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RETROSPECTIVE STUDY ON PRESCRIBING PATTERN OF GUILLAIN BARRE SYNDROME, A BINATIONAL STUDY, INDIA AND IRAN

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ABSTRACT

Introduction: Guillain-Barre syndrome (GBS) is an autoimmune and post-infectious immune disease. Guillain-Barre syndrome (GBS) is an acute-onset, monophasic, immune-mediatedpolyneuropathy that often follows an antecedent infection. Guillain-Barre syndrome is a peripheral neuropathy that causes acute neuromuscular failure. The syndrome includes several pathological subtypes. The diagnosis relies heavily on the clinical impression obtained from the history and examination, although cerebrospinal fluid analysis and electrodiagnostic testing usually provide evidence supportive of the diagnosis. Misdiagnosis is common and can be fatal because of the high frequency of respiratory failure, which contributes to the 10% mortality seen in prospective studies. There are currently two treatments commonly used to interrupt immune-related nerve damage. One is plasma exchange (PE, also called plasmapheresis); the other is high-dose immunoglobulin therapy (IV-Ig). Both treatments are equally effective if started within two weeks of onset of GBS symptoms, but immunoglobulin is easier to administer.

Objectives:

- To use suitable assessment for prognosis of disease by means of validated GB Syndrome disability scale.
- To report effectiveness and safety of Guillain Barre Syndrome treatment.
- To investigate the typical electrical diagnostic features in management of Guillain Barre Syndrome.

Methodology: A retrospective study was conducted on patients admitted to the department of general medicine, from the respective hospitals - Bangalore Baptist Hospital, Hebbal (Bangalore, India); Sagar Hospital (Bangalore, India); Shohadaye Ashayer Hospital (Iran);

Data was collected from medical records from each of the mentioned hospital. The collected data includes the relevant information of patient demographics, diagnosis, GBS occurrence its onset and duration, its progression, lab diagnostic values and other diagnostic features and treatment details. Based on the collected data, analysis was carried out and differentiated between Indian and Iranian demographics, on CSF protein levels, treatment chart and GBS Scale.

Result: The retrospective study on GB syndrome for the following parameters such as age, gender discrimination, major symptoms, differential diagnosis, level of protein in CSF, types of GB, morbid and non co-morbid condition and GB scale grade at the time of admission and at the discharge among both the Indian and Iranian population were analyzed using simple proportion ratio and filtration method and summation and presenting them in graphical and tabular form for easy understanding and discrimination. The analyzed data shows that female are more prone to GB compared to male and the age of onset of GB is between 21-30 years, due to GB scale the grade severity can be accessed.

Conclusion: Finally, I conclude that due to GB scale for severity, grades can help in accessing the early onset of disease and based on the study the onset of disease is between the age of 21-30years which helps in early treatment and decrease in progression of disease and since it was retrospective study on finding out the pattern of prescribing IGg IV therapy is best for all kinds of GBS and the differential diagnosis helps in easy detection of GB using ENMG. It is better to create awareness about the GB syndrome since female are more prone to GBS.

LIST OF ABBREVATIONS

AIDP -acute inflammatory demyelinating polyneuropathy.

AMAN - acute motor axonal neuropathy.

AMSAN - acute motor and sensory axonal neuropathy.

CMV- cytomegalovirus.

CSF- cereberospinal fluid.

CIDP- chronic inflammatory demyelinating polyneuropathy.

CT- computed tomography.

ECHO- electrocardiography.

ENMG- electroneuromyography.

GBS - Guillain-Barré syndrome.

IVIg –intra venous immunoglobulin.

LOS - lip oligosaccharide.

MFS - Miller-Fisher syndrome.

MCV - meningococcal conjugate vaccine.

MRI- magnetic resonance imaging.

NCV - nerve conduction velocity.

PE - plasma exchange.

SIADH – syndrome of inappropriate anti diuretic hormone.

INTRODUCTION

The **Nervous system** is the part of an animal that coordinates its actions by transmitting signals to and from different parts of its body. The nervous system detects environmental changes that impact the body, then works in tandem with the endocrine system to respond to such events. In vertebrates it consists of two main parts, the central nervous system (CNS) and the Peripheral Nervous System (PNS). The CNS consists of the Brain and Spinal cord. The PNS consists mainly of nerves, which are enclosed bundles of the long fibers or axons, that connect the CNS to every other part of the body. Nerves that transmit signals from the brain are called motor or efferent nerves, while those nerves that transmit information from the body to the CNS are called sensory or afferent.[1] Spinal nerves serve both functions and are called mixed nerves. The PNS is divided into three separate, the somatic autonomic, and enteric nervous systems. Somatic nerves mediate voluntary movement. The autonomic nervous system is further subdivided into the sympathetic and the parasympathetic nervous systems. The sympathetic nervous system is activated in cases of emergencies to mobilize energy, while the parasympathetic nervous system is activated when organisms are in a relaxed state.[2]

Guillain-Barre Syndrome is a problem with your nervous system. Guillain–Barre syndrome (GBS) is a rapid-onset muscle weakness caused by the immune system damaging the peripheral nervous system. Guillain-Barré syndrome (GBS) is a rare neurological disorder in which the body's immune system mistakenly attacks part of its peripheral nervous system—the network of nerves located outside of the brain and spinal cord.[3] Guillain-Barre (gee-YAH-buh-RAY) syndrome is a rare disorder in which your body's immune system attacks your nerves. Weakness and tingling in your extremities are usually the first symptoms. The initial symptoms are typically changes in sensation or pain along with muscle weakness, beginning in the feet and hands.[4][5] This often spreads to the arms and upper body, with both

sides being involved. The symptoms develop over hours to a few weeks. During the acute phase, the disorder can be life-threatening, with about 15% developing weakness of the breathing muscles requiring mechanical ventilation. Some are affected by changes in the function of the autonomic nervous system, which can lead to dangerous abnormalities in heart-rate and blood pressure. It can cause muscle weakness, reflex loss, and numbness or tingling in parts of your body.[6] It can lead to paralysis, which is usually temporary. These sensations can quickly spread, eventually paralyzing your whole body. Most people with the condition must be hospitalized to receive treatment. The exact cause of Guillain-Barre syndrome is unknown. But it is often preceded by an infectious illness such as a respiratory infection or the stomach flu.[7]

There's no known cure for Guillain-Barre syndrome, but several treatments can ease symptoms and reduce the duration of the illness. Most people recover from Guillain-Barre syndrome, though some may experience lingering effects from it, such as weakness, numbness or fatigue. GBS can range from a very mild case with brief weakness to nearly devastating paralysis, leaving the person unable to breathe independently. Fortunately, most people eventually recover from even the most severe cases of GBS. After recovery, some people will continue to have some degree of weakness.[8]

Guillain-Barré syndrome can affect anyone. It can strike at any age (although it is more frequent in adults and older people) and both sexes are equally prone to the disorder. GBS is estimated to affect about one person in 100,000 each year.[9] The exact cause of GBS is not known. It is not contagious or inherited. The affected person's immune system begins to attack the body itself. It is thought that, at least in some cases, this immune attack is initiated to fight an infection and that some chemicals on infecting bacteria and viruses resemble those on nerve cells, which, in turn, also become targets of attack.[10] Since the body’s own immune system does the damage, GBS is called an autoimmune disease (“auto” meaning “self”). Normally the immune system uses antibodies (molecules produced in an immune response) and special white blood cells to protect us by attacking infecting microorganisms (bacteria and viruses). In Guillain-Barre syndrome, however, the immune system mistakenly attacks the healthy nerves.[11]

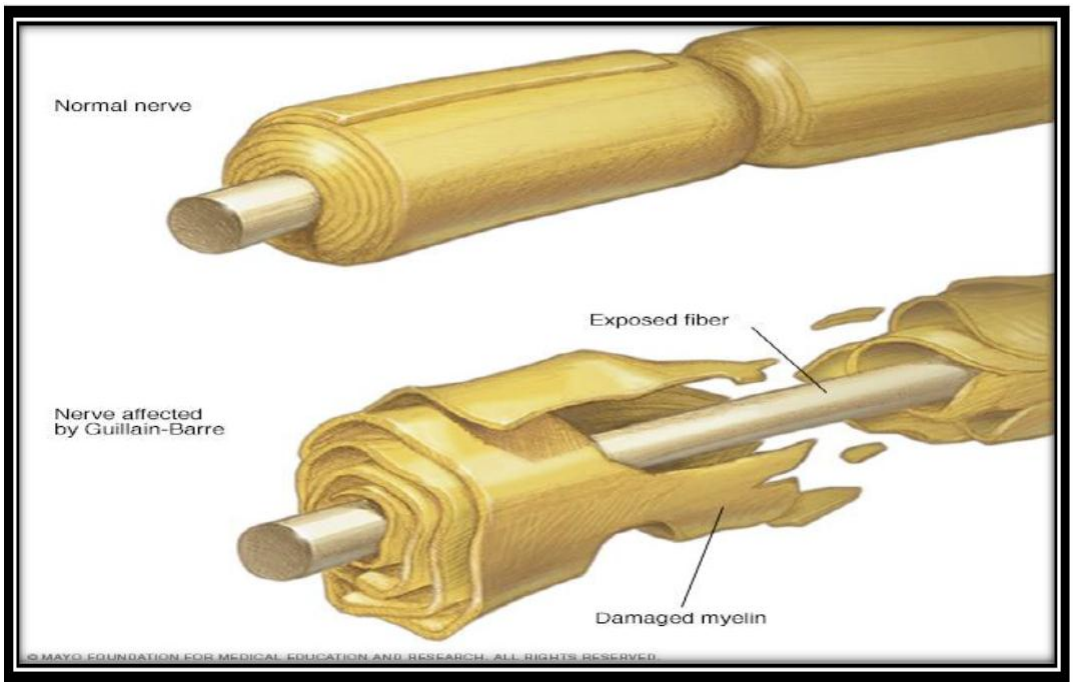


Figure I: Nerve and damaged myelin sheath. Guillain-Barre syndrome destroys the protective covering of the peripheral nerves (myelin sheath), preventing the nerves from transmitting signals to the brain.

The term GBS is often considered to be synonymous with acute inflammatory demyelinating polyradiculoneuropathy (AIDP), but with the increasing recognition over the past few decades of variants, the number of diseases that fall under the rubric GBS has grown to include axonal variants and more restricted variants such as Miller Fisher syndrome (MFS). Most cases usually start a few days or weeks following a respiratory or gastrointestinal viral infection. Occasionally surgery will trigger the syndrome. In rare cases vaccinations may increase the risk of GBS.[12] Recently, some countries worldwide reported an increased incidence of GBS following infection with the Zika virus.

Most people recover, even those with severe cases. In fact, 85% of people with GBS make a full recovery within 6 to 12 months. Once you get better, the chance of it returning is very small. The cause is unknown. The underlying mechanism involves an autoimmune disorder in which the body's immune system mistakenly attacks the peripheral nerves and damages their myelin insulation. Sometimes this immune dysfunction is triggered by an infection or, less commonly by surgery and rarely by vaccination.[13] The diagnosis is usually made based on the signs and symptoms, through the exclusion of alternative causes, and supported by tests such as nerve condition studies and examination of the cerebrospinal fluid. There are a

number of subtypes based on the areas of weakness, results of nerve conduction studies and the presence of certain antibodies. It is classified as an acute polyneuropathy.[14][15][16]

Recovery may take weeks to years. About a third has some permanent weakness. Globally, death occurs in about 7.5% of those affected. Guillain–Barré syndrome is rare, at one or two cases per 100,000 people every year. Both sexes and all parts of the world have similar rates of disease. The syndrome is named after the French neurologists Georges Guillain and Jean Alexander Barre, who described it with French physician Andre Strohl in 1916.[17]

EPIDEMIOLOGY

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. 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 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 pneumonia, 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). However, the incidence of GBS after immunization was not different from the background incidence of GBS, thereby precluding any firm conclusions about the significance of these findings.[18]

CAUSES

The exact cause of Guillain-Barre syndrome isn't known. The disorder usually appears days or weeks after a respiratory or digestive tract infection. Rarely, recent surgery or immunization can trigger Guillain-Barre syndrome. Recently, there have been a few cases reported following infection with the Zika virus. In Guillain-Barre syndrome, your immune system which usually attacks only invading organisms begins attacking the nerves.[9] In AIDP, the most common form of Guillain-Barre syndrome in the U.S., the nerves protective covering (myelin sheath) is damaged. The damage prevents nerves from transmitting signals to your brain, causing weakness, numbness or paralysis. Risk factors: Guillain-Barre syndrome can affect all age groups. But you're at slightly greater risk if:

Figure II: Age group affected by GB syndrome PC: college of medicine, University of Ibadan.

