International Journal of Contemporary Pediatrics
Gupta A et al. Int J Contemp Pediatr. 2025 Sep;12(9):1538-1542
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20252606

Case Series

Study of clinical profile of Guillain-Barré syndrome in children upto 12 years during an epidemic outbreak

Anuradha Gupta, Manjunath Awadkhan, Sangita Kumbhojkar, Maheshwari Jadhav*, Bhushan Miraje

Department of Pediatrics, RCSM GMC, Kolhapur, Maharashtra, India

Received: 15 July 2025
Accepted: 07 August 2025

***Correspondence:**
Dr. Maheshwari Jadhav,
E-mail: mahi.kims44@gmail.com

ABSTRACT

Guillain-Barré syndrome (GBS) is an acute inflammatory polyneuropathy and leading cause of Acute flaccid paralysis (AFP) in children. Outbreaks are often linked to infections, but regional data on paediatric GBS in India remains limited. To describe the clinical presentation of paediatric GBS cases admitted in RCSM GMC Kolhapur during an outbreak that occurred in Maharashtra, India in 2025. To correlate electrophysiological findings with clinical profile and outcomes of paediatric GBS cases admitted in RCSM GMC Kolhapur during an outbreak that occurred in Maharashtra, India in 2025. This is a descriptive observational case series of 5 children admitted with GBS during an outbreak. Cases were diagnosed using Brighton Collaboration criteria and nerve conduction velocity (NCV) studies. Clinical features, antecedent infections, CSF (Cerebrospinal fluid) analysis, and treatment responses were recorded. Samples were tested at the National Institute of Virology (NIV), Pune. Outcomes were assessed using the Hughes Functional Grading Scale. Five cases (3 male, 2 female) showed variable severity, with antecedent infections in four. NCV revealed AIDP (4 cases) and AMAN (1 case). Two patients required ventilation, one developed bradycardia, another ventilator-associated pneumonia. Intravenous Immunoglobulin (IVIG) (2 g/kg) was effective, with three achieving full recovery and two having residual weakness. *Campylobacter jejuni* was detected in one case. Paediatric GBS during outbreaks presents with diverse severity but responds well to IVIG. Early diagnosis and monitoring of respiratory/cranial involvement are crucial. Strengthening surveillance and investigating infectious triggers is recommended.

Keywords: GBS, IVIG, Nerve conduction velocity study, AFP

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive ascending weakness, areflexia, and sensory disturbances.[1] It is the most common cause of acute flaccid paralysis (AFP) in children, often preceded by infections such as *Campylobacter jejuni*, cytomegalovirus, or respiratory and gastrointestinal pathogens.[2] Although GBS is rare in children, its clinical presentation and outcomes can vary significantly, particularly during outbreaks linked to infectious triggers.[3] In recent years, outbreaks of GBS have been reported in association with viral infections, including Zika virus, dengue, and chikungunya, raising concerns about geographical and seasonal variations in disease presentation.[4] In India, post-infectious GBS has been documented following enterovirus, dengue, and other arboviral infections, with varying degrees of severity.[5]

The clinical course in children is milder than in adults, with a higher probability of complete recovery but also potential for severe complications such as respiratory failure and autonomic dysfunction.[6] Kolhapur, a region in Maharashtra, has witnessed sporadic cases of GBS, often coinciding with seasonal outbreaks of infectious diseases.

However, there is limited data on pediatric GBS cases from this region, particularly during outbreak periods. Understanding the clinical spectrum, antecedent infections, and outcomes in children is crucial for early diagnosis, appropriate management and prognostication.[7] This case series aims to describe the clinical presentation, electrophysiological findings, and outcomes of five pediatric GBS cases during an outbreak in Kolhapur, Maharashtra in 2025 to enhance local awareness and management strategies. Samples of all these patients were processed at National institute of virology (NIV), Pune in order to find the cause of the outbreak.

The is a descriptive observational case series conducted at a tertiary care hospital RCSM GMC Kolhapur, Maharashtra. The study proceeded with detailed clinical evaluation of 5 cases including history of antecedent infections, neurological examination, laboratory investigations, complications and outcome. Nerve conduction velocity (NCV) studies were performed in all cases to confirm the diagnosis and classify GBS subtypes. Additionally, serum, stool and CSF samples were sent to NIV, Pune for virological testing to identify potential infectious triggers. The findings were compared with existing literature to assess regional variations in GBS presentation during outbreaks.

