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Novel treatment (new drug/intervention; established drug/procedure in new situation)

Hydroxocobalamin treatment of acute cyanide poisoning with a jewellery-cleaning solution

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Summary

Acute cyanide poisoning by ingestion is often severe and lethal among jewellery industry workers. Clinical experience with hydroxocobalamin alone in severe acute cyanide poisoning by ingestion remains limited. This case concerns a 50-year-old goldsmith who tried suicide by ingestion of a jewellery cleaner solution containing approximately 1.2 g of potassium cyanide. He presented unconsciousness, with severe lactic acidosis and arteriolisation of venous blood gases. Following hydroxocobalamin treatment, neurologic and metabolic disorders rapidly improved. He was discharged home 4 days later, without neurological sequelae. The case reinforces the safety and effectiveness of hydroxocobalamin in acute cyanide poisoning by ingestion.

BACKGROUND

Acute cyanide poisoning by ingestion is often severe and lethal among jewellery industry workers. Therefore, to treat cyanide poisoning efficiently means to wage a battle against time. Clinical experience of the use of hydroxocobalamin alone in severe cyanide poisoning by ingestion remains limited. In this case report, we describe the successful use of hydroxocobalamin as the single antidote treatment of acute cyanide poisoning by ingestion of a jewellery-cleaner solution.

CASE PRESENTATION

A 50-year-old man goldsmith with a past history of depression and alcoholism was transported to the emergency department by ambulance after suddenly collapsing at home. On the day of admission, according to his wife and son, the patient began to complain of nausea and dyspepsia at 01:10. At 02:00, he was found unconscious on the floor of his residence. The emergency medical service was called at 02:10. On arrival, at 02:18, the patient was noted to be comatose and gasping for breath. Systolic blood pressure was 138 mm Hg and pulse rate 46 beats/min. The patient had an unpleasant breath odour. He was intubated and ventilated with 100% oxygen. Gastric lavage was carried out with approximately 30 ml of gastric residue recovered. A bottle of unknown origin that, according to the patient's son, smelled like cyanide was found on the floor near the patient. The patient was transported to the emergency department at 03:05. On arrival, at 03:20, the patient had a Glasgow Coma Scale score of 3, blood pressure at 118/75 mm Hg, pulse rate at 91 beats/min and body temperature at 36.0°C. The pupils were noted to be 6 mm and sluggishly reactive bilaterally. Equal breath sounds were present bilaterally. Intermittent decerebrate posturing was noted.

INVESTIGATIONS

Initial arterial blood gas test revealed pH 7.10, $PaCO_2$ 26.5 mm Hg, PaO_2 613 mm Hg, SaO_2 100%, HCO_3^- 9.9 mmol/l, lactate 20 mmol/l and anion gap 27 mmol/l. Venous blood oxygen saturation measured on the external jugular vein was 100%. Other laboratory data were haemoglobin, 14.5 g/dl; white blood cell count, 16×10^9/l; platelets 275×10^9/l; sodium, 134 mEq/l; potassium, 4.8 mEq/l; chloride, 98 mEq/l; blood urea, 0.13 g/l; plasma creatinine, 10.9 mg/l; glucose 2.79 g/l; serum osmolality, 286 Osm/kg; aspartarte aminotransferase, 98 U/l; alanine aminotransferase, 19 U/l; γ glutamil transferase, 106 U/l; alcaline fosfatase, 112 U/l; and myoglobin 543 ng/ml. Routine urine toxicology screen was negative. Whole-blood cyanide and plasma cyanocobalamin samples were not available. The chest x-ray was unremarkable.

TREATMENT

The patient was initially treated with intravenous sodium bicarbonate (cumulative dose of 450 mEq), with no success. Seizure activity was treated with intravenous diazepam.

Hydroxocobalamin (Cyanokit; Merck Santé, Semoy, France), 5 g intravenous infusion during 30 min, was started empirically at 05:40. (figure 1).

OUTCOME AND FOLLOW-UP

Eight hours after admission, the patient was transferred to the intermediate care unit without any ventilatory, renal or hemodynamic support. Adverse events related with hydroxocobalamin were chromaturia and pink-to-red skin colour discoloration. Chromaturia and skin discoloration resolved within 3 days. The patient was discharged from the hospital 4 days after admission and without neurological sequelae (assessed by routine neurological

