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RESPONSE TO LETTER

Guillain-Barré Syndrome, Subtype AMAN, Can Only Be Attributed to Lupus Erythematosus if All Infectious Causes Have Been Ruled Out [Response to Letter]

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Dear editor

We appreciate Dr. Finsterer's interest in our paper titled "From Nerve to Autoimmunity: Acute Guillain-Barré Syndrome in a 4-Year-Old with Early-Onset Pediatric Systemic Lupus Erythematosus" and his comments regarding our case study. We will be using this opportunity to clarify critical points related to the infectious workup, diagnostic evaluation, electrophysiological interpretation, and long-term follow-up of our patient.

GBS Infectious Triggers

We agree with Dr. Finsterer, particularly in pediatric patients, infections are considered the most common triggers for the development of Guillain-Barré syndrome (GBS).[1] Nevertheless, current available literature underscores the clinical diagnosis nature of GBS supported by electrophysiological findings and cerebrospinal fluid (CSF) results.[2,3] In addition, emphasizing that extensive infectious screening is not a routine if there is no clinical indication or epidemiological suspicion.[4]

In our patient, no history of recent respiratory or gastrointestinal symptoms or infections, negative travel history as well as full vaccination history were reported.[5] Furthermore, available infectious investigations were performed during hospitalization period including; stool testing, Epstein–Barr virus, Mycoplasma, cytomegalovirus testing, three blood cultures and urine culture, which were found negative. With an unremarkable chest x-ray on admission.

Regarding the febrile episode with dysuria our patient developed on day 38 post-admission, as demonstrated in Figure 2 (original study), the onset was several weeks after the neurological manifestations, diagnosis of GBS, administration of intravenous immunoglobulin therapy, and the start of neurological recovery.[5] Thus, we believe such a clinical event should not be considered as an infectious trigger for the initial GBS presentation. Even though no microbiological evidence was identified supporting the urinary tract infection, both urine analysis and culture prior to the administration of empirical ceftriaxone, reported negative results.

Electrophysiological Findings and AMAN Subtype Interpretation

Although the patient initially demonstrated clinical sensory symptoms involving both upper and lower extremities, those disturbances resolved early after initiation of treatment protocol. However, objective nerve conduction studies presented preserved sensory action potential peak latencies and amplitudes when assessing both upper and lower limbs, preserved motor conduction velocities, normal distal motor latencies, absent F-waves in the lower limbs, and decreased compound muscle action potential amplitudes for both tibial and peroneal nerves. Based on such findings, our consulting neurologist

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interpreted the pattern mostly consistent with acute motor axonal neuropathy (AMAN), primarily affecting the lower limbs.

We totally agree that the presence of sensory disturbances may lead to forming an overlap between AMAN and acute motor-sensory axonal neuropathy (AMSAN) diagnosis. Nonetheless, GBS variants – electrophysiological classification – mainly depend on nerve conduction study results rather than transient clinical sensory manifestations. With previous published studies demonstrating clinical-electrophysiological discrepancies in axonal GBS variants.[6–9]

It is also important to mention that intravenous immunoglobulin administration in our case was done prior to nerve conduction study, which may have affected the interpretation.

We also agree with Dr. Finsterer that AMAN is not restricted to the lower limbs only and may involve upper extremities, respiratory muscles, and cranial nerves.[10] Our goal was not to imply otherwise but to highlight the predominant anatomical distribution of abnormalities in our patient.

Regarding autonomic system involvement, no major manifestations were reported such as; persistent tachycardia, blood pressure variation, urinary retention, constipation or sweating abnormalities.

Long-Term Follow-Up and Clinical Outcome

Concerning the extended follow-up data in the original paper, at time of manuscript submission and publication parts of patient's following care and long-term outcomes had been continued at external centers and institutions, minimizing our ability to provide comprehensive long-term follow-up details during manuscript writing.

Nevertheless, upon further clinic visits, complete neurological recovery was achieved in the patient, with restoration of normal tendon reflexes, and resolution of motor deficits. Currently, our patient is able to fully and independently ambulate – walk and run normally without limitation. No recurrence of previous presentations was documented over a three-years period of follow-up.

The patient undergoes regular follow-up clinic visits to monitor systemic lupus erythematosus (SLE) status – with hematology, rheumatology, and nephrology teams. Stable chronic disease with no abnormalities detected in the follow-up laboratory investigations were achieved using the following maintenance regimen; enalapril, hydroxychloroquine, mycophenolate mofetil, and low-dose prednisolone.

Additional Diagnostic Investigations

We acknowledge Dr. Finsterer's comments related to performing additional investigations as; ganglioside antibody testing and contrast-enhanced spinal magnetic resonance imaging (MRI). In our reported case, we performed non-contrast spinal MRI which demonstrated no structural vertebral, spinal cord, or compressive defects. Thus, Contrast-enhanced MRI was not deemed useful subsequently, especially with the low clinical suspicion for alternative pathology. Additionally, there is limited availability of ganglioside antibody testing in the middle-income country the patient got treated in. All in all, the diagnosis of GBS had been supported by the clinical presentation, CSF findings and electrophysiological studies, with the Brighton diagnostic criteria for GBS achieved.[11]

We sincerely thank the Editor-in-Chief and Dr. Finsterer for the opportunity to further discuss and clarify important aspects of our paper. We completely agree that infections should always be taken into consideration in evaluating pediatric patients with GBS presentation.[12] However, in our patient, the sequence of events, the absence of microbiological evidence, autoimmune serology, electrophysiological study, renal involvement, and biopsy confirmed lupus nephritis supported the consideration of SLE-associated GBS. Given the rarity of such association, we believe our paper remains clinically helpful and contributes to the reported literature regarding neuroimmunological association in the pediatric population.

Disclosure

The authors report no conflicts of interest in this communication.

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