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Unmasking a Silent Threat: Early Detection of Elevated HbCO Levels When the Story Doesn't Add Up

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Abstract:

Carbon monoxide (CO) is difficult to detect in the surrounding environment due to its colorless, odorless, and tasteless properties. Patients acutely poisoned with CO can have a myriad of presentations including headache, dizziness, and critical illness— all with a mortality rate up to 3%. When concerned for CO poisoning, blood co-oximeter testing should be performed, with treatment surrounding the idea of providing supplemental oxygen The efficacy of hyperbaric oxygen (HBO) treatment remains unclear. Here we describe an atypical scenario of a patient ultimately found to have CO poisoning, and the importance of early identification and treatment of this elusive diagnosis to provide for a better neurological outcome.

Case Presentation:

A 31-year-old male with unknown past medical history presented to the ED by EMS for evaluation of "unresponsiveness." The patient was last seen normal the night prior, where he was seen entering a turned off vehicle outside with a companion. The next morning, neighbors found the patient along with his companion still in the vehicle and called 911. Per EMS report, both the patient and companion were found in the same unresponsive state, the vehicle was cold and turned off, and there were empty beer bottles present. There was no evidence of drug paraphernalia or trauma on scene. The patient was given 8mg of Narcan intranasally by police and another 2mg of Narcan IV by paramedics without change in condition. EMS reported stable vital signs during transport. In route to the hospital, paramedics witnessed decerebrate posturing. Despite normal pulse oximeter readings, the patient underwent rapid sequence intubation (RSI) with Ketamine and Rocuronium due to concern for airway compromise. The patient also received 500mL of normal saline intravenously prior to arrival.

On presentation, his vital signs were blood pressure of 110/66 mmHg, heart rate of 116 beats per minute, respiratory rate of 20 breaths per minute, temperature 98.1 degrees Fahrenheit orally, and a pulse oximetry of 100% on 70% FiO2.

Physical examination revealed an ill-appearing young male, currently intubated. His pupils were fixed 3mm bilaterally, nonresponsive to light stimuli. He was tachycardic and had equal bilateral breath sounds. Neurologically, he was nonresponsive to painful stimuli, and had no spontaneous movement of extremities. The remainder of the physical exam was unremarkable.

An X-ray of the chest and CT of the cervical spine were obtained which showed no acute abnormality. A CT of the head was performed which showed hypoattenuation in the globus pallidus bilaterally, suggesting hypoxic ischemic encephalopathy and/or carbon oxide poisoning. An electrocardiogram showed sinus tachycardia at a rate of 116 beats per minute with a normal QTc, without any ischemic changes. An arterial blood gas (ABG) was immediately obtained revealing an elevated carboxyhemoglobin (HbCO) of 16.1%. A complete blood count revealed a mild leukocytosis of 12.6. A high sensitivity troponin was tested and was initially 203. The repeat high sensitivity troponin two hours later was elevated at 396. A creatine kinase level was tested which was 1940. Lastly, a urine drug screen was positive for cocaine and amphetamines. Once the carboxyhemoglobin level was found to be elevated, the patient was placed on 100% FiO2 on the ventilator and a repeat ABG 2 hours later showed a carboxyhemoglobin of 5.3%. The patient was given 2L of IV normal saline for his diagnosis of acute rhabdomyolysis. Of note, 1 hour after arrival, the RSI medications wore off and the patient began moving all four extremities and biting his endotracheal tube. His Glasgow Coma Scale (GCS) was 5 (E1-V1-M3). The patient was then given Precedex and Vecuronium to ease his agitation and facilitate obtaining CT imaging.

Given the elevated HbCO on arrival and the level of obtundation in the setting of CT findings consistent with hypoxic encephalopathy, the prompt decision was made to transfer to a hyperbaric chamber. The patient was airlifted to the hyperbaric oxygen center, where he underwent prophylactic bilateral myringotomies and 9 days later was extubated. The patient was admitted for a total of 32 days and was eventually discharged home. He had a follow up appointment with neurology 6 months later, where his only concerns were mild hearing loss and intermittent leg weakness, ultimately making a remarkable recovery from his initial bleak neurological status and condition.

