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GUILLAIN–BARRÉ SYNDROME IN SARS-COV-2; A CASE REPORT

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ABSTRACT:
Multiple systems have been affected by COVID-19 infection, and neurological symptoms are not uncommon. A 45-year-old male presented to us in the emergency department without focal neurological deficits. He developed ascending paralysis soon after his admission, which was followed by worsening respiratory symptoms. The family of the patient refused a lumbar puncture, and his NCS/EMG showed features of acute motor axonal neuropathy (AMAN), a variant of Guillain-Barré syndrome (GBS). After being admitted on the 4th day, the patient developed type 2 respiratory failure and died on the 5th day of admission before any treatment could be started. Patients with COVID-19 may worsen their respiratory compromise due to a rare yet treatable neurological disease that should be addressed immediately to prevent complications and death.

INTRODUCTION

Globally, more than 100 million cases of SARS-CoV-2 infections have been reported so far with just under 2 million deaths since the outbreak in the Chinese city of Wuhan in December of this year(1). The evidence also indicates that it may lead to neurological diseases (2). These include headaches, anosmia, and ageusia (3). In 214 hospitalized patients infected with SARS-CoV-2 in Wuhan, China, neurological symptoms were assessed. 36.4% of those 214 patients had a neurological manifestation such as headache, hyposmia, hypogeusia, muscle damage, dizziness, hemorrhagic and ischemic stroke (4). GBS is an immune-mediated disease wherein peripheral nerves and nerve roots are affected (polyradiculoneuropathy), and the disease is often preceded by various viral or bacterial infections (5). A symmetrical, progressive, ascending flaccid limb paralysis is usually associated with areflexia or hyporeflexia. Cranial nerve involvement may or may not be present. The disease usually progresses over a period of several weeks (5) followed by a plateau and a recovery phase. GBS symptoms usually appear two to four weeks before gastrointestinal or respiratory infections are prevalent in 60 to 70% of patients (6). This is a rare case of COVID-19 infection that later manifested as GBS.

CASE PRESENTATION

A 45-year-old male presented to us with a five-day history of high-grade fever, cough, body aches, fatigue, and shortness of breath. With the exception of 10 pack-years of smoking, he had no previously known co-morbidities. In the emergency room, the patient had a temperature of 103.5F, a pulse of 112 beats per minute, a blood pressure of 110/65mm of Hg, and a respiratory rate of 32 breaths per minute. On room air, the patient had a saturation of 73% on a pulse oximeter and was severely short of breath at presentation. The chest auscultation revealed coarse crackles bilaterally. During the time of presentation, his neurological examination was normal, and he was following commands. After HRCT Chest (Figure-1) and positive RT-PCR for SARS-CoV-2, he was admitted to an isolation ward for treatment of severe COVID-19 Pneumonia and was given high flow oxygen, tocilizumab, and remdisivir along with symptomatic management.

After the third day of admission, the patient developed weakness in his lower limbs ascending. The power in the lower limb was 1/5 distally and 3/5 proximally. In addition to decreased tone, his lower limb reflexes were absent, and his upper limb reflexes were depressed. Planters were mute on both sides and sensations were normal throughout. A lumbar puncture was not performed due to the patient's clinical condition. The next day, the patient's weakness progressed to the upper limbs, and power was noted to be 2/5 distally in the hands and 4/5 proximally. Immediately, nerve conduction studies were performed which showed decreased amplitudes with slight reductions in conduction velocities (Figure-2) in multiple nerves both in the upper and lower limbs while his EMG demonstrated neuropathic changes in all muscles sampled.

Patient was diagnosed with Guillain-Barré syndrome and given the treatment options of intravenous immunoglobulin and plasmapheresis. Patient condition worsened on day 4 of admission, and he developed type 2 respiratory failure. On the following day, he died at COVID-19 ICU.

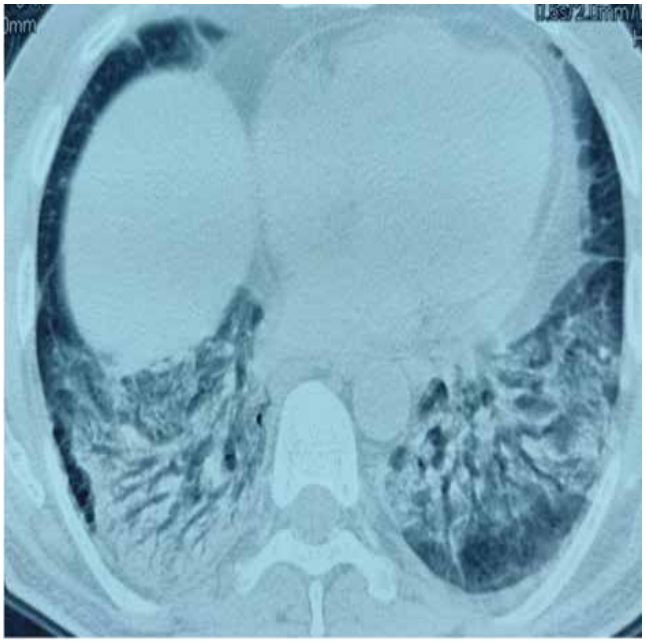

Figure-1: HRCT chest; Axial image showing dense peripheral based patches of consolidations with air bronchogram and few small peripheral ground glass opacities.

