Post-COVID-19 Guillain-Barre syndrome with good response to intravenous immunoglobulin

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The novel coronavirus severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) and its association with Guillain-Barre syndrome (GBS) were reported after the outbreak of the pandemic in 2019. We are reporting a case of post-COVID-19 GBS with a good response to intravenous immunoglobulins (IVIg).

A 60-year-old right-handed gentleman presented with bilateral lower limb weakness of 3 days duration with no pain, tingling, or numbness of the limbs. The weakness was gradual in onset, initially involving the calf muscles and then progressed to involve the thigh. He was initially able to walk with support and then was completely bedbound. On examination, his heart rate was 82 beats/min, blood pressure was 132/82 mmHg on the right upper limb in the supine position, respiratory rate was 14 cycles/min, and the temperature was 37° centigrade in the right axilla. Central nervous system examination revealed normal cognition with both lower limbs power of 3/5, deep tendon reflexes were normal in upper limbs and absent in bilateral knees and ankle. Sensory examination was normal and there were no cerebellar or meningeal signs, other systems examination was normal. Magnetic resonance imaging brain and spinal cord were within normal limits. Nerve conduction studies revealed the motor axonal type of demyelination. He had moderate COVID-19 25 days back and had completely recovered. He was initiated on plasma exchange (PLEX). In the next 2 days, his muscle weakness continued to progress in spite of PLEX involving the upper limb and respiratory muscles. Hence, he was intubated and initiated on mechanical ventilation. After 4 cycles of PLEX, he was started on IVIg due to progression of muscle weakness to 1/5 in both upper and lower limbs. Post 2 g/kg of IVIg over 5 days, his muscle power gradually improved. He was tracheostomized on day 7 of his intensive care unit stay and was gradually weaned off the ventilator. His repeat nerve conduction study done after 20 days of illness showed improvement in conduction velocity. He was transferred to the ward after 30 days of stay in intensive care.

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Recently, many cases of GBS were reported during the SARS-CoV-2 epidemics all over the world. Patients with COVID-19 typically have a fever and respiratory illness; in addition, a wide range of other symptoms has been described in the literature. While the neurological sequelae of the virus remain poorly understood, there are a growing number of reports of neurological manifestations of COVID-19 [1]. GBS associated with SARS-CoV-2 infection usually follows the typical post-infectious pattern, GBS has also been reported as part of the "long COVID-19 syndrome" [2,3]. The exact pathogenesis of COVID-19-related neurological damage is still largely unknown. Molecular mimicry between SARS-CoV-2 and various human organs and tissues has been hypothesized as a potential trigger of multiorgan autoimmunity in COVID-19 [4-6]. Furthermore, the inability to detect the SARS-CoV-2 in the majority of the CSF patient samples suggests an immune mechanism rather than direct invasion [7].

In a study conducted by Lucchese and Flöel, sequence analysis of the 41 human proteins associated with acute and chronic immune-mediated neuropathies revealed that SARS-CoV-2 contained two immunologically related hexapeptides (KDKKKK in nucleocapsid and EIPKEE in Orf1ab) with the human heat shock proteins 90 (HSP90B and HSP90B2) and 60 (HSP60), respectively [8]. They hypothesized that SARS-CoV-2 infection triggers an adaptive immune response in which T cell-B cell interactions lead to the production of specific antibodies similar to ganglioside-peptide sequences or structures, which are located on the membranes of neurons and the Schwann cells and act as receptors for anti-ganglioside antibodies, which turn them into targets for autoimmune-mediated destruction of myelin sheaths. Dufour *et al*. reported the first case with COVID-19-related GBS with a positive GM1 antibody [9]. Treatment with IVIg or PLEX is the standard management approach, along with supportive care. Majority of the studies have not shown difference in outcomes with IVIg or PLEX in patients with GBS. In a systematic review of 41 patients with post-COVID-19 GBS, the majority (83.3%) received IVIg, while 11.1% received PLEX and 5.6% were treated with both the treatment modalities [10]. In our case, the patient responded to IVIg and hence IVIg may be

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superior as compared to PLEX in post-COVID-19 GB syndrome patients. Further studies are needed to substantiate the same.

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