Case Report

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Marchiafava-Bignami Disease: A Rare Case Report and a Review of Literature

Internal Medicine Section

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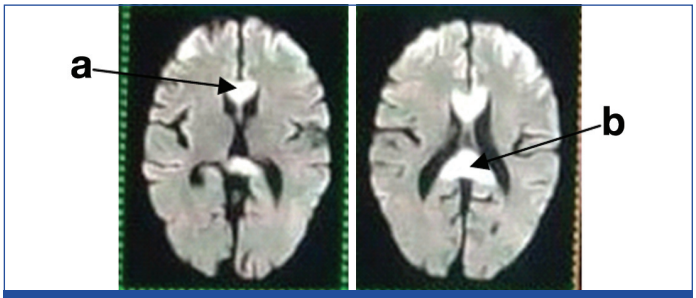
ABSTRACT

Marchiafava-Bignami Disease (MBD) is an unusual neurological illness caused by prolonged alcoholism that is distinguished by demyelination of the corpus callosum. Clinicians encounter a wide range of alcohol-related diseases in their practice, and MBD is a rare condition that requires a high clinical suspicion due to its potential to mimic withdrawal symptoms, Wernicke's-Korsakoff's Psychosis, and various other neurological conditions, leading to confusion in diagnosis. It is a toxic-demyelinating syndrome that typically affects chronic alcoholics, but it has been described in rare instances in chronically malnourished teetotalers. The symptoms and warning signs are diffuse, and the onset could be abrupt or gradual. This is a case of a 54-year-old chronic alcoholic who presented with neurological symptoms. Magnetic Resonance Imaging (MRI) of the brain revealed typical features of MBD. The patient was treated with high doses of thiamine along with folic acid and other symptomatic treatments, after which significant improvement was observed. This case report reviews past reported cases to shed light on the varied presentation features of the disease. It adds to the limited corpus of MBD literature and highlights the intricate diagnostic process and management complexities associated with this condition. By integrating clinical observations, neuroimaging findings, and treatment outcomes, this report underscores the importance of maintaining a high clinical suspicion in cases involving alcohol abuse and the significance of tailored therapeutic strategies. As MBD continues to present diagnostic challenges, this report contributes valuable insights into its presentation, progression, and potential pathways to recovery. Therefore, it requires a high clinical suspicion for both clinical and radiological diagnosis.

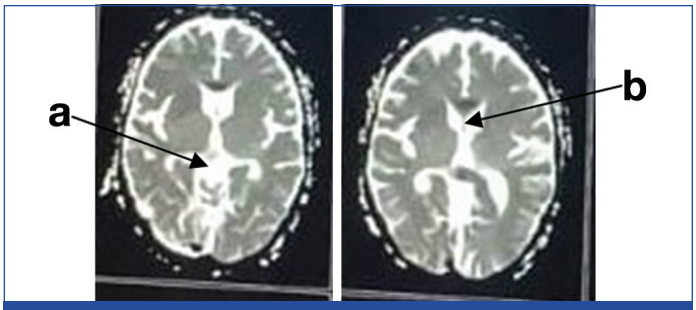
Keywords: Alcohol, Corpus callosum, Magnetic resonance imaging, Thiamine

