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# GUILLAIN-BARRE SYNDROME: A REVIEW ARTICLE

Microbiology

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## ABSTRACT

Guillain-Barré syndrome is a sporadic, auto-immune disease of the peripheral nerves triggered by infections. It can be a life threatening condition but many aspects of its nature continue to elude our comprehension therefore GBS remains a hard nut to crack. This review summarises current knowledge and best available evidence to clinicians for overhauling the disease progression and treatment.

## KEYWORDS

Guillain-Barré Syndrome; Auto-immune Disease and Peripheral Nerve Damage

## INTRODUCTION

Guillain-Barré syndrome (GBS) is a neurological disorder often linked to viral or bacterial infection. The term GBS signifies a spectrum of acute idiopathic, usually monophasic peripheral neuropathies. GBS is usually preceded by infection or other immune stimulation that induces an aberrant autoimmune response targeting peripheral nerves and their spinal roots. The syndrome can affect the nerves that control muscle movement as well as those that transmit pain, temperature and touch sensations. This can result in muscle weakness, loss of sensation in the legs and/or arms, and problems with swallowing or breathing. Demyelinating and axonal forms of the syndrome occur in varying proportions across different geographical regions, and clinical variants, such as Miller Fisher syndrome, are readily definable.[1] It is a rare condition, while it is more common in adults and in males but people of all ages can be affected.

## EPIDEMIOLOGY

GBS is a sporadically occurring disease. GBS incidence varies worldwide, ranging from 0·38 to 2·53 per 100,000, with most studies reporting 1·1 to 1·8 per 100,000. In the post-polio era, GBS was most common cause of acute flaccid neuromuscular paralysis. The mortality rates range from 3−7%. In western populations, the median incidence rate of GBS has been reported at 1·10 per 100,000 person-years in America, 1·6 in Canada, 1·33 in the UK, and 1·59 in Denmark [2].

Estimates of yearly incidence (per 100,000 people) are lowest in Japan (0.44) and highest in Chile (2.12) and Bangladesh (3.25), likely due to differences in exposure to infectious organisms. The spikes of GBS have been reported following infectious outbreaks, most notably in relation to *Campylobacter jejuni* and Zika virus [3].

According to a WHO report (1993), it was estimated that 138 cases of GBS were seen annually in seven major teaching hospitals, with approximately 75% of cases occurring in adults. While studies across the world have shown numbers to rise in seasons of weather change.

In India, the first case of GBS in Mumbai was reported on February 7, 2025. 160 cases of GBS were reported in Pune since January. There have been five deaths. Currently, 48 patients are in intensive care, 21 on ventilator, and 38 have been discharged, according to official figures. Till February, 203 suspected cases are reported out of which 8 are deceased.

According to latest news and reports other states like Assam, West Bengal, Telangana and Maharashtra have reported cases of GBS, with Maharashtra having the highest toll of suspected cases i.e, 170.

## ETIOLOGY

The exact cause for its occurrence is still not known yet. However, it commonly starts a few days or weeks following a respiratory or gastrointestinal bacterial or viral infection.

GBS and its variants are considered post-infectious, immune-mediated neuropathies. Evidence from animal models suggests a key role of molecular mimicry. In *Campylobacter jejuni* gastrointestinal infections, a lipooligosaccharide present in the outer membrane of the bacteria is similar to gangliosides which are components of the peripheral nerves. Therefore, an immune response triggered to fight infection can lead to a cross-reaction on host nerves. Additionally, the complement cascade is activated and plays a key role in the pathogenesis. Other frequent infectious causes include Cytomegalovirus, Epstein-Barr virus and Mycoplasma, Pneumoniae [2].

It may also transpire after immunization with certain vaccines like vaccine for the swine flu, the recombinant zoster vaccine, and the adenovirus vector SARS CoV 2 vaccines.

## SIGNS AND SYMPTOMS (5)

- The most common initial symptom of GBS is acroparesthesia with little objective sensory loss. Severe radicular back pain and neuropathic pain affects most of the cases. Within a few days, weakness ensues a symmetric “ascending pattern”. Most patients present initially with leg and arm weakness (32%) or selective proximal and distal leg weakness (56%) often spreading to the arm while some have onset of weakness in the arms (12%).
- GBS presents with acute, rapidly progressive flaccid paralysis of arms and legs, and decreased or absent deep tendon reflexes.
- The respiratory muscles are commonly affected and 25% of patients require artificial ventilation. In up to two thirds of patients, autonomic dysfunction occurs and manifests as cardiac arrhythmia with asystole, arterial hypertension or hypotension.
- Trouble in chewing, swallowing, or speaking.
- Difficulty with vision and eye muscles.
- Digestion and/or bladder control problems.

## VARIANTS OF GBS (6)

### Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)

The most common type of GBS in North America and Europe. In this type, the immune system damages the myelin sheath. Muscle weakness usually starts in the lower body and spreads upwards.

### Miller Fisher syndrome (MFS)

A rare variant of GBS that affects the head and face. Symptoms include paralysis of the eye muscles, difficulty swallowing and speaking, and loss of tendon reflexes.

### Acute motor axonal neuropathy (AMAN)

An axonal disorder where the immune system may damage the axons.

