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Neurological Betrayal: Case Series on the Diagnosis and Management of Guillain Barré Syndrome

Dr Jyoti Milind Kharche[1], Dr. Shantanu Ashok Shelke[2], Dr. Kaivalya Sudhir Gojamgunde[3], Dr. Mehul Rajesh Thakker[4], Dr. Sahil Sheth[5], Dr. Sara Ajay Patil[6], Dr. Sai Bedmuttha[7], Dr. Saurin Swapnil Shah[8], Dr. Shubham Jain[9], Dr. Khushbu Manish Agarwal[10], Dr. Kulbhushan Vinod Arbat[11], Dr. Rab Syed Anjum Faizy[12]

[1]Professor, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[2]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[3]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[4]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[5]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[6]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[7]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[8]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[9]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[10]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[11]Resident, Department of Medicine, MGM Medical College and Hospital, Aurangabad, India.
[12]Resident, Department of Medicine, MGM Aurangabad, India.

Correspondence:
Dr. Kaivalya Sudhir Gojamgunde

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Abstract

Background: Guillain–Barré syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy and the leading cause of acute flaccid paralysis worldwide. It exhibits marked clinical heterogeneity, ranging from mild limb weakness to severe respiratory failure, often precipitated by infectious, metabolic, or systemic triggers. Early recognition and timely immunomodulatory therapy are critical to improving outcomes. ***Objective:*** To describe the clinical spectrum, diagnostic challenges, management strategies, and outcomes of patients with Guillain–Barré syndrome presenting to a tertiary care center. ***Methods:*** This case series includes six patients diagnosed with GBS, representing diverse clinical presentations and comorbid conditions. Detailed clinical evaluation, cerebrospinal fluid analysis, nerve conduction studies, and multidisciplinary management were undertaken. Treatment strategies and outcomes were analyzed descriptively. ***Case Presentation:*** The cohort included patients aged 29–70 years with varied presentations, including acute inflammatory demyelinating polyneuropathy (AIDP), overlap syndromes, recurrent GBS progressing to chronic inflammatory demyelinating polyneuropathy (CIDP), and GBS associated with pregnancy, thyroid storm, diabetic ketoacidosis, and chronic hepatitis B infection. Respiratory failure requiring mechanical ventilation occurred in multiple cases. Intravenous immunoglobulin (IVIg) was the primary immunotherapy used, with adjunctive ventilatory support, metabolic stabilization, antiviral therapy, and long-term immunosuppression when indicated. Most patients demonstrated favorable neurological recovery with timely intervention, although disease course and recovery varied depending on severity and associated systemic conditions. ***Conclusion:*** This case series highlights the broad clinical spectrum and complexity of Guillain–Barré syndrome, emphasizing the importance of early diagnosis, vigilant respiratory monitoring, prompt immunotherapy, and multidisciplinary care. Recognition of systemic triggers and overlap with chronic immune-mediated neuropathies is essential for optimizing outcomes and guiding long-term management.

Keywords: Guillain–Barré syndrome, Acute inflammatory demyelinating polyneuropathy, Diabetes mellitus, Thyroid storm, Diabetic ketoacidosis, Respiratory failure.

INTRODUCTION

Guillain–Barré syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy characterized by rapidly progressive, symmetrical limb weakness, hyporeflexia or areflexia, and varying degrees of sensory, autonomic, and cranial nerve involvement. Since the fall of poliomyelitis, it is the most frequent cause of acute flaccid paralysis globally.

Neurological impairments usually grow over days to weeks in the monophasic course of the condition, followed by a plateau period and eventual recovery.[1]

According to estimates, there are roughly 10–20 cases of GBS per million people worldwide each year, with a small male predominance and an increasing prevalence as people age.[2] Reported incidence rates in India vary from 1.2 to 2.3 per 100,000 people per year, which may be influenced by genetic, infectious, and regional factors.[2] An antecedent infection is the most prevalent triggering event for GBS. Mycoplasma pneumoniae, cytomegalovirus, Epstein-Barr virus, Campylobacter jejuni, and more recently, SARS-CoV-2 have all been identified as significant precipitating factors.[3] In certain areas, seasonal variation has been noted, providing more evidence for the involvement of viral triggers in the pathophysiology of disease.

