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Titolo

Carbon monoxide poisoning with bilateral globus pallidus lesions on MRI: cases report

Identificativo

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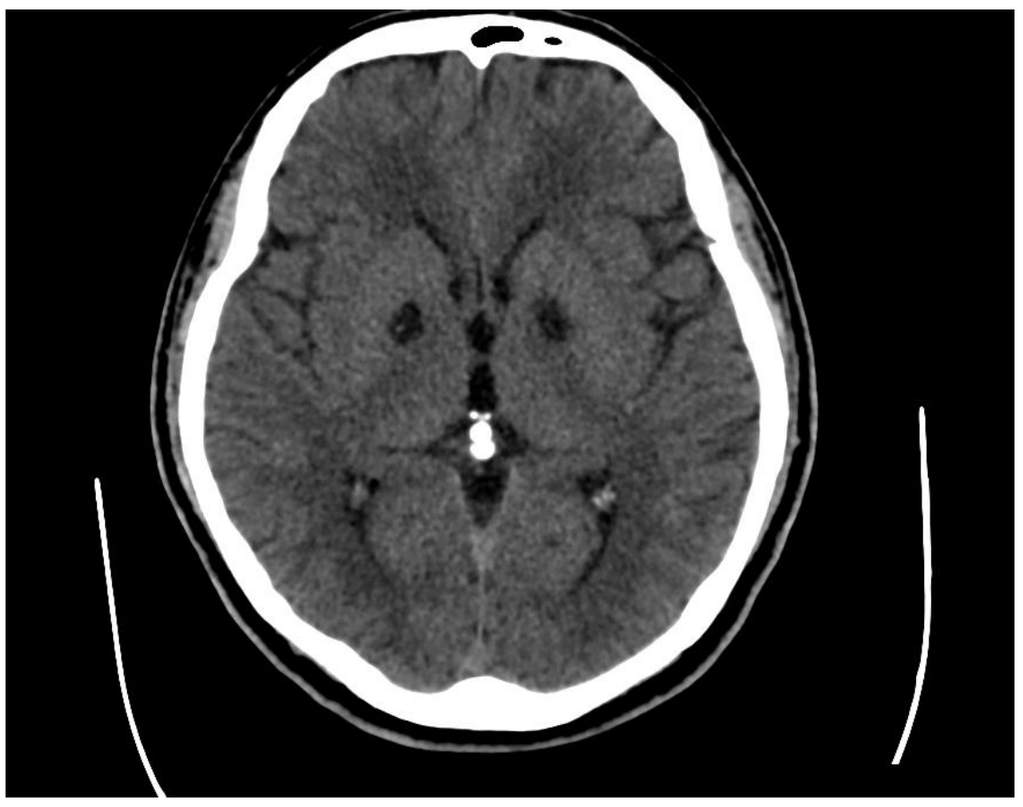
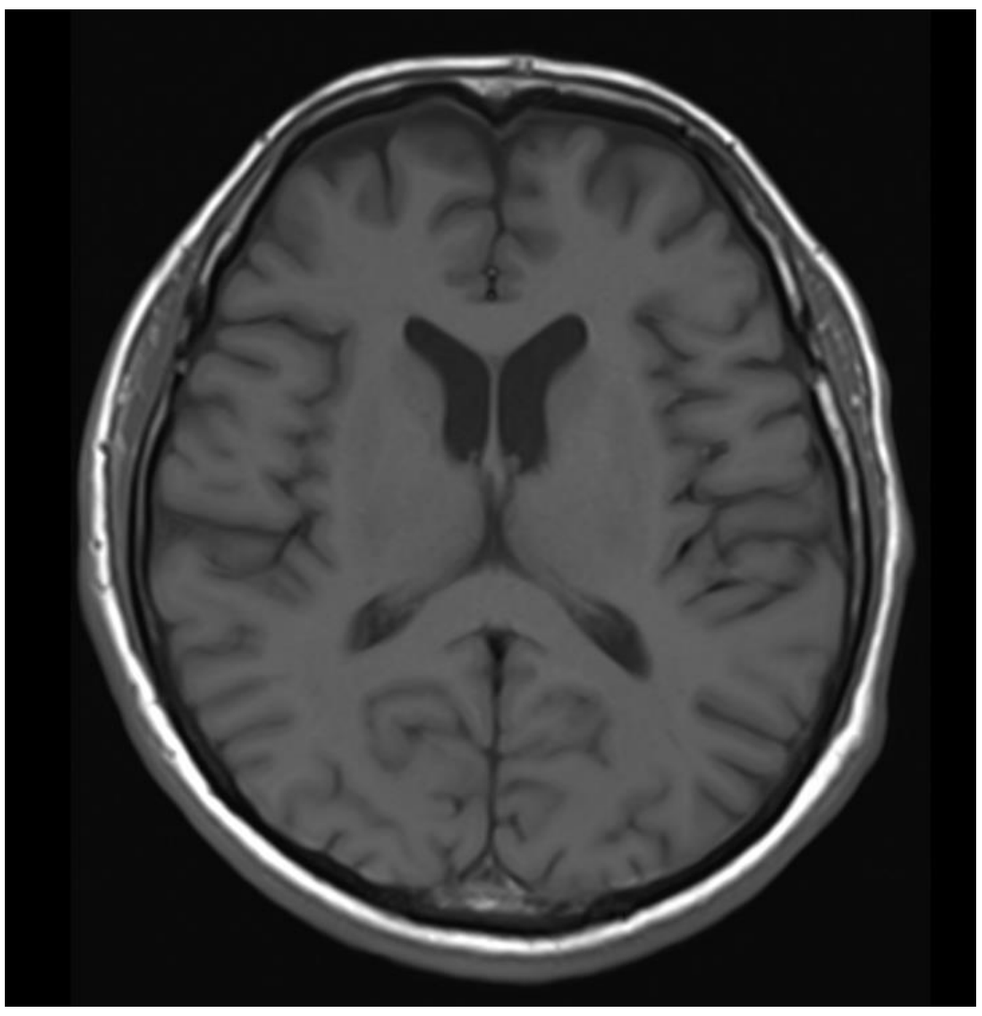
BACKGROUND: Carbon monoxide (CO) is a colorless, tasteless, odorless gas that is imperceptible to humans(1). Carbon monoxide (CO) has been the leading cause of poisoning mortality in many countries and hyperbaric oxygen (HBO) is a widely accepted treatment for CO poisoning.

