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Original Research Article

Guillain barre syndrome and its association with covid-19 infection – A clinical case series

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ABSTRACT

Background: A novel coronavirus (COVID-19) pandemic is caused by severe acute respiratory syndrome coronavirus 2 (SARs-CoV-2). This pandemic has been globally alarming in the current period. Several neurological manifestations are reported occurring with the infection. Guillain barre syndrome (GBS) or acute onset inflammatory polyradiculoneuropathy has been among the frequent manifestations observed among them.
Objectives: To know the pattern and outcome of GBS in COVID-19 affected individuals.
Methods and Methods: We have taken six individuals admitted with flaccid quadriparesis in the last two months. All were affected recently by COVID 19 infection, which RT PCR of the nasopharyngeal swab confirmed. The study participants have undergone nerve conduction studies and have been diagnosed with Guillain Barre syndrome using Brighton criteria. We did cerebrospinal fluid (CSF) analysis after admission. We initiated all patients on Intravenous immunoglobulin according to body weight (2g/kg divided over five days). We used the Barthel index score to assess the outcome of the individuals.
Results: We observed a mean duration of 18.25 days between the COVID-19 infection and the onset of symptoms. Apart from motor quadriparesis and sensory symptoms being in common, we also noticed cranial nerves and autonomic involvement. We made the diagnosis using the nerve conduction studies and Brighton criteria. After initiating intravenous immunoglobulin, all patients had a good outcome, and quality of life was better after two months of follow up.
Conclusion: Guillain Barre syndrome is one of the neurological manifestations of COVID-19 and has a dramatic response with intravenous immunoglobulin and better outcome with treatment.

1. Introduction

Novel coronavirus disease 2019 (COVID-19) is caused by severe acute respiratory syndrome coronavirus 2 (SARs-CoV-2). It has been one of the major concerning ongoing pandemics worldwide. Multiple systemic involvements apart from the respiratory system are seen with the novel virus. The nervous system has been reported to be frequently involved.[1] Both the central and peripheral nervous systems were known to be affected by the novel coronavirus.[2] The varied presentations include hyposmia, hypogeusia, encephalitis, meningitis, stroke and myopathy. Guillain barre syndrome (GBS) is an autoimmune demyelinating polyradiculoneuropathy characterised by ascending motor weakness and areflexia.[3] Recent infections or vaccinations usually trigger it. Here we report a series of cases affected with GBS following COVID-19 infection. We want to highlight the association of both and their response and outcome to the treatment through this report.

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2. Subjects and Results

2.1. Case 1

A 64-year-old female presented to us on October 24^{th} 2020, with a history of difficulty walking for ten days. Recently, she had undergone treatment for SARs-CoV-2 pneumonia on September 22^{nd} 2020. She had a computed tomography (CT) CO-RADS [coronavirus disease 2019 Reporting and Data System] score of five and a severity score of 25/25. The Nasopharyngeal swab - real-time Polymerase Chain Reaction (RT-PCR) was positive done on September 22^{nd} was positive. She was a known case of diabetes mellitus and hypothyroidism for ten years on regular medication. Clinically, she was moderately built and nourished. Her blood pressure was 100/60mm of Hg in both upper limbs with no postural drop. She had neck and truncal weakness. She had quadriparesis (lower limbs – 2/5 more than upper limbs – 3/5), proximal being involved more than distal, with absent deep tendon reflexes and bilateral plantar withdrawal. Sensory and rest of the examination were normal. Her single breath count was ten.

Her complete blood picture was hypochromic microcytic anaemia (haemoglobin – 7.7g/dl) with normal counts (9300 per cubic millimetre) and platelet count (209000 per microlitre). Metabolic profile showed normal electrolytes (Sodium – 137, Potassium – 3.96 mEq/L; Magnesium – 1.63mEq/L; Calcium – 1.07mmol/L). Creatine phosphokinase levels were 214 U/L. Her thyroid-stimulating hormone levels were 5.62 micro IU/ml. Liver functions showed normal enzymes with hypo-albuminemia (A: G reversal) [albumin – 2.4g/dl; total protein – 4.8g/dl]. Urine complete showed proteinuria. Nerve conduction studies showed reduced motor amplitudes with normal distal latencies in the bilateral median, ulnar nerves and inelicitable in bilateral peroneal and tibial nerves. H reflex was absent, and conduction velocities were normal in the tested nerves. F-wave latencies were prolonged in the peroneal and tibial nerves. Sensory stimulation showed inelicitable sural nerves. (Table 1 A) Cerebrospinal fluid analysis was normal (cell count – 2; Protein – 17.8mg/dl; Glucose – 112mg/dl). She was diagnosed with Guillain Barre syndrome and treated with intravenous immunoglobulin of 140gm over five days. She received limb physiotherapy and rehabilitative measures. She was able to walk with two-person support at the time of discharge. After two months of follow up, she was able to walk without or with minimal support with a modified Barthel index of 65.

