Carbon Monoxide Poisoning Following Use of a Water Pipe/Hookah
Joscha von Rappard, Melanie Schönenberger, Lorenz Bärlocher

SUMMARY

Background: Water pipe (hookah) smoking has become a common activity in Germany, particularly among adolescents and young adults; in 2011, its lifetime prevalence was as high as 68.8%. Similar trends can be seen in other European countries. Water-pipe smokers are exposed to the same health-endangering substances as cigarette smokers, and the inhaled amount of carbon monoxide (CO) can be as much as ten times as high. In CO intoxication, carboxyhemoglobin is formed and causes direct injury at the cellular level, leading to hypoxia and nonspecific neurological manifestations. There have only been ten reported cases around the world of CO intoxication due to the use of a water pipe, and none of these were fatal. It should be recalled, however, that accidental CO intoxication is common and is associated with high morbidity and mortality.

Case presentation and course: We present a series of four young adults, aged 16 to 21, three of whom were hospitalized because of transient unconsciousness. The carboxyhemoglobin (CO-Hb) content of the blood in the symptomatic patients ranged from 20.1% to 29.6%, while the asymptomatic patient had a CO-Hb content of 16.7%. Water-pipe smoking was the cause of CO intoxication in all four cases. The CO-Hb values were successfully brought down by the administration of highly concentrated oxygen and all patients were discharged in asymptomatic condition.

Conclusion: This case series reveals that CO intoxication due to water-pipe smoking is probably more common than is generally realized. Emergency room staff should be aware of this problem and inquire specifically about water-pipe smoking in patients with nonspecific neurological manifestations.

► Cite this as:
Information from other individuals revealed no evidence of an epileptogenic event, clinical examination was normal, and there was no evidence of any focal neurological deficits. Laboratory tests showed no abnormalities. ECG revealed incomplete right bundle branch block.

Arterial blood gas analysis showed an FCOHb level of 20.1% (normal range 0 to 5%, maximum 10% in heavy smokers). The patient was administered high-dose (above 10 L/min) oxygen via a non-rebreather mask. After two hours the patient’s FCOHb level was 4.8% and he was discharged home with no further manifestations.

Case 2:
An 18-year-old female patient was brought to the emergency room by paramedics following transient loss of consciousness. Her medical history was normal. She stated that she had taken neither recreational drugs nor medication.

Before this event the patient had been smoking a hookah with friends in a hookah lounge for approximately one hour. When she left the lounge she experienced a severe headache and paresthesia. Her vision was also blurred, and she finally collapsed and lost consciousness.

When paramedics arrived the patient’s circulation was stable and she was assigned a score of three on the Glasgow Coma Scale (GCS). The recorded loss of consciousness lasted approximately 15 minutes. On arrival at the emergency room she was given a GCS score of 15. Her circulation was stable and both clinical and neurological status seemed normal. There was no evidence of an epileptogenic event. ECG revealed no abnormalities. Cranial CT ruled out hemorrhage.

Arterial blood gas analysis showed an FCOHb level of 25.7% and she was administered high-dose (above 10 L/min) oxygen via a non-rebreather mask. Two hours later her FCOHb level was 8.4% and both his headache and his lethargy had resolved. The patient was therefore discharged home with no further manifestations.

Case 3:
A 17-year-old female patient accompanied her friend (case 2) to the emergency room. She had also smoked a hookah in the hookah lounge. Although she had no manifest problems, on learning of her friend’s diagnosis she was afraid of also having carbon monoxide poisoning.

Arterial blood gas analysis was performed and showed an FCOHb level of 16.7%. Although she had no manifest problems, she was also administered high-dose (above 10 L/min) oxygen via a non-rebreather mask. Four hours later her FCOHb level was 5% and she was discharged home, still with no manifest problems.

Case 4:
A 21-year-old male patient was brought to the emergency room by paramedics following recurrent transient loss of consciousness. His medical history was normal. He stated that he had taken neither recreational drugs nor medication.

The patient had been smoking a hookah for several hours while visiting friends. Afterwards he complained of nausea and headaches. When he collapsed on his way home and subsequently lost consciousness three times, his friends contacted the emergency services.

In the emergency room he presented with stable circulation and was assigned a GCS score of 15. He reacted slowly and lethargically while his clinical history was being taken. His leading symptom was headache. Clinical and neurological examination revealed no abnormalities and his ECG was normal. There was no evidence of an epileptogenic event.

