ISSN: 2278-2648

*International Journal of Research in Pharmacology & Pharmacotherapeutics (IJRPP)*

*IJRPP |Vol.14 | Issue 2 | Apr - Jun -2025*

**www.ijrpp.com**

DOI : https://doi.org/10.61096/ijrpp.v14.iss2.2025.511-517

**Review**

# A Comprehensive Review on Guillain-barre Syndrome: Pathophysiology, Diagnosis and Management

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Published on: 25 Jun 2025

Published by:
DrSriram Publications



## Abstract

Guillain-Barré syndrome (GBS) is a very rare immune mediated disorder which is associated with demyelination of peripheral nervous system and progressive muscle weakness that occurs mostly in previously healthy individuals. Guillain-Barré Syndrome is a life-threatening, demyelinating, autoimmune condition in which the body's immune system attacks the myelin of the peripheral nervous system. Guillain-Barré Syndrome is characterized by ascending motor weakness and acute fl accid paralysis. Demyelination results in nerve infl ammation, numbness, tingling, muscle weakness, structural damage to the myelin sheath, and possible respiratory system complications. Treatment is largely restricted to general supportive measures, Intravenous Immunoglobulin (IVIG) and Plasma Exchange (PLEX), with no current role for oral or intravenous corticosteroids in clinical practice. Several validated prognostic-scoring systems, which can predict the probability of long-term residual disability, may assist in targeting intensive therapies to high-risk patient groups. The aim of this article is to provide a practical overview of GBS, with particular emphasis on the clinical presentation, investigation and management of this important spectrum of neurological condition.

**Keywords:** Guillian- Barre Syndrome, Plasma Exchange, Corticosteroids, Intravenous Immunoglobulin (IVIG).

## INTRODUCTION

Guillain-Barre syndrome (GBS) is an autoimmune fulminant polyradiculoneuropathy that manifests as a severe fulminant polyradiculoneuropathy. Guillain- Barre Syndrome is the most prevalent cause of acute or subacute generalized paralysis, and it used to be second only to polio in terms of prevalence. Its features are ascending motor weakness, sensory and autonomic dysfunction frequently followed by prodromal illness (usually a respiratory or gastrointestinal infection. Guaillain–Barre syndrome is a rare but serious disorder in which body's immune system attacks healthy nerve cells in the peripheral nervous system Diagnosis of GBS is primarily based

on clinical history and findings. Approximately one third of the patients requires intensive care unit (ICU), mainly because of respiratory problems. Patients can be treated with symptomatic medicines. Intravenous immunoglobulins (IVIG) and plasma pheresis, these two methods are prove to be effective in the treatment. Some additional therapies like speech therapy and physical therapy hasten the recovery.

**TYPES:**

The symptoms of GBS can vary based on the type. Guillian- Barre syndrome has several forms.
The main types are:
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Is the gram-negative diarrhea causing bacteria, Campy-lobacterjejuni.
Autoimmunity which has been a mainstay in the pathogenesis of GBS involves nit various mecha
**Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP),** the most common form in North America and Europe.The most common sign of AIDP is muscle weakness that starts in the lower part of the body and spreads upward.
**Miller Fisher Syndrome (MFS),** in which paralysis start in the eyes. MFS also is associated with an unsteady walk. MFS is less common in the U.S. but more common in Asia.
**Acute Motor Axonal Neuropathy (AMAN) and Acute Motor Sensory Axonal Neuropathy (AMSAN)** are less common in the U.S. but AMAN and AMSAN are more frequent in China, Japan, and Mexico.
GBScandamagethenervescontrollingmovements,pain,temperature,
andtouchsensations.Incriticalcases,GBSmayleadtorespiratoryfailureandcanalsobemortal.
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**Acute Motor Axonal Neuropathy (AMAN) and Acute Motor Sensory Axonal Neuropathy (AMSAN)** are less common in the U.S. but AMAN and AMSAN are more frequent in China, Japan, and Mexico. GBS can damage the nerves controlling movements, pain, temperature, and touch sensations. In critical cases, GBS may lead to respiratory failure and can also be mortal. The progression of GBS can be described in three phases:
1. Initial phase: evolution of symptoms lasting day stoup to four weeks
2. Plateau phase: lasting weeks to months
3. Recovery phase: remyelination, lasting weeks to months. Criticalpatientscantakeaminimum of 2 years or more. Full recovery is not achieved in some cases.
The exact cause is unknown but frequently is associated with a respiratory or gastrointestinal infection.
The fact that the disease results in disability inspite of all therapeutic measures is the reason to explore the role of rehabilitation through physiotherapy in the management of GBS. If it is damaged in the peripheral nervous system, i.e., the nerves of the body, Guillain-Barre disease occurs. As the disease progresses, weakness becomes paralysis. Specific management of GBS is that of immunomodulation - plasmapheresis, intravenous immunoglobulin (IVIG) and/or steroids.

