A Case Series of Guillain-Barré Syndrome After COVID-19 Infection in New York

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Although much is known about the respiratory complications of infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), a wider spectrum of neurologic manifestations are beginning to be observed.1 We report 2 patients who presented to our hospital in New York, an epicenter of the coronavirus disease 2019 (COVID-19) pandemic, with Guillain-Barré syndrome (GBS) after SARS-CoV-2 infection.

Case 1
A 68-year-old man presented with 5 days of progressive gait disturbance and paresthesias of his hands and feet. Eighteen days before the onset of these symptoms, he had a fever and upper respiratory symptoms that lasted 10 days. A nasopharyngeal swab was positive for SARS-CoV-2 infection. Initial examination was notable for 4+/5 weakness in hip flexors and absent vibratory and proprioceptive sense at the toes. Reflexes were 2+ in the arms and absent in the legs. His gait was unsteady with inability to toe or heel walk. MRI of the lumbosacral spine was unremarkable. He declined lumbar puncture and opted to be monitored conservatively without treatment. Three days after admission, he developed bilateral facial weakness, dysphagia, dysarthria, neck flexion weakness, and inability to ambulate. At this time, he consented to a lumbar puncture; his CSF results were consistent with GBS (table). He completed 5 sessions of plasmapheresis without further symptom progression. Serum ganglioside antibody testing was unremarkable. Electromyography (EMG) was deferred because of infection control measures. His examination stabilized, and he was discharged to a rehabilitation facility after the completion of therapy. His dysphagia has resolved, and 28 days after GBS symptom onset, he can now ambulate with minimal assistance.

Case 2
An 84-year-old man presented with 7 days of paresthesias of his hands and feet and 3 days of progressive gait disturbance. Twenty-three days earlier, he had a fever, and a nasopharyngeal swab was positive for SARS-CoV-2 infection. Initial examination revealed 3/5 shoulder shrug, 4-/5 hip and neck flexion, and diminished vibration and proprioception at the toes. Reflexes were 1+ in the arms and absent in the legs. He was unable to stand or ambulate independently. The CSF results were consistent with GBS (table). Thus, plasmapheresis was initiated. Despite treatment, by hospital day 3, he developed bilateral facial weakness, progressive arm weakness, autonomic dysfunction, and neuromuscular respiratory failure requiring mechanical ventilation. He completed 5 sessions of plasmapheresis without further progression but remained ventilator-dependent, so he was then
given intravenous immunoglobulin (IVIg). Serum ganglioside antibody testing showed elevated GM2 IgG/IgM antibodies. EMG was deferred. He underwent tracheotomy, and 25 days after GBS symptom onset, he remains quadriparetic with intermittent autonomic dysfunction but is slowly being weaned from the ventilator.

**Discussion**

These cases add to the existing literature on GBS associated with COVID-19.²⁻⁵

The first report of GBS and SARS-CoV-2 is from China; the patient presented with concomitant neurologic and viral symptoms.³ In subsequent reports from Italy, Iran, and Pennsylvania, 7 patients developed GBS symptoms less than 2 weeks after the onset of respiratory symptoms.²,⁴,⁵ In our cases, both patients did not develop GBS symptoms until 3 weeks after the initial onset of viral symptoms. For GBS associated with a preceding infection (respiratory or gastrointestinal), the time interval between infection and onset of neurologic symptoms varies, ranging from 3 days to 3 weeks.⁶ We speculate that our patients’ onset of GBS was protracted because of an immune-mediated mechanism as opposed to direct viral-mediated damage. Time is required to produce immunoglobulins to SARS-CoV-2 and allow them to circulate and access the peripheral nervous system. Although the Pennsylvania case is an exception and the number of reports is limited, it is interesting to note that there seems to be a progressive delay between onset of viral symptoms and development of GBS as the pandemic spread from East to West.

GBS can be diagnosed clinically, and although an EMG may be helpful, it is not essential to make this diagnosis⁶; thus, an EMG was not performed for our patients because of the pandemic conditions. The CSF, however, was obtained, as we queried whether these patients had a postinfectious process or a process mediated by ongoing viral infection. CSF analysis in each case demonstrated albuminocytologic dissociation commonly seen in GBS, but SARS-CoV-2 was negative; these findings were similar to the CSF results included in the Italian series.⁷

Although we used plasmapheresis as our first-line intervention for both patients and only administered IVIg after decline despite plasmapheresis, all patients presented in other case reports were initially treated with IVIg.²⁻⁵ Both therapies are known to accelerate time to recovery in patients with GBS with a symptom duration of under 4 weeks.⁷ However, we chose plasmapheresis as the initial treatment because of the concern that both IVIg and SARS-CoV-2 can increase the risk of hypercoagulability.⁸

SARS-CoV-2 infection can cause GBS. The presentation, diagnostic methodology, and treatment for SARS-CoV-2 induced GBS around the world during the present pandemic seem to vary.

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**Disclosure**

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**References**


### Table CSF Results

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<th>Protein (mg/dL)</th>
<th>Glucose (mg/dL)</th>
<th>COVID-19 PCR (CSF)</th>
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<td>Case 1</td>
<td>3</td>
<td>226</td>
<td>56</td>
<td>Negative</td>
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<tr>
<td>Case 2</td>
<td>1</td>
<td>67</td>
<td>58</td>
<td>Negative</td>
</tr>
</tbody>
</table>

Abbreviations: COVID-19 = coronavirus disease 2019; WBC = white blood cells.

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**Appendix Authors**

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<tr>
<th>Name</th>
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<th>Contribution</th>
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<tbody>
<tr>
<td>Monica Chan, MD</td>
<td>NYU Langone Medical Center</td>
<td>Treated patient; drafted, revised, and edited the manuscript</td>
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