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Letter to the Editor

Acute Motor and Sensory Axonal Neuropathy with Bulbar and Ocular Involvement can Mimic Brainstem Bickerstaff Encephalitis and Miller-Fisher Syndrome

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Letter to the Editor

We read with interest the article by Shen *et al*. about a 56-year-old female patient diagnosed with a triple immunoneuropathy – Guillain-Barré syndrome (GBS), Miller Fisher syndrome (MFS), and brainstem Bickerstaff encephalitis (BBE). Clinically, the disease manifested with dizziness, lightheadedness, diplopia, facial palsy, ophthalmoparesis, dysphagia, tetraparesis, and respiratory muscle weakness [1]. The patient's recovery was incomplete following treatment with intravenous immunoglobulins (IVIG), steroids, ventilation, and physiotherapy [1]. The study is promising, but some points require discussion.

First, two of the three immunological diagnoses are questionable [1]. BBE is unlikely because tendon reflexes were diminished and no brainstem lesion was detected on MRI [1]. Diagnosing BBE based solely on the Odaka criteria is insufficient; Cerebral imaging should be included in the diagnostic workup. BBE is also frequently associated with elevated GQ1b antibodies [2]. MFS is unlikely, as antibodies against GQ1b were normal, but are elevated in 90–100% of MFS cases [3]. Therefore, the patient should be diagnosed with acute motor and sensory axonal neuropathy (AMSAN) involving the pharyngeal, respiratory, and ocular muscles.

The second point is that the trigger for GBS has not been identified [1]. Since the patient had a respiratory infection one week prior to the onset of GBS [1], it is conceivable that a viral or bacterial infection triggered the development of GBS [4]. Was the patient tested for viral or bacterial lung infection using appropriate tests? Was the viral panel negative? Were there any indications of bronchitis or pneumonia upon admission?

The third point is that ganglioside antibodies are a non-specific finding in GBS. Different combinations of ganglioside antibodies can be found in various GBS subtypes, but these patterns are not consistent and therefore not diagnostic. They also vary considerably between patients and GBS subtypes. Only in patients with confirmed MFS are GQ1b antibodies consistently elevated. However, other GBS subtypes can also occasionally be associated with elevated GQ1b antibodies [3].

The fourth point is that the patient's medication during hospitalization was not documented in detail. Since mechanical ventilation usually requires the use of muscle relaxants, anesthetics, analgesics, or sedatives, it cannot be ruled out that at least some of the clinical manifestations are due to the medications used rather than to AMSAN.

The fifth point concerns the unclear reason for administering glucocorticoids to the patient [1]. Steroids are known to be ineffective in GBS and often cause more side effects than benefits. Regarding the suspected ineffectiveness of IVIG: its effect can often be delayed, as in the present patient. If IVIG remains ineffective even after a second cycle or switching to pentaglobin, plasmapheresis or immunoadsorption should be attempted [5].

The sixth point concerns the discrepancy between the statement that the patient was "fully conscious" upon admission and the statement that "the diagnosis of GBS was based on an altered level of consciousness." Did the altered level of consciousness develop during the hospital stay and was it due to the required sedatives?

The seventh point concerns electromyography (EMG), which should not be confused with neurological examinations (NCV) [1]. According to the description, the patient underwent an NCV but no EMG [1].

Finally, it should be explained why it took 14 days for the cerebrospinal fluid analysis to be performed. Since GBS was suspected from the time of admission, and GBS can be a medical emergency, confirming the diagnosis as early as possible is crucial.

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