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Case Report

Guillain-Barre Syndrome: A Rare Complication of Covid-19

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ABSTRACT

Background:

Guillain-Barré syndrome (GBS) is an acute, frequently severe, and fulminant polyradiculoneuropathy that is auto immune in nature and often related to a previous infectious exposure. GBS emerged as a potentially serious complication of corona virus disease 2019 (COVID-19) ever since its declaration as a global pandemic. Recently, there have been many case reports describing the association between COVID-19 and GBS; but much remains unknown about the strength of this association and the features of GBS in this setting. We report the first case of GBS in a patient of covid-19 from Mahatma Gandhi Hospital, Bhilwara.

Case Presentation:

A 55-year-old female presented with fever, cough and shortness of breath; three weeks before the onset of acute progressive and ascending lower limbs weakness. She was admitted in Mahatma Gandhi Hospital, Bhilwara. She developed these symptoms with HRCT-15/25 and RT PCR Covid-19 positive. Electrophysiological studies showed acute motor axonal polyneuropathy (lower limbs & upper limbs). She was treated with intravenous immunoglobulin (IVIG).

Conclusions:

Physicians and neurologists should be aware of GBS as a rare but serious complication associated with COVID-19. Diagnosis is challenging and can be delayed, especially in asymptomatic patients or those with mild upper respiratory infection weeks earlier. Early diagnosis and management can improve clinical outcome.

KEYWORDS: Acute inflammatory demyelinating polyneuropathy, AIDP, Covid-19, IVIG, RT PCR

BACKGROUND

The first unexplained pneumonia cases occurred in Wuhan, China, and quickly spread to other countries. It was later revealed that these unexplained pneumonia cases had been caused by a new coronavirus. It has been stated that the symptoms of this new coronavirus infection are very similar to those of SARS-CoV which spread in 2003[1]. Both act on the same receptor, namely the angiotensin-converting enzyme 2. This virus is called SARS-CoV-2 and has been called by the WHO as the coronavirus disease 2019 (COVID-19)[2]. COVID-19, like SARS-CoV and MERS-CoV, affects the nervous system. The neurological manifestations of the COVID-19 infection are due to its effects on the central nervous system (CNS)[3], peripheral nervous system (PNS) and skeletal muscles.

Guillain Barre Syndrome (GBS) is an inflammatory disease of the PNS, characterized by rapidly progressive, symmetrical, and typically ascending weakness of the limbs with reduced or absent deep tendon reflexes, and paresthesia and sensory symptoms at the onset. Cranial nerves involvement can also be present in GBS patients, with facial and bulbar muscles often being affected[4]. GBS can be classified into different distinct clinical variants including classical sensorimotor, paraparetic, pure motor, pure sensory, Miller Fisher syndrome (MFS), pharyngeal-cervical-brachial variant (PCB), bilateral facial palsy with paresthesia, and Bickerstaff brainstem encephalitis[5]. Another classification of GBS based on the electromyography (EMG) findings has also been described, with acute inflammatory demyelinating polyneuropathy (AIDP) being the most common variant. Other EMG variants of GBS according to this classification include acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN)[6].

Participation of different viral infections, such as influenza virus, cytomegalovirus (CMV), Epstein–Barr virus (EBV), Zika virus, and MERS virus (Middle East respiratory syndrome), has been considered in the pathogenesis of GBS[7]. The incidence of GBS may increase during outbreaks/ pandemics of infectious diseases, as was previously observed during the Zika virus epidemic, in French Polynesia, in 2013.

CASE PRESENTATION

A 55-year-old female with known case of hypertension and CAD, was admitted in Medicine Department with fever, dry cough and shortness of breath since 4 days. She was conscious, oriented and hemodynamically stable and was suffering from Covid-19, confirmed by Covid-19 RT PCR and HRCT. Her Covid Score was 15/25 and Inflammatory Markers were CRP 38 mg/l, ESR 57 mm/hr, IL-6 167 pg/ml, ferritin 761 ng/ml, D-dimer 2090 ng FEU/ml, LDH 700 U/L. She was treated with Remdesivir, Ivermectin, Doxycycline, Enoxaparine and Oxygen by NRBM. Fever with chills and rigors, was low grade, intermittent type, subsided but oxygen requirement was gradually increasing.

