

Original Research Article

Clinical profile, electrophysiological pattern and predictors of short-term outcome of Guillain-Barré syndrome in children

Sufia Khatun Sumi*, Farzana Binta Rashid, M. Samsun Nahar Sumi, Nusrat Shams, Tahsina Jasmin, Bithi Debnath, Narayan Saha

Department of Paediatric Neurology, National Institute of Neuro Sciences and Hospital, Dhaka, Bangladesh

Received: 11 June 2026

Revised: 09 July 2026

Accepted: 14 August 2026

*Correspondence:

Dr. Sufia Khatun Sumi,

E-mail: kariul@hotmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Guillain-Barré syndrome (GBS) is a rare but potentially life-threatening immune-mediated disorder affecting peripheral nerves, causing acute flaccid paralysis with variable clinical severity. Its diverse presentation and disease course make diagnosis, management, and prognostication difficult, particularly in children. This study aimed to assess the clinical profile, electrophysiological pattern, and predictors of short-term outcomes in pediatric GBS.

Methods: This observational study was conducted at the National Institute of Neurosciences and Hospital, Dhaka, Bangladesh, from September 2018 to August 2019. A total of 93 children diagnosed with GBS were enrolled by consecutive sampling. Clinical characteristics, electrophysiological findings, treatment modalities, and short-term functional outcomes were analyzed. Outcome was assessed after 3 months of follow-up.

Results: Children aged 5-10 years were most commonly affected, with a male-to-female ratio of 1.7:1. Quadriplegia was the predominant presentation (87.1%), while 19.4% required mechanical ventilation. Electrophysiological studies showed acute motor axonal neuropathy (AMAN) in 65.6%, acute inflammatory demyelinating polyneuropathy (AIDP) in 31.2%, and acute motor-sensory axonal neuropathy (AMSAN) in 3.2% of cases. Patients receiving intravenous immunoglobulin with supportive care had shorter hospital stays than those receiving supportive care alone. At 3 months, 41.9% achieved favorable functional recovery (GBS disability grade 0-2), with no mortality recorded. Poor outcome was significantly associated with quadriplegia, bulbar involvement, neck flexor weakness, low baseline Medical Research Council score, and need for mechanical ventilation.

Conclusions: Quadriplegia, bulbar involvement, neck flexor weakness, poor baseline muscle strength, and mechanical ventilation requirement are significant predictors of poor short-term functional outcomes in pediatric GBS.

Keywords: Neurological presentation, Guillain-Barre syndrome, Children, Quadriplegia, Paraplegia

INTRODUCTION

Guillain-Barré syndrome (GBS) is a common cause of acute flaccid paralysis in healthy children, with a worldwide incidence rate of 1 to 2 per 100,000 per year.^{1,2} In the Iranian population, the rate ranges from 1.5 to 3.4 per 100,000.³ While GBS can occur in all age groups, it predominantly affects adults, especially males, and is more frequent in children aged 1-5 years.⁴ GBS is

believed to be an autoimmune disorder triggered by an immune response to infectious agents like *Campylobacter jejuni*, *Cytomegalovirus*, Epstein-Barr virus, *Mycoplasma pneumoniae*, and HIV.^{5,6} Molecular mimicry from these infections may lead to a cross-reaction with peripheral nerve components, resulting in different GBS types. Triggering factors such as immunization, surgery, trauma, and bone marrow transplantation may also play a role in GBS pathogenesis.^{6,7} Clinical presentations of

GBS in children commonly include pain, progressive muscle weakness, and reduced deep tendon reflexes.^{8,9} In younger children under 4 years, leg pain and refusal to walk are prevalent features. Variability exists in the epidemiology and clinical features of GBS across different regions. Understanding these variations can enhance our comprehension of disease pathogenesis, risk factors, and prognosis.¹⁰ Diagnostic criteria, established by the National Institute of Neurological and Communicative Disorders and Stroke in 1978 and updated by Asbury and Cornblath in 1990, emphasize symmetric progressive weakness with hypo or areflexia. Notably, the criteria do not encompass GBS variants.^{11,12} Albumin-cytological dissociation, crucial for diagnostic confirmation, typically occurs in the second or third week. Neurophysiological studies, particularly detecting H-reflex abolition, demonstrate high performance from the second week onward, aiding in root involvement confirmation and observing nerve conduction velocity changes in AIDP and Miller Fisher syndrome (MFS).^{13,14}

This study aimed to assess the clinical profile, electrophysiological pattern, and predictors of short-term outcomes in pediatric GBS.

