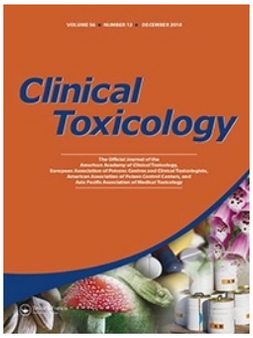

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CLINICAL RESEARCH

Comparison between carbon monoxide poisoning from hookah smoking versus other sources

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ABSTRACT

Introduction: Carbon monoxide exposure is a relatively unknown risk of smoking hookah. Dozens of cases of hookah-associated carbon monoxide toxicity have been described over the past decades, but smoking hookah is generally perceived as safe. Only recently have larger series of hookah-associated carbon monoxide toxicity been published. This study evaluates the incidence of hookah-associated carbon monoxide toxicity over 4 years, and compares the exposures from hookah against other carbon monoxide sources.

Methods: This is a retrospective cohort study of all patients with carbon monoxide toxicity referred for hyperbaric oxygen therapy at an urban hyperbaric oxygen referral center from January 2015 through December 2018. Cases of hookah-associated carbon monoxide toxicity were compared to patients exposed to other carbon monoxide sources, with an analysis of patient comorbidities, symptomatology, and laboratory evaluation.

Results: Over a 48-month period, 376 patients underwent hyperbaric oxygen therapy for carbon monoxide exposure. After exclusions, 265 patients with carbon monoxide toxicity from various sources were analyzed. There were 58 patients with hookah-associated carbon monoxide toxicity (22%). The proportion of hookah-associated carbon monoxide cases increased markedly in the latter years: 2015: 9.5%, 2016: 8.6%, 2017: 24.1%, 2018 41.6%. In the final 2 years analyzed, hookah smoking was the most frequent source of carbon monoxide toxicity referred for therapy. Hookah-associated carbon monoxide patients were younger
(28.1 vs. 45.0 years, mean difference 16.8 years, 95% confidence interval: 11.5, 22.1 years, $p < 0.001$) and more likely to be female (60% vs. 46.6%, $p = 0.06$) than patients exposed to other carbon monoxide sources. The mean difference in carboxyhemoglobin concentration between hookah associated and those exposed to other carbon monoxide sources was 4.6% (mean 20.1% vs. 24.6%, 95%CI: 1.7, 7.5, $p = 0.002$).

Conclusion: A substantial portion of patients with severe carbon monoxide toxicity was exposed through smoking hookah. The incidence of hookah-related carbon monoxide toxicity appears to be increasing.

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KEYWORDS
Hookah; carbon monoxide; hyperbaric oxygen; smoking; tobacco

Introduction

Hookah, the instrument and associated practice of smoking flavored tobacco *via* water pipe, has existed for centuries. Hookah-associated carbon monoxide (HACO) poisonings have been described sporadically in the medical literature; only in recent years have larger case series of HACO been published [1]. While the use of cigarettes, cigars, smokeless tobacco, pipe tobacco, and bidi cigarettes have all decreased among middle-school- and high-school-aged students between 2011 and 2016, the use of electronic cigarettes (e-cigs) and hookahs have increased in both age categories [2]. This phenomenon likely corresponds to the general perception among youths that both e-cigs and hookahs are safer than cigarettes [3].

The hazards of cigarette smoking are well known. Aside from cardiovascular disease, lung cancer, and chronic obstructive pulmonary disease [4,5], cigarette smokers commonly achieve blood carboxyhemoglobin concentrations between 6% and 10% [6,7]. Although the long-term health effects of e-cigs are incompletely described, toxicants and carcinogens exist in e-cigarette solutions, and significant public health concern exists about their effect on smoking prevalence and gateway potential to subsequent use of combustible tobacco products [8]. Hookah smoking, on the other hand, has not raised comparable public health concerns [9].

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Despite the available literature, little is known about the incidence of HACO toxicity. We are unaware of data comparing HACO exposures to conventional CO exposures (nonHACO). Our aim is to examine the differences between HACO and nonHACO cohorts, and describe the trends of HACO toxicity as a percentage of the total cohort referred for hyperbaric treatment over a 4-year span in an urban hyperbaric oxygen (HBO) treatment referral center.

