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REVIEW PAPER

Reviving hope – the journey of diagnosing and treating Guillain-Barré syndrome

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ABSTRACT

The Guillain-Barré syndrome (GBS) is a rare but serious neurological condition that continues to pose diagnostic and therapeutic challenges, particularly in paediatrics. Although most paediatric patients achieve full recovery, some experience persisting complications such as chronic pain, paraesthesia, or reduced physical capacity. This study provides a comprehensive review of GBS, addressing its epidemiology, pathogenesis, clinical symptoms, as well as diagnostic and therapeutic tools. An integrated treatment approach, encompassing both causal and symptomatic therapies, plays a pivotal role in the recovery process. In recent years, novel biologic therapies, such as eculizumab and efgartigimod, have emerged as promising treatment options. However, further research is required, especially within the context of the paediatric population.

KEY WORDS:

autoimmune diseases, Guillain-Barré syndrome, peripheral nervous system diseases.

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute polyradiculoneuropathy that was first described more than 100 years ago. It is now the most common cause of acute neuromuscular paralysis worldwide, and it is the best-studied neuroinflammatory disease, providing valuable diagnostic information in neurology [1, 2]. Symptoms can range from subtle paraesthesia to severe motor and respiratory dysfunction [2]. Based on the presented symptoms, clinical course and underlying mechanisms, GBS can be divided into subtypes. The subtypes of GBS include acute inflammatory demyelinating polyradiculoneuropathy (AIDP), which is the most common, acute axonal motor neuropathy (AMAN) and Miller-Fisher syndrome (MFS), with the final two being less common [2, 3].

The cause of the disease is an immune response directed against the peripheral nervous system triggered by a preceding infection. Evidence obtained *via* animal models suggests a key role for molecular mimicry as the aetiology of the disease. Many infections have been linked to GBS; however, respiratory and gastrointestinal diseases are the most common [4].

Treatment of GBS should include individualised assessment of the patient's condition and intensive monitoring of vital functions. The possibility of respiratory failure requiring artificial ventilation should be anticipated, and immune treatment in the form of intravenous immunoglobulin (IVIG) or plasma exchange should be implemented as soon as possible [5]. Our study aims to analyse the current state of knowledge about GBS, focusing on the latest diagnostic techniques and therapies that offer hope for improved patient outcomes.

MATERIAL AND METHODS

The review was based on scientific publications obtained through a comprehensive search strategy. We used a broad set of medical subject headings key words and terms such as GBS, AIDP, demyelinating diseases, peripheral nervous system diseases, diagnosis, treatment,

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and pathophysiology. The search was conducted using PubMed, Scopus and The Wiley Library databases. Of the results, we excluded those that were abstracts, were not in Polish or English, and were duplicate studies. Finally, 45 articles, either in Polish or English, were selected, with the emphasis on the most recent information on the topic, 44 of which were published no earlier than in 2016.

EPIDEMIOLOGY OF GUILLAIN-BARRÉ SYNDROME

Acute flaccid paralysis has historically been most commonly associated with poliomyelitis in infants and children who were previously healthy. However, that inglorious title was taken over by GBS after the eradication of the polio virus [6]. As an illness that is frequently preceded by an infection, most notably Campylobacter jejuni, the incidence rate varies around the world as exposure to certain infections varies in different regions [7, 8].

In the research papers analysed, the incidence rate of paediatric GBS varied and fell between 0.34 per 100 000 to 1.34 per 100 000 with a male-to-female ratio of 3 : 2 [6, 9]. However, such differences in gathered data can be easily explained, since as mentioned before, the incidence rate varies in different regions due to different pathogen exposures [8]. Excluding the age factor and including adults in the statistics, we can determine that the morbidity of GBS is the greatest in Bangladesh (3.25), followed by Chile (2.12) and the lowest in Japan (0.44), Europe and North America with an incidence rate of 0.8–1.9 (children 0.6); the values given per 100 000 [1, 8]. The previously mentioned statistics were calculated as a generalisation disregarding age ranges, however, a positive correlation can be found between the age of the patient and the incidence rate, roughly described as a 20% increase in the incidence rate with every additional 10 years to age. Additionally, the mentioned incidence rate for children is found to be lower [7]. On the other hand, a similar correlation does not seem to exist between the ages of 0 and 15. In a Danish research paper, the morbidity of GBS was shown to peak twice at different age ranges, a larger peak at the age of two and a smaller one at 14 [10]. Seasonal fluctuations of GBS incidence rates are very often shown by researchers and can be explained by seasonal infections. In Western countries, a peak is observed during the colder seasons, while in Bangladesh and China, during the warmer seasons [7].