The central nervous system is the most vulnerable structure in acute CO poisoning due to its high energy demands(2). MRI and related imaging modalities, the commonly used clinical imaging method, is important in assessing the severity of brain damage from CO poisoning and, to some extent, can predict the prognosis of CO poisoning brain damage (3). It has been reported that the bilateral basal ganglia, especially the hippocampus, and the centrum semiovale are the most common sites of lesion in acute CO poisoning MRI(3,4). Some patients with CO poisoning will still develop neurocognitive sequelae regardless of HBO therapy, which can persist since CO poisoning or be present days to weeks after a recovery from CO poisoning. HBO has been used in the prevention and treatment of neurocognitive sequelae.





CASE PRESENTATION: We report a case of a 38-year-old male who presented to the emergency department with loss of consciousness and unresponsive to verbal stimuli in the house. The diagnosis of CO poisoning was confirmed on the basis of their detailed history, physical examination and laboratory tests. According to the investigation, he had suffered accidental and prolonged CO poisoning. He was exposed in CO for more than 24 h. There was vomit beside him and he was not experiencing seizures. Then, he was transported to the hospital. In the emergency room, his vital

signs were normal with the Glasgow coma score (GCS) 4 with an intact pupillary light response and he was intubated. His arterial carboxyhemoglobin (COHb) was measured at 18%. Doctors treated him with hyperbaric oxygen therapy (HBOT) on 3 times. The patient became conscious 48 h after admission, his neurological function was largely restored. However, during of hospitalization, the patient suddenly developed neurological symptoms. He exhibited bradykinesia, apathetic attitude, frontal executive deficit (some difficulty in following orders given during the visit). Doctors considered him to have delayed neurological syndrome caused by CO poisoning. The MRI scan of his brain showed bilateral necrosis of the Globus pallidus and small area of left parietal cortical gliosis.

Neurocognitive assessment with Montreal Cognitive Assessment test and Raven matrices showed a deficit.

CONCLUSION: Long-term neurocognitive deficits occur in 15–40% of patients; imaging studies reveal cerebral white matter hyperintensities, with delayed posthypoxic leukoencephalopathy or diffuse brain atrophy. Conventional therapy is limited to normobaric and hyperbaric oxygen, with no available antidotal therapy. Although hyperbaric oxygen significantly reduces the permanent neurological and affective effects of CO poisoning, a portion of survivors still have substantial morbidity. This case explains how the severity of initial symptoms does not necessarily correlate with the development of longer-term neurological issues. The most common MRI findings are generally white matter hyperintensities (WMHs) and hippocampal atrophy(5,6). Although the metabolically active, ischemia-sensitive globus pallidus can be involved and it is not the most common site of abnormalities.

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Affiliazioni

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