Case Report
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A rare case of acute cyanide poisoning from cyanmethemoglobin reagent ingestion

Kolar Vishwanath Vinod*, Kalyappan Anupriya, Vivekanandan M

Dept. of General Medicine, Jawaharlal Institute of Postgraduate Medical Education and Research [JIPMER], Dhanvantrinagar, Puducherry, Pondicherry, India
*drkvv@rediffmail.com

Abstract

Acute poisoning from cyanides frequently gives rise to lethal toxicity and rapid death. Although many cases of acute cyanide poisoning resulting from inhalation of hydrogen cyanide and ingestion of inorganic cyanide salts have been reported, acute poisoning resulting from ingestion of cyanmethemoglobin reagent (also called Drabkin's reagent, contains potassium cyanide) has most probably not been reported so far. Here we report a rare case of acute cyanide poisoning in a 30-year-old female laboratory technician resulting from deliberate ingestion of commercially available, ready to use liquid cyanmethemoglobin reagent used for colorimetric determination of hemoglobin in blood. She made an uneventful recovery with treatment.

Keywords: Acute poisoning, cyanides, Drabkin's reagent, cytochrome oxidase

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1. Introduction

Acute poisoning from cyanides frequently gives rise to lethal toxicity and rapid death. Because of rarity of this poisoning, difficulty in diagnosis and its fulminant course, the mortality is frequently high.Inhalation and ingestion are the most frequent modes of acute poisoning,[1,2] which can result accidentally or from deliberate exposure. Cyanides have gained notoriety because of their reported use during mass executions inside gas chambers in Nazi concentration camps, homicides, suicides and because of their potential to be used in chemical warfare and terrorist acts. Here we report a rare case of acute cyanide poisoning in a 30-year-old female laboratory technician resulting from suicidal ingestion of commercially available,ready to use liquid cyanmethemoglobin reagent used for the colorimetric determination of hemoglobin in blood. The cyanmethemoglobin reagent (also called Drabkin's reagent) contains potassium cyanide, potassium ferricyanide and sodium bicarbonate in 1:4:20parts respectively.

Case report

A 30-year-old woman, working as a laboratory technician, was rushed to our emergency department with alleged suicidal ingestion of ready to use liquid Drabkin's reagent (manufactured by BeneSphera™Diagnostic Solutions, Avantor Performance Materials India Limited, Dehradun, India), about an hour before. She had vomiting 3-4 times on the way to hospital, but there was no history of headache, loss of consciousness, seizures and breathing difficulty. At admission, she was conscious, confused and agitated (Glasgow coma scale score of 12/15) with pulse rate of 116/ minute, blood pressure of 100/70 mm Hg and respiratory rate of 26/ minute. Finger pulsoximetry revealed oxygen saturation of 91%. She was administered high flow oxygen (6l/ minute) and a suspension of 60 g activated charcoal through nasogastric tube. Otherwise, systemic examination was unremarkable. Subsequently she complained of dizziness and burning sensation all over body and again had episodes of vomiting and loose stools.Arterial blood gas analysis showed mild metabolic acidosis and hypoxia (p^H:7.30, P_aO_2:66mm Hg, SaO_2:92%,

* *Author for correspondence*

$PaCO_2$:30mm Hg, serum bicarbonate: 15mEq/l, serum lactate: 1.9 mmol/l and anion gap: 16mEq/l). Laboratory evaluation revealed hemoglobin: 12.7g/dl, leucocyte count: 13,800/µl, platelet count: 386,000/ µl, plasma glucose: 108mg/dl, blood urea nitrogen: 28mg/dl, serum creatinine: 1.2mg/dl, serum sodium: 128mEq/l, serum potassium: 3.6mEq/l and normal liver function. Electrocardiogram was normal, except for sinus tachycardia. Chest X-ray was normal. We could not estimate the serum cyanide levels, because of lack of laboratory testing facility. She received intravenous fluids and hydroxocobalamine 10mg. Close monitoring of vital parameters and oxygenation was continued. After 4 hours, she became fully conscious and oriented and remained hemodynamically stable, with oxygen saturation of 93%. She continued to show gradual improvement in oxygenation and her oxygen saturation normalized after 7 hours.Upon recovery, she admitted to ingestion of about 400-500ml of cyanmethemoglobin reagent. She was kept under observation for further 48 hours and was discharged later in a stable condition, without sequelae.

