Guillain–Barré Syndrome: A Rare Autoimmune Neurological Condition

Malapati Poojitha[1], Malapati Kavitha[2]
[1,2]*Research scholars,Department of Pharmacy Practice,Santhiram College of pharmacy,Nandyal*

Abstract—**Guillain–Barré syndrome (GBS) is an acute immune-mediated disorder of the peripheral nervous system characterized by rapidly progressive muscle weakness, reduced or absent tendon reflexes, and, in severe cases, respiratory and autonomic dysfunction. It commonly develops following an infection, suggesting an abnormal immune response against peripheral nerve components. This review aims to summarize the clinical features, pathogenesis, diagnosis, and treatment of Guillain–Barré syndrome based on published literature. GBS comprises several clinical variants, including acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute motor-sensory axonal neuropathy (AMSAN). Among the associated infections, Campylobacter jejuni has an important role in disease development. Molecular mimicry between bacterial antigens and peripheral nerve gangliosides may stimulate cross-reactive antibodies, resulting in nerve damage and neurological impairment. Clinical examination forms the basis of diagnosis, supported by cerebrospinal fluid analysis and nerve conduction studies. Early recognition is essential because the disease may progress rapidly and cause serious complications. Intravenous immunoglobulin (IVIG) and plasma exchange are established immunotherapeutic approaches that can improve recovery. In addition, supportive management, including respiratory monitoring, autonomic function assessment, prevention of complications, and rehabilitation, plays an important role in patient care. The clinical course and recovery vary depending on disease severity and individual patient characteristics. Understanding the immunological mechanisms and clinical manifestations of GBS can support timely diagnosis and appropriate management. Further research into disease mechanisms, prognostic indicators, and targeted therapeutic strategies may help improve treatment outcomes and reduce long-term disability.**

Index Terms— **Guillain–Barré syndrome, peripheral neuropathy, molecular mimicry, Campylobacter jejuni, IVIG, plasma exchange.**

I. INTRODUCTION

Guillain- Barre Syndrome (GBS) is a rare sudden, uniphasic autoimmune demyelinating polyradiculoneuropathy that transmitters great patient morbidity, and marked mortality world-wide [1, 8]. Its initial characteristics are increasing motor weakness, sensory and autonomic dysfunction commonly followed by early illness, typically a respiratory infection (difficulty in breathing, weakness of respiratory muscles) and gastrointestinal infections (vomiting, diarrhoea, abdominal pain) [2, 17]. The derive immunopathogenic mechanisms of GBS are comparatively unknown, but it is viewed as a Paradigmatic post- infections auto immune disease. Different mechanisms, such as humoral and biological level immune reactions, autoantibodies and enhance, activated immune cells macrophages (phagocytes/scavenger cells) and lymphocytes (T-cells and B-cells) have been involved in GBS [3, 4]..Gbs is part of the acute flaccid paralysis syndrome in individuals.1st depicted in 1916 in two soldiers by French neurologists Georges Guillain, Jean-Alexander Barre and Andre strohl.The most common infection bacteria associated with GBS are Campylobacter Jejuni. Most infections are caused due to contaminated food, water, Ingestion of raw or under-cooked poultry and unpasteurized milk. Additionally other bacteria include Mycoplasma pneumonia, that cause respiratory illness, Haemophilus influenza, and Borelli aburgorferi [5, 17]. GBS is the most commonly known as acute paralytic neuropathy. It commonly occurs with an acute and fast progression of symmetric fatigue of limbs, reduced reflex response (Hyporeflexia) or no reflex response (Areflexia) and with or without involvement of intercostal muscles or cranial nerve. The most of patients have an infection prior to the

initiation of GBS that is within 4 weeks, most frequently in the form of respiratory problems or gastroenteritis infections. The diagnosis of GBS is based on presentation supported by cerebrospinal fluid (CSF) test and nerve conduction studies described by variable degree of motor weakness, usually present with quadriparesis. Moreover, they present with sensory characteristics, cranial nerve connection, and autonomic disturbances. Men are 1.5 times more probable to be affected than women's and the prevalence, with age from 1 per 100000 in those aged less than 30 years. Approximately 4 cases per 100000 in those older than 75 years [6, 7, 19]. In China the prevalence in adults is 0.66 cases per 100000. The common subtypes of GBS classified based on clinical manifestations, pathophysiology, and electrophysiology findings are shown in ~Table-1. The Generalized treatment in GBS involves general supportive care, specific management with immunotherapy and corticosteroids [3, 8].

