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A Rare Case of Acute Carbon Monoxide Toxicity Mimicking Stroke and Successfully Managed with Hyperbaric Oxygen Therapy

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Abstract

Acute carbon monoxide (CO) poisoning significantly impacts neurological function, stemming from incomplete combustion of carbon-containing materials, posing a substantial risk. Symptoms range from mild headaches to severe neurological complications, complicating diagnosis. Primary treatment involves supplemental oxygen via a nonrebreather mask. Hyperbaric oxygen therapy (HBOT), though debated, initiated within six hours, may enhance carboxyhemoglobin (CO-Hb) elimination and tissue oxygenation, even with decreased CO-Hb levels. Our report outlines the case of an elderly woman who arrived at the emergency department with symptoms initially suggestive of a cerebrovascular event. However, further history and examination revealed indications of acute CO poisoning, likely due to exposure to a charcoal-burning heater. Despite stable vital signs, her CO-Hb and lactates levels were high. Treatment with normobaric oxygen therapy resulted in some improvement, but significant neurological recovery was achieved with HBOT. This case represents the first documented instance of successful HBOT treatment for acute CO toxicity in Oman.

Keywords: Acute carbon monoxide poisoning, Hyperbaric Oxygen Therapy, poisoning, neurological manifestation

Introduction

Acute carbon monoxide (CO) poisoning is a well-known cause of both mortality and morbidity, particularly affecting neurological and cardiological functions. (1,2)CO is colourless, odourless, and extremely toxic, with common sources including engines, non-electric space heaters, furnaces, fire smoke, fuel combustion, vehicle exhaust, and industrial accidents. (1,2)Globally, the incidence of acute CO poisoning varies. In the United States, approximately 50,000 emergency department (ED) visits occur annually, resulting in 400 to 500 deaths.(3) The incidence of CO poisoning in Middle Eastern countries varies, with several studies highlighting the regional burden of this health issue. Saudi Arabia experiences around 1,000 cases of CO poisoning annually, with many cases linked to the use of gas heaters and poor ventilation. (4,5)In Lebanon, a smaller study reported around 200 cases annually, with incidents frequently linked to the use of generators during power outages). (5,6)

CO binds to hemoglobin with an affinity up to 200 times greater than that of oxygen, forming CO-Hb. (1,2)It also attaches to cytochrome c-oxidase in the brain, disrupting mitochondrial function and ATP production. This leads to platelet-neutrophil aggregation and neutrophil degranulation, causing brain inflammation and delayed neurological manifestations. (2,7)By displacing oxygen, CO diminishes the blood's ability to carry and deliver oxygen to tissues, resulting in severe cellular hypoxia and acidosis, especially in critical organs like the brain and heart. Additionally, CO-Hb causes a leftward shift in the oxygen dissociation curve, reducing oxygen release to tissues and exacerbating hypoxia. (1,7,8)Symptoms of acute CO poisoning range from mild headaches to severe, life-threatening complications such as cardiac ischemia, confusion, neurological deterioration, and seizures. Delayed neuropsychiatric sequelae, affecting 10-30% of CO poisoning victims, can manifest as cognitive impairment, psychosis, dementia, and Parkinsonism. (1,2,8)Diagnosis is typically confirmed through blood co-oximetry.(1,2) The primary treatment for CO poisoning involves immediate removal from the contaminated environment and proper airway management. Administering supplemental oxygen, typically through a nonrebreather mask, is a crucial initial step. In severe cases, hyperbaric oxygen therapy (HBOT) may be considered to expedite CO elimination and reduce tissue damage.(1,7)

