Journal of Young Pharmacists, 2024; 16(1):130-133.
https://www.jyoungpharm.org

Case Report

Guillain-Barré Syndrome Post COVID-19 Infection: A Case Report

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ABSTRACT

COVID-19 primarily impacts the respiratory system, with neurological manifestations often being thrombotic and affecting the nervous system. However, the specific presentation of demyelinating manifestations remains less clearly defined. While recent research has established a connection between COVID-19 and Guillain-Barré syndrome-a complex neurological disorder characterised by acute or chronic degeneration-the extent of this association and the distinctive features of GBS within this context remain uncertain. In this report, we present the case of acute GBS suspected to be induced by a COVID-19 infection. Notably, this patient did not exhibit any preceding respiratory, gastrointestinal, or systemic illnesses. Consequently, this case underlines the critical importance of acknowledging the potential risk of Guillain-Barré Syndrome following a COVID-19 infection, making a significant contribution to raising awareness about this potential association.

Keywords: Guillain-Barré syndrome, COVID-19, Polyradiculoneuritis, SARS-CoV-2.

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Received: 07-10-2023;
Revised: 18-10-2023;
Accepted: 15-11-2023.

INTRODUCTION

Guillain-Barré Syndrome (GBS) is a rare but life-threatening autoimmune neurological condition characterized by sudden flaccid paralysis and occasionally accompanied by rapid onset of muscular weakening and paralysis. GBS is defined as an acquired demyelinating polyneuropathy, which occurs when the immune system incorrectly targets peripheral nervous system components, resulting in severe inflammation, demyelination, and axonal damage. The disease is usually caused by an infectious trigger, most commonly viral or bacterial infections, but it can also occur after immunisation or surgery.[1,2] GBS pathogenesis includes a complicated interaction between the immune system, peripheral nerves, and the infectious agent. It is well known that molecular mimicry plays a critical role in causing GBS, where a structural resemblance between particular pathogen-derived antigens and components of peripheral neurons leads to cross-reactive immune responses. Activated immune cells, such as T-cells and B-cells, invade peripheral nerve tissue, causing inflammation and demyelination of nerve fibres, as well as axonal injury in some circumstances.[3] Numerous infectious agents have been proposed as potential triggers for GBS, encompassing pathogens such as Campylobacter jejuni, Epstein-Barr Virus (EBV), Cytomegalovirus (CMV), and Zika virus.[4] Neurological symptoms and complications associated with SARS-CoV-2 have been documented as well.[5] Several occurrences of Guillain-Barré Syndrome (GBS) have been documented after COVID-19 infection, as evidenced by various case reports and series. These instances shed light on a plausible correlation between the two conditions. Notably, these reports have highlighted that GBS tends to manifest several weeks after the initial COVID-19 infection, suggesting a potential association with a post-infectious immune system disturbance prompted by the preceding bout of COVID-19.[6,7] We describe a noteworthy and clinically rare case involving a 68 years old male patient who presented with indications of a potential COVID-19 infection. This patient's chief complaints revolved around the gradual onset of weakness in both his upper and lower limbs. After describing this case, we will provide insightful remarks and observations.

CASE PRESENTATION

A male patient aged 68, devoid of any known comorbidities and without any relevant history of medication use, arrived at the emergency department of Vivekananda General Hospital in Hubballi, Karnataka, India. He presented with complaints of vomiting and fever spanning three days, accompanied by a progressive weakening of both his upper and lower limbs. The patient exhibited afebrile status and was oriented to time, place, and person at presentation. His vitals were as follows: pulse rate: 100 beats per min, respiratory rate: 16 breaths per min, blood

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DOI: 10.5530/jyp.2024.16.17

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pressure: 90/40 mmHg, and his oxygen saturation level registered at 92% while breathing ambient air. The patient's Glasgow Coma Scale (GCS) score was 13 out of 15. In the view of hypotension, 1 unit of noradrenaline infusion was started. A COVID-19 real-time Reverse Transcription-Polymerase Chain Reaction (RT-PCR) test yielded negative results.

According to the patient's caregiver, the patient experienced the onset of a fever approximately one month prior. The fever was mild and sporadic and lacked association with chills, rigours, or an elevation in temperature in the evening. The patient was afflicted by a dry cough and a persistent headache as well. In response, a local physician prescribed paracetamol 650 mg twice daily for three days and a course of azithromycin 500 mg taken once daily for three days. However, at that time, the patient had not undergone COVID-19 testing. A month later, the patient experienced a recurrence of fever along with the emergence of weakness in both his upper and lower limbs. Subsequently, the patient was referred to our hospital to receive comprehensive care and management.

