Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Letter to the Editor

□ SARS-COV-2-ASSOCIATED GUILLAIN-BARRE SYNDROME REQUIRES APPROPRIATE EXCLUSION OF POSSIBLE DIFFERENTIALS

□ To the Editor:

We read with interest the article by Yakoby et al. about a 35-year-old man who received a diagnosis of Guillain-Barre syndrome (GBS) 16 days after onset of coronavirus disease 2019 (COVID-19) (1). Severe acute respiratory syndrome *coronavirus 2* (SARS-CoV-2)–associated GBS (SC2-GBS) was suspected on clinical presentation and the temporal link between the viral infection and the GBS (1). Quadriparesis and sensory disturbances started 9 days after onset of COVID-19. The patient benefited from immunoglobulins (1). It was concluded that emergency physicians need to be alert for SC2-GBS (1). The study is appealing but has several limitations that raise some concerns.

The first limitation of the study is that the diagnosis of GBS was not confirmed by electrophysiologic investigations, such as nerve conduction studies (NCSs) or needle electromyography. GBS is usually diagnosed using the validated Brighton criteria and requires the results of NCSs. Because results of NCSs were not available, the diagnosis of GBS remains uncertain and classification of the GBS subtype is not feasible. GBS subtypes that should be considered in the index patient include acute, demyelinating, inflammatory polyneuropathy; acute, motor, axonal neuropathy; and acute, motor and sensory, axonal neuropathy. Other GBS subtypes are implausible, given the clinical presentation.

A second limitation is that no reference limits for laboratory parameters were provided. It is impossible to assess the amount of blood and cerebrospinal fluid (CSF) abnormality reported in the index patient.

A third limitation is that the CSF was not investigated for the presence or absence of SARS-CoV-2 RNA, or for cytokines, such as interleukin (IL)-8, IL-6. IL-1β, or tumor necrosis factor–α, which have been reported to be elevated in the CSF of patients with neuro-COVID (2).

A fourth limitation is that there was no discussion about the contribution of vitamin B-12 deficiency, which was diagnosed in the index patient during hospitalization; the clinical presentation and pathophysiology of neuro-COVID; and the outcome of the index patient.

An explanation for why the patient presented with resting tremor of both upper limbs, fasciculations of the left thigh muscles, and an asymmetry with regard to tendon reflexes on the lower limbs was missing. There was also no explanation for the elevated level of antinuclear antibodies.

An explanation of the term *electromyelogram* is required. Do the authors mean electromyography or myelography? An explanation for why there was no enhancement of the motor roots on spinal magnetic resonance imaging with contrast medium was missing. We should know whether the right patella tendon reflex was still exaggerated on follow-up.

Overall, this interesting study had several limitations that challenge the results and their interpretation. To establish the correct diagnosis and to broaden the knowledge about manifestations of immune-mediated neuro-COVID, it is crucial to investigate SC2-GBS patients extensively and sufficiently.

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