"A STUDY ON GUILLAIN BARRE'S SYNDROME - CLINICAL PROFILE AND TREATMENT OUTCOME"

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"learn to heal"

CERTIFICATE

This is to certify that the dissertation entitled "A study on Guillain Barre's Syndrome - clinical profile and treatment outcome" is the bonafide original work of Dr. P. Saravanan in partial fulfillment of the requirements for D.M. Branch-I (Neurology) examination of THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY to be held in August 2008. The period of post-graduate study and training was from August 2005 to July 2008.

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DECLARATION

I **Dr. P. SARAVANAN,** solemnly declare that this dissertation entitled,

"A STUDY ON GUILLAIN BARRE'S SYNDROME - CLINICAL PROFILE AND TREATMENT OUTCOME" is a bonafide work done by me at the Department of Neurology, Madras Medical College and Government General Hospital during the period 2005 – 2008 under the guidance and supervision of the Professor and Head of the department of Neurology of Madras Medical College and Government General Hospital, Professor **DR.A.V.SRINIVASAN M.D.D.M.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-I)** in Neurology.

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Dr. P. SARAVANAN

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CONTENTS

INTRODUCTION

INTRODUCTION

Guillain Barre Syndrome (GBS) is an acute, self limited, inflammatory, autoimmune disorder of the peripheral nervous system triggered usually by a bacterial or viral infection or other antecedent events. It affects 0.9 to 2/100,000 persons in a year, with a worldwide distribution and a slight male preponderance. Generally at the end of one year of illness, 5% of the patients had expired and 15% might be unable to walk. Hence it causes large loss of productivity and burdens the health care due to its prolonged morbidity. It is a heterogeneous disorder in its type, severity, pathogenesis and prognosis.

GBS is characterised by a rapidly Progressive weakness of all 4 limbs with or without sensory loss, evolving within 4 weeks, followed later by slow clinical and electrophysiological recovery. The subtypes of GBS are several. Among those which produce weakness, the common one are Acute Inflammatory Demyelinating polyradiculopathy (AIDP), Acute Motor Sensory Axonal Neuropathy (AMSAN) and Acute Motor Axonal Neuropathy (AMAN) and the rare one are pharyngo-cervico Brachial variant, Bilateral foot drop, and bifacial weakness. Among those which do not produce weakness the common one is Miller. Fischer syndrome (MFS) and the rare ones are Pure sensory variant, and acral parasthesia with areflxia.

Neurophysiologic abnormalities are often very mild or occasionally normal in the early stages of GBS and hence may not correlate well with clinical disability. AIDP is characterized classically by conduction block with also prolongation of CMAP latency and f-wave latency but a normal amplitude. AMAN and AMSAN are characterized by reduction or absence of amplitude of CMAP and both CMAP and SNAP respectively.

Experimental evidence implicates autoantibodies to gangliosides as the cause of the axonal subgroup of GBS and MFS. These antibodies may be generated by the immune response to an infective organism such as Campylobacter jejuni, cross- reacting with the epitopes on the axon. The resemblance of AIDP to experimental auto immune neuritis suggests pathogenetic mechanisms involving T-cell induced, macrophage associated demyelination. This proposed autoimmune etiology lead to the induction of immunotherapy.

Intravenous Immunoglobulin (IvIg) and plasma exchange (PE) are the standard treatment options available at present. Though both have similar outcome measures many centers prefer the former because of the convenience and safety. Several studies show no or less improvement with steroids when compared to placebo. But in reality the use of steroids is prevalent especially when the definite or standard treatment options are either not affordable or not

available, but at the same time the physicians are under pressure to use "some specific drugs" having anti inflammatory properties. In the present study also, several patients were treated with injection methyl prednisolone and an attempt is made to analyze and compare the outcome measures of the patients treated with different modalities.

Most of the previous studies had utilized the 7 point (0-6) Hughe's disability grade scale to evaluate the disability at admission and to assess the outcome. In addition to the above scale, we also utilize the MRC disability scale (0-10) for assessment. Those who attain walking with or without support are graded to have good outcome (<3 in GBS (Hughe's) disability scale and <5 in MRC disability scale) and the poor outcome are for those who are unable to walk, ventilator dependence or who had expired.

It is also important to know, when to transfer the patient to intensive care unit and who may require mechanical ventilation. In this study we have also analyzed the association of some of the clinical features related to poor outcome, including death, ventilatory support and tracheostomy.

AIM OF STUDY

AIM OF STUDY

1. To study the demographic variables, clinical features and electrophysiological findings in patients with various subtypes of GBS.

2. To assess the predictors of respiratory failure.

3. To analyze the influence of poor prognostic factors on the outcome of GBS.

4. To compare the outcome measures of GBS after various treatment modalities.

5. To analyze and quantify the mean improvement in GBS after various treatment modalities.

REVIEW OF THE LITERATURE

REVIEW OF LITERATURE

The term Guillain Barre syndrome (GBS) defines a recognizable clinical entity that is characterised by rapidly evolving symmetrical limb weakness, loss of tendon reflexes, absent or mild sensory signs, and variable autonomic dysfunctions. Since the virtual elimination of poliomyelitis, GBS has become the leading cause of acute flaccid paralysis in western countries.[1] The condition, however, occurs worldwide, affecting patients of all ages and both sexes. In the majority of cases, the neuropathy is triggered by a bacterial or viral illness. Weakness can develop acutely (within days) or subacutely (up to 4 weeks) and reaches a plateau, with subsequent spontaneous resolution of paralysis. Although the pathogenesis of GBS remains incompletely defined, there is increasing support for the concept that GBS results from an aberrant organ-specific immune response.[2]

The diagnosis of GBS remains descriptive. To facilitate epidemiological research and outcome assessments of therapeutic trials, a set of generally accepted clinical, laboratory, and electrodiagnostic criteria has been set forth, delineating the prevailing clinical presentation- acute inflammatory demyelinating polyneuropathy (AIDP;[3,4] panel). It has become apparent, however, that the clinical spectrum of GBS comprises a heterogeneous group of

pathological entities, each with distinctive clinical features and most probably with its own pathogenesis.

Diagnostic criteria for Guillain Barre Syndrome[3]

Features required for diagnosis

- Progressive weakness of both the legs and arms
- Areflexia

Clinical features strongly supportive of diagnosis

- Progression of symptoms over days to 4 weeks
- Relative symmetry of signs
- Mild sensory symptoms or signs
- Cranial nerve involvement (bilateral facial palsies)
- Recovery beginning 2-4 weeks after progression ceases
- Autonomic dysfunction
- Absence of fever at onset
- Elevated cerebrospinal fluid protein with <10 cells/μl
- Typical electrodiagnostic features

Features excluding the diagnosis

- Diagnosis of botulism, myasthenia, poliomyelitis or toxic neuropathy
- Abnormal porphyrin metabolism
- Recent diphtheria
- Purely sensory symptoms without weakness

Epidemiology

GBS has been the subject of 35 population-based surveys from defined geographical areas of Europe, Australia, and North and Latin America during the past 40 years. In the past 20 years, accuracy of case ascertainment and collection have improved. All epidemiological studies, however, continue to be hampered by the absence of a reference diagnostic test that would allow a positive confirmation of the diagnosis. Nevertheless, most reports document similar figures for annual incidence. Such observations indicate that GBS occurs evenly throughout the western hemisphere, without geographical clustering and with only minor seasonal variations.

GBS is known to occur at all ages, though it is rare in infancy. The incidence remains almost uniform below the age of 40, ranging from 1-3 to 1-9 per 100 000 annually. Most surveys show a slight peak in late adolescence and young adulthood, coinciding with an increased risk of infections with

cytomegalovirus and *Campylobacterjejuni,* and a second peak in the elderly.[8-10] A hospital record-based study from the USA, published in 1997, measured the annual incidence as 8-6 per 100 000 in people over the age of 70; GBSassociated morbidity and mortality increased in parallel.[11] These observations require confirmation and remain unexplained. A failing of immune-suppressor mechanisms in elderly people has been postulated as an explanation for increased susceptibility to autoimmune disorders.

Preceding events

Antecedent infections

GBS is the prototype of a postinfectious illness; two-thirds of patients report an antecedent, acute infectious illness, most commonly a respiratory-tract infection or gastroenteritis that has resolved by the time neuropathic symptoms begin. The interval between the prodromal infection and the onset of GBS symptoms varies between I week and 3 weeks, occasionally longer; it averaged I I days in several large series.[12] In many instances, the pathogen that caused the prodromal illness remains unidentified. Although various infections and events such as surgery have been put forward as possible triggers, the link with GBS is not firmly established and remains anecdotal.

