CASE REPORT

A CASE OF SODIUM CYANIDE POISONING IN A YOUNG MALE

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ABSTRACT

Cyanide poisoning is frequently lethal, because of the early onset of severe symptoms and difficulty in the diagnosis. But early institution of supportive care, especially in mild to moderate poisoning can be life-saving. We describe one such case of a 26 year old male with history of ingestion of concentrated (98%) sodium cyanide tablets, used in the cleaning and electroplating of jewellery. The antidote was not available commercially, and early supportive care was therapeutic. Clinicians should be aware that cyanide is used commercially in a wide array of industries and although it is not easily available on retail, prompt and correct diagnosis and treatment of poisoning can be beneficial, even in the absence of antidotes. This case demonstrates that mild to moderate cyanide poisoning can be treated with supportive care.

Keywords: Sodium Cyanide Poisoning, Antidote

INTRODUCTION

Cyanide is traditionally known as a poison and has been used in mass homicides, in suicides and as a weapon of war. It was used as an agent of genocide in gas chambers by the Nazis during World War II. Sodium and potassium cyanide salts are widely used in many industries like ore extracting processes for the recovery of gold and silver, electroplating, case-hardening of steel, dyeing, printing, photography and in the synthesis of organic and inorganic chemicals (e.g., nitriles, amides, esters and amines) and the production of chelating agents. Industry widely uses nitriles as solvents and in the manufacturing of plastics. Nitriles may release HCN during burning or when metabolized following absorption by the skin or gastrointestinal tract. A number of synthesized (eg, polyacrylonitrile, polyurethane, polyamide, urea-formaldehyde, melamine) and natural (eg, wool, silk) compounds produce HCN when burned.

Acute cyanide poisoning in humans is rare and is predominantly caused by smoke inhalation from fires and much more rarely by intentional ingestion of cyanide salts as in suicide or homicide attempts (1,2). Other sources of exposure could occur by accidental contact in laboratory workers and iatrogenic exposures of the antihypertensive drug Sodium Nitroprusside, which is 44% cyanide by molar weight. Cyanide groups released from the nitroprusside molecule enzymatically, serve as potential cyanogenic agents.

Symptoms of cyanide poisoning can occur within seconds of inhalation of hydrogen cyanide gas, within minutes following oral ingestion of cyanide salts, and within hours of organic cyanide ingestion. Sodium and potassium salts of cyanide are lethal to adults in doses of 150 – 200 mg.(3,4) Because of the early onset of severe symptoms and difficulty in diagnosis, cyanide poisoning is frequently fatal.

A situation of panic often develops if a case of cyanide poisoning is reported to a hospital. Although effective antidotes are available, general supportive measures should not be ignored and may be life-saving.

CASE REPORT

A 26 year old previously healthy male was brought to the emergency ward with alleged history of ingestion of sodium cyanide tablets. He was employed in the cleaning of jewellery in an electroplating industry, which involved the use of sodium cyanide tablets (of very high purity – 98%) besides hydrochloric acid and some other chemicals. According to co-workers, he had accidentally ingested a small amount of sodium cyanide about 1½-2 hours prior to arrival in the hospital. This history seemed doubtful and ingestion was most probably intentional. Immediately the patient had vomited thrice and started developing agitation and irritable behavior.

On arrival, after a quick physical examination, a nasogastric tube was inserted to collect gastric lavage for anaylsis of the aspirate for cyanide. Activated charcoal suspension was administered and supportive care in the form of I/V fluids and high flow O_2 @ 4L/min was started. Blood samples were taken for arterial blood gas (ABG) analysis and a toxicology consult was requested. Simultaneously, a search for cyanide antidotes (Hydroxycobalamin or Sodium thiosulphate and nitrite) was initiated.

Physical examination showed a confused state with a GCS score of 12 (E3M5V4), P: 126/min, RR: 34/min,

SpO_2 – 94% on room air, BP: 104/66 mm Hg. Pupils were normal sized and equally reacting to light. There was no evidence of cherry red discoloration of his skin, with his dark skin tone, and neither his breath, nor did the aspirate smell of the characteristic bitter almond aroma. The systemic examination was unremarkable. ECG was normal, except for sinus tachycardia. Hb-18.2 gm%, TC-34,400 (N68 L27 E1 M4), PC 2.99 lakh, Na^+ 144, K^+ 3.9, Mg^{++} 2.6 mg% (1.8-2.6), SGPT-54 IU/L (5-45) Creat-1.8 mg%. ABG revealed severe metabolic acidosis (Table-1). Supportive treatment was continued in the form of I/V fluids and oxygen and patient was given I/V Sodium bicarbonate in a dose of 100 mEq over 10 min followed by 20 mEq/hr.

