

Doença de Marchiafava-Bignami: Uma Complicação Rara do Abuso do Álcool

Marchiafava-Bignami Disease: A Rare Complication of Alcohol Abuse

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Introduction

Chronic alcohol abuse is a common disorder. Its effects include neurologic complications through both direct and indirect effects on the central and peripheral nervous systems. Marchiafava-Bignami disease (MBD) is caused by demyelination and necrosis of the corpus callosum and it has long been considered to be of either a toxic or nutritional etiology.¹

history of illicit drug use. On initial assessment his mini mental state scale was 19/30, he was disorientated in time and place, with reduced speech output, psychomotor retardation, but with no focal signs. His blood pressure was normal and he had no fever. Cerebral computerized tomography revealed a callosal hypodensity and global atrophy, with no acute vascular lesions (Fig. 1A). Laboratory studies disclosed mild macrocytic anemia and elevated liver enzymes; renal, thyroid and liver function were normal; systemic inflammatory biomarkers and serum ethanol were negative. Abdominal ultrasonography showed signs of steatosis, with no splenomegaly or ascites. Brain magnetic resonance imaging (MRI) confirmed the callosal lesion, with restricted diffusion (Fig. 2B) and showed T2/FLAIR hyperintensities

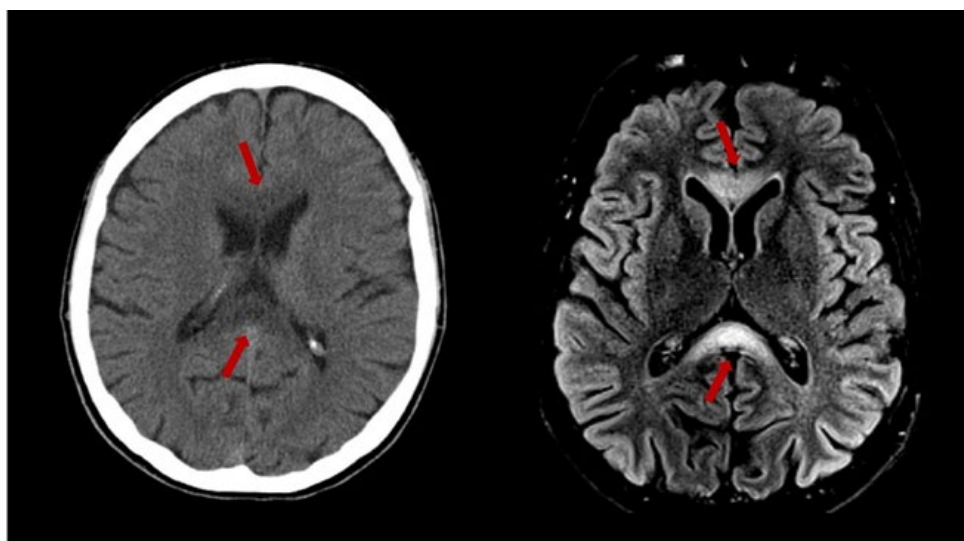


Figure 1: Axial computed tomography (CT) and FLAIR.

Case Report

A 43-year-old man, with chronic alcoholism (over 120 g daily), was brought to the emergency department after a two-week period of behavioral changes. There was no

concerning callosal genu, body and splenium (Figs 1A and 2A), suggesting MBD. He was started on several vitamin supplementation (including thiamine), with a good clinical outcome.

Discussion

MBD pathophysiology includes cytotoxic edema, demyelination and callosal necrosis, presumably attributed to the combination of alcohol-induced neurotoxicity and deficiency

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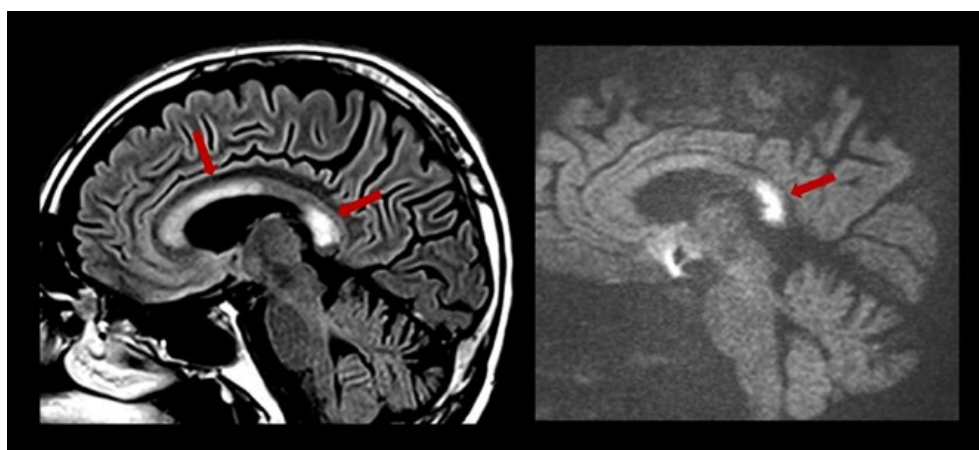


Figure 2: Sagittal FLAIR and DWI.

of B-complex vitamins.²⁻⁵ The clinical course is broad and some cases may evolve to rapid mental state deterioration and death.¹

There are no treatment guidelines but some reports suggest a favorable response to thiamine, enhancing the synergism between alcohol neurotoxic effects and hypovitaminosis B1 (responsible for Wernicke encephalopathy).⁴ Our patient improved with thiamine reposition which shows the importance of early management. ■

Responsabilidades Éticas

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Proteção de Pessoas e Animais: Os autores declaram que os procedimentos seguidos estavam de acordo com os regulamentos estabelecidos pelos responsáveis da Comissão de Investigação Clínica e Ética e de acordo com a Declaração de Helsínquia da Associação Médica Mundial.

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Ethics of the World Medical Association (Declaration of Helsinki).

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