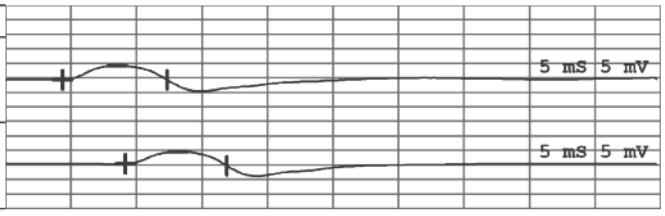
After three weeks of onset of symptoms, she developed paraplegia. Based on neurological examination, patient was conscious and oriented and her Glasgow Coma Score was E4V5M6. The Power of bilateral upper extremities was 5/5 and the power of bilateral lower extremities was 2/5. Tone of bilateral upper extremities was normal but bilateral lower extremities decreased and deep tendon reflex of knee, ankle and planter was absent. Sensory examination was within normal limits. After two days of onset of paraplegia, Nerve conduction study was conducted which was suggestive of predominantly motor axonal polyneuropathy involving (lower limbs > upper limbs) (below figures).

Motor Nerve Studies

Upper Limb

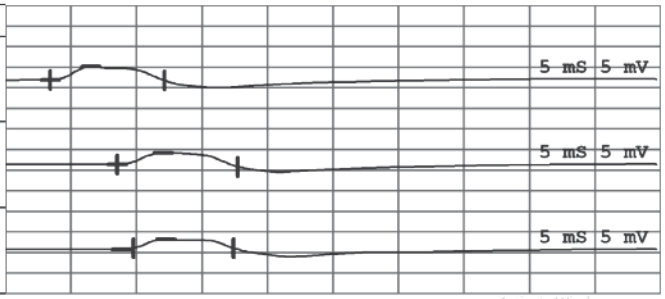
Nerve:Lt- Median

Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Wrist	4.38	8.02	4.7 mV	
Elbow	9.17	7.81	4.3 mV	48.02



Nerve:Lt- Ulnar

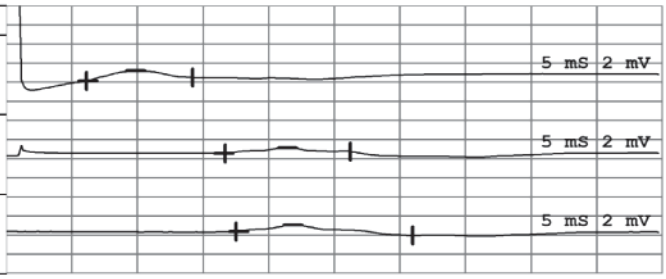
Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Wrist	3.44	8.75	3.2 mV	
BE	8.54	9.27	2.8 mV	37.25
AE	9.79	7.71	2.5 mV	72.00



Lower Limb

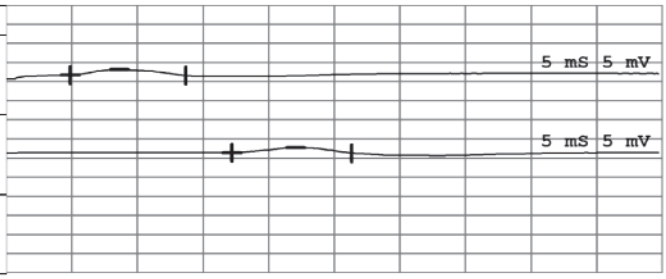
Nerve:Rt- Peroneal

Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Ankle	6.15	8.13	1.1 mV	
B-K	16.77	9.58	0.6 mV	30.13
A-K	17.60	13.54	0.7 mV	96.39