C *jejuni,* a major cause of bacterial gastroenteritis worldwide, has become recognised as the most frequent antecedent pathogen for GBS. The association has been documented in many case reports and in 14 large series of GBS patients that were collected prospectively, together with appropriate case controls. Serological or culture evidence of a recent C *jejuni* infection ranged from 26% to 41% in series of sporadic GBS cases from the UK, the Netherlands, the USA, and Japan.[13-16] This gastrointestinal pathogen was also strongly associated with an acute motor-axonal neuropathy variant of GBS observed in yearly summer epidemics among rural children in northern China.[17] In a 2-year prospective study from Hebei Province, China, serological evidence of a recent C*jejuni* infection was found in 66% of GBS patients, as opposed to only 16% of village controls.[17] Moreover, C *jejuni* infections are also the most frequent trigger of the Miller Fisher syndrome, (MFS), a variant of GBS characterised by ophthalmoplegia, ataxia, and areflexia.[18,19]

The organism may be cultured from stool for several weeks after the end of the diarrhoeal illness. In Japan, the majority of C*jejuni* isolates from GBS patients were of Penner serotype 19 (HS-19), whereas this serotype was respiratory-tract infection or gastroenteritis that has resolved by the time neuropathic symptoms begin. The interval between the prodromal infection and

the onset of GBS symptoms varies between I week and 3 weeks, occasionally longer; it averaged I I days in several large series.[12] In many instances, the pathogen that caused the prodromal illness remains unidentified. Although various infections and events such as surgery have been put forward as possible triggers, the link with GBS is not firmly established and remains anecdotal. Those pathogens for which there is convincing and statistically valid evidence of an association with GBS are listed in the table.

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frequent trigger of the Miller Fisher syndrome, (MFS), a variant of GBS characterised by ophthalmoplegia, ataxia, and areflexia.[18,19]

Cytomegalovirus infections, experienced clinically as upperrespiratory-tract infection, pneumonia, or nonspecific flu-like illness, account for the most common viral triggers of GBS, ranging from 10% to 22% in several large series.[9.12.27] Cytomegalovirus is particularly common in young female GBS patients, and the clinical picture is notable for prominent involvement of the sensory and cranial nerves.[9] Many such patients have high serum titres of antibodies reacting with GM_2 gangliosides and with sulphated glycolipids.[28-30] The specificity of such antibodies and their significance for the pathogenesis of GBS remains unknown. Associations of GBS with Epstein-Barr virus (10%) or varicellazoster virus are more common than in matched populationsY The association of GBS and HIV-l is well recognized and occurs usually around the time of seroconversion.[31] Clinical presentation does not differ from ordinary AIDP; lymphocytic pleiocytosis in the cerebrospinal fluid should raise suspicion of HIV-1 infection, prompting the search for confirmation.

GBS and vaccine

Several anecdotal case reports or small case series have linked GBS to vaccinations on the grounds of a mere temporal association, but no causal relation has been established and potentially confounding coincidental infections were not ruled out. There is, however, no doubt that rabies vaccines prepared from the infected brain tissues of adult animals carried an increased risk of inducing GBS, probably because of contamination with myelin antigens.[32] Controversy surrounded the alleged association of GBS and receipt of swine-flu influenza vaccine, administered to 45 million Americans in 1976 and 1977. After re-examination of the data, a panel of experts concluded that a small excess risk of developing GBS existed for up to 6 weeks after the immunisation.[33] The cause was never established. Carefully conducted surveillance studies of subsequent mass influenza-vaccination programmes of the US Army found no increased incidence of GBS.[34] The possibility that GBS might be triggered by live, attenuated, oral poliovirus vaccine was suggested in a report from Finland.[35] It described an unusually high incidence of GBS within weeks of a national campaign of vaccination with oral polio vaccine. The observation remains unique. Moreover, a careful epidemiological re-evaluation identified a coincidental influenza epidemic and widespread persistence of the wild-type poliovirus during the relevant period. Both could have contributed as

potential triggers to the transient GBS peak occurrence. In addition, the number of GBS cases had started to rise before the vaccination campaign. Thus, the causal relation between GBS and administration of oral polio vaccine is questionable. In addition, a large survey of GBS among children in South America showed no temporal association or increased incidence of GBS during programmes of mass immunisation with oral poliovaccine. Altogether, whether oral poliovaccine is associated with increased risk of GBS is still uncertain.[36] Most other currently used vaccines do not seem to be associated with any increased risk. Surveillance during a mass measles-vaccination programme of more than 70 million children in South America found no increased risk of GBS.[37] Two case-control surveys of approximately 200 GBS patients from southeast England, which included individuals immunised with influenza, typhoid, cholera, and diphtheriatetanus-pertussis vaccines, did not show any significant association between occurrence of GBS and a previous immunization.[38] These observations do not exclude an association, but the investigators judged that any increase in absolute risk was unlikely to be greater than five-fold. Therefore, in any person who has recovered from GBS, the risk of any vaccination should be weighed against the risk of exposure.

Antecedent events for Guillain Barre Syndrome[75]

Infections

Viral:

- EBV
- CMV
- HIV
- Influenza virus
- Coxsackie virus
- Herpes simplex
- Hepatitis A and C viruses

Bacterial:

- Campylobacter jejuni
- Mycoplasma pneumonia
- Escheritia coli

Parasites:

- Malaria
- Toxoplasmosis

Systemic illnesses

Hodgkin's lymphoma

Chronic Lymphocytic Leukemia

Hyperthyroidism

Collagen vascular disorders

Sarcoidosis

Renal disease

Other medical conditions

Pregnancy

Surgical procedures

Bone marrow transplants

Immunizations

Envenomation

Clinical spectrum

Acute inflammatory demyelinating polyradiculoneuropathy

Until very recently, the eponym Guillain Barre syndrome was used interchangeably with AIDP, which refers to the salient pathological findings: the early lymphocytic infiltrates in spinal roots and peripheral nerves, and the subsequent macrophage-mediated segmental stripping of myelin. Such segmental loss of the insulating properties of myelin is known to cause profound defects in the propagation of electrical nerve impulses, resulting eventually in conduction block and in the functional correlate of flaccid paralysis.[39] AIDP is the most prevalent form of sporadic GBS in western countries and accounts for 85-90% of cases.[13] It is generally viewed as an autoimmune disorder, triggered in most cases by an antecedent bacterial or viral infection. The target of the aberrant immune response seems to be within the Schwann-cell surface membrane or the myelin, resulting in primary inflammatory demyelination as the major pathological finding.[40,41] Humoral immune responses seem to be of particular importance, but there is also clear evidence of T-cell activation.

The precise target epitopes of the immune reaction are not known. Early in the course of AIDP there is infiltration of nerves by lymphocytes and, in particular, the deposition of activated complement components along the. outer Schwann-cell surface membrane of myelinated nerve fibres. The myelin sheaths of such fibres undergo a process of vesicular disruption, progressing from outside inward. These fine structural changes occur before the recruitment of macrophages and their invasion of nerve fibres. These findings led to the idea that binding of complement-fixing antibodies to epitopes exposed on the outer Schwann-cell surface membrane might lead to complement activation, which, in turn, would initiate the disruption of compact myelin; the recruitment of macrophages would then complete the process of segmental demyelination.[42] These observations identify the Schwann cell or myelin as the target of the immune reaction, underscoring the importance of circulating antibodies in the pathogenesis of GBS. Various antibodies to nerve-cell components, notably antiglycolipids such as anti-GMI, have been detected in serum from GBS patients,[14,17,20,43] but a direct causal link to the neuropathy has not yet been shown.

AIDP is viewed as a reactive, self-limited, autoimmune disease. The primary consequence of the immune process is the multifocal disruption of myelin segments, which leads to characteristic electrophysiological findings: slowing of nerve-conduction velocities, prolongation of distal and F-wave

latencies, and conduction block. Once the immune reactions come to a halt, repair and remyelination set in promptly, which correlates with a quick and, in most cases, complete recovery from the flaccid paralysis. In many AIDP patients, however, particularly those with severe disease, inflammatory demyelination is accompanied by variable disruption and loss of nerve axons.[44,45] Breakdown ofaxons in this setting is thought to be a secondary "bystander" event, caused possibly by intense inflammation, oedema, and swelling of nerves.[44-47] The degree of complicating axonal loss in AIDP is an important determinant of the speed of recovery, the lasting deficits, and the ultimate prognosis.[48]

Acute motor-sensory axonal neuropathy

Over the years, some case reports alluded to the possibility that the clinical spectrum of GBS is more heterogeneous. Based on clinical, electrophysiological, and pathological observations, the suggestion was made that a similar clinical presentation might result from a primary immune attack directed towards nerve axons.[49-51] This idea, which became the subject of much controversy, is now supported by direct evidence.[41,52,53]

Feasby and colleagues[49] drew attention to the unusual findings in seven of their GBS patients who presented with fulminant onset of paralysis after a

diarrhoeal or flu-like illness. All had severe generalised paralysis and six needed assisted ventilation within 2-4 days from onset of neurological symptoms. Serial electrophysiological examinations, within 2-7 days, showed very reduced or absent evoked responses on distal supramaximal stimulation of motor and sensory nerves, progressing rapidly to total loss of electrical excitability. This pattern was most consistent with findings observed in nerve fibres undergoing acute axonal degeneration.[51] Accordingly, patients showed severe, generalised muscle atrophy with delayed and very poor recovery. Examination of nerve tissue taken by biopsy early in the disease course and in two patients at necropsy after 1 month and 19 months from onset of the illness, disclosed severe axonal degeneration of motor and sensory nerve fibres with only scant lymphocytes and little demyelination. Changes extended to the most proximal portions of nerve roots, yet parent neurons were spared and retained the capacity for regeneration.[46] The pathological findings indicated a severe and probably primary insult to motor and sensory nerve axons and led to the concept of an acute axonal form of GBS.[49] The observations were subsequently confirmed and extended by Griffin and colleagues in detailed analysis and morphological study of similar case presentations from northern China.[52,53] The disorder was notable for the fulminant onset of severe paralysis and sensory deficits. Detailed immunopathology and examination of fine structure in very early disease stages provided strong evidence for a primary immune attack on nerve axons. Griffin and colleagues introduced the descriptive term now

generally used: acute motor-sensory axonal neuropathy (AMSAN).

