

## CASE REPORT

# Tobacco hookah smoking-induced carbon monoxide poisoning: A case report of non-ambient exposure

Reem Maalem<sup>1</sup>  | Abdulaziz Alali<sup>2</sup> | Saeed Alqahtani<sup>3,4</sup> 

<sup>1</sup>College of Medicine, Alfaisal University, Riyadh, Saudi Arabia

<sup>2</sup>Department of Emergency Medicine, King Faisal Specialist Hospital & Research Center, Riyadh, Saudi Arabia

<sup>3</sup>Department of Emergency Medicine, King Abdulaziz Medical City, Riyadh, Saudi Arabia

<sup>4</sup>Division of Clinical Pharmacology and Toxicology, University of Toronto, Toronto, Canada

## Correspondence

Reem Maalem, College of Medicine, Alfaisal University, P.O. Box 50927, Takhasusi Road, 11533, Riyadh, Saudi Arabia.  
Email: reemoallim@gmail.com

## Abstract

Carbon monoxide (CO) toxicity should be considered in patients presenting to the emergency department with any acute vague manifestations after hookah smoking; furthermore, smoking in an open space does not eliminate the risk of CO toxicity. High-flow supplemental oxygen should be provided immediately while further investigations and management options are considered.

## KEY WORDS

carbon monoxide poisoning, carboxyhemoglobin, hookah smoking

## 1 | INTRODUCTION

Smoking tobacco is common, and it became more approachable through different methods including hookah smoking, which is also known as Narghila or Shisha. It is a famous and flavored method of smoking tobacco through a water pipe where smoke is inhaled through a mouthpiece.<sup>1,2</sup> Up to 400 years ago, hookah has been used especially among the Middle Easterners and North Africans but became an increasingly popular way of tobacco use worldwide, in the recent years.<sup>1,3</sup> The inhaled smoke of a 60-minute session of Hookah contains at least 145 mg of carbon monoxide (CO), that is eight folds the concentration in a single cigarette, and greater quantities of nicotine, tar, chrysene, phenanthrene and fluoranthene.<sup>1</sup> CO binds rapidly to hemoglobin creating carboxyhemoglobin (COHb) in the blood, which is used as a tool to detect its concentration and the extent of toxicity.<sup>2,3</sup> We present a case of significant CO exposure from hookah smoking in an outdoor setting manifested as CO toxicity. We

sought to highlight the implication of hookah smoking as a significant source of CO exposure and toxicity through the inhaled smoke from within the water pipe circuit rather than the ambient CO exposure. Although reporting CO poisoning due to hookah smoking is not new in the medical literature, our case is unique in reporting CO toxicity from an outdoor hookah smoking session. The closed-space environment, the presence of the burning charcoal in the area, and the resultant ambient CO level have been proposed previously as contributive factors in cases of CO poisoning. To our knowledge, this is the first case report of acute CO poisoning in an outdoor hookah smoking.

## 2 | CASE PRESENTATION

### 2.1 | Clinical findings

A 43-year-old man, known to be healthy, presented to the emergency department (ED) in an ambulance with an episode

of confusion and generalized body weakness. The patient was smoking hookah outdoors when he experienced a gradual, diffused headache associated with nausea and one episode of nonbloody, nonbilious vomiting, generalized body aches, and unilateral left side body weakness. The family denied any history of seizure, fever, trauma, facial droop, palpitations, diaphoresis, or chest pain. He denied any alcohol or drug abuse. It was his first time to present with these symptoms. When his confusion subsided, his main complaint was the general muscular pain. He did not have any significant past medical or surgical history. He was not on any medications. His family history was unremarkable for stroke, heart attack, or dyslipidemia. Regarding social history, the patient was an occasional smoker of hookah. The patient has no known allergies and denied any recent travel. On physical examination, the patient was mildly confused with a Glasgow Coma Scale (GCS) of 14. His oral temperature was 36.7°C, heart rate was 71 beats per minute, oxygen saturation was 98% in room air, systolic blood pressure was 134 mm Hg over 67 mm Hg diastolic. His respiratory rate was 22 breaths per min. The patient showed no signs of rash or cyanosis. His capillary refill was <3 seconds. Both pulmonary and cardiovascular examination were within normal limits. The abdomen was soft, lax, and nontender. His neurological examination revealed generalized fatigue and drowsiness, with mild left-sided weakness. Sensation was intact, reflexes were preserved, and no cerebellar disturbance signs.

