WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.63
Volume 15, Issue 13, 1834-1849. Research Article ISSN 2277–7105

THE STUDY OF GUILLAIN BARRE SYNDROME (GBS)- A RARE BUT SERIOUS CONDITION

J. Lurdhu Mary*[1], Dr. Kumar Raja Jayavarapu[1], B. Sagar[2], L. Nandini[3], M.D. Saleha Farheen[4], M.D. Nazmeen[5], Sana[6], S.K. Heena Afsar[7], B. Divya[8], T. Keerthi[9], S. Aravind Kumar[10], B. Muralidhar[11]

[1*]Asst. Professor of Dhanvanthari Institute of Pharmaceutical Sciences.

[1]Principal of Dhanvanthari Institute of Pharmaceutical Sciences.

[2,3,4,5,6,7,8,9,10,11]B. Pharmacy IV Year Students Of Dhanvanthari Institute of Pharmaceutical Sciences, Sujatha Nagar, Kothagudem.

Article Received on 05 June 2026,
Article Revised on 25 June 2026,
Article Published on 01 July 2026

https://doi.org/10.5281/zenodo.21155541

***Corresponding Author**

J. Lurdhu Mary

Asst. Professor of Dhanvanthari Institute of Pharmaceutical Sciences.

How to cite this Article: J. Lurdhu Mary*[1], Dr. Kumar Raja Jayavarapu[1], B. Sagar[2], L. Nandini[3], M.D. Saleha Farheen[4], M.D. Nazmeen[5], Sana[6], S.K. Heena Afsar[7], B. Divya[8], T. Keerthi[9], S. Aravind Kumar[10], B. Muralidhar[11] (2026). The Study Of Guillain Barre Syndrome (Gbs)- A Rare But Serious Condition. World Journal of Pharmaceutical Research, 15(13), 1834-1849.

ABSTRACT

Guillain -Barre syndrome (GBS) is an immune-mediated disorder of nervous system and a recognized cause of generalized progressive paralysis worldwide. The most common symptoms of GBS include Quadriparesis, paresthesia of both lower and upper limbs which is ascending in nature and difficulty in standing from sitting position. GBS is rare, incidence worldwide is estimated to be 0.6-4/100,000 person/year. The commonly recognized variants of GBS are AIDP, AMAN, AMSAN and Miller - Syndrome (MFS). In these variants AIDP is the most prevalent form and accounts for 70-90 per cent of cases. Many antecedent events of GBS Ave been identified including Cytomegalovirus infection, Campylo bacter jejune, gastroenteritis, Mycoplasma pneumonia, influenza virus and Epstein Barr virus infections. Awareness of GBS complications, their detection and management may help to prevent the morbidity rate of GBS. The diagnosis and management of GBS can be complicated as its clinical presentation and disease course are heterogeneous. The first symptoms of this disorder include varying degrees of weakness or tingling sensations in the legs. The common manifestations is limb weakness, more proximal than distal. This study was aimed to document the clinical findings, assess the risk factors, diagnosis and treatment

pattern in variants of GBS (i.e. Acute Demyelinating Polyneuropathy (AIDP), Acute Motor Axonal Neuropathy(AMAN), and Acute Motor Sensory Axonal Neuropathy (AMSAN) among the patients with GBS during the hospital stay.

KEYWORDS: Guillain-Barre syndrome, Acute inflammatory demyelinating polyneuropathy, (AIDP), AcuteMotor Sensory Axonal Neuropathy (AMSAN), Retro-Prospective study.

INTRODUCTION

Guillain –barre syndrome is an auto immune disorder of peripheral nervous system, often it presents with acute onset characterized by generalized progressive weakness of upper and lower limbs, paresthesia and complete areflexia. In this autonomic dysfunction are common with conventional manifestations as loss of vasomotor control with wide fluctuation in blood pressure, postural hypotension and cardiac arrhythmias. Oropharyngeal weakness and respiratory failure may require mechanical ventilation in about one – third of hospitalized patients making it a disease of vital importance for early management.The report occurrence for GBS is 1-2 per 100,000 population and increases linearly with age, and men are about 1.5 times more affected than women.[1] Primary infections such as upper respiratory tract infections are well known preceding event in the development of GBS have been identified including Campylobacter jejune gastroenteritis, Cytomegaly virus, Mycoplasma Pneumoniae, Epstein Barr virus and Influenza virus infections. The common types of GBS are acute inflammatory demyelinating polyneuropathy. (AIDP), acute motor axonal neuropathy, (AMAN), acute motor sensory axonal neuropathy (AMSAN) and Miller Fisher Syndrome (MFS). AIDP is the most prevalent form and accounts for 70-90 per cent of cases. Primary infection such as upper respiratory tract infection is well known event in the pathophysiology of GBS. Many antecedent events are associated withGBS such as Campylobacter genus gastroenteritis, Mycoplasma pneumonia, Cytomegalovirus, Influenza virus and Epstein Barr virus infections.The clinical manifestations of GBS is ascending, progressive, symmetrical flaccid limbs paralysis, cranial nerve involvement and hyporeflexia which is progress over the course of days to several weeks.[2]

SIGNS AND SYMPTOMS: The primary symptoms of this disease include varying degrees of weakness or tingling sensation in the legs. The commonest manifestation is limb weakness, more proximal than distal.Facial palsy is the commonest type of cranial nerve involvement

followed bybulbar weakness, ophthalmoplegia and tongue weakness. In many cases, the weakness and abnormal sensations spread to the upper body.