METHODS

This observational study was conducted at the National Institute of Neurosciences and Hospital, Dhaka, Bangladesh from September 2018 to August 2019. As the study subjects, a total of 93 cases less than 16 years of age were admitted in the mentioned hospital with childhood GBS during the study period and such cases who were directly admitted to HDU, ICU, and Pediatric Indoor because of impending or established respiratory failure respectively were enrolled by using a consecutive sampling technique. The ethical committee of the mentioned hospital approved the study. Proper written consent was obtained from all the participants before data collection. All the demographic and clinical information of the participants was recorded in a predesigned questionnaire. As per the inclusion criteria of this study, children and adolescents aged <16 years and diagnosed as GBS by Asbury and Cornblath criteria were included. On the other hand, according to the exclusion criteria of this study, GBS patients having co-morbidities like cerebral palsy, hereditary neuropathy, and congenital myopathy were excluded. They were subjected to NCS and CSF studies, treated with supportive therapy and IVIG (where indicated), monitored regularly, and transferred to the ICU if needed. They were followed up during their hospital stay, on the day of discharge, after 1 month and 3 months consecutively, using clinical parameters and a functional grading scale. All data were analyzed by using the MS Office program.

RESULTS

In this study, participants had a mean age of 7.73 ± 3.80 years, with 41.9% aged 5-10 years and 31.2% aged 1-5

years. The male-to-female ratio was 1.7:1, indicating male preponderance. Antecedent illnesses were reported in 64.5%, including 26.9% with diarrhea and 20.4% with respiratory infections. Fever or mild symptoms were reported in 17.2%, while 35.5% had no preceding symptoms. Seasonal distribution was 34.4% in winter, 33.3% in summer, and 32.3% in the rainy season. GBS subtypes based on electrophysiological abnormalities included 65.6% AMAN, 31.2% acute inflammatory demyelinating polyradiculoneuropathy, and 3% acute motor and sensory axonal neuropathy (AMSAN). Axonal types (AMAN + AMSAN) prevalence was 68.8%, exceeding the 31.2% demyelinating type. Neurological presentations indicated that the majority of patients had quadriplegia (87.1%) and cranial nerve dysfunction (44.1%). Other features included pain (58.1%), nasal intonation (23.7%), neck flexor weakness (22.6%), breathing difficulty (22.6%), and autonomic dysfunction (7.5%). MRC scoring revealed that 67.2% had a score between 21 and 40 at admission. Initially, most patients were wheelchair-bound with a baseline HMS score of G-4.

HMS score analysis showed that all patients had a baseline disability score of G-3 or more. Among them, 69.9% were wheelchair-bound (HMS-4), 19.4% needed mechanical ventilation (HMS-5), and 10.8% couldn't walk without support (G-3). Ventilation was withdrawn during discharge, improving from G-5 to G-4, with no mortality. At one-month follow-up, 9.7% walked unaided (G-2), and 1 had mild symptoms (G-1). After 3 months, 41.9% had a favorable outcome (G 0-2), with 5% fully recovered (G-0), while 58.1% had a poor outcome.

The total GBS disability score was gradually decreasing in follow up and the p value was significant (<0.001). Although mechanical ventilation was needed in 27.7% of cases in the intravenous immunoglobulin and supportive care group, they required a relatively shorter hospital stay compared to the only supportive care group. At admission, HMS scoring indicated G-5 (requiring mechanical ventilation) in 20% and 17.9% of patients in the combination and supportive care groups, respectively. HMS scoring improved at the 3-month follow-up in both groups. There was a significant difference regarding the need for mechanical ventilation, hospital stay, and short-term outcomes among the groups.