CASE REPORT

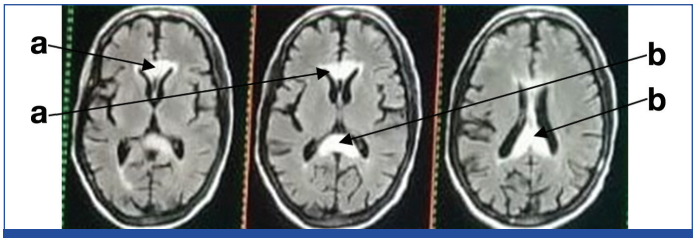
A 54-year-old male with a known case of hypertension and diabetes for two years (taking tablet amlodipine 5 mg and tablet metformin 500 mg twice daily) and a chronic alcoholic (500 mL of country liquor per day for 25 years) arrived with right hemiparesis, aphasia for five days, and alteration of sensorium for three days, with a history of two episodes of Generalised Tonic-Clonic Seizures (GTCS) two days earlier. He was drowsy and arousable on clinical examination, with a Glasgow Coma Scale score of E2V1M3, pupils bilateral 2 mm reacting to light equally, no cranial nerve involvement, power in the right upper and lower limbs was 1/5, and the right plantar had an extensor reaction. Lab investigations revealed a haemoglobin of 15.4 g%, mean corpuscular volume of 98 fL, aspartate transaminase of 98 U/L, alanine transaminase of 40 U/L, prothrombin time with an international normalised ratio of 1.5, lactate dehydrogenase of 150 U/L, normal kidney function tests, cerebrospinal fluid examination revealed a protein of 78 mg/dL, sugar of 98 mg/dL, 10 cells with 98% lymphocytes, and a negative Gram stain. Blood culture was sterile, random blood sugar was 180 mg/dL, and urine culture and sensitivity revealed no growth. Magnetic Resonance Imaging (MRI) revealed hyperintensity on Diffusion-Weighted Imaging (DWI) [Table/Fig-1], T1-weighted imaging, a drop in Apparent Diffusion Coefficient (ADC) values [Table/Fig-2], hyperintensity on Fluid-Attenuated Inversion Recovery (FLAIR) sequence [Table/Fig-3], sagittal FLAIR [Table/Fig-4] and on T2-weighted image [Table/Fig-5] in the splenius and body of corpus callosum. These radiological features were suggestive of MBD. The patient was given intravenous thiamine 500 mg eight hourly, followed by 100 mg eight hourly, intravenous levetiracetam 1 gram stat followed by 500 mg 12 hourly, and tablet aspirin 150 with atorvastatin 10 mg once a day, intravenous fluids, ceftriaxone 1 gram twice daily, and sugar monitoring. He was followed-up after six weeks with complete resolution of his symptoms; hence, MRI was not repeated.



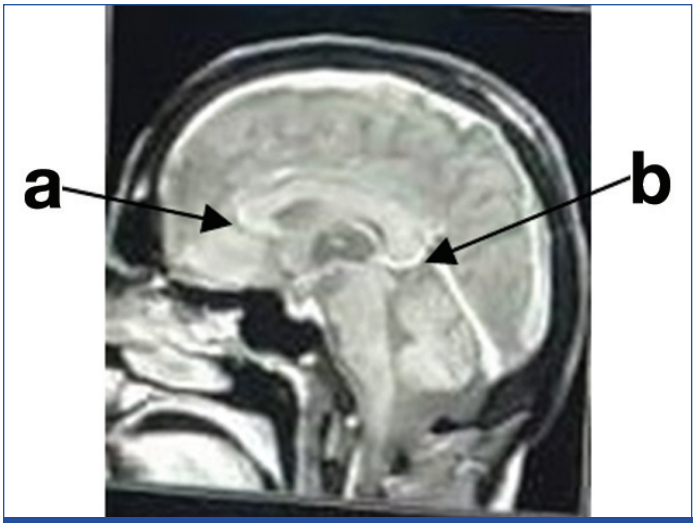
[Table/Fig-1]: Diffusion Weighted Image (DWI) showing hyperintensity of (a) Genu of corpus callosum (b) Splenium of corpus callosum.



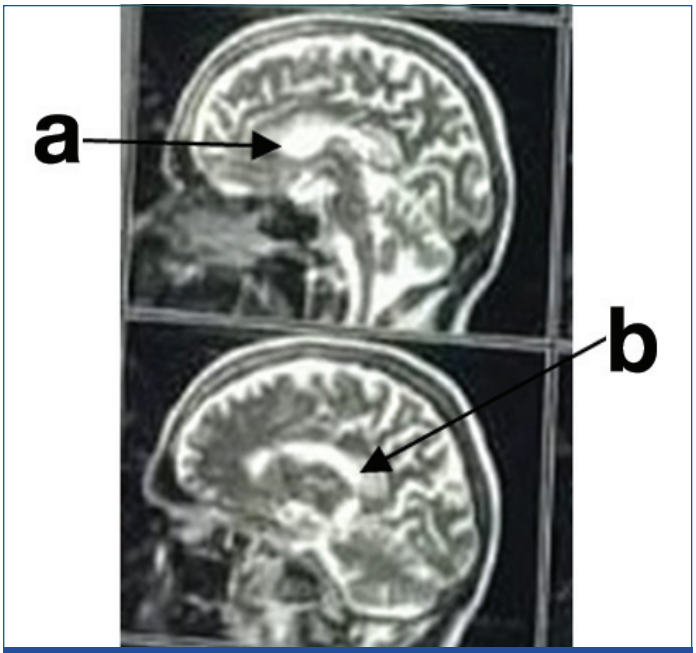
[Table/Fig-2]: Apparent Diffusion Coefficient (ADC) showing hyperintensity of (a) Splenium of corpus callosum (b) Genu of corpus callosum.