2.2. Case 2

On October 08th 2020, a 68-year-old female presented to us with difficulty walking for two weeks and fever with burning micturition for one week. She was recently treated for SARs-CoV-2 pneumonia in August 2020. She had a CO-RADS score of five with a CT severity score of 16/25. She was discharged and was able to mobilise well till two weeks back. Her past revealed she is a known case of diabetes mellitus for three years with poor drug compliance. At the time of presentation, she was conscious, alert, oriented and had bilateral pitting type of pedal oedema up to the knee. Her pulse rate was 110 beats per minute, and her blood pressure was 120/70 mm of Hg in bilateral upper limbs with no postural drop. Her respiratory rate was 23 per minute, and oxygen saturation was 97 per cent at room air. Neurological examination showed mild neck flexion weakness flaccid quadriparesis (lower limbs being more affected with a power of 2/5 than the upper limbs, with a power of 3/5), and proximal weakness was more than distal. Deep tendon reflexes were absent on both sides, and bilateral plantar reflex was flexor. The sensory system, cranial nerves and rest of the examination were within normal limits.

Her complete blood picture revealed a neutrophil predominant leucocytosis with thrombocytopenia (Haemoglobin - 13.6, Total counts - 32500, platelet count 119000/microlitre). Urinalysis showed the presence of leukocytes (urine cell count of 232cells) and proteinuria. Renal function tests revealed mild acute kidney injury (urea -101, creatinine-2.92). Liver function showed normal enzymes with hypoalbuminemia ((total protein-6.0, serum albumin-1.4, serum globulin-4.6) and reversal of albumin: globulin ratio (A: G reversal). Her electrolytes - sodium-130, potassium-4.5, chloride-103, bicarbonate-13.6, ionised calcium-1.007). Serum procalcitonin was elevated (serum procalcitonin - 2.06). Magnetic Resonance Imaging (MRI) of the brain and spine showed normal parenchyma with no significant cord changes. Nerve conduction studies of all four limbs – motor conduction studies showed normal distal latencies with reduced motor amplitudes in upper limb [bilateral median, ulnar, radial nerves] and inelicitable response in bilateral peroneal and tibial nerves. F wave latencies were prolonged in peroneal and tibial nerves and absent H-reflex. Sensory nerve conductions showed unrecordable bilateral sural nerves being non-recordable, with the rest of the tested nerves were normal. (Table 1B). She was diagnosed with Guillain Barre syndrome and started on intravenous immunoglobulin after obtaining patient and family's consent. The patient was treated with intravenous immunoglobulin (2 gram per kilogram) a total of 200 grams was given over five days. The patient's power had improved in both upper limbs and lower limbs and mobilised to the chair at the time of discharge. After two months of follow up, she was able to walk with minimal support – attributing secondary to osteoarthritis of both knees with her Barthel index of 65 on follow up.

2.3. Case 3

Here we describe a 41-year-old male presented with weakness of all four limbs for three days since November

Table 1: Nerve conduction studies (NCS) of Case 1 and Case 2.