2. Discussion

Cyanides feature among the most potent and toxic poisons and have been used both for homicidal and suicidal purposes throughout history. The severity and the speed of onset of toxic manifestations depend not only on the compound and the dose, but also on route of exposure. Inhalation of fire smoke and toxic fumes containing hydrogen cyanide and ingestion of sodium and potassium salts of cyanide are the most frequent causes of poisoning.[2,3,4] Inorganic cyanide salts lead to more rapid manifestations compared to organic cyanide compounds. Direct skin contact with cyanide compounds can cause poisoning through cutaneous absorption, which can occur through intact skin. Inhalation of hydrogen cyanide gas leads to manifestations within seconds and ingestion of inorganic salts of cyanides, such assodium and potassium cyanides,causes manifestations within minutes. However, the clinical manifestations remain same irrespective of cyanide compound and route of exposure.

Acute exposure to cyanides results in inhibition of mitochondrial cytochrome oxidase, thereby interfering with oxidative phosphorylation and ATP (adenosine triphosphate) generation.[2] This results in inhibition of oxidative metabolism, intracellular hypoxia and consequently lactic acidosis from excessive anaerobic glycolysis. Because of high reliance on aerobic metabolism,the central nervous system is most frequently involved in acute poisoning, followed by cardiovascular and respiratory systems.The manifestations of toxicity include nausea, vomiting, dizziness, confusion, excitement, hallucinations, seizures, loss of consciousness/ coma, tachycardia or bradycardia, cardiac arrhythmias, hypotension/ shock from myocardial depression, respiratory distress, non-cardiogenic pulmonary oedema and profound respiratory depression.[2,4]

Cyanide poisoning, either accidental or intentional, is more frequently encountered in persons engaged in occupations such as dyeing, photography, pest control, mineral refining, printing and electroplating, extraction of gold and silver from low grade ores and firefighting,because of occupational exposure and ease of access to cyanides. Chemists and jewellers who use inorganic cyanide salts for gold polishing are also prone for cyanide poisoning.[4,5,6] The index patient, being a laboratory technician, had easy access to cyanmethemoglobin reagent. To the best of our knowledge, this is probably the first report of cyanide poisoning resulting from deliberate ingestion of cyanmethemoglobin reagent. Although acute cyanide poisoning from potassium ferricyanide is less likely, potassium cyanide in the Drabkin's reagent can cause fatal toxicity, when consumed in sufficiently high doses. The potassium cyanide content in Drabkin's reagent is around 50mg /l. Based on studies of intentional and accidental poisonings, the average calculated lethal oral dose of cyanides in humans at the time of death was1.40mg/ kg and lowest fatal oral dose reported is 0.54mg/kg (calculated as hydrogen cyanide).[1]The index patient had mild poisoning, probably because she ingested sublethal dose of potassium cyanide.

Although cyanide antidote kit,[4,7]comprising of amyl nitrite, sodium nitrite and sodium thiosulfate is an effective antidote useful in severe poisoning, it is not readily available all over India. Amyl nitrate is to be administered emergently by inhalation, followed by sequential intravenous administration of the latter two. Hydroxocobalamine in high doses of 5g, given by intravenous infusion, has been approved as an effective and safer antidote for cyanide poisoning.[4,8] Because of its efficacy and safety, urgent empiric treatment with hydroxocobalamine has been advocated in suspected cases of acute cyanide poisoning, as treatment delays may prove

costly.[4]The latter antidote is also not readily available in the prescribed high doses. Serum lactate level has been reported to be useful for diagnosis as well as assessment of severity in acute cyanide poisoning.[9]Close monitoring of vitals and aggressive supportive care in intensive care unit are also crucial for successful outcomes.

To conclude, cyanmethemoglobin reagent ingestion can rarely be a cause of acute cyanide poisoning and emergency care physicians should be aware of diagnosis and management of acute cyanide poisoning. Measures should be taken to ensure ready access to the two orphan antidotes throughout the country.

3. Conflict of Interest

None Declared

4. References

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