Journal of Applied Pharmaceutical Research
Volume 13 Issue 4, Year of Publication 2025, Page 16 – 35
DOI: https://doi.org/10.69857/joapr.v13i4.1091

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Review Article

JOURNAL OF APPLIED PHARMACEUTICAL RESEARCH | ***JOAPR***
www.japtronline.com ***ISSN: 2348 – 0335***

SCIENTIFIC PERSPECTIVES ON GUILLAIN-BARRÉ SYNDROME (GBS): A COMPREHENSIVE REVIEW FOR SENTIENCE AFTER EARLY 2025 GBS OUTBREAK IN AN INDIAN STATE

Sarika J. Patil[1], Rohit R. Bhosale[2]*, Dhanashri D. Chavan[3], Akshay R. Yadav[4], Swapnil S. Patil[5]

Article Information
Received: 13th April 2025
Revised: 29th June 2025
Accepted: 19th July 2025
Published: 31st August 2025

Keywords
Guillain-Barré Syndrome (GBS), Pathophysiology, Risk factors, Diagnosis, Treatment options, Current research advances

ABSTRACT
Background: Guillain-Barré Syndrome (GBS) is an acute, self-limiting, and rare neurological disorder wherein the body's immune system mistakenly attacks the peripheral nervous system (PNS). A report, published in February 2025 by the Indian newspaper 'The Times of India', highlighted a significant outbreak of GBS in the Indian state of Maharashtra, owing to the *Campylobacter jejuni* (*C. jejuni*) infection. The surge in cases has been considered as one of the most significant recorded GBS outbreaks globally, which underscores the need to raise GBS awareness. **Method:** This article provides an in-depth scientific perspective on GBS, drawing on literature from scientific databases such as PubMed and ScienceDirect. It aims to enhance awareness among science-related students, researchers, medical and paramedical professionals, and the general public. **Result and discussion:** GBS is an acute polyneuropathy characterized by limb weakness with hyporeflexia or areflexia. In severe forms, respiratory and bulbar paralysis can occur, requiring mechanical ventilatory support. It is the commonest cause of acute neuromuscular paralysis. The basic underlying mechanism of the disease is a localized attack against the myelin sheath of the peripheral nerves and nerve roots, with secondary axonal damage. It is believed that the bacterial antigens have a close molecular mimicry with neural antigens. As a result, the response generated against these antigens cross-reacts with the neural cells. Plasma exchange, immunoglobulin infusion, and plasmapheresis are the mainstays of treatment for GBS. **Conclusion:** A thorough understanding of GBS is essential, including its pathophysiology, underlying causes, risk factors, symptoms, diagnostic methods, treatment strategies, and the latest advancements.

[1]*Department of Pharmaceutics, Krishna Institute of Pharmacy, Krishna Vishwa Vidyapeeth (Deemed to be University), Malkapur, Karad-415539, Maharashtra, India.*
[2]*Department of Pharmaceutics, Krishna Charitable Trust's Krishna College of Pharmacy, Malkapur, Karad–415539, Maharashtra, India.*
[3]*Department of Pharmacology, Krishna Institute of Medical Sciences, Krishna Vishwa Vidyapeeth (Deemed to be University), Malkapur, Karad-415539, Maharashtra, India.*
[4]*Department of Pharmaceutical Chemistry, Krishna Charitable Trust's Krishna College of Pharmacy, Malkapur, Karad-415539, Maharashtra, India.*
[5]*Department of Pharmaceutics, Annasaheb Dange College of B. Pharmacy, Ashta-416301, Maharashtra, India.*

For Correspondence: **bhosalerohit707@gmail.com**

INTRODUCTION

Guillain-Barré syndrome (GBS) is a rare autoimmune disorder affecting the peripheral nervous system. Globally, it's estimated that around 150,095 cases of GBS occurred in 2019, with a point prevalence of 1.9 per 100,000 people. The disease burden is increasing, with a 6.4% rise in age-standardized prevalence since 1990. Originally identified by Georges Guillain and Jean Alexandre Barré in 1916, this condition is also referred to as acute inflammatory demyelinating polyradiculoneuropathy (AIDP). GBS is a rare but serious condition that affects the peripheral nervous system (nerves outside the brain and spinal cord). It can cause muscle weakness, paralysis, and problems with coordination and balance [1,2]. Most people do make a good recovery from GBS, which can involve hospital treatment with care from various specialists. However, it can take a long time. In a few cases, it can be life-threatening [3]. For reasons that are not yet fully understood, GBS is more common in adults than in children, and although it may happen at any age, it is only really seen in very young or elderly people in extremely rare cases [4]. The first sign of GBS is often a tingling or pain in the arms and legs, which could be followed by muscle weakness or difficulty moving. For a small minority of people, it could lead to paralysis of the body, requiring full support of breathing by a ventilator [5].