After arterial blood gas analysis showed an FCOHb level of 29.6%, he was administered high-dose (above 10 L/min) oxygen via a non-rebreather mask. Two hours later his FCOHb level was 8.4% and both his headache and his lethargy had resolved. The patient was therefore discharged home with no further manifestations.

The main findings are summarized in Table 1.

Discussion
Carbon monoxide (CO) is a colorless, odorless gas produced during the incomplete combustion of hydrocarbons. Carbon monoxide poisoning is Germany’s leading cause of accidental poisoning, accounting for approximately 3700 hospital admissions and around

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Background of migration</th>
<th>Leading symptom</th>
<th>COHb (%) on admission</th>
<th>O2 therapy</th>
<th>COHb (%) on discharge</th>
<th>Delayed effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>16</td>
<td>M</td>
<td>No</td>
<td>Loss of consciousness</td>
<td>20.1</td>
<td>Yes</td>
<td>4.8</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>18</td>
<td>F</td>
<td>Yes</td>
<td>Loss of consciousness</td>
<td>25.7</td>
<td>Yes</td>
<td>7</td>
<td>No</td>
</tr>
<tr>
<td>3</td>
<td>17</td>
<td>F</td>
<td>Yes</td>
<td>None</td>
<td>16.7</td>
<td>Yes</td>
<td>5</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>21</td>
<td>M</td>
<td>Yes</td>
<td>Loss of consciousness</td>
<td>29.6</td>
<td>Yes</td>
<td>8.4</td>
<td>No</td>
</tr>
</tbody>
</table>

1 All patients are of Caucasian ethnicity. In three of the four cases the patients’ parents were born in Southeastern Europe
2 Information obtained from patients and others, e.g. paramedics’ records
3 Oxygen at atmospheric pressure using high flow (>10 L) via a non-rebreather mask
4 Information obtained during a telephone interview six to twelve months after hospital admission
Following inhalation, carbon monoxide diffuses rapidly across the alveolar membrane and binds reversibly—with 240 times the affinity of oxygen—to divalent heme iron, forming carboxyhemoglobin (COHb). Allosteric conformational change shifts the oxygen dissociation curve to the left. At the cellular level, the binding of CO to the cytochrome leads to dysfunction of the respiratory chain in the mitochondria. Formation of reactive oxygen species gives rise to neuronal and myocardial necrosis and apoptosis (10, 11).

There is only a weak correlation between symptoms of acute carbon monoxide poisoning and COHb level; symptoms are better correlated with duration of exposure. In addition to nonspecific symptoms such as fatigue, nausea, and headaches, there is also a possibility of loss of consciousness, seizures, cardiac arrhythmia, myocardial ischemia, and death (7). In addition, the severity of toxicity is determined essentially by pre-existing comorbidities (cardiovascular diseases, pulmonary diseases, anemia) and age. Age over 65 is associated with the highest mortality (12).

Carbon monoxide poisoning causes both immediate neurological symptoms and delayed neuropsychological effects. The latter can develop even after an asymptomatic latency phase lasting several weeks, and some can be long-term (13, 14). Symptoms that have been described range from mild cognitive effects (memory and concentration problems) via affective disorders (depression, anxiety disorders) through to gait and balance disorders, tremor, and loss of hearing in rare cases (15).

Approximately 100 million people smoke hookahs worldwide, particularly in North Africa and the Arab world (16). However, in Germany too, hookahs—also referred to as water pipes, narghiles, arghilas, shishas, and Hubble Bubble—are becoming increasingly popular among adolescents and young adults. This is because they are thought to provide “healthier” nicotine exposure than cigarettes (1, 3). Physicians face new challenges in the form of altered consumption behavior and a variety of new designer drugs, the manufacture of which exploits legal loopholes by processing existing substances (e.g. synthetic cannabinoids, known as “spice”) (17).

A hookah consists of a glass water container, a clay or metal tobacco bowl, a pipe stem, and a hose with a mouthpiece. A sheet of perforated aluminum foil is placed on the clay or metal tobacco bowl, and a piece of burning charcoal is placed on the foil. Inhaling through the hose draws smoke through the water container and into the mouth of the smoker (Figure).

