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Case Report

High-Dose Hyperbaric Oxygen Treatment in A Carbon Monoxide Intoxication Patient Attending Acute Smoke Inhalation and Burn

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Abstract

Carbon monoxide (CO) poisoning is a serious emergency that may result with death in our country. When CO is inhaled, it connects with hemoglobin and forms carboxyhemoglobin, which impairs oxygen transport and metabolism. In acute cases of smoke inhalation, CO and cyanide intoxication are associated with. A 35-year-old male patient weighing 90 kg was evaluated with a diagnosis of acute smoke inhalation from burns caused by fire in the office. We applied the recommended treatment modalities of up to three sessions per day for the CO intoxication patient admitted to our clinic. He was an acute smoke inhalation case exposed to long-term CO poisoning due to the intervention of a delayed fire. Applying early and high doses of HBO treatment reduces the possibility of sequels.

Keywords: Carbon monoxide poisoning, hyperbaric oxygenation, smoke inhalation injury

Introduction

Carbon monoxide (CO) poisoning is a serious emergency that can result in death in our country. When CO is inhaled, it binds to hemoglobin and forms carboxyhemoglobin (COHb), causing the degradation of the oxygen transport metabolism. It leads to hypoxic symptoms that reduce the amount of arterial oxygen. These symptoms are often headache, nausea and vomiting, dizziness, weakness, malaise, and mental disorders (1). Acute smoke inhalation is often associated with respiratory tract burns. CO and cyanide toxicity are also seen in patients. Hyperbaric oxygen (HBO) therapy is a procedure in which a patient whose body is fully taken in the cabin inhales 100% oxygen under a pressure higher than 1 atmosphere pressure (2). This therapy reduces the half-life of COHb in the body. In some experimental studies, the removal of CO from cytochrome oxidase was demonstrated by increasing the partial pressure of oxygen, thereby reducing lipid peroxidation of the brain and inhibiting leukocyte adhesion (3). The formation of sequelae in the central nervous system (CNS) is prevented in this way through HBO therapy. HBO therapy should immediately be started in these patients, and complications and sequelae such as barotrauma, pneumothorax, and oxygen toxicity should be carefully observed during therapy.

Case Report

A 35-year-old 90 kg male was brought to the emergency service at 05:00 with a diagnosis of burnt and acute smoke inhalation due to a fire at work. The patient had remained in the fire for approximately 2 h and was unconscious. His systolic/diastolic blood pressure was 100/50 mmHg. His pulse was filiform and 85/min, and his fever was 37°C. He had pupillary anisocoria, and his arms and legs were in flask style. His respiration was superficial and tachypneic (29/min). His deep tendon reflexes were total hypoactive, Glasgow Coma Scale (GCS) was 5 (E1, V1, and M3), and blood gas parameters were pH, 7.29; COHb, 38%; pO2, 32 mmHg; and HCO3, 21.5 mEQ/l. There were first-degree burns on his face and bilateral hands. According to the rule of nines, there was a first-degree burn at a rate of approximately 12%. Endotracheal intubation was applied; his chest X-ray was normal, and his pre-treatment brain CT was normal, except for edema. He was immediately taken for HBO therapy unconscious at 06.00 after providing consent (Figure 1). The treatment protocol selected was 2.8 ATA of 100% oxygen for 120 min. The conservative approach was adopted in the mechanical ventilator settings with a suspicion of burns in the lungs. The respiratory rate was selected as 14 breaths/min with the mechanical ventilator able to work in a hyperbaric environment, the tidal volume as 450 mL, the inspiration:expiration ratio as 1:2, and the pressure limit as 15 mmH2O. The patient, whose spontaneous breathing started after five sessions of HBO therapy, was extubated, but his sleepiness and headache persisted (Figure 2). His arterial blood gas values after arrival and after five sessions of HBO therapy are shown in Table 1, and comparative GCS values are shown in Table 2. The patient had a GCS of 11 (E3, V3, and M5) after therapy on the first day and was