CASE SERIES

Case 1

Ten years old male presented with progressive symmetrical weakness, initially in both lower limbs and later in upper limbs, following acute gastroenteritis 8 days prior. On admission, he was alert with SpO_2 90% on room air. Neurological examination showed bilateral lower motor neuron (LMN) type facial palsy, hypotonia, areflexia, power in lower limbs was MRC (Medical Research Counsil) grade 0/5 and in upper limbs 2/5. No sensory, bladder or autonomic disturbance noted. NCV suggested the AIDP variant of GBS. CSF and labs were normal. He was given IVIG (1 gm/kg/day for 2 days). The weakness progressed to involve bulbar and respiratory muscle, requiring mechanical ventilator support on 3rd day of admission. Developed bradycardia on day 4. He gradually improved and was extubated on Day 6. Facial palsy resolved, and he was discharged on day 34 with mild residual weakness. All microbial tests including CSF samples sent to NIV, Pune were negative. Physiotherapy was continued post- discharge.

Case 2

An 11-year-old female child presented with acute onset bilateral lower limb weakness and pain during movement of lower limbs, following a recent febrile episode. Examination revealed power 2/5 in lower limbs, 4/5 in upper limbs, with preserved reflexes and no cranial nerve involvement. There was no response to analgesics. NCV study suggested AMAN (acute motor axonal neuropathy) variant of GBS. She was treated with IVIG (1gm/kg/day for 2 days). Samples sent to NIV, Pune were negative. The child demonstrated steady improvement and was discharged after 16 days with full recovery.

Case 3

A 7-year-old female was admitted with rapid ascending quadriparesis, respiratory difficulty, and swallowing and speech difficulty within 12 hours of onset. Examination showed generalized hypotonia, 0/5 motor power in all limbs, trunk and neck, no facial expression except eye opening, absent reflexes, evidence of bulbar palsy and SpO_2 of 88% on room air. She was intubated and managed with IVIG (1gm/kg/day for 2 days). NCV findings were consistent with AIDP (Acute inflammatory demyelinating polyneuropathy). *Campylobacter jejuni* was isolated from stool. Despite aggressive treatment, she required tracheostomy due to prolonged ventilatory support. On Day 24, she developed ventilator-associated pneumonia caused by Klebsiella, managed with antibiotics. She also had one episode of convulsion.

Improvement began in the sixth week. Recovery followed a descending pattern with first noticed in face. She was weaned off gradually and T-piece trials started after 2 months. Gradually she was shifted to oral feeds from nasogastric tube feeding. At around 100th day of admission she was off oxygen support and the tracheostomy tube was removed. She was discharged after 110 days of hospital stay (Intensive care unit+ward) with residual weakness.

Case 4

An 11-year-old male presented with symmetrical ascending weakness involving lower limbs, trunk, upper limbs and neck, one day after onset. He had a febrile illness 10 days prior. Vitals were stable; neurological exam showed MRC power of 3/5 in limbs and 2/5 in neck with preserved reflexes and normal sensory examination. NCV revealed absent motor responses in multiple nerves and reduced amplitudes with prolonged latencies in others, indicating AIDP with axonal features. He was treated with IVIG (1gm/kg/day for 2 days). CSF study and all samples sent to NIV, Pune were normal. Rapid neurological recovery was observed, and the child was discharged after 14 days with complete resolution of symptoms.

Case 5

An 8-year-old male presented with a 10-day history of progressive ascending weakness involving lower limbs, upper limbs, and trunk. He had a history of acute gastroenteritis 22 days prior. On examination, he was conscious and hemodynamically stable. Muscle power was 4/5 distally and 3/5 proximally in lower limbs and 3/5 in upper limbs, with preserved reflexes and no cranial nerve involvement. Routine investigations were

unremarkable. NCV findings indicated AIDP variant of GBS. He was treated with IVIG (1gm/kg/day for 2 days). The patient responded well and by day 14, he was discharged with no residual deficits.

Table 1: Summary of findings of each case.

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5
Age/Sex	10 years/ Male	11 years/Female	7 years/Female	11 years/Male	8 years/ Male
Preceding illness	yes	yes	None significant	yes	yes
Onset duration	Progressive over several days	1 day	Rapid (within 12 hrs)	1 day	10 days
Symptoms at presentation	Ascending weakness, bilateral LMN facial palsy	Lower limb weakness and pain	Quadriparesis, bulbar and respiratory involvement	Ascending weakness including trunk and neck	Progressive ascending weakness
Bowel-bladder involvement	No	No	yes	No	No
NCV findings	AIDP	AMAN	AIDP	AIDP	AIDP
Infective workup (NIV)	Negative	Negative	*Campylobacter jejuni*	Negative	Negative
Treatment given	IVIG 2g/kg	IVIG 2g/kg	IVIG 2g/kg	IVIG 2g/kg	IVIG 2g/kg
Autonomic disturbance	Yes	No	No	No	No
Complications	Bradycardia	None	VAP with Klebsiella and convulsion.	None	None
Outcome (Hughes scale)	Discharged with mild weakness (3)	Complete recovery (0)	Discharged with Residual weakness (4)	Complete recovery (0)	Complete recovery (0)

