Respiratory Dysfunction In Guillain-Barré Syndrome (Gbs): A Narrative Review

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ABSTRACT: Guillain-Barré Syndrome (GBS) is an acute, progressive autoimmune disorder of the peripheral nervous system that leads to bilateral limb weakness and paralysis. Respiratory dysfunction is one of the most serious and life-threatening complications, affecting approximately 20–30% of patients and significantly contributing to morbidity and mortality. This narrative review explores the causes and predictors of respiratory dysfunction in GBS and emphasizes the importance of early recognition and intervention for improved outcomes. An extensive literature search was conducted using databases such as PubMed, Google Scholar, ScienceDirect, and Medscape, reviewing studies published between 2019 and 2025, including cases related to COVID-19 and pediatric populations.
Respiratory failure in GBS primarily results from weakness of the diaphragm and accessory respiratory muscles, cranial nerve involvement, and bulbar dysfunction. Key predictors of the need for mechanical ventilation include low Medical Research Council (MRC) scores, facial and bulbar weakness, autonomic dysfunction, and axonal variants such as Axonal Neuropathy (AMAN) Acute Motor and Acute Motor and Sensory Axonal Neuropathy (AMSAN). Pediatric patients, especially those with the AMAN subtype, are at higher risk. Early identification using tools such as the Hughes GBS Disability Scale (HGDS) and biomarkers like the C-reactive protein to albumin ratio (CAR) can guide timely respiratory support.
In conclusion, respiratory dysfunction in GBS is multifactorial and frequently necessitates intensive care. Early detection and intervention are crucial to improving prognosis and reducing respiratory complications.

Index Terms: Guillain–Barre Syndrome, Respiratory dysfunction, mechanical ventilation AMAN, AMSAN, demyelinating polyneuritis.

INTRODUCTION

Guillain-Barre Syndrome (GBS) is an autoimmune condition that is characterized by sudden muscle paralysis because of the immune system attacking the peripheral nervous system. Demyelinating polyneuritis is also another name for Guillain-Barré syndrome. [1] It affects one in every 100,000 people, both adults and children, and is frequently triggered by acute infections, resulting in an immune-mediated response that reacts with the peripheral nerves' myelin sheath, causing demyelination and axonal injury. In severe cases GBS may lead to respiratory failure and even death.[2] GBS initially manifests as pain, paraesthesia, numbness and other symptom like bulbar muscle weakness, respiratory failure, generalized axial and peripheral limb paralysis, with gradual increase in limb weakness and a decrease in or elimination of the tendinous reflex are the usual clinical symptoms. About 30% of Guillain-Barre Syndrome (GBS) patients have respiratory failure, necessitating the need of mechanical ventilation (MV) support and endotracheal intubation. Consequently, respiratory failure is the leading cause of death for people with GBS, making it a serious and potentially fatal condition. Severe GBS patients also need to be closely monitored in an intensive care unit (ICU) and may require artificial ventilation to survive. Determining the disease's severity early on and establishing suitable guidelines for allocating GBS patients to the general ward or ICU are crucial for reducing the risks of respiratory distress and death.[3]

EPIDEMIOLOGY

A comprehensive epidemiological analysis from 2009, primarily based on data from North America and Europe, revealed that Guillain-Barré Syndrome (GBS) occurs in approximately 1.1 to 1.8 individuals per 100,000 adults annually. The incidence is lower in children under the age of 16. The condition tends to be more common in males, and the likelihood increases with age, with an increase in risk per decade. Seasonal patterns have also been noted, with higher rates of occurrence during winter, likely due to rise of infections.[4]

In India, GBS affects an estimated 6 to 40 individuals per million each year. A distinct seasonal trend is evident, with a noticeable increase during the monsoon season. For a population of **1.2 million**, this corresponds to approximately 7 to 48 cases per year, or **0.58** to 4 per month. In some reports, this is **1.4 to 10** times higher than the expected average, with 7 cases translating to 5.8 per million. [5]

SUBTYPES

Guillain-Barre syndrome (GBS) has several subtypes, each having its own clinical characteristics and each subtype varies in prognosis and risks.

