JPSIM Journal of Pakistan Society of Internal Medicine

Review Article

A Clinical Review for Guillain-Barré Syndrome

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Abstract

GB syndrome is a common clinical presentation in medical emergency wards and the ICU. It can lead to higher mortality. Trainees find it hard to differentiate GB syndrome from other causes of weakness, especially spinal cord pathologies. The complications of the disease can be devastating if not identified & managed in a timely and appropriate manner. As the initial investigations may not confirm the diagnosis, it remains a clinical judgement for the early course of the disease.

This review focuses on the various clinical, radiological, and histological clues, especially for our junior colleagues, based on international guidelines. The purpose is to provide a clear understanding and approach to managing such cases.

Keywords: GB syndrome, poly-radiculopathy, acute peripheral neuropathies.

How to cite this:

Hussain A, Avais J. A Clinical Review for Guillain-Barré Syndrome. J Pak Soc Intern Med. 2026;7(4): 284-293

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Received: 12-06-2026 **Revised:** 21-08-2026 **Accepted:** 28-10-2026 **DOI:** https://doi.org/10.70302/jpsim.v7i4.2658

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Introduction

GBS is an immune-mediated cross-reaction against the myelin sheath of the peripheral nervous system. It's more common in men than women (immune disease, which is more common in females). It is not an autoimmune disease per se, as the immune system is not attacking itself; rather, it's triggered to attack. This cross-reaction may be a reason that steroids are not effective in GB.[1]

Its annual incidence rate is approximately 1-2/100000, and it's a global disease. It's slightly more (1.1,5 times) common in men than women. The incidence increases with age, with a peak onset b/w 50-70 years of age. There are two subtypes: demyelinating and axonal damage. The demyelinating variety is more common in Europe and America, but the axonal variety is more prevalent in Asians. This could be due to variations in neuronal structures, genetic variations, and likely a different serotype of the culprit infectious agent (s) triggering the immune response.

Trigger Factors

- The trigger factor may not be evident and is not essential for diagnosis. Many times, a subclinical infection or subtle exposure may have triggered the disease process. The most common triggers are infections.
- Common infections include Campylobacter jejuni, accounting for almost 30-40% of cases. It also causes the most severe form of GBS.
- Mycoplasma and various viruses can also trigger GBS. Viruses involved are HEV, EBV, CMV, Chikungunya, Dengue, etc.
- Vaccines may be associated with GBS, but no causal relation has been established.
- Non-infectious triggers are trauma, surgery, pregnancy, drugs, autoimmune diseases, etc.[1-3]

Pathogenesis: The cross-reaction of infectious or other antigens with neuronal antigens, such as GM1, GQ, etc., causes antibody formation against neuronal antigens in the myelin sheath and/or axons. T-helper cell infiltration is also present. Antibodies activate the complement system to produce a membrane attack complex (MAC). These immune inflammatory attacks occur more at the roots and distal parts of the nerves, as the blood-nerve barrier is the weakest at these two locations.

- Damage to the myelin sheath occurs. Macrophages remove damaged myelin sheaths. The regeneration of the myelin sheath is achieved through the migration of basal Schwann cells to form a new myelin sheath. However, these regenerating

Schwann cells form multiple nodes at shorter distances within previous nodes. These various nodes reduce conduction velocity, persisting on NCS even when the patient has recovered. Regeneration may not be complete, and neurological deficits may persist.

- AMAN and ASMAN are neuropathies (a subtype of axon damage), which means antibodies against Na channels in the node area of the axons cause damage.[1-7]

Clinical Manifestations

Disease classification and course

- GBS has many clinical variations based on demyelination or axonal damage. It can also involve motor, sensory, and/or autonomic nerves. Anatomically, it can be generalized or focal: distal involving limbs, bulbar involving the brainstem, or both.
- Also, CSF & NCS (nerve conduction studies) may be normal in the first week.

Therefore, GBS can be classified based on NCS or clinical findings.

- NCS classifies it as axonal or demyelinating.
- Clinically, it can be sensory, autonomic, &/or motor. When weakness isn't present or isn't the predominant finding, it is sensory or autonomic variety.
- Generalized or involves only some body parts. Examples of focal forms are paraparesis GBS, bifacial GBS, bulbar GBS, pharyngocervicobrachial GBS, Miller-Fischer, Bickerstaff encephalitis, etc.

