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Urgent Hyperbaric Oxygen Therapy (HbO2) And Acute Carbon Monoxide Poisoning (Case Report)

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ABSTRACT

Carbon monoxide (CO) poisoning is a significant cause of morbidity and mortality in our country, especially in winter, because of poorly functioning heating systems. We present a case with severe acute carbon monoxide poisonıng. 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 her 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. Glascow score was 4. His arterial blood gas (ABG) sample analysis revealed metabolic acidosis and hipoxemia. 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.

KEYWORDS Unconscious, Acute carbon monoxide poisoning, Hyperbarıc oxygen therapy, ICU

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 nonspesific, 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 nonspesific 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 nonspesific 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 poisonıng 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 exogeneous 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 HBO_2 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 poisonıng.

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. There was no significant finding in his history obtained from family members. He was taken to a local hospital and received oxygen via nasal cannula (10 L/minute) within 30 minutes. First CT findings were unremarkable other than brain edema and he was given one dose of 20 % mannitol. He was admitted to intensive care unit (ICU). Noninvasive monitoring measurements were blood pressure= 160/85 mmHg, heart rate= 149 beats/min, respiratory rate= 32/min, body temperature= 36.7°C. No verbal communication was present. Despite of oxygen administration with 10 L/min, ABG sample analysis revealed metabolic acidosis and hipoxemia with pH= 7.1, PaO2= 48 mmHg, PaCO2= 44.8 mmHg, HCO_3^- mmol/L= 17.4, Bex mmol/L=-9.5, lactate 76 mg/dL. There he was noticed to be metabolic acidosis, which was controlled with HCO_3^- i.v. infusion. His Glascow score was 4, modified Acute Physiology and Chronic Health Evaluation II (APACHE II) score was 26 and multiple organ dysfunction syndrome (MODS) score was 6. COHb level was 59.4 % and electrocardiography showed a mild ST- segment depression over anterior leads, suggestive of myocardial ischemia. Routine chest X-ray, serum biochemistry and complete blood counts were unremerkable. The ophthalmologist's assessment revealed right optic neuropaty with Marcus Gunn pupil of the right eye, which resolved 3 days later. His visual acute started to improve 5 days later and was completely resolved one weeks later, followed by a near-normal visual field after another week. There he was noticed to be confused and irritable, which was controlled with midazolom i.v. infusion. HBO_2 therapy was immediately initiated within 4 hours after exposure to CO in a multiplace chamber. HBO_2 therapy was withheld after completing nine session. His symptoms im-

proved after first HBO_2 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 unremerkable. 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.

DİSCUSSİON

CO poisonıng is one of the leading causes of injury and death owing to poisonıng worldwide. CO poisonıng has no pathognomic 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 poisonıng is one of the principal indications for HBO_2 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_2 therapy (5,7).

HBO_2 therapy is the fastest life saving procedure in acute CO poisonıng. It has been reported that ıts useful in eliminating acute and chronical effects of CO poisonıng (6,7). Thom et al (9) in their study with same patient population showed, in the terms of neuropsychological late phase sequela, HBO_2 therapy is much more effective than NBO therapy. In some other studies, it has been noted that regardless of etiology and severity of poisonıng HBO_2 therapy is very useful.

Generally accepted indications of HBO_2 therapy in patients with CO poisonıng are as follows: severe neurologic symptoms on presentation, syncope, continued neurologic symptoms and findings after several hours of NBO therapy, myocardial ischemia and cardiac dysrhythmias, abnormal neuropsychiatric findings, high COHb level, and infants under six months of age with symptoms of lethargy, irritability, and poor feeding (5-7). During pregnancy, high COHb levels (>15-20%), and symptoms of CO poisonıng or evidence of fetal distress were determined as accepted or recommended criteria. Untreated pneumothorax represents an absolute contraindication (6,7).

It has been reported that HBO_2 therapy is more effective within 4-6 hours of the initial exposure to the CO and that optimal freqeuncy is at least two sessions (5-7). The side effects of HBO_2 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 poisonıng with three HBO2 therapy sessions within a 24-hour period appeared to reduce the rate of cognitive sequela 6 weeks and 12 months later and supported the use of HBO_2 therapy. Yang et al (10) have applied HBO_2 therapy to a 53 years old patient with acute CO poisonıng immediately after diagnosis, they made 3 cure and discharged her in 15th days without any sequela. Çekmen et al (11) have applied HBO_2 therapy to a 17 years old patient with acute CO poisonıng immediately after diagnosis, they applied totally ten cure and discharged our patient without any sequela.. In our case, we also started HBO_2 therapy immediately after diagnosis, we applied totally nine cure and discharged our patient without any sequela.

CONCLUSİONS

In conclusion, detailed history, physical examination, and suspicion are important for the diagnosis of CO poisonıng. It has been shown that HBO_2 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.

Declaration of conflicting interest

The authors declare that there are no conflicts of interest.

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Authors' Contributions

All authors contributed to the medical management of the patient and preparation of the manuscript. All authors have read and approved the content of the manuscript.

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