

## A case of guillain-barré syndrome associated with post-covid infection during 2<sup>nd</sup> wave of coronavirus in India

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### ABSTRACT

The novel coronavirus-COVID-19 was detected in Wuhan city of Hubei Province in China on December 31, 2019. Coronaviruses are known to infect multiple organ systems, with respiratory complications being the most obvious symptoms. Although, neurological manifestations are quite rarely reported in cases of COVID-19. In this report, we present the case of a 57-year-old male patient reported with complaints of acute progressive symmetric quadriparesis and recently recovered from COVID-19. Two weeks prior to hospitalization, the patient suffered from cough and fever. The reverse transcriptase-polymerase chain reaction test for COVID-19 infection was positive. Electrodiagnostic tests showed that the patient had acute motor and sensory axonal neuropathy variant of Guillain-Barré Syndrome (GBS). COVID-19 virus stimulates the inflammatory cells and as a result, creates immune-mediated processes. GBS is an immune-mediated disorder. It is not clear whether COVID-19 infection induces the production of antibodies against specific gangliosides. Further investigations should be conducted in regards to the mechanism of GBS in patients with COVID-19 in the future.

**Key words:** Autoimmune disorder, Coronavirus disease 2019, Guillain-Barré syndrome, Quadriparesis

The novel coronavirus-COVID-19 was detected in Wuhan city of Hubei province in China on December 31, 2019 [1]. COVID-19 is a beta-coronavirus that enters the cell by fusion with Angiotensin-Converting Enzyme (ACE-2) receptor [2]. Chen *et al.* reported that most of the severely infected patients suffer from pre-existing diseases, including hypertension, cardiovascular disease, and diabetes mellitus [3]. The most prevailing symptoms at the onset of the disease (incubation period - 5.2 days) are fever, cough, dyspnea, myalgia, and diarrhea [4]. Along with respiratory complications, coronavirus can affect multiorgan systems. In the second wave of COVID-19 in India, the majority of symptoms included gastrointestinal symptoms, acute cardiac damage, and acute renal failure. Mao *et al.* evaluated neurological symptoms in 214 hospitalized patients infected with COVID-19, of which 36.4% of them had nervous system manifestations including dizziness, hypogeusia, hyposmia, muscle damage, symmetric limb paralysis, and cerebrovascular accidents [5].


Guillain-Barré Syndrome (GBS) is an acute, immune-mediated disease of peripheral nerves and nerve roots (polyradiculopathy) elicited by various infections and characterized by progressive

symmetrical quadriparesis [6]. Interestingly, this rare disorder has been increasingly reported during the second wave in India. Hence, we present a case of neurological manifestation of COVID-19 sequelae-GBS, to provide an overview on its diagnosis and management in post-COVID-19 patients.

### CASE REPORT

A 57-year-old male patient was admitted to the emergency department with symptoms of acute progressive symmetrical quadriparesis for 5 days. Two weeks before hospitalization, the patient suffered from cough and fever. Reverse transcriptase-polymerase chain reaction test for COVID-19 infection came positive. Neurological manifestations of the patient were observed to begin with acute progressive weakness of distal lower extremities over a period of 5 days.

At the time of presentation, the symptoms progressed from distal limbs to proximal limbs, in both bilateral lower limbs and upper limbs, with muscle strength of 3/5. Additional symptoms included numbness, tingling, and paraesthesia of affected extremities. On arrival, the vitals were as follows: Temperature – 98.4°F, Blood Pressure – 136/86 mmHg, Pulse rate – 102/min, Respiratory rate – 28/min, and Blood oxygen saturation levels – 98% at 2 L/min O<sub>2</sub>. The next

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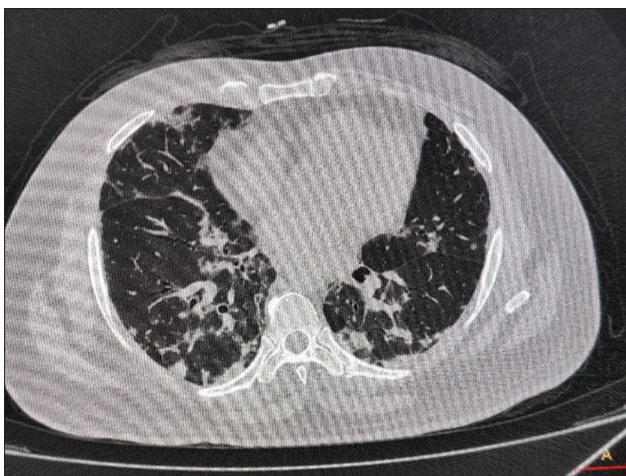
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day, there was a change in his examination findings, with diminished deep tendon reflexes when compared to the previous day. With the patient exhibiting ascending weakness with diminished deep tendon reflexes, cerebrospinal fluid analysis was planned but was not performed due to the lack of consent. On further neurological examination, he had no abnormalities at spinal sensory levels, no loss of bowel and bladder sphincteric reflexes, no meningeal irritation signs, and upper motor neuron disorders were negative. The laboratory examination findings were as follows: Serum glucose 150 mg/dL, blood urea nitrogen=18 mg/dl, serum creatinine=0.8 mg/dL, alanine aminotransferase=35 IU/L, aspartate aminotransferase=47 IU/L, sodium=135 mmol/L, and potassium=3.9 mmol/L. The blood investigations show white blood cells count of 4700 cells per microliter (neutrophil 82.7% and lymphocytes 10.4%), erythrocyte sedimentation rate of 72 mm/h, hemoglobin of 11.6 g/dL, and C-reactive protein of 2.2 mg/L. The presence of glucose and ketones in complete urine analysis was negative.