In the 3-month follow-up of the study, 41.9% of patients showed a favorable outcome (HMS 0-2), while 58.1% had a poor outcome (HMS 3-5). Univariate analysis identified significant differences in time to peak deficit, cranial nerve involvement, bulbar involvement, neck flexor weakness, low MRC scoring, ventilator support, and treatment modalities, all associated with poor functional outcomes. Binary logistic regression highlighted quadriplegia, bulbar involvement, neck flexor weakness, poor baseline MRC score, and the need for mechanical ventilation as predictors of poor functional outcomes in pediatric GBS.

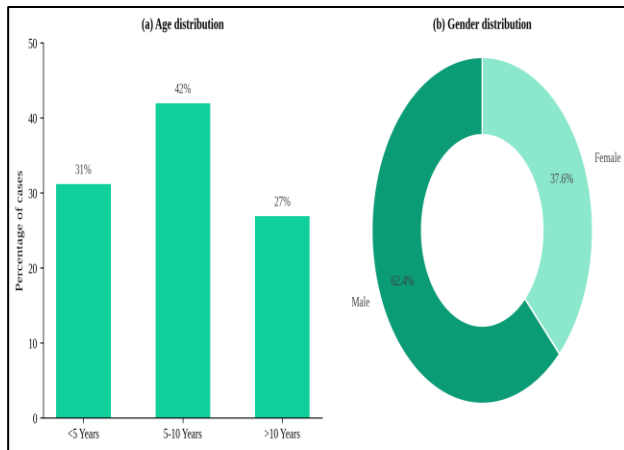


Figure 1: Age and gender distribution of cases, (n=93).

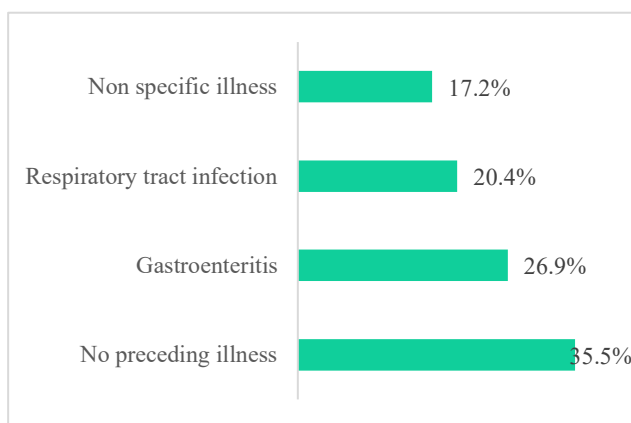


Figure 2: Antecedent events.

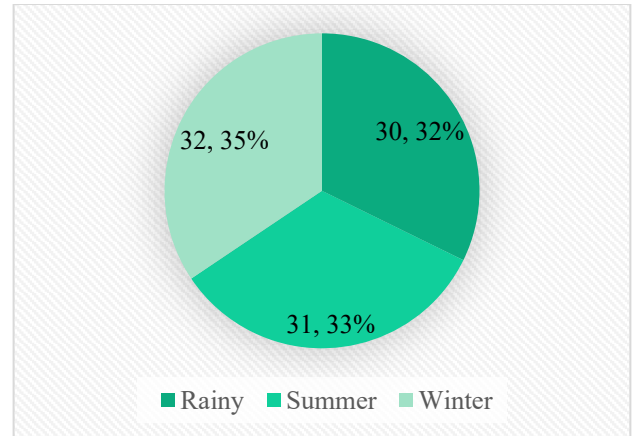


Figure 3: Seasonal variations.

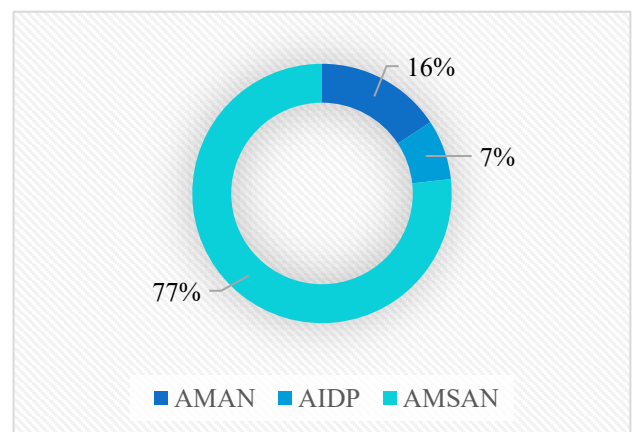


Figure 4: Varieties of GBS based on electrophysiological pattern.

