

Guillain-Barré syndrome following COVID-19 infection in an elderly patient: a case report

Enkhmaa Luvsannyam^{1*}, Arathi Jayaraman², Molly Sanjay Jain², Karan Sharma³, Manoj Reddy Somagutta¹, Rathna Kumar Yallapragada⁴

European Journal of Medical Case Reports

Volume 5(8):242–245

<https://doi.org/10.24911/ejmcr/173-1618006223>



This is an open access article distributed in accordance with the Creative Commons Attribution (CC BY 4.0) license: <https://creativecommons.org/licenses/by/4.0/> which permits any use, Share — copy and redistribute the material in any medium or format, Adapt — remix, transform, and build upon the material for any purpose, as long as the authors and the original source are properly cited. © The Author(s) 2021

ABSTRACT

Background: Coronaviruses can affect multiple body systems and respiratory failure is the most common complication. Since the outbreak of coronavirus disease-2019 (COVID-19) in January 2020, the association between COVID-19 and Guillain-Barre syndrome (GBS) has been growing. GBS is known to be triggered by an antecedent infection, mostly viruses.

Case Presentation: We present a case of GBS in an 83-year-old female patient with a confirmed COVID-19 infection. The patient initially presented with fever, cough, and fatigue. She was treated with intravenous fluid and symptomatic treatment and discharged home after stabilization. Several weeks after her initial encounter, she experienced bilateral paresthesias as well as numbness and tingling in her lower extremities. The patient's neurological symptoms were not alleviated with standard intravenous immune globulin (IVIG) therapy; however, her symptoms significantly improved with subsequent plasmapheresis therapy.

Conclusion: Based on the emerging evidence of recent studies, there is a possible connection between COVID-19 and GBS. Clinicians should be aware of the neurological manifestations of COVID-19 infection. Early diagnosis and proper treatment of COVID-19 and its neurological symptoms are crucial to increase the chance of a successful recovery.

Keywords: COVID-19, SARS-CoV-2, coronavirus, Guillain-Barre syndrome, acute inflammatory demyelinating polyneuropathy, case report.

Received: 9 April 2021

Accepted: 31 July 2021

Type of Article: CASE REPORT

Specialty: Internal Medicine

Correspondence to: Enkhmaa Luvsannyam

*Avalon University School of Medicine, Willemstad, Curacao

Email: luvsenkh@isu.edu

Full list of author information is available at the end of the article.

Background

Coronavirus disease-2019 (COVID-19) is caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which is a single-stranded positive linear RNA beta coronavirus with an envelope and spike proteins on the outer surface [1,2]. COVID-19 symptoms can include fever, cough, and shortness of breath, along with several nervous system presentations, such as hypogeusia, hyposmia, dizziness, and headache [1-5]. The degree of symptom severity depends on various factors, including age and comorbidities that have weakened the immune system. Extreme symptom severity has led to death.

Guillain-Barré syndrome (GBS) is a demyelinating autoimmune disorder in which the body's immune system attacks the myelin sheath of the peripheral nervous system [6]. It commonly presents a few days after upper respiratory tract infections or gastroenteritis. The most common presentation is ascending paralysis starting from the lower limbs which slowly progresses to involve the breathing muscles causing respiratory failure in a matter of a few

weeks only [2,7]. Other symptoms include weakness in coordination, hyporeflexia, weakness in eye muscles, and autonomic dysfunction affecting the heart rate and blood pressure [7]. Some pathogens associated with GBS are *Campylobacter jejuni*, Cytomegalovirus, Epstein-Barr virus, human immunodeficiency virus, Zika virus, and *Mycoplasma pneumoniae* [4,5,7].

The association between COVID-19 and GBS is novel, and the mechanism of action is still being investigated with a variety of theories. Regardless of the mechanisms involved, patients with a recent history of COVID-19 have developed GBS. The case report presented herein contributes to the vital data collection necessary to help the scientific community further understand the epidemiology and prognosis of GBS sequelae of COVID-19.

