



Received: 23-09-2026
Accepted: 03-10-2026

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Letter to the Editor

Painful and Restricted Eye Movements, Headaches, Loss of Consciousness and a History of Thrombosis are More Indicative of Cavernous Sinus Thrombosis than Miller Fisher Syndrome

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Letter to the Editor

We read with interest the article by Beshr *et al.* about a 36-year-old woman with arterial hypertension, diabetes mellitus, and recurrent venous thrombosis. The patient suddenly developed severe headaches, was unconscious for 15 minutes, and presented with ptosis, ophthalmoparesis, and tetraparesis ^[1]. She was diagnosed with Miller Fisher syndrome (MFS) and treated with intravenous immunoglobulins (IVIG) ^[1]. At the two-month follow-up, the patient had almost completely recovered ^[1]. The study is promising, but several points warrant discussion.

First, we disagree with the diagnosis of MFS ^[1]. Since the patient developed tetraparesis, Guillain-Barré syndrome (GBS) with extraocular muscle involvement should be diagnosed, rather than MFS. Furthermore, urinary incontinence and dysphagia are not classic features of MFS. In this context, GQ1b antibodies are absent ^[1]. Since they are elevated in up to 100% of MFS patients ^[2], it would have been helpful to confirm the diagnosis by detecting elevated GQ1b antibodies.

Second, we disagree with the assumption that the loss of consciousness was due to MFS. MFS is not usually associated with syncope. However, there are a few cases of MFS with the addition of Bickerstaff encephalitis, in which loss of consciousness was part of the clinical presentation ^[3]. Was the cerebral MRI performed with contrast to rule out Bickerstaff encephalitis?

Third, the lack of cerebrospinal fluid (CSF) analysis - although acknowledged as a limitation - represents a significant shortcoming of the study. To rule out Bickerstaff encephalitis, viral, bacterial, or parasitic meningitis, and subarachnoid hemorrhage, it would have been essential to know the presence of pleocytosis, cytoalbumin dissociation, antiviral antibodies or a positive viral PCR test (including SARS-CoV-2), elevated cytokines, chemokines, or glial factors, xanthochromia, or elevated ganglioside antibodies.

Fourth, cavernous sinus thrombosis was not adequately ruled out ^[1]. Was magnetic resonance venography (MRV) performed with contrast medium? It is not mentioned whether the D-dimer level was elevated. Since cavernous sinus thrombosis can manifest with altered mental status, particularly painful eye movements, and headaches ^[4], and since the patient had a history of recurrent venous thrombosis, it would have been essential to rule out cavernous sinus thrombosis. Furthermore, it should have been determined whether the patient suffered from a hereditary or acquired coagulation disorder.

The fifth point is that an epileptic seizure was not sufficiently ruled out ^[1]. Were there signs of tongue or lip biting or involuntary urinary incontinence? Even if there was no postictal confusion or tonic-clonic components, it cannot be excluded that the 15-minute loss of consciousness was due to an atonic seizure or non-convulsive status epilepticus. Was creatine kinase measured and was it elevated postictally? In this context, the results of the EEG recordings are missing.

The sixth point is that the nerve conduction studies could also be due to diabetic polyneuropathy and do not necessarily indicate GBS. The patient had poorly controlled diabetes for years (HbA1c 9).

Finally, magnetic resonance angiography is lacking to rule out reversible cerebral vasoconstriction syndrome (RCVS), vasculitis, or cerebral artery dissection. Since RCVS is associated with severe throbbing headaches ^[5], it is crucial to rule out this possible cause of the index patient's headaches.

Acknowledgements

Funding: No funding was received.

Data Access Statement: All data are available from the corresponding author.

Ethics Statement: Not applicable.

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

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.

Data Access Statement: Not applicable.

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: Miller-Fisher Syndrome, Cavernous Sinus Thrombosis, Tetraparesis, Headache, Ophthalmoparesis

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