Case Report

Marchiafava-Bignami syndrome with Wernicke-Korsakoff overlap: A rare case of post-binge encephalopathy in a chronic alcoholic

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ABSTRACT

Marchiafava-Bignami syndrome (MBS) is a rare demyelinating disorder of the corpus callosum, predominantly seen in chronic alcoholics and malnourished individuals. It is characterized by demyelination and necrosis of the corpus callosum, often presenting with neuropsychiatric manifestations. We report a 32-year-old male with a long history of alcohol abuse who developed altered sensorium and cognitive impairment following a binge-drinking episode. Magnetic resonance imaging revealed diffuse T2/fluid-attenuated inversion recovery hyperintensities involving the genu and splenium of the corpus callosum, bilateral thalami, internal capsules, and brainstem, suggestive of MBS with associated Wernicke-Korsakoff overlap. Cerebrospinal fluid analysis and oligoclonal bands were negative. The patient showed partial improvement in sensorium following thiamine therapy. This case highlights the importance of early recognition of alcohol-related demyelinating encephalopathies to prevent irreversible neurological damage.

Key words: Chronic alcoholism, Corpus callosum, Marchiafava-Bignami syndrome, Magnetic resonance imaging brain, Thiamine deficiency, Wernicke-Korsakoff syndrome

Marchiafava-Bignami syndrome (MBS) is a rare [1], potentially fatal disorder characterized by demyelination and necrosis of the corpus callosum, particularly the central fibers of the body, genu, and splenium. First described in 1903 [2] in Italian red wine drinkers, MBS is strongly associated with chronic alcoholism and malnutrition. It often presents with neuropsychiatric features, cognitive impairment, and interhemispheric disconnection. MBS can occur as an isolated entity or overlap with Wernicke-Korsakoff syndrome (WKS), resulting in a spectrum of alcohol-related encephalopathies. Neuroimaging, particularly magnetic resonance imaging (MRI), plays a pivotal role in diagnosis.

We report a case of a young male chronic alcoholic presenting with altered behavior and cognitive decline, later diagnosed as MBS with Wernicke-Korsakoff overlap.

CASE REPORT

A 32-year-old male with a 10–15-year history of chronic alcohol consumption (4–5 drinks daily) presented with altered behavior, drowsiness, and progressive memory impairment following a binge-drinking episode 1.5 months prior. The patient had a smoking history of approximately 8 pack-years as well. The patient had no known history of hypertension, diabetes, tuberculosis, chronic liver disease, seizures, psychiatric illness, or prior neurological disorders. He was not on any long-term medications and had no known drug allergies. There was no similar neurological or psychiatric illness in the family. The patient belonged to an upper-middle-class background according to the modified Kuppuswamy socioeconomic scale. After the episode, he developed confusion, forgetfulness for recent and remote events, and difficulty recognizing friends and relatives. He also reported decreased oral intake over the preceding 2 weeks.

On examination, the patient was conscious but disoriented, not following verbal commands. Cranial nerve examination was unremarkable. Muscle tone and deep tendon reflexes were normal with bilateral flexor plantar response. No signs of meningeal irritation were present. Systemic examination was within normal limits. On admission, vitals were stable with a blood pressure of 114/85 mmHg, pulse rate of 84/min, Temperature of 99°F, and SpO_2 of 99% on room air. Neurological examination was done, which showed phonation was absent initially for 2–3 days. The patient gradually developed some phonation, and by the 10th day, he was able to articulate small words and form sentences with improved language fluency. Attention was initially untestable due to altered

Access this article online	
Received - 14 October 2025 Initial Review - 29 October 2025 Accepted - 17 November 2025	Quick Response code
DOI: 10.32677/ijcr.v11i12.7903	

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sensorium. By the 7th day, digit span was 2–3 forward and 0 backward, improving to 5 and 3, respectively, at discharge. Initially, ocular movements could not be assessed, but later became full and normal as the sensorium improved.

Routine laboratory investigations revealed mild elevation of transaminases (serum glutamic-oxaloacetic transaminase 84 U/L and serum glutamic-pyruvic transaminase 88 U/L), normal renal function, and normal electrolytes except mild hyponatremia (sodium 127 mEq/L) at admission. Viral markers (Human immunodeficiency virus, hepatitis B surface antigen, and hepatitis C virus) were negative. Cerebrospinal fluid examination showed glucose 92 mg/dL, protein 94 mg/dL, and 2–5 cells/mm^3; oligoclonal bands were negative. Visual evoked potentials (VEPs) were normal.