RISK FACTORS: Age: risk increases with age. Sex: males are higher risk than female to GBS. Campylobacter jejune bacterial infection, HIV, Epstein Barr virus (EBV), these are associated with cases of GBS. History of surgeries and vaccination are also linked to GBS in rare cases.

Table 1: Clinical Features Of Gbs.

MOTOR DYSFUNCTION	SENSORY DYSFUNCTION	AUTONOMIC DYSFUNCTION
Areflexia Neck muscle weakness Cranial nerve palsies Symmetrical limb weakness	Pain Ataxia Numbness Paresthesia Loss of joints position sense Touch and pain sensation	Hyper salivation Tonic pupils Gastric disturbances Hypertension and postural hypotension Urinary sphincter disturbances Wide fluctuation of pulse and blood pressure Cardiac arrhythmia

PATHOPHYSIOLOGY

The exact pathophysiology of GBS is still not clear, but molecular mimicry shows a possible mechanism of pathogenesis.The important surface molecules of the nervous system is Gangliosides. Based on the concept of molecular mimicry, antibodies are formed against the Gangliosides in the lipopolysaccharide moiety of Campylobacter jejune, which cross react with peripheral nerves causing damagen. Incytomegalovirus infection more often Anti-GM2 antibodies are found.[3]

AIDP

The classic pathological picture of Guillain -Baree syndrome is of multifocal mononuclear cell infiltration throughout the peripheral neurons system in which the distribution of inflammation corresponding to the clinical shortage. Then macrophages invade the myelin sheaths and deprive the axons. Based on one hypothesis, the activated macrophages are targeted to antigens, on the surface of the myelin sheath of Schwann cells by activated T lymphocytes, which are major actors in autoimmune neuritis.[4] The primary invasion of the Schwann cell membrane is a result of toxic nitric oxide radicals, matrix metalloproteinases and other mediators released by activated macrophages. In severe lesions, the axons are also

damaged likelihood as a secondary or "bystander'' results of the radicals and toxic enzymes by the immune mediated inflammatory response directed against the myelin sheath.[5]

AMAN

The pathological mechanism is different in AMAN. Certainly targeted by their Fc-receptor-mediated binding of antibodies directed against ganglioside antigens on the axolemma. Primarily the macrophages invade the nodes of Ranvier where they insert between the surrounding Schwann cell axolemma and the axon, leaving the myelin sheath intact. In severe cases, severe degeneration of the whole axon may occur due to the axons are damaged in the ventral root. In AMAN patient are quickly recover than patient in AIDP. This is because of in AMAN the pathological process blocks conduction but not severe the axon.[6]

AMSAN

The pathology in AMSAN resembles that in AMAN, with the same pattern of macrophage invasion of the perinodal space. However, with AMSAN, the dorsal, as well as the ventral, roots are affected.

Fisher's syndrome

The pathophysiology of the Fisher's syndrome is not clear so far. Since it is benign condition, uncomplicated cases do not come to autopsy, and the affected parts of the nervous system cannot be biopsied. The primary electro physiological finding in Fisher's syndrome is an abnormality of sensory conduction. Sensory nerve action- potential amplitudes initially fall and then return to normal along with clinical improvement. The time course of these changes is consistent with sensory peripheral nerve demyelination or conduction failure along the axon, not axonal loss followed by regeneration.[7]

TABLE 2.

VARIANTS OF GBS	RELATED ANTIBODIES
Acute inflammatory demyelinating polyneuropathy (AIDP)	Unknown
Acute motor axonal neuropathy (AMAN)	GM1, GM1b, GD1a, GalNac-GD1a
Acute motor sensory axonal neuropathy (AMSAN)	GM1, GM1b, GD1a.
Fisher's syndrome	GQ1b, GD1a
Fisher's syndrome overlaps with Guillain-Barre syndrome	GQ1b, GM1, GM1b, GD1a, GalNac-GD1a

DIAGNOSIS

GBS is a clinical diagnosis, with areflexia and progressive weakness evolving over less than four weeks. The first symptoms of the Guillain –Barre syndrome are paresthesia, weakness, pain in the limbs, numbness or some combination of these symptoms. The primary features are progressive bilateral and relatively symmetric weakness of the limbs, and the weakness progresses over a period of 12 hours to 28 days before a plateau is reached to.[8]

Typical clinical features

These are bilateral progressive weakness of the arms and legs, without CNS involvement. Often GBS present with sensory loss or Paresthesia followed by weakness that starts in lower limbs and progresses to the upper limbs and cranial muscles.Reflexes are absent or decreased in most of the patients in GBS.[9] Dysautonomia is common and it can include pupillary disfunction, heart rate or blood pressure instability, and bladder or bowel dysfunction. Pain is frequently reported and can be radicular, muscular and neuropathi. Within two weeks the maximum disability is reached.

