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Letter to the Editor

Before a Patient with Progressive Tetraparesis is Diagnosed with MADSAM, All Differential Diagnoses must be Thoroughly Ruled Out

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Letter to the Editor

We read with interest the article by Khan *et al.* about a 55-year-old woman with progressive, ascending tetraparesis and sensory disturbances following a lung infection, who was diagnosed with Lewis-Sumner syndrome (LSS), also known as multifocal acquired demyelinating sensory and motor neuropathy (MADSAN), after she had initially been misdiagnosed with systemic lupus erythematosus and treated with chloroquine for a year [1]. The patient benefited from repeated intravenous immunoglobulins (IVIGs) [1]. The study is interesting, but some points should be discussed.

The first point is that we disagree with the diagnosis of LSS (MADSAM) [1]. LSS is characterized by predominantly distal muscle weakness and conduction blocks [2]. LSS also predominantly affects the upper extremities and not the lower extremities, as was the case with the index patient [1]. Since the index patient showed proximal muscle weakness in the clinical neurological examination, the lower extremities were predominantly affected, and no conduction blocks were described in the nerve conduction studies (NCSs), it is rather unlikely that the patient described actually suffered from LSS. Another argument against MADSAN is that there was no elevated protein content in the cerebrospinal fluid.

The second point is that the symptoms and signs were not described in sufficient detail [1]. The patient initially complained of sensory disturbances and pain. In which areas did the numbness and pain occur? What type of pain did the patient report? Furthermore, the muscle weakness was not quantified [1]. The muscular research council (MRC) grades should be specified in order to assess the distribution and extent of the muscle weakness.

The third point relates to the discrepancy between the description that the patient was able to stand but was unable to walk [1]. This discrepancy should be clarified. Did the patient use a walking aid, crutches, or a rollator to walk?

The fourth point is that it was not clarified why the patient lost 13.6 kg in weight in six months [1]. Were there signs of dysphagia, did the patient aspirate? In cases of bulbar involvement, GBS patients may exhibit dysarthria or dysphagia [3]. Was there any indication of diarrhea or depression in the patient's medical history?

The fifth point is that the patient was described as having proximal muscle weakness as well as reduced hand strength [1], which indicates both proximal and distal weakness. Was the proximal weakness predominant over the distal weakness, or vice versa?

Finally, it is not clear why a patient with progressive paraparesis of the lower extremities was not referred to a neurologist. Although the patient reportedly underwent a comprehensive examination at the onset of paraparesis [1], it is unclear whether these examinations also included a neurological examination, NCSs, and CSF examinations. A patient with progressive, ascending paraparesis or tetraparesis must be referred to a neurologist immediately and undergo an appropriate neurological examination. Was the pathogen that caused the respiratory infection before the onset of paraparesis identified? Did the patient have an infection with influenza, Mycoplasma pneumoniae, cytomegalovirus, or SARS-CoV-2 [4]?

In summary, patients with progressive, ascending paraparesis should be referred to a neurologist immediately and undergo a neurological examination. Examination of paraparesis should not be delayed in order to ensure appropriate treatment and avoid misinterpretation. Delayed examination can complicate diagnosis, as important characteristics of a disease may disappear over time.

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References
1. Khan S, Wennberg B, Hooda F, Witkowska M. Delayed treatment and diagnostic challenges in differentiating multifocal acquired demyelinating sensory and motor neuropathy from lupus: A case report and literature review. AME Case Rep, Oct 24, 2025; 9:111. Doi: 10.21037/acr-2025-82
2. Viala K, Renié L, Maisonobe T, Béhin A, Neil J, Léger JM, *et al*. Follow-up study and response to treatment in 23 patients with Lewis-Sumner syndrome. Brain, Sep 2004; 127(Pt 9):2010-2017. Doi: 10.1093/brain/awh222
3. Cao Q, Chu H, Fu X, Yao J, Xiao Z, Lu Z. Case Report: Acute Bulbar Palsy Plus Syndrome: A Guillain-Barré Syndrome Variant More Prone to Be a Subtype than Overlap of Distinct Subtypes. Front Neurol, Nov 24, 2020; 11:566480. Doi: 10.3389/fneur.2020.566480
4. Finsterer J. Triggers of Guillain-Barré Syndrome: *Campylobacter jejuni* Predominates. Int J Mol Sci, Nov 17, 2022; 23(22):14222. Doi: 10.3390/ijms232214222