[Table/Fig-3]: Fluid-Attenuated Inversion Recovery (FLAIR) axial showing hyperintensity of (a,b) Genu and splenium of corpus callosum.



[Table/Fig-4]: Sagittal FLAIR image showing hyperintensity of (a) Genu of corpus callosum (b) Splenium of corpus callosum.



[Table/Fig-5]: T2 Weighted image showing hyperintensity of (a) Genu of corpus callosum (b) Splenium of corpus callosum.

DISCUSSION

Two Italian pathologists, Ettore Machiafava and Amico Bignami, first identified MBD. Even though clinical signs may be exceedingly diverse and ambiguous, MBD should be evaluated in patients with chronic alcoholism or malnourishment who present with specific neurological symptoms [1]. Recognising neuroradiologic features is crucial for making an early diagnosis. Among the remedies include thiamine, folate, vitamin B12, and steroid therapy [2]. In rare situations, other CNS structures such as the optic chiasma, optic tracts, putamen, cerebellar peduncles, anterior commissure, cortical grey matter, and U fibers may be damaged.

Additionally, a review article by Li J and Li F suggested that Hashimoto's Encephalopathy is better termed autoimmune encephalopathy associated with thyroid antibodies since antithyroid antibodies are essential laboratory features for the diagnosis of Hashimoto's Encephalopathy [3]. Seizures were observed in approximately 60-70% of patients, with many presenting as the initial indication of the condition. During the acute phase, the corpus callosum shows hyperintensity on T2-weighted imaging but hypo-intensity on T1-weighted imaging and proton density-weighted MR images. Cystic lesions and small foci of T2 hypointensity may be identified during the subacute phase, primarily due to haemosiderin deposition. Changes in signal intensity are less apparent in the chronic stage, although persistent atrophy of the affected structure may still be visible [4]. Presentations of MBD in similar published cases are provided below in [Table/Fig-6] [2,4-20].

The most recent classification categorises MBD into two subgroups. Type A has a poor prognosis and symptoms related to the pyramidal tract, limb hypertonia, seizures, and hyperintense enhancement of the corpus callosum on T2 sequence MRI. Type B is characterised by normal or slight disorientation, dysarthria, gait disturbance, and hyperintense lesions on T2-weighted MR sequences of the corpus callosum. The prognosis for type B is favourable, with lesions potentially reversible, suggesting underlying oedema rather than demyelination [21,22]. FLAIR scans reveal a central hypointense signal surrounded by a peripheral hyperintense ring, forming the "sandwich sign," which is one of the diagnostic criteria for MBD. The central hypointense signal indicates necrosis, while the central hyperintense signal points to gliosis. Uniform hyperintense lesions suggest a combination of demyelination and oedema [23].

Case report	Age (years)/ Gender	Duration of alcohol intake	Presentation	Imaging	Prognosis/Follow-up
Gupta M et al., [2]	45/M	More than 100 gm country liquor for last three years	Known case of Diabetes Mellitus Type II, with no history of alcoholism or malnourished with progressive weakness and altered consciousness, had a history of lower limb weakness for two months, which progressed rapidly making him wheelchair-bound, had signs of respiratory distress on presentation to Emergency Medicine Department (EMD), which led to intubation and transfer to the Intensive Care Unit (ICU)	Magnetic Resonance Imaging (MRI) {T2 and Fluid-Attenuated Inversion Recovery (FLAIR)} revealed hyper-intensities involving cerebral and middle cerebellar peduncles, the parietal region of the corpus callosum, and the part of basal ganglia. DWI showed a symmetrical hypersignal throughout the lesion, with a commensurate drop in ADC values	Clinically, there was no improvement. The patient passed away on the 125th day of his hospitalisation.
Seneviratne K et al., [4]	53/M	Chronic 35 units per week	Confused and had impaired memory	MRI of his brain showed dilatation of the ventricles with cerebral atrophy. Confluent white matter signal abnormalities were present in the periventricular regions	Intravenous thiamine B1 and chlordiazepoxide, discharged with improved gait and balance.
Wais AA et al., (MBD) [5]	45/M	10 years	Impaired unconscious and weakness in upper and lower limbs	MRI revealed hyperintensity signals on T2 images, followed by isointense to hypointensity signals on T1 images of the whole corpus callosum on D1W	Improved with Thiamine and folic acid; and was discharged; but was lost to follow-up.
Wang J et al., [6]	62/M	20 years	Headache and mild cognitive impairment	MRI revealed symmetrical hypodensity on the T1 image and hyperintensity on the T2 image in the splenium and corpus callosum	Was able to speak with others fluently but had mild memory loss following a week of treatment.
Dabla S et al., [7]	45/M	10 years	Multiple non specific neuropsychiatric manifestations	MRI demonstrated altered signal intensity in the parietal subcortical white matter	A relative improvement in his behaviour with Thiamine was seen on discharge but was lost to follow-up.

