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Original Research Paper

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GUILLAIN-BARRÉ SYNDROME FOLLOWING COVID-19: A NEWLY EMERGING COMPLICATION

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ABSTRACT

BACKGROUND: Severe acute respiratory syndrome coronavirus 2 (SARS-cov-2) is causing a worldwide pandemic of COVID-19. As case numbers increase, the body of research around SARS-cov-2 and the pathophysi- ology of the disease process it causes (COVID-19), expands daily. Patients with COVID-19 typically present with fever and respiratory illness; however, a wide range of other symptoms have been described. While the neurological sequelae of the virus remain poorly understood, there are a growing number of reports of neurological manifestations of COVID-19.A recent study from Wuhan reported that 78 (36.4%) out of 214 patients admitted with COVID-19 had neurological symptoms ranging from anosmia and taste disturbances to cerebrovas- cular to strokes and seizures.

Additionally, there is increasing recognition Of a link between COVID-19 and Guillain-Barré syndrome (GBS), with three international case reports and one case series of five patients are the only published cases to date. To help add to this small but developing body of evidence, this is the first published case of GBS secondary to COVID-19 in the North east India

KEYWORDS :

CASE PRESENTATION

On 10 December 2020, a 28-year-old, right-handed female was refered from a District Hospital to our tertiary health care centre with 1 week history of progressive limb weakness and foot dysaesthesia. On the evening prior to admission, in the District Hopital she noted that she had some difficulty standing in unaided and noticed some tingling sensations in her feet. The next morning, he was unable to stand, and her arms felt weakvand for which she was taken to district hospital Incidentally she also reported a 1 week history of cough and headache, myalgi and fever. bladder. She smoke, and drank alcohol occasionally. On Presentation to our institute, she was febrile, tachycardic (heart rate 110 bpm) and had bilateral crepitations to the mid-zones on lung auscultation. Her oxygen saturation was 88% on room air and respiratory rate was 28 breaths/min. Limb examination revealed reduced tone with symmetrical weakness of 4/5 on the Medical Research Council (MRC) Power Grading scale in upper and lower limbs (table 1). She had diminished tricep reflexes and absent bicep, supinator, knee and ankle reflexes bilaterally. Pinprick sensation was impaired to the right midfoot and left ankle, with. Cranial nerves were intact.

After 72 hours, her lower limb power had reduced to 3/5 proximally and 2/5 distally (table 1) and all limb reflexes were absent. The overall impression was of a progressive flaccid symmetrical sensory and motor neuropathy was considered initially

Table 1 Examination of motor function on admission and after 24 hours

	power (MRC grading)			
	on admission		After 72 hours	
	Right	Left	Right	Left
Upper limb				
Shoulder abduction	4	4	3	3
Elbow flexion	4	4	3	3
Elbow extension	4	4	3	3
Wrist extension	4	4	3	3
Wrist flexion	4	4	3	3
Finger extension	4	4	3	3
Finger flexion	4	4	3	3
Thumb abduction	4−	4−	3	3
Lower limb				
Hip flexion	4	4	3	3
Hip extension	4	4	3	3
Knee flexion	4	4	3	3
Knee extension	4+	4+	3	3
Dorsiflexion	3	3	2	2
Plantarflexion	3	3	2	2
Great toe dorsiflexion	3	3	1	1

Investigations

On admission, blood tests were significant, thrombo cythaemia (490×10^9/L) and a raised C reactive protein (25 mg/L). Renal profile, electrolyte, serum thyroid and clotting functions were all within the normal range. Antinuclear antibody, antineutrophil cytoplasmic antibodies, virus screen (HIV, hepatitis B and hepatitis C) were negative. Her chest X-ray on admission () showed no convincing consolidative change, infiltrates or ground-glass shadowing. Her SARS-CoV-2 (COVID-19) RNA nasopharyngeal swab was positive.

She was managed for covid-19 according to institute protocol She was found to be negative for COVID-19 on 7th day of admission in our institute and she was shifted non-covid ICU for futher management

A lumbar puncture was performed 9.days after admitting in our institute which revealed high cerebrospinal fluid (CSF) protein (73mg/dl) with normal glucose and cell counts. No organisms were found on gram staining

Nerve conduction studies were carried out on day 12 of admissions. F-wave latencies, when present, were measured from the onset of the compound motor action potential to the onset of the F-wave.

