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Guillain Barre Syndrome: Symptoms, Diagnosis and Treatment

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ABSTRACT

Guillain Barre Syndrome (GBS) is found to be a rapidly progressing defence mediated disorder that usually affects the extremities. The most prevalent form seen among the class of GBS is acute inflammatory demyelinating polyneuropathy (AIDP). Cerebrospinal fluid (CSF) evaluation and electrophysiological findings are used mainly to diagnose GBS. The cerebrospinal fluid evaluation shows increased protein levels. Nerve conduction studies show a possible blockage, of conduction. Plasma exchange and intravenous immunoglobulin play a vital part in the treatment of GBS. Supportive therapy includes controlling pain with nonsteroidal anti-inflammatory drugs, carbamazepine, or gabapentin; monitoring for respiratory complications and preventing venous thrombosis. GBS is a serious illness and the long-term impact is 3-6 years in GBS patients after the onset of the disease. It will take a long time to recover. In 20-30% of adult patients, chronic illness can be seen, but in children, it is less common. Extreme fatigue is seen in one-third of patients and long term residual problems involve fatigue, pain and psychological disability.

Key Words: Polyradiculoneuropathy, Covid-19, Immunotherapy, Campylobacter jejuni, Plasma exchange, Fatigue

INTRODUCTION

Guillain Barre Syndrome (GBS) is an intense, heterogeneous, defence-mediated outlying nerve fibre and nerve root disorder. ¹This syndrome had been named after French physician George Guillain and Jean Alexander Barre in 1916.²

GBS is usually introduced by an infection or any immune stimulation which induces an infection targeting peripheral nerve and spinal roots. Patients with GBS develop increasing paralysis that begins in the legs and extends to the arms. GBS is the usual cause of acute flaccid immobility distinguished by uniform fragility of the extremities and lack of reflex or reflex which reaches its maximal in 1 month.³

Respiratory depression develops in 25% of patients and cranial nerve involvement is frequent. Ventilator assistance is necessary for one-third of hospitalized patients with respiratory decline.⁴

There are mainly 4 types of GBS, acute inflammatory demyelinating polyneuropathy (AIDP), acute motor and sensory axonal neuropathy (AMSAN) and acute motor axonal neuropathy (AMAN) are more common GBS

types while Miller Fisher syndrome (MFS) occurs less commonly.⁵

Clinical characteristics, electrophysiological tests, and cerebrospinal fluid studies are the basis of GBS diagnosis. Common subtypes of GBS include classic AIDP which on electrophysiological studies show changes in the myelin status of peripheral nerves which is demyelination. AMAN shows axonal dysfunction of the peripheral nervous system. In AMSAN (acute sensory and motor axonal neuropathy) both motor and sensory axons are affected.

Another familiar GBS variant is Miller Fisher Syndrome (MFS) involving ataxia, areflexia, and ophthalmoplegia. Other less common GBS variants are GBS paretic weakness, pharyngeal-cervical-brachial or bifacial weakness with a distal tingling sensation, acute pandysautonomic, cranial polyneuritis, pure sensory neuropathy, and oropharyngeal variants.

Epidemiology of GBS

The yearly incidence of GBS varies from 0.34 to 1.34 cases/100000 person, increases linearly with age and the incidence

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are lower in children than in adults. Men are affected 1.5 times more often than women. 70-90 per cent of cases are reported with AIDP. GBS can occur in any season.⁶

Aetiology of GBS

The exact cause of GBS is unknown. GBS occurs due to autoimmune infection on peripheral nerves. GBS is known to develop after a bacterial infection like campylobacter jejuni and mycoplasma pneumonia or virus infections such as cytomegalovirus, Epstein-Barr virus, influenza-like illness, HIV, Zika virus, etc.⁷ Campylobacter infection is the most commonly found microorganism in 30%-35% of GBS cases. Rarely recent surgery, trauma, bone marrow transplantation, or vaccination can trigger GBS.⁸ Recently GBS cases reported following infection with Zika virus and COVID 19.⁹

