Recurrent Carbon Monoxide Poisoning From Cigarette Smoking

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Abstract: Carbon monoxide intoxication remains a major cause of morbidity and mortality in the United States with an estimate of 50,000 cases annually in emergency departments nationwide (Weaver, N Engl J Med. 2009;360:1217–25). Sources of carbon monoxide most often include car exhaust, malfunctioning heating systems and inhaled smoke. It has been well established that there is a dose-dependent increase in carboxyhemoglobin (COHb) concentration with tobacco use. It is generally accepted that heavy smokers have COHb levels <10% to 15% (Ernst and Zibrak, N Engl J Med. 1998;339:1603–8). The authors report a 48-year-old woman with significant tobacco abuse who presented with COHb levels as high as 24.2% in the face of tobacco use.

Key Indexing Terms: Tobacco abuse; Carboxyhemoglobin; Carbon monoxide. **[Am J Med Sci 2010;340(5):427–428.]**

CASE REPORT

A 48-year-old white female with a longstanding history of tobacco abuse presented with a 1-day history of headache and dizziness. Initial evaluation revealed an increased carboxyhemoglobin (COHb) level of 21.8% and hypoxemia with a PO_2 of 46 mm Hg. The patient denied any other complaints. Vital signs were stable with 97% oxygen saturation by pulse oximetry. On examination, the patient had diminished breath sounds with diffuse wheezing and significant digital clubbing in the upper extremities. Additional diagnostic studies including complete blood count, computed tomography of head, electrocardiogram, chest radiograph, and computed tomography of chest were unrevealing. She was treated with 100% supplemental oxygen and abstinence from tobacco, and the COHb level decreased steadily to 1.9%. She was subsequently discharged from the hospital.

One week later, the patient returned to the hospital with similar complaints and a COHb level of 24.2%. She denied any known carbon monoxide (CO) exposures, despite extensive efforts of identifying a source by the local gas company; however, she did admit to continued tobacco abuse with upward of 2 packs per day. Repeat treatment with 100% oxygen therapy and abstinence from smoking decreased her COHb level to 3.2%. Because of the patient and primary medical providers' concern that smoking could be related to her marked carboxyhemoglobinemia, a level was drawn before tobacco use and after heavy tobacco use of approximately 16 cigarettes in 2 hours. Her COHb level increased from 13.6% to 19.6%, which confirmed that her tobacco abuse had a significant role in causing her symptomatology and carboxyhemoglobinemia. Subsequently, complete tobacco cessation was recommended.

DISCUSSION

CO poisoning is a common problem in the United States with >50,000 emergency room visits annually. The source of CO is often identified as a malfunctioning furnace, inadequate ventilation from heating sources, motor exhaust and, rarely, excessive tobacco use alone. In fact, a recent literature search performed using medical subject headings COHb, tobacco use and CO poisoning did not reveal any reports of COHb levels reaching the level we report in this case secondary to tobacco use. The lethality of CO stems from its greater affinity to bind hemoglobin compared with oxygen. As a result, CO shifts the oxygen-hemoglobin dissociation curve to the left. This phenomenon, also known as the Haldane effect, leads to tissue hypoxia.

The clinical symptoms of CO poisoning are nonspecific. Patients may be asymptomatic at COHb levels <10%. With COHb levels between 10% and 20%, patients may experience headache and dyspnea. At levels between 20% and 30%, patients generally complain of nausea, vomiting, throbbing headache and fatigue. Levels greater than 30% can produce severe symptoms such as confusion, syncope, palpitations, chest pain, tachypnea, respiratory arrest, cardiac arrest, seizures or death. It is important to note that COHb levels may not correlate well to symptomatology; therefore, one must have a high index of suspicion. The diagnosis can be supported with increased COHb levels.[3,4]

The treatment of CO intoxication first requires the patient be removed from the source of CO production. Subsequently, high-flow 100% normobaric oxygen should be administered until COHb is normalized. Hyperbaric oxygen therapy may be considered, but its indications for use remain controversial. Current evidence suggests hyperbaric oxygen can reduce cognitive sequelae of CO intoxication if administered within 24 hours of an acute episode of CO intoxication.[3,4]

It is also important to note that certain patient populations are at greater risk for CO intoxication—namely adults with chronic cardiopulmonary disease, sickle cell disease, thalassemias and smokers. In a 25-year prospective cohort study,[1] smokers using >25 cigarettes per day had mean COHb levels of 6.02% (SD of 2.86) when compared with lifelong nonsmokers with COHb levels of 1.59% (SD of 1.72). In addiion, Garcia et al[2] observe a maximal COHb level of 9.6% in smokers using >20 cigarettes a day. Our patient showed repeated levels much greater than expected based on these studies. In the absence of any other diagnosis to explain the patient's findings, it is our supposition that the increased COHb levels were secondary to excessive tobacco

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inhalation. In turn, these increased COHb levels led to chronic hypoxemia and, likely, resulted in clubbing of the digits. Thus, the treatment modality recommended to our patient was smoking cessation.

KEY POINT

- Significant tobacco use can increase COHb levels and lead to signs and symptoms consistent with CO poisoning.

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