HBOT, recognized as an effective treatment for acute CO poisoning, operates through various mechanisms. This medical intervention involves inhaling pure oxygen in a pressurized

chamber, and is used to treat conditions like CO poisoning, decompression sickness, non-healing wounds, radiation injury, and certain infections.(9,10) It is typically administered at pressures between one and three atmospheres absolute (ATA), creating elevated pressure and hyperoxia, which produce mechanical and physiological effects. In cases of CO poisoning, HBOT is recommended for patients with severe poisoning, CO-Hb levels of ≥ 25%, and cardiac or neurological symptoms.(3,7,9) It plays a critical role by rapidly eliminating CO from the body, increasing blood oxygen concentration, displacing CO from hemoglobin, and enhancing tissue oxygenation. Research has shown that HBOT administered within 24 hours can reduce cognitive sequelae and long-term neurological deficits.(7,11) In the Sultanate of Oman, currently only two facilities offer HBOT, with considerable experience in managing acute CO poisoning cases. While there have been a few news reports of such cases, unfortunately, there is a lack of case reports or medical literature on CO poisoning in the country.(12,13) This case involves an elderly woman presenting with altered mental status and slurred speech, diagnosed with acute CO toxicity, and successfully treated with HBOT.

Case Report

An 82-year-old woman with a medical history of hypertension, dyslipidemia, and ischemic heart disease presented to the ED with progressively worsening mental status (GCS: 9/15; E2V2M5) and dysarthria, which developed 6 hours before presentation. Her son reported that she used a charcoal-burning heater for warmth for unknown duration and was found nearly unconscious about 2 to 3 meters from the burning-charcoal, with drooling saliva. On arrival at the ED, she was hemodynamically stable (HR: 92/min; SBP:110mmHg; RR: 22/min; SP02: 98% RA) with a temperature of 38.2 degrees Celsius. Neurological examination revealed a drowsy patient with localization of response to painful stimuli, motor weakness (2/5) in all limbs, and normal reflexes. Pupillary reactions were intact. Respiratory examination revealed bilateral equal air entry and bi-basal crepitations, suggestive of pulmonary edema. Systemic examinations were otherwise unremarkable, except for facial puffiness and blister formation around her left eye, with no evidence of burns.

An electrocardiogram (ECG) revealed normal sinus rhythm with nonspecific T-wave inversions in the inferior leads. Venous blood gas analysis showed a CO-Hb level of 33.5% and a lactate level of 3 mmol/L. She was immediately started on high-flow oxygen therapy, along with supportive care including antipyretics and antibiotics. A computed tomography (CT) scan effectively ruled out any acute brain insult, initially considered as a potential diagnosis given

her presentation, while chest X-rays revealed diffuse reticular shadowing indicative of pulmonary edema. Initial blood investigations, including a coagulation profile, were normal, except for a significant elevation in troponin levels (ng/L) compared to baseline [203 (day 0) < 592 (day 0 - 3rd hour) < 748 (day 1) > 553 (day 1 - 12th hour) > 413 (day 2) < 416 (day 3) < 341 (day 4)], suggesting possible type II myocardial infarction secondary to cellular hypoxia. Despite a drop in CO-Hb levels to 9.1% after 2 hours of normobaric oxygen therapy, there was minimal clinical improvement, with vital signs remaining stable (HR: 88/min; SBP:118mmHg; RR: 17/min; SP02: 98% RA). After consulting with a toxicologist, HBOT was initiated for 2.5 hours, resulting in a remarkable improvement in her neurological status (GCS: 15/15; E4V5M6), while maintaining stable vital signs (HR: 85/min; SBP: 115mmHg; RR: 15/min; SpO2: 98% RA). Subsequent blood gas analysis, post-HBOT, showed a drop in CO-Hb level from 9.1% to 1.7%. Additionally, serial troponin levels, initially elevated, began to decline by day 2, as previously mentioned. The patient remained hemodynamically stable, and oxygen therapy was gradually tapered, being discontinued by day 5. The patient was discharged with a plan to continue her regular medications. At the three-month follow-up, she had no cognitive or neurological sequelae and remained hemodynamically stable.

The authors confirm that they have acquired all necessary patient consent forms, including consent from the patient's relatives, to share the patient's clinical information in this report. The patient and her relatives acknowledge that the patient's names and initials will not be disclosed, and every effort will be made to protect her identity, although complete anonymity cannot be assured.

