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*Case Report*

# Clinical Repercussions of Guillain-Barré Syndrome: A Case Study and Systematic Review

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**Abstract:** Guillain-Barré Syndrome (GBS) is part of a heterogeneous group of immune-mediated polyneuropathies characterized by segmental demyelination accompanied by mononuclear infiltrates in the endoneural tissue and myelin sheaths. It is a rare neurological disorder that primarily affects adults. Its origin is often post-infectious, triggered by an autoimmune response against the peripheral nervous system, which leads to the classic triad: paresthesia, progressive and ascending muscle weakness with areflexia, and, in severe cases, paralysis. Its onset is related to previous infections, often caused by *Campylobacter jejuni*, cytomegalovirus, or the Epstein-Barr virus. The diagnosis is generally straightforward and is determined through a complete medical history, a neurological examination focusing on identifying the triad, and complementary tests such as electromyography and cerebrospinal fluid analysis. Within days, the cerebrospinal fluid often shows elevated protein levels, while the cellular content remains largely unchanged, resulting in albuminocytologic dissociation. Treatment includes respiratory support in severe cases, intravenous immunoglobulin, or plasmapheresis. Recovery depends on several factors and may take weeks or even months. This study presents the case of a 46-year-old man who developed GBS following a prior infection. The case highlights the diagnostic challenges, clinical progression, and the implementation of treatment.

**Keywords:** Guillain-Barre Syndrome; Clinical Diagnosis; Immunotherapy.

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## 1. Introduction

Guillain-Barré Disorder (GBS) is an intense immune-mediated polyradiculoneuropathy characterized by dynamic extreme shortcomings and, in a few cases, total loss of motion. The pathogenesis of GBS is complex, including numerous immune mechanisms that influence peripheral nerves. GBS is categorized into subtypes based on its pathological features: acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute motor and sensory axonal neuropathy (AMSAN) [1]. The worldwide frequency of GBS shifts from 0.5 to 2 cases per 100,000 person-years, with a higher prevalence in older adults, especially those over 80 years of age, where the rate reaches 2.7 cases per 100,000 person-years. In children under two years of age, it is significantly rarer, with a reported frequency of around 0.6 per 100,000 children. Epidemiological data suggest a higher predisposition among males, with a male-to-female ratio

ranging from 1.1:1 to 2:1 [2]. In Brazil, an estimated annual rate of 0.6 per 100,000 individuals was reported between 1995 and 2002. Seasonal and geographical variations have also been observed, with certain outbreaks linked to preceding infectious epidemics, such as the Zika virus epidemic in Latin America [3].

The key pathological mechanism of AIDP involves antibody- and complement-mediated demyelination, leading to the destruction of myelin and macrophage activation. In axonal subtypes such as AMAN and AMSAN, anti-ganglioside antibodies, such as GM1 and GD1a, play a fundamental role by directly attacking axons and Schwann cell membranes [4]. One of the central pathological mechanisms in GBS is the interaction between antibodies and antigens in peripheral nerves, resulting in complement-mediated damage. Anti-ganglioside antibodies are critical pathogenic factors but are not present in all patients. Nodal dysfunction, or "nodopathy," occurs when an immune response affects the axonal sheath at the nodes, causing reversible conduction damage, axonal degeneration, or segmental demyelination [5]. Additionally, GBS can be triggered by a preceding infection, such as Campylobacter jejuni, which induces a cross-reactive immune response to gangliosides in the nerve through molecular mimicry. Other common infectious agents include cytomegalovirus, Epstein-Barr virus, and influenza virus. Epidemiological studies have also linked GBS outbreaks to emerging viral diseases, including SARS-CoV-2 and Zika virus [6].

Classically, GBS presents ascending weakness, accompanied by sensory deficits and areflexia, with rapid progression over days or weeks. Neurological symptoms are commonly preceded by respiratory or gastrointestinal infections. Diagnosis is primarily clinical and supported by cerebrospinal fluid examination, which typically reveals albuminocytologic dissociation, and electrodiagnostic studies showing characteristics of demyelination or axonal involvement [7]. This case report describes a 46-year-old male who initially presented with nonspecific symptoms such as fever, rash, and arthralgia, which resolved spontaneously within a few days. After a latency period of approximately 15 days, the patient developed subacute neurological symptoms, including paresthesias, progressive weakness, and areflexia, with a sensorimotor distribution consistent with a "stocking and glove" pattern. The clinical diagnosis of GBS was supported by laboratory findings of albuminocytologic dissociation in cerebrospinal fluid (CSF) [8].

