Case Report

Acute cyanide Intoxication: A rare case of survival

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ABSTRACT

A 30-year-old male jewellery factory worker accidentally ingested silver potassium cyanide and was brought to the emergency department in a state of shock and profound metabolic acidosis. This patient was managed hypothetically with use of injection thiopentone sodium intravenously until the antidote was received. Cyanide is a highly cytotoxic poison and it rapidly reacts with the trivalent iron of cytochrome oxidase thus paralysing the aerobic respiration. The result is severe lactic acidosis, profound shock, and its fatal outcome. The patient dies of cardio-respiratory arrest secondary to dysfunction of the medullary centres. It is rapidly absorbed, symptoms begin few seconds after exposure and death usually occurs in <30 min. The average lethal dose for potassium cyanide is about 250 mg. We used repeated doses of thiopentone sodium till the antidote kit was finally in our hands, hypothesising that it contains thiol group similar to the antidote thiosulphate. Moreover, it is an anticonvulsant. We were successful in our attempts and the patient survived though the specific antidotes could be administered after about an hour.

Key words: Cyanide, cytochrome oxidase, cytotoxic, metabolic acidosis, thiopentone

INTRODUCTION

Cyanide is a highly cytotoxic poison[1] and its intoxication may be acute or chronic. Acute intoxication is lethal if not immediately diagnosed and treated within 30 minutes. Mode of poisoning is usually inhalation of smoke, absorption from contaminated skin; accidental oral ingestion is less common. Population at risk include those working in photography, electroplating, chemical, plastic industries and those involved in polishing of gold and silver jewellery. Diagnosis is difficult and is usually based on circumstantial evidence. It is mandatory for emergency department of each hospital as well as the employer to be well equipped with antidote kit and first aid provision for timely management of such cases. We are reporting a case of acute cyanide intoxication managed with symptomatic support and administration of intravenous thiopentone sodium till the antidote kit was available which took almost 45-50 minutes.

CASE REPORT

A healthy male aged 30 years working in a jewellery factory was brought to the emergency department approximately 10 min after accidental ingestion of cyanide. On admission, he was in severe shock. He was immediately resuscitated and put on mechanical ventilation. SpO_2 did not rise beyond 60% in spite of intermittent positive pressure ventilation with 100% oxygen.

Arterial blood gas (ABG) report revealed high anion gap metabolic acidosis and injection sodium bicarbonate was given stat and infusion was started. Electrocardiogram (ECG) showed global ST depression pattern with right bundle branch block (RBBB).

Meanwhile, the attendants confirmed the substance ingested was silver potassium cyanide used to polish metal plates required for artificial jewellery. The source of poisoning was the beverage (tea), which was kept open nearby thus unknowingly got contaminated. Its antidotes were not available at any of the nearby pharmacy and patient was not improving. The patient's attendants were asked to go to the same factory he was working in, for seeking the antidote kit.

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Suddenly patient started having convulsions hence injection thiopentone sodium was administered intravenous (iv). To our surprise patient's SpO_2 started improving and reached 90% then again started falling after 5-7 min. We repeated injection thiopentone sodium every10 min to maintain SpO_2 around 90-92%. Total dose of 450 mg was needed over a period of 45-50 min. Patient's vitals were stable. There were no convulsions. By this time attendants were back with the antidote kit. Amyl nitrite inhalation was given via endotracheal tube for 15 s with intermittent interval of 15 s of 100% oxygen which was followed by administration of sodium nitrite 300 mg at 5 ml/min (10 ml) followed by sodium thiosulphate 12.5 g iv stat slowly (25 ml).

Patient's condition started stabilising: SpO_2 improved to 96%, systolic blood pressure (B.P.) to 100-110 mm Hg. And mean B.P. to 70-80 mm Hg and patient regained consciousness with slight drowsiness.

After 2 h ABG was repeated and there was marked improvement in metabolic acidosis. Repeat ECG showed normal ST segment but persistence of RBBB.

After 24 h patient was haemodynamicaly stable, was given weaning trial and was extubated. Patient was able to maintain SpO_2 100% on oxygen support by mask and was conscious, oriented. He was shifted to ward, 48 h after admission to Intensive Care Unit and sent home after 5 days of admission to hospital.