The examination of the central nervous system revealed indications of quadriparesis. The examination revealed an absence of deep tendon reflexes, while the plantar reflex elicited a flexor response on both sides. Additionally, the patient exhibited paraesthesia, primarily affecting the distal lower limbs on both sides. The patient's superficial sensory functions displayed no abnormalities. The patient did not exhibit any impairment of deep sensory functions. The examination of cranial nerves yielded normal outcomes, and there were no indications of sensory spinal level engagement or any signs suggestive of meningeal irritation.

On evaluation, his arterial blood gas showed metabolic acidosis with an increased lactate of 8.8 mmol/L. Haematological tests showed Hb levels of 12.2 gm%, TC was 12160, Platelet count was 17,100 cells/mm^3, and ESR levels of 70 mm/hr. His biochemical parameters revealed CRP levels of 300 mg/L, and LDH levels of 312 U/L. The electrolyte profile revealed sodium levels of 135 mmol/L and chloride levels of 96 mmol/L. LFT showed increased levels of total bilirubin (6.8 mg/dL), direct bilirubin (5.2 mg/dL), AST (136 U/L), ALT (84 U/L), ALP (203 U/L), Total protein (5.7 g/dL), and Albumin (2.7 g/dL). The procalcitonin level of the patient was 100 ng/mL. His RFT was abnormal (Urea: 87 mg/dL, creatinine-serum: 2.87). Dengue serology, Malaria, and Weil Felix tests were negative and HIV I and II, HBsAg and HCV were non-reactive.

X-ray of the chest showed multifocal homogeneous opacities in bilateral lung fields. Computed tomography of the thorax revealed multifocal areas of consolidation and ground glass densities with interstitial thickening and subpleural bands, predominantly in the peripheral distribution of both lungs involving all lobes-features were suspicious for COVID-19 infection (CO-RADS score of 4). The total CT severity score was 13/25. A 2D Echo showed LVEF: 56%, no RWMA. The results of the Nerve Conduction Study (NCS) showed that Distal Motor Latency (DML) was prolonged along with the diminished amplitude of Compound Muscle Action Potentials (CMAP) across both the median and ulnar nerves on both sides of the body, as well as in the peroneal and tibial nerves. Additionally, F-wave latency was prolonged in both of his upper extremities. These observed outcomes collectively suggested the presence of a demyelinating polyneuropathy that affected both upper and lower limbs. Notably, the lower limbs exhibited a more prominent impact compared to the upper limbs.

A diagnosis of GBS-Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) due to suspected COVID-19 infection was made based on the findings described above. Because the patient presented early to the hospital, his progressive lower limb weakness was treated with IV Immunoglobulin (IVIG) at 0.4 g/kg/day for 6 days. Because of acute renal injury, two sessions of dialysis were done. He was given 1 unit of Single Donor Platelet (SDP) and 6 units of Random Donor Platelet (RDP) transfusion to treat thrombocytopenia. The patient was started on IV fluids, IV antibiotics (Meropenem), IV Proton Pump Inhibitors, IV Corticosteroids, IV Analgesics and other supportive care. He underwent thorough physiotherapy throughout his hospitalization period. The patient was stabilized. His condition improved symptomatically and was discharged with appropriate medical advice.

DISCUSSION

During the recent pandemic, there is also mounting evidence that SARS-CoV-2 infection is linked to immune-mediated neurological sequelae, such as GBS.[8,9] A study conducted in Northern Italy reported a notably elevated occurrence of patients with GBS during the COVID-19 outbreak, as well as an increased presence of GBS patients diagnosed with COVID-19. This underscores the potential role of SARS-CoV-2 in triggering GBS.[10,11] The ongoing research aims to unravel the underlying pathophysiology connecting COVID-19 and GBS. GBS is a type of inflammatory condition which is distinguished by an unusual host reaction towards infections, which results in molecular mimicry and causes nerve root damage. This deviant host response involves processes such as activating the complement system, followed by macrophage infiltration, and inflammation of the nerve roots and cell bodies peripherally.[12,13] GBS majorly affects males (68.9%), underscoring the gender-specific epidemiological pattern associated with COVID-19. The median age of affected patients spanned from 49 to 70 years.[14] GBS is recognized to manifest in four distinct clinical forms or variants and the most prominent form is Acute Inflammatory Demyelinating Polyradiculoneuropathy or AIDP.[15,16] Our diagnosis was verified by a nerve conduction investigation, which indicated an AIDP form of GBS.