The most common (85-90% of cases) type of GBS is bilateral sensorimotor findings starting from the feet and progressing upward (typical GBS). Other varieties are less common.

Sensory Symptoms are dysaesthesia in the feet and back pain (due to root inflammation). Sensory signs are minimal or absent in the classic cases. Sensory symptoms & signs are absent in motor neuron disease, myasthenia, and muscle diseases such as hypocalcemic paralysis. Rarely, GB involves only sensory, but no motor, so only sensory loss is noted. Sometimes, any combination of motor and sensory involvement can be seen. GB can also be purely sensory or even autonomic, but these are rare patterns.

Lower motor pattern limb weakness: As lumbar roots are the longest, they are typically involved first, and sacral roots are the shortest, so they are rarely involved. Hence, GBS mostly starts as a lower motor lesion in the legs, and it rarely involves sphincters (cauda equina syndrome is a lower motor lesion but involves sphincters, while a spinal cord lesion is an upper motor lesion and involves sphincters). Classically, GBS starts from the feet and progresses upward to involve the legs, thighs, upper limbs, bulbar muscles & respiratory muscles.

- Less commonly: GB doesn't ascend, so only B/L foot drop is there; it's not progressing upwardly, or GB involves only lower limbs, not upper or cranial nerves.
- One golden rule of GB is that whatever pattern it follows, ankle jerk is almost always absent in all patterns right from the beginning.
- GB is a lower motor disease (hyporeflexia, hypotonia) polyradiculopathy. It is also a peripheral nervous system disease (the roots of cranial and spinal nerves are called polyradiculopathy). The myelin sheath of peripheral nerves and their roots is formed by Schwann cells; an immune attack on Schwann cells. GBS never involves.
- Reflexes, especially ankle jerks, are often absent (>95% of cases). It's rare to have GBS with normal ankle jerks. However, Bickerstaff encephalitis involving the brainstem causes hyperreflexia due to the pyramidal tract involved at the brainstem level. Almost 50% of Bickerstaff cases also have hyporeflexia due to associated ganglionopathy or large fiber neuropathy.[8-12]

Bulbar weakness is a bad prognostic sign and causes dysphonia, dysphagia, diplopia, ophthalmoplegia, weakness of Jaw muscles, bilateral facial nerve weakness, absent jaw jerk & absent gag reflex, etc. Respiratory muscle weakness is seen in severe cases, and ventilatory support is needed. Respiratory muscle weakness doesn't cause dyspnoea (as there is no effort) but starts with hypoxia and hypercapnia. Monitoring respiratory rate, chest expansion, FVC (forced vital capacity), and blood gas levels is essential in patients with progressive GB. Ventilatory support is often needed in such cases: NIV doesn't work well as the patient's respiratory drive is poor.

Dysautonomia is seen in almost 30% of cases. Parasympathetic nerves (pupil, vagus, and sacral) are involved more than sympathetic nerves due to less myelin sheath on the latter. Therefore, most findings are due to the unopposed sympathetic system and loss of parasympathetic supply. These include sinus tachycardia, tachyarrhythmia, hypertension, postural

hypotension, pupillary dilatation, urinary retention, constipation, etc. Hypertension can rarely cause hypertensive emergencies such as papilledema, heart failure, AKI, &/or posterior reversible encephalopathy syndrome (PRES). Postural hypotension is due to a lack of further increase in the sympathetic drive. Constipation and urinary retention may confuse it with spinal cord pathology or Cauda equine. However, spinal cord pathology has sensory levels, and plantar reflexes are upgoing. Cauda equina syndrome is always below L1 (never involves upper limbs, trunk, or L1 area), mixed sensory & motor, and may be an asymmetric patchy disease. Monitoring is essential. The use of antihypertensive drugs and antiarrhythmic therapy should be done with caution, as bradyarrhythmia or hypotension can also occur rapidly.

