Bratisl Lek Listy 2009; 110 (11)
716 – 718

CASE REPORT

Urgent hyperbaric oxygen therapy (HBO_2) for acute carbon monoxide poisoning

Nedim Cekmen, Ozcan Erdemli

Güven Hospital Department of Anesthesiology and Intensive Care, Ankara, Turkey. **nedimcekmen@yahoo.com**

Abstract: *Background:* A case with severe acute carbon monoxide poisoning is presented the 17-year-old female was previously healthy and non-smoker. She was found lying unconscious on the floor. Although her father smelled a pungent odor and felt headache, dizziness, agitation, and dyspnea after entering the room, he had realized that she was apneic and than he gave her mouth-to-mouth respiration for 10 minutes before breathing resumed. She 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. She was admitted to an intensive care unit. No verbal communication was present. Her Glascow score was 6, modified APACHE II score was 24 and MODS score was 6. Arterial blood gas (ABG) sample analysis revealed metabolic acidosis and hypoxemia with pH 7.2. Carboxyhemoglobin (COHb) level was 51.4 % 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 unremarkable. HBO_2 therapy was immediately initiated within 4 hours after exposure to CO in a multiplace chamber. HBO_2 therapy was withheld after completing ten session. Her symptoms improved after first HBO_2 therapy and COHb level was 24 %. She was discharged on day 4. She had a normal follow-up six weeks after discharge. It has been shown that HBO_2 therapy has provided prominent improvement in the early and late effects of carbon monoxide poisoning and this improvement is more quick and more effective in acute phase *(Ref. 10)*. Full Text (Free, PDF) *www.bmj.sk.*
Key words: acute carbon monoxide poisoning, hyperbaric oxygen therapy, unconscious.

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). CO poisoning is common in the United States, accounting for an estimated 50 000 emergency department visits for diagnosed cases annually. It accounts for approximately 3.800 deaths in the United States per annum. Because the signs and symptoms of CO poisoning are nonspesific, it is likely that much more cases are unsuspected or attributed to other etiologies and therefore go undiagnosed (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. Ýnitial 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). Children may be at greater risk of injury after CO exposure as a result of their high respiration rate, high oxygen metabolism, and immature central nervous system. After apparent recovery from the acute CO intoxication, delayed neurologic and/or psychiatric symptoms are more frequently reported in adults than children (1, 2, 4). Elevated blood carboxyhemoglobin (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 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 for direct management (3–5). The Undersea and Hyperbaric Medical Society recommends hyperbaric oxygen (HBO_2) therapy for CO-poisoned individuals based upon the clinical severity of illness irrespective of the degree of elevation of their COHb measurements (6–8). A case with severe acute CO poisoning is presented.

Güven Hospital Department of Anesthesiology and Intensive Care, Ankara, Turkey

Address for correspondence: Nedim Cekmen, MD, Güven Hospital Department of Anesthesiology and Intensive Care, 72. Sok. 66/4 Emek Ankara, Turkey.
Phone: +90.3122132057, GSM: +90.05337133150

Case report

A 17-year-old female was previously healthy and non-smoker. She was found lying unconscious on the floor. Although her fa-

ther smelled a pungent odor and felt headache, dizziness, agitation, and dyspnea after entering the room, he had realized that she was apneic and than he gave her mouth-to-mouth respiration for 10 minutes before breathing resumed. There was no significant finding in his history obtained from family members. She 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 she was given one dose of 20 % mannitol. She was admitted to an intensive care unit. Noninvasive monitoring measurements were blood pressure= 170/90 mmHg, heart rate=160 beats/min, respiratory rate=30/min, body temperature=36.5 °C. No verbal communication was present. Despite of oxygen administration with 6 L/min, arterial blood gas (ABG) sample analysis revealed metabolic acidosis and hypoxemia with pH 7.2, PaO_2=54 mmHg, $PaCO_2$=34.8 mmHg, HCO_3^- mmol/L=14.6, Bex mmol/L=-11.5, lactate 55 mg/dL. She was diagnosed metabolic acidosis, which was controlled with HCO_3^- i.v. infusion. Her Glascow score was 6, modified APACHE II score was 24 and MODS score was 6. COHb level was 51.4 % 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 2 days later. Her visual acuse started to improve 4 days later and was completely resolved one week later, followed by a near-normal visual field after another week. There she started 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 ten session. Her symptoms improved after first HBO_2 therapy and COHb level was 24 %. She could speak and opened her eyes spontaneously 8 hours after the incident, although she still had poor memory. She was oriented, but psychomotor slowness was noted. On day 2 after CO exposure, she 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. She was discharged on day of 4. She had a normal follow-up six weeks after discharge. There was no residual neurologic and psychologic deficit at 2 months.

