Treatment of Severe Carbon Monoxide Poisoning Using a Portable Hyperbaric Oxygen Chamber

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We report the first case of suspected carbon monoxide poisoning treated by hyperbaric oxygen therapy by using a portable hyperbaric stretcher. A 40-year-old British man in Kabul, Afghanistan, was found unresponsive in his apartment. Initial treatment consisted of oxygen by mask at a Combat Support Hospital for several hours, with minimal improvement. Operational security and risk prevented his immediate evacuation to the nearest fixed hyperbaric facilities. He was subsequently treated twice using an Emergency Evacuation Hyperbaric Stretcher, according to the *US Navy Diving Manual* treatment Table 9. The patient showed marked neurologic improvement after the first treatment and experienced near complete recovery before eventual evacuation. This case illustrates the practical use of portable chambers for the treatment of suspected cases of carbon monoxide poisoning in an austere environment. [Ann Emerg Med. 2006;xx:xxx.]

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INTRODUCTION

The Emergency Evacuation Hyperbaric Stretcher and Treatment System, manufactured by SOS Limited (London, UK), is a portable, single-occupant hyperbaric decompression chamber primarily used for the emergency treatment of diving injuries and decompression sickness (Figure). We report the first use of this device for treatment of suspected carbon monoxide poisoning.

CASE REPORT

A 40-year-old British man in Kabul, Afghanistan, was found unresponsive in his apartment after being absent from a morning meeting. Elevation was 3,000 feet, ambient temperatures in the region ranged 30°F (-1°C)to 40°F (4°C), and a portable propane space heater was being used to heat the apartment. According to his associates, the patient appeared well without complaint on the evening before his illness and throughout his assignment to the region. There was no history or evidence of trauma or ingestion. The patient had no significant medical history and was taking no medications.

When found, he was transported to a local hospital and subsequently transferred, while receiving high-flow oxygen, to a combat support hospital at Bagram Airfield in Afghanistan (elevation 5,000 ft). At that time, vital signs were normal: blood pressure 123/60 mm Hg, pulse rate 83 beats/min, respiratory rate 14 breaths/min, temperature 98.4° F (36.8°C), SaO₂ 100% on oxygen at 15 L per minute delivered by nonrebreather mask. Physical examination was remarkable for a Glasgow Coma Scale (GCS) score of 7 and pupils that were reactive to light. Diagnostic testing revealed normal serum electrolyte levels, urine toxicology screen results, and noncontrast head computed tomography results. Arterial blood gas showed pH 7.43, paCO₂ 25.8, paO₂ 300 mm Hg, and base excess of -7 mmol/L. CBC count was normal, except for an elevated WBC count of $18.1 \times 10^3/\text{mm}^3$.

Administration of 2 mg of naloxone intravenously did not improve his condition. With other common causes of altered mental status such as alcohol, opiate abuse, uremia, electrolyte disturbance, endocrine disorder, sepsis, trauma, and psychiatric and neurologic disorders considered, carbon monoxide poisoning was the presumed diagnosis. Confirmatory testing for carboxyhemoglobin was not available, and high-flow-oxygen therapy was continued.

The diving medical officer was consulted. After a combined 6 hours of high-flow surface oxygen therapy during ambulance transport and at the combat support hospital, the patient exhibited a GCS score of 9 (Eyes 2 Verbal 2 Motor 5), gross auditory deficits, symmetric 2 to 3 out of a possible 5 in strength in bilateral upper and lower extremities, dysmetria, dysdiadochokinesis, and poor immediate and remote recall. Operational security and risk prevented emergency transport to



Figure. The Emergency Evacuation Hyperbaric Stretcher (length 88.5 in/2.245 m; diameter 23.5 in/595 mm; weight 110 lbs/50 kg) Courtesy of S.O.S. Hyperlite.

the nearest fixed hyperbaric facility. The portable hyperbaric chamber was declared the best available option for immediate treatment (Figure).

