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Revenge of the Barbecue Grill
Carbon Monoxide Poisoning

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CARBON MONOXIDE (CO) poisoning can be difficult to recognize because it often has a presentation that suggests a viral illness, food poisoning, gastroenteritis, or even a functional illness. Because CO is the leading cause of death due to poisoning in the United States,[1-3] emergency department physicians should suspect CO poisoning when a similar illness occurs simultaneously in two or more persons.

Report of Cases

Four members of a non-English-speaking family presented to a university hospital emergency department, all complaining of headache, lethargy, abdominal cramping, and nausea. They related a history of drinking unrefrigerated milk the evening before presentation. Two patients had mild orthostatic blood pressure changes. They were treated with intravenous fluids and discharged with the diagnosis of infectious gastroenteritis. One family member remained in the emergency department with persistent symptoms.

Several hours later, four other members of the same family presented to the emergency department with similar complaints. They were evaluated and treated with intravenous fluids, and all were discharged, including the patient from group 1. Several hours later, four more members of the same family presented to the emergency department with similar complaints. One patient had not ingested the suspected milk, but she suffered from migraine headache and thought that this was one of her headaches.

Further questioning through an interpreter revealed that all patients were members of the same household. Specific questions about home heating systems did not suggest mechanical problems; in fact, it had not been used. An unaffected relative, who lived elsewhere, admitted to cooking indoors with charcoal briquets in the fireplace after originally stating that the cooking was done outdoors.

Specimens were drawn for arterial blood gas determinations and carboxyhemoglobin (COHb) concentrations. Results were 0.069, 0.151, 0.171 and 0.174 (normal < 0.150).* The four patients in the emergency department were treated with 100% oxygen for two hours with symptomatic improvement. The COHb concentration was rechecked in the patient who had a 0.174 level, and it had fallen to 0.044 (4.4%). Fire fighters were dispatched to check the house and reported a barbecue grill in the middle of the living room and evidence of smoke damage.

Discussion

Carbon monoxide is the leading cause of death due to poisoning in the United States.[1-3] It is also the most common cause of death in combustion-related inhalation injury. Claude Bernard in 1857 provided the first accurate description of CO poisoning. Since then many advances in our understanding of the pathophysiology have been achieved.

Carbon monoxide combines preferentially with hemoglobin to produce COHb, displacing oxygen and reducing the systemic arterial oxygen content. Possible mechanisms of toxicity include a decrease in the oxygen-carrying capacity of blood, alteration of the dissociation characteristics of oxyhemoglobin so that delivery to the tissues further decreases, and a decrease in cellular respiration by binding with cytochrome a_3.

Goldbaum and co-workers postulated that high dissolved CO concentrations develop only at the alveolar-blood interface when CO is inhaled. Dissolved CO delivered to tissues, especially heart and brain, causes most of the CO toxicity by binding with the cytochrome a_3.[4] Hence, it is dissolved CO, not COHb, that causes toxic reactions.

The half-life of COHb is 320 minutes for young healthy volunteers breathing room air. Administering 100% oxygen at 1 atm reduces the half-life to 80.3 minutes, whereas 100% oxygen at 3 atm will reduce the half-life to 23.3 minutes.[5]

Carbon monoxide binds to cardiac and skeletal myoglobin and hemoglobin. Cardiac myoglobin binds three times more CO than skeletal myoglobin.[6] Because carboxy-

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*In Système International units, these values are expressed as a fraction of 1. In conventional units, they would be 6.9%, 15.1%, 17.1%, and 17.4%.

myoglobin dissociation is slower than COHb due to its increased affinity for myoglobin, a "rebound effect" with a delayed return of symptoms can occur.[7]

Carbon monoxide poisoning does not cause dyspnea or tachypnea until it causes hypoxemia from circulatory dysfunction or lactic acidosis from tissue hypoxia. At low levels, chronic cardiopulmonary problems, for example, angina and chronic obstructive pulmonary disease, may be exacerbated because cardiac myoglobin binds with great affinity and rapidly reduces myocardial oxygen reserve.