DISCUSSION

Cyanide is a rapidly acting and highly toxic chemical and its acute intoxication is fatal.[1] The outcome depends on the dose ingested and the availability of the antidote. Common sources of acute cyanide poisoning include smoke inhalation during the burning of rubber, plastic, silk etc., Occupational hazards are common in people working in industries such as photography, chemical research, synthetic plastics, metal processing, electroplating, gold and silver industries, jewellery polishing etc.[2]

Cyanide is a highly cytotoxic poison. It rapidly reacts with the trivalent iron of cytochrome oxidase thus paralysing the mitochondrial electron transport chain and aerobic respiration. The result is severe lactic acidosis, profound shock and a fatal outcome.[3] The patient dies of cardio-respiratory arrest secondary to dysfunction of the medullary centres. It is rapidly absorbed, symptoms begin a few seconds after exposure and death usually occurs in <30 min. The average lethal dose for potassium cyanide is about 250 mg.[4]

Cyanide poisoning is difficult to detect. The most important clue to diagnosis is the circumstantial evidence, rather than the signs or symptoms. Person's breath can smell like bitter almonds, but this is not easy to detect and is not conclusive. Serum cyanide levels though difficult to assess confirm the diagnosis. In our case, the history given by his coworkers and the symptoms were clue to the diagnosis.[5]

The hallmark in the treatment of acute cyanide intoxication is administration of specific antidotes. It is available as a kit which contains the following: Amyl nitrite, sodium nitrite, and sodium thiosulphate.[6] Sodium thiosulphate acts by binding to the mitochondrial enzyme rhodanase thus facilitating conversion of cyanide to thiocyanate. A newer antidote being used is hydroxocobalamin which, combines with cyanide and forms cyanocobalamin. In animal models hydroxocobalamin is synergistic with thiosulphates. Although not 100% successful, these antidotes can often prevent the cyanide from further poisoning the victim. Multiple antidotes are available worldwide. The most widely used are nitrites with thiosulphate.[7] Due to toxicity of the nitrites, there is always a search for safer alternative. The recent studies have shown that combination of hydroxocobalamine with thiosulphate is considered a better choice.[8-10]

Although no literature is available regarding use of thiopentone in treatment of cyanide intoxication, we hypothetically used it and could sustain the patient until antidote kit was available. We chose thiopentone [Figure 1] because of its anticonvulsant effects, reduced cerebral blood flow and cerebral metabolic rate of oxygen consumption. Although it contains thio group, it is not similar to the cyanide antidote thiosulphate as is evident in their chemical structure. Sodium thiosulfate contains a sulphane sulphur moiety [Figure 2] (a divalent sulphur bound only to another sulphur moiety) and the availability of such is the rate-limiting step in the enzymatic conversion of cyanide to much less toxic and renally-excreted thiocyanate (by endogenous rhodanase). No such

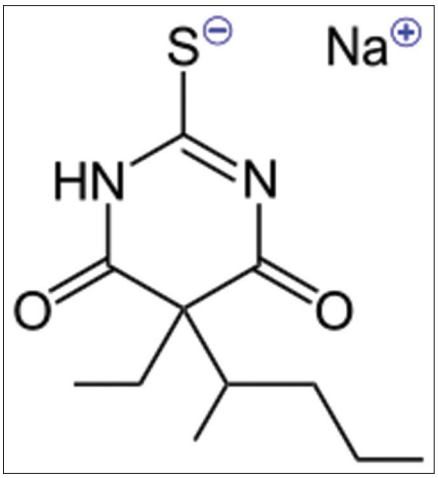

Figure 1: Thiopentone sodium

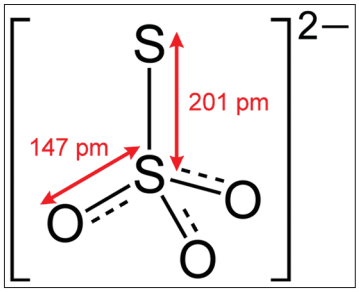

Figure 2: Thiosulphate ion

moiety is found in thiopentone sodium. Thus the mechanism may not be related to the sulphur moiety of thiopentone.

It is important to note that cyanide is rapidly acting poison and once signs and symptoms appear, death ensues within 30 min. However, we received the antidote kit after around 60 min. Patient fully recovered and was sent home hale and hearty.

CONCLUSION

Control of convulsions with thiopentone can have a role in sustaining victims of acute cyanide intoxication until the definitive antidotes arrive. Employees routinely working in at-risk industries using cyanide must be informed of the hazards from exposure to the contaminants and should be trained in appropriate procedure in handling the cyanide. Antidote kit should always be available at the place of cyanide exposure and should be sent to the emergency department along with the patient.

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