ACCIDENTAL CYANIDE POISONING FROM INHALATION OF GOLD POLISHING CHEMICAL: AN UNUSUAL CASE REPORT

Dr. Monisha Pradhan, Assistant Professor,*
Dr. Anand Pawar, Junior Resident,*
Dr. Suresh Chand, Junior resident,*
Dr. C. Behera, Associate Professor,*
Dr P.C Dikshit, Director Professor & Head,*
*Department of Forensic Medicine, Maulana Azad Medical College, BSZ Marg, New Delhi

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Corresponding author
Dr. Monisha Pradhan
Phone: +919899682111
Email: monishapr@yahoo.com

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Abstract
Cyanide toxicity can be traced back to antiquity and its use as a suicidal agent is well known. However, fatal accidental poisoning by cyanide at the workplace is an uncommon event. Here we report a case of accidental cyanide exposure causing death of a 40 year old male, jeweller by profession, at his shop. The route of exposure was inhalation of a powdered chemical containing potassium cyanide which is used for polishing gold jewellery. The manner of exposure, clinical features and autopsy findings are discussed in this paper.

Introduction
Cyanide is a known as a highly toxic and deadly substance and has been used as a poison for thousands of years. The effects of a high dose of cyanide are quick, and death occurs within minutes. It has been used as a chemical warfare agent and for many military purposes mainly in the form of volatile liquids like hydrocyanic acid. It is notoriously used in executions, homicides, and suicides. In civilian life, poisoning with cyanide and its compounds can occur from its use in chemical syntheses, electroplating, plastics processing, tanning, metallurgy, and as a fumigant [1]. It is also used as a part of gold polishing chemical which is available in powdered form (Fig.1). Combustion of synthetic products that contain carbon and nitrogen, such as plastics, synthetic fibres and cigarette smoke causes release of cyanide. The release of cyanide and cyanogenic compounds from combustion of such products is the most common source of human exposure to cyanide [1,2,3]. It is also present in food like chokecherries, bitter almonds, apricot pits, lima beans and cassava beans [1]. We are presenting a case of cyanide poisoning through inhalation of a gold polishing chemical in powder form containing potassium cyanide.

Case Report
A 40 year old male was brought to the emergency with history of accidental poisoning with gold polishing chemical. According to his statement, he owned a jewellery shop where regularly worked with a gold plating powder which contains potassium cyanide. On that fatal day, at around 8:20 pm, while working with the powder at his shop, he switched on an overhead fan by mistake. The powdered chemical dispersed in air and was inhaled by him. Immediately then he started complaining of nausea, vomiting and feeling of burning sensation all over the body. He was rushed to the hospital by his relatives.

On arrival, he was conscious, oriented, with pulse rate 80/minute and BP 100/60 mmHg. His chest and abdominal examination was normal. Deceased had 4-5 episodes of loose stools and dizziness after reaching the hospital. There was no shortness of breath, bleeding, altered sensorium or seizures. His blood pressure started to decline and was 60 mmHg of systole after 2 hours. His ABG recording showed pH 7.30, pO2 50, pCO2 23, HCO3 11.6, SO2 79 which was suggestive of type 1 respiratory failure. Deceased was managed with oxygen inhalation, IV fluids, ionotropes, antiemetics, anacids, metronidazole, injection hydroxycobalamine 2 cc intramuscularly, injection human insulin 4 units. As sodium thiosulphate and sodium nitrate was unavailable, it was not administered. Deceased was a known diabetic and hypertensive.

The deceased’s condition deteriorated with loss of consciousness and declining blood pressure and pulse till the BP and pulse were not recordable. Cardiopulmonary resuscitation was started and continued as per protocol. However, deceased could not be resuscitated and was declared dead. The survival time after the poisoning was about 6 hours.

