AHEAD OF PRINT

Prognostic factors associated with worse outcomes in patients with GBS: A systematic review

Marta Grelowska[1,B–D], Katarzyna Logoń[1,B–D], Edyta Dziadkowiak[2,A,D–F]

[1] Student Scientific Association of Neurology, Wroclaw Medical University, Poland
[2] Department of Neurology, Wroclaw Medical University, Poland

A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation;
D – writing the article; E – critical revision of the article; F – final approval of the article

Advances in Clinical and Experimental Medicine, ISSN 1899–5276 (print), ISSN 2451–2680 (online) *Adv Clin Exp Med*. 2025

Address for correspondence
Edyta Dziadkowiak
E-mail: edyta.dziadkowiak@umw.edu.pl

Funding sources
None declared

Conflict of interest
None declared

Received on November 30, 2023
Reviewed on March 23, 2024
Accepted on April 8, 2024

Published online on October 2, 2024

Abstract

Guillain–Barré syndrome (GBS) is an autoimmune polyradiculoneuropathy with diverse clinical subtypes, characterized by rapidly evolving motor weakness, sensory disturbances and areflexia. The global prevalence of GBS has been steadily increasing, with regional disparities. Mortality rates vary but remain elevated in patients requiring mechanical ventilation. This systematic review aimed to evaluate the predictive risk factors for the severity of the disease and poor short- and long-term outcomes of GBS. The literature search was conducted using the PubMed database by 2 independently working researchers. After a screening process of studies published before November 2023, a total of 109 articles were selected. Original articles, systematic and narrative reviews, meta-analyses, and editorials were selected based on their clinical relevance. The exclusion criteria included patients under 18 years of age, pregnant women and articles in languages other than English and Polish. Long-lasting GBS complications included pain, fatigue and persistent neurological deficits, affecting patients for years after recovery. Identifying the appropriate therapeutic methods, risk factors and prognoses of GBS at an early stage is crucial. Various risk factors for death and poor functional outcomes were found, regarding patient characteristics, the clinical course of GBS, laboratory and neurographic results, as well as treatment methods.

Key words: treatment outcome, risk factors, prognosis, Guillain–Barré syndrome

Cite as
Grelowska M, Logoń K, Dziadkowiak E. Prognostic factors associated with worse outcomes in patients with GBS: A systematic review [published online as ahead of print on October 2, 2024]. *Adv Clin Exp Med*. 2025. doi:10.17219/acem/186949

DOI
10.17219/acem/186949

Introduction

Guillain–Barré syndrome (GBS) is an autoimmune inflammatory polyradiculoneuropathy affecting peripheral nerves.[1] It is characterized by rapidly evolving ascending motor weakness, areflexia and sensory disturbances that develop within 4 weeks.[2] Guillain–Barré syndrome often follows infections, but it can also occur after vaccinations, surgeries or during pregnancy.[3] The main variants of GBS include acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN), and Miller–Fisher syndrome (MFS).[2] Acute inflammatory demyelinating polyradiculoneuropathy manifests as a sensorimotor form that can co-occur with cranial nerve deficits and autonomic dysfunction. Acute motor axonal neuropathy is a pure motor form in which the cranial nerves are intact. Acute motor and sensory axonal neuropathy (AMSAN) is a condition that shares similarities with the AMAN pattern, but it additionally affects sensory nerves.[4] Miller–Fisher syndrome is less common and is characterized by ataxia, ophthalmoplegia and areflexia.[5]

The age-standardized prevalence of GBS is the highest in high-income Asia Pacific and North American countries, especially Japan and Singapore. East Asia and Oceania have the lowest GBS prevalence rates.[6] The AIDP type is significantly more common in Europe and North America, while AMAN occurs more frequently in East Asia.[5]

The prevalence of GBS has continued to increase globally over the years. In 1990, the global prevalence per 100,000 persons was 3.6%, and in 2019 it reached 9.5%.[6] In a 2009 study, the global incidence of GBS was estimated between 1.1 and 1.8 cases per 100,000 persons/year.[7] In the recent 2021 meta-analysis, the incidence of GBS among the cohort studies was higher and varied from 0.30 to 6.08 cases per 100,000 persons and 0.42 to 6.58 cases per 100,000 person-years.[8] Guillain–Barré syndrome is slightly more frequent in men than in women and its incidence tends to increase with age.[9]

The mortality rates of GBS vary significantly between studies and range between 1–18%.[10–12] They remain higher (12–20%) in patients requiring endotracheal intubation and mechanical ventilation (MV).[13,14]

Guillain–Barré syndrome is associated with long-lasting complications, such as pain, fatigue, disability, and impaired psychosocial functioning.[15] Persistence of moderate-to-severe pain was reported in different studies after 1 or 2 years in over 1/3 of patients.[16,17] Patients, after recovering from GBS, still report neurological deficits. Many studies described deficits in ambulation and sensation occurring 1 year after illness onset.[18,19] Motor and sensory disturbances were reported quite commonly even 10 years later.[20] In a study by Durand et al., after 6 months, almost 1/3 of patients had a disability grade ≥2 (Plasma Exchange/Sandoglobulin Guillain–Barré Syndrome Trial Group, 1998: 0 = healthy, no signs or symptoms of Guillain–Barré syndrome; 1 = minor symptoms or signs and able to run; 2 = able to walk 5 m across an open space without assistance; 3 = able to walk 5 m across an open space with the help of 1 person and waist-level walking-frame, stick, or sticks; 4 = chairbound/bedbound: unable to walk as in 3; 5 = requiring assisted ventilation (for at least part of day or night); 6 = dead).[21] In another study, at 3–5 years after GBS onset, 20% of patients had a disability grade of 2 and 10% had a disability grade of 3.[22] In a recent long-term study, approx. 10% of patients exhibited disability by the end of the study period. Of these, 5% demonstrated moderate disability, while 5.2% exhibited severe disability.[23]

The outcomes of GBS differ between the GBS subtypes. A study by Zhang et al. found that the prognosis of AMAN patients was poorer than that of AIDP patients,[24] which was confirmed in a 2020 study where AMAN was found to be an independent predictor of an unfavorable outcome.[25] A recent 2022 study reported worse outcomes in patients with AMAN and AMSAN compared to those with AIDP.[26] Patients with MFS usually have a good natural recovery, and almost no residual deficits were left at follow-up, regardless of the treatment.[27]

The pathophysiology of GBS is based on the phenomenon of molecular mimicry. Depending on the site on the nerve cell where the antibody attack occurs, GBS assumes a specific clinical form. The autoimmune process is usually initiated by an infection. Figure 1 shows these processes in a clinical form.

The figures were drawn with Procreate v. 5.3.3 (Savage Interactive, Hobart, Australia). Parts of the Fig. 1 were made using pictures from Servier Medical Art, which is licensed under a Creative Commons Attribution 3.0 Unported License (https://creativecommons.org/licenses/by/3.0/). The diagnosis and management of GBS should be based on guidelines published in 2023 by van Doorn et al.[28] The diagnosis is established regarding the patient's history and neurological, electrophysiological and cerebrospinal fluid (CSF) examinations. An alternative diagnosis for the weakness must be excluded.[29,30] Guillain–Barré syndrome should be taken into account in patients who have rapidly progressive symmetric motor weakness of the legs and/or arms in the absence of other apparent causes, especially if there is a history of recent diarrhea or respiratory infection.[28] Patients with the classic sensorimotor form present with distal paresthesias or sensory loss, ascending weakness, and a loss of reflexes. Symptoms develop within no more than 4 weeks and in most patients within 2 weeks.[28,30,31] Cerebrospinal fluid analysis is valuable and usually shows an elevated protein level and a normal cell count, known as albuminocytologic dissociation.[32] In standard conduction velocity tests, prolongation of distal latencies, slowing of conduction velocities mostly in motor fibers, and prolongation or absence of F-waves are observed.[33]

Electrodiagnostic studies are also helpful in differentiating between the 4 subtypes of classical GBS: AIDP, AMAN, AMSAN, and MFS.[34] The criterion for the diagnosis