In actual fact, hookah smoking gives rise to the same harmful substances as cigarette smoking (in particular tar, nicotine, and carbon monoxide) (19). In addition to the type of tobacco used and the different temperatures at the combustion area, there are qualitative and quantitative differences in inhaled toxins, caused in particular by the differing duration of inhalation (approximately five minutes for cigarettes and 50 for hookahs). It should be stressed that both conventional tobacco blends and alternative, nicotine-free herb blends differ only in their nicotine content, not in the quantities of other toxic substances (carbon monoxide, tar, polycyclic hydrocarbons) inhaled (18, 20). As a result of the combustion of charcoal, a hookah smoker inhales more than 10 times more carbon monoxide than a cigarette smoker (19).

Table 2 provides an overview of all case reports of carbon monoxide poisoning associated with hookah use published in PubMed up to July 2014.

The dates and geographical distribution of the case reports (no cases before 2009, two in 2009, one in 2010, two in 2011, two in 2012, three in 2013; six case reports in the Middle East/Southeast Asia, four in Europe/the USA) may reflect the prevalence and increasing incidence of hookah smoking, as well as increasing awareness among physicians. The symptoms most frequently described are nonspecific and include headache, dizziness, nausea, and vomiting; five cases of transient loss of consciousness have also been recorded in connection with hookah smoking. With one exception, all patients were treated with oxygen at atmospheric pressure via a non-rebreather mask.

No follow-up was performed in any of the cases described. However, in one case prolonged neurological complaints such as dizziness and headaches were reported over a period of several weeks (30).

Although there have not yet been any recorded cases of fatal carbon monoxide poisoning following hookah smoking, approximately 370 fatalities per year (9). Although it is most commonly caused by smoke inhalation from fires, accidental carbon monoxide poisoning has also been reported in connection with fireplaces, hot water boilers, and charcoal table barbecues (9).

FIGURE

Schematic representation of a hookah
use, carbon monoxide poisoning is one of the leading causes of fatal accidental poisoning worldwide (31). Because its spectrum of clinical symptoms is broad and nonspecific, diagnosis is often subject to substantial delay.

As the photometric absorption of COHb is similar to that of oxyhemoglobin, COHb cannot be detected using standard pulsoximetry. Carbon monoxide poisoning can therefore only be confirmed by venous or arterial blood gas analysis.

Treatment options include high-concentration oxygen via a non-rebreather mask and hyperbaric oxygen therapy in a pressure chamber. The former allows administration of 90% FiO2 using high flow (10 to 15 L/min), provided the fit of the mask is airtight (32). The half-life of COHb can be reduced from three to four hours in ambient air (21% O2) to 40 to 80 minutes using a non-rebreather mask, or 15 to 30 minutes in hyperbaric conditions (2.5 bar) (33).

As mentioned above, initial COHb level is only weakly correlated with both the severity of symptoms and delayed neurological/neuropsychiatric effects. In addition, different studies have different inclusion and exclusion criteria. The studies currently available therefore provide a heterogeneous range of opinions concerning the best treatment (34, 35). As high-concentration oxygen is safe, universally available, and cost-effective, while hyperbaric therapy can cause complications (barotrauma, perforated eardrum, seizure, air embolism), the latter should be reserved for patients with severe symptoms and for carbon monoxide poisoning during pregnancy (13, 35).

Conclusions

As hookah smoking is widespread, and the observation period in this case report was short (four cases in six months), it is suspected that many cases of carbon monoxide poisoning following hookah use remain hidden. The majority of those affected are probably not admitted to a hospital because their symptoms are nonspecific. In addition, the fact that high COHb levels can be found even in completely asymptomatic patients (see case 3) demonstrates the dangerous and unpredictable nature of carbon monoxide poisoning.