With acute exposure, blood levels correlate with symptoms and signs but do not reflect tissue CO levels. Arterial blood gas levels are used to determine the degree of carbon monoxide intoxication and treatment.[8,9] With levels less than 0.10 (10%), patients are usually asymptomatic. As the COHb concentration increases above 0.20 (20%), headache, dizziness, confusion, and nausea may develop. Chest pain due to myocardial ischemia may occur, as can cardiac arrhythmias. Coma and seizures due to cerebral edema are common with concentrations of greater than 0.40 (40%), and death is likely with more than 0.60 (60%).[10-12]

The differential diagnosis includes viral illnesses, food poisoning, depression, transient ischemic attacks, coronary artery disease, arrhythmias, and functional illnesses, among others. The most common misdiagnosis is a "flu-like" syndrome.[9]

The mainstay of therapy is supplemental oxygen and monitoring for cardiac arrhythmias. The goal of oxygen therapy is to improve the oxygen content of the blood by maximizing the fraction dissolved in plasma (Pao_2).[13] The advantage of hyperbaric oxygen therapy is a more rapid reduction in COHb levels. Disadvantages of this treatment modality include few available treatment centers and the requirement for patient transport. Patients with severe neurologic or cardiovascular symptoms or very high COHb concentrations would benefit from hyperbaric oxygen, but it is these very sick patients for whom transport is most hazardous.

Once treatment begins, oxygen therapy and observation must continue long enough to rule out delayed sequelae as the carboxymyoglobin unloads.

The initial presentation of our patients was consistent with gastroenteritis, for which they were given intravenous hydration and other supportive measures. We initially did not consider inhaled toxins. Once an adequate history, which included indoor cooking, and after arterial blood gas results were obtained, they were placed on 100% oxygen therapy with a complete resolution of the symptoms. They were discharged to a safe environment within three hours of the correct diagnosis.

This case illustrates the importance of always considering CO poisoning when two or more patients are similarly or simultaneously sick. The diagnosis must be excluded by a directed history and physical examination. If suspicion remains, COHb testing should be done and oxygen therapy should be started empirically while results are pending. If CO poisoning is confirmed, the source must be identified and recommendations for correction or avoidance made.

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Primary Tuberculous Thyroid Abscess Mimicking Carcinoma Diagnosed by Fine Needle Aspiration Biopsy

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TUBERCULOSIS AFFECTING the thyroid gland is a rare condition. Exact figures for the incidence of tuberculosis of the thyroid are unavailable. The infection may present first in the thyroid gland or appear secondary to a tuberculous process elsewhere in the body. Barnes and Weatherstone noted that 7% of cases of miliary tuberculosis involve the thyroid gland.[1] The gland can also become involved with focal caseating tuberculosis, which can be blood-borne,[2,3] or by direct extension from the larynx and cervical lymph nodes. The pathologic forms include multiple thyroidal granulomata, goiter with caseation, cold abscess, chronic fibrosing thyroiditis, and, least common, acute abscess.[4] Patients present with a thyroid nodule,[5] abscess,[4] thyroiditis,[6] or findings suggesting carcinoma[7] of the thyroid gland.

A tuberculous abscess of the thyroid gland as the sole manifestation of tuberculosis is even more uncommon. Swelling of the neck is the most consistent sign and dysphagia the most common complaint.[3] The diagnosis of a tubercular abscess is rarely made clinically. In the case reported here, the clinical presentation suggested an undifferentiated thyroid cancer or granulomatous disease of the thyroid. Fine needle aspiration biopsy revealed milky fluid, and subsequent cultures confirmed the diagnosis preoperatively.

Report of a Case

The patient, a 61-year-old, asymptomatic, married white woman, was referred to the University of California, San Francisco, Medical Center because of a right neck mass of 1½ months' duration, clinically suggestive of anaplastic

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