The case illustrates the typical clinical progression of GBS and underscores the importance of a detailed evaluation for the differential diagnosis of polyradiculoneuropathies. It also highlights the need for early therapeutic interventions, including plasmapheresis or intravenous immunoglobulin (IVIG). This report further addresses the functional impact of GBS, the importance of multidisciplinary management, and the critical role of psychological support in the patient's recovery [9].

## 2. Materials and methods

This is a descriptive and analytical study based on the analysis of a clinical case involving a 46-year-old male patient with subacute neurological symptoms. The case was initially managed at a Health Center and later referred to a tertiary hospital with a Neurology service. Information was obtained through a detailed anamnesis, including the history of initial symptoms and their temporal progression, pre-existing conditions, occupational history, and exposure to risk factors. An assessment of potential recent infections or medication use was also conducted.

A comprehensive neurological examination was performed, with an emphasis on assessing muscle strength (using the MRC scale), deep tendon reflexes (classified according to intensity), tactile and pain sensitivity (focusing on the "stocking and glove" pattern), and the identification of autonomic signs. More sensitive and specific tests were conducted based on the diagnostic hypothesis, and clinical and laboratory findings were correlated, confirming the diagnosis of Guillain-Barré Syndrome.

In addition to presenting the clinical case report, this study integrates a systematic review of the available scientific literature, following the PRISMA protocol guidelines

(Preferred Reporting Items for Systematic Reviews and Meta-Analyses) (Figure 1). The research was conducted in the PubMed, SciELO, and BVS databases. Using the Boolean operators AND and OR, the search included the terms "Guillain-Barré Syndrome", "Clinical Diagnosis", and "Immunotherapy" in various combinations, prioritizing studies focused on Guillain-Barré Syndrome (GBS), particularly its clinical manifestations, diagnostic criteria, treatments, and prognosis.

The inclusion criteria selected for this review encompassed original studies, systematic reviews, case reports, and case series published in the last 15 years. Only full-text publications in English, Spanish, and Portuguese were considered. Conversely, studies that did not present clinical data or that were limited to experimental aspects without direct implications for clinical practice were excluded. Additionally, duplicate publications or those with insufficient methodological quality, as determined by a critical evaluation, were not included.

This study adhered to the ethical principles of the Declaration of Helsinki, and informed consent was obtained from the patient for the use of clinical data in the case report.

## 3. Case Report

A 46-year-old man arrived at the hospital's emergency department after experiencing symptoms for two days. He initially presented with mild, nonspecific pain in his thighs and legs, accompanied by tingling paresthesias in his feet and slight difficulty walking due to decreased distal strength in his lower extremities. He denied any recent infections or pathological history, with the only notable medical history being a case of gouty arthritis diagnosed and treated five years ago.

On physical examination, the patient had preserved muscle strength except for mild weakness at the distal level of his lower limbs, which caused slight difficulty walking. Osteotendinous reflexes were normal in all four extremities, cranial nerves were intact, and a bilateral Lasegue sign was positive (+). The initial diagnostic considerations included radicular syndrome and an unspecified myopathy. Consequently, a Creatine Phosphokinase (CPK) test was ordered, which returned normal results (18.9 IU/L), along with a complete blood count and serum electrolytes, which were also within normal limits. The patient was prescribed analgesics and anxiolytics and was discharged with instructions for reevaluation if needed.

After three months, the patient returned to the emergency department of the same hospital with a slight worsening of symptoms, including increased difficulty walking and the acute onset of right-sided peripheral facial paralysis. Muscle strength in the lower limbs was graded as 4/5 in the thighs and legs and 3/5 in the feet. Osteotendinous reflexes remained normal, except for a slightly decreased Achilles reflex. A lumbar puncture was performed, revealing albuminocytologic dissociation in the cerebrospinal fluid (Table 1). Based on these findings, the patient was diagnosed with acute demyelinating polyneuropathy or atypical Guillain-Barré Syndrome and was hospitalized for specific treatment with intravenous immunoglobulin (IVIG) for five days.

**Table 1.** Results of the cerebrospinal fluid examination performed on the day of admission to hospital. Albumino-cytological dissociation is observed.

| Cerebrospinal Fluid Examination | Exam Result |
|---|---|
| Cell count | 0 to 2 $mm^3$ |
| Glucose | 74 mg/dL |
| Total protein | 110 mg/dL |
| Result | Proteinorachie with low glucose, suggesting GBS |