G B & raised ICP: Hypertension or high CSF proteins can raise intracranial pressure, causing papilledema in GB (the optic nerve itself is not involved in GB),[13-21]

Diagnosis of typical GBS: Diagnosis requires bilateral limb involvement (feet progressing upward) and absent/diminished reflexes bilaterally (especially ankle jerk). Additional findings favoring GBS include CSF showing high protein, NCS findings of demyelination or axonal damage, progression within 4 weeks, symmetrical signs, sensory symptoms such as dysaesthesia but lack of sensory signs, bilateral facial palsy, dysautonomia, etc.[13-17]

Onset & timeframe for progression: GBS can start within 12-24 hours or over days. Triggering causes may have prodromal clues before the neurological symptoms. Such prodromal symptoms may be respiratory or GI symptoms.

- GBS neurological symptoms and signs peak within four weeks. Sometimes, the onset is rapid, and the peak may be reached within 12-24 hours or a few days.
- Once the peak is achieved, a stable phase of neurological findings (plateau phase) may last 2-4 weeks. Then, the recovery phase starts after the plateau phase. Recovery may range from almost back to normal to minimal or no recovery at all. Recovery depends on the regeneration of the myelin sheath or axons.
- Plateau or recovery phase may have treatment-related worsening in almost 10% of cases, most likely due to rapid clearance of the treatment drugs, including IVIG, from the blood. It responds well to the reintroduction of the same treatment. Unlike relapses, these worsening episodes are usually short-lived and respond quickly to the treatment.
- True recurrence is seen in approximately 5% of cases. Relapse should raise suspicion about CIDP.

Miller-Fischer Syndrome (MFS) & Bickerstaff Encephalitis {Table 1]

- Eye muscles and brainstem are rich in gangliosides. Therefore, anti-ganglioside antibodies often involve these areas.
- Miller-Fischer is a descending variant of GBS that involves cranial nerves right from the beginning, alone or in combination with peripheral nerve roots.
- The patient presents with acute-onset symmetric cranial neuropathy, especially involving ocular, vestibulocochlear, IX & X cranial nerves.
- Clinical findings are ophthalmoplegia, dilated pupils, ptosis, dysphagia, dysarthria, etc. Areflexia and ataxia are there, too, in full-blown Miller-Fischer syndrome.
- Areflexia or hyporeflexia are seen in 90-95% of cases of GBS (except Bickerstaff encephalitis). It's due to a break in the reflex arc for stretch reflexes.
- Ataxia in Miller-Fisher is due to either peripheral nerve issues affecting the dorsal root ganglion, position, and vibration of large fiber sensory nerves or cerebellar involvement in Miller-Fisher.
- Incomplete Miller-Fischer may have some or any combination of cranial nerve involvement, e.g., ptosis, ophthalmoplegia, dilated pupil, dysphagia, or ataxia.
- Patients with Miller-Fischer have positive anti-ganglioside antibodies (anti-GQ1) in 85-90% of cases.
- The brainstem involvement in patients with Miller-Fisher is called Bickerstaff Encephalitis. These patients have hyperreflexia instead of Areflexia due to pyramidal tract involvement in the brainstem. Also, these patients have impaired consciousness due to involvement of the reticular formation.[8-16]

Axonal Variants of GBS

- These are variants of nodal diseases (see details under neuropathies).
- AMAN is a motor-only and milder form,

which often triggers Diarrhea during the summer season in Asian countries. It's a variant of GBS with Campylobacter infections in 60-70% of cases and positive antibodies against nodal structures (GM1, GD, Neurofascin-186, &/or gliomedin). It shows conduction blocks, delayed latencies (thought to be due to demyelination), and reduced CMAP (axonal). However, some patients with AMAN only have nodal dysfunction due to these antibodies causing conduction blocks. In contrast, others also have axonal damage: both pathologies continue along the same spectrum. AMAN cases have neuropathy, which may progress to complete axonopathy.

- Disruption of myelin attachment at the paranode explains latencies.
- AMAN cases with the disease limited to neuropathies usually have reversible conduction blocks (CB) and reduced CMAP, both of which resolve, and patients recover well.
- AMAN cases where the disease progresses from the neuropathies to axonal damage usually have poor recovery, and CMAP remains reduced or absent.

