

Neurophysiological Subtypes and Short-Term Outcomes of Paediatric Guillain-Barré Syndrome – A Tertiary Care Experience

DOI: 10.5281/zenodo.18014338

Humaira Rafiq Quaderi¹, Kinkar Ghosh²

Received: 03 Dec 2025
Accepted: 08 Dec 2025
Published: 17 Dec 2025

Published by:
Gopalganj Medical College, Gopalganj,
Bangladesh

Correspondence to
Humaira Rafiq Quaderi

ORCID
<https://orcid.org/0000-0003-2029-167X>

Copyright © 2025 The Insight



This article is licensed under a Creative Commons Attribution 4.0 International License.



ABSTRACT

Background: Guillain-Barré syndrome (GBS) is the leading cause of post-infectious acute flaccid paralysis in children, typically presenting with rapidly progressive, bilateral weakness beginning in the lower limbs and ascending proximally. It includes several neurophysiological subtypes—both demyelinating and axonal—that differ in clinical features and outcomes. **Objective:** To determine the frequency of neurophysiological subtypes of paediatric GBS and assess their short-term clinical outcomes. **Methods & Materials:** This retrospective study reviewed medical records of children diagnosed with GBS at Bangladesh Shishu Hospital from January to December 2024, using the Brighton diagnostic criteria. Socio-demographic data, cerebrospinal fluid findings, and nerve conduction study-based subtypes were documented. Functional outcomes were assessed using the Hughes GBS Disability Scale at discharge and at 3-month follow-up, categorizing scores 0–3 as good and 4–6 as poor outcomes. Statistical analysis was performed using SPSS version 20, with $p < 0.05$ considered significant. **Results:** Among 45 children, the most common subtype was AMAN (53%), followed by AMSAN (31%) and AIDP (16%). The AMSAN subtype demonstrated significantly greater disease severity, including longer duration of illness, non-ambulatory presentation, progressive course, and higher ICU admission ($p = 0.01, 0.003, 0.01, 0.03$). Although discharge outcomes did not vary significantly across subtypes, AMSAN was associated with poorer 3-month recovery ($p = 0.04$). IVIG use correlated with worse outcomes ($p = 0.017$), reflecting its use primarily in more severe cases. **Conclusion:** The AMSAN subtype was linked to more severe clinical manifestations and poorer short-term functional outcomes compared to other subtypes. Early neurophysiological classification may help predict prognosis and highlight the need for closer monitoring and targeted rehabilitation in children with AMSAN.

Keywords: Guillain-Barré Syndrome, Neurophysiological Subtypes, Nerve Conduction Studies, Short-Term Outcome

(The Insight 2025; 8(3): 604-608)

1. Department of Epidemiology & Research, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh
2. Associate Professor, Department of Paediatric Neuroscience, Bangladesh Shishu Hospital & Institute, Dhaka, Bangladesh

INTRODUCTION

Guillain-Barre Syndrome (GBS) is the most common cause of acute flaccid paralysis in children. It is characterized by the rapid onset of bilateral and symmetrical weakness, typically beginning in the lower limbs and ascending to involve the upper limbs.^[1] The severity of motor weakness can vary significantly, ranging from mild gait disturbances to complete quadriplegia. Involvement of motor cranial nerves and life-threatening respiratory muscle weakness occurs in approximately 10–25% of cases, necessitating ventilator support.^[2] The incidence of GBS in children under the age of 15 is estimated to be between 0.34 and 1.34 per 100,000 per year.^[3] The onset is often preceded by an upper respiratory or gastrointestinal infection, which acts as a trigger for the immune-mediated response that leads to nerve damage. The commonly identified antecedent pathogens are *Campylobacter jejuni*, cytomegalovirus, Epstein-Barr virus, and *Mycoplasma pneumoniae*.^[4] GBS is broadly classified into demyelinating and axonal forms. These subtypes differ in terms of clinical features, electrophysiological patterns, and prognostic implications.^[5] Electrophysiological studies help differentiate these subtypes and guide clinical management. The most common variant in Southeast Asia is acute motor