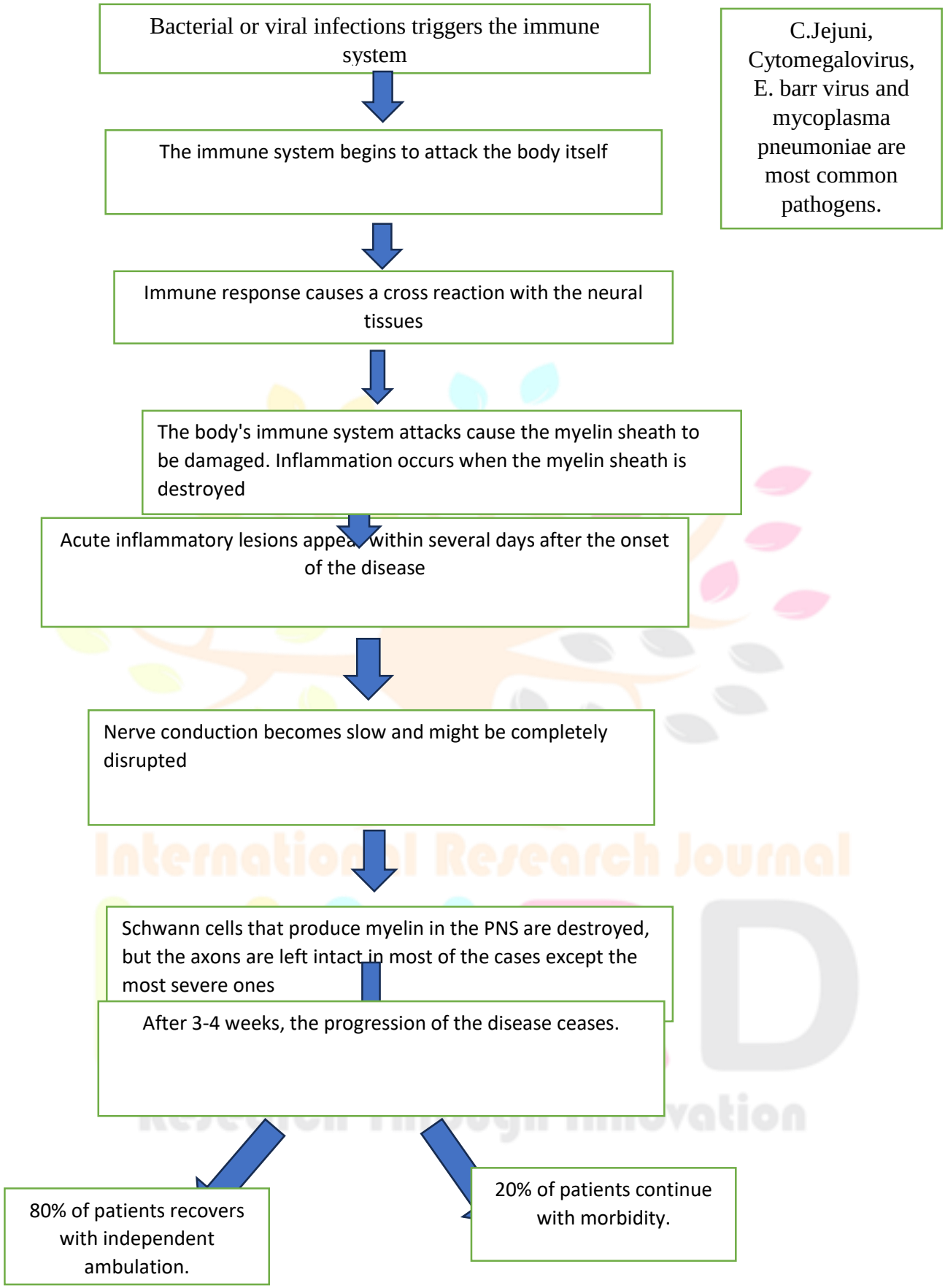
- Acute inflammatory demyelinating polyneuropathy (AIDP)
- Acute Motor Axonal Neuropathy (AMAN)
- Acute Motor and Sensory Axonal Neuropathy (AMSAN)
- Miller Fisher Syndrome (MFS)
- Isolated Bilateral Facial Paralysis (IBFP)