Existing literature demonstrates that the prevailing manifestation of COVID-19-induced GBS is the conventional sensorimotor GBS type. Furthermore, the majority of cases fulfilled the electrophysiological criteria aligning with the AIDP diagnosis. Although a few instances reported GBS with a para-infectious onset, the bulk of patients exhibited a post-infectious GBS onset. Consequently, GBS stemming from COVID-19 appears to adhere to similar patterns observed in the conventional post-infectious GBS attributed to other infectious agents.[17] IVIG or plasma exchange are two therapy possibilities. IVIG is thought to function by regulating the immune system. IVIG is given at a dose of 2 g/kg every five days. In contrast to the current patient, who exhibited significant improvement following the Intravenous Immunoglobulin (IVIG) treatment, a patient in the United Kingdom diagnosed with para-infectious GBS attributed to COVID-19 did not respond favourably to IVIG. Instead, this patient experienced a progression of weakness, and a decline in respiratory vital capacity, and necessitated intubation with Intensive Care Unit (ICU) admission. The exacerbation of such cases might stem from either a direct infiltration of toxins from the COVID-19 virus or could be linked to the severity of pneumonia. However, it is noteworthy that the response to IVIG or plasma exchange did not exhibit a distinction between para-infectious and post-infectious GBS in COVID-19 cases, based on the limited number of cases documented in the available literature.[6,18]

Due to the rising number of individuals experiencing post-COVID-19 sequelae, it is imperative to exercise caution when dealing with patients who exhibit neurological symptoms. While the exact nature of the connection between COVID-19 and GBS remains uncertain, a mere correlation should not be equated with a definitive cause.[18] The current case implies that GBS might emerge as a neurological complication after a COVID-19 infection. It is noteworthy that despite our patient testing negative for the virus through RT-PCR testing, his CT scan exhibited notable COVID-19-related abnormalities (CO-RADS score of 4).

CONCLUSION

With the rising recognition of post-COVID-19 Guillain-Barré Syndrome (GBS), there is a growing need for more comprehensive studies to comprehensively define the nature of this condition, including its clinical manifestations. Particularly, a thorough investigation into the efficacy of Intravenous Immunoglobulin (IVIG) or plasma exchange in both para-infectious and post-infectious GBS cases is warranted. Consequently, it holds crucial significance to actively document instances of GBS triggered by COVID-19. These case reports contribute to the accumulation of a global body of evidence, imperative for formulating the most effective management strategies for similar cases in the future.

ACKNOWLEDGEMENT

The authors would like to thank the invaluable contributions of Dr. Bharati Kangrali (MBBS, MD, General Medicine) in the thorough clinical assessment and clerking of the case presented in this case report.

CONFLICTS OF INTEREST

The authors declare that there is no conflict of interest.

PATIENT CONSENT

The patient's written informed consent was obtained for the publication of this case report.

ABBREVIATIONS

AIDP: Acute Inflammatory Demyelinating Polyradiculoneuropathy; **ALP:** Alkaline phosphatase; **ALT:** Alanine transaminase; **AST:** Aspartate transaminase; **CMAP:** Compound Muscle Action Potentials; **CMV:** Cytomegalovirus; **CO-RADS:** COVID-19 Reporting and Data System; **CRP:** C-reactive Protein; **CT:** Computed Tomography; **EBV:** Epstein-Barr virus; **ESR:** Erythrocyte Sedimentation Rate; **GBS:** Guillain-Barré syndrome; **GCS:** Glasgow Coma Scale; **Hb:** Haemoglobin; **IVIG:** Intravenous Immunoglobulin; **LDH:** Lactate dehydrogenase; **LFT:** Liver Function Test; **NCS:** Nerve Conduction Studies; **RFT:** Renal Function Test; **RT-PCR:** Real-Time Reverse Transcription-Polymerase Chain Reaction; **TC:** Total Count.