PATHOGENESIS AND MECHANISMS

Guillain-Barré syndrome symptoms are usually preceded by an infection, generally Campylobacter jejuni [7]. Different pathogens often associated with GBS include viruses such as Epstein-Barr virus, influenza A virus, the Zika virus, cytomegalovirus, hepatitis E virus (HEV) and bacteria including *Mycoplasma pneumoniae*, *Haemophilus influenzae* [1]. The reason for such statement suggests the fact that molecular mimicry exists between microbial antigens and human nerve cells [7]. The pathogens' surface lipo-oligosaccharides trigger the production of cross-reactive antibodies targeting gangliosides like GM1, GD1a, and GQ1b [1]. This immune response leads to demyelination or axonal injury, with recovery occurring through slow axonal regeneration or faster peripheral remyelination. The balance of these processes contributes to different GBS subtypes: AIDP, AMAN, and MFS [7].

To investigate AMAN, a rabbit was immunised with GM1. In the said model, macrophages strip myelin, and antibodies attack Schwann cells, leading to their degeneration. It is also theorised that antibodies might affect nerve regeneration. Patients with AMAN are primarily dealing with axonal injury without demyelination or T-cell inflammation [7].

Acute inflammatory demyelinating polyradiculoneuropathy has been investigated in rat models of experimental autoimmune neuritis. These studies confirmed that segmental demyelination and remyelination result in slowed nerve conduction, prolonged distal latencies, and temporal dispersion [7, 8].

CLINICAL PRESENTATION

Guillain-Barré syndrome typically begins with rapidly progressing, symmetrical weakness starting in the legs and advancing to the arms and, in some cases, the cranial nerves. Pain, especially in children, may be neuropathic, radicular, or musculoskeletal. Deep tendon reflexes are often lost early, with most patients exhibiting absent reflexes as symptoms peak. Motor symptoms progress quickly, usually reaching their peak within two weeks [11, 12]. Autonomic dysfunction is a major complication, affecting both sympathetic and parasympathetic systems, leading to cardiovascular instability, abnormal pupil responses, and bowel or bladder dysfunction [13, 14]. These symptoms are common but often underdiagnosed in children [13]. Guillain-Barré syndrome can present atypically, with asymmetrical weakness affecting different limbs or severe pain preceding motor symptoms. In young children (under 6 years old), this syndrome may show nonspecific or atypical symptoms, such as diffuse pain, refusal to bear weight, irritability, signs of meningitis, or unsteady walking [14]. The severity varies from mild weakness to respiratory failure, with 15% of paediatric patients requiring mechanical ventilation. However, most children recover well, with minimal impairment after 1–4 months, and over 90% regain full function [15].

DIAGNOSIS AND DIAGNOSTIC TOOLS

Confirming a diagnosis of GBS is challenging due to the limited number of definitive tests. Diagnosis primarily relies on clinical symptoms, a history of prior infections, and a selection of laboratory tests. Early-stage GBS can

be particularly difficult to identify as its symptoms often resemble those of other neurological disorders [16]. A review of the literature revealed that diagnostic recommendations are largely based on findings from adult patients with GBS, for whom a higher number of relevant studies are available compared to the paediatric population [17]. The clinical diagnosis of GBS needs to be supported by the results of cerebrospinal fluid (CSF) and nerve conduction studies (NCS) [18].

LABORATORY TESTS AND CEREBROSPINAL FLUID

Blood tests have limited diagnostic value, typically showing only mild inflammation. Cerebrospinal fluid analysis may reveal elevated protein without lymphocytosis in about 80% of paediatric cases and can be tested for anti-ganglioside antibodies [15]. Cerebrospinal fluid examination is recommended when the diagnosis is unclear or alternative conditions need to be ruled out. Normal CSF protein levels in the first week do not exclude GBS, while a white blood cell count > 50 cells/μl should prompt consideration of other diagnoses [2].

