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COgnitive crisis: unveiling neurological consequences of carbon monoxide poisoning: a case report

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Abstract: Carbon Monoxide (CO) is commonly emitted by vehicles, industrial processes, and heating systems. It can lead to unintentional poisoning in enclosed spaces. Common findings include tachycardia, hypotension, hypoxemia, headache, dizziness, seizures, or coma. A thorough history is essential to suspect potential CO poisoning. Confirmatory tests include measuring carboxyhemoglobin levels in blood. The first step of management is to remove the patient from the source, followed by oxygen supplementation. In severe cases, hyperbaric oxygen therapy may be indicated. Prognosis depends on the level of exposure, as well as the promptness of treatment. Early intervention leads to better outcomes. Delayed neurological deficits are potential long-term outcomes. Prognosis for recovery improves significantly with the use of CO detectors in homes and education about the risks of CO exposure.

We present a case of a 15-year-old male who presented with acute encephalopathy. Upon physical examination, laboratory study, and neuroimaging, a diagnosis of CO poisoning was made. The patient was intubated, placed on a ventilator, and managed conservatively with remarkable recovery.

Keywords: Brain Changes; Carbon Monoxide Poisoning; Hyperbaric Oxygen Therapy; MRI; Neuroimaging; Toxic Encephalopathy

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1. Introduction

Carbon monoxide (CO) poisoning is a leading cause of poisoning-related morbidity and mortality worldwide. It occurs when CO binds to hemoglobin, reducing oxygen delivery to tissues and leading to hypoxic injury, particularly in the brain. Neuroimaging, especially magnetic resonance imaging (MRI), has become essential in evaluating patients with suspected CO toxicity. MRI can reveal specific findings that correlate with the severity of the poisoning and the neurological sequelae.

2. Case presentation

A 15 -year-old male was found unconscious inside a closed room along with four other family members. The patient was transferred to the emergency department immediately. A malfunctioning heating system was in place, which caused the CO buildup. Initial assessment showed a feeble pulse with undetectable blood pressure and oxygen saturation. Neurological examination showed generalized hypotonia with bilateral plantar reflexes being mute and Glasgow coma scale (GCS) being E1V1M1. Arterial blood gas analysis revealed a PCO2 level of 54 mmHg (normal: 35 -45 mmHg) with a PO2 of 190 mmHg (normal: 80-100 mmHg). COoximetry was performed, which showed fraction of carboxy hemoglobin (FCOHb) of 0.8% (normal values: non-smokers: <1%, smokers: 2-10%) and fraction of methemoglobin (FMetHb) performed post-ventilation of 0.8% (normal range:<1-2%). Other laboratory results were insignificant. The diagnosis of CO poisoning was confirmed. The patient was immediately intubated and placed on mechanical ventilation in view of a GCS of 3. Ionotropic support started for hypotension along with intravenous fluids. The patient responded favorably to ventilation, with subsequent arterial blood gas analyses showing a significant reduction in PCO2 to levels of 38 mmHg and decreased need for inotropic support. The patient was successfully extubated on day 5, following an improvement. Brain MRI was performed after 1 week to assess potential brain injury. It showed T2-weighted and fluid-attenuated inversion recovery (FLAIR) hyperintensity of the genu, splenium, bilateral centrum semiovale, left basifrontal region, periventricular white matter of both frontal lobes, and left occipital lobe. Multiple microhemorrhages are seen in the subcortical and deep white matter of the bilateral cerebral hemispheres and along the corpus callosum (Figure 1).

Post-extubation, the patient was closely monitored for 3 days, during which no symptoms worsened. Consequently,