ETIOLOGY AND PATHOPHYSIOLOGY

The etiology of Guillain-Barre Syndrome (GBS) is complex and multifactorial. GBS is an immune-mediated disease targeting the peripheral nervous system. [10,11,12]

- Antecedent Infections: Patients say up to two-thirds had a prior bacterial or viral disease before neurological symptoms started. The most prevalent pathogens are Campylobacter jejuni, Epstein-Barr Virus, Cytomegalovirus, Mycoplasma pneumoniae, Hepatitis A and B &Varicella zoster.
- Immunological Mechanisms; It is not exactly understandable, but studies suggest that molecular mimicry plays a key role. This occurs when the immune system unknowingly attacks the peripheral nervous system.
- Specific Antigens and Antibodies; Certain antigens like Lipopolysaccharides and Gangliosides are thought to be triggering the immune response. The resulting antibodies cross-reacts with myelin sheath leading to demyelination and axonal damage.

Understanding the etiology of GBS is important to develop appropriate and effective prevention and treatment strategies.



Around 70% of individuals with Guillain barre syndrome (GBS) have been reported to have antecedent infections. Molecular mimicry thus aids in the understanding of GBS, particularly the axonal variant. The gangliosides found in plasma membranes of the peripheral nerves are like the lipo-oligosaccharides of Campylobacter jejuni. Numerous peripheral neurons have been demonstrated to be the target of ganglioside antibodies. The anti-GQ1B antibody, for instance, is found in patients with Miller-Fisher syndrome. The

anti-GM1 antibodies may be linked to this variant. Acute inflammatory demyelinating polyneuropathy (AIDP), the most prevalent kind of GBS in the United States, has the least understood pathogenesis.[13]

SIGNS AND SYMPTOMS:

Guillain-Barré Syndrome (GBS) typically manifests 2-4 weeks after an infection and weakness can advance over a couple of hours to days. [11] It presents with rapid progressive, limb weakness that begins in the lower extremities and ascends proximally. Areflexia or hyporeflexia is characteristic, and sensory symptoms such as paraesthesia, numbness, sensory impairment and limb pain may precede weakness. Cranial nerve involvement of the facial and bulbar muscles can lead to dysphagia, dysarthria, and facial drooping. In severe cases, weakness of the diaphragm and intercostal muscles causes respiratory compromise, often requiring ventilatory support. Autonomic dysfunction presents as tachycardia or bradycardia, blood pressure fluctuations, or urinary retention. Pain and fatigue are common during both acute and recovery phases.

DIAGNOSIS AND TREATMENT

The diagnosis of GBS is primarily clinical, supported by cerebrospinal fluid (CSF) analysis and electrodiagnosis. Clinically, patients present with progressive, bilateral weakness of limbs accompanied by diminished or absent deep tendon reflexes, typically reaching maximal severity within four weeks. CSF examination often reveals albuminocytological dissociation, characterized by elevated protein levels with normal or low cell counts. Nerve conduction studies (NCS) and electromyography (EMG) confirm demyelination or axonal degeneration, The presence of anti-ganglioside antibodies, particularly anti-GQ1b, may aid diagnosis in specific variants such as Miller Fisher Syndrome. [14,15]

Patients with GBS are prone to complications, some potentially fatal, making continuous monitoring crucial. Those with respiratory failure or autonomic dysfunction should be managed in an intensive care unit (ICU) for respiratory and haemodynamic support. [4] Plasmapheresis and intravenous immunoglobulin (IVIg) are the only proven treatments for GBS, though optimal dosing and duration remain unclear. Disease severity varies, complicating treatment decisions.

A significant prognostic indicator of severe GBS is the requirement for mechanical ventilation and endotracheal intubation. early identificati**on** of patients likely to require ventilatory support is critical. [3]

So, the objective of this review is to identify Respiratory dysfunction of Guillain barre syndrome (GBS) patient.