De Ryck H et al., [8]	66/M	Chronic	Subacute disorientation, which had been preceded by modest gait issues for a week. The clinical examination revealed an upset patient with bilateral abnormal plantar reflexes. During the observation, his condition deteriorated, and he fell into a coma a day later. The patient had many generalised epileptic seizures on the second day of hospitalisation, for which antiepileptic medication was started. Intubation and artificial ventilation were required due to the sedative impact of the antiepileptic medication	MRI Brain revealed symmetric hyperintensity of white matter on T2 images, most notably in the corpus callosum	MRI of the brain after one week revealed significant improvement in the white matter lesions. Two weeks later, he presented with icterus and was diagnosed with Epstein-Barr Virus (EBV) hepatitis; lymph node biopsies revealed EBV-positive Diffuse Large Cell B-Cell Lymphoma (DLCBL).
Yang L et al., [9]	49/M	10 years	Altered mental state and loss of muscle strength on the right side for four days. Was diagnosed with hypertension a month ago and had a history of dizziness 15 days ago	FLAIR pictures demonstrated several hyperintense lesions in the frontoparietal-occipital subcortical areas, right basal ganglia, and the genu, body, and splenium of the corpus callosum. DWI images revealed hyperintensity in the corpus callosum's genu and body. T2WI pictures in the sagittal plane showed high-intensity signals across the corpus callosum	Gradually regained consciousness and the paresthesia improved. MRI Brain done after 38 days, revealed linear necrosis in the central layer of the body apart from the dorsal and ventral layers whereas the other lesions in the brain had nearly completely disappeared.
Khiew MA [10]	21/M	Two years	Agitation, severe attention deficit, instability, postural tremor, a broad-based gait, and disorientation in time, location, and person. He described hallucinations, early sleeplessness, and lower-limb paresthesias	MRI revealed bilateral ovaloid hyperintense spots on T2-weighted images and hypointense areas on T1-weighted images in the corpus callosum splenium. There was also noticeable atrophy in the callosal commissure's posterior body	Was clinically healthy and abstinent at the six-month follow-up.
Takahashi N et al., [11]	68/F	Nil	Inability to eat had articulation issues	MRI brain showed symmetrical high-intensity areas on T2 and DWI in the genu, stem, and splenium of the corpus callosum	Significant neurological improvement after 15 days of treatment.
Bhavani KG et al., [12]	43/M	20 years	Seizures and vomiting	MRI revealed hypointensity in the corpus callosum's body and splenium	Improved on treatment.
Hui PS et al., [13]	41/M	25 years	Psychosis and minimal cognitive impairment	MRI brain indicated shrinkage of the corpus callosum	With medication, his psychotic symptoms gradually improved.
Bachar M et al., [14]	44/F	Nil	Anorexia, growing weakness, and trouble walking for two weeks, was obese and had a history of bipolar disorder; had Roux-en-Y gastric bypass surgery 23 years ago	MRI revealed abnormalities in the splenium of the corpus callosum and the posterior limb of both internal capsules. Hypoglycaemic encephalopathy, extrapontine myelinosis, and stroke were ruled out. Her MRI was suggestive of type B MBD (focal Callosal involvement)	Encephalopathy was completely resolved after 10 days of treatment. A repeat MRI brain done 15 days following the original scans revealed that her splenium and internal capsule abnormalities had been resolved.
Matsuura H and Shindo K [15]	58/M	Chronic	Acute onset aphasia, dysphasia, dysarthria, and disorientation, was asthmatic, but he had no previous cardiovascular or gastrointestinal problems. Neurological examination revealed left unilateral spatial neglect, left/right confusion, verbal paraphasia, and apraxia	FLAIR and T2-weighted MRI revealed substantial brain atrophy and hyperintense signal changes in the corpus callosum splenium	Lost to follow-up.
Vanhove F [16]	46/F	Nil	Altered sensorium, mutism, dysarthria, left upper limb weakness and left seventh nerve paralysis	MRI brain revealed severe cerebral atrophy and a hyperintense signal on FLAIR images of the entire corpus callosum, with diffusion-restricted lesions on diffusion-weighted imaging. These lesions were not seen on T1-weighted images and had no contrast enhancement	A six-day follow-up MRI scan revealed decreased FLAIR hyperintensity and diffusion limitation. After 26 days, a follow-up MRI revealed hypointense lesions throughout the whole corpus callosum on T1-weighted sequences.
Simões I et al., [17]	52/M	Chronic	Agitation, severe impairment of attention, imbalance, postural tremor, a wide-based gait, and disorientation in time, space, and person. He reported zoopsias, initial insomnia, and paresthesias of the lower limbs	The MRI revealed bilateral ovaloid hyperintense areas on T2-weighted images and hypointense areas on T1-weighted images in the splenium of the corpus callosum. There was also a discrete atrophy in the posterior body of the callosal commissure	A 7-day course of high-dose parenteral thiamine followed by oral thiamine 300 mg/d and was discharged. At six months follow-up visit, the patient was clinically well and abstinent.
Cui Y et al., [18]	62/M	Nil	Dizziness and gait instability that persisted for >10 days	The MRI of this patient revealed a solitary lesion in the corpus callosum	A diet rich in vitamins to improve the brain's blood and oxygen. Two weeks after admission, delusions of the patient had improved markedly. At the follow-up after two months, he could walk freely.
Sunil Kumar K et al., [19]	55/M	25 years, 750 mL country liquor daily	Altered sensorium followed by loss of consciousness for one day	MRI showed low signal intensity in T1WI and high signal intensity in T2WI in the central portion of the genu, body, and splenium of the corpus callosum with relative sparing of dorsal and ventral layer characterising Sandwich sign. The corresponding region in the FLAIR image showed a hyperintense rim and hypointense core suggestive of a gliotic rim with central necrosis	Multivitamins, corticosteroids, stabilisation of plasma glucose, and supportive care.

