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ASSESSMENT OF CLINICAL PROFILE DATA AND TREATMENT PATTERN OF GBS PATIENTS.

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1] ABSTRACT

Introduction: Guillain-Barré syndrome (GBS) is a very rare immune mediated disorder which is associated with demyelination of peripheral nervous system and progressive muscle weakness that occurs mostly in previously healthy individuals. It usually presents with ascending paralysis and is severe enough to warrant hospital admission for its management. Methodology: The current study is a retrospective, observational study was conducted for the assessment of clinical profile data and treatment pattern of Guillain-Barré syndrome. Result: According to our data, we observe that treatment like IVIG, Plasmapheresis, Nutrition therapy; Analgesic etc. are the common medication prescribed by the physicians as per patient condition. AIDP is the most common sub-type of GBS was observed in patients, with about 60% AIDP, 20%AMAN, 20% MFS patients were observed. The mostly observed symptoms in the GBS are weakness of limbs, bifacial weakness, slurring of speech and fever. As per our data about 72% of males and 28% of females were diagnosed with GBS. Conclusion: Both IVIG and Plasmapheresis treatment showed significant improvement within our study setting IVIG was the preferred treatment option due to low side effect profile and ease of administration however cost of treatment is higher compared to Plasmapheresis.

Keywords: Guillain-Barré syndrome, immune mediated disorder, Plasmapheresis

French neurologists Georges Guillain, Jean-Alexandre Barre and Andr'e Strohl, in the year 1916, observed acute areflexic paralysis in two individuals which recovered with time. They found an increased protein concentration in the cerebrospinal fluid Called as Guillain–Barre syndrome (GBS), it was described as a clinical condition characterised by rapid spread of ascending upper or lower limb weakness, defective tendon reflexes, weak sensory signs and possible autonomic dysfunctions. In the western countries, post-polio eradication, GBS is a prominent cause of acute flaccid paralysis, bearing resemblance with poliomyelitis and many other non-specific conditions like AIDS post-surgery, etc. Inflammation of peripheral nerves, to a larger extent, causes GBS. Sensory information

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like pain, temperature is conveyed from the body to the brain and motor signals like movements, etc., are conveyed from the brain to the body by these nerves. A tingling sensation in the legs and arms is felt in GBS. This could be severe and could lead to an ascending paralysis. Legs become weak and the paralysis then sets in the upper limbs and the face. During this time, there is usually a complete loss of deep tendon reflexes. Infections with a number of bacteria and viruses are linked to the onset of GBS, of which the most common is the gram negative diarrhoea causing bacteria, Campylobacter jejuni. Autoimmunity which has been a mainstay in the pathogenesis of GBS involves in it various mechanisms depending on the sub-types of GBS. [1]

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyneuropathy (AIDP), an autoimmune disease affecting the peripheral nervous system that is usually triggered by an acute infectious process. It is frequently severe and usually presents as an ascending paralysis marked by weakness in the legs that spreads to the upper limbs and the face along with complete loss of deep tendon reflexes. The exact cause of GBS is unknown, but it is sometimes triggered by a respiratory infection or the stomach flu. This potentially deadly disorder is relatively rare, occurring worldwide in only one or two people per 100,000, with slightly more males than females affected. All age groups can be affected; the incidence rises with age, and there is a minor peak among young adults. There is no cure for the disorder, but several treatments can ease symptoms and reduce the duration of the illness. Most people recover completely from even the most severe cases of GBS. [2]

2.1] PATHOPHYSIOLOGY

GBS is a post infectious disorder. Two-thirds of patients report symptoms of a respiratory or gastrointestinal tract infection before the onset of GBS. In about half of patients with GBS, a specific type of preceding infection can be identified and *C. jejuni* is responsible for at least one-third of these infections. [3-6]

Estimates show that infections contribute to the development of GBS as previous investigations have shown that in 70% of cases with GBS, patients had a history of infections. [7, 8]

Guillain-Barré syndrome (GBS) is an important cause of acute neuromuscular paralysis. Molecular mimicry and a cross-reactive immune response play a crucial part in its pathogenesis, at least in those cases with a preceding Campylobacter jejuni infection and with antibodies to gangliosides. [9]

Recent neurophysiological and pathological studies have led to a reclassification of the diseases that underlie Guillain-Barré syndrome (GBS) into acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor and sensory axonal neuropathy (AMSAN) and acute motor axonal neuropathy (AMAN). The Fisher

