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Letter to the Editor

GBS Associated with Encephalitis and Myelitis Necessitates the Identification and Treatment of the Triggering Pathogen

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Letter to the Editor

We read with interest the article by Numan *et al*. regarding a previously healthy 31-year-old man who developed Guillain-Barré syndrome (GBS), subtype acute inflammatory demyelinating polyneuropathy (AIDP), accompanied by encephalitis and myelitis, which necessitated long-term mechanical ventilation [1]. The patient's response to treatment with intravenous immunoglobulins (IVIG), plasmapheresis, glucocorticoids, and rituximab was inadequate, and the clinical course had an unfavourable outcome [1]. While the study is noteworthy, several aspects warrant further discussion.

First, the investigation into the triggering factors for the GBS and the concurrent encephalitis and myelitis was too limited [1]. More comprehensive testing might have identified the causative pathogen, which would have facilitated not only the management of the neurological complications but also the treatment of the underlying infection itself. Specifically, the diagnostic workup lacked screening for EBV, SARS-CoV-2, hepatitis A and E, chikungunya virus, West Nile virus, influenza viruses, dengue virus, Japanese encephalitis virus, *Klebsiella pneumoniae*, *Treponema pallidum*, *Borrelia burgdorferi*, *Campylobacter jejuni*, and *Haemophilus influenzae* [2].

Second, the cerebral vasculature was not evaluated [1]. To rule out vasculitis, it would have been necessary to perform magnetic resonance angiography (MRA) with "black-blood" sequences, computed tomography angiography (CTA), perfusion imaging, or digital subtraction angiography (DSA) to determine whether the CNS lesions were associated with vasculitis [3]. Furthermore, connective tissue diseases, which can also be associated with vasculitis, were not sufficiently ruled out. Were ANCA antibodies tested, and if so, were the results abnormal?

Thirdly, potential cardiac involvement was not adequately investigated [1]. Since a bradycardia-tachycardia syndrome can stem from various causes, not just autonomic dysfunction, it would have been crucial to exclude alternative triggers for the cardiac arrhythmias. This would have required an echocardiogram, a cardiac MRI (with contrast agent), and long-term ECG monitoring to rule out myocarditis or a cardiotoxic effect of the administered medications on the cardiac conduction system. If a viral trigger was considered, it would have been particularly important to rule out viral myocarditis.

The fourth point is that no MRI examination of the anterior and posterior nerve roots using a contrast agent was performed. This method allows for the assessment of whether there is contrast enhancement of the nerve roots or if they appear thickened [4]. Both are typical features of an inflammatory process, such as that seen in radiculitis.

The fifth point is that the concept of combined central and peripheral demyelination (CCPD) cannot be substantiated unless the demyelination within the central nervous system (CNS) has been confirmed via autopsy or brain biopsy. Since the CNS imaging in Fig 2 shows only T1 and T2 sequences, it cannot be ruled out that the observed lesions have a cause other than demyelination.

The sixth point is that there is no information regarding the long-term clinical course [1]. Was the patient successfully weaned off mechanical ventilation, and was the tracheostomy removed? Did needle electromyography show any signs of reinnervation? Did the lesions in the brain and spinal cord regress on follow-up MRI scans?

Finally, several aspects were not addressed [1]. Was the patient tested for ganglioside antibodies or antibodies associated with autoimmune encephalitis? Was a paraneoplastic syndrome sufficiently ruled out? Did subsequent nerve conduction studies (NCS) show any signs of recovery?

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References
1. Numan M, Alhatou M, Hussain Z, Hussain M, Alhatou A, Hamad W. One trigger, two systems: Post-infectious severe Guillain-Barre syndrome with concurrent central nervous system demyelination": A case report. Oxf Med Case Reports. 2026; 2026(10):omag202. Doi: 10.1093/omcr/omag202
2. Finsterer J. Triggers of Guillain-Barré Syndrome: *Campylobacter jejuni* Predominates. Int J Mol Sci. 2022; 23(22):14222. Doi: 10.3390/ijms232214222
3. Berlit P. Diagnosis and treatment of cerebral vasculitis. Ther Adv Neurol Disord. 2010; 3(1):29-42. Doi: 10.1177/1756285609347123
4. Alkan O, Yildirim T, Tokmak N, Tan M. Spinal MRI findings of guillain-barré syndrome. J Radiol Case Rep. 2009; 3(3):25-28. Doi: 10.3941/jrcr.v3i3.153