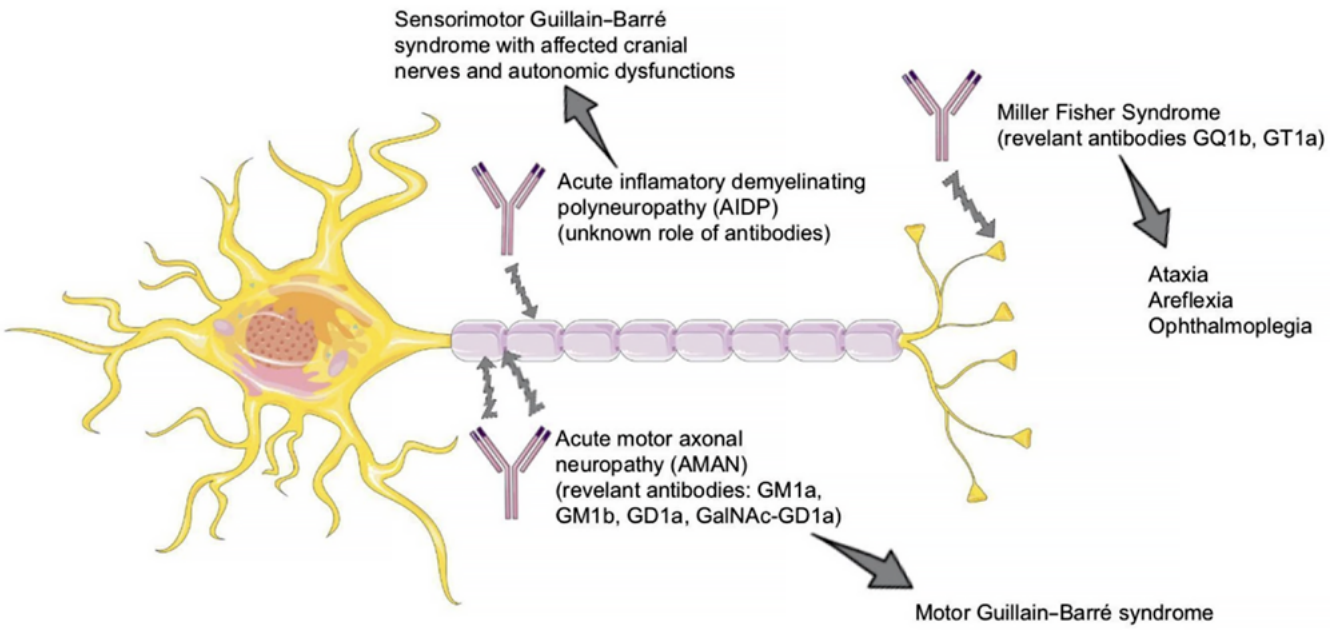

Fig. 1. Pathomechanism of GBS subtypes

of AIDP is the electrophysiological confirmation of a decrease in the conduction velocity of 2 or more motor nerves, suggesting an immune-mediated demyelinating process involving the membrane of Schwann cells or myelin.[35] Acute motor axonal neuropathy is distinguished from AIDP due to the occurrence of axonal involvement without demyelination. The diagnosis of AMAN is based on the finding of reversible conduction failure due to axonal conduction block at the nodes of Ranvier or the motor nerve terminal without axonal degeneration or extensive axonal degeneration.[28] There are also rarer types of GBS, such as AMSAN and MFS. The first of them concerns changes, the basis of which lies in the axonal degeneration of both motor and sensory fibers. The latter is characterized by a characteristic triad of clinical symptoms, which includes ophthalmoplegia, ataxia and areflexia, which is closely related to the presence of specific antibodies against ganglioside GQ1b.[36]

It is currently believed that the best effects in GBS therapy are achieved through the use of intravenous immunoglobulin (IVIG) 0.4 g/kg within 2 weeks of the onset of symptoms for 5 days.[37] Good results are also achieved by performing plasmapheresis in the amount of 4–6 treatments. The key variable influencing the effectiveness of therapy is the time of initiation of therapy, which should be started as soon as possible, up to 12 h after the onset of symptoms.[38]

Objectives

This study aimed to undertake a new review of the up-to-date literature concerning the risk factors regarding patient characteristics, the course of GBS, and laboratory and neurographic test results. The efficacy of the possible treatments was also discussed.

Materials and methods

A review of scientific articles published in the PubMed database between 1981 and 2023 was performed. Data were collected in September 2023 by 2 independently working researchers. The following filters were used in the PubMed database: ((GBS) OR (Guillain–Barré syndrome)) AND (long-term) AND ((disability) OR (outcomes) OR (mortality)), ((GBS) AND (risk factors)) and ((GBS) AND (predictors)) for a total of 1,384 results. Of these, 944 articles were removed after reviewing the title or abstract, since they were unrelated to the topic of the research. The exclusion criteria were patients under 18 years of age and pregnant women. Conference abstracts and articles in languages other than English and Polish were excluded as well. Ultimately, 73 articles were qualified for analysis. Additionally, 36 papers were used that did not appear in the automatic search but were considered relevant. The summary of the results for unfavorable outcomes is provided in Table 1. The summary of the studies mentioned in this review is provided in Table 2.[39–92] Figure 2 depicts the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) chart of evaluated studies. All figures and tables were prepared manually Servier Medical Art and Procreate software.

Risk factors: Patient characteristics

Risk factors for death regarding patient characteristics are older age and pre-existing comorbidities, such as organ dysfunction (including cardiac and pulmonary disease), diabetes mellitus and coronary artery disease.[39–41] In a study by Dhar et al., advanced age was the strongest predictor of poor outcomes.[40,42] Van den Berg et al. found

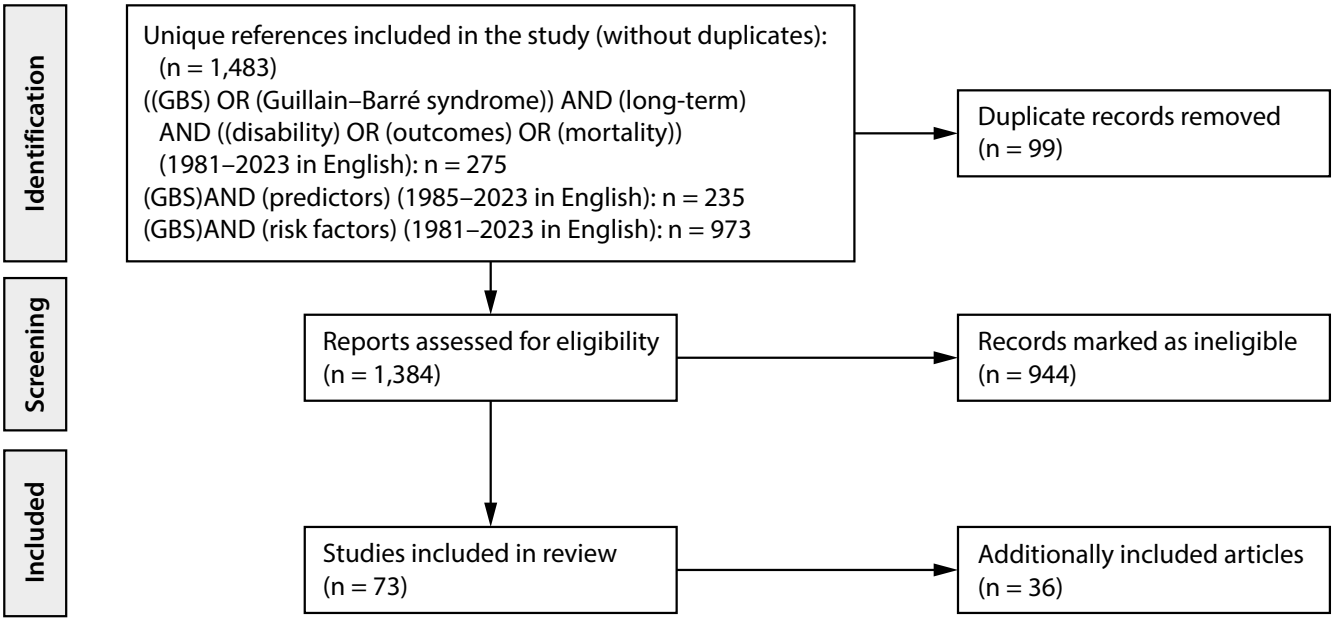

Fig. 2. Identification of studies via database and registers

Table 1. Overview of prognostic factors for death and disability in Guillain–Barré syndrome

