Case Report

Getting paralysed after COVID: Guillain–Barre syndrome

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Abstract

Neurological involvement after coronavirus disease (COVID-19) pneumonias is common and occurs in almost one-third of the patients. The commonest neurological symptoms are ageusia, anosmia, headache, nausea, vomiting, dizziness, and myalgia. Guillain–Barre syndrome (GBS) is a rare manifestation of severe acute respiratory syndrome-coronavirus 2 (SARS-CoV-2) infection; whereas the common neurological manifestations of the SARS-CoV-2 infection occur with the onset of the respiratory symptoms and may be due to the direct invasion of the nervous system by the virus, GBS in COVID-19 follows a time lag of 1–4 weeks and may be attributable to the immune mechanism of molecular mimicry. Here we report a case of GBS in a patient of COVID-19 which occurred on the 22nd day after the onset of the disease. The patient recovered completely and went home walking.

Keywords: COVID-19, Guillain-Barre syndrome, SARS-CoV-2

Introduction

The COVID-19 pandemic, caused by the SARS-CoV-2 infection, started from Wuhan province of China and spread globally with almost no country remaining unaffected. It has myriad symptoms and signs, involving almost all the organ systems but predominantly affecting the respiratory system. Neurological involvement has been seen in almost one-third of patients with ageusia and anosmia as the most common specific manifestations. Other common neurological manifestations include headache, vomiting, nausea, dizziness, and disorders of consciousness.[1] All these symptoms are the consequences of direct invasion of the nervous system by the virus. Less common neurological manifestations such as acute cerebrovascular disease and impaired consciousness have also been seen in some cases.[1]

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Rare neurological manifestations like GBS as a complication of COVID-19 have also been reported from various parts of the world with almost 42 such cases being reported in the literature.[2] GBS in COVID-19 may be attributable to the secondary immune mechanism like antigen mimicry just like other bacterial and viral infections. However, it still cannot be conclusively stated that whether the neurological symptoms associated with SARS-CoV-2 are attributable to an abnormal immune response or direct injury by the virus.

Case History

An 84-year-old male with a medical history of adequately treated hypertension and hypothyroidism presented to the emergency department with complaints of fever, cough, and breathlessness for 5 days. Chest X-ray revealed bilateral pneumonia and throat swab RTPCR for COVID-19 was positive. The patient turned hypoxic and was admitted in the critical care unit where he received targeted therapy for COVID-19 in the form of high-flow nasal oxygen, ventilatory support, intravenous remdesivir, steroid, convalescent plasma and subcutaneous low molecular weight heparin (LMWH). He showed high values of serum ferritin,

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C- reactive protein (CRP), and IL-6 [Table 1]. He also received tocilizumab in view of the cytokine storm. He was shifted out of the CCU on day18 when the pneumonia resolved.

On 22nd day, patient developed bilateral lower limb weakness with power of 3/5 (according to the medical research council grading of muscle power (MRC) scale) at all joints. On the next day, patient developed both upper limb weakness with a power of 4/5 at all the joints [Table 2]. Plantars were mute and all deep tendon reflexes were absent. There was no sensory loss and no bladder or bowel involvement. MRI spine was normal. Cerebrospinal fluid examination revealed albuminocytological dissociation. Nerve conduction studies revealed reduced conduction velocities and prolonged distal latencies in motor nerves in both upper and lower limbs. There was impersistent F waves and decreased compound muscle action potential in both lower limbs. According to the diagnostic Brighton Collaboration Criteria, he was diagnosed as acute demyelinating polyradiculoneuropathy. He was treated with intravenous immunoglobulin (IvIg) 0.4 gm/kg/day for 5 days. He developed transient thrombocytopenia and leucopenia after IvIg which improved spontaneously. He improved with treatment and on discharge, went home walking without any support.

Discussion

GBS commonly follows many bacterial and viral infections like Epstein–Barr virus, campylobacter jejuni, cytomegalovirus, influenza A virus, haemophilus influenza, and mycoplasma pneumoniae. Previous types of coronavirus (SARS-CoV and MERS) and Zika virus have been associated with GBS as well. Neurological involvement is common in COVID-19, with ageusia and anosmia as the most common specific manifestations. Other neurological manifestations include headache, vomiting, nausea, dizziness, and disorders of consciousness.

There have been reports of several cases of GBS in COVID-19 but the exact prevalence is still not known. Most literature reported affected males over 50 years[3] and the incidence of GBS rises with age reflecting the fact that older age and male gender are risk factors for more severe COVID-19.[4] All major subtypes of GBS such as acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor sensory axonal neuropathy (AMSAN), and Miller Fisher syndrome (MFS) have been reported in COVID-19. However, most reports are of AIDP, like in our case.