axonal neuropathy (AMAN). While in North America and Europe, acute inflammatory demyelinating polyneuropathy (AIDP) is more frequently observed. Acute motor-sensory axonal neuropathy (AMSAN) is another recognized variant, though less common in paediatric populations.^[5] Early identification of GBS subtypes in children is essential for anticipating disease course, adapting treatment strategies, and informing prognosis. The cornerstone of treatment includes supportive care, with specific therapies such as intravenous immunoglobulin (IVIG) or plasmapheresis.^[6] Children with bulbar involvement or respiratory compromise often require intensive care unit (ICU) management, including mechanical ventilation. Overall, the prognosis of GBS in the paediatric population is more favourable than in adults. Mortality is rare, and 90–95% of affected children achieve full recovery within 3 to 12 months.^[7] While the clinical and electrophysiological aspects of GBS are well-studied in adults, data in paediatric populations remain limited, particularly in low-resource and developing countries. A better understanding of neurophysiological subtypes and their outcomes in children is essential for optimizing care and improving long-term functional outcomes.

METHODS & MATERIALS

This retrospective study was conducted in the Paediatric Neuroscience Department of Bangladesh Shishu Hospital and Institute over one year (January 1, 2024–December 31, 2024). Medical records of children aged <18 years, diagnosed with Guillain-Barré Syndrome (GBS), based on the Brighton diagnostic criteria,^[8] were reviewed. Patients with incomplete medical records or who were lost to follow-up were excluded from the analysis. For each patient, the information regarding gender, age at the onset of clinical presentation, antecedent events, neurological signs and symptoms, electrophysiological findings, and laboratory findings (i.e., cerebrospinal fluid examination results), treatment, and clinical course (progressive/non-progressive) based on Erasmus GBS Respiratory Insufficiency Score (EGRIS) was collected from the record book. A score, 0-4 was considered non-progressive, and a score of >4 was considered progressive disease.^[9]

Hughes Scale^[10]

Grade	Description
0	Healthy
1	Minor symptoms or signs of neuropathy, but capable of manual work/capable of running
2	Able to walk without the support of a stick (5m across an open space) but incapable of manual work/running
3	Able to walk with a stick, appliance, or support (5m across an open space)
4	Confined to bed or chair-bound
5	Requiring assisted ventilation (for any part of the day or night)
6	Death

**Note-score- 0-3-Good outcome, score 4-6-poor Outcome*

STATISTICAL METHOD

Data were processed and analyzed by SPSS 23 (Statistical Program for Social Sciences). Categorical variables were presented in frequency and percentage. Pearson chi-square or Fisher’s exact test, as appropriate, was applied for qualitative variables. A P-value less than 0.05 was considered to be statistically significant.

RESULTS

A total of 45 GBS patients, most of the children (76%), were referred from the inpatient department, with a significant proportion (51%) being under five years old. The gender distribution was nearly equal. A notable finding was the predominance of patients from middle-income families (40%) (Table I). All patients underwent nerve conduction study, and electro-physiological subtypes showed AMAN (53%), AMSAN (31%), and AIDP (16%) (Figure 1). Among 45 patients, the disease was found to be progressive in 38% of patients (Table II). While need for ICU care was 31%. The clinical profiles of GBS patients were analysed for associations with the three main electro-clinical subtypes: AMAN, AMSAN, and AIDP. The key findings from this analysis are shown in Table II. For the 31 children who were non-ambulatory at presentation, the AMAN subtype was the most frequent (61.2%), followed by AMSAN (35.4%) and AIDP (3.4%). This association was statistically significant (p=0.003). The duration of illness <15 days was more in AMAN (66.6%, n=14) compared to AIDP and AMSAN (23.8% and 9.6%). While more than 15 days of illness (n=24), the distribution shifted, with the AMSAN subtype (50%, n=12) (p=0.01). The clinical course of the disease was significantly associated with the subtype (p=0.01). Among the 17 patients, the AMSAN were the most progressive (47%, n=8). For the 28 patients with a non-progressive course, the AMAN subtype was the most common (60.8%, n=17), then AIDP (17.8%, n=5). (Table II) The need for Intensive Care Unit (ICU) admission was significantly associated with the GBS subtype (p= 0.03). Out of 14 patients, the majority of the AMSAN subtype required ICU care (57%, n=8). In contrast, a large proportion of patients (n=31) who did not require ICU care were of the AMAN subtype (64.5%, n=20) (p=0.03). The presence of cranial nerve palsy was not found to be

