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Case Report

# Marchiafava–Bignami disease in chronic alcoholic patient

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ABSTRACT

Marchiafava–Bignami disease is a rare toxic encephalopathy seen mostly in chronic alcoholics due to progressive demyelination and necrosis of the corpus callosum. It may involve adjacent white matter and subcortical regions. We present here the magnetic resonance imaging findings of Machiafava–Bignami disease in a chronic alcoholic patient. In 1903, Italian pathologists Marchiafava and Bignami described 3 alcoholic men who died after having seizures and coma. All 3 patients were chronic alcoholics and had consumed considerable amounts of red wine.



## History

A 45-year-old chronic alcoholic patient who was taking alcohol for 20 years and presented with altered sensorium and seizures.

Patient's family reported that he had consumed a daily average of 250 mL of liquor for 2 weeks. The patient was in poor physical condition and seemed malnourished. The patient is diagnosed case of liver cirrhosis since 5 years. On examination, the patient was confused. Although there was no weakness in his extremities, he demonstrated lack of motor coordination. Electroencephalogram showed diffuse slow waves of 5-8 Hz without epileptiform discharge.

Laboratory test results revealed hyperosmosis of 370 mmol/L (normal, 290 mmol/L), high blood creatinine (2.1 mg/dL; normal, 0.5-1.2 mg/dL), and high blood urea nitrogen (40.0 mg/dL; normal, 8-23 mg/dL). Sodium and potassium levels were, respectively, 146 mmol/L and 4.3 mmol/L. Cerebrospinal fluid studies revealed no abnormalities. There are low serum levels of vitamin B12, folic acid, and albumin seen.

## Imaging findings

Cranial computed tomography showed diffuse corpus callosal and bilateral periventricular hypodensity.

### *Protocol*

Magnetic resonance imaging (MRI) was performed on a 1.5 T magnet (GE Signa, 1.5 T, General Electric). Fast spin-echo T1-weighted (repetition time [TR]: 650 ms, echo time [TE]: 14 ms), T2-weighted (dual-echo TR, 2014 ms; TE, 30 and 100 ms), and fluid attenuation and inversion recovery (FLAIR; Turbo Spin Echo; turbo factor 11; TR, 5496 ms; TE, 100 ms; inversion time, 2000 ms) images in axial and sagittal planes were acquired. Diffusion-weighted imaging (DWI) scans were acquired with diffusion gradients along each of the 3 principal axes with 3 different values (0, 187, and 757 s/mm$^2$). Postcontrast T1-weighted (TR, 650 ms; TE, 14 ms) images were acquired after intravenous administration of 0.2 mL/kg body weight of

