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Case Report

Guillain Barre Syndrome in a Young Man with SARSCoV-2: A Rare Association

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A B S T R A C T

Guillain Barre Syndrome (GBS) is usually a post-infectious autoimmune disease that manifests as acute ascending flaccid paralysis. The disease is usually uncommon. However, recently it was reported in a few COVID-19 cases before complete resolution of COVID symptoms. An association between olfactory-gustatory disturbances and sensory abnormalities is frequently observed in GBS with COVID-19. The electrophysiological studies usually reveal a demyelinating pattern. Respiratory involvement, as part of respiratory muscle paralysis or COVID-19 pneumonia, is associated with poor recovery in affected patients. Here, we present a case of a young man, pre-morbid healthy, who presented with GBS with mild COVID-19 infection. He successfully recovered after treatment with Intravenous immunoglobulin IVIg.

Keywords: COVID-19, Guillain Barre Syndrome (GBS), AIDP, Paralysis, Steroids, IVIg

Introduction

Coronavirus disease (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is typically associated with respiratory illness (mild to severe), anosmia and/ or non-specific systemic complaints. Neurological manifestations of this disease are less commonly reported than other manifestations. Headache, stroke, seizure, encephalitis, vertigo, and paresthesia have been reported earlier. However, Guillain Barre syndrome (GBS) has been reported only on an occasional basis.[1]

GBS is an autoimmune polyneuropathy caused by infectious agents like *Campylobacter*, Influenza virus, Epstein Barr virus and Zika virus. The incidence of GBS was higher when associated with a COVID-19 positive state as compared to a negative state, suggesting probable neurotropism of this virus.[2,3] We present below a case of SARS-CoV-2 infection in a young male who developed GBS and was successfully managed with intravenous immunoglobulin (IVIg).

Case Report

A 28-year-old man was hospitalized with tingling sensations in both legs for seven days, weakness of legs for four days, and difficulty in phonation and swallowing for the last one day. The tingling sensation began on both legs and was perceived in the right arm for the last two days. The weakness in legs had an ascending progression with initial distal muscles weakness, marked by slipping of footwear, gradually progressing upwards to involve the proximal muscles of the legs. The weakness also increased in severity with the patient not being able to sit unsupported or lift himself up from the bed. He denied any weakness in the upper limbs at admission. Since last one day, he had also developed difficulty in phonation and swallowing with nasal regurgitation of liquids. He complained of pooling of secretions in his throat. However, there was no difficulty in sensation or voiding of urine. He was afebrile at admission but gave a history of undocumented low-grade fever for

the last three-four weeks. He also reported dysgeusia for the last seven days, however, there was no cough, throat pain, anosmia or shortness of breath. There was no history of rash, diarrhoea, vomiting, or jaundice. He was tested positive for SARS Cov-2 infection on 20th day of symptoms as part of screening, when admitted to the hospital with the above neurological complaints, and was referred to our centre. He had a past history of fistula-in-ano which was treated conservatively two months back, and was healed at present. There was no underlying co-morbidity. He was a non-smoker and non-alcoholic and a student by profession.

At admission, he was conscious and oriented with a blood pressure of 138/88 mmHg, heart rate of 94/minute, respiratory rate of 18/min without any use of accessory muscles and oxygen saturation on room air was 97%. General physical examination was unremarkable. Central nervous system examination revealed normal sensorium and intact memory. He had absent gag reflex with a nasal twang in his voice and pooling of secretions in the oropharynx. Rest of the cranial nerves were normal on clinical testing. Motor system examination revealed decreased tone in bilateral lower limbs with loss of muscle power (3/5) in muscle groups at hip, knee, and ankle. There was no weakness in the upper limb muscles. There was no involvement of the respiratory muscles or diaphragm. Single breath count was 22. The deep tendon reflexes were absent in both upper and lower limbs with absent Babinski reflex. Sensory system examination was normal for touch, vibration, temperature and pressure. There were no cerebellar or meningeal signs. Rest of the systemic examination was unremarkable.

A provisional diagnosis of acute ascending flaccid paralysis with bulbar palsy was kept. Nerve conduction study was performed which revealed prolonged distal latency in bilateral common peroneal and right posterior tibial nerve, but normal distal latency in median and ulnar nerves. Compound Muscle Action Potential (CMAP) amplitude was normal in the left posterior tibial nerve with reduced conduction velocity. F-wave was absent in bilateral common peroneal and posterior tibial nerves but normal in ulnar and median nerves. H-reflex was absent bilaterally in lower limb nerves, suggestive of Acute Demyelinating Inflammatory Polyneuropathy (AIDP). The laboratory investigations after admission showed haemoglobin of 14.1 g/dL, total leucocyte count of 7800/mm^3 ($P_{31}L_{63}M_3E_1$) with normal renal function tests. Serum sodium, potassium and calcium were 134 meq/L, 4.4 meq/L, and 10.2 mg/dL, respectively. Liver enzymes were mildly elevated with alanine transaminase of 57 IU/L and aspartate transaminase of 69 IU/L.