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syndrome of ophthalmoplegia, ataxia and areflexia is the most striking of several related conditions. [10]

It is also likely to be a consequence of the immunogenetic background of the patient since few patients develop GBS after infection even with one of these strains. Attempts to match the subtypes of GBS to the fine specificity of anti-ganglioside antibodies and to functional effects in experimental models continue but have not yet fully explained the pathogenesis. T cells are also involved in the pathogenesis of most or perhaps all forms of GBS. T cell responses to any of three myelin proteins, P2, PO and PMP22, are sufficient to induce experimental autoimmune neuritis. [10]

Strong evidence supports an important role for antibodies to gangliosides in pathogenesis. In particular antibodies to ganglioside GM1 are present in 14-50% of patients with GBS, and are more common in cases with severe axonal degeneration associated with any subtype. Antibodies to ganglioside GQ1b are very closely associated with Fisher syndrome, its formes frustes and related syndromes. Ganglioside-like epitopes exist in the bacterial wall of C. jejuni. Infection by this and other organisms triggers an antibody response in patients with GBS but not in those with uncomplicated enteritis. [10]

Antiglycolipid antibodies are most pathogenic factors in the development of GBS, but not found in 40% of patients with GBS. One of the principal target regions in GBS is the node of Ranvier where functional molecules including glycolipids are assembled. Nodal dysfunction induced by the immune response in nodal axolemma, termed "nodopathy," can electrophysiologically show reversible conduction failure, axonal degeneration, or segmental demylination. [11]

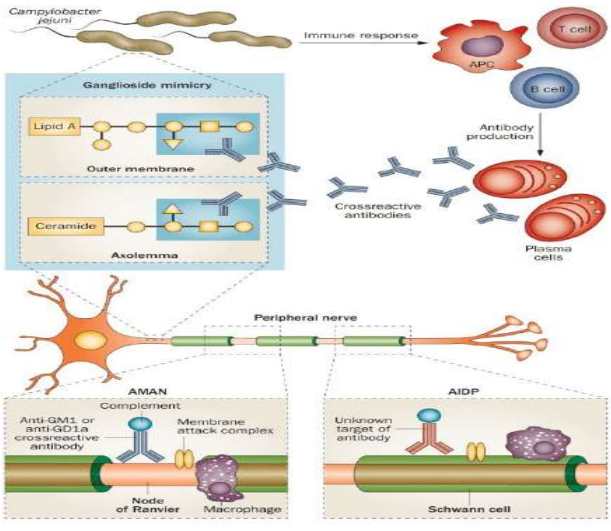

Figure 1: Acute motor axonal neuropathy caused by Campylobacter jejuni. [61]

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AMAN

In the acute motor axonal neuropathy (AMAN) form of GBS, the infecting organisms probably share homologous epitopes to a component of the peripheral nerves (molecular mimicry) and, therefore, the immune responses cross-react with the nerves causing axonal degeneration; the target molecules in AMAN are likely to be gangliosides GM1, GM1b, GD1a and GalNAc-GD1a expressed on the motor axolemma. [12]

AIDP

In the acute inflammatory demyelinating polyneuropathy (AIDP) form, immune system reactions against target epitopes in Schwann cells or myelin result in demyelination; however, the exact target molecules in the case of AIDP have not yet been identified. AIDP is by far the most common form of GBS in Europe and North America, whereas AMAN occurs more frequently in east Asia (China and Japan). [12,14-16]

AMSAN

Acute motor and sensory axonal neuropathy (AMSAN) is a variant of GBS in which both motor and sensory fibers are involved and which can be demonstrated on electrophysiological studies. It is more severe and associated with prolonged or even partial recovery. Clinical features are similar to AMAN but also involve sensory symptoms. The underlying pathological process is similar to that for AMAN (i.e. antibody mediated axonal damage). [13]

MFS

Miller Fisher syndrome (MFS) presents with ataxia, areflexia and ophthalmoplegia. 25% of patients may develop limb weakness. Electrophysiological studies show primarily sensory conduction failure. Anti_ganglioside antibodies to GQ1b are found in 90% of patients and are associated with ophthalmoplegia. There have been limited pathological studies in MFS but demyelination of nerve roots has been demonstrated. [13]