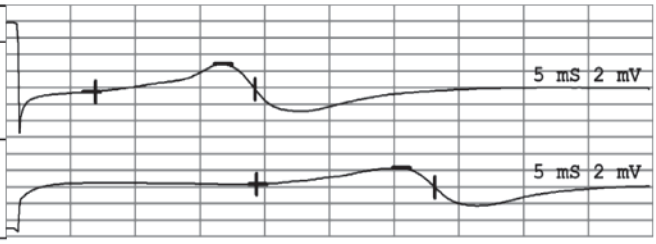
Nerve:Lt- Peroneal

Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Ankle	4.90	8.85	1.4 mV	
B-K	17.29	9.17	1.3 mV	33.09
A-K				



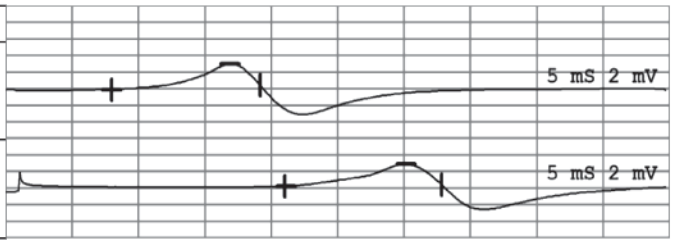
Nerve:Rt- Tibial

Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Ankle	6.98	12.40	3.3 mV	
Knee	19.48	13.85	2.0 mV	30.40



Nerve:Lt- Tibial

Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Ankle	8.02	11.25	3.1 mV	
Knee	21.15	11.88	2.7 mV	28.94

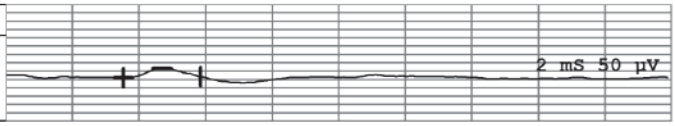


Sensory Nerve Studies

Upper Limb

Nerve:Lt- Median

Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Wrist	3.54	2.33	55.5 µV	45.20



Nerve:Lt- Ulnar

Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Wrist	2.83	2.33	31.4 µV	49.47

Lower Limb

Nerve:Rt- Sural

Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Mid Calf	4.54	1.83	17.5 µV	33.04

Nerve:Lt- Sural

Site	Lat1(mS)	Dur(mS)	Amp	NCV(m/S)
Mid Calf	3.63	1.00	15.4 µV	41.44

She required ventilator support. After four days of onset of paraplegia, IVIG 2 gm/kg was administered for 5 days. On fifth day of paraplegia, CSF study report show clear appearance, 00 cell/cumm, sugar 86 mg/dl and protein 21 mg/dl. HRCT score increased to 23/25 after one month of onset of symptoms. Weakness was noted in bilateral upper extremities (power 4/5) after ten days of onset of paraplegia. Right pneumothorax had developed during mechanical ventilation and was managed with ICTD. Power of upper extremities gradually decreased to 3/5 within twenty days of onset of paraplegia. She died due to both severe grade Covid-19 and its complication like GBS.

DISCUSSION

In this presented case, the patient developed paraplegia after three weeks of COVID-19 infection confirmed by RT PCR and finally diagnosed as a classic form of GBS. Abdullahi *et al.* conducted a systematic review of 83 cases of GBS patients after SARS-CoV-2 infection, which revealed that GBS had preceded COVID-19 in only two cases. Therefore, attempt should be made to the exclude COVID-19 infection in any patient with GBS during the COVID-19 pandemic[8]. In the present case, the diagnosis of COVID-19 does not raise any doubts. The disease was confirmed with COVID-19 RT PCR and HRCT thorax with inflammatory markers. The symptoms of COVID-19 were not completely subsided till she developed GBS in which the ENG study revealed features of mixed demyelinating and axonal polyneuropathy. In the study by Abu-Rumeileh *et al.*, which comprised of an analysis of 73 cases, it was found that the classic form of GBS, with sensorimotor presentation and acute inflammatory demyelinating polyneuropathy, was the most frequently described variant (51/73, 70% of cases)9. GBS symptoms developed on an average of 14 days from the onset of symptoms of COVID-19 infection[9]. In the present case this

time was 21 days. Usually patients experience fever, cough, and other symptoms of respiratory tract infections, such as dyspnea.

