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A Rare Case of Stroke Following Carbon Monoxide Poisoning from Residential Fire

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Abstract
Carbon monoxide (CO) poisoning is a significant global public health concern and a leading cause of accidental poisoning. Neurological complications are well documented, but acute ischemic stroke is a rare but potentially devastating outcome. We report the case of a 51-year-old male who developed an acute left middle cerebral artery (MCA) territory infarct following CO poisoning during a domestic fire incident. The case underscores the importance of early neuroimaging in suspected CO poisoning with persistent focal deficits. This report highlights a rare but serious manifestation of CO toxicity.
Keywords: Carbon monoxide, smoke inhalation, neurological complications, ischaemic stroke

INTRODUCTION

Carbon monoxide is a colorless, odorless, gas produced by the incomplete combustion of carbon-containing fuels. Its toxic effects primarily arise from the formation of carboxyhemoglobin, which impairs oxygen delivery to tissues, leading to hypoxia. The brain and heart are particularly vulnerable due to their high oxygen demand.

CO poisoning is frequently associated with nonspecific symptoms such as headache, confusion, and dizziness. In severe cases, it may cause seizures, coma, and death. Although hypoxic encephalopathy is the most recognized neurological complication, ischemic stroke remains an underreported and rare outcome. The underlying pathophysiology is believed to involve cerebral hypoxia, vasospasm, oxidative stress, and endothelial injury.

Case Presentation

A 57-year-old male, with no known comorbidities and nonalcoholic and non smoke, was brought to the Emergency room in an unconscious state, after being rescued from his burning home. History obtained from patient's neighbour, revealed, that the patient had been trapped inside a smoke-filled room for 30–40 minutes. On arrival, patient was drowsy and arousable. His pulse rate was 110 bpm, blood pressure as 100/60 mmHg, and SpO_2 of 89% on room air. Temperature was 101 degrees Fahrenheit and blood glucose was 122 mg/dl. Glasgow Coma Scale (GCS) was 9/15. Examination of cardiovascular and respiratory system were normal. Patient's ABG analysis was done and showed Ph of 7.4, PCO2 of 42 mmhg, PO2 of 78 mmHg, HCO3 of 22, FCO2Hb was 22. Patient was started on 100% oxygen support (normobaric oxygen therapy) after which his consciousness had improved. After improvement in patient's GCS, patient was found to have slurred speech and right-sided hemiparesis with power of 3/5 in both right upper limb and lower limb and left sided power 5/5. Tone was normal in all 4 limbs and bilateral plantar had flexor response and rest of the neurological exam was normal. Bilateral Pupils were equal and reactive to light.Investigations revealed a carboxyhemoglobin level of 21%. CT brain was done which showed a hypodensity in the left MCA territory, and MRI brain confirmed acute CVA with right hemiparesis, non-hemorrhagic infarct

involving the left MCA territory. Etiology was suspected to be carbon monoxide poisoning and hence started on management accordingly. He received oxygen support along with Neuro-protective

medication and symptomatic management throughout the course in hospital. Physiotheray was done regularly. Vitals were routinely monitored and charted. Over 14 days, the patient showed significant neurological recovery and was discharged with residual right-sided weakness and mild speech deficits.

DISCUSSION

Ischemic stroke secondary to carbon monoxide (CO) poisoning is a rare but increasingly recognized complication with potentially severe outcomes. While the predominant neurological sequelae of CO poisoning involve global hypoxic-ischemic encephalopathy and delayed neuropsychiatric symptoms, focal ischemic events—especially in the middle cerebral artery (MCA) territory—have been documented in isolated case reports and small series (1,2). These focal infarcts can significantly worsen the patient's neurological prognosis if not promptly diagnosed and managed.

The pathophysiology of ischemic stroke in the setting of CO poisoning is multifactorial. CO binds with high affinity to hemoglobin, displacing oxygen and forming carboxyhemoglobin, which impairs oxygen delivery and causes cellular hypoxia (3). In addition to hypoxic stress, CO triggers endothelial dysfunction, promotes platelet aggregation, and generates reactive oxygen species (ROS), leading to oxidative injury to cerebral vasculature (4,5). These mechanisms may result in vasospasm, microvascular thrombosis, or direct endothelial injury, culminating in ischemic injury to vulnerable brain regions (6).

Neuroimaging plays a pivotal role in identifying CO-related cerebral complications. While non-contrast CT may be normal or nonspecific in the early stages, MRI—especially diffusion-weighted imaging (DWI)—is more sensitive in detecting acute infarcts and subtle white matter changes (7). Common findings include bilateral globus pallidus lesions, but focal infarcts in the cortical or subcortical regions may also be visualized, particularly in cases with focal deficits (8).

Hyperbaric oxygen therapy (HBOT) remains the cornerstone of treatment in moderate to severe CO poisoning. It facilitates rapid dissociation of CO from hemoglobin and restores oxygenation to hypoxic tissues. Moreover, it has been shown to mitigate oxidative damage and may reduce the incidence of delayed neurologic sequelae (9,10). Though in our case, normobaric oxygen therapy had been given which is also an effecive alternative therapy (12). However, evidence regarding its impact specifically on ischemic stroke outcomes remains limited and mixed, necessitating further research (11).

CONCLUSION

Although rare, ischemic stroke represents an important neurological complication of carbon monoxide poisoning. Clinicians should maintain a high index of suspicion in patients with persistent focal neurological symptoms following CO exposure. Early neuroimaging, particularly MRI, is crucial in detecting ischemic changes. Timely initiation of hyperbaric oxygen therapy and a multidisciplinary approach involving neurology and critical care can improve outcomes. Further prospective studies are needed to clarify the incidence, risk factors, and optimal treatment strategies for stroke in the context of CO toxicity.

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