

Original Research Article

Guillain–Barre syndrome - clinical profile, diagnosis and recovery in a tertiary care setting

Dipali Y. Uikey*, Anita Paritekar

Rajarshree Chattrapati Shahu Maharaj Government Medical College and Chhatrapati Pramilitai Raje Hospital, Kolhapur, Maharashtra, India

Received: 15 August 2025

Revised: 18 September 2025

Accepted: 17 October 2025

*Correspondence:

Dr. Dipali Y. Uikey,

E-mail: uikeydipali@gmail.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–Barre syndrome (GBS) is an autoimmune disease and a recognized cause of generalized progressive paralysis worldwide. The present study was aimed to document the clinical finding, diagnostic challenges, and management outcome amongst the patients with GBS during the hospital stay.

Methods: A retrospective analysis of 22 cases diagnosed as GBS was conducted. Medical records and the data related to age, sex, antecedent illness, muscle power graded by the Medical Research Council scale, functional scores, details of intensive care unit complications and need for ventilation were obtained.

Results: GBS seems to affect all age groups with male preponderance. The male-to female ratio was 2.5:1. Maximum number of patients (50 %, n=11) were in the age group of 60 years and above. The next common age group was 13-30 years in which 31.8% (n=7) patient. Nearly 72% patients (n=16) had history of preceding illness. Paraparesis as a common clinical feature 54.5% (n=12). The majority (n=20) 90.9% were found to be of AMSAN followed by AIDP variants (n=2) 9.09%. About 59.09% patient (n=13) improved at the time of discharge on their F-scores with mild disability.

Conclusions: GBS remains a neurologic emergency that requires early recognition and prompt management to improve patient outcomes. Our study highlights the diverse clinical presentations and underscores the importance of supportive care and timely initiation of immunotherapy. While most patients show favourable recovery with appropriate treatment, residual deficits and complications can persist, emphasizing the need for ongoing rehabilitation and follow-up. Increased awareness among clinicians, especially in primary care and emergency settings, is crucial for early diagnosis. Further research is warranted to explore predictive markers of prognosis and optimize long-term care strategies.

Keywords: Acute motor axonal neuropathy, Guillain–Barre syndrome

INTRODUCTION

Guillain–Barré syndrome (GBS), sometimes referred to as Landry's paralysis, is an acute or subacute, immune-mediated disorder of the peripheral nervous system. It most often presents with progressive weakness of the limbs, abnormal sensations such as tingling, and a marked reduction or absence of tendon reflexes.^{1,2} In many patients, cranial nerves are affected, producing facial or bulbar muscle weakness. The weakness generally follows an ascending pattern, beginning in the legs and spreading

upward over several days to weeks, resulting in flaccid paralysis.

Autonomic involvement is frequently observed, commonly manifesting as unstable blood pressure, postural hypotension, and cardiac rhythm disturbances. Approximately one-third of hospitalized patients require ventilatory support because of respiratory muscle failure or bulbar dysfunction, highlighting the importance of timely recognition and treatment.^{2,3} Evidence suggests that GBS is not a single entity but rather a spectrum of acute

neuropathic conditions, most often triggered by immune-mediated inflammation affecting spinal nerve roots, peripheral nerves, and areas prone to entrapment.²

The global incidence of GBS is estimated at 1–2 cases per 100,000 people annually.⁴ The likelihood increases with age, and men are affected more often than women. Infections preceding the onset of neurological symptoms are a well-established risk factor, most commonly occurring 1–2 weeks prior to disease onset.³ Pathogens linked with GBS include *Campylobacter jejuni*, cytomegalovirus, *Mycoplasma pneumoniae*, Epstein–Barr virus, and influenza virus, among others.^{2,4} Diagnostic confirmations may be achieved with cerebrospinal fluid (CSF) analysis and electrodiagnostic studies, although both may appear normal during the early course of the disease.^{3,4}

Several clinical subtypes of GBS are recognized. These include acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN), and Miller–Fisher syndrome.^{5,6} Among these, AIDP is the most common, accounting for the majority of cases worldwide. Although GBS can develop throughout the year, its seasonal distribution may reflect peaks in triggering infections. In parts of Asia, for example, cases are more frequently reported in the summer months.^{7,8}

Treatment relies on early immunotherapy combined with comprehensive supportive care.⁹ Both therapeutic plasma exchange (TPE) and intravenous immunoglobulin (IV Ig) have been shown to improve outcomes when administered within the first few weeks of symptom onset.^{10,11} Supportive strategies, such as careful respiratory monitoring, prevention of thromboembolic complications, pain control, and early physical rehabilitation, are also crucial to reducing complications and improving recovery.^{12,13} The objective of this study is to evaluate the clinical presentation, diagnostic finding, therapeutic interventions, and outcome in patients diagnosed with GBS in a tertiary care centre.

