"A STUDY ON THE CLINICAL AND ELECTRODIAGNOSTIC PROFILE OF GUILLAINE BARRE SYNDROME AND ITS CORRELATION WITH EARLY PREDICTORS OF PROGNOSIS IN A TERTIARY CARE CENTRE

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CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON THE CLINICAL AND ELECTRODIAGNOSTIC PROFILE OF GUILLAINE BARRE SYNDROME AND ITS CORRELATION WITH EARLY PREDICTORS OF PROGNOSIS IN A TERTIARY CARE CENTRE"** is a bonafide work done by **DR. KARTHIKA.R**, Post Graduate Student, Institute of Internal Medicine, Madras Medical College, Chennai-3, in partial fulfillment of the University Rules and Regulations for the award of MD Branch – I General Medicine, under our guidance and supervision, during the academic year 2012 - 2015.

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I, **DR. KARTHIKA.R,** solemnly declare that the dissertation entitled **"A STUDY ON THE CLINICAL AND ELECTRODIAGNOSTIC PROFILE OF GUILLAINE BARRE SYNDROME AND ITS CORRELATION WITH EARLY PREDICTORS OF PROGNOSIS IN A TERTIARY CARE CENTRE"** is done by me at Madras Medical College, Chennai-3 during April 2014 to September 2014 under the guidance and supervision of Prof. K. S. CHENTHIL, M.D., to be submitted to The Tamilnadu Dr. M.G.R Medical University towards the partial fulfillment of requirements for the award of M.D. DEGREE IN GENERAL MEDICINE BRANCH-I.

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ABBREVIATIONS

GBS	Guillain Barre syndrome
AIDP	Acute inflammatory demyelinating polyneuropathy.
NCS	Nerve conduction study.
EMG	Electromyography.
IVIG	Intravenous immunoglobulin..
CMAP	Compound muscle action potential.
AGE	Acute gastroenteritis.
C.jejuni	Campylobacter jejuni.
AMAN	Acute motor axonal neuropathy.
AMSAN	Acute motor sensory axonal neuropathy.
MFS	Miller Fisher variant.
CSF	Cerebrospinal fluid.
SIADH	Syndrome of inappropriate antidiuretic hormone secretion.
LL	Lower limb
UL	Upper limb

CONTENTS

INTRODUCTION

Guillain Barre Syndrome (GBS) is an acute, autoimmune, frequently severe, polyradiculoneuropathy. GBS affects all ages and it has a slight male preponderance. It is a symmetrical, predominantly motor, flaccid and areflexic paralysis with a clinical progression of the disease usually for a period of 4 weeks.

GBS has an incidence of around 0.4-1.9 per one lakh population. The neuropathic symptoms are preceded by mild upper respiratory or gastrointestinal symptoms in most of the cases by 1-3 weeks in 50-70% of cases. Recently enteric organisms Campylobacter jejuni serological studies are done and it is identified as the causative organism in a small proportion of cases. Multiple cranial nerve palsy involving facial, bulbar and ocular muscle has been reported in 10-60% of cases with papilledema being uncommon. Respiratory failure occurs in 10-30% of cases.

GBS is a self limiting disorder associated with recovery in upto 70% of cases but the morbidity and mortality is bestows is also a concern as the sequelae of the disease is associated with significant motor handicap in up to 7% of cases. Mortality usually reaches up to 14% in few studies.

GBS subtypes are recognized on the basis of electro diagnostic and pathologic distinctions and it includes acute inflammatory demyelinating polyneuropathy (AIDP), the most common variant, followed by two axonal variants –Acute motor sensory axonal neuropathy (AMSAN) and Acute motor axonal neuropathy(AMAN). Regional variants include 1. Miller Fisher syndrome,2. Pandysautonomia, 3.pure sensory syndrome, ophthalmoplegia with anti-GQ1b antibodies, 4.GBS with severe bulbar and facial palsy.

Cerebrospinal fluid analysis typically reveals ‘albumino-cytological dissociation. Nerve conduction studies shows reduced conduction velocity with conduction block, accompanied with increased distal latency. Prolonged or absent F waves in two or more motor nerves

is characteristically seen. Sensory nerve conduction delay occurs in up to 60% of cases.

Immunomodulation is the treatment of choice, done with either plasmapharesis or intravenous immunoglobulin (IVIG) with both modes having equal efficacy in terms of treatment.

AIM AND OBJECTIVES OF STUDY

The purpose of this study is about the clinical profile with electro diagnostic features of GBS in a South Indian population admitted at a tertiary care Centre in Madras Medical College, to correlate the clinical and electro diagnostic features with disability of the disease and thereby to identify the poor prognostic clinical and nerve condition features in a much earlier course of the disease so that ,those groups can be treated and monitored more scrupulously during the course of the disease and hence attenuate the morbidity and mortality associated with the disease.

REVEIW OF LITERATURE

GENERAL CONSIDERATION

Guillain-Barre Syndrome (Landry-Guillain-Barre-Strohl Syndrome) also known as acute inflammatory demyelinating polyneuropathy, (AIDP) is a common cause of acute or sub-acute generalized paralysis with close competence to Polio at a time in history[1]. It has a worldwide distribution without much seasonal variation, though certain studies show an increased incidence of the disease in winter and few temperate countries show an increased trend in the rainy months[1-5]. Respiratory and gastro intestinal infections can occur at the offset the disease in up to 70% of cases[7]. They are followed by neurological symptoms and signs 1-3 weeks or sometimes even longer in around 60% of cases.Though the infections are trivial but often febrile. Other antecedent events associated with GBS are immunization at one point or another and can even sometimes occur as coincidence though details are not

available. Few microbes are associated with GBS. Recent studies can demonstrate serological markers of enteric organisms such as Campylobacter jejuni associated with causation of the disease but has been proved as a causative agent only in small proportion of the cases up to 32%[10]. The clinical symptoms include that of watery diarrhea and abdominal cramping. It produces motor axon degeneration associated with increased anti-GM1 antibodies and often shows delayed but incomplete recovery of GBS[11]. Other organisms implicated are viruses like Cytomegalovirus which is associated with sensory and cranial nerve dysfunction and elevated anti GM2 antibodies, Epstein-Barr-Virus, Human immunodeficiency Virus, bacteria's such as Mycoplasma pneumonia and Borrelia burghdoferi. Lymphomas such as Hodgkin Lymphoma and exposure to thrombolytic agents have been rarely associated with GBS[5]. Vaccines such as the outdated anti-rabies vaccine[23] developed with the nervous tissue and the swine influenza vaccine, also known as the New Jersey vaccine given in late 1976

showed a slight increase in the number of GBS cases then[21-28]. Trauma and surgery are very rarely know to precede the neurological symptoms of GBS but clear cut causal relation is yet to be proved. GBS has also been reported along with autoimmune conditions like systemic lupus erythematosus very rarely[19]. Other much rarer association include carcinoma lung, paraproteinemia and sarcoidosis. GBS has also been reported in the postpartum period.

HISTORY OF GBS

The first report of generalized paralysis was found in the description given by Wardrop and Ollivier in the year 1934. Later in the year 1859 Landry reported a case of acute ascending motor paralysis associated with respiratory failure culminating in death. It was the French neurologist trio of Guillaine, Barre and Strohl described the acute flaccid paralysis that developed in two French soldiers and found to resolve spontaneously without any treatment

or complication. They even reported the albumino-cytological dissociation found in the CSF analysis of those patients which was quiet

unique compared to the flaccid paralysis of Polio. The pathophysiology of GBS was described by Haymaker and Kernohan in the year 1949 who emphasized the edema of the nerve roots during the initial stages of the disease. In the year 1969 the hallmark of the disease was studied and found to be perivascular mononuclear inflammatory infiltration of the nerve roots which was described extensively by Asbury, Arnason and Adams[1-5]. Though the disease has no specific diagnostic tests it remains a descriptive disorder characterized by rapidly progressive weakness usually symmetrical involving both legs and arms, with hyporeflexia or areflexia and a flaccid type of paralysis. More recently complement deposition of the myelin sheath have been demonstrated as the earliest clinical event taking place.

INCIDENCE

The incidence of GBS ranges from 0.4-1.8 per one lakh population per year[6-10]. Most studies found a male preponderance of the disease with

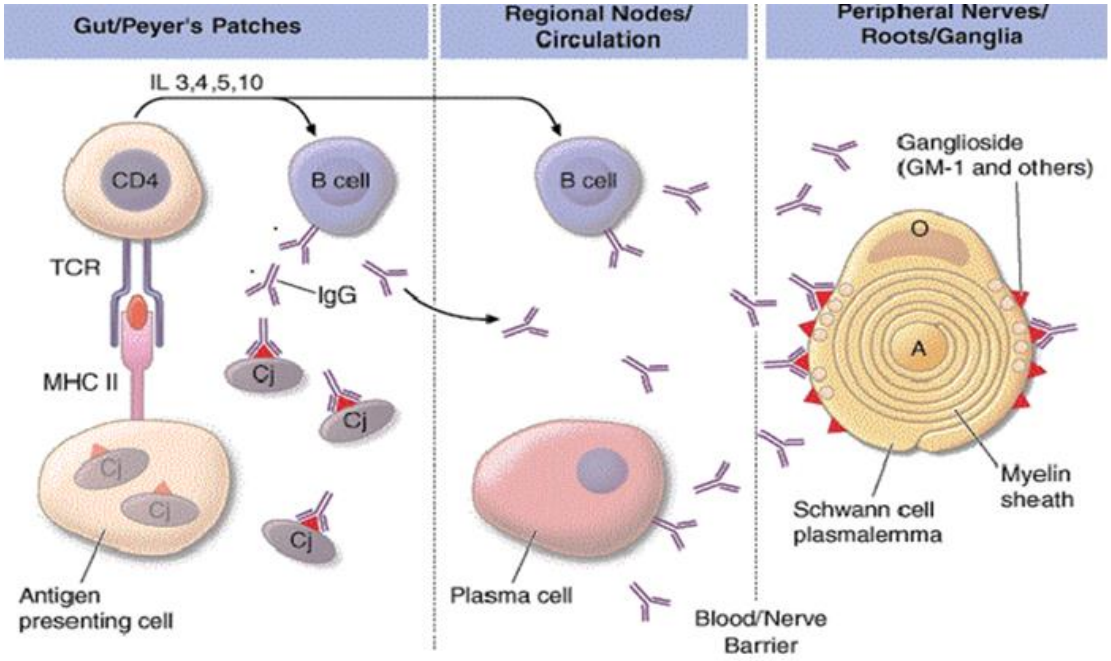
a male is to female ratio of 3:2. Regarding the seasonal variation, is one of the conspicuous reports from China with axonal variants in the rural rice fields during the years of 1991-1992. The disease is prevalent in both extremes of the age group with mean age of the population being 44.

PATHOPHYSIOLOGY

Several studies demonstrate the autoimmune nature of the disease process with the cell mediated immunologic reactions directed against the peripheral nerves. Experimentally induced peripheral nerve disease also known as experimental allergic neuritis or EAN was demonstrated by Adams et al in animals 2 weeks after immunization[14]. The basic protein designated as P2 found only in the peripheral nerves was demonstrated by Brostoff

and his colleagues. High levels of soluble interleukin IL-2 receptors shed from the activated T cells and IL-2 itself found in high levels in GBS patients have been found out. It involves all the cell mediated,

humoral and complement mediated immune system in the pathogenesis of the disease process[32]. Postulated immunopathogenesis of GBS which is associated with Campylobacter jejuni infections has been demonstrated below in the pictorial representation of the disease pathophysiology. The glycoconjugates on the C.jejuni cross reacting with the ganglioside of the Schwann cell surface receptors and adjacent peripheral myelin sheath are recognized by the B cells and these B cells along with those activated by the T cell and independent of T cell activation secrete the IgG and IgM antibodies respectively. CD4 cells also take part in this process of T helper cell action by the protein of C.jejuni presented by the antigen presenting cells. The crucial event taking place is the escapade of the activated B cells from the gut Peyers patches into the regional lymphnodes. Along with the activated T cells, the activated B cells break the blood nerve barrier allowing the deposition of auto antibodies into the myelin sheath and the perivascular area. This is characterized by edema between the myelin lamellae and vesicular disruption demonstrated in the the form of circular blebs along the outer surface of the myelin sheaths. The membrane attack complex consisting of the C5b-C9 allows the calcium entry into the myelin sheaths along with other mediators of myelin damage such as the TNF-Tumor necrosis factor[33].



I. ANTIGANGLIOSIDE ANTIBODIES

Various ganglioside antibodies like GM1, GM1b, GM2, GD1a, GD1b, GQ1b, GT1a[32-35] etc, have been distributed in a specialized fashion in the nerve sheaths known as **lipid rafts**. These antibodies are associated with variants of GBS. GM1 and GD1a antibodies are associated with pure motor or axonal variants whereas GD3, GT1a and GQ1b are associated with the Miller Fisher variant. Glycolipid directed antibodies and even antibodies to the peripheral nerves have been identified. In few variants of GBS such as the sensory motor variant the antibody target is yet to be identified

II. MOLECULAR MIMICRY AND CROSS REACTIVITY

The immune response towards the non-self antigens are directed or misdirected towards the host nerve tissue because of a epitope resemblance known as molecular mimicry[38]. Glycoconjugates present in the neural tissue are the targets. To be more specific the gangliosides which are the complex glycosphingolipids with sialic acid residues and participates in cellular interaction is mimicked by the lipooligosaccharides which are expressed by C.jejuni. Gangliosides are present in abundance in the plasma membrane of cells and also in the human nervous tissue at the Nodes of Ranvier. GM1 antibodies are the most common type directed against the gangliosides of the nervous tissue in C. jejuni infections[42]. These antibodies are present in 20-50% of cases.

Sialic acid residues of these pathogenic C.jejuni can also trigger the dendritic cells and hence the toll like receptors and resulting in B cell activation and hence humoral autoimmunity. H.influenza is even known to demonstrate molecular mimicry strategies.

Few reports of the purified bovine brain gangliosides which are used in the treatment of various neuropathic disorders are shown to inflict GBS in few group of patients in Europe and these patients demonstrate high level of anti GM1 antibodies targeted against the Nodes of Ranvier.

III. COMPLEMENT MEDIATED ACTIVATION

Complement activation have been shown to produce nerve damage by the anti GM1 antibodies around the Nodes of Ranvier[86], disrupting the sodium channels resulting in conduction blocks at these sites[56-57]. Experimental models reveals the similarity of complement activation to that of a alpha latrotoxin like effect which is associated with dramatic release of acetylcholine and neurotransmitter depletion at these sites and nerve muscle paralysis. Interestingly these neurotoxic effects can be inhibited by the immunoglobulins and complement inhibitors like eculizumab[75].

Miller Fisher Syndrome is associated with Anti GQ1b IgG antibodies which are found in 90% of these cases[40,73]. These antibodies are found early in the course of the disease and also are specific to this particular variant unless the other variants involve the extra ocular motor nerve involvement. The extra ocular nerves are abundant in the GQ1b antibodies compared to other organs.

Though these observations are strong enough to suggest an autoimmune basis as a strong pathogenic cause in GBS yet are found to be inconsistent. This is because antibodies are also demonstrated against the Schwann cells and nerve growth cone regions but these antibodies were not able to produce disease when direct passive transfer was done to

a naïve host. On the other hand trans placental transfer of antibodies resulting in GBS in the fetus has also been demonstrated[51].

Complement mediated activation occurs first which results in vesicular disintegration of the myelin sheath and macrophage recruitment at these sites thus producing collateral damage to the axon and myelin sheaths. Anti GD1a antibodies found in AMAN variant are deposited in the large motor axons and sparing the sensory roots as well although both motor and sensory nerves express the gangliosides.

Conduction block is the hallmark of demyelinating forms of GBS resulting in flaccid paralysis and sensory disturbances. Since axonal connections remain intact the recovery can take place rapidly as remyelination can occur[69]. But once axonal degeneration sets in the recovery rate can be much slower and secondary degenerative changes in the axons can result in residual disability. When axons have become degenerated and disintegrated demonstrable by the electrophysiological studies they much regenerate for recovery to occur. Other alternatives for regeneration includes collateral sprouting and reinnervation from motor axons can occur over a period of several months.

