RESEARCH PAPER

Electrophysiological Correlation and Clinical Outcomes in Guillain–Barré Syndrome: A Prospective Cohort Study

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INTRODUCTION

Guillain–Barre syndrome (GBS) is the leading cause for acute neuromuscular weakness worldwide. After the introduction of vaccine for poliomyelitis it is the commonest cause of acute flaccid paralysis in the world.[1]

This is the most common cause of acute or subacute generalized paralysis in practice, It occurs in all parts of the world and in all seasons, affecting children and adults of all ages and both sexes.[2] It has been classically described as an acute ascending paralysis with areflexia, associated with antecedent infective illness and cerebrospinal fluid albumin-cytological dissociation.[3] The disease progresses through a phase of progressive weakness lasting for up to four weeks, followed by a variable plateau phase and then a recovery phase.[4]

Guillain Barre Syndrome (GBS) is classically a clinical diagnosis but electrophysiology and nerve conduction studies help in supporting the diagnosis, making a distinction between axonal and demyelinating variants as well as helping in prognostication. The characteristic findings supportive of acute inflammatory demyelinating polyradiculo-neuropathy include prolonged or absent *F* wave latencies, conduction blocks at non- entrapment sites, prolonged distal motor latencies, reduced conduction velocities, and temporal dispersion. [5]

The abnormalities in NCS may be recorded by the first week of illness and are most pronounced by the second week after the onset of weakness. To increase the certainty of diagnosis with NCS and to increase the diagnostic yield, recordings at least four motor nerves, three sensory nerves, *F* waves, and H- reflexes should be obtained.[2] The electrophysiological study may play important role in further investigation of the pathogenesis and assessment of prognosis.[6]

Treatment of GBS is mainly by supportive care, plasma exchange or intravenous immunoglobulin has favorable outcome and complete recovery in most individuals.

MATERIALS AND METHODS

This prospective longitudinal cohort study was conducted between April 2024 and March 2025 in the Department of Neurology at a tertiary care center. The hospital is a high-volume referral center catering to both urban and rural populations. Patients were recruited using a convenience sampling method. The study protocol was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrollment. The study was conducted in accordance with the Declaration of Helsinki. This was a single-study dataset, and no prior publication or future reuse of the data is planned.

STUDY POPULATION

A total of 26 consecutive patients diagnosed with Guillain–Barré Syndrome (GBS) were included in the study. Diagnosis was based on clinical criteria consistent with Asbury's guidelines, including progressive weakness of less than 4 weeks duration, areflexia, and with or without cranial nerve involvement. Patients presenting with clinical variants of GBS were also included.

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Patients were excluded if they had early bladder or bowel involvement, marked asymmetry of symptoms, or alternative diagnoses such as myasthenia gravis, botulism, porphyria, or drug-induced neuropathy. Patients below 18 years of age were also excluded.

CLINICAL ASSESSMENT

All patients underwent detailed clinical evaluation at baseline using a structured proforma. Data collected included demographic characteristics, antecedent illness, clinical presentation, cranial nerve involvement, autonomic dysfunction, and treatment details.

Neurological assessment included evaluation of motor strength using the Medical Research Council (MRC) sum score and disability assessment using the Guillain–Barré Syndrome Disability Scale (GBSD).

ELECTROPHYSIOLOGICAL ASSESSMENT

Nerve conduction studies were performed using standard techniques. At least three motor nerves (median, ulnar, and common peroneal) and three sensory nerves (median, ulnar, and sural) were evaluated. Parameters assessed included distal latency, compound muscle action potential (CMAP) amplitude, sensory nerve action potential (SNAP) amplitude, conduction velocity, and F-wave latency.

Patients were classified into electrophysiological subtypes—acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute motor sensory axonal neuropathy (AMSAN)—based on established criteria.

FOLLOW-UP

All patients underwent repeat clinical and electrophysiological evaluation after 2 months. Changes in MRC sum score, GBSD score, and electrophysiological parameters were recorded and compared with baseline findings.