Hashino Y et al., [20]	51/M	Chronic	Dysarthria and dysphagia on admission was alert with mild cognitive dysfunction. The facial expression was flat, and there was weakness of the orbicularis oris bilaterally. Speech was slurred, had difficulty swallowing, and the gag reflex and palate elevation were poor. The jaw jerk reflex was brisk and the snout reflex was positive	MRI revealed hyperintense lesions in the splenium of the corpus callosum and the primary motor cortices bilaterally	After vitamin B therapy for 17 days, the neurological symptoms were alleviated concurrently with the disappearance of the lesions on MRI.

[Table/Fig-6]: Varied presentations of Marchiafava Bignami Disease (MBD) in previously reported cases [2,4-20].

Lesions exhibit hyperintensity on DWI, accompanied by varying ADC values. In the early stages, cytotoxic oedema may underlie the condition, leading to hyperintense lesions with a low ADC on DWI. Increased ADC values in later stages could be attributed to demyelination without axonal damage [24]. Additional imaging modalities include magnetic resonance spectroscopy, which demonstrates an increase in choline and a rise in the Choline/Creatine (Cho/Cr) ratio during the acute phase.

Lactate peaks are commonly observed during the acute or subacute stages of demyelination. Single-Photon Emission Computed Tomography (SPECT) scans may reveal bilateral decreased cerebral blood flow [25].

Some conditions that mimic MBD include Wernicke encephalopathy, Wernicke-Korsakoff's Psychosis, epileptic seizures, acute meningoencephalitis, acute disseminated encephalomyelitis, delirium tremens/alcohol withdrawal syndrome, stroke (recurrent Heubner artery infarction), multiple sclerosis, malignancies (astrocytoma, lymphoma), and other demyelinating diseases (progressive multifocal leukoencephalopathy) [26-28].

Most MBD case reports have shown a favourable response to intravenous injections of Vitamin B1, folic acid, and high-dose corticosteroids. Some case reports have indicated significant improvement with high-dose intravenous thiamine (500 mg every 8 hours), oral vitamin B complex, amantadine, and folate [2,3,7-10]. Management also involves intensive nutritional supplementation, with a focus on alcohol withdrawal. Additionally, management includes stabilising blood glucose levels and providing supportive care. Early detection and treatment can help reverse the disease [25,26].

CONCLUSION(S)

In the case discussed here, a patient with persistent alcohol intake presented with seizures and altered sensorium, and neuroimaging enabled early detection of MBD, facilitating the early initiation of treatment. Recognising MBD imaging features is crucial for radiological diagnosis. When identified early and treated with high-dose Thiamine and steroids, patients have a better prognosis.

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