DISCUSSION

The present case series describes five pediatric patients diagnosed with Guillain-Barré Syndrome (GBS) during a localized outbreak in 2025 in Kolhapur, Maharashtra. All cases were diagnosed using Brighton Collaboration criteria and NCV studies.[8] Although, modified Asbury criteria can also be used for diagnostic purposes.[9] These cases showcase the classical as well as variant clinical profiles of GBS in children with variable outcomes. The observed clinical patterns are consistent with established literature while offering some unique epidemiological insights in an Indian pediatric cohort.

Preceding infections are important trigger in the pathogenesis of GBS, as seen in four out of five cases. In one case (case 3), *Campylobacter jejuni* was isolated from stool samples. This finding was consistent with other cases in regions like Pune in the outbreak. This aligns with the findings by Kalra et al, in a North Indian case-control study which demonstrated a significant association between Campylobacter infection and GBS in children, with 55% of cases showing recent exposure to the pathogen compared to 6% of controls.[2] Similarly, Sharma et al, found a high rate of antecedent infections in their pediatric GBS cohort (83.3%) in Rajasthan, predominantly respiratory and gastrointestinal.[4] Dutta et al in their analysis of GBS patients in Western India also reported that 64% of patients had antecedent illness, supporting our findings.[6] The clinical presentation in our series was predominantly in the form of progressive ascending weakness, consistent with classic GBS. All cases except case 2 developed varying degrees of quadriparesis. Case 3 presented with fulminant progression within 12 hours, accompanied by respiratory and bulbar involvement, requiring mechanical ventilation and later tracheostomy, which represents the severe spectrum of the disease. A similar pattern was observed by Verma et al in a study from North India, where 22.6% of pediatric GBS cases presented with respiratory distress and 9.7% required ventilator support.[3]

In the study, two cases (40%) required respiratory support, which may reflect a more severe disease spectrum during the outbreak. Nerve conduction velocity (NCV) study showed AIDP (acute inflammatory demyelinating polyneuropathy) in four cases (1, 3, 4, and 5), while one case (2) showed features of AMAN (Acute motor axonal neuropathy). The predominance of the AIDP subtype (80%) is consistent with several Indian studies. In Verma et al's cohort, 71.6% of children had the AIDP variant, and only 15% showed axonal forms such as AMAN or AMSAN.[3] Shrivastava et al in their Indian perspective review also reported that AIDP remains the most common variant in India, observed in nearly 60–70% of cases across multiple studies.[7] Interestingly, case 2, which showed AMAN on NCV, had a relatively milder presentation and complete recovery was achieved within two weeks. This finding contrasts with reports suggesting that AMAN often results in more severe deficits and slower recovery. However, as highlighted by Verma et al, axonal variants may have a variable prognosis and, in some cases, recover faster than demyelinating forms, especially with early treatment.[3]

Cranial nerve involvement was seen in two patients. Case 1 presented with bilateral lower motor neuron facial palsy, while case 3 showed bulbar palsy. This was consistent with findings in a study by Kalita et al, where 36% of children with GBS had facial nerve involvement, and bulbar weakness was seen in 20% of cases.[10] It is noteworthy that cranial involvement was more common in those requiring respiratory support, underlining its association with severe disease.

Reflex examination showed that only two cases (1 and 3) had absent reflexes, while the others had preserved reflexes. Though areflexia is a hallmark of GBS, preserved or even brisk reflexes have been documented, particularly in early stages or in certain variants like AMAN. This was also supported in Sharma et al.'s series, where deep tendon reflexes were preserved in 14% of patients.[4] Therefore, absence of reflexes, though supportive, is not universally necessary for diagnosis. CSF analysis in our series was largely normal. Classical albumin-cytologic dissociation (raised protein with normal cells) may be absent early in the disease.

According to Willison et al, CSF abnormalities are seen in only 50–60% of patients in the first week of illness and rise to over 80% after two weeks.[1] Hence, normal CSF in early stages should not rule out the diagnosis. Treatment in all five cases consisted of IVIG at 2 g/kg over 2 days, the current standard of care. All cases responded favorably to the treatment. This is in line with findings from a multicenter study by Dutta et al, which reported favorable outcomes in 76% of pediatric patients treated with IVIG alone, and only 8% had poor outcomes at discharge.[6] Verma et al, also noted that early IVIG administration (<7 days from symptom onset) significantly correlated with better outcomes.[3]

Complications were noted in two patients. Case 1 developed bradycardia on day 4, a manifestation of autonomic dysfunction, which is seen in up to 20% of pediatric GBS cases. Autonomic disturbances have been well documented in Indian series, including that by Sharma et al, where autonomic dysfunction was observed in 13% of children.[4] Case 3 developed ventilator-associated pneumonia due to Klebsiella, emphasizing the risk of nosocomial infections in patients requiring prolonged ventilation and ICU stay.