Type	Prognostic factor
Demographic	older age pre-existing comorbidity: pulmonary disease, cardiac disease, dyslipidemia, diabetes recent history of surgery
Clinical	higher severity of weakness at entry mechanical ventilation lack of mechanical ventilation when needed increased delay from onset of weakness to entry voiding difficulty longer time to peak disability autonomic dysfunction bulbar nerve involvement papilledema neck flexor weakness the type of the antecedent disorder: gastroenteritis pulmonary infection long duration stay in hospital chief complaint: weakness
Laboratory	presence of anti-GD1a/GD1b and/or anti-GD1b/GT1b antibodies hyponatremia low serum albumin levels higher neutrophil/lymphocyte ratio (NLR) and elevated C-reactive protein (CRP) elevated protein levels elevated neurofilament light protein (NFL) lower folate levels higher fasting blood glucose (FPG) levels increased cerebrospinal fluid total protein (CSF-TP) higher protein-to-albumin ratio (CAR) elevated CRP
Neurographic	markedly attenuated compound muscle action potentials inexcitable motor nerves denervation changes lack of electrical activity in the quadriceps femoris muscle on the 10th day lower deltoid muscle strength decreased intraepidermal nerve fiber density (IENFD)

that 73% of the deceased patients had a history of pulmonary or cardiac disease.[43] This is consistent with other studies, in which mortality was significantly associated with underlying cardiopulmonary diseases.[13,44] A recent 2022 study found a correlation between dyslipidemia and the severity of GBS.[45] Furthermore, the recent history of surgery is associated with an unfavorable short-term prognosis and disease severity.[41]

Risk factors: Clinical course of the disease

Several risk factors for death and poor functional outcome regarding the clinical course of GBS were found, including the severity of weakness at entry, MV, delay from onset of weakness to entry, voiding difficulties, and time to peak disability.[43,46,47,93] The time between onset of disease and death is highly variable. In a study by van den Berg et al., the median time was 76 days (ranging from 23–152 days). Sixty-seven percent of patients died in the recovery phase, 20% in the acute progressive phase and 13% during the plateau phase.[43] The severity of the disease is usually assessed using the Medical Research Council (MRC) sum score or GBS disability score.[94,95] In a recent 2023 study, the best predictor of clinical rating scores using the Hughes Disability Scale (HDS) and Overall Neuropathy Limitation Scale (ONLS) was a low MRCSS on the 10th day of treatment.[48] A study of Bangladeshi patients revealed that MV and the absence of ventilator support when it was required were risk factors for death. The unavailability of MV for patients with acute respiratory failure was identified as the most important risk factor that accounted for 20% of deaths.[49] The need for MV is correlated with longer hospital stay and and reduced rate of recovery up to 1 year after the onset of disease.[50]

Table 2. A summary of the studies mentioned in a review

Author	Year of study	Number of patients	Type of study	Estimated factor
Shangab et al.[39]	2020	82 GBS patients	retrospective study	older age, requirement for MV, axonal type of nerve injury, severity of weakness at entry
Dhar et al.[40]	2008	77 GBS patients	retrospective study	advanced age, prolonged MV, ICU complications (mostly pneumonia)
Wen et al.[41]	2021	155 GBS patients	retrospective study	recent history of surgery, older age, cranial nerve impairment, elevated levels of liver enzymes, lower MRC score, requirement for MV, pneumonia
Zhang et al.[42]	2017	535 GBS patients	retrospective study	older age, lower MRC score at nadir
van den Berg et al.[43]	2013	527 GBS patients	prospective study	older age, severity of weakness at entry, requirement for MV, delay from onset of weakness to entry, longer time to peak disability
Serrano and Rabinstein[44]	2010	85 patients admitted to the intensive care unit with acute neuromuscular respiratory failure	retrospective study	older age, longer MV, longer ICU stay
Ding et al.[45]	2022	147 GBS patients and 153 healthy individuals	case–control study	dyslipidemia
González-Suárez et al.[46]	2013	106 GBS cases	retrospective study	older age, severe deficits at onset, injured cranial nerves, requiring MV, axonal lesion patterns
Park et al.[47]	2016	47 GBS patients	retrospective study	older age, severity at admission, voiding difficulty, MV
Khedr et al.[48]	2023	62 GBS patients	prospective study	older age, the presence of an antecedent event particularly diarrhea, low MRC score at the 10th day, elevated CRP, hyponatremia, cytoalbuminous dissociation
Ishaque et al.[49]	2017	407 GBS patients	prospective study	lack of MV when it was required, autonomic dysfunction, bulbar nerve involvement, MV, longer progressive phase
Shangab and Al-Kaylani[50]	2021	82 GBS patients	retrospective study	need for MV
Verma et al.[51]	2013	90 GBS patients	prospective study	autonomic dysfunction, neck flexor weakness, MV requirement, lower MRC score on admission, axonal pattern on electrophysiological assessment
Paul et al.[52]	2012	138 GBS patients	retrospective and prospective study	presence of bulbar weakness
Beghi et al.[53]	1996	297 GBS patients	multicentre prospective study	older age, antecedent gastroenteritis, electrophysiological signs of axonopathy, latency to nadir
Walgaard et al.[54]	2011	397 GBS patients	prospective study	older age, preceding diarrhea, low MRC score at admission and at 1 week
Kobori et al.[55]	2017	4,132 GBS patients	retrospective study	coexisting cytomegalovirus, herpes simplex virus infections on admission
Di et al.[56]	2023	62 GBS patients	retrospective study	pneumonia, hyponatremia, hypoalbuminemia
Nasiri et al.[57]	2018	57 GBS patients	retrospective study	autonomic dysfunction
Alloush et al.[58]	2019	20 GBS patients	analytical observational study	need for MV, longer stay at the hospital
Wang et al.[59]	2017	523 GBS patients	retrospective study	chief complaint of weakness
Kaida et al.[60]	2007	234 GBS patients	retrospective study	ganglioside complexes (GSCs)
Lardone et al.[61]	2010	34 GBS patients	prospective study	specificity of anti-GM1 antibodies
Koga et al.[62]	2003	134 GBS patients	retrospective study	IgG1 and IgG3 subclass of anti-GM1 antibody
Bech et al.[63]	1997	17 GBS patients	prospective study	IgM anti-GM1 antibodies
Wu et al.[64]	2012	1,590 GBS patients	meta-analysis	TNF-α 308A allele
Safa et al.[65]	2020	669 GBS patients	literature review	a.o. TNF-α 308A allele
Tunç[66]	2019	81 GBS patients	retrospective study	decreased albumin and sodium levels, increased CSF protein levels, higher age, elevated NLR, higher CRP levels
Sipilä et al.[67]	2017	69 GBS patients	retrospective study	low plasma sodium level
Saifudheen et al.[68]	2011	50 GBS patients	retrospective study	age >50, ventilatory support, hyponatremia, and bulbar weakness

Table 2. A summary of the studies mentioned in a review – cont.

