Dear Editor,

We express our sincere appreciation to Finstere et al., for their deep consideration on our article entitled: Guillain-Barré syndrome associated with COVID-19: a case report study by J. Hosseini Nejad et al.

They have argued some concerns regarding the Guillain-Barré syndrome (GBS) report. Here, we intend to respond those point by point unbiasedly to clarify all aspects.

Their letter and our corresponding response can make accessible a perfect concept about the title and solve probable challenge for readers.

Finstere et al. have stated that 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.

In response, it should be noted that NCSs were not carried out because the patient was in critical condition and there was not possible to transfer him to the NCS ward. Certainly, while the NCS was not carried out, the subtypes will not be stratified.

Finstere et al. have argued that diagnosis of GBS is only upon the clinical presentation and CSF investigations are 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.

We say that it is reasonable that Guillain-Barré syndrome cannot be diagnosed by relying solely on CSF (Korinthenberg et al. 2020). But we did not do that. Patient history, patient clinical examination confirming Areflexia, and high level of protein detected in CSF were our criteria for GBS confirmation. In accordance with the text, all of them especially high level of protein provide more than 90% probability and indication of GBS (Walling and Dickson 2013). Although, it would have been better if the NCS had been done, but because of the patient critical condition, it was not possible.

In the other episode, Finstere et al. have issued 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?

Yes, we agree, it was a major limitation of the study.

A further limitation that have been mentioned by Finstere et al. is that the cause of cardiac arrest remained unexplained. The readers should be informed about the previous history, the current medication prior to hospitalization, and results of the autopsy findings. Additionally, readers 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 predisposing

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factor for Takotsubo syndrome (TTS), readers should be told if there were any indications for TTS, particularly if creatine-kinase, proBNP, or troponin was elevated.

Our response is that the most important factor for a GBS patient who suddenly experiences cardiac arrest is autonomic neuropathy. So, this dysfunction in GBS disease may lead to cardiac arrest (Zaeem et al. 2019). The patient underwent cardiac arrest during transfer to the ward for further clinical procedures.

Finstere et al. have declared that they did not agree that GBS is a rare neurological complication of a SARSCoV-2 infection. GBS is increasingly recognized as a neuro-immunological complication of COVID-19. In a recent systematic review, 220 patients with SARS-CoV-2 associated GBS were reported. Since then, a number of further cases with SARS-CoV-2 associated GBS have been published.

Maybe, it is a unilateral true statement. During the time of data gathering, report written, and publication, several reports were presented. However, at the time of case happening, very few cases of GBS were reported in relation with COVID-19. So, it was one of the first cases to be discovered. But on the one hand, we still believe that GBS is still one of the rare complication of COVID-19 because in the hundreds of millions of people who are infected, GBS is still very rare and is a rare complication.

Finstere et al. stated that it is unclear if respiratory distress was attributed to COVID-19 pneumonia, 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 etiology.

We also agree with this statement.

Finstere et al. have asked that, why did it take four days until the patient underwent lumbar puncture?

The response is that for two reasons:

(A) GBS was not of probable issue in the early days, meaning that 2 days had elapsed since the patient was admitted to the ICU when neurological counseling was requested.

(B) Contrary to the authors’ claim that the medical work-up should be taken immediately, the text mentions that CSF protein in the GBS may not be too high in the first week of the GBS may not aid in diagnosis.

Evidence suggests that after 4 days, the likelihood of a positive level of protein in CSF and its help to diagnose GBS increases. So, if the patient is undergone LP on the first day, the chances of failing to diagnose GBS were high.

The last issue that they have stated is that how can the patient cough on day 6 although he had been intubated on day 4?

Response is that it is a common frequent phenomenon in ICU. During the intubation, respiratory efforts and high irrigation caused by endotracheal tube lead to cough.

We once again thank Dr. Finstere research team for a thorough review of our article. We hope that these scientific discussions will pave the way for the transparency of the documents and the guidance of the readers.

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