Relationship between GBS & COVID 19 infections

SARS-COV 2 can infect the nervous system, skeletal muscles and respiratory tract. In severe cases, the neurological involvement is greater, which includes acute cerebrovascular disease, disturbances of consciousness and skeletal muscle damage.¹⁰ COVID-19 and GBS may have a possible correlation between them. The case report suggests COVID 19 may cause peripheral nervous system damage meeting diagnostic criteria for acute sensory and motor poly-radiculonephritis.^{11, 12, 13}

Risk factors of GBS

- The risk of developing GBS increases with age.
- Men are more likely to cause GBS than women.
- GBS can be easily stimulated mainly by Campylobacter infection found in undercooked poultry.
- Pneumonia - a bacterial infection in the lungs
- Surgery that can lead to GBS
- Hodgkin lymphoma can lead to GBS
- Vaccination against influenza or childhood vaccines can also lead to GBS

Classification of GBS

GBS variants /subtypes of GBS are

Common variants.¹⁴

1. AIDP
2. AMSAN
3. AMAN
4. MFS
5. Pharyngeal -cervical-brachial variant
6. Paraparetic variant

Other variants.¹⁵

1. Acute pandysautonomia
2. Pure sensory GBS
3. Facial dysphagia and distal limb paraesthesia
4. Acute bulbar palsy with areflexia

5. Sixth nerve palsy and distal paraesthesia
6. Bickerstaff encephalitis

Various forms of immunoglobulins can be found in different GBS subtypes such as anti-GM1 and GD1a in AMAN, anti-GD1a IgG in serious axonal motor GBS, anti-GQ1b IgG in MFS, anti-GT1b ganglioside and poly-sialoganglioside IgG in the variants of cranial nerve (Table 1). Anti-GD1b-IgG in pure ataxic sensory GBS and anti-GM2-IgM in serious GBS along with past cytomegalovirus inflammation.^{16, 17, 18}

Table 1: Association of anti-ganglioside IgG immunoglobulins with subtypes of Guillain-Barre syndrome.

Variants	IgG antibodies developed
AIDP	None
AMAN	GM1 and GD1a
AMSAN	GM1 and GD1a
Acute motor conduction blocks neuropathy	GM1 and GD1a
Pharyngeal -cervical -brachial (PCB) variant	GT1a (less frequently with GQ1b and GT1a)
Miller Fisher Syndrome (MFS)	GQ1a and GT1b
Acute ataxic neuropathy (without ophthalmoplegia)	GQ1b and GT1a
Pure sensory ataxic variant	GD1b (less frequently with GQ1b and GT1a)
Bickerstaff brainstem encephalitis	GQ1b and GT1a

External and internal ophthalmoplegia, cerebellar-like ataxia and oropharyngeal palsy have all been linked to IgG antibodies GQ1b and GT1A. Sensory ataxia is related to IgG anti -GD1b antibodies that do not cross-react with GM1.

Pathology of GBS

The pathology includes lymphocytic infiltration by the vertebral roots and peripheral nerves followed by multifocal stripping mediated by myelin macrophages and further resulted in the absence or delay in the spread or conduction of the electrical nerve impulse causing flaccid paralysis. Recovery is associated with remyelination. As a secondary consequence, axonal disruption and loss is observed in patients with severe GBS.¹⁹

Pathological studies in AMAN show a small amount of inflammatory infiltration with axon destruction and in AMSAN similar pathological changes have been observed but include motor and abdominal nerve roots.²⁰

Pathogenesis of GBS

The defence system attacks the outlying nervous system and inactivates the immune system in GBS. For example, C. jejuni indicates lipooligosaccharides in its cell wall proximal

to nerve myelin or axonal segments. This, in turn, produces antiganglioside antibodies throughout the body, attacking nerves and myelin sheaths with outer nerve fibres and nerve root injuries in vulnerable individuals.²¹

