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Acute Inflammatory Demyelinating Polyneuropathy: a comprehensive literature review on Guillain-Barré Syndrome

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Summary

Introduction and purpose

Guillain-Barré Syndrome (GBS) is an acute, immune-mediated polyneuropathy characterized by rapid-onset muscle weakness and areflexia. It typically follows an infection and can lead to severe complications, such as respiratory failure or autonomic dysfunction. It is a significant concern in neurology and critical care due to its unpredictable course and potential to cause long-term disability. This article aims to present a comprehensive review of Guillain-Barré Syndrome, focusing on its key clinical and prognostic aspects, according to current literature.

Description of the state of knowledge

Guillain-Barré Syndrome is a complex condition with diverse clinical presentations and pathophysiological mechanisms. It is widely recognized as an immune-mediated disorder, often triggered by infections such as *Campylobacter jejuni* or cytomegalovirus. The key pathological process involves molecular mimicry, leading to an autoimmune attack on peripheral nerves, resulting in demyelination or axonal damage. Immunotherapy with intravenous immunoglobulin (IVIG) or plasma exchange (PE) is the foundation of treatment, while supportive care is essential for managing respiratory failure and autonomic dysfunction.

Conclusions

GBS remains a complex condition requiring rapid diagnosis and management. While immunotherapy significantly improves recovery, challenges persist, particularly in the most severe cases. Further research into pathophysiological mechanisms and experimental therapies is essential to refine treatment approaches and improve patient outcomes.

Key words

Guillain-Barre Syndrome; Acute Inflammatory Demyelinating Polyneuropathy; Immunotherapy

Introduction and purpose

Guillain-Barré Syndrome is a complex immune-mediated neurological disorder characterized by acute, progressive peripheral nerve demyelination and/or axonal damage, leading to paralysis and hyporeflexia. Although its exact etiology is not fully understood, GBS often follows infections such as *Campylobacter jejuni*, Epstein-Barr virus, cytomegalovirus, or Zika virus, which trigger abnormal immune responses.

These responses involve molecular mimicry, in which antibodies generated against pathogens cross-react with peripheral nerve gangliosides, disrupting normal nerve function. Variants of GBS include the classic acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), Miller-Fisher syndrome (MFS), and Acute Motor-Sensory Axonal Neuropathy (AMSAN) and each of these variants has different clinical and electrophysiological patterns.[1,2]

From an epidemiological perspective, Guillain-Barré Syndrome affects individuals across all age groups, with a median incidence of approximately 1 per 100,000 people per year. The risk increases significantly with age, rising by about 20% for every additional decade. Males exhibit a slightly higher susceptibility compared to females. Nearly 30% of patients may need mechanical ventilation.[3] The diagnosis should be established as early as possible, relying on the presence of characteristic findings such as albuminocytologic dissociation in the cerebrospinal fluid and reduced nerve conduction velocities. The current therapeutic options, including intravenous immunoglobulin (IVIG) and plasma exchange (PE), have proven effective in accelerating recovery. However, despite their efficacy new interventions are continuously being looked for to further optimize treatment strategies and improve patient outcomes. [4,5]

These aspects make Guillain-Barré Syndrome an important subject for advancing the understanding of immune-neurological interactions and optimizing clinical care.[6]

The purpose of this article is to provide an overview of GBS, including its epidemiology, pathophysiology, clinical presentation, diagnostic criteria, and treatment options, while highlighting key aspects of prognosis and patient outcomes.

Materials and methods

This literature review on Guillain-Barré Syndrome utilized peer-reviewed articles published between 1988 and 2024, sourced from databases including PubMed, Scopus, and Web of Science. The review focused on studies addressing the pathophysiology, clinical presentation,

diagnosis, treatment, and prognosis of GBS. A total of 35 articles were analyzed to provide a comprehensive synthesis of current knowledge.

Description of the state of knowledge

Medical history

The history of Guillain-Barré Syndrome diagnosis dates back to the early 20th century when Georges Guillain, Jean Alexandre Barré, and André Strohl described two soldiers with acute areflexic paralysis, emphasizing the role of cerebrospinal fluid (CSF) albuminocytologic dissociation without cellular response. This landmark discovery provided the diagnostic foundation for what is now recognized as a spectrum of disorders affecting the peripheral nervous system. Over time, electrophysiological studies have clarified the subtypes of GBS, including Acute Inflammatory Demyelinating Polyneuropathy (AIDP) and Acute Motor Axonal Neuropathy (AMAN). AIDP is characterized by immune-mediated demyelination, which leads to slowed nerve conduction and progressive muscle weakness. In contrast, AMAN primarily affects motor axons, resulting in rapid and severe limb weakness without significant sensory involvement. These distinctions have facilitated more precise diagnostic approaches.[7,8]