Autopsy Findings
At autopsy, the deceased was a 40 year old male, heavy built measuring 165 cm in length and weighing 110 kg. His face was congested and cyanosis was present in the fingers and toe nails.
Post-mortem staining was distinctly bright red in colour and was present over the back of the body except over pressure areas (Fig.3). Rigor mortis was developed all over the body. There was brownish yellow fluid coming out of the nose and mouth. There was no external injury present over the body. Internally, the mucosal wall of the respiratory tract was congested. Lungs were hyper-inflated, congested and oedematous. The right lung weighed 512 gm and the left weighed 500 gm. Both the lungs had multiple subpleural petechial haemorrhages over the lungs surfaces, more marked over the basal surfaces (Fig.4). The heart weighed 360 gm and was grossly normal with patent coronaries. Liver weighed 1526 gm and showed macro-nodular cirrhotic changes with yellowish discoloration. Stomach contained about 100 ml of brownish fluid mixed with mucus and yellowish discoloration. Mucosa was normal. Both kidneys showed chronic hypertensive changes. On opening the skull, the dura was intact, the meninges appeared normal and the body except over pressure areas (Fig.3). Rigor mortis was developed all over the body. Internally, the mucosal wall of the respiratory tract was congested. Lungs were hyper-inflated, congested and oedematous. The right lung weighed 512 gm and the left weighed 500 gm. Both the lungs had multiple subpleural petechial haemorrhages over the lungs surfaces, more marked over the basal surfaces (Fig.4). The heart weighed 360 gm and was grossly normal with patent coronaries. Liver weighed 1526 gm and showed macro-nodular cirrhotic changes with yellowish discoloration. Stomach contained about 100 ml of brownish fluid mixed with mucus and yellowish discoloration. Mucosa was normal. Both kidneys showed chronic hypertensive changes. On opening the skull, the dura was intact, the meninges appeared normal and the brain showed congestive changes with patchy subarachnoid haemorrhages over the cerebral convexities (Fig.5). The viscera were sent for chemical analysis.

Discussion

Cyanide is among the most potent and deadly poisons and human exposure to it can occur in numerous ways. Use of cyanide as suicidal agent has been reported since ancient times. People who use cyanide to commit suicide often have ready access to the poison through their occupations. These occupations include chemists, jewelers, pest controllers, and people working in mineral refining, photography, electroplating, dyeing, printing, and salmon poaching industries. Jewelers use cyanogenic compounds like potassium or sodium cyanide, mercuric cyanide and silver cyanide to rid gold of tarnish. Accidental poisoning can be suicidal or accidental. Most of the cases reported were suicidal in nature and occurred through ingestion[2, 6, 10-12]. Inhalation exposures to cyanide usually occur through smoke from fires and from fumigation vapours [1-3, 8]. Accidental inhalation of cyanide compounds in powder or dust form have not been reported in literature.

In our report, the deceased was a jeweller by profession and had been using potassium cyanide to clean gold jewellery. On that fatal day, he accidentally inhaled the gold polishing chemical containing potassium cyanide. He survived for a few hours to give details about his exposure. It is not uncommon for jewelers to be exposed to cyanide compounds, but the manner of exposure in this report, which was inhalational, is rather rare.

Though the manner of exposure maybe different, the manifestations are similar through all routes of exposure as cyanide is rapidly absorbed from all mucous surfaces, and even from unabraded skin. The minimum fatal dose of pure potassium cyanide is about 0.2-0.5g and for sodium cyanide, it is about 0.15 g. The onset of signs and symptoms is usually less than 1 minute after inhalation and within a few minutes after ingestion. On inhalation, death occurs immediately when the dose of cyanide is 270 ppm, and after 10 minutes if the dose is 181 ppm [13]. However, death may be delayed for several hours, if small dose is taken [3, 9]. In our report, the amount of the dose inhaled was not known, however it is likely the amount was not as much as that could be taken intentionally.

The clinical manifestations of cyanide poisoning are largely due to intracellular hypoxia as it interferes with the action of an enzyme cytochrome oxidase. Exposure to low concentrations may result in a range of non-specific features including headache, dizziness, throat discomfort, chest tightness, skin itching and eye irritation and hyperventilation. As hypoxia progresses, patients may experience progressively lower levels of consciousness, seizures, and coma [3, 8]. In this case, the deceased complained of nausea, vomiting, burning sensation over the body, loose stools and dizziness. He developed hypotension and respiratory failure following which there was loss of consciousness and ultimately cardiac arrest. The history, clinical symptoms and autopsy findings in this report were supportive of death due to cyanide poisoning the findings in the respiratory tract were indicative of the inhalational manner of exposure.

This paper aims to make the clinicians aware of occupations using cyanide, so that there is immediate suspicion of a possible cyanide poisoning and provide prompt treatment and care to the patient. Though such cases are rare, the emergency antidotes should be made available at all levels of the healthcare system so that such fatalities are preventable. Also, the forensic pathologist should give due attention to the background and occupation of the deceased while conducting autopsies in cases of poisoning.

Conflict of Interest

None Declared

References


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