Atypical clinical presentation

GBS can also involve in an atypical manner. Sensory signs and weakness primarily start in the arms and legs or synchronously in all limbs. Children who have less than 6 years old can present with features of atypical manner such as irritability and poorly localized pain. Delay in diagnosis of GBS might causes by failure to recognize these signs as an early presentation.

CSF ANALYSIS

In this test an elevated protein levels and fewer mononuclear cells/mm3 are strongly support the diagnosis.

During the first week CSF protein levels are normal. In one of the studies 12% of patients were found to have more than 5 cells / microliter in the CSF.[10]

NERVE CONDUCTION STUDIES

These studies are assists to diagnose Guillain -Barre syndrome in clinical practice. It’s needed to meet all criteriaof Brighton for Guillain Barre syndrome.[11] NCS are essential for classification of Guillain Barre syndrome in AIDP or AMAN.

In this AIDP features of demyelination is prolonged distal motor latency, decreased motor nerve conduction velocity, temporal depression, conduction blocks and increased F – wave

latency. In other hand AMAN shows no features of demyelination, one demyelinating feature in one nerve, if distal CMAP amplitude is less than 10%LLN, can be found; distal CAMP amplitude less than 80% LLN in at least two nerves. Conduction block of Transient motor nerve might be present.[12]

LLN- lower limit of normal

CAMP – compound muscle action potential

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of CNS Encephalitis, progressive limb weakness, transverse myelitis, acute disseminated encephalomyelitis, leptomeningeal malignancy.

MOTOR NEURONS

Poliomyelitis, amyotrophic lateral sclerosis, West Nile virus anterior myelitis, progressive spinal muscular atrophy.

Plexus

Diabetes mellitus Nerve roots, acute onset chronic inflammatory demyelinating neuropathy, cytomegalovirus related radiculitis, Lyme disease, leptomeningeal malignancy and HIV – related radiculitis.[13]

Peripheral nerves

Guillain Barre syndrome, Lyme disease, porphyria, diphtheria, vasculitis, chronic inflammatory demyelinating neuropathy, metabolic disorders.

Neuromuscular junction

Myasthenia gravis, intoxication of muscles, acute cardiomyopathy, dermatomyositis.[14]

Diagnostic criteria for Guillain -Barre syndrome

1. Progressive weakness in arms and legs
2. Areflexia weak limbs.

Additional symptoms

1.Mild sensory symptoms absence in AMAN)

2. Progressive phase lasts days to 4 weeks.[15]

TABLE 3: Electrophysiological features of GBS.

Reduced conduction velocity. Conduction block or abnormal temporal dispersion. Prolonged terminal latency. Absent F wave or prolonged F wave latency.	Absent or reduced compound muscle action potential (CAMP) amplitude. Normal motor terminal latency and conduction velocity. Normal sensory nerve action potential(snap).	Absent or reduced SNAP amplitude. Absent or reduce CAMP amplitude. Normal motor terminal latency and conduction.

TREATMENT

Both plasma therapy and IVIG shows good outcomes in GBS.

About plasma exchange

In 1978, Brittle et al first observed the outcomes of plasma exchange in a patient following with Guillain -Barre syndrome.[16] In the first two weeks of the illness, plasma exchange is beginning it decreased the period of hospital stay, time to reach ambulation and mechanical ventilation. In a Guillain -Barre syndrome study the French cooperative group of plasma exchange recommends four exchanges in moderate to severe cases and two exchanges in mild cases, based on their over 500 patients involving.[17] This study includes the plasma exchange complications are septicemias, hypocalcemia, hypotension, and abnormal clotting.[18]

About Intravenous Immunoglobulin therapy

A retrospective study proved that the accelerated children recovery in Guillain -Barre syndrome with IVIG who were unable to walk. Currently the preferred choice of Guillain -Barre syndrome in treating is remains IVIG.[19]

IVIG is used in the treatment of several immunologically mediated disorders. It is supposed to act through several mechanisms including anti-idiotypic suppression of autoantibodies. The IVIG mechanism of action is probably multifactorial and uncertain, including the provision of anti-idiotype antibodies, interference with complete activation and blocked of Fc receptors. Antibodies increased catabolism may also play a part.[20] A large, randomized, multicenter trial compared Intravenous Immunoglobulin,plasma exchange, and combination of both there was no significant difference in efficacy between plasma exchange and IVIG. No evidence was in significant advantage in combined treatment. 0.4 mg/kg / day for five days gives significant outcomes in GBS.[21]

TABLE 4.

Contraindications of IVIG	Adverse effects of IVIG
Severe congestive cardiac failure	Nausea and vomiting
Selection IgA deficiency	Myalgia
Anaphylaxis following previous	Aseptic meningitis
Renal insufficiency	Fever and chills

Corticosteroids

Methylprednisolone (MP) is mostly used in the treatment of GBS. Few studies suggest that MP along with IVIG shows better outcomes in GBS.