ASMAN is a sensory and motor variant with sensory findings. It is more severe and often occurs after a respiratory infection, with an early peak and prolonged course.[1-5]

D/D of GBS: [1-5,8-12,17-18]

The following clues should alert us to other possibilities

1. Fever (infections such as spinal osteomyelitis).
2. Sensory signs (CIDP, cord, Cauda Equina, peripheral neuropathies)
3. Upgoing plantar responses (cord or brainstem)
4. Intact reflexes, especially ankle jerk or hyperreflexia (cord or brainstem)
5. Sphincter involvement (cord or Cauda Equina)
6. Fasciculations (MND).
7. No sensory symptoms or signs (think about conditions with pure motor findings)
8. Sensory level (cord)
9. Asymmetrical findings (stroke, tumor, infection more likely)
10. Rapid resolution within hours to a few days (metabolic causes are more likely).
11. Progressive disease even after 4 weeks (acute CIDP)

Multiple recurrences (acute CIDP)

Diseases to Consider

GBS, spinal cord or Cauda Equina

- The spinal cord and Cauda Equina are the main and very important D/D. Sensory level, sphincter involvement, and mixed sensory-motor signs are the clues.
- Cauda Equina never involves L1, thoracic, and cervical roots. It's often patchy.
- The spinal cord gives signs at the level and below the level of the lesion, but the cord above the lesion is often normal (unless the disease is multifocal).
- MRI spine often detects the lesions in the cord or at the Cauda Equina.
- GBS doesn't involve sphincters unless there is dysautonomia. GBS has absent reflexes with down-going plantar responses, whereas cord lesions have up-going plantar responses and hyperreflexia.
- GBS has no sensory level, and significant sensory signs are rare.

Brainstem vs. GBS

- Brainstem stroke, encephalitis, abscesses, tumors, etc., can be a D/D of GBS involving the brainstem (Bickerstaff Encephalitis). Listeria is known to cause brainstem encephalitis with ring abscesses, and the rabies virus can also cause it.
- Unilateral or asymmetrical signs often indicate lesions other than the GB variant.
- Down-going plantar reflexes are never seen in brainstem diseases except in GBS, which may have hyporeflexia due to concomitant involvement of dorsal root ganglia or peripheral nerves.

MRI brainstem with contrast is almost always abnormal in these lesions and can exclude these possibilities. However, an MRI may be normal for Bickerstaff Encephalitis.

Acute onset CIDP vs. GBS

- The main D/D, besides cord and Equina lesions, is acute-onset CIDP.
- Continued progression of neurological deficit beyond four weeks is against GBS and suggests acute onset of CIDP.
- Similarly, frequent relapses (3 or more) after achieving their peak during the plateau or

recovery phase also suggest CIDP.

- Acute-onset CIDP is less likely to have the following: preceding infection, autonomic dysfunction, bulbar involvement, facial involvement, & ventilation requirement.
- CIDP has prominent sensory signs (sensory symptoms can be present in AIDP, but sensory signs are rare).
- CIDP involves proximal and distal equally and bilaterally.

Table 1: *Variants & D/D of GB syndrome.*

GBS Variants:

Criteria	Types	Clues
NCS	Axonal vs demyelination	Axonal can be AMAN or ASMAN. These are considered Nodopathies & antibodies against nodal structures (GM1, GD, Neurofascin-186, &/or gliomedin) are positive in 70% of cases. NCS shows conduction blocks, delayed latencies (thought to be due to demyelination), and reduced CMAP (axonal).
Clinically	Motor, sensory, autonomic	**Generalized Ascending** GBS is ascending paralysis. **Localized GBS:** Paraparesis GBS, bifacial GBS, bulbar GBS, pharyngocervicobrachial GBS
Anti-GQ1b positive GBS	Miller-Fischer Bickerstaff	Brainstem involvement is Bickerstaff, and brainstem sparing is Miller-Fischer.

