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SARS-CoV-2 Definitively Causes Guillain-Barre Syndrome

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LETTER TO THE EDITOR

We read with interest Sirin *et al*'s editorial on the question of whether SARS-CoV-2 causes Guillain-Barre syndrome (GBS) or not Şirin, N. G. Several studies reporting increased incidence of GBS during the pandemic and some decreased incidence were discussed [Şirin, N. G, 2023]. The authors concluded that the causal relation between SARS-CoV-2 infection and GBS remains controversial and that case-control studies are needed to assess whether GBS is indeed causally related to SARS-CoV-2 [Şirin, N. G, 2023]. The study is impressive, but several points require discussion.

Arguments for a causal relationship between SARS-CoV-2 and GBS are that there is often a close temporal connection between onset of SARS-CoV-2 and the onset of GBS, that cerebrospinal fluid (CSF) studies often show elevated cytokines, chemokines, or glial factors, and that common triggers of GBS such as Campylobacter jejuni, Haemophilus influenzae, Mycoplasma pneumoniae, Epstein-Barr virus, cytomegaly virus, hepatitis-E, or Zika are negative. Another argument is that patients who were completely healthy suddenly become neurologically ill shortly after a SARS-CoV-2 infection.

The incidence rates reported in the literature have several limitations. First, the number of patients included is often small and the region examined is not representative. Second, patients with mild GBS are often not hospitalised, particularly during the pandemic, and therefore some patients may not be recorded in GBS statistics. Third, GBS can be easily misdiagnosed when the clinical manifestations are mild or when the clinical presentation is atypical. GBS can be easily overlooked, particularly in patients with pure or dominant dysautonomia, or cranial nerve involvement without peripheral nerve impairment, or in patients with predominant sensory symptoms. Also in ICU patients GBS can be easily overlooked and misinterpreted as critically ill neuropathy. Fourth, GBS patients not receiving intravenous immunoglobulins (IVIGs) are missed when IVIG registries are used for incidence calculation.

A clinical manifestation of GBS not included in the editorial is autonomic involvement. GBS can also manifest with pure dysautonomia without motor or sensory symptoms. Autonomic dysfunction as a manifestation of GBS is crucial to consider as it is easily overlooked and can manifest in various different presentations such as anhydrosis or hyperhidrosis, anxiety, blurred or double vision, bowel incontinence, brain fog, constipation, dizziness, dysphagia, exercise intolerance, insomnia, arterial hypotension, orthostasis, syncope, tachycardia, tunnel vision, urinary incontinence/retention, or weakness. The most common presentations of dysautonomia in GBS are postural tachycardia syndrome (POTS) and orthostasis.

A second manifestation of GBS not considered is mall fiber neuropathy (SFN) [Binder, A. *et al*., 2010]. SFN in GBS manifests as focal, regional, or global pain syndrome, sensory disturbances (hypoesthesia, dysesthesia, paresthesia) or autonomic dysfunction [Yuki, N. *et al*., 2018].

In summary, there is ample evidence that SARS-CoV-2 infection can cause GBS. GBS incidence rates may not have increased during the pandemic because GBS was misdiagnosed, mild cases not hospitalised, or IVIG registries were used to calculate incidence rates. Patients with SARS-CoV-2 associated GBS should be taken seriously, and diagnosed and treated adequately and quickly.

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