In addition to AIDP and AMAN, Miller-Fisher Syndrome (MFS) was identified as a distinct variant, characterized by the triad of ophthalmoplegia, ataxia, and areflexia, and strongly associated with anti-GQ1b antibodies. [9] Another less common subtype, Acute Motor-Sensory Axonal Neuropathy (AMSAN), involves severe axonal damage affecting both motor and sensory nerves, leading to a prolonged recovery process.[8] The refinement of diagnostic criteria through advances in imaging, immunology, and the detection of antiganglioside antibodies has deepened our understanding of the autoimmune mechanisms underlying these variants.[10]

Epidemiology and risk factors

Guillain-Barré Syndrome is a disease characterized by an overall incidence rate of approximately 1 to 2 cases per 100,000 people yearly worldwide. The risk increases significantly with age, rising by about 20% for every additional decade, and males exhibit a slightly higher susceptibility compared to females. Almost 30% of patients require mechanical ventilation, which is associated with a mortality rate of 5%–12%, slower recovery, and an increased risk of residual disability.[3] In approximately two-thirds of cases, the onset

is preceded by an infection, often a mild and unrecognized respiratory or gastrointestinal illness. Infectious agents such as *Campylobacter jejuni*, cytomegalovirus, and Zika virus are frequently identified as key triggers, particularly in regions where these pathogens are endemic. These associations underline the critical role of infections in the pathogenesis of GBS. [1,2,11]

Genetic predisposition also plays a role, with specific HLA haplotypes associated with heightened susceptibility.[12]

Epidemiological surveillance and a deeper understanding of these risk factors are essential for improving early diagnosis, guiding targeted prevention strategies, and optimizing management, particularly in high-risk and susceptible regions.[13]

Clinical presentation and diagnostic criteria

Guillain-Barré Syndrome is an acute-onset, immune-mediated polyneuropathy characterized by symmetrical muscle weakness and areflexia, typically progressing in an ascending pattern from the lower extremities.[14] Patients typically report initial symptoms such as paresthesia and numbness in the hands and feet, weakness, and limb pain, which progressively worsen over time. Respiratory involvement may necessitate mechanical ventilation, making early recognition of ventilatory impairment crucial.[15,16]

Pain is a prevalent and significant symptom in GBS, reported in up to 89% of patients at various stages of the disease. It commonly presents as deep, aching discomfort affecting the back, lower and upper limbs, often intensifying during the acute phase. Pain severity is correlated with disease progression and may persist for months, adversely impacting the quality of life.[17,18]

Cranial nerve involvement, such as facial palsy and bulbar dysfunction, is another common manifestation of Guillain-Barré Syndrome, often leading to difficulties with swallowing and speech. Additionally, approximately two-thirds of patients experience autonomic dysfunction of varying degree, which may present as tachycardia, blood pressure instability, or gastrointestinal issues, further complicating the disease course. [14,19]

Guillain-Barré Syndrome diagnosis involves clinical evaluation, cerebrospinal fluid (CSF) analysis, and electrophysiological studies. The characteristic clinical features include symmetrical progressive weakness and areflexia, typically reaching their maximum severity within four weeks. Autonomic dysfunction, such as cardiac arrhythmias and blood pressure instability, further supports the diagnosis.[20]

The common diagnostic features include albuminocytologic dissociation in the cerebrospinal fluid, characterized by an elevated protein level with a normal white cell count, as well as slowed nerve conduction velocities observed in electrophysiological studies. Early recognition of these clinical and diagnostic features dictates timely therapeutic intervention and reduces complications.[21,22]

The Brighton criteria provide a structured approach to diagnosis by integrating clinical features, CSF findings, and electrophysiological data. As part of differential diagnosis, infectious diseases, malignancies, and disorders of the neuromuscular junction, must be excluded to ensure an accurate diagnosis and appropriate treatment.[14,23]

Pathophysiology

The pathophysiology of Guillain-Barré Syndrome involves immune-mediated damage to peripheral nerves, typically occurring after an infection. *Campylobacter jejuni* has been considered the most common bacterial pathogen preceding its onset. However, other infections, including viral agents such as Epstein-Barr virus, cytomegalovirus, and more recently SARS-CoV-2, may also induce GBS.[14,15,24]