Discussion

This case report details the successful treatment of acute CO poisoning in an elderly woman who exhibited acute neurological and cardiac manifestations. Initially found semi-conscious near a charcoal burner, she exhibited symptoms resembling an acute cerebrovascular event. The other differentials considered include metabolic and endocrine disorders such as hypoglycemia, hyperglycemia, electrolyte imbalances, liver failure, renal failure, and thyroid dysfunction, which were ruled out by initial laboratory tests. Sepsis, meningitis, encephalitis, and systemic infections were excluded based on history, radiological findings, and laboratory markers. Neurologic conditions like non-convulsive status epilepticus, post-ictal states, and increased intracranial pressure, along with psychiatric causes and cardiovascular issues, were also considered and ruled out. The blood gas analysis revealed a concerning CO-Hb level of

33.5%, approaching the fatal concentrations typically ranging from 50-60%. The brain and heart, with their high oxygen demand, are particularly vulnerable to tissue hypoxia induced by acute or chronic CO poisoning. (1,8)Without timely intervention, neurological symptoms of CO poisoning can progress from non-specific symptoms including headache and nausea to severe symptoms including loss of consciousness, seizures, coma, and death.(7,9) Additionally, CO poisoning can cause delayed neurological sequelae (DNS) characterized by cognitive impairment, memory deficits, personality changes, and movement disorders, which may appear days to weeks after the initial exposure.(1,8) Previous study have shown a significant association has been found between CO poisoning and an increased risk of ischemic stroke, with the incidence being 2.5 times higher for those exposed to CO. (14) Notably, half of the CO-poisoned patients developed ischemic strokes before the age of 50. The age-specific hazard ratio (HR) for CO-poisoning to non-CO-poisoning was four times greater in younger populations compared to the elderly, indicating a stronger impact on younger individuals.(14) Studies have shown that hyperbaric oxygen therapy (HBOT) administered within the first few hours following carbon monoxide (CO) exposure significantly reduces the risk of delayed neurological sequelae (DNS) and improves long-term neurological outcomes. (3,10) While HBOT's neuroprotective and therapeutic effects have been well-documented in experimental models, its efficacy in improving cognitive function in neurological disorders remains debated.(15) Some studies report improvements in cognitive function, while others yield inconclusive results.

Our patient developed transient myocardial ischemia evident by non-specific ECG changes and serial rise in Troponin which later improved to her baseline after HBOT. Elevated troponin levels in such cases are predictive of short-term mortality and indicate myocardial injury. Reports suggest that up to one-third of patients with moderate or severe CO poisoning exhibit some degree of myocardial injury includes angina, arrhythmias, and myocardial infarction. (7,8)This is due to the inhibition of oxidative phosphorylation and the direct binding of CO to myoglobin, which has a 60-fold greater affinity for CO than oxygen.(4,8) These interactions can lead to myocardial infarction and cardiac dysfunction, even in the absence of underlying coronary disease. Additionally, CO's impact on mitochondrial respiration could potentially cause myocardial stunning.(1,2,8) The prompt initiation of HBOT in our patient likely played a role in reducing the injury severity, as evidenced by the gradual decrease in troponin levels and marked improvement in neurological status to normal.

Overall, this case emphasizes the importance of early and effective treatment for CO poisoning, highlights the role of HBOT in mitigating neurological and cardiac damage, and raises awareness of the potential long-term risks such as ischemic stroke associated with CO exposure. Further large-scale studies are needed to confirm these findings and optimize treatment protocols.

Conclusion

HBOT can be an effective modality of treatment for acute severe CO poisoning presenting with neurological and cardiac manifestations. This case report adds valuable insights to the existing medical literature by demonstrating successful management with HBOT.

Authors' Contribution

YL and DH conceptualized the work, curated the data. YL, DH, AA and SA drafted the manuscript. DH, AA and SA reviewed and edited the manuscript. DH contributed to resource allocation. AA and SA supervised the work. All authors approved the final version of the manuscript.

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