Mortality in Guillain-Barre syndrome dramatically dropped with the advent of intensive care and safe ventilation. Clinical studies document pulmonary emboli, infections, and cardiac arrhythmias as the major causes of death.[22]

When to admit in the ICU

The following are reasons to admit patients to intensive care unit (ICU)

Severe swallowing dysfunction, respiratory insufficiency, rapid progression of weaknesses and severe automatic cardiovascular dysfunction.[23]

Respiratory Care

Respiratory failure is also a life threatening and most common complication of GBS. The mortality rate in GBS exceeded 30%, prior to mechanical ventilation mostly from respiratory failure. The higher incidence of ventilator – associated pneumonia is associated with delaying of tracheostomy >14 days after intubation.[24]

Table 5: Factors associated with poor outcome of GBS.

Etiology Cytomegalovirus Previous gastro intestinal infection Campylobacter jejune, infection	**Clinical features** Older age Longer time to clinical improvement Greater disability and disease sever
Biochemical markers Neuro specific enolase and S-100b protein in CSF	**Electrophysiology** In exit table nerve
Anti-GM1 antibodies	Absentor reduced CAMP (<20% of the lower limit of normal)

Dysautonomia

Autonomic dysfunction occurs to some degree in 65% of patients with GBS. The including manifestation are Brady or tachy-arrhythmias, abnormal hemodynamic responses to

vasoactive medications, sweating abnormalities and gastrointestinal dysfunction.[25] Autonomic dysfunction has 2 important implications. Patients who are at risk for automatic complications are at risk for produce other complications such as cardiovascular collapse, arrhythmia, and blood pressure fluctuations. In some patients, gastrointestinal autonomic dysfunction results in gastric, constipation and ileus. A Retrospective study has found that up to 15% patients with GBS needing ICU care in ileus.[26]

MATERIALS AND METHODS

This study on Guillain-Barre Syndrome was conducted using a hospital-based observational research design to evaluate the clinical features, diagnosis, treatment, and outcomes of patients affected by the disease.[27] Patients diagnosed with Guillain-Barre Syndrome according to established clinical and neurological criteria were included in the study after obtaining informed consent. Demographic details, medical history, presenting symptoms, neurological findings, laboratory reports, treatment information, and follow-up data were collected using structured case record forms. The materials used for the study included patient medical records, clinical assessment tools, laboratory equipment, cerebrospinal fluid [CSF] collection kits, blood collection materials, biochemical analyzers, and electrophysiological instruments for nerve conduction studies and electromyography.[28]

Clinical assessment was performed to evaluate muscle weakness, sensory disturbance, loss of deep tendon reflexes, cranial nerve involvement, respiratory difficulties, and functional disability. Laboratory investigations included protein blood tests such as complete blood count, blood glucose estimation, liver and renal function tests, and electrolyte analysis. Cerebrospinal fluid examination was carried out through lumbar puncture to identify the characteristic finding of albumin-cytological dissociation, which is marked by elevated protein levels with a normal or slightly increased cell count. Electrophysiological studies, including nerve conduction studies and electromyography, were conducted to confirm the diagnosis, determine the severity of nerve damage, and classify the various subtypes of Guillain-Barre Syndrome.[29]

Data regarding treatment modalities such as intravenous immunoglobulin therapy, plasmapheresis, supportive care, and mechanical ventilation when required were also collected and analyzed. Patients were followed throughout their hospital stay and during follow-up visits to assess recovery, improvement in muscle strength, mobility status, duration of hospitalization, complications, and overall clinical outcomes. The collected data were

entered into statistical software such as SPSS and analyzed using appropriate descriptive and inferential statistical method. Ethical approval for the study was obtained from the institutional ethics committee, and all patient information was kept confidential. The results obtained from the collected clinical, laboratory, and treatment data were analyzed and interpreted to provide a comprehensive understanding of the diagnosis, management, and prognosis of Guillain-Barre Syndrome.[30]

CONCLUSION

In this six months study we found NCSs are play vital role in clinics to know the severity of disease and variant of GBS.By assessing the risk factors and antecedent events the cause of GBS can be assessed.Based on the findings from the study,in few patients along with medications adherence,complete stoppage of alcohol abuse is very important to recover quickly from the condition and non-pharmacological measures to be taken to prevent further complications and recurrent onsets of GBS.In this study corticosteroids also shows better outcomes in all variants of GBS.Few patients who are not respond to corticosteroids, were treated with IVIG or combination of both.

RESULTS TABLE

Table 6: Gender Wise Distribution.