There is much that is still unknown about what happens inside the body during recovery from paralysis after GBS. GBS is a rare neurological disorder also called acute inflammatory demyelinating polyradiculoneuropathy (AIDP), a devastating disease and the most common cause of subacute paralysis in Western countries [7]. It is estimated that the disease affects one or two people per 100,000. In most cases of GBS, between a third and a half of a patient's progressive limp is no longer persistent, but death can occur in approximately five percent of cases [8,9]. It is essential to diagnose GBS early for rapid treatment with the available therapies that can mitigate its symptoms [10]. Specifically, it seems that the use of plasma exchange and intravenous immunoglobulin may reduce the severity and duration of illness for those who treat patients with GBS [11,12]. It is essential to recognize that GBS is not a single disease, even though it is usually treated in the same way. There are several variants and types of GBS, the most common being AIDP, which is characterized by widespread inflammation and loss of myelin in multiple peripheral nerves [13]. The disease usually occurs a few days or a few weeks after a minor infection, such as a cold, sore throat, or gastrointestinal illness. The sudden onset is the initial symptom of GBS, with a viral or bacterial infection as the most common trigger. The problem begins with weakness and numbness in the legs, which can spread to the chest and arms. The body's muscles often don't respond to commands from the brain, and the patient can gradually lose muscle function [14]. Usually, the infection is over by the time the patient is sick enough to develop paralysis. Reports indicate that about a third of the patients have difficulty swallowing and that a quarter have to be placed on a respirator at some point during the course of the disease. Limited control of eye muscles and abnormal heart rhythms are additional problems that arise in a small proportion of patients [15,16]. As it is a rare disease, the treatment and follow-up can be costly; however, the cost for the patient as well as society may be reduced by learning more about what causes this disease and by developing better treatment options [17-19].

The GBS outbreak, initially centered in Pune and spreading to other states, had resulted in over 200 reported cases and 23 deaths. The World Health Organization (WHO) had to step in to assist Indian health authorities in managing the outbreak. This surge in cases emphasized the need for better understanding and preparedness to manage GBS not just within India but on a global scale.

PATHOPHYSIOLOGY

The pathophysiologic hallmark of GBS is an inflammatory attack by the immune system on the peripheral nervous system (PNS), specifically against the myelin sheath of the peripheral nerves and nerve roots, with secondary axonal damage; wherein an immunological trigger, such as an infection, sets off the immune response, followed by damage to the axons and/or myelin. GBS and its variants are widely recognized as immune-mediated neuropathies that typically arise following an infection. Research, particularly from animal studies, points to molecular mimicry as a crucial factor in their development. A well-documented example involves *Campylobacter jejuni (C. jejuni)*, a bacterial pathogen associated with GI infections. This bacterium possesses a lipooligosaccharide in its outer membrane that closely resembles gangliosides, which are essential components of peripheral nerves. As a result, the immune system, in its effort to eliminate the infection, may mistakenly target the body’s own nerve tissues, leading to neurological damage.

Moreover, Epstein-Barr virus, cytomegalovirus, and *Mycoplasma pneumoniae* (*M. pneumonia*) are commonly identified antecedent pathogens responsible for developing GBS. A wide range of infections has been implicated in triggering GBS, with gastrointestinal and respiratory infections being the most frequently reported precursors. Studies indicate that up to 70% of individuals diagnosed with GBS recall experiencing an illness within one to six weeks before the onset of symptoms. Notably, during the Zika virus epidemic, there was a surge in reported GBS cases, further reinforcing the link between viral infections and the syndrome. Additionally, various case reports have suggested other potential triggers, including certain medications and surgical procedures, though these associations require further investigation [20-22]. This complex interplay between infection, immune response, and nerve damage highlights the need for continued research into the underlying mechanisms of GBS and its various subtypes [23].

In the acute inflammatory state of the PNS, antiganglioside antibodies have been found in some human patients, providing direct evidence for an autoimmune etiology. Cross-reactivity of antimyelin/antiganglioside B cell and T cell clones suggests that molecular mimicry is the leading mechanism. A related approach uses engineered T cell receptors specific for myelin epitopes to investigate the role of molecular mimicry in GBS. This early stage in disease development is an attractive target for treatment strategies aimed at modulating the very early stage of autoreactivity in GBS and perhaps other T cell-mediated autoimmune diseases as well. T cells from cytokine-deficient donors have been shown to limit the development of autoimmune diseases in several mouse models.

By exploiting oligonucleotide-oligosaccharide conjugates acting as ligands for antigen-specific T cells, it was demonstrated that selective, transient intravenous injection of peptide-bound antigenic stimuli prevents autoimmune responses leading to autoimmune diseases by inducing intensive apoptosis of cognate autoreactive T cells [24,25]. This induction of antigen-specific apoptosis removes T cells with the precise antigen specificity revealed by the therapeutic interventions, as verified by the absence of re-populating antigen-specific T cells after stimulation of the thymus with the whole antigen [26]. T cell deletion by this treatment selectively occurs among T cells in the very sensitive naive state of the autoantigenic T cell precursors [27].

Immune system mechanisms involved

The immune response depends on the distinction between self and non-self-cells, and several mechanisms are in place to prevent the activation of an immune response to one's own body components [28,29]. In normal conditions, healthy but activated immune cells are eliminated by the immune system. The same phenomenon can exist to maintain the tolerance of the host to the endogenous components of the peripheral nerve [30]. However, in autoimmune diseases, activated immune cells against host tissue antigens escape elimination, attack, and damage this tissue. A challenging question is the triggering event of the autoimmune response that opens the way to pathogenic T cells [31]. Interactions of lymphocytes with their target antigen are necessary for their activation and proliferation. This happens in lymphoid organs that harbor large numbers of both T cells and antigen-presenting cells. Cells of the peripheral immune system, T lymphocytes, and others recognize the antigen because they have a receptor specific for this antigen; here, the nerve components "weakly cell" or "cryptically cell" processing and presentation by immunocompetent cells of the immune system are other components of the system's authentic branch point for T cell activation [32]. T cells act as the final effector cells to inflict damage; that is, the cells responsible for tissue injury. Up to this point, there is a cascade of primary immune mechanism actions involved, including the T cells, previously sensitized in the presence of an effective Antigen-presenting cell (APC). These interactions can lead to T cells becoming activated and ultimately mount nervous tissue damage [33,34].

