

Early Changes in Motor and Sensory Nerve Conduction Study Parameters and Prognosis of Pediatric Patient

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Abstract:

Guillain-Barre Syndrome (GBS) is a temporary autoimmune disorder affecting the peripheral nervous system. Typically, it is instigated by viral or bacterial infections or other factors. This condition manifests as sensory alterations or pain, primarily in the back, and entails muscle weakness that originates in the hands and feet before progressing to the upper body.

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Introduction

Guillain-Barre Syndrome (GBS) is a temporary autoimmune disorder affecting the peripheral nervous system. Typically, it is instigated by viral or bacterial infections or other factors. [1] This condition manifests as sensory alterations or pain, primarily in the back, and entails muscle weakness that originates in the hands and feet before progressing to the upper body. [2] GBS is considered a rare ailment, with an annual incidence rate of 0.9 to 2 cases per 100,000 individuals, and it slightly affects more males than females. [3]

While paralytic illnesses with ascending symptoms have been recognized for many centuries, the first documented description of an ascending generalized paralysis dates to 1834. [4] However, it was not until the 20th century, with the introduction of diagnostic lumbar puncture, that the critical features of the illness were fully understood. [5]

In the vast majority of GBS patients, prompt initiation of treatment is crucial. If the patient has already entered the plateau stage, treatment may no longer be necessary. The abnormalities in nerve conduction studies (NCS) can be detected by the end of the first week of illness and are most notable by the second week after the initial weakness sets in. [6] Currently, during the first week of symptom onset, there is no definitive diagnostic method for GBS.

However, a meta-analysis of randomized clinical trials reveals that treatment can reduce the need for mechanical ventilation by nearly 50% and increase the chances of complete recovery after one year. Significant functional improvement may occur toward the end of the first week of treatment.

Therefore, early diagnosis and timely initiation of treatment are of utmost importance. [7]

Previous studies have shown that Nerve conduction studies can be used as a tool for the diagnosis of GBS within the first week of symptom onset. This novel study aims to study early nerve conduction changes in the paediatric population of GBS. Investigating early nerve conduction changes in the paediatric population with Guillain-Barré Syndrome can contribute to our understanding of the disease and potentially improve diagnostic capabilities in this specific group.

Studying the paediatric population specifically is important because GBS can present differently in children compared to adults. The clinical manifestations, disease progression, and response to treatment may vary in pediatric patients. Additionally, it may provide insights into the patterns of nerve conduction abnormalities observed early in the disease course, potentially aiding in differentiating GBS from other neurological conditions. [8]

The findings from this novel study have important implications for early diagnosis and intervention in paediatric GBS cases. Early detection can lead to the timely initiation of appropriate treatments such as intravenous immunoglobulin (IVIg) or plasmapheresis, which have been shown to improve outcomes in GBS. [7] Moreover, understanding the early nerve conduction changes specific to the paediatric population may contribute to refining diagnostic criteria and optimizing management strategies for this age group.

Material and Methodology

This was an observational prospective study of Guillain-Barré syndrome and its variants in children conducted from January 2022 to March 2023 in the Physiology and Pediatrics Department (only inpatient) at AIIMS Patna Hospital.

Ethical Consideration:

The protocol of the study was approved by the Institutional Research Cell (Ref No. AIIMS/Pat/IRC/2020/PGTh/Jan21/24) and Institutional Ethics Committee of AIIMS Patna (Ref. No. AIIMS/Pat/IEC/PGTh/Jan21/24). Informed written consent was obtained from parents of all enrolled children.

Inclusion Criteria:

1. All patients of GBS based on Asbury's criteria which included ascending areflexic quadriparesis, with or without cranial nerve dysfunction, evolving within a period of four weeks. [9]
2. Age ≥ 1 year to 15 years
3. Patients not already on a mechanical ventilator

Exclusion Criteria:

1. Age ≤ 1 year or ≥ 15 years
2. Miller-Fischer syndrome
3. Atypical GBS
4. Features of other diseases like myasthenia gravis, botulism, poliomyelitis, porphyria, and diphtheria
5. Drug or toxin-induced acute neuropathy.

