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Review Article

Overview on Guillain-Barre's Syndrome (Guillain GBS)

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Abstract

Guillain Barre's Syndrome (GBS) is a rare neurological disorder and is the most common and most severe acute paralytic neuropathy, with about 100,000 cases every year. Pathogen *Campylobacter jejuni* (*C. jejuni*) is the most frequently responsible for the initial infection that leads to GBS. Abnormal heart rate or blood pressure, weakness, sensation changes, coordination problems are some of the symptoms that are seen in GBS. A knowledge of variants of GBS is important in differentiating GBS from other mimics. There are three classic variants of GBS - clinical, electrophysiological and sensory variants. GBS is an immune-mediated attack against various parts of peripheral nerve - myelin or axon as a result of molecular mimicry. Disease activity and treatment response are currently monitored through interval neurological examination and outcome measures. Novel treatment strategies are in development depending on the pathophysiology of GBS variants. In this article we review the number of cases, history, etiology, symptoms, classification, pathophysiology, diagnosis and treatments of GBS.

Keywords: *Guillain Barre's Syndrome (GBS); Paralytic Neuropathy; Immunity; Novel Treatment; Neuropathy*

Introduction

Guillain-Barré syndrome (GBS) is a rare neurological disorder in which a person's immune system mistakenly attacks part of their peripheral nervous system-the network of nerves that carries signals from the brain and spinal cord to the rest of the body. Guillain-Barré syndrome (GBS) is the most common cause of acute neuromuscular paralysis worldwide [1]. GBS is a postinfectious, monophasic, immune-mediated polyradiculoneuropathy, and its diagnosis is largely based on clinical patterns with or without the support of laboratory findings and electrophysiology. Under the umbrella term of Guillain-Barré syndrome are several recognisable variants with distinct clinical and pathological features. The severe, generalised manifestation of Guillain-Barré syndrome with respiratory failure affects 20-30% of cases. The syndrome is named after the French physician Georges Guillian and Jean Alexandre Barré who described it in 1916. It has been more than 100 years when Guillain-Barré and Strohl, reported two cases of acute flaccid paralysis with albumino-cytological dissociation that has come to be known as Guillain-Barré syndrome [2]. GBS begins suddenly and can increase in intensity over a period of hours, days, or weeks until certain muscles cannot be used at all. Some cases of GBS are very mild and only marked by brief weakness. Others cause nearly devastating paralysis, leaving the person unable to breathe on their own. In these cases, the disorder is life-threatening-potentially interfering with breathing blood pressure, or heart rate. Fortunately, most people eventually recover from even the most severe cases of GBS (Figure 1). After recovery, people may continue to have some weakness.

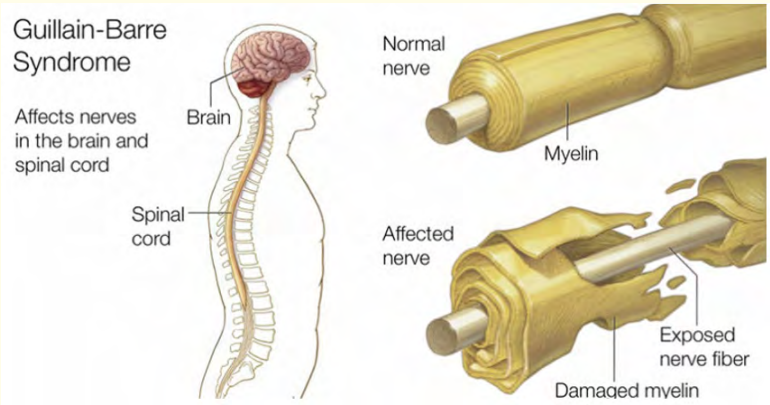

Figure 1: *GBS affected nerve.*

Epidemiology

There are 100,000 new cases of GBS every year [3]. Most studies that estimate incidence rates of Guillain Barré syndrome were done in Europe and North America, and showed a similar range of 0.81 -1.91 (median 1-1) cases per 100000 people per year [3]. Estimates of yearly incidence (per 100,000 people) are lowest in Japan (0.44) [4], China (0.67) [5], Tanzania (0.83) [6], and Finland (0.84) [7], and highest in Chile (2.12) [8] and Bangladesh (3.25) [8], likely due to differences in exposure to infectious organisms. Seasonal variations are described [10], and spikes of GBS have been reported following infectious outbreaks (Figure 2), most notably in relation to *Campylobacter jejuni* [11] and Zika virus (ZIKV) [12,13].