CLINICAL FEATURES OF GBS

GBS is rapidly evolving in nature with features of paresthesia and slight numbness of the toes and fingers being the earliest manifestation. However the areflexic motor paralysis evolving over a period of several day to 4 weeks is the course of the illness. The paralysis is ascending type and the patient characteristically describes their symptoms as rubbery legs. The legs are affected more than arms and both proximal and distal group of muscles are affected. The trunk, intercostal, neck and cranial muscles may be affected later[4-7]. Facial paresis usually bilateral occurs in 50% of cases. Bulbar palsy and lower cranial nerve involvement are also frequently reported and associated with a delayed recovery or respiratory failure but these can be mistaken for brainstem ischemia[90]. In upto 5% percent of the patients the disease can result in total paralysis with respiratory failure occurring in a few days. Ocular nerve palsy with unreactive pupils can also occur.

Pain and aching discomfort involving the hips, thighs and back or even diffusely over the entire spine can occur in about 50% of the cases and these are mistaken for back strain, lumbar disc disease and even other orthopedic diseases. Even few patients describe burning sensation involving the toes and fingers as their earliest symptom and it can become a persistent problem. Vibration and proprioception are reduced in

the toes and fingers are reduced in the initial weeks and touch-pressure vibration tends to be more affected than pain and temperature.

Deep tendon reflexes are reduced or absent and these findings are usually consistent. It is due to desynchronization of afferent impulses in reflex arc due to non – uniform demyelination. Rarely only the ankle reflexes are lost in the initial weeks of the illness. The need for mechanical ventilation is associated with rapid progression of the disease process often associated with bulbar and facial muscle weakness. Fever and other constitutional symptoms are absent at the onset of the illness.

Bladder dysfunction may occur in the much severe cases but it is usually transient in the initial weeks, if bladder dysfunction is found to be persistent than the diagnosis of GBS is to be doubted. The plateau of the disease is reached within a period of 4 weeks in most cases

Disturbance in autonomic functions can occur even in milder cases of GBS and include the following features of sinus tachycardia, less often bradycardia, fluctuating blood pressure, facial flushing, loss of sweating or episodic profuse diaphoresis and cardiac arrhythmias. These symptoms infrequently persist for more than a week. Pain which is a dysesthetic pain can occur and its responsive to the usual pain medication. Lymphadenopathy and splenomegaly should prompt the diagnosis of viral infection associated with the disease.

VARIANTS OF GBS

- REGIONAL

- Miller Fisher syndrome of ophthalmoplegia, ataxia and areflexia
- Cervico-brachial –pharyngeal, associated with ptosis
- Oculopharyngeal weakness
- Predominantly paraparesis
- Bilateral facial or abducens weakness with distal paresthesias
- Ophthalmoplegia with GQ1b autoantibodies

- FUNCTIONAL

- Generalized ataxia without dysarthria or nystagmus
- Pure sensory
- Pure motor
- Pan dysautonomia
- Axonal

ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY

The most common variant of is **Acute inflammatory Demyelinating polyneuropathy.**

Adults are most commonly affected

90% occurs in western world

Recovery is rapid usually

Anti-GM1 antibodies is present in less than 50%

Electrophysiological pattern shows a demyelinating pattern

Schwann cells are affected first followed by widespread myelin damage, macrophage activation with lymphocytic infiltration and axonal damage.

ACUTE MOTOR AXONAL NEUROPATHY (AMAN)

This variant was described in detail by Feasby and his colleagues of an acute areflexic polyneuropathy with widespread and severe axonal degeneration[35].

Predominantly children and young adults are affected and has been prevalent in China and Mexico associated with rapid evolution of symptoms with very slow and poor recovery.

Muscle atrophy becomes evident relatively early in the course of the disease.EMG shows numerous electrically in excitable motor nerves with signs of extensive denervation. Post mortem examinations show severe axonal degeneration in nerves and roots with minimal inflammatory changes.

Complement depositions are prominent and macrophage has been demonstrated in the periaxonal space. Visser et al reported similar findings in a series of polyneuropathies from Holland. C.jejuni has been found to be associated with this type of GBS. Circulating antibodies to anti GM1 and anti GD1a have been demonstrated[31].

AMAN and AMSAN are equivalent to axonal GBS. Characterized by symmetric limb weakness, diffuse areflexia, facial and oropharyngeal muscle weakness and respiratory insufficiency. Extraocular muscles are typically spared

Another variant of this illness has been described in several instances as an acute multifocal neuropathy with electrophysiologic motor conduction block where the reflexes are unaltered described by Caposso et al. A chronic form of this is termed as Multifocal motor neuropathy as it is commonly understood.

It is not known whether the acute axonal polyneuropathy is a distinct entity of GBS or a variant39. These axonal neuropathies have poor response to immunetherapy[91]. Sometimes the electrically unexcitable nerves can be due to conduction block rather than axonal degeneration. The differential diagnosis of critical illness polyneuropathy and rarer entities of porphyric neuropathy and tick paralysis should be considered.

ACUTE MOTOR SENSORY AXONAL NEUROPATHY(AMSAN)

It is a motor sensory axonal variant of GBS with an annual incidence of 1.1 per one lakh population. First described by Feasby and his colleagues with severe disease progression[69] . The disease process is characterized by quadriparesis with areflexia, distal sensory loss and respiratory failure. Upto 30-50% of cases have been reported in Asia and Latin America. Electrodiagnostic profile shows a diffuse loss of motor and sensory potentials with diffuse degeneration without evidence of demyelination. As with other types of GBS an infectious disease precedes the onset of limb weakness in majority of cases. It is associated with the following antibodies anti-GM1/GD1a/GM1b/GalNac-GD1a).

MILLER FISHER VARIANT

Is known for its classic triad of ophthalmoplegia, ataxia and areflexia which was described by C.Miller Fisher in 1956.

Usually accounting for 5% of the GBS cases and in some studies upto 12% have been described .in south Asian population.

Diplopia is the usual initial symptom followed by limb or gait ataxia associated with mild sensory symptom, swallowing difficulties or proximal muscle weakness occurring in upto one half of the cases.

Abducent nerve palsy is the first extraocular muscle palsy to occur and it may progess to complete ophthalmoplegia. Though ptosis is frequent, pupillary affliction is rare.

Asymmetric limb involvement can occur in few cases with limb and gait ataxia which can be confused with cerebellar disease. CSF protein elevation is much less compared to the typical GBS. Adults and Children are affected with antibodies directed against GQ1b antibodies.

Electrodiagnostic studies are variable with loss of sensory potentials, mostly demyelinating in nature sometime alternating with milder forms of axonal degeneration. May be confussed sometimes with encephalitis or cerebrovascular accidents.

Motor nerve terminal blockade explains generalized weakness when associated with reversible failure of Acetylcholine release from presynaptic motor nerves.

PURE MOTOR VARIANT

Usually acute and progressive without sensory loss which has been reported in upto 18% in one case series. Predominantly involves the distal limbs but cranial nerve sparing and elevated anti GM1 titres and serology positive for C.jejuni infection. Course and recovery is similar to GBS.CSF proteins are usually elevated.

Electrodiagnostic profile shows both axonal and demyelinating variety sometimes with marked axonal degeneration making recovery slower and associated with much more disability.

Poliomyelitis, acute myasthenia gravis and tick paralysis can be considered as the differential diagnosis.

PURE SENSORY VARIANT

Relatively rare form of polyneuropathy with rapid onset large fiber sensory loss with the predominant symptom being sensory ataxia.Tremors, positive Romberg sign, pseudoathetosis and dysautonomia can occur. In severe cases sensory dysfunction may

involve face and torso. CSF proteins are typically elevated and Electrodiagnostic study shows a demyelinating pattern

Cervical myelopathy, Sjogren's syndrome , Malignant and non malignant sensory neuropathy should be considered as the differential diagnosis.

PURE DYSAUTONOMIC VARIANT

Considered to be a much rarer variant of GBS. Initial symptoms being abdominal pain, vomiting , diarrhea and constipation with possible history of preceding viral infection

Sometime gastroparesis with abdominal distention and ileau has been reported. Orthostatic hypotension associated with syncope, urinary urgency, retention, vasomotor instability and erectile dysfunction has been reported.

Cardiac arrhythmias warrant intensive care monitoring of the disease process. Motor involvement is rare with milder forms of distal sensory symptoms and areflexia is usually present.

Electrodiagnostic study is normal showing autonomic variability, tilt table testing, sympathetic skin response and abnormal sweat testing(

QSART). Plateau is after a week and recovery is slow involving several months.

PARAPARETIC VARIANT

One of the types of regional variant with isolated leg weakness and areflexia. Typically upper limbs, cranial nerves and spinchters are spared with radicular pain being the most common symptom. Electrodiagnostic study is demyelinating. MRI studies are done to rule out spinal cord disease such as disc disease or caudaequina syndrome.

PHARYNGEAL CERVICAL BRACHIAL VARIANT

Another regional variant affecting cervical, brachial and oropharyngeal muscles exclusively with antibodies against GT1a showing a higher level. May initially involve the pharyngeal and neck muscles with later spread to the limbs and lowerlimbs are typically spared.

As it involves facial weakness with ptosis and ophthalmoparesis it can be mistaken for myasthenia gravis. Electrodiagnostic study shows a demyelinating pattern and the recovery is usually is late and incomplete.

OTHER LESS COMMON VARIANTS

- Bilateral foot drop with upper limb paresthesias
- Acute ataxia without ophthalmoplegia
- Facial diplegia or abducent palsies with distal paresthesias
- Acral paresthesias with diminished reflexes in either arms or legs.
- Isolated post infectious ophthalmoplegia.

DIAGNOSTIC FEATURES OF ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY[12-13]

- Progressive weakness of limbs from mild paresis to complete paralysis
- Generalized hypo or areflexia
- Rapidly progressive motor weakness which reaches nadir in 2 weeks in 50% , 3 weeks in 80%, 4 weeks in 90%\
- Mild to moderate sensory loss
- Symmetrical limb involvement
- Cranial nerve involvement-facial nerve paralysis which occurs in 50% and it is bilateral and asymmetrical, cranial nerves XII,X, III,IV,VI and XI can be involved
- Recovery begins in 2-4 weeks
- Autonomic dysfunction which includes tachycardia, arrhythmias, postural hypotension and vasomotor symptoms
- Proceeding infections which may be gastrointestinal or respiratory tract infection.
- Elevated CSF proteins
- CSF counts which are <10 mononuclear cells/mm3

- Nerve conduction studies show slowing or conduction block in 80% of cases
- Nerve conduction velocity is reduces in 60% and it can be patchy
- Distal motor latency increases and it can reach 3 times the normal value.\
- F-Waves indicate proximal NCV slowing.
- NCV findings are normal in 15-20% of patients
- No abnormalities can be found for several weeks in nerve conduction studies.

FINDINGS REDUCING POSSIBILITY OF DIAGNOSIS

- When asymmetrical weakness is present
- Bladder and bowel symptoms which fail to resolve
- Severe bladder and bowel involvement at the initiation of disease
- Greater than 50 mononuclear cells /mm3 in CSF
- Well demarcated sensory level

EXCLUSION CRITERIA

Other causes of acute neuromuscular weakness like myasthenia gravis, botulism, poliomyelitis, toxic neuropathy. Abnormal CSF cytology suggesting carcinomatous invasion of the nerve roots.

LABORATORY FINDINGS

CSF findings which are distinctive and consisting of elevated protein which may reach up to 100-1000mg/dl without pleocytosis. CSF may be normal upto 48 hours; it is only by the end of first week when the protein levels are elevated. The cell count may reach upto 10-100 cells per microliter. Persistent CSF pleocytosis suggests alternate diagnosis like viral myelitis, unrecognized HIV infection, leukemia or lymphomas or even neurosarcoidosis. The number of cells reduces rapidly in a matter of 2-3 days. The CSF proteins are normal during the first few days of symptoms but begin to rise later and reaching a peak in 4-6 weeks. The increase in proteins reflects a widespread inflammatory disease of nerve roots, but higher values does not have any prognostic significance. The CSF proteins may remain normal in few cases of GBS all throughout the disease. It particularly occurs in patients with Fisher syndrome or other axonal forms of GBS, where the CSF proteins may be normal or only slightly elevated.

NERVE CONDUCTION STUDY ABNORMALITIES

The most frequent early electrodiagnostic feature of GBS are reduction in the amplitude of muscle action potentials, slowing of conduction velocity and conduction blocks. Prolongation of distal latencies and absent F waves which reflect involvement of proximal nerve roots reflecting focal demyelination. Delayed H-reflex is almost always present. Disordered conduction is the hallmark of the disease which may be present more prominently as the disease progresses but widespread axonal damage even early in the course of the disease always been a poor prognostic factor. Acute GBS patients shows gadolinium enhancement of the cauda equine roots on magnetic resonance imaging and it can also prove as a diagnostic factor in complicated cases.

METABOLIC ABNORMALITIES

Liver function abnormalities have been found to be abnormal in fewer than 10 percent of the patients which indicates a recent or ongoing viral hepatitis probably due to CMV or EBV infections. T wave and other ECG changes of minor degree are reported frequently but these are only temporary. The Erythrocyte sedimentation rate is normal and is elevated only when there is additional process of infection, neoplastic or autoimmune factor which can coexist with GBS like SLE.

Hyponatremia occurs in a small proportion of the cases after the first week especially in patients on mechanical ventilation and it is

usually attributable to the syndrome of inappropriate antidiuretic hormone secretion (SIADH) but the atrial natriuretic effect also contributes to the natriuretic effect. Hyponatremia was noted in upto 48% of cases in one case series. Pseudohyponatremia from the intravenous immunoglobulin administration have also been described in few studies. Pseudo hyponatremia usually occurs because of increase protein and lipid concentration during the measurement of sodium using ion selective electrodes. The indirect analytical methods usually exclude the lipids and proteins occupying the plasma sample and thereby produce a falsely low sodium levels and it can solved by using the direct ion selective electrodes. The intravenous immunoglobulins are liable to cause hyponatremia due the large amount of proteins it carries. But this cannot be the only reason as true hyponatremia has also been demonstrated using the direct ion selective electrodes. This true hyponatremia has been attribute to osmotic translocation of water from intracellular to extracellular space mediated by increase in intravascular osmolality due to sucrose based immunoglobulin prepartions. Diabetes insipidus is rare and it is usually unexplained. High rates of proteinuria and glomerulonephritis have been reported in a few studies and the cause is usually unknown.

Hypokalemia has also been reported in few cases of GBS when the diagnosis can be really perplexing as both can cause flaccid paralysis. Hypomagnesemia has also been reported in GBS and it can occur due to the process of plamapharesis which can remove magnesium from the plasma pool.

DIFFERENTIAL DIAGNOSIS OF GBS

GBS is the most common type of acute generalized polyneuropathy as well as the most frequently evolving and even potentially fatal form of polyradiculoneuropathy. It is the form of polyradiculoneuropathy which can bring the patient to the brink of death and even produce respiratory failure within days to weeks. But there are other neurologic mimickers which can even produce such degree of flaccid paralysis and respiratory failure.

The most important problem is that of spinal cord disease to differentiate from GBS using a definite sensory level and there is marked bladder and bowel involvement. The spinal cord disease can be usually multifactorial and the usual preceding factors can be that of trauma or

even long standing lower back ache. The preceding viral infection in transverse myelitis should be kept in mind as even GBS has the same preceding cause but the major sphincter involvement and the disease can be proven by MR imaging which can be associated with significant demyelination. There can be certain confounding factors like loss of tendon reflexes which can occur in the initial stages of the spinal cord disease due to spinal shock.