Methods

Overview

We reviewed the institutional database of consecutive patients treated with HBO for CO exposure/toxicity at a 457-bed hospital in the New York City region from January 2015 through December 2018. The hospital serves as the regional burn and hyperbaric center. The study was approved by the School of Medicine Institutional Review Board.

Study population

We reviewed all patients referred for HBO therapy. We included all patients who met the predefined inclusion criteria: carboxyhemoglobin concentration >25%, or carboxyhemoglobin concentration >5% and symptoms consistent with CO toxicity (syncope, seizure, new objective neurologic deficit, ongoing chest pain, or evidence of myocardial damage). We excluded patients from the analysis who were less than 13 years old, who were transferred with an undocumented carboxyhemoglobin concentration, or who had CO exposure but did not meet the predefined criteria for toxicity. Children younger than 13 years were excluded to focus on a population that plausibly could be hookah smokers. Exposed pregnant patients were routinely treated with HBO regardless of carboxyhemoglobin concentrations or symptoms and were therefore excluded from analysis.

Data elements

The retrospective chart review was performed in accordance with methods described by Gilbert et al. [10] and Kaji et al. [11] MS, an emergency medicine resident blinded to the study hypothesis, was trained to abstract predefined variables on a set of "practice" medical records. The abstracted data were entered into a shared electronic form. The following predefined variables were abstracted from the electronic medical record (QuadraMed CPR, QuadraMed Corporation, Plano, TX) for all patients: age, gender, all medical comorbidities, cigarette and tobacco smoking history, co-ingestants, carboxyhemoglobin concentration at arrival hospital, CO exposure source and setting, symptoms, triage vital signs, interpretation of initial ECG, echocardiogram results, peak troponin concentration, peak lactate concentration, complications of HBO therapy, and patient disposition. All carboxyhemoglobin values were obtained from blood gas analysis from the initial presenting hospital. Abstraction performance was monitored by reassessing a 20% random sample. Conflicting or ambiguous data were reconciled through consensus.

Data analysis

Descriptive statistics were calculated for all variables, using means, standard deviations and 95% confidence intervals (CIs). Student's *t* test was used to compare means for continuous data. Chi-square and Fisher exact tests were used to compare the categorical outcomes. Differences between groups are presented as means with corresponding 95% CIs.

Results

Between January 2015 and December 2018, 376 patients underwent HBO therapy for CO exposure. Figure 1 describes the flow of patients through the study. One hundred-eleven

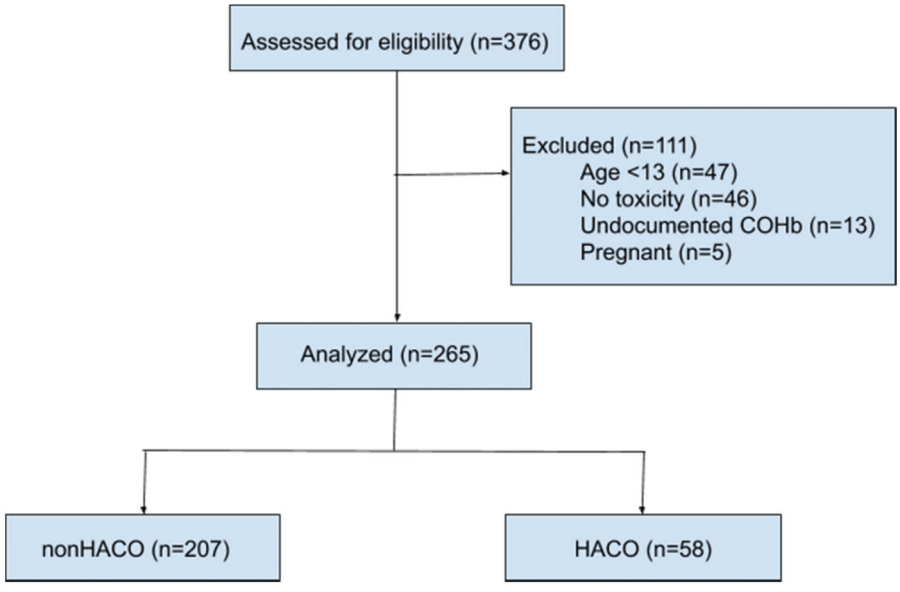

Figure 1. Flow diagram. HACO: hookah-associated carbon monoxide toxicity; nonHACO: carbon monoxide toxicity from other sources.

patients were excluded from the final analysis, with age less than 13 years being the most common reason for exclusion. Two hundred sixty-five patients with CO toxicity were analyzed. Of this group, 58 patients (22%) had HACO toxicity.