STATISTICAL ANALYSIS

Data were entered into Microsoft Excel and analyzed using appropriate statistical methods. Continuous variables were expressed as mean ± standard deviation, and categorical variables were expressed as percentages.

Comparisons between baseline and follow-up clinical scores were performed using the Wilcoxon signed-rank test. Differences among electrophysiological subtypes were analyzed using analysis of variance (ANOVA) for continuous variables and the Kruskal–Wallis test where appropriate. A p-value of < 0.05 was considered statistically significant.

RESULTS

A total of 32 patients with suspected Guillain–Barré Syndrome (GBS) were evaluated during the study period. Six patients were excluded for the following reasons: (i) alternative diagnoses such as myasthenia gravis or metabolic neuropathy (n = 2); (ii) early bladder or bowel involvement suggestive of alternate pathology (n = 1); (iii) marked asymmetry of weakness (n = 1); and (iv) refusal to participate or incomplete data (n = 2). Finally, 26 patients were included in the analysis.

EPIDEMIOLOGICAL FEATURES

The mean age of the study population was 50.04 ± 15.34 years, with a male predominance (61.5%). The majority of patients were from rural areas (69.2%). A preceding illness was reported in a significant proportion of patients, with diarrhea (30.8%) and upper respiratory tract infection (19.2%) being the most common antecedent events.

CLINICAL CHARACTERISTICS

At presentation, all patients had progressive limb weakness with areflexia. Cranial nerve involvement was observed in 53.8% of patients, most commonly affecting facial nerves. Sensory symptoms were present in 52.8% of patients, while autonomic dysfunction was noted in a subset of cases.

The mean Medical Research Council (MRC) sum score at baseline was 42.73, which improved to 49.35 at 2-month follow-up. Similarly, the Guillain–Barré Syndrome Disability (GBSD) score improved significantly from 3.35 at baseline to 2.23 at follow-up.

TREATMENT PROFILE

Most patients received intravenous immunoglobulin (IVIg) therapy (69.2%), while the remaining patients underwent plasma exchange (30.8%). Both treatment modalities were associated with clinical improvement.

ELECTROPHYSIOLOGICAL FINDINGS

At baseline, electrophysiological evaluation showed that acute inflammatory demyelinating polyradiculoneuropathy (AIDP) was the most common subtype (50%), followed by axonal variants including acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN).

At follow-up, the proportion of patients classified as AIDP increased to 61.5%, while the number of equivocal or normal studies decreased, indicating improved diagnostic clarity with serial nerve conduction studies.

Motor nerve abnormalities included prolonged distal latencies, reduced conduction velocities, and abnormal F-wave latencies, while sensory nerve involvement was variable. Axonal variants demonstrated reduced compound muscle action potential amplitudes with relatively preserved conduction velocities.

OUTCOME ANALYSIS

Significant improvement was observed in clinical parameters between baseline and follow-up. The improvement in MRC sum score and GBSD score was statistically significant ($p < 0.05$).

When analyzed by subtype, patients with AIDP demonstrated better recovery compared to axonal variants, particularly AMSAN, which was associated with poorer outcomes. AMAN showed intermediate recovery patterns.

DISCUSSION

Guillain–Barré Syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy and remains the leading cause of acute neuromuscular paralysis in the post-polio era.[7-9] The pathogenesis is largely attributed to molecular mimicry, particularly following infections such as *Campylobacter jejuni*, resulting in cross-reactive immune responses against peripheral nerve components, as described by Hughes et al. and van Doorn et al.[10-12]

The clinical profile observed in our study, characterized by progressive, symmetric limb weakness with areflexia, is consistent with classical descriptions. However, variability in presentation, including cranial nerve involvement and sensory symptoms, contributes to diagnostic challenges, especially in atypical cases. In such scenarios, nerve conduction studies play a pivotal role in confirming diagnosis and enabling subtype classification into acute inflammatory demyelinating polyradiculoneuropathy, acute motor axonal neuropathy, and acute motor sensory axonal neuropathy, as proposed by Hadden et al.[13]