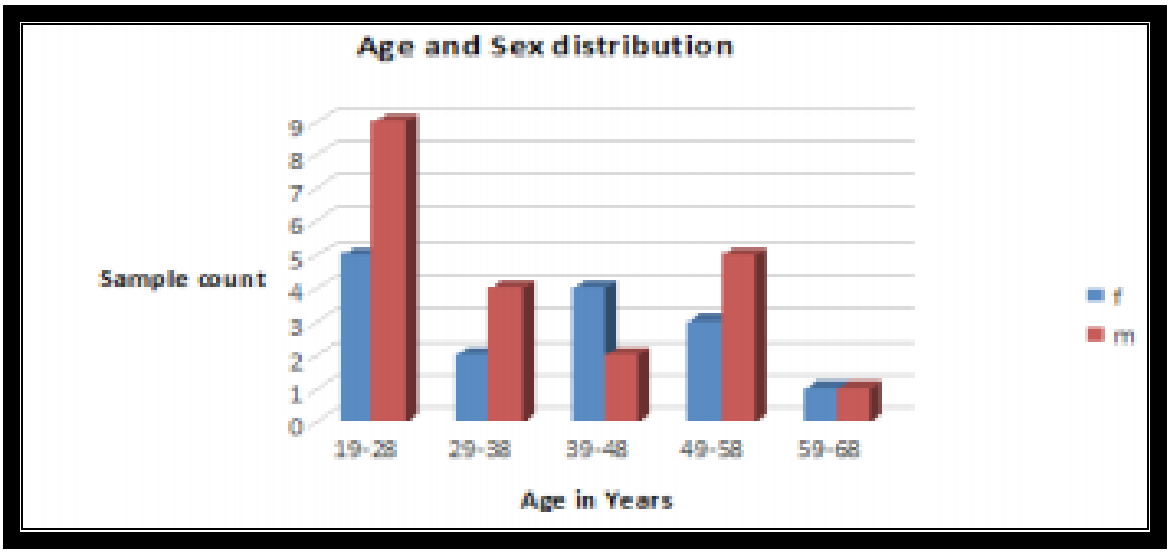


Figure III: Demographic Characteristics.

Guillain-Barre syndrome may be triggered by:

- Most commonly, infection with campylobacter, a type of bacteria often found in undercooked poultry
- Influenza virus
- Cytomegalovirus
- Epstein-Barr virus
- Zika virus
- Hepatitis A, B, C and E
- HIV, the virus that causes AIDS
- Mycoplasma pneumonia
- Surgery
- Hodgkin's lymphoma. Rarely, influenza vaccinations or childhood vaccinations

SYMPTOMS

Guillain-Barre syndrome often begins with tingling and weakness starting in your feet and legs and spreading to your upper body and arms. In about half of people with the disorder, symptoms begin in the arms or face. As Guillain-Barre syndrome progresses, muscle weakness can evolve into paralysis. Unexplained sensations often occur first, such as tingling in the feet or hands, or even pain (especially in children), often starting in the legs or back. Children will also show symptoms with difficulty walking and may refuse to walk.[11] These sensations tend to disappear before the major, longer-term symptoms appear. Weakness on both sides of the body is the major symptom that prompts most people to seek medical attention. The weakness may first appear as difficulty climbing stairs or with walking. Symptoms often affect the arms, breathing muscles, and even the face, reflecting more widespread nerve damage. Occasionally symptoms start in the upper body and move down to the legs and feet.[19]

Most people reach the greatest stage of weakness within the first two weeks after symptoms appear; by the third week 90 percent of affected individuals are at their weakest.

In addition to muscle weakness, symptoms may include:

- Difficulty with eye muscles and vision.
- Prickling, pins and needles sensations in your fingers, toes, ankles or wrists. Weakness in your legs that spreads to your upper body.

- Unsteady walking or inability to walk or climb stairs.
- Difficulty with eye or facial movements, including speaking, chewing or swallowing. Severe pain that may feel achy or cramp like and may be worse at night.
- Difficulty with bladder control or bowel function. Rapid heart rate.
- Low or high blood pressure. Difficulty breathing.
- Pain that can be severe, particularly at night. Choking on saliva.

These symptoms can increase in intensity over a period of hours, days, or weeks until certain muscles cannot be used at all and, when severe, the person is almost totally paralyzed. In these cases, the disorder is life-threatening potentially interfering with breathing and, at times, with blood pressure or heart rate. Guillain-Barre syndrome is a serious condition that requires immediate hospitalization because it can worsen rapidly.[20] The sooner appropriate treatment is started, the better the chance of a good outcome. People with Guillain-Barre syndrome usually experience their most significant weakness within two to four weeks after symptoms begin.

VARIABLE	RESPIRATORY(VA&VB) (n=10)	NON RESPIRATORY (n=26)	P VALUE
AGE			
<40	4(40%)	18(69.2%)	0.21
>40	6(60%)	8(30.7%)	
SEX			
MALE	5(50%)	16(61.5%)	0.80
FEMALE	5(50%)	10(38.46%)	
MRC GRADING			
0-10	6(60%)	0	0.000083
11-30	3(30%)	17(65.3%)	
31-40	1(10%)	9(34.6%)	
CSF Study Done (33)	N=10	N= 23	
PLEOCYTOSIS	8(80.00%)	18(69.00%)	0.91
ELECTRO DIAGNOS-TIC STUDY	9(90%)	25(96.1%)	0.90
AIDP	5(55.5%)	7(26.9%)	0.45
AMAN	2(22.2%)	7(26.9%)	
AMSAN	1(11.1%)	8(30.7%)	
MIXED	1(11.1%)	3(15.3%)	

Figure IV: Baseline characteristics among respiratory and non respiratory failure group of GBS.

Table I: Types of GB Syndrome.

Types	Symptoms
Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP)	Most common variant, 85% of cases. Primarily motor inflammatory demyelination ± secondary axonal damage ('bystander effect'). Maximum of 4 weeks of progression.
Acute Motor-Sensory Neuropathy (AMSAN) Axonal	Motor and sensory involvement with severe course respiratory and bulbar involvement. Primary axonal degeneration with poorer prognosis.
Acute Motor Axonal Neuropathy (AMAN) Miller-Fisher Variant	Motor only with early and severe respiratory involvement. Primary axonal degeneration. Often affects children, young adults. Up to 75% positive C. jejuni serology, often also anti-GM1, anti-GD1a positive Ophthalmoplegia, sensory ataxia, areflexia. 5% of all cases. 96% positive for anti-GQ1b antibodies.
Pharyngeal-Cervical-Brachial Variant	Often associated with IgG anti-GT1a.Presents with proximal descending weakness. Must distinguish from botulism and diphtheria.
Acute Pandysautonomia	Widespread sympathetic and parasympathetic failure.

Guillain-Barre syndrome is now known to occur in several forms. Guillain-Barre syndrome is one of several disorders involving weakness due to peripheral nerve damage caused by the person's immune system. While GBS comes on rapidly over days to weeks, and the person usually recovers, other disorders develop slowly and can linger or recur. Related peripheral nerve disorders with slow onset and persisting or recurrent symptoms include chronic inflammatory demyelinating polyneuropathy (CIDP) and multifocal motor neuropathy. CIDP features weakness that can recur, repeatedly, over the course of years. Multifocal motor neuropathy typically affects many different muscles in a small part of a limb or limbs. Usually the symptoms are more severe on one side of the body.[17]

The main types are:

Acute inflammatory demyelinating polyradiculoneuropathy (AIDP), the most common form in the U.S. The most common sign of AIDP is muscle weakness that starts in the lower part of your body and spreads upward.

Miller Fisher syndrome (MFS), in which paralysis starts in the eyes. MFS is also associated with unsteady gait. MFS occurs in about 5 percent of people with Guillain-Barre syndrome in the U.S. but is more common in Asia.

Acute motor axonal neuropathy (AMAN) and acute motor-sensory axonal neuropathy (AMSAN) are less common in the U.S. But AMAN and AMSAN are more frequent in China.

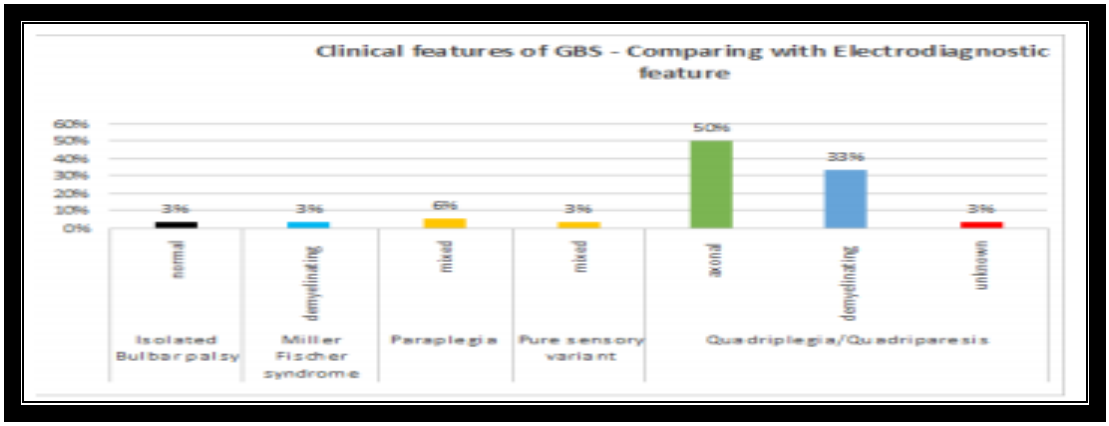


Figure V: Clinical Features

Complication

VARIABLE	DEMYELINATING (n=12)	AXONAL (n=18)(AMSAN+AMAN)	P VALUE
MEAN AGE	46	35	V
SEX RATIO(M:F)	0.7:1	2.6:1	0.09
Male	5	13	
Female	7	5	
ANTECEDENT INFECTION	4(33.3%)	4(22.2%)	0.11
RESPIRATORY ILLNESS	1(8.3%)	7(38.8%)	
GIT	0	2(11.1%)	
NONSPECIFIC	7(58.3%)	5(27.7%)	
NIL			
DURATION OF ILLNESS	7(58.3%)	7(38.8%)	0.29
<1 week	5(41.6%)	6(33.3%)	0.64
<2 weeks	0	3(16.6%)	0.13
<3 weeks	0	2(11.1%)	0.23
<4 weeks			0
Motor deficits	2(16.6%)	3(16.6%)	1
Mild MRC(31-40)	7(58.3%)	11(61.1%)	0.87
Moderate MRC(11-30)	3(25%)	4(22.2%)	0.86
Severe MRC(0-10)			
SENSORY DEFICITS	12(100.00%)	10(55.50%)	0.007
FACIAL PALSY	9(75.00%)	11(61.10%)	0.42
AUTONOMIC DYSFUNC-TION	6(50.00%)	6(33.30%)	0.36
RESPIRATORY FAILURE	5(41.60%)	3(16.60%)	0.12
HUGHES GRADING	0	0	0.08
Grade 1	0	1(5.5%)	
Grade 2	5(41.6%)	6(33.5%)	
Grade 3	1(8.3%)	8(44.4%)	
Grade 4	6(50%)	3(16.6%)	
5A(without ventilator)	0	0	
5B(with ventilator)	0	0	
Grade 6			
CSF ALBUMINO CYTO-LOGICAL DISSOCIATION	11(91.6%)	11(61.1%)	0.06
CSF ALBUMIN>100	4(36.3%)	3(27.2%)	
CSF ALBUMIN<100	7(63%)	8(72.7%)	0.64

Figure VI: Comparison between demyelinating and axonal variant of GBS.

Guillain-Barre syndrome affects your nerves. Because nerves control your movements and body functions, people with Guillain-Barre may experience:

Breathing difficulties: The weakness or paralysis can spread to the muscles that control your breathing, a potentially fatal complication. Up to 30 percent of people with Guillain-Barre syndrome need temporary help from a machine to breathe when they're hospitalized for treatment.

Residual numbness or other sensations: Most people with Guillain-Barre syndrome recover completely or have only minor, residual weakness, numbness or tingling.

Heart and blood pressure problems: Blood pressure fluctuations and irregular heart rhythms (cardiac arrhythmias) are common side effects of Guillain-Barre syndrome.