GENDER	TOTAL NO. OF PATIENTS
MALE	19(76%)
FEMALE	6(24%)

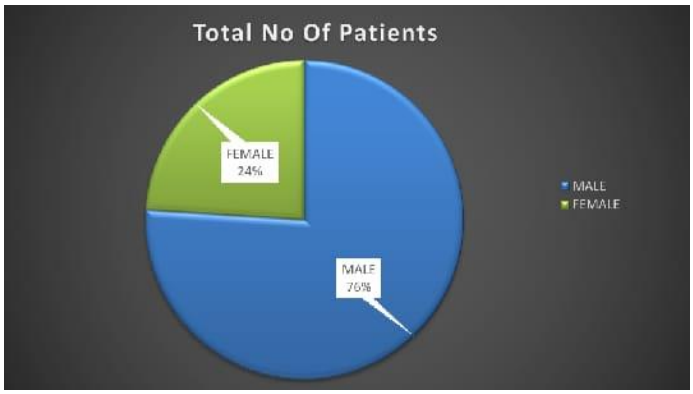


TABLE 7: AGE WISE DISTRIBUTIONS.

AGE	MALE	FEMALE	TOTAL
15- 40	12(48%)	3(12%)	15(60%)
41-60	5(20%)	2(8%)	7(28%)
61-80	2(8%)	1(4%)	3(12%)

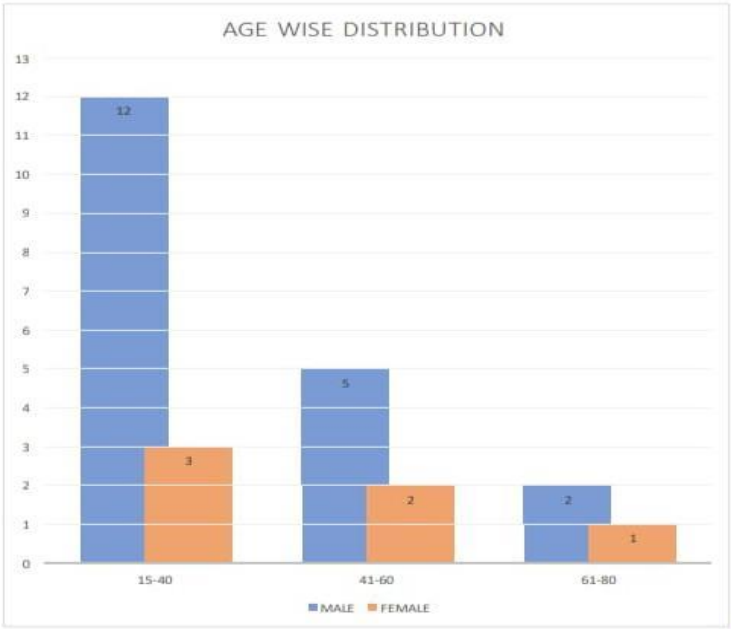


Table 8: Antecedent Events Oberved In Patients.

ILLNESS	MALE	FEMALE	TOTAL
None	13(52%)	5(20%)	18(72%)
Diarrhea	1(4%)	1(4%)	2(8%)
Flu like symptoms	3(12)	2(8%)	5(20%)
History of surgery	2(8%)	1(4%)	3(12%)
Recurrent onset of GBS	1(4%)	0(0%)	1(4%)

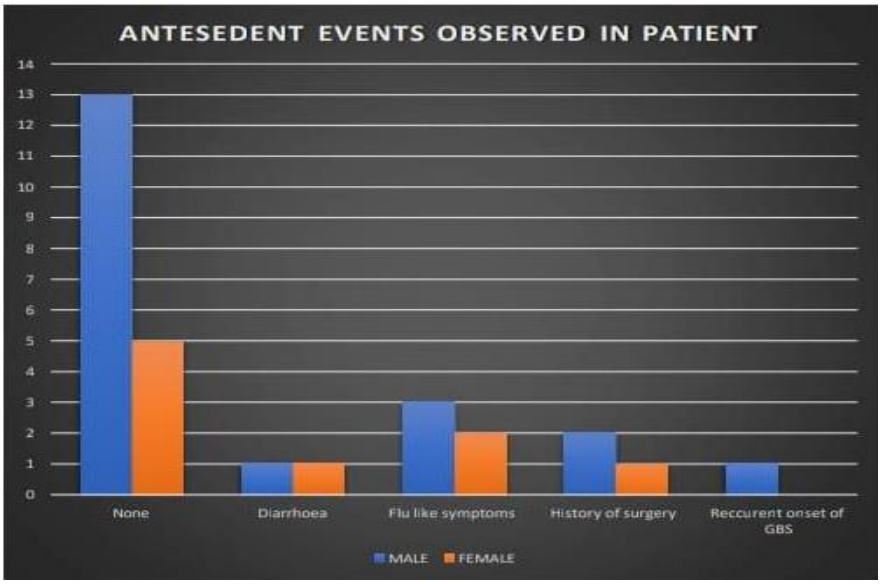


Table 9: Signs And Symptoms In Patients.

