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Case Report

Laminar Cortical Necrosis Due to CO Poisoning in Young Adult

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Abstract

Carbon monoxide (CO) exposure is a common cause of toxic brain damage, ranging from transient neurological dysfunction to coma and death. Consequently, hypoxic brain damage is most notable in regions like the globus pallidus, basal ganglia, cerebral white matter, and the cerebral cortex. Diagnosis necessitates a heightened level of suspicion, alongside clinical assessment, analysis of patient carboxyhemoglobin (COHb) levels. Additionally, cardiology evaluation and neurological assessment, encompassing brain imaging especially Brain MRI, are essential components of the diagnostic process. This article reports a case of a young patient exposed to CO because of an apartment house's faulty gas heater. The MRI revealed cerebral lesions characterized as extensive and isolated laminar cortical necrosis (LCN), affecting both cerebral hemispheres.

Keywords: *Carbon Monoxide; Brain Toxicity; Laminar Cortical Necrosis; Magnetic Resonance Imaging*

Abbreviations

CO: Carbon Monoxide; COHb: Carboxyhemoglobin; MRI: Magnetic Resonance Imaging; LCN: Laminar Cortical Necrosis

Introduction

Laminar cortical necrosis is neuropathologically defined as focal or diffuse necrosis of one or more cortical lamina. It has been described in association with a variety of disorders, most frequently with cerebral hypoxia and cerebral ischemia [1], while isolated LCN due to Carbon monoxide (CO) exposure is an uncommon condition, often leading to coma and death [2,3]. MRI is the preferred diagnostic modality for accurately diagnosing and precisely localizing the location and extent of lesions.

Case Presentation

A 28-year-old young patient with no significant medical history was admitted to the intensive care unit in our hospital due to consciousness impairment following carbon monoxide (CO) poisoning. Upon clinical examination, a comatose state was observed, with a Glasgow Coma Scale score of 8.

Following appropriate management and treatment with immediate O_2 breathing and hyperbaric oxygen therapy (HBO), the progression was characterized by delayed awakening.

A cerebral MRI was conducted using T1-weighted, T2-weighted, FLAIR, and diffusion-weighted sequences. The MRI revealed the presence of a hyper-intense signal in FLAIR sequences (Figure 1) with diffusion restriction (Figure 2), these lesions showed a hypo-intense signal in T1 with no evidence of haemorrhage in T2* sequence (Figure 3). These lesions involved parieto-temporo-occipital cortical region of both hemispheres, sparing gray nuclei. These findings are suggestive of a diagnosis of cortical laminar necrosis. The patient's condition deteriorated, ultimately leading to their demise.

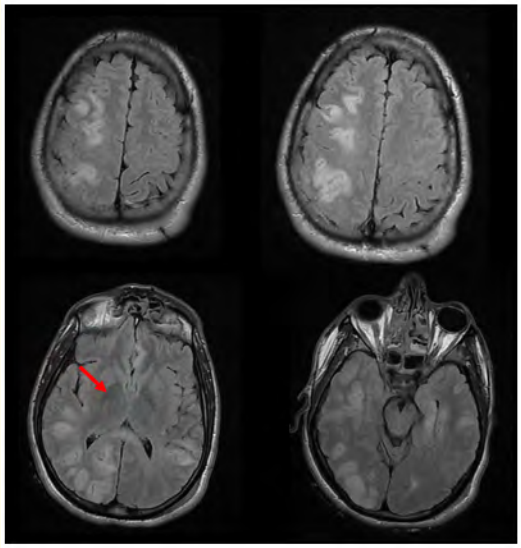

Figure 1: *Axial FLAIR images showing the hyper-intense signal of the cortical lesions sparing the gray nuclei (red arow).*

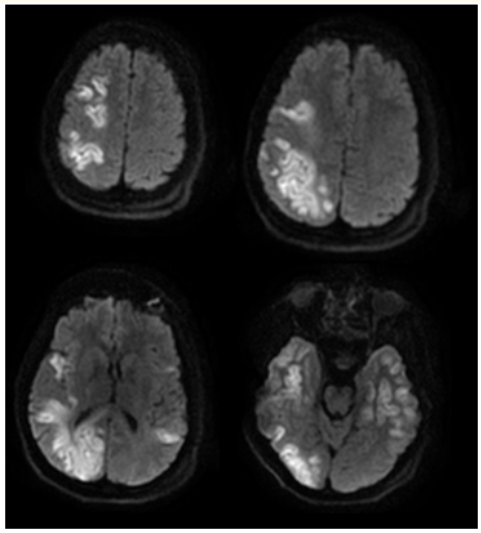

Figure 2: *Diffusion sequence images showing the diffusion restriction of the parieto-temporo-occipital cortical lesions.*