The demographic findings in our cohort, with a mean age of approximately 50 years and a male predominance, are consistent with those of previous studies by Sudulagunta et al. and Shahrizaila et al.[7,14] Although GBS affects all age groups, a higher incidence in adults and elderly populations has been consistently reported by Hughes et al.[10-11]

Antecedent infections were noted in a significant proportion of patients, particularly diarrheal illness and upper respiratory tract infections. Similar patterns have been described in earlier studies by Sharma et al. and Sinha et al., reinforcing the role of immune activation in disease onset. The immunopathological cascade involves activation of T-cells, cytokine-mediated disruption of the blood–nerve barrier, complement activation, and macrophage-mediated demyelination, as elaborated by McGrogan et al.[9]

This can further be substantiated by the following list of infections reported in the previous studies.

Study	Preceding illness				
	URTI	Gastro intestinal	Diarrhoea	Fever	Others
Current study	19.2%	-	30.8%	19.2%	-
Sharma A et al [15]	14%	32 %	47 %	12 %	-
Sinha S et al [16]	11%	-	20 %	15 %	Campylobacter infection-41
Sudulagunta et al [7]	35 %	47 %	-	43 %	Others-4
Pieter A van Doorn et al [12]	Cough-48 % Sore throat- 39 % Nasal discharge- 30%	-	27 %	52 %	-
Nortina Shahrizaila [14]	48 %	-	0.05 %	0.05 %	-

Cranial nerve involvement was observed in more than half of our patients, with facial nerve palsy being the most common, consistent with existing literature by van Doorn et al. and Sudulagunta et al.[7,12] Sensory disturbances were also frequently noted, with pain and paresthesia being predominant, reflecting both nociceptive and neuropathic mechanisms during different disease phases.

A summary of the findings found in different studies is as follows:

Study	AIDP	AMAN	AMSAN
Guptha D et al [17]	85%	11 %	-
A Sharma et al [15]	60%	34%	6%
S Sinha et al [16]	43%	33%	24%
Kannan et al [8]	49%	44%	-
Nortina Shahrizaila [14]	81%	4.2%	4.3%
Pokalkar D et al [18]	56%	14%	20%

Electrophysiological evaluation revealed acute inflammatory demyelinating polyradiculoneuropathy as the most common subtype, both at baseline and follow-up, consistent with global and Indian data reported by Gupta et al. and Sharma et al.[15,17] Serial nerve conduction studies demonstrated evolution of findings over time, with increased detection of abnormalities at follow-up and reclassification of some cases. Similar subtype conversion has been reported by Hiraga et al. and Vyas et al.[19-20], emphasizing that early electrophysiological studies may underestimate disease severity or subtype. Reduced compound muscle action potential amplitude was the most frequent abnormality, while conduction velocity slowing was less commonly observed, particularly in early stages.

Clinical outcomes assessed using the GBS Disability Scale and Medical Research Council sum score showed significant improvement at 2 months. These findings are in line with previous studies by Arami et al.[21],

demonstrating favorable recovery with appropriate treatment. Subtype-wise analysis revealed better outcomes in demyelinating variants compared to axonal variants, particularly acute motor sensory axonal neuropathy, which showed greater disability. Similar observations have been reported by Smith et al.[22], highlighting poorer prognosis in axonal forms. However, studies by Hadden et al.[13] have shown variability in outcomes, particularly in acute motor axonal neuropathy, possibly due to reversible conduction failure.

A comparison of the follow-up GBSD scores on each grade from the present and the other studies are as follows:

Study	Frequency distribution (%) of patients within different grades in GBS disability scale					
	1	2	3	4	5	6
Present study	26.9	23.1	34.6	11.5	0	0
Vyas A et al [19]	0	3.57	28.57	57.14	10.71	0
Ropper et al [23]	0	11.6	16.5	47.5	33.9	0
Nortina Shahrizaila et al [14]	0	4.7	9.4	52.3	33.33	0
Kannan et al [8]	11.32	38.67	15.09	17.92	38.50	4.11

Both intravenous immunoglobulin and plasma exchange were effective treatment modalities in our study, with no significant difference in outcomes. This is consistent with findings by Bril et al., Visser et al., and Romano et al.,[24-26] which demonstrated comparable efficacy between the two therapies. Therefore, treatment decisions should be individualized based on patient characteristics, availability, and contraindications.