SIGNS AND SYMPTOMS	MALE	FEMALE	TOTAL
QUADRIPARESIS	19(76%)	6(24%)	25(100%)
PARAESTHESIA	14(56%)	4(16%)	18(72%)

DIFFICULTY IN WALKING	15(60%)	1(4%)	16(64%)
DIFFICULTY IN STANDING FROM SITTING POSITION	15(60%)	4(16%)	19(76%)
BREATHLESSNESS IN WALKING	8(32%)	2(8%)	10(40%)
FEVER	3(12%)	2(8%)	5(20%)

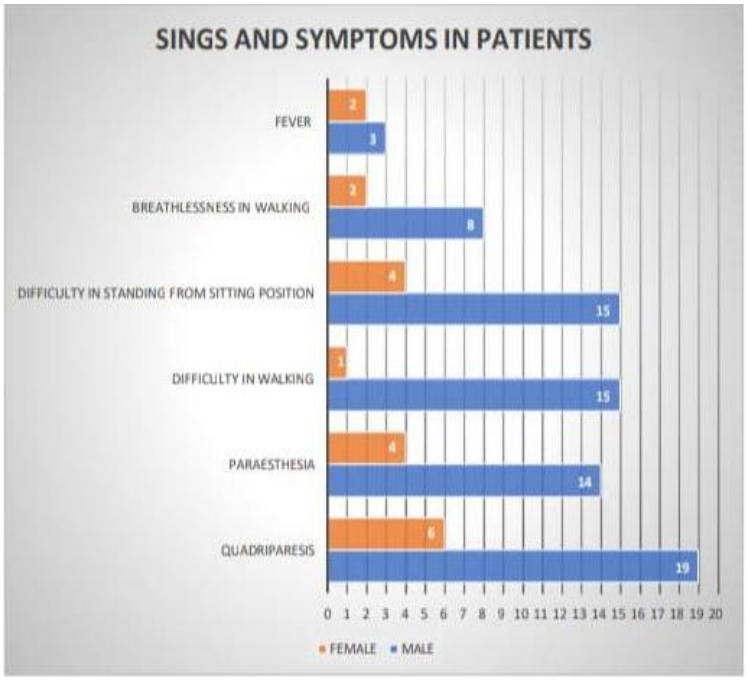


Table 10: Medication Adherence.

DIAGNOSTIC PARAMETERS	NO OF PATIENTS
NCS	18(72%)
CSF	2(8%)
BOTH	5(20%)

NOTE: NCS (Nerve conduction study)

CSF (Cerebrospinal Fluid)

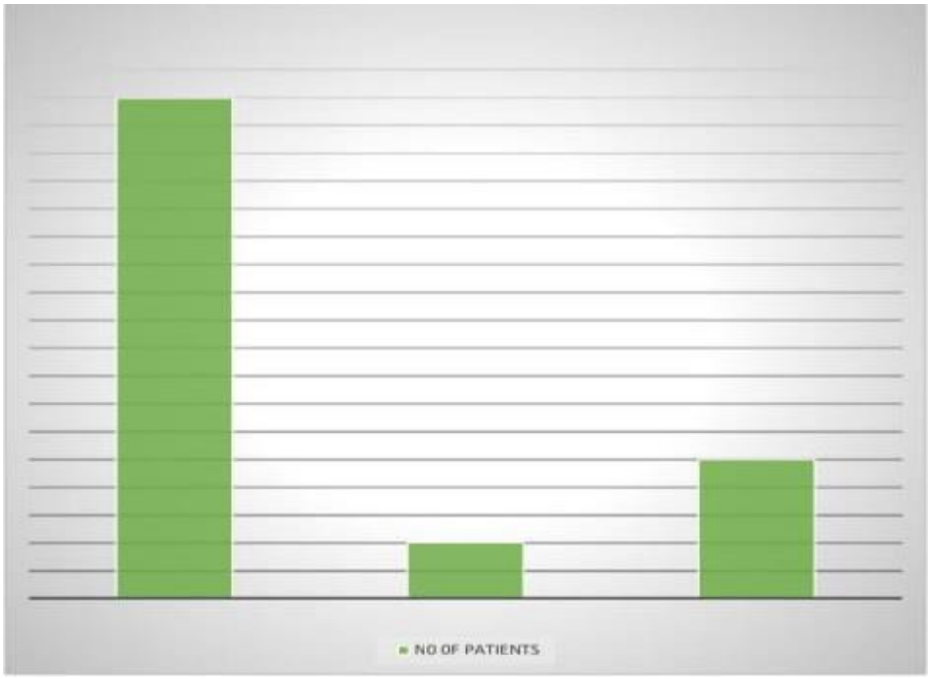


Table 11: Risk Factors Observed In Patients.