2.2] EPIDEMOLOGY

Based on well-controlled population-based studies, the incidence of GBS in Europe is 1.2–1.9 cases per 100,000, whereas worldwide, the incidence is 0.6–4 cases per 100,000. Atypical presentations, such as Fisher syndrome, are much less frequent, with an incidence of 0.1 per 100,000. Men are 1.5 times more likely to be affected than women, and the incidence increases with age from 1 per 100,000 in those aged below 30 years to about 4 cases per 100,000 in those older than 75 years. [17]

In China, the incidence in adults is 0.66 cases per 100,000. About two thirds of GBS cases have an antecedent infection within six weeks prior to symptom onset, generally an

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upper respiratory tract infection or gastroenteritis. Although the pathological organism is not often identified, the usual infectious agents associated with subsequent GBS include Epstein-Barr virus, Mycoplasma pneumoniae, Campylobacter jejuni and cytomegalovirus. In China, summer epidemics of the AMAN form of GBS were found to be secondary to infection with Campylobacter jejuni. In addition to antecedent infections, GBS develops after vaccination. Concerns about vaccine-induced GBS were first raised following the 1976–77 influenza vaccinating season, when a statistically significant increased risk of GBS was reported within 6–8 weeks of receiving the ‘‘swine flu” vaccine. Subsequently, studies that investigated the relationship between GBS and influenza immunization reported low relative risks that were not statistically significant. A combined analysis of the 1992–93 and 1993–94 vaccine campaigns in the USA reported a marginally increased risk of GBS (1 extra case of GBS for every 1 million vaccines) following influenza vaccination during the 6 weeks following immunization, a result recently confirmed in a Canadian study. Further, GBS has been reported after immunization with the hepatitis vaccine and the meningococcal conjugate vaccine (MCV4). [18-25].

2.3] ETIOLOGY

Guillain Barre Syndrome is often proceded by an infection. This could be bacterial and viral infection. Guillain Barre Syndrome may also be triggered by vaccine administration or surgery.

In the context of zika virus infection, unexpected increase in cases of Guillain Barre Syndrome has been described in affected countries. The most likely explanation of available evidence from outbreaks of zika virus infection and Guillain Barre Syndrome is that zika virus infection is a trigger of Guillain Barre Syndrome.

2.4] TREATMENT & DIAGNOSIS

Guillain-Barré syndrome (GBS) is an acute polyneuropathy with a variable degree of weakness that reaches its maximal severity within 4 weeks. The disease is mostly preceded by an infection and generally runs a monophasic course. Both intravenous immunoglobulin (IVIG) and plasma exchange (PE) are effective in GBS. Rather surprisingly, steroids alone are ineffective. Mainly for practical reasons, IVIG usually is the preferred treatment. [38]

The prognosis of GBS is generally favourable, but it is a serious disease with a mortality of approximately 10% and approximately 20% of patients are left with severe disability. Treatment of GBS is subdivided into: (i) the management of severely paralysed patients with intensive care and ventilator support; and (ii) specific immunomodulating treatments that shorten the progressive course of GBS, presumably by limiting nerve damage.

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High-dose intravenous immunoglobulin (IVIG) therapy and plasma exchange aid more rapid resolution of the disease. [9]

The type of preceding infection and patient-related host factors seem to determine the form and severity of the disease. Intravenous immunoglobulin (IVIG) and plasma exchange are effective treatments in GBS; mainly for practical reasons, IVIG is the preferred treatment. Whether mildly affected patients or patients with Miller Fisher syndrome also benefit from IVIG is unclear. Despite medical treatment, GBS often remains a severe disease; 3-10% of patients die and 20% are still unable to walk after 6 months. In addition, many patients have pain and fatigue that can persist for months or years. [12]

Many patients have pain, fatigue or other residual complaints that may persist for months or years. Pain can also be very confusing in making the diagnosis, especially when it precedes the onset of weakness. Advances in prognostic modelling resulted in the development of a simple prognostic scale that predicts the chance for artificial ventilation, already at admission; and in an outcome scale that can be used to determine the chance to be able to walk unaided after 1, 3 or 6 months. [38]

2.5] SUPPORTIVE THERAPY

- **NUTRITION THERAPY**

➢ Diet is important consideration when dealing with autoimmune disorders such as Gillian-Barre Syndrome.

➢ Consuming more fermented foods, and leafy vegetables can help boost your immune system.

➢ High energy and High protein diet have been recommended so as to reduce muscle wasting assist respiratory weaning.

