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REVIEW ON CYANIDE POISONING

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Abstract

Cyanide is one of the most lethal poison. It leads to death within a few minutes to a few hours sometimes. Depending upon the dose and route of administration or exposure the symptoms develop. The area of exposure is manufacturing and industrial sources such as insecticides, photographic solution, fumigation and electroplating work, plastic manufacturing and jewelry cleaners etc. History shows the common use of cyanide poisoning in suicidal and homicidal cases and also use as chemical warfare agent for terrorist attack. Incidence of cyanide poisoning is rare but the occurrence of death is seen instant in some cases. It causes histotoxic anoxia and 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 binds to the enzyme cytochrome C oxidase and blocks mitochondrial transport chain. This results in cellular hypoxia and the depletion of ATP occur, leading to metabolic acidosis. Symptoms such as headache, dizziness, vertigo, spasmodic closure of jaw and clawing of hands, tonic type of convulsions of the limb and trunk, muscular weakness and flaccidity, muscular paralysis, intense cyanosis, hypertension followed by hypotension, coma etc leads to death. Death is mainly due to cardiovascular failure and respiratory failure. Thus, rapid treatment to be started in such patients. The 100 % oxygen support and rapid therapy of antidotal treatment is necessary for life saving. Very efficient antidote is Hydroxocobalamin and other antidotes are also important in cyanide poisoning in the treatment as life saver. Survivors of cyanide poisoning may develop neuropathies.

Keywords: *Lethal Poison, Cardiovascular failure, Rapid treatment, Hydroxocobalamin*

Introduction

Cyanide is an irritant inorganic metal poison also known as Acid of sugar. Cyanide is a highly cytotoxic poison. It is available in different forms they are (a) Gaseous form – Hydrogen cyanide b) Liquid form – Hydrocyanic acid or prussic acid c) Crystalline powder – Salts of cyanide, i.e., sodium, potassium or calcium cyanide. Hydrogen cyanide was first isolated from Prussian blue dye in 1786, and it was first extracted from almonds around 1800. Naturally cyanide is found in some foods such as lima beans, almonds, cassava roots, apples, flax etc. Cyanide is also found in manufacturing and industrial sources such as insecticides, photographic solutions, plastics manufacturing, and jewelry cleaner etc . Cyanide reacts at a higher level with high stomach acidity thus readily absorbs and causes fatality. In past it has been used as a poison in mass homicides and suicides. On 26 January 1904, company promoter and swindler Whitaker Wright committed suicide by ingesting cyanide in a court anteroom immediately after being convicted of fraud. Jolly Thomas of Kozhikode, Kerala, India, was arrested in 2019 for the murder of 6 family members. Murders took place over 14-year period and each victim ate a meal prepared by the killer. The murders were allegedly motivated by wanting control of the family finances and property.[77]Dhillon, Amrit (7 October 2019). "Woman in India admits poisoning six family members with cyanide". The Guardian. Archived from the original on 8 October 2019. Retrieved 8 October 2019...

Mode of Action

The gas is absorbed through skin when it is moist with perspiration, mucous membrane and through respiratory tract. In the stomach by the action of HCL acid, cyanide salts are changes into HCN and subsequently absorbed as cyanide ion (CN-). So, fatal period of HCN is much less than cyanide salts. That is why achlorhydric (no free hydrochloric acid in the gastric juice) persons are resistant to cyanide salts. But water in gastric juice and stomach contents can hydrolyse cyanide salts to liberate hydrocyanic acid. It inhibits the cytochrome oxidase system. It results in histotoxic anoxia, reducing O2 carrying capacity of blood, preventing utilization of oxygen by dissociating tissue cells from the oxyhaemoglobin of the red cells – thus results in bright red colouration of the venous blood. It blocks the final step of oxidative phosphorylation and prevents the formation of ATP and its use as energy source.The greater part of it is converted into thiocyanate by an enzyme rhodanese (present in mitochondria of liver and kidney) which needs sodium thiosulphate. Some cyanides are converted to cyanocobalamin (vitamin B12) in the presence of hydroxocobalamin (vitamin B12a). It is

mostly excreted through urine and a small part is excreted unchanged in breath and sweat producing characteristic bitter almond smell.

Fatal Dose

Pure Hydrocyanic acid – 50 to 60 mg, Diluted Hydrocyanic acid – 30 to 50 drops (1.5 drachm), 50 to 60 drops of oil of bitter almond, Cyanide salts (of sodium, potassium or calcium) – 200 to 300mg, Exposure to concentration of 1 part of hydrocyanic acid in 2000 parts of air can cause instantaneous death of adults. 1 part in 10,000 parts of air within few monutes, 1 part in 50,000 parts of air within few hours. Permissible concentration of HCN in air is 1 part per 1,00,000.

Fatal Period

The fatal period is seen Exposure to HCN gas 2 to 10 minutes, if not immediate, Sodium and potasssium cyanide : 15 to 30 minutes.

