

Case report

A Pediatric Case of Post-SARS-CoV-2 Guillain Barre Syndrome of Pure Axonal Variety

Sanghamitra Ray', Manish Kumar²

¹Assistant Professor, Department of Paediatrics, Chacha Nehru Bal Chikitsalaya, Delhi, India.

INFO

Corresponding Author:

Sanghamitra Ray, Department of Paediatrics, Chacha Nehru Bal Chikitsalaya, Delhi, India.

E-mail Id:

dr.sanghamitra.ray@gmail.com
Orcid Id:

http://orchid.org/0000-0002-8247-9283 **How to cite this article**:

Ray S, Kumar M. A Pediatric Case of Post-SARS-CoV-2 Guillain Barre Syndrome of Pure Axonal Variety. Postgrad J Pediatr Adol Med. 2025;1(1):62-64.

Date of Submission: 2025-03-15 Date of Acceptance: 2025-04-25

ABSTRACT

Guillain-Barre syndrome (GBS) is a common entity in the pediatric population and a major cause of flaccid paralysis worldwide. Recently, during the COVID-19 pandemic, a surge in GBS cases has been observed, primarily in the adult population. Here, we present a case of a child with post-SARS-CoV-2 infection GBS of a rare variety. A five-year-old female child presented with weakness in both lower limbs for two months and complaints of numbness in her lower limbs. She had a history of fever three weeks prior to this admission. Along with universal areflexia and reduced power in both lower limbs, the cerebrospinal fluid analysis showed albuminocytological dissociation. A nerve conduction study (NCS) was performed, which was typical of GBS of the severe acute motor and sensory axonal neuropathy (AMSAN) type. The anti-COVID-19 antibody (IgM) was significantly high. The patient was successfully managed with plasmapheresis and showed complete recovery. This is a rare pediatric case of post-COVID-19 GBS of the AMSAN variety treated successfully with plasmapheresis.

Keywords: GBS, Pure Axonal Variety, Plasmapheresis, Universal Areflexia

Introduction

The most prevalent polyneuropathy in the pediatric population is Guillain Barre syndrome (GBS). Being post-infectious in origin, many infections have been implicated as causative factors of GBS over time. Many case reports have been published of GBS following SARS-CoV-2 infection in the adult population since the onset of the COVID-19 pandemic. This is a rare case of pediatric GBS of the AMSAN (acute motor and sensory axonal neuropathy) variety post-SARS-CoV-2 infection.

Case Presentation

A five-year-old girl presented in November 2021 with weakness in both lower limbs for 2 months, which was progressive for the initial month and static thereafter.

The child was developmentally normal with no significant past illness. The weakness was ascending, and at the time of admission, the child was unable to walk even with support, along with numbness and tingling in her lower limbs. There was no bladder or bowel involvement and no history suggestive of seizures or altered sensorium. She had a history of fever almost three weeks before the onset of weakness in her limbs. There was no history of animal bites or recent immunization. There was no family history of documented COVID-19 infections, but both parents had fever with cough for a week four weeks prior.

On examination, there was no cranial nerve involvement. There was generalized areflexia and reduced power in both lower limbs (3/5), whereas the power of the upper limbs was normal. Superficial reflexes were present, and

Postgraduate Journal of Pediatrics and Adolescent Medicine

²Professor, Department of Paediatrics, Chacha Nehru Bal Chikitsalaya, Delhi, India.

the plantar reflex was down-going. Based on the history and examination, a provisional diagnosis of acute flaccid paralysis with the first possibility of GBS was made.

A stool sample for polio was sent, which was negative. CSF examination revealed albuminocytological dissociation with a CSF protein level of 115 mg/dL and absent cells in the CSF. Subsequently, a nerve conduction study was done, which suggested the presence of severe acute motor and sensory axonal neuropathy. Sensory nerve action potential (SNAP) was absent in the bilateral sural nerve, and compound motor action potential (CMAP) was absent in the peroneal and tibial nerves.

To evaluate the cause of post-infectious polyneuropathy, an anti-COVID-19 antibody test was conducted as SARS-CoV-2 was the most common infection prevalent during that time period, along with other investigations such as viral markers and HIV. Blood culture and CSF cultures were sterile. Though the SARS-CoV-2 RT-PCR was negative in this case, the anti-COVID-19 antibody (IgM) was very high at 348 AU/mL (positive > 50 AU/mL), leading to a final diagnosis of post-COVID-19 GBS. A panel of inflammatory markers showed no evidence of MIS-C (multi-system inflammatory syndrome in children). Subsequently, plasmapheresis was planned as intravenous immunoglobulin (IVIG) was not available at our hospital due to a high demand for IVIG for MIS-C cases during that time. The patient underwent 5 cycles of alternate-day plasmapheresis with 1.5 times plasma volume exchange along with intensive physiotherapy, during which she showed gradual improvement in power. She was discharged after 3 weeks and is under regular follow-up. Within 1 month of discharge from the hospital, she regained normal power in both lower limbs and is asymptomatic at present after 3 months of follow-up.

