Acute Cyanide Poisoning:
Case Report of the Use of Hyperbaric Oxygen

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Davis FM, Ewer T. Acute cyanide poisoning: case report of the use of hyperbaric oxygen. J Hyper Med 1988; 3(2):103–106.—A 22-yr-old male suffered severe acute cyanide poisoning from self-ingestion. After a poor response to standard methods of care over about 2 h, he underwent hyperbaric oxygen therapy (HBO) at 2.8 ATA. Full consciousness was regained rapidly during the first 20 min HBO. He made an uneventful recovery.

clinical, cyanide, human, hyperbaric oxygen, poisoning

Introduction

Cyanides are among the most rapidly acting poisons and may be absorbed by ingestion, inhalation, or (less rapidly) through the skin. Life-threatening cyanide poisoning is an infrequent condition which, however, may be averted by immediate recognition and prompt therapy with specific measures. The place of antidotes and hyperbaric oxygen therapy (HBO) remains controversial (1). Since HBO has only been reported in a few cases, we present a patient with severe self-inflicted cyanide poisoning in whom it was successfully applied.

Case Report

A 22-yr-old foreman welder was witnessed by his father at about 1730 h to drink half a wineglass of a mixture which he then admitted contained a large quantity of cyanide taken from his electrical engineering workplace. An ambulance was immediately called and he reached the emergency room at 1815 h. On arrival he was deeply unconscious (Glasgow Coma Scale = 5), with fixed, dilated pupils. Hypertonia, hyperreflexia, withdrawal responses to painful stimuli, tachypnea, and tachycardia were present. His airway was well maintained with minimal assistance, and supine blood pressure was 160/80 mmHg. In addition, his face was flushed and edematous, with epiphora, rhinorrhea, and sialorrhea.

Two, 0.3 ml ampuls of amylnitrate were given immediately by inhalation, followed by 100% oxygen administered by mask using an anesthetic circle system. An i.v. line was established, and 300 mg dicobalt edetate, 10 ml sodium nitrite 3%, 50 ml sodium thiosulfate 25%, and 50 ml dextrose 50% were given. Gastric lavage was performed in the left lateral head-down position without

endotracheal intubation, and amorphous carbon was instilled into the stomach. Despite these procedures, there was little improvement in his condition over an hour, at which stage he was transferred to the hyperbaric chamber at another hospital 5 km away. During transfer a further 300 mg dicobalt edetate was administered i.v. On arrival at the chamber at 1945 h he remained unconscious but by this time had reactive pupils and spontaneous limb and head movements, but no vocalization. Withdrawal responses to pain, hypertonia, and hyperreflexia persisted.

Bilateral myringotomies were performed at chamber-side under direct vision, and he was compressed to 2.8 bar on 100% oxygen via a Scott mask, HBO being started at 2010 h. Within the first 15 to 20 min of HBO the patient became fully conscious but rather agitated, complaining of eye and tooth pain. He vomited 3 times during HBO. He settled after i.v. 50 mg demerol, 10 mg, metoclopramide, and 10 mg diazepam. Despite the use of these sedatives and opioids, he was fully conscious and oriented in time, place, and person when he emerged from the chamber at 2124 h, after 70 min HBO. Examination at this time was unremarkable apart from facial edema and a palpable bladder. He was transferred back to the intensive care unit of the referring hospital for observation.

Routine investigations taken at the time of admission revealed an elevated hemoglobin 189 g • liter^{-1}, packed cell volume 0.57, and white count 16.8 × 10^9 • liter^{-1} with a low erythrocyte sedimentation rate at 1 mm • liter^{-1}. His automated biochemistry was normal, apart from a serum potassium of 2.6 mmol • liter^{-1}. Tests were highly positive for cyanide in his plasma and gastric contents, but quantitative assay could not be performed. A post-HBO chest x-ray revealed increased opacification of the right middle and lower zones consistent with early toxic injury or aspiration. These signs had resolved the following day.

He made an uneventful recovery and was discharged after psychiatric assessment. He did not admit to previous attempts of parasuicide, his present attempt appearing to be related to a number of factors, including marital problems, death of a close friend, conviction for a traffic violation, etc., and a final precipitating event of emotional discord with his father. He refused subsequent psychiatric assistance and was able to return to his previous work as a foreman electrician.

