International Journal of Community Medicine and Public Health
Ali BM et al. Int J Community Med Public Health. 2023 Dec;10(12):5038-5043
http://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20233808

Review Article

Guillain-Barré syndrome: a review of immunotherapy, rehabilitation, and long-term outcomes

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Received: 16 November 2023
Accepted: 20 November 2023

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ABSTRACT

Guillain-Barré syndrome (GBS) is a complex autoimmune disorder characterized by the rapid onset of motor weakness and sensory disturbances, often precipitated by preceding infections. GBS is considered a rare disorder, with an estimated annual incidence ranging from 0.6 to 4.0 cases per 100,000 individuals. It constitutes a medical emergency due to its potential for severe disability or mortality. The hallmark clinical features of GBS include progressive symmetric muscle weakness and sensory disturbances. Diagnosis relies on clinical evaluation, cerebrospinal fluid analysis, and electrodiagnostic studies, with the Brighton criteria providing standardized diagnostic criteria. The hallmark clinical features include progressive symmetric muscle weakness, sensory deficits, and areflexia or hyporeflexia. The management of GBS primarily involves supportive care and immunomodulatory therapies. Plasmapheresis, or plasma exchange, and intravenous immunoglobulin (IVIG) are the mainstay treatments aimed at modulating the immune response and shortening the illness duration. Rehabilitation, encompassing physical and occupational therapy, is pivotal for restoring functionality and improving long-term outcomes.

Keywords: Guillain-Barré syndrome, Autoimmune disorder, Immunotherapy, Plasmapheresis, Intravenous immunoglobulin, Rehabilitation, Autoimmune response, Neuropathy

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute, immune-mediated neuropathy characterized by the rapid onset of ascending motor weakness and sensory deficits. GBS is considered a medical emergency, as it can lead to severe disability or even death if not promptly diagnosed and treated. The hallmark clinical features of GBS include progressive symmetric muscle weakness and sensory disturbances.[1] The weakness typically begins in the lower extremities and ascends to involve the upper limbs and, in severe cases, respiratory muscles. Patients often report paresthesias, such as tingling or numbness, which may precede weakness. GBS may also manifest as areflexia or hyporeflexia, with absent or diminished deep tendon reflexes.[2] Autonomic dysfunction, such as fluctuations in

blood pressure and heart rate, may complicate the clinical picture.

GBS is an autoimmune disorder believed to result from molecular mimicry, where an immune response to an infectious agent cross-reacts with components of peripheral nerve myelin.[3] The exact trigger varies among individuals, with preceding infections commonly associated with GBS, including *Campylobacter jejuni*, Epstein-Barr virus, and cytomegalovirus. Immune-mediated destruction of the myelin sheath and, in some cases, axonal injury lead to impaired nerve conduction and clinical deficits. The presence of anti-ganglioside antibodies further supports the immune-mediated nature of GBS.

The diagnosis of GBS relies on clinical evaluation, cerebrospinal fluid (CSF) analysis, and electrodiagnostic studies.[4] The Brighton Criteria, which stratifies diagnostic certainty into levels 1-3, provides a standardized approach to diagnosis. CSF analysis typically reveals elevated protein levels with a normal white blood cell count. Electrodiagnostic studies, such as nerve conduction studies and electromyography, help confirm the presence of peripheral nerve abnormalities and distinguish GBS from other neuropathies.

GBS is considered a rare disorder, with an estimated annual incidence ranging from 0.6 to 4.0 cases per 100,000 individuals.[5] While GBS can affect individuals of all ages, it exhibits a bimodal age distribution, with peaks in children and adults over 50 years old.[6] Male preponderance has been observed in some studies. Notably, GBS may follow various infections, and geographical variations in its incidence have been reported.

Management of GBS primarily involves supportive care and immunomodulatory therapies. Plasmapheresis and intravenous immunoglobulin (IVIG) are the mainstay treatments that aim to modulate the immune response and shorten the duration of illness.[7] Additionally, respiratory support may be required in cases of severe weakness affecting the respiratory muscles. Physical therapy plays a crucial role in the rehabilitation phase, helping patients regain strength and functionality. Recent research in GBS has shed light on the underlying immunopathogenic mechanisms, including the role of specific autoantibodies and the gut microbiota.[8,9] Advances in supportive care and immunomodulatory treatments have improved patient outcomes, emphasizing the importance of early diagnosis and intervention. Furthermore, ongoing research is exploring novel therapeutic targets and potential biomarkers for GBS.

METHODOLOGY

This study is based on a comprehensive literature search conducted on 17 October 2023, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed immunotherapy, rehabilitation, and long-term outcomes in relation to Guillain-Barré syndrome. There were no restrictions on date, language, participant age, or type of publication.

