Carbon Monoxide Toxicity after Lighting Coals at a Hookah Bar

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Abstract

Introduction Unintentional non-fire-related (UNFR) carbon monoxide (CO) poisonings continue to account for a significant health and economic burden in the United States. While most of these poisonings are related to faulty central heating or water heaters in private dwellings, less common sources should also be considered when a patient presents with any signs or symptoms suggestive of CO toxicity.

Case Report The authors present a case where a patient was found to have severe CO poisoning, a COHgb level of 33.8 %, after lighting coals for a water pipe called a hookah. The patient was initially unconscious and was found to have electrocardiogram (ECG) changes consistent with cardiac ischemia that resolved following treatment with hyperbaric oxygen therapy.

Discussion In recent years, hookah bars have gained in popularity, especially in urban areas and around college campuses. This was the first case to identify the potential occupational exposure of employees working at hookah bars to CO. Furthermore, the patient's COHb level of 33.8 % was higher than any previously reported in the literature with exposure via hookah pipe. The practitioner should consider CO poisoning in patients who smoke tobacco via a hookah and consider early hyperbaric oxygen therapy in those experiencing significant symptoms.

Keywords Carbon monoxide · Water pipe · Hyperbaric oxygen · Hookah · Shisha

Introduction

In the United States, there is a significant health and economic burden caused by unintentional non-fire-related (UNFR) carbon monoxide (CO) poisonings each year. In 2007, there were greater than 230,000 emergency department visits and over 22,000 hospitalizations, producing an estimated cost of \$26 million from admissions alone [1, 2]. Unintentional CO poisoning is preventable, and yet, each year an average of 439 persons die in the United States due to UNFR CO poisonings [3]. Symptoms of CO poisoning may include headache, dizziness, nausea, vomiting, fatigue and confusion, progress to loss of consciousness, cardiorespiratory failure, and death [1]. Carbon monoxide poisoning can also cause delayed neurological sequelae [4]. A tobacco smoker may have ambient levels of COHb as high as 10 %. There have been previously reported CO poisonings from smoking tobacco via hookah—however, not to the level or symptom severity achieved in this patient who also had accompanying ECG changes, with resolution of symptoms after receiving hyperbaric oxygen (HBO) therapy.

A hookah is a water pipe that originated in India and Persia in the sixteenth century where users smoke tobacco called shisha or narghile. In recent years, there has been a dramatic increase noted in hookah smoking [5]. In 2004, between 200 and 300 new hookah bars had opened in the United States, the majority in big cities or near college campuses [6]. Smoking tobacco out of hookah is perceived as being less harmful than cigarette smoking; however, the water pipe smoker may inhale as much smoke during one session as a cigarette smoker would inhale consuming 100 or more cigarettes [5–7]. Ironically, the first water pipe was invented by a physician as a less harmful method of tobacco use [5]. The practitioner

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must maintain vigilance to identify and aggressively treat these patients.

Case Report

A 24-year-old previously healthy male presented via ambulance after falling unconscious at work. The patient worked at a hookah bar and admitted to lighting approximately 30–40 hookahs filled with flavored tobacco during his shift. The patient would hold a butane lighter next to a standard coal and inhale through the hose to draw the flame across to light the coals. Witnesses at the hookah bar reported that the patient fell to the floor and remained on the ground until paramedics arrived. The patient believed he was on the ground for 10 min. The patient reported nausea, headache, diffuse substernal chest pressure, generalized weakness, and paresthesias of bilateral shoulders upon waking and while in the Emergency Department (ED). He denied smoking tobacco products outside of lighting coals at work. He admitted to lighting more coals than he usually did during a shift and denied prior headaches, episodes of dizziness, near syncope, or syncope while working.

Emergency Department vital signs revealed a temperature of 98.3 °F, pulse rate of 105/min, respiratory rate of 18/min, blood pressure 134/71 mmHg, and pulse oximetry of 98 % on 15 L O₂/min nonrebreather mask. On physical exam, the patient was awake but slow to respond to questions. He was oriented to person, place, and time with a Glasgow Coma Scale of 15. Neurological exam revealed intact cranial nerves and no focal neurological deficits including normal finger to nose test. There were no identifiable injuries from the patient's fall to the ground.

Initial diagnostic studies revealed an abnormal ECG showing normal sinus rhythm, incomplete right bundle branch block (RBBB), left anterior fascicular block (LAFB), and nonspecific ST-T segment changes (Fig. 1). Bedside

glucometer reading was 114 mg/dl. Portable chest radiograph, urine toxicology panel (amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine, opiates, and phencyclidine), serum alcohol, serum chemistry panel, cardiac enzymes, and complete blood count were all unremarkable. An arterial blood gas was drawn demonstrating a COHb level of 33.8 %, pH 7.43, pCO₂ 35.2 mmHg, and pO₂ 530 mmHg.

