

COVID-19-induced Guillain-Barre Syndrome: A Rare Complication of SARS-CoV-2 Infection

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ABSTRACT

SARS-CoV-2 causing COVID-19 initially began in Wuhan, China and now has been declared a pandemic by the World Health Organization (WHO). In addition to respiratory symptoms, it can cause various complications ranging from neurological to myocardial injuries. Guillain-Barre Syndrome (GBS) is an acute polyradiculoneuropathy affecting more often lower limbs than upper limbs and is often related to previous infectious diseases. We, herein, describe a case of a young female who presented with typical symptoms of GBS after having COVID-19 and was later on confirmed with nerve conduction study and lumbar puncture.

Key Words: COVID-19, SARS-CoV-2, Guillain-barre syndrome.

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INTRODUCTION

Coronavirus disease 2019 (COVID-19) caused by SARS-CoV-2 initially began in Wuhan, China in December 2019 and now has been declared as a pandemic by the WHO. COVID-19 patients mostly present with fever, headache, dyspnea and fatigue but can manifest various neurological and myocardial complications.^{1,2} Only a few cases of COVID-19-associated Guillain-Barre Syndrome (GBS) have been reported so far. For instance, Sedaghat Z *et al.* reported a COVID-19 induced GBS in which they presented a 65-year-old male patient who came to Emergency Department (ED) with acute progressive symmetrical quadriparesis and later on GBS was diagnosed by nerve conduction study.³ Five other patients having features of GBS secondary to COVID-19 have been reported from Italy. Four out of these presented with lower-limb paralysis and one patient had facial diplegia on presentation and later on ataxia and paresthesia developed. The latency period among all the patients from the onset of COVID-19 symptoms to the development of GBS symptoms was 5 to 10 days.⁴ Here, we present such a rare complication of COVID-19 in a 32-year female who developed GBS two weeks after SARS-CoV-2 infection.

CASE REPORT

A 32-year female having a history of polymerase chain reaction (PCR)-proven COVID-19 three weeks previously, presented to the ED with a history of bilateral lower limb weakness from the past 5 days. On examination, she was vitally stable with a blood pressure of 140/90 mmHg, a pulse of 80/min, and oxygen saturation of 97%. Neurological examinations revealed diminished knee reflexes in both lower limbs. Motor examination showed decreased power in lower limbs (3/5) with normal power (5/5) in upper limbs. She was admitted as a suspected case of GBS and baseline investigations were sent, results of which were unremarkable. A nerve conduction study was performed, which showed features of acute generalised sensory-motor polyneuropathy, of mixed variety, predominantly of demyelinating type, predominantly affecting the lower limbs. Lumbar puncture showed a high level of protein of 195 mg/dL, normal glucose, 73 mg/dL and a cell count of less than 5/mm³ (Table I). She was planned for plasmapheresis therapy. After three sessions of plasmapheresis, she improved clinically and was discharged home. She was advised a follow-up after one month.

Table I: Laboratory findings of CSF analysis.

Appearance	Clear
CSF Glucose	73 mg/dL
CSF Protein	195 mg/dL
Cell count	<05/mm ³
Giemsa Stain	Occasional lymphocytes seen
Gram Stain	No microorganisms seen
ZN Stain	No AFB seen

DISCUSSION

GBS is an acute flaccid paralytic condition that commonly manifests as progressive symmetric weakness; and patients usually

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develop these symptoms within three days to six weeks after diarrheal type condition or upper respiratory infection. *Campylobacter jejuni* is the most common bacterial infectious agent associated with GBS. Other bacterial and viral pathogens involved in GBS development are mycoplasma pneumonia, haemophilus influenza, cytomegalovirus, Epstein-Barr virus, human immunodeficiency virus, and Zika virus.^{5,6} GBS, which is an autoimmune disease, occurs when antibodies against surface glycoproteins of the offending pathogen cross-react with protein structures of peripheral nerve components (molecular mimicry). Other coronaviruses such as *MERS-CoV* and *SARS-CoV* infections are associated with GBS. Kim *et al.* described a case of GBS in a patient infected with *MERS-CoV*, while neurologic manifestations in *SARS-CoV* infected patients were revealed by Tsai *et al.*^{7,8} *SARS-CoV-2* enters the human cells by binding to the angiotensin-converting enzyme 2 (ACE2) receptors, producing a wide range of manifestations. The most common symptoms are fever, cough, dyspnea, myalgia, headache, and diarrhea.^{1,4} Zhao *et al.* reported the first case of GBS in *SARS-CoV-2* infected patients.⁹ Scheidl *et al.* reported a 54-year-old woman presenting with upper limb weakness after three weeks of having mild COVID-19. She was diagnosed as a case of GBS secondary to *SARS-CoV-2* infection after ruling out other possibilities.¹⁰

Multiple theories have been put forward for the pathogenesis of GBS development in COVID-19 patients; but the exact mechanism is not known, yet. One theory states that nervous tissue injury can be associated with direct neuronal invasion by the virus through its direct binding with ACE2 receptors. Another hypothesis explains the immune-mediated indirect injury to neurons, stating that the immune system is overstimulated with increased formation of interleukin-6 (IL-6) and to the generation of an autoimmune reaction. It has been stated that *SARS-CoV-2* stimulates excessive immune reaction, activates inflammatory cells with an increased formation of cytokines such as IL-6, leading to neuronal tissue damage.³ It is thus possible that these immunological processes are responsible for the neurological manifestations in COVID-19 patients. It has also been stated in literature that COVID-19 patients with severe symptoms and quickly worsening clinical condition are more prone to develop grave neurological events. It is still unknown whether COVID-19 prompts the formation of autoantibodies against specific gangliosides, which are normally seen with other forms of GBS. Further studies need to be done regarding the pathophysiologic mechanism of GBS in patients with COVID-19 in the future.

Although *SARS-CoV-2* primarily causes respiratory and gastrointestinal symptoms, but physicians should be aware of the neurological manifestations of GBS possibly related to *SARS-CoV-2* infection. There is limited data available in the literature regarding the pathogenesis of GBS in COVID-19 patients.

Further large-scale studies should be conducted to understand the pathogenesis of this association.

PATIENT'S CONSENT:

A written informed consent has been taken from the patient.

CONFLICT OF INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

MH: Collected the data.

MH, AWK, MAK, FS, SJK: Wrote the initial manuscript.

MH, AWK, SJK: Critically revised the manuscript.

All authors have read the final manuscript.

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