A lumbar puncture with CSF protein analysis and cell count measurement is an auxiliary test in the diagnosis of GBS. Normal protein levels in the CSF within the first two weeks do not exclude GBS, while elevated protein levels with a normal cell count may suggest the disease. If initial CSF findings are inconclusive, a repeated lumbar puncture after 10–14 days may provide additional diagnostic value [19]. A study compared 156 children with GBS to 26 children with acute flaccid myelitis (AFM) linked to enterovirus D68. Lumbar punctures were performed in most cases. Elevated CSF protein levels were more common and higher in GBS than in AFM. Cytoalbuminologic dissociation, a key diagnostic feature of GBS, was more frequently observed in the GBS group [16, 20].

ELECTROPHYSIOLOGICAL STUDIES

Electrophysiological studies are necessary to confirm the diagnosis of GBS and to identify its numerous variants. It is recommended to base the diagnosis on clinical criteria supported by the results of CSF analysis and electrophysiological studies [19].

THE ROLE OF NEUROIMAGING

Neuroimaging plays a role in ruling out alternative diagnoses, such as a spinal cord tumour. Magnetic resonance imaging of the spine with gadolinium contrast can reveal nerve root enhancement in 80–90% of paediatric cases [15].

BIOMARKERS

Neurofilament light chain (NfL) is a promising biomarker for axonal damage. Previous studies have reported that both NfL and neurofilament heavy chain are elevated in the CSF of patients with GBS and are associated with clinical outcomes. Recent research has shown that in most patients with GBS, serum NfL was elevated in the early stage of the disease, during the first two weeks, despite IVIG treatment. Additionally, high serum NfL levels are associated with a more severe clinical course and a less favourable prognosis in GBS [21].

THE BRIGHTON CRITERIA

The Brighton criteria are used for diagnosing GBS and help distinguish high-risk patients from low-risk patients. The mentioned criteria include all the characteristics and initial symptoms of the disease, as well as diagnostic tools that aid in its identification and monitoring [22]. The Brighton criteria for diagnosing GBS include the absence of alternative diagnoses for the weakness, diminished or absent deep tendon reflexes in weak limbs, a monophasic course with onset to nadir occurring within 12 hours – 28 days, bilateral and flaccid limb weakness, CSF cell count less than 50 cells/microl, CSF protein concentration above normal, and NCS findings that align with one of the GBS subtypes. The lack of awareness and use of the Brighton criteria makes it challenging for physicians to diagnose ambiguous cases of GBS, which may contribute to higher morbidity and mortality rates [22].

TREATMENT OPTIONS

Guillain-Barré syndrome typically shows spontaneous recovery, and treatments aim to shorten recovery, manage symptoms, and restore pre-illness physical function [7, 17]. Treatment can be divided into causal and symptomatic. Causal therapy targets GBS's underlying cause and includes immunological therapies like plasma exchange, IVIG, and antibiotics. Symptomatic treatment involves pain management, respiratory support, thrombosis prevention, physiotherapy, psychological support, and constant monitoring of vital functions to minimise negative outcomes [17, 23–25].

Every patient with a probable or definite diagnosis of GBS should be hospitalised as the progression and severity of the illness cannot be predicted [24]. The primary actions undertaken after hospitalisation should consist of setting up tracking machinery/monitoring devices in order to measure vital signs such as heart rate, blood pressure and vital lung capacity [17, 24, 25]. Monitoring of the heart function allows for a rapid response in the case of tachyarrhythmia, bradyarrhythmia and hypertension caused by autonomic neuropathy experienced by approximately 50% of paediatric GBS patients. Low molecular weight heparin is advised in immobilised patients as potential blood clots can lead to a pulmonary embolism or even a stroke [17, 25]. Furthermore, monitoring lung function is crucial for the survival of patients

as the dysfunction of respiratory muscles is common in GBS and, if untreated, will lead to suffocation and death. Through monitoring vital capacity, it is possible to initiate mechanical ventilation and eliminate respiratory failure till the end of the recovery period [17, 23–26]. In addition to mechanical ventilation, mucolytic drugs can be administered, as well as suction of the respiratory tract if necessary [17, 24].