Acute motor-axonal neuropathy

The concept of axonal variant forms of GBS was further supported by case reports of sporadic acute, purely motor-axonal neuropathies, now termed AMAN, which were triggered in many cases by an enteric infection with *Cjejuni.* Serum samples from such patients contained high titres of antibody to gangliosides (GM}, GDla, and GDlb) and these paralleled the clinical course.[50,54,55] Sporadic AMAN cases have been observed worldwide; they represent 10-20% of GBS patients in contemporary prospective series.[14] The term AMAN was introduced originally with the case descriptions of acute ascending paralysis that had been observed among rural children in northern China, occurring annually as a summer epidemic. 76% of Chinese AMAN cases were also seropositive for C*jejuni* and a substantial number had IgG antibodies to GM1.[17] Electro-physiological examination and necropsy in some cases confirmed a pure motor and axona neuropathy pattern.[41,52,53].

Electrophysiological studies showed a reduction or absence of distally evoked compound motor-action potentials--early signs of denervation on needle electromyography-but normal condttction velocities and normal action potentials in sensory nerves. These observations were also typical for sporadic

AMAN cases. The findings suggest that the axonal degeneration primarily involves the motor-nerve terminals. These predicted changes were demonstrated in muscle and nerve tissue from a sporadic AMAN case. The biopsy samples showed severe and selective loss of terminal motor axons, whereas the distal sensory fibres were completely intact.[56] Yet, in severe and advanced AMAN cases studied by detailed necropsy, the axonal pathology was much more severe and widespread. Motor axons were shown to have degenerated along their entire length.[41] The earliest demonstrable pathological change seemed to be the binding of IgG and activated complement components to the axolemma at nodes of Ranvier in large motor fibres.[52] Macrophages became attracted to such nodes and tracked underneath the detached myelin lamellae along the periaxonal space, dissecting the axon from the overlying Schwann cell and compact myelin. Axolemma, in contact with invading macrophages, was focally destroyed; axons showed progressive degenerative changes to the point of total disintegration.[53] In some patients, however, who had died early, the morphological changes were very scant despite severe clinical paralysis.

On the basis of these observations, the sequence of events has been postulated to take place as follows. C *jejuni* strains associated with the AMAN pattern of GBS are known to have in their liposaccharide membrane GM1-like epitopes that contain the Gal((31-3)GaINac moiety.[25] The host generates antibodies against GM1 or related gangliosides that bear Gal((31-3)GaINac, the

terminal disaccharide that is a candidate epitope. Axolemma at nodes of Ranvier and at terminal motor axons are enriched with Gal(pa-3)GaINac.[57,58] Binding of cross-reacting complement-fixing antibodies to these epitopes on axolemma might initially result in potentially reversible physiological failure of conduction without morphological change.[59,6o] Subsequent activation of complement could induce the observed early structural changes in nerve axons and initiate recruitment of macrophages, which then cause further axonal damage. Severity of axonal destruction might vary, depending on the vigorousness of the immune response; it could range from limited degeneration of motor terminals to generalised and more widespread Wallerian-like degeneration of motor fibres.[52] The time span of recovery would vary accordingly. Regeneration of motor-nerve terminals over the required short distance can happen quickly because the potential for nerve regeneration is probably greatest in childhood, which could explain the rapid recovery from paralysis in many children with AMAN and their overall good prognosis.[61]

Miller Fisher syndrome

Another variant form of GBS-the Miller Fisher syndrome (MFS)-has distinct immunological and pathological features. The MFS pattern is triggered by certain C*jejuni* strains that give rise to a characteristic pattern of antibodies to GQ1bganglioside.[22,23] IgG antibodies to GQ1bare seen in 96% of MFS cases

and parallel the disease course. The antibodies recognise epitopes that are expressed specifically in the nodal regions of oculomotor nerves, but also in dorsal-root ganglion cells and cerebellar neurons.[26,62] This pattern corresponds with the clinical features of ophthalmoplegia, ataxia, and areflexia. Anti-GQlb-containing serum from MFS patients interfered with neuromuscular transmission in a mouse phrenicnerve/diaphragm preparation, probably by blocking the release of acetylcholine from motor-nerve terminals.[63] The effect seemed specific, and may offer an explanation for the motor weakness seen in patients with MFS. Antibodies to GQlb cross-reacted with epitopes contained in the liposaccharide of MFS-associated C *jejuni* strains, again suggesting the possibility of molecular mimicry.[19]

There are other GBS variants which are relatively rare.[76]

Pure sensory variant

It is charectorised by a rare occurrence of acute sensory polyneuropathy with elevated csf proteins and demyelinating features on electrodiagnostic studies. There is a rapid onset of large fibre sensory loss with resultant sensory ataxia, positive Romberg sign, pseudoathetosis, tremor, lesser involvement of small fibre sensoryfunction. The important differential diagnosis

to be considered are Sjogren syndrome and paraneoplastic sensory ganglionopathy

Pure Dysautonomia

It is a rare variant of GBS, with initial symptoms pertaining to gastrointestinal tract such as abdominal pain, vomiting and diarrhea or constipation. There may be possible history of viral infection. Orthostatic hypotension and syncope may the disabling features. Although areflexia and mild sensory symptoms may be evident, there is no motor weakness. Routine electrodiagnostic studies are normal, hence autonomic testing such as heart rate variability, tilt-table testing, sympathetic skin response(SSR), and sweat testing (QSART) may be needed. Most people recover slowly after few months.

Pharyngo Cervico Brachial variant

It is a rare regional GBS variant, affecting predominantly, cervical, brachial or oropharyngeal muscles. Some studies have documented high titres of GT1a antibodies. Patients may initially suffer with neck and pharyngeal weakness which may involve later the upper but not the lower limbs. Electrodiagnostic studies may show demyelinating changes in the upper limbs.

Other less common entities are:

1. Paraparetic variant
2. Acral parasthesias with diminished reflexes in either arms or legs.
3. Facial diplegia or abducens palsies with distal parasthesias
4. Isolated post infectious ophthalmoplegia
5. Bilateral foot drop with upper limb parasthesias.
6. Acute ataxia without ophthalmoplegia.

Laboratory Studies

Cerebrospinal fluid studies

Approximately 90% of patients with GBS demonstrate spinal fluid protein elevation without leucocytosis at the time of maximal weakness. Though the range is broad, values greater than 1.0 gm/ dl are rare and suggest another diagnosis. Although there are usually less than 10 cells / mm3 spinal fluid, it is important to remember that a pleocytosis of 10-20cells/mm3 is seen in approximately 5% of patients and should not dissuade one from the diagnosis if the clinical and electrophysiological features are otherwise typical[77]. A spinal fluid cell count of more than 50 cells/mm^3 suggests infection with human immunodeficiency virus.

Electro diagnostic studies

AIDP

Several sets of electrodiagnostic guide lines for the identification of peripheral nerve demyelination in GBS have been published, and the number of patients diagnosed with AIDP can vary greatly depending on which criteria for demyelination are applied[78-80]. Majority of AIDP patients will fulfill the criteria by the end of fourth or fifth week, it is more important to have an appreciation for the earlier and sequential changes that are likely to be encountered in patients with AIDP. Conduction block is the hallmark of a demyelinating lesion accounting for the weakness and sensory loss in AIDP. Brown and Feasby found partial motor conduction block in one or more motor nerves in nearly three fourths of AIDP patients within 2 weeks of the onset of paralysis. To find this high frequency of partial motor conduction block, however, needle electrode stimulation at proximal sites is required. About 50% of AIDP patients demonstrate prolonged distal motor and F-wave latencies when first studied[80]. Conduction velocities in the demyelinating range occur mostly in third or fourth weeks.