Necrotizing myelopathy which is quite common in the tropical regions can even mimic GBS which can be associated with absent tendon reflexes due to extensive destruction of the grey matter of the spinal cord. Still more confusing factor is the early and transient urinary retention which can occur in smaller portion of GBS patients, but bladder and bowel involvement is not so extensive as in spinal cord diseases which helps to differentiate between the two.

Another common spinal cord disease which can be confused with GBS include cervical myelopathy. In GBS there is generalized paralysis with facial and respiratory muscle involvement. The fingertips are

paresthetic once the sensory system is involved and ascended to the calf level. Sensory loss involving the periphery and the trunk occurs rarely in early course of the disease but the reflexes are lost invariably in all limbs in comparison to cervical myelopathy which can be brisk.

Poliomyelitis is another differential diagnosis which can be considered and compared with GBS as there is predominant motor involvement in either cases. Other viral muscular paralysis which occurs in the western world due to the west nile virus and the enterovirus are notable examples other than polio. These infections are viral and are usually accompanied with significant findings of fever, meningo-encephalitis, early and significant pleocytosis in the spinal fluid and motor paralysis particularly with rather asymmetric involvement

Carcinomatous meningitis is another entity should be considered which is characterized by sub acute and symmetric involvement but more importantly involves the distal musculature and producing distal weakness more than proximal weakness. Comparing the proximal and distal group of muscles there is usually irregular involvement and the

facial muscles are usually spared in comparison with Guillaine Barre syndrome. The limb involvement is also not simultaneous and symptoms occur in a one limb following the other and is due to the neoplastic infiltration of nerve roots. Sciatica may be an accompanied finding in either cases but radicular pain is unusual in GBS. Spinal fluid analysis for the carcinomatous cells solves the dispute and is diagnostic for carcinomatous meningitis.

Another interesting differential diagnosis for generalized GBS with ophthalmoparesis or Fisher variant form of GBS is Basilar artery thrombosis. The differentiating features include brisk reflexes with Babinski sign being positive in the case of basilar artery thrombosis in comparison with GBS which is characterized by reactive pupils, areflexia and F wave abnormalities in the latter.

Acute myasthenia gravis is another important differential diagnosis for GBS with ophthalmoparesis or the Fisher variant which is characterized by ophthalmoplegia where as myasthenia gravis include ptosis, ophthalmoplegia , bulbar paralysis and respiratory muscle

paralysis. The important feature to differentiate is absence of sensory symptoms as wells as the tendon reflexes which are impaired. The mandibular muscles are very rarely involved in GBS in comparision with myasthenis gravis where the excercised jaw hangs open. Neostigmine improves the condition of myasthenia gravis whereas it has no role in GBS.

Botulism may also simulate GBS but there is usually bradycardia which is unusual for GBS. Tick paralysis, which affects the pediatric population of the western world but can affect both the young and old in the eastern part of the world is nearly impossible to differentiate from GBS unless one find the tick. Both share ascending paralysis and sometimes can even produce ataxia and ophthalmoplegia but sensory loss is not a feature of tick paralysis and the CSF protein invariably remains the same.

Few cases of shellfish or reef fish consumption contaminated with saxitoxin, ciguatoxin, or even tetrodotoxin which occurs in neurotoxic shellfish poisoning can produce acute facial-brachial paresthesias,

weakness, tachypnea and even iridoplegia which may last for a few days and can mimic GBS with generalized weakness or the Fisher variant.

Neuromuscular disorders in critically ill patients with systemic comorbid medical condition makes it difficult to distinguish it from GBS. Some of the following includes polyneuropathy in the critically ill, metabolic causes like the accelerated neuropathy of renal failure which is seen predominantly in the diabetic population who receive peritoneal dialysis or acute hypophosphatemia induced by hyperalimentaion can also mimic GBS. Drug induced polymyopathy due to drugs like steroids and the prolonged effects of neuromuscular blocking agents which may get accumulated in certain conditions like renal failure and acidosis can also simulate GBS. episodic porphyria are painful polyradiculopathy associated with paralysis.

CRITICAL ILLNESS POLYNEUROPATHY

It is an acute or sub acute symmetrical polyneuropathy which occurs in critically ill and septic patients especially in patients with multiorgan failure. It complicates weaning in mechanically ventilated patients even when the multiorgan failure is brought under control. The similarities with GBS include predominant motor paralysis and can include few electrophysiological abnormalities like conduction blocks and can even result in respiratory failure. Sensory symptoms can occur in various degrees but is usually mild. But cranial nerve involvement is rarer as is the autonomic dysfunction which seldom occurs. The EMG findings shows early denervation and normal CSF distinguish it from the demyelination associated with GBS. There are no inflammatory nerve changes. But the axonal form of GBS and critical illness neuropathy bears close resemblance and the confounding factors of drugs and nutritional deficiencies are attributable to the critical illness neuropathy. Many mediators of inflammation particularly the tumor necrosis factor has been implicated in the causation of critical illness neuropathy. Another close differential diagnosis is acute quadriplegic myopathy

which can occur in the critically ill. It has been attributed due to high dose of steroids and neuromuscular blocking agents but it can be differentiated using serum creatine kinase which may reach concentration up to several thousand units and the electro diagnostic studies reveal a unique pattern producing degeneration of myofilaments in all the muscles.

ACUTE UREMIC POLYNEUROPATHY

The well-known chronic sensory neuropathy associated with chronic kidney disease can sometimes mimic GBS by its accelerated process culminating in sub-acute and acute weakness of muscles. But it occurs predominantly in diabetic patients with end stage renal failure and who has been treated with peritoneal dialysis. In some ways the illness simulates GBS like generalized weakness and paresthesia predominantly distal. Change to hemodialysis produces little effect but renal transplantation is curative. The electrophysiological pattern mimics GBS producing a demyelinating pattern but there are no conduction blocks. The CSF proteins are also elevated. Few case series have reported

features both clinical and electrophysiological quiet indistinguishable from GBS and even some produce rapid clinical response to plasma exchange or gamma globulin infusion.

DIPHTHERITIC POLYNEUROPATHY

Corynebacterium diphtheria produces an exotoxin paralyses pharyngeal and laryngeal muscles within 1-2 weeks after the onset of infection and produces blurring of vision due to paralysis of accommodation. It produces a polyneuropathy beginning 5 to 8 weeks, acute or subacute limb weakness with paresthesias and distal loss of vibratory and position sense. The weakness involves the extremities predominately. After a few days the patient becomes bedridden and it can even produce respiratory muscle paralysis. The CSF protein is usually elevated 50-200 mg/dl. The pharyngeal infections are associated with cardiomyopathy and severe polyneuropathy with respiratory paralysis. It usually produces segmental demyelination without inflammatory changes involving the sensory roots and sensory ganglia. Treatment is with Diphtheria antitoxin given within 48h of onset of infection but is of little

value when polyneuropathy develops. The prognosis is usually excellent when respiratory paralysis is overcome.

PORPHYRIC POLYNEUROPATHY

It is a severe rapidly advancing symmetrical and motor polyneuropathy often with abdominal pain and psychosis and convulsions. It is inherited as an autosomal dominant trait. The metabolic defect is present in the liver and is due to the increased production and increased urinary excretion of delta amino levulinic acid. The peripheral and the central nervous system is affected usually in the variegate type of porphyria and is associated with marked skin sensitivity to light and trauma. The first study was made by Waldenstrom in 1937. The classic symptom is moderate to severe colicky pain of the abdomen. Frequently associated with constipation and ileus and vomiting. The disease is characterized by recurrent attacks and precipitated by sulfonamides, griseofulvin, estrogen, barbiturates, phenytoin and succinimide anticonvulsants. These drugs should be avoided in patients with porphyria and seizure disorder. The neurologic manifestation typically

involves motor nerves more severely than the sensory nerves. The symptoms usually involve the feet and ascend upward. The proximal muscles are more often involved with loss of knee jerk with preservation of ankle jerk. Facial palsy, dysphagia and ocular palsy occurs in severe cases. The symptoms vary from mild to severe fatal respiratory paralysis or cardiac paralysis. Confusion, delirium, visual defects and convulsion occur and can progress to severe neuropathy. Autonomic symptoms like tachycardia and hypertension are present in the acute phase of the disease. The prognosis is usually excellent. To summarize it is characterized by relapsing nature, acute onset, and abdominal pain, psychotic symptoms with predominant motor neuropathy often with bibrachial distribution of weakness, truncal sensory loss and tachycardia.

The pathologic finding is insignificant ,rarely associated with demyelination. Diagnosis is confirmed by demonstration of large amounts of porphobilinogen and delta aminolevulinic acid in the urine. The urine turns dark when standing due to formation of porphobilin. Treatment involves intravenous glucose and intravenous hematin. Other aspects of

treatment of respiratory support, use of beta blocking agent if tachycardia and hypertension are severe and intravenous glucose suppress the heme biosynthetic pathway and pyridoxine is also helpful. Prevention is the most important strategy before the attack can be precipitated.

VASCULITIC POLYNEUROPATHIES

Associated with systemic lupus erythematosus, polyarteritis nodosa and related disorders develop rapidly and is difficult to differentiating is helpful to differentiate between the two disorders from GBS[101]. Careful clinical and electrophysiological testing is needed to differentiate between the two. In one study three patients with polyarteritis nodosa and one with Churg-Strauss became completely paralysed within a week and one died of intestinal perforation. However most cases evolve rapidly and are usually asymmetrical and multifocal distribution. Even few patients with alcoholism, occult carcinoma, Hodgkin disease or renal transplantation who developed an acute polyneuropathy as rapid in its evolution as GBS has also been described in Refsum disease.

TREATMENT OF GBS

GENERAL MEDICAL ASPECTS

Respiratory assistance and careful nursing form the corner stone of the treatment as the disease remits naturally and the outlook looks favorable in most of the cases[98]. About one quarter of the patients

requires mechanical ventilation in recent era. As the patient's condition fluctuated widely and unpredictably and more over rapidly in the first few days of the illness virtually all cases should be admitted in the hospital for close monitory of respiratory, autonomic and motor dysfunction.

Maximal respiratory force and expiratory vital capacity is enough for the bedside assessment of diaphragmatic strength and respiratory function. These measurements help to assess the likelihood of respiratory failure. The strength of the neck muscles and trapezii which share the same segmental innervation as the diaphragm helps to assess the diaphragm function. Single breath count can be used to assess the respiratory function. The ability to reach 20 generally corresponds to a vital capacity of greater than 1.5 L. If a downward trend in these measurements is observed then endotracheal intubation and mechanical ventilation should be considered. Incipient respiratory failure should be suspected even when there is minor degree of tachypnea and when the partial pressure of PaO2 falls below 85 reflecting pulmonary atelectasis.

Those patients with oropharyngeal weakness required endotracheal intubation as early as possible but mechanical ventilation is not always required in these kind of cases. These treatments are so demanding that these kind of patients should be admitted in intensive care units with extensive monitoring by the personal skilled physicians[99].

The other major aspects of the treatment in severely debilitated patients are the management of cardiac autonomic instability and prevention of the medical problems that arise in the critically ill patients. Hypotension can occur in 10 percent of the patients and is treated with vasopressor and intravenous fluid infusion for a short period of time. Hypertension is usually managed with short acting beta blockers like labetalol, but care should be taken since precipitous fall in blood pressure can be dangerous enough.

Prevention of electrolyte imbalance, gastrointestinal hemorrhage and pulmonary embolism in patients who are bedbound by the use of subcutaneous heparin or pneumatic compression boots require careful

monitoring. Adynamic ileus is a problem which can occur and can even lead to perforation in few cases.

Hyponatremia is another common entitity encountered and is usually due to SIADH, and the drop in serum sodium can be produces or exaggerated by positive pressure mechanical ventilation. Fluid restriction in the case of SIADH or salt supplemention in the case of sodium loss.

Waking dreams or hallucinations can occur after weeks of immobilization.

Failure to effectively clear the tracheobronchial secretions and the need for prolonged ventilation are the usual indications for tracheostomy. In most cases this can be postponed until the third week of intubation. However rapidly quadriplegic patients and ventilator dependent patients may require tracheostomy earlier. Once tracheostomy is performed care should be given for adequate tracheal toileting and pulmonary and urinary tract infections with adequate antibiotics[101].

The decisions to wean and discontinue respiratory aid are based on the degree and timing of recovery of respiratory function. The weaning process generally begins when the vital capacity reaches 10 ml/kg or when comfortable breathing is sustained for a few minutes. Physiotherapy should be done to prevent pressure palsies and pressure sores.

PLASMA EXCHANGE AND IMMUNOGLOBULIN

The specific treatment includes plasma exchange and intravenous immunoglobulin. (IVIG) [101-104]. If the patient becomes unable to walk unaided or if he shows a significant reduction in vital capacity or severe oropharyngeal weakness plasmapharesis or IVIG[105] is instituted as promptly as possible. Three large randomized trials comprising more than 500 patients have established the usefulness of plasma exchange administered during the evolving phase of GBS. In the patients who are treated within 2 weeks of the onset of symptoms, the hospitalization time is halved, mechanical ventilation time is also halved and is the time required to achieve ambulation[104]. However if the plasma exchange is delayed more than two weeks then the procedure is of little value.

However if the patient still continues to proceed in the third or fourth week of illness then the treatment can be instituted. Young patients respond well to plasmapharesis and the preserved compound muscle action potential have better prognosis than decreased CMAP. The advised regimen is to remove 200-250 ml/kg of plasma in 4-6 treatments on alternative days or even in shorter days if there is no [coagulopathy106].

The replacement fluid is saline combined with 5% albumin. The need for larger bore needle usually necessitates the implantation of a double lumen subclavian or internal jugular catheters and this may lead to complications such as pneumothorax, infection and hemorrhage. In some cases the treatment can be completed with the antecubital vein. Hypotension, hypoprothrombinemia and cardiac arrhythmias can occur as complication to plasma exchange. Hepatitis and HIV can be avoided if the plasma is replaced with albumin and normal saline rather with pooled plasma.

IVIG is also effective with the dosage of 0.4g/kg per day for 5 consecutive days and is both easier and safer. In one large study plasma

exchange was compared with IVIG and there was better result in patients treated with plasma exchange but the difference failed to reach any statistical significance. Renal failure, proteinuria and aseptic meningitis manifested by headache are the complication of IVIG. The gamma globulin can cause anaphylaxis and deep venous thrombosis.

5-10 percent of the patients treated with either plasmapharesis or IVIG enter into relapse and becomes apparent several days to 3 weeks after treatment. If there was good response to initial therapy then the same treatment can be given for the successive relapse. The clinical response that occurs in patients treated with IVIG or PE cannot be readily discerned in an individual patient.

The value of corticosteroids alone in the treatment of GBS has been disputed. However none of the randomized controlled studies have shown the beneficial effect of corticosteroid. Although corticosteroid can no longer be recommended as routine treatment for acute GBS few instances have shown that intravenous high doses of corticosteroids seemingly halted the progress of acute disease[106].

PROGNOSIS

3-5 percent of the patients do not survive the disease even in the best of the hospitals. Death is most often due to cardiac arrest, perhaps related to dysautonomia, adult respiratory distress syndrome, pneumo or hemothorax or accidental machine failure[95].

Pulmonary embolism or even sepsis due to prolonged immobilization can occur but respiratory failure remains as the main cause of death.

The majority of the patients nearly recover completely with mild motor deficits or sensory complaints in the feet or legs. In about 10 percent of the patients residual disability is more. This occurs in most severe and rapidly evolving cased with widespread axonal damage and in those requiring prolonged and early ventilation[92].

Widespread denervation in EMG associated with decreased compound muscle action potential less than 10 percent was associated with more severe damage. The average period for ventilator has been

around 20 days. The older patients recovered more slowly than the younger patient.

The most common residual defects are weakness of the lower leg muscles, numbness of toes and feet, and mild facial weakness. Few patients had sensory ataxia. Distal neuropathic pain and persistent autonomic problems can occur but infrequently. Fatigue, asthenia, muscle cramps, dizziness, pain and breathlessness can also occur. Depression is an important associated symptom[98].

The speed of recovery varies but the pace is steady and can occur within a few weeks to months. If the axons are degenerated it can take up to 6-18 months to recover.