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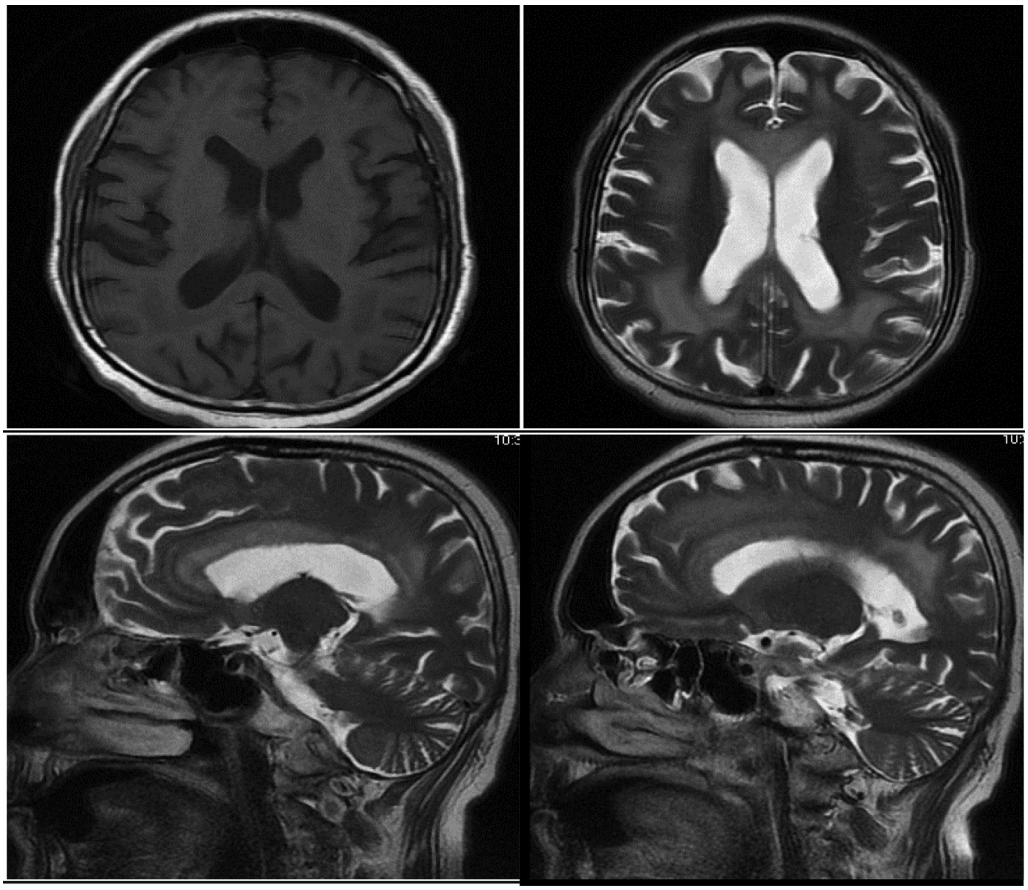

**Fig. 1 – In a 45-year-old chronic alcoholic patient presented with seizures, altered sensorium, and bilateral lower limb paresis, T1W (upper row right) image shows hypointense corpus callosum and periventricular white matter with corresponding T2 (upper row left and middle row) and FLAIR (lower row right images shows hyperintense signal with true restricted diffusion in DWI image (lower row left). There is no postcontrast enhancement seen (last image).**

gadodiamide (Omniscan) at a rate of 4 mL/s with a delay of 10 minutes in the axial and sagittal planes.

Figure 1 shows on T2-weighted and FLAIR images show diffuse hyperintensity in corpus callosum and bilateral periventricular white matter with corresponding T1-hypointense signal. On DWI images restricted diffusion seen in bilateral peritrigonal white matter and middle layer of splenium of corpus callosum with corresponding drop in signal on apparent diffusion coefficient seen. There is no evidence of increased abnormal signal in the subcortical white matter seen. There is no evidence of postcontrast enhancement.

Marchiafava–Bignami disease (MBD) was diagnosed based on the clinical and imaging features.

## Discussion

MBD is a rare complication of chronic alcoholism characterized by demyelinization and necrosis of the entire length and middle layer of corpus callosum with extension into hemispheric white matter [1,2]. Occasionally, other structures of the central nervous system may be involved, such as the optic chiasm and tracts, putamen, cerebellar peduncle, and anterior commissure. Cortical gray matter and subcortical *U* fibers are involved rarely [3,4].

Most accepted etiologic factor is the in multiple vitamin B deficiency [5].

*Clinical manifestations [6]:*

(1) Acute state: seizures, alterations of consciousness, and death may occur.
(2) Subacute state: characterized by mental confusion, behavioral disorders, memory deficits, cerebellar signs, and interhemispheric disconnection.
(3) Chronic state: mild dementia.

*Clinicoradiological subtypes of MBD [6,7]:*

(1) Type A: characterized by alterations of consciousness and diffuse swelling of the entire corpus callosum on imaging.
(2) Type B: mild impairment of consciousness and small callosal lesions associated with good prognosis.

On MRI images [8], T1W shows confluent hypointense signal intensity of corpus callosum.

T2 and FLAIR shows hyperintense signal in middle layer of corpus callosum (Sandwich sign).