Electromyographic findings depend on the extend and severity of axonal involvement. Early in the course, abnormal spontaneous activity is absent and motor unit potentials are normal. But volitional contraction may reveal a pattern of fast firing motorunits typical of neurogenic recruitment. Fibrillations

and sharp waves develop after the second week depending on the degree of axonal disruption.

AMSAN

Electrophysiological studies in patients with AMSAN are indicative of axonal loss at both acute and chronic stages. The characteristic feature is marked reduction in the compound muscle action potential amplitude or electrical inexitability of motor nerves, which can be found as early as 3-5 days of onset[46]. Sensory nerve action potential are also lost. Abundant fibrillation potentials and positive sharp waves can appear quite early.

AMAN

In patients with AMAN, the main abnormality in motor conduction studies is reduced compound muscle action potential amplitudes and absent F-wave responses.[83,84] Nerve conduction velocity, distal latency and F-minimum latency are normal. Partial motor conduction block or abnormal temporal dispersion is absent. Sensory nerve conduction studies are normal. Needle EMG examination shows fibrillations and positive sharp waves in the affected muscles by 2-3 weeks after the onset of weakness.

Course of illness

Most patients with AIDP become maximally weak within 11-12 days of onset and essentially all reach a nadir by 4 weeks.

Those with AMSAN and AMAN usually reach their nadir within 6 days. Occasional patients may have stepwise or stuttering course. Despite improvement in supportive and immunomodulating therapy, the mortality rate remains 3-5% for GBS with predominant weakness.

Prolonged disability occurs in a surprisingly high percentage of cases, especially in those with AMSAN. Many of these patients are still unable to walk, one year after the onset of their illness. Permanent disability, usually affecting the lower limbs and requiring arthrodesis of ankle and foot occur in about 10% of patients. A smaller percentage of patients may have residual disability, for years with wheelchair dependence and impaired quality of life.[85]

In a large series involving almost 300 patients, the mean time to onset of recovery was 28 days, while the mean time to complete recovery in those with a complete response was 200 days. Rates of clinical recovery at 12 and 24 weeks were 70% and 80% respectively. This indicates that about 20% of patients will have a recovery period extending beyond 6 months. The time and extend of

recovery are similar for both AMAN and AIDP.[61] Whereas patients with AMSAN usually have more prolonged periods of recovery and more severe neurological residual deficits.

Approximately 10% of GBS patients may have a malignant course characterized by prolonged stays in the intensive care units, ventilatory dependence (extending 4-6 months) and longer periods of rehabilitation. These patients usually have AMSAN, with rapid onset of quadriplegia, severe axonal changes with reduced motor action potentials.

In general poor prognostic factors identified are[75]

1. Older age.
2. Rapid onset prior to presentation.
3. Ventilator dependence.
4. Inexcitable or reduced amplitude motor evoked responses.
5. No treatment.
6. Preceding diarrheal illness

In order to document the stage of illness and to assess a particular effect of treatment appropriate scales has to be applied. In GBS studies the 7 point Hughes GBS disability scale is the most popularly used. Modified Rankin's

disability scale, MRC disability scale and functional evaluation by Barthel Index are also used for disability assessment.

The following are some of the scales used.

Guillain Barre Syndrome disability Scale[86]

0 Healthy

1 Minor symptoms or signs of neuropathy but capable of manual work/capable of running.

2 Able to walk without support of a stick (5m across an open space), but incapable of manual work or running.

3 Able to walk with a stick, appliance or support (5m across an open space).

4 Confined to bed or chairbound

5 Requiring assisted ventilation (for any part of the day or night)

6 Death

MRC Disability Scale[87]

0 Normal

1 No disability, minor sensory signs or areflexia.

2 Mild disability; ambulatory for 200m; mild weakness in one or more limbs and sensory impairment.

3 Moderate disability; ambulatory for 50m without stick;moderate weakness MRC grade 4 and sensory impairment.

4 Severe disability; able to walk 10m with support of stick; motor weakness MRC grade 4 and sensory impairment.

5 Requires support to walk 5m; marked motor and sensory signs.

6 Cannot walk 5m, able to stand unsupported and able to transfer to wheelchair, able to feed independently.

7 Bedridden, severe quadriparesis; maximumstrength MRC grade3.

8 Respirator and/or severe quadriparesis; maximumstrength MRC grade 2.

9 Respirator and quadriplegia.

10 Death.

Rankin's disability scale[88]

1 No disability

2 Slight disability;unable to carryout some previous activities but looksafter own affairs without assistance

3 Moderate disability; needs some help but walks without assisstence.

4 Moderately severe disability; unable to walk and do bodily care without help.

5 Severe disability; bedridden, incontinent; constant nursing care needed.

Treatment

Patients with GBS need to be admitted to hospital for close observation. Care for these patients is best provided in tertiary centers, with intensive-care facilities and a team of medical professionals who are familiar with the special needs of GBS patients. The evolution and severity of the neuropathy is variable; it can happen with alarming speed so that intubation and mechanical ventilation may be necessary 24-48 h from onset of symptoms. Admission to an intensive-care unit and ventilatory support is needed in 33% of GBS patients, who will often also show haemodynamic instability and autonomic dysfunction. Utmost vigilance and anticipation of potential complications are necessary to optimise the chances of a favourable outcome. Areas to be aware of include: prevention of thromboembolic complications; online cardiac monitoring; serial assessments of the ventilatory reserve, oropharyngeal weakness, and airway protection; appropriate bowel care and pain management; adequate nutrition, and psychological guidance and support.

Progression of disease varies in duration: about 75% of patients reach their nadir within 2 weeks; 92% within 3 weeks and 94% within 4 weeks.[64] After a brief plateau phase, improvement begins with gradual resolution of paralysis over weeks to months. Outcome is generally favourable. An epidemiological survey in 1993-94 of 140 GBS patients in southeast England

showed that 70% had made a complete recovery 1 year later, 22% were unable to run, and 8% were unable to walk unaided. In this series, ten patients (7%) died and three patients remained bedridden or ventilator-dependent at 1 year; all 13 patients were over 60 years old.[65]Similar figures were reported in other series. Several clinical factors have been identified that assist in the early prediction of outcome. The most reliable indicators for significant residual disability at 12 months from onset are: age over 60 years, rapid disease progression to quadriparesis in less than 7 days, need for ventilator support, and a mean distal motor amplitude of less than 20% of the lower limit of normal. A preceding diarrhoeal illness adds to a poor prognosis.13 The mortality rate remains at 5-8%, even with the most modem intensive-care medicine.[66] Prognosis is better in children, who need less time on ventilation and show a more rapid recovery from paralysis.[67]

Plasma exchange

Three large, multicentre, controlled trials have demonstrated unequivocal benefit from plasma exchange when it is used within the first 2 weeks of disease. In the North American trial of 245 patients with severe GBS, 122 patients were randomly assigned plasma exchange (five exchanges of 50 mL/kg bodyweight each, given over 7-14 days), and 123 were assigned conventional treatment. On average, patients treated by plasma exchange improved more

rapidly, could be weaned from assisted ventilation earlier (24 vs 48 days), and reached ambulation 1 month earlier (53 vs 85 days). This meant a considerable saving, because patients spent less time in intensive-care units and hospital. Plasma exchange was ineffective when started later than 2 weeks from onset of symptoms. These results were corroborated by two French studies that also established that plasma exchange is beneficial in milder GBS and that it improves long-term outcome. At 1 year, 71% of patients treated by plasma exchange recovered full motor strength, as opposed to 52% of controls.[68,69] Observations were consistent and reproducible, which addresses the criticism that has been voiced over the lack of a sham-pheresis control group and thereby the lack of allocation concealment.

Within 1-2 weeks of initial improvement after plasma exchange, secondary worsening may be seen in about 10% of patients.[70] These limited relapses may be due to persistent active disease or to antibody rebound; additional treatments by plasma exchange lead to renewed improvement.[71] The current recommendations are to use two plasma-exchange treatments for mild GBS and four or five for severe GBS, starting as soon as possible on a schedule of alternating days. Should the patient show secondary worsening, it is usually best to resume additional plasma-exchange treatments, although intravenous infusion with Ig may be used as an alternative. Plasma exchange is reasonably safe, but not totally free of risk, particularly in haemodynamically unstable GBS

patients. Such risks, the high cost, and the limited availability of plasma exchange facilities prompted the search for alternative treatments.