Table 1: Neurological presentation.

Parameters	N	Percentages
Variables		
Quadriplegia	81	87.10%
Paraplegia	12	12.90%
Cranial nerve involvement	41	44.10%
Bulbar involvement	39	41.90%
Facial palsy	2	2.10%
Breathing difficulty	21	22.60%
Nasal intonation	22	23.70%
Autonomic involvement	7	7.50%
Pain	54	58.10%
Neck flexor weakness	21	22.60%
Baseline MRC sum score, (n=64)		
Severe weakness (0-20)	13	20.30%
Moderate weakness (21-40)	43	67.20%
Mild weakness (41-60)	8	12.50%
Baseline HMS score, (n=93)		
Walk 5-meter support (G-3)	10	10.80%
Wheelchair-bound (G-4)	65	69.90%
Ventilation support (G-5)	18	19.40%
(Mean±SD)	4.09±0.54	

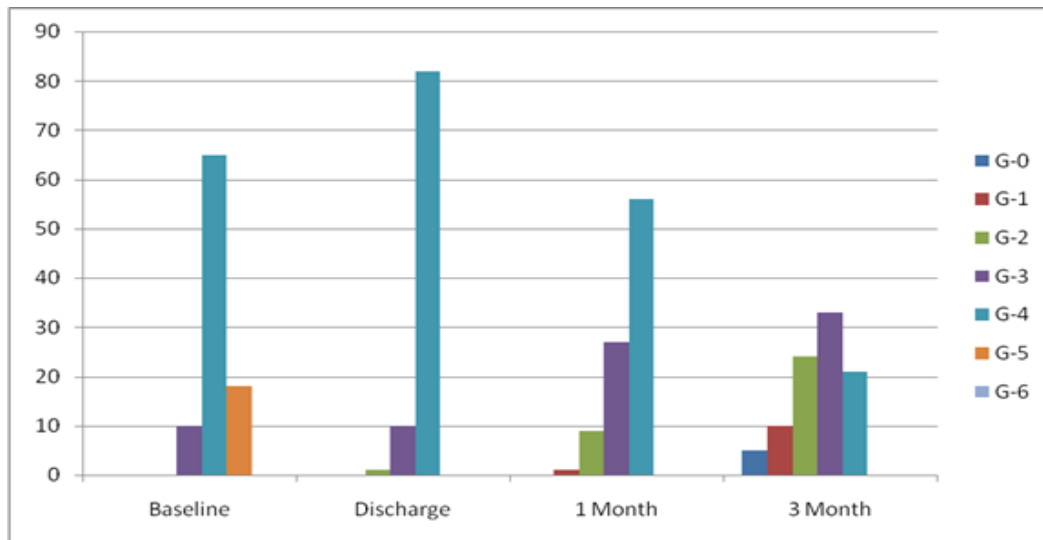


Figure 5: HMS score at different periods.

Table 2: Comparison of outcomes of study subjects regarding treatment modalities.

Variables	Groups				P value
	IVIG and SC, (n=65)		SC group, (n=28)		
	N	%	N	%	
Mechanical ventilation					
Yes	18	27.70	0	0.00	0.001
No	47	72.30	28	100	
Immediate outcome: Hospital stays (Days)					
<7	29	44.60	12	42.90	0.001
>7	36	55.40	16	57.10	
HMS (Admission)					
G 3	7	10.80	3	10.70	0.001
G 4	45	69.20	20	71.40	
G 5	13	20.00	5	17.90	
After 3 months					
G 0	3	4.60	2	7.10	0.001
G 1	7	10.80	3	10.70	
G 2	17	26.20	7	25.00	
G 3	23	35.40	10	35.70	
G 4	15	23.10	6	21.40	

*IVIG: Intravenous immunoglobulin, SC: Supportive care

Table 3: HMS score at baseline, discharge, 1 month and 3 months among the study cases (n=93).