Table 1: Common subtypes of Guillain-Barré syndrome [9, 13]

II. PATHOPHYSIOLOGY

Figure 1. Proposed pathophysiological sequence in Guillain–Barré syndrome

The classic pathological picture of Guillain Barre Syndrome is of multifocal mononuclear cell infiltration throughout the peripheral nervous system in which the distribution of inflammation corresponds to the clinical deficit [10]. The triggering events which involve the campylobacter jejuni infection, viral respiratory infection,

COVID-19, autoimmune neuritis. Macrophages invade the myelin sheath & denude the axons. The infectious agent contains antigens that resemble component of peripheral nerves. The antibody &T-cells generated against the pathogen mistakenly attack the body's own peripheral nerves [10, 11]. This targets mainly Schwann cells membrane, Myelin sheath of peripheral nerves, spinal nerve roots, this may lead to loss of myelin which produces the slow nerve conduction, delayed reflex transmission, which shows the ascending weakness, Areflexia, sensory symptoms. [11, 12, 14].

AMAN (Acute Motor Axonal Neuropathy):

The triggering infections (like Campylobacter jejuni infection, cytomegalovirus, Epstein–Barr virus, and Mycoplasma pneumoniae) cause the bacterial surface to contain lipooligosaccharides (LOS) that resemble gangliosides on peripheral motor nerves. [10, 17]. Probably targeted by Fc receptor-mediated binding of antibodies directed against ganglioside antigens on the axolemma, macrophages invade the nodes of Ranvier, where they penetrate between the axon and the surrounding Schwann cell membrane while leaving the myelin sheath relatively intact. [10, 11].Axonal conduction failure with intact myelin leads to motor weakness and paralysis, which may also cause possible distal axonal degeneration in severe cases. [11, 13].

Miller Fisher Syndrome (MFS):

Miller Fisher Syndrome is a distinctive variant of Guillain-Barré Syndrome that mainly affects the nerves responsible for eye movements, coordination, and protective reflexes. In many cases, an infection occurring before the neurological symptoms can trigger an abnormal immune response. [10, 11].

Antibodies produced against the infectious agent may mistakenly recognize similar components present on peripheral nerves, particularly gangliosides such as GQ1b. This immune-mediated process interferes with nerve signal transmission and produces the characteristic clinical features of MFS: ophthalmoplegia (weakness of eye movements), ataxia (loss of coordination), and areflexia (reduced or absent reflexes). Unlike the classical form of GBS, prominent limb weakness may be less marked in MFS.

III. DIAGNOSIS

The diagnosis of Guillain–Barré syndrome (GBS) is primarily clinical, based on progressive bilateral limb weakness and reduced or absent deep tendon reflexes. Cerebrospinal fluid (CSF) analysis typically shows albuminocytological dissociation, characterized by elevated protein levels with a normal or mildly increased white blood cell count. However, CSF protein may be normal during the early stages of the disease [2,3].

Nerve conduction studies and electromyography help confirm the diagnosis and identify electrophysiological subtypes. The Brighton criteria

provide standardized levels of diagnostic certainty based on clinical features, CSF findings, and electrophysiological evidence [17,18]. Differential diagnoses include transverse myelitis, myasthenia gravis, spinal cord compression, and other peripheral neuropathies. Early assessment of respiratory function and autonomic stability is essential to prevent life-threatening complications [2,3].

