The diagnosis of COVID Guillain-Barré syndrome requires the exclusion of differential diagnoses

El diagnóstico de COVID – Síndrome Guillain-Barré requiere la exclusión de diagnósticos diferenciales

Dear Editor,

We read with interest the article by Cea et al. about three females with SARS-CoV-2 associated Guillain-Barre syndrome (GBS) developing 5 before and 4 and 26 days after onset of COVID-19 respectively a pharyngeal swab PCR positive for SARS-CoV-2 (case 1, case 2, case 3)[1]. Only case 2 required mechanical ventilation. All three patients recovered partially upon application of intravenous immunoglobulins (IVIGs)[1]. The study is appealing but raises concerns that should be discussed.

We should be told how the authors knew that respiratory failure in the one patient with a need for mechanical ventilation (case 2) was due to pulmonary compromise from the SARS-CoV-2 infection and not from involvement of the respiratory muscles in GBS. COVID-19 pneumonia not necessarily requires mechanical ventilation in each case. Were there any indications for involvement of the axial or respiratory muscle in GBS in case 2?

Because case 2 had involvement of the VIIth, IXth, Xth, and XIIth cranial nerves, Bickerstaff encephalitis (BBE) should be ruled out. An MRI with contrast medium may either show an enhancing lesion in the brainstem or enhancing roots of the lower cranial nerves[2].

How can the authors be sure that case 1 had GBS and not deterioration of pre-existing diabetic polyneuropathy? Cerebrospinal fluid investigation did not show a dissociation cyto-albuminique and the patient had poorly controlled diabetes[1]. We should know the HbA1c values on admission. Diabetes control may have further deteriorated during the SARS-CoV-2 infection. We also should know the results of nerve conductions studies (NCSs) prior to SARS-CoV-2 associated GBS. These results should be compared with NCSs carried out one month after discharge.

The cause of coma in case 2 had not been clarified[1]. A normal computed tomography (CT) scan of the brain does not rule out brainstem immune encephalitis, brainstem stroke, or BBE. We should know if a cerebral MRI had been performed and with what result. We should know the pH value on admission. After which time did the patient recover from coma? How severe was ketoacidosis on admission and after awaking from coma?

We should know if the cerebro-spinal fluid (CSF) was positive for SARS-CoV-2 in any of the three patients. Though usually negative in SARS-CoV-2 associated GBS, a PCR for SARS-CoV-2 should have been performed in the CSF.

Patients with SARS-CoV-2 associated GBS may have elevated cytokines, chemokines, or glial factors in the CSF[3]. We should know if any of the three patients underwent determination of the cytokine profile in the CSF.

In case 3 GBS developed 26 days after onset of COVID-19[1]. This is a fairly long latency why alternative triggers of GBS should be considered. The patient was diagnosed with non-specific colitis. Was an infection with campylobacter jejuni or any other infectious agent ruled out?

We disagree with the argument that the prevalence of GBS did not increase because there was a general decrease of GBS due all other infections[1]. There are no indications that the infectious causes of GBS declined during the pandemic.

Overall, the interesting study has some limitations that call the results and their interpretation into question. Clarifying these weaknesses would strengthen the conclusions and could improve the study.

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