Author	Year of study	Number of patients	Type of study	Estimated factor
Wang et al.[69]	2015	55 GBS patients	prospective study	hyponatremia
Rumalla et al.[70]	2017	54,778 GBS patients	multicentre retrospective study	hyponatremia
Ozdemir[71]	2016	62 GBS patients	retrospective study	albumin levels, NLR and PLR
Jahan et al.[72]	2023	140 GBS patients	prospective study	elevated NLR
Sun et al.[73]	2023	136 GBS patients	retrospective study	elevated NLR
Ning et al.[74]	2021	426 GBS patients	retrospective study	NLR and PLR
Ning et al.[75]	2021	200 GBS patients	retrospective study	CAR and CRP levels
Sahin et al.[76]	2017	24 GBS patients	retrospective study	CSF protein level; NLR
Gonzalez-Quevedo et al.[77]	2009	53 GBS patients	prospective study	B-CSFB dysfunction
Bourque et al.[78]	2020	173 GBS patients	retrospective study	CSF-TP values
Bae et al.[79]	2016	85 GBS patients	prospective study	chronic inflammation and nerve ischaemia in diabetes mellitus
Wang et al.[80]	2015	304 GBS patients	prospective study	higher level of fasting plasma glucose (FPG)
Peric et al.[81]	2017	257 GBS patients	retrospective study	presence of diabetes mellitus independently of age
Gao et al.[82]	2018	112 GBS patients	retrospective study	serum folate levels
Petzold et al.[83]	2006	23 GBS patients	prospective study	high CSF NfH levels
Axelsson et al.[84]	2018	18 GBS patients	pilot study	high NFL in CSF
Martín-Aguilar et al.[85]	2020	98 + 24 samples of GBS patients	prospective study	increased sNfL levels
López-Hernández et al.[86]	2022	153 GBS patients	ambispective cohort study	deltoid muscle strength
Sundar et al.[87]	2005	46 GBS patients	retrospective study	abnormal H reflex and F waves
Miller et al.[88]	1988	60 GBS patients	prospective study	mean compound muscle action potential amplitude
Ruts et al.[89]	2012	32 GBS patients	prospective study	intraepidermal nerve fiber density (IENFD)
Grimm et al.[90]	2016	27 GBS patients	prospective study	ultrasonographic detection of cervical spinal nerve and vagus nerve enlargement
França et al.[91]	2005	18 GBS patients	retrospective study	elderly age is associated with complications after plasmapheresis
Wang et al.[92]	2017	186 GBS patients	retrospective study	no correlation between treatment options and long-term improvement

MRC – Medical Research Council; MV – mechanical ventilation; NLR – neutrophil-to-lymphocyte ratio; PLR – platelet-to-lymphocyte ratio; CAR – C-reactive protein-to-albumin ratio; CRP – C-reactive protein; CSF – cerebrospinal fluid; NLR – neutrophil/lymphocyte ratio; CSF-TP – CSF total protein; B-CSFB – blood-CSF barrier; CSF NfH – cerebrospinal fluid neurofilament; NLP – neurofilament light protein; sNfL – serum neurofilament light chain.

The probability of developing respiratory insufficiency within the 1st week can be assessed with the Erasmus GBS Respiratory Insufficiency Score (EGRIS).[96] It employs time between the onset of weakness and admission, facial and/or bulbar weakness, and MRC scores to divide patients into 3 groups according to their risk. In 2023, Luijten et al. published a modified EGRIS, which requires less information for a prediction, can be used at multiple time points, and is used in less severe cases.[97] The inability to walk unaided at 4 and 26 weeks in GBS patients can be predicted using the modified Erasmus GBS Outcome Score (mEGOS).[98]

Autonomic dysfunction, bulbar nerve involvement, papilledema, and neck flexor weakness have also been identified as factors associated with adverse outcomes in GBS patients.[48,49,51] Bulbar palsy and neck flexor weakness are often correlated with respiratory compromise and the need for MV.[51,52] Durand et al. reported that bulbar palsy was present in 38% of ventilated patients and in 10% of non-ventilated patients,[21] while Paul et al. found bulbar involvement in 92.5% of ventilated patients compared to 28.2% of non-ventilated patients.[52]

The Italian Guillain–Barré Study Group observed that the type of antecedent disorder influenced the chances of clinical recovery. Patients who experienced gastroenteritis prior to the onset of symptoms took the longest time to achieve clinical recovery, with an average duration of 292 days, whereas those with an upper respiratory infection averaged 193 days, and patients with influenza took an average of 123 days to recover.[53] This was later confirmed in studies by van Koningsveld et al., Walgaard et al. and Khedr et al., in which preceding diarrhea was an unfavorable factor for recovery at 3 and 6 months.[48,54,99] Moreover, coexisting cytomegalovirus (CMV) and herpes simplex virus (HSV) infections on admission may correlate with a higher risk of respiratory failure.[55]

The study by Dhar et al. stated that for the occurrence of severe complications, the risk difference did not reach statistical significance in terms of final recovery. However, serious ICU complications were associated with a longer time to recover.[40] A recent 2023 study found that pulmonary infections can be used as an independent predictor for a poor early prognosis in patients with GBS.[56]

A prolonged hospital stay was found to be significantly associated with a poorer prognosis. This may be attributed to the higher incidence of complications commonly associated with prolonged hospital stays, including pneumonia, sepsis and respiratory distress syndrome (RDS).[58,58]

Wang et al. found that the chief complaints of GBS patients could be clinic predictors of disease severity, the need for MV and short-term outcomes. Patients presenting with weakness as a main complaint were more likely to experience a severe disease progression and have a worse short-term outcome, while a chief complaint of numbness and cranial nerve involvement was a promising predictor.[59]

In a study conducted by Lopez–Hernandez et al., the AMAN subtype was found to be a predictor of worse short-term outcomes.[25]

Risk factors: Laboratory tests

For the clinician, the most important factor is the susceptibility of the GBS variant to standard treatment regimens. A direct predictor of therapeutic problems is the need for MV. Patients who showed the presence of anti-GD1a/GD1b and/or anti-GD1b/GT1b antibodies were most likely to have GBS with impaired spontaneous breathing.[60] The presence of antibodies against ganglioside complexes (GSCs) also determines the occurrence of symptoms such as ophthalmoplegia and lower cranial nerve deficits.[60] Gangliosides such as GD1a may interact with GM1 in cell membranes to regulate the binding and biological activity of some anti-GM1 antibodies. However, studies have shown that the high specificity of anti-GM1 antibodies in GBS is a factor defining the disease severity.[61] It is worth paying attention to the presence of anti-GM1 antibodies (immunoglobulin g; IgG) in patients with GBS due to the selection of treatment. Intravenous immunoglobulins have been proven to be more effective than plasmapheresis in patients with these antibodies.[62] Studies also show that monitoring anti-GM1 IgM levels can predict clinical status and recovery in patients with GBS.[63]

Despite the relatively small number of available studies, it should be remembered that the presence of the tumor necrosis factor alpha (TNF-α) 308A allele may be a moderate risk factor for GBS.[64] Additionally, it has been noted that GBS patients show abnormal expression of immune-related genes. Identification of GBS risk alleles may help identify risk groups, avoid triggers and design personalized therapeutic approaches.[65]

Moreover, researchers are using serum C3 complement levels as a biomarker in GBS. Higher C3 levels are associated with longer hospitalizations and more frequent treatment-related fluctuations. These patients also presented lower MRCSS and higher GBS disability scores (GBS-SDS). The clinical severity of GBS occurs with longitudinal change in C3 levels.[100]

Other important parameters that will indicate difficulties in treatment are sodium, albumin, neutrophil/lymphocyte ratio (NLR), C-reactive protein (CRP), and protein concentrations in the CSF.[48,66] Hyponatremia occurs during more severe GBS episodes but is not directly correlated with or directly specific to them. Its occurrence is probably related to the disturbance of body homeostasis.[66–68] However, analyses have proven a relationship between the results of the HDS and the ONLS and sodium concentrations in patients with a poor prognosis.[48,67,69] Sodium levels should be monitored, especially in patients with other risk factors, as they can directly affect outcomes.[70]

Low serum albumin levels accompanied more severe forms of GBS. Researchers believe that their level is a protective factor. Serum albumin plays a strong antioxidant role by inhibiting free hydroxyl radicals that are produced in the process of inflammation, demyelination and axonal damage. Therefore, it was found to be beneficial to administer human albumin in patients with GBS and hypoalbuminemia.[55,66]

Neutrophil/lymphocyte ratio and CPR are considered non-specific parameters of blood tests. However, they can confirm the diagnosis, and at the end of the 1st month of the disease, their levels have a prognostic value for a more severe course.[66,71,72] Neutrophil/lymphocyte ratio can be considered an independent risk factor for GBS.[73,74]

According to studies, a CRP >5 and protein-to-albumin ratio (CAR) >0.21 are independently associated with the occurrence of respiratory failure in patients with GBS, while a CRP >5 and CAR >0.19 predict poorer short-term outcomes in patients with GBS. The researchers suggest measuring the CAR on admission as it may be a better predictor of complications such as risk of respiratory failture than only CRP results.[75]