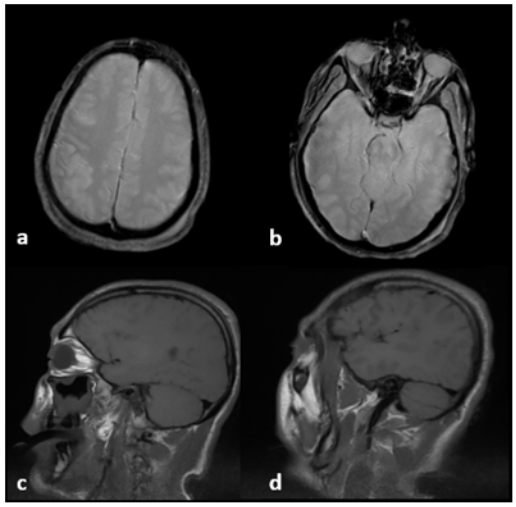

Figure 3: *Axial T2* sequence images (a, b) and para sagittal T1 images (c, d) showing the absence of brain haemorrhage.*

Discussion

CO intoxication is one of the most common types of poisoning in the world. It is a highly toxic gas. The incidence of CO poisoning is unknown and many cases probably go unrecognized [2]. CO binds rapidly to Hb, since the affinity of hemoglobin for CO is 210 times higher than its affinity for oxygen, leading to the formation of carboxyhemoglobin (COHb). The oxygen carrying capacity of the blood decreases, causing tissue hypoxia [2,3].

Acute carbon monoxide intoxication primarily leads to impairments in brain function. Additionally, delayed neurological consequences can manifest. Tissue hypoxia is the ultimate outcome of CO poisoning [3]. Certain brain areas are particularly vulnerable to hypoxic injury, encompassing, the basal nuclei, the cerebral cortex, specifically its second and third layers, the white matter, and the Purkinje cells of the cerebellum [2,3].

The gray matter, being more susceptible than the white matter; as neurons have a higher metabolic demand, it can undergo isolated damage, defining as laminar cortical necrosis [1,2]. This term denotes a form of neuronal ischemic remodelling that results most frequently from hypoxia. Among the cortical layers, the third layer is particularly vulnerable. Histologically, cortical laminar necrosis corresponds to the preferential loss of neurons in layers III and V. Depending on the severity of the involvement, the extent of cortical necrosis can vary, often accompanied by the presence of macrophages, gliosis, and vascular neogenesis [1,4]. in case of LCN due to CO poisoning, next to hypoxia, another proposed mechanism for ongoing inflammation in the brain is an immunologic response [3].

On MRI, laminar cortical necrosis is characterized by permanent, focal or diffuse cortical high signals on T1-weighted and fluid-attenuated inversion-recovery (FLAIR) images, which follow the gyral anatomy of the cerebral cortex. The lesions exhibit hyperintensity on the diffusion sequence, within the initial 24 hours and throughout the first three weeks. The T2 gradient echo sequences do not reveal any cortical lesion hypointensity, effectively excluding a hemorrhagic origin [1,5,6]. An indication of a ruptured blood-brain barrier, indicated by cortical contrast enhancement, becomes noticeable within the initial weeks and can persist for up to nine months. In the chronic stage cortical atrophy may develop [5].

Beyond hypoxia, which can arise from cardiac failure or intoxication, the causes of LCN are varied and represented by ischemia, hypoglycemia, extended arterial hypotension, epileptic episodes, renal or hepatic dysfunction, immunological triggers like antiphospholipid syndrome or lupus, as well as involvement during encephalitis of infectious origin [7-10]. Clinically, the manifestation is marked by disruptions in consciousness, at times accompanied by motor or sensory deficits [8].

The prognosis of LCN is unfavorable, marked by elevated morbidity and mortality rates. Unfortunately, there is no specific curative treatment available; the approach primarily hinges on the underlying cause [1,9]. The primary treatment approach for CO poisoning hinges on administering 100% normobaric oxygen and hyperbaric oxygen therapy, especially when the patient experiences any period of unconsciousness [2,3]. Our patient received both forms. However, due to the extensive cortical lesions, the course of progression was marked by delayed awakening, worsening condition, and ultimately, the unfortunate outcome of death.

Conclusion

Laminar cortical necrosis is a severe complication resulting from cerebral hypoxia, which can be caused by various underlying factors. MRI is the preferred diagnostic method for confirming this condition and assessing the extent of lesions, which ultimately influences the prognosis. In our case, the cause was carbon monoxide (CO) intoxication. Unfortunately, the outcome proved fatal due to the extensive nature of the lesions.

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