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Table 1. The detailed comparison of arterial blood gas values in the first 24 h

Arterial blood gas parameters	Before HBO therapy	After five sessions of HBO therapy on the first day
pH	7.29	7.37
pO_2	32 mmHG	63 mmHG
pCO_2	32.7 mmHG	24.5 mmHG
sO_2	29.9%	84.8%
COHb	38%	1.9%
Lactate	6.4 mEQ/l	3.9 mEQ/l
HCO_3	21.5 mEQ/l	27 mEQ/l
Base deficit	-6.9 mmol/l	-2.2 mmol/l

HBO: hyperbaric oxygen

Table 2. The comparison of Glasgow Coma Scales after five sessions of HBO therapy

Glasgow Coma Scale	Before therapy	After five sessions of HBO therapy
Ability to open the eyes (E)	1	3
Verbal response (V)	1	3
Motor response (M)	3	5

HBO: hyperbaric oxygen

Figure 1. The patient was immediately taken for therapy after being intubated. He was unconscious and had a Glasgow Coma Scale of 5

Figure 2. After five sessions, the patient was extubated. He was conscious and cooperative. He had a Glasgow Coma Scale of 11

admitted to the burn unit, where he underwent two sessions of HBO therapy per day; he received 20 sessions of therapy in total and was discharged with a GCS of 15 and without neurological sequelae. In terms of advanced neuropsychiatric findings, there was no pathologic finding in his examination that was controlled 1 month later.

Discussion

The excretion of COHb from the body is accelerated by HBO therapy. The amount of COHb excreted from the body as a result of 90-min normobaric oxygen therapy is approximately one-third the amount excreted in 3 ATA of 90-min HBO therapy. Daniel Mathieu usually recommended 2.5 ATA of 90-min treatment and 1–3 sessions per day (4). The Undersea and Hyperbaric Medical Society reported treatment as 2.4–3 ATA of 100% O_2 for 120 min and suggested repetition every 6–8 h as long as the patient continued recovering (5). A study conducted in 2002 showed that three sessions of HBO therapy in the first 24 h reduced the risk of cognitive sequelae in the 6-week and 12-month evaluations (6). The recommended treatment modalities are generally applied only for patients who are evaluated for CO poisoning, and we apply treatment for up to three sessions per day in patients with CO poisoning. The study by Weaver has been a guide for our treatment application (6). Ours was a CO poisoning patient who was exposed to acute smoke inhalation for a long time and for whom there was delayed intervention because of the fire. The treatment was required to be applied early and the dose was required to be high because poisoning was severe according to the GCS and because it was accompanied by burns and smoke inhalation. Administering oxygen to the patient through a routine mask will take quite a long time for excreting CO. For this reason, the likelihood of early or late-term occurrence of sequelae increases. The early- and high-dose administration of HBO therapy reduces the likelihood of sequelae. The clinical condition of our patient rapidly improved after he showed a positive response to our treatment. While the GCS was 5 when the patient was brought to the emergency service, it increased to 11 at the end of five sessions in the first 24 h. In our center, the patient was monitored and followed up, and neurological examinations were performed. The patient, who was clinically evaluated on the first day after the treatment, was followed up at the burn unit and completed HBO therapy in 20 sessions.

During the treatment of the patient, a risk of CNS oxygen toxicity was assumed. Nevertheless, the patient did not have a tonic–clonic epilepsy seizure, which is a sign of CNS oxygen toxicity during treatment. The indications for CNS oxygen toxicity induced by hyperoxia are clear, but the cause of this condition is not well known; seizures are reversible and do not cause permanent damage (7). This toxicity is rare. Yıldız et al. who used the same HBO therapy modality as ours showed that CNS oxygen toxicity, an extremely rare complication, was encountered three times among 36500 patients, including all patients in their center between 1996 and 2003 (8). Hampson encountered toxicity once in 3338 patients in his center. By modifying the treatment, Hampson continued treating 5 patients out of 6 who had seizures (9). It is appropriate to apply high-dose HBO therapy to patients because intervention against seizures is possible by means of internal assistance.