METHODS

The prospective observational study was carried out at department of medicine RCSM GMC and CPR Hospital, Kolhapur post obtaining institutional ethical committee approval. 20 cases with the diagnosis of GBS admitted to RCSM and CPR Hospital Kolhapur from January 2025 to April 2025 during the GBS outbreak in India. The data related to age, sex, date of admission, antecedent illness, duration of symptoms before admission, muscle power graded by the Medical Research Council (MRC) scale, Hughes' functional scores (F-scores), details of intensive care unit (ICU) complications if any, need for ventilation, details of investigations including cerebrospinal fluid (CSF) and electrodiagnostic analysis, complete blood profile, lipid profile, serum electrolytes, coagulation profile, blood grouping.^{14,15} Critical and supportive care

comprising respiratory care including mechanical ventilation as and when required, cardiac monitoring, DVT prophylaxis, management of infections, nutritional care and physiotherapy were integral part of the treatment. Patients were classified according to MRC manual muscle testing grading system (0-5) and functional grading scales: grade 0-healthy, grade 1-minor symptoms and signs of neuropathy, grade 2-able to walk five min without assistance, grade 3-able to walk five min with assistance, grade 4-confined to bed or chair bound, and grade 5-requiring assisted ventilation.

Statistical analysis

The collected data were compiled and analysed using the statistical package for the social sciences (SPSS) version 25.0. Descriptive statistics were used to summarize the baseline characteristics of the study population. Continuous variables such as age and MRC muscle power scores were expressed as mean±standard deviation (SD) or median with interquartile range (IQR), depending on the distribution of data. Categorical variables such as sex, antecedent illness, presence of ICU complications, and requirement of mechanical ventilation were presented as frequencies and percentages. The Chi-square test or Fisher's exact test (where applicable) was used to examine associations between categorical variables such as sex, antecedent illness, and the need for mechanical ventilation.

RESULTS

In this study, overall 22 patients enrolled, 16 were male. The male to female ratio was 2.5:1. Maximum number of patients (50 %, n=11) were in the age group of 60 years and above. The next common age group was 13-30 years in which 31.8 % (n=7) patients were seen (Table 1). Nearly 72 % patients (n=16) had history of preceding illness. Gastroenteritis was found to be the most common antecedent event preceding GBS in 45% patients (n=10) followed by flu-like illness as evidenced by fever and cough 18.6% patients (n=4). Two patient presented after unusual presentation, one presented after myocardial ischaemia attack and another one after the herpes zoster infection (Table 2).

Table 1: Age and sex distribution of Guillain–Barre syndrome patients.

Age (years)	Male (N)	Female (N)	Total (%)
13-30	5	2	7 (31.8)
31-59	2	2	4 (18.2)
>60	10	1	11 (50)

All patients developed neurological illness within two weeks of the onset of the symptoms. Majority of the patients were admitted to the hospital with paraparesis as a common clinical feature 54.5% (n=12) followed by progressive weakness in all four limbs (quadriplegia) in 45.5% patients (n=10). Dysphagia and respiratory distress were noted in 10 patients (45.4%) each. None of the

patients were found to have bladder and bowel involvement. All patients had areflexia. All patients underwent nerve conduction velocity testing as the diagnostic testing. The majority (n=20, 90.9%) were found to be of AMSAN followed by AIDP variants (n=2, 9.09%). CSF analysis done but there is no significant albuminocytologic dissociation. Although we used IVIg 72.7% (n=16) and plasmapheresis 27.2% (n=6). During the course of admission and treatment 45.5% (n=10) required ventilator support due to respiratory failure, out of which 3 patients still admitted with tracheostomised. Both therapies are known to accelerate time to recovery in patient with GBS with a symptom's duration of under 4 weeks. The mean duration of hospital stay was 2 weeks. On presentation, there was a diffuse weakness of all four extremities. The lower limbs were more severely involved than the upper limbs and distal extremities were affected more than the proximal extremities. Most of the patients were reported to be active on their F-scores before the onset of GBS. The F-score on admission and discharge were 4 and 3, respectively. About 59.09% patient (n=13) improved at the time of discharge on their F-scores with mild disability. Three patient required ventilator support and they had tracheostomy in situ; patient died during hospital stay 27.7% (n=6) (Table 3).

Table 2: Antecedent events observed in patients with GBS.

Illness	No of patients	Percentage
None	6	27
Gastroenteritis	10	45
Flu like illness	4	18
Herpes zoster	1	4.5
Myocardial infraction	1	4.5

Table 3: Clinical symptoms/sign, treatment, variants and complications seen in patients.