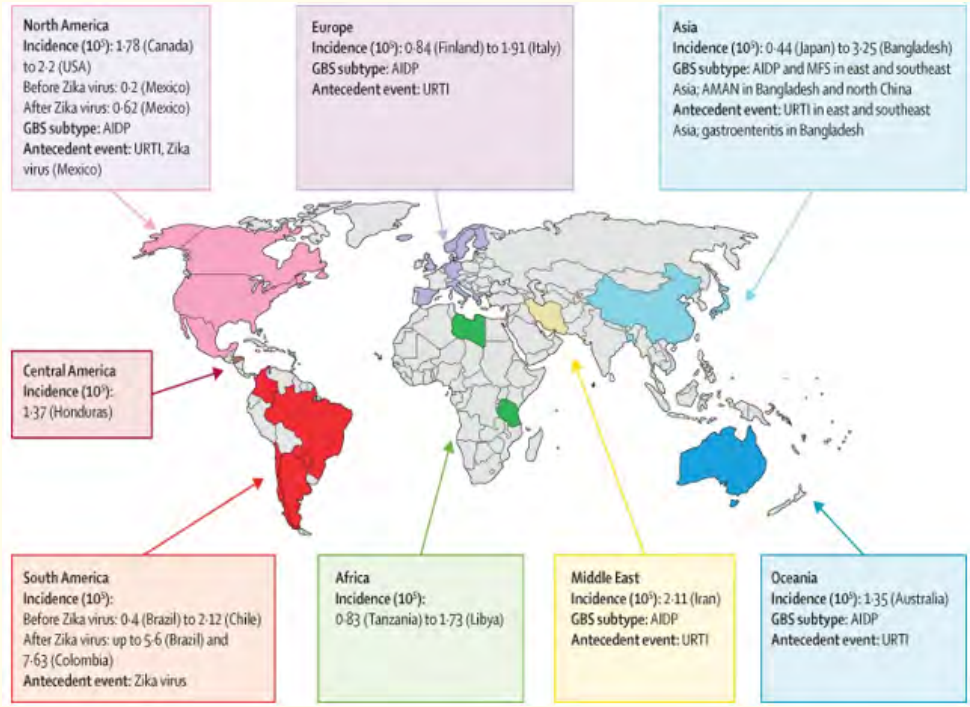

Figure 2: *GBS incidence rates.*

The incidence of GBS in India is estimated to be between 1 to 2 cases per 100,000 people per year. India has recently experienced an outbreak of GBS primarily affecting the state of Maharashtra, with cases also appearing in other states. The outbreak has been linked to contaminated water sources, specifically a bacteria called *Campylobacter jejuni*. As of March 2025, around 300 cases and 23 deaths have been reported across eight states. Maharashtra has seen the highest number of cases, with Pune being the initial epicenter. Cases have also been reported in Andhra Pradesh, Assam, Tamil Nadu, and West Bengal. Research indicate that the covid-19 pandemic had an impact on the clinical characteristics and presentation of GBS cases in India (Figure 3). COVID-19 is associated with an increased risk of Guillain-Barré syndrome (GBS), both during active infection and after recovery.

