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POST COVID-19 INFECTION AND GUILLAIN BARRE SYNDROME: A CASE REPORT

Bismi S.* and Sreevidya S.

6th Year Pharm D, Ezhuthachan College of Pharmaceutical Sciences, Trivandrum, Kerala, India.

***Corresponding Author: Bismi**

6th Year Pharm D, Ezhuthachan College of Pharmaceutical Sciences, Trivandrum, Kerala, India.

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ABSTRACT

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy that is often related to a previous infectious exposure. GBS emerged as a potentially serious complication of coronavirus disease 2019 (COVID-19) since its declaration as a global pandemic. Novel outbreak with coronavirus 2019 began since 31 December 2019. Coronaviruses can cause multiple systemic infections that respiratory complications are the most obvious symptoms. In this report, we describe the symptoms of Guillain Barre syndrome (GBS) in one infected patient with COVID-19, for the first time. We reported a 65-years- old male patient with complaints of acute progressive symmetric ascending quadriparesis. Two weeks prior to hospitalization, the patient suffered from cough, fever, and RT-PCR was reported positive for COVID-19 infection. The electrodiagnostic test showed that the patient is an AMSAN variant of GBS. COVID-19 stimulates inflammatory cells and produces various inflammatory cytokines and as a result, it creates immune-mediated processes. GBS is an immune-mediated disorder and molecular mimicry as a mechanism of autoimmune disorder plays an important role in creating it. It is unclear whether COVID-19 induces the production of antibodies against specific gangliosides. Further investigations should be conducted about the mechanism of GBS in patients with COVID-19, in the future.Clinical manifestations of COVID-19 are known to be variable with growing evidence of nervous system involvement.

KEYWORDS: COVID-19; Case report; Guillain Barre syndrome; Neuropathy; Novel coronavirus.

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute immune-mediated disease of peripheral nerves and nerve roots that is usually preceded by respiratory or gastrointestinal infection. It presents with progressive, ascending, symmetrical limb weakness, and paresthesia with diminished or absent deep tendon reflexes, with or without respiratory and cranial nerves involvement. On 31 December 2019, a novel coronavirus (COVID-19) was detected in Wuhan City, Hubei Province of China.[1] COVID-19 is a new beta coronavirus, which enters the cell via fusion with angiotensin-converting enzyme 2 (ACE2) receptor.[2] The symptoms of COVID-19 are dependent on the age and the patient's underlying medical illness and also the condition of the immune system.[3] These manifestations involved both central and peripheral nervous systems and ranged from a simple headache and dizziness to a more sinister presentation such as encephalitis or cerebrovascular stroke. Coronaviruses can cause multiple systemic infections that 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.[5] Some studies reported gastrointestinal complications, acute cardiac damage, and acute renal failure due to COVID-19 infection.[6,7] Mao and et al evaluated neurological symptoms in 214 patients infected with COVID-19 (8). Of 214 hospitalized patients, 36.4% had nervous system manifestations including dizziness, headache, hypogeusia,,hyposmia, muscle damage, ischemic and hemorrhage stroke.[8] In our knowledge, up to now, no reported neuropathy and/or Guillain–Barré syndrome (GBS) due to COVID-19 infection. GBS is an acute immune-mediated disease of the peripheral nerves and nerve roots (polyradiculoneuropathy) that is usually elicited by various infections.[9] The classic clinical manifestations of GBS is 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.[9] Two-thirds of patients usually report respiratory tract or gastrointestinal infection 2–4 weeks prior to the onset of neurological symptoms of GBS.[10] In this report, we describe GBS symptoms in one infected patient with COVID-19, for the first time. Guillain-Barré syndrome (GBS) is an acute immune-mediated disease of peripheral nerves and nerve roots that is usually preceded by respiratory or gastrointestinal infection. It presents with progressive, ascending,

symmetrical limb weakness, and paresthesia with diminished or absent deep tendon reflexes, with or without respiratory and cranial nerves involvement.[2]