Nerve conduction studies revealed reduced conduction

velocity and prolonged distal motor latencies in motor and sensory nerves in the upper and lower limbs with more marked

Table 2 Motor and sensory nerve conduction studies

nerve	Latency (ms)	Amplitude (mV)	Conduction velocity (m/s)	F-wave latency	Comments
Motor nerve conduction studies					
Medianus motor right					
Wrist–APB	7.20	4.3			
Elbow–wrist	14.7	1.86	41.3	Absent	Dispersed potential
Ulnaris motor right					
Wrist–ADM	4.12	6.2		40.5	
Ab. elbow–wrist	12.0	2.9	44.4		Dispersed potential
Peroneus motor left					
Ankle–EDB	6.67	2.7		Absent	
Pop fossa–ankle	20.8	1.01	30.4		Dispersed potential
Peroneus motor right					
Ankle–EDB	7.23	2.6		Absent	
Pop fossa–ankle	25.7	0.23	22.3		Dispersed potential
Tibialis motor left					
Ankle–AH	10.4	1.68		Absent	
Knee–ankle	30.0	0.38	22.4		Dispersed potential
Tibialis motor right					
Ankle–AH	9.39	1.66		Absent	
Knee–ankle	24.2	0.77	29.7		Dispersed potential
Medianus sensory right					
Digit II–wrist	–	–	–		Absent sensory nerve action potential
Ulnaris sensory right					
Digit V–wrist	2.45	4.0	40.8		
Radialis sensory right					
Forearm–dorsum	1.64	30.8	54.9		
Suralis sensory left					
Calf–latmalleolus	2.47	12.4	44.5		
Suralis sensory right					
Calf–lat. malleolus	3.74	7.9	34.8		
Peroneus superfic sensory left					
Lower leg–dorsum	3.54	12.0	31.1		
Peroneus superficialis sensory right					
Lower leg–dorsum	3.84	7.3	28.6		

Treatment:

Intravenous immunoglobulin (IVIG) 0.4g/kg everyday for a period of 5 days was started on 15th day of admission in our institute(delayed due to problem in procuring the IvIg) Physiotherapy was initiated on 15th day of admission.

Outcome And Follow-up

On day 20th day of admission, the limb weakness started reducing and she improved substantially and weakness gradually improved and she was discharged on 32nd day of admission in our institute and she was kept in regular followup. Her weakness gradually improved and at the end of 6 weeks and she was able to walk with support

DISCUSSION

GBS is an autoimmune condition characterised by rapidly progressive limb weakness, often with sensory and cranial nerve deficits, and can result in significant morbidity and mortality. The syndrome is typically post-infectious, with two-thirds of adult patients reporting respiratory or gastrointestinal infections in the 6 weeks prior to presentation, which are thought to trigger an immune response leading to a neuropathy.[7] A number of well-recognised antecedent infections have been identified in case–control studies, including bacteria such as *Campylobacter jejuni* and viruses such as *Cytomegalovirus* and Epstein-Barr virus. In the case presented, the patient developed significant neurology only 2 week after the development of her cough and myalgia,. This raises the possibility of a parainfectious course, similar to GBS cases described in association with Zika virus.[8] While parainfectious neuropathies may develop as an unusual hyperimmune response, they could also represent a direct toxic or neuropathic effect. Further research is needed to differentiate between these two possibilities in COVID-19 patients.

The Italian series reported that 5 (0.42%) out of 1200 patients admitted to their hospitals with COVID-19 presented with GBS, which is disproportionately high for a rare disease that affects 1.6 per 100,000 person-years (matched for the average age of their cohort).[4 9] This case report is adding evidence to the increasing recognition that COVID-19 could be an infectious trigger for GBS. The interval between the onset of symptoms of COVID-19 and the first symptoms of GBS was approximately 7 days, and neurological symptoms evolved rapidly over 3 days. These time windows are in keeping with the Italian series.[5]

The clinical manifestations of GBS are varied, from mild limb weakness to respiratory muscle involvement requiring mechanical ventilation. Studies have found that the severity of GBS is associated with the causative organism, demonstrated by the higher rates of severe axonal forms following *C. jejuni* infection.[10] As such, it is important to further research the link between COVID-19 and GBS to help with diagnosis and prognostication. Of importance, half of the currently reported cases (4/8) have needed mechanical ventilation, higher than the recognised 20%–30% in all GBS cases. Despite the small sample size, this could represent an interaction between the COVID-19 pneumonitis and GBS increasing the likelihood of needing respi- ratory support. Alternatively, this may suggest that COVID-19 is a trigger for a more severe and rapidly progressing neuropathy.

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