At the end of the first hour, the patient started to develop obtundation, and GCS score decreased to 11 (E2M5V3). P-144/min, RR-40/min, SpO_2 – 89%, BP-110/74 mm Hg. High flow O_2 was increased to 10 L/min and bicarbonate infusion was continued. The search for cyanide antidote kit proved futile as it was not available anywhere. Even the company manufacturing the sodium cyanide tablets was contacted for the antidotes, which was unavailable there as well.

After 2 hours of treatment, patient still had sinus tachycardia (P-120/min, RR-30/min, BP-130/80 mm Hg), but sensorium started improving to a GCS score of 13 (E4M5V4) and SpO_2 increased to 93%. A repeat ABG was ordered (Table-1), which showed improvement in the metabolic acidosis. The bicarbonate infusion was halved to 10mEq/hr.

After 5 hours, patient was fully conscious, P – 90/min, RR – 24/min, BP – 130/80 mm Hg and SpO_2 - 100% on oxygen at 10 lit/min. Bicarbonate replacement was stopped. He was kept under observation for two days. By the second day, oxygen was no more required to maintain SpO_2. His CBC also showed marked improvements – Hb – 15.2 gm%, TC – 14,700 (N76 L19 E3 M3) and PC – 2.03 lakh. With an uneventful stay, he was discharged with stable hemodynamics on the next day.

Table 1 : Serial ABG Analysis (ISTAT1-Abott-USA)

Parameter	(Units)	2 hours (on admission)	4 hours	7 hours
pH		7.074	7.288	7.404
PCO_2	(mmHg)	24.8	39.6	39
PO_2	(mmHg)	71	110	174
HCO_3	(mmol/L)	7.2	19	24.4
BEecf	(mmol/L)	(-23)	(-8)	0
Total CO_2	(mmol/L)	8	20	26
SaO2	(%)	87	98	100
Serum Na^+	(mmol/L)	142	143	145
Serum K^+	(mmol/L)	3.7	4.7	4.2
Ionic Calcium	(mmol/L)	1.19	1.16	1.19
Hct	(%PCV)	54	49	48
Hb	(g/dl)	18.4	16.7	16.3

DISCUSSION

Cyanide has a special affinity for ferric ions found in cytochrome oxidase, the terminal oxidative respiratory enzyme within the mitochondria and an essential catalyst for tissue utilization of oxygen. When cytochrome oxidase (at cytochrome a3) is inhibited by cyanide, cellular respiration is inhibited due to uncoupling of mitochondrial oxidative phosphorylation and histotoxic anoxia occurs as aerobic metabolism becomes inhibited. As anaerobic metabolism continues, there is a lactic acid accumulation, producing severe metabolic effects.(6,7) The tissues with the highest oxygen requirements (brain and heart) are the most profoundly affected.

Although effective antidotes against cyanide (i.e. Hydroxocobalamin and Cyanide Antidote kit) are available, general supportive measures should not be ignored and may be life-saving. According to Jacobs' experience of 104 industrial poisoning cases, the use of specific antidotes is only indicated in cases of severe poisoning with signs of deep coma, with wide non-reactive pupils and respiratory insufficiency with circulatory insufficiency.(5) Aggressive airway management with delivery of 100% oxygen can be lifesaving.(6,8,10) It can also treat concomitant carbon monoxide exposure. Oxygen accelerates the reactivation of cytochrome oxidase and protects against cytochrome oxidase inhibition by cyanide.(11) Even if a patient is unconscious, an antidote does not necessarily have to be administered immediately unless vital signs deteriorate.(9)

Supportive care should include:

- Airway control, ventilation, 100% oxygen delivery (because cyanide causes a decrease in oxygen utilization).
- Crystalloids and vasopressors, as needed, for hypotension.
- Sodium bicarbonate according to ABG and serum bicarbonate level.

CONCLUSION

This report aims to highlight that the prognosis in cyanide toxicity is reasonably good if rapid supportive intervention and effective antidotal therapy are provided. Mild to moderate poisoning can be managed with prompt and effective supportive care. Although the incidence of acute cyanide poisoning is rare, the availability of its antidotes is even rarer. And, in spite of cyanide being a very potent poison, its use in various industries is high.

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