Clinically, a wide range of phenotypes are included in GBS. While axonal variations like Acute Motor Axonal Neuropathy (AMAN) and Acute Motor Sensory Axonal Neuropathy (AMSAN) are more commonly observed in Asian cultures, Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is the most common type in Western countries. Miller Fisher syndrome is a separate but related clinical condition that is characterised by ophthalmoplegia, ataxia, and areflexia.[1] Clinical characteristics, cerebrospinal fluid (CSF) study showing albuminocytological separation, and electrophysiological investigations supporting subtype classification and prognostication are all necessary for an accurate diagnosis.[4]

Reducing morbidity and mortality requires early detection and timely immunomodulatory treatment beginning. The cornerstones of treatment continue to be intravenous immunoglobulin (IVIg) and plasma exchange, which are equally effective when given during the first two weeks following the beginning of symptoms.[9] Since up to one-third of patients may need ventilatory support, supportive care—especially careful respiratory monitoring and control of autonomic dysfunction—is critical to patient outcomes.[6]

The purpose of this case series is to emphasise the heterogeneity in presentation and illness course while illuminating the clinical spectrum, diagnostic difficulties, and therapeutic results of patients with Guillain-Barré syndrome treated at our tertiary care facility.

CASE PRESENTATION

Case 1: Acute Inflammatory Demyelinating Polyneuropathy with Respiratory Failure

A 45-year-old male with no significant past medical history presented with rapidly progressive bilateral lower limb weakness over three days, associated with difficulty standing and climbing stairs. Neurological examination revealed symmetric flaccid paralysis of the lower limbs with absent deep tendon reflexes, while cranial nerves were initially spared. Nerve conduction studies demonstrated mixed demyelinating and axonal sensorimotor polyneuropathy, consistent with AIDP. Cerebrospinal fluid (CSF) analysis showed albuminocytologic dissociation with elevated protein (72 mg/dL) and normal cell counts.

As the patient's forced vital capacity decreased and their respiratory distress worsened during their hospital stay, non-invasive ventilation had to be escalated to invasive mechanical ventilation. Intravenous immunoglobulin (IVIg) was started early, coupled with neuro-physiotherapy and extensive respiratory care. Gradual neurological improvement was seen over the next two weeks, enabling effective ventilation weaning. The patient was recommended systematic therapy and follow-up, and upon release, they were able to walk about with little help.

Case 2: Severe GBS with Prolonged Ventilation and Tracheostomy (Overlap Syndrome)

A 70-year-old male presented with acute-onset neck weakness, bilateral ptosis, and rapidly progressive respiratory distress. The initial diagnostic dilemma included GBS relapse versus myasthenia gravis due to prominent bulbar and ocular involvement. Neurological examination revealed generalized areflexia. He developed hypercapnic respiratory failure requiring mechanical ventilation and later tracheostomy due to prolonged ventilatory dependence.

Acetylcholine receptor antibodies were found to be borderline positive, but repeated nerve stimulation was not definitive, and a CT scan of the chest did not detect thymoma. Overall, the clinical picture suggested either developing chronic inflammatory demyelinating polyneuropathy (CIDP) or overlap syndrome. The patient was successfully extubated, decannulated, and had a slow neurological improvement. Mycophenolate mofetil and tapering corticosteroids were used for long-term immunomodulation. His ambulatory discharge highlights the difficulty of distinguishing between acute neuromuscular diseases in older people.

Case 3: GBS in a Patient with Diabetes and Cardiac Disease

A 52-year-old woman with known coronary artery disease, hypertension, and type 2 diabetes mellitus first had low back discomfort six days prior to developing increasing lower limb paralysis. A neurological examination showed that the lower

limbs had symmetric weakness and no reflexes. Acute inflammatory demyelinating polyneuropathy was established by nerve conduction tests.
In addition to routine neuro-physiotherapy, she got IVIg for six days. She was extensively watched for autonomic instability, arrhythmias, and glycaemic variations due to her comorbidities. Throughout the hospital stay, strict metabolic and cardiovascular management was enforced. The patient continued to progress neurologically and did not have respiratory compromise. Her stable condition and notable muscle strength improvement upon discharge underscore the significance of careful supportive treatment for GBS patients with several comorbidities.