Figure 2. Clinical assessment and diagnostic work-up for Guillain–Barré syndrome

IV. CLINICAL MANIFESTATIONS

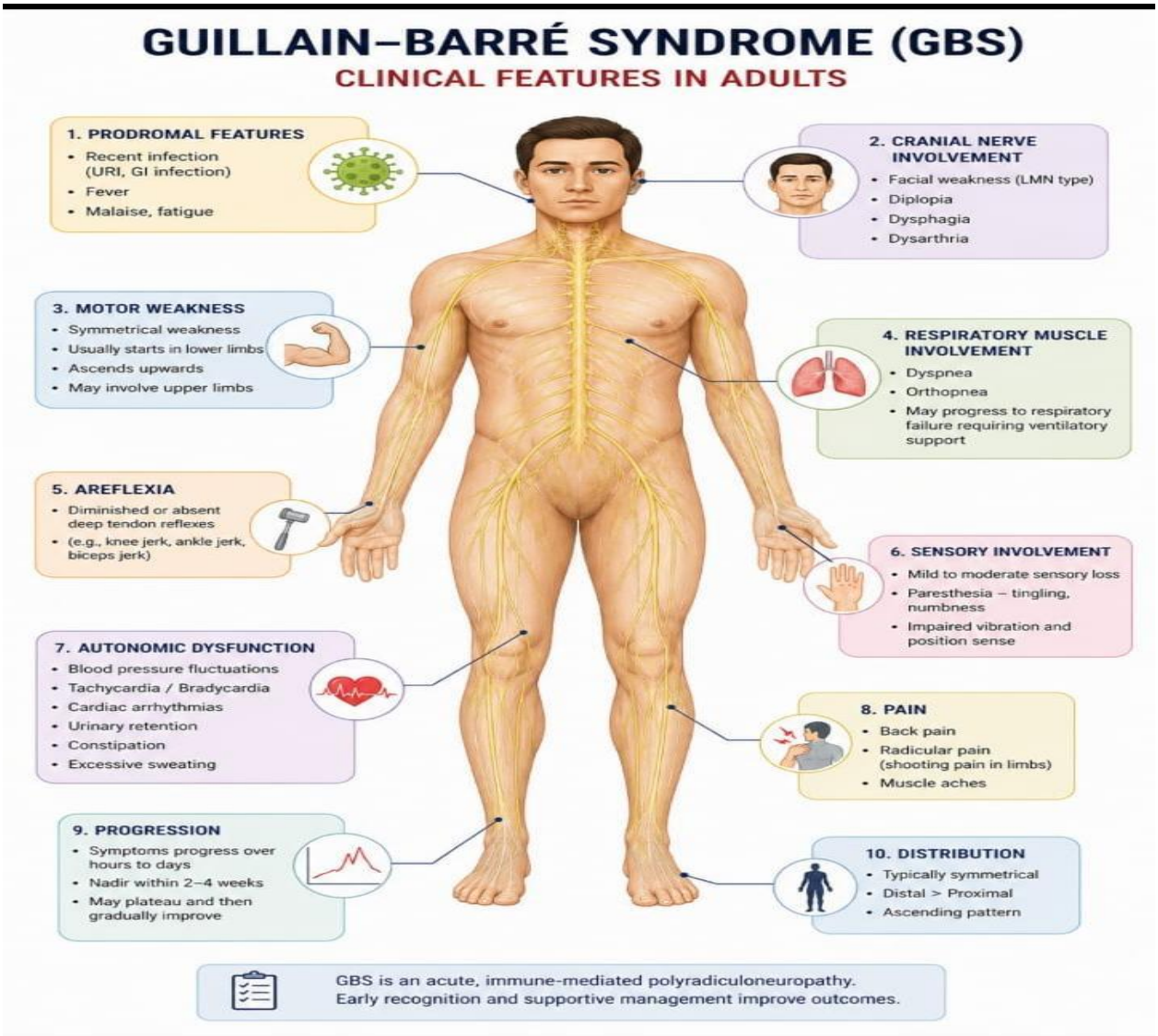


Table 2. Clinical manifestations of Guillain–Barré syndrome

V. TREATMENT

Table 3. Overview of Guillain–Barré syndrome management

Treatment Algorithm:

Figure 3. Treatment algorithm for Guillain–Barré syndrome

VI. CONCLUSION

Guillain–Barré syndrome (GBS) is a rare but potentially life-threatening autoimmune disorder affecting the peripheral nervous system. It is commonly associated with preceding infections, particularly Campylobacter jejuni, which may trigger

an abnormal immune response through molecular mimicry. Early recognition of progressive muscle weakness, reduced or absent tendon reflexes, and other neurological manifestations is essential for timely diagnosis and management. Clinical assessment, cerebrospinal fluid analysis, and nerve conduction studies play important roles in confirming the diagnosis and identifying disease subtypes. Intravenous immunoglobulin (IVIG) and plasma exchange are established treatment options, while respiratory monitoring, autonomic support, prevention of complications, and rehabilitation are essential components of patient care. Although many patients recover with appropriate treatment and supportive management, recovery may vary depending on disease severity and individual factors. Further research into the immunopathogenesis, prognostic indicators, and novel therapeutic approaches is needed to improve clinical outcomes and reduce long-term disability in patients with GB.

VI. TABLES AND FIGURES

The tables and figures below are placed after the References section, as requested. Please insert the original figure images in the marked locations if they are available separately.

Table 1. Common subtypes of Guillain–Barré syndrome

Subtype	Abbreviation
Acute inflammatory demyelinating polyradiculoneuropathy	AIDP
Acute motor axonal neuropathy	AMAN
Acute motor-sensory axonal neuropathy	AMSAN
Miller Fisher syndrome	MFS

Legend: Clinical variants listed in the manuscript. Source citation in text: [9, 13].

Table 2. Clinical manifestations of Guillain–Barré syndrome

Clinical feature	Description / key characteristics	Approximate frequency
Progressive weakness	Symmetric ascending weakness; progresses over hours to days, with peak within 2–4 weeks.	90–95%
Paraesthesia	Tingling, numbness, or pins-and-needles, usually in feet and hands; may precede weakness.	60–70%
Hyporeflexia / areflexia	Diminished or absent deep tendon reflexes; an early and prominent finding.	90–100%
Cranial nerve involvement	May include facial weakness, ocular movement problems, bulbar weakness, and difficulty swallowing or speaking.	20–30%
Autonomic dysfunction	May include blood-pressure or heart-rate fluctuations, arrhythmia, urinary retention, constipation, sweating changes, or dry skin.	20–70%
Respiratory muscle involvement	Dyspnoea or shallow breathing; may progress to respiratory failure requiring ventilation.	20–30%
Pain	Muscle, back, or radicular pain, often affecting the lower back and legs.	30–50%

Legend: Frequencies and descriptions are reproduced from the original manuscript and should be checked against the cited source [8, 14] before submission.

Table 3. Overview of Guillain–Barré syndrome management

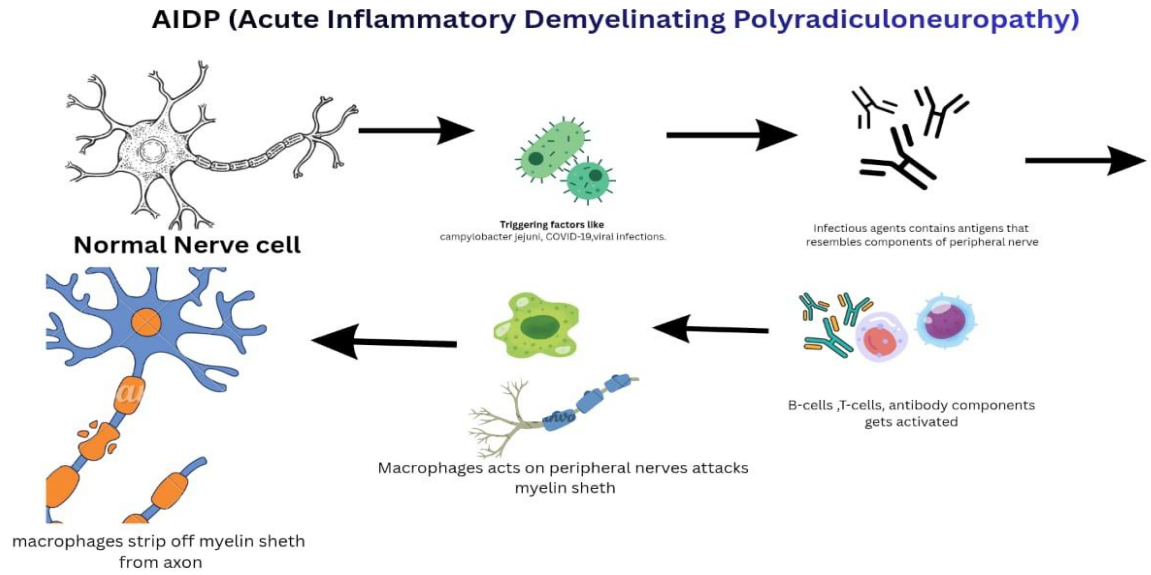
Management area	Interventions listed	Purpose / indication	Notes
Immunotherapy	Intravenous immunoglobulin (IVIG); plasma exchange	Moderate-to-severe GBS	The manuscript states these are effective options and should not be used together.
Supportive care	Analgesia, DVT prophylaxis, gastrointestinal protection, nutrition	Symptom relief and prevention of complications	Individualize care.
Respiratory support	Oxygen therapy; mechanical ventilation when required	Respiratory failure or inadequate ventilation	Monitor respiratory function closely.
DVT prophylaxis	Enoxaparin; compression stockings	Immobile patients	Assess bleeding risk and clinical suitability.

