Stomach :-
Yellowish Brown digested food emitting strong Garlicky odour with Mucosa congested, eroded & Haemorrhagic.

- **Additional Findings**

D1 : Small Intestine & Large Intestine - Mucosa edematous & Congested
D2 : Trachea & Bronchi - Regurgitated food particles
D4 : Trachea & Bronchi - White fine froth

Chemical Analysis

Viscera [Stomach & its Contents, Liver, Kidneys, Blood] sent for chemical analysis to Telangana State Forensic Science Laboratories, Hyderabad

Time Since Death

12hrs to 18hrs prior to postmortem examination.

Summary Of Fsl Report

"CYANIDE POSITIVE" detected in all samples .

Final Opinion

Cause Of Death in all deceased is due to "CYANIDE POISONING"

DISCUSSION

- According to Henderson & Haggard Classification Cyanide is a Systemic Asphyxiant.
- "CYANOGEN " - Normal constituent of Body is 15 to 30 mcg.
- Widely Distributed in Nature.
- Mode of Death :-
- Central Respiratory arrest –Histotoxic Anoxia
- Fatal period – 2 to 10 minutes / Immediate
- Fatal Dose

i. Bitter Almonds: 50 to 80
ii. Hydrocyanic acid 50 to 60 ml
iii. Cyanogen – 100 to 200ppm
iv. KCN/ NaCN – 150 to 300mg

- **Classical Characteristics**
- **Odour :** BITTER ALMOND
- **Brick Red** : Skin & Mucous membranes
- Cyanosis

- Post mortem examination Changes :-
- Rigor Mortis - sets early lasts long
- Post mortem lividity – Bright Red [due to Cyan meth Hb]Stomach / Duodenum – Bright Red to Brown [due to KCo3]