Case 4: GBS Associated with Diabetic Ketoacidosis and Chronic Hepatitis B
Diabetic ketoacidosis (DKA) was diagnosed in a 29-year-old male patient with type 2 diabetes mellitus and chronic hepatitis B-related liver damage. He experienced increasing bilateral limb weakness with areflexia after metabolic stabilisation. Sensorimotor polyneuropathy was detected by nerve conduction investigations, and albuminocytologic dissociation (protein 360 mg/dL, 2 cells) was clearly visible in CSF examination. The patient had respiratory failure, necessitating the use of mechanical ventilation. Tenofovir antiviral medication was started in order to avoid viral reactivation during immunotherapy due to the high hepatitis B viral load. IVIg and vigorous physical therapy were used to treat him. Successful extubation was made possible by progressive neurological and respiratory improvement. His ambulatory discharge highlights the possible significance of viral and metabolic stresses as GBS precipitants.

Case 5: GBS in Pregnancy Associated with Thyroid Storm
A 31-year-old primigravida at 28 weeks of gestation presented with rapidly progressive bilateral lower limb weakness and respiratory distress. Neurological examination revealed flaccid paralysis with generalized areflexia. Nerve conduction studies demonstrated sensorimotor polyneuropathy consistent with Guillain–Barré syndrome. She developed acute respiratory failure and required endotracheal intubation and mechanical ventilation.

During hospitalization, she exhibited persistent tachycardia (heart rate >140/min), hyperthermia, and autonomic instability. Thyroid function tests revealed suppressed TSH with markedly elevated free T3 and free T4 levels, consistent with thyroid storm. A diagnosis of GBS complicated by thyroid storm in pregnancy was made.

In addition to intensive endocrine therapy, which included propranolol, antithyroid medications (methimazole/PTU according to institutional protocol), and intravenous corticosteroids, she was treated with intravenous immunoglobulin (IVIg). An emergency lower-segment caesarean section was carried out after intrauterine fetal demise was discovered despite extensive care. The patient's neurological and thyroid conditions gradually improved after surgery. She was successfully weaned off mechanical ventilation and extubated. She was instructed to continue neurological and endocrine follow-up at release because she was hemodynamically stable and had improved motor strength. The intricate relationship between pregnancy, thyroid storm, and Guillain-Barré syndrome is highlighted in this instance, underscoring the importance of early detection and a multidisciplinary therapeutic strategy combining obstetrics, neurology, endocrinology, and critical care specialists.

Case 6: Recurrent GBS Progressing to CIDP / Overlap Syndrome
Recurrent lower limb weakness was reported by a 37-year-old lady with a history of GBS. A neurological examination showed areflexia and symmetric weakness. Nerve conduction investigations revealed mixed demyelinating and axonal sensorimotor neuropathy, while CSF examination revealed increased protein with normal cell counts. The results of a thorough autoimmune and infectious workup were negative. IVIg was used to treat her at first, and she showed some improvement. A diagnosis of developing CIDP or overlap syndrome was taken into consideration in light of the relapsing history and electrophysiological results. For long-term immunosuppression, she was switched to corticosteroids and mycophenolate mofetil. With preparations for nodopathy panel testing and rituximab consideration in the event of any relapses, the patient was discharged stable.

Table 1: Comprehensive Comparative Analysis of all the Cases

Case	Age/Sex	Diagnosis	Comorbidities / Triggers	Treatment / Key Features
1	45/M	AIDP (GBS)	None significant	IVIG, mechanical ventilation, physiotherapy
2	70/M	GBS vs MG overlap	None significant	Ventilation, MMF + steroids
3	29/M	AIDP (GBS)	DKA, chronic HBV	IVIG, Tenofovir, ventilation
4	52/F	AIDP (GBS)	DM, HTN, CAD	IVIG ×6 days, physiotherapy
5	31/F	AIDP (GBS)	Pregnancy, thyroid storm	IVIG, endocrine stabilization, LSCS
6	37/F	Recurrent GBS / CIDP	Previous GBS	IVIG → steroids + MMF