Pain management	Paracetamol; gabapentin	Muscular or neuropathic pain	Monitor response.
Physiotherapy / rehabilitation	Passive and active exercise; physiotherapy; occupational therapy	Functional recovery and mobility	Progress according to tolerance.

Legend: This table summarizes the management options described in the manuscript; it is not a substitute for current clinical guidance. Verify drug details and recommendations before publication.

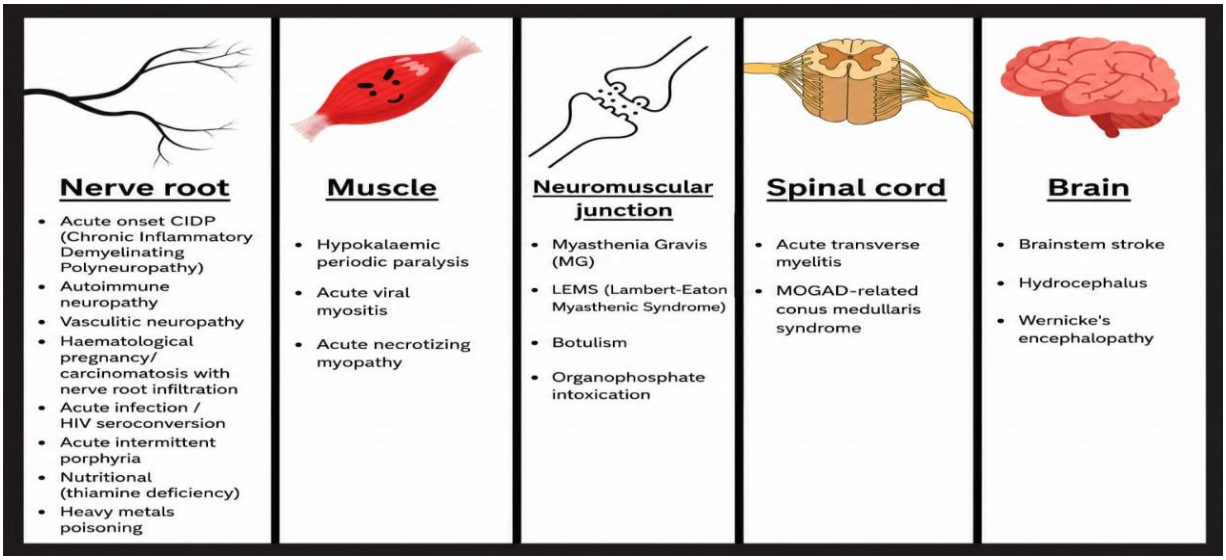
Figures

Figure 1. Proposed pathophysiological sequence in Guillain–Barré syndrome



Legend: A preceding infection may trigger an immune response that cross-reacts with peripheral nerve components, leading to demyelination or axonal injury and neurological weakness.