High-dose intravenous immunoglobulin

Intravenous Ig is a promising therapy in various disorders with a presumed autoimmune basis, and has the advantage of low risk and ease of application. This therapy was therefore introduced as an alternative to plasma exchange. The two treatments were compared for their effectiveness in a multicentre study of 150 GBS patients in the Netherlands.[72] Intravenous Ig was given at a dose of 0-4 g/kg bodyweight for 5 days consecutively, and plasma-exchange treatments followed the conventional schedule. At 4 weeks significantly more patients showed functional improvement with intravenous IgG (p=0-024) and the investigators concluded that the two treatments were of equal efficacy. However, the two groups were not equally matched and the study lacked masking. Therefore, these two treatments were assessed again in a large, multicentre, randomised trial coordinated by Hughes.[73] Plasma exchange was compared with intravenous IgG (Sandoglobulin, 0-4 g/kg bodyweight for 5 days) and with a combined treatment of plasma exchange (five times over 10-14 days), followed by intravenous IgG (0-4 g/kg bodyweight for 5 days) in 379 adult patients with severe GBS. At 4 weeks from randomisation, the functional disability-measured by a seven-point disability scale--was assessed by an

observer unaware of treatment allocation. On analysis, the three groups did not differ significantly in this outcome criterion, nor did they differ significantly in any of the secondary outcome measures (time to recover unaided walking; time to discontinue ventilation; recovery from disability during 48 weeks). The study concluded that plasma exchange and intravenous IgG had equivalent efficacy and that combination of the two treatments did not confer a significant advantage. Because of the ease of application, intravenous IgG is currently the preferred treatment of GBS. Limited relapses may also be observed in about 10% of patients treated with intravenous IgG; most respond equally well to a series of repeated infusions.

Corticosteroids

Contrary to expectation, corticosteroids proved to be of no benefit in GBS. In a large, double-blind, placebo-controlled, multicentre trial of methyl prednisolone (500 mg intravenously for 5 days within 2 weeks of onset) versus placebo, the groups did not differ significantly in any of the outcome measures. A continuing, multicentre trial in the Netherlands is examining the effects of intravenous IgG alone or with high-dose intravenous methyl prednisolone. The study is based on pilot observations that suggested a beneficial interaction between intravenous IgG and steroids.[74]

Without doubt, there will be advances in treatment as the pathogenesis of GBS is further elucidated. These advances should address the needs of the patients who are left with severe motor sequelae 1 year from onset- about 20% of the total. Recovery of these patients depends on axonal regeneration, which might be promoted by administration of nerve-growth factors. Immunoregulatory cytokines that are involved in terminating the disease process are being discovered and may become useful in treatment. More importantly, preventive measures that control, or even eliminate certain C*jejuni* infections will be the best means to lower the incidence of GBS.

MATERIALS AND METHODS

MATERIALS AND METHODS

This is a prospective study, conducted during the period of January 2006 to December 2007. Those patients who had been admitted with the diagnosis of GBS, in the medical, neuromedical, emergency medical or intensive medical care unit of Madras Medical College in this period were included in the study. The approval of the Ethical committee of our institute was obtained for the conduction of the study.

The inclusion criteria consists of patients who presented with features of GBS based on Asbury's criteria which included ascending areflexic quadriparesis, with or without cranial nerve dysfunction, evolving within a period of four weeks. We also included patients who presented with features of GBS subtypes without prominent weakness.

The exclusion criteria consists of

1. Early and prominent bladder and bowel dysfunction
2. Marked and persistent asymmetry of symptoms and signs
3. Presence of persistent sharp sensory level
4. Features of other diseases like myasthenia gravis, botulism, poliomyelitis, porphyria and diphtheria
5. Drug or toxin induced acute neuropathy.

Data regarding the demographic features like age, sex distribution and month of occurrence, clinical features like antecedent illness, involvement of cranial nerves and autonomic dysfunction were collected.

Electro diagnostic studies were performed on patients using the NMS machine. As far as possible bilateral median, ulnar, tibial and peroneal motor and F-waves and median, ulnar and sural sensory conduction were done on all patients. The amplitude and latency of CMAP (Compound Muscle Action Potential) and SNAP (), conduction velocity of motor and sensory nerves and persistence and minimum latency of F-waves were recorded. Nerve conduction studies helped to confirm and categorize the diagnosis and subtypes of GBS.

The basic biochemical and clinicopathological tests and chest x ray were done for all patients. Cerebrospinal fluid analysis was done whenever possible and relevant. Investigations like liver function tests, thyroid function test, CPK, HbsAG, HIV and ABG were done on patients as per the need.

During admission patients were analyzed for their disability using the GBS disability scale and MRC disability scale. For patients with disability grade of > 3 in GBS disability scale and for those with progressively increasing weakness, the definite treatment options (IvIg or plasma exchange) were started. Due to non-availability some patients received only injection methyl prednisolone.

Patients were followed up throughout their stay in the hospital. Intensive medical care was provided for those patients with advanced stage of disease. Elective intubation was done for those patients who had poor single breath count estimation and reduced peak expiratory flow rate and for those with neck muscle weakness and poor cough reflex. Ventilatory support was provided for those in need. Tracheostomy was performed on those patients who tend to require ventilatory support for more than 10 – 14 days.

Complications of prolonged immobility, ventilation, drug side effects and catheterization like aspiration and ventilator associated pneumonia, pressure sores, UTI psychosis and DVT were noted and treated accordingly.

Periodic assessment of their clinical status and disability was done and their peak disability was noted. At the end of 8 weeks duration, reassessment was done in their clinical status and prevailing disability score was noted for further analysis.

Microsoft excel was used to record the data obtained. The SSEP package was used for statistical analysis. The mean and standard deviation was calculated for certain variables, which follow normal distribution. The association of two categorical variables was evaluated by chi-square tests. The significance of association of certain factors like the treatment adopted and poor prognosticators with the outcome variables like death, ventilator need, tracheostomy and bedridden state was measured by stepwise logistic regression analysis. Statistical significance was considered when the p-value was < 0.05. The mean improvement of disability score was calculated for each of the treatment modality.

RESULTS

RESULTS

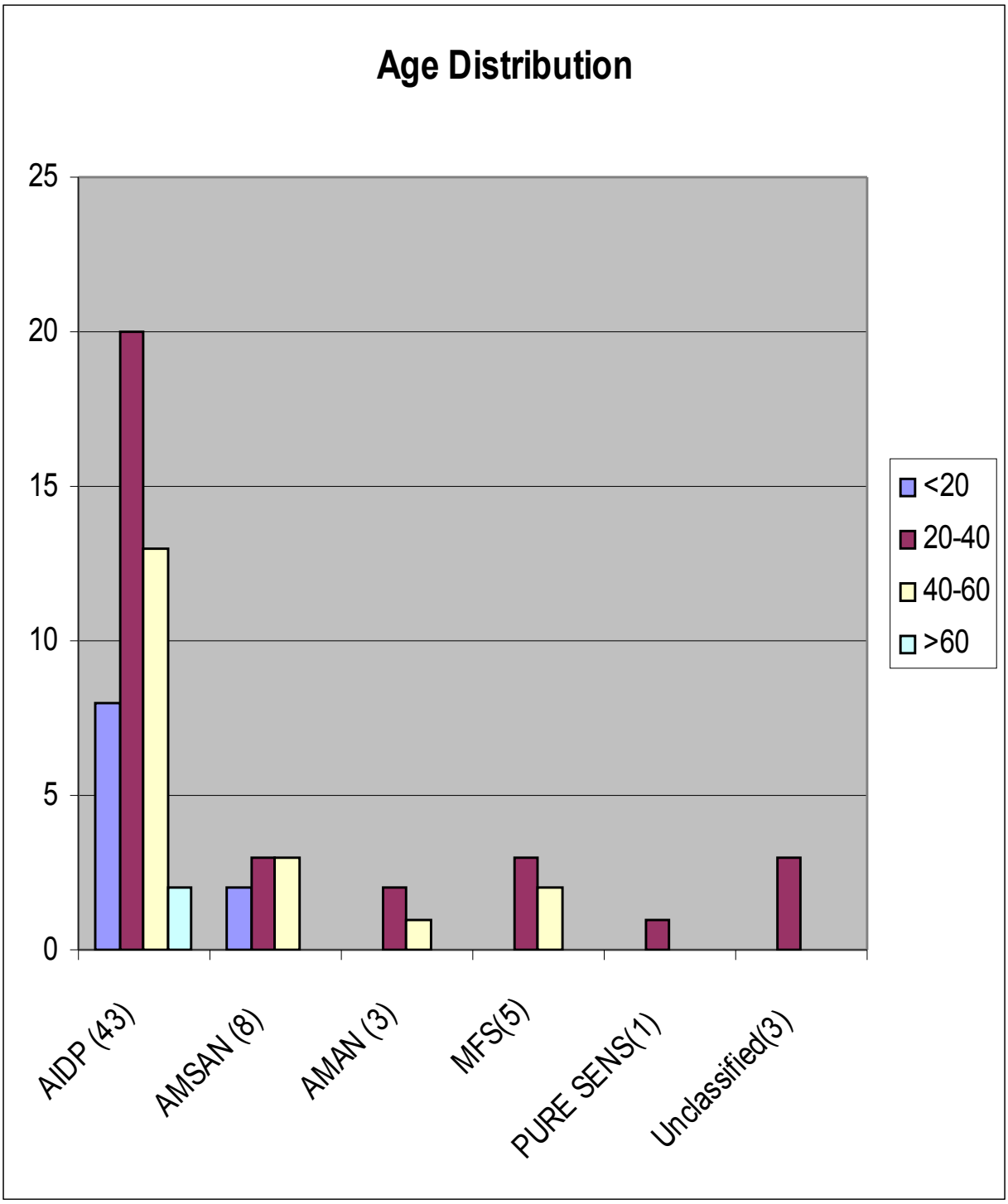
In our study, totally 63 patients were registered from January 2006 to December 2007. These patients included those who were admitted in Medical Wards, Emergency Wards, Intensive Medical Care Unit and in the Neurology Ward.

