Case Report

A Report on Survived cases of Carbon-monoxide Toxicity

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ABSTRACT

Two young hostel boarders admitted to the emergency department with the complaints of sudden onset headache, nausea, vomiting, breathing difficulty; and, one of them had an episode of syncope with urinary incontinence. On further detailed history, we found that they had burned charcoal in their room to combat the cold weather. They fell asleep, after about three hours both of them experienced the above symptoms, however another roommate had only a headache. On further investigation the diagnosis was made in the line of carbon monoxide poisoning. The diagnosis of carbon monoxide (CO) toxicity usually missed by the clinicians and occurs commonly because of the vague and broad spectrum nature of complaints. Specifically inquiring about possible exposures when considering the diagnosis is important. Patients with suspected carbon monoxide poisoning should be treated immediately with normobaric oxygen (with a fraction of inspired oxygen as high as possible) which speeds up the excretion. The source of CO should be identified and appropriate measures should be taken to mobilize patients or source. Considering partial recovery after treatment in CO toxicity, patients should be cautioned for resurgence of symptoms and advised to revisit to the emergency department without delay.

Keyword: carbon monoxide; toxicity; headache; poisoning; normobaric.

INTRODUCTION

The deadly effect of carbon monoxide has been known since as long as Greek and Roman times, when the gas was used for executions.[1] In 1857 Claude Bernard postulated that its noxious effect was caused by reversible displacement of oxygen from hemoglobin to form carboxy.[2] In 1926 it became apparent that hypoxia was caused not only by deficient oxygen transport but also by poor tissue uptake. Warberg used yeast cultures to show that cellular uptake of oxygen was inhibited by exposure to a large amount of carbon monoxide.[3] Carbon monoxide is known as the silent killer as it has no color or smell. Some poisonings are caused by self-harm but most are accidental. In the 1960s and 1970s the conversion from coal gas to carbon-monoxide-free natural gas caused a dramatic reduction in poisoning.[4,5,6,7] The diagnosis is made by prompt measurement of carboxyhemoglobin levels.Treatment consists of the patient's removal from the source of exposure and the immediate administration of 100% supplemental oxygen in addition to aggressive supportive measures. The use of hyperbaric oxygen is not beneficial. In our Emergency Department we came across two survived cases of carbon monoxide poisoning, due to combustion of charcoal in a closed space. They presented with typical symptoms and were treated conservatively with high flow oxygen. Subsequently these patients improved and were discharged.

CASE-REPORT

Two young female patients aged 23 years (patient1) and 25 years (patient 2), working in a software company, staying in a hostel presented to the emergency. They had a history of sudden onset headache, nausea, vomiting, breathing difficulty and one of them had an episode of syncope with urinary incontinence. On further detailed history, we found that they had burned charcoal in their room to combat the cold weather. They fell asleep, after about three hours both of them experienced the above symptoms, however another roommate had only a headache. Hence they both visited the Emergency Department at night. On general physical examination (Table 1 and 2) there was no cyanosis, no hypoxia, and no tachypnea

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Table 1: General physical examination findings of patient 1.

Examination Findings	Results
Pulse Rate	98/min
Respiratory Rate	18/min
Temperature	Afebrile
SpO_2	98% RA
Blood Pressure	120/70mmHg

Table 2: General physical examination of patient 2.

Examination Findings	Results
Pulse Rate	110/min
Respiratory Rate	20/min
Temperature	afebrile
SpO_2	98% RA
Blood Pressure	110/70mmHg

Table 3: Systemic examination of patient 1.

Respiratory System	Normal vesicular breath sounds
Cardiovascular system	No murmurs
Per Abdomen	Soft and non-tender
Central Nervous System	No deficits

Table 4: Systemic examination of patient 2.

Respiratory System	Normal vesicular breath sounds
Cardiovascular system	No murmurs
Per Abdomen	Soft and non-tender
Central Nervous System	No deficits

A provisional diagnosis of "Carbon Monoxide poisoning" was made and they were treated with high flow oxygen via a NRB (Non-Rebreathing) mask. To measure carbon monoxide levels co oximetry was done which showed the following results (Table 5 and Table 6) and the diagnosis confirmed. Both patients were treated symptomatically with anti-emetics, analgesics and high flow oxygen. Both patients had increased levels of carboxy-hemoglobin levels in co oximetry. They were observed for 8 hours in ED, and discharged once their symptoms improved.

Table 5: Arterial blood gas profile with CO oximetry values of Patient 1.

	Observed value	Normal value
pH	7.407	7.350-7.450
pCO2	31.8mmHg	32.0-45.0
pO2	469 mmHg	83.0-108
sO2	99.7 mmHg	95.0-99.0
COHb	9.2%	0.9-1.0
HCO3	19.6mmol/L	
Lactate	0.7mmol/L	0.5-1.6
Hb	10.4 g/dl	12.0-16.0

CoHb: Carboxyhemoglobinemia; Hb: Hemoglobin; Hco3: serum bicarbonate; pCO2: Partial pressure of carbon dioxide; pO2: partial pressure of oxygen.