Electrophysiological studies were performed using conventional procedures. Motor conduction studies were performed on the median, ulnar, tibial, and fibular nerves. Sensory nerve studies were performed on the median, radial, and sural nerves. The amplitude of the negative phase was measured for compound muscle action potentials (CMAPs) and sensory nerve action potentials (SNAPs). H-reflexes were recorded from the soleus and rectus femoris muscles, and F-waves were recorded from the median or ulnar nerves. Functional outcomes were assessed using the Hughes GBS Disability Scale,^[10] at discharge and 3-month follow-up at the paediatric neuroscience department. A Hughes score of 0–3 was considered a good outcome, while 4–6 indicated a poor outcome. Clinical presentation, CSF findings, treatment, and Outcomes were compared across neurophysiological subtypes of GBS.

statistically significantly associated with any specific subtype (p=0.3). (Table II). About 21 out of 45 patients (46.7%) had a good outcome at discharge, and 24 (53.3%) had a poor outcome. In contrast, after 3 months of follow-up, 62.2% had a good outcome, and 37.7% had a poor outcome. (Figure II) Treatment of GBS consists of supportive management along with specific therapy by IVIG or plasmapheresis when indicated (progressive disease, respiratory failure, cranial nerve palsy). In this study, those who required IVIG, 8 (42.1%), had a good outcome, while 11 (57.9%) had a poor outcome. Conversely, among those who did not require IVIG, 20 (76.9%) experienced a good outcome and 6 (23.1%) had a poor outcome (Table III). A Pearson’s chi-square test demonstrated a statistically significant association between IVIG administration and clinical outcome (p = 0.017), suggesting that outcomes differed depending on IVIG use (Table III). Based on Table IV, the association between electro-clinical subtypes of Guillain-Barré Syndrome (GBS) and disease outcomes (both at discharge and after 3 months) in a paediatric population of 45 patients was analysed and interpreted. There was no statistically significant association between the GBS subtype and the patient's outcome at discharge (p=0.5). After 3 months post-discharge, 28 patients (62.2%) had a good outcome, and 17 (37.8%) had a poor outcome. In contrast to the discharge outcome, a statistically significant association was observed between the GBS subtype and the outcome at 3 months post-discharge (p=0.04). Of the 28 patients who achieved a "Good" outcome at 3 months, the majority (64.2%, n=18) belonged to the subtype AMAN. Conversely, among the 17 patients with a "Poor" outcome at 3 months, the AMSAN subtype was the most prominent, accounting for 53% (n=9) of these cases. In the AIDP group, 5 patients (17.9%) showed good recovery, while 2 (11.8%) had a poor outcome. This significant finding highlights that, during discharge, the prognosis may be similar across subtypes, but after 3 months, the AMSAN subtype is associated with a higher likelihood of a poor outcome, emphasizing the importance of electro-clinical classification in long-term prognostication. (Table IV).