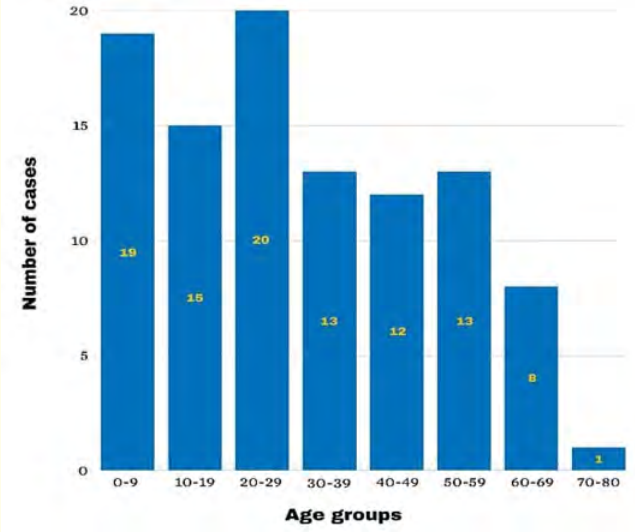

Figure 3: *GBS outbreak in Pune age wise distribution.*

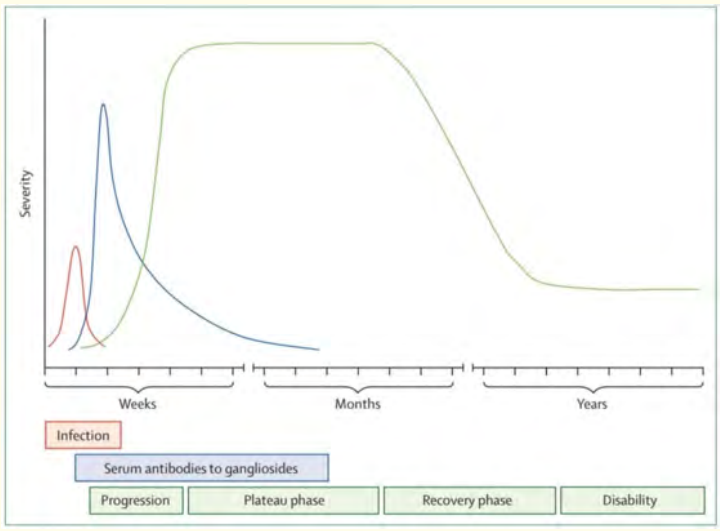

Figure 4: *Guillain Barre' Syndrome time course.*

Guillain-Barré syndrome increases with age (0.6 per 100000 per year in children and 2.7 per 100000 per year in elderly people aged 80 years and over) and the disease is slightly more frequent in males than in females [10]. Many different preceding infections have been identified in patients with the disorder, but only for a few microorganisms has an association been shown in case-control studies. *C. jejuni* is the predominant infection, found in 25-50% of the adult patients, with a higher frequency in Asian countries [14,15]. Other infections associated with Guillain Barré syndrome are cytomegalovirus (CMV), Epstein Barr virus, influenza A virus, *Mycoplasma pneumoniae*, and *Haemophilus influenza* [16,17]. An association of Guillain-Barré syndrome with hepatitis E has been identified in patients from both the Netherlands and Bangladesh [18,19]. An emerging relation between Guillain-Barré syndrome and acute arbovirus infection including Zika and chikungunya is being closely monitored and is the subject of major interest as the global epidemic spreads. Cases of Guillain-Barré syndrome have also been reported shortly after vaccination with Semple rabies vaccine and various types of influenza A virus vaccine. During the 1976 vaccination campaign for H1N1 influenza A virus, roughly one in 100000 people who had been vaccinated developed Guillain-Barré syndrome [20]. Although a similar association was suggested for the H1N1 influenza A vaccination in 2009, extensive studies showed only 1.6 excess cases of Guillain-Barré syndrome per 1000000 people vaccinated, a frequency similar to all seasonal vaccinations [21,22]. Vaccination might, in fact, reduce the chance of an individual developing Guillain Barré syndrome after natural infection with influenza A, which is itself a possible candidate to precipitate the disorder. A commonly asked clinical question is whether vaccination increases the risk of Guillain-Barré syndrome recurrence in previously affected individuals; this hypothesis seems not to be the case [23]. In a survey, none of the 106 patients with Guillain-Barré syndrome who had been vaccinated against influenza (range of vaccinations per person 1-37 times, total 775 vaccinations reported are occurrence [24]. Guillain-Barré syndrome increases with age (0.6 per 100000 per year in children and 2.7 per 100000 per year in elderly people aged 80 years and over) and the disease is slightly more frequent in males than in females of Guillain-Barré syndrome after the recurrence of Guillain-Barré syndrome after the vaccination.

