

# Guillain-Barre syndrome associated with COVID-19 infection

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## ABSTRACT

**Background:** Coronavirus disease 2019 (COVID-19) typically causes breathing issues that can range from flu symptoms to extreme pneumonia, but it can also impair extra respiratory systems and cause multisystemic failure, including neurological complications.

**Case Presentation:** A 55-year-old male with acute progressive symmetrical ascending quadriparesis complaints was admitted to the hospital. Twelve days prior to hospitalization, the patient with taste disruption, myalgia, fever, and polymerase chain reaction with reverse transcription was confirmed to have been positive for COVID-19 infection. The neurophysiological findings were consistent with the diagnosis of Guillain-Barré syndrome (GBS).

**Conclusion:** COVID-19 activates inflammatory cells and creates a number of inflammatory cytokines and eventually produces immune-mediated processes. Both cell and humoral-dependent pathways of GBS pathogenesis are believed to be related. The peripheral nervous system, myelin, axons, and in some cases, both immune-mediated attacks are believed to be the cause of molecular expression. COVID-19 is believed to induce antibody formation against particular gangliosides. Further study is needed to understand the role of GBS caused by infection with COVID-19.

**Keywords:** Guillain-Barré syndrome, COVID-19, plasmapheresis, neurologic emergencies, critical illness.

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## Background

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) originated in Wuhan City, China, and is now expanding across the world [1]. The new coronavirus (CoV) named Coronavirus disease 2019 (COVID-19) can grow with different symptoms depending on the patient's medical background, age, and immune system status by entering the cell using the angiotensin-converting enzyme 2 (ACE2) receptor. Patients who have chronic disorders such as diabetes mellitus, asthma or cardiovascular disease are compromised and affected by COVID-19 more than stable patients [2]. It occurs mainly with respiratory-related symptoms such as cough and difficult breathing, but it may also occur with other certain systemic symptoms such as high fever, exhaustion, diarrhea, stomach pain, and altered consciousness. While neurological signs are increasingly seen, not all of them can be identified as yet [3]. As a consequence of CoV infection, neurological complications such as febrile seizures, encephalitis, and mental status fluctuations may occur. Coronaviruses are believed to have neurotropic and neuroinvasive properties. Coronaviruses can induce inflammation and demyelination in the central nervous system (CNS) through the olfactory bulb after nasal infection [4].

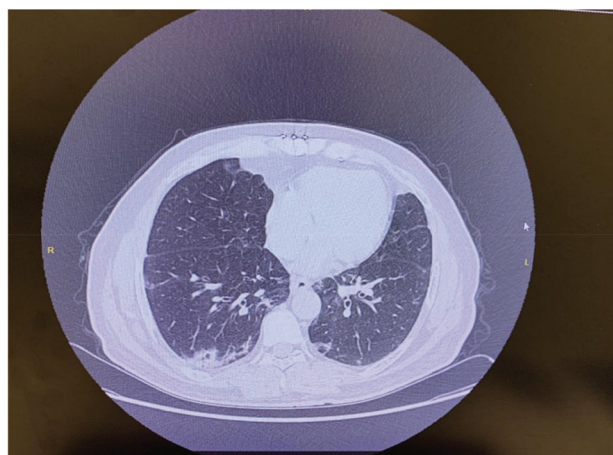
In 1916, Georges Guillain, Jean-Alexandre Barré, and Andre Strohl described a condition that can be seen with symptoms such as acute, inflammatory, demyelinating polyradiculoneuropathy [5]. Autoimmunity is believed to be responsible for this peripheral nerve activity and ascending symmetrical limb fatigue, sensory dysfunction and reduced deep tendon reflexes [5]. Guillain-Barré syndrome (GBS) is seen with signs such as quickly developed weakening in limbs, such as arms and legs, as well as bulbar, facial, and respiratory muscles, and is the most frequent cause of acute flaccid paralysis [6]. People infected by GBS often recover without any disability or sensory impairment; nevertheless, some patients have to be followed-up for a long time in an intensive care unit and some may be discharged with limb fatigue, sensory dysfunction, and symptoms of paresthesia or dysesthesia [6]. In addition, approximately 5% of the patients die from complications such as respiratory failure, pneumonia, and arrhythmias [6]. GBS is a medical emergency including high morbidity and lower life expectancy [6]. 2-4 weeks before the onset of neurological signs of GBS, two-thirds of patients typically experience respiratory tract or gastrointestinal infection [2]. We explain the

symptoms of GBS in one infected patient with COVID-19 in this article.