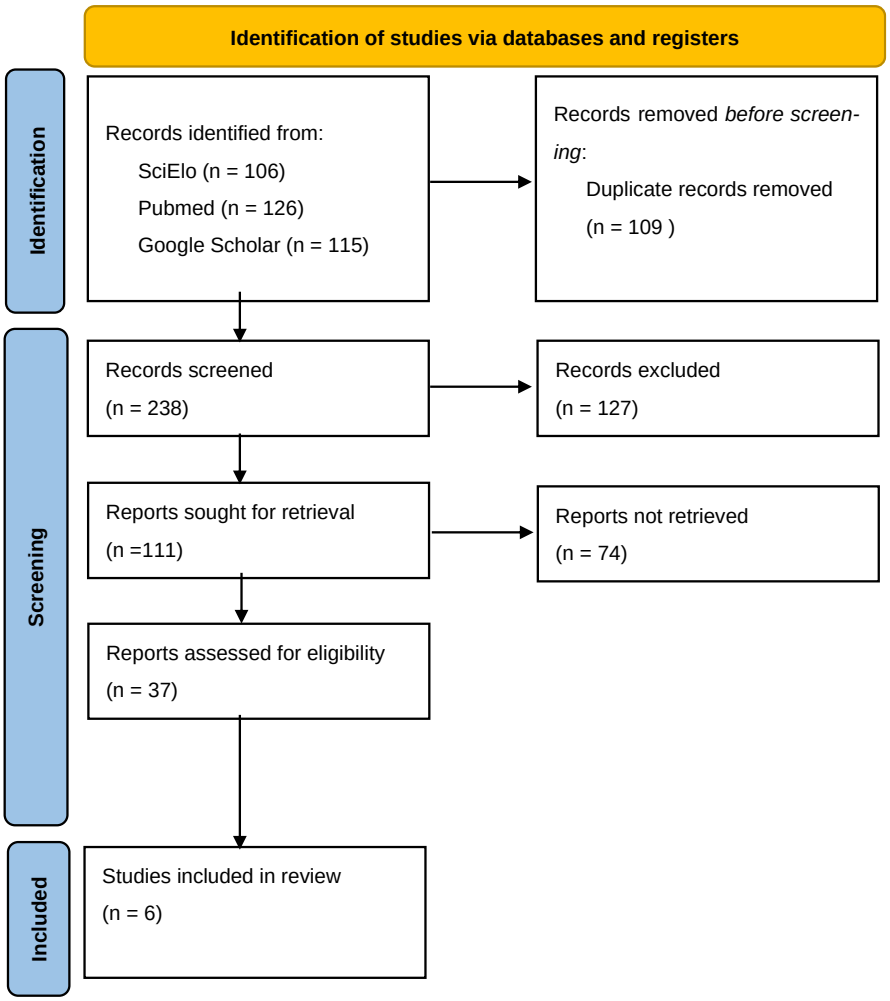
Following the completion of treatment, the patient was discharged with slight clinical improvement and without any hospital-related complications. In post-discharge follow-

up, he reported continued symptom improvement and the complete resolution of both radicular pain and facial paralysis.

## 4. Review

According to the study results, to enhance understanding and ensure transparency in the selection method, the flowchart of scientific articles was constructed following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Figure 1). The first phase involved searching for the PubMed, SciELO, and BVS databases, yielding a total of 347 articles. In the second phase, 109 duplicate articles were removed. In the third phase, titles and abstracts were screened, resulting in the selection of 238 articles. In the final phase, an exploratory, selective, and analytical reading of all studies was conducted, and excerpts related to Guillain-Barré Syndrome were identified and categorized. Ultimately, six articles were selected for discussion.

**Figure 1.** Schematic representation of synthesis and analysis of results (PRISMA).




## 5. Discussion

Guillain-Barré syndrome (GBS) is an acute paralytic disease that causes the rapid onset of limb weakness and often affects the facial, swallowing, and respiratory muscles. Tingling and numbness usually occur in the limbs simultaneously. The disease is typically caused by multifocal inflammation of the spinal roots and peripheral nerves, particularly

their myelin sheaths. Axonal damage often occurs as a secondary consequence of the inflammatory response, though in some cases, the axons themselves are the primary target of the immune attack [8].

GBS is a rare neurological condition characterized by progressive muscle weakness and paralysis, often triggered by viral or bacterial infections. Its incidence varies globally, ranging from 0.5 to 2 cases per 100,000 person-years, with a significant increase in older age groups. Among individuals over 80 years of age, the incidence reaches 2.7 cases per 100,000 person-years. In children under 2 years of age, it is even rarer, with a rate of 0.6 per 100,000 children. In Brazil, between 1995 and 2002, the estimated annual incidence was 0.6 per 100,000 people. Studies indicate that men are more susceptible to GBS than women, with a male-to-female ratio ranging from 1.1:1 to 2:1. However, there is no definitive explanation for this male predominance to date [9].