HMS scoring	Baseline	Discharge	1 month	3 months	P value
Total score, mean±SD	4.09±0.54	3.87±0.36	3.48±0.71	2.59±1.11	<0.001

Table 4: Factors affecting functional outcome in GBS patients at 3 months.

Parameters	HMS (0-2), (n=39)	HMS (3-6), (n=54)	P value
Age (in year)			
<5	12 (30.8%)	17 (31.5%)	0.929
5-10	17 (43.6%)	22 (40.7%)	
>10	10 (25.6%)	15 (27.8%)	
Gender			
Male	28 (71.8%)	30 (55.6%)	0.113
Female	11 (28.2%)	24 (44.4%)	

Continued.

Parameters	HMS (0-2), (n=39)	HMS (3-6), (n=54)	P value
Seasons			
Rainy	15 (38.5%)	15 (27.8%)	0.638
Summer	10 (25.6%)	21 (38.9%)	
Winter	14 (35.9%)	18 (33.3%)	
Time to peak deficit			
<3 d	9 (23.1%)	18 (33.3%)	0.029
3-5 d	11 (28.2%)	22 (40.7%)	
6-10 d	11 (28.2%)	10 (18.5%)	
>10	8 (20.5%)	4 (7.4%)	
Weakness			
Quadriplegia	29 (74.4%)	52 (96.3%)	0.003
Paraplegia	10 (25.6%)	2 (3.7%)	
Cranial nerve involved	11 (28.2%)	29 (53.7%)	0.015
Bulbar involvement	9 (23%)	30 (55.5%)	0.003
Autonomic involved	3 (7.7%)	6 (11.1%)	0.536
Nasal intonation	5 (12.8%)	17 (31.5%)	0.098
Neck flexor weakness	2 (5.1%)	19 (35.2%)	0.001
MRC scoring (Baseline)			
0-20	1 (3.7%)	12 (32.4%)	0.001
21-40	18 (66.7%)	25 (67.6%)	
41-60	8 (29.6%)	0 (0%)	
Subtypes of GBS			
AMAN	21 (53.8%)	40 (74.1%)	0.056
AMSAN	2 (5.1%)	1 (1.8%)	
AIDP	16 (41.1%)	13 (24.1%)	
CSF protein (n=82)			
<100 mg/dl	12 (36.4%)	15 (30.6%)	0.637
≥100 mg/dl	21 (63.6%)	34 (69.4%)	
Ventilator support			
MV group	2 (5.1%)	16 (29.6%)	0.003
Non-MV group	37 (94.9%)	38 (70.4%)	
Hospital stays			
<7 days	21 (53.8%)	20 (37%)	0.139
≥7 days	18 (46.2%)	34 (63%)	
Treatment given			
IVIG +SC	22 (56.41%)	43 (79.63%)	0.022
Supportive care only	17 (43.58%)	11 (20.37%)	

Table 5: Independent predictor of poor functional outcome in GBS patients, (n=93).

Variables	Odd ratio	P value	95% CI
Age (in years)	0.967	0.942	0.397-2.356
Gender	0.491	0.113	0.204-1.184
Quadriplegia	8.966	0.007	1.838-43.731
Bulbar involvement	4.167	0.002	1.664-10.436
Neck flexor weakness	10.043	0.003	2.178-46.313
Poor baseline MRC score	12.48	0.019	1.509-103.504
Less time to peak deficit	1.574	0.329	0.634-3.912
Prolonged hospital stays	2.000	0.105	0.864-4.629
Need for mechanical ventilation	7.789	0.009	1.673-36.266
Supportive care only	0.331	0.018	0.132-0.827
High CSF protein	1.295	0.587	0.509-2.396
Axonal variety	2.194	0.084	0.899-5.355

