

## Post-Infectious Guillain Barré Syndrome Related to COVID-19: Unusual Case Report

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DOI: [10.36347/sjmcr.2024.v12i06.008](https://doi.org/10.36347/sjmcr.2024.v12i06.008)

| Received: 21.04.2024 | Accepted: 29.05.2024 | Published: 01.06.2024

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### Abstract

### Case Report

Guillain-Barré Syndrome (GBS) is a rare immune mediated inflammatory disease of the peripheral nervous system that is presumed to be triggered by preceding infections. In the era of pandemic COVID-19 (coronavirus disease), there is an emerging case of para/post infectious GBS associated to COVID-19 that are described worldwide. Here, we report a 55-years-old man who was admitted for fatigue, fever and cough related to covid-19. Later, after disappearing of covid-19 symptoms he began presenting an ascending areflexic tetraparesis. CSF and NCS were in favour of GBS diagnosis, in particular pure motor AIDP subtype. Antigangliosides antibodies were negative. Lumbosacral MRI showed contrast enhancement of cauda equina roots. The outcome was good after receiving intravenous immunoglobulins treatment.

**Keywords:** Guillain-Barré Syndrome, COVID-19, post-infectious, Sars-cov2, virus.

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## INTRODUCTION

Coronavirus disease (COVID-19) is an infectious disease caused by a novel member of human RNA coronavirus called SARS-Cov-2 (severe acute respiratory syndrome coronavirus 2). It's a global pandemic disease that emerged from Wuhan city, Hubei province of China in December of 2019 [1]. The phenotypes of COVID-19 range from asymptomatic patients to severe life-threatening respiratory failure, systemic dysfunction, and death [1]. The most common symptoms include mild flu-like syndrome [2]. The neurological complications related to covid19 are reported worldwide and could involve peripheral and central nervous system [3]. Guillain-Barré Syndrome (GBS) is an inflammatory polyradiculoneuropathy that has been described in association with COVID-19 in several case reports and small series [4]. Filosto and et al. reported an increasing of GBS cases during the outbreak of COVID-19 in northern Italy between March 2020 and April 2020 and they were greater than five times that reported in the last 3 years [5]. The cases of GBS associated to COVID-19 are emerging but they are described especially in country with high and upper middle income [4]. However, this association is under reported in developing countries like Morocco [4]. To date, a single case of this association has been published in April 2020 that concerned a 70-years-old woman with

axonal subtype in Morocco [6]. Here, we report a second documented case of post infectious GBS related to COVID-19 and it's the first demyelinating subtype in our country.

## CASE REPORT

A 55-years-old man with controlled diabetes mellitus presented to the emergency department with history of fever, cough and fatigue that were evolving for 4 days. On clinical examination, he was hemodynamically stable with 99% oxygen saturation on room air. Lung CT scan showed bilateral ground glass opacities (fig. 1). COVID-19 testing was positive by reverse-transcriptase polymerase-chain-reaction (RT-PCR) technique in rhinopharyngeal swab. The patient was treated with Hydroxychloroquine and azithromycine. Clinical and biological improvement was seen at the fifth day of admission. Two days later (11 days after the onset of COVID-19 symptoms), the patient had begun experiencing progressive lower limb weakness that evolved to the upper limbs within 5 days. There was no suggestive history of sensory symptoms, fever, respiratory difficulty, bowel and bladder symptoms or swallowing disturbance. Eight days after the onset of limb weakness, the patient had lost the ability to walk independently. The Medical Research Council (MRC) Scale grade for muscle strength was 2/5 for

proximal and 1/5 for distal muscles of the lower limbs, and 3/5 for proximal and 2/5 for distal muscles of the upper limbs (MRC sum score was 25/60). Deep tendon reflexes were absent in all extremities with generalized hypotonia and positive Lasègue sign. Cranial nerves and sensory examination were normal. The study of CSF demonstrated a typical albumino-cytologic dissociation (ACD) (white-cell count of less than 5 per cubic millimetre, level of proteins at 0,75g/l (N < 0,45g/l)). The patient was tested for COVID-19 and has negative RT PCR in nasopharyngeal swab, and had positive serology for both Ig G and Ig M antibodies.