Case Presentation

An 83-year-old Caucasian woman with a past medical history significant for chronic atrial fibrillation, dyslipidemia, and hypertension presented to the emergency

department (ED) with a low-grade fever, cough, extreme fatigue, and weakness for 2 weeks. Due to her symptoms, rapid SARS-CoV-2 testing was performed and results came back positive. In the ED, her temperature was 99.9 F, respiration rate was 20 per minute, and oxygen saturation was 95%. WBC count was 5.16 k/ μ l, hemoglobin was 15.1 g/dl, and troponin level was elevated at 20 and 24 ng/l. Electrocardiogram (ECG) and chest X-ray were also done which were consistent with atrial fibrillation and early bilateral interstitial infiltrates. The patient was admitted and treated with intravenous (IV) and oral fluids with minerals, multivitamin supplements, pain, and pyrexia management. IV anticoagulants and antiviral medications were not given as the patient was not hypoxicemic. Post-acute care management of the COVID-19 infection, the patient was sent to the skilled nursing facility for rehabilitation and medical management. One week after her transfer, the patient developed new symptoms of severe bilateral lower extremity (LE) pain, numbness, tingling, and difficulty walking. Neurologic examination revealed decreased sensations, 0/5 strength, and diffuse areflexia in bilateral lower limbs. Computed tomography and ultrasound of the bilateral LEs were performed and no significant abnormalities were seen. Due to her symptoms concerning GBS, electromyography and nerve conduction velocity (EMG/NCV) study were performed which showed abnormalities highly suggestive of an acute inflammatory demyelinating polyneuropathy (AIDP) process/GBS (Figure 1). The patient also underwent a fluoroscopy-guided lumbar puncture which showed pink cerebrospinal fluid (CSF) with an RBC count of 1,106, no WBC, glucose level of 50 mg/dl, and elevated protein of 64 mg/dl consistent with albuminocytologic dissociation.

The patient was started on intravenous immune globulin (IVIG) 0.5 gram/kg for 5 days and continued with physical and occupational therapy (PT/OT). After 5 days of IVIG treatment, the patient continued to have numbness and weakness in her LEs. Subsequent magnetic resonance imaging of the brain and the entire spine were unremarkable. Due to no clinical improvement in her symptoms after a course of IVIG treatment, plasmapheresis was initiated. After a total of three plasmapheresis treatments, the patient's symptoms have subsided significantly with improved sensations and strength in her LEs. Following improvement, the patient was sent to the acute rehabilitation unit for further PT/OT and performed gradual improvement of her motor strength.

Discussion

In this study, we report AIDP in a patient recently diagnosed with COVID-19. In AIDP, acute demyelination causes classic ascending motor paralysis and sensory abnormalities, resulting in death if the diaphragmatic muscles are also affected [4]. The diagnosis of GBS starts with the detailed history of infection onset and resolution in the patient and constitutes findings from spinal fluid and neurophysiology. The characteristic findings in CSF include a protein level greater than 0.55 g/l and fewer than 10 WBCs commonly known as "albuminocytological dissociation" [6]. EMG/NCV testing could be further used to exclude other causes of muscle weakness from GBS [7]. These diagnostic techniques used in our patient showed results that are highly suggestive of AIDP/GBS. The management of GBS is mainly immunotherapy treatments with plasmapheresis and IVIG [5]. Both treatments work to neutralize the harmful antibodies attacking the