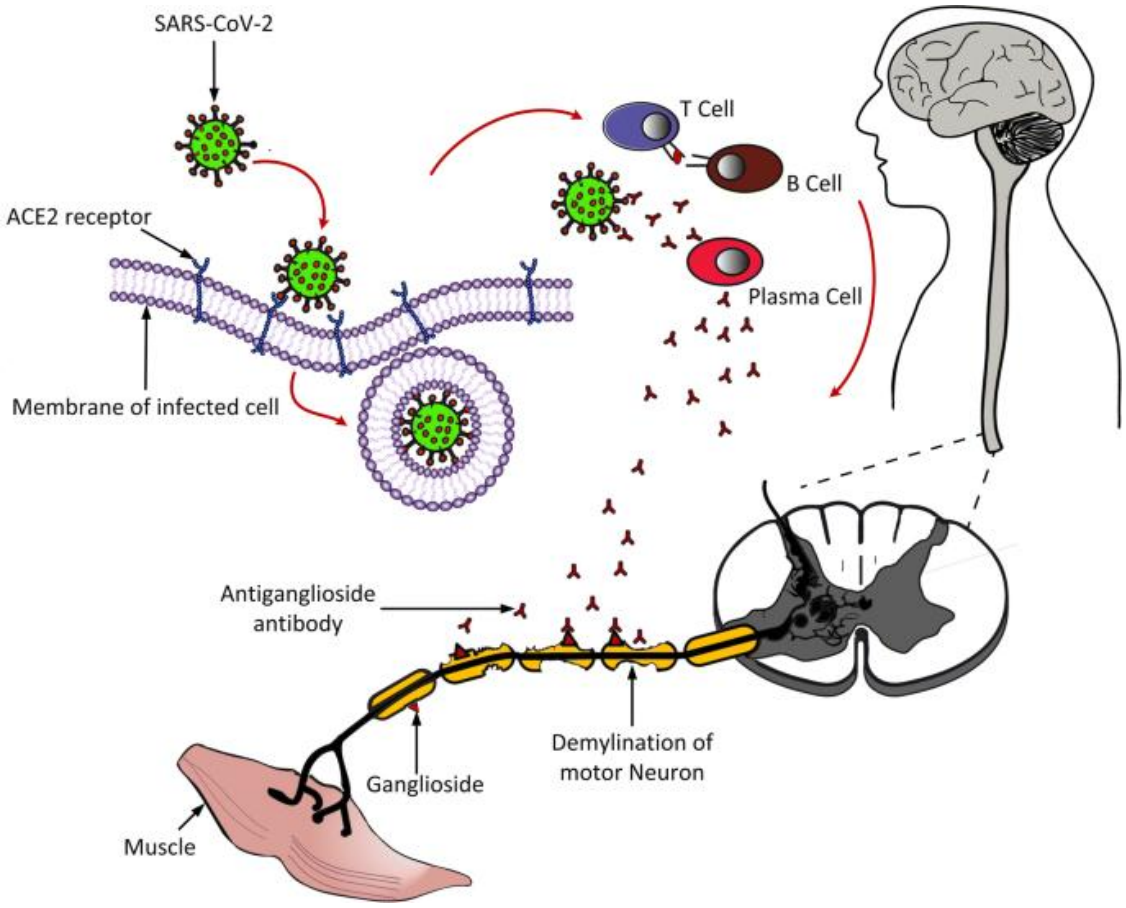

Figure 1: A schematic representation of the likely pathophysiology of COVID-19-associated GBS.

SARS-COV-2 has a high affinity for the angiotensin-converting enzyme 2 (ACE2) receptor, located on nasal and oral mucosa , neurons, glia cells, and blood vessels of the central nervous system .During an infection, SARS-COV-2 binds this receptor and is endocytosed. Due to similarity in the peptide sequences or epitopes of SARS-Cov-2 and gangliosides (molecular mimicry), the antibodies formed against the virus, through the T Cell-B cell interactions, may bind the gangliosides located on the peripheral neurons. This may result in an autoimmune response that destroys the myelin and/or axons. The demyelination or axonal damage disrupts neural transmission, which causes the GBS symptoms such as muscle weakness, paralysis, coordination problems, breathing difficulties, and autonomic dysfunction.