MRI of the brain demonstrated multiple T2/fluid-attenuated inversion recovery (FLAIR) hyperintense lesions involving the genu and splenium of the corpus callosum, bilateral periventricular deep white matter, posterior limbs of the internal capsule, thalami, anterior commissure, pons, and medulla. Diffusion restriction was noted in the posterior limb of the left internal capsule. The corpus callosum appeared bulky with effacement of the lateral ventricles. These findings were consistent with MBS. Whole-spine MRI revealed hyperintensity in the proximal cervical cord at C1–C2. No abnormal leptomeningeal enhancement was seen (Figs. 1 and 2).

The patient was treated with high-dose intravenous thiamine (500 mg TDS), methylcobalamin (1 g daily), multivitamin supplements, and supportive care. Over the course of treatment, his sensorium improved partially, with better orientation and reduced confusion. However, residual cognitive deficits persisted at discharge. At discharge, his sensorium improved to E4V5M6. The patient, however, required assistance with daily activities. On follow-up after 7 days, he was able to perform basic self-care tasks such as eating, brushing, dressing, and using his phone, but still needed help for mobility and complex tasks such as cooking or climbing stairs.

DISCUSSION

MBS represents a spectrum of alcohol-related demyelinating encephalopathies (Table 1). The pathogenesis is thought to involve nutritional deficiency, particularly thiamine, leading to disruption of myelin integrity in the corpus callosum. The disease is classified into acute, subacute, and chronic forms, with the acute variant often presenting with coma and seizures, and the subacute form presenting with progressive cognitive impairment and gait ataxia. In this case, the patient presented subacutely after a binge episode, a typical setting for MBS. In this patient, the overlap with WKS was evident both clinically and radiologically. Clinically, the patient demonstrated features of Wernicke's encephalopathy, such as confusion and ataxia, along with cognitive decline and memory impairment typical of Korsakoff psychosis. Radiologically, MRI findings showed callosal demyelination consistent with MBS and bilateral thalamic involvement suggestive of WKS, confirming an overlap pathology.

MRI findings are diagnostic in most cases [3]. The hallmark is symmetrical lesions in the corpus callosum, typically the body and splenium, which appear hyperintense on T2 and FLAIR sequences. Involvement of extracallosal structures [4] such as the internal capsules, thalami, and cerebellar peduncles, as in this patient, is less common but reported [5]. Differentials such as Wernicke's encephalopathy, multiple sclerosis, and acute disseminated encephalomyelitis (ADEM) were

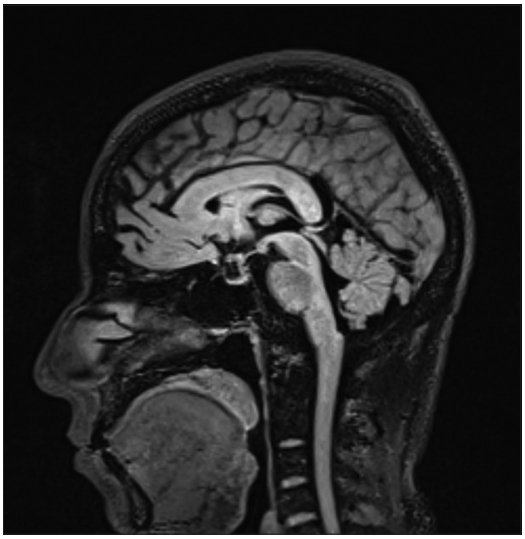

Figure 1: Sagittal T1-weighted magnetic resonance imaging brain showing diffuse thickening and hypointensity of the corpus callosum, particularly involving the genu and splenium, causing effacement of the body of the lateral ventricles. These findings are suggestive of demyelination and edema characteristic of Marchiafava-Bignami syndrome