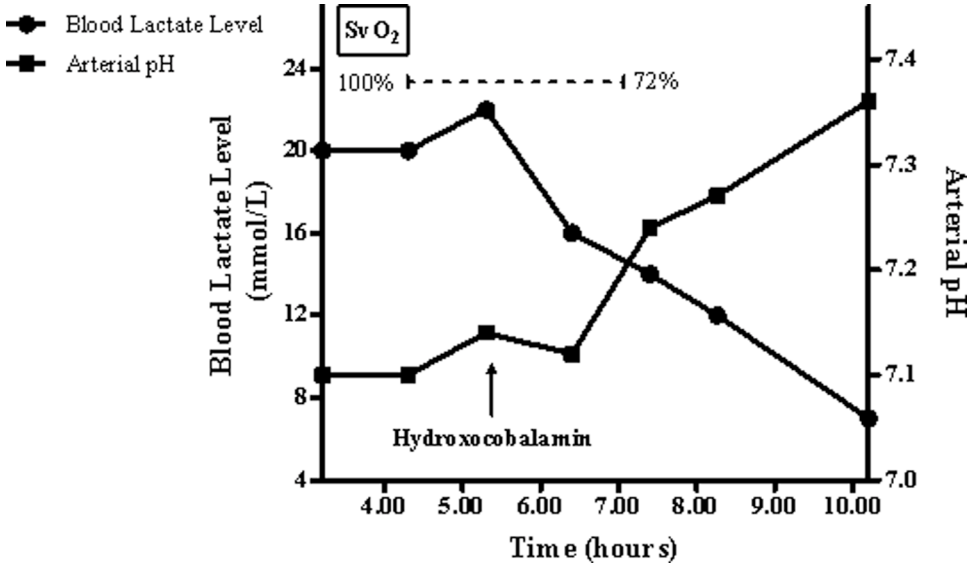

Figure 1 Summary of blood lactate concentrations, arterial pH and SvO_2 values over time. SvO_2, venous oxygen saturation (external jugular vein).

examination). Afterwards, the patient provided us very useful information regarding the dose of potassium cyanide ingested. He and his son were goldsmiths in a jewellery company. They were routinely using a jewellery-cleaner solution containing 80 g potassium cyanide in 5 l of water, for gold-stripping process. On that night, at 01:00, the patient said he filled up an empty bottle with that solution. He acknowledged ingesting approximately 50–100 ml of the bottle content, approximately 1.2 g of potassium cyanide, in a suicide attempt.

DISCUSSION

Cyanide salts are commonly used in the jewellery industry but are not commonly available by the general population in retail markets. Gold-stripping process involves the use of potassium cyanide, hydrogen peroxide (35%) and water. The gold is stripped and made brighter in appearance (1% gold is lost during the stripping process). Almost every company in the gold-manufacturing community, especially in small- and medium-sized operations, uses this process.

Geller and Alsop[1] reported three lethal cases of acute cyanide poisoning by ingestion of a metal polish cleaner solution containing cyanide salts. The patients belonged to a community who had liberal access to cyanide salts. Hydroxocobalamin was not available.

Reports describing suicide attempts through ingestion of cyanide usually do not report the range of supposed ingested dose. Nevertheless, it has been reported that the ingestion of 50–100 mg of cyanide salt is followed by almost instantaneous collapse and cessation of respiration.[2]

Initial clinical manifestations of acute cyanide poisoning are non-specific. A blood-lactate concentration of greater than or equal to 8 mmol/l is a relatively sensitive marker of cyanide poisoning, defined as a blood-cyanide concentration greater than or equal to 39 µmol/l.[3] The presence of lactic acidosis and arteriolisation of venous blood gases may serve as an early clue to the diagnosis of cyanide poisoning.[4] In fact, there is a general consensus that emergent care of the patient should be based on the clinical presentation rather than on blood-cyanide concentrations.[3]

On arrival to the emergency department, our patient presented with deep metabolic acidosis with high anion gap, a blood lactate concentration more than double the level observed in severe intoxications and a venous blood oxygen saturation of 100%. Therefore, though information regarding blood-cyanide levels was not available, the suspicion of severe acute cyanide poisoning was high. Afterwards, it was confirmed by the patient himself.

Hydroxocobalamin is a haeme-like molecule with a complexed cobalt atom. Each hydroxocobalamin molecule binds 1 cyanide ion to form non-toxic cyanobobalamin. It is well tolerated, with no known major toxicities in doses up to 10 g administered to healthy volunteers.[5] Borron *et al*[6] reported a survival rate of 71% in a group of 14 patients diagnosed for acute cyanide poisoning and treated with hydroxocobalamin. In this study, 4 of the 12 patients who ingested a cyanide salt were in cardiac or respiratory arrest and received hydroxocobalamin 0.5–4 h after ingestion. Unfortunately, three of them died.

We report on a case of severe acute cyanide poisoning by ingestion successfully treated with hydroxocobalamin, regardless of the delay of approximately 4.5 h between ingestion and administration. Although we believe supportive care only could have been enough to save our patient, hydroxocobalamin treatment clearly hastened his clinical improvement (figure 1).

The limitations of this case report are both the absence of blood cyanide concentrations determination and the measurement of the quantity of cyanocobalamin eliminated in urine.

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Learning points

- Acute cyanide poisoning requires a high index of suspicion for diagnosis in a patient with unexplained severe lactic acidosis. Physicians should be aware that cyanide is generally available to members of the jewellery industry community, a fact which should be considered while treating those suspected of attempting suicide by consuming cyanide.
- Hydroxocobalamin is a safe and highly effective therapy for acute cyanide poisoning by ingestion. Moreover, this case report supports the existence of a window of time for antidote administration.

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Competing interests None.

Patient consent Obtained.

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