Introduction
Guillain-Barre syndrome (GBS) is a rare autoimmune-mediated polyradiculoneuropathy that is often preceded by a viral infection. GBS has emerged as a complication of coronavirus disease 2019 (COVID-19). Here we report a case of GBS in a patient with symptoms that started on the 3rd day after the diagnosis of COVID-19 was made. The patient had a prolonged course of acute hospitalization and rehabilitation, however demonstrated significant functional gains by the time of discharge. This clinical scenario is important to recognize to help guide diagnosis and facilitate proper intervention.

Case Description
A 62-year-old male with history of sarcoidosis, ulcerative colitis, and degenerative joint disease initially presented to the emergency department with a 2-day history of ambulatory dysfunction due to profound lower extremity weakness. He was notably diagnosed with COVID-19 three days prior. He was evaluated by the neurology team. Workup was performed, including a lumbar puncture (LP) and brain/C-spine MRI’s. The LP revealed cytoalbumic dissociation, consistent with GBS. He was noted to have sensory motor quadriparesis, facial diplegia, and ophthalmoplegias, and was suspected to have the axonal sensory motor subtype of GBS. He received two rounds of intravenous immune globulin (IVIG).

Discussion
Since the emergence of COVID-19 and its declaration as a global pandemic, it has been associated with various neurological manifestations involving both the central and peripheral nervous systems. GBS is an acute autoimmune disease of the peripheral nervous system, usually preceded by a respiratory or gastrointestinal viral infection. Clinical manifestations of GBS range from mild cases of brief weakness to more severe cases of complete paralysis, including respiratory failure. Neuromuscular involvement presents as progressive, ascending, symmetrical weakness, as well as paresthesia’s with diminished or absent deep tendon reflexes. Thus, it is imperative to consider GBS as a potentially serious sequela of COVID-19 and include it in the differential in patient with COVID-19 with subsequent neurological changes, including weakness and ambulatory dysfunction, as early diagnosis and management can improve clinical outcomes.

He was then deemed an appropriate candidate for acute inpatient rehabilitation after a prolonged hospital course of nearly two months. On admission, manual muscle testing revealed the following strength grades: bilateral hip flexors (HF) 2/5, bilateral knee extendors (KE) 3/5, bilateral dorsiflexors (DF)/plantarflexors (PF) 0/5. Sensation was diminished to light touch in bilateral feet. He was unable to walk. While at rehab, he continued to improve with therapies over the course of approximately six weeks. He was started on Gabapentin for management of neuropathic pain in his lower extremities. By the time of discharge, manual muscle testing showed improvement in the majority of the tested muscle groups with the following strength grades: left HF 3/5, left KE 4/5, right HF 4/5, and right KE 4/5. He continued to have foot drop, however was able to walk out of rehab utilizing a rolling walker with bilateral dorsiflexion assist braces.

Conclusions
While a link between COVID-19 and GBS has not yet been fully elucidated, it is important to keep this on the differential diagnosis of someone with new neurologic symptoms following a COVID-19 infection. In our patient, it is hard to ignore the temporal relationship of a relatively healthy individual becoming profoundly weak only a few days after contracting COVID-19. However, with a structured, intensive rehabilitation program, patient’s can still recover from the GBS and be able to return home at a functional level.

References