Carbon Monoxide Poisoning: Bilateral Globus Pallidus Lesions as a Key Imaging Clue

Intoxicação por Monóxido de Carbono: Lesões Bilaterais nos Globos Pálidos como Pista Imagiológica Clássica

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Palavras-chave: Globo Pálido/diagnóstico por imagem; Intoxicação por Monóxido de Carbono/diagnóstico por imagem.

Teaching Point

1. Imaging is essential for the diagnosis of carbon monoxide poisoning. Bilateral globus pallidus lesions are a classic imaging finding.

A 67-year-old woman, together with two relatives, developed weakness and drowsiness. They were evaluated in the emergency department, where a viral illness was suspected, and were discharged. Two days later, the patient, previously independent, was found prostrate and was brought to the emergency department.

On examination, she was drowsy but arousable to verbal stimuli and capable of answering simple questions. She was disoriented in time and place and vital signs were normal. Arterial blood gas analysis on room air showed: pH 7.50, pO_2 62 mmHg, pCO_2 35 mmHg, and HCO_3^- 27 mmol/L, consistent with mild respiratory alkalosis and hypoxemia.

An emergent brain computed tomography (CT) showed bilateral globus pallidus hypodensities (Fig. 1), a pattern suggestive of carbon monoxide (CO) poisoning. Subsequent brain magnetic resonance imaging (MRI) confirmed bilateral T1 hypointensity (Fig. 2a), T2-FLAIR hyperintensity (Fig. 2b), and restricted diffusion (Fig. 2c–d), consistent with acute injury, with no additional findings.

Hypoxic–ischemic injury was considered in the differential diagnosis, but the absence of lesions in other vascular territories is unusual. The bilateral distribution of lesions suggested a toxic or metabolic aetiology. Further investigation identified a domestic water heater as the probable source of carbon monoxide exposure, and the diagnosis was supported by the imaging findings and anamneses.

Figure 1: Brain CT: bilateral hypodensity of globus pallidus.

CO is a frequent cause of toxic neurological injury because of its high affinity for haemoglobin, resulting in impaired oxygen delivery, tissue hypoxia, and cellular death.[1]

Clinical manifestations are nonspecific and include dizziness, confusion, and coma,[2] making early suspicion challenging. Imaging plays a key diagnostic role: CT may not show abnormalities, so MRI is the gold standard.[2] *Globi pallidi* lesions are a classic finding, due to its vulnerability to hypoxia.[2]

Management consists of airway protection and administration of 100% oxygen.[3] Survivors of CO exposure are at risk of secondary parkinsonism, related to injury of the globi pallidi.[2] The patient developed dystonia and tremor and was treated with botulinum toxin, with improvement of symptoms. ■

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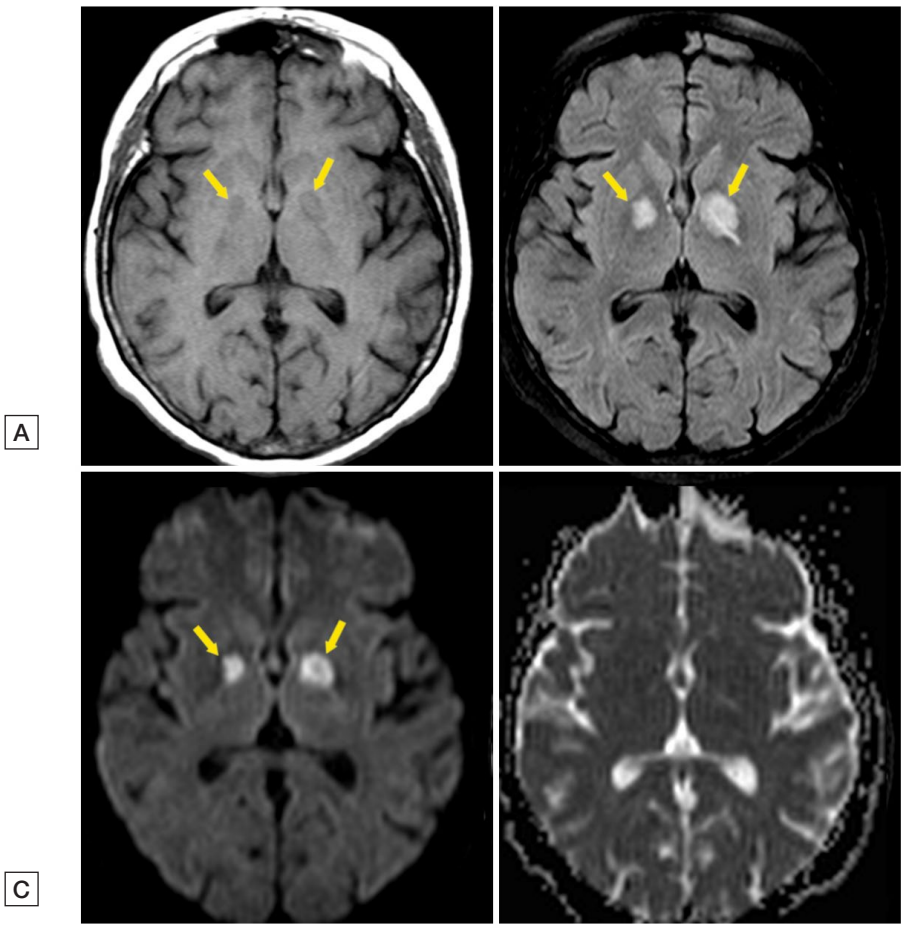

Figure 2: MRI shows **[A]** T1-weighted bilateral hypointensity, with **[B]** T2-FLAIR hyperintensity, and **[C-D]** restricted diffusion in globus pallidus.

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