DISCUSSION

In this study, 93 patients under 16 years were included, with the majority (41.9%) aged 5-10 years (mean age 7.73 ± 3.80 years). Koul et al reported a predominance of children below 4 years in their study of 61 children under 15 years.¹⁵ Kumbhar et al found that 50% of their patients were between 6 months to 5 years of age.¹⁶ Males were more prevalent (58%, male to female ratio 1.7:1), consistent with the study by Sarkar et al in Kolkata where 69.06% were male.¹⁷ The most common prior illness was gastroenteritis (26.9%), followed by respiratory tract infection (20.4%), although the organism was not identified. Studies in the Asian subcontinent consistently found gastroenteritis as the most frequent preceding event.^{18,19} Islam et al in Bangladesh also reported that GBS was frequently preceded by enteric infection (46%).²⁰ While GBS is considered sporadic without seasonal preference, one-third (34.4%) of GBS patients in our study presented in the winter season.²¹ Similar results were also found in the study by Sandhya et al.²² In our study, nerve conduction studies (NCS) were performed in all patients, revealing that AMAN was the most common variant of GBS (65.6%), followed by AIDP (28%) and AMSAN (3.2%). These findings align with the study conducted by Ropper et al.²³ However, it's worth noting that the AMAN variety is now considered the most common form of GBS in Asian countries.²⁴ Kalita et al study reported that 66.8% of patients had an antecedent illness, with only 19.4% having a diarrheal illness, and diarrhea was more commonly associated with the AMAN variety.²⁵ Progressive ascending symmetrical muscular weakness with areflexia or hyporeflexia was observed in 90% of GBS cases in various studies, including Haden et al.²⁶ Similar results were seen in our study, where 87.1% of patients presented with flaccid quadriparesis and 44.1% with cranial nerve involvement. This presentation is also consistent with the study done by Habib et al who observed quadriparesis and cranial nerve palsy in 84% and 48% of patients, respectively.²⁷ Acute respiratory failure was observed in 18 (19.4%) of patients in our study, requiring mechanical ventilation with no mortality, similar to the study done by Koul et al.²⁸ Both bulbar and neck flexor weakness are usually correlated with respiratory compromise, probably due to impaired oral and pharyngeal protective reflexes in such patients. Paul et al also found that bulbar involvement was present in 92.5% and 86.6% of patients, respectively, although our study showed 42% of patients had bulbar involvement.²⁹ The acute phase of GBS can be life-threatening, with complications such as respiratory paralysis, secondary infections, bulbar weakness, multisystem involvement, and dysautonomia.²⁸ In our study, acute respiratory failure occurred in 18 (19.4%) patients requiring mechanical ventilation, and there were no fatalities, aligning with findings from Koul et al study.¹⁵ Autonomic dysfunction, observed in 9 (9.7%) patients in our study, contrasted with Rajesh et al study, which reported a higher prevalence in adults (34.4%).³¹ In our study, cerebrospinal fluid analysis, performed in 89.2%

of patients around day 10, revealed albumin-cytological dissociation in all cases. The mean hospital stay duration in our study was 10.03 days, contrasting with Koul et al study, where it exceeded 20 days.¹⁵ The present study identified higher peak disability scores (HMS) in the axonal than the AIDP group, with slower recovery in the former, consistent with findings in Gupta et al study.³¹ In our study, the axonal group consistently exhibited higher peak disability scores during discharge, with a statistically significant difference found in HMS scoring at one-month follow-up compared to the AIDP group ($p=0.006$). Gupta et al study also reported a significant difference in mean discharge disability scores between the AMAN and AIDP groups ($p=0.025$).³¹ Supportive therapy, as advised by the Department of Physical Medicine and Rehabilitation, was provided to all patients in our study. In contrast, Sadek et al study in Egypt administered IVIG to all 50 children, regardless of disease grade, followed by physiotherapy.³² Plasma pheresis was not administered to our patients due to the unavailability of children in our institute. Koul et al prospective study demonstrated improved recovery time and a favorable hospital stay duration with IVIG treatment compared to the pre-IVIG era.³³ Data from similar reports found that IVIG had shortened the time to the first improvement and to regain independent walking.³⁰ Our study also showed a statistically significant difference regarding the need for mechanical ventilation, duration of hospital stays, and outcome after 3 months in the IVIG with supportive care group in comparison to only supportive care group. In our study, 39 (41.9%) patients showed a favorable outcome (HMS 0-2) after 3 months of follow-up. In our study, univariate analysis revealed that clinical parameters such as time to peak deficit, cranial nerve involvement, neck flexor weakness, bulbar involvement, requirement of ventilatory assistance, treatment modalities, and MRC sum score on admission were significantly associated with poor functional outcomes at 3 months, consistent with findings by Verma et al.³⁰ In contrast, Kalita et al multivariate analysis identified peak disability and dysautonomia as independent predictors of poor recovery at 3 months.²⁵ However, in our binary logistic regression analysis, we identified quadriplegia, bulbar involvement, neck flexor weakness, low MRC score, and the need for mechanical ventilation as independent predictors of poor outcomes in childhood GBS.