Signs and Symptoms of cyanide poisoning

Acute poisoning : The poisoning is very rapid in its action. However , onset of action depends upon the physical form and the dose. Due to inhalation of gas or ingestion of massive dose, consciousness is lost instantaneously and death is very rapid within 1 to 2 seconds. Inhalation causes a sense of constriction in the throat and chest, vertigo, dizziness, giddiness, confusion, insensibility, unconsciousness, weak and quick pulse, acute respiratory distress and death occurs following respiratory centre paralysis and circulatory failure.

Due to ingestion of large doses, symptoms appear at once or within a few seconds. At times symptoms may appear as late as 1 to 2 minutes and during that period the person can perform some conscious acts like corking of the bottle, throwing it or putting it in its previous position or walking for some distance. At times, even when not inhaled, the victim may die very rapidly without any symptoms.

Depending upon the dose of ingestion , either during ingestion or within 1 to 2 minutes of ingestion, there will be headache, dizziness, giddiness, vertigo, excitement, anxiety, confusion, drowsiness, spasmodic closure of jaw and clawing of hands, tonic type of convulsions of the limb and trunk, muscular weakness and flaccidity, muscular paralysis leading the victim unable to stand, fine froth at the angles of mouth, intense cyanosis, relaxation of the sphincters, eyes remain wideopen, fixed and glistening, pupils dilated and not reacting to light, skin is cold and bathed wth perspiration, pulse becomes feeble, imperceptible, weak and quick, smell of bitter almond in the breath, initially dyspnoea and tachypnoea later on

becomes slow and shows severe respiratory distress, initially there may be hypertension followed by hypotension. Death occurs due to respiratory failure.

Chronic Poisoning

It occurs under the following circumstances - Persons engaged in electroplating, tanning and dye industries, photography, glinding etc. Persons suffering from congenital deficiency of rhodanese – optic nerves becomes sensitive to cyanides. In heavy smokers – there occurs progressive loss of visual functions. Due to consumption of large quantity of cassava or tapioca (contains cyanogens like linamarin and lataustralin). Inhalation of cyanide gas in sublethal concentration for some time. Persons handling potassium cyanide or other salts.

Signs and Symptoms of chronic cyanide poisoning

The victim complain of headache, vertigo, nausea, vomiting, anorexia, glossitis, stomatitis, foetid smell in the breath, anaemia, loss of weight, hoarseness of voice, conjunctivitis, progressive loss of visual function, acute visual failure (due to congenital deficiency of rhodanese), optic atrophy, deafness, constipation or diarrhoea, dermatitis, skin eruptions or bullae formation, desquamation of skin from hand and fingers (from handling cyanides), ataxia, peripheral sensory neuropathy.

Treatment

Due to rapid fatality in this poisoning, victim usually dies before starting of treatment. If the person is available alive, treatment should be started immediately , and the aim of the treatment is to reverse the cyanide-cytochrome combination.As the cyanide has greater affinity for methaemoglobin, by administering nitrites hemoglobin may be transformed into methaemoglobin which will combine with cyanide to form cyanmethemoglobin which is non toxic.10 ml of 3% sodium nitrite (0.3 gm in 10 ml sterile water) is given through intravenous route slowly (over a period of 2 to 3 minutes). Through the same needle, 50 ml of 25% sodium thiosulphate is given over a period of 10 to 15 minutes. This combination therapy is considered to be effective antidote for hydrocyanic acid and potassium cyanide poisoning. Repeat the nitrite-thiosulphate therapy every hourly in half of their doses, till the patient recovers. Cyanide is converted into non-toxic thiocyanate. But Sodium nitrite may cause formation of an excessive amount of methaemoglobin which has no oxygen carrying capacity, and causes a fall of blood pressure. When concentration of methaemoglobin becomes more than 50% , blood transfusion or exchange transfusion is required. Amyl nitrate (after breaking an ampule of 0.2 ml) soaked in cotton wool may be held before the nostrils for inhalation for a period of 30

seconds at an interval of 2 to 3 minutes may be continued if nitrite thiosulphate is not available ready at hand or till they are available for use.

Previously, intravenous infusion of 50 cc of 1 % sterile aqueous solution of methylene blue (Methylthionine chloride) was being used as an antidote because of its power to convert haemoglobin into methaemoglobin which had the scope to combine with free cyanide, thus preventing cyanide to act adversely in the system. As methaemoglobins has no oxygen carrying capacity, it may be accumulated in the circulation. So, it must not be used as an antidote , for its inherent danger.

Antidotes:- (a) Dicobalt EDTA (Kelocyanor) 600 mg (20 ml) or dissolved in 100 ml of 5% glucose may be given slowly through an intravenous route and may be repeated in half of its dose till recovery.

(b) 4-dimethyl aminophenol (4-DMAP) 3 mg/kg of body weight may also given slow I.V., considered to be the antidote of choice to induce methemoglobinemia.

(c) Hydroxocobalamin (Vit. B12 precursor) – 4 to 5 gm (50 mg/kg of body weight), 1000 mg/ml in 3 to 3.5 liter infusion intravenously may be effective. P-amino-propiophenone (PAPP) may also be used for its sustained action , though not acting as quickly as nitrites. In addition to precaution , patients who are treated with hydroxocobalamin should avoid sun exposure to prevent photosensitivity.