History

Guillain-Barré syndrome (GBS) is named after the French physicians Georges Guillain, Jean-Alexandre Barré, and André Strohl, who first described it in 1916, though Jean-Baptiste Octave Landry first reported on a similar condition in 1859.

YEAR	
1859	Jean-Baptiste Octave Landry described an ascending paralysis, which is now recognized as an early description of GBS.
1916	Guillain and Barré were medical students together at the Saltpetriere in Paris at the turn of the century and specialised in neurology. During the First World War, they were both serving as doctors in the French Army. They noted the cases of two soldiers who had become partially paralyzed. One, in particular, had fallen over when he had put his pack on and had been unable to get up. Both the soldiers quickly recovered, possibly assisted by treatments with pork chops and claret. Together with Strohl, they published their classic paper in 1916. It was noted that reflexes were reduced and that the protein level in the cerebrospinal fluid was raised though this was not accompanied with a high white blood cell count. This was a crucial discovery as two common infections of the time, syphilis and tuberculosis, would have shown such an increase. After World War One, doctors were faced with three similar conditions with slightly different definitions: Landry's ascending paralysis, acute febrile (or perhaps infectious) neuropathy and the radiculoneuritis described by Guillain et al.
1927	It was in 1927 when the term Guillain-Barré syndrome was first used at a presentation by Dragonescu and Claudian. Their presentation was introduced by Barré himself but Strohl's name was omitted not only from the title of the presentation but also from the list of authors in the reference to the 1916 paper.
1949	In 1949 Haymaker and Kernohan suggested a wider definition of the illness, suggesting that Landry's ascending paralysis and Guillain-Barré syndrome were indistinguishable and called the condition Landry-Guillain-Barré syndrome.
1953	Guillain's last paper was in 1953. He believed the syndrome to be generally benign though the death of a patient, who after postmortem was found to have extensive peripheral nerve damage, had necessitated a shift in his position. Guillain still suspected an unknown infection as the cause and dismissed suggestions, made ten years earlier (Bannwarth), that the cause was due to allergy.

YEAR	LATER DEVELOPMENTS
1956	Charles Miller Fisher, a US doctor, described three patients with acute external ophthalmoplegia (eye paralysis), sluggish pupil reflexes, ataxia (lack of balance) and areflexia (absent tendon reflexes). Two patients had no weakness; the other had a facial palsy and possible weakness. All three recovered spontaneously . Because some patients with GBS had ophthalmoplegia and there were other similarities, Dr Fisher concluded that these patients had suffered a disorder akin to GBS.
1958	A paper was published by Dr JH Austin who described a chronic form of GBS. Austin's paper was based on a review of 30 cases, the earliest of which went back to 1894, and on two of his own. This chronic form has gone through a variety of names and attempts to define it though it now generally known as CIDP (Chronic Inflammatory Demyelinating Polyneuropathy).
1970's & 1980's	Diagnostic criteria were developed, and the effectiveness of plasmapheresis and intravenous immunoglobulin therapy was established.
1980's	The acute axonal type of GBS was first recognized, expanding the understanding of the disease beyond the previously recognized demyelinating form.

