Journal of NeuroVirology (2022) 28:622–623
https://doi.org/10.1007/s13365-022-01067-2

LETTER TO THE EDITOR

SARS-CoV-2–associated Guillain–Barre syndrome requires extensive pre- and post-mortem examinations

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Received: 8 June 2021 / Revised: 8 June 2021 / Accepted: 28 February 2022 / Published online: 10 March 2022

With interest we read the article by Nejad et al. about a 70-year-old male who was diagnosed with Guillain–Barre syndrome (GBS) being attributed to a concomitant infection with SARS-CoV-2 (Nejad et al. 2021 May). GBS was diagnosed upon the clinical presentation and the cerebrospinal fluid (CSF) findings and treated with immunoglobulins. GBS was complicated by the development of autonomic neuropathy and the patient died from cardiac arrest despite artificial ventilation (Nejad et al. 2021 May). The study is appealing but has several limitations which raise the following comments and concerns.

The main limitation of the study is that nerve conduction studies (NCSs) were not carried out. GBS is usually diagnosed according to the Brighton criteria, which require the results of NCSs. NCSs are also required to assess which subtype of GBS (acute, inflammatory demyelinating polyneuropathy (AIDP), acute motor, axonal neuropathy (AMAN), acute motor and sensory, axonal neuropathy AMSAN) was present. Diagnosing GBS only upon the clinical presentation and CSF investigations is insufficient as differentials, such as critical neuropathy, pressure palsies, compartment syndrome, or neuropathy due to toxicity of anti-COVID-19 drugs, were not appropriately excluded. Treatment of the latter conditions is at variance from treatment of GBS.

Another limitation is that autonomic neuropathy was diagnosed but it was not specified how autonomic neuropathy manifested clinically and upon which methods and diagnostic criteria it was diagnosed. Were there pupillary or secretory abnormalities, decreased heart rate variability, voiding problems, or sexual dysfunction?

A further limitation is that the cause of cardiac arrest remained unexplained. Thus, we should be informed about the previous history, the current medication prior to hospitalisation, and results of the autopsy findings. Additionally, we should know the ECG and echocardiography findings prior to death. Did the patient develop heart failure or were pro-brain natriuretic peptide (pro-BNP) values and troponin values elevated? Since autonomic neuropathy is regarded as a predisposing factor for Takotsubo syndrome (TTS) (Khalid et al. 2015 Nov), we should be told if there were any indications for TTS, particularly if creatine-kinase, proBNP, or troponin were elevated.

We do not agree that GBS is a rare neurological complication of SARS-CoV-2 infection. GBS is increasingly recognised as a neuro-immunological complication of COVID-19. In a recent systematic review, 220 patients with SARS-CoV-2–associated GBS were reported (Finsterer and Scorza 2021). Since then, a number of further cases with SARS-CoV-2–associated GBS have been published.

It is unclear if respiratory distress was attributed to COVID-19 pneumonia or to affection of the respiratory muscles in GBS, or to both? Knowing the cause of respiratory distress is crucial as treatment depends on the underlying aetiology.

There are some inconsistencies. Why did it take 4 days until the patient underwent lumbar puncture? If GBS is suspected, immediate work-up has to be initiated. How can the patient cough on day 6 although he had been intubated on day 4?

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Overall, the interesting report has limitations which challenge the results and their interpretation. Diagnosing GBS requires appropriate work-up and exclusion of various differentials. The cause of cardiac arrest needs to be specified and autonomic neuropathy needs to be appropriately diagnosed.

Author contribution JF: design, literature search, discussion, first draft, critical comments.

Availability of data and material All data reported are available from the corresponding author.

Declarations

Ethical approval and consent to participate Not applicable.

Consent for publication Not applicable.

Competing interests The authors declare no competing interests.

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