	A. **Case 1 NCS**			B. **Case 2 NCS**		
Motor Nerve conduction study						
Nerve	**Latency (ms)**	**Amplitude (mV)**	**CV (m/s)**	**Latency (ms)**	**Amplitude (mV)**	**CV (m/s)**
Left median Wrist Elbow	3.0 9.12	2.2 2.4	42.3	3.6 9.1	4.6 2.0	39.0
Right median Wrist Elbow	3.25 8.12	2.6 2.2	52.9	4.3 10.0	2.2 1.7	38.0
Left Ulnar Wrist Below elbow	2.25 7.25	8.0 14.5	55.8	2.4 7.8	3.3 2.1	42.0
Right ulnar Wrist Below Elbow	2.75 7.12	7.37 12.00	62.7	3.3 8.7	2.1 1.5	38.0
Left peroneal Ankle Head of fibula	NR NR	NR NR	NR	NR NR	NR NR	NR
Rightperoneal Ankle Head of fibula	NR NR	NR NR	NR	NR NR	NR NR	NR
Left tibial Ankle Popliteal fossa	NR NR	NR NR	NR	NR NR	NR NR	NR
Right tibial Ankle Popliteal fossa	NR NR	NR NR	NR	NR NR	NR NR	NR
F waves		**Latency (ms)**			**Latency (ms)**	
Left median		31.4			32.7	
Right median		32.4			31.4	
Left ulnar		22			**NR**	
Right ulnar		33.9			33.3	
Left peroneal		**NR**			**NR**	
Rightperoneal		**NR**			**NR**	
Left tibial		**NR**			**NR**	
Right tibial		**NR**			**NR**	
		Sensory nerve conduction study				
Sensory nerves	**Peak Latency**	**Amplitude**	**CV**	**Peak Latency**	**Amplitude**	**CV**
Right median	3.15	10.8	53.5	5.2	25	46
Left median	3.15	9.5	54.9	4.1	15	56
Right ulnar	2.30	9.4	50.4	4.6	16	52
Left ulnar	2.25	8.9	58.3	4.1	28	55
Right sural	**NR**	**NR**	**NR**	NR	**NR**	NR
Left sural	**NR**	**NR**	**NR**	**NR**	**NR**	**NR**

NCS – Nerve Conduction studies; ms – millisecond; mV – millivolt; m/s – metre/second; CV – conduction velocity; NR – not recordable.

01^{st} 2020. He had recent COVID-19 infection two weeks back – his real-time Polymerase Chain Reaction (RT-PCR) of the nasopharyngeal swab was positive, and his CT chest showed a CORADS score of four. On examination, his BP was 130/90 mm Hg in bilateral upper limbs in the supine position with no postural drop, and pulse rate (PR) was 112 beats per minute with normal body temperature. He had a bilateral facial weakness with neck muscle weakness (neck flexion and extension – weak). He had flaccid quadriparesis (lower limbs – 2/5; upper limb – 2/5) with absent deep tendon reflexes with bilateral plantar flexor. The rest of the examination was normal. His single breath count was twelve. During the hospital stay, he had autonomic dysfunction like tachycardia, profuse sweating and paralytic ileus.

Laboratory investigations showed normal electrolytes (Sodium – 137mEq/L, Potassium – 4.23mEq/L, Calcium – 4.5mg/dl, Magnesium – 1.8mg/dl). Creatine phosphokinase levels were normal (78U/L). Nerve conduction studies (Table 3) showed that motor conduction studies showed reduced amplitudes and conduction velocities with normal distal latencies in bilateral median, ulnar and radial nerves and inelicitable in peroneal and tibial nerves. H-reflex was absent. F wave latencies and SNAPS were absent in tested nerves. (Table 2 A). Cerebrospinal fluid analysis showed albumin-cytological dissociation (protein – 407.8mg/dl; sugar – 117mg/dl, total cell count – 10cells/cubic mm). According to Brighton criteria – with clinical and electrophysiological aspects, we diagnosed him with Guillain Barre syndrome. We initiated him with intravenous immunoglobulin (IVIg) according to the weight of 150grams over five days. He received limb and respiratory physiotherapy. He was able to walk with two-person support at the time of discharge. After two months of

Table 2: Nerve conduction studies of Case 3 and Case 4.

	C. Case 3 NCS			D. Case 4 NCS		
Motor Nerve conduction study						
Nerve	**Latency (ms)**	**Amplitude (mV)**	**CV (m/s)**	**Latency (ms)**	**Amplitude (mV)**	**CV (m/s)**
Left median						
Wrist	6.14	3.1	36.4	3.62	18.3	52.5
Elbow	13.44	2.3		8.75	16.5	
Right median						
Wrist	6.88	0.9	38.9	4.12	14.3	53.4
Elbow	13.75	0.6		8.12	13.3	
Left Ulnar						
Wrist	4.06	4.0	44.8	2.25	15.6	67.0
Below elbow	10.21	3.3		6.50	14.7	
Right ulnar						
Wrist	5.42	3.9	48.6	3.12	10.8	72.0
Below Elbow	11.56	2.5		7.12	9.7	
Left peroneal						
Ankle	5.21	0.8	33.8	5.87	5.2	56.0
Head of fibula	16.56	0.5		12.75	3.5	
Right peroneal						
Ankle	4.90	1.3	39.2	3.75	1	51.8
Head of fibula	14.58	0.6		11.25	0.3	
Left tibial						
Ankle	8.23	0.2	33.0	4.87	12	45.1
Popliteal fossa	20.52	0.3		14.12	4.9	
Right tibial						
Ankle	9.48	0.5	35.1	4.12	9.5	48.1
Popliteal fossa	21.15	17.1		12.75	3	
F waves		**Latency (ms)**			**Latency (ms)**	
Left median		36.4			32.7	
Right median		NR			31.4	
Left ulnar		NR			NR	
Right ulnar		NR			33.3	
Left peroneal		NR			NR	
Right peroneal		NR			NR	
Left tibial		NR			NR	
Right tibial		NR			NR	
		Sensory nerve conduction study				
Sensory nerves	**Peak Latey**	**Amplitude**	**CV**	**Peak Latency**	**Amplitude**	**CV**
Right median	NR	NR	NR			
Left median	NR	NR	NR			
Right ulnar	NR	NR	NR			
Left ulnar	NR	NR	NR			
Right sural	NR	NR	NR	NR	NR	NR
Left sural	NR	NR	NR	NR	NR	NR