In this case series 75% of patients had a background of migration. A possible association has already been

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>No. of cases</th>
<th>Patient age</th>
<th>Leading symptoms</th>
<th>COHb (%)</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lim et al.</td>
<td>2009</td>
<td>Singapore</td>
<td>1</td>
<td>19</td>
<td>Dizziness</td>
<td>27.8</td>
<td>Oxygen at atmospheric pressure</td>
</tr>
<tr>
<td>Uyanýk et al.</td>
<td>2009</td>
<td>Turkey</td>
<td>1</td>
<td>25</td>
<td>Headache, vomiting, dizziness</td>
<td>28.7</td>
<td>Oxygen at atmospheric pressure</td>
</tr>
<tr>
<td>Cavus et al.</td>
<td>2010</td>
<td>Turkey</td>
<td>1</td>
<td>25</td>
<td>Loss of consciousness</td>
<td>31.1</td>
<td>Oxygen at atmospheric pressure</td>
</tr>
<tr>
<td>Arziman et al.</td>
<td>2011</td>
<td>Turkey</td>
<td>5</td>
<td>17 to 27</td>
<td>3 × nausea, dizziness, 1 × loss of consciousness</td>
<td>11.4 to 24.3</td>
<td>Oxygen at atmospheric pressure</td>
</tr>
<tr>
<td>La Fauci et al.</td>
<td>2011</td>
<td>Italy</td>
<td>1</td>
<td>16</td>
<td>Confusion</td>
<td>24</td>
<td>Hyperbaric oxygen</td>
</tr>
<tr>
<td>Clarke et al.</td>
<td>2012</td>
<td>UK</td>
<td>11</td>
<td>17 to 34</td>
<td>Headache, dizziness</td>
<td>7.3 to 21</td>
<td>Oxygen at atmospheric pressure</td>
</tr>
<tr>
<td>Ashurst et al.</td>
<td>2012</td>
<td>USA</td>
<td>1</td>
<td>21</td>
<td>Vomiting, loss of consciousness</td>
<td>15.3</td>
<td>Oxygen at atmospheric pressure</td>
</tr>
<tr>
<td>Karaca et al.</td>
<td>2013</td>
<td>Turkey</td>
<td>1</td>
<td>20</td>
<td>Loss of consciousness</td>
<td>31.1</td>
<td>Oxygen at atmospheric pressure</td>
</tr>
<tr>
<td>Ozkan et al.</td>
<td>2013</td>
<td>Turkey</td>
<td>1</td>
<td>19</td>
<td>Loss of consciousness</td>
<td>32.7</td>
<td>Oxygen at atmospheric pressure</td>
</tr>
<tr>
<td>Bens et al.</td>
<td>2013</td>
<td>The Netherlands</td>
<td>3</td>
<td>*</td>
<td>Dizziness, headache</td>
<td>5.7 to 22</td>
<td>Oxygen at atmospheric pressure</td>
</tr>
</tbody>
</table>
described in the literature (1, 36) and may be due to dif-fering smoking habits in different cultures.

All patients were asked about any delayed effects during a telephone interview conducted in July 2014. In particular, they were asked about neurological/neuropsychological symptoms such as headache, dizziness, vision problems, concentration problems, forgetfulness, and depression. With the exception of patient two, who had suffered respiratory difficulties for several weeks, all the patients reported that they had had absolutely no symptoms following their event.

Although this case series did not provide any evidence of delayed neurological consequences, swift diagnosis and treatment is extremely important in cases of carbon monoxide poisoning.

Oxygen therapy at atmospheric pressure seems to be a cost-effective, safe, effective treatment in emergency care.

This case series provides evidence that the number of cases of carbon monoxide poisoning in connection with hookah smoking is underestimated. This should prompt emergency admission staff to ask targeted questions regarding hookah smoking for patients with non-specific neurological symptoms.

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**KEY MESSAGES**

- Hookah smoking gives rise to the same harmful substances as cigarette smoking. The inhaled quantity of carbon monoxide (CO) can actually be up to 10 times greater.

- Hookah smoking can lead to significant carbon monoxide poisoning. Clinically, it can be associated with transient loss of consciousness.

- Because the photometric absorption spectrum of carboxyhemoglobin is similar to that of oxyhemoglobin, carbon monoxide poisoning can only be confirmed using a special pulsoximeter or by venous or arterial blood gas analysis.

- Swift administration of high-dose (>10 L/min) oxygen via a non-rebreather mask is a cost-effective, safe, effective treatment. In patients with severe symptoms and for carbon monoxide poisoning during pregnancy, therapy in hyperbaric conditions should be considered as a treatment option.

- In cases of non-specific neurological symptoms, targeted questions should be asked regarding hookah smoking, and carbon monoxide poisoning should be ruled out using blood gas analysis.

**Conflict of interest statement**
The authors declare that no conflict of interest exists.

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