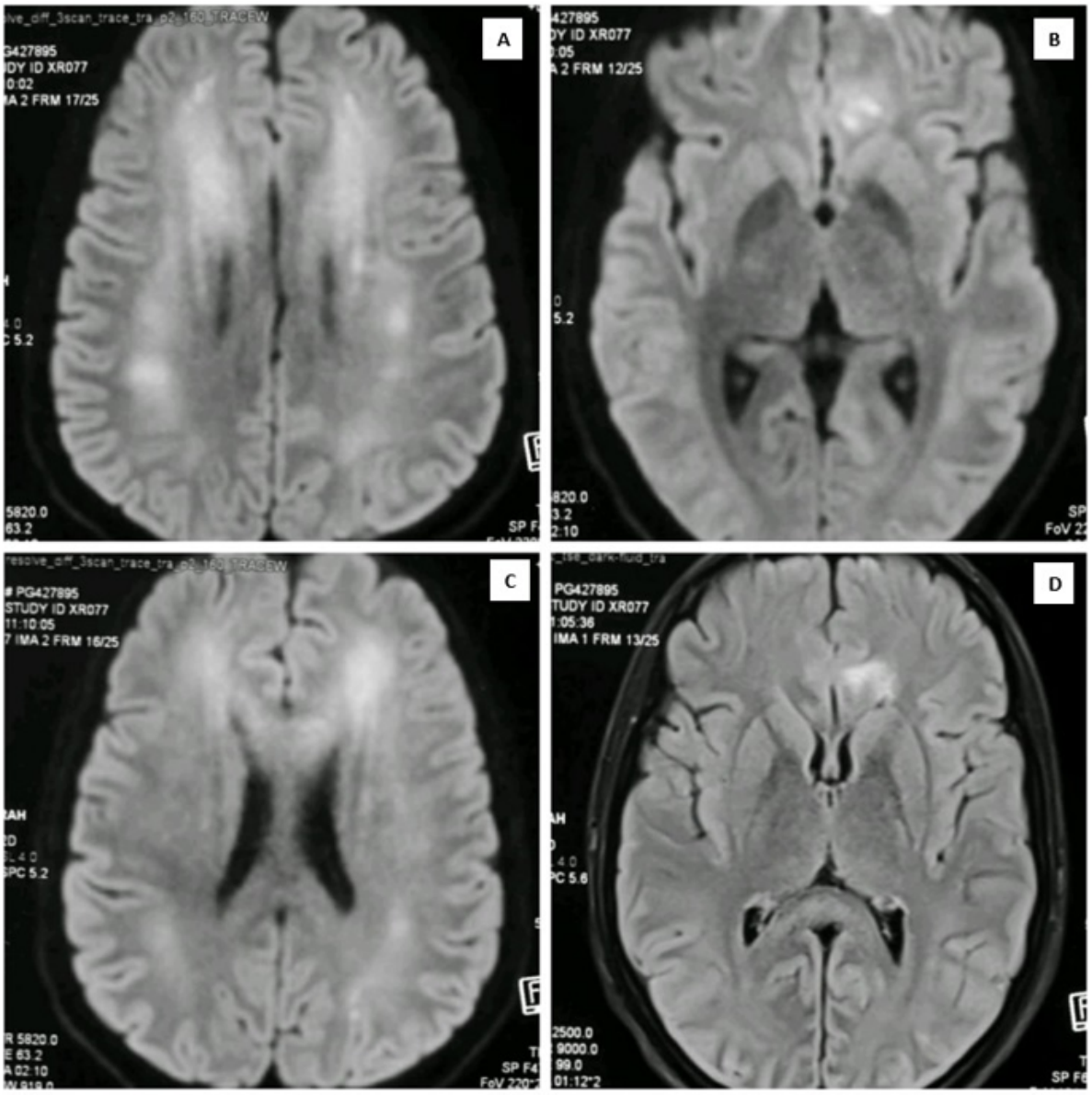

Figure 1 Brain MRI showing the following changes

Figure 1A Altered signal intensity in the form of T2-weighted and fluid-attenuated inversion recovery (FLAIR) hyperintensity with diffusion restriction involving the genu and splenium of corpus callosum, bilateral centrum semiovale, left basifrontal region, periventricular white matter of both frontal lobes, subcortical U fibers of left occipital lobe

Figure 1B A focus of T1 hyperintensity and T2/FLAIR hypointensity with surrounding edema showing susceptibility changes is seen in the genu of the corpus callosum on the left side, suggestive of acute microhemorrhage

Figure 1 C & 1D Multiple microhemorrhages are seen in the subcortical and deep white matter of the bilateral cerebral hemispheres and along the corpus callosum

the patient was discharged in a hemodynamically stable condition and continued to do well during follow-up appointments without any residual neurological deficit.