After 10 hours of continuous surface oxygen therapy, the patient had persistent neurologic impairment (Table), but he was able to safely cooperate with hyperbaric treatment. The first hyperbaric treatment was abbreviated and a lower dose than originally intended because of patient discomfort at 34 feet seawater pressure, or approximately 2.0 atmospheres absolute. One hundred percent oxygen between 1.6 and 2.0 atmospheres absolute was provided for a total of 40 minutes, with irregular air break periods. At posttreatment examination, the patient was awake but drowsy, with a GCS score of 15. He complained of a frontal headache and difficulty hearing. Physical examination revealed Teed class I middle ear barotrauma (erythema and injection at the handle of the malleus) to bilateral tympanic membranes. The patient continued to receive oxygen by mask.

Reassessment 24 hours after the initial hyperbaric treatment demonstrated persistent deficits. The patient was treated with a complete US Navy treatment Table 9 to a maximum pressure of 2.4 atmospheres absolute, with 3 30-minute oxygen breathing periods separated by 5-minute air-breathing periods.¹ He tolerated the procedure well and exhibited significant neurologic improvement (Table). Subsequent ancillary testing results were unremarkable. The peripheral leukocytosis returned to normal, and initial culture results were negative.

Table. Chronological neurologic assessment of carbon monoxide poisoning victim treated with emergency evacuation hyperbaric

 stretcher.*

Measurement	Presentation to Combat Support Hospital	Pretreatment, Day 1	Posttreatment, Day 1	Pretreatment, Day 2	Posttreatment, Day 2
Time	2:30 рм	10:30 рм	11:30 рм	10 рм	11:45 рм
Level of consciousness	Coma	Lethargic	Drowsy	Alert	Alert
GCS score	7 E2V1M4 ^{\dagger}	11 E3V3M5	15	15	15
Folstein Mental Status Examination	N/A	10/30 [*]	23/30	26/30 (-) 4 Serial 7s [§]	29/30 (-) 2 Serial 7s
Gross auditory testing	Absent	Gross impairment R > L	Subjective Impairment R > L	Mild subjective impairment on right; sensorineural hearing loss, Weber/Rinné	Mild subjective impairment on right
Strength	N/A	2-3/5	4/5	5/5 Subjective right hip flexor weakness	5/5
Coordination testing	N/A	Poor, deficits in finger-nose-finger, heel-shin, and rapid alt. movements	Retarded but improved	Normal	Normal
Sensation testing	N/A	N/A	Left L-2 distribution diminished light touch	Normal	Normal

GCS, Glasgow Coma Scale, R>L, right is greater than left; N/A, not performed; alt., alternating; L-2, second lumbar sensory dermatone.

*Failure to obtain significant improvement in the patient's neurologic status after 10 hours of treatment with 100% normobaric oxygen resulted in the treatment with hyperbaric oxygen and improvement of neurologic status in the clinical variables outlined.

⁺EVM refers to Eyes, Verbal and Motor responses. Alternatively, EVM refers to scores used for Glasgow Coma Scale pertaining to responses for eye opening, verbal and motor skills, respectively.

*Fractions such as 10/30 represent a score out of 30 possible points.

[§]26/30 (-) Serial 7s indicates deductions were made from only the Serial 7 section of the Folstein Mental Status Examination.

DISCUSSION

Carbon monoxide poisoning remains a significant threat worldwide.² Oxygen is the accepted antidote of choice; however, determining which patients should receive hyperbaric oxygen, when they should receive it, and which hyperbaric profile to use remains controversial despite several randomized trials.³⁻⁵ Carboxyhemoglobin's half-life in vivo is reduced significantly with oxygen therapy.⁶ Clinical studies and guidelines support the use of hyperbaric oxygen for treatment of moderate to severe carbon monoxide poisoning. The number of treatments performed and specific treatment table varies by facility, but current literature suggests that patients with moderate to severe carbon monoxide poisoning and associated neurologic manifestations receive greater benefit from hyperbaric oxygen therapy than with normobaric oxygen alone.^{7,8} In both short- and long-term analysis, patients treated with hyperbaric oxygen experienced decreased neurologic sequelae resulting from early hyperbaric treatments.9,10

