Carbon monoxide (CO) poisoning is a significant cause of morbidity and mortality in our country, especially in winter, because of poorly functioning heating systems (1). Because the signs and symptoms of CO poisoning are nonspecific, it is likely that many more cases are unsuspected or attributed to other etiologies and therefore go undiagnosed. It accounts for approximately 3,800 deaths in the United States per annum (1-3). The sources of exogenous CO that cause poisoning include motor vehicle exhaust fumes, poorly functioning heating systems (gas heaters, catalytic gas ovens or stoves), improper use of coal or wood stoves and inhaled smoke. The manifestations of acute CO poisoning are nonspecific and severity of symptoms ranges from mild to severe, such as coma, respiratory depression and hypotension. Coma, confusion, seizures, syncope and death can occur in patients with prolonged or severe CO exposure. Initial symptoms such as headache, dizziness, nausea, vomiting, and malaise may mimic a nonspecific viral illness. In younger children, these effects may be more difficult to recognize (2-4). After apparent recovery from the acute CO intoxication, delayed neurologic and psychiatric symptoms are more frequently reported in adults than children (1,2,4,5). Elevated blood COHb measurements are used to confirm a clinical diagnosis of exposure to CO and, in some instances, assess the severity of poisoning. When CO poisoning is suspected clinically, measurement of blood COHb is typically performed. An elevated COHb level (>2% for nonsmokers and >9% for smokers) strongly suggest exposure to exogenous CO and supports clinical diagnosis of CO poisoning. Many feel that the degree of elevation of COHb level does not correlate well with the patient’s presenting clinical picture and do not use it to direct management (1,3-5). The Undersea and Hyperbaric Medical Society recommends HBO2 therapy for CO-poisoned individuals based upon the clinical severity of illness irrespective of the degree of elevation of their COHb measurements (5-8). We present a case with severe acute CO poisoning.

INTRODUCTION

CO poisoning is a significant cause of morbidity and mortality in our country, especially in winter, because of poorly functioning heating systems (1). Because the signs and symptoms of CO poisoning are nonspecific, it is likely that many more cases are unsuspected or attributed to other etiologies and therefore go undiagnosed. It accounts for approximately 3,800 deaths in the United States per annum (1-3). The sources of exogenous CO that cause poisoning include motor vehicle exhaust fumes, poorly functioning heating systems (gas heaters, catalytic gas ovens or stoves), improper use of coal or wood stoves and inhaled smoke. The manifestations of acute CO poisoning are nonspecific and severity of symptoms ranges from mild to severe, such as coma, respiratory depression and hypotension. Coma, confusion, seizures, syncope and death can occur in patients with prolonged or severe CO exposure. Initial symptoms such as headache, dizziness, nausea, vomiting, and malaise may mimic a nonspecific viral illness. In younger children, these effects may be more difficult to recognize (2-4). After apparent recovery from the acute CO intoxication, delayed neurologic and psychiatric symptoms are more frequently reported in adults than children (1,2,4,5). Elevated blood COHb measurements are used to confirm a clinical diagnosis of exposure to CO and, in some instances, assess the severity of poisoning. When CO poisoning is suspected clinically, measurement of blood COHb is typically performed. An elevated COHb level (>2% for nonsmokers and >9% for smokers) strongly suggest exposure to exogenous CO and supports clinical diagnosis of CO poisoning. Many feel that the degree of elevation of COHb level does not correlate well with the patient’s presenting clinical picture and do not use it to direct management (1,3-5). The Undersea and Hyperbaric Medical Society recommends HBO2 therapy for CO-poisoned individuals based upon the clinical severity of illness irrespective of the degree of elevation of their COHb measurements (5-8). We present a case with severe acute CO poisoning.