Variables	No. of patients	Perce -tage
Clinical symptoms/sign		
Quadripareisis	10	45.4
Paraparesis	12	54.5
Areflexia	22	100
Variants		
AMSAN	20	90
AIDP	2	9.09
Treatment		
IV Ig	16	72.7
Plasmapheresis	6	27.2
Complications		
Respiratory failure	10	45.4
Required ventilator support	10	45.4
Death	6	27.2
Discharge	13	59.09
Still admitted tracheostomy	3	13.6

DISCUSSION

GBS is an uncommon but potentially life-threatening neurological disorder, where early recognition and intervention are crucial. In tertiary care centres, access to specialized investigations and therapies plays an important role in determining patient outcomes. The present study evaluated the clinical features, diagnostic hurdles, treatment approaches, and prognosis of individuals diagnosed with GBS in our institution.

Confirmation of GBS relied on standard diagnostic methods. CSF assessment frequently demonstrated albuminocytologic dissociation, while electrophysiological studies helped differentiate demyelinating from axonal subtypes. In selected cases, magnetic resonance imaging (MRI) assisted in excluding alternative conditions such as myelopathies or acute inflammatory neuropathies. Regarding treatment, intravenous immunoglobulin (IVIg) and therapeutic plasma exchange (TPE) remained the most widely used options, both with established efficacy in modifying disease progression. Our findings suggest that starting these therapies promptly was linked with shorter hospital stays and better functional recovery. Supportive measures, such as ventilatory assistance for respiratory failure and structured physiotherapy programs for rehabilitation, were also critical in overall management.

A male predominance was noted in our study, aligning with previous reviews which have reported higher incidence in men and increased frequency among older adults. Progressive weakness, particularly in the lower limbs, was the most frequent presenting symptom, and gastrointestinal illness or flu-like episodes were common antecedent events. Among disease variants, AMSAN was observed most often, followed by AIDP.

The role of specific therapies in GBS has long been debated. Hughes et al concluded that both IVIg and TPE provide comparable benefit, and other studies support the effectiveness of TPE across several neurological conditions, including GBS.^{2,11} In our series, most patients received IVIg, with favourable outcomes in the majority. However, some developed respiratory compromise requiring mechanical ventilation. Predictors of respiratory failure reported in prior literature include rapidly progressive weakness, bulbar dysfunction, ineffective cough, and declining vital capacity.¹⁶ Admission to an intensive care setting is therefore recommended for patients at high risk of respiratory involvement.²⁵

Beyond immunotherapy, comprehensive care remains indispensable. Hughes et al highlighted the role of rehabilitation as a key element of management.¹³ In our experience, preventive nursing practices such as regular repositioning to avoid pressure sores, along with physiotherapy to maintain muscle tone and enhance recovery, were integral. All patients were referred for physiotherapy at discharge, emphasizing the importance of

early and sustained rehabilitation in improving outcomes.²⁶

Limitations

This study has several limitations. The relatively small sample size may restrict the strength and generalizability of the findings. Additionally, variability in individual treatment decisions among physicians could have influenced outcomes. Finally, as the study was conducted in a single tertiary care hospital, results may not be directly applicable to other settings.

CONCLUSION

GBS seems to affect all age groups with male preponderance. Most common antecedent event and presenting feature were gastroenteritis and paraparesis, respectively. AMSAN was the most common variant. GBS remains a neurologic emergency that requires early recognition and prompt management to improve patient outcomes. Our study highlights the diverse clinical presentations and underscores the importance of supportive care and timely initiation of immunotherapy. While most patients show favourable recovery with appropriate treatment, residual deficits and complications can persist, emphasizing the need for ongoing rehabilitation and follow-up.