Clinical manifestations of GBS

The acute or rapid development of paralysis progresses with the loss of deep tendon reflexes or areflexia over days or weeks. Distal paraesthesia and pain precede muscle fatigue that increases quickly from the lower to the upper limb. In 50 per cent of cases, pain occurs early in low back neck and shoulders. Facial bulbar weakness is common. Rapid deterioration to respiratory failure may develop within hours and in case of respiratory weakness, 20 per cent need ventilator support. In late and severe cases bladder dysfunction can occur. Sensory symptoms in the legs usually mark the disease onset. Lumbar pain commonly represents inflammation in the nerve root. Cranial nerve involvement is found. The common manifestations reported are pyrexia, cough, pharyngitis, and other upper respiratory symptoms.²² GI symptoms may precede acute motor subtypes of motor-sensory axonal neuropathy. The most important symptoms seen in GBS patients are the bilateral increasing, uniform weakness of limbs. (Table 2)

Table 2: Symptoms seen in various variants are as follows.

Variants	Clinical Features
Acute inflammatory demyelinating Poly-radiculoneuropathy	Progressive symmetrical weakness, hyporeflexia or Areflexia
AMAN	Only motor symptoms
AMSAN	Similar to AMAN, with predominant sensory involvement
Classic sensorimotor GBS	Uniform weakness and sensory signs develop rapidly with absent or diminished tendon reflexes and usually reach a nadir within 2 weeks
Pure motor	Motor weakness without sensory signs
Para paretic	Paresis restricted to legs
Pharyngeal –cervical –brachial variant	pharyngeal, cervical, and brachial muscle fragility without involving lower limb weakness
Bilateral facial palsy with paraesthesia	Bilateral facial weakness, paraesthesia, and decreased reflexes
Pure sensory	Intense or sub-acute sensory neuropathy without other defects
MFS	Ophthalmoplegia, ataxia, and Areflexia
Bickerstaff brainstem encephalitis	Ataxia, Ophthalmoplegia, areflexia, pyramidal tract signs and diminished consciousness

Guillain-Barré syndrome (GBS) is characterized by rapidly evolving ascending weakness, mild sensory loss and hyporeflexia, progressing to a nadir over up to four weeks.

Diagnosis of GBS

Two important characteristic standards for GBS are evolved by the National institute of neurological disorders and stroke (NINDS) (Table 3) in 1978(revised in 1990) and Brighton collaboration in 2011 (Table 4).²³

Table 3: National Institute of neurological disorders and stroke (NINDS)

Essential Characteristics
Progressive weakness in both limbs
Areflexia (or hyporeflexia).
Features supportive of the diagnosis
Increase in clinical manifestations over days to 4 weeks
Moderate sensory clinical manifestations
Cranial nerve participation, mainly bilateral facial frailty
Reclamation starts 2 to 4 weeks after progression stops.
Autonomic dysregulation
Absence of pyrexia in the beginning
Typical CSF (albumin-cytologic dissociation)
EMG/nerve conduction findings (characteristic signs of a demyelinating process in the peripheral nerves)
Features casting doubt on the diagnosis
Asymmetrical weakness
Persistent bladder and bowel dysfunction
Bladder or bowel dysfunction at the onset
>50 mononuclear leukocytes/mm ³ or presence of polymorph nuclear leukocytes in CSF
Distinct sensory level.
Features that rule out the diagnosis
Hex carbon abuse
Abnormal porphyrin metabolism
Recent diphtheria infection
Lead intoxication
Other similar conditions: poliomyelitis, botulism, hysterical paralysis, toxic neuropathy.

Criteria developed by the National Institute of Neurological Disorders and Stroke

Table 4: Brighton Diagnostic Criteria

	Level of diagnostic certainty			
DIAGNOSTIC CRITERIA	1	2	3	4
bilateral and flaccid limb weakness	+	+	+	+/_
Reduced deep tendon reflexes in weak limbs	+	+	+	+/_
Monophasic course and time between onset –nadir 12 h to 28 days	+	+	+	+/_
CSF cell count less than 50 /μl	+	+	-	+/_
CSF protein concentration greater than normal value	+	+/_	-	+/_

Table 4: (Continued)

	Level of diagnostic certainty			
NCS findings consistent with one of the variants of GBS	+	+/-	-	+/-
Absence of alternative diagnosis of weakness	+	+	+	+

+ Present, - absent, +/- present or absent

NCS –nerve conduction studies, GBS-Guillain barre syndrome

The determination of GBS depends on the medical history and assessment along with complementary tests such as CSF and electrodiagnostic studies. The clinical history includes antecedent infection symptoms, distal paraesthesias and progression to radii in 12 h to 28 days. The examination includes symmetrical weakness, ataxia, and hyporeflexia in 90 % of patients. Laboratory tests for electrolytes, glucose, liver enzymes and renal function are done on suspected individuals.