- **EXERCISE**

➢ Exercise are essential for people with GBS to regain strength, coordination and functional independance.

➢ Aerobic exercise such as walking atleast 20 mins three times per week may help to improve aerobic capacity.

- **PHYSIOTHERAPY**

➢ The physical therapist will help to improve your ability to walk using techniques such as strenghtning exercise , walking training and balance activities

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3] REVIEW OF LITERATURE

Guillain-Barré syndrome (GBS) and its subtypes are correlated with distinct anti-ganglioside antibodies. Hence, we aimed to evaluate the frequency of anti-ganglioside antibodies, nerve conduction profile and its correlation with clinical profile and functional outcome in patients with GBS. Evidence shows that anti-ganglioside antibodies play an important role in the immunopathogenesis of GBS. [39] Most common antibodies that are identified are against GM1, GD1a, GD1b, GT1a, and GQ1b gangliosides. One of the proposed mechanisms is molecular mimicry in which antecedent infection produces specific antibodies, which in turn cross-reacts with peripheral nerve components because of the sharing of cross-reactive epitopes. [40] These antibodies can be directed against myelin or the axon. Antecedent infection with *Campylobacter jejuni* produces high titers of antibodies to GM1, GD1a, GalNAc-GD1a, and GD1b, which are strongly associated with the AMAN variant. [41] Similar association with *Campylobacter jejuni* infection and antibodies to GQ1b ganglioside has been reported in Miller Fischer syndrome (MFS).[42] Antibodies are more associated with the AMAN variant of GBS than with acute inflammatory demyelinating polyneuropathy (AIDP).[43] Very few studies have addressed the outcome of GBS in relation to anti-ganglioside antibodies.[44,45] In the present study, we aimed to assess the frequency of anti-ganglioside antibodies and the correlation of these antibodies with clinical features, electrophysiological patterns, and outcome in patients with GBS.
GBS subtypes are also classified according to nerve conduction study (NCS) results. GBS electrodiagnostic criteria (referred to as ‘Hadden criteria’) distinguish primary demyelinating (acute inflammatory demyelinating polyradiculoneuropathy) from primary axonal [acute motor axonal neuropathy (AMAN) or acute motor sensory axonal neuropathy] subtypes, as well as inexcitable and equivocal forms. [46-48] Moreover, a modified set of electrodiagnostic criteria (referred to as ‘Rajabally criteria’) has recently been proposed [49], enabling improved early electrodiagnosis based on a single NCS.

Thus, the aim of the present study is to document the clinical and electrophysiological features of GBS and its variants, and also to assess the suitability of different diagnostic criteria, in a cohort of GBS patients.

It has been proposed that anti-ganglioside antibodies contribute to the immunopathogenesis of GBS and certain other inflammatory neuropathies. Arguments against the importance of these antibodies in GBS have been raised because anti-ganglioside antibodies such as IgM anti-GM1 are also observed in apparently healthy individuals and in patients with multifocal motor neuropathy with conduction block other than GBS. [50, 51] The mechanism by which anti-ganglioside antibodies production is triggered is not very well known. However, it has been proposed that

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production of anti-ganglioside antibodies may be triggered by viruses or bacteria through a molecular mimicry mechanism or epitope sharing. Anti-ganglioside antibodies bind to the nodal axolemma or the paranodal myelin, thus leading to axonal degeneration or demyelination. Research has shown that the clinical features of GBS and its subtypes are closely associated with some specific anti-ganglioside antibodies. Four gangliosides – GM1, GD1a, GT1a, and GQ1b – described in GBS differ with regard to the number and position of their sialic acids, in which M, D, T, and Q represent mono-, di-, tri-, and quadri-sialosyl groups, respectively. IgG anti-ganglioside antibodies to GM1 and GD1a are more commonly associated with AMAN, AMSAN, and acute motor conduction block neuropathy, but not with AIDP. [52] Motor and sensory nerves express similar quantities of GM1 and GD1a; however, their expression within various tissues may differ. It has been suggested that IgG anti-GD1a antibodies bind to the motor nerve terminals where the blood nerve barrier is deficient and cause pure motor neuropathy. [53] This could explain the preferential motor axon injury seen in AMAN.