Elevated protein levels in the CSF are detected during inflammation of the nervous system. It has been noted that the lower the protein values at the beginning of a GBS episode, the better the prognosis.[76] High values may indicate destruction of the blood–nerve barrier.[77] It has been observed that there are higher absolute values of CSF total protein (CSF-TP) in classic sensorimotor GBS and local GBS compared to MFS and motor GBS. However, due to the weak correlation of CSF-TP and disability in GBS, it cannot be used as a factor for the modification of treatment plans.[78]

Diabetes exacerbates the clinical and electrophysiological symptoms of GBS and affects long-term disability due to the presence of chronic low-grade inflammation (elevated inflammatory markers: CRP, TNF-α and interleukin 6; IL-6).[79] Higher fasting blood glucose (FPG) levels on admission were associated with a poorer short-term

prognosis as measured using the MRCSSs and GBS disability scale at discharge. However, the development of disability is not related to blood HbA1c or CSF glucose concentrations.[80] Additionally, some diabetic patients may have pre-existing nerve damage, which exacerbates the reduced rate of nerve regeneration. It is also noted that patients with GBS and diabetes are more likely to develop the axonal form of the disease, and the electrophysiological changes in these patients are more pronounced.[81]

A significant relationship was demonstrated between folate deficiency at the time of admission and the duration of GBS progression. The exact role is unclear, but it is known that folate is essential for peripheral nerves, and its deficiency is associated with axonal sensory polyneuropathy.[82]

Long-term symptoms of GBS are caused by axonal damage. The presence of elevated levels of neurofilaments (NfH), a biomarker indicative of axonal damage, has been demonstrated to possess prognostic value in the context of GBS. Additionally, CSF NfH levels correlated with F scores and MRCSSs. These were higher in patients with neurophysiological features of axonal degeneration. The cutoff point for poorer motor and functional outcomes was defined as >0.73 ng/mL of NfH in the CSF.[83] Additionally, research shows that neurofilament light protein (NFL) should be included as an early indicator of patients requiring extensive medical and rehabilitation interventions for the long term. Patients who are severely disabled at the onset of GBS but have low concentrations of NFL in their CSF are considered to have a significantly greater chance of recovery.[84,85]

Risk factors: Neurographic tests

Neurophysiological tests can be successfully used to establish the initial diagnosis. Attempts to use them to determine the prognosis raises many doubts. There is no shortage of voices claiming that the lack of electrical excitability of the motor nerves and the lack of electrical activity in the quadriceps femoris muscle on the 10th day after the onset of the disease are independent factors for a more severe course and more difficult treatment.[101] However, more recent studies have shown that the diagnostic value of neurophysiological methods increases in proportion to the time since the onset of the disease.[102] Besides the quadriceps femoris muscle, deltoid muscle strength may also have a predictive value.[86]

Studies show that patients with markedly attenuated compound muscle action potentials (CMAPs), inexcitable motor nerves, and denervation changes on electromyography will be required to undergo MV. Nevertheless, the most prevalent abnormalities observed in both patients requiring and not requiring ventilation are abnormal H reflexes and F waves.[87,88]

Another predictive factor was found to be the intraepidermal nerve fiber density (IENFD), which correlates (decreases early and stays low for a long time) with pain intensity in the acute phase and may predict long-term disability.[89] In USG, there can be detected vagus nerve or cervical spinal nerve hypertrophy and regression of these changes within 6 months indicates a better prognosis.[90]

Risk factors: Treatment course

In the treatment of GBS, it is important to use IVIGs or plasmapheresis, which, according to researchers, maximizes survival potential.[103,104] There are also no significant differences between the use of plasma exchange and IVIG. Nevertheless, control using the ONLS indicates the advantage of treatment with IVIG.[48,91] Long-standing improvements may not be directly related to IVIG treatment but are caused by self-limitations. Despite this, studies prove that this treatment had a long-term effect on both mild and moderate-to-severe GBS.[92] It also does not seem that more intensive treatment has a significant impact on improvement in patients with advanced degrees of disability. Studies have shown that patients unable to walk on their own did not show improvement after an additional course of immunotherapy.[105]

Limitations

This systematic review has several limitations. First, only articles in English and Polish were included. No searches were made for scientific reports in other languages. Furthermore, the simultaneous appearance of some conditions or diagnostic test results and GBS episodes can be coincidental. Moreover, the heterogeneity of patients makes it difficult to combine and interpret the results, limiting conclusions. It was not possible to fully verify this data due to insufficient information about the patients.

Conclusions

This review of the literature focused on identifying prognostic factors associated with a worse outcome in patients with GBS. Scales to identify patients at high risk of mortality have also been developed to assess the course of GBS. Knowledge of these prognostic factors may, in the future, make it possible to modify the current treatment regimens for these patients.

ORCID iDs

Marta Grelowska https://orcid.org/0009-0001-6634-512X
Katarzyna Logoń https://orcid.org/0000-0002-9038-2940
Edyta Dziadkowiak https://orcid.org/0000-0002-9618-9308

References

1. Arcila-Londono X, Lewis R. Guillain–Barré syndrome. *Semin Neurol*. 2012;32(3):179–186. doi:10.1055/s-0032-1329196
2. Dimachkie MM, Barohn RJ. Guillain–Barré syndrome and variants. *Neurol Clin*. 2013;31(2):491–510. doi:10.1016/j.ncl.2013.01.005