If our patient had undergone HBO therapy for 2–3 sessions per day, the risk of CNS oxygen toxicity may have decreased and he would be likely to recover, though late. However, in such a case, the possibility of early and late sequelae would have been quite high. The patient who regained consciousness late would have been admitted to the intensive care unit instead of the burn unit. Considering the risk of infection, possibilities of neurological sequelae, and costs of intensive care, the efficacy and benefit of HBO therapy in the patient is clearly seen.

Hyperbaric oxygen therapy also quickly healed the burn wounds of our patient. It accelerates wound healing by increasing the production of oxygen-dependent collagen and has also been shown to prevent the formation of infections in the wound site by reducing the expression of the interleukin-2 receptor, which increases the formation of infections in the burn area (10). The risk of infection decreased in the scar and burn sites of the patient.

Conclusion

There are no plans for 5-day HBO therapy modalities in clinical trials conducted. We believe that the period of clinical improvement and development of the patient should be monitored by performing additional sessions or by increasing the oxygen periods of current sessions in selected patients. In patients with concomitant burns and smoke inhalation, a hyperbaric medical specialist should change treatment plans whenever necessary. The treatment of patients whose treatment findings improve during the treatment process should be extended. The CNS should be monitored via an internal assistant in terms of oxygen toxicity, and the first intervention should be performed if necessary. If the patient does not show any improvement according to the follow-up by the specialist physician during treatment, routine HBO therapy and close follow-up should be continued in the intensive care unit. We can help patients with CO poisoning recover without neurological sequelae by attempting different HBO therapy modalities. For new information, there is a need for multicenter studies, in which large case series are available, with control groups.

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References

1. Hart GB, Strauss MB, Lennon PA, Whitcraft DD. Treatment of smoke inhalation by hyperbaric oxygen. J Emerg Med 1985; 3: 211-5. [CrossRef]
2. Gill AL, Bell CN. Hyperbaric oxygen: its uses, mechanisms of action and outcomes. QJM 2004; 97: 385-95. [CrossRef]
3. Thom SR. Functional inhibition of leukocyte B2 integrins by hyperbaric oxygen in carbon monoxide-mediated brain injury in rats. Toxicol Appl Pharmacol 1993; 123: 248-56. [CrossRef]
4. Mathieu D. Handbook on Hyperbaric Medicine, Springer, The Netherlands, 2006, 253-54. [CrossRef]
5. Hampson NB. Hyperbaric Oxygen Therapy Committee. Hyperbaric Oxygen Therapy: 1999 Committee Report. Kensington, MD: Undersea and Hyperbaric Medical Society; 1999.
6. Weaver LK, Hopkins RO, Chan KJ, Churchill S, Elliott CG, Clemmer TP, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. N Engl J Med 2002; 347: 1057-67. [CrossRef]
7. Plafki C, Peters P, Almeling M, Welslau W, Busch R. Complications and side effects of hyperbaric oxygen therapy. Aviat Space Environ Med. 2000; 71: 119-24.
8. Yildiz S, Ay H, Qyrdedi T. Central nervous system oxygen toxicity during routine hyperbaric oxygen therapy. Undersea Hyperb Med 2004; 31: 189-90.
9. Hampson N, Atik D. Central nervous system oxygen toxicity during routine hyperbaric oxygen therapy. Undersea Hyperb Med 2003; 30: 147-53.
10. Xu N, Li Z, Luo X. Effects of hyperbaric oxygen therapy on the changes in serum sIL-2R and Fn in severe burn patients. Zhonghua Zheng Xing Shao Shang Wai Ke Za Zhi 1999; 15: 220-3.