Guillain-Barré syndrome is a rare autoimmune disease of the peripheral nervous system with considerable mortality and morbidity. AIDP is the most common variant of GBS found in the study. However, there is high prevalence of axonal variant (40%) as compared to Western (6%-7%) population. AMAN had a significantly worse prognosis as compared to AIDP. Most patients (85%) had good outcome. [54]

The recent outbreak of the coronavirus disease 2019 (COVID 19) has caused an unprecedented worldwide pandemic characterized by an overwhelming health burden and serious socioeconomic consequences. This disease is typified by respiratory symptoms, ranging from mild flu-like symptoms to a severe highly lethal pneumonia. [55, 56] The current coronavirus pandemic is caused by the newly identified severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which is a highly pathogenic coronavirus that is associated with rapid infectivity rates. The structure of SARS-CoV-2 and its mechanism of infection are similar to those associated with other coronaviruses, including the severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV). [55, 57] In this regard, SARS-CoV-2 has a 75–80% identical genome sequence with MERS-CoV and SARS-CoV [58]

Evidence from the different study suggests that SARS-COV-2 is a potential trigger of GBS, which not only manifests in patients with severe but also those with mild or absent prior COVID-19 symptoms. Although GBS is typically regarded as a post infectious neurological disorder, a large number of COVID-19 patients present with an atypical para-infectious profile, and the majority of cases, including those with a post infectious profile, have short time latency between the onset of COVID-19 and GBS symptoms. This short time interval increases the likelihood for symptomatic overlap between para-infectious manifestations and a COVID-19-GBS, which not only complicates treatment but could also result in a poor prognosis characterized by severe autonomic

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dysfunction in the form of respiratory failure, heart failure, gastroplegia, multiple organ failure, and other signs of severe neuromuscular weakness or paralysis. These severe symptoms increase the odds for admission into the intensive care unit, need for SN Compr. Clin. Med. (2020) 2:2702–2714 2711 mechanical ventilation, extended recovery period, higher mortality rate, and permanent disability. Thus, given the high infectibility rates of the novel SARS-COV-2, COVID-19- associated GBS should be a major public health concern. GBS symptoms can progress very rapidly, and in our study, the two fatalities occurred within 12 to 24 h mainly caused respiratory failure and multi-organ failure. Paying particular attention to potential signs of GBS among COVID-19 patients and intervening at an early stage may improve treatment outcomes and minimize on morbidity, disease burden, and fatalities. Findings suggest that inclusion of neurological assessment in the COVID-19 diagnostic process for timely identification and effective symptom management may yield better treatment outcomes. Although the exact relationship between the novel COVID-19 and comorbid GBS remains incompletely defined, systematic follow-up of the patients could aid in the quick identification of relapse cases, given that some patients continue to test positive for SARS-CoV-2 after a prolonged period of time. [59]

Still many health care professionals are not aware of this rare disease and give wrong medication therapy to patient and worsen patient condition.

4] AIM AND OBJECTIVE

Aim

To assess clinical profile data and treatment pattern of Gbs patients.

Objective

1) To assess clinical profile data of Guillain barre syndrome [GBS] patients

2) To study treatment pattern of patients diagnosed with Guillain Barre Syndrome [GBS].

5] PLAN OF WORK

1. Literature survey
2. Discussion with doctor
3. Concern of patient
4. Collection of data
5. Assessment of data

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6] METHODOLOGY

The current study, "A retrospective, observational study was conducted for the assessment of clinical profile data and treatment pattern of Gillian barre syndrome" was conducted at tertiary care hospitals in Pune district Maharashtra.

This study is observational study of patients with Guillain-Barre syndrome.

The data regarding clinical profile and treatment pattern was collected and analysed from the case records of patients diagnosed with GBS at tertiary care hospitals.

1. Study design: An observational study.

2. Data collection: In the current study data was collected retrospectively.

3. Sample size: According to the availability of data (n=25) at different levels at hospital.

4. Selection of participants: The patient suffering from GBS

5. Ethical Issues:

- Prior permission and inform written consents was taken from each respondent.
- Privacy and Confidentiality of each respondent was maintained.

7] RESULT AND DISCUSSION

7.1] TREATMENT PATTERN:

According to our data, we observe that treatment like IVIG, Plasmapheresis, Nutrition therapy, Analgesic, Antibiotics etc. are the common medication prescribed by the physicians as per patient condition.

Following are the common treatment pattern for GBS Patients.

1) IVIG
2) Plasmapheresis
3) Proton pump inhibitors
4) Laxatives
5) Antibiotics
6) Vitamin supplements
7) Analgesics

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IVIG: The most commonly used treatment for Guillain-Barré syndrome is intravenous immunoglobulin (IVIG). IVIG is a treatment made from donated blood that contains healthy antibodies.

