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Letter to the Editor

The Diagnosis of AMASN with Bickerstaff Encephalitis Requires that the Diagnostic Criteria are Met for Both

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Letter to the Editor

We read with interest the article by Aziz *et al.* about a 14-year-old boy who developed progressive tetraparesis after a febrile infection with abdominal pain and general fatigue over 15 days ^[1]. The patient was diagnosed with Guillain-Barré syndrome (GBS) with symptoms of Bickerstaff encephalitis (BBE). After plasmapheresis, he made an incomplete recovery within three months ^[1]. The study is insightful but requires further discussion.

First, the diagnosis of GBS, subtype acute motor axonal neuropathy (AMSAN), is questionable ^[1]. Nerve conduction velocity measurements of the motor nerves were normal, initial cerebrospinal fluid (CSF) analysis showed no increase in CSF protein, and no contrast-enhanced magnetic resonance imaging (MRI) of the nerve pathways was performed to document swelling or contrast enhancement of the lumbar nerve roots. No follow-up nerve conduction velocity (NCV) measurements were performed to document any deterioration or improvement in sensory NCV or involvement of the motor nerves. Furthermore, there is a discrepancy between normal motor NCV measurements and muscle weakness, as well as between reduced sensory action potentials and the absence of sensory disturbances. Did the GBS also manifest with autonomic dysfunction?

The second point is that the search for the causative agent was not sufficiently thorough ^[1]. Only dengue fever and malaria could be ruled out. Although staphylococci were detected in the blood and CSF cultures, no antibiotic therapy was initiated. Since GBS can be caused by infectious agents, and the patient's medical history contained no evidence of vaccinations, trauma, surgery, sepsis, or immunological disorders, the patient should have been comprehensively tested for all possible GBS pathogens. These include viruses, bacteria, protozoa, helminths, and parasites ^[2]. As *Campylobacter jejuni* is the most common causative agent of GBS worldwide, the patient should have been tested for it by culture, PCR, or antigen detection, especially since he had developed abdominal pain before the onset of the neurological impairment ^[2]. Other possible triggers of GBS that should have been ruled out include *Haemophilus influenzae*, *Mycoplasma pneumoniae*, *Ehrlichia chaffeensis*, *Orientia tsutsugamushi*, *Escherichia coli*, varicella-zoster virus, HIV, Zika virus, Epstein-Barr virus, influenza virus, SARS-CoV-2, hepatitis A, B, C, D, and E viruses, cytomegalovirus, rubella virus, West Nile virus, dengue virus, Japanese encephalitis virus, and chikungunya virus ^[2].

Thirdly, NCV measurements were only performed in a rudimentary manner ^[1]. Neither F-waves nor needle electromyography were recorded. These investigations would have been essential to clarify the cause of the muscle weakness and, if applicable, to detect a radicular lesion. Even with normal creatine kinase levels, a myogenic cause of tetraparesis, especially myositis following a viral infection, must be ruled out. Myositis can occur with normal creatine kinase levels ^[3].

The fourth point is that the diagnosis of BBE-like presentation is not justified ^[1]. The patient's cerebral MRI was unremarkable with contrast ^[1]. In BBE, MRI typically shows T2/FLAIR hyperintensities in the brainstem (e.g., pons, midbrain, cerebral peduncles, cerebellum, thalamus), DWI hyperintensities in the same distribution, or contrast enhancement on T1-weighted imaging ^[4]. BBE is also associated with CSF changes such as elevated CSF protein, lymphocytic pleocytosis, and albumin cytology dissociation ^[5]. Another argument against BBE is that BBE is usually associated with elevated GQ1b antibodies, which were normal in the index patient. More likely than BBE, the index patient suffered from GBS involving cranial nerves IX and X and with positive GD1q antibodies.

Finally, there is a discrepancy between the muscle strength on the neurological examination (5/5 in the upper extremities and 4/5 in the lower extremities) and the patient's history, which also describes upper extremity weakness. Did the upper extremity weakness resolve spontaneously prior to admission? There is also a discrepancy between the patient's history, which reports being bedridden, and the clinical examination, which shows an MRC score of 4 in the lower extremities. Patients with an MRC

score of 4 are usually able to stand and walk with assistance. These discrepancies should be investigated.

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