Duration of hospital stay ranged from 14 to over 40 days. The two patients requiring ventilation (cases 1 and 3) had prolonged stays. Mean hospital stay in similar Indian studies ranges from 10 to 30 days depending on severity. Dutta et al reported a mean stay of 22 days, and Sharma et al, observed a mean stay of 17 days.[4,6] Our findings are consistent, though case 3 had extended hospitalization due to respiratory failure and superadded infection. Outcomes at discharge were generally favorable, with three patients achieving complete recovery and two patients having residual weakness. These outcomes are comparable to Indian literature. Verma et al observed that 74.2% of children regained independent ambulation within one month of illness, while 25.8% took longer.[3] Factors associated with poor outcome include bulbar involvement, respiratory failure, and absent reflexes-all seen in our more severely affected patients. Our findings underscore the prognostic value of these clinical features.

The predominance of male patients (3 out of 5) is consistent with the well-established male bias in GBS. Shrivastava et al, noted a male to female ratio of 1.7:1 in Indian studies.[7] Hormonal and immunological factors may contribute to this disparity. Age distribution in our series ranged from 7 to 11 years, which is typical for pediatric GBS, commonly affecting school-aged children.

CONCLUSION

This case series highlights the variable clinical presentation, electrophysiological patterns and outcomes of pediatric Guillain-Barré Syndrome in children during an outbreak in Kolhapur, Maharashtra. Most cases follow an antecedent infection and respond well to IVIG therapy. AIDP is the predominant subtype, and early recognition of severe features like cranial or respiratory involvement is critical. Timely intervention results in favorable outcomes. The study fulfills its objective of improving awareness regarding early management of GBS in children, strengthening surveillance, role of early NCV access and investigating infectious triggers during seasonal spikes of acute flaccid paralysis.

ACKNOWLEDGEMENTS

We thank Dr. Satyawan More (Dean) and Dr. Shishir Mirgunde (Associate Professor, Paediatrics) for supporting us.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. Lancet. 2016;388:717-27.
2. Kalra V, Chaudhry R, Dua T, Dhawan B, Sahu JK, Mridula B. Association of Campylobacter jejuni infection with childhood Guillain-Barré syndrome: a case-control study. J Child Neurol. 2009;24(6):664-8.
3. Verma R, Chaudhari TS, Raut TP, Garg RK. Clinico-electrophysiological profile and predictors of functional outcome in Guillain-Barré syndrome in children. J Pediatr Neurosc. 2013;8(2):94-8.
4. Sharma CM, Kumawat BL, Ralot T, Tripathi G, Dixit S. Guillain-Barre syndrome occurring during dengue fever. J Indian Med Assoc. 2011;109(9):675-82.
5. Pari H, Amalnath SD, Dhodapkar R. Guillain-Barre Syndrome and Antibodies to Arboviruses (Dengue, Chikungunya, and Japanese Encephalitis) in Southern India. Ann Indian Acad Neurol. 2022;25(2):203-6.
6. Dutta D, Debnath M, Nagappa M, Das SK, Wahatule R, Sinha S, et al. Antecedent infections in Guillain-Barré syndrome patients from south India. J Peripher Nerv Syst. 2021;26(3):298-306.
7. Shrivastava M, Nehal S, Seema N. Guillain-Barre syndrome: Demographics, clinical profile & seasonal variation in a tertiary care centre of central India. Indian J Med Res. 2017;145(2):203-8.
8. Roodbol J, Wit MY, Berg B, Kahlmann V, Drenthen J, Catsman-Berrevoets CE, et al. Diagnosis of Guillain-Barré syndrome in children and validation of the Brighton criteria. J Neurol. 2017;264(5):856-61.
9. Monique M. Ryan. Guillain-Barre syndrome. In:Kliegman RM, St. Geme JW III,editors. Nelson Textbook of Pediatrics. 22nd ed. Elsevier; 2024. Chapter 656:3900-6.
10. Kalita J, Misra UK, Das M. A study of clinical, electrophysiological and prognostic profiles of Guillain-Barré syndrome. Acta Neurol Scand. 2004;110(2):106-13.

Cite this article as: Gupta A, Awadkhan M, Kumbhojkar S, Jadhav M, Miraje B. Study of clinical profile of Guillain-Barré syndrome in children upto 12 years during an epidemic outbreak. Int J Contemp Pediatr 2025;12:1538-42.