HACO patients were younger than non-HACO patients by almost 17 years (28.2 vs. 45.0 years; mean difference 16.8 years, 95%CI: 11.5, 22.1 years, $p < 0.001$; Table 1). HACO patients were generally healthier (i.e., without comorbidities), although this did not achieve statistical significance [HACO, 36/58 (62.1%) vs. nonHACO, 86/173 (50%); $p = 0.103$]. About 60% of the HACO patients were female [35/58 (60.3%) vs. 96/207 (46.4%), $p = 0.06$].

Five (8.6%) of the HACO patients reported working in a hookah bar/lounge at the time of symptom onset (i.e., occupational or ambient exposure). Three of these 5 reported either simultaneously smoking hookah while working, or "priming" the hookah for guests. The carboxyhemoglobin concentrations of these five patients were similar to the remainder of the HACO cohort (carboxyhemoglobin, 19.5%). The location of hookah use was documented in two-thirds of the HACO cases, with 92% of these toxicities occurring in enclosed/indoor environments. Approximately two-thirds of the HACO toxicities occurred in a hookah bar/lounge, and one-third in a home setting.

The percentage of patients referred for HBO who were HACO-related increased markedly in each of the last 3 years of the study period (Figure 2). In the first 24 months analyzed, HACO patients made up 9.1% of patients receiving emergency HBO therapy. The following year in 2017, the percentage increased to 24.1%. In 2018, 41.6% of all patients with CO toxicity were exposed *via* hookah smoking. This represented a nearly 4-fold increase in the number of HACO cases in the first half versus the last half of the study period.

CO exposure from malfunctioning furnaces (23.7%) and fires (22.2%) remained the most common sources of CO exposure in the nonHACO cohort. Unknown source of CO exposure represented 21.3% of the cohort. Hookah smoking represented the third most common source of CO exposure over the entire period analyzed. However, in the final 2 years of analysis hookah smoking became the leading single cause of CO toxicity. In 2018, the number of HACO toxic patients outnumbered documented exposures from malfunctioning furnaces and fires combined.

Patients with HACO toxicity presented to the ED with lower carboxyhemoglobin than non-HACO toxic patients (mean 20.1% vs. 24.6%), with a difference of carboxyhemoglobin of 4.6% (95%CI: 1.7, 7.5, $p = 0.002$). However, CO toxicity from smoking hookah resulted in more neurologic symptoms than toxicity from other CO sources (Figure 3). HACO toxic patients were more likely to suffer syncope [54/58 (93.1%) vs. 146/207 (70.5%), $p < 0.001$] and seizures [6/58 (10.3%) vs. 6/207 (2.9%), $p = 0.016$) compared to nonHACO toxic patients. NonHACO patients were more likely to report shortness of breath or be in respiratory distress [18/207 (8.7%) vs. 0/58 (0%), $p = 0.02$], and more likely to suffer associated burn injuries [16/207 (7.7%) vs. 0/58 (0%), $p = 0.03$] compared to HACO patients. NonHACO patients were also more likely to present with an altered mental status or confusion (AMS), although this did not reach statistical significance [17/206 (8.2%) vs. 1/58 (1.7%), $p = 0.083$]. Because of this difference in respiratory distress and AMS, all intubated patients [nonHACO 59/207 (28.5%) vs. HACO 0/58 (0%)] and nearly all hospitalized patients [nonHACO 87/207 (42%) vs. HACO 1/58 (0%)] were part of the nonHACO cohort.

Seventy-eight percent of intubations were related to fires or smoke inhalation injuries. The remaining intubations were associated with suicides, automobile exhaust, malfunctioning furnaces or generators. Cardiac enzymes and lactates were drawn on a minority of HACO patients. HACO patients were more likely to be cigarette smokers [21/50 (42%) vs. 26/145 (17.9%), $p = 0.001$]. The presence of co-ingestants was recorded in 55% of the HACO cohort, with alcohol being the most common co-ingestant [alcohol 25/32 (78%), marijuana 1/32 (3%), none 7/32 (22%)].