CASE PRESENTATION

A 65-years- old male patient was admitted to the emergency department, with symptoms of acute progressive symmetric ascending quadriparesis. Neurological manifestations of the patient began with acute progressive weakness of distal lower extremities, five days before admission. At that time, the symptoms progressed from distal limbs to proximal limbs and he had been quadriplegia one day before admission. There was facial paresis bilaterally. He had no urinary and fecal incontinence. Two weeks prior to hospitalization, the patient suffered from cough, fever and sometimes dyspnea. At that time, he referred to an infectious disease specialist and was diagnosed with COVID-19 after examining oropharyngeal sampling, and chest computer tomography (CT). Reverse transcription-polymerase chain reaction (RT-PCR) for COVID-19 was positive and the patient was treated with hydroxychloroquine, Lopinavir/Ritonavir (LPV/RTV) and Azithromycin. In the past medical history, the patient was a well-known case of type 2 diabetes mellitus and was treated with metformin medication.

On physical examination, the patient was afebrile with blood pressure 120/80 mm/hg, heart rate 73 beats/minute, respiratory rate 18/minute, and oxygen saturation of 95% 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 2/5 in proximal, 3/5 in distal of the upper extremities and 1/5 in proximal, 2/5 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–Brackmann grade 3). He had no spine sensory level. Meningeal irritation signs and upper motor neuron disorder signs were negative. The laboratory examination results were follows: serum glucose 159 mg/dL; blood urea nitrogen: 19 mg/dL; creatinine 0.8 mg/dL; alanine aminotransferase 35 IU/L; aspartate aminotransferase 47 IU/L; sodium 135 mmol/L; potassium 3.9 mmol/L; white blood cell count 14,700 cells per microliter (neutrophils = 82.7%; lymphocytes = 10.4%); Erythrocyte sedimentation rate 72 mm/hour, C-reactive protein 2+, hemoglobin 11.6 g/dL and negative glucose and ketone in complete urinalysis. Cervical and brain magnetic resonance imaging (MRI) was done and showed a normal finding except for mild herniation of two intervertebral discs. Lung CT showed diffused consolidations and ground-glass opacities in both lungs, and bilateral pleural effusion (Fig. 2).

