AUTHORS' REPLY TO SARS-COV-2 AND ANTI-COVID VACCINES TRIGGER GUILLAIN-BARRÉ SYNDROME

Dear Editor,

We greatly appreciate the comments and observations on our review article by Dr. Finsterer[1]. Concerning the epidemiological differences in electrophysiological variants of Guillain-Barré syndrome (GBS), we are aware that in Caucasians (persons from Europe and North America), acute inflammatory demyelinating neuropathy is the predominant variant, an epidemiological observation mentioned in our article[1]. Nonetheless, as we stated in the title of our article, we aimed to discuss mainly GBS epidemiology in Mexico, where acute motor axonal neuropathy is the prevailing electrophysiological variant[2].

The cited case report regarding cytokine elevation in cerebrospinal fluid is quite interesting[3]. We acknowledge the information of GBS cases occurring within close temporality to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections. Moreover, potential connection with plausible mechanistic associations has been made throughout the ongoing coronavirus disease 2019 (COVID-19) pandemic. However, despite these possible links, large-scale studies have failed to demonstrate a causal association between SARS-CoV-2 and GBS. We base this statement on the results of the largest epidemiological study on GBS and COVID-19 conducted to date by Lunn and colleagues in 2021, where they found no relationship between GBS and SARS-CoV-2 infections across the United Kingdom during the pandemic's first wave[4]. The aforementioned is supported by the fact that as the number of COVID-19 cases increased, the number of GBS cases did not, compared to pre-pandemic reports, a finding that reinforces the need for further population-based studies to establish a potential causal link[4].

It is important to note that the worldwide incidence of GBS decreased during the pandemic, possibly related to a reduction in the number of non-SARS-CoV-2 respiratory infections promoted by the widespread use of facemasks, suggesting that sporadic cases of GBS may occur even with close temporality to SARS-CoV-2 infection. Furthermore, regarding the inclusion of the pure dysautonomic clinical variant in our figure, we decided not to include it since the recognition of this variant is still in debate, as most of these patients during their disease course usually develop motor or sensory symptoms[5]. Moreover, the case report cited by Dr. Finsterer also states that this variant is still under debate[6]. Finally, it is well-known that case reports represent the lowest level of evidence; hence, we cannot establish a causal relationship without large-scale or population-based epidemiological studies.

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