All patients underwent one HBO session of approximately 60 min (4-8 min descent, 46 min of 100% oxygen at 2.8 atmospheres absolute with a 5-minute air break after 23 min,

Table 1. Baseline characteristics.

	nonHACO	HACO	*p* Value
N	207	58	–
Age	45.0	28.2	**<0.001**
Male (%)	53	40	0.06
Smoker (%)	17.9	42.0	**0.001**
Comorbidities (%)			
None	49.7	62.1	0.10
Hypertension	26.4	3.4	**<0.001**
Diabetes	14.4	12.1	0.66
Asthma/COPD	8.0	12.1	0.36
Psychiatric disorder	6.9	5.2	0.64
Dyslipidemia	5.7	1.7	0.21
Seizure disorder	2.3	1.7	0.79

Bolded values represent statistically significance.
HACO: hookah-associated carbon monoxide toxicity; nonHACO: carbon monoxide toxicity from other sources.

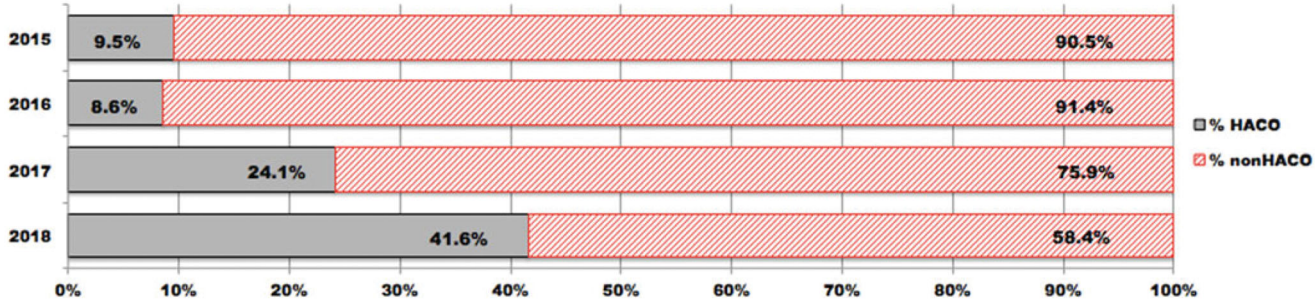

Figure 2. Percentage of hookah-associated carbon monoxide (HACO) toxicity vs. carbon monoxide toxicity from other sources (nonHACO), by year.