Discussion

GBS is a polyradiculoneuropathy of post-infectious in origin and the causative agents are mostly some microbes causing gastrointestinal or respiratory infections. There is a presence of autoantibodies in GBS, which in turn, is the consequence of an immunological response of the body towards a microbiological agent which subsequently cross-reacts with an antigenically similar structure of peripheral nerve, resulting in polyradiculoneuropathy. Many microbiological agents have been associated with GBS, including Campylobacter jejuni, Cytomegalovirus, Epstein-Barr virus, Mycoplasma pneumoniae, etc.

Since early 2020, the onset of the pandemic caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2); a wide spectrum of diseases involving both central and peripheral nervous systems have been reported as a part of SARS-CoV-2 infection or as its aftereffects. From chronic headache to encephalitis, stroke are being reported

worldwide mostly in adults and a few cases in children. Coronaviruses are thought to cause GBS either directly through neuroinvasion (ACE2 receptors on neurons) or through aberrant immunological response.^{1,2}

The first ever post-SARS-CoV-2 GBS was reported in an elderly female who developed GBS of demyelinating variety after a visit to pandemic epicentre Wuhan.² Subsequently there were few reported in children.^{3–5} Recently a case of post-COVID acute motor axonal neuropathy was reported in an adolescent male from India.⁶

Plasmapheresis is a recommended therapeutic modality in GBS as per the standard recommendation. Intravenous immunoglobulin and plasmapheresis are equally effective in the treatment of GBS, and five sessions of plasma exchange are ideally recommended on an alternate-day regimen. Gajjar et al. published data on therapeutic plasma exchange in 40 pediatric patients of GBS from India which was shown to be an effective and safe method in this cohort of children.

As our patient presented with a longer duration of illness and with a less favourable axonal subtype, rapid and complete recovery with plasmapheresis has been a matter of hope. Further studies in similar kind of cases will help us gain further.

Conclusion

This case is one of the rarest cases of post covid-19 GBS of AMSAN variety in a child, who showed complete recovery with therapeutic plasma exchange. This case highlights the fact of considering COVID-19 as a preceding cause of GBS and the beneficial role of plasmapheresis wherever available.

Acknowledgement

SR collected data. SR and MK were both involved in patient care and manuscript preparation.

Conflict of Interest: None

References

- Zhou Z, Kang H, Li S, Zhao X. Understanding the neurotropic characteristics of SARS-CoV-2: from neurological manifestations of COVID-19 to potential neurotropic mechanisms. J Neurol. 2020;267(8):2179-84. [PubMed] [Google Scholar]
- Zhao H, Shen D, Zhou H, Liu J, Chen S. Guillain-Barre syndrome associated with SARS-CoV- 2 infection: causality or coincidence? Lancet Neurol. 2020;19(5):383-4. [PubMed] [Google Scholar]
- Curtis M, Bhumbra S, Felker MV, Jordan BL, Kim J, Weber M, Friedman ML. Guillain-Barré syndrome in a child with COVID-19 infection. Pediatrics. 2021;147(4):e2020015115. [PubMed] [Google Scholar]

- 4. Khalifa M, Zakaria F, Ragab Y, Saad A, Bamaga A, Emad Y, Rasker JJ. Guillain-Barré syndrome associated with severe acute respiratory syndrome coronavirus 2 detection and coronavirus disease 2019 in a child. J Pediatric Infect Dis Soc. 2020;9(4):510-3. [PubMed] [Google Scholar]
- 5. Rafiei Tabatabaei S, Shariatpanahi G, Azadkiya R, Karimzadeh P, Karimi A. Guillain-Barre syndrome in a child infected with COVID-19. Arch Pediatr Infect Dis. 2022;10(Suppl):e113564. [Google Scholar]
- Krishnakumar A, Kewalramani D, Mahalingam H, Manokaran RK. Guillain-Barré syndrome with preserved reflexes in a child after COVID-19 infection. Indian J Pediatr. 2021;88(8):831-2. [PubMed] [Google Scholar]
- 7. Padmanabhan A, Connelly-Smith L, Aqui N, Balogun RA, Klingel R, Meyer E, Pham HP, Schneiderman J, Witt V, Wu Y, Zantek ND, Dunbar NM, Schwartz J. Guidelines on the use of therapeutic apheresis in clinical practice—evidence-based approach from the Writing Committee of the American Society for Apheresis: the eighth special issue. J Clin Apher. 2019;34(3):171-354. [PubMed] [Google Scholar]
- 8. Malek E, Salameh J. Guillain-Barre syndrome. Semin Neurol. 2019;39(5):589-95. [PubMed] [Google Scholar]
- Gajjar M, Patel T, Bhatnagar N, Solanki M, Patel V, Soni S. Therapeutic plasma exchange in pediatric patients of Guillain-Barre syndrome: experience from a tertiary care centre. Asian J Transfus Sci. 2016;10(1):98-100. [PubMed] [Google Scholar]