



Commentary

Guillain-Barre syndrome is a definite complication of SARS-CoV-2



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Letter to the Editor,

With great interest we read the article by El Aidouni et al. about a 49 years old male who developed quadriparesis with sensory disturbances in the lower limbs, and sphincter dysfunction two weeks following a mild COVID-19 infection [1]. Upon work-up by nerve conduction studies (NCSs), cerebro-spinal fluid (CSF) investigations and cerebral magnetic resonance imaging (MRI), the patient was diagnosed with Guillain-Barre syndrome (GBS) and treated with intravenous immunoglobulines (IVIG), which were ineffective [1]. The patient did not start to recover before application of plasmapheresis [1]. The study is appealing but raises the following concerns.

We do not agree with the notion that SARS-CoV-2 “potentially” triggers GBS [1]. There is meanwhile clear evidence that there is a pathophysiological link between SARS-CoV-2 and GBS from >300 cases reported with SARS-CoV-2 associated GBS as per the end of July 2021 [unpublished results]. Furthermore, GBS may not only be triggered by the viral infection but even by SARS-CoV-2 vaccinations [2]. Since diagnosing SARS-CoV-2 associated GBS can be difficult in patients requiring intensive care unit (ICU) treatment and since mild cases may occur, which are frequently missed, the true prevalence of SARS-CoV-2 associated GBS is likely to be higher than so far anticipated.

We do not agree with the definition of GBS in the introduction. GBS is not rare. GBS does not only manifest with muscle weakness but also with sensory disturbances or autonomic dysfunction. Autonomic dysfunction may be even the exclusive manifestation of GBS [3]. GBS may exclusively manifest in the cranial nerves (polyneuritis cranialis), only in with dysphagia and weakness of neck and upper limb muscles (PCB) or may manifest in the brainstem as Bickerstaff encephalitis (BBE). There is also the GBS subtype Miller-Fisher syndrome (MFS), which does not follow the definition of GBS provided in the introduction [1].

Missing is the classification of the GBS subtype in the index patient. We should be told if NCSs were indicative of acute, inflammatory, demyelinating neuropathy (AIDP) or acute, motor (and sensory), axonal neuropathy (AMAN/AMSAN). Knowing the GBS subtype is crucial as treatment and treatment response may vary between these two subtypes.

Missing is the information if CSF investigations revealed the presence

of SARS-CV-2 RNA or not. Since only three patients with SARS-CoV-2 associated GBS have been reported in whom virus RNA was found in the CSF [4,5], we should be told if GBS in the index patient was rather interpreted as immunogenic or as infectious.

How can the authors be sure that the index patient did not present with involvement of the respiratory muscles in GBS? Affection of the respiratory muscles can be only ruled out after application of NCSs to the phrenic nerve and lung function tests.

GBS is diagnosed according to the Brighton criteria [6]. Since NCSs did not unequivocally indicate GBS, level-1 of the diagnostic criteria is not accomplished.

Regarding the dimension of the amplitude of the sensory nerve potentials, we should know if the authors truly mean “mV”. Given the numbers in this column, we suspect that the authors rather mean “microV” than “mV”.

Overall, the elegant case report has several limitations which should be addressed before drawing final conclusions. There is a need to closely follow-up a SARS-CoV-associated GBS patient to assess when full recovery is achieved.

Statement of ethics

Was in accordance if ethical guidelines.

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Informed consent

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Ethical approval

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Author contribution

Josef Finsterer was responsible for design, literature search, discussion, first draft, critical comments, and final approval.

Registration of research studies

- 1 Name of the registry:
- 2 Unique Identifying number or registration ID:
- 3 Hyperlink to your specific registration (must be publicly accessible and will be checked):

Guarantor

J Finsterer.

Declaration of competing interest

None.

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