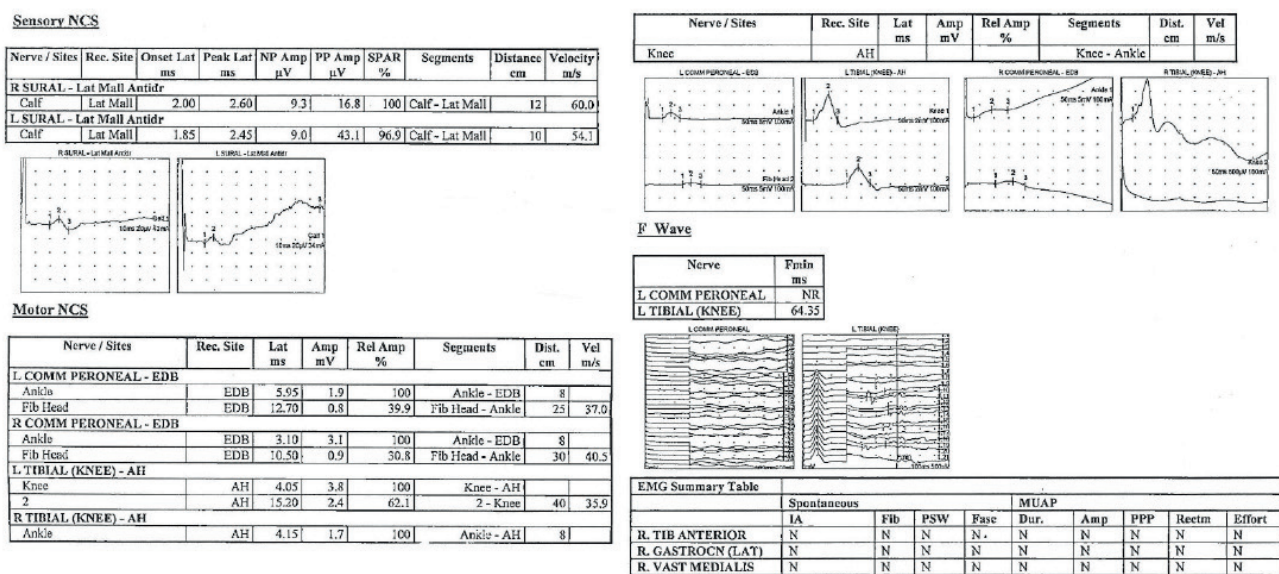


Figure 1. EMG/NCV study of the patient demonstrating abnormal partial and complete conduction block in multiple motor nerves and sparing of the sural nerves highly suggestive of an AIDP/GBS. Lat Mall: lateral malleolus, EDB: extensor digitorum brevis, AH: abductor hallucis, MUAP: motor unit potential, IA: insertional activity Fib: fibrillation potentials, PSW: positive sharp waves, Fasc: fascination potential, Dur: duration, Amp: amplitude, PPP: polyphasic potentials, Rectum: recruitment pattern.

peripheral nervous system and are effective modalities. Our patient was treated with both therapies, unfortunately, with her aging and immunocompromised nature, the IVIG did not work efficiently. However subsequent plasmapheresis therapy showed adequate improvement in her symptoms.

COVID-19 infection stimulates the inflammatory cytokines causing endothelial cell injury resulting in acute respiratory distress syndrome as the major cause of death in many patients [7]. However, this virus has been rapid in affecting almost every other body system including the nervous system causing initial neurological symptoms as anosmia and ageusia [6]. There have been many cases reported of COVID-19 association with GBS having a wide spectrum of etiologies [7]. The mechanism of action is still unclear. The explanation of other viral and bacterial causes of GBS is post-infectious molecular mimicry and cross-reactivity [1-7]. The view applied here would be that SARS-CoV-2 has certain gangliosides on its surface, which resemble gangliosides found in the peripheral nervous system [6]. The host body inadvertently attacks the gangliosides on the peripheral nervous system when it mounts an immune response to the SARS-CoV-2 surface ganglioside [6]. A recent study by Keddie et al. has partially challenged this theory by showing no significant homology between the SARS-CoV-2 and human linear protein structures; however, the same study suggested viral protein modifications within host cells leading to surface protein homology [8]. In our patient, the common respiratory flu-like illness that turned out positive for COVID-19 further led to having triggered GBS within a few weeks.

It is an interesting finding that many patients affected with COVID-19 were found to have elevated troponin levels [9]. This finding has a direct association with cardiac injury thereby highly associated with mortality rates in COVID-19 patients [9,10]. The theory relies on the role of ACE2, the binding receptor for SARS-CoV-2 cellular entry [9]. Angiotensin-converting enzyme 2 (ACE2) is highly expressed in cardiac pericytes and pulmonary cells serving a protective role in their pathophysiology [9]. In addition to using ACE2 as an initial entry point, the virus also downregulates its expression resulting in reduced conversion of angiotensin II (Ang-II) to angiotensin I (Ang-I) [10]. This leads to detrimental cardiac injury by affecting the renin-angiotensin-aldosterone system leading to complications such as myocarditis, congestive heart failure, and even myocardial infarction in COVID-19 patients [10]. Similarly, in our patient, elevated troponin levels were found which could have a role in the rapid weakness and deterioration of her condition. It could also be a possible reason that the cardiac injury made her immune system too weak to respond to the initial treatment of GBS leading to its failure.

Conclusion

The acute phase of COVID-19 infection usually affects the respiratory system and cardiovascular system as seen in our patient. The case report also indicates that GBS is considered as a neurological complication seen in convalescent phase or post recovery phase of an acute COVID-19 infection. With the increasing correlation between COVID-19 and GBS, clinicians must be aware of and include GBS in the differential diagnosis when a patient with history of COVID-19 infection presents with neurological symptoms. It is also essential for clinicians to diagnose GBS early and treat it properly, especially in elderly patients and patients with comorbidities, to improve the chance of successful recovery in view of potential complications of acute respiratory and cardiovascular failures.

What is new?

The association between COVID-19 infection and Guillain-Barre syndrome has been growing since the outbreak in January 2020. More severe presentation and prolonged recovery are seen in elderly/immunocompromised patients; therefore, prevention and treatment in a timely manner are crucial. Clinicians should be aware of neurological complications of an acute COVID-19 infection and include GBS in the differential diagnoses.