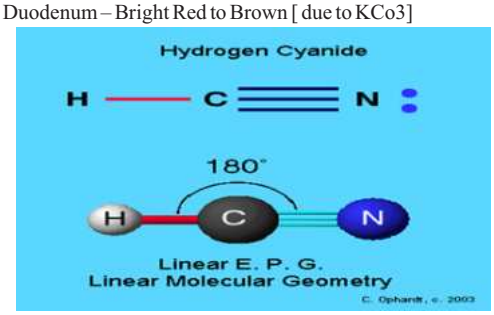

Fig.1. Molecular Geometry of HCN

Fig.2. Molecular structure of HCN & KCN

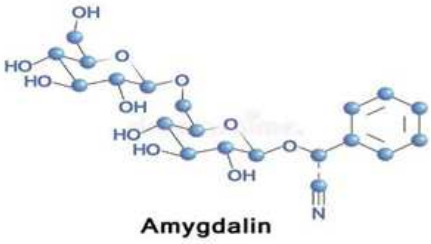

Fig.3. Amygdalin Structure

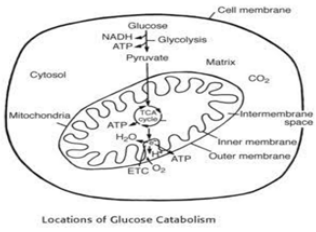

Fig.4. Mechanism of action sites:Cyanide

Fig.5. Inhibition of Cytochrome-c-oxidase by Cyanide

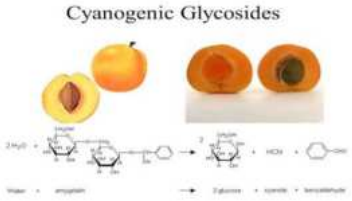

Fig.6. Cyanogenic Glycosides mechanism of action to produce Cyanide

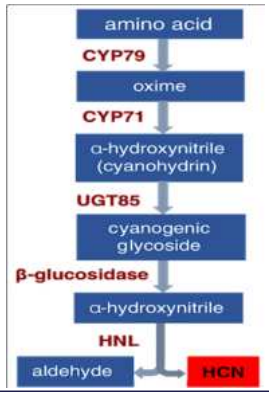

Fig.7. Cyanide production from Cyanogenic glycosides : aminoacids

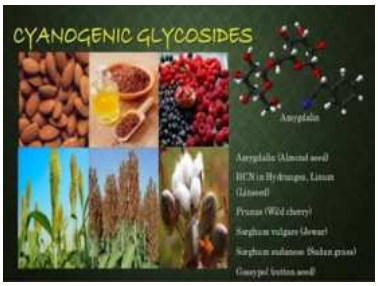

Fig.8. Examples of Cyanogenic Glycosides

Fig.9. Plants producing Cyanogenic glycosides

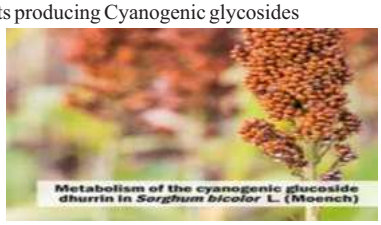

Fig.10. Metabolism of Dhurrin: Cyanogenic glucoside

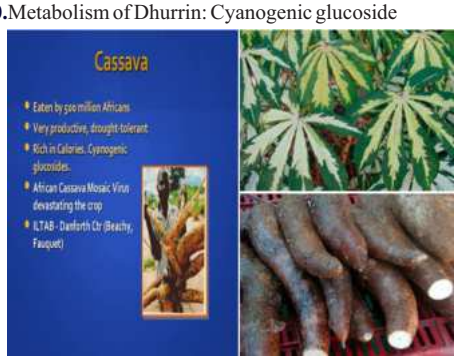

Fig.11. Cassava: Cyanogenic glucoside

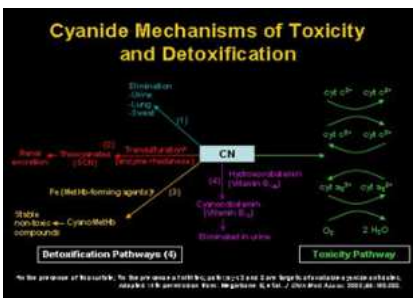

Fig.12. Mechanism of Toxicity & Detoxification of Cyanide

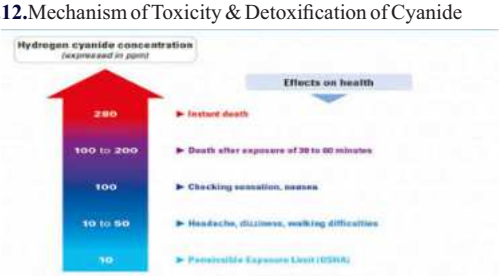

Fig.13. Concentration of HCN Vs Health effects

Fig.14. Mitochondrial Cytochrome Heme group

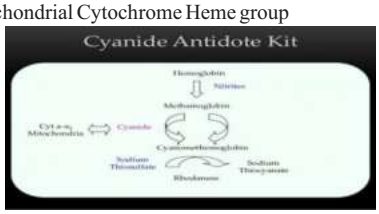

Fig.15. Mechanism of Action of Cyanide Antidote Kit

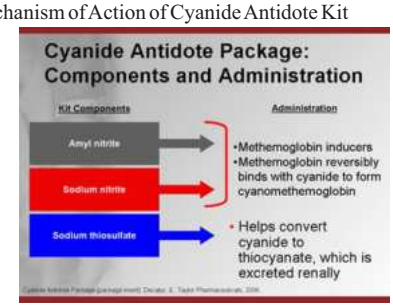

Fig.16. Components & Administration of Cyanide Antidote Kit

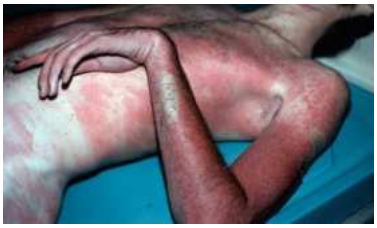

Fig.17. Cherry red Postmortem staining in Cyanide Poisoning

Fig.18. Haemorrhagic Gastric Mucosa in Cyanide poisoning

Fig.19. Crime Scene - Source: News Paper Clippings from Investigating Officer

Fig.20. Deceased Photographs From Investigating Officer

Table 1: Uses Of Cyanide Salts

S.No.	Salts	Uses	Properties
1.	Potassium cyanide, Sodium cyanide, Mercuric cyanide, Silver cyanide	Photography, Electroplating, Hardening of steel, Silver & Gold processing, Dyeing	Alkaline in reaction, soluble in water, highly poisonous
2.	Calcium cyanide	Mining Industry	Cheap
3.	Magnesium cyanide [CYAMAG], Cyanogen chloride	Insecticides	---

Table 2: Details of Cyanogenic plants

S.No.	Cyanogenic Plants	Toxic Part	Cyanogenic Glycoside
1.	Prunus species – cherry laurel, plum choke berry, peach mountain mahogany, Apricot, Wild black cherry	Leaf, Bark, Seed	Prunasin/Amygdalin
2.	Sorghum species – sorghum, sudan grass, johnson grass, arrow grass	Grain, Shoot	Dhurrin
3.	Apple,Pear,Crab apple	Seed	Amygdalin
4.	Cassava, lima beans	Bean, Root	Linamarin
5.	Miscellaneous – Christmas berry, Velvet grass, jet berry bush, elder berry bamboo, cycad nut	Bead, Leaf, Shoot, Sprout	---