Effect on the peripheral nerves

GBS is an autoimmune condition that affects the PNS and occurs more commonly in males than in females, typically starting in their 40s. The progression of the disease continues to worsen for about three to four weeks before stabilizing [35]. The loss of function experienced by patients usually escalates over a span of 12 hours but can happen over the course of many days. Symptoms may continue to develop for up to two weeks, and spastic paralysis transpires faster than flaccid paralysis [7,36]. The syndrome can damage the sense of touch as well as other functions of our body’s muscles, such as the autonomic nervous system. This system is responsible for regulating involuntary body functions, including the heart rate, blood flow, temperature regulation, digestion, and bladder control [37]. The lack of blood flow stimulated by the disease is not the direct cause of its neuropathy; rather, the issue lies in the sugar molecules in the

blood that become absorbed into intracellular molecules. This impedes the transport of materials from the cell body to the axon and results in an inability of the cell body to repair the cell wall [11]. In general, the axon experiences more wall damage as a result of GBS, and there is a possibility of singular axon destruction due to the removal of the protective myelin layer. This, in turn, prompts decomposition of the nerve and stops the axon from growing back [38]. Figure 1 represents the pathophysiology of GBS logically and straightforwardly.

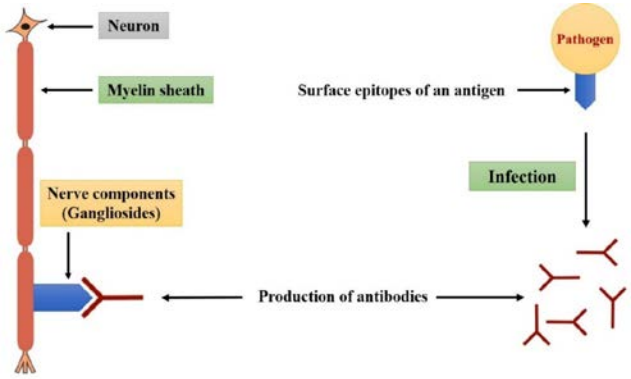

Figure 1: Pathophysiology of GBS.

CAUSES AND RISK FACTORS

The most commonly identified trigger for possible development of GBS is gastrointestinal infection with *C. jejuni*, one of the most common causes of food poisoning. Also, as mentioned in the pathophysiology section, a well-documented example involves infection caused by *C. jejuni*, including Epstein-Barr virus, cytomegalovirus, and *M. pneumoniae*, commonly identified as antecedent pathogens responsible for developing GBS. It's known that GBS sufferers come down with Guillain-Barré in the aftermath of some infectious illness, whether it is the flu, bronchitis, or some unforeseen stomach or intestinal infection. The symptoms of a *Campylobacter* infection range from cramping, diarrhea, fever, irritability, and overall malaise (as is the case in children). For fifty percent of those who come down with GBS, it all starts with some bacterial-related livestock [6,20,21].

Bacterial infections

Infections from different bacteria have been shown to precede GBS, however, *C. jejuni* is responsible for up to 40% of GBS cases and is the primary antecedent infection described. *C. jejuni* has a presence in the intestines of all birds, although chickens and turkeys are well-known members. The relative risk of developing GBS after an infection with *C. jejuni* is estimated to be 100-fold higher compared to the general population [39-41]. However, overestimated associations are also reported. This organism is also found in the feces of approximately 25% of cattle, pigs, and birds, the primary sources of human exposure, with infection due to food-borne contaminated products or consumption of unpasteurized milk [42]. Serological profiles that follow Campylobacteriosis with a strong antibody response against lipopolysaccharide core are closely linked to the development of GBS. However, a problem that often arises is that islet-activating effector T cells contribute to the severity of campylobacteriosis by questioning the antibody-related immunity model [43,44]. Probably, only a subset of individuals exposed to *C. jejuni* infection will develop GBS, and require some form of immune dysfunction to promote the development of GBS. Enterotoxigenic Bacteroides fragilis (ETBF), a toxin-producing strain of the bacteria *Bacteroides fragilis* (*B. fragilis*) that can cause diarrhea in humans, has also been described in relation to the previous onset of GBS [45,46]. Moreover, enteritis leads to an increased serum immune response triggered by the patient-specific strain. Several molecular mimicry epitopes found in GBS patients and reported clinical signs and symptoms were identified from the immune response [47]. Moreover, in some healthy controls, pre-existing cellular immunity against a molecular mimicry epitope has been detected, suggesting that colonization or hidden infection may lead to immune suppression or inhibitory response in GBS patients. In contrast, in healthy controls, protective immunity is maintained [48]. Thus, a relationship exists where colonization of *B. fragilis* may lead to the formation of host or grouped; in response to colonization or a previous infection event, there is substantial evidence that this type of event may be solved with the epitope-specific T cell-mediated induction of an immune suppression response [49,50]. Other bacterial infections, often involving acute gastroenteritis, including *Mycoplasma pneumoniae*, *Helicobacter pylori* (*H. pylori*), *Chlamydia trachomatis*, *Haemophilus influenzae*, and *Neisseria spp.*, have also been leading infections. *H. pylori*, which causes gastric or peptic ulcers, esophageal cancer, and primary thrombocytopenia, has been thought to link the relationship between GBS and other infectious diseases caused by *H. pylori*-induced idiopathic thrombocytopenic clinical death [51,52].