## 2.2 | Diagnostic assessment

Regarding the history, the patient was an occasional hookah smoker. He was smoking his hookah in a backyard where he gradually started to have the symptoms. The laboratory findings showed Blood Glucose level of 5.8 mmol/L and normal complete blood count results. In regard to venous blood gases, pH was 7.39,  $P_{CO_2}$  was 42 mm Hg,  $P_{O_2}$  was 20 mmHg, and  $HCO_3$  was 25.4 nmol/L. The anion gap was of 11.0 and  $TCO_2$  of 26.7. The patient's COHb level was elevated 38.8%. The reference range of COHb is  $\leq 2\%$  in nonsmokers and  $\leq 10\%$  in smokers.<sup>4</sup> The patient's electrocardiogram (ECG) revealed normal sinus rhythm with no evidence of ischemia. A noncontrast Computed Tomography (CT) of the brain was done and revealed no neurological underlying etiology.

## 2.3 | Clinical course

The patient was diagnosed with CO toxicity secondary to hookah smoking, completing a triad consistent with CO poisoning: (i) symptoms suggestive of CO poisoning such as: headache, the most common presentation, dizziness, fatigue, nausea/vomiting, altered mentation, and loss of consciousness. (ii) History of recent CO exposure (hookah smoking). (iii) Elevated COHb level.<sup>4</sup>

## 2.4 | Therapeutic interventions

The patient refused the Hyperbaric Oxygen (HBO) Therapy; therefore, he was put on high-flow oxygen ( $O_2$ ) via nonre-breather mask and admitted to the hospital. An hour and a half after the initiation of the treatment his vital signs were stable and his VBG showed COHb level of 23.20%, an hour and half later on the same oxygen flow, the patient recorded 14.70% CoHb, 3 hours later it dropped down to 6.40%. The patient gradually regained his muscular power and full awareness; therefore, he was discharged with a COHb of 2.80% after 10 hours of high-flow oxygen.

## 3 | DISCUSSION

Carbon monoxide (CO) poisoning is known as the most common fatal inhaled intoxicating gas. It is odorless and colorless.<sup>4</sup> It binds to several heme proteins, such as hemoglobin (Hgb), and myoglobin in heart and skeletal muscles. Compared to oxygen, CO has 300 folds higher affinity to bind Hgb, which eventually affects the oxygen delivery to body tissues leading to tissue hypoxia despite the normal  $PaO_2$  on arterial blood gases, measure of the dissolved  $O_2$ , and the normal  $O_2$  saturation ( $SpO_2$ ) which is usually calculated from  $PaO_2$  by the regular bedside pulse oximeter. Patients who were exposed to CO are usually asymptomatic until carboxyhemoglobin (COHb) values in the blood reaches 10% in smokers and 2% in nonsmokers.<sup>4</sup> Symptoms of CO intoxication vary in severity, starts with as simple as diffuse headache to loss of conscious. Symptoms mimicking stroke and myocardial infarction might be present. In severe cases, both cardiac and neurological manifestations can occur.<sup>1,2,4,5</sup> Smoking leads to 4.9 million deaths per year globally.<sup>1,3</sup> The duration of smoking, volume of nicotine smoked per puff and the temperature of which the nicotine is being burnt play a role to why hookah smoking has around 10 times the amount of CO combusted compared to a single regular cigarette.<sup>1,2,6</sup> Five minutes of hookah smoking produces four times the CO of an entire cigarette.<sup>6</sup> When measured among hookah smokers in a field study, an average of 38.5 parts per million (ppm) of CO was present with an increase of 300% in CO for their samples, comparing pre- and posthookah session.<sup>7</sup> Furthermore, regarding nicotine, studies have shown that the peak nicotine concentration between hookah and cigarettes were equal, but the duration (45 minutes) of hookah smoking resulted in an increased exposure to nicotine that was almost double the dose of exposure.<sup>6</sup> There is more CO than nicotine in hookah, an approximation of 50 to 1 in comparison to 16 to 1 in cigarettes; therefore, a hookah smoker would be exposed to higher CO concentration in order to get nicotine satisfaction.<sup>8</sup> According to Undersea and Hyperbaric Medical Society, generally in cases where COHb is more