Age distribution

Out of the 63 patients, 20 patients were in the age group below 20 years, 32 patients were in the age group between 20-40 years, 19 patients were in the age group between 40-60 years. Only 2 patients were in the age group above 60 years. The distribution is also reflected in the subtypes of GBS. The following table and diagram illustrate the details.

Table-1 Age distribution

Age Group	<20	20-40	40-60	>60
Total - 63	10 (15.8%)	32 (50%)	19 (30%)	2 (4%)
AIDP [43]	8	20	13	2
AMSAN [8]	2	3	3	-
AMAN [3]	-	2	1	-
MFS [5]	-	3	2	-
Pure Sens [1]	-	1	-	-
Unclassified [3]	-	3	-	-


Age Distribution
25
20
15
10
5
0
AIDP (43)
AMSAN (8)
AMAN (3)
MFS(5)
PURE SENS(1)
Unclassified(3)
<20
20-40
40-60
>60

Sex distribution

Regarding the sex distribution there is a slight male preponderance of 35 males and 28 females among the 63 patients totally registered. Similar feature is noted in the subtypes also.

Table-2 Sex Distribution

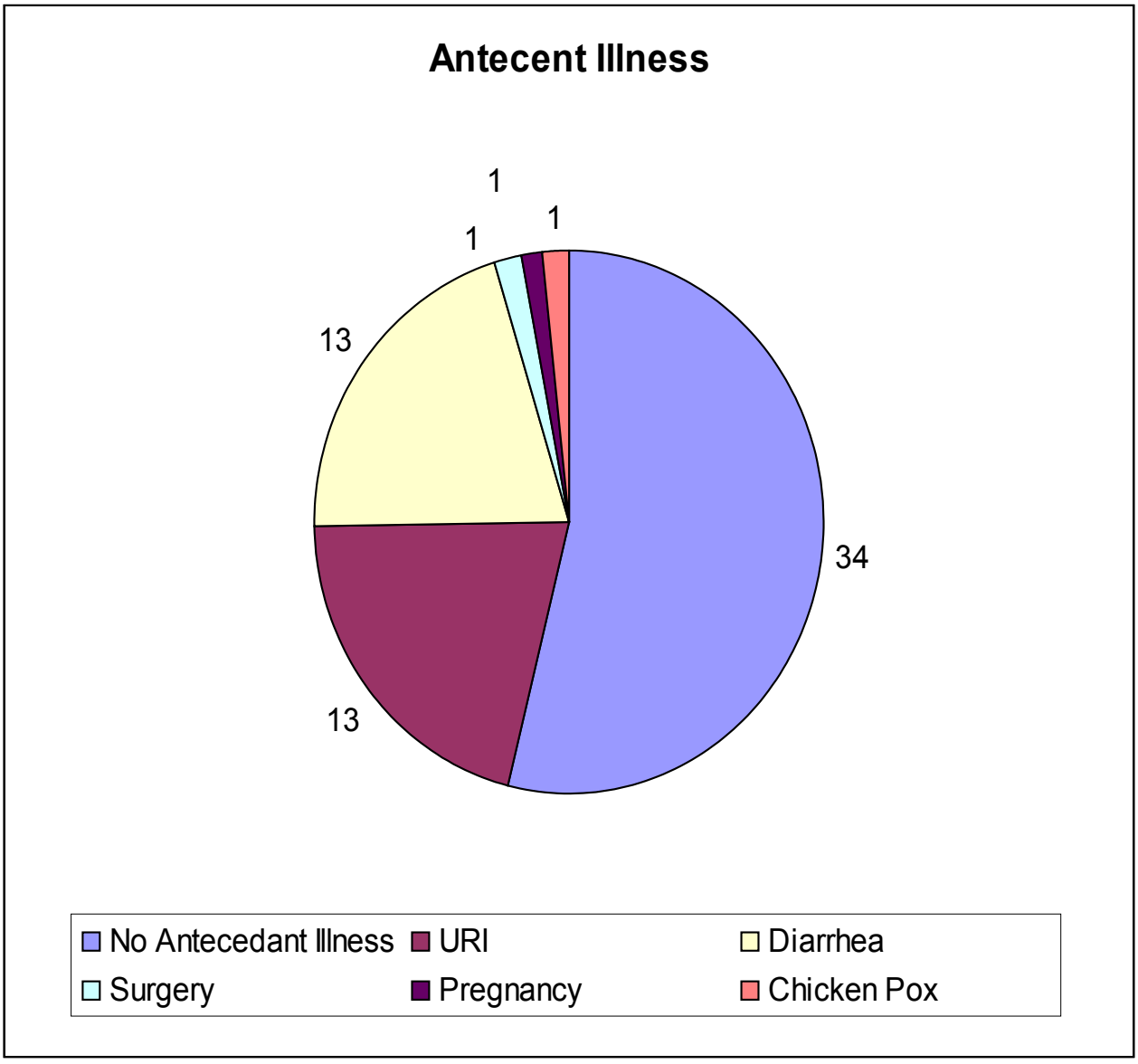
Total - 63	Male [35] (56%)	Female [28] (44%)
AIDP [43]	23`	20
AMSAN [8]	5	3
AMAN [3]	2	1
MFS [5]	3	2
Pure Sens [1]	-	1
Unclassified	2	1

Antecedent Illness

29 patients in the study had history of antecedent illness preceded by the occurrence of GBS. Upper respiratory infection and diarrhea were noted in each of 13 patients. One patient had undergone appendicectomy and 1 had chicken-pox infection. One patient was pregnant during the occurrence of the illness. The following table shows the distribution among the subtypes of GBS.

Table-3 Antecedent illness

Total 29/63 (46%)	URI [13] 21%	Diarrhea [13] 21%	Surgery [1]	Others [2]
AIDP [21] (48%)	20	8	1 (Appendicectomy)	1-pregnancy 1-chicken pox
AMSAN [4] (50%)	2	2	-	-
AMAN [2] (66%)	-	2	-	-
MFS [1] (20%)	1	-	-	-
Unclassfied [1] (33%)	-	1	-	-


Antecent Illness
1
1
1
13
34
13
No Antecedant Illness
URI
Diarrhea
Surgery
Pregnancy
Chicken Pox

Cranial Nerve Involvement

Uni or bilateral facial nerve involvement was observed in 22 patients and bulbar weakness was observed in 13 patients. 8 patients had both the features. Oculomotor weakness was noted in 5 patients who belonged to Miller Fischer Syndrome group. The following table and diagram shows the details.

Table-4 Cranial Nerve Involvement

Total 32 (42.9%)	Facial Only [14]	Bulbar Only [5]	Both (F+B) [8]	Oculomotor [5]
AIDP [21]	12	5	4	-
AMSAN [2]	1	-	1	-
AMSAN [1]	1	-	-	-
MFS [5]	-	-	-	5
Pure Sens [0]	-	-	-	-
Unclassified [3]	-	-	3	-

Autonomic Dysfunction

Autonomic disturbances, which are generally considered bad prognosticators were noted in totally 27 patients. They were tachy or bradycardias, heart blocks and postural hypotension. Table 5 shows the details.