### Case Presentation

A 55-year-old man with the onset of acute progressive symmetrical ascending quadriparesis was admitted to the emergency department. Neurological manifestations of the patient began with acute progressive weakness of the distal lower extremities 1 week prior to admission. The signs moved from distal limbs to proximal limbs at that stage, and bilateral facial paresis occurred. He did not have urinary and fecal incontinence. The patient had suffered from taste disruption, myalgia, and fever, 12 days before hospitalization. At that point, after an oropharyngeal sampling and tomography of the chest, he reported to an infectious disease specialist and was diagnosed with COVID-19. The polymerase chain reaction with reverse transcription of COVID-19 was positive and the patient was given hydroxychloroquine and favipiravir for treatment. Recently, the patient had a well-known diagnosis of type 2 diabetes mellitus and was treated with metformin and empagliflozin. He was also awaiting heart surgery and was taking aspirin and statin drugs.

At the time of hospitalization, the patient was informed and had no dyspnea. The muscle strength test revealed bilateral lower extremity deficiency with the Medical Research Council of 5/5 in the proximal and distal upper extremities and 1/5 in the proximal and 2/5 in the distal lower limbs. Deep tendon reflexes were not present in the lower limbs. The vibration and fine touch feeling distal to the ankle joints was diminished, as well as bifacial nerve paralysis (House-Brackmann grade 4). He did not have a spine sensory level. Signs of meningeal irritation and symptoms of upper motor neuron disease were negative. The findings of the experimental test were as follows: serum glucose 165 mg/dl; blood urea nitrogen 77 mg/dl; creatinine 0.64 mg/dl; alanine aminotransferase 48 IU/l; aspartate aminotransferase 21 IU/l; sodium 138 mmol/l; potassium 4.2 mmol/l; white blood cell count 8,060 cells per microliter (neutrophils = 74.5%; lymphocytes = 15.8%); erythrocyte sedimentation rate 72 mm/hour, C-reactive protein 2+, hemoglobin 16.7 g/dl; and ++glucose and +ketone in complete urinalysis. COVID-19 *Immunoglobulin G + Immunoglobulin M* was >10.00 (reactive). Brain computed tomography was carried out and revealed a normal result. Lung CT revealed diffuse consolidations and ground-glass opacities and bilateral pleural effusion in the lungs (Figure 1). Lumbar puncture was carried out on day 7 and the result of cerebrospinal fluid (CSF) protein was 2,103 mg/l (150-400), and CSF cell count was zero. The CSF analysis indicated albuminocytological dissociation. Our patient underwent plasmapheresis for a period of 7 days based on clinical manifestations linked to GBS. He also received pregabalin 150 mg twice daily for neuropathic pain and low weight heparin for prophylaxis of deep venous thrombosis. The neurophysiological analysis was conducted



**Figure 1.** Chest computer tomography revealing ill-defined opacities in both lungs.

after plasmapheresis therapy. Electrodiagnostic parameters displayed reduced amplitude at the potential for compound muscle activity and no response at the potential for sensory nerve action. Electromyography showed a decline in recruiting. These findings are associated with acute motor-sensory neuropathy of the axon. Following plasmapheresis therapy, he was transferred to the Physical and Rehabilitation Center.

### Discussion

Our study details a case of GBS related to COVID-19 infection. Coronaviruses are zoonotic pathogens found in humans and multiple species with a wide variety of clinical characteristics, from asymptomatic to hospitalization requirements in the intensive care unit with respiratory, urinary, hepatic, and neurological symptoms. They were not considered to be so pathogenic to humans until they were first seen in Guangdong, China, in 2002 and 2003 with severe acute respiratory syndrome (SARS). Approximately 10 years after SARS, this time, another extremely pathogenic CoV, Middle East Coronavirus Respiratory Syndrome (MERS-CoV), had arisen in the Middle Eastern countries. In December 2019, the novel coronavirus, another public health problem, was the focus of worldwide attention due to a pneumonia epidemic of uncertain origin [7]. It was recognized that human coronavirus viruses have the elevated degree of mutations and may invade the CNS and can be associated with neurological signs [8].