Viral infections

Viral infections are also linked to GBS, but well-documented viral-trigger cases are relatively uncommon. The viral-induced demyelination can generate antigens to initiate the immune-

activated pathology. Still, these antigens might not be present for long enough to induce an immune response, and the cellular response to the viral infection might be initiated too late in the reaction to initiate cross-reactivity and antiganglioside antibody generation. The most common viral triggers of GBS are associated with 5% of triggered GBS cases [7-11]. The viruses share common antigens with gangliosides, and the immune response to the virus can cross-react with the antigens, generating antibody or T cell activation. The anti-gangliosides can bind directly to neurons and initiate autoimmune responses, whereas activation of the immune cells leads to the T cell-mediated pathology characteristic of AIDP [53,54].

Respiratory and gastrointestinal infections

Respiratory or gastrointestinal tract infection, particularly of the upper airways, frequently precedes GBS. After a specific viral infection, patients may develop nonspecific symptoms like those of the disease. Respiratory illness is more frequent with *M. pneumophila* and less frequently associated with *S. pneumoniae* in adults, and such patients are certainly at risk for a bacterium, where *G. lamblia* is frequently associated with waterborne disease, and *S. gondii* may be asymptomatic as well [55,56]. In the pediatric population, the injury is typically less severe than among adults, and respiratory or gastrointestinal infection precedes most illnesses [57].

Surgery and trauma

Surgery and trauma can trigger the onset of GBS. As with bacterial infection, there is no direct evidence that bacteria enter the bloodstream and trigger an immune response. The likely explanation is that infections cause weakening of the gut lining, enabling small molecules to seep through the gut wall. Usually, large molecules and food cannot get through the blood-brain barrier and enter the spinal column [58]. Once inside, a normally secluded protein in the immune system can be recognized by its components. Over the next few weeks, antibodies are produced or their numbers increase, and they happen to cross-react with the myelin sheath's key structural protein. Now, when the skin or the lungs are pricked, the T cells that are reactive with myelin also respond [59,60]. More specifically, damage of some type to the myelin component that is recognized as foreign triggers antibody-producing cells to shift into high gear. They produce more antibodies, and the free T cells in the spinal column become overloaded with these copies, leading to the destruction of myelin around the axon [61]. Antibodies are released and self-produce more copies of themselves. As a consequence, other T cells are made that recognize the foreign substance as the common structure. On balance, the body sees a bacterial or other infection, and the onset of nerve damage can follow. Beyond active immunization, passive immunization has also been observed in some patients [55,61].

Vaccination

There has been speculation that GBS is caused by vaccinations for the eradication of infectious diseases, such as hepatitis B, whooping cough, diphtheria, polio, enterovirus, and influenza. The controversy surrounding the additional risk of GBS in vaccine recipients has made it difficult for public health officials to eradicate or reduce infectious diseases [62]. It was reported in 1978-79 that the National Influenza Immunization Program Administration in the US increased cases of GBS 8-10 fold over background rates, resulting in epidemiological studies that were only performed retrospectively [63]. It has become apparent that the H1N1 vaccine can provoke central respiratory failure and death in some cases of GBS. Even though the US Department of Defense published a protocol on how to manage GBS if it arose from the H1N1 vaccine, saying that since August 2012, the United States has received 24 vaccinations of H1N1, which resulted in the onset of GBS within 6 weeks of immunization, there are still centers to this day that are not linking the vaccine as a cause [11,64]. Figure 2 summarizes the causes of GBS.

SYMPTOMS AND VARIANTS

Emergency medical treatment should be sought if muscle weakness or an acute onset of unusual sensations occurs. The earlier GBS is diagnosed and treatment is begun, the more successful the outcome [65,66]. Tests performed may include MRI of the brain and/or MRI of the spine, lumbar puncture, and electromyography. Once the diagnosis is confirmed, IV immunoglobulin or plasma exchange therapy is initiated [67,68]. These therapies aim to stop the immune attack upon the nervous system and prevent long-term weakness. In severe cases, weakness becomes so great that the person is unable to breathe on their own, and a breathing machine will be necessary [69,70]. This is a life-threatening situation, and the person is admitted to the intensive care unit, likely for several weeks or longer [71,72]. Moreover, symptoms include muscle weakness or numbness/ tingling sensations, usually starting in the legs and spreading upward [73]. Furthermore, there are variants of GBS, which form a separate group of disorders with less weakness.

Bickerstaff's brainstem encephalitis (BBE) is a variant of GBS. In addition to leg, arm, and sometimes respiratory weakness, BBE has a disturbance in consciousness with drowsiness, and the eyes have a jerky movement called nystagmus and a slow to-and-fro movement called ophthalmoplegia [74,75]. There are also problems with swallowing and the development of oculopalatal myoclonus (a rare disorder causing rhythmic eye movements and simultaneous soft palate contractions). Some people with BBE have no arm weakness, distinguishing it from those with the Miller Fisher syndrome (MFS) who have, in addition to ophthalmoplegia and ataxia, no arm weakness [76,77]. People with polyneuritis cranialis present with an inability to move many facial muscles, and the sensation of the face is often abnormal. They sometimes have trouble closing their eyes completely, with a reduction in tearing, and taste is usually altered or reduced [78]. They can also develop limb weakness. Such people have cranial nerve dysfunction so severe that little or no leg weakness develops, and it can be weeks before some people with *polyneuritis cranialis* develop weakness of the muscles of breathing [79]. The most significant degree of neurotoxicity seems to be in small neuronal cells; the sensory ganglia, dorsal root ganglia, and cranial nerves and facial muscles depend on these ganglia for motor control [80]. Table 1 provides some other variants of GBS.