List of Abbreviations

ACE2	Angiotensin-converting enzyme 2
AIDP	Acute inflammatory demyelinating polyneuropathy
COVID-19	Coronavirus disease 2019
CSF	Cerebrospinal fluid
EMG/NCV	Electromyography/nerve conduction velocity
GBS	Guillain-Barré syndrome
LE	Lower extremity
PT/OT	Physical and occupational therapy
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2

Conflict of interests

The authors declare that there is no conflict of interests regarding the publication of this case report.

Funding

None.

Consent for publication

Written informed consent was taken from the patient.

Ethical approval

Ethical approval is not required at our institution for publishing an anonymous case report.

Author details

Enkhmaa Luvannyam¹, Arathi Jayaraman², Molly Sanjay Jain², Karan Sharma³, Manoj Reddy Somagutta¹, Rathna Kumar Vallapragada⁴

1. Internal Medicine, Surgery Department, Avalon University School of Medicine, Willemstad, Curacao
2. Internal Medicine, Saint James School of Medicine Park Ridge, Illinois

3. Internal Medicine, Windsor University School of Medicine, Cayon Saint Kitts and Nevis
4. Internal Medicine, AMITA Health Adventist Medical Center Hinsdale, Illinois

References

1. Virani A, Rabold E, Hanson T, Haag A, Elrufay R, Cheema T, et al. Guillain-Barré syndrome associated with SARS-CoV-2 infection. *IDCases*. 2020;20:e00771. <https://doi.org/10.1016/j.idcr.2020.e00771>
2. Sedaghat Z, Karimi N. Guillain Barre syndrome associated with COVID-19 infection: a case report. *J Clin Neurosci*. 2020;76:233–5. <https://doi.org/10.1016/j.jocn.2020.04.062>
3. Zhao H, Shen D, Zhou H, Liu J, Chen S. Guillain-Barré syndrome associated with SARS-CoV-2 infection: causality or coincidence? *Lancet Neurol*. 2020;19(5):383–4. [https://doi.org/10.1016/S1474-4422\(20\)30109-5](https://doi.org/10.1016/S1474-4422(20)30109-5)
4. Dalakas MC. Guillain-Barré syndrome: the first documented COVID-19-triggered autoimmune neurologic disease: more to come with myositis in the offing. *Neurol Neuroimmunol Neuroinflamm*. 2020;7(5):e781. <https://doi.org/10.1212/NXI.0000000000000781>
5. Rahimi K. Guillain-Barre syndrome during COVID-19 pandemic: an overview of the reports. *Neurol Sci*. 2020;41(11):3149–56. <https://doi.org/10.1007/s10072-020-04693-y>
6. Bueso T, Montalvan V, Lee J, Gomez J, Ball S, Shoustari A, et al. Guillain-Barre syndrome and COVID-19: a case report. *Clin Neurol Neurosurg*. 2021;200:106413. <https://doi.org/10.1016/j.clineuro.2020.106413>
7. Abu-Rumeileh S, Abdelhak A, Foschi M, Tumani H, Otto M. Guillain-Barré syndrome spectrum associated with COVID-19: an up-to-date systematic review of 73 cases. *J Neurol*. 2021;268(4):1133–70. <https://doi.org/10.1007/s00415-020-10124-x>
8. Keddie S, Pakpoor J, Mousele C, Pipis M, Machado PM, Foster M, et al. Epidemiological and cohort study finds no association between COVID-19 and Guillain-Barré syndrome. *Brain*. 2021;144(2):682–93. <https://doi.org/10.1101/2020.07.24.20161471>
9. Majure DT, Gruberg L, Saba SG, Kvasnovsky C, Hirsch JS, Jauhar R et al. Usefulness of elevated troponin to predict death in patients with COVID-19 and myocardial injury. *Am J Cardiol*. 2021;138:100–6. <https://doi.org/10.1016/j.amjcard.2020.09.060>
10. Tersalvi G, Vicenzi M, Calabretta D, Biasco L, Pedrazzini G, Winterton D. Elevated troponin in patients with coronavirus disease 2019: possible mechanisms. *J Card Fail*. 2020;26(6):470–5. <https://doi.org/10.1016/j.cardfail.2020.04.009>

Summary of the case

1	Patient (gender, age)	Female, 83 years old
2	Final diagnosis	COVID-19 infection, GBS
3	Symptoms	Fever, cough, fatigue, LE pain, numbness and tingling
4	Medications	IV fluids with minerals, multivitamin, analgesic, antipyretic, IVIG
5	Clinical procedure	Chest radiography, ECG, CT and US of LE, EMG/NCV, lumbar puncture, MRI of the brain and entire spine, plasmapheresis
6	Specialty	Internal medicine