Table - I: Socio-demographic characteristics of the patients (n=45)

Variables	Category	Frequency (n)	Percentage (%)
Age	<5Y	23	51
	5Y-10Y	13	29
	>10Y	09	20
Sex	Male	23	51
	Female	22	49
Income	Lower income	15	33
	Middle Income	18	40
	Higher income	12	27
Referral Unit	Outpatient department	11	24
	Inpatient Department	34	76

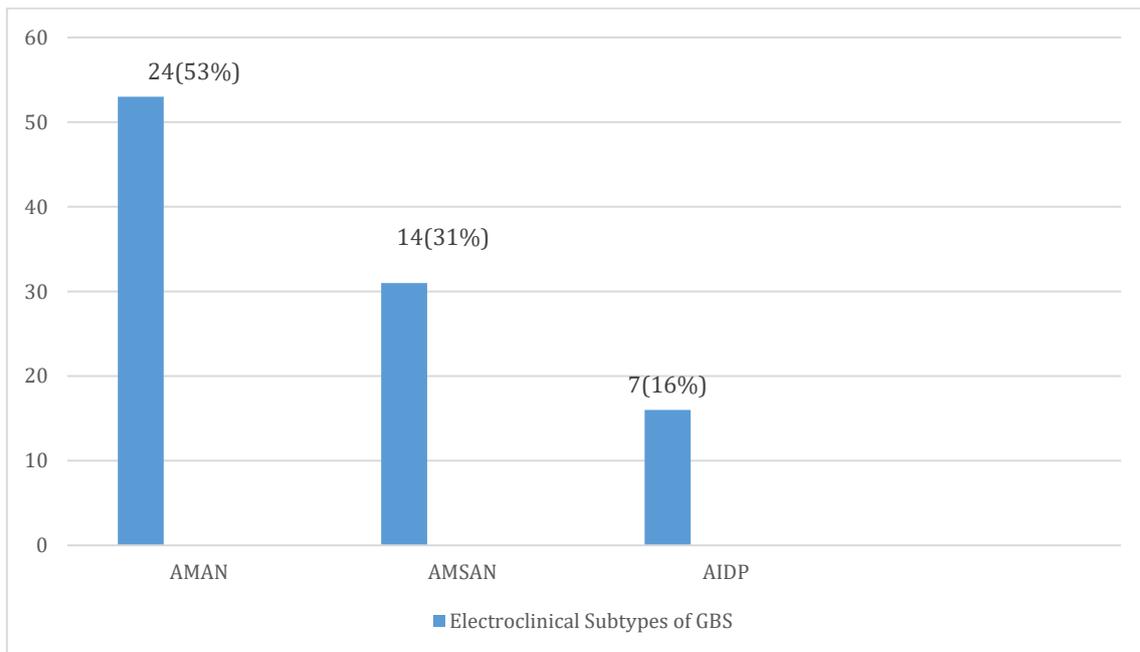


Figure - 1: Electroclinical subtypes of GBS

Table - II: Association between disease profile and Electro-clinical subtypes of GBS

Variable	Electro-clinical Subtypes			Total	P value
	AMAN	AMSAN	AIDP		
Gait					
Ambulatory	05(35.7%)	03(21.4%)	06(42.9)	14(31.12%)	0.003
Non-ambulatory	19(61.25)	11(35.45)	01(3.2%)	31(68.8%)	
Cranial nerve palsy					
Yes	03(50%)	03(50%)	0	06(13.4%)	0.3
No	21(65.6%)	11(34.3%)	07(21.8%)	39(86.6%)	
Duration of illness during NCS					
<15days	14(66.6%)	02(9.6%)	5(23.8%)	21(46.6%)	0.01
>15days	10(41.6%)	12(50%)	2(8.4%)	24(53.3%)	
Course of disease					
Progressive	07(41.2%)	8(47%)	2(11.8%)	17(37.7%)	0.01
Non-Progressive	17(60.8%)	6(21.4%)	5(17.8%)	28(62.3%)	
Need for ICU					
Yes	04(28.6%)	08(57%)	2(14.3%)	14(31%)	0.03
No	20(64.5%)	06(19.35%)	5(16.1%)	31(69%)	