Limitations

This study's limitations include its scope, demanding larger-scale, multicenter, population-based research to fully discern the pattern of childhood GBS. Additionally, further investigation is advised to ascertain the long-term outcomes and quality of life following childhood GBS, addressing current gaps in understanding. These recommendations underscore the need for more extensive, targeted studies to enhance the depth and breadth of knowledge in this field.

CONCLUSION

In pediatric GBS, there is a notable prevalence of axonal subtypes, which is twice that of demyelinating subtypes. Predictors of functional outcomes in pediatric GBS include quadriplegia, bulbar involvement, neck flexor weakness, a suboptimal baseline Medical Research Council score, and the need for mechanical ventilation. Understanding these patterns and predictors is crucial for diagnosis and prognosis in pediatric GBS.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

- Yuki N, Hartung HP. Guillain-Barré syndrome. *N Engl J Med*. 2012;366(24):2294-304.
- Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. *Neuroepidemiology*. 2011;36(2):123-33.
- Berzegar M, Davari F S, Dastagiri S, Malekian A, Toopchizadeh V. Childhood Guillain Barre Syndrome in the Iran's east Azerbhaijan province: 2001-2005. *Iran J Child Neurol*. 2008;2(4):25-30.
- Mohammad B, Saeed D, Mohammad HK, Ali V. Childhood Guillain-Barre syndrome in the North-West of Iran. *BMC Neurol*. 2007;7:22.
- Ansar V, Valadi N. Guillain-Barré syndrome. *Prim Care*. 2015;42(2):189-93.
- Dash S, Pai AR, Kamath U, Rao P. Pathophysiology and diagnosis of Guillain-Barré syndrome-challenges and needs. *Int J Neurosci*. 2015;125(4):235-40.
- Fujimura H. The Guillain-Barré syndrome. *Handb Clin Neurol*. 2013;115:383-402.
- Arcila-Londono X, Lewis RA. Guillain-Barré syndrome. *Semin Neurol*. 2012;32(3):179-86.
- Korinthenberg R, Schessl J, Kirschner J. Clinical presentation and course of childhood Guillain-Barré syndrome: a prospective multicenter study. *Neuropediatrics*. 2007;38(1):10-7.
- Roodbol J, de Wit MC, Walgaard C, de Hoog M, Catsman-Berrevoots CE, Jacobs BC. Recognizing Guillain-Barre syndrome in preschool children. *Neurology*. 2011;76(9):807-10.
- National Institute of Neurological and Communications Disorders and Stroke. Criteria for diagnosis of Guillain Barré syndrome. *Ann Neurol*. 1978;3(6):565-6.
- Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. *Ann Neurol*. 1990;27(4):21-4.
- Mogale KD, Antony JH, Ryan MM. The pharyngeal-cervical-brachial form of Guillain-Barré syndrome in childhood. *Pediatr Neurol*. 2005;33(4):285-8.
- Legido A, Tenenbaum SN, Katsekos CD and Menkes J. Autoimmune and post-infectious diseases. En: Menkes J, Sarnat HB, María BL, editors. *Child Neurology*. 7th edition. Philadelphia; Lippincott Williams and Wilkins. 2006;557-657.
- Kaul RL, Alfutaisi A. Prospective study of children with Guillain-Barre syndrome. *Indian J Pediatr*. 2008;75(8):787-90.
- Kumbhar SG, Kumbhar SK, Sale MS, Kawada R. Clinical Profile of childhood Guillain Barre Syndrome- A Retrospective study. *Int J Healthcare Biomed Res*. 2016;04(03):19-25.