3. Walling AD, Dickson G. Guillain–Barré syndrome. *Am Fam Physician*. 2013;87(3):191–197. PMID:23418763.
4. Van Den Berg B, Walgaard C, Drenthen J, Fokke C, Jacobs BC, Van Doorn PA. Guillain–Barré syndrome: Pathogenesis, diagnosis, treatment and prognosis. *Nat Rev Neurol*. 2014;10(8):469–482. doi:10.1038/nrneurol.2014.121
5. Kuwabara S. Guillain–Barré syndrome: Epidemiology, pathophysiology and management. *Drugs*. 2004;64(6):597–610. doi:10.2165/00003495-200464060-00003
6. Bragazzi NL, Kolahi AA, Nejadghaderi SA, et al. Global, regional, and national burden of Guillain–Barré syndrome and its underlying causes from 1990 to 2019. *J Neuroinflammation*. 2021;18(1):264. doi:10.1186/s12974-021-02319-4
7. McGrogan A, Madle GC, Seaman HE, De Vries CS. The epidemiology of Guillain–Barré syndrome worldwide. *Neuroepidemiology*. 2009;32(2):150–163. doi:10.1159/000184748
8. Wachira VK, Farinasso CM, Silva RB, Peixoto HM, De Oliveira MRF. Incidence of Guillain–Barré syndrome in the world between 1985 and 2020: A systematic review. *Glob Epidemiol*. 2023;5:100098. doi:10.1016/j.gloepi.2023.100098
9. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain–Barré syndrome: A systematic review and meta-analysis. *Neuroepidemiology*. 2011;36(2):123–133. doi:10.1159/000324710
10. Levison LS, Thomsen RW, Andersen H. Increased mortality following Guillain–Barré syndrome: A population-based cohort study. *Eur J Neurol*. 2022;29(4):1145–1154. doi:10.1111/ene.15204
11. Fragiel M, Miró Ò, Llorens P, et al. Incidence, clinical, risk factors and outcomes of Guillain–Barré in Covid-19. *Ann Neurol*. 2021;89(3):598–603. doi:10.1002/ana.25987
12. Palaiodimou L, Stefanou M, Katsanos AH, et al. Prevalence, clinical characteristics and outcomes of Guillain–Barré syndrome spectrum associated with COVID-19: A systematic review and meta-analysis. *Eur J Neurol*. 2021;28(10):3517–3529. doi:10.1111/ene.14860
13. Lawn ND, Wijdicks EFM. Fatal Guillain–Barré syndrome. *Neurology*. 1999;52(3):635–635. doi:10.1212/WNL.52.3.635
14. Alshekhlee A, Hussain Z, Sultan B, Katirji B. Guillain–Barré syndrome: Incidence and mortality rates in US hospitals. *Neurology*. 2008;70(18):1608–1613. doi:10.1212/01.wnl.0000310983.38724.d4
15. Khan F, Pallant JF, Ng L, Bhasker A. Factors associated with long-term functional outcomes and psychological sequelae in Guillain–Barré syndrome. *J Neurol*. 2010;257(12):2024–2031. doi:10.1007/s00415-010-5653-x
16. Ruts L, Drenthen J, Jongen JLM, et al. Pain in Guillain–Barré syndrome: A long-term follow-up study. *Neurology*. 2010;75(16):1439–1447. doi:10.1212/WNL.0b013e3181f88345
17. Forsberg A, Press R, Einarsson U, De Pedro-Cuesta J, Widén Holmqvist L. Impairment in Guillain–Barré syndrome during the first 2 years after onset: A prospective study. *J Neurol Sci*. 2004;227(1):131–138. doi:10.1016/j.jns.2004.09.021
18. Meythaler JM. Rehabilitation of Guillain–Barré syndrome. *Arch Phys Med Rehabil*. 1997;78(8):872–879. doi:10.1016/S0003-9993(97)90203-3
19. Bernsen RAJAM, De Jager AEJ, Van Der Meche FGA, Suurmeijer TPBM. How Guillain–Barré patients experience their functioning after 1 year. *Acta Neurol Scand*. 2005;112(1):51–56. doi:10.1111/j.1600-0404.2005.00429.x
20. Forsberg A, Press R, Holmqvist LW. Residual disability 10 years after falling ill in Guillain–Barré syndrome: A prospective follow-up study. *J Neurol Sci*. 2012;317(1–2):74–79. doi:10.1016/j.jns.2012.02.026
21. Durand MC, Porcher R, Orlikowski D, et al. Clinical and electrophysiological predictors of respiratory failure in Guillain–Barré syndrome: A prospective study. *Lancet Neurol*. 2006;5(12):1021–1028. doi:10.1016/S1474-4422(06)70603-2
22. Bersano A, Carpo M, Allaria S, Franciotta D, Citterio A, Nobile-Orazio E. Long term disability and social status change after Guillain–Barré syndrome. *J Neurol*. 2006;253(2):214–218. doi:10.1007/s00415-005-0958-x
23. Yi SW, Lee JH, Hong JM, Choi YC, Park HJ. Incidence, disability, and mortality in patients with Guillain–Barré syndrome in Korea: A nationwide population-based study. *J Clin Neurol*. 2022;18(1):48. doi:10.3988/jcn.2022.18.1.48
24. Zhang G, Li Q, Zhang R, Wei X, Wang J, Qin X. Subtypes and prognosis of Guillain–Barré syndrome in Southwest China. *PLoS One*. 2015;10(7):e0133520. doi:10.1371/journal.pone.0133520
25. López-Hernández JC, Colunga-Lozano LE, Garcia-Trejo S, et al. Electrophysiological subtypes and associated prognosis factors of Mexican adults diagnosed with Guillain–Barré syndrome: A single center experience. *J Clin Neurosci*. 2020;80:292–297. doi:10.1016/j.jocn.2020.04.059
26. Kiraz M, Yılgör A, Milanlıoğlu A, Çilingir V, Çağaç A, Özkan S. Clinical subtypes, seasonality, and short-term prognosis of Guillain–Barré syndrome in an Eastern city of Turkey. *NeuroAsia*. 2022;27(4):937–944. doi:10.54029/2022wak
27. Bai HX, Wang Z, Tan L, Xiao B, Goldstein JM, Yang L. The effectiveness of immunomodulating treatment on Miller–Fisher syndrome: A retrospective analysis of 65 Chinese patients. *J Peripher Nerv Syst*. 2013;18(2):195–196. doi:10.1111/jns5.12030
28. Van Doorn PA, Van den Bergh PYK, Hadden RDM, et al. European Academy of Neurology/Peripheral Nerve Society Guideline on diagnosis and treatment of Guillain–Barré syndrome. *Eur J Neurol*. 2023;30(12):3646–3674. doi:10.1111/ene.16073
29. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain–Barré syndrome. *Ann Neurol*. 1990;27(Suppl 1):S21–S24. doi:10.1002/ana.410270707
30. Sejvar JJ, Kohl KS, Gidudu J, et al. Guillain–Barré syndrome and Fisher syndrome: Case definitions and guidelines for collection, analysis, and presentation of immunization safety data. *Vaccine*. 2011;29(3):599–612. doi:10.1016/j.vaccine.2010.06.003
31. Nomura K. Guillain–Barré syndrome: Clinical features, immune mechanisms, and therapies [in Japanese]. *Rinsho Shinkeigaku*. 1996;36(12):1367–1369. PMID:9128414.
32. Guillain G, Barré JA, Strohl A. Radiculoneuritis syndrome with hyperalbuminosis of cerebrospinal fluid without cellular reaction: Notes on clinical features and graphs of tendon reflexes (1916) [in French]. *Ann Med Interne (Paris)*. 1999;150(1):24–32. PMID:10400560.
33. Vucic S, Cairns KD, Black KR, Tick Chong PS, Cros D. Neurophysiologic findings in early acute inflammatory demyelinating polyradiculoneuropathy. *Clin Neurophysiol*. 2004;115(10):2329–2335. doi:10.1016/j.clinph.2004.05.009
34. Rajabally YA, Durand MC, Mitchell J, Orlikowski D, Nicolas G. Electrophysiological diagnosis of Guillain–Barré syndrome subtype: Could a single study suffice? *J Neurol Neurosurg Psychiatry*. 2015;86(1):115–119. doi:10.1136/jnnp-2014-307815
35. Prineas JW. Pathology of the Guillain–Barré syndrome. *Ann Neurol*. 1981;9(Suppl 1):6–19. doi:10.1002/ana.410090704
36. Yuki N, Taki T, Takahashi M, et al. Molecular mimicry between GQ_{1b} ganglioside and lipopolysaccharides of *Campylobacter jejuni* isolated from patients with Fisher's syndrome. *Ann Neurol*. 1994;36(5):791–793. doi:10.1002/ana.410360517
37. Hughes RA, Swan AV, Van Doorn PA. Intravenous immunoglobulin for Guillain–Barré syndrome. *Cochrane Database Syst Rev*. 2014;2014(9):CD002063. doi:10.1002/14651858.CD002063.pub6
38. Chevret S, Hughes RA, Annane D. Plasma exchange for Guillain–Barré syndrome. *Cochrane Database Syst Rev*. 2017;2017(2):CD001798. doi:10.1002/14651858.CD001798.pub3
39. Shangab M, Al Kaylani M. Clinical course and predictors of poor functional outcome in Guillain–Barré syndrome: A retrospective study. *Dubai Med J*. 2020;3(3):93–98. doi:10.1159/000510443
40. Dhar R, Stitt L, Hahn AF. The morbidity and outcome of patients with Guillain–Barré syndrome admitted to the intensive care unit. *J Neurol Sci*. 2008;264(1–2):121–128. doi:10.1016/j.jns.2007.08.005
41. Wen P, Wang L, Liu H, et al. Risk factors for the severity of Guillain–Barré syndrome and predictors of short-term prognosis of severe Guillain–Barré syndrome. *Sci Rep*. 2021;11(1):11578. doi:10.1038/s41598-021-91132-3
42. Zhang B, Wu X, Shen D, et al. The clinical characteristics and short-term prognosis in elderly patients with Guillain–Barré syndrome. *Medicine (Baltimore)*. 2017;96(1):e5848. doi:10.1097/MD.0000000000005848
43. Van Den Berg B, Bunschoten C, Van Doorn PA, Jacobs BC. Mortality in Guillain–Barré syndrome. *Neurology*. 2013;80(18):1650–1654. doi:10.1212/WNL.0b013e3182904fcc
44. Cabrera Serrano M, Rabinstein AA. Causes and outcomes of acute neuromuscular respiratory failure. *Arch Neurol*. 2010;67(9):1089–1094. doi:10.1001/archneurol.2010.207
45. Ding Y, Wang L, Sun J, et al. Remnant cholesterol and dyslipidemia are risk factors for Guillain–Barré syndrome and severe Guillain–Barré syndrome by promoting monocyte activation. *Front Immunol*. 2022;13:946825. doi:10.3389/fimmu.2022.946825