Other than preventing complications such as thrombosis and respiratory failure, it is important to maintain the well-being of patients. This can be achieved through hydration, i.v. administering of saline, electrolytes, and nutrition, with the latter being advised in cases of mechanically ventilated patients and such where the oropharyngeal nerves are affected [23, 24]. In addition, pain management is another factor in the patient's comfort and overall health. When considering pain medication, drugs should be administered with respect to the analgesic ladder, beginning with non-opioids such as paracetamol [17, 24, 26, 27]. According to the guidelines from 2020, 70% of paediatric patients suffering from GBS experience neuropathic pain with which even potent opioids may not be effective, in such situations, anticonvulsants such as carbamazepine and gabapentin are advised [17, 24, 27]. However, the use of corticosteroids is not advised in paediatric patients as no studies confirmed their statistically relevant effectiveness and also such drugs have serious side effects involving the growth period [17, 23, 24]. The course of illness progression can be very rapid and severe, which correlates with a downfall in the patient's mental health, therefore, psychological support is as essential in the process of recovery as the administering of medication [17].

Causal treatment consists of distributing antibiotics and immunotherapy. Antibiotics are chosen in correlation with the bacteria which caused the primary infection, thereby triggering the onset of GBS through molecular mimicry [17, 23, 28]. However, the main treatment method for GBS, which has been confirmed by multiple studies to shorten the recovery period, is plasma filtration and immunoglobulin transfusions. Plasma filtration was considered as a way of treating GBS based on the molecular mimicry theory, therefore, through limiting the flow of corrupted immune cells, it is possible to slow the process of neurodegeneration [23]. The guidelines for undergoing plasmapheresis are to exchange 40–50 ml/kg of mass over 3–5 times every 2 days, where the substitute fluid is 5% albumin [17, 24, 25]. Plasmapheresis is an invasive form of treatment and is the most effective when administered in the first 7 days of the illness's onset [23, 25]. After plasmapheresis, control of blood morphology and IgG level is advised [25]. The guidelines on the use of therapeutic apheresis from 2020 suggest a different form of plasma filtration based on a system of binding immunoglobulins, which eliminates the need for any substitute fluids known as immunoadsorption systems [23]. Contrary to plasma filtration, another effective way of treating GBS is administering a purified form of human plasma consisting mainly of IVIG [17, 29]. Based on most recent guidelines, IVIG is to be administered at a dose of 0.4 g/kg of body mass over five consecutive days, however, when considering paediatric patients, a dose of 2 g/kg over two consecutive days is found to be more effective, such a large dose can only be considered in patients with no renal or cardiac dysfunctions [17, 24, 25, 29, 30]. A study conducted in India in 2022 compared the long-term outcomes of treating GBS with IVIG compared to the natural course of the illness only supported with symptomatic treatment. The results were similar, with outcomes depending on the type and severity of GBS. The treatment was not found to be as altering in the course of mild GBS. Also, the study evaluated the outcomes on paediatric patients; the first six months of recovery were comparable in both groups, and the treatment was ineffective in paediatric patients with AIDP GBS. Intravenous immunoglobulin showed limited effects in mild GBS and AIDP in paediatric patients but was more effective in motor-sensory GBS [29]. Furthermore, a retrospective cohort study and a double-blind, randomised study conducted in the Netherlands researched the effectiveness of administering a second dose of IVIG and its correlation with a more prosperous and faster recovery. Both studies concluded the ineffectiveness of a second dose and also the study conducted in the Netherlands presented that the second dose of IVIG correlated with serious complications such as thrombotic events, which occurred twice as many times when compared to the placebo group, other side effects involved pneumonia, asystole, infections and pulmonary embolism. However, the patients subject to the second dose of IVIG were found to have a 50% decrease in the duration of mechanical ventilation compared to the placebo group [30, 31].