DISCUSSION

GBS is an autoimmune disorder characterized by peripheral nerve demyelination or axonal injury, leading to muscle weakness and sensory disturbances. The immune system's aberrant response to various antecedent infections is thought to be the primary trigger for GBS.[8] Immunotherapy plays a crucial role in managing GBS by modulating the immune response, mitigating the disease's severity, and accelerating recovery.[10] The rationale for immunotherapy in GBS lies in its autoimmune nature, where the immune system erroneously targets peripheral nerve components. By reducing or modulating this immune response, immunotherapy aims to attenuate the progression of the disease, limit nerve damage, and expedite recovery. Immunotherapy is particularly vital in severe GBS cases, where rapid intervention can prevent respiratory compromise and improve long-term outcomes.

Plasmapheresis, also known as plasma exchange, is an established immunotherapy modality for GBS.[11] It involves removing the patient's plasma, which contains pathogenic antibodies and immune complexes, and replacing it with donor plasma or a plasma substitute. Plasmapheresis is thought to work by removing detrimental antibodies and inflammatory mediators while providing essential factors for nerve regeneration. Multiple studies have demonstrated its efficacy in reducing disability and shortening the duration of GBS symptoms.[12,13] This includes a randomized controlled trial with 245 GBS patients, which confirmed plasma exchange as an effective therapy.[13] Plasma exchange emerged as the established standard of care for treating GBS and gained endorsement from the American Academy of Neurology guidelines. These guidelines recommend plasma exchange for GBS patients who are unable to walk within 4 weeks of symptom onset for non-ambulatory patients and within 2 weeks for ambulatory patients. The treatment typically involves a series of five exchanges, replacing one plasma volume over 1 to 2 weeks. For milder cases, two exchanges may suffice, while moderate or severe cases may require a minimum of four exchanges.[12,14] Plasma exchange has demonstrated its effectiveness in reducing the need for mechanical ventilation, expediting the ability to walk with assistance, and enhancing long-term muscle strength recovery.[15] Notably, continuous flow machines and the use of albumin as an exchange fluid have proven to be effective methods.

The exact mechanism of action behind plasma exchange remains unclear, but it is believed to remove harmful antibodies, immune complexes, complement factors, cytokines, and other pro-inflammatory substances that contribute to GBS.[16] This process reduces demyelination or axonal injury and facilitates clinical recovery.[17] However, it is important to note that plasma exchange is associated with significant side effects, including hemodynamic instability, coagulation problems, hypocalcemia, septicemia, thrombosis, pneumonia, complications related to central venous access, and allergic reactions.[18] The infusion of citrate in the exchange fluid can lead to metabolic acidosis or hypocalcemia. Relative contraindications include hemostatic disorders, unstable cardiovascular status, active infections, and pregnancy.[19] Limited access to specialized equipment and expertise, the need for close monitoring, and potential adverse effects have limited the widespread use of plasma exchange for GBS. Additionally, extended hospitalization and associated higher costs have further restricted its utilization.

IVIg is a well-established immunotherapy for GBS, derived from pooled purified immunoglobulins obtained from numerous blood donors. The first randomized controlled trial (RCT) of IVIg for GBS confirmed its efficacy, comparable to PE, in 1992.[20] The American Academy of Neurology recommended IVIg for GBS patients needing assistance to walk within 2 weeks (Level A, Class I evidence) or 4 weeks (Level B recommendation) of symptom onset.[17] IVIg therapy has been observed to expedite recovery in children compared to supportive therapy, based on limited evidence from three open-label trials. The typical IVIg dosage for GBS is 0.4 grams per kilogram of body weight daily for five consecutive days, adding up to 2 grams per kilogram.[21] Administering the total dose over 2 days is equally effective. Research has indicated that patients with higher increases in serum IgG levels after IVIg treatment, particularly at two weeks post-treatment, are more likely to achieve independent ambulation after 6 months. This suggests that a higher dose or a second course of IVIg may be beneficial for patients with lower serum IgG increases.[22] The exact mechanism of IVIg action in GBS remains unclear, but it is believed to modulate the immune system in various ways, including suppressing autoantibody production, inhibiting complement activation, and affecting Fc receptor expression and T-cell functions.[23-25] The overall effect is thought to reduce nerve damage and expedite recovery. It is important to note that IVIg is not a standardized drug, and its composition may vary depending on the manufacturer and donor source. Adverse events associated with IVIg are typically minor and rare, occurring in less than 10% of GBS patients. Severe reactions reported include myocardial infarction, renal failure, vomiting, and headaches.[17,20] Conditions such as elevated serum viscosity, high triglycerides, or hypergammaglobulinemia are relative contraindications due to an increased risk of thromboembolic events.[23,26] IVIg should be used cautiously in patients with specific medical conditions, and careful monitoring can help mitigate the risks associated with its use.

Therefore, despite the proven efficacy of plasmapheresis and IVIG, challenges in optimizing immunotherapy for GBS persist. These challenges include determining the appropriate timing of treatment, addressing variability in patient responses, and managing potential adverse effects.[27] Emerging strategies aim to tackle these issues, such as identifying predictive biomarkers to guide treatment decisions and developing targeted immunotherapies designed to modulate specific immune pathways implicated in GBS. One potential therapy under investigation is Eculizumab, a monoclonal antibody that targets complement component 5 (C5) and inhibits the production of pro-inflammatory factors associated with nerve damage in GBS[28,29] Clinical trials in the UK and Japan are assessing its efficacy, guided by promising results from animal studies. Several other biologic agents are also being considered as potential future GBS therapies, drawing from data from animal models and small case reports.[30] Given the differing disease mechanisms between GBS variants involving nerve demyelination and those involving nerve damage, future clinical trials may involve randomization based on electrophysiologic characteristics or subgroup analyses, considering factors like variant prevalence and disease severity measures.

