Int. j. adv. multidisc. res. stud. 2023; 3(2):1047-1048

Received: 04-04-2023
Accepted: 14-04-2023

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Letter to the Editor

Marchiafava Bignami disease and its differentials

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We read with interest the article by Vikhe *et al*. on a 40 years-old male who was diagnosed with Marchiafava-Bignami disease (MBD) based on the clinical presentation (memory loss for 1 month, altered sensorium for 10 days, irrelevant talk for 10 days, dysarthria, spasticity) and cerebral MRI (multifocal T2 and fluid attenuated inversion recovers (FLAIR) hyperintense lesions in both frontal lobes and a T2 hyperintense lesion in the left body of corpus callosum) [1]. His individual history was positive for chronic alcoholism, diabetes, and polyneuropathy [1]. Treatment included thiamine, vitamin-B12, folic acid, insulin, antibiotics, and pregabalin. The outcome was not reported [1]. The study is excellent but has limitations and raises concerns that should be discussed.

We disagree with the diagnosis MBD. The hallmark of acute MBD is the "sandwich sign" [2], characterised by a symmetrical lesion encompassing the body of the corpus callosum's central region but leaving out the ventral and dorsal layer [2]. The "sandwich sign" is usually best visible on sagittal T1-weighted MRI images [2]. Except for the small T2 hyperintensity in the left anterior portion of the body of corpus callosum, no other callosal lesion was described in the index patient.

Another limitation is that various differential diagnoses were not sufficiently ruled out. Differential diagnoses that could explain the clinical presentation and the imaging findings include Wernicke encephalopathy, diabetic encephalopathy (hyperglycemic and hypoglycemic crises), hepatic encephalopathy, septic encephalopathy, paraneoplastic encephalopathy, autoimmune encephalitis (AIE), non-convulsive epileptic status, demyelinating disease, such as multiple sclerosis, myelin oligodendrocyte glycoprotein (MOG)-associated disease (MOGAD), acute disseminated encephalomyelitis (ADEM), infarction of the recurrent artery of Heubner, and neoplasia, such as lymphoma or astrocytoma [2]. Missing in this respect is the application of contrast medium, application of perfusion-weighted MRI images, magnetic resonance angiography (MRA), recording of an electroencephalogram (EEG), measurement of NH3, determination of antibodies associated with AIE, determination of MOG and aquaporin-4 (AQ4) antibodies, documentation of oligoclonal bands (OCBs)), and measurement of pro-calcitonin.

Another limitation of the study is that the patient's serum thiamine and vitamin-B12 levels were not measured. The patient was treated with thiamine (100mg/d) and methyl cobalamin (500microg/d), and folic acid (5mg/d) without any proof that these vitamins were deficient. Before substituting vitamins, their reduction should be demonstrated.

There is a discrepancy between the absence of fever and the diagnosis pneumonia [1]. We should be told why a patient with pneumonia and a white blood cell count of 17500 has not fever. Absence of fever is not comprehensible, particularly in the light of the history in the days before admission (altered sensorium, irrelevant talk).

Overall, the interesting study has limitations that call the results and their interpretation into question. Clarifying these weaknesses would strengthen the conclusions and could improve the study. MBD should be diagnosed only after extensive work-up and exclusion of all possible differential diagnoses.

Declarations
Acknowledgements: None.

Disclosures: The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author contribution: JF: design, literature search, discussion, first draft, critical comments, final approval.

Compliance with Ethics Guidelines: This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors.

Keywords: White Matter Lesion, Chronic Alcoholism, Thiamine, Marchiavafa-Bignami, Electroencephalography

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