Table 1: *History of GBS.*

Etiology and developments

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. 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. 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. 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. The Vaccine Adverse Event Reporting System defines vaccine associated GBS as the emergence of GBS symptoms within 6weeks of vaccination. According to certain studies, vaccinations such as the meningococcal vaccine, poliovirus vaccine, vaccine, and rabies vaccine are capable of triggering GBS. A study revealed that the monovalent inactivated influenza A (H1N1) 2009 vaccine had been associated with a slight spike in the incidence of GBS. The precise mechanism of GBS after receiving the COVID-19 vaccine has not been determined yet. There may be a molecular mimicry mechanism functioning in COVID-19 vaccine-associated GBS which needs to be investigated with importance.

Clinical symptoms

Weakness: The weakness seen in GBS usually comes on quickly and worsens over hours or days. Often, feet are affected first, and weakness may move up the body to eventually impact the legs, arms, face, and breathing muscles. The person may first notice unexpected difficulty climbing stairs or walking. Less commonly, symptoms start in the face and move down to the legs and feet. Most people reach the greatest stage of weakness within the first two weeks after symptoms appear by the third week 90% of affected people are at their weakest.

Sensation changes: In GBS, the brain may receive abnormal sensory signals from the rest of the body due to the nerve damage associated with the condition. This results in unexplained, spontaneous sensations, called paraesthesias, that the person may feel as tingling, a sense of insects crawling under the skin (called formications), and pain. Some people with GBS feel a deep muscular pain in the back and/or legs. Unexplained sensations often happen first, such as tingling in the feet or hands or even pain (especially in children), often starting in the legs or back. Children will also begin to have difficulty walking and may refuse to walk. These sensations tend to disappear before the major longer term symptoms appear.

Other symptoms of Guillain-Barré syndrome may include (Figure 5):

- Difficulty with eye muscles and vision
- Difficulty swallowing, speaking, or
- chewing Pricking or pins and needles in the hands and feet
- Pain that can be severe, particularly at night
- Coordination problems and unsteadiness
- Abnormal heart rate or blood pressure
- Problems with digestion and/or bladder control.

Figure 5: *Sign and symptoms of GBS.*

Classification of GBS

There are three variants of GBS (Figure 6 and 7):