RISK FACTORS	MALE	FEMALE	TOTAL
None	12(48%)	3(12%)	15(60%)
Alcoholism	7(28%)	0(0%)	28%)
Smoking	5(20%)	0(0%)	5(20%)
Tobacco chewer	3(12%)	0(0%)	3(12%)
Hypothyroidism	0(0%)	2(8%)	2(8%)

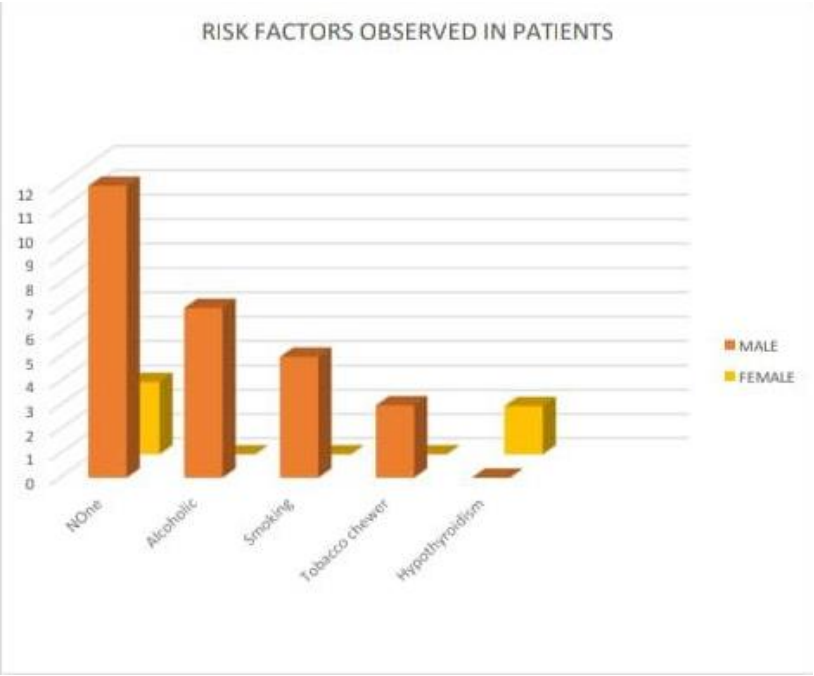


Table 12: Variants Of Gbs.

VARIANT	MALE	FEMALE	TOTAL
AIDP	12(48%)	3(12%)	15(60%)
AMAN	1(4%)	0(0%)	1(4%)
AMSAN	1(4%)	1(4%)	2(8%)
POST COVID GBS	2(8%)	2(8%)	4(16%)
RECURRECT GBS	1(4%)	0(0%)	1(4%)

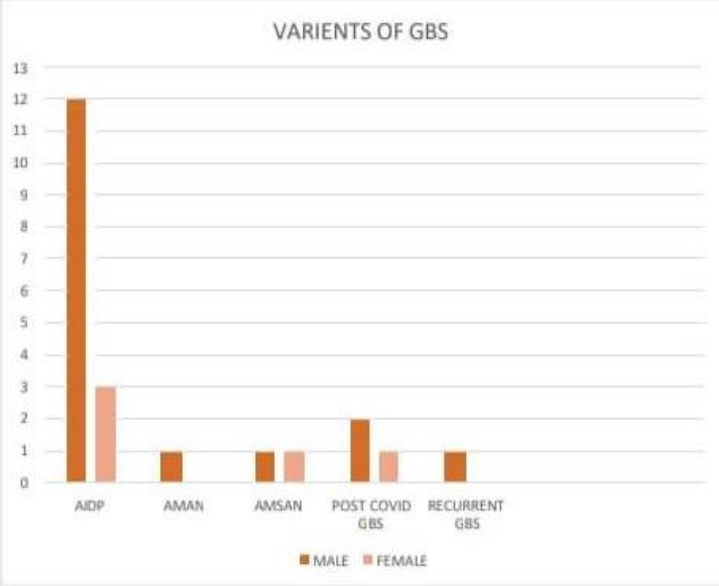
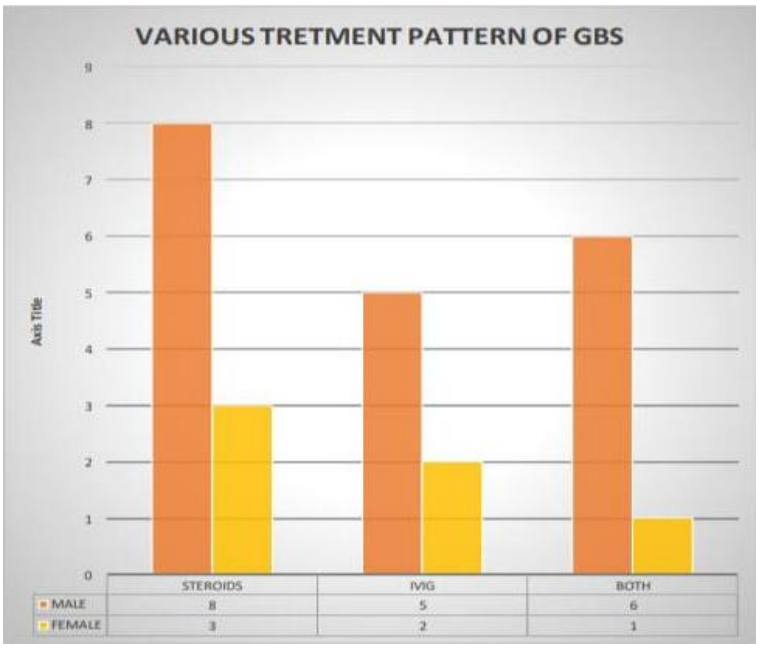


Table 13: Treatment Pattern.