- Sarkar UK, Menon L, Sarbapalli D, Pal R, Kar S, Singh J, Mondal M. Spectrum of Guillain Barre Syndrome in tertiary care hospital at Kolkata. *JNSBM*. 2011;2(2):211-5.
- Veena K, Naveen S, Suvasini S, Sheffali G, Rama C, Benu D. Outcome in Childhood Guillain-Barré Syndrome. *Indian J Pediatr*. 2009;76(8):795-9.
- Kundu NC. Electrophysiology in the Guillain-Barré syndrome: study of 30 cases. *J Bangladesh College Physicians Surgeons*. 2006;24:54-60.
- Islam Z, Jacobs BC, van Belkum A, Mohammad QD, Islam MB, et al. An axonal variant of Guillain-Barre' syndrome associated with *Campylobacter* infection in Bangladesh. *Neurology*. 2010;74(7):581-7.
- Van Doorn PA, Ruts L, Jacobs BC. Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome. *Lancet Neurol*. 2008;7(10):939-50.
- Sandhya M, Snehalatha IJB, Devender A, Balarami R, Narayana P. Guillain-Barré syndrome: Clinical profile and Consensus to revise Hughes grade 5. *Int J Med Publ Health*. 2016;6(4):193-9.
- Ropper AH. Electrodiagnostic Abnormalities in 113 Consecutive Patients with Guillain-Barre Syndrome. *Arch Neurol*. 1990;47(8):881-7.
- Ye Y, Wang K, Deng F, Xing Y. Electrophysiological subtypes and prognosis of Guillain-Barré syndrome in northeastern China. *Muscle Nerve*. 2013;47(1):68-71.
- Kalita J, Misra UK, Goyal G, Das M. Guillain-Barré syndrome: Subtypes and predictors of outcome from India. *J Peripher Nerv Syst*. 2014;19(1):36-43.
- Haden RDM, Hughes RAC. Guillain-Barre Syndrome recent advances. *Arch Neural*. 2008;41:511-14.
- Habib R, Saifuddin M, Islam R, Rahman A, Bhowmik NB, Haque MA. Clinical Profile of Guillain Barre's Syndrome-Observations from a Tertiary Care Hospital of Bangladesh. *BIRDEM Med J*. 2017;7(1):38-42.
- Rees JH, Thompson RD, Smeeton NC, Hughes RAC. Epidemiological study of Guillain-Barré syndrome in southeast England. *J Neurol Neurosurg Psychiatr*. 1998;64(1):74-7.
- Paul BS, Bhatia R, Prasad K, Padma MV, Tripathi M, Singh MB. Clinical predictors of mechanical ventilation in Guillain-Barré syndrome. *Neurol India*. 2012;60:150-3.

30. Verma R, Chaudhari TS, Raut TP, Garg RK. Clinico-electrophysiological profile and predictors of functional outcome in Guillain–Barre syndrome (GBS). *J Neurological Sci*. 2013;335:105-11.
31. Gupta P K, Sankhyan N, Singhi S and Singhi P. A Comparison of Clinical Profile and Short-term Outcome of Demyelinating and Axonal Subtypes of Guillain-Barré Syndrome in Children. *EC Paediatr*. 2019;8(10):1064-73.
32. Sadek AA, Taleb AA, Ali WA. Outcome of Guillain-Barré Syndrome in Children: A prospective cohort study in a tertiary hospital in Upper Egypt. *Electronic Physician*. 2016;8(12):3318-24.
33. Koul R, Chacko A, Ahmed R. Ten-year prospective study (Clinical Spectrum) of childhood Guillain-

Barré Syndrome in the Arabian Peninsula: Comparison of outcome in patients in pre- and post-intravenous immunoglobulin eras. *J Child Neurol*. 2003;18(11):767-71.

Cite this article as: Sumi SK, Rashid FB, Sumi SN, Shams N, Jasmin T, Debnath B. Clinical profile, electrophysiological pattern and predictors of short-term outcome of Guillain-Barré syndrome in children. *Int J Contemp Pediatr* 2026;13:1886-93.