Case Report

Rare Case of Carbon Monoxide Poisoning in a South Indian Town

Sadananda Naik B

ABSTRACT
A case of accidental carbon monoxide (CO) poisoning in a semi urban area of South India is reported here. Carbon monoxide poisoning is a rather uncommon type of poisoning in this region of India.

Key Words: Carbon monoxide, CO, South India, Semi-urban area

Introduction

Carbon monoxide poisoning is one of the most common causes of morbidity and mortality in Western countries such as the United States of America.[1,2] Case may be intentional or accidental, and exposure may be lethal. Approximately 600 accidental deaths occur due to carbon monoxide poisoning every year in the United States.[3] However, such cases are not very common in India, and the few cases that are reported are due to the use of inappropriate room heating appliances kept in the bed room, which may be ill-ventilated with all the windows and doors closed.[4] But CO poisoning is virtually unheard of in the coastal areas which generally have warm weather all the year around. We report a case of accidental carbon monoxide poisoning in a semi-urban area of a coastal region of South India.

The Case

A 20 year-old male, goldsmith by profession, was brought to the emergency department of our hospital early one morning with a history of unresponsive deep sleep. He was habituated to work in his shop late into the night and subsequently sleep there. As he had not responded to repeated knocks and shouts in the morning, his friends had broken open the door only to find him unconscious in the bed. The furnace in the shop was emitting smoke and all the windows were closed.

On examination, the victim appeared to be a well built young male, smelling of smoke. He manifested tachycardia (110/min), and virtually normal blood pressure (130/80 mm Hg). Cyanosis was not apparent. He had tachypnoea with a respiratory rate of 20/min, and was in deep coma. Decerebrate rigidity could be elicited on painful stimuli. His pupils were mildly dilated and reacted sluggishly to light; the fundii were normal. There were no other focal neurological signs. Lungs were clear, and CVS status was normal except for the tachycardia. Investigations revealed a normal blood count, urine examination, blood sugar, and renal and liver parameters. X-ray chest and ECG were also within normal limits. Specific tests for detecting carbon monoxide could not be done owing to lack of such a facility.

The patient was treated as a case of severe CO poisoning, with endotracheal intubation and administration of 100% oxygen. Supportive measures including IV dextrose, mannitol and dexamethasone were undertaken. The patient regained consciousness after one hour, and after a period of three hours he was fully conscious and oriented. Later, he was re-evaluated in detail and demonstrated no neurological or mental aberrations. Supportive treatment and nasal oxygen therapy was continued for another 24 hours, and the patient was then discharged. He was followed up periodically, for a period of three

Consultant Physician, Alvas Health Centre, Moodabidri 574227, Dakshina Kannada, Karnataka, India. Email: sadanandanaik@sancharnet.in

months and did not have any delayed mental or neurological sequelae.

Discussion

Carbon monoxide (CO) is a colourless and nonirritant toxic gas that is usually produced by the incomplete combustion of hydrocarbons. Exposure (suicidal) to the CO in motor vehicle exhaust fumes account for the majority of deaths in the United States, while accidental CO exposure from gasoline powered generators is the second commonest cause.[1] In India, accidents occur in connection with incomplete combustion of wood, charcoal, and coal in ill-ventilated rooms.[4]

Symptoms in CO poisoning range from mild [constitutional symptoms] to severe [coma, respiratory depression, and hypotension].[5] Our patient had severe neurological symptoms. Cardiac manifestations such as angina, arrhythmias, and pulmonary oedema can also occur. However, our patient did not have any cardiac manifestations.The classical findings of cherry red extremities, cyanosis, and retinal haemorrhages were also not seen.[6] Delayed neuropsychiatric syndromes in the form of cognitive and personality changes, dementia, psychosis and parkinsonism etc., after apparent recovery from acute intoxication have been described.[7,8] Over a six-month follow-up period, our patient did not reveal any such abnormalities.

Carbon monoxide toxicity appears to result from a combination of tissue hypoxia and direct carbon monoxide-mediated damage at cellular level. Carbon monoxide competes with oxygen for binding to haemoglobin. The affinity of haemoglobin for carbon monoxide is 200 to 250 times as great as its affinity for oxygen. Because of this, the oxygen-haemoglobin dissociation curve is shifted to the left, leading to impaired release of oxygen at the tissue level and cellular hypoxia.[9]

Because carbon monoxide poisoning has no pathognomonic signs or symptoms, a high index of suspicion is essential for making the diagnosis.[5] Venous samples are adequate for measurement of carboxyhaemoglobin.[10] Pulse oximetry cannot distinguish between carboxyhaemoglobin and oxyhaemoglobin.[11,12]. Measurement of elevated levels of CO in exhaled air of the patient, or in the ambient air at the scene of exposure can sometimes help to confirm the diagnosis.[5] However, these facilities are not routinely available, and clinicians will have to depend on their acumen, circumstantial evidence, and simple tests such as the well known Kunkel's test.[4] In accidental exposures to CO, the victim should be immediately evacuated from the scene of exposure and 100% oxygen should be administered. Hyperbaric oxygen therapy is indicated in patients with deep coma. The half-life of CO is 4 to 6 hours when the patient is breathing room air, 40 to 80 minutes when the patient is breathing 100 % oxygen, and only 15 to 30 minutes when the patient is breathing hyperbaric oxygen.[13] Our patient showed a dramatic recovery following 100 % oxygen administration.

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