



FROM BOOZE TO BRAIN FOG- A THIAMINE TALE OF SURVIVAL

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ABSTRACT Marchiafava-Bignami disease (MBD) is a rare, alcohol-related neurological disorder characterized by demyelination and necrosis of the corpus callosum. We present a case of a 46-year-old chronic alcoholic male who developed acute neurological deficits following a fall. MRI revealed characteristic lesions in the splenium of the corpus callosum. Early intervention with high-dose thiamine and multidisciplinary care resulted in complete neurological recovery. This case highlights the critical role of timely thiamine repletion, the importance of neuroimaging in diagnosis, and the need for a structured rehabilitation approach in MBD management.

KEYWORDS :**INTRODUCTION**

Marchiafava-Bignami disease (MBD) is a rare condition predominantly affecting chronic alcoholics, with pathological features resembling Wernicke's encephalopathy. The disease involves selective demyelination and necrosis of the corpus callosum, leading to a spectrum of neurological deficits.¹ Early recognition and treatment are crucial to prevent irreversible damage

CASE PRESENTATION

A 46-year-old male with a long-standing history of heavy alcohol use presented to the emergency department following a fall from stairs, sustaining head trauma to the left parieto-temporal region. Post-injury, he developed:

- Right-sided hemiparesis (upper and lower limbs)
- Altered sensorium (disorientation, confusion)
- Dysarthria (slurred speech)

Diagnostic Workup

1. MRI Brain with MRA:
 - Revealed hyperintensities in the splenium of the corpus callosum on T2/FLAIR sequences, suggestive of demyelination and necrosis.
 - No evidence of acute infarction or hemorrhage.
2. CSF Analysis:
 - Normal cell count, protein, and glucose—ruling out meningitis or encephalitis.
3. 2D Echocardiography:
 - Normal cardiac function (EF 60%), no structural abnormalities.
4. USG Abdomen & Pelvis:
 - Findings consistent with alcoholic liver disease (fatty infiltration, hepatomegaly).
5. Additional Findings:
 - Cellulitis in lower limbs (likely due to poor hygiene and malnutrition).

MANAGEMENT

1. MRI Brain with MRA:
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DISCUSSION

Pathophysiology of MBD

MBD is strongly linked to chronic alcoholism and malnutrition, particularly thiamine (B1) deficiency. The corpus callosum is highly susceptible to metabolic insult, leading to demyelination, edema, and necrosis.² The exact mechanism remains unclear but may involve:

- Oxidative stress from alcohol metabolites.
- Microvascular injury due to chronic toxicity.

- Glutamate excitotoxicity in vulnerable white matter tracts.

Key Diagnostic Clues

1. Neuroimaging (MRI):
 - T2/FLAIR hyperintensities in the corpus callosum (splenium most common).
 - Diffusion restriction in acute phases.³
2. Exclusion of Mimics:
 - Stroke (ischemic or hemorrhagic).
 - Infectious encephalopathy (e.g., HSV encephalitis).
 - Metabolic encephalopathies (e.g., hepatic encephalopathy).

Therapeutic Approach

- Immediate thiamine replacement (500 mg IV TID in severe cases).⁴
- Multidisciplinary care:
 - Neurology for seizure prophylaxis if needed.
 - Nutrition for long-term vitamin repletion.
 - Physical therapy to prevent contractures and improve mobility.

Prognostic Factors

- Early thiamine administration correlates with better outcomes.⁵
- Extent of corpus callosum involvement predicts residual deficits.
- Concurrent liver disease may complicate recovery.

CONCLUSION

This case underscores the diagnostic and therapeutic challenges of Marchiafava-Bignami disease. Key takeaways include:

1. High clinical suspicion in chronic alcoholics with acute neurological decline.
2. MRI is the gold standard for diagnosis.
3. Aggressive thiamine therapy can reverse deficits if initiated early.
4. Long-term abstinence and nutritional support are essential to prevent relapse.

A coordinated team approach involving neurologists, internists, physiotherapists, and dietitians is critical for optimal recovery.

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