Lung computed tomography scan revealed bilateral ground-glass opacities (Fig. 1). Brain magnetic resonance imaging revealed normal findings. A neurophysiological study was performed. Electrodiagnostic parameters demonstrated decreased amplitude of compound muscle action potential and no response at sensory nerve action potential, suggestive of mixed sensorimotor neuropathy (Table 1). On the basis of available evidence (ascending paralysis, loss of deep tendon reflexes in weak limbs, and the results of electrophysiological studies), a final diagnosis of GBS was made according to Brighton criteria, with LEVEL-2 of certainty. Electrodiagnostic tests showed that patient had an acute motor and sensory axonal neuropathy variant of GBS.

The patient was initially hemodynamically stabilized and started on empirical intravenous antibiotics, IV fluids, antacids, and anticoagulants. After confirmation of the diagnosis,



**Figure 1:** High resolution computed tomography scan of chest showing bilateral ground glass opacities obtained during patient's COVID-19 illness, that is, 2 weeks prior admission. Source: Department of Radiology, Care Hospitals, Banjara Hills, Hyderabad

intravenous immunoglobulin (IVIG) was administered at a dose of 0.4 g/kg/day, for a total duration of 5 days, as per hospital protocol of GBS management. The patient showed significant symptomatic improvement within 1 week of IVIG therapy. The patient underwent physiotherapy sessions. Improvement in power and tone was noticed. The patient was discharged with regular follow-up advice.

## DISCUSSION

The classic clinical manifestations of GBS are progressive, ascending, symmetric, flaccid limbs paralysis, along with areflexia or hyporeflexia, with or without cranial nerve involvement, which can progress over the course of days to several weeks [6]. Two-thirds of patients usually report respiratory tract or gastrointestinal infections, 2–4 weeks before the onset of neurological symptoms of GBS [7]. In this case report, we reported GBS in a patient with a COVID-19 infection.

COVID-19 is a beta-coronavirus akin to severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS), such that fever, cough, and difficulty in breathing are the first symptoms that are commonly reported in infected patients with those viruses [7]. Neurological manifestations associated with COVID-19 infection have been reported by Mao *et al.* study [8]. The neurological manifestations have also been reported in other beta-coronaviruses (SARS and MERS), including polyneuropathy, myopathy, stroke, and GBS [9]. To the best of our knowledge, neuropathy and GBS have not been widely reported in association with COVID-19 infections in India. This patient had typical symptoms of GBS, which presented approximately 2 weeks succeeding the respiratory tract infection with COVID-19. A provisional diagnosis of GBS was made on the basis of preceding infection history and relative symmetric limb weakness with a monophasic course, which was further confirmed with electrophysiological studies.

Studies on coronaviruses have shown that these viruses have neurological and neuro-invasive characteristics [10]. Both SARS and COVID-19 attack the ACE-2 receptors [4,11] This receptor is detected in the cell membrane of human organs including lungs, kidneys, liver, nervous system, and skeletal muscles. The mechanism of GBS formation in patients with COVID-19 has not yet been investigated. COVID-19 stimulates inflammatory cells and produces various inflammatory cytokines and as a result, it creates further immune-mediated processes [4]. GBS is an immune-mediated disorder, and the mechanism of molecular mimicry has an important role in creating this autoimmune disorder.

It is unclear whether COVID-19 infection induces the production of antibodies against specific gangliosides that usually appear with certain forms of GBS. Further investigations should be conducted about the mechanism of GBS in patients with COVID-19 in the future.

Table 1: Nerve conduction study parameters in the patient with GBS

Nerve Stimulated	Stimulation Site	*Amplitude		Latency (ms)		Conduction velocity		F wave	
		RT	LT	RT	LT	RT	LT	RT	LT
Median (s)	Wrist	NR	NR	NR	NR	NR	NR		
Ulnar (s)	Wrist	NR	NR	NR	NR	NR	NR		
Sural (s)	Calf	NR	4.4	NR	2.01	NR	34		
Median (m)	Wrist	2.2	1.9	6.70	7.58			48	51
	AF	1.8	1.4	13.71	15.1	32	42.1		
Ulnar (m)	Wrist	4.6	4.31	4.20	4.7			42	48
	BE	4.5	3.42	8.32	8.61				
	AE	4.4	3.5	9.80	10.10	45	38		
Tibial (m)	Ankle	1.10	0.3	9.90	12.25			NR	NR
	Popliteal F.	0.91	0.3	20.65	27.02	30	22		
Peroneal (m)	Ankle	NR	NR	NR	NR	NR	NR		

\*Amplitude motor=mV, Sensory=mV, m=Motor study, s=Sensory study, RT: Right; LT: Left, AF: Antecubital fossa, BE: Below elbow, AE: Above elbow, BF: Below fibula, LP: Lateral popliteal fossa, NR: No response, GBS: Guillain-Barre syndrome

## CONCLUSION

In summary, to the best of our knowledge, this is among the various reported cases of GBS in a patient infected with COVID-19, given that most common symptoms of COVID-19 infection reported and two-thirds of GB patients usually mention the findings of preceding respiratory tract infection.

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## AUTHOR CONTRIBUTIONS

Dr. Anil Kumar Behera - Concept and design of study, revision, and final approval of the version. Dr. Hassan - Drafting the article, data analysis, and revision of the article. <sup>2</sup>Dr. Chaitanya Challa - Final approval of the version. Dr. Divya - Drafting the article and data analysis. Dr. Madhusudhan Reddy - Interpretation of data. Dr. Bharat - Revision of article for intellectual content. Dr. Bharadwaj - Design of study and concept. Dr. Athallah - Acquisition of data. Dr. Manaswini - Acquisition of data.

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