REFERENCES

1. Huzar TF, George T, Cross JM: Carbon monoxide and cyanide toxicity: etiology, pathophysiology and treatment in inhalation injury. Expert Rev Respir Med. 2013, 7:159-170. 10.1586/ers.13.9
2. Kerns II, WP,Kirk MA,Cyanide and hydrogen sulfide.in: Flomenbaum NE Goldfrank LR Hoffman RS Howland
3. Parker-Cote JL, Rizer J, Vakkalanka JP, Rege SV, Holstege CP. Challenges in the diagnosis of acute cyanide poisoning. Clin Toxicol (Phila). 2018 Jul;56(7):609-617.
4. Simeonova FP, Fishbein L. Hydrogen cyanide and cyanides: human health aspects. 2004 [cited 2018 August 4]. Available from: http:// www.who.int/iris/handle/10665/42942.
5. Agency for Toxic Substances and Disease Registry. Public health statement for cyanide. 2006 [cited 2018 August 4]. Available from: https://www.atsdr.cdc.gov/phs/phs.asp?id=70&tid=19.
6. Lee SK, Rhee JS, Yum HS. Cyanide poisoning deaths detected at the national forensic service headquarters in Seoul of Korea: a six year survey [2005~2010]. Toxicol Res. 2012;28(3):195–9.
7. Morocco AP. Cyanides. Crit Care Clin. 2005;21(4):691–705 vi. 5. Eckstein M. Cyanide as a chemical terrorism weapon. JEMS. 2004;29(8):suppl 22–31.
8. Director of National Intelligence. Terror franchise: the unstoppable assassin. 2015 [cited 2018 August 4]; Available from,http://www.dni.gov/files/documents/ubl/english/TerrorFranchise.
9. Moore J. ISIS supporters call for poisoning of food in grocery stores across U.S. and Europe. Newsweek 2007 [cited 2018 August 2]. Available from: http://www.newsweek.com/isis-supporters-callpoisoning-grocery-stores-us-and-europe-660750.
8. Hodoh O, Dallas CE, Williams P, Jaine AM, Harris C. A benchmark system to optimize our defense against an attack. Am J Dis Med. 2015;10(3):177–88
9. Parker-Cote JL, Rizer J, Vakkalanka JP, Rege SV, Holstege CP. Challenges in the diagnosis of acute cyanide poisoning. Clin Toxicol [Phila]. 2018;56(7):609–17.
10. Agency for Toxic Substances and Disease Registry. Toxicological profile for cyanide. 2006 [cited 2018 July 30]. Available from: https://www.atsdr.cdc.gov/ ToxProfiles/tp.asp?id=72&tid=19
11. RTI International. Cyanide: understanding the risk, enhancing preparedness. Clin Toxicol. 2006;44(suppl 1):47–63
12. Lee J, Mahon SB, Mukai D, Burney T, Katebian BS, Chan A, et al. The vitamin B12 analog cobinamide is an effective antidote for oral cyanide poisoning. J Med Toxicol. 2016;12(4):370–9.
13. Ma J, Dasgupta PK. Recent developments in cyanide detection: a review. Anal Chim Acta. 2010;673(2):117–25. 27.
14. Jackson R, Logue BA. A review of rapid and field-portable analytical techniques for the diagnosis of cyanide exposure. Anal Chim Acta. 2017;960:18–39
15. F J Baud, Cyanide: critical issues in diagnosis and treatment,Hum Exp Toxico, 2007 Mar;26(3):191-201. doi: 10.1177/0960327107070566.
16. Yasuhiko Kaita 1, Takehiko Tarui 2, Takahiro Shoji 2, Hiroshi Miyauchi
17. Logue B, Hinkens DM, Baskin SI, Rockwodd GA. The analysis of cyanide and its breakdown products in biological samples. Crit Rev Anal Chem. 2010;40(2):122–47.
18. Ma J, Dasgupta PK. Recent developments in cyanide detection: a review. Anal Chim Acta. 2010;673(2):117–25.
19. Yoshihiro Yamaguchi, Cyanide poisoning is a possible cause of cardiac arrest among fire victims, and empiric antidote treatment may improve outcomes, Am J Emerg Med 2018 May;36(5):851-853. doi: 10.1016/j.ajem.2018.01.054. Epub 2018 Feb 1.
20. RTI International. Cyanide: understanding the risk, enhancing preparedness. Clin Toxicol. 2006;44(suppl 1):47–63. 34. Gleick PH. Water and terrorism. Water Policy. 2006;8(6):481–503.