Abbreviations: AMAN (acute motor axonal neuropathy), ASMAN (acute motor sensory axonal neuropathy), CMAP (compound motor action potential), EMG (electromyography), NCS (nerve conduction studies), SAP (sensory action potential)

D/D for GBS:

Disease	Clues against GBS	Clues favoring GBS
Spinal cord	Sensory level, sphincter involvement, and mixed sensory-motor signs. Upgoing planters and hyper-reflexia.	No sensory level Sphincter involvement is rare in GBS—absent reflexes and down-going planters.
Clues Cauda equina	Never involve L1, thoracic, and cervical roots. It's often patchy, mixed moor & sensory loss. Sphincters are involved.	GBS often extends to L1 & above. GB is diffuse. Motor predominant. Sphincters are rarely involved.
Brainstem	Unilateral or asymmetrical signs. Cranial nerves, upgoing planters, impaired consciousness, brainstem signs. MRI abnormalities.	Never unilateral. Planters are down going in 50% BSE. BSE may have a normal MRI.
Acute CIDP	Progresses beyond 4 weeks. Relapses are common. Absence of autonomic, respiratory, cranial nerves, or brainstem disease. Proximal = distal, sensory & motor signs, all 4 limbs. Response to steroids	Peaks within 4 weeks Relapse is rare. Preceding infection, autonomic dysfunction, bulbar involvement, facial involvement, & ventilation requirement. Ascending. No response to steroids.

Abbreviations: BSE (Bickerstaff Encephalitis).

- CIDP responds well to immunosuppressants such as corticosteroids.

Other D/Ds

1. Diabetic polyradiculopathy can mimic GBS, and it's important to exclude diabetes, even if the patient isn't known to have diabetes.
2. Hypo- or hyperkalemia can cause rapid-onset muscle weakness. These may have abnormal K levels, but sometimes, serum values may be normal. The response is rapid, with recovery in 12-24 hours, unlike GBS, which takes weeks to recover. Sensory clues are absent as it's pure muscle tissue. Reflexes may be present unless it's very severe paralysis.
3. Toxins (drugs, botulism, Bacillus cereus, diphtheria) can also cause neuromuscular junction issues with pure motor weakness: tubocurarine poisoning, insecticide poisoning, muscle relaxant overdose, anesthetic overdose, aminoglycosides.
4. Tick paralysis (tick bite).
5. Botulism: clues are canned food intake, no GIT s/s, descending paralysis, clear CSF, and normal nerve conduction, which is normal as the problem is at the NMJ. Botulism is due to impaired release of acetylcholine from nerve endings.
6. Diphtheria toxin-induced neuropathy: pseudomembrane on tonsils, skin infection, or infection in the larynx, depending on the location of the diphtheria infection (tonsil, skin, or larynx).
7. Metabolic toxins such as lead (Pb) often cause motor neuropathy.

Pure motor Findings without sensory findings

- Muscle diseases involve proximal muscles symmetrically, and reflexes are normal.
- NMJ disease, such as myasthenia, is fatigable, involves oculobulbar muscles more, produces positive acetylcholine receptor antibodies, and responds to acetylcholine-like drugs.
- MND is a slow-onset disease with pure motor signs, including fasciculations. It progresses slowly over months to years, and the CSF is normal.
- Polio is an acute-onset, pure motor but asymmetrical disease involving one or two limbs.

Investigations for GBS[1-10, 15-20]: Investigations are mostly needed to exclude infections, metabolic & electrolyte abnormalities, and other diagnostic possibilities.

CSF

- The focus of GB is the roots, and roots are bathed in CSF. When the immune system attacks roots, immunoglobulins are present in CSF, which explains the high CSF protein in GB. As antibodies mainly attack roots, cells are usually absent or very low in CSF. This is protein-cell dissociation in CSF (Infection in CSF is rich in cells).
- It helps exclude other possibilities, such as infections, etc.
- Proteins are usually high due to the increased permeability of the blood-brain barrier at the roots located within the CSF. High protein levels are more common in a demyelinating pattern, which has more extensive inflammation of the myelin sheath (than the axonal variety, which often is limited to node areas). High protein levels indicate the likelihood of a severe course. These high CSF proteins can lead to CSF drainage block and raised intracranial pressure, causing papilledema. Papilledema can be seen due to high CSF proteins, & hypertensive encephalopathy due to autonomic involvement (see below). However, proteins may be normal in almost half of the patients during the first week. Therefore, normal CSF proteins are possible if LP is done during the first week. More than 90% of cases have elevated proteins by the end of the second week.
- Cells are normal, or there is mild CSF pleocytosis. Cells, if high, are mostly 10-20 (always below 50) and are lymphocytes. Cell counts of more than 50 or cells other than lymphocytes should raise suspicion of infectious or other causes.