than 25%, hyperbaric oxygen (HBO) therapy is the recommended treatment in the acute setting, despite the controversy regarding its role in preventing delayed neurological sequelae. However, due to the physiologic restraints in pregnant patients and children, lower threshold of COHb level, 15%, is the cutoff for the HBO therapy consideration. Otherwise, and in the absence of cardiac ischemia or significant neurological dysfunction, normobaric oxygen therapy in the mean of high-flow O<sub>2</sub> via nonrebreather mask, is the mainstay of treatment.<sup>3-5</sup> The reliability of the historical data is a major limitation in our case report. It is dependent on the historian at the time of assessment. We do not know if there was any other significant source of CO in the reported outdoor setting, which in our case was in a house backyard.

## 4 | CONCLUSION

Carbon monoxide (CO) toxicity should be considered in patients presented to the emergency department with any acute neurological manifestations or any vague symptoms after hookah smoking. The CO exposure among hookah smokers is significant and can occur through the inhaled smoke from within the water pipe circuit rather than the ambient CO exposure. Therefore, smoking in a non-closed-space environment does not eliminate the risk of CO toxicity. If CO toxicity is suspected; high-flow supplemental oxygen should be provided immediately while further investigations and management options are considered.

## CONFLICT OF INTEREST

None declared.

## AUTHOR CONTRIBUTIONS

RM, AA and SA: the authors have fulfilled the criteria and should qualify for authorship. RM: contributed to the conception and design, data acquisition, drafting the manuscript, and approving the revised and final version of the manuscript. The author has managed the manuscript submission process. AA: contributed to the conception and design, drafting the

manuscript, and approving the revised and final version of the manuscript. SA: contributed to the conception and design, critically revising the manuscript, and approving the revised and final version of the manuscript. The author has supervised the manuscript submission process.

## ORCID

Reem Maalem  <https://orcid.org/0000-0002-3296-8580>

Saeed Alqahtani  <https://orcid.org/0000-0002-8408-9537>

## REFERENCES

1. Aslam H, Saleem S, German S, Qureshi W. Harmful effects of shisha: literature review. *Int Arch Med*. 2014;7(1):16.
2. Zahran FM, Ardawi MS, Al-Fayez SF. Carboxyhemoglobin concentrations in smokers of sheesha and cigarettes in Saudi Arabia. *BMJ*. 1985;291(6511):1768-1770.
3. Ashurst J, Urquhart M, Cook M. Carbon monoxide poisoning secondary to hookah smoking. *J Am Osteopath Assoc*. 2012;112:686-688.
4. Erratum: carbon monoxide poisoning: pathogenesis, management, and future directions of therapy. *Am J Respir Crit Care Med*. 2017;196(3):398-399.
5. Rappard JV, Schönenberger M, Bärlocher L. Carbon monoxide poisoning following use of a water pipe/hookah. *Deutsch Aerztebl Int*. 2014;111(40):674-679.
6. Cobb CO, Shihadeh A, Weaver MF, Eissenberg T. Waterpipe tobacco smoking and cigarette smoking: a direct comparison of toxicant exposure and subjective effects. *Nicotine Tob Res*. 2010;13(2):78-87.
7. Barnett TE, Curbow BA, Soule EK, Tomar SL, Thombs DL. Carbon monoxide levels among patrons of hookah cafes. *Am J Prev Med*. 2011;40(3):324-328.
8. Shihadeh A, Saleh R. Polycyclic aromatic hydrocarbons, carbon monoxide, "tar", and nicotine in the mainstream smoke aerosol of the narghile water pipe. *Food Chem Toxicol*. 2005;43(5):655-661.

**How to cite this article:** Maalem R, Alali A, Alqahtani S. Tobacco hookah smoking-induced carbon monoxide poisoning: A case report of non-ambient exposure. *Clin Case Rep*. 2019;7:1178–1180. <https://doi.org/10.1002/ccr3.2173>