REVIEW OF LITERATURE

Author (Year)	Study Objective / Title	Sample / Study Design	Key Findings	Relevance to Present Study
Galassi et al. (2023)	Predictors of mechanical ventilation in GBS	230 adult GBS patients, multicenter retrospective	13.9% required MV; 84% of those within 7 days. Dysautonomia and axonal subtype were independent predictors; neck/facial/bulbar weakness predicted tracheostomy.	Directly supports prevalence and predictors reported in Results/Findings.
Melone et al. (2020)	Early mechanical ventilation in high-risk GBS patients	56 adults, randomized trial (EMV vs control)	Early MV did not significantly change mortality, pneumonia rate, or long-term	Provides evidence for individualized ventilatory decision-making

	(randomized trial)		outcomes despite higher MV use in EMV group.	referenced in Results.
Yao et al. (2023)	Early predictors of mechanical ventilation in GBS (HGDS and albumin)	145 GBS patients, retrospective	Higher Hughes GBS Disability Scale (HGDS) scores and shorter onset-to-admission times predicted MV requirement; low albumin associated with poor prognosis.	Supports use of HGDS and albumin combined as prognostic markers in Results.
Ning et al. (2021)	Predictors for mechanical ventilation and short-term prognosis in GBS (CAR)	200 patients, observational	C-reactive protein-to-albumin ratio (CAR) >0.19 associated with worse outcomes and higher risk of respiratory failure.	Underpins CAR discussion as a biochemical predictor in Results.
Debnath et al. (2022)	Subtype distribution and ventilatory needs in pediatric GBS	83 pediatric patients, cohort (Bangladesh)	AMAN subtype associated with higher rates of respiratory distress and greater need for assisted ventilation; longer recovery compared with AIDP.	Provides pediatric and subtype-specific data referenced in Results.
Maskin et al. (2021)	Risk factors for respiratory failure among hospitalized GBS patients	113 patients, observational	Cranial nerve involvement, facial palsy, bulbar weakness, and low MRC scores significantly predicted respiratory failure (ORs reported).	Offers validated clinical predictors consistent with Results/Findings.
Islam et al. (2019)	Risk factors for respiratory failure in GBS (Bangladesh cohort)	507 GBS patients, prospective	22% required MV; bulbar palsy, dysautonomia, and facial weakness were significant predictors.	Large cohort evidence supporting predictor prevalence used in Results.
Gonzalez et al. (2019)	Risk factors for respiratory failure among hospitalized GBS patients	135 patients, cohort (South America)	25.9% required ICU/respiratory support; lower limb areflexia and facial weakness useful for triage and risk stratification.	Supports triage and bedside predictor points in Results.
Wang et al. (2023)	Clinical predictors of respiratory	455 adult patients, retrospective	129 patients developed respiratory muscle	Adds large-sample confirmation of clinical scoring

	muscle paralysis in GBS		paralysis; higher EGRIS and Hughes scores, lower MRC, and bulbar palsy predicted paralysis.	and predictors discussed in Results.
Yadav et al. (2021)	Assessment of inflammatory markers and ventilatory needs in GBS	86 adult patients, observational	39.5% required invasive MV; AMSAN subtype had highest ventilation need; developed predictive models for IMV.	Supports subtype-related risk (axonal/AMSAN) and predictive modelling in Results.
Patel et al. (2022)	Respiratory distress in GBS: predictors from a tertiary center in India	94 patients, retrospective	27.6% developed respiratory distress; dysautonomia and bulbar palsy were key predictors.	Regional data aligning with Results' predictor findings.
Cheng et al. (2022)	Clinical predictors of respiratory failure in GBS	252 patients, retrospective	12.3% required MV; male sex, shorter onset-to-admission, lower MRC sum score, NLR, and cranial deficits predicted MV.	Supports multiple clinical and lab predictors included in Results.
Michel Chávez et al. (2023)	Invasive mechanical ventilation in GBS: retrospective observational study	86 patients, retrospective	39.5% required IMV; most received IVIg or plasma exchange; detailed ventilation outcomes reported.	Provides data on MV rates and management outcomes referenced in Results.
Notarnicola et al. (2023)	Predictors of respiratory failure in GBS: 22-year retrospective cohort	187 patients, retrospective	30% developed respiratory failure; inability to raise arms and bulbar symptoms were risk factors; bedside indicators can predict MV need.	Long-term cohort data reinforcing bedside predictor utility cited in Results.

