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COVID-19 CASE PRESENTING WITH GUILLAIN BARRE SYNDROME (GBS).

Medicine

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ABSTRACT

Coronaviruses can cause multiple organ involvement, however respiratory complications are most common. In this report, we describe the symptoms of Guillain Barre syndrome (GBS) in a patient with COVID-19. We report a 65-years- old gentleman with complaints of acute progressive symmetric ascending quadriparesis. Two weeks prior to hospitalization, the patient suffered from diarrhea, fever, slurring of speech and dysphagia and an RT-PCR was reported positive for COVID-19 infection. The electrodiagnostic test showed that the patient is an Acute motor sensory axonal neuropathy (AMSAN) variant of GBS. COVID-19 stimulates inflammatory cells that produce various pro-inflammatory cytokines which may lead to cytokine storm and maladaptive immune responses. GBS is an immune-mediated disorder and molecular mimicry as a mechanism of autoimmune disorder plays an important role in causing it. It is unclear whether COVID-19 induces the production of antibodies against specific gangliosides. More data needs to be compiled to look at the incidence of GBS type presentation in patients with COVID 19. Further investigations should be conducted about the mechanism of GBS in patients with COVID-19, in the future.

KEYWORDS

Novel coronavirus, COVID-19, Neuropathy, Guillain Barre syndrome, Case report

INTRODUCTION

COVID-19 is a disease about which practitioners and researchers are still learning signs/symptoms, risk factors, comorbidities and outcomes. The first case of COVID-19 in India, which originated from China, was reported on 30th January 2020. India currently has the largest number of confirmed cases in Asia,[1] and has the third highest number of confirmed cases in the world after the United States and Brazil.[2] In this report, we describe for the first time in India GBS symptoms in a patient with COVID-19.

Coronaviruses can cause multiple systemic infections however respiratory complications are the most recognizable symptoms similar to severe acute respiratory syndrome coronavirus (SARS-CoV). The most prevailing symptoms at the onset of disease, after an incubation period of approximately 5.2 days, are fever, cough, dyspnea, myalgia, headache, and diarrhea.[3] Some studies reported gastrointestinal complications, acute cardiac damage, and acute renal failure due to COVID-19 infection.[4&5] Mao and et al evaluated neurological symptoms in 214 patients infected with COVID-19.[6] Of 214 hospitalized patients, 36.4% had nervous system manifestations including dizziness, headache, hypogeusia, hyposmia, muscle damage, ischemic and hemorrhage stroke.[6] COVID19 causes neurological complications including encephalopathy, meningoencephalitis, ischemic stroke and Guillain-Barré Syndrome (GBS). Neuropathy and/or Guillain–Barré syndrome (GBS) due to COVID-19 infection has been rarely reported. GBS is an acute immune-mediated disease of the peripheral nerves and nerve roots (polyradiculoneuropathy) that is usually elicited by various infections.[7] The classic clinical manifestation of GBS is a progressive, ascending, symmetrical flaccid limbs' paralysis, along with areflexia or hyporeflexia and with or without cranial nerve involvement, which can progress over the course of days to several weeks[7]. Two-thirds of patients usually report respiratory tract or gastrointestinal infection 2–4 weeks prior to the onset of neurological symptoms of GBS[8].

Case Presentation

A 66-years- old gentleman was admitted to the internal medicine department with symptoms of acute progressive symmetric ascending quadriparesis. Three weeks prior to hospitalization, the patient suffered from watery diarrhea and low grade intermittent fever. Neurological manifestation of the patient began with acute progressive weakness of distal lower extremities, ten days before admission. At that time, the symptoms progressed from distal limbs to proximal limbs and he developed quadriplegia one day before admission. There was facial paresis bilaterally. He had no urinary and fecal incontinence. At that time, he was diagnosed with COVID-19 after examining oropharyngeal sampling, and chest computed tomography (CT). Reverse transcription-polymerase chain reaction (RT-PCR) for COVID-19 was positive and the patient was treated with hydroxychloroquine and Azithromycin. In the past medical history, the patient was a known case of type 2 diabetes mellitus and hypertension for 6 months and was on medications for the same.

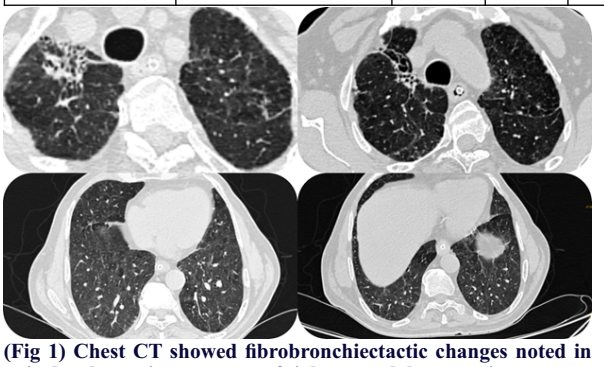
On physical examination, the patient was afebrile with blood pressure 142/82 mm/hg, heart rate 82 beats/minute, respiratory rate 18/minute, and oxygen saturation of 98% on room air. The patient was conscious and had no dyspnea, at the time of hospitalization. The muscle strength examination showed weakness in four limbs with a Medical Research Council (MRC) scale of 3/5 in proximal & distal of the upper extremities and 4/5 in proximal, 3/5 in distal of the lower extremities. Deep tendon reflexes were absent generally. There was a reduction in the vibration and fine touch sensation distal to the ankle joints and also bifacial nerve palsy (House–Brackman grade 4). He had no spinal sensory level. There were no signs of meningeal irritation signs or upper motor neuron disease.