A study by Lapidurou et al. on the experiences and perceptions of GBS patients concluded that their psychological needs are often unmet by healthcare services. Maintaining a positive attitude was identified as crucial for patients to successfully cope with and recover from GBS. Therefore, incorporating psychological therapy into the treatment regimen could be beneficial if needed and desired by patients. This underscores the importance of an interdisciplinary care model involving physicians, physiotherapists, psychologists, and other healthcare professionals, who can provide more holistic support. Such an approach not only facilitates physical recovery but also addresses emotional and social needs.

This study reports the clinical case of a 46-year-old male patient who initially presented with exanthema, pruritus, fever, and arthralgia, which progressed to paresthesias in both the lower and upper limbs, ultimately leading to difficulty walking, holocranial headache, and intestinal constipation. Studies conducted in Latin America indicate that GBS prevalence is highest in individuals aged 20 to 60 years, with males being more affected than females [11]. However, other studies suggest that GBS can occur at any age, except in infants, with a higher prevalence in adults over 50 years old, and is more common in men than in women [12].

The typical form of GBS presents as a combination of motor and sensory symptoms, usually starting with paresthesia or loss of sensation in the distal extremities, followed by progressive weakness that initially affects the legs, then extends to the arms and cranial muscles, in an ascending pattern [11]. However, according to recent findings, weakness and sensory loss—though always affecting both sides of the body—may present asymmetrically (atypical form) or predominantly involve proximal or distal parts of the extremities, beginning in the legs, arms, or simultaneously in all extremities. Additionally, some patients experience severe, widespread pain or isolated cranial nerve dysfunction before muscle weakness appears [11].

In atypical cases, where only motor impairments are evident, a subtype of acute motor neuropathy may be present. In such cases, electrophysiological reflexes may remain normal, even during disease progression [11]. Generally, GBS is triggered by a preceding infection, which initiates an aberrant immune response. For an accurate diagnosis, the clinical history and a complete neurological examination must be supported by electrophysiological studies and cerebrospinal fluid (CSF) analysis. A hallmark CSF finding is elevated protein levels with a normal cell count, known as albuminocytologic dissociation. However, in early disease stages, CSF findings may still be normal, meaning a negative result does not exclude GBS [4].

Electrophysiological studies can demonstrate peripheral nervous system involvement, typically revealing increased distal motor nerve latencies and reduced conduction velocity in motor or sensory nerves [12]. Regarding neuroimaging, T1-weighted MRI with contrast of the cervical or lumbar spine may show thickening and enhancement of nerve roots or the cauda equina [11].

The Brighton Criteria are another essential tool used to aid in GBS diagnosis. They help differentiate between low- and high-risk patients, allowing for early and rapid diagnosis. These criteria also guide treatment decisions based on patient severity. In under-equipped clinical settings, the Brighton Criteria play a crucial role in detecting GBS [12]. Studies indicate that intravenous immunoglobulin (IVIg) is most effective when started within two weeks of symptom onset, while plasma exchange (PE) is effective when initiated within four weeks [13]. However, further research is needed to compare these two treatments and determine whether one offers superior benefits over the other [13]. Some studies suggest that patients with severe symptoms and rapid progression should immediately receive either IVIg or PE to halt disease progression [13]. Moreover, studies indicate that the combination of IVIg and corticosteroid therapy does not provide additional benefits over IVIg alone. Future research should focus on addressing existing knowledge gaps and identifying new therapeutic strategies for GBS.

## 6. Conclusion

Guillain-Barré Syndrome (GBS) is a severe and multifaceted neurological condition characterized by complex immunological mechanisms that can lead to demyelination or axonal degeneration. The clinical case reported in this article exemplifies the typical progression of the disease, from initial nonspecific symptoms, potentially triggered by a preceding infectious episode, to the development of acute polyradiculoneuropathy, confirmed through clinical and laboratory findings, such as albuminocytologic dissociation in cerebrospinal fluid.

This case study highlights key aspects of GBS, including the importance of recognizing its triggering factors, the role of prior infections, and the manifestation of sensorimotor deficits in a "stocking and glove" distribution. The temporal progression of the case, with a latency period between the initial symptoms and the onset of neurological impairment, underscores its relationship with immunological mechanisms, such as molecular mimicry and antibody-antigen interactions in peripheral nerves.

Furthermore, clinical management, which included immunomodulatory therapy with plasmapheresis, demonstrates the importance of early intervention in preventing complications and optimizing the patient's functional recovery. This study also highlights the relevance of a multidisciplinary approach, incorporating physiotherapeutic and psychological support, which are essential components of a comprehensive rehabilitation process that addresses both the physical and emotional impacts of the syndrome.

Thus, this report not only reinforces established knowledge about GBS but also emphasizes the need for an integrated and personalized approach to patient management, focusing on functional recovery and quality of life. The case presented contributes to clinical understanding and underscores the importance of ongoing research to deepen insights into the pathophysiological and therapeutic foundations of the syndrome.

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