5-10 percent of the patients have recurrences. An illness that can occur as acute demyelinating polyneuropathy may fail to stabilize and continue to progress steadily or even may have incomplete remission and can progress to develop chronic inflammatory neuropathy. Many grading

systems have been developed to study the disability that is associated with GBS. The scoring systems which has been used to monitor the recovery of the patients is the disability scoring system by Hughes which comprises of the following parameters and grading.

HUGHES DISABILITY SCORING[102]

Grade 0	Normal functional state
Grade 1	Able to run with minor signs and symptoms
Grade 2	Able to walk 5 m independently
Grade 3	Able to walk 5 m with aid
Grade 4	Bed- or chair-bound
Grade 5	Requires assisted ventilation

MATERIAL AND METHODS

SETTING:

This study was conducted at the Institute of Internal Medicine, IMCU, Rajiv Gandhi Government General Hospital and Madras Medical College.

ETHICAL COMMITTEE APPROVAL:

Obtained.

STUDY DURATION:

This study was conducted over a period of six months.

STUDY POPULATION:

Patients admitted with Guillain Barre Syndrome in medical wards, IMCU, Institute of Internal medicine.

SAMPLE SIZE:

Fifty cases of Guillain Barre Syndrome

TYPE OF STUDY:

Cross sectional study-Prospective and Retrospective.

INCLUSION CRITERIA

- Acute inflammatory demyelinating polyneuropathy.

EXCLUSION CRITERIA:

- Hypokalemic periodic paralysis.
- Diphtheritic paralysis.
- Traumatic Quadriparesis.
- Paraneoplastic neuropathy.
- Vasculitic polyneuropathy.
- Botulism.
- Vascular occlusion of spinal artery.

- Degenerative quadriparesis.
- Acute transverse myelitis
- Infectious paraspinal abscess and fractures.
- Acute disseminated encephalomyelitis.
- Porphyria induced polyneuropathy.

DATA COLLECTION AND METHODS

Informed consent was obtained from each patient or the relative.

Patients had their history taken according to a Questionnaire and were subjected to clinical examination.

Patients were subjected to routine blood investigations [renal function tests (including electrolytes), liver function tests], Electrocardiography, complete blood count, chest xray, HIV and HbsAg ELISA.

Cerebrospinal fluid analysis done including CSF sugar, protein, LDH, cell count, cytology and cultures.

Nerve conduction study analysis was done and the conduction velocity, latency, blocks, F waves, compound muscle action potentials were calculated.Patients were classified based on their disability at 15 days and at discharge using Hughes Disability scoring scale.

Various parameters like age, sex, month of admission, preceding events, predominant symptoms, cranial nerve involvement, dysautonomia, requirement of mechanical ventilation, electrodiagnostic patterns, decreased Compound muscle action potential less than 10 percent, CSF proteins, presence of albuminocytological dissociation , disability grading was done and was correlated with outcome of the disease depending on whether there is complete recovery of the disease , residual deficit or death.

All the data were entered in the proforma (enclosed)Data were analyzed using SPSS package and ANOVA.

OBSERVATION AND RESULTS

TABLE 1 AGE DISTRIBUTION

		Frequency	Percent
Valid	21-30	19	38.0
	31-40	14	28.0
	41-50	10	20.0
	51-60	4	8.0
	61-70	3	6.0
	Total	50	100.0

TABLE-2 SEX DISTRIBUTION

		Frequency	Percent
Valid	Male	33	66.0
	Female	17	34.0
	Total	50	100.0

TABLE-3 ADMISSION MONTH

		Frequency	Percent
Valid	June	14	28.0
	July	18	36.0
	August	15	30.0
	September	3	6.0
	Total	50	100.0

TABLE-4 PROCEEDING EVENT

	Frequency	Percent

Valid	RTI	12	24.0
	Age	10	20.0
	No	28	56.0
	Total	50	100.0

TABLE-6 PRESENTING SYMPTOM

		Frequency	Percent
Valid	LL weakness	30	60.0
	Sensory symptoms	15	30.0
	UL weakness	5	10.0
	Total	50	100.0

TABLE-7 SENSORY SYMPTOM

		Frequency	Percent
Valid	LL weakness	30	60.0

	Sensory symptoms	15	30.0
	UL weakness	5	10.0
	Total	50	100.0

TABLE-8 CRANIAL NERVE INVOLVEMENT

		Frequency	Percent
Valid	Yes	21	42.0
	No	29	58.0
	Total	50	100.0

TABLE-9 DYSAUTONOMIA

		Frequency	Percent
Valid	Yes	18	36.0
	No	32	64.0
	Total	50	100.0

TABLE-10 MECHANICAL VENTILATION

		Frequency	Percent
Valid	Yes	12	24.0

	No	38	76.0
	Total	50	100.0

TABLE-11 ALBUMINOCYTOLOGICAL DISSOCIATION

		Frequency	Percent
Valid	Yes	39	78.0
	No	11	22.0
	Total	50	100.0

TABLE-12 ELECTRODIAGNOSTIC VARIANT

		Frequency	Percent
Valid	Normal	5	10.0
	Demyelinative	30	60.0
	AMAN	10	20.0
	AMSAN	5	10.0
	Total	50	100.0

TABLE-13 DECREASED CMAP<10% OF LOWERLIMIT

		Frequency	Percent
Valid	Yes	9	18.0

	No	41	82.0
	Total	50	100.0

TABLE-14 TREATMENT GIVEN

		Frequency	Percent
Valid	Plasmapharesis	19	38.0
	IVIg	31	62.0
	Total	50	100.0

TABLE-15 AVERAGE LATENCY TO START TREATMENT

		Frequency	Percent
Valid	3	33	66.0
	4	16	32.0
	5	1	2.0
	Total	50	100.0

TABLE-16 OUTCOME

		Frequency	Percent
Valid	3	33	66.0
	4	16	32.0
	5	1	2.0
	Total	50	100.0

TABLE-17 HUGHES DISABILITY SCORING AT 15 DAYS

		FREQUENCY	PERCENT
VALID			
	GRADE 0	-	-
	GRADE 1	-	-
	GRADE 2	4	8.0
	GRADE 3	23	46.0
	GRADE 4	11	22.0
	GRADE 5	12	24.0
	TOTAL	50	100.0

AT DISCHARGE/DEATH

		Frequency	Percent
Valid	Grade 0	29	58.0
	Grade 1	5	10.0
	Grade 2	6	12.0
	Grade 3	5	10.0
	Grade 5	5	10.0
	Total	50	100.0

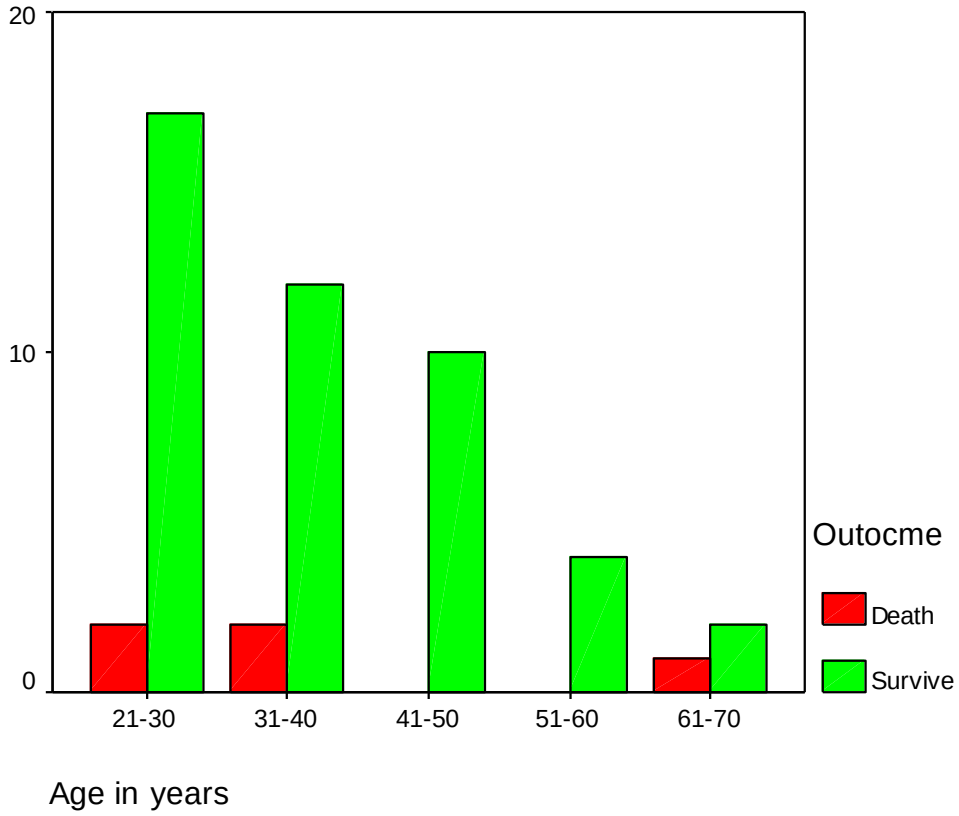
TABLE-18 RECOVERY

		Frequency	Percent
Valid	Complete Recovery	27	54.0
	Residual Deficit	17	34.0
	Total	45	90.0
Total		50	100.0

COMPARISON BETWEEN THE ABOVE PARAMETERS AND OUTCOME OF THE DISEASE

TABLE-19 AGE DISTRIBUTION AND OUTCOME(DEATH)

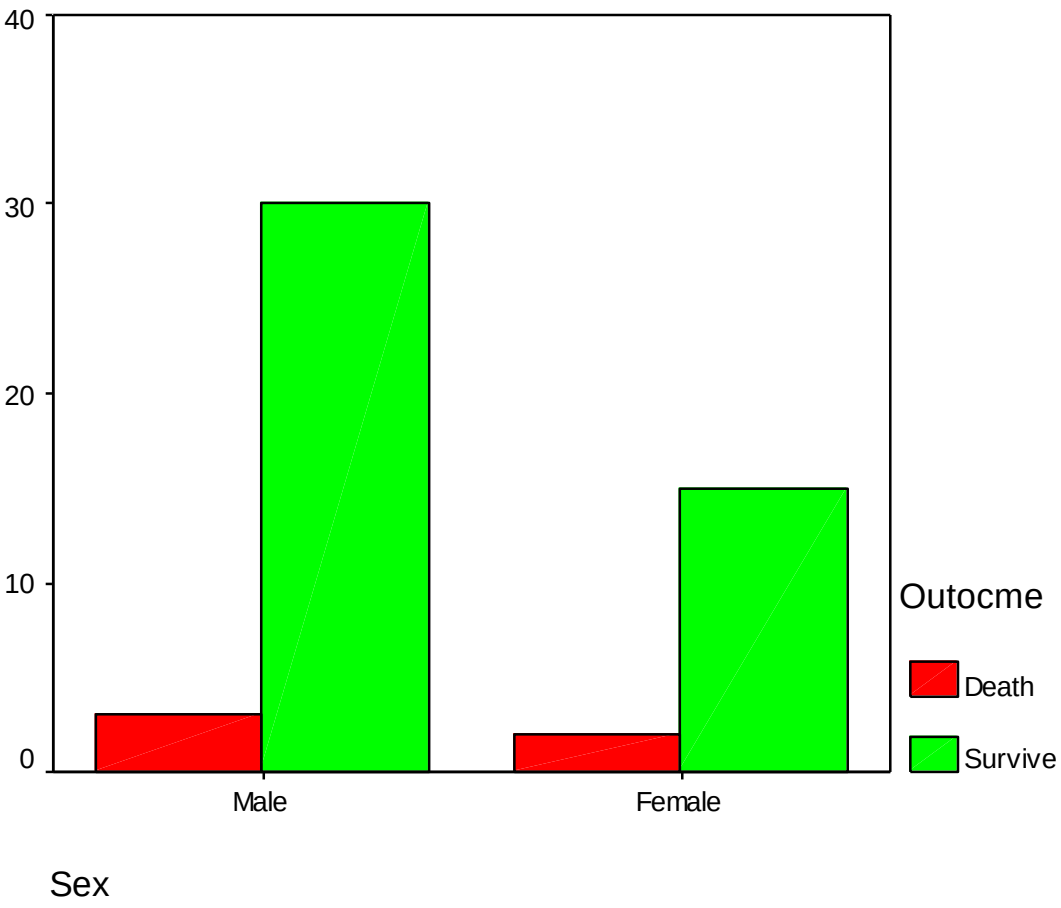
			Outcome		Total	P value
			Death	Survive		
Age in years	21-30	Count	2	17	19	0.454
		% within Age in years	10.5%	89.5%	100.0%	
		% within Outcome	40.0%	37.8%	38.0%	
	31-40	Count	2	12	14	
		% within Age in years	14.3%	85.7%	100.0%	
		% within Outcome	40.0%	26.7%	28.0%	
	41-50	Count	0	10	10	
		% within Age in years	.0%	100.0%	100.0%	
		% within Outcome	.0%	22.2%	20.0%	
	51-60	Count	0	4	4	
		% within Age in years	.0%	100.0%	100.0%	
		% within Outcome	.0%	8.9%	8.0%	
	61-70	Count	1	2	3	
		% within Age in years	33.3%	66.7%	100.0%	
		% within Outcome	20.0%	4.4%	6.0%	
Total		Count	5	45	50	
		% within Age in years	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	



In our study group, the relationship between age and outcome (death) is insignificant with the **p value of 0.454**

TABLE-20 SEX DISTRIBUTION AND MORTALITY

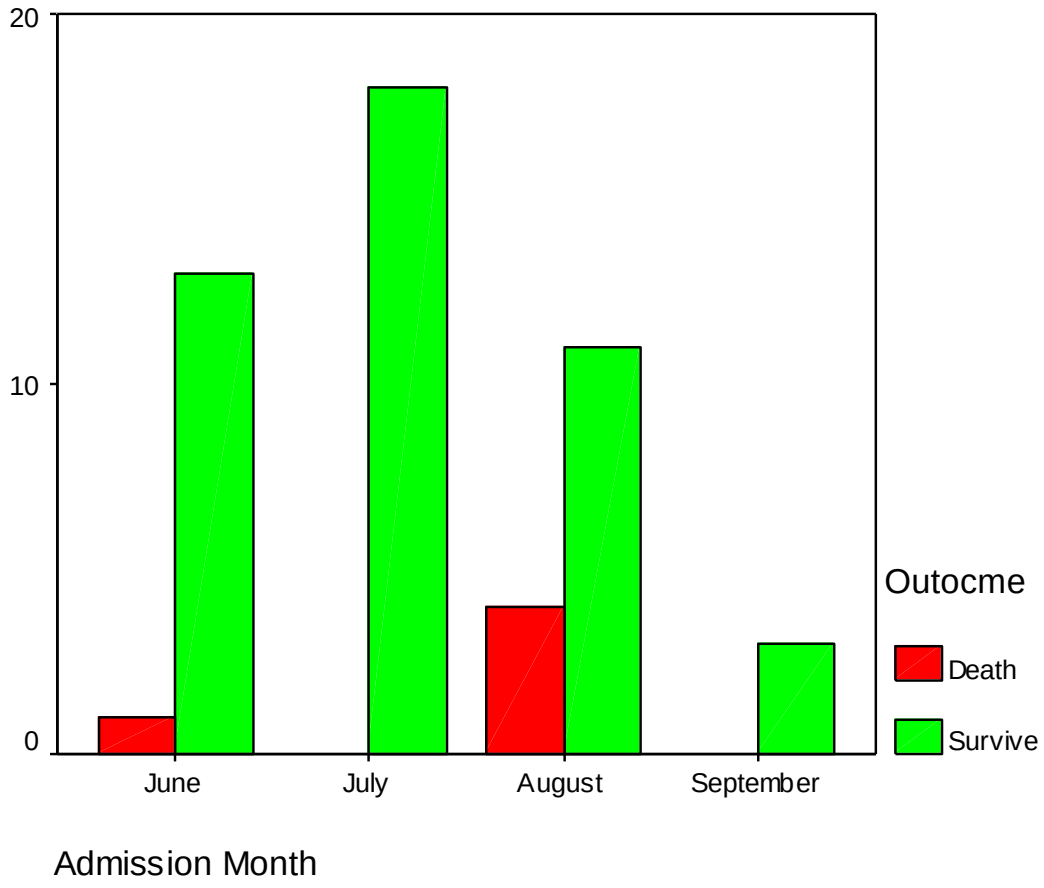
			Outcome		Total	P value
			Death	Survive		
Sex	Male	Count	3	30	33	
		% within Sex	9.1%	90.9%	100.0%	
		% within Outcome	60.0%	66.7%	66.0%	
	Female	Count	2	15	17	
		% within Sex	11.8%	88.2%	100.0%	0.560
		% within Outcome	40.0%	33.3%	34.0%	
Total		Count	5	45	50	
		% within Sex	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	