Discussion

CO poisoning is one of the leading causes of injury and death from poisoning worldwide. CO poisoning 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 poisoning is one of the principal indications for HBO_2 therapy. Crush injury, traumatic ischemia, compartment syndrome, gas gangrene, necrotizing fasciitis, refractory osteomyelitis, massive air embolism, purpura fulminans and decompression sickness constitute other accepted indications for HBO_2 therapy (6, 7).

HBO_2 therapy is the fastest life saving procedure in acute CO poisoning. It has been reported that ýt is useful in eliminating acute and chronical effects of CO poisoning (6, 7). Thom et al (9) in their study inthe same patient population showed, in the terms of neuropsychological late phase consequences, 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 poisoning HBO_2 therapy is very useful.

Generally accepted indications of HBO_2 therapy in children with CO poisoning 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 poisoning or evidence of fetal distress were determined as accepted or recommended criteria. Untreated pneumothorax represents an absolute contraindication (6, 7).

HBO_2 therapy is only available at a few centers, making distance and transport of unstable patients major concerns with respect to HBO_2 therapy. 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 frequncy is at least two sessions (5–7).

Weaver et al (7) reported that treatment of adult patients with acute symptomatic CO poisoning 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 poisoning immediately after diagnosis, they made 3 courses and discharged her on day 15 without any sequela. In our case, we also started HBO_2 therapy immediately after diagnosis, we applied 10 courses and discharged our patient without any consequences.

The side effects of HBO_2 are related to pressure/volume changes and to oxygen toxicity. Middle ear, sinuses, and lung may be affected by pressure changes, and central nervous system and lung by oxygen toxicity (6).

After apparent recovery from the acute CO poisoning, delayed neurologic and/or psychiatric symptoms may be seen in 2.8–10.7 % of children after a lucid interval of 2 to 51 days. In children, delayed neurologic sequelae include transient deterioration, memory difficulties, decline in school performance, mental retardion, mutism, urinary and fecal incontinence, various motor abnormalities, facial palsy, psychosis, chronic headaches, seizure, and epilepsy (5–8).

Conclusions

In conclusion, especially in winter because of the poorly functioning heating systems, acute CO poisoning is continuing to be an important health problem in adults and children in our country. The spectrum of CO poisoning symptoms may be wide and

variable in children. Detailed history, physical examination, and suspicion are important for the diagnosis of CO poisoning. It has been shown that HBO_2 therapy has provided a prominent improvement in the early and late effects of CO poisoning and this improvement is more rapid and more effective when applied in the acute phase.

References

1. Weaver LK, Valentine KJ, Hopkins RO. Carbon monoxide poisoning: risk factors for cognitive sequalae and the role of hyperbaric oxygen. Amer J Resp Crit Care Med 2007; 176 (5): 491—497.

2. Wolf SJ, Lavonas EJ, Sloan EP et al. Clinical policy: Critical issues in the management of adult patients presenting to the emergency department with acute carbon monoxide poisoning. Ann Emerg Med 2008; 51 (2): 138—152.

3. Raphael JC. Acute carbon monoxide poisoning. Rev Prat 2008; 58 (8): 849—854.

4. Choi IS. Carbon monoxide poisoning: Systemic manifestations and complications. J Korean Med Sci 2001; 16: 253—261.

5. Hampson NB, Hauff NM. Carboxyhemoglobjn levels in carbon monoxide poisoning: do they correlate with the clinical picture? Amer J Emerg Med 2008; 26 (6): 665—669.

6. Stoller KP. Hyperbaric oxygen and carbon monoxide poisoning: a critical review. Neurol Res 2007; 29 (2): 146—155.

7. Weaver LK, Hopkins RO, Chan KJ et al. Hyperbaric oxygen for acute carbon monoxide poisoning. New Engl J Med 2002; 347 (3): 1057—1067.

8. Butler FK Jr, Hagan C, Murphy-Lavoie H. Hyperbaric oxygen therapy and the eye. Undersea Hyperb Med 2008; 35 (5): 333—387.

9. Thom SR, Taber RL, Mendiguren II et al. Delayed neuropsychologic sequelae after carbon monoxide poisoning: Prevention by treatment with hyperbaric oxygen. Ann Emerg Med 1995; 25: 474—480.

10. Yang CC, Ger J, Li CF. Formic acid: a rare but deadly source of carbon monoxide poisoning. Clin Toxicol (Phil) 2008; 46 (4): 287—289.

Received April 20, 2009.
Accepted August 18, 2009.