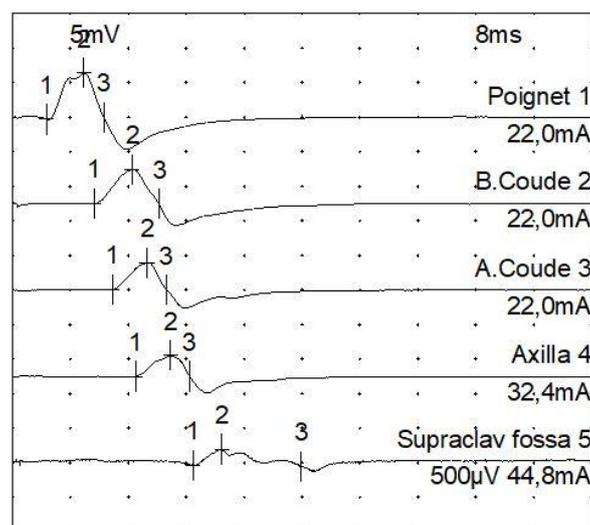
Routine blood investigations including complete blood count, serum sodium and potassium, creatine kinase enzyme, serum B12 levels, liver, kidney and thyroid function tests were normal. Viral markers including Human Immunodeficiency Virus, Hepatitis B Surface antigen and Hepatitis C Virus antibody were negative.

Electrophysiological study was performed (10 days after the onset of paralysis) using a Nicolet Viking EMG device. Motor Nervous Conduction Study has demonstrated demyelinating features (table 1): prolonged motor distal latencies, temporal dispersion, conduction blocks (fig. 2), and very slow velocities. F wave responses were absent in lower limbs, and prolonged in the upper limbs. The sensory nerve conduction study was normal. Therefore, a pure motor Acute Inflammatory Demyelinating Polyneuropathy (AIDP) diagnosis was evoked. Our patient was tested for anti-gangliosides antibodies that were absent in serum. Contrast-enhanced MR imaging of lumbosacral spine revealed enhancement of the cauda equina roots (Fig. 3). Brain MRI didn't show any significant abnormalities.

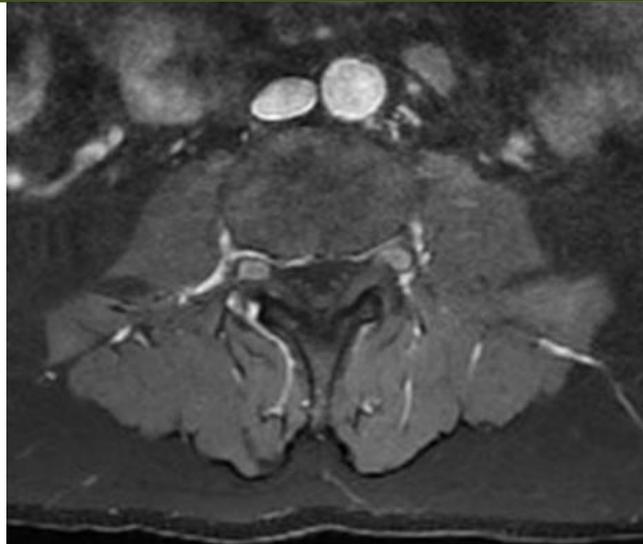
On day eleven after the onset of paralysis, the patient was treated with intravenous immunoglobulin (IVIg) at the dose of 0,4g/kg/day given over the course of 5 days. One week after treatment with IVIg, he showed a significant improvement of muscle strength and his MRC sum score at discharge was 43/60.



**Figure 1: Axial image of chest computed tomography revealing ground-glass opacities in both lungs**



**Figure 2: Motor Nervous Conduction Study of right ulnar nerve showing conduction block in below elbow-wrist segment and temporal dispersion in the erb's point-axilla segment**



**Figure 3: Axial T1 FAT-SAT post-contrast MRI of lumbosacral spine showing enhancement of cauda equina roots**

## DISCUSSION

We describe a case of GBS that appeared eleven days after confirmed COVID-19 infection. Neurological complications of COVID-19 are reported in several studies and among them we found numerous cases of GBS [7]. GBS is an immune-mediated response that may be preceded by an upper respiratory infection or gastroenteritis in two-thirds of the time [8]. The authors suggest that these infections incite an aberrant immune response against peripheral nerves and/or spinal nerve roots due to molecular mimicry [8].

The majority of cases published in literature had either para-infectious or post-infectious profile. Recently, the authors suggest a post-infectious immune-mediated mechanism, rather than a para-infectious etiology [4]. Our patient was recovering from COVID-19 and was asymptomatic for 2 days before developing areflexic ascending tetraparesis at the 11<sup>th</sup> day of covid-19 symptoms onset. During progression of paralysis, he was controlled negative for COVID-19 and had both Ig G and Ig M antibodies in serum. Giving these arguments and presence of ACD in CSF, a post-infectious process was more accurate.