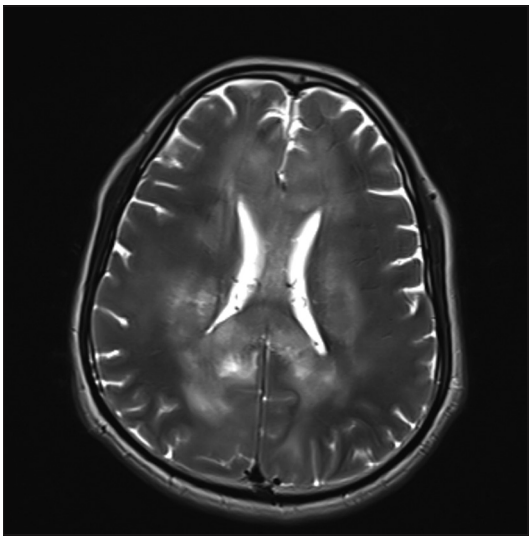

Figure 2: Axial T2-weighted magnetic resonance imaging brain demonstrating bilateral symmetrical hyperintense lesions involving the body and splenium of the corpus callosum and adjacent periventricular white matter. No blooming or hemorrhage noted. Findings are consistent with alcohol-related demyelination seen in Marchiafava-Bignami syndrome

Table 1: Alcohol-related neurological disorders

Category	Disorder	Key clinical features
Acute	Wernicke's encephalopathy	Confusion, ophthalmoplegia, and ataxia due to thiamine deficiency
Acute	Alcohol withdrawal delirium	Tremors, agitation, hallucinations, and autonomic instability
Acute	Alcoholic hallucinosis	Predominantly auditory hallucinations with clear sensorium
Subacute	Marchiafava-bignami syndrome	Cognitive decline, interhemispheric disconnection, andcallosal demyelination
Chronic	Korsakoff psychosis	Anterograde amnesia, confabulation, and mammillary body atrophy
Chronic	Alcoholic cerebellar degeneration	Truncal ataxia, dysarthria, and cerebellar vermis atrophy
Chronic	Peripheral neuropathy	Stocking-glove sensory loss, areflexia, and distal weakness
Chronic	Alcoholic myopathy	Proximal muscle weakness due to ethanol toxicity

considered but excluded based on negative oligoclonal bands and normal VEP findings [6].

Clinically, MBS differs from other alcohol-related encephalopathies by exhibiting interhemispheric disconnection and severe cognitive dysfunction. Radiologically, it typically shows symmetrical T2/FLAIR hyperintensities in the corpus callosum (especially body and splenium), while Wernicke's encephalopathy involves the periaqueductal gray matter, mammillary bodies, and thalami, subacute combined degeneration of the spinal cord, multiple sclerosis, and ADEM. Differentiation is based on lesion distribution and clinical context, as MBS predominantly affects the corpus callosum [7,8].

Management primarily involves prompt thiamine replacement (200–500 mg IV TDS), nutritional rehabilitation, and abstinence from alcohol. Early intervention may reverse callosal demyelination, while delayed treatment often results in irreversible necrosis and callosal atrophy. The partial recovery observed in this patient reflects the benefit of early thiamine therapy, though the presence of extensive callosal and extracallosal lesions likely contributed to residual cognitive impairment [9].

Prognosis depends on the stage and extent of lesions. The acute form may be fatal, whereas subacute and chronic forms have variable outcomes. MRI follow-up often shows regression of lesions with treatment, although callosal atrophy may persist. Similar overlap cases have been described by Heinrich *et al*. [1], Hillbom *et al*. [3], and Singh [7], all underscoring the benefit of early thiamine supplementation. In our case, although partial cognitive recovery was noted, the persistence of deficits likely reflected extensive callosal and extracallosal involvement.

CONCLUSION

MBS should be suspected in chronic alcoholics presenting with neurocognitive dysfunction or altered mental status. MRI plays a key diagnostic role by revealing characteristic callosal and extracallosal lesions. Early thiamine therapy remains the cornerstone of management. This case emphasizes the importance of early recognition and intervention in preventing permanent neurological sequelae.

ACKNOWLEDGMENT

The authors thank the Department of Neurology and Department of Radiology, Safdarjung Hospital in their assistance in clinical assessment and MRI interpretation. We also thank the hospital staff for their valuable assistance in the diagnosis and management of this case.

AUTHOR'S CONTRIBUTIONS

Namit Gupta: Concept and design, data acquisition, and drafting the manuscript; Pankaj Sampatlal Gurjar: data acquisition; Ayushi Daga: Data acquisition; and Shilpi Rani: Critical review.

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Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Gupta N, Gurjar PS, Daga A, Rani S. Marchiafava-Bignami syndrome with Wernicke-Korsakoff overlap: A rare case of post-binge encephalopathy in a chronic alcoholic. Indian J Case Reports. 2025;11(12):656-658.