Table-5 Autonomic Dysfunction

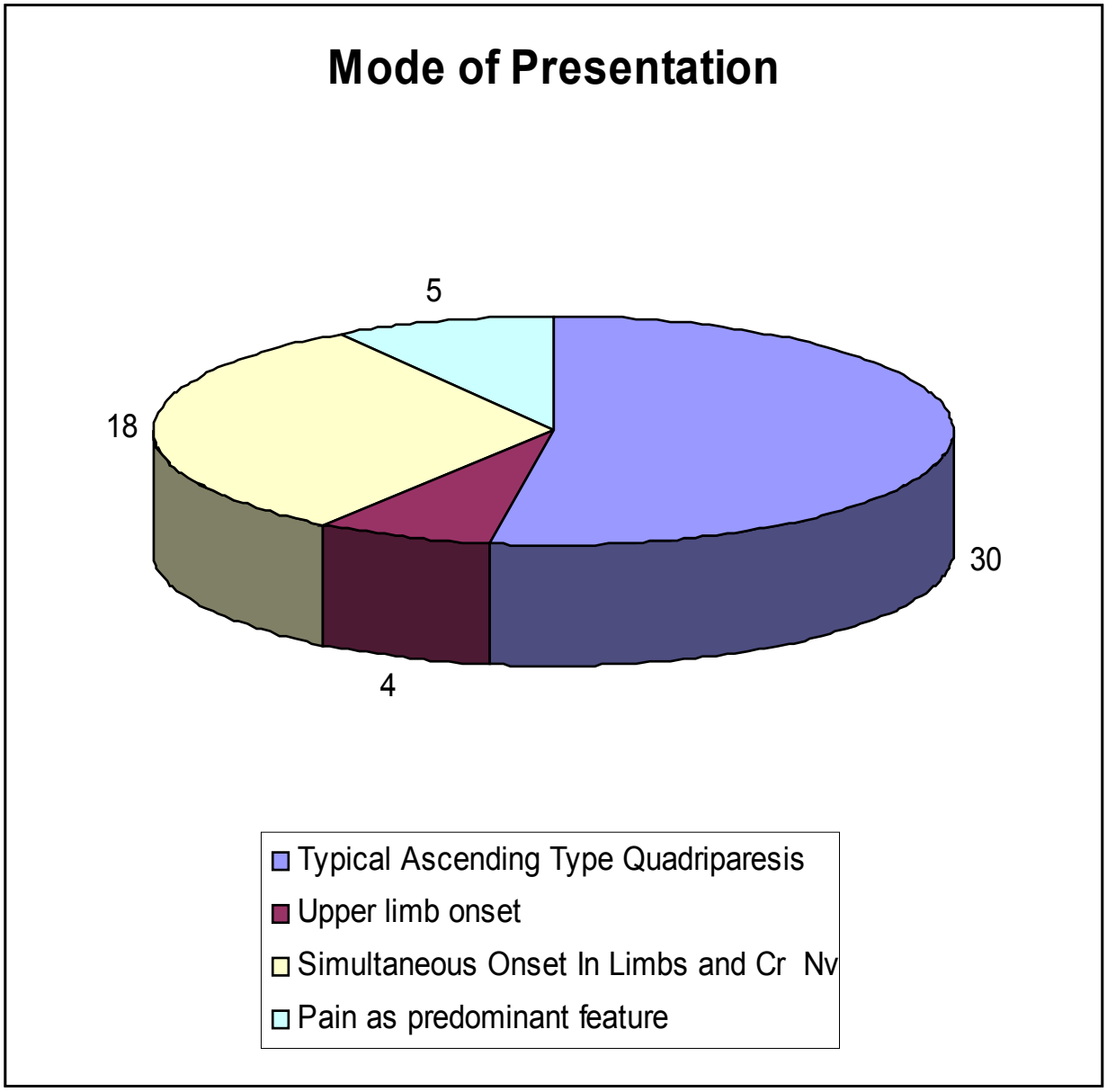
Total-23	Tachycardia [17]	Brady Cardia [8]	Postural Hypotension [5]	Heart Block [3]	Sudden Cardiac Death [1]
AIDP [18]	13	3	2	1	1
AMSAN [5]	2	3	1	1	-
AMAN [1]	-	1	-	1	-
MFS [1]	-	1	-	-	-
Pure Sens [0]	-	-	-	-	-
Unclassified [2]	2	-	2	-	-

Clinical features

Among the 63 patients totally registered, 35 had typical onset of ascending quadriparesis with areflexia. 4 patients had onset of their symptoms in upper limb. In 18 patients, the onset of weakness was simultaneous in all 4 limbs and in cranial nerves. Pain was the predominant presenting feature in 5 patients though they had weakness and areflexia. Positive sensory parasthesias like pins and needles sensations were noted in 8 patients. The following table gives details about the presenting feature.

Table-6 Mode of Presentation

	Typical Ascending Type Quadriparesis	Upper limb onset	Simultaneous Onset In Limbs and Cr Nv	Pain as predominant feature	presence of sensory parasthesia
AIDP	23	3	6	5	8
AMSAN	4	1	3	-	-
AMAN	-	-	3	-	-
MFS	3	-	2	-	-
Pure Sens	-	-	1	-	-
Unclassified	-	-	3	-	-


Mode of Presentation
5
18
30
4
Typical Ascending Type Quadriparesis
Upper limb onset
Simultaneous Onset In Limbs and Cr Nv
Pain as predominant feature

Month wise distribution

Table 7 details the occurrence of GBS in various months which shows that maximum cases are noted in June to October months.

Table-7 Distribution in months

	J	F	M	A	M	Jn	Jly	A	S	O	N	D
Total	4	5	4	4	4	6	6	8	8	8	3	3
AIDP	3	2	1	3	4	6	6	3	5	6	2	2
AMSAN	-	2	1	-	-	-	-	2	-	2	1	-
AMAN	-	-	-	-	-	-	-	1	1	-	-	1
MFS	1	-	1	1	-	-	-	1	1	-	-	-
Pure Sens	-	1	-	-	-	-	-	-	-	-	-	-
Unclassified	-	-	1	-	-	-	-	1	1	-	-	-

CSF Analysis

CSF analysis was done in 11 patients in the first week and in 24 patients in the second week. CSF analysis was normal in 10 patients during 1st week and in 24 patients during the 2nd week period.

Table-8 CSF results

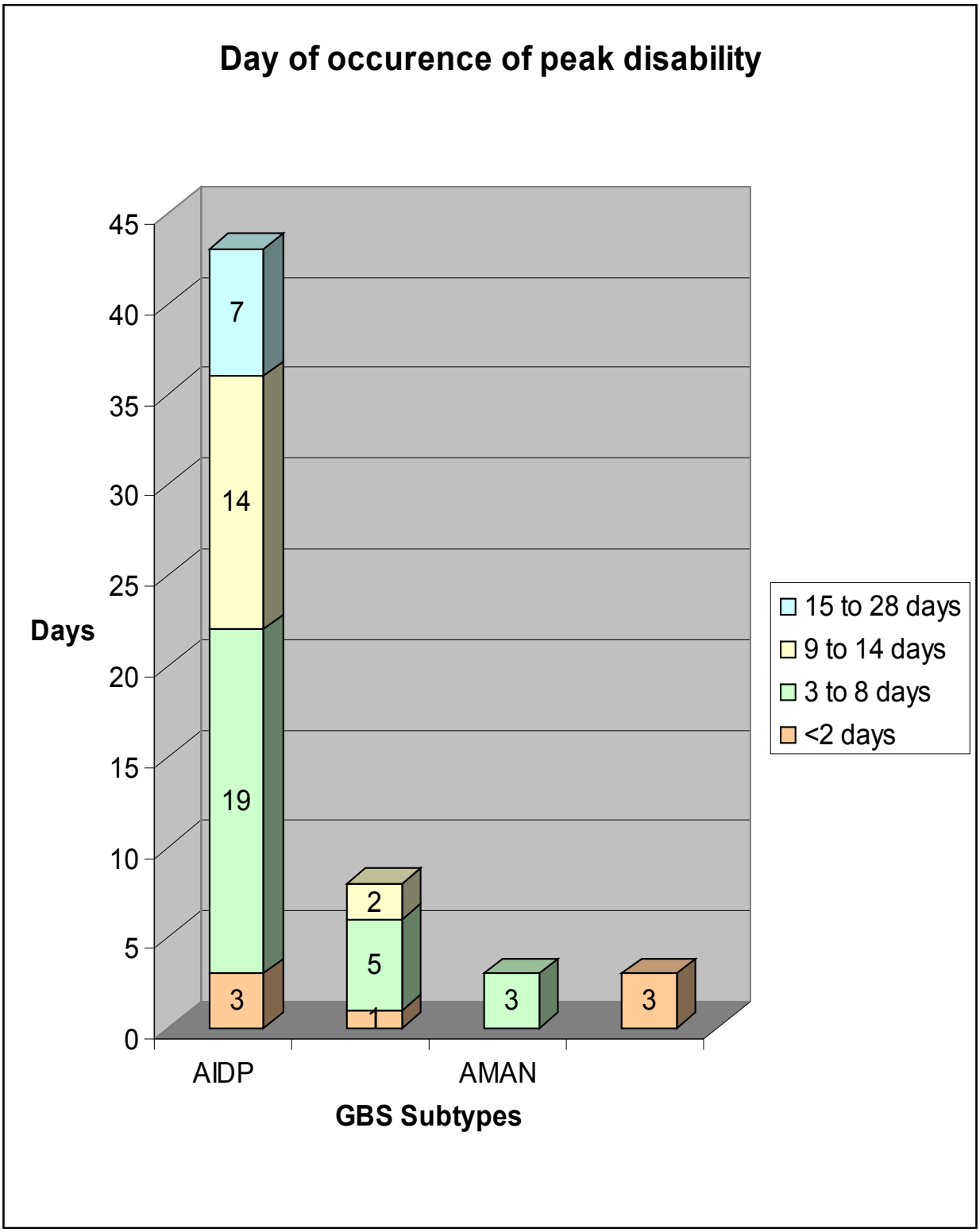
Total – 35/63	Done in I Week [11]		Done in II week [24]	
	Normal	Abnormal	Normal	Abnormal
AIDP	8	1	8	7
AMSAN	-	-	3	1
AMAN	-	-	1	1
MFS	2	-	2	1
Pure Sens	-	-	-	-
Unclassified	-	-	-	-

Peak disability after onset of symptoms (in days)

The timing of occurrence of peak disability from the onset of symptoms was noted in all patients. Generally the rapidity of attaining peak disability is a poor prognostic sign. It was grouped in 4 categories i.e. <2, 3-8, 9-14 and 15-28 days.