**PATHOPHYSIOLOGY OF GBS**

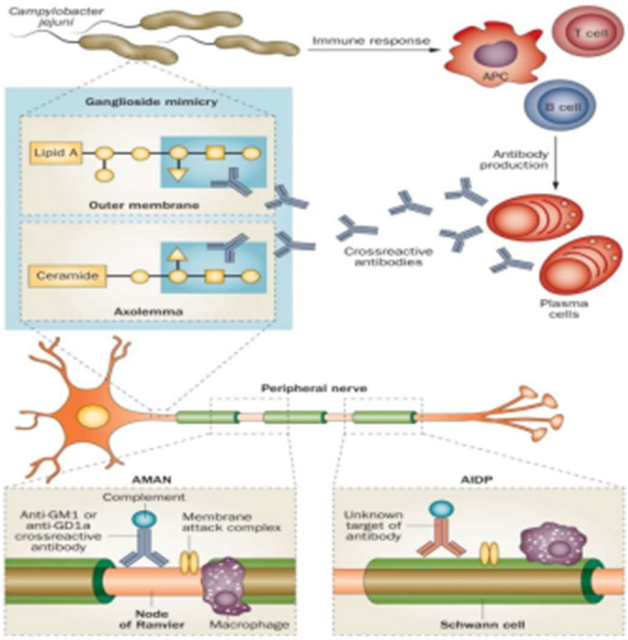
**1.Immune Trigger:** GBS is thought to be triggered by a preceding infection (bacterial or viral) or, less commonly, by surgery, vaccination, or other factors.

**2. Autoimmune Response:** The body's immune system, which normally protects against foreign invaders, begins to attack its own nerve cells, specifically the myelin sheath, which insulates nerve fibers and speeds up nerve impulse transmission.

**3. Inflammation and Demyelination:** The immune response causes inflammation, and the myelin sheath is damaged or destroyed in areas throughout the peripheral nervous system.

**4. Nerve Conduction Blockage:** Demyelination impairs nerve conduction, and in some cases, the axons themselves may be damaged, further slowing or blocking nerve signals.

**5. Clinical Presentation:** The resulting muscle weakness, numbness, and paralysis typically begin in the legs and can spread upwards, affecting the arms, trunk, and even the facial muscles and breathing muscles.




**6. Recovery:** In many cases, nerve damage is reversible, and the body can regenerate the myelin sheath (re-myelination) over time. However, in severe cases, the damage to the axons may be more permanent, leading to longer-lasting or incomplete recovery.

**RISK FACTORS**

While studies are ongoing, a myriad of genes, gene networks and canonical pathways, including transcription factors and inflammatory cytokines, have been revealed in Guillain-Barré Syndrome pathogenesis. A study by Chang et al. found 256 genes and 18 gene networks significantly associated with Guillain-Barré Syndrome including four significantly upregulated functional genes, FOS, PTGS2, HMGB2 and MMP9, involved in inflammatory response, infectious and respiratory diseases. Associated gene networks included hub genes MMP9, PTGS2 and CREB1. The gonadotrophin-releasing hormone pathway, corticotrophin-releasing hormone pathway and ERK/MAPK were the most significant canonical pathways involved in Guillain-Barré Syndrome. Molecular and cellular functions associated with Guillain-Barré Syndrome included cellular development, movement and cell death. Hematological system development and function, immune cell tracking and organismal survival were the most significant functions involved in Guillain-Barré Syndrome physiological development. A DNA analysis from 2002 revealed a deletion at chromosomal locus 17p12, typical of Hereditary Neuropathy with Liability to Pressure Palsies (HNPP). The exact cause of Guillain-Barre syndrome isn't known. It usually appears days or weeks after a respiratory or digestive tract infection. Rarely, recent surgery or vaccination can trigger Guillain-Barre syndrome. In Guillain-Barre syndrome, your immune system — which usually attacks only invading organisms — begins attacking the nerves. In AIDP, the nerves' protective covering, known as the myelin sheath, is damaged. The damage prevents nerves from transmitting signals to your brain, causing weakness, numbness or paralysis.