NCS & EMG

Demyelination

- Absent F waves or increased latency of F waves indicates root involvement.
- Distal parts of nerves have increased latency, conduction blocks, and broad (prolonged duration> 8 ms) compound muscle action potentials due to variable conduction of motor axons in the nerve (some axons are normal, and others have delayed conduction).

- EMG usually shows fewer muscle fibers activated (low recruitment) as low-amplitude compound action potentials.
- Denervation can lead to increased irritability of muscle cells with fibrillations.
- Regeneration of the myelin sheath with multiple nodes slows the motor conduction velocity.

AMAN & AMSAN have staged changes on serial NCS

1. First, the motor amplitude is decreased with normal sensory amplitude. Latency and conduction velocities may be affected as well. F wave may be absent, but without any delay in latencies. Usually, no temporal dispersion (widening of the action potential) occurs if conduction is affected, as in a demyelinating pattern.
2. Later, the amplitude is reduced or absent without conduction delays.
3. Sensory amplitude, as is the motor, is also reduced in cases with AMSAN.

MRI of the brain &/or spine

- MRI is needed to exclude other possibilities affecting the brainstem, spinal cord, or cauda equina. Infections, stroke, or space-occupying lesions.
- In addition, MRI with contrast can show thickening &/or enhancement of roots (spinal &/or cranial nerve roots).
- Miller-Fischer may also have a thickening of the posterior column.
- MRI may be normal in almost half of the cases of Bickerstaff Encephalitis (BSE) and may not detect abnormalities in the brainstem—the silence of MRI in the setting of brainstem presentation favors BSE. However, brainstem edema, or contrast enhancement, may be found when MRI is positive for encephalitis.

Treatment of GBS[15-21]

IVIG or Plasma Exchange (PLEX): It's mainly used for progressive limb disease, dysautonomia, and respiratory or bulbar involvement. Response to IVIG or PLEX isn't immediate and may take weeks. The response may not be complete.

IVIG

- IVIG dose is 2gm/kg, which can be given in 2 doses (1gm /day) or 5 doses (0.4gm /kg/day).
- IVIG is the preferred treatment in pregnancy, immunocompromised states, and if there is an active infection.
- IVIG can cause volume overload, thrombotic episodes due to hyperviscosity, or aseptic meningitis. Allergic reactions such as anaphylaxis or serum sickness may also occur.
- IgA-deficient patients should receive IVIG without IgA to avoid anaphylaxis.
- Cost and availability are other issues.

PLEX

- PLEX involves 200-250 ml/kg plasma given in 5 divided doses over 10 days.
- Response is expected in 1-2 weeks.
- Infections, protein deficiency, clotting factor deficiency, thrombocytopenia, risk of infections, etc., are common.

Supportive Care

- Cardiac monitoring for arrhythmia.
- Blood pressure management
- Fluid intake-output balance.
- Serial neurological and respiratory assessments are done frequently, 2- 4-hourly, in acute cases. It can be less frequent once the disease plateaus or improves.
- Such intense monitoring may be avoided for those with mild, especially non-progressive limb disease (without dysautonomia, bulbar, and respiratory issues). Such cases may be kept in medical wards.

Ventilation & respiratory support: Respiratory assessment is essential, especially when rapidly progressive limb involvement can extend to respiratory muscles, a bulbar disease affecting the respiratory or vasomotor center, bulbar involvement, or dysautonomia.

The following are the tools for assessing respiratory compromise.