The relationship between sex and outcome (death) is insignificant with the **p value of 0.564**

TABLE-21 ADMISSION MONTH AND MORTALITY

			Outcome		Total	P value
			Death	Survive		
Admission Month	June	Count	1	13	14	0.069
		% within Admission Month	7.1%	92.9%	100.0%	
		% within Outcome	20.0%	28.9%	28.0%	
	July	Count	0	18	18	
		% within Admission Month	.0%	100.0%	100.0%	
		% within Outcome	.0%	40.0%	36.0%	
	August	Count	4	11	15	
		% within Admission Month	26.7%	73.3%	100.0%	
		% within Outcome	80.0%	24.4%	30.0%	
	September	Count	0	3	3	
		% within Admission Month	.0%	100.0%	100.0%	
		% within Outcome	.0%	6.7%	6.0%	
Total		Count	5	45	50	
		% within Admission Month	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	



Admission month had no significance on the mortality of the patients **p value=0.069**

TABLE 22-PRECEDING EVENT AND MORTALITY

			Outcome		Total	P value
			Death	Survive		
Preceding Event	RTI	Count	4	8	12	0.006
		% within Preceding Event	33.3%	66.7%	100.0%	
		% within outcome	80.0%	17.8%	24.0%	
	AGE	Count	1	9	10	
		% within Preceding Event	10.0%	90.0%	100.0%	
		% within outcome	20.0%	20.0%	20.0%	
	No	Count	0	28	28	
		% within Preceding Event	.0%	100.0%	100.0%	
		% within outcome	.0%	62.2%	56.0%	
Total		Count	5	45	50	
		% within Preceding Event	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	

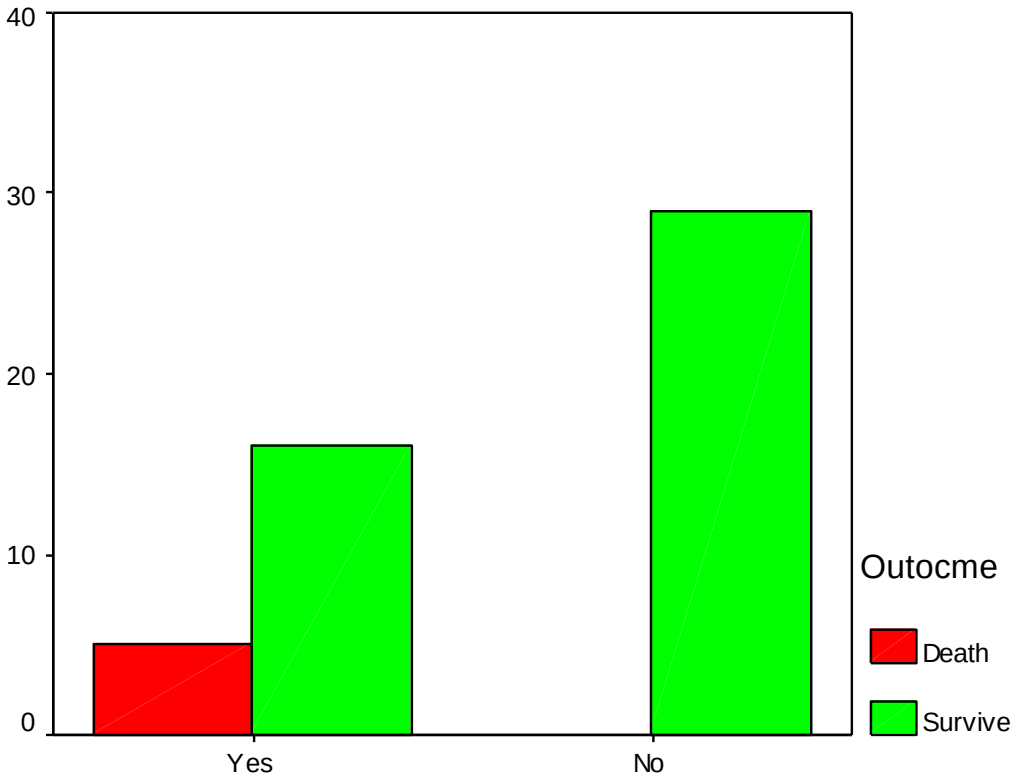
TABLE-23 PRESENTING SYMPTOM AND MORTALITY

			Outcome		Total	P value
			Death	Survive		
Presenting Symptom	LL weakness	Count	5	25	30	0.157
		% within Presenting Symptom	16.7%	83.3%	100.0%	
		% within outcome	100.0%	55.6%	60.0%	
	Sensory symptoms	Count	0	15	15	
		% within Presenting Symptom	.0%	100.0%	100.0%	
		% within outcome	.0%	33.3%	30.0%	
	UL weakness	Count	0	5	5	
		% within Presenting Symptom	.0%	100.0%	100.0%	
		% within outcome	.0%	11.1%	10.0%	
Total		Count	5	45	50	
		% within Presenting Symptom	10.0%	90.0%	100.0%	
		% within outcome	100.0%	100.0%	100.0%	

P value =0.157

TABLE-25 CRANIAL NERVE INVOLVEMENT AND MORTALITY

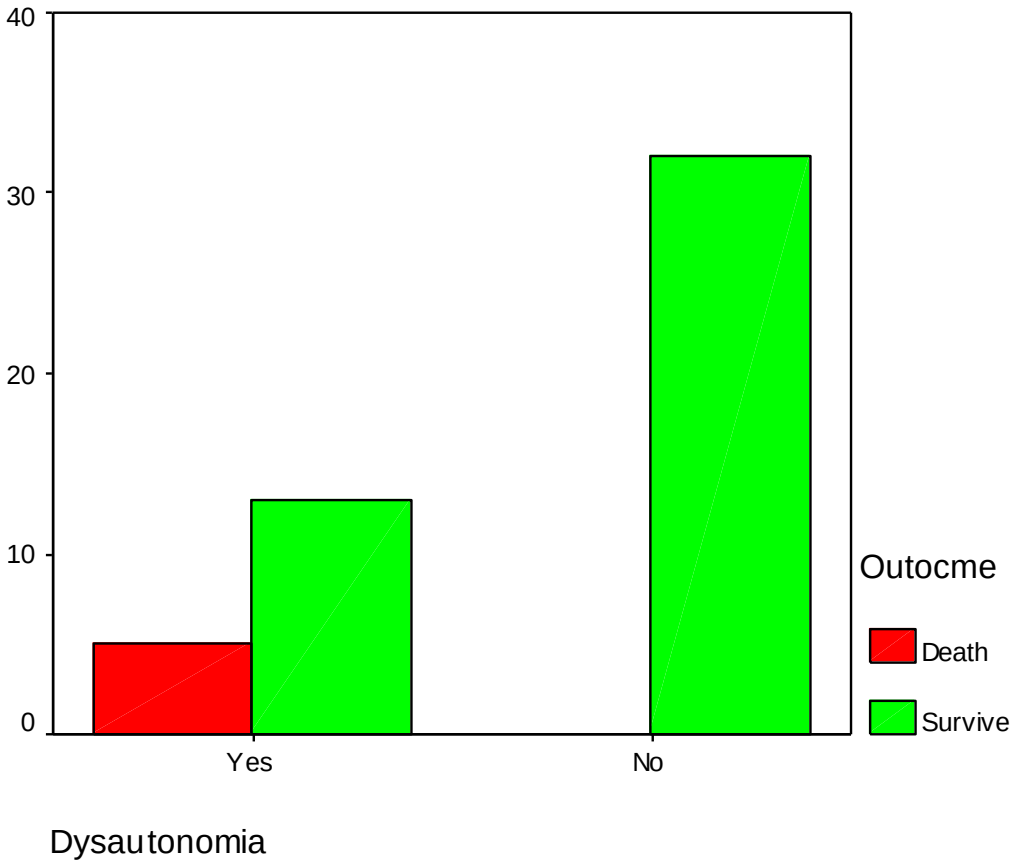
			outcome		Total	P value
			Death	Survive		
Cranial Nerve Involvement	Yes	Count	5	16	21	0.010
		% within Cranial Nerve Involvement	23.8%	76.2%	100.0%	
		% within outcome	100.0%	35.6%	42.0%	
	No	Count	0	29	29	
		% within Cranial Nerve Involvement	.0%	100.0%	100.0%	
		% within outcome	.0%	64.4%	58.0%	
Total		Count	5	45	50	
		% within Cranial Nerve Involvement	10.0%	90.0%	100.0%	
		% within	100.0%	100.0%	100.0%	



Cranial nerve involvement was found to have significant relationship with outcome- death of the patient. **p value 0.010-** highly significant

TABLE-26 DYSAUTONOMIA AND MORTALITY

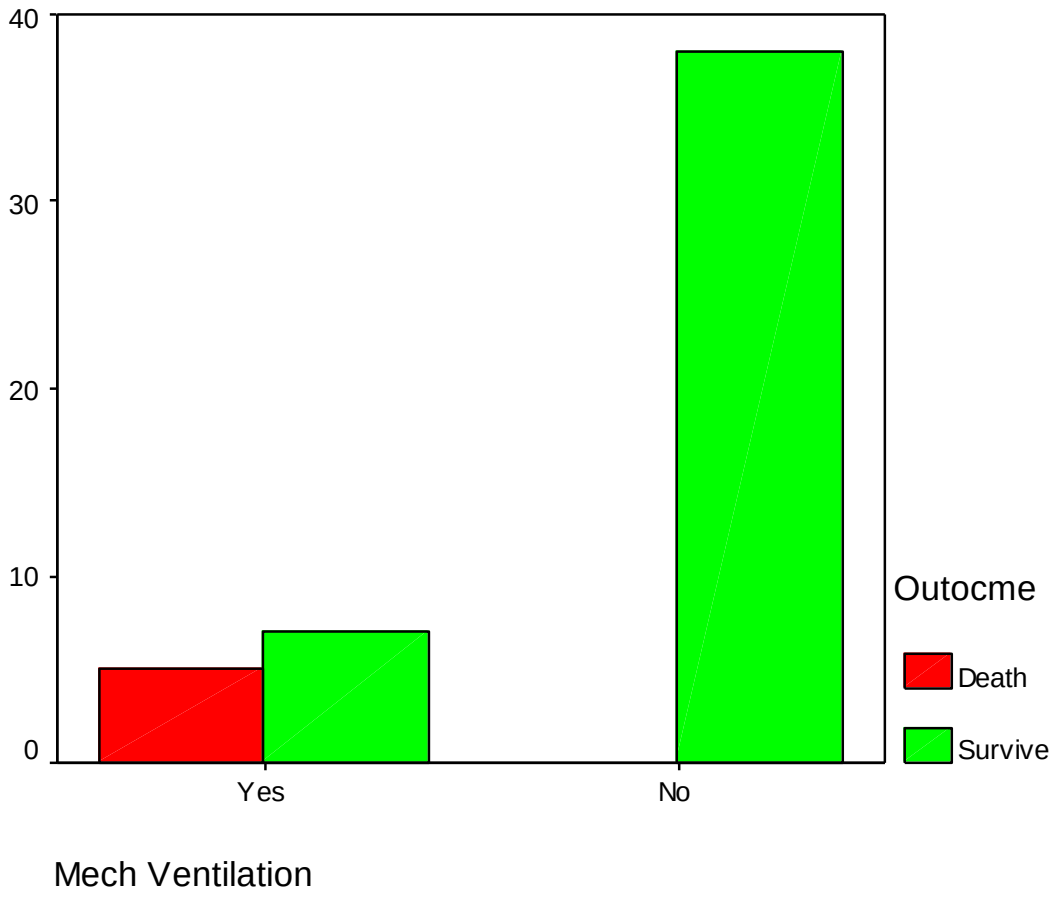
			Outcome		Total	P value
			Death	Survive		
Dysautonomia	Yes	Count	5	13	18	0.004
		% within Dysautonomia	27.8%	72.2%	100.0%	
		% within Outcome	100.0%	28.9%	36.0%	
	No	Count	0	32	32	
		% within Dysautonomia	.0%	100.0%	100.0%	
		% within Outcome	.0%	71.1%	64.0%	
Total		Count	5	45	50	
		% within Dysautonomia	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	



Dysautonomia and mortality had strong correlation with the **p value=0.004**

TABLE-27 MECHANICAL VENTILATION AND MORTALITY

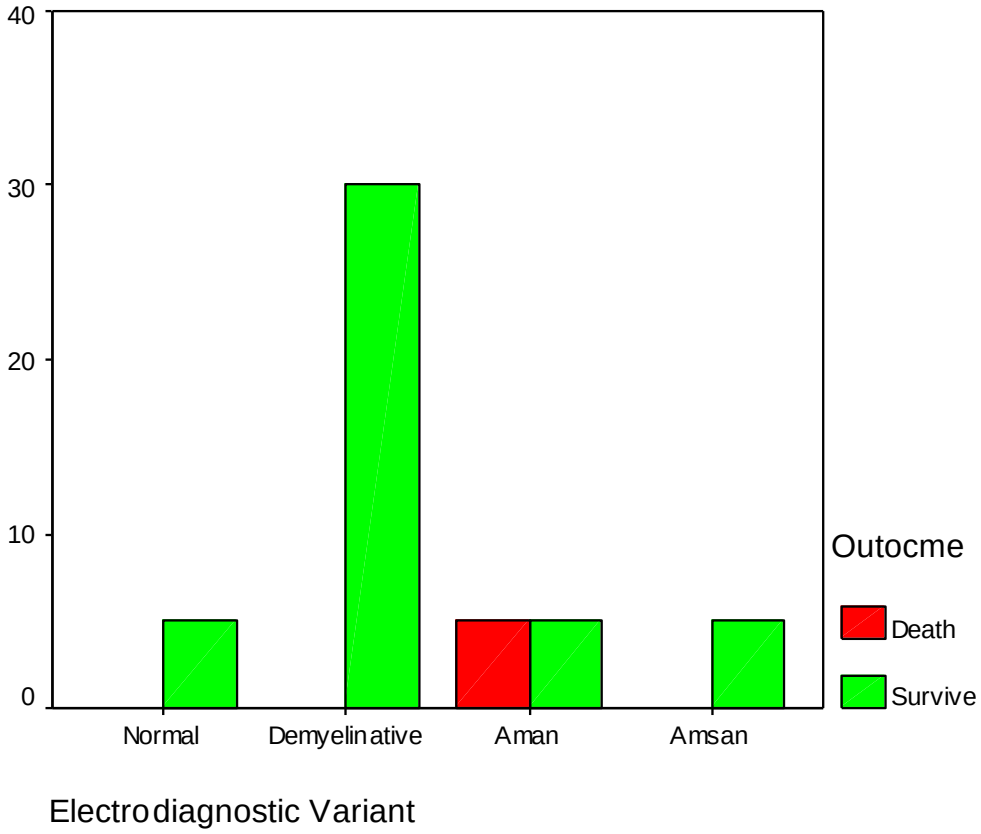
			Outcome		Total	P value
			Death	Survive		
Mechanical Ventilation	Yes	Count	5	7	12	0.000
		% within Mechanical Ventilation	41.7%	58.3%	100.0%	
		% within outcome	100.0%	15.6%	24.0%	
	No	Count	0	38	38	
		% within Mechanical Ventilation	.0%	100.0%	100.0%	
		% within Outcome	.0%	84.4%	76.0%	
Total		Count	5	45	50	
		% within Mechanical Ventilation	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	



Mechanical ventilation had a very high significance with the mortality of the disease with p value being highly significant of <0.001.