DRUG	MALE	FEMALE	TOTAL
CORTICOSTEROIDS	8(32%)	3(12%)	11(44%)
IVIG	5(20%)	2(8%)	7(28%)
BOTH	6(24%)	1(4%)	7(28%)



REFERENCES

1. Bellanti R, Rinaldi S. Guillain-Barré syndrome: a comprehensive review. *European Journal of Neurology*., 2024; 31(8): 16365.
2. Hughes RAC. Guillain-Barré syndrome: History, pathogenesis, treatment, and future directions. *European Journal of Neurology*., 2024; 31(11): 16346.
3. Yuki N, Hartung HP. Guillain-Barré Syndrome. *New England Journal of Medicine*., 2012; 366(24): 2294–2304.
4. van den Berg B, Walgaard C, Drenthen J, et al. Guillain–Barré syndrome: pathogenesis, diagnosis, treatment and prognosis. *Nature Reviews Neurology*., 2014; 10(8): 469–482.
5. Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. *Lancet*., 2016; 388(10045): 717–727.
6. 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.
7. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome. *Neuroepidemiology*., 2011; 36(2): 123–133.
8. McGrogan A, Madle GC, Seaman HE, de Vries CS. The epidemiology of Guillain-Barré syndrome worldwide. *Neuroepidemiology*., 2009; 32(2): 150–163.
9. Wijdicks EFM, Klein CJ. Guillain-Barré syndrome. *Mayo Clinic Proceedings*., 2017; 92(3): 467–479.
10. Ropper AH, Samuels MA. *Adams and Victor's Principles of Neurology*. 11th ed. New York: McGraw-Hill; 2019.
11. Ropper AH. The Guillain-Barré syndrome. *New England Journal of Medicine*., 1992; 326: 1130–1136.
12. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. *Annals of Neurology*., 1990; 27: 21–24.
13. Cornblath DR, Hughes RAC. Guillain-Barré syndrome and variants. *Neurologic Clinics*., 2013; 31(2): 491–510.
14. Hughes RAC, Cornblath DR. Guillain-Barré syndrome. *Lancet*., 2005; 366(9497): 1653–1666.
15. Jacobs BC, Rothbarth PH, van der Meché FGA, et al. The spectrum of antecedent infections in Guillain-Barré syndrome. *Neurology*., 1998; 51(4): 1110–1115.
16. Willison HJ, O'Hanlon GM. The immunopathogenesis of Miller Fisher syndrome and Guillain-Barré syndrome. *Journal of Neuroimmunology*., 1999; 100(1-2): 3–12.

17. Nachamkin I, Allos BM, Ho T. Campylobacter species and Guillain-Barré syndrome. *Clinical Microbiology Reviews*., 1998; 11(3): 555–567.
18. Dimachkie MM, Barohn RJ. Guillain-Barré syndrome and variants. *Neurologic Clinics*., 2013; 31(2): 491–510.
19. van Doorn PA. Treatment of Guillain-Barré syndrome and CIDP. *Journal of the Peripheral Nervous System*., 2005; 10(2): 113–127.
20. Hughes RAC, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. *Cochrane Database of Systematic Reviews*., 2014.
21. Kuwabara S. Guillain-Barré syndrome: epidemiology, pathophysiology and management. *Drugs*., 2004; 64(6): 597–610
22. Chevret S, Hughes RAC, Annane D. Plasma exchange for Guillain-Barré syndrome. *Cochrane Database of Systematic Reviews*., 2017.
23. Lawn ND, Wijdicks EFM. Intensive care management of Guillain-Barré syndrome. *Muscle & Nerve*., 1999; 22(8): 1022–1030.
24. Walgaard C, Lingsma HF, Ruts L, et al. Prediction of respiratory insufficiency in Guillain-Barré syndrome. *Brain*., 2010; 133(3): 670–677.
25. Uncini A, Kuwabara S. Electrodiagnostic criteria for Guillain-Barré syndrome. *Clinical Neurophysiology*., 2012; 123(8): 1487–1495.
26. Rodríguez Y, Rojas M, Pacheco Y, et al. Guillain–Barré syndrome, transverse myelitis and infectious diseases. *Cellular & Molecular Immunology*., 2018; 15(6): 547–562.
27. Kieseier BC, Hartung HP. Therapeutic strategies in Guillain-Barré syndrome. *Seminars in Neurology*., 2004; 24(2): 187–194.
28. Vucic S, Kiernan MC, Cornblath DR. Guillain-Barré syndrome: an update. *Journal of Clinical Neuroscience*., 2009; 16(6): 733–741.
29. Fokke C, van den Berg B, Drenthen J, et al. Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. *Brain*., 2014; 137(1): 33–43.
30. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barré syndrome. *Lancet*., 2021; 397(10280): 1214–1228.