**Guillain-Barre syndrome may be trigerred by:**

- Most commonly, an infection with campylobacter, a type of bacteria often found in undercooked poultry.
- Influenza virus
- Cytomegalo virus
- Epstein-Barr virus
- Zika virus
- Hepatitis A, B, C and E
- HIV, the virus that causes AIDS.
- Mycoplasma pneumonia

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**COMPLICATIONS**

Guillain-Barre syndrome affects your nerves. Because nerves control your movements and body functions, people with Guillain-Barre syndrome may experience:

- **Trouble breathing.** Weakness or paralysis can spread to the muscles that control your breathing. This can potentially be fatal. Up to 22% of people with Guillain-Barre syndrome need temporary help from a machine to breathe within the first week when they're hospitalized for treatment.
- **Residual numbness or other sensations.** Most people with Guillain-Barre syndrome recover completely or have only minor, residual weakness, numbness or tingling.
- **Heart and blood pressure problems.** Blood pressure fluctuations and irregular heart rhythms are common side effects of Guillain-Barre syndrome.
- **Pain.** One-third of people with Guillain-Barre syndrome experience nerve pain, which may be eased with medicine.
- **Trouble with bowel and bladder function.** Sluggish bowel function and urine retention may result from Guillain-Barre syndrome.
- **Blood clots.** People who are not mobile due to Guillain-Barre syndrome are at risk of developing blood clots. Until you're able to walk independently, you may need to take blood thinners and wear support stockings to improve blood flow.
- **Pressure sores.** You may be at risk of developing bedsores, also known as pressure sores, if you're not able to move. Changing your position often may help avoid this problem.
- **Relapse.** A small percentage of people with Guillain-Barre syndrome have a relapse. A relapse can cause muscle weakness even years after symptoms ended.

When early symptoms are worse, the risk of serious long-term complications goes up. Rarely, death may occur from complications such as respiratory distress syndrome and heart attacks.

**How to Prevent GBS?**

Since GBS is mainly a sequel to infections in most persons, reducing infection can be protective, including.
Washing your hands frequently to prevent bacterial and viral infections
Cooking the food, especially poultry, thoroughly to avoid Campylobacter infections.
Get vaccinated against influenza, COVID-19, and other preventable diseases.
Drink safe, clean water to avoid contaminated sources.
Practice mosquito protection in areas where Zika virus is common.
Boost your immune system with a healthy diet, exercise, and proper hygiene.

**INFECTIONS**

- **Bacterial:** Campylobacter jejuni is a frequent trigger, often associated with food poisoning.
- **Viral:** Influenza, cytomegalovirus (CMV), Epstein-Barr virus, and Zika virus have all been linked to GBS.
- **Other:** Mycoplasma pneumoniae, hepatitis viruses, and even COVID-19 have also been associated

Other Potential Triggers:

- **Vaccines:** While rare, certain vaccines, like the flu vaccine, have been associated with a slight increase in GBS risk.
- **Surgery or Trauma:** In some cases, GBS can develop after surgery or a traumatic injury.
- **Environmental Factors:** Environmental factors like pollution and climate change may also play a role in immune system dysregulation, potentially contributing to GBS.

- **Genetic Susceptibility:** Some studies suggest that certain genetic factors might make individuals more susceptible to GBS.