NCS – Nerve Conduction studies; ms –millisecond; mV – millivolt; m/s – metre/second; CV – conduction velocity; NR –not recordable

Table 3: Nerve conduction studies of Case 5 and Case 6.

	Case 5 NCS			Case 6 NCS		
Motor Nerve conduction study						
Nerve	**Latency (ms)**	**Amplitude (mV)**	**CV (m/s)**	**Latency (ms)**	**Amplitude (mV)**	**CV (m/s)**
Right median						
Wrist	5.63	6.0	46.0	3.54	2.3	46.73
Elbow	10.63	5.4		8.44	2.2	
Left median						
Wrist	5.94	8.0	48.0	4.69	2.6	55.91
Elbow	10.73	6.3		8.75	1.9	
Left Ulnar						
Wrist	3.85	7.6	51.2	1.25	4.0	30.8
Below elbow	8.54	4.5		9.27	3.1	
Right ulnar						
Wrist	3.33	8.8	51.2	2.40	5.0	47.9
Below Elbow	8.02	7.5		7.29	4.1	
Left peroneal						
Ankle	6.46	1.0	32.0	4.06	1.9	36.1
Head of fibula	15.21	0.6		13.44	1.3	
Right peroneal						
Ankle	6.15	3.1	36.0	3.85	1.3	36.9
Head of fibula	13.65	2.0		13.02	1.1	
Left tibial						
Ankle	6.88	2.0	36.1	5.21	4.9	32.7
Popliteal fossa	17.40	1.8		16.35	3.5	
Right tibial						
Ankle	6.35	3.6	41.0	4.79	4.1	35.0
Popliteal fossa	15.63	2.5		15.83	2.6	
F waves		**Latency (ms)**			Latency (ms)	
Left median		36.7			25.4	
Right median		35.8			26.8	
Left ulnar		36.3			35.4	
Right ulnar		31.5			30.6	
Left peroneal		NR			25.7	
Right peroneal		NR			26.6	
Left tibial		70.8			66.4	
Right tibial		66.8			69.1	
			Sensory nerve conduction study			
Sensory nerves	**Peak Latency**	**Amplitude**	**CV**	**Peak Latency**	**Amplitude**	**CV**
Right median	4.54	11.2	30.8	3.75	12.3	41.1
Left median	4.46	8.2	31.4	3.08	21.4	50.3
Right ulnar	2.5	23.4	44.0	2.5	5.0	53.2
Left ulnar	2.58	15.6	42.6	2.33	14.3	57.5
Right sural	3.88	8.9	41.2	2.88	6.9	43.4
Left sural	4.04	7.0	37.1	2.67	3.3	45.7

NCS – Nerve Conduction studies; ms –millisecond; mV – millivolt; m/s – metre/second; CV – conduction velocity; NR –not recordable

Table 4: Summary of the cases studied and their outcome

Case Number / Age / sex	Day of Onset of symptoms after testing positive for COVID	Comorbidities	NCS	MRI brain and Spine	Treatment /Recovery
1 / 64 / F	21	Diabetes mellitus, Hypothyroidism, Anaemia of Chronic disease	AMAN	Not done	IVIg / Good Recovery
2 / 68 / F	28	Diabetes mellitus	AMAN	Normal	IVIg / Good Recovery
3 / 41 /M	14	None	AIDP	Not done	IVIg / Good Recovery
4 / 35 / M	10	None	AMAN	Normal	IVIg / Good Recovery
5 / 50 / M	10	None	AIDP	Normal	IVIg / Good recovery
6 / 72 / F	10	Diabetes mellitus, Systemic hypertension	AMSAN	Normal	IVIg / Good recovery

NCS – Nerve conduction studies; MRI – Magnetic Resonance Imaging; F – female; M- Male; COVID – Coronavirus Disease 2019; AMAN – Acute Motor Axonal Neuropathy; AIDP – Acute Inflammatory Demyelinating Polyradiculoneuropathy : AMSAN – Acute Motor-Sensory Axonal Neuropathy; IVIg – Intravenous immunoglobulin.

follow up, he was able to walk with minimal support with a modified Barthel index of 75.