All Patients Underwent:

- 1) Pediatric Neurological Sheet was checked and General and Neurological history was recorded.
- 2) General and Neurological Examination
- 4) Electrophysiological investigations such as:

Nerve conduction studies were performed on all patients using the Neurosoft Neuro MEP-NET machine and following the standard protocol. NCS

- a) All electrophysiological studies were performed using Neurosoft Neuro MEP-NET machine electromyography machine with

surface recording and stimulating electrodes.

- b) Nerve conduction studies were conducted three times for each patient: first within 3 days of admission, second between 3-7 days and lastly between 7-14 days of admission.
- c) Motor nerve conduction studies of median, ulnar, radial, deep peroneal, and posterior tibial, were recorded bilaterally.
- d) Sensory conduction study of median, ulnar, radial, superficial peroneal nerve, and sural nerves were also recorded bilaterally. Amplitude of sensory nerve action potential (SNAP), peak latency and CV was measured in sensory nerves.
- e) Values were defined as abnormal if they were outside of the age corrected normal range and expressed as a percentage of the upper (ULN) or lower (LLN) limit of normal.
 - As subjects were children of age group 5 years or older, adult nerve conduction values were taken as normal because the nerve conduction parameters of children approach adult value by that age.
- f) Based on nerve conduction study (NCS), patient were classified into: AIDP, AMAN, AMSAN, Inexcitable and equivocal (Appendix 3). [11]

Results

During the study period, 11 patients were recruited who were diagnosed to have GBS and admitted in department of Pediatrics, AIIMS Patna. There were 5 female patients and 6 Male patients. We found a seasonal clustering pattern in this study, with majority of cases occurring in winter (44.12%), followed by summer (29.41%).

NCSs were performed in 11 children. Three readings of NCS were taken. First within 3 days of admission, second between 3-7 days and last between 7-14 days of admission. All neurophysiological examinations were abnormal. Seven (63.64%) out of 11 children had demyelinating (AIDP) pattern, 04 (36.36%) had axonal (AMAN) pattern. No AMSAN or inexcitable electrophysiological subtypes were seen in this study as in Table (3).

Table 3. Electrophysiological subtypes of childhood GBS.

	GBS Electrophysiological subtypes	Numbers (%)
Total		11 (100)
	AIDP	7 (63.63%)
	AMAN	4 (36.36%)

The data presented in Table 4 reveals that a comprehensive total of 110 nerve conduction studies were undertaken.

Table 4. Electrophysiological Abnormality in childhood GBS.

GBS Electrophysiological Abnormality	Numbers
Motor NCS	110 (100%)
Abnormal total	92(83.63%)
Abnormal CMAP amplitude	45(40.9%)
Abnormal MCV	40 (36.36%)
Abnormal distal latency	23 (20.9%)
Conduction block	11 (10%)
Sensory NCS	110(100%)
Abnormal total	7 (8.18%)
Abnormal peak latency	0 (0%)
Abnormal SNAP amplitude	7 (8.18%)
Abnormal SCV	0 (0%)

Among these studies, an overwhelming 83.63% exhibited abnormalities, underscoring the significant prevalence of motor dysfunction as a primary characteristic. In contrast, sensory studies predominantly demonstrated normal results, with a mere 8.18% of the total 110 studies indicating abnormalities. It is worth noting that the detected abnormalities primarily centered around the SNAP (Sensory Nerve Action Potential) amplitude.