Discussion

The clinical toxicology of cyanide has recently been extensively reviewed in detail elsewhere (1, 2) and will not be reviewed here. Cyanide in high dose produces a histotoxic (intracellular) hypoxic poisoning by the binding of cyanide ion to the ferric iron of mitochondrial cytochrome oxidase, inhibiting the reoxidation of cytochrome a, and thus blocking the production of high energy phosphate aerobic metabolism. It also binds with hemoglobin to form

cyanhemoglobin, which cannot transport oxygen, leading to an "oxygen saturation gap" (1).

Specific chemical antidotes are the mainstay of therapy in cyanide poisoning and should be administered early. However, it is important to distinguish trivial symptoms and signs from those of early severe poisoning, since inappropriate or excessive therapy is also hazardous. In New Zealand, standard protocols are recommended by the Department of Health (3), both for immediate first aid treatment in the field and for in-hospital care. While the use of amylnitrite inhalation is recommended for immediate use, it has poor storage properties, is relatively ineffective, and its use in some other conditions causing unconsciousness, such as cerebral hemorrhage, may be lethal.

Dicobalt edetate (Kelocyanor), 300 mg i.v. given over 1 min, is considered the antidote of first choice (3). This may produce hypotension, tachycardia, and sometimes retching. A 2nd dose may be given immediately if there is no or inadequate response to the first. If there is no response to this within 5 min then a 3rd dose may be given. Alternatively, the i.v. combination of 10 ml 3% sodium nitrite given over 2 min followed immediately by 50 ml 25% sodium thiosulfate may be used. In our emergency room, a combination of both therapies with only a single dose of Kelocyanor initially has been used with success for a number of years. For the patient described, this routine was followed by a stomach washout and charcoal instillation to reduce any further absorption of cyanide from the gut. Initial management of this man, however, may be criticized on two counts. First, endotracheal intubation should have been performed for airway protection, since on the subsequent chest radiograph there was evidence of a probable aspiration pneumonitis. Second, the 2nd dose of Kelocyanor should have been given earlier than it was.

It is not clear what the mechanism for a beneficial effect from HBO might be. It is hypothesized that it is based on a cyanide-oxygen interaction at the cytochrome level, possibly combined with enhanced respiratory excretion of cyanide or its metabolites by displacing cyanide from its binding sites (2). In their detailed review of the laboratory and animal data on the role of oxygen, Litovitz et al. (2) concluded that these "demonstrated an unequivocal indication for oxygen administration to all cyanide poisoned patients," but that convincing data on the value of HBO over and above this were lacking. This view was supported by Hall and Rumack (1).

Clinical experience with HBO in cyanide poisoning is limited (2, 4, 5). Trapp (4) reported the successful early use of HBO in reversing coma in 1 patient following no response to amylnitrite and sodium thiosulfate. Litovitz et al. (3) had no success with one deeply comatose woman who had suffered cardiorespiratory arrest and who eventually died. Hart et al. (5) reported on 5 smoke inhalation victims suffering combined carbon monoxide and cyanide poisoning, 4 of whom responded well to HBO.

In our patient, at the time HBO was commenced, nearly 2.5 h had passed since the cyanide ingestion, and, while some improvement in neurologic status had been observed, the patient remained deeply comatose at this time. The rapid return of consciousness once HBO was commenced was in clear contrast to his prior clinical progression up to that point. Although a true cause-and-effect relationship cannot be argued conclusively, the response in this case left little doubt in our minds as to the beneficial effects of HBO.

This raises the question of the current role of HBO in acute cyanide poisoning. We would concur with the view of Hall and Rumack (1) that, where hyperbaric facilities are available, "it would currently seem appropriate to administer HBO to cyanide poisoning victims who do not have an adequate clinical response to supportive measures, 100% normobaric oxygen and antidote." Clearly, further laboratory studies of cyanide-oxygen interactions on cytochrome oxidase are needed, while HBO merits further clinical evaluation in acute severe cyanide poisoning.

References

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