The laboratory examination results were as follows: serum glucose 192 mg/dL; HBa1c 8.2%, urea 42.9 mg/dL; creatinine 0.72 mg/dL;ALT 34 U/l, AST 48 U/l; ALP 78 U/l; sodium 130 mmol/L; potassium 4.6 mmol/L; white blood cell count 11210 cells per microliter (neutrophils = 72%; lymphocytes = 18%); serum ferritin 313 ng/ml, Erythrocyte sedimentation rate 50 mm/hour, C-reactive protein 2.4 mg/l , hemoglobin 13.2 g/dL and glucose +2 and absent ketones in complete urinalysis. Cervical and brain magnetic resonance imaging (MRI) was done which was suggestive of chronic small vessel ischemic changes (FAZEKA GRADE 4). Chest CT showed fibrobronchiectactic changes noted in various lobes(Fig 1). On day 10, after complain of imbalance while walking , a neurophysiological study was done which showed absent CMAP in bilateral tibial nerves and conduction block with markedly reduced velocity in both peroneal nerves, there was absent SNAP in all sensory sample nerves of upper and lower limb and an H reflex was absent bilaterally. These findings are consistent with acute motor-sensory axonal neuropathy (table 1). Cerebrospinal fluid (CSF) analysis showed albuminocytological dissociation (protein 70mg/dl, glucose 120mg/dl, total cell 4/mm3, polymorphs 01%, lymphocytes 99%). Our patient received 0.40 g/kg/day intravenous Immunoglobulin for a duration of five days according to clinical manifestations related to GBS. After successful treatment supports are gradually weaned off and Patient is discharged after 20 days with full recovery with normal power in all limbs.

Table No. 01 MNC STUDY

NERVE	STIMULATION SITE	LAT 1 (mS)		LAT 2 (mS)		AMP (mV)		DIST (mm)		CV(m/s)	
		RT	LT	RT	LT	RT	LT	RT	LT	RT	LT

Median	wrist	4.38	4.63	16.0	15.88	8.25	7.14				
	Elbow	9.38	9.75	21.13	21.13	6.93	5.45	240	260	48	50.73
Peroneal	Ankle	4.13	6	15.38	16.25	1.82	2.25				
	Below fib head	17.13	17.63	31.13	28.38	0.53	0.85	360	350	27.69	30.11
	Above fib head	20.38	19.88	28.13	34.25	0.63	1.18	80	70	24.62	31.11
Tibial	Ankle	0.0	0.0	0.0	0.0	0.0	0.0				
	Popliteal fossa	0.0	0.0	0.0	0.0	0.0	0.0				
Ulnar	wrist	3	3.5	16.13	18	5.31	3.53				
	Below elbow	8	7.5	21.38	22.25	4.32	4.97	240	230	48	57.5
	Above elbow	9.63	9.0	23.0	23.68	4.11	4.41	80	80	49.23	53.33



(Fig 1) Chest CT showed fibrobronchiectactic changes noted in apical and anterior segments of right upper lobe, superior segment of right lower lobe and apicoposterior segment of left upper lobe. Fibrotic changes were noted in the posterior segment of right upper lobe, superior segment, anterior basal and medial basal segment of right lower lobe and anteromedial basal segment of left lower lobe. Centrilobular and paraseptal emphysematous changes were noted in bilateral lung fields

DISCUSSION

COVID19 causes various neurological complications including encephalopathy, meningoencephalitis, ischemic stroke and Guillain-Barré Syndrome (GBS). Radiological series have shown infarcts, microhemorrhages, features of posterior reversible encephalopathy syndrome, or nerve root enhancement CNS demyelination post-COVID-19. The postulated mechanisms of the various neurological syndromes include, either individually or in combination, direct viral neuronal injury, a secondary hyperinflammation syndrome, para and post-infectious inflammatory or immune-mediated disorders, or the effects of a severe systemic disorder with the neurological consequences of sepsis, hyperpyrexia, hypoxia, hypercoagulability and critical illness.

GBS syndrome is typically post-infectious, thought to be due, in great part, to molecular mimicry,[9] wherein antibodies created in response to various bacterial or viral infectious (Campylobacter jejuni, Epstein–Barr virus, cytomegalovirus, and Zika virus.)cross-react with neurons and the myelin sheath, causing demyelination and/or axonal damage.[9-12] GBS is typically multiphasic, with rapid progression, a prolonged plateau, and variable recovery with approximately 15%–20% permanent disability at 1 year.[9-12] GBS-related mortality is approximately 5%, often due to neuromuscular respiratory failure and autonomic dysfunction.[9-12.] To date, there have been nine published cases of GBS associated with COVID–19 (The patients were from one each from Iran [13.] China [14], Spain [15], US [16] and five patients from Italy[17]; the Spanish team reported the Miller Fisher case). Our patient developed significant neurologic weakness only 2weeks after the development of fever and diarrhea, This raises the possibility of a postinfectious GBS. The temporal course of events in our case highlight 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.[17]

It is crucial that clinicians are aware of this association of COVID-19 & GBS to avoid delays in diagnosis and to promote early initiation of treatment and supportive care for a condition associated with significant morbidity and mortality. There has been extensive evidence between Zika virus and GBS.[18-20] Whether COVID-19 patients are also at high risk of GBS, is largely unknown so further epidemiological studies are needed to determine if there is a true increase in incidence of GBS due to the COVID-19 pandemic. Our patient is one of the few cases of COVID 19 presenting as GBS to be reported from the subcontinent.

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