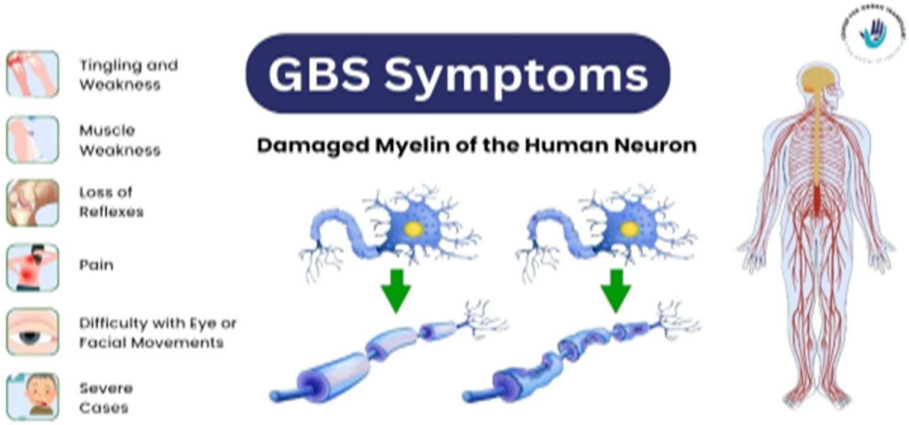
**SYMPTOMS**

Guillain-Barre syndrome often begins with tingling and weakness starting in the feet and legs and spreading to the upper body and arms. Some people notice the first symptomsin the arms or face. As Guillain-Barre syndrome progresses, muscle weakness can turn into paralysis.

Symptoms of Guillain-Barre syndrome may include:

- A pins and needles feeling in the fingers, toes, ankles or wrists.
- Weakness in the legs that spreads to the upper body.
- Unsteady walk or not being able to walk or climb stairs.
- Trouble with facial movements, including speaking, chewing or swallowing.
- Double vision or inability to move the eyes.
- Severe pain that may feel achy, shooting or cramp like and may be worse at night.
- Trouble with bladder control or bowel function.
- Rapid heart rate.
- Low or high blood pressure.
- Trouble breathing.

People with Guillain-Barre syndrome usually experience their most significant weakness within two weeks after symptoms begin. Since GBS is mainly a sequel to infections in most persons, reducing infection can be protective, including




**PREVENTION AND MANAGEMENT**

**Management and Treatment of Guillain-Barré Syndrome**

There's no specific cure for GBS, but treatment focuses on managing symptoms and speeding recovery. The primary treatments include:

- **Plasma exchange**: This medical procedure removes harmful antibodies from the blood.
- **Intravenous immunoglobulin (IVIG)**: This involves administering healthy antibodies to help block the damaging antibodies attacking the nerves.
- **Supportive care**: This includes monitoring vital signs, managing pain, and preventing complications like blood clots and pneumonia.

Recovery from GBS can be a long process, and physical therapy is essential to regain strength and mobility.

**Can I Prevent Guillain Barre Syndrome?**

Unfortunately, there's no guaranteed way to prevent Guillain-Barré Syndrome (GBS). While the exact cause remains unknown, it's believed to be triggered by an immune response following an infection. However, certain steps can help bolster your overall health and potentially reduce the risk of infections that might precede GBS:

1. **Maintain good hygiene:** Regular handwashing, especially during flu season, can help prevent infections.
2. **Healthy lifestyle**: Eating a balanced diet, getting enough sleep, and regular exercise can strengthen your immune system.
3. **Stay updated on vaccinations**: Ensure you're up-to-date on recommended vaccinations, including flu shots.

4. **Prompt treatment of infections**: If you develop an infection, seek medical attention promptly to prevent complications.

These measures are general health recommendations and do not guarantee the prevention of GBS. If you experience symptoms of GBS, seek immediate medical attention. Early diagnosis and treatment of Guillain Barré Syndrome is crucial for recovery.

**INVESTIGATIONS**

**Serum biochemistry**

Urea and electrolytes are usually normal but may have evidence of the syndrome of inappropriate ADH secretion (SIADH) or renal dysfunction. ALT and gamma GT may be raised in 33% of patients. Creatine kinase may be raised.

**Inflammatory markers:**

Erythrocyte sedimentation rate is usually raised and C- reactive protein is sometimes elevated.

**Anti-ganglioside antibodies:** Anti-GM1 is positive in 25% of patients and is associated with a worse outcome. Anti-GD1a is associated with AMAN subtype of GBS. Anti-GQ1b is associated with Miller-Fisher syndrome.

**Infection screen:** Serology tests for Campylobacter jejuni, Cytomegalovirus, Epstein-Barr virus, Herpes simplex virus, Mycoplasma pneumonia, HIV antibodies should be considered. Stool cultures looking for evidence of gastrointestinal infections particularly Campylobacter jejuni.

**Radiological:** A CT brain is indicated to exclude other causes of symptoms and evidence of raised intracranial pressure prior to performing a lumbar puncture. An MRI of the spine may show selective anterior spinal nerve root enhancement with gadolinium and will exclude cervical nerve impingment.