REFERENCES

1. Kaeley N, Kabi A, Pillai A, Shankar T, Ameena M S S. Post-COVID-19 Guillain-Barré syndrome: a case report with literature review. Cureus. 2022;14(1):e21246. doi: 10.7759/cureus.21246, PMID 35178309.
2. Yuki N, Hartung HP. Guillain-Barré syndrome. N Engl J Med. 2012;366(24):2294-304. doi: 10.1056/NEJMra1114525, PMID 22694000.
3. van den Berg B, Walgaard C, Drenthen J, Fokke C, Jacobs BC, van Doorn PA. Guillain-Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. Nat Rev Neurol. 2014;10(8):469-82. doi: 10.1038/nrneurol.2014.121, PMID 25023340.
4. Levin KH. Variants and mimics of Guillain Barré syndrome. Neurologist. 2004;10(2):61-74. doi: 10.1097/01.nrl.0000117821.35196.0b, PMID 14998436.
5. Paterson RW, Brown RL, Benjamin L, Nortley R, Wiethoff S, Bharucha T, *et al.* The emerging spectrum of COVID-19 neurology: clinical, radiological and laboratory findings. Brain. 2020;143(10):3104-20. doi: 10.1093/brain/awaa240, PMID 32637987.
6. Webb S, Wallace VC, Martin-Lopez D, Yogarajah M. Guillain-Barré syndrome following COVID-19: a newly emerging post-infectious complication. BMJ Case Rep. 2020;13(6):e236182. doi: 10.1136/bcr-2020-236182, PMID 32540883.
7. Zito A, Alfonsi E, Franciotta D, Todisco M, Gastaldi M, Cotta Ramusino M, *et al.* COVID-19 and Guillain-Barré syndrome: a case report and review of literature. Front Neurol. 2020;11:909. doi: 10.3389/fneur.2020.00909, PMID 32973665.
8. Frye BC, Meiss F, von Bubnoff D, Zissel G, Müller-Quernheim J. Vasoactive intestinal peptide in checkpoint inhibitor-induced pneumonitis. N Engl J Med. 2020;382(26):2573-4. doi: 10.1056/NEJMc2000343, PMID 32579820.
9. Munhoz RP, Pedroso JL, Nascimento FA, De Almeida SM, Barsottini OGP, Cardoso FEC, *et al.* Neurological complications in patients with SARS-CoV-2 infection: a systematic review. Arq Neuro Psiquiatr. 2020;78(5):290-300. doi: 10.1590/0004-282x20200051, PMID 32490966.
10. Filosto M, Cotti Piccinelli S, Gazzina S, Foresti C, Frigeni B, Servalli MC, *et al.* Guillain-Barre´ Syndrome and COVID-19: an observational multicentre study from two Italian hotspot regions. J Neurol Neurosurg Psychiatry. 2021;92(7):751-6. doi: 10.1136/jnnp-2020-324837, PMID 33158914.

11. Luijten LWG, Leonhard SE, Van Der Eijk AA, Doets AY, Appeltshauser L, Arends S, *et al.* Guillain-Barré syndrome after SARS-CoV-2 infection in an international prospective cohort study. Brain. 2021;144(11):3392-404. doi:10.1093/brain/awab279, PMID 34553216.
12. Zuo Y, Yalavarthi S, Shi H, Gockman K, Zuo M, Madison JA, *et al.* Neutrophil extracellular traps in COVID-19. JCI Insight. 2020;5(11). doi: 10.1172/jci.insight.138999, PMID 32329756.
13. Moore JB, June CH. Cytokine release syndrome in severe COVID-19. Science. 2020;368(6490):473-4. doi: 10.1126/science.abb8925, PMID 32303591.
14. Papri N, Hayat S, Mohammed A, Afsar MNA, Hasan I, Rahman A, *et al.* Guillain-Barré syndrome associated with SARS-CoV-2 infection: A case report with long term follow up. J Neuroimmunol. 2021;356:577590. doi: 10.1016/j.jneuroim.2021.577590, PMID 33957540.
15. Expósito J, Carrera L, Natera D, Nolasco G, Nascimiento A, Ortez C. Síndrome de Guillain-Barré y otras neuropatías autoinmunes: tratamiento actual [Guillain-Barré syndrome and other autoimmune neurophaties: current therapy]. Med (B Aires). 2022; 82;Suppl 3: 82-8. Spanish. PMID 36054864.
16. Hadden RDM, Cornblath DR, Hughes RAC, Zielasek J, Hartung H-P, Toyka KV, *et al.* Plasma exchange/Sandoglobulin Guillain-Barré syndrome trial group. Ann Neurol. 1998;44(5):780-8. doi: 10.1002/ana.410440512, PMID 9818934.
17. 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. doi: 10.1007/s00415-020-10124-x, PMID 32840686.
18. Carpenter K, Iqbal A, Singh R, Deepika K, Koritala T, Jain N, *et al.* COVID-19 infection and Guillain-Barre syndrome: a case series. Cureus. 2022;14(2):e21998. doi: 10.7759/cureus.21998, PMID 35282522.

Cite this article: Hegde M, Raj S, Tikadar D, Nyamagoud SB. Guillain-Barré Syndrome Post COVID-19 Infection: A Case Report. J Young Pharm. 2024;16(1):130-3.