METHODOLOGY

A literature search was conducted using Google Scholar, PubMed, Medscape, ScienceDirect, and major neurology journals. Advanced and manual searches identified relevant studies using the keywords Guillain-Barrie syndrome, respiratory dysfunction, Demyelinating polyneuritis.

Inclusion criteria: Studies from 2019–2025 involving GBS with COVID-19, pediatric cases, RCTs, and cohort studies were included.

Exclusion criteria: Studies before 2019, non-randomized trials, meta-analyses, and research involving GBS with comorbidities such as diabetes or hypertension were excluded.

RESULTS/FINDINGS

Respiratory dysfunction is the most life-threatening complication of Guillain-Barre Syndrome, affecting around 20-30% of the patients. It primarily results from weakening of the diaphragm, intercostal, and accessory respiratory muscles combined with bulbar and cranial nerve involvement, which impairs the airway, increasing the risk of aspiration and pneumonia. This review integrates findings from various studies and case reports to outline the characteristics of the patients who are most at risk for respiratory failure. Consistent predictors across the studies include bulbar weakness, facial palsy, neck flexor weakness, dysautonomia, and lower Medical Research Council (MRC) scores.

Respiratory dysfunction can affect approximately 14% of GBS patients, with Mechanical Ventilation (MV) often required within the first week of symptom onset [Galassi et al., 2023]. Predictive factors such as axonal subtype (particularly AMAN), autonomic dysfunction, cardiovascular comorbidities, and cranial and bulbar weakness are associated with high risk of respiratory failure, highlighting the need for early detection and tailored interventions. However, despite the high risk, early mechanical ventilation (EMV) has not consistently improved clinical outcomes, indicating that ventilatory decisions should be tailored to each patient's needs. [Melone et al., 2020].

Recent studies have explored clinical scoring system and biomarkers to enhance early detection. The Hughes GBS Disability Scale (HGDS) has been shown to accurately predict respiratory involvement, particularly in patients requiring mechanical ventilation. Patients with higher HGDS scores at admission and discharge had reportedly poorer prognosis. HGDS alongside albumin levels may be a valuable marker for early recognition of high-risk patients [Yao et al.,2023].

Additionally, the C-reactive protein to albumin ratio (CAR) is a new predictive metric that has clinical value in identifying patients at higher risk for worse outcomes, particularly if > 0.19 [Ning et al., 2021]. For paediatric cases, especially with AMAN variants, there are higher frequencies of respiratory, sensory, and gastrointestinal symptoms, requiring ventilatory support and taking significantly longer to recover than AIDP patients [Debnath et al., 2022]. Despite these findings, gaps still persist in clinical practice. The decision-making process regarding the ventilatory support for respiratory dysfunction remains inconsistent among different medical centers.

This review has identified that respiratory dysfunction in GBS is both common and multifactorial, demanding early identification, risk stratification, and multidisciplinary intensive-care management to reduce morbidity and mortality.

CONCLUSION

GBS is a severe autoimmune disorder that mainly affects the peripheral nervous system which results in generalized limb weakness and paralysis. It also causes respiratory failure in almost 30% of patients and needs ventilatory assistance as the disease is caused by immune mediated damage to the nerves. This review highlights the essential importance of identifying the disease in early stages and prompt appropriate treatments like plasmapheresis, IVimmunoglobulin, mechanical ventilations to improve the recovery and recognizing early diagnosis indicators like high disability scores, axonal variants and the involvement of cranial nerves. Therefore, improving results requires prompt respiratory assessment, predictive modelling, and interdisciplinary management. Improving these prediction instruments, evaluating the long-term impacts of respiratory failure, developing standardized procedures for ventilatory support in GBS, and lessening the effects of respiratory issues in GBS should be the goals of future research.

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