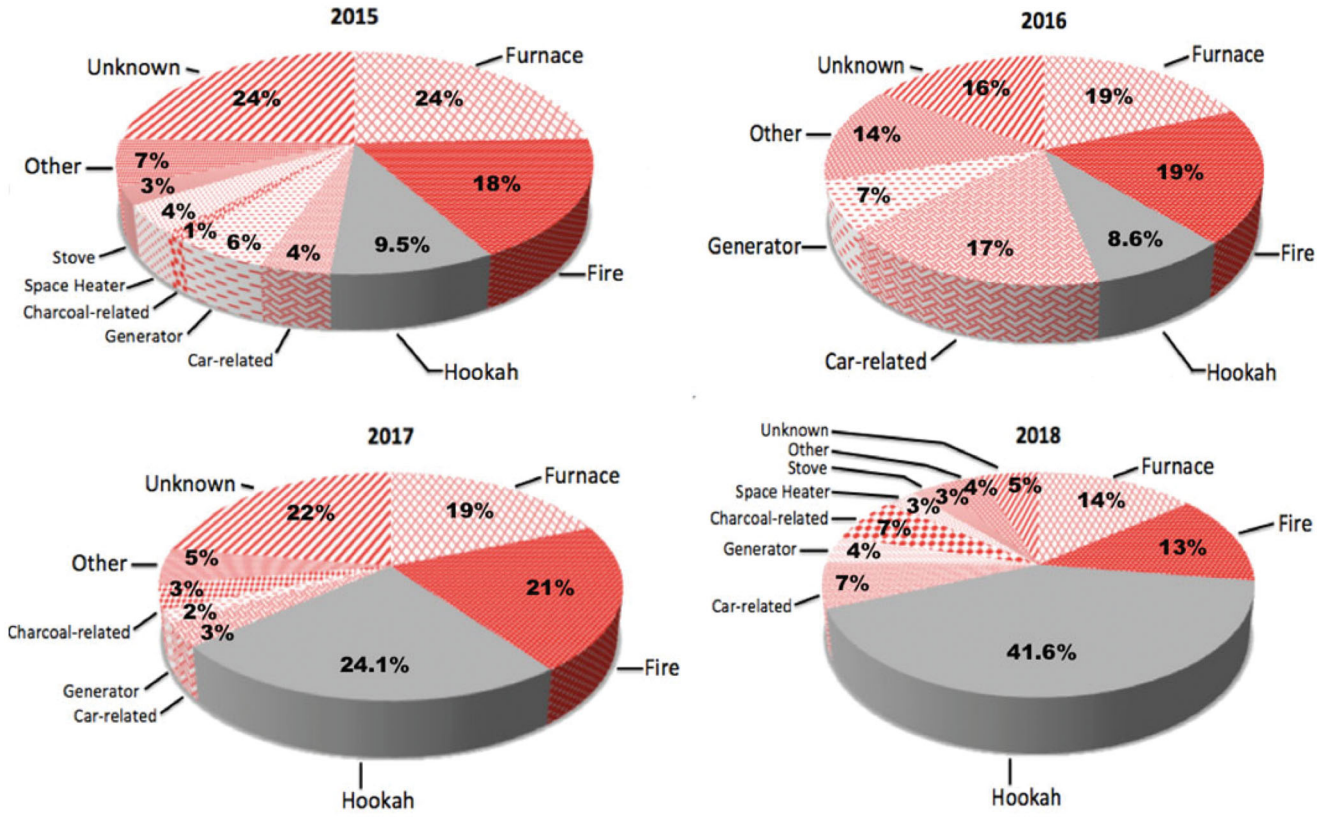

Figure 3. Exposure sources of carbon monoxide toxicity, by year.

4-8 min ascent). No patients reported complications from HBO therapy. Patients were then admitted or discharged depending on the severity of their symptoms. One patient in the HACO group required admission for evaluation of her initial presentation consistent with stroke.

Limitations

All CO toxic patient referrals were consulted by a specially trained, on-call HBO Emergency Medicine physician, and the institutional database included standard CO exposure questions, but we did not use a study-specific questionnaire. It is possible that there are baseline differences between groups not accounted for by the data collected. Drug and alcohol co-ingestants and medication co-exposures were not always documented. It may be clinically important that more of the HACO cohort were cigarette-smokers, with an elevated baseline COHb concentration that would contribute to the overall concentration. About one out of five patients within the cohort had an unknown source of CO exposure. Our data set did not attempt to evaluate the effects of HBO therapy on HACO toxicity.

Nevertheless, citywide HBO referral criteria did not change during the study period. Additionally, since carboxyhemoglobin concentrations are not universally sent on all ED patients, not all CO toxic patients may have been identified. These limitations could shift the prevalence of HACO toxicity higher or lower.

Discussion

In this retrospective analysis conducted at a regional burn and HBO center, we identified a marked increase in hookah-related carbon monoxide toxicity among young, otherwise healthy patients. Hookah smoking has often been compared favorably to cigarette smoking. Over its 4-centuries of use, hookah smoking has not been framed as a significant public health problem [1]. Only 115 cases of HACO poisoning had been reported in the medical literature [12]. Even with scattered subsequent HACO case reports, our 58 cases from a single study site, would contribute to cases in the medical literature by over 50%. This would suggest significant under-reporting and under-recognition.

Whereas cigarette smoking prevalence has continued to decline, hookah smoking prevalence among certain populations is increasing [2,13,14]. In 2016, more than 10% of US middle and high school students reported smoking hookah in their lifetime [15], and an estimated 1 million reported recent past 30-day hookah use [2].

Increases in reported HACO cases likely mirror the increased popularity of hookahs. Cases and small series of HACO had been sporadically reported until 2018 when Eichhorn and colleagues published a 4-year case series from Germany, with 61 HACO patients. However, the total number of CO-poisoned patients was not given in the study, and the incidence of HACO not reported.