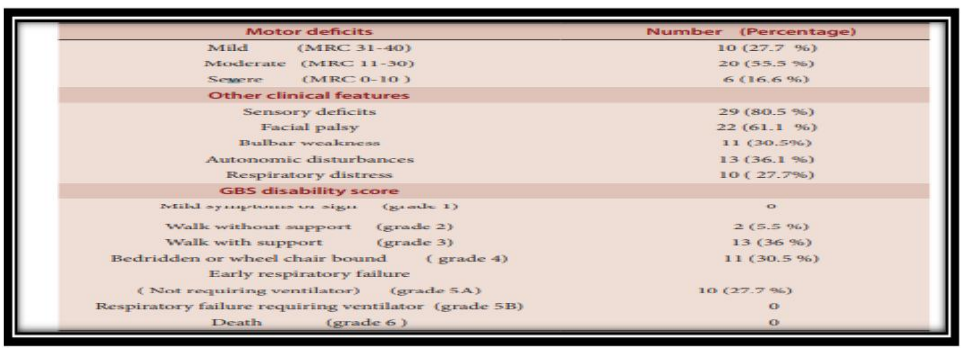
Pain: Up to half of people with Guillain-Barre syndrome experience severe nerve pain, which may be eased with medication.

Bowel and bladder function problems: Sluggish bowel function and urine retention may result from Guillain-Barre syndrome.

Blood clots: People who are immobile due to Guillain-Barre syndrome are at risk of developing blood clots. Until you're able to walk independently, taking blood thinners and wearing support stockings may be recommended.

Pressure sores: Being immobile also puts you at risk of developing bedsores (pressure sores). Frequent repositioning may help avoid this problem.

Relapse: Around 3 percent of people with Guillain-Barre syndrome experience a relapse.



Motor deficits	Number (Percentage)
Mild (MRC 31-40)	10 (27.7 %)
Moderate (MRC 11-30)	20 (55.5 %)
Severe (MRC 0-10)	6 (16.6 %)
Other clinical features	
Sensory deficits	29 (80.5 %)
Facial palsy	22 (61.1 %)
Bulbar weakness	11 (30.5%)
Autonomic disturbances	13 (36.1 %)
Respiratory distress	10 (27.7%)
GBS disability score	
Mild symptoms or sign (grade 1)	0
Walk without support (grade 2)	2 (5.5 %)
Walk with support (grade 3)	13 (36 %)
Bedridden or wheel chair bound (grade 4)	11 (30.5 %)
Early respiratory failure	
(Not requiring ventilator) (grade 5A)	10 (27.7 %)
Respiratory failure requiring ventilator (grade 5B)	0
Death (grade 6)	0

Figure VII: Neurological deficit and guillain-barre disability score.

Severe, early symptoms of Guillain-Barre syndrome significantly increase the risk of serious long-term complications. Rarely, death may occur from complications such as respiratory distress syndrome and heart attack.

Pathophysiology

Guillain-Barre syndrome is preceded by a viral or bacterial infection it is possible that the infecting agent has changed the chemical structure of some nerves. The immune system treats these nerves as foreign bodies and mistakenly attacks them. It is also possible that the virus makes the immune system itself less discriminating and no longer able to recognize its own nerves. Some parts of the immune system special white blood cells called lymphocytes and macrophages perceive myelin as foreign and attack it. Specialized white blood cells called T lymphocytes (from the thymus gland) cooperate with B lymphocytes (that originate in bone marrow) to produce antibodies against the person's own myelin and damage it.[12]

In some forms of GBS, antibodies made by the person to fight a Campylobacter jejuni bacterial infection attack axons in the motor nerves. This causes acute motor axonal neuropathy, which is a variant of GBS that includes acute paralysis and a loss of reflexes without sensory loss. Campylobacter infections can be caused by ingesting contaminated food or from other exposures.[16] The infected person's body then makes antibodies against Campylobacter. Some Campylobacter molecules resemble molecules in the person's nerve axons, so when the person's antibodies fight the Campylobacter bacteria they also attack the look-alike axons. This slows nerve conduction and causes paralysis. Scientists are investigating various GBS subtypes to find why the immune system reacts abnormally in this syndrome and other autoimmune diseases.

DIAGNOSIS

The initial signs and symptoms of GBS are varied and there are several disorders with similar symptoms. Guillain-Barre is difficult to diagnose at first. This is because the symptoms are very similar to those of other neurological disorders or conditions that affect the nervous system, such as botulism, meningitis, or heavy metal poisoning. Therefore, physicians find it difficult to diagnose GBS in its earliest stages. Physicians will note whether the symptoms appear on both sides of the body (the typical finding in Guillain-Barre syndrome) and the speed with which the symptoms appear (in other disorders, muscle weakness may progress over months rather than days or weeks). In GBS, deep tendon reflexes in the legs, such as knee jerks, are usually lost.[21] Reflexes may also be absent in the arms. Because the signals

traveling along the nerve are slow, a nerve conduction velocity test (NCV, which measures the nerve's ability to send a signal) can provide clues to aid the diagnosis. There is a change in the cerebrospinal fluid that bathes the spinal cord and brain in people with GBS. Researchers have found the fluid contains more protein than usual but very few immune cells (measured by white blood cells). Therefore, a physician may decide to perform a spinal tap or lumbar puncture to obtain a sample of spinal fluid to analyze. In this procedure, a needle is inserted into the person's lower back and a small amount of cerebrospinal fluid is withdrawn from the spinal cord. This procedure is usually safe, with rare complications. The following findings include:

- Recent onset, within days to at most four weeks of symmetric weakness, usually starting in the legs.
- Abnormal sensations such as pain, numbness, and tingling in the feet that accompany or even occur before weakness.
- Absent or diminished deep tendon reflexes in weak limbs.
- Elevated cerebrospinal fluid protein without elevated cell count. This may take up to 10 days from onset of symptoms to develop.
- Abnormal nerve conduction velocity findings, such as slow signal conduction.
- Sometimes, a recent viral infection or diarrhea.

The following tests are used to help confirm a diagnosis:

Spinal tap: A spinal tap involves taking a small amount of fluid from spine in lower back. This fluid is called cerebrospinal fluid. Cerebrospinal fluid is then tested to detect protein levels. People with Guillain Barre typically have higher-than-normal levels of protein in their cerebrospinal fluid. This test is also referred to as a lumbar puncture.

Electromyography: An electromyography is a nerve function test. It reads electrical activity from the muscles to help your doctor learn if your muscle weakness is caused by nerve damage or muscle damage. ERVE conduction tests: It is used to test how well nerves and muscles respond to small electrical pulses.[22]

TREATMENT

There is no known cure for Guillain-Barré syndrome. However, some therapies can lessen the severity of the illness and shorten recovery time. There are also several ways to treat the complications of the disease. Because of possible complications of muscle weakness,

problems that can affect any paralyzed person (such as pneumonia or bed sores) and the need for sophisticated medical equipment, individuals with Guillain-Barré syndrome are usually admitted and treated in a hospital's intensive care unit.[13] The symptoms can quickly get worse and can be fatal if they aren't treated. In severe cases, people with Guillain-Barré can develop full-body paralysis. Guillain-Barré can be life-threatening if paralysis affects the diaphragm or chest muscles, preventing proper breathing. Guillain-Barre is an autoimmune inflammatory process that's self-limiting, it will resolve on its own. The goal of treatment is to lessen the severity of the immune attack and support your body functions (for example, lung function) while nervous system recovers.

Acute care

There are currently two treatments commonly used to interrupt immune-related nerve damage. One is plasma exchange (PE, also called plasmapheresis); the other is high-dose immunoglobulin therapy (IVIg). Both treatments are equally effective if started within two weeks of onset of GBS symptoms, but immunoglobulin is easier to administer.

Plasmapheresis (plasma exchange)

The immune system produces proteins called antibodies that normally attack harmful foreign substances, such as bacteria and viruses. Guillain-Barré occurs when your immune system mistakenly makes antibodies that attack the healthy nerves of your nervous system. Plasmapheresis is intended to remove the antibodies attacking the nerves from your blood. During this procedure, blood is removed from your body by a machine. This machine removes the antibodies from your blood and then returns the blood to your body. In the process of plasma exchange, a plastic tube called a catheter is inserted into the person's veins, through which some blood is removed. The blood cells from the liquid part of the blood (plasma) are extracted and returned to the person.[23] This technique seems to reduce the severity and duration of the Guillain-Barre episode. Plasma contains antibodies and PE removes some plasma; PE may work by removing the bad antibodies that have been damaging the nerves.[24]

Intravenous Immunoglobulin

Immunoglobulins are proteins that the immune system naturally makes to attack infecting organisms. IVIg therapy involves intravenous injections of these immunoglobulins. The immunoglobulins are developed from a pool of thousands of normal donors.[25] When IVIg is given to people with GBS, the result can be a lessening of the immune attack on the nervous

system. The IVIg can also shorten recovery time. Investigators believe this treatment also lowers the levels or effectiveness of antibodies that attack the nerves by both "diluting" them with non-specific antibodies and providing antibodies that bind to the harmful antibodies and take them out of commission. High doses of immunoglobulin can also help to block the antibodies causing Guillain-Barre. Immunoglobulin contains normal, healthy antibodies from donors. Miller-Fisher syndrome is also treated with plasmapheresis and IVIg. Anti-inflammatory steroid hormones called corticosteroids have also been tried to reduce the severity of Guillain-Barré syndrome. However, controlled clinical trials have demonstrated that this treatment is not effective.[24,25]

Supportive care is very important to address the many complications of paralysis as the body recovers and damaged nerves begin to heal. Respiratory failure can occur in GBS, so close monitoring of a person's breathing should be instituted initially. Sometimes a mechanical ventilator is used to help support or control breathing. The autonomic nervous system (that regulates the functions of internal organs and some of the muscles in the body) can also be disturbed, causing changes in heart rate, blood pressure, toileting, or sweating. Therefore, the person should be put on a heart monitor or equipment that measures and tracks body function. Occasionally GBS-related nerve damage can lead to difficulty handling secretions in the mouth and throat. In addition to the person choking and/or drooling, secretions can fall into the airway and cause pneumonia.

Rehabilitative care

As individuals begin to improve, they are usually transferred from the acute care hospital to a rehabilitation setting. Here, they can regain strength, receive physical rehabilitation and other therapy to resume activities of daily living, and prepare to return to their pre-illness life. Complications in GBS can affect several parts of the body. Often, even before recovery begins, caregivers may use several methods to prevent or treat complications. Injections of blood thinners can help prevent dangerous blood clots from forming in leg veins. Inflatable cuffs may also be placed around the legs to provide intermittent compression. All or any of these methods helps prevent blood stagnation and sludging (the buildup of red blood cells in veins, which could lead to reduced blood flow) in the leg veins. Muscle strength may not return uniformly; some muscles that get stronger faster may tend to take over a function that weaker muscles normally perform called substitution. The therapist should select specific exercises to improve the strength of the weaker muscles so their original function can be

regained. Occupational and vocational therapy help individuals learn new ways to handle everyday functions that may be affected by the disease, as well as work demands and the need for assistive devices and other adaptive equipment and technology.[23],[24][25]

Table II: Guillain-Barre Syndrome Disability Scale.

0	Healthy
1	Minor symptoms or signs of neuropathy but capable of manual work/running
2	Able to walk without support (5meter)but incapable of manual work/running
3	Able to walk with support (5meter)
4	confined
5	Requiring assisted ventilation(any part of the day or night)
6	Death

GBS disability scale is used to identify the severity of the GBS syndrome. It is used to identify the mild, moderate or severe of GB syndrome. Based on the signs and symptoms, we classify GBS scale in to 6 grades, which helps in easy detection of severity. Our study aims in Identifying the grade of GBS, and treatment effectiveness and differentiation between Indian and Iranian demography and epidemiology.

OBJECTIVES

1. To use suitable assessment for prognosis of disease by means of validated GB Syndrome disability scale.
2. To report effectiveness and safety of Guillain Barre Syndrome treatment.
3. To investigate the typical electrical diagnostic features in management of Guillain Barre Syndrome.