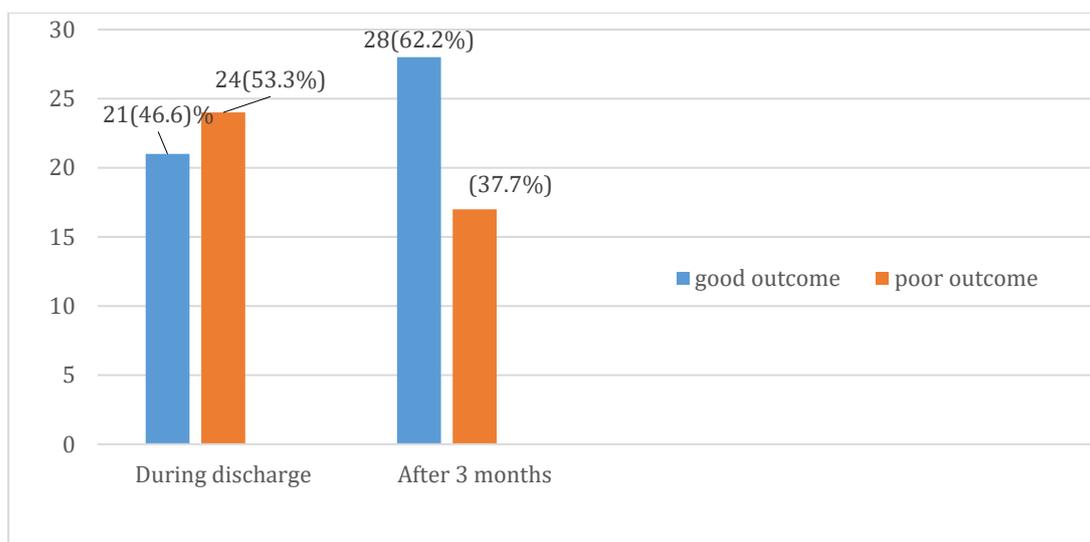


Figure - 2: Outcome of GBS during discharge and 3 months after discharge

Table - III: Association between IVG requirement and outcome of GBS after 3 months

Variables	Outcome after 3 months		Total	p-value
	Good	Poor		
IVIG				
Required	08(42.1%)	11(57.9%)	19	0.01
Not required	20(76.9%)	06(23.1%)	26	
Total	28	17	45	

Table - IV: Association between disease outcome and electro-clinical subtypes of GBS

Variables	Electro-clinical Subtypes			Total	P value
	AMAN	AMSAN	AIDP		
Outcome at discharge					
Good	12(57.1%)	05(23.8%)	04(19%)	21	0.5
Poor	12(50%)	09(37.5%)	03(12.5%)	24	
Total	24	14	07	45	
Outcome at 3 months					
Good	18(64.2%)	05(17.9%)	05(17.9%)	28	0.04
Poor	06(35.2%)	09(53%)	02(11.8%)	17	
Total	24	14	07	45	

DISCUSSIONS

In Bangladesh, nerve conduction studies (NCS) are becoming more available and advanced, helping doctors better diagnose and treat Guillain-Barré Syndrome (GBS). Of the 45 children, the male was 23(51%), the female was 22(29%), and 51% of the children were less than 5 years of age. These findings are similar to many studies where males were predominant, and this corresponded to the increased risk of GBS in males worldwide, and the mean age of presentation was around 5 years.^[11-12] Middle-income families were predominant (40%), then lower-income families (33%). These findings were supported by many studies in Bangladesh, where poor hygiene and sanitation, unsafe drinking water, and frequent exposure to pathogens in low and middle-income families are highly vulnerable to outbreaks of infectious diseases that are capable of triggering GBS.^[13-14] Other studies found that socioeconomic status influenced health-seeking patterns and time to diagnosis in GBS cases.^[15] The current study identified the subtypes of GBS as AMAN (53%), AMSAN (31%), and AIDP (16%). This pattern aligns with other Asian studies reporting a higher prevalence (30–65%) of AMAN and AMSAN.^[13] but differs from Western reports, where AIDP is more common^[10,15] The higher prevalence of AMAN and AMSAN in Asia may be attributed to increased *C. jejuni* infections, a