While immunotherapy addresses the underlying autoimmune response, rehabilitation plays an integral role in GBS management by addressing the functional impairments and disabilities that result from nerve damage. Firstly, physical therapy is a cornerstone of GBS rehabilitation, focusing on restoring and maintaining muscle strength, range of motion, and functional mobility.[31] Early mobilization is critical to prevent contractures and muscle atrophy, as GBS patients are often immobilized during the acute phase of the illness.[32] Therapists employ a range of exercises, gait training, and assistive devices to help patients regain independence and optimize their physical function. Further, occupational therapy addresses the challenges GBS patients may face in performing activities of daily living (ADLs) and vocational tasks. Therapists work to improve fine motor skills, hand dexterity, and adaptive techniques to enhance patients' autonomy. Home modifications and assistive devices are often recommended to facilitate independent living. Furthermore, respiratory dysfunction can be a life-threatening complication of GBS, particularly in cases of severe muscle weakness involving the diaphragm. Respiratory therapists play a crucial role in monitoring respiratory function, providing ventilatory support when necessary, and implementing strategies to prevent complications such as pneumonia.[33] Breathing exercises and pulmonary hygiene techniques are also employed to optimize lung function.

Interdisciplinary collaboration is fundamental in GBS rehabilitation. A team of healthcare professionals,

including physical therapists, occupational therapists, respiratory therapists, nurses, and physicians, work together to develop individualized care plans that address the diverse needs of GBS patients.[34] Regular assessments and communication among team members ensure comprehensive care and timely adjustments to treatment plans. Further, rehabilitation for GBS patients extends beyond physical and functional aspects. Psychosocial support, including counseling and psychological interventions, plays a crucial role in addressing the emotional and mental health challenges that often accompany GBS. Coping strategies, stress management, and support groups can help patients and their families navigate the psychological impact of the disease.[35] The ultimate goal of rehabilitation for GBS patients is to optimize long-term outcomes. This involves not only restoring physical function but also promoting community reintegration and enhancing quality of life. Rehabilitation efforts may continue for several months or even years, adapting to the evolving needs of the patient.

While the focus of GBS management typically centers on acute treatment and stabilization, understanding the long-term outcomes is essential for both patients and healthcare providers. Recovery in GBS is highly variable and influenced by several factors. Early intervention with immunotherapy, such as plasmapheresis and intravenous immunoglobulin (IVIG), has been associated with improved outcomes. The initial severity of muscle weakness, subtype of GBS (e.g., acute inflammatory demyelinating polyneuropathy, axonal variants), age, and underlying medical conditions also play critical roles in determining the extent and rate of recovery.[36] While many GBS patients experience significant improvement in muscle strength and function, a substantial proportion continues to grapple with persistent disabilities. These disabilities may manifest as residual weakness, sensory deficits, pain, fatigue, and difficulties with activities of daily living.[37] Some patients may require ongoing physical or occupational therapy to manage these challenges.[38]

GBS can have a profound impact on patients' quality of life, even in cases where physical function partially or fully recovers. Chronic pain, residual weakness, and sensory disturbances can lead to decreased mobility, limited social participation, and psychological distress. Addressing the psychosocial aspects of GBS recovery is crucial in improving the overall well-being of affected individuals. Recent research in GBS has shed light on potential strategies to improve long-term outcomes. This includes investigations into the role of neurotrophic factors, such as brain-derived neurotrophic factor (BDNF), in nerve regeneration and functional recovery.[39,40] Additionally, efforts to identify predictive biomarkers for long-term prognosis may aid in tailoring treatment and rehabilitation plans to individual patients.[41] Long-term management of GBS often involves ongoing rehabilitation and support. Physical therapy, occupational therapy, and assistive devices can help patients maximize their functional abilities and adapt to persistent disabilities. Psychological support and counseling can address the emotional challenges associated with living with GBS-related disabilities.

CONCLUSION

GBS presents a significant medical challenge, demanding early recognition and intervention. Immunotherapies like plasmapheresis and IVIG have proven effective, but optimizing treatment remains a challenge. Rehabilitation plays a pivotal role in long-term recovery. Research into prognostic factors and emerging therapies is ongoing, offering hope for improved GBS management. Comprehensive, interdisciplinary care is key to maximizing outcomes and enhancing the quality of life for GBS patients.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Ali BM, Alghamdi AA, Alanazi WR, Alsamti AA, Allahyani AF, Altalhi MM, et al. Guillain-Barré syndrome: a review of immunotherapy, rehabilitation, and long-term outcomes. Int J Community Med Public Health 2023;10:5038-43.