- Breathing difficulties,
- Weak Cough,
- Unable to count to 15 with a single breath,
- Rapid respiratory rate,
- Using accessory muscles,
- Declining chest expansion,
- Hypoxemia (PaO 2 56 or below),
- Hypercapnia (PaCO2 48 or more),

Table 2: *NCS, CSF & MRI*

Disease	NCS & EMG	CSF	MRI
Ganglionopathy	Conduction and latencies are normal. SAP reduced or absent. CMAP-normal. Reduced or absent activity of ascending spinal sensory tracts (DCT & STT).	Normal Underlying cause changes	Thickening Enhancement Cord atrophy.
GBS	Normal initially or in mild disease Absent F waves or increased latency of F waves. Delayed conduction and increased latencies (demyelination). Conduction blocks in the distal part of nerves. EMG: low-amplitude compound action potentials. Denervation: fibrillations. Myelin regeneration: multiple nodes slow motor conduction velocity.	High CSF proteins in GB. Protein-cell dissociation in CSF (Infection in CSF is rich in cells)	May show thickening &/or enhancement of roots.
CIDP	Normal initially or in mild disease Latencies are increased & conduction velocities are reduced in both sensory and motor nerves, and in both proximal and distal parts. Motor dispersion & prolonged duration of compound motor action potential (CMAP) F-waves may be absent.	Proteins are elevated in 90% CSF cells are often normal. 10-50 cells may be present. All should be lymphocytes.	It may show thickening/contrast enhancement of the affected roots/nerves.
Plexopathy	Locate the lesions and differentiate them from mononeuritis, mononeuropathy, or polyradiculopathy. Sensory deficit in the arm with preserved sensory NCS excludes plexus or nerve issues but suggests sensory root problems. Assessed if the nerves are still reaching the muscles Regenerative changes Axonal damage (low or absent amplitude) or demyelinating (slow conduction velocity) disease. The severity of the injury is indicated by absent action potentials (axonal damage), severe fibrillation (denervation), and absent or minimal recruitment. EMG can detect dorsal root involvement (paraspinal muscles), severe or acute damage (absent OR reduced CMAP), regenerative changes (recruitment, broad, high-amplitude CMAP), denervation changes (fibrillation),	CSF is often normal	It may show thickening or enhancement.

- Reduced FVC (forced vital capacity)

Low maximum inspiratory or expiratory pressure, etc.

Abbreviations: CMAP (compound motor action potentials), DCT (dorsal column tract), EMG (electromyography), GBS (Guillain-Barré syndrome), NCS (nerve conduction studies), SAP

(sensory action potential), STT (spinothalamic tract)

NIV isn't a good choice, as these patients don't get better in a short period. NIV often isn't effective, as respiratory muscle effort and respiratory center drive may be compromised, too.

Elective intubation is preferred to avoid worsening of dysautonomia with emergency intubation. Muscle-paralyzing drugs may exacerbate the weakness. Denervation-related muscle hypersensitivity can cause hyperkalemia with succinylcholine.

Tracheostomy may help to assist extubation for those who are on a ventilator for 10 or more days.

When is ICU needed?

- Deteriorating respiratory parameters
- Dysautonomia, especially with abnormal hemodynamics (BP, heart rate)
- Cardiac arrhythmia or failure
- Rapidly progressive limb weakness,
- Bulbar involvement,
- Bickerstaff encephalitis, &/or
- Sepsis
- IVIG or PLEX should also be administered in the ICU setting.

Additional Supportive Care:

- Care for nutrition, swallowing assessment
- Speech therapy
- Prevention of bed sores and DVT
- Physiotherapy,
- Occupational therapy.
- A tracheostomy and PEG tube for feeding may be needed.
- Rehabilitation is needed to maximise recovery and restore a normal life.

Prognosis for GBS[18-21]

- Almost 80% can walk, and over half recover completely within 6-12 months.
- Prognosis is poor in those with axonal disease, especially for severe cases. Other poor prognostic factors are older age, comorbidities, and involvement of the respiratory, bulbar, or autonomic system.
- Mortality varies from 3-7%, but it goes up to 20% for those who need ventilatory support.

Mortality is higher in resource-limited countries due to a lack of adequate diagnostic and therapeutic options.

Conflict of Interest: None

Funding Source: None

Authors' Contribution

AH: Conception.

JA: Design of the work.

AH, JA: Data acquisition, analysis, or interpretation.