Figure 2: Causes of GBS

Table 1: Variants of GBS

Variant	Description	Treatment Approaches	Ref.
Acute Inflammatory Demyelinating Polyneuropathy (AIDP)	The most common form in the Western world involves inflammation & demyelination of nerves.	Plasma Exchange (PE) & IV Immuno globulins (IVIG) are commonly used	[81]
Miller Fisher Syndrome (MFS)	Characterized by ataxia, areflexia, and ophthalmoplegia (eye muscle weakness).	IVIG is the preferred treatment. PE can also be used if IVIG is not effective	[82]
Acute Motor Axonal Neuropathy (AMAN)	Involves damage to the axons of the nerves, primarily affecting motor function	IVIG is the primary treatment. PE can be considered in severe cases	[83]
Acute Motor and Sensory Axonal Neuropathy (AMSAN)	Similar to AMAN, but also affects sensory nerves.	IVIG is the primary treatment. PE can be considered in severe cases	[84]
Pharyngeal-Cervical-Brachial (PCB) Weakness	Affects the nerves controlling the muscles of the throat, neck, and upper limbs.	IVIG is the primary treatment. PE can be considered in severe cases	[85]

DIAGNOSIS

Careful examination of patients and a thorough understanding of their signs and symptoms are necessary for a medical provider to make a diagnosis of GBS. Various tests may be followed, including a lumbar puncture or spinal tap to collect spinal fluid, which can be tested for indicators that may suggest GBS, and electromyography to determine the presence of nerve damage [86-88]. The criteria for the diagnosis of GBS have remained essentially the same since its inception. The spectrum of GBS includes clinical variants such as recurrent GBS and an axonal variant subsumed under the rubric of AMAN. The requirement of elevated cerebrospinal protein gives strong support to the hypothesis that GBS is an autoimmune disease, as do the findings in some patients of antiglycolipid antibodies [89]. Electromyography (EMG) evidence of demyelination in the early days of illness is characteristic and may be fulminant. The albuminocytologic dissociation of increased cerebrospinal protein compared to a normal cell count must be present. AMAN, unlike the demyelinating form of GBS, has a subacute onset, less severe weakness, and does not have EMG or cerebrospinal fluid evidence of demyelination. The required acute length-dependent, multifocal demyelination and conduction block were demonstrated by nerve biopsy in a patient with GBS [90]. Additional diagnostic tests that may be performed when GBS is suspected include nerve conduction studies (NCS) and lumbar puncture. In an NCS, a harmless electrical stimulation is given to a nerve, and the response is measured. Patients with GBS have distinct changes in their NCS that help to confirm the diagnosis. In a lumbar puncture, also

known as a spinal tap, a small needle is placed in another layer of tissues that covers the spinal cord. A sample of cerebrospinal fluid is then removed and studied for signs of GBS or another condition that may mimic GBS. This test is also used to detect elevated protein levels, which can help confirm a GBS diagnosis [91-93].

TREATMENT OPTIONS

Though there is no cure for GBS, researchers have demonstrated that the syndrome is very responsive to the correct type of attention and treatment. The most dangerous symptom of GBS is that the paralysis can spread to the muscles that control breathing [94]. Fortunately, modern medicine has some advanced life-support equipment, and the majority of people who contract GBS make a full recovery. In fact, the most common treatment is rather conservative and supportive in the form of around-the-clock monitoring [95-97]. In severe cases, a person who has contracted the disease is administered artificial respiration. Monitoring for life-threatening conditions, such as blood clots or hypertension, is also necessary. For most individuals, alternating sessions of exercise and rest while receiving professional care will suffice. Intensive physical therapy is the most effective treatment for nerve recovery [98,99]. One of the most frustrating aspects of GBS is that it can occur at any age, and children are no exception. Hence, drug therapies may be sufficient for adults, whereas children with GBS typically require plasmapheresis [100].

No matter the method used, the key aspect to successful treatment is to initiate it as soon as GBS is strongly suspected. As with any illness, every person's sensitivity to viral attacks greatly differs [101]. Some individuals do not develop GBS until weeks after the body has resolved a bacterial or viral infection. During the recovery period, one may need to use special equipment, such as crutches or wheelchairs, to assist in movement. Since the pace of recovery is different for each individual, the use of prescribed physical aids will vary as well [102]. However, those who have suffered from GBS fall back into the same categories as normal individuals once they recover [103,104].

Plasma exchange (plasmapheresis)

In this procedure, blood is removed from the body, then processed to separate blood cells from other blood components. These other blood components, also called plasma, contain antibodies responsible for damaging nerve fibers. The separated blood cells are then returned to the body, where blood plasma is quickly replaced. The role of plasma exchange in the treatment of GBS is to remove those antibodies believed to cause the autoimmune response [105]. Numerous medical studies show that this approach is highly effective and greatly speeds recovery compared to simply watching and waiting due to the natural progression of the disease. Due to the high cost of plasma exchange, resulting from the need to pay for plasma and equipment used in a hospital environment, a method of selectively removing plasma antibodies was developed that became a replacement for plasma exchange [106,107]. Plasma exchange poses a greater risk to less stable patients with GBS [108]. Certain other medical conditions may also prevent safe use. Plasma exchange may require the handling of bulky equipment and the return of processed components to the correct blood vessel. Sometimes, surgical placement of a temporary blood vessel is associated with the risk of blood clots and infection [109].