A comparison of the scores from the current and the other study findings is as follows:

Study	Findings
Present study	The disability for the 3 groups was- AIDP<AMAN<AMSAN
Prem Chand et al [27]	The disability for the 3 groups was- AIDP<AMSAN<AMAN
Smith et al [25]	Axonal pattern had the worst prognosis
Hadden et al [13]	Axonal pattern showed both faster and slower recovery but demyelinating pattern showed only slower recovery.
Vyas A et al [22]	77% of axonal and only 42.86% of demyelinating showed recovery with better prognosis for axonal

Recovery and prognosis have been better for AIDP than the axonal types in most of the cases.

Complications related to prolonged immobilization, including bedsores, aspiration pneumonia, and muscle wasting, were observed in a few patients, highlighting the importance of comprehensive supportive care.[28-30]

CONCLUSION

Serial nerve conduction studies significantly enhance diagnostic accuracy in Guillain–Barré Syndrome and improve subtype classification over time. Acute inflammatory demyelinating polyradiculoneuropathy was the most common electrophysiological subtype observed in this study. Demyelinating variants demonstrated a better prognosis compared to axonal variants. A strong correlation was noted between electrophysiological findings and clinical recovery. Follow-up nerve conduction studies are essential for accurate diagnosis and prognostication.

LIMITATIONS

This study was limited by a relatively small sample size, its single-center design, and a short duration of follow-up, which may affect the generalizability of the findings.

CLINICAL IMPLICATIONS

The findings support the important role of drug delivery strategies such as intravenous immunoglobulin and plasma exchange in the management of Guillain–Barré Syndrome. The study emphasizes the importance of early initiation and appropriate timing of therapy. It also highlights the need for a subtype-based therapeutic approach guided by electrophysiological classification to optimize patient outcomes.

REFERENCES

1. Stephan L, Hauser A, Asbury A. Guillain–Barré syndrome and other immune mediated neuropathies. In: Harrison's Principles of Internal Medicine. 16th ed. 2005;2:2513-6.
2. Ropper AH, Brown RH. Adams and Victor's Principles of Neurology. 8th ed. New York: McGraw-Hill; 2005.
3. Guillain G, Barré JA, Strohl A. Sur un syndrome de radiculonevrite avec hyperalbuminose du liquide céphalo-rachidien sans réaction cellulaire. Bull Mem Soc Med Hop Paris. 1916;40:1462-70.