Plasmapheresis: A plasma exchange, also called plasmapheresis, is sometimes used instead of IVIG. This involves being attached to a machine that removes blood from a vein and filters out the harmful antibodies that are attacking your nerves before returning the blood to your body.

Proton pump inhibitors: Proton pump inhibitors reduce the amount of acid made by your stomach. They are commonly used to treat acid reflux and ulcers of the stomach and part of the gut called the duodenum.

Laxatives: Constipation is a common condition among patients with Guillain-Barré Syndrome. It is believed, that the constipation is caused by autonomic neuropathy. Laxatives are a type of medicine that can help you empty your bowels if you're having trouble going to the toilet. They're widely used to treat constipation.

Antibiotics: Penicillin is the most common and most effective antibiotic given for GBS. Although Vancomycin may be adequate for treatment of GBS meningitis in patients who are allergic to penicillin, skin testing and desensitization for penicillin therapy might be considered.

Vitamin supplements: Vitamins B1, B6, and B12 is the supportive therapy in cases of GBS that can help minimize or prevent the progression of the disease.

Analgesics: The most commonly prescribed analgesics are non-steroidal anti-inflammatory drugs (NSAIDs) and opioids. Anticonvulsants and antidepressants are also very commonly used by specialists.

Table 1: Table for Drugs Used for GBS Patient Treatment.

TREATMENT	BRAND NAME
Proton pump inhibitors	Pantop
Antibiotics	Monocef-O, Tab Taxim O
Laxatives	Syrup Duphalac, Syrup Looz
Anti-emetics	Tab. Emeset
Vitamin supplements	Inj optineuron, Cap Rejunex CD3
ANTI- pyretic	Inj Kabimol
Anti-epileptic	Inj Levipil
Anti-fungal	Inj Cospofungin
Anticoagulant	Inj Clexane
Nutrition supplements	Inj Otaski lamp
Diuretics	Inj Lasix
Anti-inflammatory	Inj Effcorlin
Anti-histamines	Inj Avil 1 amp stat

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Anti-diabetic	Tab Glycomet
Corticosteroids	Inj Hydrocort
Antihypertensive	Tab Stamlo
Anti convulsants	Tab Pregalin

7.2] CLINICAL PROFILE

The following clinical profile data was observed on the evidence of collected data on the basis of the symptoms, distribution, recovery rate etc.

7.2.1] GBS Variants

Guillain-Barré syndrome (GBS) is an important cause of acute neuromuscular paralysis. Molecular mimicry and a cross-reactive immune response play a crucial part in its pathogenesis, at least in those cases with a preceding Campylobacter jejuni infection and with antibodies to gangliosides.

Guillain-Barré syndrome (GBS) reclassified into acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor and sensory axonal neuropathy (AMSAN) and acute motor axonal neuropathy (AMAN) and Miller Fisher syndrome (MFS).

According to our data, we observed that there are 60% of AIDP, 20% of AMAN, and 20% of MFS patient were there. AIDP is the most common sub-type of GBS was observed in patients.

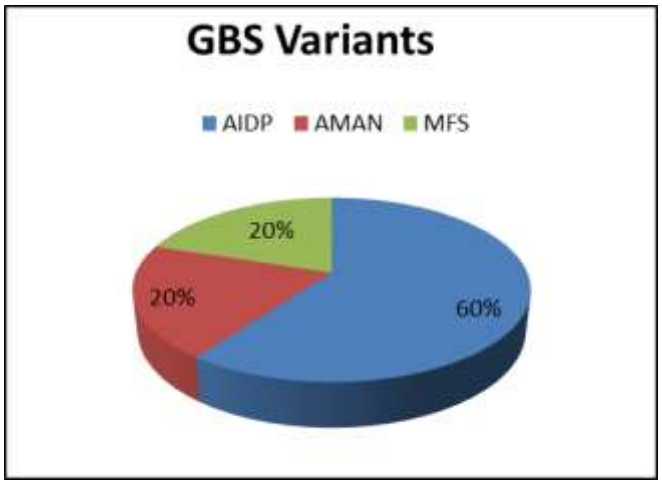

Figure 2: Pie chart of GBS Variants

7.2.2] Recovery Rate

Many patients have pain, fatigue or other residual complaints that may persist for months or years. Pain can also be very confusing in making the diagnosis, especially when it precedes the onset of weakness. Advances in prognostic modelling resulted in the

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development of a simple prognostic scale that predicts the chance for artificial ventilation, already at admission; and in an outcome scale that can be used to determine the chance to be able to walk unaided after 1, 3 or 6 months. As per the recovery rate of the 18 months follow up of GBS patients. According to our data, about 61% patients of GBS has full recovery rate and 25% and 14 % with good recovery and partial recovery were observed.