In previous publications, the results of the nerve conduction study were most often typical for the demyelinating form of GBS, and it was found in 77.4% (48/62) of patients, whereas 14.5% (9/62) of cases were diagnosed with the axonal subtype of GBS, and 8.1% (5/62) with the mixed subtype[9]. In the presented case, the ENG study revealed features of mixed demyelinating and axonal polyneuropathy. Existing CSF case studies have displayed albumin–cytologic dissociation (ACD). In published case reports, 71.2% (42/59) of the cases reported ACD with a mean protein concentration of 100 mg/dL (in our study, it was 21.2 mg/dl). The most common findings in biochemical and serological tests were leukopenia and an increased level of C-reactive protein (CRP)[9]. In the presented case, CRP was increased to 37.8 mg/dl (laboratory reference range: <5 mg/dl); no leukopenia was found.

From the cases reported in the literature, the majority of patients (85.7%; 60/70) were treated with IVIG or TPE, and glucocorticosteroids (GCS). It should be noted that GCS are not recommended as a therapeutic option for GBS. Mechanical ventilation or non-invasive respiratory support was required in 21.4% (15/70) of patients[9]. A total of 72.1% (49/68) of patients with post-COVID-19 GBS achieved a partial or complete remission, whereas 5% (4/68) died9. In the study by Aladawi *et al.*, which reviewed 109 cases of post-COVID-19 GBS, 40/99 patients required intensive care treatment, 33.3% (33/99) required mechanical ventilation, and 6.1% (6/99) of patients' died[10]. A worse course of GBS was observed in older patients. Moreover, in the relatively young age group, the mild course of COVID-19, and the prompt treatment could positively affect the course of GBS. Aladawi et al. described classic sensorimotor form (64/99; 64.5%) in NCS AIDP (59/77, 76.6%) but in our case predominantly motor axonal polyneuropathy was seen. The presented case has many features in common with those described previously in the literature.

To date, most patients have been treated with IVIG, and in the presented case, we also administered IVIG. The patient showed response to treatment after IVIG, but death occurred due to severe grade of covid-19 with GBS and comorbidities like CAD and hypertension. A previous COVID-19 infection with severe respiratory symptoms favors a worse prognosis; and if associated with GBS, it increases the risk of respiratory failure and the need for mechanical ventilation due to possible weakening of the respiratory muscles. The available treatment options for GBS increase the chance of clinical improvement. In most of the cases described so far (approximately 2/3), post-COVID-19 GBS achieved a good result (GBS disability score of ≤2); a worse course was most often associated with old age[9]. However, long-term prognosis is still uncertain.

ABBREVIATIONS

GBS: Guillain-Barré syndrome, COVID-19: Corona virus disease 2019, IVIG: intravenous immunoglobulin, AIDP: Acute inflammatory demyelinating polyneuropathy, PNS: Peripheral nervous system, AMAN: Acute motor axonal neuropathy, AMSAN: Acute motor and sensory axonal neuropathy, CMV: Cytomegalovirus, EBV: Epstein–Barr virus, MERS: Middle East respiratory syndrome, ACD: Albumin–cytologic dissociation, GCS :Glucocorticosteroids

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AUTHOR CONTRIBUTIONS:

DS: conceived, designed and performed the study; first draft of the manuscript and illustrations; analysis

VS: Study Design,

JK: critical review of the manuscript.

The final manuscript was critically reviewed and approved by all authors. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE:

All procedures were performed in accordance with the ethical standards of the institutional and national research committee

CONFLICT OF INTEREST: None

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