46. González-Suárez I, Sanz-Gallego I, Rodríguez De Rivera FJ, Arpa J. Guillain–Barré syndrome: Natural history and prognostic factors. A retrospective review of 106 cases. *BMC Neurol*. 2013;13(1):95. doi:10.1186/1471-2377-13-95
47. Park SH, Kim NH. Early prediction factors of poor outcome in Guillain–Barré syndrome. *Soonchunhyang Med Sci*. 2016;22(2):79–82. doi:10.15746/sms.16.018
48. Khedr EM, Mohamed MZ, Shehab MMM. The early clinical and laboratory predictors of GBS outcome: Hospital-based study, Assiut University, Upper Egypt. *Egypt J Neurol Psychiatry Neurosurg*. 2023;59(1):45. doi:10.1186/s41983-023-00646-2
49. Ishaque T, Islam MB, Ara G, et al. High mortality from Guillain–Barré syndrome in Bangladesh. *J Peripher Nerv Syst*. 2017;22(2):121–126. doi:10.1111/jns.12215
50. Shangab M, Al Kaylani M. Clinical predictors for mechanical ventilation and prognosis in patients with Guillain–Barré syndrome: A 10-year experience. *Neurol Sci*. 2021;42(12):5305–5309. doi:10.1007/s10072-021-05251-w
51. Verma R, Chaudhari TS, Raut TP, Garg RK. Clinico-electrophysiological profile and predictors of functional outcome in Guillain–Barré syndrome (GBS). *J Neurol Sci*. 2013;335(1–2):105–111. doi:10.1016/j.jns.2013.09.002
52. Paul BS, Bhatia R, Prasad K, Padma MV, Tripathi M, Singh MB. Clinical predictors of mechanical ventilation in Guillain–Barré syndrome. *Neurol India*. 2012;60(2):150. doi:10.4103/0028-3886.96383
53. Beghi E. The prognosis and main prognostic indicators of Guillain–Barré syndrome: A multicentre prospective study of 297 patients. *Brain*. 1996;119(6):2053–2061. doi:10.1093/brain/119.6.2053
54. Walgaard C, Lingsma HF, Ruts L, Van Doorn PA, Steyerberg EW, Jacobs BC. Early recognition of poor prognosis in Guillain–Barré syndrome. *Neurology*. 2011;76(11):968–975. doi:10.1212/WNL.0b013e3182104407
55. Kobori S, Kubo T, Otani M, et al. Coexisting infectious diseases on admission as a risk factor for mechanical ventilation in patients with Guillain–Barré syndrome. *J Epidemiol*. 2017;27(7):311–316. doi:10.1016/j.je.2016.07.003
56. Di X, Wang J, Li L, Liu L. Establishment of a single-center-based early prognostic scoring system for Guillain–Barré syndrome. *BMC Neurol*. 2023;23(1):97. doi:10.1186/s12883-023-03143-4
57. Nasiri J, Ghazavi M, Yaghini O, Chaldavi M. Clinical features and outcome of Guillain–Barré syndrome in children. *Iran J Child Neurol*. 2018;12(2):49–57. PMID:29696046. PMCID:PMC5904738.
58. Alloush T, Fahmy NA, Fouad MM, Albaroudy HO, Hamdy M, Salem HH. Prediction of outcome in patients with Guillain–Barré syndrome: An Egyptian study. *Neurosci Med*. 2019;10(3):232–246. doi:10.4236/NM.2019.103018
59. Wang Y, Shang P, Xin M, Bai J, Zhou C, Zhang HL. The usefulness of chief complaints to predict severity, ventilator dependence, treatment option, and short-term outcome of patients with Guillain–Barré syndrome: A retrospective study. *BMC Neurol*. 2017;17(1):200. doi:10.1186/s12883-017-0982-3
60. Kaida K, Morita D, Kanzaki M, et al. Anti-ganglioside complex antibodies associated with severe disability in GBS. *J Neuroimmunol*. 2007;182(1–2):212–218. doi:10.1016/j.jneuroim.2006.09.013
61. Lardone RD, Yuki N, Odaka M, Daniotti JL, Irazoqui FJ, Nores GA. Anti-GM1 IgG antibodies in Guillain–Barré syndrome: Fine specificity is associated with disease severity. *J Neurol Neurosurg Psychiatry*. 2010;81(6):629–633. doi:10.1136/jnnp.2009.183665
62. Koga M, Yuki N, Hirata K, Morimatsu M, Mori M, Kuwabara S. Anti-GM1 antibody IgG subclass: A clinical recovery predictor in Guillain–Barré syndrome. *Neurology*. 2003;60(9):1514–1518. doi:10.1212/01.WNL.0000061615.77865.83
63. Bech E, Ørntoft TF, Andersen LP, Skinhøj P, Jakobsen J. IgM anti-GM1 antibodies in the Guillain–Barré syndrome: A serological predictor of the clinical course. *J Neuroimmunol*. 1997;72(1):59–66. doi:10.1016/S0165-5728(96)00145-2
64. Wu LY, Zhou Y, Qin C, Hu BL. The effect of TNF-alpha, FcγR and CD1 polymorphisms on Guillain–Barré syndrome risk: Evidences from a meta-analysis. *J Neuroimmunol*. 2012;243(1–2):18–24. doi:10.1016/j.jneuroim.2011.12.003
65. Safa A, Azimi T, Sayad A, Taheri M, Ghafouri-Fard S. A review of the role of genetic factors in Guillain–Barré syndrome. *J Mol Neurosci*. 2021;71(5):902–920. doi:10.1007/s12031-020-01720-7
66. Tunç A. Early predictors of functional disability in Guillain–Barré syndrome. *Acta Neurol Belg*. 2019;119(4):555–559. doi:10.1007/s13760-019-01133-3
67. Sipilä JOT, Kauko T, Soilu-Hänninen M. Admission sodium level and prognosis in adult Guillain–Barré syndrome. *Int J Neurosci*. 2017;127(4):344–349. doi:10.3109/00207454.2016.1163551
68. Saifudheen K, Jose J, Gafoor VA, Musthafa M. Guillain-Barré syndrome and SIADH. *Neurology*. 2011;76(8):701–704. doi:10.1212/WNL.0b013e31820d8b40
69. Wang Y, Liu J. Hyponatremia is a predictor for poor outcome in Guillain–Barré syndrome. *Neurol Res*. 2015;37(4):347–351. doi:10.1179/1743132814Y.0000000455
70. Rumalla K, Reddy AY, Letchuman V, Mittal MK. Hyponatremia in Guillain–Barré syndrome. *J Clin Neuromuscul Dis*. 2017;18(4):207–217. doi:10.1097/CND.0000000000000157
71. Ozdemir HH. Analysis of the albumin level, neutrophil-lymphocyte ratio, and platelet-lymphocyte ratio in Guillain–Barré syndrome. *Arq Neuropsiquiatr*. 2016;74(9):718–722. doi:10.1590/0004-282X20160132
72. Jahan I, Ahmed R, Ahmed J, et al. Neutrophil-lymphocyte ratio in Guillain–Barré syndrome: A prognostic biomarker of severe disease and mechanical ventilation in Bangladesh. *J Peripher Nerv Syst*. 2023;28(1):47–57. doi:10.1111/jns.12531
73. Sun S, Wen Y, Li S, Huang Z, Zhu J, Li Y. Neutrophil-to-lymphocyte ratio is a risk indicator of Guillain–Barré syndrome and is associated with severity and short-term prognosis. *Heliyon*. 2023;9(3):e14321. doi:10.1016/j.heliyon.2023.e14321
74. Ning P, Yang B, Yang X, et al. Lymphocyte-based ratios for predicting respiratory failure in Guillain–Barré syndrome. *J Neuroimmunol*. 2021;353:577504. doi:10.1016/j.jneuroim.2021.577504
75. Ning P, Yang B, Yang X, et al. Clinical value of C-reactive protein/albumin ratio in Guillain–Barré syndrome. *Neurol Sci*. 2021;42(8):3275–3283. doi:10.1007/s10072-020-04930-4
76. Sahin S, Cinar N, Karsidag S. Are cerebrospinal fluid protein levels and plasma neutrophil/lymphocyte ratio associated with prognosis of Guillain–Barré syndrome? *Neurol Int*. 2017;9(2):7032. doi:10.4081/ni.2017.7032
77. Gonzalez-Quevedo A, Carriera Fernandez R, O'Farrill Lestayo Z, Luis Suarez I, Mustelier Becquer R, Luis Gonzalez RS. An appraisal of blood-cerebrospinal fluid barrier dysfunction during the course of Guillain–Barré syndrome. *Neurol India*. 2009;57(3):288. doi:10.4103/0028-3886.53282
78. Bourque PR, Brooks J, Warman-Chardon J, Breiner A. Cerebrospinal fluid total protein in Guillain–Barré syndrome variants: Correlations with clinical category, severity, and electrophysiology. *J Neurol*. 2020;267(3):746–751. doi:10.1007/s00415-019-09634-0
79. Bae JS, Kim YJ, Kim JK. Diabetes mellitus exacerbates the clinical and electrophysiological features of Guillain–Barré syndrome. *Eur J Neurol*. 2016;23(3):439–446. doi:10.1111/ene.12885
80. Wang Y, Li G, Yang S, et al. Fasting glucose levels correlate with disease severity of Guillain–Barré syndrome. *PLoS One*. 2015;10(12):e0145075. doi:10.1371/journal.pone.0145075
81. Peric S, Bozovic I, Bjelica B, et al. Diabetes mellitus may affect short-term outcome of Guillain–Barré syndrome. *J Peripher Nerv Syst*. 2017;22(2):127–130. doi:10.1111/jns.12206
82. Gao Y, Zhang HL, Xin M, et al. Serum folate correlates with severity of Guillain–Barré syndrome and predicts disease progression. *Biomed Res Int*. 2018;2018:5703279. doi:10.1155/2018/5703279
83. Petzold A, Hinds N, Murray NF, et al. CSF neurofilament levels: A potential prognostic marker in Guillain–Barré syndrome. *Neurology*. 2006;67(6):1071–1073. doi:10.1212/01.wnl.0000237334.69665.92
84. Axelsson M, Sjögren M, Andersen O, Blennow K, Zetterberg H, Lycke J. Neurofilament light protein levels in cerebrospinal fluid predict long-term disability of Guillain–Barré syndrome: A pilot study. *Acta Neurol Scand*. 2018;138(2):143–150. doi:10.1111/ane.12927
85. Martín-Aguilar L, Camps-Renom P, Lleixà C, et al. Serum neurofilament light chain predicts long-term prognosis in Guillain–Barré syndrome patients. *J Neurol Neurosurg Psychiatry*. 2021;92(1):70–77. doi:10.1136/jnnp-2020-323899
86. López-Hernández JC, Jorge De Saráchaga A, Briseño-Godínez ME, et al. Deltoid muscle strength and autonomic dysfunction as independent risk factors for invasive mechanical ventilation in patients with Guillain–Barré syndrome. *Int J Neurosci*. 2023;133(12):1403–1410. doi:10.1080/00207454.2022.2082963