REVIEW OF LITERATURE

R.A.C. Hughes, E.F.M. Wijdicks, R. Barohn, et.al conducted a study on Practice parameter: Immunotherapy for Guillain–Barré syndrome, their objective was to provide an evidence-based statement to guide physicians in the management of Guillain–Barre syndrome (GBS). They followed a methodology by doing a literature search and deriving of evidence-based statements concerning the use of immunotherapy, and analysed that treatment with plasma exchange (PE) or IV immunoglobulin (IVIg) hastens recovery from GBS and concluded that

combining the two treatments is not beneficial. Steroid treatment given alone is not beneficial and finally recommended the following:

1) PE is recommended for non-ambulant adult patients with GBS who seek treatment within 4 weeks of the onset of neuropathic symptoms. PE should also be considered for ambulant patients examined within 2 weeks of the onset of neuropathic symptoms;
2) IVIg is recommended for non-ambulant adult patients with GBS within 2 or possibly 4 weeks of the onset of neuropathic symptoms. The effects of PE and IVIg are equivalent;
3) Corticosteroids are not recommended for the management of GBS;
4) Sequential treatment with PE followed by IVIg, or immuno-absorption followed by IVIg is not recommended for patients with GBS; and
5) PE and IVIg are treatment options for children with severe GBS.[26]

Ambed Mishra, Sai Krishna G and Komal Krishna, did a study on Gullian Barre syndrome which describes Guillain-Barré syndrome (GBS), an unusual illness of the nervous system in which a person's own immune system damages the nerve cells, causing muscle weakness, and sometimes, paralysis. This condition has an expected incidence of 1-2 cases per 100,000 individuals and the mortality rate is high due to misdiagnosis. They explained that though the expected number of cases to that are reported vary from underdeveloped and developing countries, and are very less. The actual reported cases of occurrence of GBS is nearly negligible from these countries which may be attributed to the disease misdiagnosis and reported that misdiagnosis occurs due to lack of knowledge of GBS in many practitioners, where they misdiagnose GBS to be a case of paralysis which becomes a serious issue as GBS may cause death when untreated within few hours to days, this review highlights the major symptoms including causes of misdiagnosis due to similar presenting signs and symptoms to other diseases and treatment of this rarely reported diseaseand concluded that this review was to pass a safety message to all the healthcare practitioners regarding GBS and to aid them in recognizing the disease better and thereby decreasing the mortality and misdiagnosis.[28]

Payam Khomand, Sajjad Abdolmaleki, Ebrahim Ghaderi et.al; did a retrospective study on Guillain-Barre Syndrome: A Retrospective Study of Clinical and Epidemiological Features in Kurdistan, West of Iran, From 2005 To 2014, the aim of their study was to conduct a 10-year survey on epidemiological and clinical features of GBS in Tohid and Besat hospitals, Sanandaj, Iran, from 2005 to 2014,Since the study was a retrospective study, based on medical records, in which 98 hospitalized cases in Tohid and Besat hospitals (tertiary referral

hospitals), Sanandaj, Iran, between 2005 and 2014 were investigated. The epidemiological and clinical information was obtained from eligible cases. Data analysis was performed using SPSS version 16. Chi-square and *t* test were used for analyses. The significant level was considered at $P < 0.05$and reported that the mean age of cases with GBS was 22.16 years. Among final 69 patients who were studied, 36 cases (52.2%) were male and 33 cases (47.8%) were female. Most cases of disease occurred in the spring. Thirty-nine patients (56.52%) had risk factors like history of gastrointestinal infections, respiratory infections, and surgery 2-4 weeks before the disease onset. Four cases (10.25%) needed mechanical ventilation. The most common protocol of treatment was IVIg (n = 47, 68%). More than half of the patients (52.2%) achieved relative recovery. In 6 patients, (8.7%) relapse was occurred and finally concluded that study showed that there was a significant relationship between sensory- motor involvement, gender and age. Moreover, the relationship between gender and prognosis was indicated.[29]

Ted M. Burns did a study on Guillain-Barre syndrome (GBS) which is an acute-onset, monophasic, immune-mediated polyneuropathy that often follows an antecedent infection, and the diagnosis relies heavily on the clinical impression obtained from the history and examination, and concluded that cerebrospinal fluid analysis and electrodiagnostic testing usually provide evidence supportive of the diagnosis, and the clinician must also be familiar with mimics and variants to promptly and efficiently reach an accurate diagnosis, finally Intravenous immunoglobulin and plasma exchange are efficacious treatments and requires supportive care during hospitalization.[32]

Anand B. Pithadia, Nimisha Kakadia did a study on GBS an autoimmune and post-infectious immune disease. The syndrome includes several pathological subtypes, the most common of which is a multifocal demyelinating disorder of the peripheral nerves. In the present review, the main clinical aspects and the basic features of GBS are discussed along with approaches to diagnosis and treatment. Furthermore, the Pathophysiology of GBS is reviewed, with an emphasis on the production of symptoms and the course of the disease.[33]

Dana L. Newswanger, Charles R. Warren did a study on GBS Guillain-Barre syndrome (GBS) is a group of autoimmune syndromes consisting of demyelinating and acute axonal degenerating forms of the disease. Nerve conduction study helps differentiate the heterogeneous subtypes of GBS. Patients exhibit a progressive paralysis that reaches a plateau phase. In most patients, resolution is complete or near complete. Mortality from GBS

most often is associated with dysautonomia and mechanical ventilation finally, concluded that GBS usually is associated with an antecedent infection by one of several known pathogens and cross-reactivity between the pathogen and the nerve tissue sets up the autoimmune response, the treatment consists of supportive care, ventilatory management (in about one third of patients), and specific therapy with intravenous immunoglobulin or plasmapheresis, and suggested neurologist consultation.[40]

Payam Khommad, Sajjad Abdolmaleki Ebrahim Ghaderi, et al; carried out a retrospective study of clinical and epidemiological features in GB Syndrome The aim of their study was to conduct a 10-year survey on epidemiological and clinical features of GBS in Tohid and Besat hospitals, Sanandaj, Iran, from 2005 to 2014. It was a retrospective study, based on medical records, in which 98 hospitalized cases in Tohid and Besat hospitals (tertiary referral hospitals), Sanandaj, Iran, between 2005 and 2014 were investigated. The epidemiological and clinical information was obtained from eligible cases. Data analysis was performed using SPSS version Chi-square and t test were used for analyses. The significant level was considered at $P < 0.05$, the results obtained was the mean age of cases with GBS was 22.16 years. Among final 69 patients who were studied, 36 cases (52.2%) were male and 33 cases (47.8%) were female. Most cases of disease occurred in the spring. Thirty-nine patients (56.52%) had risk factors like history of gastrointestinal infections, respiratory infections, and surgery 2-4 weeks before the disease onset. Four cases (10.25%) needed mechanical ventilation. The most common protocol of treatment was IVIg (n = 47, 68%). More than half of the patients (52.2%) achieved relative recovery. In 6 patients, (8.7%) relapse was occurred, Finally their study showed that there was a significant relationship between sensory- motor involvement, gender and age. Moreover, the relationship between gender and prognosis was indicated.[54]

Ilknur Kozanoglu, Yerdelen Deniz, Nurhilal Buyukkurt,et al carried out a retrospective study on Patients with Guillain-Barré Syndrome treated with therapeutic plasma exchange and other treatment options. Therapeutic plasma exchange (TPE) has been shown to hasten recovery in Guillain-Barré syndrome (GBS). In this study, their objective was to show the outcome of disability grade in a retrospective analysis of data of clinical experience of TPE using the COBE Spectra Apheresis system and other treatment options in selected patients from a series of 56 patients with GBS at a single treatment centre in Turkey. Ten patients had the acute motor axonal neuropathy (AMAN) subtype; 46 had the acute inflammatory

demyelinating polyneuropathy (AIDP) subtype of GBS. Three hundred and eighteen TPE procedures were performed taking 2 to 3 hours: in 6.3 % of them a peripheral catheter was used; in 93.7 % of them a central catheter was used. Replacement fluids were fresh frozen plasma (FFP), lactated Ringer's solution or 3% hydroxyethyl starch (HES). Among the patients, 12 (21.4 %) who had severe disease course received additional treatment to TPE – this was intravenous immunoglobulin (IVIG) in 11 patients. One patient was treated with steroids after rheumatology consultation due to another autoimmune disease. After 2 weeks, the mean GBS disability scores had significantly decreased from 3.75±0.48 to 2.44±0.96 (p=0.0001) and mean Medical Research Council (MRC) muscle strength scores significantly increased from 2.07±0.89 to 3.54±0.88 (p=0.0001). No difference in efficacy was observed between AMAN and AIDP subtypes. Adverse events occurred in 20 procedures (6.3 %) of TPE and were mostly transient hypocalcaemia and allergic reactions that did not necessitate treatment discontinuation. Difficulty in venous access was observed in 3.14 % of procedures they finally, concluded that TPE using the COBE Spectra Apheresis system provides effective treatment of GBS with an acceptable safety profile using various replacement fluids and is an essential part of disease management. Although the benefit is controversial, other treatment options may be applied as an additional therapy in selected patients.[55]

Jean-Marc Léger, LuisQuerol, Mazen M Dimachkie carried out a study on Immunomodulatory role of therapeutic plasma exchange in peripheral nervous system and neuromuscular diseases, They reported that the therapeutic plasma exchange (TPE) removes pathogenic antibodies and immune complexes from the plasma. However, TPE may also impact a number of other immune-modulatory pathways that mediate cellular immunity. Data from clinical trials support the effectiveness of TPE in Guillain-Barré syndrome (GBS) and chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). However, to date, the use of TPE for the treatment of chronic myasthenia gravis (MG) is not supported by large clinical studies and there are discrepancies between guidelines and clinical practice. More clinical trials are needed to understand the role of TPE in MG, GBS, and CIDP, as well as other neuromuscular diseases in which it is used, or which may represent potential targets for TPE.[61]

A. Chiò, D. Cocito, M. Leone, M.T. Giordana, G. Mora, R. Mutani carried a prospective study on population-based incidence and outcome survey of GB syndrome with an objective of the incidence and long-term prognostic factors of Guillain-Barré syndrome (GBS) in a

population-based study. They followed a methodology of patients with GBS diagnosed according to National Institute of Neurological and Communicative Disorders and Stroke criteria in the 2-year period 1995 to 1996 in two Italian regions were prospectively followed up for 2 years after onset of GBS and gave a result of a total of 120 patients, corresponding to a crude annual incidence rate of 1.36/100,000 population (95% CI, 1.13 to 1.63). A total of 7 (5.8%) patients, all but one with axonal or mixed EMG pattern, died acutely within 30 days from the onset of the disease. Acute mortality was due to respiratory involvement and intensive care unit complications. In multivariate analysis, a worse 2-year outcome (Hughes score ≥2) was related to a higher Hughes grade at nadir, axonal or mixed EMG, age ≥50 years, and absence of respiratory infections preceding GBS. The persistence of disability 2 years after the acute phase was related to axonal involvement and a worse status at nadir and finally concluded that after adjustment to US population, the incidence rates for GBS from different countries showed no significant differences. Both acute mortality and long-term disability in GBS seem to be related to an axonal involvement and a Hughes grade ≥2 at nadir.[65]

Hemal Tande, Jigar Vanza, Nilima Pandya carried out a study on Guillain-Barre Syndrome (GBS) Review. It usually presents with ascending paralysis and is severe enough to warrant hospital admission for its management. The incidence of GBS is 1.1-1.8 cases in 100,000 per year and the incidences increases with age. GBS clinical spectrum is heterogeneous and encompasses Acute Inflammatory Demyelinating Polyneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN), Acute Motor and Sensory Axonal Neuropathy (AMSAN) and Miller Fisher Syndrome (MFS). The disease is typically characterized by a rapid onset of symmetrical limb weakness, which progresses over days to 4 weeks, and occurs in patients of all ages. Most patients also have sensory disturbances such as tingling or dull feelings. In developed countries GBS has become the most common cause of acute flaccid paralysis. Finally concluded, despite improved recognition and treatment, GBS continues to be a severe disease. Efficacious treatments include intravenous immunoglobulin and plasma exchange but supportive care during and following the hospitalization is also very much crucial.[66]

A Hiraga**,** M Mori**,** Ogawara et.al; carried out a study on Recovery patterns and long term prognosis for axonal Guillain–Barre syndrome with an objective to clarify the long term prognosis for patients with AMAN, followed a methodology of clinical recovery and outcome in consecutive GBS patients were reviewed, and found out a result that

electrodiagnostic criteria showed that 44 patients (45%) had AMAN, 33 (34%) had AIDP, and 20 (21%) were unclassified. Most of the severely affected patients had received plasmapheresis or immunoglobulin therapy. Slow recovery (inability to walk independently at six months after onset) was found in six of the AMAN patients (14%) and in two of the AIDP patients (6%). Of the six AMAN patients, four could walk independently one year after the onset, and the other two could walk independently at 28 and 57 months after onset. Of the two AIDP patients, one could walk at nine months after the onset while the other died of pneumonia seven months after onset Finally concluded, AMAN electrodiagnosis is not always a marker of poor recovery. Almost all the severe AMAN patients who had slow recoveries over the first six months could eventually walk independently, although some required several years.[67]

METHODOLOGY

Patients admitted with GB syndrome and with co morbid condition in the following hospital

1. Bangalore Baptist Hospital, Hebbal. (Bangalore, India)
2. Sagar Hospital Kumaraswamy Layout (Bangalore, India)
3. Sagar Hospital Jayanagar (Bangalore, India)
4. Shohadaye Ashayer Hospital (Iran); are taken in to the consideration including the records from MRD from the following respective hospital for my project. The duration of my study is 6 months from July 2018-December 2018. The Inclusion criteria for my study were patients admitted to the above mentioned hospitals and also the records from MRD of those hospitals from July to December. The exclusion criteria for my study were OPD patient are not included and emergencies condition (less than 24hr of hospitalization) are not considered. The prescription guidelines, Micromedex, Medscape and references books were used as tools to review the prescription and case charts. The data stored confidentially and will be subjected to further analysis using appropriate software. I have collected all the relevant information of the patient starting from the day of admission to the day of discharge- the data includes patient demographics, history of present illness, chief complaints, past history of patient illness, other co-morbid conditions, signs and symptoms, laboratory data which specifically correlates with GBS, the levels of proteins in CSF, and scan reports of Magnetic resonance Imaging (MRI), Computed Tomography (CT), Electrocardiogram (ECHO) and Electroneuromyography (ENMG).