The patient was started on 100 % oxygen via a high flow, nonrebreather mask upon receipt of the toxic COHb level, 38 min after arrival to the ED. The hyperbaric specialist was contacted, and the patient underwent HBO therapy, approximately 100 min after ED arrival, given severe symptoms of CO toxicity including syncope, persistent chest pain, and ECG abnormalities. Following HBO treatment at 2.4 atmospheres, 100 % FiO₂ for 105 min, the patient reported resolution of headache and chest pain. A subsequent ECG and treadmill stress test demonstrated persistence of the RBBB, but resolution of the LAFB and nonspecific ST-T segment changes (Figs. 1, 2, and 3). The fire department was contacted and obtained normal ambient airborne CO concentrations at the hookah bar, thus suggesting that the patient primarily was exposed via smoking the hookah.

Discussion

Carbon monoxide is an odorless, colorless gas that is produced anytime a fossil fuel is burned. CO causes tissue hypoxia by binding to hemoglobin, forming carboxyhemoglobin (COHb), with 200–300 times more affinity than oxygen; thus, displacing oxygen from hemoglobin [8]. The half life of COHb is 4–5 h at room air, 60 min breathing 100 % FiO₂ at sea level and 30 min with HBO therapy [8]. The majority of UNFR CO poisonings occur as a result of faulty central heating or water heaters in private dwellings [9]. Treatment of CO poisoning remains controversial and is debated in the literature.

Fig. 1 Initial ECG obtained upon arrival

Fig. 2 Subsequent ECG obtained 15 min after initial

A randomized, double-blinded control study demonstrated that HBO therapy reduced the cognitive sequelae measured 6 weeks from symptomatic CO poisoning to 24 % compared to 46 % in the control group [10]. Cognitive functioning tests at 12 months continued to show significant differences between HBO and normobaric oxygen (NBO) treatment groups. Interestingly, many of the patients in the study treated with HBO had normal COHb levels just prior to the treatment, suggesting a mechanism independent of elevated COHb levels at the time of HBO therapy [10]. Further analysis by Weaver et al. has identified several risk factors leading to worse cognitive sequelae, including patient age of 36 years or older, exposure time periods 24 h or longer, loss of consciousness, and COHb levels greater than or equal to 25 %; these patients may benefit most from HBO therapy [11].

The benefits of HBO therapy should be carefully weighed with the risks. The most common side effects are reversible, including myopia, fatigue, headache, and nausea [12, 13]. Other complications, such as barotrauma to the middle ear, sinuses, and lungs as well as oxygen toxicity leading to seizures, pulmonary edema, fibrosis, and hemorrhage have greater morbidity. Fires in the hyperbaric chamber are the

most common fatal complication [12, 13]. Large prospective randomized trials, as well as several expert reviews, have demonstrated no benefit of HBO over NBO therapy for CO poisoning [14–19]. Among these, a recent Cochrane review concluded that present data do not demonstrate a reduction in adverse neurologic outcomes from HBO therapy in patients with CO poisoning [20].

Prior research has linked significant CO toxicity to hookah use. Several case reports described patients with COHb levels ranging from 15.3 % to 31.1 % who were treated with normobaric oxygen therapy [21–24]. One prior case report in which a 16-year-old female had a COHb level of 24.0 % used HBO (2.8 atmospheres for 2 h) [25]. The patient presented here had a COHb level of 33.8 %, higher than any previously reported in the literature with exposure via hookah pipe. ECG changes were also identified in our patient, likely attributed to the cardiotoxic effects of CO that resolved following HBO therapy. Furthermore, this was the first case to highlight the potential occupational risk of CO exposure posed to employees working at hookah bars. To our knowledge, the previously reported CO poisonings from hookah pipes were from patrons smoking tobacco via hookah and

Fig. 3 ECG obtained following hyperbaric oxygen therapy

not employees lighting coals [21–25]. This potential occupational hazard is an area that requires further investigation as hookah bars and use continue to grow in popularity.

This case demonstrates the potential dangers of hookah smoke, as well as the danger posed to employees at hookah bars who light coals by inhaling through the pipe while torching the coal. Further research is needed to delineate the dangers posed to employees at hookah bars required to light coals. The practitioner should maintain a high index of suspicion for CO poisoning in patients with a history of hookah use, and early HBO therapy should be carefully considered in all symptomatic patients found to have CO poisoning.

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