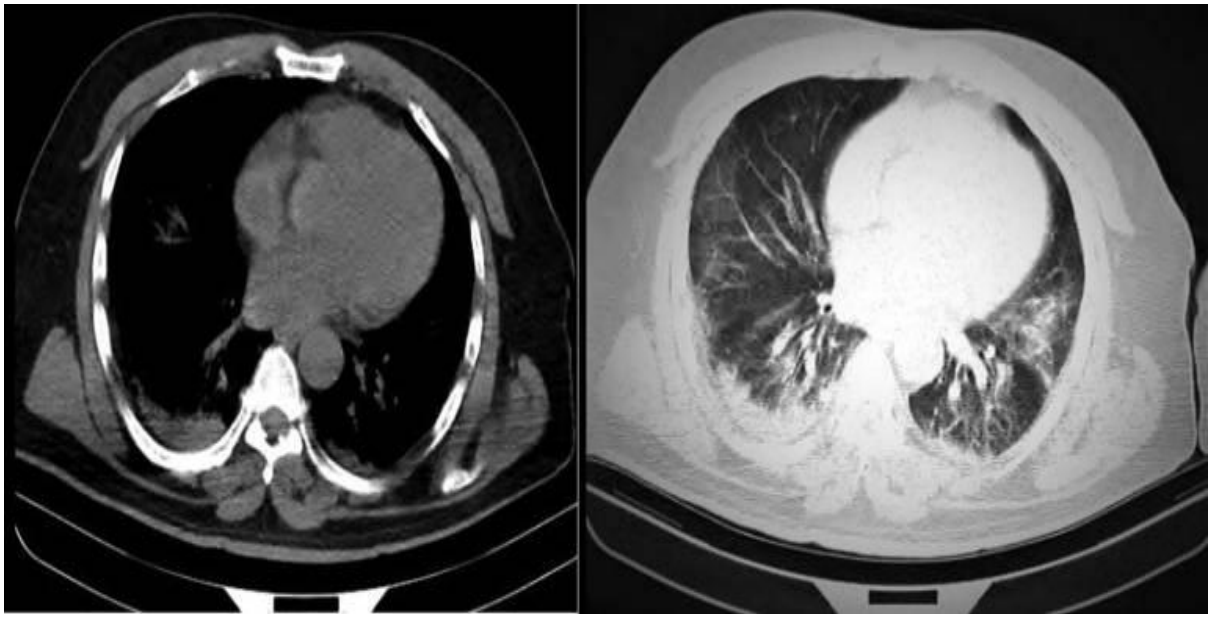

Figure 2: Lung CT showed diffused consolidations and ground-glass opacities in both lungs, and bilateral pleural effusion.

DISCUSSION

SARS-CoV-2 frequently afflicts the respiratory system and gastrointestinal tracts. It shares its identity with other human coronaviruses including SARS-CoV and Middle East respiratory syndrome coronavirus. In this group of viruses, the respiratory system is commonly affected but they have also shown the involvement of the nervous system.[4] Increasing reports of neurologic manifestations of COVID-19 are emerging, but only a few cases of GBS associated with this virus have been established. GBS is an immune-mediated response, likely from a recent infection, where the immune system attacks the peripheral nerves due to a molecular mimicry phenomenon. This has preceded two-thirds of the times by an upper respiratory infection or gastroenteritis.

The case series by Mao *et al* in Wuhan, China, was one of the first studies that showed neurologic manifestations in patients with COVID-19. They concluded that patients with more severe COVID-19 illness were more likely to have neurologic symptoms.[5] In contrast, our patient's respiratory status was relatively stable.

In Italy, a series of five patients were diagnosed with GBS 5–10 days after a viral illness from COVID-19. Similar to our patient, they did not show typical MRI findings of GBS including surface thickening and contrast enhancement on the conus medullaris and the nerve roots of the cauda equina. Only one of the five patients had a functional recovery to the point of ambulation. We cannot yet conclude the severity of neurologic injury with COVID-19-associated GBS.[6]