4. Van den Berg B, Walgaard C, Drenthen J, Fokke C, Jacobs BC, van Doorn PA. Guillain–Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. Nat Rev Neurol. 2014;10:469-82.
5. Nagappa M, Netto AB, Taly AB, Kulkarni GB, Umamaheshwara Rao GS, Periyavan S, Rao S. Electrophysiological observations in critically ill Guillain–Barré syndrome. Neurol India. 2016;64:914-20.
6. Willison HJ, Jacobs BC, van Doorn PA. Guillain–Barré syndrome. Lancet. 2016; pii:S0140-6736(16)00339-1.
7. Sudulagunta SR, Sodalagunta MB, Sepehrar M, et al. Guillain–Barré syndrome: clinical profile and management. Ger Med Sci. 2015;13:1-15.
8. 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.
9. McGrogan A, Madle GC, Seaman HE, De Vries CS. The epidemiology of Guillain–Barré syndrome worldwide. Neuroepidemiology. 2009;32(2):150-63.
10. Hughes RA, Charlton J, Latinovic R, Gulliford MC. No association between immunization and Guillain–Barré syndrome in the United Kingdom, 1992 to 2000. Arch Intern Med. 2006;166:1301-4.
11. Hughes RA, Rees JH. Clinical and epidemiologic features of Guillain–Barré syndrome. J Infect Dis. 1997;176(Suppl 2):S92-8.
12. Van Doorn PA, Ruts L, Jacobs BC. Clinical features, pathogenesis, and treatment of Guillain–Barré syndrome. Lancet Neurol. 2008;7(10):939-50.
13. Hadden RD, Karch H, Hartung HP, Zielasek J, Weissbrich B, Schubert J, et al. Preceding infections, immune factors, and outcome in Guillain–Barré syndrome. Neurology. 2001;56(6):758-65.
14. Shahrizaila N, Goh KJ, Abdullah S, Kuppusamy R, Yuki N. Two sets of nerve conduction studies may suffice in Guillain–Barré syndrome. Clin Neurophysiol. 2013;124(7):1456-9.
15. Sharma A, Lal V, Modi M, Vaishnavi C, Prabhakar S. Campylobacter jejuni infection in Guillain–Barré syndrome. Neurol India. 2011;59(5):717.
16. Sinha S, Prasad KN, Jain D, Pandey CM, Jha S, Pradhan S. Preceding infections and antiganglioside antibodies in Guillain–Barré syndrome. Clin Microbiol Infect. 2007;13(3):334-7.
17. Gupta D, Nair M, Baheti NN, Sarma PS, Kuruvilla A. Electrodiagnostic and clinical aspects of Guillain–Barré syndrome. J Clin Neuromuscul Dis. 2008;10:42-51.
18. Pokalkar D, Narisetty V, Polusani R, Kamera S, Poosarla SS. Topographic variants of Guillain–Barré syndrome. IAIM. 2015;2:32-8.
19. Vyas A, Swami S, Jaiswal K. Serial electrophysiological changes in Guillain–Barré syndrome. Int J Adv Med. 2016;3(3):552.
20. Hiraga A, Mori M, Ogawara K, Kojima S, Kanesaka T, Misawa S, et al. Recovery patterns in axonal Guillain–Barré syndrome. J Neurol Neurosurg Psychiatry. 2005;76(5):719-22.
21. Arami MA, Yazdchi M, Khandaghi R. Epidemiology of Guillain–Barré syndrome in northwest Iran. Ann Saudi Med. 2006;26:22-7.
22. Smith TA. Prognosis in Guillain–Barré syndrome using clinical and neurophysiological features. Ugeskr Laeger. 2000;162(36):4805-7.
23. Ropper AH, Wijdicks EF, Shahani BT. Electrodiagnostic abnormalities in Guillain–Barré syndrome. Arch Neurol. 1990;47(8):881-7.
24. Bril V, Ilse WK, Pearce R, Dhanani A, Sutton D, Kong K. Immunoglobulin versus plasma exchange in Guillain–Barré syndrome. Neurology. 1996;46(1):100-3.
25. Visser LH, Van der Meché FG, Meulstee J, Rothbarth P, Jacobs BC, Schmitz PI, Van Doorn PA. Cytomegalovirus infection and Guillain–Barré syndrome. Neurology. 1996;47(3):668-73.
26. Romano JG, Rotta FT, Potter P, Rosenfeld V, Santibanez R, Rocha B, Bradley WG. Relapses in Guillain–Barré syndrome after treatment. Muscle Nerve. 1998;21(10):1327-30.
27. Chand P, Jan F, Kaleem S, Yousafzai MT, Ibrahim S. Clinical features of Guillain–Barré syndrome using Hughes scoring system. Asia Pac J Clin Trials Nerv Syst Dis. 2017;2(2):45.
28. Tekgul H, Serdaroglu G, Tutuncuoglu S. Clinical features and prognosis of Guillain–Barré syndrome in children. Pediatr Neurol. 2003;28(4):295-9.
29. Walgaard C, Lingsma HF, Ruts L, Drenthen J, van Koningsveld R, Garssen MP, et al. Prediction of respiratory insufficiency in Guillain–Barré syndrome. Ann Neurol. 2010;67(6):781-7.
30. Van Koningsveld R, Steyerberg EW, Hughes RA, Swan AV, van Doorn PA, Jacobs BC. A clinical prognostic scoring system for Guillain–Barré syndrome. Lancet Neurol. 2007;6(7):589-94.