1. Clinical variants
2. Electrophysiological variants
3. Sensory variants.

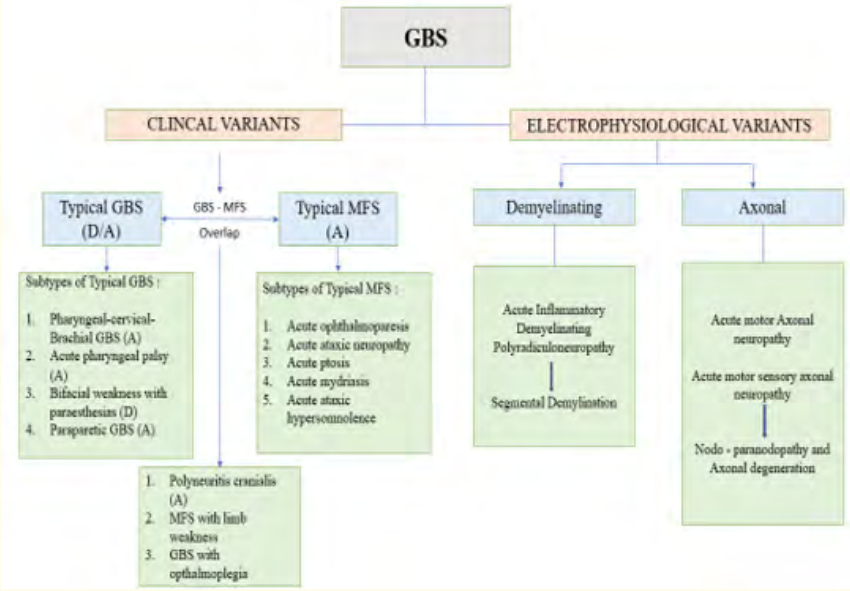

Figure 6: *Phenotypic and electrophysiological classification of GBS.*

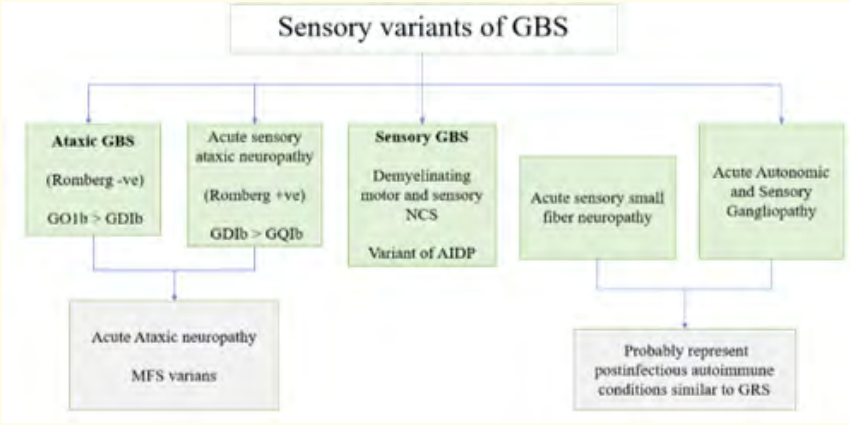

Figure 7: *Classificatoin of sensory variants of GBS.*

Pathophysiology and clinical finding

GBS is triggered 1-3 weeks prior of gastrointestinal or respiratory infection that shows molecular mimicry. Molecular mimicry plays a substantial role in our understanding of GBS, particularly the axonal variant. The lipooligosaccharides of *Campylobacter jejuni* is similar to the gangliosides of peripheral nerve membranes. Gangliosides antibodies have been shown to have different peripheral nerve targets.

In molecular mimicry, ganglioside antigens are shared between peripheral nerve and pathogen coat proteins. This triggers immune response that cross-reacts with peripheral nerves, beginning at nerve roots that increases permeability of blood nerve barrier at level of proximal nerve roots which increases protein in cerebrospinal fluid. Immune response that cross-reacts with peripheral nerves causes demyelination (antibodies attack Schwann cells) that furthers cause secondary damage - Axonal damage (antibodies attack nodes of Ranvier). Demyelination is responsible for AIDP that along with AMAN and AMSAN by axonal damage, leads to Acute immune-mediated polyneuropathy (Figure 8-10).

