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Letter to the Editor

Autoimmune Hepatitis can Present with an Axonal Polyneuropathy Mimicking Guillain-Barre Syndrome

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Letter to the Editor

With reference to the article by Abdelmotelib *et al.* [1] knowledge of the trigger of Guillain-Barre syndrome (GBS) is needed for appropriate treatment and a good outcome. Triggers of GBS can be infections with neurotropic viruses or bacteria, vaccinations, malignancy, trauma, ganglioside infusion, or transplantation [2]. If GBS was diagnosed during the pandemic, SARS-CoV-2 infection should be considered a possible trigger.

The diagnosis of GBS, subtype acute motor axonal neuropathy, should be based on established criteria such as the Brighton-criteria or the EAN-criteria. According to the Brighton-criteria level-1 GBS is diagnosed if there is bilateral weakness, decreased/absent tendon reflexes, a monophasic course, normal cerebrospinal fluid (CSF) cell count, elevated CSF-protein, and nerve conduction studies consistent with one of the GBS subtypes [3].

In patients with autoimmune hepatitis (AIH), tetraparesis, increased CSF-protein, and normal cell count, GBS is unlikely, as AIH has been reported in association with axonal polyneuropathy [4] but not with GBS. Differentiation between radiculitis and length-dependent axonal neuropathy can be achieved by an MRI of the spinal cord, showing thickened and enhancing nerve roots in case of GBS [5]. Regarding the pathophysiological link between AIH and axonal neuropathy, an immune-mediated injury affecting peripheral nerves or small fibres can be considered.

In order to assess the effect of intravenous immunoglobulins (IVIGs) it is useful to specify the MRC-classification of tetraparesis before and after treatment. Careful monitoring of neurological recovery will help illustrate the treatment response.

In summary, clinicians should be aware that AIHs may mimic GBS through an axonal polyneuropathy pattern, and careful neurodiagnostic testing can prevent misclassification.

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