The collected data was analyzed based on age discrimination, gender discrimination both Inter and Intra among India and Iran, and on major symptoms observed in GBS, levels of protein in CSF(Cerebrospinal Fluid), co morbid and non co morbid condition, types of GBS observed among admitted individual or MRD records, treatment pattern and GBS scale. The analysis was done by taking all the data into consideration, followed by applying the filtration method in MS excel sheet, filtering the specific data required for the specific discrimination for an age, demographics, treatment, type of GBS, population distribution and GBS scales (Huges scale) – a standard scale of GBS severity and carried out the percent calculation or the number of patient involved, then it is exported in to graphs and tables and correlated with the other data. Since my study was an retrospective, I have correlated the factors involved in GBS from the old relevant studies also, I in a simple method had done a distribution ratio for the following factors in GBS in order to give a brief description about GBS in the following respective hospital. My view in following this simple method is first to understand the normal overview of GBS in both the countries in a particular site.

RESULT AND DISCUSSION

Table-3 and Figure 8 suggest the details of age distribution of patients both in Iran and India. The highest percent of patients affected with GBS are at the age of 21-30 years. The secong most affected age group is 61-70 years.

Out of 86 patients 19(22%), 17(19.7%), 13(15.1%), 12(13.9%), 8(9.3%), 6(6.9%), 6(6.9%), 3(3.4%) and 2(2.3%) are belongs to age of 0-90 years respectively as shown in figure.

Table-3: Details of age distribution of patients.

Age	Number of Patients	Percentage (%)
0-10 Years	6	6.9
11-20 Years	2	2.3
21-30 Years	19	22.0
31-40 Years	8	9.3
41-50 Years	13	15.1
51-60 Years	12	13.9
61-70 Years	17	19.7
71-80 Years	6	6.9
81-90 Years	3	3.4

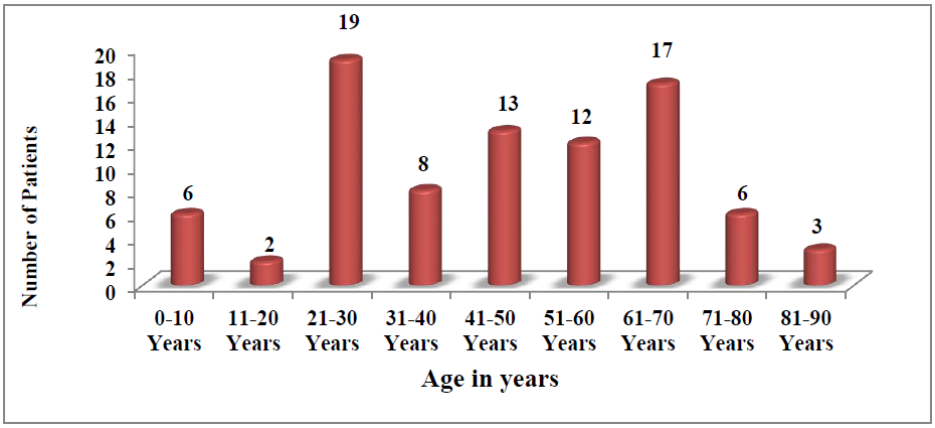


Figure-8: Graph showing details of Age distribution of patients.

Table 3 and figure 9 indicates gender distribution of patients both in Iran and India. The most affected gender are females compared to males. Out of 86 patients 49(56.9%) were females and 37(43.02%) were males.

Table-4: Details of gender distribution of patients.

Gender	Number of patients	Percentage (%)
Female	37	56.9
Male	49	43.02
Total	**86**	**100**

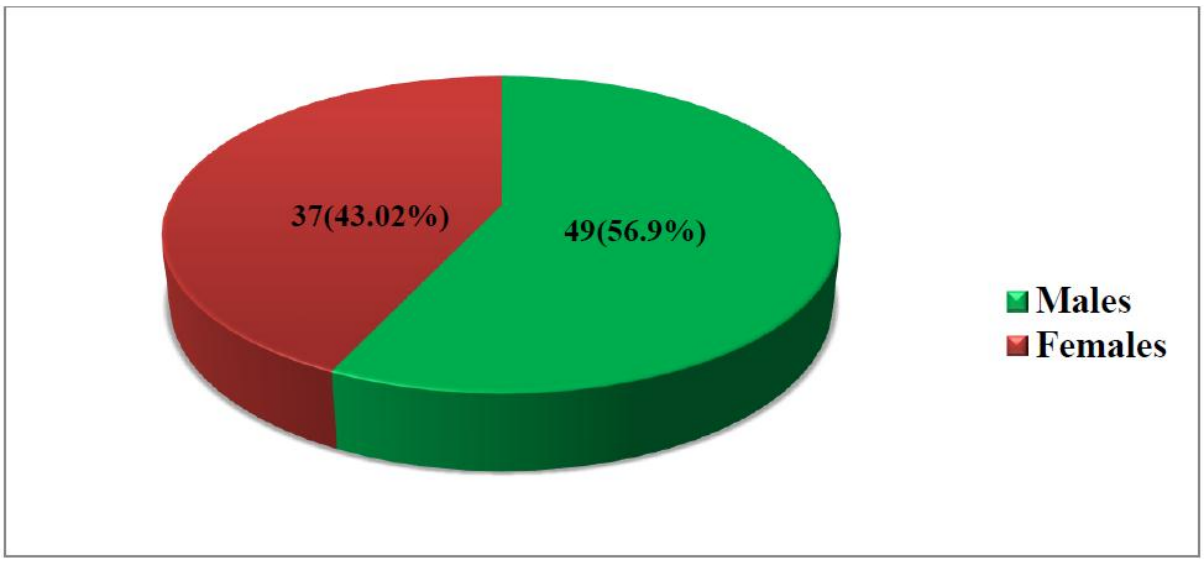


Figure-9: Graph showing details of gender distribution of patients.

Table 5 and figure 10 shows the distribution ratio of patient in both Iran and India. Total number of patients are 86 out of which 63(73.25%) belongs to India and 23(26.7%) belongs to Iran.

Table-5: Details of country distribution of patients.

Demographic data	Number of patients	Percentage (%)
India	63	73.25
Iran	23	26.7
Total	**86**	**100**

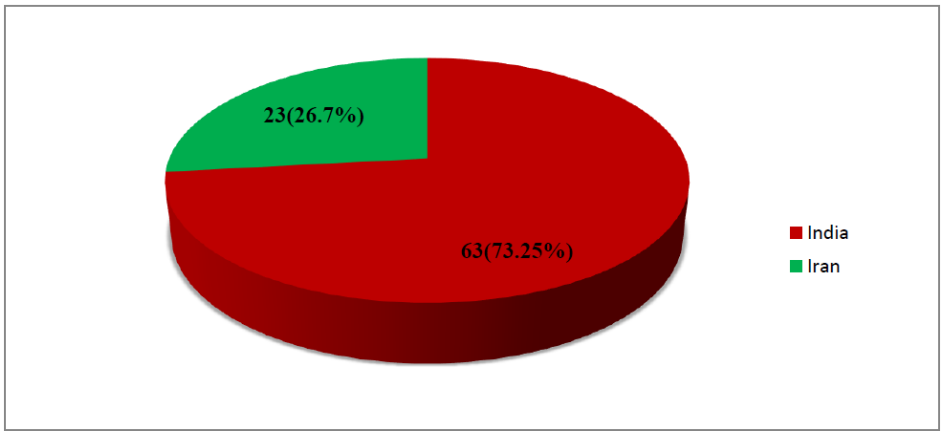


Figure-10: Graph showing details of country distribution of patients.

Table 6 and figure 11 shows the details of demographical data of patient enrolled in both countries .Out of 86 patients 23(26.7%) patients belongs to Iran out of which 24(60.8%) females and 9(39.1%) are males. Out of 63(73.25%) Indian patients, 14(60.8%) females and 9(39.1%) are males, and total of 37 females and 49 males.

Table-6: Details of demographical countries and patients enrolled.

Gender	Number of patients		Percentage (%)
	Females	Males	
Iran	14(60.8%)	9(39.1%)	26.7%
India	23(36.5)	40(63.5%)	73.25%
Total	**37**	**49**	**86(100%)**

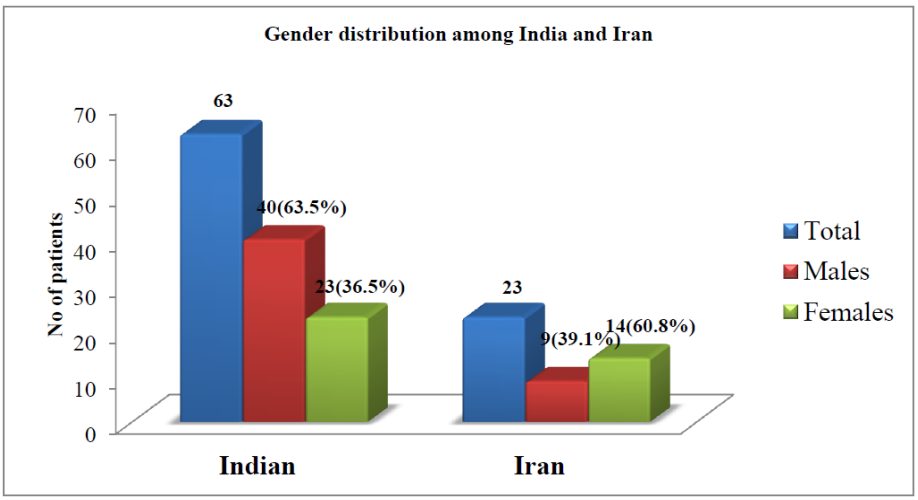


Figure 11: Graph showing details of gender distribution of patients.

Table 7 and figure 12 shows the details of major symptoms observed in GBS. Out of 86 patients, 72 had progressive weakness in both arms and legs, 41 had mild sensory deficits, 35 had fever, 24 had facial palsy, 23 with areflexia.

Table-7: Major symptoms observed in GBS patient.

Major symptoms	Total	India	Iran
PROGRESSIVE WEAKNESS IN BOTH ARMS AND LEGS	72	53	19
AREFLEXIA	23	11	12
Mild sensory deficits	41	32	9
Facial palsy	24	19	5
Fever	35	28	7
Total number of patient's	86	63	23

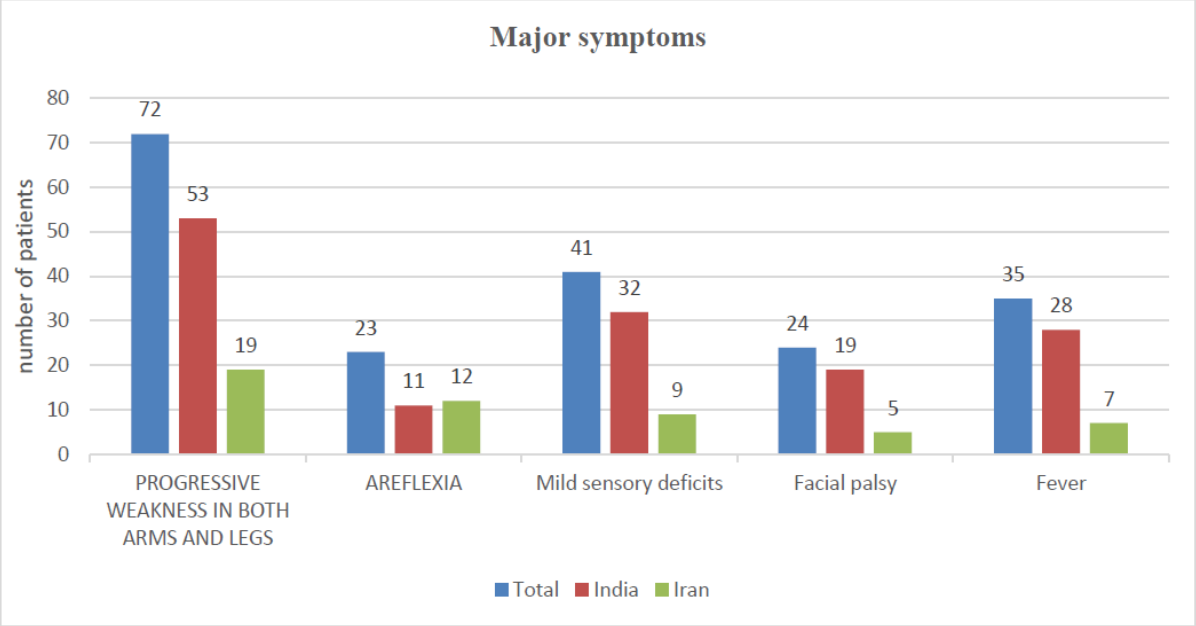


Figure 12: Graph shows the Major symptoms observed in GBS patient.