Our study contributes one of the largest reports of HACO toxic patients, and is the only to compare characteristics of HACO with nonHACO patients. We report on a considerable increase in HACO incidence, with the number of HACO

patients more than doubling in each subsequent year. In the final year of analysis, more than four out of every 10 patients treated for CO toxicity were exposed from hookah smoking. To contrast, in 2000, a report from Saudi Arabia noted only one out of 24 CO-related intoxications from various origins was due to water pipe smoking [16]. Hookah smoking was the leading cause of CO toxicity at our HBO referral center in the final year analyzed, outnumbering traditional sources such as malfunctioning furnaces, fires, cars, and gas generators. This would suggest a need to devote comparable resources for public health risk characterization posed by this practice and mitigation efforts.

The nearly 4-fold increase in HACO cases between the first half versus the last half of the study may be related to an increase in hookah establishments. In New York City alone, the number of establishments serving hookah in 2017 numbered nearly 400, four times higher than in 2012 [17]. The population of New York City increased by 2.4% in the same period [18].

Studies have attempted to quantify the hazards of hookah smoking relative to cigarettes. Hookahs deliver significantly higher CO levels and hookah smokers absorb more CO than habitual smokers of other combustible tobacco products [19].To contrast, other experiments suggest that the water-filled chamber acts as an efficient filter medium for smoke condensate [20], resulting in a smoke that is much less complex than that of cigarettes [21], and containing far fewer toxic compounds within its smoke [22]. Our study clearly shows that CO is insufficiently filtered.

Carboxyhemoglobin concentrations are lower in our HACO cohort. Our finding that HACO patients present with a nearly five percentage point lower concentration could be due to HACO exposure causing more severe symptoms at lower carboxyhemoglobin values. The difference could also be due to HACO patients presenting to medical care later, or having their carboxyhemoglobin test drawn later in their evaluation. It would seem unlikely, however, that patients who have syncope or seizures would present to medical care in a delayed fashion.

Carboxyhemoglobin concentrations alone are difficult to interpret, and have not been shown to correlate well with patient symptoms or outcomes [23]. Admission concentrations are inaccurate predictors of peak levels, and carboxyhemoglobin half-lives are affected by the CO source, exposure time, and oxygen treatment [23,24].

It is unknown if the pattern of CO exposure associated with HACO toxicity results in delayed neurologic sequelae (DNS). However, we found important differences between the presentation of HACO toxicity and non-HACO toxicity. Whereas nonHACO toxic patients presented with varied symptoms, HACO toxic patients exhibited almost exclusively neurologic symptoms. The central nervous system is the most sensitive to CO poisoning. Syncope and seizures have long been identified as particularly poor prognostic indicators of more severe neuronal damage and risk for DNS [24,25].

HACO toxicity appears to be multifactorial. To evaluate the etiology of HACO toxicity and the reason for its rise, experiments would have to examine (1) product-specific details, (2) patient-specific vulnerabilities, (3) the patient–-product interaction, and (4) combustion source. Product-specific questions should include the smoking material and additives, the heat source, and liquids or substances placed in the base. Charcoal-heated hookah has been shown to produce exhaled CO concentrations 9- to 10-fold greater than electrically heated hookah [24]. Charcoal-heated hookah also induced marked vasodilation compared to electrically heated hookah [24], perhaps elucidating our clinical observations of syncope and seizures in HACO patients.

There may be patient-specific vulnerabilities to developing HACO toxicity. A report of a single patient who developed HACO toxicity on multiple occasions [26] suggests that there may be predispositions to HACO toxicity. While female gender may be protective in CO poisoning, depending on age [27], gender difference in hookah-related morbidity has not been previously noted, and our report shows a trend toward more females developing HACO toxicity. Further research is needed to explore biological factors that may attribute to HACO toxicity.

The patient–product interaction including length of time smoked, communal smoking, smoking location, and co-ingestions needs to be scrutinized. Interestingly, only 5 (8.6%) patients developed HACO toxicity while working within a hookah bar/lounge. It is somewhat surprising that there were not more bar/lounge patrons affected if high ambient CO concentrations are an important factor. Additionally, two HACO patients reported smoking hookah in an outdoor environment, further suggesting ambient CO concentration is not the only determinant of HACO toxicity.

Conclusions

In our retrospective review, within a short time span, hookah smoking became a substantial contributor to CO toxicity. Its rapid increase is of particular concern, outnumbering traditional sources of CO toxicity. Frontline physicians should ask about hookah exposures in patients with CO toxicity, and individuals who smoke hookah should be warned about its health risks. We urge public health authorities to undertake the necessary prospective investigations and risk mitigation measures.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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