**Lumbar puncture:** Increased protein levels and ell levels in CSF are indicative of GBS.

**Nerve conduction studies:** Findings depend on subtype of GBS. The majority show demyelinating pattern while some patients may show evidence of axonal loss with little or no demyelination.

**Respiratory function test:** These may show reduced vital capacity, maximal inspiratory and expiratory pressures. Arterial blood gases may indicate progressive respiratory failure.

**Etiologies and development of GBS**

GBS is a syndrome that can arise from a range of circumstances, including numerous infectious diseases and vaccinations, among others. The exact cause and progression of GBS have not yet been conclusively established. This complicates the process of determining the exact mechanisms or pathways of development. Nevertheless, certain likely reasons and mechanisms have been identified and will be further elucidated in this discussion.

The pathogen most frequently responsible for the initial infection that leads to GBS is Campylobacter jejuni (C. jejuni).C. jejuni is most commonly spread to people through their diet, specifically by the ingestion of raw or undercooked poultry meat and fish, unpasteurized milk, or water contaminated with the bacteria.The elevated incidence of C. jejuni infection in various geographical areas can potentially be attributed to factors such as the quality of their sanitation systems, environmental conditions, and host-related variables, which may encompass dietary practices. A 45-year-old man who acquired GBS with irreparable neurological impairment 2 weeks after contracting C. jejuni-associated gastroenteritis was the first to demonstrate the correlation between C. jejuni infection and the onset of GBS in 1982.

As previously stated, GBS is an immune-mediated, post-infectious disorder; it is not known whether a specific disease-causing agent is responsible for GBS; therefore, the condition is referred to as a syndrome rather than a disease. Immune systems that are cellular and humoral are likely involved in the development of GBS. The majority of patients claim to have had an infectious disease in the weeks before developing GBS. Numerous infectious agents have been identified, and it is believed that many of these agents cause the body to produce antibodies against particular gangliosides and glycolipids found in the peripheral nerve system myelin, including GM1 and GD1b. Molecular mimicry is the most widely accepted theory for how autoimmune diseases arise. Antibodies are generated in response to C. jejuni infection, triggering the complement system and ultimately resulting in phagocytosis of the bacteria. However, in very uncommon events, antibodies generated against particular C. jejuni antigens will also bind to gangliosides of nervous tissue, leading to complement activation and destruction by phagocytes. Damage to the peripheral nervous system causes demyelination and axonal damage.

Antibodies targeting ganglioside GM1 have been identified in 20%–71% of patients with C. jejuni-associated GBS, a significantly higher occurrence compared to other GBS cases. The probable cause of this discovery is the occurrence of molecular mimicry between ganglioside GM1 and the lipopolysaccharide of C. jejuni. Another instance is the Miller-Fischer syndrome variant's target, the GQ1b ganglioside. It is believed that the presence of particular antigens in C. jejuni's capsule that are related to nerves is what gives the organism its pathogenicity. Antibodies produced by immune reactions against the capsular elements interact with myelin to trigger demyelination. The peripheral nervous system appears to be immunologically damaged by ganglioside GM1, which appears to cross-react with C. jejuni lipopolysaccharide antigens. The final outcome of autoimmune

attacks is muscle paralysis along with possible sensory or autonomic abnormalities when the immune system incorrectly targets the peripheral nervous system**.** Autoimmune attacks can also produce myelin inflammation and conduction blockage.

## CONCLUSION

Gullain-Barre syndrome is a postinfectious, immune- mediated peripheral neuropathy. Guillain Barre-Syndrome is a neurological disorder. The peripheral region is disrupted in this disorder. The lower extremities grow weak as a result of this condition. The patient cannot walk, stand, or run because of a medical condition. GBS remains a significant worldwide cause of rapidly progressive muscular paralysis. Although it is predominantly a clinical diagnosis, neurophysiology, CSF analysis and neuroimaging are all helpful in excluding potential mimics (and corroborating the diagnosis), which may otherwise lead to diagnostic conundrums and therapeutic dilemmas. The method of electrodiagnosis helps in the diagnosis. Definitely, immunotherapy is proven to make a huge difference in the recovery of GBS patients and both plasma exchange (PE) and intravenous immunoglobulin (IVIg) are effective equally. Because it has a fewer side effect profile and easier to be administered, IVIg may be preferred. A severe axonal injury early prevention in the disease course is an important major focus, for the reason that it is an important limiting factor for achieving proper, longterm outcomes.

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