TABLE 30-ELECTRODIAGNOSTIC VARIANT AND MORTALITY

			Outocme		Total	P value
			Death	Survive		
Electro diagnostic Variant	Normal	Count	0	5	5	0.000
		% within Electrodiagnostic Variant	.0%	100.0%	100.0%	
		% within Outcome	.0%	11.1%	10.0%	
	Demyelinative	Count	0	30	30	
		% within Electro diagnostic Variant	.0%	100.0%	100.0%	
		% within Outcome	.0%	66.7%	60.0%	
	AMAN	Count	5	5	10	
		% within Electrodiagnostic Variant	50.0%	50.0%	100.0%	
		% within Outcome	100.0%	11.1%	20.0%	
	AMSAN	Count	0	5	5	
		% within Electrodiagnostic Variant	.0%	100.0%	100.0%	
		% within Outcome	.0%	11.1%	10.0%	
Total		Count	5	45	50	
		% within Electrodiagnostic Variant	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	



The electrodiagnostic variants of AMAN and AMSAN had significant relationship with outcome **p value=<0.001.**

TABLE-31 DECREASED CMAP<10% LOWERLIMIT AND MORTALITY

			Outcome		Total	P value
			Death	Survive		
Decreased CMAP<10% of Lowerlimit	Yes	Count	5	4	9	0.000
		% within Decreased CMAP<10% of Lowerlimit	55.6%	44.4%	100.0%	
		% within Outcome	100.0%	8.9%	18.0%	
	No	Count	0	41	41	
		% within Decreased CMAP <10% of Lowerlimit	.0%	100.0%	100.0%	
		% within Outcome	.0%	91.1%	82.0%	
Total		Count	5	45	50	
		% within Decreased CMAP <10% of Lowerlimit	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	

Decreased CMAP of <10% of lower limit had significant correlation with outcome with **p value=<0.001**

TABLE-32 TREATMENT AND MORTALITY

			Death	Survive		P value
Treatment given	Plasmapharesis	Count	5	14	19	
		% within Treatment given	26.3%	73.7%	100.0%	
		% within Outocme	100.0%	31.1%	38.0%	
	IVIg	Count	0	31	31	
		% within Treatment given	.0%	100.0%	100.0%	0.003
		% within Outocme	.0%	68.9%	62.0%	
Total		Count	5	45	50	
		% within Treatment given	10.0%	90.0%	100.0%	
		% within Outocme	100.0%	100.0%	100.0%	

P value 0.003

TABLE-34 HUGHES DISABILITY SCORE AT 15 DAYS AND MORTALITY

			Outcome		Total	P value
			Death	Survive		
Hughes Disabilty Grading Score at 15 Days	Grade 2	Count	0	4	4	
		% within Hughes Disability Grading Score at 15 Days	.0%	100.0%	100.0%	
		% within Outcome	.0%	8.9%	8.0%	
	Grade 3	Count	0	23	23	
		% within Hughes Disability Grading Score at 15 Days	.0%	100.0%	100.0%	
		% within Outcome	.0%	51.1%	46.0%	
	Grade 4	Count	0	11	11	
		% within Hughes Disability Grading Score at 15 Days	.0%	100.0%	100.0%	0.001
		% within Outcome	.0%	24.4%	22.0%	
	Grade 5	Count	5	7	12	
		% within Hughes Disability Grading Score at 15 Days	41.7%	58.3%	100.0%	
		% within Outcome	100.0%	15.6%	24.0%	
Total		Count	5	45	50	
		% within Hughes Disability Grading Score at 15 Days	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	

P value significant<0.001

TABLE-35 HUGHES DISABILITY SCORING AT DISCHARGE/DEATH

			Outcome		Total	P value
			Death	Survive		
At discharge/ death	Grade 0	Count	0	29	29	
		% within At Discharge /Death	.0%	100.0%	100.0%	
		% within Outcome	.0%	64.4%	58.0%	
	Grade 1	Count	0	5	5	
		% within At Discharge /Death	.0%	100.0%	100.0%	
		% within Outcome	.0%	11.1%	10.0%	
	Grade 2	Count	0	6	6	
		% within At Discharge Death	.0%	100.0%	100.0%	
		% within Outcome	.0%	13.3%	12.0%	
	Grade 3	Count	0	5	5	0.000
		% within At Discharge /Death	.0%	100.0%	100.0%	
		% within Outcome	.0%	11.1%	10.0%	
	Grade 5	Count	5	0	5	
		% within At Discharge /Death	100.0%	.0%	100.0%	
		% within Outcome	100.0%	.0%	10.0%	
Total		Count	5	45	50	
		% within At Discharge	10.0%	90.0%	100.0%	
		% within Outcome	100.0%	100.0%	100.0%	

P value is significant <0.001

TABLE-36- AGE DISTRIBUTION AND RECOVERY/DISABILITY

			Recovery		Total	P value
			Complete Recovery	Residual Deficit		
Age in years	21-30	Count	9	8	17	0.476
		% within Age in years	52.9%	47.1%	100.0%	
		% within Recovery	32.1%	47.1%	37.8%	
	31-40	Count	7	5	12	
		% within Age in years	58.3%	41.7%	100.0%	
		% within Recovery	25.0%	29.4%	26.7%	
	41-50	Count	8	2	10	
		% within Age in years	80.0%	20.0%	100.0%	
		% within Recovery	28.6%	11.8%	22.2%	
	51-60	Count	2	2	4	
		% within Age in years	50.0%	50.0%	100.0%	
		% within Recovery	7.1%	11.8%	8.9%	
	61-70	Count	2	0	2	
		% within Age in years	100.0%	.0%	100.0%	
		% within Recovery	7.1%	.0%	4.4%	
Total		Count	28	17	45	
		% within Age in years	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Age had no significance with degree of recovery **p value=0.476**

TABLE-37 SEX DISTRIBUTION AND RECOVERY/DISABILITY

			Recovery		Total	
			Complete Recovery	Residual Deficit		P value
Sex	Male	Count	22	8	30	0.033
		% within Sex	73.3%	26.7%	100.0%	
		% within Recovery	78.6%	47.1%	66.7%	
	Female	Count	6	9	15	
		% within Sex	40.0%	60.0%	100.0%	
		% within Recovery	21.4%	52.9%	33.3%	
Total		Count	28	17	45	
		% within Sex	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Sex and degree of recovery had grade 2 degree of significance with

p value=0.033

TABLE-38 ADMISSION MONTH AND RECOVERY/DISABILITY

			Recovery		Total	
			Complete Recovery	Residual Deficit		P value
Admission Month	June	Count	8	5	13	0.661
		% within Admission Month	61.5%	38.5%	100.0%	
		% within Recovery	28.6%	29.4%	28.9%	
	July	Count	11	7	18	
		% within Admission Month	61.1%	38.9%	100.0%	
		% within Recovery	39.3%	41.2%	40.0%	
	August	Count	8	3	11	
		% within Admission Month	72.7%	27.3%	100.0%	
		% within Recovery	28.6%	17.6%	24.4%	
	September	Count	1	2	3	
		% within Admission Month	33.3%	66.7%	100.0%	
		% within Recovery	3.6%	11.8%	6.7%	
Total		Count	28	17	45	
		% within Admission Month	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Admission month had no significance with degree or recovery

p value=0.661

TABLE 39-PRECEDING EVENT AND RECOVERY/DISABILITY

			Recovery		Total	
			Complete Recovery	Residual Deficit		P value
Preceding Event	RTI	Count	3	5	8	0.196
		% within Preceding Event	37.5%	62.5%	100.0%	
		% within Recovery	10.7%	29.4%	17.8%	
	Age	Count	5	4	9	
		% within Preceding Event	55.6%	44.4%	100.0%	
		% within Recovery	17.9%	23.5%	20.0%	
	No	Count	20	8	28	
		% within Preceding Event	71.4%	28.6%	100.0%	
		% within Recovery	71.4%	47.1%	62.2%	
Total		Count	28	17	45	
		% within Preceding Event	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Preceding event had no significance with degree of recovery with

p value =0.196

TABLE-40 PRESENTING SYMPTOM AND RECOVERY/DISABILITY

			Recovery		Total	P value
			Complete Recovery	Residual Deficit		
Presenting Symptom	LL weakness	Count	18	7	25	0.089
		% within Presenting Symptom	72.0%	28.0%	100.0%	
		% within Recovery	64.3%	41.2%	55.6%	
	Sensory symptoms	Count	6	9	15	
		% within Presenting Symptom	40.0%	60.0%	100.0%	
		% within Recovery	21.4%	52.9%	33.3%	
	UL weakness	Count	4	1	5	
		% within Presenting Symptom	80.0%	20.0%	100.0%	
		% within Recovery	14.3%	5.9%	11.1%	
Total		Count	28	17	45	
		% within Presenting Symptom	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

*LL-LOWERLIMB UL-UPPERLIMB

Preceding symptom had no significance with degree of recovery with

p value=0.089

TABLE- 41CRANIAL NERVE INVOLVEMENT AND RECOVERY/DISABILITY

			Recovery		Total	P value
			Complete Recovery	Residual Deficit		
Cranial Nerve Involvement	Yes	Count	2	14	16	0.000
		% within Cranial Nerve Involvement	12.5%	87.5%	100.0%	
		% within Recovery	7.1%	82.4%	35.6%	
	No	Count	26	3	29	
		% within Cranial Nerve Involvement	89.7%	10.3%	100.0%	
		% within Recovery	92.9%	17.6%	64.4%	
Total		Count	28	17	45	
		% within Cranial Nerve Involvement	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Cranial nerve involvement has higher significance with pattern of recovery with **p value of <0.001**

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TABLE-42 DYSAUTONOMIA AND RECOVERY/DISABILITY

			Recovery		Total	
			Complete Recovery	Residual Deficit		P value
Dysautonomia	Yes	Count	2	11	13	<0.001
		% within Dysautonomia	15.4%	84.6%	100.0%	
		% within Recovery	7.1%	64.7%	28.9%	
	No	Count	26	6	32	
		% within Dysautonomia	81.3%	18.8%	100.0%	
		% within Recovery	92.9%	35.3%	71.1%	
Total		Count	28	17	45	
		% within Dysautonomia	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Dysautonomia had grade 1 significance with degree of recovery with

p value of <0.001

TABLE-43 MECHANICAL VENTILATION AND RECOVERY/DISABILITY

			Recovery		Total	
			Complete Recovery	Residual Deficit		P value
Mechanical Ventilation	Yes	Count	0	7	7	<0.001
		% within Mechanical Ventilation	.0%	100.0%	100.0%	
		% within Recovery	.0%	41.2%	15.6%	
	No	Count	28	10	38	
		% within Mechanical Ventilation	73.7%	26.3%	100.0%	
		% within Recovery	100.0%	58.8%	84.4%	
Total		Count	28	17	45	
		% within Mechanical Ventilation	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Requirement of mechanical ventilation had significant correlation with recovery with **p value=<0.001**

TABLE-45 ELECTRODIAGNOSTIC VARIANT AND RECOVERY/DISABILITY

			Recovery		Total	P value
			Complete Recovery	Residual Deficit		
Electrodiagnostic Variant	Normal	Count	4	1	5	
		% within Electrodiagnostic Variant	80.0%	20.0%	100.0%	
		% within Recovery	14.3%	5.9%	11.1%	
	Demyelinative	Count	23	7	30	
		% within Electrodiagnostic Variant	76.7%	23.3%	100.0%	
		% within Recovery	82.1%	41.2%	66.7%	
	Aman	Count	0	5	5	0.002
		% within Electrodiagnostic Variant	.0%	100.0%	100.0%	
		% within Recovery	.0%	29.4%	11.1%	
	Amsan	Count	1	4	5	
		% within Electrodiagnostic Variant	20.0%	80.0%	100.0%	
		% within Recovery	3.6%	23.5%	11.1%	
Total		Count	28	17	45	
		% within Electrodiagnostic Variant	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Electrodiagnostic pattern with axonal variant had significant correlation with recovery with **p value =0.002**

TABLE-46 DECREASED CMAP<10% OF LOWERLIMIT AND RECOVERY/DEATH

			Recovery		Total	P value
			Complete Recovery	Residual Deficit		
Decreased CMAP<10% of lower limit	Yes	Count	0	4	4	
		% within Decreased CMAP<10% of lower limit	.0%	100.0%	100.0%	0.007
		% within Recovery	.0%	23.5%	8.9%	
	No	Count	28	13	41	
		% within Decreased CMAP<10% of lower limit	68.3%	31.7%	100.0%	
		% within Recovery	100.0%	76.5%	91.1%	
Total		Count	28	17	45	
		% within Decreased CMAP<10% of Lower limit	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Decreased CMAP <10% of lower limit had significant correlation with recovery

P value=0.007

TABLE-48 HUGHES DISABILITY GRADING SCORE AT 15 DAYS AND RECOVERY/DISABILITY

			Recovery		Total	
			Complete Recovery	Residual Deficit		P value
Hughes Disabilty Grading Score at 15 Days	Grade 2	Count	4	0	4	
		% within Hughes Disability Grading Score at 15 Days	100.0%	.0%	100.0%	
		% within Recovery	14.3%	.0%	8.9%	
	Grade 3	Count	19	4	23	<0.001
		% within Hughes Disability Grading Score at 15 Days	82.6%	17.4%	100.0%	
		% within Recovery	67.9%	23.5%	51.1%	
	Grade 4	Count	5	6	11	
		% within Hughes Disability Grading Score at 15 Days	45.5%	54.5%	100.0%	
		% within Recovery	17.9%	35.3%	24.4%	
	Grade 5	Count	0	7	7	
		% within Hughes Disability Grading Score at 15 Days	.0%	100.0%	100.0%	
		% within Recovery	.0%	41.2%	15.6%	
Total		Count	28	17	45	
		% within Hughes Disability Grading Score at 15 Days	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

HUGHES DISABILITY AT DISCHARGE AND RECOVERY/DISABILITY

			Recovery		Total	P value
			Complete Recovery	Residual Deficit		
At Discharge/Death	Grade 0	Count	28	1	29	<0.001
		% within At Discharge/Death	96.6%	3.4%	100.0%	
		% within Recovery	100.0%	5.9%	64.4%	
	Grade 1	Count	0	5	5	
		% within At Discharge/Death	.0%	100.0%	100.0%	
		% within Recovery	.0%	29.4%	11.1%	
	Grade 2	Count	0	6	6	
		% within At Discharge/Death	.0%	100.0%	100.0%	
		% within Recovery	.0%	35.3%	13.3%	
	Grade 3	Count	0	5	5	
		% within At Discharge/Death	.0%	100.0%	100.0%	
		% within Recovery	.0%	29.4%	11.1%	
Total		Count	28	17	45	
		% within At Discharge/Death	62.2%	37.8%	100.0%	
		% within Recovery	100.0%	100.0%	100.0%	

Hughes disability scoring had significant correlation with recovery at 15 days and at discharge with **p value=<0.001**

TABLE-49 RESULTS

The mean age group of our study population was 35.77 years. The respective age group related percentages are as follows. 38% of the patients were in the age group of 21-30 years, 28% of the patients in the age group of 31-40 years, 20% in 41-50 years, 8% in 51-60 years, 6% in 61-70 years. 66% of the patients were males and 34% of the patients were females.

GBS cases occurred in maximum number during our study period of July comprising 36%, August-30%, June-28%, September-6%. The average duration of hospital stay was 21.2 days.

22% of the patients had preceding events. RTI was present in 55% of the patients and AGE was present in 10% of the patients.