2.4. Case 4

A 35-year-old male with no known comorbid illness in the past had presented with acute onset weakness of all four limbs with breathing difficulty for ten days. He initially consulted at a local hospital for the same – he had respiratory failure, was intubated and put on artificial ventilation. He had undergone a tracheostomy after two days and came to us for further management. He underwent treatment for COVID-19 pneumonia 10 days before the onset of the symptoms. Clinically he had neck muscle weakness on tracheostomy with minimal ventilatory support. He had bilateral facial palsy, with the rest of the cranial nerves being normal. He had quadriparesis (2/5 of all four limbs) with absent deep tendon reflexes and bilateral plantar mute. His sensory and rest of the examination was normal. An echocardiogram showed mild left ventricular dysfunction with elevated troponin T levels. MRI brain with the whole spine showed a tiny right cerebellar infarct with a normal spine.

Considering an acute onset myocardial infarction with stroke secondary to COVID 19 infection, we started him on a newer anti-coagulant (Dabigatran) and aspirin. Nerve conduction studies showed – motor conduction studies showed normal distal latencies with reduced amplitudes in the bilateral median, ulnar, radial, peroneal and tibial nerves. F wave latencies and SNAPs were normal in tested nerves with inelicitable H reflex (Table 2 B). He was treated with intravenous immunoglobulin of 130grams over five days. He had a hypersensitivity reaction to IVIg, which got resolved with antihistamines and steroids. He had good limb and respiratory muscle physiotherapy during the hospital stay. During two months of follow up, he was able to do his regular activities with a Barthel index of 80.

2.5. Case 5

Here, a 50-year-old male with no known comorbid illnesses presented to the emergency department (ED) with an acute onset of right-sided facial weakness for seven days, followed by walking difficulty for two days. Initially, he was evaluated elsewhere for the same. His brain imaging (MRI brain with magnetic resonance angiogram of intracranial vessels) done was normal. The nerve conduction studies (NCS) of all four limbs showed prolonged motor latencies and F wave latencies in the bilateral median, bilateral tibial and left ulnar nerves with absent F wave latencies in bilateral peroneal nerves. He had reduced motor amplitudes in bilateral tibial and left peroneal nerves, with normal SNAPs in tested nerves (Table 3 A). Findings were suggestive of demyelinating motor polyradiculoneuropathy. He was recently diagnosed with COVID -19 pneumonia with RT PCR positive on April 24^{th}, 2021, and HRCT lung showed a CORADS score of 2 and CT severity score of 6/25. He was treated for the same and discharged on April 29^{th}, 2021. Following a day later, he developed the presenting symptoms. Clinically, he had bifacial lower motor neuron type weakness with quadriparesis (limb power – lower limbs – 3/5; upper limb – 4+/5 with proximal more than distal involvement). He had absent deep tendon reflexes with bilateral plantar flexor. Neck muscle strength was normal, and the single breath count was 12. Blood investigations like serum electrolytes, creatinine phosphokinase and thyroid profile were normal. Echocardiogram was normal. He was treated with intravenous immunoglobulin (according to weight – 2g/kg – 150grams over five days). His limb power improved by the third day of immunoglobulin. He was able to walk without support by the third day of immunoglobulin. Lumbar puncture showed zero cells with normal protein and glucose. His CSF was negative for infections. He was discharged after ten days of treatment, able to walk without

support with mild facial weakness. After two months of discharge, he resumed his routine and has a Barthel index of 100.

2.6. Case 6

We had a 72-year-old female who presented with acute onset right-sided hemiparesis for ten days. Recently she was diagnosed positive for novel coronavirus (COVID 19) pneumonia on April 16^{th}, 2021, by RT PC. Her radiological evidence showed a CORADS score of five and a severity score of 12/25. She was tested negative after ten days of illness. Following a day later, she noticed the presenting symptoms. She was a known diabetic and hypertensive for three years with good compliance. Clinically she had quadriplegia with a power of left upper limb – 4/5, left lower limb of 3/5, right upper limb 3/5 and right lower limb of 2/5 with absent reflexes and bilateral plantar flexor. Her neck muscle was weak with no facial or bulbar weakness. The bowel and bladder were continent.