Table 8 and figure 13 indicates the co morbid and non co morbid conditions observed among 86 patients, Out of 86 patients, 54(62.79%) had co morbid condition and 32(37.25%) had non co morbid condition.

Table 8: Co-morbid conditions.

Condition	Total	India	Iran
Comorbid conditions	54(62.79%)	37(58.73 %)	17(73.91%)
Non-Comorbid conditions	32(37.2%)	26(41.26%)	6(26.08%)
Total Number of Patients	**86**	**63**	**23**

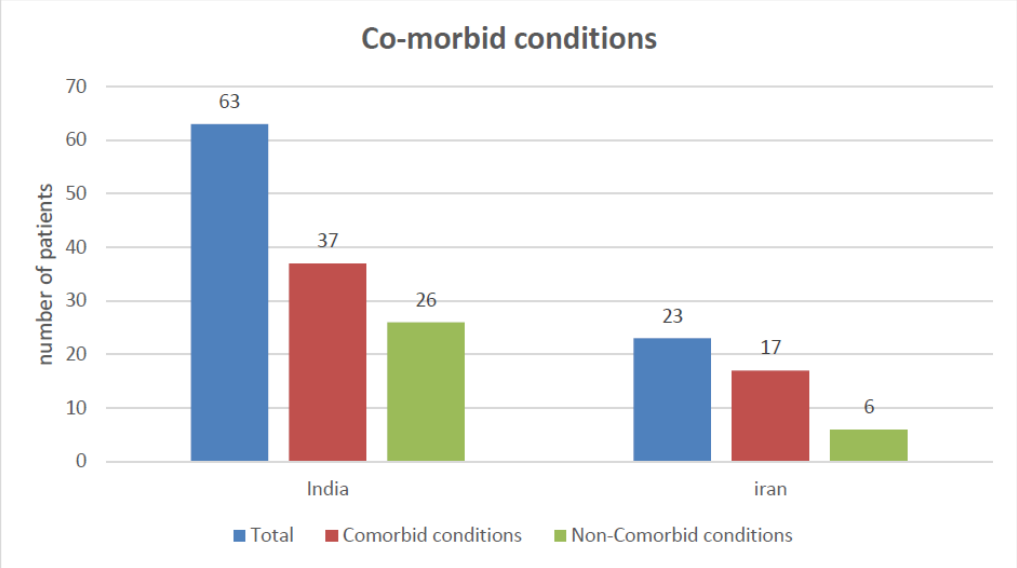


Figure 13: Graph showing Comorbid and non comorbid conditions observed in GBS patients.

Table 9 and figure 14 shows the levels of proteins in CSF normal range(15-45mg/dl). Out of 86 patients diagnosed with GB, 30(34.8%) shows high levels of protein with 56(65.11%) as normal range of protein in CSF.

Table-9: Protein levels in CSF in GBS patient.

Protein levels in CSF	Number of Patients
High levels	30(34.8%)
Within normal range	56(65.11%)
Total	**86**

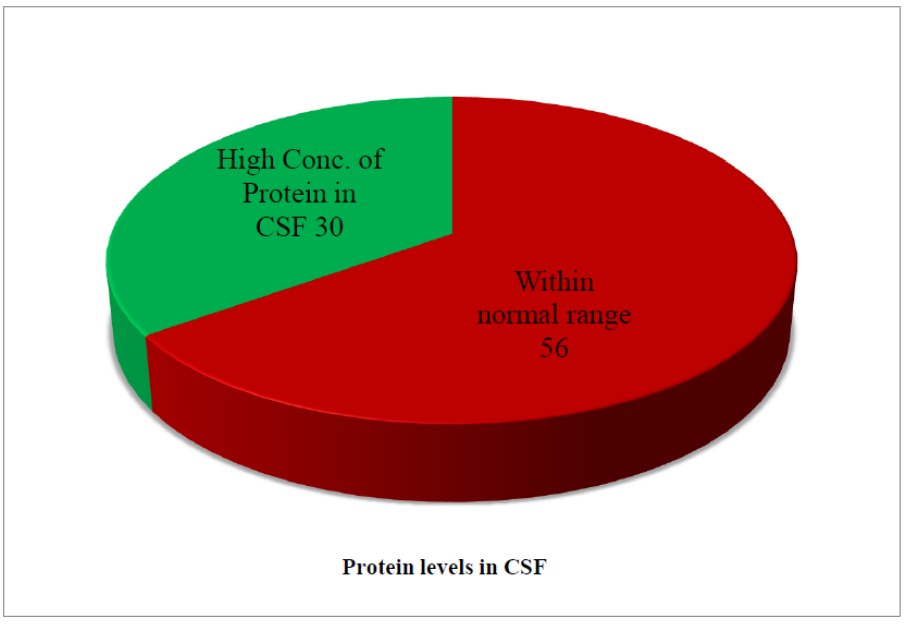


Figure 14: Graph shows Protein level in CSF in GBS.

Table 10 and figure 15 shows the treatment chart as majority prescribed in GBS patient. Out of 86 patient, 53 patient were given IV IGg, 36 patient had plasmapheresis, 27 patient were given corticosteroids, 33 were given antibiotics for treating the infection causing GB.

Table-10: Primary prescribing treatment pattern in GBS.

Treatment	Total	India	Iran
IgG	53	35	18
Plasmapheresis	36	26	10
Corticosteroids	27	19	8
Antibiotics	33	20	13
Total Number of Patients	**86**	**63**	**23**

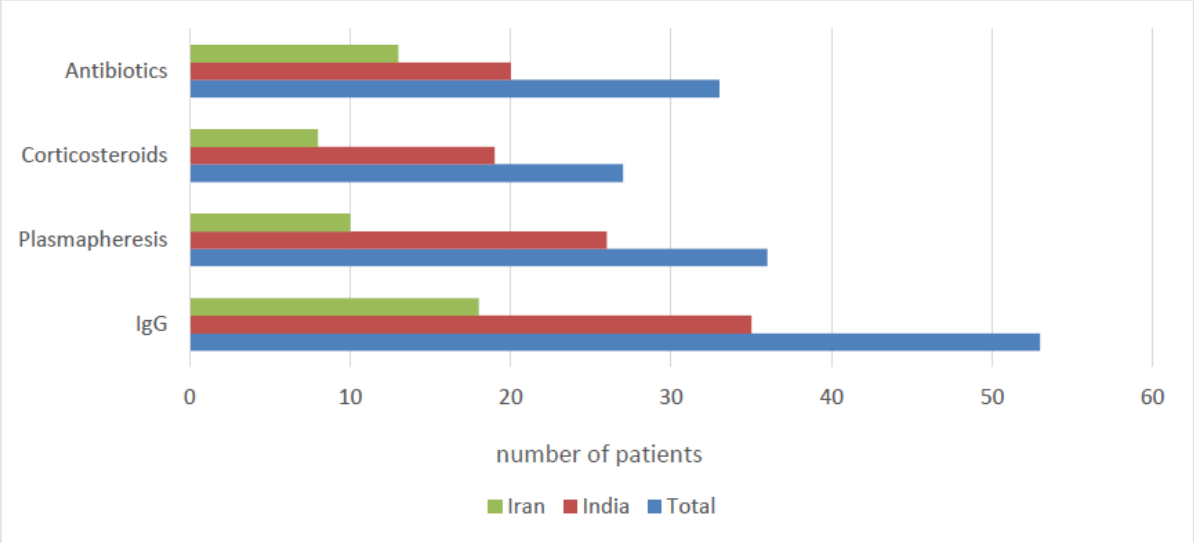


Figure 15: Graph showing details of primary prescribing treatment pattern in GBS.

Table 10 shows the treatment chart prescribed in GBS patient. Out of 86 patient, 53 patient were given IV IGg, 36 patient had plasmapheresis, 27 patient were given corticosteroids, 33 were given antibiotics for treating the infection causing GB.

Table-11: Prescribing treatment pattern in GBS.

Drugs	Number of patients received various classes of drugs		
	India (n:63)	Iran(N:23)	Total(n:86)
Antiemetics	11(17%)	2(8%)	13(15%)
Analgesics	27(42%)	6(26%)	33(38%)
Antibiotics	20(31%)	13(56%)	33(38%)
Antihypertensive	27(21%)	5(26%)	32(37%)
Antidiabetics	14(22%)	15(26%)	19(22%)
Anti Asthmatics	7(8%)	2(11%)	9(10%)
Antihistamines	3(4%)	7(30%)	10(11%)
Antiplatelet	12(19%)	11(47%)	23(26%)
Anticoagulant	25(39%)	12(52%)	37(43%)

Antilipidemic	10(15%)	7(30%)	17(19%)
Antipsychotic	1(2%)	3(13%)	4(5%)
Antidepressant	3(5%)	0(0%)	3(4%)
Hormones	9(15%)	1(4%)	10(11%)
Diuretic	4(7%)	1(4%)	5(6%)
Antianxiety	3(4%)	0(0%)	3(4%)
Proton pump inhibitor	53(84%)	16(69%)	69(80%)
Anticonvulsant	15(23%)	11(47%)	26(30%)
Corticosteroids	19(30%)	8(34%)	27(31%)
Immunoglobulins	35(55%)	18(70%)	53(60%)
Plasmapheresis	26(41%)	10(40%)	36(41%)

Figure 16 shows Various classes of drugs prescribed in GBS patients in india and iran. The IV IGg plasmapheresis, corticosteroids and antibiotics are the major drugs prescribed for management of GBS patients.

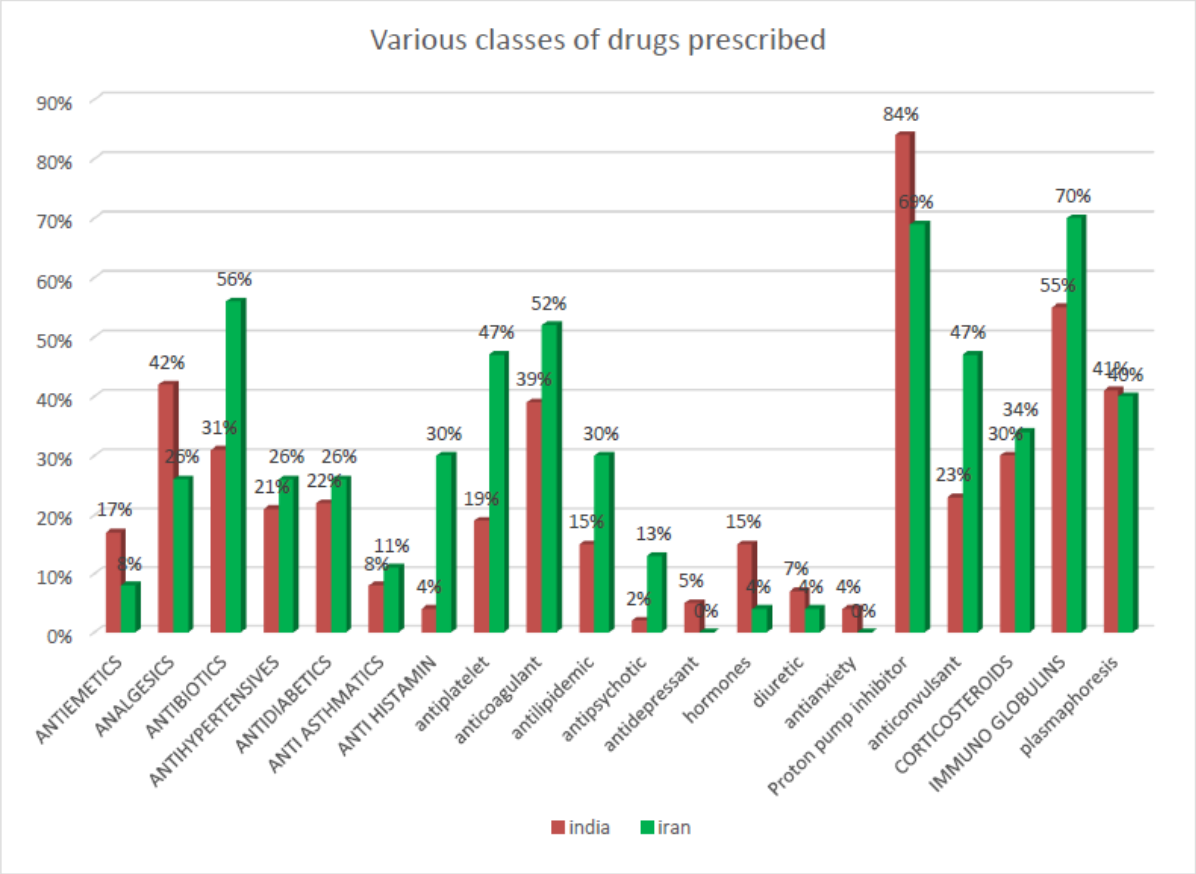


Figure 16: Graph showing details of prescribing treatment pattern in GBS in India and Iran.