Intravenous immunoglobulin (IVIg)

Immunoglobulins are the antibodies, the body's natural defense against invading organisms. GBS occurs when the body's immune system begins to attack the body itself, and in this case, the nerve fibers of the peripheral nerves. In the treatment of GBS, large doses of immunoglobulin are given to the patient through a vein in the arm. The donor of the immunoglobulin may be a pool of donors who have donated blood before, and these blood donations are then used as a source of immunoglobulin [110]. When the immunoglobulin is administered to a patient with GBS, it reacts with antibodies produced by the patient's immune system against the patient's own nerve fibers, causing them to malfunction. In the majority of cases, this stops the progression of the disease. At least one study has shown that a high dose of immunoglobulin given to the patient within the first week of the onset of GBS had a better outcome compared to those who received a low dose of immunoglobulin [111]. In the case of GBS and the treatment with immunoglobulin, minor side effects such as headache and/or fever can develop [112]. More serious side effects are unusual but have been reported, such as kidney damage or an overload of fluid, among a very small number of patients who are treated. One of the essential roles of the physician when caring for the patient with GBS is to decide whether the benefits of treatment with immunoglobulin outweigh the possible risks [113-115].

Medications

Physical and ergotherapy, along with medical procedures such as plasmapheresis or removal of contaminated fluids from the body, lessen the severity of GBS and accelerate the healing process, thereby reducing the complications of GBS [116]. Temporary airway support is required in ventilated patients, and comprehensive pulmonary care minimizes the probability of pneumonia [117]. In terms of medications, nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids can be used to relieve pain, anticonvulsants and antidepressants can be used to treat neuropathic pain, and selective serotonin reuptake inhibitors (SSRIs) and anxiolytics can be used to treat anxiety.

Physical therapy

The purpose of physical therapy in the acute phase of GBS is to provide sensory stimulation, maintain flexibility and skin integrity, and prevent contracture development. This is primarily accomplished with the use of positioning, passive stretching, rigid posterior stays or splints, serial casting, and range of motion [116-120]. The patient is limited in where these activities can occur due to sensory and motor involvement. Fatigue is also a significant factor. Patients should be allowed periods of rest between repetitions of exercises or therapies [121-126]. Physical therapy in the chronic phase also incorporates a range of motion and strength exercises, but the primary goal is to maintain active shortening in muscles and tendons [127-129]. Figure 3 summarizes the available treatment options for managing GBS.

PROGNOSIS AND OUTCOMES

The potential outcomes from GBS are variable due to the spectrum of motor and autonomic manifestations, variability associated with prior conditions, and variable temporal course of the disease. Consequently, patients who survive the acute episodes of the illness have heterogeneous functional recovery [130-133]. Indeed, despite initial severe quadriplegia and respiratory failure, many GBS patients may eventually recover almost normal nervous system function. For example, an adult GBS patient may return to an everyday life and have few, if any, lingering abnormalities [131,134-136].

On the other hand, GBS patients may have significant paresthesia, areflexia, and fatigue, which may be symptomatic and remind them of their acute illness throughout their lives. Long-term survival, as well as whether a patient will have lingering deficits, depends primarily on whether or not they suffer from secondary complications such as infection or the need for tracheostomy placements and prolonged ventilator support [132,134].

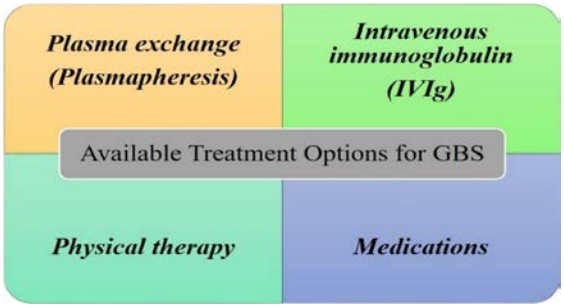

Figure 3: Available treatment options for managing GBS

Approximately 15-20% of treated cases remain significantly disabled, and 5% die. Furthermore, the mortality, particularly during the acute phase, depends on several factors, including the severity of weakness, as well as which specific treatment measures are implemented for each individual case [137-143]. Patients who have had GBS may continue to experience problems when the syndrome is active and during the recovery period.

These can be relieved or improved, but need to be identified and managed appropriately [144]. Possible support for the period of acute GBS and during recovery might include psychological support and rehabilitation services, including mobility aids, adaptive technology, support from neurophysiotherapy, self-help groups and others who have experienced GBS, advice from a specialist in neurological occupational therapy, especially for posture or joint concerns to help in maintaining independence and mobility, and also support for swallowing problems through a dietitian or speech therapist as well as psychological therapy in the case of depression or anxiety [145,146]. In the longer term, patients with GBS might need to be monitored and receive medical support because they can experience other diseases associated with age, particularly heart problems, vascular problems, high or low blood pressure, lung problems, blood clotting, and thyroid deficiencies [147,148].

Furthermore, incomplete recovery is common in patients with GBS, even though it is a neurological emergency. Immunotherapy is widely used for the treatment of GBS. Plasma exchange can shorten the duration of mechanical ventilation and decrease serum nerve growth factor levels, but their relationships with long-term outcomes and mechanisms remain

unclear [149,150]. Plasma exchange, hemoperfusion, and hemofiltration may reduce the chance of receiving mechanical ventilation therapy and decrease the time of recovery, but immunotherapy may have no significant effect on long-term outcomes. Enteral nutrition should be promoted for the prevention of malnutrition. Because severe acute respiratory syndrome has a higher possibility of incomplete recovery, the respiratory function of these patients should be given more attention [151].