The autoimmune response is essentially driven by molecular mimicry. This occurs when the immune system mistakenly identifies myelin sheath components or axonal elements of peripheral nerves as foreign after recognizing similar structures from the surface of an infectious agent. Molecular mimicry between bacterial or viral antigens and components of nerves, particularly gangliosides on the surface of peripheral nerves, triggers immune activation. This process involves the activation of T cells and the production of antibodies.[25,26] The autoimmune response targets the myelin sheath, leading to demyelination, which impairs nerve conduction and results in the characteristic symptoms of weakness, sensory loss, and in severe cases, paralysis. Sometimes, it also damages the axons themselves, leading to axonal GBS, which can result in more severe and longer-lasting neurological deficits.[15]

Treatment

The primary aims of treatment are to suppress the autoimmune response and to support the patient through the acute phase of illness. Immunotherapy is the foundation of GBS management, with intravenous immunoglobulin (IVIG) and plasma exchange (PE) being the two primary therapeutic options. IVIG, administered at a dose of 0.4 g/kg/day for five days, works by neutralizing autoantibodies and inhibiting the activation of complement, leading to

reduced nerve damage and improved recovery. Plasma exchange functions by removing pathogenic antibodies and immune complexes, which reduces the autoimmune attack on peripheral nerves and mitigates inflammation. This therapy is typically conducted in five sessions over 14 days and is particularly effective in patients with severe or rapidly progressing symptoms. Both are most effective when initiated within the first two weeks of symptom onset and seem equally effective in decreasing symptom severity and duration.[15,27]

Supportive care is critical in managing complications, particularly weakness of the respiratory muscles, which affects approximately 20–30% of patients and requires mechanical ventilation.[16,28] Another significant complication is autonomic dysfunction, observed in nearly two-thirds of patients. Continuous monitoring for cardiac arrhythmias and blood pressure fluctuations is necessary, as these manifestations can lead to life-threatening events. Beyond cardiovascular symptoms, autonomic dysfunction in GBS often manifests as thermoregulatory abnormalities, leading to excessive sweating or anhidrosis, and gastrointestinal disturbances, resulting in reduced motility, constipation, or diarrhea. These systemic manifestations of autonomic involvement underscore the importance of comprehensive supportive care and vigilant monitoring throughout the course of the disease.[19]

Pain management using neuropathic pain agents, such as gabapentin or pregabalin, is vital for enhancing patients' comfort. Additionally, early mobilization and physical therapy during the recovery phase enhance functional outcomes and reduce the risk of long-term disability.[29,30,31]

Corticosteroids have historically been used in the treatment of GBS, but current evidence indicates they are ineffective and are no longer recommended as a standard therapy.[32]

Prognosis

Negative prognostic factors in Guillain–Barré Syndrome include advanced age (>60 years), rapid progression of weakness within seven days of symptom onset, severe weakness at admission, need for mechanical ventilation, preceding diarrhea, and severe neuropathy evident on electrophysiological studies.[1]

The prognosis presents a complex recovery process, with most patients initiating improvement within 28 days and achieving near-complete recovery within 200 days in 80% of cases. However, 65% experience minor residual symptoms, such as sensory disturbances or mild motor deficits.[33] Permanent disabling weakness, imbalance, or sensory deficits are

reported in 5–10 percent of patients. Mortality rates range from 3–8%, often due to complications like sepsis or respiratory failure, predominantly in older patients.[34]

GBS is associated with a range of psychological complications that significantly impact patients' quality of life, both during the acute phase and throughout the recovery period. Fatigue, which affects up to 60% of patients, is a significant challenge, particularly during recovery, contributing to physical and mental strain.[35]

Conclusions

Guillain-Barré Syndrome remains a complex neurological disorder, characterized by acute immune-mediated damage to the peripheral nervous system. This condition presents with a wide spectrum of clinical manifestations, including ascending paralysis, sensory disturbances, and autonomic dysfunction. Advances in immunotherapy, supportive care, and diagnostic techniques have significantly improved patient outcomes, but the disease continues to pose challenges due to its complex pathophysiology and potential for severe complications.

The prognosis for GBS is generally favorable, with most patients achieving substantial recovery. However, residual deficits persist in some cases, highlighting the importance of early intervention and multidisciplinary care, including physical therapy and rehabilitation.

Early and accurate diagnosis, supported by clinical, cerebrospinal fluid, and electrophysiological findings, is crucial to initiating timely immunotherapy with intravenous immunoglobulin or plasma exchange. Further research into novel therapeutic strategies and better predictive tools is essential to optimize care and enhance recovery in affected individuals.

Disclosure

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