DISCUSSION

The current case series' clinical variability and epidemiological trends closely resemble those seen in national reviews and previous population-based research. The frequency of Guillain-Barré syndrome is believed to be rather similar worldwide, with regional differences caused by environmental variables, genetic predisposition, and viral exposures. As observed in our group, seasonal clustering and post-infectious onset have been repeatedly documented, supporting the immune-mediated pathogenesis of GBS caused by systemic stresses or antecedent infections.[1,2]

The most common electrophysiological subtype in this series was acute inflammatory demyelinating polyneuropathy (AIDP), which is consistent with data from Western nations where demyelinating variations account for the majority of cases.On nerve conduction tests, however, a number of patients showed axonal or mixed demyelinating-axonal characteristics[1]. The correlation between severe illness and subsequent axonal degeneration, which is becoming more well acknowledged in Asian and developing-country cohorts, is reflected in this study as well as geographical diversity.[7] Additionally, delayed presentation and increased disease severity have been associated with mixed electrophysiology, which may have an impact on prognosis.

In GBS, respiratory failure continues to be the most important factor influencing intensive care unit (ICU) admission, morbidity, and death. Several patients in this cohort needed ventilatory assistance, highlighting the significance of early respiratory monitoring using metrics including negative inspiratory force (NIF) and forced vital capacity (FVC). In line with other research highlighting the importance of careful critical care management in enhancing results, prompt escalation to non-invasive or invasive ventilation in conjunction with immunotherapy was linked to a favourable neurological recovery.[8]

The mainstay of GBS treatment is still immunotherapy using intravenous immunoglobulin (IVIg) or plasma exchange (PLEX). Both methods have shown similar effectiveness when used during the first two weeks of symptom onset.[9] IVIg is frequently chosen in clinical practice because it is easier to administer, more tolerable, and appropriate for certain groups, such pregnant women. Certain instances, such as those with IVIg contraindications or insufficient response to initial treatment, may be saved for plasma exchange.[1] Thyroid storm, diabetic ketoacidosis (DKA), and active hepatitis B infection were among the serious systemic conditions that many of the patients in this group had. These circumstances either made the clinical course more difficult or probably served as GBS antecedent triggers. GBS has been linked to viral disorders like hepatitis B and metabolic stress conditions like diabetic ketoacidosis (DKA), which supports the theory that immunological dysregulation in both conditions may cause peripheral nerve damage with thyroid storm.[10]

Pregnancy-related GBS, as seen in one instance, is uncommon but involves increased risks for both the mother and the foetus, especially because of respiratory impairment and autonomic instability. It is crucial to use multidisciplinary care that includes neurology, obstetrics, endocrinology, and critical care. IVIg is the preferred immunotherapy during pregnancy, according to previous systematic reviews, and it has been shown to improve mother neurological outcomes when paired with careful obstetric and intensive care support.[11]

The known overlap between acute and chronic immune-mediated neuropathies is seen in one patient's recurrent GBS and progression to chronic inflammatory demyelinating polyneuropathy (CIDP). Even though recurrence is rare, these individuals need to be closely monitored and may require long-term immunomodulation with corticosteroids, steroid-sparing medications, or biologic treatments like rituximab. In these situations, customised treatment approaches are emphasised in current neurology recommendations.[12] All things considered, the management strategy used in this series—early immunotherapy, careful respiratory monitoring, timely critical care support, and organised rehabilitation—closely complies with current guideline recommendations and produced positive functional outcomes across a wide range of disease severity.[8]

CONCLUSION

From simple AIDP to severe respiratory failure, pregnancy-associated illness, endocrine emergency and recurring immune-mediated neuropathies, this case series illustrates the wide range of clinical manifestations and consequences of Guillain-Barré syndrome. Positive results were mostly dependent on timely diagnosis, early immunotherapy treatment, careful respiratory support, and multidisciplinary management. Optimising patient recovery and lowering morbidity requires an understanding of systemic triggers and the possible requirement for long-term immunomodulation in recurring or overlapping disorders.

