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A Case Report of Carbon Mono-Oxide Poisoning

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ABSTRACT

According to the Centers for Disease Control and Prevention, carbon monoxide poisoning (COP) causes around 50000 visits to the emergency department (ED) each year in the USA and correlates with roughly 2700 deaths. After carbon dioxide, carbon monoxide (CO) is the 2nd most common toxin in the air. It is odorless, tasteless, and colorless. Because the symptoms of COP are nonspecific, it is frequently misdiagnosed in clinical practice. To avoid potential patient complications and mortality, it is vital for the healthcare professional to recognize and treat COP as soon as possible. This article presents a case study of a patient with CO toxicity after smoking hookah, which had COP, and discusses the pathophysiology, diagnosis, treatment, and nursing care. The electrocardiogram, chest X-ray, and 2-D echocardiography were normal. He was given high-flow O_2 and supportive treatment. He was observed for 6h with no recurrence of his symptoms and no medical treatment. This case demonstrates how CO poisoning's dampening influence can be restored in the short run with appropriate drug therapy and maintained in the long run without it.

Keywords: Carbon Monoxide Poisoning (COP); Carbon Monoxide.

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DIAGNOSTIC TESTING AND EVALUATION

Underdiagnosis in COP is reported by Gulati, Bledsoe and McEvoy, who claim that 30%- 50% of patients seeking therapy for other problems may not be diagnosed with COP. This is because indications are frequently linked to other causes. As a result, getting a complete medical record is critical for detecting COP, especially in individuals who have unclear symptoms. Patients with heating systems or other COP risk factors should be asked about by attentive nurses in critical care.[1] Determining specifically, the place of the onset of symptoms helps identify a CO source, whether in their home, work, or automobile, or during extracurricular activities (such as boating), or when using appliances, such as generator/ home water heaters as car exhaust fumes, and tobacco smoke. COP is differentiated from viral illness by the onset of symptoms in family members. In viral illness, family members develop symptoms serially, while in COP patients exhibit symptoms within a similar time frame. Flu-like symptoms, key features of viral origin such as fever, myalgia, or cough are absent in carbon monoxide poisoning. Diarrhea is also atypical symptoms of COP. Another, key point to ask for diagnosis is whether any domestic pets have changed their behavior. Because pets' foods are different from humans and do not spread infections So, an unwell pet in the same household at the same time could signify COP.[2]

CASE REPORT

A male patient, 55 years old, unknown to be hypertensive or diabetic but he is known case of hyperlipidemia on statin therapy presented to ED with complaint of dizziness and transient numbness of the lower limbs and left upper limb. He admits he was smoking shisha in a closed place with his friends for nearly about 90 min. Then he felt unwell, he left the place and went back home and on arrival he felt fainted and about to lose his consciousness with ascending numbness started from the lower limbs to his left upper limb, which resolved spontaneously after some time. He was shifted to ED by ambulance. On admission, his vitals were stable, and CT brain was normal and neurological examination was normal.

He was put on high-flow oxygen (O2) via a nonrebreather mask and admitted to the hospital. He was discharged with a COHB of 1.9%

Laboratory Findings

His Venous blood gas showed a critically elevated carbon monoxide level at 25%.

Table 1: Case findings

pH Art POC	7.38	7.35–7.45
pCO_2 Art POC	49.8 mmHg	35.0–45.0
pO_2 Art POC	19.9 mmHg	50.0–100.0
HCO_3 (Act) Art POC	28 mmol/L	22-26
BE (B) Art POCT	3 mmol/L	-2.00-2.00
THB Art POC	139 g/L	120–170
O_2 Sat Art POC	52.9%	95.0–99.0
O_2HB Sat Art POC	39.1%	>=95.0
COHB Art POC	25.3	0.0–2.0
HHb Art POC	4.0	
MetHb Art POC	0.7	0.0-1.5
K Art POC	3.3 mmol/L	3.4–5.1
Cl Art POC	105 mmol/L	98–107
Na Art POC	142 mmol/L	136–145

Electrocardiogram: Normal.

Chest X-ray: Normal.

2-D echocardiography: Normal.

TREATMENT CONSIDERATIONS

The 1st line of intervention by the emergency professional should be the initial evaluation of the patient and safety. The 2nd line of intervention should be to lower the COHb level below 5% by administering 100% Normobaric O_2 with high flow. As delivering 100% O_2, Outside ER is difficult, responders must first decide to intubate the patient endotracheally depending on a consciousness level and severity of respiratory distress. Because 100% oxygen delivery outside the operating theater is difficult to achieve, the first responders must decide whether to endotracheally intubate the patient based on their level of consciousness and the severity of their respiratory distress. Oxygen treatment is indicated irrespective of pulse oximetry or oxygen arterial pressure.

