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LETTER

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

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Dear editor

We read with interest the article by Kanan et al about a 4-year-old boy with systemic lupus erythematosus (SLE) which was made responsible for triggering consecutively the acute motor axonal neuropathy (AMAN) subtype of the Guillain-Barré syndrome (GBS).[1] The patient recovered from GBS after repeated administration of intravenous immunoglobulins (IVIG).[1] The article is compelling, but some points require further discussion.

First, alternative causes of GBS were not sufficiently ruled out in the described patient.[1] Even with a negative history of recent pulmonary or gastrointestinal infection, it would have been necessary to exclude the most common infectious causes of GBS.[2,3] These include infections with Campylobacter jejuni, Haemophilus influenzae, Mycoplasma pneumoniae, enterococci, typhoid fever, E. coli, hepatitis A, B, C, D and E, varicella-zoster virus, HIV, Zika, Epstein-Barr virus, influenza, SARS-CoV-2, rubella, West Nile virus, Japanese encephalitis and chikungunya.[2] Infections can be asymptomatic in children and adults, which is why the causative pathogens often go undetected.[3] Since treating the causative infection is part of the therapy for GBS, it is important to carefully screen for subclinical or mild infections. An indication of an infectious cause in the index patient is the diagnosis of a urinary tract infection on day 38 of hospitalization and the improvement of his condition only after treatment of the urinary tract infection with ceftriaxone.[1] Which pathogen was detected in the urine culture?

Second, we disagree with the diagnosis of AMAN.[1] AMAN is defined as purely motor axonal neuropathy.[4] However, the patient reported sensory disturbances, and the case report describes a loss of sensation in the upper and lower extremities, which, according to Figure 2,[1] resolved after 7–10 days. This discrepancy needs to be addressed. Even with normal nerve conduction velocity (NCV) measurements of the sensory nerves, the diagnosis of AMAN cannot be made and must be changed to acute motor and sensory axonal neuropathy (AMSAN). Furthermore, it should be determined whether autonomic fibers are affected.

Third, we disagree with the assumption that AMAN affects only the lower extremities.[1] In general, AMAN can affect all motor nerves, including those of the upper extremities, respiratory muscles, and head.

The fourth point is that the long-term course was not reported.[1] While recovery from GBS is mentioned in the abstract, the case report does not detail whether and after what period all GBS manifestations have completely resolved.[1] To assess whether the patient has fully recovered from SLE and GBS, the results of the 6- and 12-month follow-up examinations should be available. The immunosuppressive therapy the patient received for systemic lupus erythematosus (SLE) should also be reported.

Finally, some important information is missing. A contrast-enhanced magnetic resonance imaging (MRI) scan of the spine to document swelling and contrast enhancement of the nerve roots is missing. Ganglioside antibodies are missing, no reference values were provided in Table 2, detailed NCV measurements are missing, and no NCV reference values were reported.

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Overall, the GBS in the index patient was most likely triggered by a subclinical infection, probably in the bladder, and not necessarily by the SLE. The kidney involvement in the SLE may have predisposed the patient to the development of a urinary tract infection.

Disclosure

The author reports no conflicts of interest in this communication.

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