Table 6: Arterial blood gas profile with CO oximetry values of Patient 2.

	Observed value	Normal value
pH	7.428	7.350-7.450
pCO2	32.6mmHg	32.0-45.0
pO2	401 mmHg	83.0-108
sO2	99.6 mmHg	95.0-99.0
COHb	11%	0.9-1.0
HCO3	21.2mmol/L	
Lactate	1.1 mmol/L	0.5-1.6
Hb	11.8 g/dl	12.0-16.0

CoHb: Carboxyhemoglobinemia; Hb: Hemoglobin; Hco3: serum bicarbonate; pCO2: Partial pressure of carbon dioxide; pO2: partial pressure of oxygen.

DISCUSSION

Carbon monoxide (CO) is an odorless, tasteless, colorless, nonirritating gas formed by hydrocarbon combustion. The atmospheric concentration of CO is generally below 0.001 percent, but it may be higher in urban areas or enclosed environments. CO binds to hemoglobin with much greater affinity than oxygen, forming carboxyhemoglobin (COHb) and resulting in impaired oxygen transport and utilization. CO can also precipitate an inflammatory

cascade that results in CNS lipid peroxidation and delayed neurologic sequelae. The overall case-fatality rate for CO poisoning ranges from 0 to 31 percent.[8,9,10] Smoke inhalation is responsible for most inadvertent cases of CO poisoning. Other potential sources of CO include poorly functioning heating systems, improperly vented fuel-burning devices (eg, kerosene heaters, charcoal grills, and camping stoves[11] Carbon monoxide (CO) diffuses rapidly across the pulmonary capillary membrane and binds to the iron moiety of heme (and other porphyrins) with approximately 240 times the affinity of oxygen.[1] The degree of carboxyhemoglobinemia (COHb) is a function of the relative amounts of CO and oxygen in the environment, duration of exposure, and minute ventilation. Nonsmokers may have up to 3 percent carboxyhemoglobin at baseline; smokers may have levels of 10 to 15 percent.[12] Severe chronic obstructive pulmonary disease can cause a modest but significant elevation in carboxyhemoglobin levels, even among patients without exposure to tobacco smoke. The mechanism and clinical significance of this finding is unclear.[13] The clinical findings of carbon monoxide (CO) poisoning are highly variable and largely nonspecific.[14,15] Moderately or mildly CO-intoxicated patients often present with constitutional symptoms, including headache (the most common presenting symptom), malaise, nausea, and dizziness, and may be misdiagnosed with acute viral syndromes.[13] In addition to current symptoms, the clinician should specifically inquire (of the patient and/or witnesses) about loss of consciousness. In the absence of concurrent trauma or burns, physical findings in CO poisoning are usually confined to alterations in mental status, so a careful neurologic examination is crucial. Patients may manifest symptoms ranging from mild confusion to coma. Specific cognitive testing, such as the Carbon Monoxide Neuropsychological Screening Battery.[16] is usually not used in the acute setting and, in any event, is not universally endorsed due to its inability to discriminate between the effects of CO and those of other intoxicants.[17] Although some textbooks describe a "cherry red" appearance of the lips and skin as indicative of CO poisoning, this is an insensitive sign.[17] Severe CO toxicity can produce neurologic symptoms such as seizures, syncope, or coma, and also cardiovascular and metabolic manifestations such as myocardial ischemia, ventricular arrhythmias, pulmonary edema, and profound lactic acidosis. Hyperbaric O2 therapy is logical, but of no proven benefit for CO poisoning. Transfer to a hyperbaric chamber and pressurization may take hours and so hyperbaric treatment may be no more effective than ventilation on 100% normobaric O_2 Consider hyperbaric treatment if a patient has been unconscious at any time, has COHb >20%, is pregnant, or has cardiac complications or neurological or psychiatric features. Although the administration of normobaric oxygen hastens the elimination of carbon monoxide, one trial did not show a reduction of cognitive sequelae after the inhalation of normobaric oxygen, as compared with no supplemental oxygen therapy.[14] However, since normobaric oxygen is safe, readily available, and inexpensive, it should be provided until the carboxyhemoglobin level is less than 5%.

CONCLUSION

Misdiagnosis of carbon monoxide (CO) toxicity commonly occurs because of the vague and broad spectrum nature of complaints. Specifically inquiring about possible exposures when considering the diagnosis is important. Patients with suspected carbon monoxide poisoning should be treated immediately with normobaric oxygen (with a fraction of inspired oxygen as high as possible) which speeds up the excretion. The source of CO should be identified and appropriate measures should be taken to mobilize patients or source. Considering partial recovery after treatment in CO toxicity, patients should be cautioned for resurgence of symptoms and advised to revisit to the emergency. Patients should be educated to minimize the risks of carbon monoxide exposure, including avoiding the operation of combustion engines, combustion of charcoal, petroleum products indoors, and performing periodic furnace inspections.

CONFLICTS OF INTEREST

Declared none.

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