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LETTER TO THE EDITOR

Could your acute stroke patient be carbon monoxide poisoning?

Ömer Taşkın[1] · **Nezihat Rana Dişel**[2]

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Introduction

Carbon monoxide (CO) is a colorless, odorless, tasteless and non-irritating gas and its exposure causes 50,000 Emergency Department (ED) admissions and 1300 deaths in the United States (US) every year. Carbon monoxide binds to the heme with approximately 240 times the affinity of oxygen and this raises the level of carboxyhemoglobin (COHb). Nonsmokers may have up to 3% COHb levels while smokers may have levels of 10–15% [1].

The clinical findings of carbon monoxide poisoning (COP) are variable and nonspecific. Mild and moderate cases present with symptoms including headache, nausea, and dizziness. However, syncope, shortness of breath, loss of consciousness, chest pain, could be the symptoms of serious poisoning which might cause pulmonary edema, myocardial ischemia, ventricular arrhythmias and coma because of its effects on the heart and brain.

Patients diagnosed with COP and recovered from suffering long-term neurocognitive sequelae due to brain damage. Delayed neuropsychiatric syndrome is seen in almost 40% of patients with serious poisoning [1]. Hemiplegia and dysarthria are the symptoms of acute stroke patients in general and are very rare to be seen in COP patients. In this case report, we present a patient who presented to the ED with hemiplegia and dysartia and who was further diagnosed with COP.

✉ Ömer Taşkın
omertaskinmd@gmail.com

Nezihat Rana Dişel
ranalpay@gmail.com

[1] Emergency Service, Yüreğir State Hospital, Serinevler mahallesi, Ege Bagatur Caddesi, no: 236, 01240 Yüreğir, Adana, Turkey

[2] Emergency Medicine Department, Faculty of Medicine, Çukurova University, Adana, Turkey

Case

A 68-year-old female was admitted to the ED with complaints of slurred speech and a right-sided weakness ongoing for one hour. The patient's vitals were normal except sPO_2 of 88%. The patient had a history of hypertension, diabetes mellitus, coronary artery disease with the by-pass operation and left-sided ischemic stroke 4 years ago with sequelaes of hypoesthesia and loss of motor functions (4/5). The patient's Glasgow Coma Scale score was 12 (E4, M6, V2) and her blood glucose was 180 mg/dL. Her physical examination revealed dysarthria, retraction at the corner of the mouth, plegia on the right side (1/5), the smell of smoke on her clothes and sequelae on the left side because of the previous stroke history. Her electrocardiogram revealed a normal sinus rhythm with 90 bpm. She was started nasal oxygen (4 L/min) and blood samples for further studies are obtained as well as computed tomography (CT) scan of the head and thorax to explain her neurological symptoms and hypoxemia. Her CT scans revealed no acute pathology. We have planned a diffusion magnetic resonance imaging (MRI) for acute ischemic lesions. The MRI showed no acute ischemic lesions. Her neurological symptoms showed improvement, her second physical examination revealed 4/5 motor function on her right side and her speech was better. The arterial blood gas analysis taken at the time of the admission revealed pH: 7.36, PO_2: 90 mmHg, PCO_2: 40.65 mmHg and COHb was 28.32%. Her complete blood count, biochemistry and coagulation tests were normal except for a mild elevation in her troponin T levels, 0.020 ng/mL (0.0–0.014 ng/dL). After her symptoms improved with O_2 therapy and imaging studies showed no acute pathological lesions and with a COHb level of 28% we diagnosed the patient with COP. The patient was hospitalized for hyperbaric oxygen therapy and follow-up; after 5 days the patient was discharged with no new sequelae.

Discussion

Carbon monoxide poisoning is a combination of tissue hypoxia and direct CO-mediated damage at the cellular level. Carbon monoxide binds to the heme and this raises the level of COHb. CO increases the affinity of other sites in the Hb tetramer for oxygen, and also decreases oxygen release and transport [1]. CO bounds to molecules such as myoglobin, cytochromes, and NADPH reductase, resulting in impairment of mitochondrial oxidative phosphorylation. Since the level of this binding cannot be measured, even if the COHb levels of the patients are normal, their intoxication may be severe. The half-life of this bound is longer than the half-life of COHb as a result of this symptoms of poisoning may still occur even if COHb levels became normal [2]. The half-life of COHb in room air is about 5 h, the half-life can be decreased to about 1–1.5 h with 100% oxygen treatment, it can reduce to less than 30 min with the hyperbaric oxygen treatment [3].

The diagnosis of CO poisoning is based upon a history and physical examination in junction with elevated COHb levels. Headache, dizziness and abdominal pain could be the early symptoms and it can be easily misdiagnosed with acute viral syndromes. However, as the exposure time increases, the severity of symptoms increases such as mental status changes, syncope, seizure.

Hemiplegia and dysarthria can be seen in various conditions such as transient ischemic attack (TIA), stroke and cerebral hemorrhage. The underlying mechanism of these situations is loss of cerebral blood flow. Acute brain injury of COP is mostly observed due to hypoxia. Brain injury may be due to transient vasogenic edema or infarction without occlusion of cerebral arteries. These specific neurological symptoms are not common in COP patients. There are only a few cases reporting COP with hemiplegia [4, 5] and pathological findings in cerebral imaging's are present in almost all of them, which is different from our case.

Hemiplegia and dysarthria with normal findings in cerebral imaging, the smell of smoke on the patient's clothes with a high COHb level, obtaining a history for heating her house by a coal stove, clinical improvement with oxygen and recovery with hyperbaric oxygen therapy level led us to focus on COP instead of TIA.

Conclusion

Carbon monoxide poisoning is still an important public health issue. The symptoms are generally non-specific and may cause misdiagnoses. When emergency physicians encounter patients with atypical neurologic findings or cannot support their findings with imaging evidence, they should review the patient's history and consider COP, especially during the winter season. To the best of our knowledge, our patient with hemiplegia and dysartria without any pathological findings in cerebral imaging is the first COP case in the literature.

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Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent Informed consent was obtained from the patient included in the case report.

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