Cerebrospinal fluid examination: Elevated CSF protein level (1800mg/dl where the normal range is 15-45mg/dL) with normal CSF cell count is the classic finding in GBS (albumin-cytological discoloration)

Electro diagnostic studies: This study would show sensorimotor poly- radiculoneuropathy/polyneuropathy suggested by decreased conduction speed, decreased sensory and motor amplitudes. Axonal and demyelinating subtypes can be differentiated by nerve conduction studies (NCS). Anomalies in nerve conduction appear to peak more than 2 weeks after weakness starts. NCS anomalies dependent on subtypes of GBS (AIDP, AMAN/AMSAN)

Imaging Studies: Differential diagnoses like brain stem infection, stroke, inflammation of the spinal cord or anterior horn cell, nerve root compression or leptomeningeal malignancy may be excluded from magnetic resonance imaging (MRI).

Lumbosacral spine MRI will illustrate lumbosacral nerve root gadolinium enhancement to better diagnose GBSS.

Testing of antiganglioside: Specific antibody detection can be diagnostically important, as 90% MFS patients are seen with anti-GQ1b antibodies. In AMAN patients, anti-GM1 and anti-GD1a antibodies are commonly found.²⁴

Management strategies

Symptomatic patients with GBS who can walk unassisted for more than 5 m can be managed in peripheral centres. They need attentive cardiac and respiratory function control and disease prevention. They should be noted for disease progression if patients require ventilation in the first week after disease onset. 25% of patients require ventilation. If any of the symptoms or signs are noticed, they should be moved

immediately to specialist hospitals. In 3-6 months, 80% of patients are fully recovered, 4% die and the remaining individuals suffer from residual neurological disorders that may be serious. In the acute phase, the patients require supportive therapy and immunotherapy.²⁵

1) Supportive therapy

This includes tracheostomy, deep vein thrombosis prophylaxis, skincare, bedsore, joint physiotherapy daily. Care should be given to the patients to reduce disease-related issues or secondary complications leading to mortality.

a) Management of respiratory failure

Respiratory paralysis may be caused by GBS. One-third of patients need mechanical ventilation. Tachypnoea, increase in heart rate, brow sweating, non-simultaneous chest and abdominal movement, maximum respiratory pressure <30 mm H₂O, vital capacity < 20 ml/kg, maximum expiratory pressure <40 mm H₂O predicts respiratory failure are clinical signs. Facial fatigue, bulbar paresis, and neck weakness are elements associated with respiratory decline. Percutaneous dilation tracheostomy can be done after 2 weeks of intubation and should be done on the current status of the individual.²⁶

b) Deep vein thrombosis (DVT) prophylaxis

Immobilisation caused by GBS can lead to pulmonary embolus and DVT formation. Subcutaneous fractionated or unfractionated heparin (5000U every 12 hour) or enoxaparin (40mg every day) and support stockings are recommended for non-ambulatory patients.

c) Pain management

Oral or parenteral narcotic analgesics are required in 75% GBS patients and 30% of patients were treated with IV morphine infusions (1-7mg/hr). As opiates can intensify gastrointestinal dysmotility and bladder widening, cautious observing is required. Gabapentin (15mg/kg/day) and carbamazepine (300mg day by day) are utilized for powerful agony decrease in GBS patients. Other adjuvant treatments used for the board of neuropathic torment are mexiletine, tramadol, tricyclic stimulant prescriptions and so forth.²⁷

Table 5: GBS Disability Scale

o	A healthy state
1	Minor symptoms and will be able to run
2	Able to walk 10m or more without help but unable to run
3	Able to walk 10 m across an open space with help
4	Lack of physical function
5	Requires oxygenation fewer times in a day
6	Death