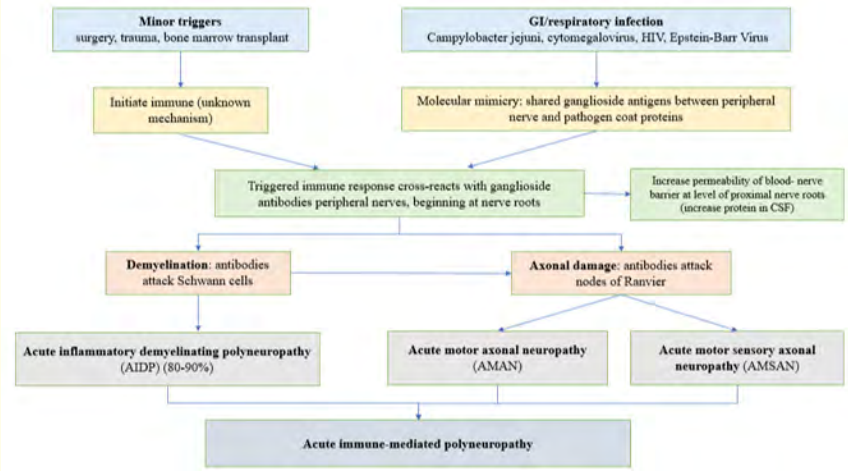

Figure 8: *Pathophysiology of GBS.*

Antibody	Target site	Variant of GBS	Notable phenotypic features
Anti-ganglioside antibodies			
Anti-ganglioside GM1a/b	Node	AMAN	Anti-GM1 Abs are linked with reversible conduction failure
Anti-ganglioside GD1a	Node	AMAN, AMSAN	More specific and sensitive than GM1 antibodies
Anti-ganglioside GalNac-GD1a	Node	AMAN	Distal dominant weakness
Anti-ganglioside GD1b	Node	Acute ataxic neuropathy	Acute onset ataxia with or without Romberg sign
Anti-ganglioside GQ1b	Paranode	MFS and subtypes	Anti-GQ1b Abs suggest good treatment response prognosis
Anti – ganglioside GT1a	Paranode	PCB variant	
Non-Ganglioside antibodies			
Neurofascin (NF186)	Node	AMAN, AIDP	
Neurofascin (NF155)	Paranode	AIDP	More likely in CIDP than in AIDP
Contactin 1(CNTN1)	Paranode	AIDP	More likely in CIDP than in AIDP

Figure 9: *Antibodies in GBS and their clinical significance.*

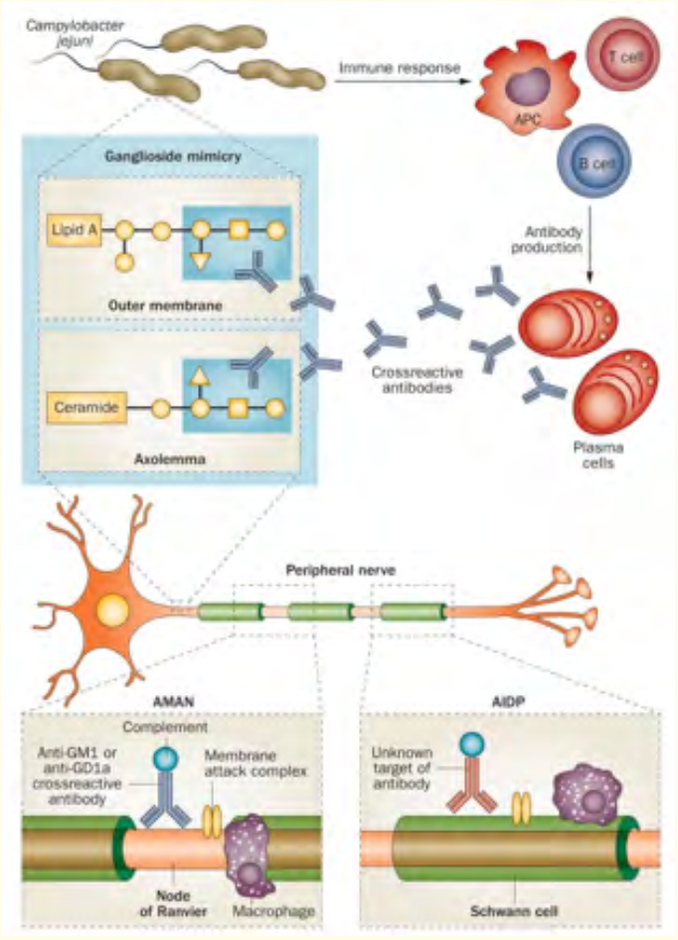

Figure 10: *Reaction of antibodies on peripheral nerve.*

Diagnosis

Guillain-Barré syndrome (GBS) diagnosis involves a combination of clinical assessment, nerve function tests, and cerebrospinal fluid analysis. A key finding is albuminocytologic dissociation (elevated protein in the cerebrospinal fluid with normal white blood cell count). Early diagnosis is crucial for timely treatment and management (Figure 11).