In recent years, there have been new approaches presented for the treatment of GBS, such as the use of antibodies like eculizumab, efgartigimod and ANX005 or immunosuppressants like ginkgolides. Eculizumab is a humanised monoclonal antibody targeting the complement protein C5. A double-blinded study was conducted in Japan in 2018 and compared the effectiveness and safety of the mentioned drug in an IVIG combined treatment [32]. The study presented that the group administered with IVIG combined with eculizumab had a more apparent long-term recovery over 24 weeks than the placebo group. The statistics differentiated the eculizumab group, with 74% of patients obtaining a GBSDS < 1, whereas only 18% of patients from the placebo group achieved such results. Furthermore, the eculizumab group showed to have a more improved motor function, was protected from respiratory muscle paralysis and the destruction of motor nerve terminals [7, 32]. Another example of an antibody proposed as a treatment for GBS

is efgartigimod, which is a human IgG antibody fragment; it is found to assimilate the degradation of IgG [33]. Two studies were conducted in recent years, and it was found that two weeks after the drug administration, IgG and albumin levels were decreased, and patients were able to walk independently after four weeks [33, 34]. Another example of potential treatment is the ANX005 drug, a humanised immunoglobulin G4 recombinant antibody against C1q, which should inhibit the complement cascade and, as the previously mentioned antibodies, should cease the progression of GBS; however, the drug is still in trial periods, and there is no reliable evidence to support its effectiveness [35]. Another way of delaying the progression of GBS could be the use of immunosuppressive drugs, such as 'ginkgolides' presented in a 2021 study, which decrease proinflammatory cytokines like IFN-y and IL-12p70 thereby down-regulating the expression of TLR; similarly to ANX005 it is also still in trial periods with positive effects presented by mice [36].

Bifidobacterium infantis is a species of the probiotic bacteria naturally occurring in the intestines of newborns; studies propose the use of this bacterium as a medium for inhibiting the proliferation of T-cells and apoptosis through the PD-1 signal track [37, 38]. The mentioned studies were conducted using the blood of GBS patients; unfortunately, the trial of the potential medication was conducted on rats. Therefore, further research involving human subjects is necessary to draw viable conclusions, although it could be a means of reversing the demyelination process undertaken during GBS progression [37, 38].

Physical rehabilitation is a crucial part of GBS treatment, starting from the onset of the disease to improve mobility, strength, balance, and patient motivation [17, 24, 25, 39–42]. A 2023 case report for an 8-year-old GBS patient included rehabilitation involving cycling, treadmill walking, strength training, breathing exercises, and balance exercises, with regular position changes for bedridden patients. In cases of extreme weakness, robot-assisted gait training can help by supporting the patient's weight and aiding muscle strengthening [41]. Additionally, the incorporation of VR technology is beneficial to maintaining the focus of younger patients [42]. Furthermore, physical therapy should be continued even after the patient is discharged from the hospital for the most promising results [39, 40]. A 2023 study in India evaluated teleconsultation-based rehabilitation, especially during COVID-19, where parents received guidance through phone consultations, presentations, and diagrams. The study found this approach highly effective (85–95% compatibility) and noted fewer hospital visits compared to traditional rehabilitation [40] (Figure 1).

PROGNOSIS AND LONG-TERM OUTCOMES

With a mortality rate of 3–5%, GBS is a serious neurological condition in children [15]. Young patients typically