KEYWORDS

Unconscious, Acute carbon monoxide poisoning, Hyperbaric oxygen therapy, ICU

CASE REPORT

The 30-year-old male was previously healthy and non-smoker. He was found lying unconscious on the floor. Although his father smelled a pungent odor and felt headache, dizziness, agitation, and dyspnea after entering the room, he had realized that he was apneic and than he gave his mouth-to-mouth respiration for 10 minutes before breathing resumed. He was taken to a local hospital and received oxygen via nasal cannula (10 L/minute) within 30 minutes. First cranial tomography (CT) findings were unremarkable other than brain edema. He was admitted to intensive care unit. Glasgow score was 4. His arterial blood gas (ABG) sample analysis revealed metabolic acidosis and hypoxemia. Carboxyhemoglobin (COHb) level was 59.4 % and electrocardiography showed a mild ST-segment depression over anterior leads, suggestive of myocardial ischemia. HBO2 therapy was immediately initiated within 4 hours after exposure to CO in a multiplace chamber. HBO2 therapy was withheld after completing nine session. His symptoms improved after first HBO2 therapy and COHb level was 23 %. He was discharged on day of 5 and a normal follow-up five weeks after discharge. It has been shown that HBO2 therapy has provided prominent improvement in the early and late effects of CO poisoning and this improvement is more quick and more effective in acute phase.
proven after first HBO therapy and COHb level was 23 %. He could speak and opened his eyes spontaneously 8 hours after the incident, although he still had poor memory. He was oriented, but psychomotor slowness was noted. On day 2 after CO exposure, he complained of severe headache, displayed aggressive behavior, and became confused. Second noncontrast CT of the brain on admission and at day 4 was unremarkable. Routine serum biochemistry, ABG and complete blood counts were unremarkable. He was discharged on day of 5. He had a normal follow-up five weeks after discharge. There was no residual neurologic and psychologic deficit at 3 months.

**DISCUSSION**

CO poisoning is one of the leading causes of injury and death owing to poisoning worldwide. CO poisoning has no pathognomonic signs or symptoms, and a high level of suspicion is essential for making the diagnosis. The most common symptoms in our patients were altered mental state, dizziness, headache, syncope, convulsion, and loss of consciousness (2-4).

Acute CO poisoning is one of the principal indications for HBO therapy. Crush injury, traumatic ischemia, compartment syndrome, necrotizing fasciitis, refractory osteomyelitis, massive air embolism, gas gangrene, purpura fulminans and decompression sickness constitute other accepted indications for HBO therapy (5,7).

HBO therapy is the fastest life saving procedure in acute CO poisoning. It has been reported that its useful in eliminating acute and chronic effects of CO poisoning (6,7). Thom et al (9) in their study with same patient population treated acute and chronical effects of CO poisoning (6,7).

In conclusion, detailed history, physical examination, and suspicion are important for the diagnosis of CO poisoning. It has been shown that HBO therapy has provided prominent improvement in the early and late effects of CO poisoning and this improvement is more quick and more effective in acute phase.

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It has been reported that HBO therapy is more effective within 4-6 hours of the initial exposure to the CO and that optimal frequency is at least two sessions (5-7). The side effects of HBO are related to pressure/volume changes and to oxygen toxicity. Middle ear, sinuses, and lung may be commonly affected by pressure changes, and central nervous system and lung by oxygen toxicity (6).

Weaver et al (7) reported that treatment of adult patients with acute symptomatic CO poisoning with three HBO therapy sessions within a 24-hour period appeared to reduce the rate of cognitive sequel 6 weeks and 12 months later and supported the use of HBO therapy. Yang et al (10) have applied HBO therapy to a 53 years old patient with acute CO poisoning immediately after diagnosis, they made 3 cure and discharged her in 15th days without any sequela. Çekmen et al (11) have applied HBO therapy to a 17 years old patient with acute CO poisoning immediately after diagnosis, they applied totally ten cure and discharged our patient without any sequela. In our case, we also started HBO therapy immediately after diagnosis, we applied totally nine cure and discharged our patient without any sequela.

**REFERENCES**