CoV experiments have demonstrated that these viruses had neurotrophic and neuroinvasive properties [9]. COVID-19 has neurological manifestations close to those of extreme acute respiratory syndrome, SARS-CoV and MERS-CoV [10]. ACE2 is commonly expressed, including in the lungs, coronary system, intestines, liver, central nervous system, and adipose tissue. ACE2 has recently been identified as the SARS-CoV-2 receptor, an infectious agent responsible for COVID-19, providing a vital link between immunity, inflammation, ACE2, and cardiovascular disease [11].

CoV and Zika virus forms that were previously discovered were also associated with GBS. The mechanism of GBS occurrence is based on molecular mimicry and anti-ganglioside antibodies following infection in genetically predisposed patients. These antibodies have the strongest relationship with certain types of GBS. An autoimmune response in which antibodies to the pathogen that are identical to the protein complexes of the peripheral nerve components cause damage to the nervous system is a potential process. "Molecular mimicry" has been established as a theoretical probability that structural resemblances between foreign and self-peptides are essential in shaping to the cross-activation in pathogen-derived peptides of self-reactive B cells or T cells [10].

Our patient had classic symptoms of GBS that began around 2 weeks after the infection of the respiratory tract with COVID-19. Since this patient had a previous history of COVID-19 and had a progressive worsening of monophasic symmetric limbs, we suggested the GBS variant as a possible diagnosis [2]. This disorder was confirmed by electrodiagnostic results, and the CSF study revealed albuminocytological dissociation seen in the GBS.

Early diagnosis of GBS and initiation of early care is very important. However, improvement ranged from complete neurological recovery to little improvement in extremity function and severe respiratory failure. As there is a risk of death due to respiratory muscle involvement in GBS, prompt diagnosis and early care are critical in all patients. Intravenous immunoglobulin (0.4/g/kg/day) or a 5-day plasmapheresis are successful medication options. Fortunately, the combination of these was not more effective when compared to the usage alone. Since he had rapid decline, we preferred plasmapheresis to our patient for a duration of 7 days and initiated pregabalin for neuropathic pain and low weight heparin for prophylaxis of deep venous thrombosis. After the treatment, we referred him to the Physical and Rehabilitation Center because he had a weakness and little improvement.

## Conclusion

According to published research studies from around the world, there are neurological symptoms consistent with COVID-19 infection. The most prevalent signs of COVID-19 infection have been characterized as respiratory infections, and two-thirds of GBS patients usually explain viral symptoms before COVID-19 symptoms so that GBS can be accepted as a neurological complication during or after COVID-19 infection. COVID-19-associated GBS is a recent established problem that clinicians should be mindful of, in order to treat cases properly. It is unknown if COVID-19 induces the manufacture of particular ganglioside antibodies that normally occur with some types of GBS. Further investigations on the mechanism of GBS in patients with COVID-19 should be carried out in the future, about the pathogenesis.

## List of Abbreviation

ACE2	Angiotensin-converting enzyme 2
CNS	Central nervous system
CoV	Coronavirus
COVID-19	Coronavirus disease 2019
GBS	Guillain-Barré syndrome
MERS-CoV	Middle East Coronavirus Respiratory Syndrome
SARS	Severe acute respiratory syndrome
SARSCoV-2	Severe acute respiratory syndrome coronavirus 2

## Conflict of Interests

The authors declare that there is no conflict of interest regarding the publication of this article.

## Funding

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## Consent for publication

Written consent was obtained from the patient.

## Ethical approval

Ethical approval is not required at our institution to publish an anonymous case report.

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### Summary of the case

1	<b>Patient (gender, age)</b>	55-year-old male patient
2	<b>Final diagnosis</b>	Guillain-Barre syndrome
3	<b>Symptoms</b>	He had progressive symmetrical ascending quadriparesis
4	<b>Medications</b>	Patient underwent plasmapheresis for a period of 7 days, received pregabalin 150 mg twice daily for neuropathic pain, and low weight heparin for prophylaxis of deep venous thrombosis
5	<b>Clinical procedure</b>	After the treatment, he was transferred to the Physical and Rehabilitation Center
6	<b>Specialty</b>	Neurology