The classical sensory-motor variants of GBS were the most reported cases related to COVID-19, while pure motor forms like that seen in our patient were described in only 4 patients [4]. Electrophysiological study of patients with GBS associated to COVID-19 had showed different subtype pattern; demyelinating, axonal and mixed forms [4]. Demyelinating forms of GBS are the most described in the context of SARS-Cov-2 infections [4].

Contrast enhancement of nerve roots may occur in GBS patients, especially in cases where the electrophysiology and spinal fluid characteristics are

equivocal for GBS [9]. In a systematic review of 73 patients with GBS associated to COVID-19, spinal MRI was performed in 23 patients and showed spinal nerve root enhancement in 8 cases [10]. Our case is a supplementary observation demonstrating that contrast enhancement of nerve roots may be seen in post infectious GBS related to COVID-19. Antigangliosides antibodies were negative in our study and most published cases [4].

Treatment of GBS associated to COVID-19 resembles to the standard treatment used in classic GBS. Among 61 patients with GBS related to SARS-Cov-2 included in meta-analysis; 23 (41,1%) patients were admitted to the Intensive care unit, 2 (3,8%) patients died while 34 (65,3%) had good outcome at discharge [4]. Our patient has shown significant improvement with IVIg and presented good outcome at discharge emphasizing their efficiency in GBS following COVID-19.

## CONCLUSION

The pandemic Covid-19 is still causing a serious overwhelming health burden and socio-economic problems. The causal relationship between SARS-cov-2 infection and GBS is not yet confirmed. However, physicians should be aware of this probable association for rapid diagnosis and early management of GBS in the context of COVID-19.

## REFERENCES

1. Guan, W. J., Ni, Z. Y., Hu, Y., Liang, W. H., Ou, C. Q., He, J. X., ... & Zhong, N. S. (2020). Clinical characteristics of coronavirus disease 2019 in China. *New England journal of medicine*, 382(18), 1708-1720.
2. Alberti, P., Beretta, S., Piatti, M., Karantzoulis, A., Piatti, M. L., Santoro, P., ... & Ferrarese, C. (2020). Guillain-Barré syndrome related to COVID-19

- infection. *Neurology: Neuroimmunology & Neuroinflammation*, 7(4), e741.
3. Korálnik, I. J., & Tyler, K. L. (2020). COVID-19: a global threat to the nervous system. *Annals of neurology*, 88(1), 1-11.
  4. Hasan, I., Saif-Ur-Rahman, K. M., Hayat, S., Papri, N., Jahan, I., Azam, R., ... & Islam, Z. (2020). Guillain-Barré syndrome associated with SARS-CoV-2 infection: A systematic review and individual participant data meta-analysis. *Journal of the Peripheral Nervous System*, 25(4), 335-343.
  5. Filosto, M., Piccinelli, S. C., Gazzina, S., Foresti, C., Frigeni, B., Servalli, M. C., ... & Uncini, A. (2021). Guillain-Barré syndrome and COVID-19: an observational multicentre study from two Italian hotspot regions. *Journal of Neurology, Neurosurgery & Psychiatry*, 92(7), 751-756.
  6. El Otmani, H., El Moutawakil, B., Rafai, M. A., El Benna, N., El Kettani, C., Soussi, M., ... & Afif, H. (2020). Covid-19 and Guillain-Barré syndrome: more than a coincidence!. *Revue neurologique*, 176(6), 518.
  7. Almqvist, J., Granberg, T., Tzortzakakis, A., Klironomos, S., Kollia, E., Öhberg, C., ... & Ineichen, B. V. (2020). Neurological manifestations of coronavirus infections—a systematic review. *Annals of clinical and translational neurology*, 7(10), 2057-2071.
  8. Willison, H. J., Jacobs, B. C., & van Doorn, P. A. (2016). Guillain-barre syndrome. *The Lancet*, 388(10045), 717-727.
  9. Gorson, K. C., Ropper, A. H., Muriello, M. A., & Blair, R. (1996). Prospective evaluation of MRI lumbosacral nerve root enhancement in acute Guillain-Barré syndrome. *Neurology*, 47(3), 813-817.
  10. Guan, W. J., Ni, Z. Y., Hu, Y., Liang, W. H., Ou, C. Q., He, J. X., ... & Zhong, N. S. (2020). Clinical characteristics of coronavirus disease 2019 in China. *New England journal of medicine*, 382(18), 1708-1720.