Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Chest Infections

SESSION TITLE: Medical Student/Resident Chest Infections Posters
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COVID-19 INFECTION COMPLICATED BY GUILLAIN-BARRÉ SYNDROME: A CASE REPORT

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INTRODUCTION: COVID-19 (SARS-CoV-2) is causing a current pandemic. It commonly manifests with fever, dyspnea, and cough. Few COVID-19 patients with Guillain-Barré Syndrome (GBS) have been reported. The severe inflammatory response and the critically-ill nature of many COVID-19 patients is a challenge to distinguish GBS from critical illness polyneuropathy and myopathy. We present a COVID-19 patient complicated by GBS.

CASE PRESENTATION: A 60-year-old woman presented with fever, cough, myalgia, and dyspnea for 10 days. A swab for SARS-CoV-2 RT-PCR was positive. CT chest revealed bilateral "ground-glass" opacities. She was started on oxygen, azithromycin, and hydroxychloroquine. Three weeks later, she developed bilateral symmetrical LE numbness and weakness that progressed to UE. Respiratory status worsened with increasing O2 requirements. Neuro exam showed weakness (2/5) in LE and (3/5) in UE. Respiratory muscle testing demonstrated a NIF of -35 cm H2O and an FVC of 1.7 L. MRI spine showed contrast enhancement of cauda equina nerve roots (Fig 1). CSF analysis revealed cytoalbuminologic dissociation (CAD) with 197 mg/dL of proteins and 0 WBC. She was diagnosed with GBS and started on intravenous immunoglobulin (IVIG) 0.4g/Kg/day for 5 days. After a week of therapy, the patient improved, recovered from COVID-19, and was discharged home.

DISCUSSION: GBS is a disorder in which the immune system attacks gangliosides on the peripheral nervous system. It presents with ascending weakness and can cause total body paralysis and respiratory failure in severe cases. It is associated with a variety of viral and bacterial infections. 12 cases of GBS have been reported in COVID-19 infection. GBS developed within 10 days of COVID diagnosis and presented with ascending progressive, flaccid quadriparesis. All except 2 patients underwent CSF analysis and 91% showed CAD. IVIG was used for all the patients, and one was started on plasmapheresis. The involvement of the PNS supports the coronavirus neurotropic invasion pathway. It is still unclear if SARS-CoV-2 can directly invade neurons and cause neuropathy. We could not test for SARS-CoV-2 in CSF in our case, but the absence of WBC in the CSF indicated an immune response typically seen in GBS rather than direct neuronal invasion, in which pleocytosis is expected. MRI usually showed contrast enhancement of cauda-equina nerve roots due to radicular irritation. CSF showing CAD is usually observed in the second week after symptom onset. IVIG is preferred over plasmapheresis for treating GBS due to fewer side effects. However, thrombotic events occur in 1–16.9%. All of the reported COVID-19 cases with GBS, including our case, received IVIG, and none of them reported thrombotic events.

CONCLUSIONS: Our case emphasizes that GBS should be considered as one of the differentials in patients with COVID-19 patients with ARDS, polyneuropathy, and difficulty weaning off ventilator.

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