common cause of gastroenteritis^[13]. In this study, the duration of illness (onset of disease to maximum severity) was less than 15 days was longer in the AMAN group (66.6%) compared to AMSAN and AIDP (p=0.01). In a similar study, the mean time to maximal weakness in the axonal group was 4.2 days (range 1–12 days).^[16] The AMSAN was found to be more progressive and needed ICU support compared to AMAN and AIDP (p=0.01, p=0.03). This is similar to a recent study by Priyadarshini *et al.*, which identified the AMSAN variant as having severe clinical manifestations compared to other electrophysiological subtypes.^[17] These findings differ from the report by Sen *et al.*^[18] Regarding management, the requirement of IVIG and associated outcomes was statistically significant (p=0.02), showing that those treated with IVIG were more likely to experience poor outcomes. The direction of this association differs from the study by Joseph *et al.*^[19]. While these findings were supported by Katila J *et al.*, who showed that IVIG therapy did not alter the outcome in AMAN but resulted in a lesser proportion of poor recovery at 6 months in AIDP (0.8% vs. 6.6%, p = 0.03).^[20] The outcome of GBS in this study showed no mortality during the study period. Good outcomes (Hughes score 0-3) were observed in 47% of patients at discharge and 62.2% after three months. Recently, Yadav *et al.* studied 36 children and reported a

recovery rate of 84.4% at a three-month follow-up.^[21] In this study, the outcome was good in AMAN, at discharge (57%) and after 3 months follow-up (64.2%), compared to AMSAN and AIDP ($p= 0.04$). This differs from other studies where AMAN has been found to have a poorer clinical outcome.^[22] However, other reports have shown that AMAN patients can also make a rapid recovery.^[23] Whereas Karolok ZS et al. found all subtypes had near equal favourable outcomes.^[11] This study had several limitations. The study was conducted at a single centre with a small sample size, which may have reduced the statistical power and reliability of the analysis. The short follow-up duration and retrospective data collection may have resulted in incomplete clinical information. In this regard, large-scale multicentre studies are required for further evaluation

CONCLUSION

This study demonstrates that the AMSAN subtype is strongly associated with greater clinical severity, including prolonged illness, non-ambulatory presentation, a progressive disease course, and higher ICU admission rates. Although outcomes at discharge did not differ significantly across subtypes, a clear divergence emerged by three months: children with AMAN showed a better recovery profile, whereas those with AMSAN had poorer functional outcomes. These findings underscore the prognostic value of early neurophysiological classification in paediatric GBS and highlight the need for closer monitoring and targeted rehabilitation for children with the AMSAN subtype to optimize recovery.

ACKNOWLEDGEMENT

I would like to express my sincere gratitude to all my colleagues in the Department of Paediatric Neuroscience and all the doctors at Bangladesh Shishu Hospital and Institute for their support and for referring patients to the neurophysiology laboratory.