RECENT ADVANCES IN RESEARCH

Several recent lab studies have pointed out some possible mechanisms of action of IV immunoglobulins in the treatment of Guillain-Barré. Muscle pain, a common symptom in patients, is becoming better understood, and a potential cause of severe muscle pain could be attributed to the necrosis of muscle spindles [152,153]. The question of whether acute misuse of muscle and muscle shortening during the course of rehabilitation is involved in the pathophysiology of the development of contractures could be due to loss of afferents that convey information regarding muscle tension [154].

Furthermore, it was shown that it is possible to stimulate muscle functionally by muscle electrostimulation with surface electrodes in varying positions over the muscle [155-158]. The genetic contribution to the risk for Guillain-Barré is being investigated. The discovery of an association between a certain polymorphism in the promoter region of the antioxidative enzyme suggests a possible mechanism of free-radical-mediated damage to axons [157,159]. The complement system also plays an important role in fiber degeneration. The hypothesis that inhibition of atypical activation of this system in peripheral nerves will prevent ongoing nerve degeneration was positively proven. This is shown step by step in rats. The clearance of myelin debris is further important for optimal recovery & remodeling of the axon & the myelin sheath, respectively [160-165]. Current research is yielding new information and advancements in treating Guillain-Barré, and clinical trials are also now underway or recently completed. The trials have their own set of specific criteria for participation. Clinical and biological characteristics are also evaluated, wherein the purpose of the study is to determine how often different biological markers of inflammation appear in the bloodstream of people with GBS to help learn more about the causes of GBS and to identify improved treatments for GBS [22,166]. Moreover, GBS in children in India is examined by taking reference from previous studies on how children are being treated, but not recruiting participants. This study aims to explore the clinical and epidemiological features, current trend of the disease, and short-term outcome of GBS in the pediatric age group in India. The study also examines the predictive factors of GBS severity and outcome, including mortality in children, and identifies therapeutic modalities [1].

Additionally, understanding and predicting immune recovery in GBS also followed, where the study recruited participants with the goal to find out more about the recovery patterns and immunology of GBS, wherein the research team followed people with GBS during their time in the hospital and after consent was given, participants visited the research center as scheduled, for one year after GBS diagnosis. With this study, the goal was to gain more information about the immune response and planned treatments for future Guillain-Barré studies [167]. Furthermore, drugs and models that accelerate GBS were evaluated to examine the extent to which investigational drugs and validated experimental models promote quick recovery from experimental GBS. The scientists conducted experiments and sought to include individuals with early GBS diagnosis to analyze their blood serum and gather information on how investigational drugs and identified molecular changes promote myelin regeneration, thereby accelerating recovery [139,142]. Studies in different clinical settings have broadened the spectrum of GBS; besides the post-infectious onset, the condition has been reported to occur postoperatively, secondary to systemic illnesses, and after specific interventions such as bone marrow transplantation [168-170]. Wider clinical recognition should lead to more insight into the pathophysiology of GBS. Animals and humans appear to become more susceptible to immune-mediated illnesses soon after the resolution of an infection, a phenomenon that raises important questions about the immune-regulatory events surrounding recovery from systemic processes and the specific niceties of diarrhea illnesses [171-172]. By identifying specific Human leukocyte antigen (HLA) haplotypes in patients with the Miller Fisher variant versus the axonal variant, some specific HLA alleles have been associated with GBS. In the Dutch population, multiple studies have associated the HLA-DQw1 allele with a risk of GBS. A focus on specific HLA alleles may disprove the longstanding hypothesis based on molecular mimicry; in this approach, immune cross-reactivity with minor differences

between the infectious agent and the neural ganglioside protein has been postulated [173,174].

Clinical profiles, management procedures, and prognostic outcomes were evaluated in Taiwan from 1998 to 2013. In this report, there were 1,017 patients from six different medical centers. Clinical features, body mass index (BMI), and prognostic factors were analyzed in laboratory, electrophysiology, and prognostic subgroups. Furthermore, a comparison with pediatric GBS patients was performed, wherein the results indicated that the occurrences surged in both 2006 and 2007, with mean annual incidence rates in the ranges of 1.11 and 1.37 per 100,000 person-years, respectively [175,176].

The GBS Foundation International Disability Scale can be used to differentiate the functional outcomes and is associated with older age, preceding diarrhea, and antecedent surgical operations. Frequent comorbidities include type 2 diabetes mellitus and infections [177,178]. The allergic reaction and irritable bowel syndrome showed a decreased risk of acute GBS. The estimated mean annual incidence of pediatric GBS was 0.35 per 100,000 persons, which is approximately one-third of the incidence and a lower percentage of adults in India [179].

The pneumococcal vaccine was the most frequent preceding event in Taiwanese children. Combined research consisted of two interlaced narratives containing abrupt outbreaks in either seasonality or individual age groups in Taiwan [180]. A thorough knowledge of geographical factors, seasons, comorbidities, and clinical phases labeled as complex GBS patients is beneficial for private practitioners or health policy administrators. Therapeutic data records were obtained on GBS in the Taiwanese population with the hope that potential developments can increase treatment benefits [181,182]. Moreover, few case studies are available, wherein a previously healthy 35-year-old woman experienced the onset of upper respiratory infection symptoms, which were promptly followed by right-sided weakness and incoordination. Electromyography supported a diagnosis of left facial diparesis, with severely reduced conduction velocities, conduction block, or slowing of conduction. Ambulation became increasingly complex, and the patient required assisted ventilation [183].