Treatment for minor symptoms usually involves four hours of high-flow oxygen, as well as a regular reassessment of symptoms in the ED and discharge to home. Yet, before discharging CO source must be located and other people who might be exposed must be examined. Unsettled complaints, diagnostic results associated with serious toxicity, or other medical or psychosocial reasons for longer surveillance are all admission requirements. In moderate to severe situations of COP, critical care professionals should consider intubation, admission to the intensive care unit for cardiac and neuromonitoring & support, and the likelihood of hyperbaric oxygen treatment.[2,4,5]

HBO2 THERAPY OVERVIEW

HBOT is a well-known yet divisive technique. HBO2 delivers pressurized 100% oxygen at 2–3 times more pressure than that of normal air pressure. The average treatment time is 90 min, although the frequency and period of treatments may vary depending on the regimen. Though 100% oxygen delivery can reduce CO removal from COHb to 80 min, as compared to 4-6 hours in room air. CO removal is further accelerated to 20-30 minutes with HBO2 therapy. During COP, it increases the concentration of dissolved O_2 in blood, reduces intracranial pressure & edema by inducing cerebral vasoconstriction, and inhibits oxidative stress. Animal studies suggest that HBO2 therapy reduces morbidity and mortality by limiting direct cytotoxic effects, lowering the risk of later neurological effects.

Indications for HBO2 Therapy

There are no universal criteria for selecting patients for HBO2 treatment in CO toxicity. However, The Undersea and Hyperbaric Medical Society recommend HBO2 therapy for individuals >36 years old with serious COP, COHb level of more than 25, exposed for 24 hours, as evidenced by the loss of consciousness neurological & CVS dysfunctioning, or severe acidosis. Indications for HBO2 therapy include disrupted cognitive condition, fainting, convulsions, and hypotension. Other relevant considerations are clinical signs that do not settle even after 4 hours of high-flow oxygen therapy via nonrebreather, mentioned in Table 1.[5]

Table 1: A summary of indications for HBO2 therapy in COP

1. Concurrent burns.	4. Pregnancy with COHb levels of more than 15%.	7. History of loss of consciousness.
2. Neurological findings. a. Altered mental status. b. Coma. c. Focal neurological déficits. d. Seizures.	5. Other considerations Cardiovascular compromise (hypotension, ischemia, infarction, dysrhythmia).	8. Metabolic acidosis. 9. Persistent symptoms despite Normobaric oxygen.
3. Extremes of age.	6. Elevated COHb level >25%.	10. Abnormal neuro-psychometric testing results.

In pregnant women, HBO2 therapy should consider more seriously, especially if foetal distress is suspected. COHb > 15% in pregnant ladies indicate serious toxicity. The only absolute contraindication to HBO2 therapy is an untreated pneumothorax.

DISCUSSION AND CONCLUSION

CO poisoning is a possibly deadly clinical situation that is frequently misdiagnosed or overlooked. When patients report to the ER with any severe neurological signs or any non-specific symptoms following hookah smoking, carbon monoxide (CO) toxicity should be considered. CO poisoning is common among hookah smokers, and it can be caused by inhaled smoke from within the water pipe circuit rather than environmental CO poisoning. As a result, smoking in an open setting does not eliminate the risk of CO poisoning. CO binds to Hb 200–300 more tightly than oxygen. So, it inhibits the dissociation of oxygen and entry into peripheral blood causing hypoxia. The half-life of COHb is 60 minutes at sea level by inhaling 100% oxygen compared to

4-5 hours at room air. The patient would be symptomatic for a long period if a proper diagnosis is not made and managed as a stable head injury. Smoking hookah, as well as Shisha nargileh and Arguileh, are one type of waterpipe smoking. According to WHO research, shisha smoking is uncommon in Singapore but prevalent in Mediterranean countries.[3] Water pipe smoking is reported to generate more smoke than cigarette smoking. According to estimates smoke exposure could be as high as 100–200 cigarettes per session.

CO poisoning is difficult to evaluate in the emergency room since the symptoms are often vague. A history of exposure to CO poisoning is, certainly, the most dependable indicator of CO toxicity. It's wise to note that when dizziness arises because of CO poisoning, it usually means a carboxyhemoglobin level of 20% or higher, which is classified as moderately serious.[6] Making a timely diagnosis can aid in speeding up the treatment of such patients. The cooperation of respiratory medicine typically provides the best chance of survival for the patient. The critical care nurse's intervention repertoire must also include patient Safety analysis and education. In this patient, the intervention was made by lowering the COHb level below 5% by administering 100% Normobaric O_2 with high flow.

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