Table 12 and figure 17 shows the type of Gb observed in GB patient. Out of 86 patients, 49 patients shown the different types of GB among which 27(69%) shown AMAN, 4(10.2%) shown AMSAN, and 8(20.5%) shown the AIDP.

Table-12: Type of GB observed in GBS patient.

Type of GBS	Number of Patients
AMAN	27
AMSAN	4
AIDP	8

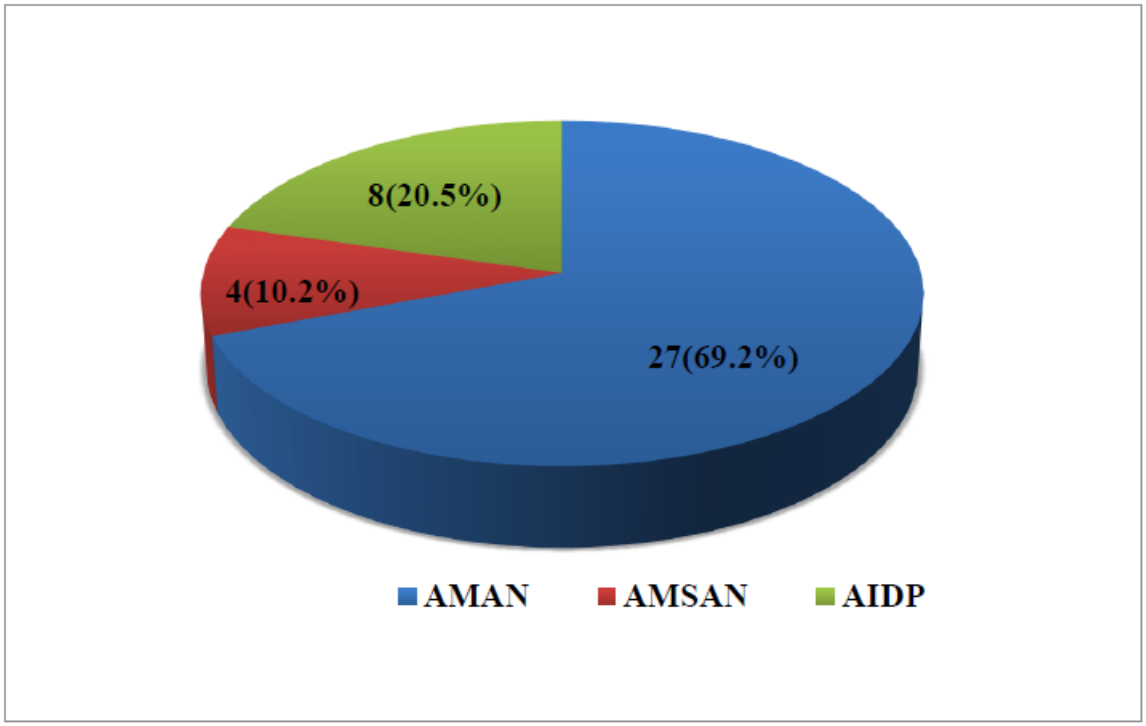


Figure 17: Graph showing details of Type of GBS observed in patients.

Table 13 and figure 18 shows the grades of GBs scale measured during admission time and discharge time Out of 86 patient, 77 patients shows 0 grade at discharge, 8 patients shows 3 grade and 1 patient showed grade 6 at discharge which shows drastic improvement in the symptoms when compared with admission time measurement of GB scale, in grade 2, 3 4, 5 shows the 20,35,22,9 patients in the following scale.

Table-13: Grades of GBS scale at the time of admission and discharge.

Grade Scale	**Total(** n=86)		**India**(n=63)		**Iran**(n=23)	
	At the time of Admission	**At the time of Discharge**	**At the time of Admission**	**At the time of Discharge**	**At the time of Admission**	**At the time of Discharge**
0	0	77(89.5%)	0	55(87.3%)	0	22(95.6%)
1	0	8(9.3%)	0	8(12.6%)	0	0
2	20(23.2%)	0	12(19%)	0	8(34.7%)	0
3	35(40.6%)	0	25(39.6%)	0	10(43.4%)	0
4	22(25.5%)	0	21(40%)	0	1(4.3%)	0
5	9(10.4%)	0	5(7.9%)	0	4(17.3%)	0
6	0	1(1.1%)	0	0	1(4.3%)	1(4.4%)

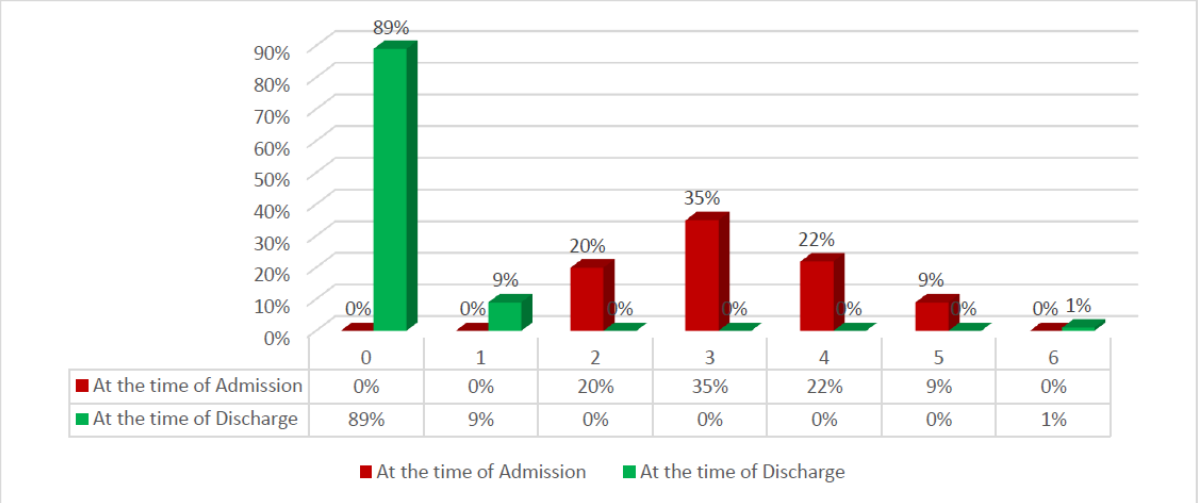


Figure 18: Graph showing details of grades of GB Scale at the time of admission and discharge.

Table 14 and figure 19 shows the differential diagnosis in GB patient out of 44 had undergone ENMG, 19 had undergone MRI scan, 7 had undergone CT scan and 1 with ECHO.

Table-14: Differential electrical diagnosis in GBS.

Differential Electrical diagnosis	Number of patients
ENMG	44
MRI	19
CT	7
Echo	1

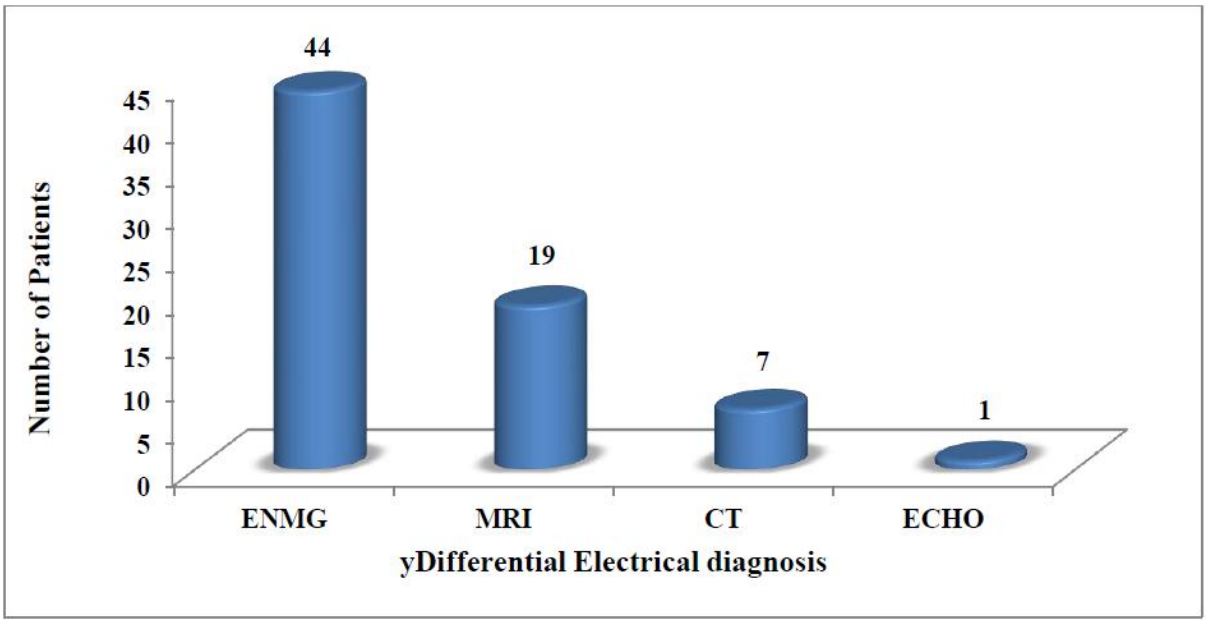


Figure 19: Graph showing details of differential electrical diagnosis in GB patients.

Table 15 shows possible drug interaction observed in medication chart of **GBS** patients. Aspirin + insulin 6(7.1%), Atorvastatin + Clopidogrel 5(5.9%), Aspirin + Enoxaparin 4(4.7%), has the most chance of occurrence among other.

Table-15: Drug interactions in GBS.

Drugs interacting	Effect of interaction	Severity	Frequency (Male)	Frequency (Female)	Frequency (Total)
Enoxaparin + Clopidogrel	Increase risk of bleeding	**Major**	1(3.5 %)	1(3.5%)	2(2.3%)
Enoxaparin + Pentoxifylline	Increase risk of bleeding	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Aspirin + Enoxaparin	Increase risk of bleeding	Moderate	1(3.5 %)	3(5.3%)	4(4.7%)
Aspirin + Clopidogrel	Increase risk of bleeding	Moderate	0(0%)	3(5.3%)	3(3.5%)
Aspirin + Fondaparinux	Increase risk of bleeding	**Major**	0(0%)	1(3.5%)	1(1.1%)
Aspirin + Methylprednisolone	Decrease the effect of Aspirin	Moderate	0(0%)	1(3.5%)	1(1.1%)
Aspirin + Telmisartan	Reduces the effect of Telmisartan	Moderate	0(0%)	1(3.5%)	1(1.1%)
Aspirin + insulin	Increase risk of hypoglycemia	Moderate	1(3.5 %)	5(8.9%)	6(7.1%)
Aspirin + losartan	Decrease the effect of Losartan	Moderate	0(0%)	2(3.5%)	2(2.3%)
Aspirin + Hydrocortisone	Gi irritation	Moderate	0(0%)	1(3.5%)	1(1.1%)
Aspirin + Valsartan	Decrease the effect of Valsartan	Moderate	0(0%)	1(3.5%)	1(1.1%)
Aspirin + Ibuprofen	Increase risk of GI ulcers	**Major**	1(3.5 %)	0(0%)	1(1.1%)
Aspirin + Ciprofloxacin	Cns side effects tremors, anxiety,	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Aspirin + Warfarin	Increase risk of bleeding	**Major**	0(0%)	1(3.5%)	1(1.1%)
Atorvastatin + Clopidogrel	Decreases effect of Clopidogrel	Moderate	2(7.1%)	3(5.3%)	5(5.9%)
Atorvastatin + Rosuvastatin	Increase risk of nerv damage	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Amlodipine + Bisoprolol	Headache and dizziness	Moderate	0(0%)	1(3.5%)	1(1.1%)
Amlodipin + Olanzapine	Headache and dizziness	Moderate	0(0%)	1(3.5%)	1(1.1%)
Amlodipine + Budesonide	Decrease the effect of **A**mlodipine	Moderate	0(0%)	1(3.5%)	1(1.1%)
Bisoprolol and Budesonide	Decrease the effect of Bisoprolol	Moderate	0(0%)	1(3.5%)	1(1.1%)
Bisoprolol + Methylprednisolone	Decrease the effect of Bisoprolol	Moderate	0(0%)	1(3.5%)	1(1.1%)