REFERENCES

1. Levison LS, Thomsen RW, Markvardsen LK, Christensen DH, Sindrup SH, Andersen H. Pediatric Guillain-Barré Syndrome in a 30-Year Nationwide Cohort. *Pediatr Neurol.* 2020;107:57–63.
2. Alsheklee A, Hussain Z, Sultan B, Katirji B. Guillain-Barré syndrome: incidence and mortality rates in US hospitals. *Neurology.* 2008;70:1608.
3. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. *Neuroepidemiology.* 2011;36:123–133.
4. Levison LS, Thomsen RW, Markvardsen LK, Christensen DH, Sindrup SH, Andersen H. Pediatric Guillain-Barré Syndrome in a 30-Year Nationwide Cohort. *Pediatr Neurol.* 2020;107:57–63.
5. Estrade S, Guiomard C, Fabry V, Baudou E, Cances C, Chaix Y, et al. Prognostic factors for the sequelae and severity of Guillain-Barré syndrome in children. *Muscle Nerve.* 2019;60:716–723.
6. Hughes MI. *Neurology. In: Forfar and Arneil's Textbook of Pediatrics.* 7th ed. p. 930.
7. DiFazio MP, Jallo GI. Pediatric Guillain-Barre Syndrome. In: *Drugs & Diseases: Neurology.* New York: Medscape; 2019 Nov 14.
8. Roodbol J, de Wit MY, van den Berg B, et al. Diagnosis of Guillain-Barré syndrome in children and validation of the Brighton criteria. *J Neurol.* 2017;264:856–861.
9. Walgaard C, Lingsma HF, Ruts L, et al. Prediction of respiratory insufficiency in Guillain-Barré syndrome. *Ann Neurol.* 2010;67:781–787.
10. van Koningsveld R, Steyerberg EW, Hughes RAC, Swan AV, van Doorn PA, Jacobs BC. A clinical prognostic scoring system for Guillain-Barré syndrome. *Lancet Neurol.* 2007;6(7):589–594.
11. Karalok ZS, Taskin BD, Yanginlar ZB, Gurkas E, Guven A, Degerliyurt A, et al. Guillain-Barré syndrome in children: subtypes and outcome. *Childs Nerv Syst.* 2018;34(11):2291–2297.
12. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. *Neuroepidemiology.* 2011;36(2):123–133.
13. Islam Z, et al. Axonal variant of Guillain-Barré syndrome associated with *Campylobacter* infection in Bangladesh. *Neurology.* 2010;74:581–587.
14. Pal M, Ayele Y, Hadush M, Panigrahi S, Jadhav VJ. Public health hazards due to unsafe drinking water. *Air Water Borne Dis.* 2018;7:1000138.
15. Koul RL, Alfutaisi A. Prospective study of children with Guillain-Barre syndrome. *Indian J Pediatr.* 2008;75(8):787–790.
16. Habib R, Saifuddin M, Islam R, Rahman A, Bhowmik N, Haque MA. Clinical Profile of Guillain-Barré Syndrome – Observations from a Tertiary Care Hospital of Bangladesh. *BIRDEM Med J.* 2017;7(1):38–42.
17. Debashree P, Anuhya V, Mahapatra A. Clinico-epidemiological profile and prediction of outcome in children with Guillain-Barré syndrome. *Ital J Pediatr.* 2025;51:179.
18. Sen S, Kumar A, Roy B. Clinical outcome of Guillain-Barre syndrome in 108 children. *Indian Pediatr.* 2021;58:833–835.
19. Joseph N, Shrigiri S. Predictors of Treatment Outcome and Clinical Profile among Guillain-Barre Syndrome Patients in South India. *Rev Recent Clin Trials.* 2023;18(4):258–268.
20. Kalita J, Misra UK, Chaudhary SK, Das M, Mishra A, Ranjan A, Kumar M. Outcome of Guillain-Barré syndrome following intravenous immunoglobulin compared to natural course. *Eur J Neurol.* 2022;29(10):3071–3080.
21. Yadav S, Jain P, Sharma S, Kumar V, Aneja S. Guillain-Barré syndrome in North Indian children: Clinical and serial electrophysiological features. *Neurol India.* 2019;67:724–727.
22. Siddiqui M, Majid S, Yusuf H, Mateen F. Electrophysiological pattern and predictors of functional outcome of patients with Guillain-Barré syndrome at a tertiary care hospital in Pakistan. *J Coll Physicians Surg Pak.* 2022;32(3):364.
23. Kuwabara S, Ogawara K, Misawa S, Mizobuchi K, Sung JY, Kitano Y, et al. Sensory nerve conduction in demyelinating and axonal Guillain-Barré syndromes. *Eur Neurol.* 2004;51:196–198.