Table 1:

Laboratory tests	At presentation	3rd day of admission	Normal Range
Hb	14.5	15.3	13 – 17 g/dL
MCV	86.6	88.2	80 – 98 fL
TLC	17250	21300	4000 – 10000 /µL
Platelets	178000	86000	150000 – 400000 / µL
ALT	53	76	4 – 42 U/L
Creatinine	1.1	1.45	0.6 – 1.3 mg/dl
Urea	58	63	13 – 43 mg/dl
CRP	247	253	<10 mg/L
D-Dimers	1784	2350	<250 ng/mL
Ferritin	2360	4559	20 – 300 ng/mL
IL-6	110.2		<7 pg/mL

admission also indicated an acute infection. In this case, GBS shows a para-infectious pattern, rather than the usual post-infectious pathology in respiratory or gastrointestinal infections. Such an association may merit further study and may require further epidemiological data.

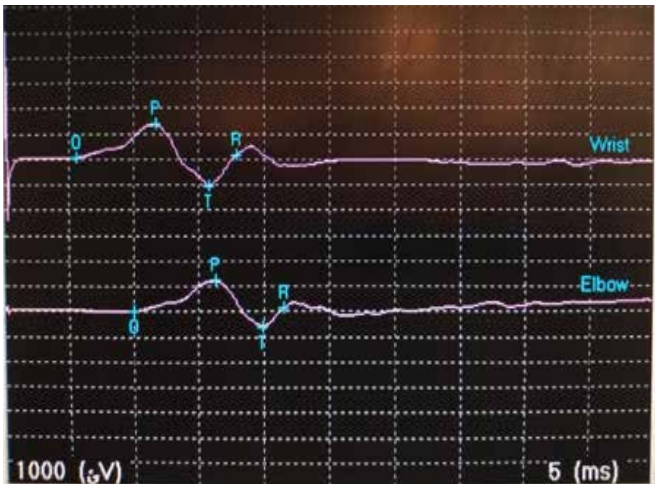

Figure-2: NCS findings.

DISCUSSION:

GBS usually manifests itself over a period of a few days to 6 weeks and is usually preceded by respiratory or gastrointestinal infections [7]. There have been numerous reports of its association with viral infections, such as HIV, EBV, and most recently Zika virus [7,8]. In most cases, it is thought to be due to an autoimmune response where antibodies are developed against the glycoprotein found on the surface of the causative organisms, which is similar to a similar protein found on peripheral nerves, resulting in neurological damage [7]. SARS-CoV-2 caused a similar reaction in our patient, resulting in GBS. The subcontinent has not yet reported any cases of GBS caused by SARS-CoV-2. To date, only one case series [9] and two case reports [10,11] have been documented worldwide.

Our case presented all the characteristics of a typical, but severe GBS. During the current infection, the patient developed a full-blown case of GBS that started out as a normal neuropathy. Because of the general condition of patients with severe SARS-COV-2 infection, neurological symptoms like those in our patient can be easily missed. As a result, physicians should pay careful attention to peripheral neuropathies and seek neurological consultation whenever such symptoms are observed in patients with SARS-CoV-2 infection.

As a rather novel virus, SARS-CoV-2 requires further study in order to understand its virulence and pathogenicity. Neurological complications like in our patient should be aggressively treated if possible, to prevent further respiratory compromise. Initially, our patient presented with symptoms of SARS-CoV-2 infection, but soon developed classic GBS symptoms. His initial and follow-up investigations on the third day of

CONCLUSION:

There is an association between SARS-CoV-2 infection and a neurological disease in this case report. This may cause rapid deterioration and even death. Clinicians are urged to keep an eye out for symptoms related to SARS-COV-2 that can easily be missed. Comprehensive systemic reviews should be conducted to ensure no symptoms are overlooked. In order to prevent further complications, patients with neuropathic association should receive aggressive treatment.

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Naveed Ullah Khan; data collection, data analysis, manuscript writing, manuscript review
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Waleed Shahzad; data collection, manuscript review
Hafsa Mobeen; data collection, manuscript review
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