Figure 2. Clinical assessment and diagnostic work-up for Guillain–Barré syndrome



Legend: Clinical features are assessed alongside cerebrospinal fluid analysis and nerve conduction studies; respiratory and autonomic function should also be evaluated.

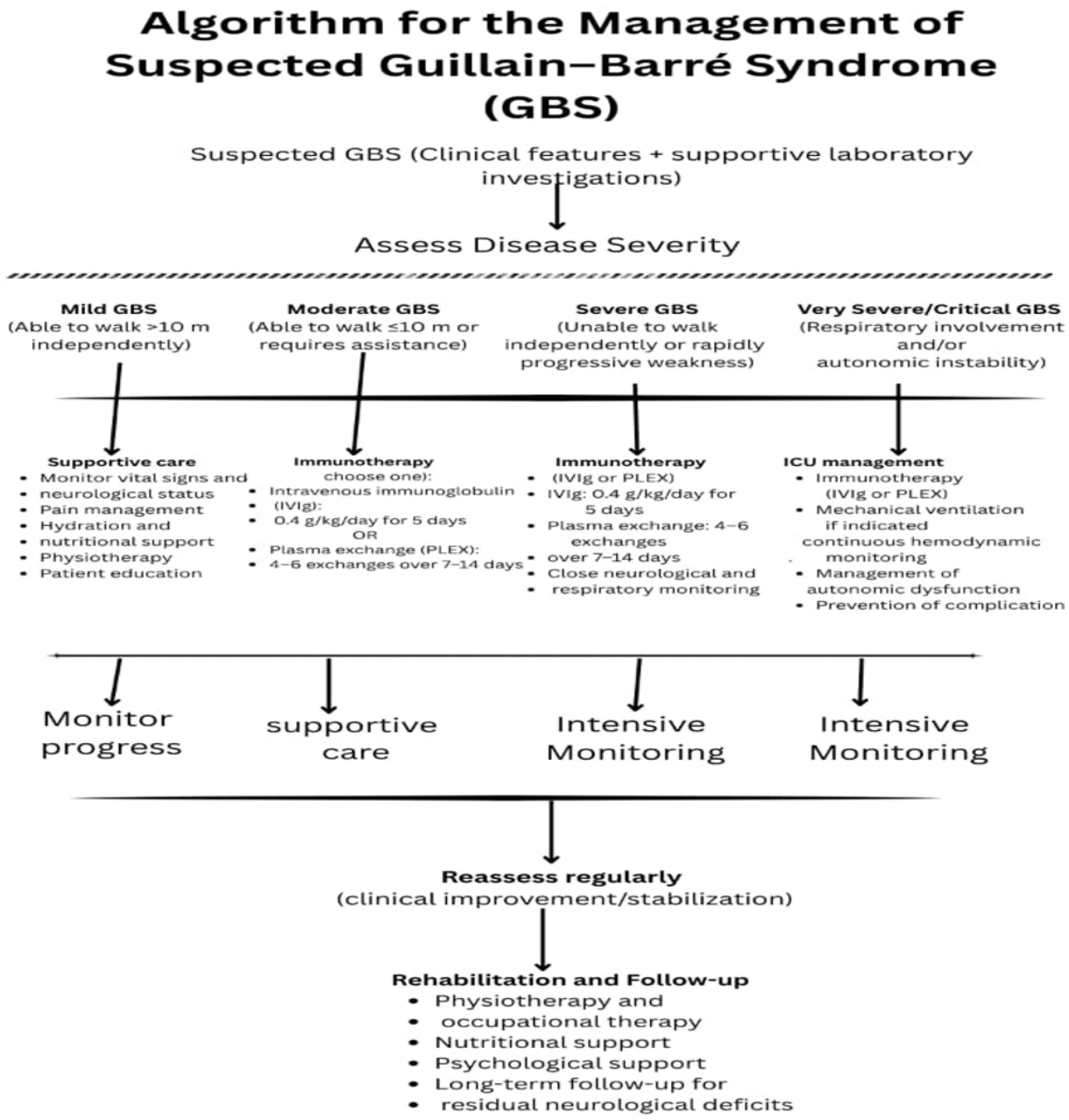


Figure 3. Treatment algorithm for Guillain–Barré syndrome
Legend: Management includes assessment of disease severity, appropriate immunotherapy, respiratory and autonomic monitoring, supportive care, and rehabilitation.