The presenting symptom was lower limb weakness in 60% of the patients, sensory symptoms in 15% of the patients and upper limb weakness in 10% of the patients. 42% of the patients had cranial nerve involvement, 36% of the patients had dysautonomia and 24% of the patients required mechanical ventilation in our study. The average duration of ventilator dependence was 14.5 days.

Average CSF protein was 76.03 mg%. Albumino-cytological dissociation was present in 78% of the cases.

Nerve conduction study revealed Demyelinative type in 60% of the patients, AMAN variant in 22% of the patients, AMSAN variant in 10% of the patients and it was normal in 8% of the patients. Decreased CMAP <10% was taken as a component of nerve conduction study as it was found to be a major determinant of prognosis in various randomized controlled study and it was found to be present in 18% of patients.

These patients were treated with plasmapharesis in 38% and IVIG was given in 62% of the patients. The average latency for starting the treatment was 3.36 days. 5 patients(10%) died in our study group and more than two thirds of the patients were associated with dysautonomia and required mechanical ventilation.

The factors affecting the outcome at discharge both morbidity and mortality were found to be

1. Presence of multiple cranial nerve involvement
2. Requirement of mechanical ventilation.
3. Presence of dysautonomia
4. features of axonapathy –AMAN and AMSAN variant in nerve conduction study
5. Decreased CMAP<10 percent in nerve conduction study.

TABLE-50 DISCUSSION

Guillaine barre syndrome is the commonest cause of acute and subacute flaccid quadriparesis. Its distribution and occurrence is worldwide and it does not have much seasonal variation and it affects both children and the adult population with no sex predilection.

Usually heralded by a preceding event of respiratory or gastrointestinal infection or immunization which precedes the disease process by 3 weeks in upto 61% of the cases. There are numerous disease association with GBS, viruses, bacteria, immunization, trauma, surgery, connective tissue disorders and even pregnancy which can be followed by acute inflammatory demyelinating polyneuropathy.

In our study group the mean age of the population is 35.77 years. The patients in the age group of 20-40 years were maximum comprising of around 66%. But in large scale prospective study done by the Italian GBS group comprising of 297 study population, maximum age group belonged to the older population of 55 years comprising of 46.8%. In the indian subcontinent, varies GBS study groups conducted at NIMHANS, kerala and other south indian counterparts correlated well with our study group with the maximum number of patients belonging to the 2^{nd} and 3^{rd} decade of life. As these study groups were smaller and due to our small sample size of 50 this age difference might have

occurred, but the middle aged population is affected more when compared to the western population

The male female ration is 3:2, demonstrated by many large randomized controlled trials in our study group the ratio was 2:1 with male population comprising of around 66% and the female population is 34%. Hughes et al study showed a male: female ratio of 1.5:1. The neurology society of Netherland showed the same along with Alshekhelee et al group demonstrated 2:1 male female ratio

The age distribution and the sex distribution did not seem to affect the outcome of the disease-death or the degree of recovery with the p value being greater than 0.05

In our study group the maximum number of cases occurred in the period of July followed by June comprising a total of 64% of the cases. Similar results were obtained in the GBS study group of China with slight increase during the summer. The Ho TW et al group in northern china reported a increased incidence of GBS in the summers and it has been associated with Campylobacter jejuni and other viral infections. The month of occurrence did not have any significance with the recovery or death of the patients. Similar pattern was observed by van Koningsweld et al on the Caribbean island during the years of 1996 but it has come down back to normal in 2006.

The most common preceding event in our study group was respiratory tract infection comprising of around 24 percent and 10 percent of the cases had preceding gastrointestinal infection. Koga M, Yuki et al in Japan observed cough (48%) and sore throat (39%) with nasal discharge (30%) and diarrhea present in 27% of the patients. The preceding events did not have significance on the morbidity but the recovery rate had significance with preceding events.

The commonest subtype of GBS in our study population was demyelinating type comprising 60% with axonal variant comprising 30%. There was no Miller fisher variant in our study population and 10% of the patients were normal. The Rong kuo Lyo et all study group had 49% of the patients which were demyelinative, 4% axonal,19% MFS and 28% were unclassified. Hughes et al study population showed a sensory motor form to be the most common axonal variant in the European and North American territory. Ho TW et al showed an the axonal variants were around 5-10%. Similar results were obtained by Mckhann GM et al., and Griffin JW et al., The variants were found to affect the recovery and mortality of the patients with poor outcome and mortality observed in axonal variants compared to demyelinating variants in our study group with the maximum death in the acute motor axonal neuropathy.

Lower limb weakness was the commonest presenting symptom in 60% of the patients, sensory involvement in 30% of the patients and Upper limb weakness was noted in 10 percent of the patients. In the Taiwan GBS study group by Rong Kuo Lyu et al., 60% has sensory involvement. In the Spanish

study group by Sedano MJ et al., 66.7% had sensory involvement. The presenting symptom did not have any effect on mortality but recovery rate seemed to have significance with the presenting symptoms with those involving the lower limb and sensory symptoms as the initial symptoms seemed to have residual deficit.

Cranial nerve involvement was present in 42% of the patients in our study group and the facial nerve paralysis was the most common observed palsy followed by Bulbar palsy and ocular palsy. The Spanish study group had 43.5% with cranial nerve involvement; the Taiwan study group had 60% of the study population with cranial nerve involvement. Ito M, Kuwabara et al., found that facial nerve involvement is the most common present in 70% with Bulbar and oculomotor nerves less affected. Bickerstaff brainstem encephalitis was the striking feature of the above study group. Cranial nerve involvement seemed have significant effect on mortality, ventilator dependence and residual deficits were more in our study group.

Dysautonomia was present in 36% of the patients in our study population and it was directly related to ventilator dependence and mortality in our study group. It occurred in less than two third of the patients in our study population and this may be due to the smaller size of our study population. Singh NK et al., Moulin et al and Winer JB showed that arrhythmias are more prevalent in the group with Dysautonomia. Sinus tachycardia was the most common

Dysautonomic feature in our study with fluctuating blood pressure. Dysautonomia was the major cause of mortality in our study group.

Mechanical ventilation was required in 24% of the patients in our study group. The average duration of ventilator dependence was 14.5 days in our study group. The Rong Kyo Lyu et al., study group had 20.9% with ventilator dependence. 10 days was the average duration on ventilator. J.H. Rees et al., study group showed 25% of the patient's required mechanical ventilation, the study was conducted in South East England with average duration of 42 days. Mechanical ventilation was directly related to mortality and poor recover in our study population.

Nerve conduction study was done and Decreased compound muscle action potential was observed. Decreased CMAP <10 percent was present in 18 percentage of the patients. It had higher significance with morbidity and mortality with P value of <0.001 . Similar results were obtained by the Spanish GBS group by Sedona MJ et al., and Taiwan group by Rong Kuo Lyo et al., Decreased CMAP is a significant electrodiagnostic finding which has greater significance with recovery and mortality.

Albuminocytological dissociation was present in 78 percent of the patients and it had no significance with morbidity or mortality of the patients in our study group. The disability was calculated using Hughes disability score and it had greater value in the predictability of morbidity and mortality in our study

group. It is a simple bedside analytical score comprising of 6 scores. Grade 0 is without symptoms, grade 6 being death. Grade 1 and 2 with mild symptoms, Grade 3 , 4 and 5 with progressive worsening of symptoms culminating in respiratory failure, moribund status and ventilator dependence. It was a reliable and quick bed side analytical index of prognosis observed by Hughes et al study group.

The patients were treated with plasmpharesis 19% and IVIG was given in 62% of the patients. In our study group the patients with severe debilitating symptoms were started directly on plasmapharesis in comparison to the IVIG treated groups, hence mortality was found to be slighter higher in the plasmapharesis treated group. Van der Meche et al., Hughes et al., and Plasma exchange /Sandoglobulin Guillain-Barre Syndrome trial found no difference in the efficacy of plasmapharesis to IVIG and combination of both were not found to be helpful or better over the other.

LIMITATION OF THE STUDY

Small study group

Absence of follow up study

TABLE-51 CONCLUSION

Guillaine Barre syndrome is the commonest cause of acute flaccid quadriparesis in our country. Early identification of poor prognostic factors is essential to predict the outcome of the disease in affected population.

Our study was undertaken to study about the clinical and electrodiagnostic presentation in our south indian population and to correlate them with prognosis of the patients.

Our study with a group of 50 patients showed that axonal variant of GBS with cranial nerve involvement, dysautonomia, dependence on mechanical ventilation and decreased CMAP on nerve conduction study to be associated with higher mortality and morbidity in comparision to other parameters.

Age of the patients, sex distribution, month of admission, preceding events of respiratory and gastrointestinal infections were not associated with mortality and morbidity of the patients with minimal effect of preceding infections on recovery of the patients.

Early identification of the severely affected group with the above mentioned adverse clinical and electrodiagnostic profile can be helpful for prompt treatment, anticipation of complication and adequate rehabilitation of the patients and thereby attenuating the morbidity and mortality associated with the same

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ANNEXURES

II. PROFORMA

Name: Patient ID No:

Age/Sex : Contact No:

Date of admission:

Date of discharge:

Occupation:

COMPLAINTS

- Weakness of lower limbs
- Weakness of upper limbs
- Numbness and paresthesia of limbs
- Difficulty in walking
- Difficulty in coordination
- Difficulty in breathing and speaking

- Difficulty in swallowing food
- Difficulty in chewing food
- Difficulty in blowing mouth
- Deviation of angle of mouth
- Nasal regurgitation
- Difficulty in raising head from bed and mainting head posture
- Difficulty of turning from side to side in bed
- Diplopia
- Excessive sweating
- Bladder and bowel disturbance
- Palpitations, giddiness and headache

PAST HISTORY

☐ Fever, URI, Diarrhoea, vaccination ☐ CAD, HTN, TYPE2DM

☐ Kidney disease ☐ STROKE/TIA

☐ Other Neurological disease

PERSONAL HISTORY

☐ Smoking

☐ Alcohol intake

GENERAL EXAMINATION

BP: Lying:

BP: Standing:

Pulse:

Weight:

Single breath count:

Resting heart rate:

Systemic examination:

CVS:

RS:

P/A:

CNS:

Higher mental functions

Cranial nerves

Spinomotor system-

Bulk:

Tone:

Reflex: superficial and deep

Power-(Medical research council grading system)

Neck muscle weakness

Plantar

Neck stiffness

Sensory system

Bladder and bowel:

Cerebellum

Spine and cranium

Disability scoring- Modified Disability Grading Scale for GBS (modified from Hughes RAC et al 1978.)

Grade 0	Normal functional state
Grade 1	Able to run with minor signs and symptoms
Grade 2	Able to walk 5 m independently
Grade 3	Able to walk 5 m with aid
Grade 4	Bed- or chair-bound
Grade 5	Requires assisted ventilation

MADRAS MEDICAL COLLEGE, CHENNAI-3

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CERTIFICATE OF APPROVAL

To
Dr. Karthika.R,
Post Graduate in MD General Medicine,
Madras Medical College,
Chennai – 600003.

Dear Dr. Dr. Karthika.R,

The Institutional Ethics Committee has considered your request and approved your study titled **"Clinical and Electrodiagnostic profile of Guillaine Barre Syndrome and its correlation with early predictors of prognosis in a tertiary care centre"** No. 45072014.

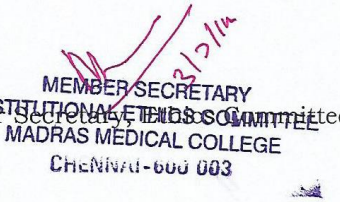
The following members of Ethics Committee were present in the meeting held on 01.07.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Dr. R. Vimala, M.D., Dean, MMC, Ch-3.	-- Deputy Chair Person
3.	Prof. Kalaiselvi, MD., Vice-Principal, MMC, Ch-3	-- Member Secretary
4.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
5.	Dr. G. Muralidharan, Director Incharge , Inst. of Surgery	-- Member
6.	Prof. Md Ali, MD., DM., Prof & HOD of MGE, MMC, Ch-3.	-- Member
7.	Prof. Ramadevi, Director i/c, Biochemistry, MMC,Ch-3.	-- Member
8.	Prof. Saraswathy, MD., Director, Pathology, MMC, Ch-3.	-- Member
9.	Prof. Tito, Director, i/c. Inst. of Internal Medicine, MMC	-- Member
10.	Thiru. Rameshkumar, Administrative Officer	-- Lay Person
11.	Thiru. S. Govindasamy, BABL, High Court, Chennai-1.	-- Lawyer
12.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist

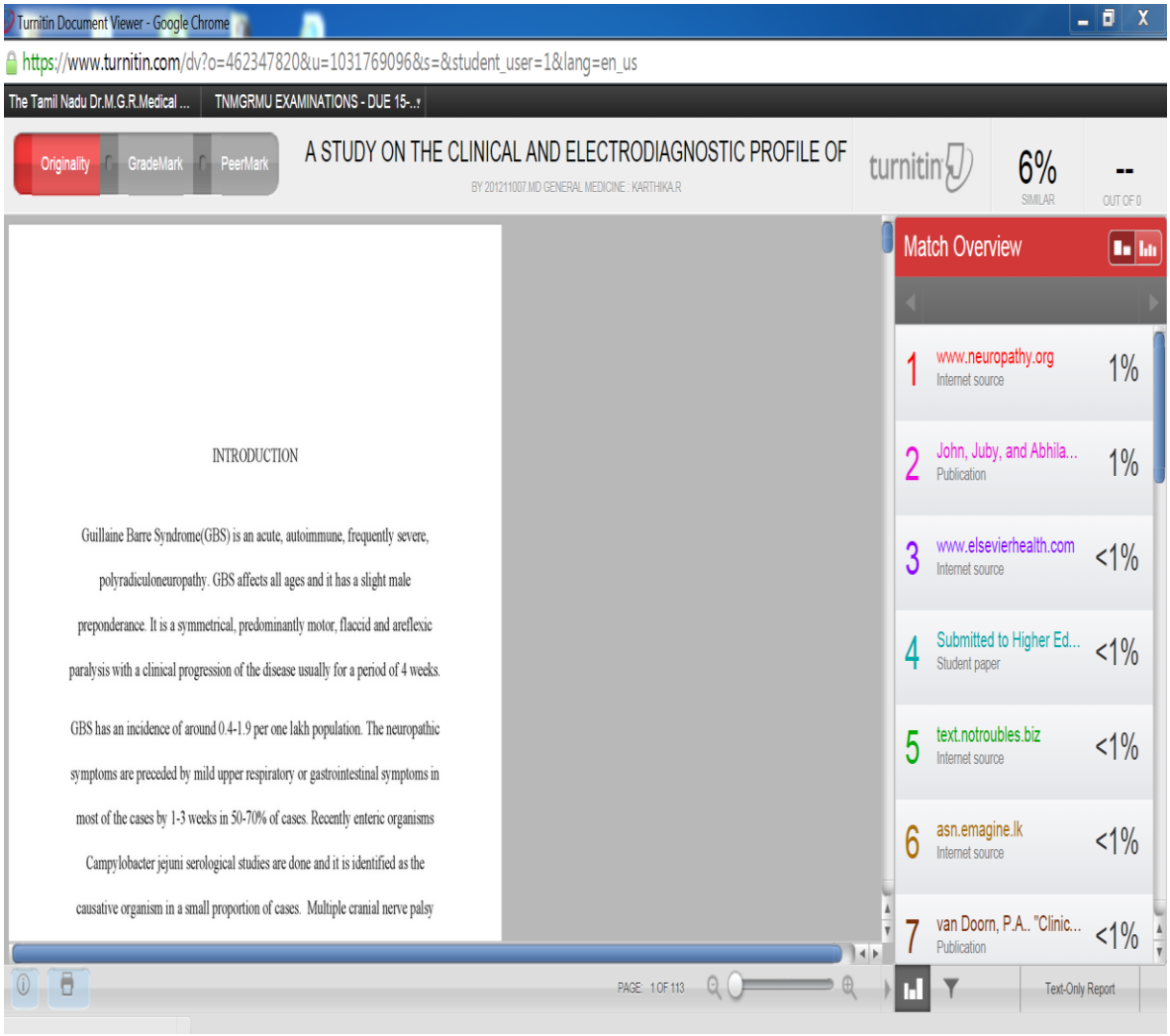
We approve the proposal to be conducted in its presented form.

Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.



Member Secretary, Ethics Committee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INTRODUCTION
Guillaine Barre Syndrome(GBS) is an acute, autoimmune, frequently severe, polyradiculoneuropathy. GBS affects all ages and it has a slight male preponderance. It is a symmetrical, predominantly motor, flaccid and areflexic paralysis with a clinical progression of the disease usually for a period of 4 weeks.
GBS has an incidence of around 0.4-1.9 per one lakh population. The neuropathic symptoms are preceded by mild upper respiratory or gastrointestinal symptoms in most of the cases by 1-3 weeks in 50-70% of cases. Recently enteric organisms Campylobacter jejuni serological studies are done and it is identified as the causative organism in a small proportion of cases. Multiple cranial nerve palsy
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INTRODUCTION

Guillaine Barre Syndrome(GBS) is an acute, autoimmune, frequently severe, polyradiculoneuropathy. GBS affects all ages and it has a slight male preponderance. It is a symmetrical, predominantly motor, flaccid and areflexic paralysis with a clinical progression of the disease usually for a period of 4 weeks.

GBS has an incidence of around 0.4-1.9 per one lakh population. The neuropathic symptoms are preceded by mild upper respiratory or gastrointestinal symptoms in most of the cases by 1-3 weeks in 50-70% of cases. Recently enteric organisms Campylobacter jejuni serological studies are done and it is identified as the causative organism in a small proportion of cases. Multiple cranial nerve palsy involving facial, bulbar and ocular muscle has been reported in 10-65% of cases with papilledema being uncommon. Respiratory failure occurs in 10-30% of cases.

GBS is a self-limiting disorder associated with recovery in upto 70% of cases but the morbidity and mortality is heinous is also a concern as the sequelae of the disease is associated with significant motor handicap in upto 7% of cases. Mortality usually reaches upto 14% in few studies.

INFORMATION SHEET

We are conducting a study on "SERUM **ZINC LEVELS IN DECOMPENSATED LIVER DISEASE AND ITS CORRELATION WITH THE STAGE OF HEPATIC ENCEPHALOPATHY**" among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

The purpose of this study is to assess the correlation between serum Zinc level in Decopensated chronic liver disease, its complications and stage of hepatic encephalopathy. We are selecting certain cases and if you are found eligible, after filling up the questionnaire, 5 ml blood will be collected. You will also undergo clinical examination, serum Zinc, LFT, RFT and PT/APTT examination. These tests and special studies do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator | Signature of Participant

Date :
Place :

PATIENT CONSENT FORM

Study Title	:	Clinical and electrodiagnostic profile of Guillaine Barre syndrome and its correlation with early predictors of prognosis in a tertiary care centre.
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Name	:	
Age/Sex	:	
Identification Number	:	

Patient may check (☑) these boxes

The details of the study have been provided to me in writing and explained to me in my own language ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological and biochemical tests. ☐

Signature/thumb impression
Patients name and address

Signature of Investigator
Study Investigator's Name:
Dr. KARTHIKA.R

ஆராய்ச்சி தகவல் தாள்

சென்னை இராஜீவ்காந்தி அரசு பொது மருத்துவனையில் திறனற்ற கல்லீரல் நோயாளிகளின் சீரம்சிங்க் அளவிற்கும் கல்லீரல் மூளை நலிவிற்கும் உள்ள தொடர்பினை பற்றிய ஆய்வாகும்.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் உங்களுடைய திசுக்களை எடுத்து சில சிறப்பு பரிசோதனைக்கு உட்படுத்தி அதன் தகவல்களை ஆராய்வோம். அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்பு ஏற்படாது என்பதையும் தெரிவித்துக்கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும்போதோ அல்லது ஆராய்ச்சியின்போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதை தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

நாள் :
இடம் :

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு

திறனற்ற கல்லீரல் நோயாளிகளின் சீரம்சிங்க் அளவிற்கும் கல்லீரல் மூளை நலிவிற்கும் உள்ள தொடர்பினை பற்றி ஆராய்தல்.

இந்த ஆராய்ச்சியில் திறனற்ற கல்லீரல் நோயாளிகளின் சீரம்சிங்க் அளவிற்கும் கல்லீரல் மூளை நலிவிற்கும் உள்ள தொடர்பினை பற்றி ஆராயப்படுகிறது. நீங்கள் தகுதியுள்ளவர் என்றால், உங்களின் முழு சம்மதத்தோடு, உங்களிடமிருந்து 5 மி.லி இரத்தம் எடுக்கப்படும். மேலும், உங்கள் இரத்தத்தில், கல்லீரல் செயல்பாடு மதிப்பீடு, சீறுநீரக செயல்பாட்டு மதிப்பீடு இரத்தம் உராய்தல் மதிப்பீடு ஆகியவற்றை செய்யப்படும் இந்த பரிசோதனைகள் உங்கள் சிகிச்சையை பாதிக்காது என்றும் உறுதி அளிக்கப்படுகிறது.

இந்த ஆராய்ச்சின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்துகொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதம்.

கையொப்பம்

KEY TO MASTER CHART

A- PATIENT NUMBER

B- AGE

C- SEX

D- ADMISSION MONTH

E- DURATION OF STAY IN HOSPITAL

F- PRECEDING EVENT RTI-RESPIRATORY INFECTION,AGE-ACUTE GASTROENTERITIS

G- PRESENTING SYMPTOM

H- SENSORY INVOLVEMENT

I- CRANIAL NERVE INVOLVENT

J- DYSAUTONOMIA

K- VENTILATOR DEPENDENCE

L- DURATION OF MECHANICAL VENTILATION

M- CSF PROTEIN IN MG%

N- ALBUMINOCYTOLOGICAL DISSOCIATION

O- ELECTRODIAGNOSTIC VARIANT D-DEMYELINATIVE, AMAN-ACUTE MOTOR AXONAL NEUROPATHY, AMSAN-ACUTE MOTOR SENSORY AXONAL NEUROPATHY

P- DECREASED CMAP<10% OF LOWER LIMIT

Q- TREATMENT GIVEN P-PLASMAPHARESIS, IVIG- IV IMMUNOGLOBULIN

R- LATENCY FOR STARTING TREATMENT

S- DEATH

T- HUGHES DISABILITY GRADING AT DAY 15

U- HUGHES DISABILITY GRADING AT DISCHARGE/DEATH

V- COMPLETE RECOVERY

W- RESIDUAL DEFICIT

A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X
1	63	M	JUNE	14	YES /RT	LL	NO	NO	NO	NO	0	67	YES	D	NO	IVIg	4		GRADE 4	GRADE 0	YES		
2	28	M	JUNE	17	YES /RT	S	YES	NO	NO	NO	0	70	YES	D	NO	IVIG	3		GRADE 4	GRADE 1		YES	
3	33	F	JUNE	21	YES /RT	LL	NO	NO	NO	NO	0	69.5	YES	D	NO	IVIG	4		GRADE 3	GRADE 0	YES		
4	37	M	JUNE	23	NO	LL	NO	YES	YES	NO	0	75.6	YES	AMAN	NO	P	5		GRADE 4	GRADE 3		YES	
5	21	M	JUNE	17	NO	S	YES	NO	NO	NO	0	78	YES	D	NO	IVIG	3		GRADE 3	GRADE 9	YES		
6	30	M	JUNE	18	NO	LL	NO	NO	NO	NO	0	79.3	YES	D	NO	IVIG	4		GRADE 4	GRADE 0	YES		
7	36	F	JUNE	22	YES/AGE	LL	NO	YES	NO	NO	0	80	NO	NORMAL	NO	IVIG	4		GRADE 4	GRADE 2		YES	
8	21	M	JUNE	15	NO	LL	NO	NO	NO	NO	0	88	YES	NORMAL	NO	IVIG	4		GRADE 3	GRADE 0	YES		
9	34	M	JUNE	18	NO	LL	NO	NO	NO	NO	0	90	YES	D	NO	IVIG	3		GRADE 4	GRADE 0	YES		
10	24	M	JUNE	14	NO	LL	NO	NO	NO	NO	0	78	NO	D	NO	IVIG	3		GRADE 2	GRADE 0	YES		
11	22	F	JUNE	27	YES/ RTI	LL	NO	YES	YES	YES	17	75.4	YES	AMAN	YES	P	3	DEATH	GRADE 5	GRADE 5			
12	47	M	JUNE	25	YES/AGE	LL	NO	YES	YES	NO	0	76	YES	AMAN	NO	P	3		GRADE 4	GRADE 2		YES	
13	25	F	JUNE	24	NO	LL	NO	YES	YES	NO	0	65	YES	AMSAN	NO	P	3		GRADE 4	GRADE 2		YES	
14	28	M	JUNE	19	YES/ RTI	LL	NO	NO	NO	NO	0	63.2	YES	D	NO	IVIG	4		GRADE4	GRADE1			
15	22	M	JULY	23	NO	S	YES	YES	YES	NO	0	69	YES	AMSAN	NO	P	4		GRADE 4	GRADE 0	YES		
16	34	M	JULY	20	NO	LL	NO	NO	NO	NO	0	88	NO	D	NO	IVIG	4		GRADE 3	GRADE 0	YES		
17	46	M	JULY	18	YES/AGE	LL	NO	NO	NO	NO	0	88.5	YES	D	NO	IVIG	3		GRADE 3	GRADE 0	YES		
18	25	M	JULY	26	NO	LL	NO	YES	YES	YES	15	87.3	YES	AMAN	YES	P	3		GRADE 5	GRADE 3		YES	
19	45	F	JULY	18	NO	LL	NO	NO	NO	NO	0	73.5	NO	D	NO	IVIG	3		GRADE 3	GRADE 0	YES		
20	25	F	JULY	23	NO	S	YES	NO	NO	NO	0	75.6	YES	D	NO	IVIG	3		GRADE 3	GRADE 1		YES	
21	35	M	JULY	17	NO	UL	NO	NO	NO	NO	0	79.8	YES	D	NO	IVIG	3		GRADE 3	GRADE 0	YES		
22	36	F	JULY	25	YES/ RTI	S	YES	YES	YES	YES	12	78.5	YES	AMSAN	NO	P			GRADE 5	GRADE 2		YES	
23	50	F	JULY	27	YES/ RTI	LL	NO	YES	YES	YES	14	73.2	YES	AMAN	YES	P	4		GRADE 5	GRADE 3		YES	
24	37	M	JULY	21	NO	LL	NO	NO	NO	NO	0	87.6	YES	NORMAL	NO	IVIG	3		GRADE 3	GRADE 0	YES		
25	58	M	JULY	22	NO	S	YES	YES	NO	NO	0	89.3	NO	D	NO	IVIG	4		GRADE 3	GRADE 1		YES	
26	42	F	JULY	19	YES/AGE	LL	NO	NO	NO	NO	0	64.3	YES	D	NO	IVIG	4		GRADE 3	GRADE 0	YES		
27	47	M	JULY	15	NO	LL	NO	NO	NO	NO	0	65.7	NO	D	NO	IVIG	4		GRADE 3	GRADE 0	YES		
28	61	F	JULY	14	NO	LL	NO	NO	NO	NO	0	87	YES	NORMAL	NO	IVIG	4		GRADE 2	GRADE 0	YES		
29	36	M	JULY	19	NO	LL	NO	NO	NO	NO	0	92	YES	D	NO	IVIG	3		GRADE 3	GRADE 0	YES		

A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W
30	39	M	JULY	18	NO	UL	NO	NO	NO	NO	0	54	NO	D	NO	IVIG	3		GRADE 3	GRADE 0	YES	
31	26	M	JULY	26	YES/AGE	S	YES	YES	YES	YES	12	55	YES	AMSAN	NO	P	3		GRADE 5	GRADE 2		YES
32	24	F	JULY	30	NO	LL	NO	YES	YES	YES	15	60.4	YES	AMAN	YES	P	3		GRADE 5	GRADE 3		YES
33	48	M	AUGUST	21	NO	LL	NO	NO	NO	NO		56.7	NO	D	NO	P	3		GRADE 2	GRADE 0	YES	
34	29	M	AUGUST	27	YES/ RTI	LL	NO	YES	YES	YES	12	89	YES	D	NO	P	3		GRADE 5	GRADE 2		YES
35	32	M	AUGUST	30	YES/ RTI	LL	NO	YES	YES	YES	16	94.3	YES	AMAN	YES	P	3	DEATH	GRADE 5	GRADE 5		
36	59	F	AUGUST	29	NO	S	YES	YES	YES	YES	13	67.9	YES	AMSAN	YES	P	3		GRADE 5	GRADE 1		YES
37	27	M	AUGUST	15	YES/AGE	LL	NO	NO	NO	NO		69.8	YES	NORMAL	NO	IVIG	3		GRADE 3	GRADE 0	YES	
38	23	F	AUGUST	14	NO	S	YES	NO	NO	NO		65.7	YES	D	NO	IVIG	4		GRADE 3	GRADE 0	YES	
39	43	M	AUGUST	14	NO	S	YES	NO	NO	NO		77	NO	D	NO	IVIG	3		GRADE 3	GRADE 0	YES	
40	57	F	AUGUST	16	YES/AGE	LL	NO	NO	NO	NO		74	YES	D	NO	IVIG	3		GRADE 3	GRADE 0	YES	
41	22	M	AUGUST	16	YES/AGE	S	YES	NO	NO	NO		75	YES	D	NO	IVIG	3		GRADE 3	GRADE 0	YES	
42	53	M	AUGUST	19	NO	UL	NO	NO	NO	NO		78	YES	D	NO	IVIG	3		GRADE 2	GRADE 0	YES	
43	40	M	AUGUST	25	NO	S	YES	YES	YES	NO		70	YES	D	NO	P	4		GRADE 4	GRADE 3		YES
44	30	F	AUGUST	28	YES/ RTI	LL	NO	YES	YES	YES	14	68	YES	AMAN	YES	P	3	DEATH	GRADE 5	GRADE 5		
45	49	M	AUGUST	20	NO	LL	NO	NO	NO	NO		69.6	YES	D	NO	IVIG	3		GRADE 3	GRADE 0	YES	
46	64	M	AUGUST	30	YES/AGE	LL	NO	YES	YES	YES	17	69	YES	AMAN	YES	P	3	DEATH	GRADE 5	GRADE 5		
47	33	M	AUGUST	31	YES/ RTI	LL	NO	YES	YES	YES	17	80	YES	AMAN	YES	P	3	DEATH	GRADE 5	GRADE 5		
48	50	M	EPTEMBE	22	NO	S	YES	YES	NO	NO		89	NO	D	NO	P	3		GRADE 3	GRADE 0	YES	
49	29	F	EPTEMBE	21	YES/AGE	S	YES	YES	NO	NO		93	NO	D	NO	IVIG	3		GRADE 3	GRADE 1		YES
50	37	F	EPTEMBE	17	YES/ RTI	S	YES	NO	NO	NO		92	YES	D	NO	IVIG	3		GRADE 3	GRADE 0		
							15	21	18	12			NO			IVIG						
					22-44%	eakness-30	30%	42%	36%	24%	14.5 DAYS	6.03 mg%	39-78%	IAN=11-2	SED CMA	P	168	EATH-5-10%			COMPLET	18-36%
	35.77%	M-33-66%	NE-14-28	1.2 DAYS	RTI -54.5	symptoms-15-30%							AMSAN-5-10%		IVIg-31-62	9.36 DAYS					RECOVERY	
	21-30-38%	F-17-34%	LY18-36%		10-AGE-45.4	eakness-5-10%							DEMYELINATIVE-30-60%								27-54%	
	31-40-28%		AUGUST-15-30%										NORMAL-8%									
	41-50-20%		SEP-3-6%																			
	51-60-8%																					
	61-70-6%																					