Most commonly used measure of levels of activity and participation

2) Immunotherapy

If given during the first week of the disease, along with plasma exchange (PE), Intravenous immunoglobulin (IVIG) are productive immunotherapies for both grown-ups and children with GBS. If the patient is unable to walk 10 m unassisted, immunotherapy is begun (GBS scale ≥ 3) (Table 5). CT shows treatment results within 14 days of onset of weakness.

a) Plasma Exchange

Also called plasmapheresis which involves the removal of small amounts of plasma (<15% of patient's total blood volume). PE within 4 weeks from onset should be applied and much effect is seen when PE starts within 2 weeks of onset. It can reduce the abnormal amount of protein in the blood. PE can benefit all patients with light, average and extreme GBS. It can also be benefited in patients who require minimal walking assistance and in patients who are slowly improving. Bed and ventilator bound patients should be instructed to do PE. PE is typically provided on 5 separate occasions as one plasma volume, 50ml/kg body weight. Adverse effects include a decrease in blood pressure, septicaemia, pneumonitis, abnormal coagulation and hypocalcaemia. Haemostatic disorders, unstable CV state, inflammation and pregnancy are mostly seen as contraindications. An alternative technique to PE is immunoadsorption. It is a therapy that excludes Ig from circulation without any supplementation of albumin.²⁸

b) Intravenous Immunoglobulin (IVIG)

Due to greater ease and availability, the dose of 0.4g/kg body weight for five consecutive days daily made IVIG preferred treatment over PE. This protects patients from infection and suppresses the pathway mediated by inflammatory and immune systems. The mechanisms involved in IVIG include reducing interleukin development and supplying an antigen that blocks the binding of autoantibodies to B lymphocytes.²⁹

3) Other treatment modalities

For GBS therapy, treatment with corticosteroids is ineffective. The Nerve axons can be substantially prevented from damage by sodium channel blockers.

Different management strategies are followed in specific patient groups.

1) MFS Patients

Patients with pure MFS can completely recover within 6 months without any therapy, but patients should be closely monitored for limb fragility, bulbar or facial immobility, or respiratory decline.

2) Pregnant females

During pregnancy, either IVIG or plasma exchange is contraindicated. As PE needs careful monitoring, IVIG may be more common.³⁰

3) Children

For children with GBS, IVIG is first-line therapy

Prognosis

The long-term impact is 3-6 years in GBS patients after the onset of the disease and 3-5% of patients die. Extreme fatigue is seen in one-third of patients. Long-term residual issues that include incomplete recovery of physical function, fatigue, pain and psychological disability may be encountered by patients with GBS. It is appropriate to carefully monitor these long-term consequences.

Physical function: An improvement program with physiotherapists, specialists and occupational therapists should be carried out to reinstitute outcomes of motor and sensory conditions. Exercises that include stationary cycling, walking, and weight training improves walking ability and self-sufficient in daily activities.

Fatigue: Fatigue is found in 60-80% of GBS patients. Programmes involving exercises benefit in reducing fatigue

Pain: A third of a patients complains of acute pain from one year of onset, which can last for more than 10 years. Chronic pain is distinguished by the muscular ache in the lower spine and extremities, hurting paraesthesia, joint stiffness and radical discomfort. Management includes packing and administration of medications for asystole or nociceptive suffering.

Psychological pain: Speedy decrease in somatic function can cause anxiety or depression. This needs to be identified and treated earlier because the mental state can influence physical recovery. Psychological or psychiatric help may be requested for patients with GBS.

CONCLUSION

GBS is a well-recognized, monophasic, damaging neurologic illness. GBS mainly causes intense, rapid and progressive ascending motor weakness. Special care and attention should be given to patients who require mechanical ventilation. Two therapeutic options such as PE and IVIG have shown an effective reduction in symptoms. Anyway, the condition has 3% to 5% mortality. Newly arising post contaminations types of GBS, for example, those related to COVID 19 should be firmly observed as worldwide pandemics spread.

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