Laboratory investigations showed normal electrolytes (potassium – 3.5mEq/L; Sodium – 142mEq/L, Calcium – 1.192mmol/L, Magnesium – 1.24mEq/L). Creatine phosphokinase was normal. She had a complete blood picture (haemoglobin – 12.4gram/dl, total leucocyte counts – 9300/microlitre and platelet counts – 257000/microlitre) in normal limits. MRI brain plain with CSF flow studies showed no acute changes with mild prominence of ventricles, and MRI whole spine showed no significant cord changes. NCS after one week of treatment showed– reduced amplitudes with decreased conduction velocities in the bilateral median, peroneal and tibial nerves. Prolonged F wave latencies were noted in bilateral tibial nerves with normal SNAPs with inelicitable H reflex (Table 3 B). Lumbar puncture showed two cells with mildly elevated protein (50mg/dl) with normal glucose. We noted no evidence of any infections in CSF. She was diagnosed with Guillain Barre syndrome post-viral illness-related and initiated intravenous immunoglobulin (120grams over five days). After IVIg and adequate physiotherapy, she walked with two-person support and was discharged after ten days of hospital stay. After two months of follow up, she was walking without support, and her Barthel index was 75.

3. Discussion

Severe acute respiratory syndrome coronavirus (SARS-CoV-2), as the name suggested, it did not solely restrict to the respiratory system. It has been reportedly evident of other organ involvement like kidney, liver, heart, skin and brain.[4] The virus enters the nervous system by the haematogenous route or by the olfactory tract.[5] The virus has a propensity for angiotensin-converting enzyme receptors present on the endothelial cells of cerebral vessels, a possible viral entry. The novel virus can be responsible for causing varied neurological presentations. They include altered mental status, seizures, headache, neuropsychiatric manifestations, cerebrovascular events (Strokes, vasculitis, cortical venous thrombosis) and cranial neuropathies. Peripherally, myopathy and motor polyradiculoneuropathy are acknowledged to occur with the novel virus. Among the neurological manifestations, here we discussed polyradiculoneuropathy occurring in affected individuals secondary to coronavirus. The minimum duration we noticed was ten days, and the maximum was 28 days. We summarised the outcome of the cases in a table (Table 4).

We observed a mean duration of 15.5 days, which was higher than a case series reported by Kaveh Rahimi from Iran[6] and Nanda S et al. from New Delhi.[7] It was similar to a multicentric study done by Dhamne Megha C et al.[8] About one out of six persons (16.67%) had needed ventilator support, with everyone having an excellent outcome after treatment with immunoglobulin with no deaths reported. This occurrence of polyradiculoneuropathy in the infected individuals was probably due to the autoimmune process and molecular mimicry. Though no specific antibodies were identified in the underlying mechanisms, its excellent response to IVIg had suggested an underlying immune-inflammatory mechanism. We could not do the autoimmune profile in the above case series. Among them, two had multiple comorbid illnesses, and the other two had no comorbid illnesses. Treating patients with Guillain barre syndrome include plasma exchange or intravenous immunoglobulin. We observed that all the patients had shown a dramatic improvement after initiation of intravenous immunoglobulin irrespective of an underlying comorbid condition. This was different from the response noted with IVIg in Italy by Toscano G et al.[9] Though the response was similar to that observed with a study done by Finister J et al. in Vienna, they observed that outcome was also dependent on the underlying comorbidities.[10] We observed all variants had a good response with the treatment, similar to a case study reported by Lascano et al.[11] Here we observed adults, and a pediatric case has been reported in Bangalore by Das KY et al.[12] Only a few case reports have been reported globally regarding the association of GBS with the SARs-CoV 2 virus. They are from Italy,[13,14] Iran,[15] United States,[16] and the first being from China by Hua Zhao et al.[17] Guillain barre syndrome with COVID-19 infection has been observed in India, and it is under-reported so far. This case series is the first to be reported in our region.

4. Conclusion

Guillain barre syndrome can occur secondary to coronavirus infection. Irrespective of comorbid illness and severity of respiratory illness secondary to coronavirus, a surpassing response has been noted with the initiation of treatment.

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6. Conflict of Interest

The author declares no potential conflicts of interest with respect to research, authorship, and/or publication of this article.

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