In a review article of 33 cases of GBS in COVID-19 by Kaveh Rahimi,[5] the average duration for neurological symptoms following COVID-19 infection was 11.92 ± 6.20 days. The mean age of the patients was 57.26 ± 15.82 years, with the youngest being 5 years and the oldest being 84 years old. Our patient was 84 years old and the quadriparesis started on 22nd day after the infection. In another article by James B. Caress *et al.*[6] (37 cases), the mean age was 59 years, the male prevalence was 65% and the mean duration 11 days. Respiratory failure was present in almost 40% of GBS related to COVID-19 which is higher than that observed in non-COVID GBS cases (20–30%).[7] This suggests that both COVID-19 pneumonia and GBS-associated respiratory muscle weakness contribute to the respiratory failure. The physicians should always consider GBS in the differential diagnosis of a respiratory insufficiency in patients with COVID-19, especially when there is a discrepancy between chest imaging and respiratory parameters.[8] Our case also had respiratory failure and required ventilatory support.

GBS in COVID-19 occurs because of the post-infectious mechanism of molecular mimicry. This is supported by delayed onset quadriparesis after SARS-CoV-2 infection and the presence of autoantibodies that result from an immune response directed to an epitope of the infectious agent that cross-reacts with a structurally similar component of peripheral nerve, resulting in delayed immune-mediated damage to peripheral nerve.[9] SARS-CoV-2 attaches to cell surfaces by the viral spike (S) protein, which binds to angiotensin-converting enzyme 2 and to gangliosides containing sialic acid residues, including the GalNAc residue of GM1.[6] Cross-reactivity between the viral protein–associated gangliosides and peripheral nerve gangliosides may result out of molecular mimicry and subsequent nerve sheath destruction.

Table 1: Serial laboratory investigations

	Day 2	Day 6	Day 11	Day 15	Day 23	Day27	Day 28	Day 29	Day 33
Hb% (gm/dl)	13.4	12.9	12.8	12.2	11.1	9.9	9.6	10.3	10.2
TLC (per cumm)	9100	9200	8200	7400	4500	3300	2300	8900	4200
Neutrophils (per cumm)	91	90	90	96	56	68	57	81	69
Lymphocytes (per cumm)	6	9	8	11	36	21	32	12	18
NLR	15.17	10	11.25	8.73	2.39	3.24	1.79	6.77	3.84
Platelet count (per cumm)	13,4000	147,000	11,2000	108,000	10,6000	95,000	91,000	98,000	105,000
LDH (U/l)	395.6	755	535	433	368	276	368	302	288
CRP (mg/dl)	14.48	20.19	2.16	0.61	0.74	0.52	0.68	0.8	0.6
IL-6 (pg/ml)	56.44	68.10	111.48	96.77	136.15	124.45	136.15	73.24	61.61
Ferritin (ng/ml)	1215	1517	3272	2879	2185	1909	1902	1720	1687
Creatinine (mg/dl)	0.80	0.78	0.71	0.70	0.76	0.63	0.80	0.62	0.60
ALT (U/l)	170	119	116	68.30	63.20	45.1	442	59.6	45.1
AST (U/l)	138	56	43	35.60	46.20	38	33.1	51.70	33.10

TLC - Total leucocyte count; NLR - Neutrophil lymphocyte ratio; LDH- Lactate dehyrogenase; IL-6 - Interleukin 6; CRP - C reactive protein; ALT - Alanine transaminase; AST - Aspartate transaminase

Table 2: Grading of power (Medical Research Council Grading)

	Right	Left		Right	Left
Upper limb			Lower limb		
Shoulder abduction	4/5	4/5	Hip flexion	3/5	3/5
Elbow flexion	4/5	4/5	Hip extension	3/5	3/5
Elbow extension	4/5	4/5	Knee flexion	3/5	3/5
Wrist extension	4/5	4/5	Knee extension	3/5	3/5
Wrist flexion	4/5	4/5	Ankle Dorsiflexion	3/5	3/5
Finger extension	4/5	4/5	Ankle Plantarflexion	3/5	3/5
Finger flexion	4/5	4/5	Great toe dorsiflexion	3/5	3/5

A parainfectious mechanism for GBS in COVID-19, mediated by the generalized, hyperinflammatory response is also suggested in some cases where symptoms of COVID-19 and GBS occurred simultaneously and autoantibodies were not detected.

Conclusion

Neurological involvement in COVID-19 is common but GBS is rare and it should be considered in a patient with quadriparesis or respiratory failure which is out of proportion to the severity of the COVID illness. In fact, in the current scenario, all patient with GBS should be screened for COVID-19. This should be kept in mind by the primary care physicians.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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