Plasma exchange therapy was instituted, and plasma IgG anti-ganglioside antibodies reactive against GMI, GM1b, or GD1a were detected. After a slow disease course, the patient improved and became functionally independent one year after onset. Furthermore, a previously healthy 38-year-old woman experienced the onset of diarrhea. Three days before admission, the patient developed muscle weakness in all extremities and rapidly progressive facial diplegia [184].

EMG findings of F wave prolongation, muscle denervation with reduced amplitude, and plasma anti-ganglioside antibodies suggested the diagnosis of acquired neuropathy. The patient's dyspnea and aspiration compelled the initiation of mechanical ventilation. Plasma exchange therapy was eventually employed. Plasma IgG anti-ganglioside antibodies reactive against GalCer and/or GalNac-GD1a were identified. With a slow recovery after prolonged intubation, the patient became fully functional one year after onset [185]. Despite the current improvements in diagnostic tools and the availability of information, it is still essential to continue to educate physicians at every level to recognize and understand the care of patients with peripheral neuropathy, myasthenia gravis, and GBS [186,187].

PATENTS

Many researchers have filed patents related to the diverse inventions for managing GBS. One of the inventions relates to methods of GBS treatment, more specifically, to methods involving the inhibition of the classical pathway of complement activation [188]. Another invention relates to the inhibition of the complement replacement pathway, especially factor B, in patients suffering from conditions and diseases associated with activation of the complement replacement pathway, such as age-related macular degeneration, diabetic retinopathy, and related ophthalmic diseases [189]. Moreover, methods of treating neuroinflammation and other disorders are reported, comprising administering to the subject an effective amount of a compound described, e.g., a SHP1 inhibitor or SHP2 inhibitor, or a pharmaceutical composition [190]. Additionally, one of the inventions discloses simple and efficient glycan- or carbohydrate-based processes or methods for the rapid identification of biological markers and therapeutic targets, especially glycan-related targets of infectious diseases, cancers, autoimmune diseases, allergies, inflammation, toxicity, obesity, and/or other disorders of humans, animals, plants, and other organisms. Therefore, novel methods and products for the diagnosis, prevention, and treatment of such diseases obtainable based on these therapeutic targets can be developed [191].

Recently, a patent was filed related to the invention based on the interaction of fibroblasts and immune cells and their use for activation for treating various conditions, including GBS [192].

FUTURE DIRECTIONS

Five areas of research are expected to provide valuable information on the causes of GBS, the reason for its increased incidence after certain infections, and the mechanisms of resistance to viral and bacterial infection by the PNS. First, the association of certain organisms with GBS suggests the involvement of an immune mechanism [193]. This association allows for the generation of specific murine monoclonal antibodies that may recognize autoantigens involved in GBS. Similar methods can be used in human studies. Second, evidence was presented that an antecedent respiratory tract infection is the most common factor preceding the development of the syndrome. Since other, more powerful experimental models typically use pathogens that preferentially infect the airways, the study of GBS may also use these disease models [194]. Third, as GBS is not only associated with infectious diseases, particularly the immune abnormalities of such diseases, a comprehensive comparison of these findings is warranted. Fourth, since an intravenous injection of certain neuroblastoma glycoproteins, these antibodies are commonly found in the serum [195].

These same antibodies are also recognized by antibodies secreted by certain organisms, although it was not established if these are the same antibodies. Creation of long-term clonal lines of these B cells may identify molecules that control the creation of these antibodies and thereby prevent induction of the post-infection autoimmune state and GBS. Finally, long-term clonal lines of certain conditions and a more rapid onset of mechanical ventilation seem to avoid the induction of the post-infection autoimmune state during a true breathing challenge. Future laboratory studies should be directed toward the long-term effects following respiratory viral infection in collaboration with researchers who study pneumonia [196-198].

CONCLUSION

Unfortunately, there’s no surefire way to stop GBS from occurring. Although the precise cause remains elusive, it’s thought to be triggered by an immune response following an infection. GBS is a potentially life-threatening cause of progressive peripheral paralysis. GBS can be asymptomatic, meaning that a patient can have zero symptoms; whereas other patients may have mild cold or flu-like symptoms, followed by severe paralysis with respiratory failure; and some patients may have difficulty swallowing or breathing. A variety of sensorimotor symptoms leading to variable disability are the hallmark of the condition, wherein the progression of disability during the first few weeks to a few months of symptoms is the most critical parameter in clinical evaluation and prognosis. That said, maintaining good overall health can be beneficial, as it may reduce the likelihood of infections that sometimes precede GBS. Simple things like practicing good hygiene, living a healthy lifestyle, staying up to date with vaccinations, and seeking prompt treatment for infections can help lower the risk. If any symptoms arise, seeking immediate medical attention is necessary. Early intervention plays a significant role in improving the chances of recovery. While many people do recover fully, the process can be difficult. Support, both physical and emotional, is critical. Regaining independence often requires a lot of patience, persistence, and regular physical therapy.

FINANCIAL ASSISTANCE

NIL

CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTION

Sarika J. Patil conceptualized the article's original idea. Rohit R. Bhosale and Dhanashri D. Chavan contributed to the data compilation and manuscript preparation. Akshay R. Yadav and Swapnil S. Patil performed the literature review. All authors have read and approved the final manuscript.

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