Tramadol + Ondansetron	serotonin syndrome	**Major**	2(7.1%)	0(0%)	2(2.3%)
Tramadol + gabapentin	Headache and dizziness	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Tramadol + pregabalin	Headache and dizziness	Moderate	2(7.1%)	0(0%)	2(2.3%)
Tramadol + Amitriptyline	Increase risk of seizure	**Major**	0(0%)	1(3.5%)	1(1.1%)
Tramadol + Escitalopram	Decrease effect of Tramadol	**Major**	1(3.5 %)	0(0%)	1(1.1%)
Gabapentin + Pregabalin	Headache and dizziness	Moderate	1(3.5 %)	1(3.5%)	2(2.3%)
Gabapentin + Amitriptyline	Headache and dizziness	Moderate	0(0%)	1(3.5%)	1(1.1%)
Levothyroxine + Glimepiride	Decrease effect of Glimepiride	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Pheniramine + Gabapentin	Headache and dizziness	Moderate	0(0%)	1(3.5%)	1(1.1%)
Pheniramine + Pregabalin	Headache and dizziness	Moderate	0(0%)	1(3.5%)	1(1.1%)
Pentoxifylline + Insulin	Increase risk of hypoglycemia	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Insulin + Metformine	Increase risk of hypoglycemia	Moderate	1(3.5 %)	1(3.5%)	2(2.3%)
Insulin + Levothyroxin	Decrease effect of Insulin	Moderate	0(0%)	1(3.5%)	1(1.1%)
Insulin + Telmisartan	Increase risk of hypoglycemia	Moderate	0(0%)	2(3.5%)	2(2.3%)
Insulin + Albuterol	Decrease effect of Insulin	Moderate	0(0%)	1(3.5%)	1(1.1%)
Insulin + Carvedilol	Increase risk of hypoglycemia	Moderate	0(0%)	1(3.5%)	1(1.1%)
Insulin + Losartan	Increase risk of hypoglycemia	Moderate	0(0%)	1(3.5%)	1(1.1%)
Insulin + Furosemide	Decrease effect of Insulin	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Digoxin + Ramipril	Increase blood level of digoxin	Moderate	0(0%)	2(3.5%)	2(2.3%)
Digoxin +Metoprolol	Decrease heart rate	Moderate	0(0%)	1(3.5%)	1(1.1%)
Metoprolol + Furosemide	Irregular heart beat,weakness	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Linezolid + Alfuzosin	Headache and dizziness	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Atropine + Hydroxyzine	Heat intolerance, Abdominal cramp	Moderate	1(3.5 %)	0(0%)	1(1.1%)
levothyroxine + Metformin	Decrease effect of Metformin	Moderate	0(0%)	1(3.5%)	1(1.1%)
Haloperidol + Labetalol	Changes in heart rate, Fainting	Moderate	0(0%)	1(3.5%)	1(1.1%)
Labetalol +	Additive effect in	Moderate	0(0%)	1(3.5%)	1(1.1%)

Alprazolam	Lowering BP				
Labetalol + Quetiapine	Additive effect in Lowering BP	Moderate	0(0%)	1(3.5%)	1(1.1%)
Quetiapine + Alprazolam	Dizziness _Confusion	Moderate	0(0%)	1(3.5%)	1(1.1%)
Nortriptyline + SERTRALINE	Constipation_ Urinary retention	Moderate	0(0%)	1(3.5%)	1(1.1%)
Ciprofloxacin + Hydrocortisone	RISK OF tendon rupture	Major	0(0%)	1(3.5%)	1(1.1%)
Midazolam + Spironolactone	additive effects in lowering BP	Moderate	0(0%)	1(3.5%)	1(1.1%)
Hydrocortisone + Spironolactone	Reduce the effects of Spironolactone	Moderate	0(0%)	1(3.5%)	1(1.1%)
Heparin + Spironolactone	kidney failure, Muscle paralysis	Moderate	0(0%)	1(3.5%)	1(1.1%)
Acetazolamide + Midazolam	Reduce the effects of Acetazolamide	Moderate	0(0%)	1(3.5%)	1(1.1%)
Furosemide + Midazolam	additive effects in lowering BP	Moderate	0(0%)	1(3.5%)	1(1.1%)
Salmeterol + Carvedilol	Reduce the benefits of both medications	Major	1(3.5 %)	0(0%)	1(1.1%)
Clonazepam + Morphine	CNS depression coma	Major	1(3.5 %)	0(0%)	1(1.1%)
Midazolam + Morphine	Dizziness, drowsiness, confusion	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Dopamine + Metformin	Decrease effect of Metformin	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Morphine + Metformin	Increase the effects of Metformin	Moderate	1(3.5 %)	0(0%)	1(1.1%)
Ibuprofen + Methylprednisolone	GI irritation	Moderate	0(0%)	1(3.5%)	1(1.1%)
Total			**28**	**56**	**84**

Table 16 and figure 20 shows History of infectious disease before symptom onset. out of 86 patients the 43(50%) had Respiratory tract infection and 12(13%) had Gastrointestinal infection.

Table-16: History of infectious disease.

	India	**Iran**	**total**
Respiratory tract infection	36	7	43
Gastrointestinal infection	9	3	12
others	3	1	4
Total number of patient	**63**	**23**	**86**

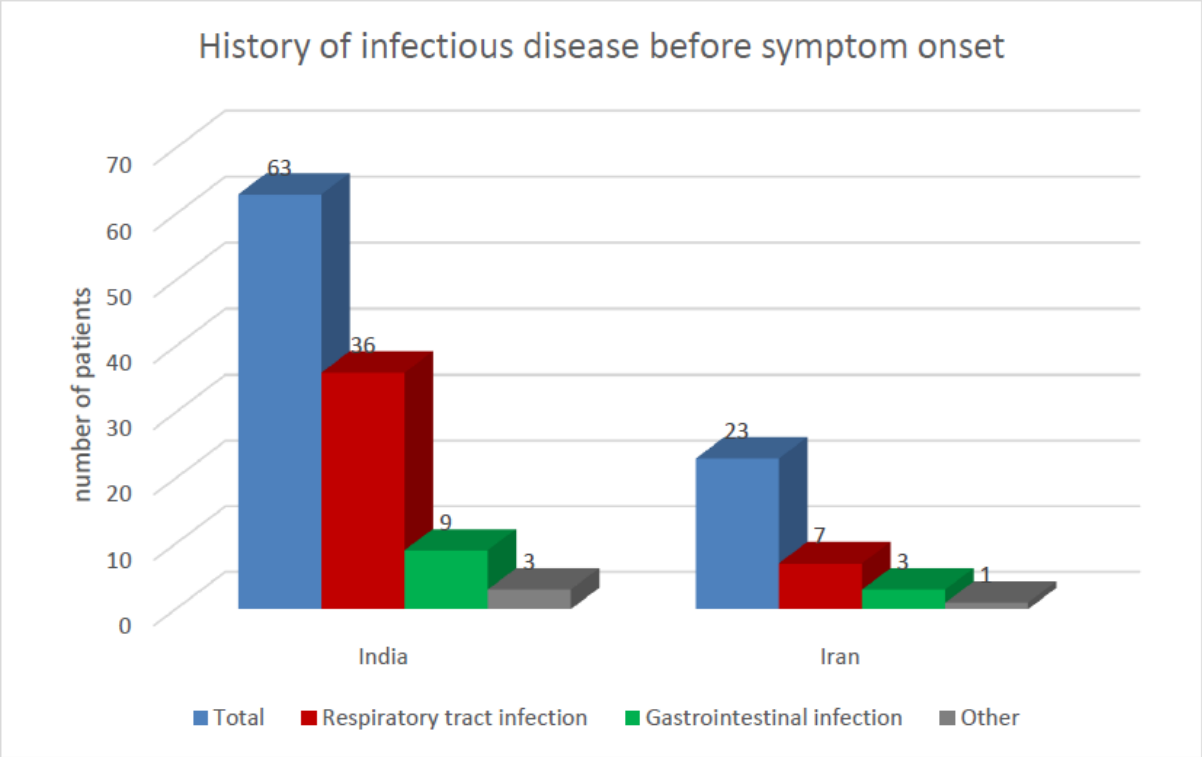


Figure 20: Graph showing details History of infectious disease in GB patients.

From the following tables and graphs, the retrospective study on GB syndrome was been carried out and the analysis was done on the following parameters such as age, gender discrimination among India and Iran and comparison between India and Iran population, major symptoms, co morbid and non co morbid conditions, type of GB, protein levels in CSF, primary treatment prescribing pattern and Grades of GBS scale at the time of admission and at the time of discharge which gives the relevant population distribution and its percent or proportion in other remarkable parameters which either helps in finding out the differential diagnosis or to improve the treatment pattern or helping in epidemiological studies.

The treatment data shows the other following treatment due to following co morbid conditions like type II diabetes mellitus, hypertension, hypothyroidism, respiratory infection, pneumonia, urinary tract infection, Gastro intestinal bleeding, gastritis, chronic renal failure, Parkinson and so on which includes the medication like antidiabetics like metformin, glimipride, glibenclamide, antihypertensivesn like atenolol, carvedilol, metoprolol, amlodipine, clonidine, telmisartan, losartan antiemetics, proton pump inhibitors, antihistamines like citirizine, levocitrizine, antiaxiety, antipsychotics, antiplatelet, anticoagulants, antibiotics, corticosteroids and so on which might causes drug drug interaction only in few scenario, the interaction might be unusual bleeding , head ache,

dizziness, either increasing the effect of drug or decreasing the effect of drug due to other drug usage, seizures, bradycardia or tachycardia, hypoglycemic effects or hypotension, electrolyte imbalance and gastrointestinal effects which might be occurred as mild, moderate sometimes as severe serious interaction.

CONCLUSION

Finally, I conclude that the retrospective study on GB syndrome helps in easy identification of GB syndrome at an early stage in order to prevent progress of disease, the GB scale which gives the grades helps in detection of mild, moderate and severity of GBS and its stages at the diagnosis time and the Typical ENMG, MRI scan helps in easy detection of demyelinated neurons or lesions and the treatment with the IV IGg has improved the quality of life of the patient compared to other treatment in majority cases. The study shows that females are more prone to GB syndrome than male and the higher incidence of occurrence of GB occurs at an age of 21- 30 years. Progressive weakness in both arms and legs 72(83%) is the higher incidence of symptoms. AMAN 27(30%) is higher incidence of subtype GBS.

Aspirin + insulin 6(7.1%), Atorvastatin + Clopidogrel 5(5.9%), Aspirin + Enoxaparin 4(4.7%), has the most chance of drug-drug interactions during management of GBS.

LIMITATIONS

- The study period was less (2016-2018) because of rarity of the syndrome covered less number of sample size to carryout in a large number of population since it’s a binational study.
- Equal partition on study period at study sites did not meet the criteria due to which the number of population in the study has diversion since it’s a binational study.

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ANNEXURE

Annexure 1: patient data collection form

IP no:	Age:	Sex:
Comorbidities:	Onset of GBS:	
	Duration stay:	
	progressive weakness in both arms and legs: present ◯ not ◯	
relative symmetry of syndrome:	Mild sensory of signs or symptoms:	
Clinical manifestations : Cranial nerve involvement Bulbar involvement Ophthalmoplegia Areflexia		
Absence of fever at onset:		
Typical electrical Diagnostic features :		
High concentration of Protein in **CSF**:		
Glucose level in **CSF**:		
Chloride level in **CSF**:		
Serum CR level:		
Subtype of **GBS** :		
Plasmapheresis :		

Corticosteroids	**Dose**	**frequency**

Immunoglobulins	Dose	frequency
Other Drugs		

Interacting Drugs			Interacting Drugs	
Effect of interaction:			**Effect of interaction:**	
Type of interaction: Major Moderate			**Type of interaction:** Major Moderate	

GBS DISABILITY SCALE:	GRADE 0 healthy	GRADE 1 Symptomatic but capable of running	GRADE 2 Incapable of running	GRADE 3 Unable to walk without assistance	GRADE 4 Confined to bed	GRADE 5 Requiring assisted ventilation	GRADE 6 death
At first							
At discharge time							