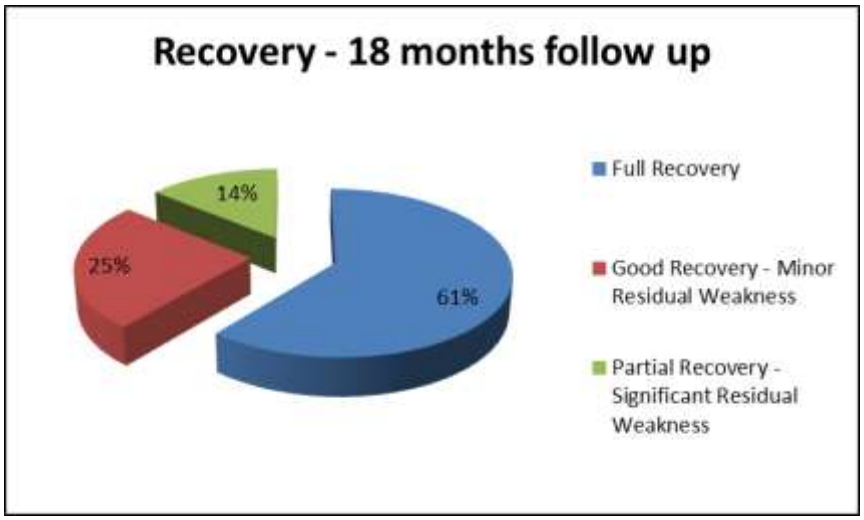

Figure 3: Pie chart of Recovery – 18 months Follow up

7.2.3] FUNCTIONAL MOTOR DEFECTS.

These are the grade scale for assessing the functional motor defects by Hughes.
As per our data, grade scale 2 was mostly observed as they were able to walk without support but not capable of manual working/running for a specific period of time.

0	Healthy
1	Minor symptoms or signs of neuropathy but capable of manual work/capable of running
2	Able to walk without support of a stick (5 m across an open space) but incapable of manual work/running
3	Able to walk with a stick, appliance or support (5 m across an open space)
4	Confined to bed or chair bound
5	Requiring assisted ventilation (for any part of the day or night)
6	Death

Figure 4: Hughes Grade Scale for Assessing Functional Motor Defects.

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7.2.4] Symptoms Observed During Treatment.

Guillain-Barre syndrome (GBS) is a rare disorder in which a person's own immune system damages their nerve cells, causing muscle weakness and sometimes paralysis. Weakness and tingling in your extremities are usually the first symptoms.

This symptoms usually last for a few weeks, GBS was described as a clinical condition characterised by rapid spread of ascending upper or lower limb weakness, defective tendon reflexes, weak sensory signs and possible autonomic dysfunctions.

According to data, these are symptoms mostly observed in the GBS Patients. Fever, weakness in limbs,and bifacial weakness symptoms having higher percentage in the observation.

Table 2: Table for Symptoms Observed During Treatment.

SYMPTOMS OBSERVED	PATIENTS %
Slurring of speech	60 %
Bifacial weakness	80%
Neck weakness	20%
Difficulty in closing eyes	70%
Weakness in limbs	80%
Fever	100%