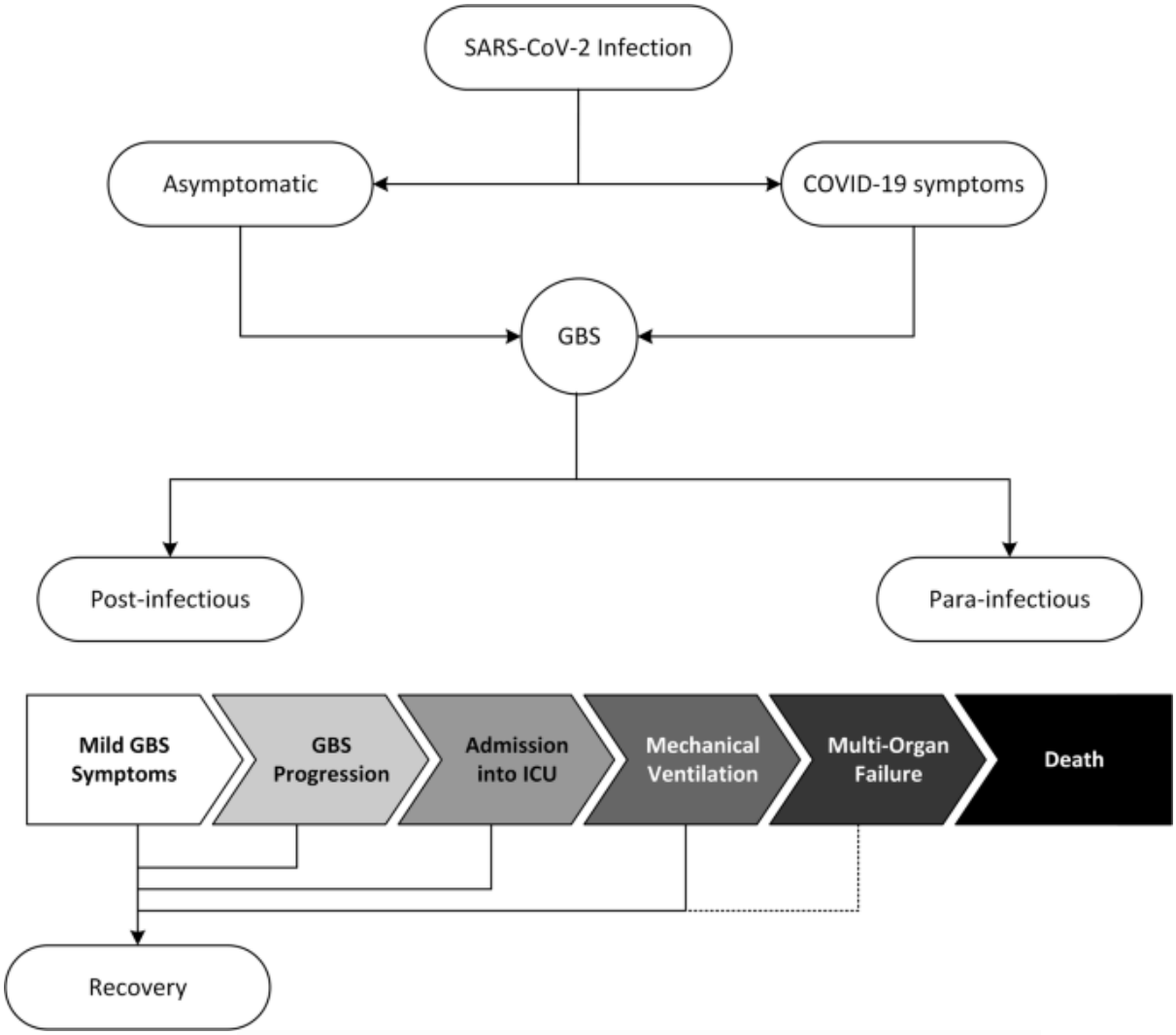

Figure 3: The atypical manifestation and prognosis of COVID-19-associated GBS.

The literature shows there is variability in the presentation of COVID-19 and GBS. Our case had a typical course of viral symptoms preceding GBS findings. However, two other case reports identified concurrent respiratory and neurologic symptoms.[7,8] Besides, the duration from onset of viral illness to neurologic manifestations have ranged from 5 to 24 days.[9] In all cases reported, treatment with IVIG was administered. However, the recovery varied from full neurologic recovery to no change in extremity function and terminal respiratory failure.[7,12]

There are several theories on how the virus attacks the nervous system. Studies postulate that the virus can infect a peripheral neuron, use an active retrograde transport mechanism across the synapse onto the cell body and reach the brain.[13] Other proposed mechanisms include direct damage through angiotensin converting enzyme-2ACE2 receptors, cytokine-related injury and hypoxia-related sequela.[14] It is unclear if the COVID-19 itself triggers the formation of antibodies again any specific forms of glycolipids seen in some forms of GBS.[15] There is a need for further investigation into how COVID-19 is related to GBS.

CONCLUSION

In summary, to our knowledge, this is the first reported case of GBS in a patient infected with COVID-19. Given that the most common symptoms of infection with COVID-19 were reported respiratory infections and two-thirds of Guillain-Barre patients usually mention respiratory infections before the onset of symptoms, hence GBS should be considered as neurological complications of infection with COVID-19. Therapy with IVIG or plasmapheresis should be initiated along with antiviral treatment.Neurologists should be aware of GBS as a rare complication associated with COVID-19. Diagnosis can be challenging and delayed, especially in asymptomatic patients or those with mild respiratory infection weeks earlier. Early diagnosis and management can improve clinical outcome. Most of the literature consist of case reports or case series, and further larger studies are needed to assess the causal relationship between COVID-19 and GBS.

Statement of Ethics

The patient gave written consent to share his case.

Conflict of Interest Statement
The authors declare that they have no conflicts of interest to disclose.

Declaration of Competing Interest
The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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