REFERENCES

1. Hosseinpour S, Pazouki R, Ashrafi MR, Bemanalizadeh M, Ghahvechi Akbari M, Rezaei S, Parvaneh N, Heidari M, Vafaee-Shahi M, Hosseini F, Bagheri S, Tavasoli AR. Exploring the Phenotypic Profile of Acute Flaccid Paralysis: Insights from a Third-Level Pediatric Emergency Room. Iran J Child Neurol. 2024 Fall;18(4):33-45. doi: 10.22037/ijcn.v18i4.43749. Epub 2024 Sep 29. PMID: 39478947; PMCID: PMC11520268.

2. Lin D, Jin Y, Shao X, Xu Y, Ma G, Jiang Y, Xu Y, Jiang Y, Hu D. Global, regional, and national burden of inflammatory bowel disease, 1990-2021: Insights from the global burden of disease 2021. Int J Colorectal Dis. 2024 Sep 7;39(1):139. doi: 10.1007/s00384-024-04711-x. PMID: 39243331; PMCID: PMC11380638.
3. Finsterer J. Triggers of Guillain-Barré Syndrome: Campylobacter jejuni Predominates. Int J Mol Sci. 2022 Nov 17;23(22):14222. doi: 10.3390/ijms232214222. PMID: 36430700; PMCID: PMC9696744.
4. Dimachkie MM, Barohn RJ. Guillain-Barré syndrome and variants. Neurol Clin. 2013 May;31(2):491-510. doi: 10.1016/j.ncl.2013.01.005. Epub 2013 Feb 19. PMID: 23642721; PMCID: PMC3939842.
5. Abbas A, Rajabally YA. Complications of Immunoglobulin Therapy and Implications for Treatment of Inflammatory Neuropathy: A Review. Curr Drug Saf. 2019;14(1):3-13. doi: 10.2174/1574886313666181017121139. PMID: 30332974.
6. Strzelec M, Detka J, Mieszczak P, Sobocińska MK, Majka M. Immunomodulation-a general review of the current state-of-the-art and new therapeutic strategies for targeting the immune system. Front Immunol. 2023 Mar 9;14:1127704. doi: 10.3389/fimmu.2023.1127704. PMID: 36969193; PMCID: PMC10033545.
7. Salvadores N, Sanhueza M, Manque P, Court FA. Axonal Degeneration during Aging and Its Functional Role in Neurodegenerative Disorders. Front Neurosci. 2017 Sep 4;11:451. doi: 10.3389/fnins.2017.00451. PMID: 28928628; PMCID: PMC5591337.
8. Pearson SD, Koyner JL, Patel BK. Management of Respiratory Failure: Ventilator Management 101 and Noninvasive Ventilation. Clin J Am Soc Nephrol. 2022 Apr;17(4):572-580. doi: 10.2215/CJN.13091021. Epub 2022 Mar 10. PMID: 35273008; PMCID: PMC8993478.
9. Wiegers EJA, Jacobs BC. New insights in the immune treatment of Guillain-Barré syndrome. Curr Opin Neurol. 2025 Oct 1;38(5):471-477. doi: 10.1097/WCO.0000000000001408. Epub 2025 Jul 21. PMID: 40748027; PMCID: PMC12419018.
10. Rathish D, Karalliyadda S. Concurrent presentation of thyroid storm and diabetic ketoacidosis: a systematic review of previously reported cases. BMC Endocr Disord. 2019 May 17;19(1):49. doi: 10.1186/s12902-019-0374-3. PMID: 31101104; PMCID: PMC6525384.
11. Sharma SR, Sharma N, Masaraf H, Singh SA. Guillain-Barré syndrome complicating pregnancy and correlation with maternal and fetal outcome in North Eastern India: A retrospective study. Ann Indian Acad Neurol. 2015 Apr-Jun;18(2):215-8. doi: 10.4103/0972-2327.150608. PMID: 26019422; PMCID: PMC4445200.
12. Inan B, Bekircan-Kurt CE, Demirci M, Erdem-Ozdamar S, Tan E. Differentiating recurrent Guillain-Barre syndrome and acute-onset chronic inflammatory polyneuropathy: literature review. Acta Neurol Belg. 2024 Oct;124(5):1467-1475. doi: 10.1007/s13760-024-02557-2. Epub 2024 Apr 25. PMID: 38664341.