Discussion

GBS can occur at any age, but it predominantly manifests in children above three years old. [12] However, there is a lack of comprehensive data on GBS in developing countries. One significant challenge in understanding GBS in children is the inconsistency in its clinical characteristics across different studies. The variations in findings could be attributed to the diverse geographical and racial backgrounds of the studied populations. This highlights the need for more extensive research to comprehend the nuances of GBS in various regions and demographics. [13]

In this investigation, all conducted neurophysiological assessments displayed abnormal findings. This observation aligns with prior research by Korinthenberg et al. [14], van Doorn et al. [15], and Devos et al. [16], wherein all electrodiagnostic (EDx) evaluations yielded abnormal results, underscoring the significance of early EDx in GBS. However, in contrast to studies conducted by Sadek et al. and Benamer & Bredan [17], where 6% and 1% of patients respectively exhibited normal nerve conduction study results, our study demonstrated a higher rate of electrophysiological abnormalities. One potential explanation for this elevated rate of abnormalities could be the extended time lapse between clinical onset and neurophysiologic examination in children, owing to the diagnostic challenges inherent in pediatric cases.

This study's noteworthy observations reveal that the most frequent anomaly detected was a reduction in Compound Muscle Action Potential (CMAP) amplitude, affecting 40.9% of patients, followed by abnormal motor conduction velocity, present in

36.36% of cases. Increased motor latencies were evident in 20.9% of patients, while conduction block was discerned in 10% of cases. This pattern concurs with findings from prior studies by Ye et al. [18] and Devos et al. [16]. The occurrence of conduction block in approximately one-third of cases during the early stages of GBS aligns with the characteristic conduction slowing associated with demyelination [19].

Shifting focus to sensory Nerve Conduction Studies (NCS), our study identified aberrant outcomes in only 8.18% of cases. This discovery echoes the results of Ye et al. [18], who noted abnormal sensory NCS in just 24% of childhood GBS cases. This pattern suggests a more pronounced involvement of motor nerves in childhood GBS.

The distribution of subtypes based on nerve conduction studies unveiled that 63.63% of patients exhibited AIDP, while 36.36% displayed AMAN, and no patients were categorized under the AMSAN or unclassified groups. This distribution closely mirrors the findings of Sadek et al. [20], who reported 52% AIDP and 36% AMAN cases. Similarly, Benamer & Bredan [17] documented 44% AIDP and 35% AMAN cases. Comparable results were also evident in various studies [21-23] conducted across Europe and the USA, where AIDP stood as the dominant subtype.

In contrast, developing and Asian countries exhibited divergences, with AMAN frequencies reported at 38% in Mexico, 27.8% in Turkey, 44.2% in southern India, 56% in Bangladesh, 35% in Iran, and 47% in Japan². Notably, our study observed a lower incidence of AMAN compared to earlier reports. These disparities suggest potential geographic and regional variations, which might be influenced by factors like genetic backgrounds and environmental exposures [19].

Conclusion

GBS affects both sexes; however, males were affected more than females in this study. AIDP was the commonest subtype in studied population, followed by AMAN variant. NCS abnormality was

seen in all the recruited subjects. The results indicate that the inclusion of nerve conduction study in the diagnostic process can enhance the accuracy and efficiency of identifying GBS cases at an early stage.