87. Sundar U, Abraham E, Gharat A, Yeolekar ME, Trivedi T, Dwivedi N. Neuromuscular respiratory failure in Guillain–Barré syndrome: Evaluation of clinical and electrodiagnostic predictors. *J Assoc Physicians India*. 2005;53:764–768. PMID:16334619.
88. Miller RG, Peterson GW, Daube JR, Albers JW. Prognostic value of electrodiagnosis in Guillain–Barré syndrome. *Muscle Nerve*. 1988;11(7): 769–774. doi:10.1002/mus.880110714
89. Ruts L, Van Doorn PA, Lombardi R, et al. Unmyelinated and myelinated skin nerve damage in Guillain–Barré syndrome: Correlation with pain and recovery. *Pain*. 2012;153(2):399–409. doi:10.1016/j.pain.2011.10.037
90. Grimm A, Décard BF, Schramm A, et al. Ultrasound and electrophysiologic findings in patients with Guillain–Barré syndrome at disease onset and over a period of six months. *Clin Neurophysiol*. 2016; 127(2):1657–1663. doi:10.1016/j.clinph.2015.06.032
91. França Jr MC, Deus-Silva L, Castro RD, et al. Guillain-Barré syndrome in the elderly: Clinical, electrophysiological, therapeutic and outcome features. *Arq Neuropsiquiatr*. 2005;63(3B):772–775. doi:10.1590/S0004-282X2005000500010
92. Wang Y, Lang W, Zhang Y, Ma X, Zhou C, Zhang HL. Long-term prognosis of Guillain–Barré syndrome not determined by treatment options? *Oncotarget*. 2017;8(45):79991–80001. doi:10.18632/oncotarget.20620
93. Rajabally YA, Uncini A. Outcome and its predictors in Guillain–Barré syndrome. *J Neurol Neurosurg Psychiatry*. 2012;83(7):711–718. doi:10.1136/jnnp-2011-301882
94. Kleyweg RP, Van Der Meché FGA, Schmitz PIM. Interobserver agreement in the assessment of muscle strength and functional abilities in Guillain–Barré syndrome. *Muscle Nerve*. 1991;14(11):1103–1109. doi:10.1002/mus.880141111
95. Hughes RAC, Newsom-Davis JM, Perkin GD, Pierce JM. Controlled trial prednisolone in acute polyneuropathy. *Lancet*. 1978;312(8093): 750–753. doi:10.1016/S0140-6736(78)92644-2
96. Walgaard C, Lingsma HF, Ruts L, et al. Prediction of respiratory insufficiency in Guillain–Barré syndrome. *Ann Neurol*. 2010;67(6):781–787. doi:10.1002/ana.21976
97. Luijten LWG, Doets AY, Arends S, et al. Modified Erasmus GBS Respiratory Insufficiency Score: A simplified clinical tool to predict the risk of mechanical ventilation in Guillain–Barré syndrome. *J Neurol Neurosurg Psychiatry*. 2023;94(4):300–308. doi:10.1136/jnnp-2022-329937
98. Doets AY, Lingsma HF, Walgaard C, et al. Predicting outcome in Guillain–Barré syndrome: International validation of the Modified Erasmus GBS Outcome Score. *Neurology*. 2022;98(5):e518–e532. doi:10.1212/WNL.0000000000013139
99. Van Koningsveld R, Steyerberg EW, Hughes RA, Swan AV, Van Doorn PA, Jacobs BC. A clinical prognostic scoring system for Guillain–Barré syndrome. *Lancet Neurol*. 2007;6(7):589–594. doi:10.1016/S1474-4422(07)70130-8
100. Min YG, Ju W, Seo JW, et al. Serum C3 complement levels predict prognosis and monitor disease activity in Guillain–Barré syndrome. *J Neurol Sci*. 2023;444:120512. doi:10.1016/j.jns.2022.120512
101. Ortiz Corredor F, Mieth Alviar KW. Prognostic factors for walking in childhood Guillain–Barré syndrome [in Spanish]. *Rev Neurol*. 2003;36(12):1113. doi:10.33588/rn.3612.2003056
102. Kroczka S, Świerczyńska, Kaciński M. Kliniczne i neurofizjologiczne następstwa zespołu Guillain-Barre. *Przegl Lek*. 2011;68(11):1098–1103. https://ruj.uj.edu.pl/server/api/core/bitstreams/859b996a-0270-4b82-a90b-244232beda4e/content. Accessed May 4, 2024.
103. Cheng B, Chang W, Chen J, et al. Long-term prognosis for Guillain–Barré syndrome: Evaluation of prognostic factors and clinical experience of automated double filtration plasmapheresis. *J Clin Apher*. 2003;18(4):175–180. doi:10.1002/jca.10066
104. Liu S, Dong C, Ubogu EE. Immunotherapy of Guillain–Barré syndrome. *Hum Vaccin Immunother*. 2018;14(11):2568–2579. doi:10.1080/21645515.2018.1493415
105. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain–Barré syndrome. *Lancet*. 2021;397(10280):1214–1228. doi:10.1016/S0140-6736(21)00517-1