AH: Draft the work.

JA: Review critically for important intellectual content.

All authors approve the version to be published.

All authors agree to be accountable for all aspects of the work.

References

1. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barré syndrome. Lancet. 2021;397(10280):1214-1228
2. Kouton L, Boucraut J, Devaux J. Electro-physiological features of chronic inflammatory demyelinating polyradiculoneuropathy associated with IgG4 antibodies targeting neurofascin 155 or contactin 1 glycoproteins. Clin Neurophysiol 2020;131:921–7
3. Israeli E, Agmon-Levin N, Blank M, Chapman J, Shoenfeld Y. Guillain-Barré syndrome--a classical autoimmune disease triggered by infection or vaccination. Clin Rev Allergy Immunol. 2012;42(2):121-30
4. Yuki N, Hartung HP. Guillain-Barré syndrome. N Engl J Med. 2012;366(24):2294-304.
5. Leonhard SE, Mandarakas MR, Gondim FAA, Bateman K, et. al. Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol. 2019;15(11):671-683
6. Oh SJ, LaGanke C, Claussen GC. Sensory Guillain-Barré syndrome. Neurology. 2001;56(1):82-6.
7. Wakerley BR, Yuki N. Isolated facial diplegia in Guillain-Barré syndrome: Bifacial weakness with paresthesias. Muscle Nerve. 2015;52(6):927-32
8. Yuki N, Kokubun N, Kuwabara S, Sekiguchi Y, et. al. Guillain-Barré syndrome associated with normal or exaggerated tendon reflexes. J Neurol. 2012;259(6):1181-90.
9. Dionne A, Nicolle MW, Hahn AF. Clinical and electrophysiological parameters distinguishing acute-onset chronic inflammatory demyelinating polyneuropathy from acute inflammatory demyelinating polyneuropathy. Muscle Nerve. 2010;41(2):202-207.
10. Leonhard SE, Mandarakas MR, Gondim FAA, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. Nat Rev Neurol. 2019;15(11): 671-683.

11. Verboon C, Doets AY, Galassi G, et al. Current treatment practice of Guillain-Barré syndrome. Neurology. 2019;93(1):e59-e76
12. Yuki N, Kokubun N, Kuwabara S, et al. GuillainBarré syndrome associated with normal or exaggerated tendon reflexes. J Neurol 2012; 259(6):1181-90.
13. Shahrizaila N, Lehmann HC, Kuwabara S. GuillainBarré syndrome. Lancet Lond Eng. 2021; 397(10280):1214-28.
14. Scarpino M, Lolli F, Carrai R, Lanzo G, et. al. Diagnostic accuracy of neurophysiological criteria for early diagnosis of AIDP: A prospective study. Neurophysiol Clin. 2016 Feb;46(1):35-42.
15. Walgaard C, Lingsma HF, Ruts L, Drenthen J, et. al. Prediction of respiratory insufficiency in Guillain-Barré syndrome. Ann Neurol. 2010;67(6):781-7.
16. WDerksen A, Ritter C, Athar P, Kieseier BC. Sural sparing pattern discriminates Guillain-Barré syndrome from its mimics. Muscle Nerve. 2014;50(5):780-4.
17. Dionne A, Nicolle MW, Hahn AF. Clinical and electrophysiological parameters distinguishing acute-onset chronic inflammatory demyelinating polyneuropathy from acute inflammatory demyelinating polyneuropathy. Muscle Nerve. 2010;41(2):202-7.
18. Chevret S, Hughes RA, Annane D. Plasma exchange for Guillain-Barré syndrome. Cochrane Database Syst Rev. 2017;2(2):CD001798.
19. Hughes RA, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. Cochrane Database Syst Rev. 2014;2014(9)-:CD002063.
20. Hughes RA, Swan AV, Raphaël JC, Annane D, van Koningsveld R, van Doorn PA. Immunotherapy for Guillain-Barré syndrome: a systematic review. Brain. 2007;130(Pt 9):2245-57.
21. van den Berg B, Bunschoten C, van Doorn PA, Jacobs BC. Mortality in Guillain-Barre syndrome. Neurology. 2013;80(18):1650-4.