(d) Alpha-ketoglutaric acid is also considered to be effective and safe.

Stimulants like coramine, atropine, lobeline, picrotoxin or amphetamine may be given to stimulate respiration. Cortison, binodrenal, noradrenaline in 5 % glucose-saline may be given to prevent cardiorespiratory collapse. Artificial respiration. In cases of delayed death or in cases of potassium cyanide or dilute hydrocyanic acid poisoning – stomach wash is given with ferrous and ferric sulphate and potassium carbonate mixture or sodium thiosulphate, hydrogen peroxide or potassium permanganate (300 mg in 500 ml). After completion of stomach wash ferrous and ferric sulphate with potassium carbonate is given by mouth. It acts as a chemical antidote and forms Prussian blue which is inert and innocuous.

In case of poisoning due to inhalation of cyanide gas - Patient is removed from the source to fresh air. Artificial respiration is given. Intravenous Sodium nitrite and sodium thiosulphate are given in the usual dose. Coramine 5 ml (25%) or other analeptics may be given. Inhalation of amyl nitrate through soaked cotton wool is started. Nitrate-thiosulphate therapy is given in usual doses.

In cases of ingestion of calcium cyanide – No specific antidote is available. Usual stomach wash and symptomatic treatment is given.

In cases of ingestion of Mercury cyanide – Gastric lavage is given along with the above noted treatment, BAL in usual dose is helpful.

Autopsy Findings

Externally, face including lips appear livid, cheeks appear cherry red in colour, jaw is firmly closed and fine froth at the angles of mouth, fingernails cyanosed, eyes are bright, prominent, glistening in appearance, pupils dilated, cornea remains clear for a long time. P.M. Stains appear cherry red or bright red in colour due to formation of cyanmethemoglobin and unutilized oxyhaemoglobin in the tissue level and presence of oxyhaemoglobin in the vessels including vein. Rigor mortis sets in early if convulsion occured prior to death and it also lasts longer.

Internally the smell of hydrocyanic acid is detected on the opening of the chest cavity and stomach. Lungs are congested, oedematous and the smell of hydrocyanic acid is detected. Right side of the heart is usually full and engorged with venous blood. All the viscera are congested. Mucous membrane of the stomach and intestine is intensely congested and red.

In case of potassium cyanide poisoning, lips , tongue, stomach and duodenum show evidence of corrosion with punctate hemorrhage underneath with crimson red colouration which may turn to brick red to rusty brown due to formation of cyanohemoglobin by the effect of cyanide on hemoglobin in presence of alkali. In cases of death following cyanide poisoning, blood concentration exceeds 1 mg%.

Like Carbon monoxide it retards putrefaction. To some extent, it acts as a preservative because of its oxygen binding affinity.

Formaldehyde, a component of embalming fluid, destroys cyanide rapidly causing difficulty in detection of the poison . So, embalming is contraindicated in a case of suspected cyanide poisoning.

Diagnosis of cyanide

Diagnosis is often difficult.[2] the person has a decreased level of consciousness, low blood pressure, or high lactic acid Cyanide poisoning. Plasma lactate levels of more than 8 millimoles per liter Trusted Source are 70% specific for cyanide poisoning

Lee-Jones test – Few crystals of ferrous sulphate are added to 5 ml of the gastric content. To it, 5 drops of sodium hydroxide are added. The mixture is boiled and cooled. To it, 10 drops of

10 % hydrochloric acid are added. Greenish blue colour indicates cyanide and purplish colour indicates salicylate.

Medicolegal Aspect

For Commercial use - In electroplating, silver plating, In silver and gold processing, In photography, For hardening of steel and iron cases, In manufacturing of dyes etc. As fertilizer, insecticide (trade name 'cymag') and rodenticide, Mining industry, metallurgy, tanning.

HCN acid is used in industries for fumigation of rooms, shipholds , For bugs, vermins, lice etc., For synthetic refining of ores, cleansing of metals, manufacturing of synthetic rubbers, plastics etc.

As medicine Laetrile (a synthetic amygdalin) used as a chemotherapeutic agent for carcinoma, sodium nitroprusside in the treatment of hypertension as laboratory reagent.

Used mostly for suicidal purpose, Cattle poisoning, Rarely used as homicidal because of its characteristic odour , perceptible taste and easy detection.

Discussion

Cyanide is a respiratory poison causing asphyxia and death in less time. The ingestion and inhalation are route of poisoning which absorbs the poison quickly and produces respiratory failure and circulatory failure. It also shows progressive neuropathy. Clinical manifestations vary widely, depending on the dose and route of exposure, and may range from minor upper airway irritation to cardiovascular collapse and death within minutes. In severe cases, rapid, aggressive therapy consisting of supportive care and antidote administration is of utmost importance.

Conclusion

Review on cyanide poisoning presents that it is a deadliest poison causing instantaneous death. Also sometimes it produces death without any symptoms. Moreover cyanide poisoning had occurence in industrial areas due to its occupational exposure. Thus, the safety and precautions should be undertaken while working in cyanide exposure areas. Treatment should be done without dealy and 100% oxygen support with antidotes is very important for saving life.

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