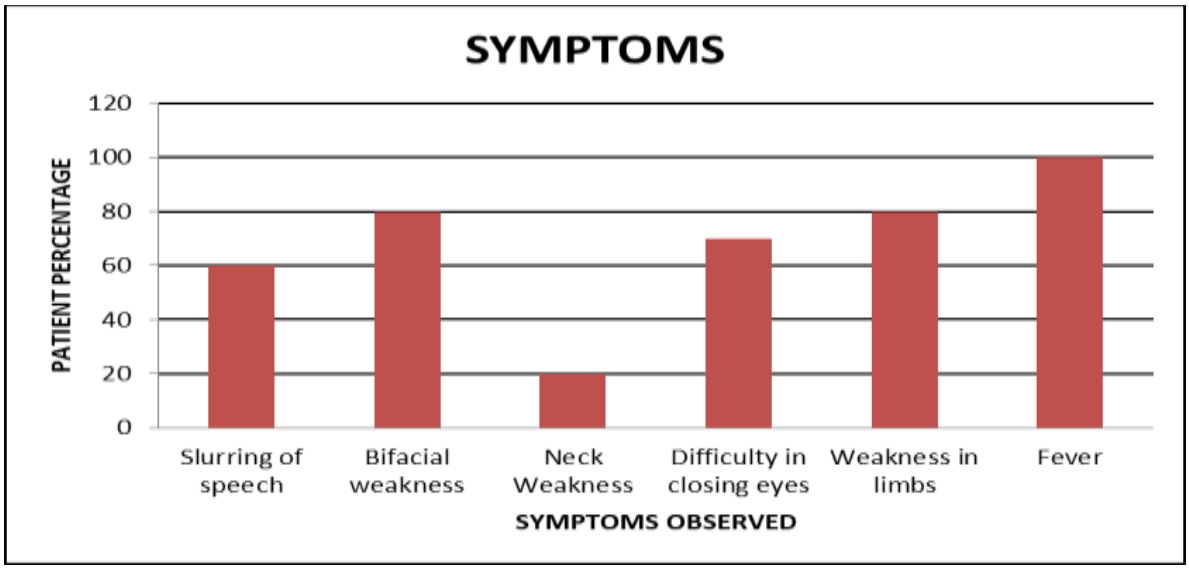

Figure 5: Bar chart of symptoms observed in GBS Patients.

7.2.5] Distribution of Respondents by Gender.

According to the data, 72% male and 28% women respondents were observed in GBS Disease. The GBS disease is mostly occurred in males as compare to females.

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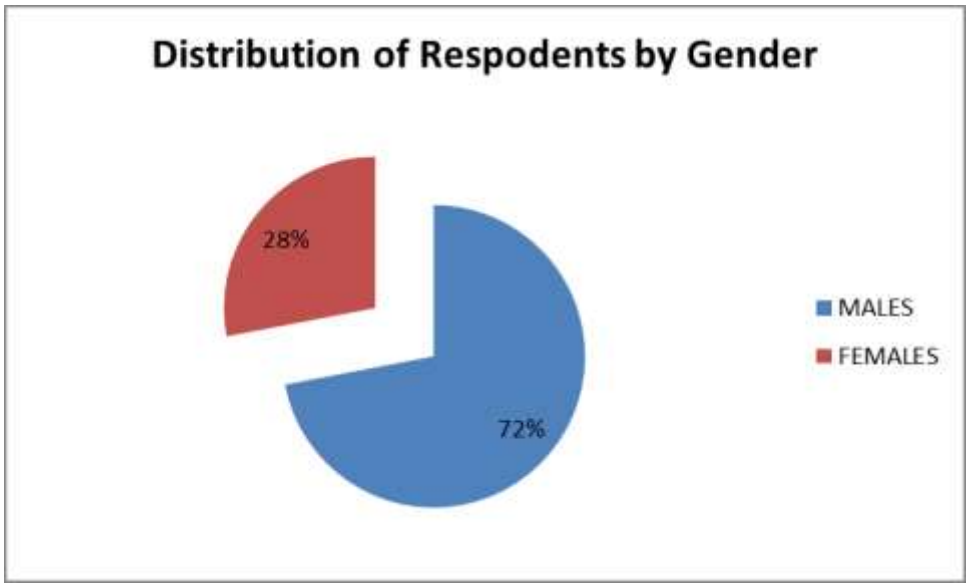

Figure 6: Pie chart of distribution of Respondents by gender.

8] CONCLUSION:

The proportion of different subtypes of Guillain-Barré syndrome (GBS) and their prognosis varied significantly among different regions. This study attempts to assess the clinical subtypes and outcome of GBS Patients.

The prognosis of different subtypes and outcome predictors were analyzed. The most common subtype of GBS was AIDP (60%), followed by AMAN (20%) and MFS (20%).

Both IVIg and plasmapheresis treatment showed significant improvement in our study setting IVIg was the preferred treatment option due to low side effect profile and ease of administration however cost of treatment is higher compared to plasmapheresis . Plasmapheresis was associated with complications such as BP, HR fluctuations.

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