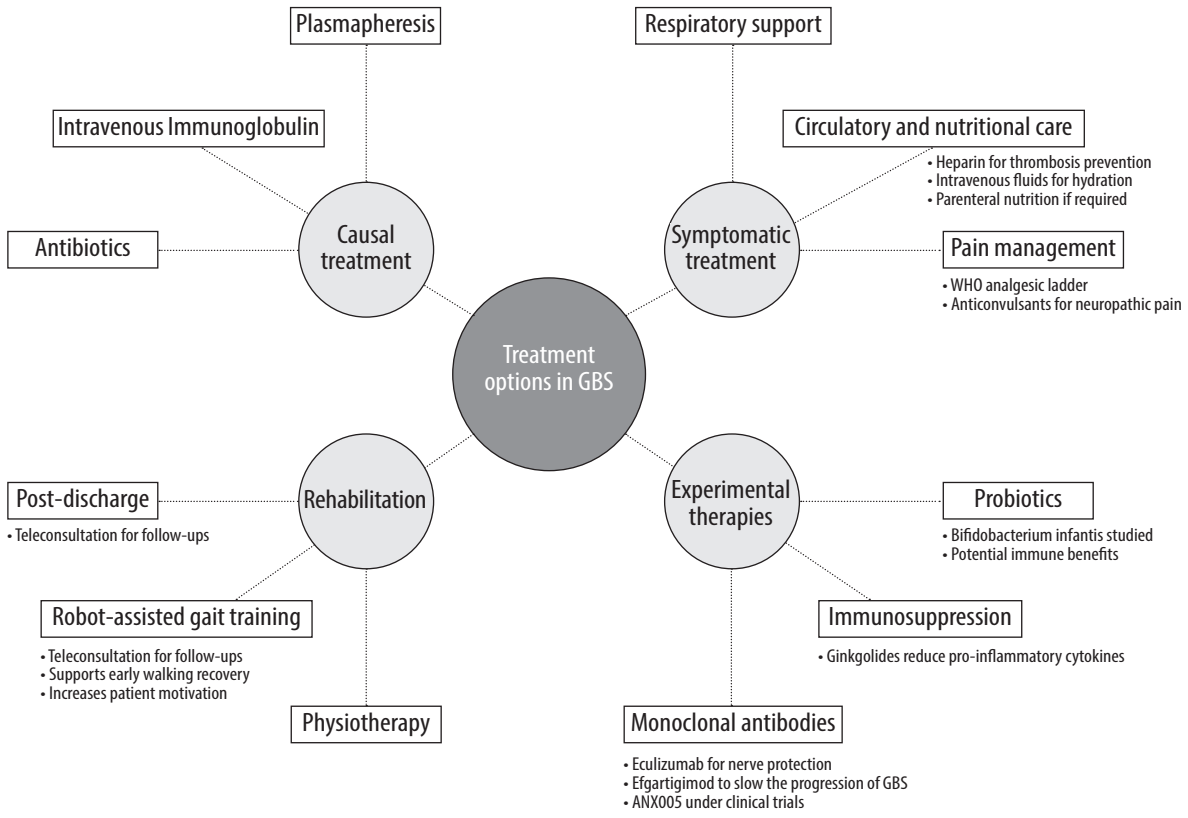

FIGURE 1. Therapeutic approaches in Guillain-Barré syndrome – causal, symptomatic, experimental, and rehabilitation strategies [17, 23–28, 32, 33, 35–42]

GBS – Guillain-Barré syndrome, WHO – World Health Organization

experience better long-term outcomes and have a more favourable prognosis than adults [11]. While most children achieve a full recovery, some may continue to face lasting challenges, such as discomfort, chronic fatigue or an inability to walk unassisted six months after symptoms begin. Although significant improvement is typically seen within the first year, full recovery has also been reported in cases where symptoms last for more than three years [15]. A recent retrospective study by Nasiri *et al*. evaluated 57 children (aged 1–13 years) with GBS, finding a 92.9% full recovery rate and a 1.8% mortality rate. The only death was caused by autonomic dysfunction, leading to tachycardia, abnormal sweating, blood pressure instability, and fatal cardiac arrhythmias [43]. In the study from 2019, 110 children were analysed, identifying two factors associated with long-term sequelae. First, axonal forms of GBS (AMAN or AMSAN) carried a higher risk, with 29% of affected children developing complications, compared to 5% with a demyelinating form. Second, a shorter interval between symptom onset and hospitalisation was linked to worse long-term outcomes [44]. Another study by Estublier *et al*. followed 51 children for a median of 6 years and 4 months (range 3–20 years) after the acute phase. It found that 67% had long-term sequelae, mostly mild, with common complaints including paraesthesia, pain, and fatigue. The risk of persistent symptoms was strongly linked to initial disease severity, particularly loss of ambulation during the acute phase, with female patients more likely to experience severe sequelae. These findings emphasise the need for long-term follow-up and improved evaluation to address the lasting impact of GBS on children's daily lives, autonomy, and mental health [45].

CONCLUSIONS

Guillain-Barré syndrome poses diagnostic and therapeutic challenges due to its diverse symptoms and similarity to other neurological disorders. Early diagnosis and timely treatment are crucial to improving prognosis and reducing complications such as chronic pain, paresthesias, and impaired mobility.

Current therapies, including plasmapheresis and intravenous immunoglobulins, are effective but vary in efficacy depending on the disease subtype and patient characteristics. Emerging biological treatments, such as eculizumab and efgartigimod, show promise, though further research is needed, especially in paediatric cases.

Advancing clinical trials on personalised therapies and identifying biomarkers for more precise diagnosis and monitoring are essential. Continued research is crucial to refining GBS management and improving patient outcomes.

DISCLOSURES

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