Impairment of cognitive functioning, including memory, perception, and attention, as seen in this case, is a common effect of carbon monoxide poisoning. A series of 26 emergency treatments with neuropsychiatric testing after a single hyperbaric treatment resulted in notable improvements in cognition with early treatment.¹⁰

Animal studies suggest that hyperbaric oxygen is the most important treatment in the management of moderate to severe carbon monoxide poisoning.^{11,12} This is evident considering the high degree of morbidity associated with late complications seen in victims not receiving hyperbaric oxygen therapy.¹³ Treatment long after the initial insult has anecdotally provided some benefit, but limiting the amount of time to treatment appears to provide immediate improvement in neurologic deficits and may decrease the permanent neurologic sequelae associated with severe exposure.^{8,9,13}

Carbon monoxide poisoning is a clinical diagnosis that is confirmed by laboratory testing. Because carbon monoxide has a relatively short half-life in vivo, the levels obtained in the emergency setting often do not correlate directly with symptoms. Patients may present with very high levels of carboxyhemoglobin and few symptoms. Conversely, they may present with severe symptoms and insignificant elevations in blood carboxyhemoglobin. We believe the history and clinical course in our patient, including the profound improvements witnessed in response to hyperbaric oxygen therapy, support the diagnosis of carbon monoxide poisoning. Although confirmation by carboxyhemoglobin testing was not available, the thorough medical evaluation and history of coma while sleeping in a closed environment with carbon-monoxide-producing hydrocarbon fuel allowed us to conclude that this was the only reasonable explanation for his condition.

There are several methods of administering portable hyperbaric oxygen using equipment such as the Transportable Recompression Chamber System (Cowan Manufacturing Party Limited, Warners Bay, Australia), the Camp D'altitude, and Gamow Bag, to name a few. Most methods, such as the Transportable Recompression Chamber System, are impractical for use by small medical teams because of their relative large size and weight in comparison to the Emergency Evacuation Hyperbaric Stretcher (Figure). The others mentioned are practical for treating altitude-related maladies but cannot achieve significant levels of pressure to allow for a treatment with one of the accepted US Navy Diving treatment tables that are used for the treatment of carbon monoxide poisoning.¹

The Emergency Evacuation Hyperbaric Stretcher is designed primarily for the emergency treatment of diving injuries such as arterial gas embolism or decompression illness. The portable single-occupant hyperbaric chamber provides 100% oxygen by mask to a single occupant at a maximum pressure of 2.8 atmospheres, gauge pressure (3.8 atmospheres absolute). The chamber is made of a flexible composite tube with a protective fabric cover and lift straps. Composite Plexiglas end domes with penetrator plates contain the connections for communications, gas supply, and vent and pressure gauges. It weighs 110 pounds (excluding the gas bottles), and the entire system can be transported by air or ground.¹⁴ Some limitations of the Emergency Evacuation Hyperbaric Stretcher include, but are not limited to, the following: the need for trained operating staff, the absence of inside tender capability, resulting in difficult patient monitoring, the need for continuous replenishment of portable pressurized oxygen and air supplies, and fragility of components, resulting in the need for great care to be taken when assembling and working with the equipment. According to the manufacturer, this case represents the first use of an Emergency Evacuation Hyperbaric Stretcher for treatment of presumed carbon monoxide exposure.

The number of facilities at which treatment for severe carbon monoxide poisoning with hyperbaric oxygen therapy is available is growing in the United States.² During disasters, when the public health risk and incidence of carbon monoxide poisoning are acutely elevated, portable hyperbaric chambers can be mobilized to an area of need to temporize the unavailability of fixed facilities. An excellent illustration is the sudden increase in poisonings in the wake of Hurricane Katrina in September 2005.¹⁵

Appropriate training, expense, and logistic support limit the accessibility to fixed hyperbaric chambers throughout the world. Portable hyperbaric chambers may represent a feasible option for treatment in these circumstances. Use of these devices in underserved regions and in support of medical crisis should be investigated. Proper training in the principles of clinical hyperbaric medicine remains vital to safe use of these devices.

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Short Abstract for Lueken, Heffner & Parks, YMEM We

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