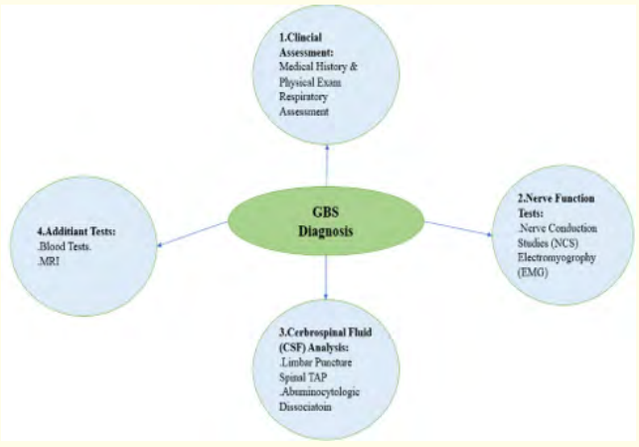

Figure 11: *Diagnosis of GBS.*

Here's a more detailed breakdown

1. **Clinical assessment:**

- **Medical history and physical exam:** Doctors will assess symptoms like progressive muscle weakness, numbness, tingling, and pain, particularly in the extremities. They will also check for diminished or absent deep tendon reflexes.
- **Respiratory assessment:** Due to the potential for respiratory muscle involvement, doctors will closely monitor breathing function.

2. **Nerve function tests:**

- **Nerve conduction studies (NCS):** These tests measure the speed at which electrical signals travel along nerves, helping to identify nerve damage patterns.
- **Electromyography (EMG):** This test assesses the electrical activity of muscles and nerves, helping to distinguish GBS from other conditions.

3. **Cerebrospinal fluid (CSF) analysis**:

- **Lumbar puncture (Spinal tap):** A small amount of CSF is collected from the lower back and analyzed.
- **Albuminocytologic dissociation:** A hallmark of GBS is elevated protein levels in the CSF with normal white blood cell counts.

4. **Additional tests:**

- **Blood tests:** These can help rule out other conditions and assess overall health.
- **MRI:** While not routinely used, MRI scans can help exclude other conditions like spinal cord inflammation or brainstem infections.

5. **Importance of early diagnosis:**

- Rapid progression.
- GBS can worsen quickly, potentially leading to respiratory failure and other complications.

Treatment

Guillain-Barré syndrome (GBS) treatment focuses on supportive care and therapies to reduce the severity and duration of the illness. Two main treatments are plasma exchange (plasmapheresis) and intravenous immunoglobulin (IVIg) therapy. Physical therapy is also crucial for recovery.

1. **Immunotherapy:**

- **Plasma exchange (Plasmapheresis):** This procedure removes harmful antibodies from the blood that are attacking the nerves.
- **Intravenous immunoglobulin (IVIg):** This treatment involves infusing healthy antibodies from donors to neutralize the harmful antibodies.

2. **Supportive care:**

- **Respiratory support:** A significant number of GBS patients require mechanical ventilation due to respiratory muscle weakness.
- **Pain management:** Pain relief is essential, especially during the acute phase.
- **Preventing complications:** Measures to prevent blood clots, pneumonia, and other complications are crucial, especially in severe cases.

3. **Physical therapy:**

- **Mobility and strength:** Physical therapy helps restore strength, flexibility, and mobility.
- **Functional training:** Therapists guide patients in regaining daily living skills such as dressing, washing, and walking.
- **Speech therapy:** If GBS affects swallowing or speaking, speech therapy can help.
- **Mobility aids:** Canes, braces, and wheelchairs can aid in mobility and reduce fatigue.

4. **Other treatments:**

- **Corticosteroids:** May be used in conjunction with other treatments to reduce inflammation.
- **Monitoring:** Regular monitoring of vital signs, respiratory function, and other parameters is essential.

5. **Recovery:**

- Recovery from GBS can be a long process, and some individuals may experience lingering effects.
- Physical and occupational therapy are crucial for regaining strength and function.
- New treatments and therapies are continuously being researched and developed.

Conclusion

Guillain-Barré Syndrome is a serious neurological condition with multifaceted etiology and variable clinical outcomes. Early recognition and treatment are crucial for improving prognosis. Treatments have been developed and proved effective, but these are not sufficient in many patients. Although there have been major steps forward, this is no time for complacency as the research area continues to face deep, unsolved issues around pathogenesis of Guillain-Barré syndrome, especially for the acute inflammatory demyelinating polyneuropathy form of the disorder. Continued research into the pathophysiology and treatment of GBS is essential for enhancing patient care and outcomes.

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