Funding: No funding sources

Conflict of interest: None declared

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

REFERENCES

- Ropper AH, Samuels M. Adams and Victor's principles of neurology. New York: McGraw-Hill Medical Publishing Division. 2005;1117-27.
- Gorson KC, Ropper AH. Guillain-Barré syndrome (acute inflammatory demyelinating polyneuropathy) and related disorders. In: Katirji B, Kaminski HJ, Preston DC, editors. Neuromuscular disorders in clinical practice. Boston: Butterworth-Heinemann. 2002;544-66.
- Burns TM. Guillain-Barré syndrome. Semin Neurol. 2008;28:152-67.
- van Doorn PA, Ruts L, Jacobs BC. Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome. Lancet Neurol. 2008;7:939-50.
- Rees JH, Soudain SE, Gregson NA, Hughes RA. *Campylobacter jejuni* infection and Guillain-Barré syndrome. N Engl J Med. 1995;333:1374-9.
- Kalita J, Misra UK, Goyal G, Das M. Guillain-Barré syndrome: subtypes and predictors of outcome from India. J Peripher Nerv Syst. 2014;19:36-43.
- Sivadon-Tardy V, Orlikowski D, Porcher R, Sharshar T, Durand MC, Enouf V, et al. Guillain-Barré syndrome and influenza virus infection. Clin Infect Dis. 2009;48:48-56.
- Larsen JP, Kvåle G, Nyland H. Epidemiology of the Guillain-Barré syndrome in the county of Hordaland, Western Norway. Acta Neurol Scand. 1985;71:43-7.
- Zaheer M, Naeem M, Nasrullah M. Seasonal variation and sex distribution in patients with Guillain-Barré syndrome. Pak J Neurol Sci. 2008;3:6-8.
- Sharma G, Sood S, Sharma S. Seasonal, age and gender variation of Guillain-Barré syndrome in a tertiary referral center in India. Neurosci Med. 2013;4:23.
- Hughes RA, Swan AV, Raphaël JC, Annane D, van Koningsveld R, van Doorn PA. Immunotherapy for Guillain-Barré syndrome: a systematic review. Brain. 2007;130(9):2245-57.
- Raphaël JC, Chevret S, Hughes RA, Annane D. Plasma exchange for Guillain-Barré syndrome. Cochrane Database Syst Rev. 2012;7:CD001798.
- Hughes RA, Wijdicks EF, Benson E, Cornblath DR, Hahn AF, Meythaler JM, et al. Supportive care for patients with Guillain-Barré syndrome. Arch Neurol. 2005;62:1194-8.
- Medical Research Council. Aids to the investigation of the peripheral nervous system. London: HMSO; 1943.
- Hughes RAC, Newsom-Davis JM, Perkin GD, Pierce JM. Controlled trial of prednisolone in acute polyneuropathy. Lancet. 1978;2:750-3.
- Sriganesh K, Netto A, Kulkarni GB, Taly AB, Umamaheswara Rao GS. Seasonal variation in the clinical recovery of patients with Guillain-Barré syndrome requiring mechanical ventilation. Neurol India. 2013;61:349-54.
- McGrogan A, Madle GC, Seaman HE, de Vries CS. The epidemiology of Guillain-Barré syndrome worldwide: a systematic literature review. Neuroepidemiology. 2009;32:150-63.
- van Koningsveld R, Rico R, Gerstenbluth I, Schmitz PI, Ang CW, Merkies IS, et al. Gastroenteritis-associated Guillain-Barré syndrome on the Caribbean Island Curaçao. Neurology. 2001;56:1467-72.
- Nachamkin I, Arzarte Barbosa P, Ung H, Lobato C, Gonzalez Rivera A, Rodriguez P, et al. Patterns of Guillain-Barré syndrome in children: results from a Mexican population. Neurology. 2007;69:1665-71.
- Gupta D, Nair M, Baheti NN, Sarma PS, Kuruvilla A. Electrodiagnostic and clinical aspects of Guillain-Barré syndrome: an analysis of 142 cases. J Clin Neuromuscul Dis. 2008;10:42-51.
- Sharma A, Lal V, Modi M, Vaishnavi C, Prabhakar S. *Campylobacter jejuni* infection in Guillain-Barré syndrome: a prospective case-control study in a tertiary care hospital. Neurol India. 2011;59:717-21.
- 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;59:727-32.
- Islam Z, Jacobs BC, van Belkum A, Mohammad QD, Islam MB, Herbrink P, et al. Axonal variant of Guillain-Barré syndrome associated with

- Campylobacter infection in Bangladesh. *Neurology.* 2010;74:581-7.
24. Borhani Haghighi A, Banihashemi MA, Zamiri N, Sabayan B, Heydari ST, Safari A, et al. Seasonal variation of Guillain-Barré syndrome admission in a large tertiary referral center in Southern Iran: a 10-year analysis. *Acta Neurol Taiwan.* 2012;21:60-3.
25. Orlikowski D, Prigent H, Sharshar T, Lofaso F, Raphael JC. Respiratory dysfunction in Guillain-Barré syndrome. *Neurocrit Care.* 2004;1:415-22.
26. Winer JB, Hughes RAC, Greenwood RJ, Perkin GD, Healy MJR. Prognosis in Guillain-Barré syndrome. *Lancet.* 1985;1:1202-3.
27. Kaya E, Keklik M, Sencan M, Yilmaz M, Keskin A, Kiki I, et al. Therapeutic plasma exchange in patients with neurological diseases: multicenter retrospective analysis. *Transfus Apher Sci.* 2013;48:349-52.

Cite this article as: Uikey DY, Paritekar A. Guillain–Barre syndrome - clinical profile, diagnosis and recovery in a tertiary care setting. *Int J Res Med Sci* 2025;13:4801-5.