The patient was kept nil per oral and started on tube feeds with frequent oral suctions. He had received one dose of injectable methylprednisolone 40 mg one day prior to admission in this hospital in view of his COVID-19 positivity. IVIg was started as 2 gm/day as a slow intravenous infusion. He also received supportive treatment for SARS-CoV-2 with antioxidants, tablet zinc, tablet ivermectin and subcutaneous low-dose heparin once a day. The lower limb weakness started to improve by the third day of IVIg infusion and he was able to stand by day 10 of hospitalization. The bulbar palsy recovered slowly and he was started on semisolid feeds on day 12 of hospitalization. His repeat RTPCR test for SARS-CoV-2 was negative on day 14 of hospitalization and he was discharged on the 20th day of hospitalization. He didn't develop any respiratory complications during the hospital stay. At discharge, he had residual bulbar weakness with minimal residual motor weakness in both lower limbs (muscle power at hip 4/5 and 5/5 in rest of the muscle groups).

Discussion

GBS can be a consequence of molecular mimicry between antibodies against SARS-CoV-2 and gangliosides on the surface of peripheral nerves. There have been recent reports of association of these two clinical conditions.[2-5] The resultant immune mediated demyelination or axonal damage results in the clinical symptoms characterized by ascending flaccid paralysis with areflexia.

Most patients with GBS developed neurological symptoms before the complete resolution of COVID-19 symptoms. The average interval between onset of symptoms of SARS-CoV2 and associated GBS varied from 14-24 days.[2,4] The index patient developed neurological symptoms in the late third week of illness. A recent parainfectious profile has been reported with SARS-CoV2 where symptoms of GBS presented at mean 8 days of COVID-19 symptoms raising doubts as to the true cause of respiratory failure between GBS and COVID-19.[5,6] The antiganglioside antibodies were negative in majority of the affected patients suggesting a probable direct neuronopathic pathophysiology instead of a postinfectious autoimmune phenomenon in COVID-19 with GBS.[5]

The presence of sensory symptoms and paraparesis/quadriparesis was commonly seen in affected patients, unlike autonomic symptoms.[4] Majority of the patients had associated pneumonia and required assisted ventilation.[2,4] An olfactory-gustatory disorder was commoner in GBS patients with COVID positive status, as also in the index case, than those with COVID-negative status. This is probably a result of olfactory nerve being directly exposed to the virus through nasal invasion.[3] However, as compared to GBS with COVID-19 negative status, those who were COVID-19 positive were more likely to have involvement of all four limbs, lower muscle strength, and requirement of intensive care.[2] On the contrary, there was no difference in the clinical

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features or recovery in 47 GBS cases with respect to their COVID-19 status reported during the pandemic in the UK, probably suggesting the role of host factors in determining the severity of autoimmune process.[7] The index patient had associated dysgeusia but did not require ventilatory support for COVID-19 or GBS.

The diagnosis of GBS is supported by albuminocytological dissociation in cerebrospinal fluid or characteristic pattern on electrophysiological studies. The pattern on electrophysiological studies can help classify acute polyneuropathy as Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN), or Acute Motor Sensory Axonal Neuropathy (AMSAN).[8] The SARS-CoV-2 associated GBS type has been reported as the AIDP variant in majority with a few patients showing AMAN or mixed variant.[2,4,9]

Almost 70% recovery rate was reported in affected cases, though old age and underlying severe COVID-19 were associated with poor prognosis.[4] IVIg was used successfully in the majority of cases with a few patients requiring plasmapheresis or steroids. IVIg was started promptly and recovery was good in the index patient. An association with influenza vaccine has been inconclusively documented earlier based on comparison of observed cases of GBS in vaccine recipients than earlier proportion.[10] However, whether a similar risk would be seen with the COVID-19 vaccine, is yet to be ascertained.

To conclude, the above case represents a relatively less frequent neurological manifestation of COVID-19 in a young previously healthy male. His COVID-19 remained mild in severity and GBS promptly responded to IVIg therapy.

Conflict of Interest: None

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