References

1. Winer JB, Hughes RA, Anderson MJ, Jones DM, Kangro H, Watkins RP. A prospective study of acute idiopathic neuropathy. II. Antecedent events. *J Neurol Neurosurg Psychiatry*. 1988 May;51(5):613–8.
2. Hughes RA, Rees JH. Clinical and epidemiologic features of Guillain-Barré syndrome. *J Infect Dis*. 1997 Dec;176 Suppl 2: S92-98.
3. Govoni V, Granieri E. Epidemiology of the Guillain-Barré syndrome. *Curr Opin Neurol*. 2001 Oct;14(5):605–13.
4. Asbury AK, McKhann GM. Changing views of Guillain-Barré syndrome. *Ann Neurol*. 1997 Mar;41(3):287–8.
5. Gregson NA, Koblar S, Hughes RA. Antibodies to gangliosides in Guillain-Barré syndrome: specificity and relationship to clinical features. *Q J Med*. 1993 Feb;86(2): 111–7.
6. Gupta D, Nair M, Baheti NN, Sarma PS, Kuruvilla A, Diplomate-American Board. Electrodiagnostic and clinical aspects of Guillain-Barré syndrome: an analysis of 142 cases. *J Clin Neuromuscul Dis*. 2008 Dec;10(2):42–51.
7. Treatment of Guillain-Barré syndrome with high-dose immune globulins combined with methylprednisolone: a pilot study. The Dutch Guillain-Barré Study Group. *Ann Neurol*. 1994 Jun;35(6):749–52.
8. Berciano J, Orizaola P, Gallardo E, Pelayo-Negro AL, Sánchez-Juan P, Infante J, et al. Very early Guillain-Barré syndrome: A clinical-electrophysiological and ultrasonographic study. *Clin Neurophysiol Pract*. 2019 Nov 30;5:1–9.
9. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. *Ann Neurol*. 1990;27 Suppl:S21-24.
10. Zhang G, Li Q, Zhang R, Wei X, Wang J, Qin X. Subtypes and Prognosis of Guillain-Barré Syndrome in Southwest China. *PloS One*. 2015;10(7):e0133520.
11. Hughes RAC, Cornblath DR. Guillain-Barré syndrome. *Lancet Lond Engl*. 2005 Nov 5;366(9497):1653–66.
12. Kannan MA, Ch RK, Jabeen SA, Mridula KR, Rao P, Borgohain R. Clinical, electrophysiological subtypes and antiganglioside antibodies in childhood Guillain-Barré syndrome. *Neurol India*. 2011;59(5):727–32.
13. Wu X, Shen D, Li T, Zhang B, Li C, Mao M, et al. Distinct Clinical Characteristics of Pediatric Guillain-Barré Syndrome: A Comparative Study between Children and Adults in Northeast China. *PloS One*. 2016;11(3):e0151611.
14. Korinthenberg R, Schessl J, Kirschner J. Clinical presentation and course of childhood Guillain-Barré syndrome: a prospective multicentre study. *Neuropediatrics*. 2007 Feb;38(1):10–7.
15. Van Doorn PA, Kuitwaard K, Walgaard C, van Koningsveld R, Ruts L, Jacobs BC. IVIG treatment and prognosis in Guillain-Barré syndrome. *J Clin Immunol*. 2010 May;30 Suppl 1(Suppl 1):S74-78.
16. Devos D, Magot A, Perrier-Boeswillwald J, Fayet G, Leclair-Visonneau L, Ollivier Y, et al. Guillain-Barré syndrome during childhood: particular clinical and electrophysiological features. *Muscle Nerve*. 2013 Aug;48(2):247–51.
17. Benamer HTS, Bredan A. Guillain-Barré syndrome in Arab countries: a systematic review. *J Neurol Sci*. 2014 Aug 15;343(1–2):221–3.
18. Ye YQ, Wang KR, Sun L, Wang Z. Clinical and electrophysiologic features of childhood Guillain-Barré syndrome in Northeast China. *J Formos Med Assoc Taiwan Yi Zhi*. 2014 Sep;113(9):634–9.
19. Singh S PL. Nerve Conduction Studies In Guillain-Barré Syndrome. *The Internet Journal of Neurology*. Volume 16(2013).
20. Sadek AA, Abou-Taleb A, Ali WA. Outcome of Guillain - Barré Syndrome in Children: A prospective cohort study in a tertiary hospital in Upper Egypt. *Electron Physician*. 2016 Dec;8(12):3318–24.
21. Lee JH, Sung IY, Rew IS. Clinical presentation and prognosis of childhood Guillain-Barré syndrome. *J Paediatr Child Health*. 2008;44(7–8):449–54.
22. Asbury AK. New concepts of Guillain-Barré syndrome. *J Child Neurol*. 2000 Mar;15(3):183–91.
23. Tekgul H, Serdaroglu G, Tutuncuoglu S. Outcome of axonal and demyelinating forms of Guillain-Barré syndrome in children. *Pediatr Neurol*. 2003 Apr;28(4):295–9.