### Acute motor-sensory axonal neuropathy (AMSAN)

An axonal disorder where the immune system may damage the axons. Symptoms tend to be more severe and prolonged than in other types.

In Asian countries, **AMAN** is the most common variant.

## DIAGNOSIS

The diagnostic criteria for GBS were devised in 1978 by the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS, now NINDS). Following are the criteria:

- Progressive weakness: Weakness that worsens over time and affects more than one limb.
- Areflexia: Loss of tendon reflexes, usually in the distal limbs and symmetrically.
- Symmetrical symptoms: Symptoms that affect both sides of the body.
- Electrodiagnostic features: Motor or sensorimotor neuropathy on nerve conduction studies.
- Cerebrospinal fluid (CSF) changes: Increased protein levels in the CSF.

### Diagnostic Test

NERVE CONDUCTION VELOCITY TEST – measures the nerve's ability to send a signal. In GBS, the signals travelling along the damaged nerves are slowed.

IMAGING – In some cases, MRI of spinal cord or the brain is done to find any other potential cause of muscle weakness.

### Differential Diagnosis

- Poliomyelitis
- Myasthenia gravis
- Viral myositis
- Chronic inflammatory demyelinating polyneuropathy
- Sarcoidosis

## TREATMENT

Treatment of GBS relies on excellent supportive care to prevent death from respiratory failure and cardiac arrhythmia and to avoid complications such as thromboembolism and infections.

In addition, specific "causal" immunomodulatory treatment consisting of plasma exchange or intravenous immunoglobulin has been shown to be beneficial in large randomized trials and these treatments are considered equal in terms of efficacy.

Intravenous immunoglobulins is given 2 grams/kilogram divided over 5 days.

Plasma exchange is thought to act by removing pathogenic antibodies, humoral mediators, and complement proteins involved in the pathogenesis of GBS.

The effect is present if either treatment is given within 4 weeks, but the stronger effect may be present if treatment is administered within 2 weeks

Corticosteroids are not recommended in GBS. Intravenous methylprednisolone does not improve neurological outcome [8].

New strategies in GBS treatment [9]

### Therapies For Antibodies

Neonatal Fc receptor (FcRn) can adjust the concentration of endogenous IgG. Inhibiting FcRn can reduce the level of IgG in the body, and it has been proven effective in animal experiments. The use of FcRn inhibitors significantly reduces the antibody levels in mice, reducing nerve damage and clinical symptoms.

### Therapies For The Complement Pathway

Application of eculizumab in GBS patients.
ANX005 is a humanized immunoglobulin G4 (IgG4) recombinant antibody against C1q that blocks the initiation of the classical complement cascade.

### Therapies Inhibiting Inflammatory Cells And Inflammatory Factors

Includes administration of dimethyl fumarate (DMF), decitabine (DAC), 2-deoxy-D glucose (2-DG), fingolimod.

## PREVENTION STRATEGIES

- **Practising good hygiene:** Wash hands frequently, especially before eating.
- **Cooking food properly:** Avoid undercooked poultry to prevent bacterial infections.
- **Getting vaccinated:** Stay up-to-date on vaccines to protect against viruses that may trigger GBS.

## CONCLUSION

GBS is a postinfectious, immune- mediated peripheral neuropathy. As our knowledge continues to expand, its pathophysiological mechanisms remain partially understood and many questions are yet to be answered. The focus of care should remain early diagnosis and treatment, to prevent severe axonal damage and minimise long- term disability. Meanwhile, novel therapies and neuropathy fluid biomarkers are under ongoing evaluation and may improve clinical management in the foreseeable future.

## REFERENCES

1. Willison H, Jacobs B, Van Doorn P, Guillain-Barré syndrome, The Lancet 2016;388(10045):717-27.
2. Lehmann HC, Hughes RA, Kieseier BC, Hartung HP. Recent developments and future directions in Guillain-Barré syndrome. J Peripher Nerv Syst. 2012 Dec;17(3):57-70.
3. Bellanti R, Rinaldi S. Guillain-Barré syndrome: a comprehensive review. Eur J Neurol. 2024;31(8):56-9.
4. Hughes RA, Hadden RD, Gregson NA, Smith KJ. Pathogenesis of Guillain- Barré syndrome. J Neuroimmunol. 1999 Dec;100(1-2):74-97.
5. Dimachkie MM, Barohn RJ. Guillain-Barré syndrome and variants. Neurol Clin. 2013 May;31(2):491-510.
6. Sriwastava S, Kataria S, Tandon M, Patel J, Patel R, Jowkar A, Daimee M, Bernitsas E, Jaiswal P, Lisak RP. Guillain Barré Syndrome and its variants as a manifestation of COVID-19: A systematic review of case reports and case series. J Neurol Sci. 202115;(1):420-5.
7. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol. 1990;27 Suppl: S21-4.
8. Nguyen TP, Taylor RS. Guillain-Barre Syndrome. [Updated 2023 Feb 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK532254/
9. Yao J, Zhou R, Liu Y, Lu Z. Progress in Guillain-Barré syndrome immunotherapy-A narrative review of new strategies in recent years. Hum Vaccin Immunother. 2023;19(2):221-6.