Bilateral Globus Pallidus T1 Hyperintensity in Hepatic Cirrhosis

Hiperintensidades de Globos Pálidos en Cirrosis Hepática

Oscar H. Del Brutto,[1] Denisse A. Rumbea,[2] Emilio Arias,[2] Eduardo J. Guzmán[3]

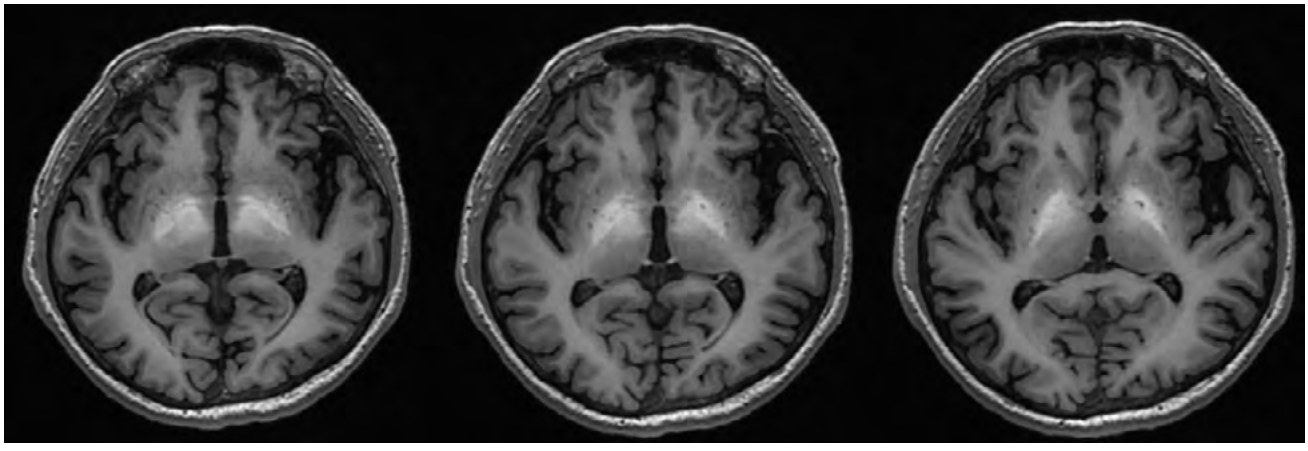

Figure 1. T1-weighted MRI showing bilateral and symmetric lesions in the globi pallidi.

Bilateral symmetric T1 hyperintensities in the globus pallidus on MRI are a hallmark of manganese deposition secondary to hepatic dysfunction, reflecting portosystemic shunting and subclinical hepatic encephalopathy.[1]

A 60-year-old man with history of diabetes mellitus type 2, and chronic liver disease, presented with troubling gait. The rest of the neurological examination was unremarkable. Cognitive performance was normal, as evidence by a Montreal Cognitive Assessment test of 25 points. T1-weighted MRI revealed bilateral and symmetric hyperintensities in the globus pallidus (Figure 1), a classic radiological finding in patients with chronic liver disease, particularly hepatic cirrhosis. Manganese is excreted via bile; however, in cirrhosis, compromised hepatobiliary function and collateral circulation allow manganese to bypass hepatic metabolism and accumulate in the brain.

This phenomenon is part of the spectrum of acquired hepatocerebral degeneration, a chronic neuropsychiatric syndrome distinct from overt hepatic encephalopathy. Patients may be asymptomatic or present with subtle cognitive deficits, extrapyramidal symptoms, or parkinsonism.[2] Importantly, the T1 hyperintensity does not necessarily correlate with serum ammonia levels or acute encephalopathy but rather reflects chronic exposure to neurotoxic substances due to liver insufficiency.[3]

The finding is non-specific but highly suggestive in the appropriate clinical context. Differential diagnoses include Wilson disease, non-ketotic hyperglycemia, and prior gadolinium exposure, but the symmetry and clinical background of cirrhosis strongly support manganese-related acquired hepatocerebral degeneration. Recognition of this imaging pattern is important, as it may prompt further evaluation of subclinical neurocognitive impairment and guide management strategies, including optimization of liver function and consideration of transplant candidacy.

[1]School of Medicine, Universidad Espíritu Santo – Ecuador. Samborondón, Ecuador.
[2]Research Center, Universidad Espíritu Santo – Ecuador. Samborondón, Ecuador.
[3]Mediscan Imaging Center. Guayaquil, Ecuador.

Correspondencia:
Oscar H. Del Brutto, MD.
Urbanización Toscana, Apt 3H, Km 4.5 vía Puntilla-Samborondón, 092301, Samborondón – Ecuador.
E-mail: oscardelbrutto@hotmail.com

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References

1. Kwong AJ, Zahr NM. Serum biomarkers of liver fibrosis identify globus pallidus vulnerability. Neuroimage Clin. 2023;37:103333. https://doi.org/10.1016/j.nicl.2023.103333
2. Kikui S, Danno D, Takeshima T. Reversible subacute parkinsonism and cognitive decline in a patients with acquired hepatocerebral degeneration: a case report. Cureus. 2025;17:e93431. https://doi.org/10.7759/cureus.93431
3. Kulisevsky J, Pujol J, Balanzó J, et al. Pallidal hyperintensity on magnetic resonance imaging in cirrhotic patients: clinical correlations. Hepatology. 1992;16:1382-1388. https://doi.org/10.1002/hep.1840160613

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