REFERENCES

[1] Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. The Lancet. 2016;388(10045):717-727. doi:10.1016/S0140-6736(16)00339-1.

[2] Leonhard SE, Mandarakas MR, Gondim FAA, et al. Diagnosis and management of Guillain-Barré syndrome in ten steps. Nature Reviews Neurology. 2019;15(11):671-683. doi:10.1038/s41582-019-0250-9.

[3] 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. European Journal of Neurology. 2023;30(12):3646-3674. doi:10.1111/ene.16073.

[4] Yuki N, Hartung HP. Guillain-Barré syndrome. New England Journal of Medicine. 2012;366(24):2294-2304. doi:10.1056/NEJMra1114525.

[5] Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barré syndrome. The Lancet. 2021;397(10280):1214-1228.

[6] McGrogan A, Madle GC, Seaman HE, de Vries CS. The epidemiology of Guillain-Barré syndrome worldwide: a systematic literature review. Neuroepidemiology. 2009;32(2):150-163. doi:10.1159/000184748.

[7] 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.

[8] van Doorn PA, Ruts L, Jacobs BC. Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome. The Lancet Neurology. 2008;7(10):939-950. doi:10.1016/S1474-4422(08)70215-1.

[9] 9. Goodfellow JA, Willison HJ. Guillain-Barré syndrome: a century of progress. Nature Reviews Neurology. 2016;12(12):723-731. doi:10.1038/nrneurol.2016.172.

[10] Yuki N, Susuki K, Koga M, et al. Carbohydrate mimicry between human ganglioside and Campylobacter jejuni lipooligosaccharide causes Guillain-Barré syndrome. Proceedings of the National Academy of Sciences USA. 2004;101(31):11404-11409. doi:10.1073/pnas.0403299101.

[11] Uncini A, Kuwabara S. Electrodiagnostic criteria for Guillain-Barré syndrome: a critical revision and the need for an updated consensus. Clinical Neurophysiology. 2012;123(8):1487-1495. doi: 10.1016/j.clinph.2012.01.025.

[12] Fokke C, van den Berg B, Drenthen J, et al. Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. Brain. 2014;137(Pt 1):33-43. doi:10.1093/brain/awt285.

[13] Dimachkie MM, Barohn RJ. Guillain-Barré syndrome and variants. Neurologic Clinics. 2013;31(2):491-510. doi: 10.1016/j.ncl.2013.01.005.

[14] Kuwabara S. Guillain-Barré syndrome. Current Neurology and Neuroscience Reports. 2007;7(1):57-64. doi:10.1007/s11910-007-0004-0.

[15] 15. Hughes RAC, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. Cochrane Database of Systematic Reviews. 2012;(7):CD002063. doi: 10.1002/14651858.CD002063.pub5.

[16] Raphaël JC, Chevret S, Hughes RAC, Annane D. Plasma exchange for Guillain-Barré syndrome. Cochrane Database of Systematic Reviews. 2012;(7):CD001798. doi: 10.1002/14651858.CD001798.pub3.

[17] Leonhard SE, van der Eijk AA, Andersen H, et al. An international perspective on preceding infections in Guillain-Barré syndrome: the IGOS-1000 cohort. Neurology. 2022;99(12): e1299-e1313. doi:10.1212/WNL.0000000000200885.

[18] Busl KM, Fried H, Muehlschlegel S, et al. Guidelines for neuroprognostication in adults with Guillain-Barré syndrome. Neurocritical Care. 2023;38(3):564-583. doi:10.1007/s12028-023-01707-3.

[19] Doets AY, Verboon C, van den Berg B, et al. regional variation of Guillain-Barré syndrome. Brain. 2018;141(10):2866-2877. doi:10.1093/brain/awy232.

[20] Al-Hakem H, Doets AY, Stino AM, et al. CSF findings in relation to clinical characteristics, subtype, and disease course in patients with Guillain-Barré syndrome. Neurology. 2023;100(23): e2386-e2397. doi:10.1212/WNL.0000000000207282.