3. Discussion

This case highlights the importance of pattern recognition by neuroimaging in diagnosing CO poisoning in the context of appropriate clinical settings and laboratory parameters.

In 2021, the global age-standardized mortality rate for unintentional CO poisoning was approximately 0.353 per 100,000 people (1). In many industrialized countries, carbon monoxide is the cause of more than 50% of fatal poisonings. Reliance on traditional heating sources, such as wood and coal, is a persistent challenge in underdeveloped and developing (1).

When multiple family members are found unconscious to-

gether, the initial suspicion often falls on poisoning, especially during the winter. However, there is misdiagnosis risk in patients with milder symptoms as carbon monoxide poisoning and other illnesses can present with headache, dizziness, nausea, and vomiting (2,3). Delayed treatment due to misdiagnosis can worsen outcomes and increase the risk of long-term health consequences (3). In carbon monoxide (CO) poisoning, pulse oximetry is unreliable because it cannot differentiate between oxyhemoglobin (HbO_2) and carboxyhemoglobin (COHb), often displaying a falsely normal or high oxygen saturation (SpO_2). This occurs because the device interprets COHb as HbO_2 due to similar light absorption characteristics (4). In contrast, CO-oximetry is the standard for detecting CO poisoning. It uses multiple wavelengths of light to accurately measure various forms of hemoglobin, including oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, and methemoglobin. In cases of CO poisoning, CO-oximetry reveals elevated COHb levels, which may exceed 10% in smokers and 15–20% in non-smokers (4). Carbon monoxide poisoning causes widespread hypoxic-ischemic injury to the central nervous system (CNS), which is often reflected in imaging findings (3). While initial clinical findings may not always correlate with the extent of brain injury, neuroimaging remains an invaluable tool for identifying the affected regions. In addition, cerebral white matter, cerebral cortex, cerebellum, hippocampus, amygdala, splenium of corpus callosum, and insula might be affected. This higher susceptibility is thought to be due to its relatively high oxygen consumption and perfusion (5,6). Non-contrast CT (NCCT) images of the brain shows bilateral symmetrical hypodensities in the globus pallidus of basal ganglia (5). MRI findings in CO poisoning can vary based on the timing of the scan and the severity of the exposure. Initially, imaging may show edema, which can evolve into necrosis or gliosis if the injury is severe. Diffusion-weighted imaging (DWI) and FLAIR sequences are particularly sensitive in detecting early changes, even when clinical symptoms may not fully correlate with the degree of brain injury (7,8).

While oxygen therapy is the mainstay treatment, hyperbaric oxygen therapy (HBOT) is used in moderate to severe carbon monoxide (CO) poisoning to rapidly eliminate COHb, improve tissue oxygenation, and reduce the risk of delayed neurological sequelae (4). It significantly shortens COHb half-life to about 20–30 minutes. HBOT is typically indicated in patients with COHb>25% (or >15% in pregnancy), loss of consciousness, neurological symptoms, cardiovascular instability, or severe acidosis. While evidence is mixed, it is considered beneficial in high-risk cases. Treatment should be individualized, considering clinical severity and resource availability (4,9).

Delayed neuropsychiatric syndrome (DNS) is a complication of carbon monoxide (CO) poisoning that occurs days to weeks after initial recovery. It is characterized by a return or emergence of symptoms such as memory loss, personality changes, mood disorders, movement abnormalities, and cognitive impairment. DNS is thought to result from delayed demyelination due to hypoxic and immune-mediated injury, often affecting brain white matter. MRI may show diffuse white matter changes or basal ganglia lesions. Risk factors include severe poisoning, prolonged exposure, older age, and high COHb levels. Recovery varies; some patients improve over time, while others may have lasting deficits. Hyperbaric oxygen therapy may help reduce the risk of DNS.

4. Conclusion

This case highlights the critical role of brain MRI in evaluating patients with CO poisoning in the context of an appropriate clinical setting. The various patterns of involvement of different regions of the brain can assist in diagnosing CO toxicity and predicting the potential for long-term neurological sequelae.

5. Declarations

5.1. Acknowledgement

None.

5.2. Authors' contribution

All authors contributed to the manuscript equally.

5.3. Conflict of interest

None.

5.4. Funding

None.

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