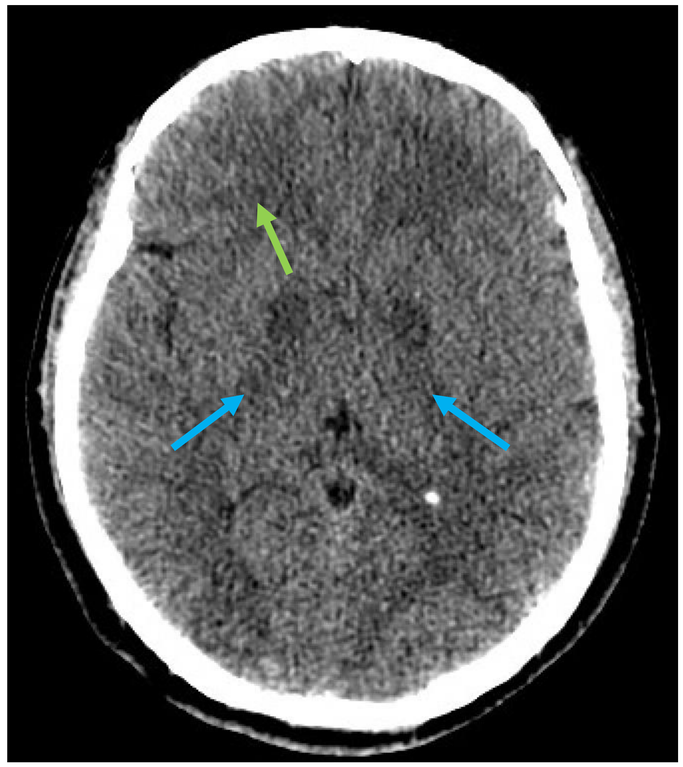

Figure 1

Figure 1: Computed tomography imaging of the head demonstrating hypoattenuation in bilateral globus pallidus (blue arrows) and some loss of grey-white matter differentiation (green arrow) suggesting hypoxic ischemic encephalopathy and edema consistent with CO poisoning

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Discussion:

It is paramount that emergency physicians have a high index of suspicion for this condition. Carbon monoxide poisoning presents a diagnostic challenge due to its range of presenting symptoms, carrying with it a high risk of mortality and neurological complications as it mimics other possible pathologies such as drug overdose and migraines, which can further delay diagnosis.

The pathophysiology of CO poisoning derives from the effect of CO on cellular mitochondrial respiration, causing free radical generation and inflammation. The two most affected organs are the brain and the heart. As a result, long term effects of CO poisoning are often seen in one-third of patients presenting as cardiac arrhythmias, ventricular dysfunction, cardiac infarction, and neurocognitive deficits [1,2].

On presentation, the patient's signs and symptoms will correlate to the severity of carbon monoxide poisoning. The first step however is to have a broad differential diagnosis, as these patients do not always present from typical textbook scenarios of house fire, a home without CO detectors with multiple sick family members, or cars that were left running in the garage. Once the differential diagnosis is established and elevated blood HbCO levels are found, it is important to obtain CT imaging and place the patient on supplemental oxygen [3].

Computed topography and MRI of the head are the imaging modality of choice to understand the extent of disease. Most often, the iron-rich globus pallidus is involved due to CO affinity for binding to the iron as seen in Figure 1 [4,5]. Symmetric hypoattenuation in the globus pallidus should alert radiologists and emergency physicians to confirm this potentially fatal condition and begin appropriate treatment.

Once identified, prompt treatment of CO poisoning begins with delivery of supplemental oxygen. In awake patients, non-rebreather mask is a great modality. In cases such as the one described here, 100% FiO2 should be delivered from the ventilator to the patient via the endotracheal tube. Although controversial, some studies have found a 100% protective effect of HBO therapy on the development of neurologic sequalae in severe CO poisoning [6]. There has also been discussion of neuroprotective effects of HBO however this topic remains understudied.

Notably, carbon monoxide exposure greater than 6 hours, GCS less than 9, lack of pupillary reflex to light, loss of consciousness, and intubation requirement correlate to higher prognostic levels of developing delayed neurologic sequalae [4]. Most commonly, psychiatric issues such as anxiety and depression, cognitive impairment, loss of motor and function are seen as consequences of CO poisoning.

Conclusions:

Carbon monoxide can lead to fatal accidental poisoning with catastrophic neurological outcomes. It should be suspected in all populations presenting with a range of complaints that can be as vague as headache and weakness, to coma and unresponsiveness. Patients with CO poisoning do not always present from a house or a car garage. Immediate testing of HbCO levels on blood co-oximetry should be performed to evaluate CO levels. CT imaging of the head is a helpful imaging tool for diagnostic confirmation. A range of neurological complications can ensue such as leg weakness as seen in this case of a 31-year-old male, to severe cognitive dysfunction and psychiatric disease. If at all suspected, CO poisoning must be diagnosed and managed promptly with supplemental O2 or HBO in this potentially lethal condition to avoid further complications.