Table-9 Peak disability after onset of Symptoms
(in days)

	<2 [7] (11%)	3-8 [29] (46%)	9-14 [20] (32%)	15-28 [7] (11%)
AIDP	3	19	14	7
AMSAN	1	5	2	-
AMAN	-	3	-	-
MFS	-	1	4	-
Pure Sens	-	1	-	-
Unclassified	3	-	-	-


Day of occurence of peak disability
Days
45
40
35
30
25
20
15
10
5
0
7
14
19
3
2
5
1
3
3
AIDP
AMAN
GBS Subtypes
15 to 28 days
9 to 14 days
3 to 8 days
<2 days

Nerve conduction study parameters

The following table details the various nerve conduction study abnormalities noted in different types of GBS. Only in 55 out of 63 patients Nerve conduction study was possible and in the rest of the patients, the study was deferred either due to early death or difficulty to mobilize. Conduction block was noted in 19 cases and F-wave abnormality in the form of either impersistance or prolongation of minimal latency was noted in 48 patients. Distal latency prolongation was noted in 39 patients.

Table-10 Nerve Conduction Study Parameters

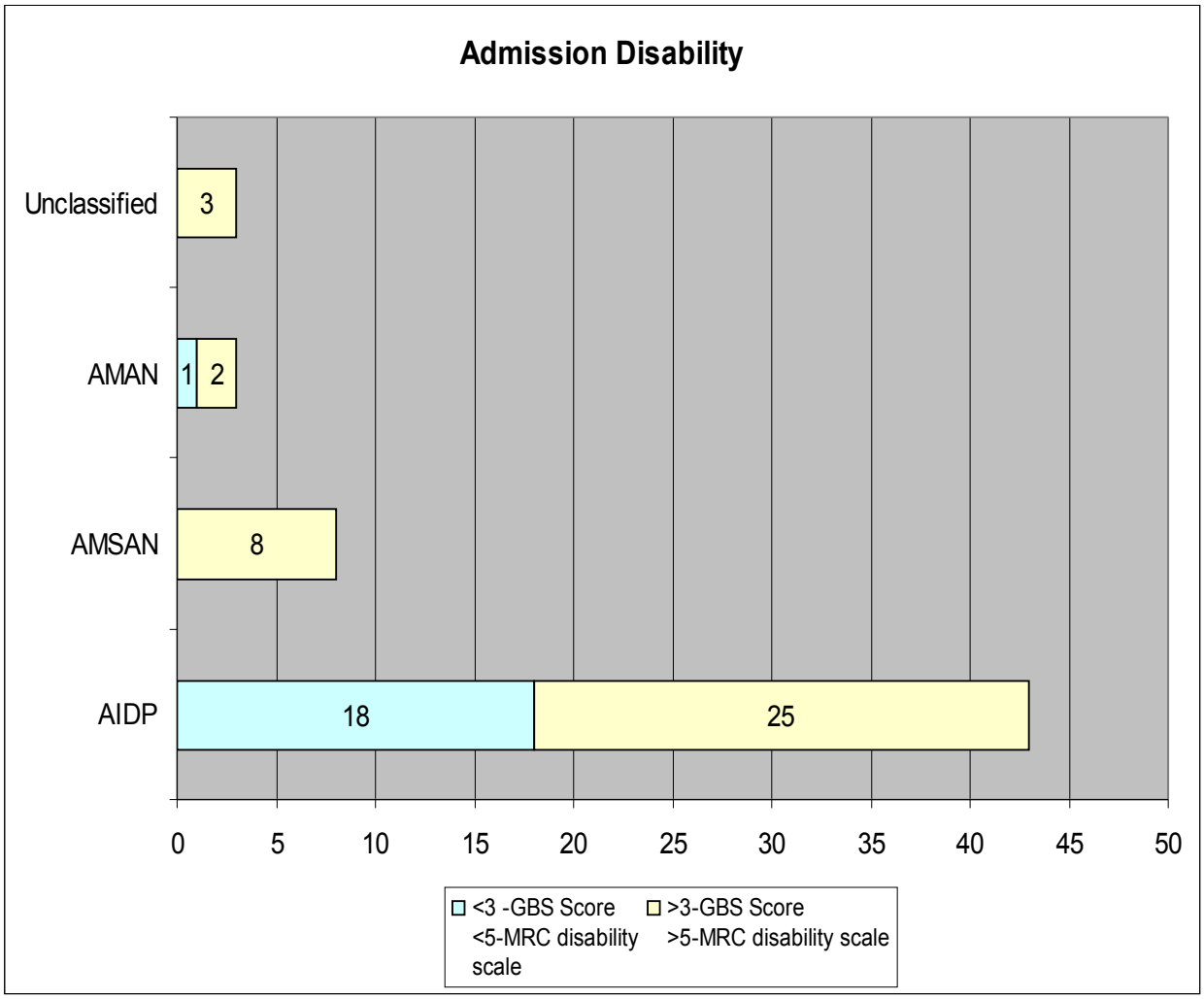
	Conduction Block (>2.Nerves) [19] (34.5%)	F-Wave Abnormality [48] (87.2%)	Distal latency Prolongation [39] (70%)	CMAP [30] (54.5%)	SNAP [11] (20%)
AIDP [40]	19 (47.5%)	37 (92.7%)	39 (97%)	21 (52.5%)	-
AMSAN [6]	-	6 (100%)	-	6/6	6/6
AMAN [3]		3	-	3	-
MFS [5]	-	2	-	-	4
Pure Sens [1]	-	-	-	-	1
Unclassfied [0]	-	-	-	-	-

Severity of disability with which patient admitted

In the present study, 25 patients were admitted with disability grade of >3 in GBS disability scale (>5 in MRC disability scale) and 18 were admitted below the score. The score is applicable for the GBS subtypes which cause weakness. The following table and figure shows the details.

Table-11 Severity of disability with which patient presented

		<3 -GBS Score <5-MRC disability scale [19] (30.2%)	>3-GBS Score >5-MRC disability scale [38] (60.3%)
AIDP	43	18	25
AMSAN	8	-	8
AMAN	3	1	2
Unclassified	3	-	3


Admission Disability
Unclassified
AMAN
AMSAN
AIDP
3
1
2
8
18
25
0
5
10
15
20
25
30
35
40
45
50
<3 -GBS Score <5-MRC disability scale
>3-GBS Score >5-MRC disability scale

Treatments given for subtypes

In our study IvIg was given to 18 patients, plasma exchange was given for 9 patients and injection methyl prednisolone was given for 25 patients. No specific treatment was provided for 4 patients who presented with very minimal disability and they improved spontaneously. Three patients in the study presented in a very acute form, with severe disability scores and died before any specific form of treatment was initiated.

Table-12 Treatments given for various subtypes of GBS

	IvIg [22] (34.9 %)	Methyl Prednisolone [25] (39.7 %)	Plasma Exchange [9] (14.3 %)	No Treatment [4] (6.3 %)	Not Applicable [3] (4.8 %)
AIDP [43]	18	15	6	4	-
AMSAN [8]	3	3	2	-	-
AMAN [3]	1	1	1	-	-
MFS [5]	-	5	-	-	-
Pure Sens [1]	-	1	-	-	-
Unclassified [3]	-	-	-	-	3

Subtypes of GBS patients who needed ventilator or ended up in death

In the present study, 16 patients needed ventilator support and 10 patients expired. Both the need for ventilator support and occurrence of death, are noted in AIDP and AMSAN group and in those patients who presented with very severe form of illness (fulminant form of illness).

Table-13 Subtypes of GBS patients who needed ventilator or ended up in death

	Ventilator [20] (31.7 %)	Death [10] (15.9 %)
AIDP	13	5
AMSAN	4	2
AMAN	-	-
MFS	-	-
Pure Sens	-	-
Unclassified	3	3

Among the 63 patients, death was the outcome for 10 patients. The following table shows the incidence of poor prognostic factors among the 10 patients who had expired.

Table-14 The poor prognosticators associated with the patients who had expired

Total -10	**%**	**Ventilator [10]**	**Autonomic disturbance [9]**	**Peak of weakness ≤ 8 days [10]**	**Bulbar dysfunction [9]**	**Elderly Population [2]**	**High grade disability* at presentation [9]**	**Diarrhea [4]**
AIDP [5]	11.6	5	4	5	3	2	4	2
AMSAN [2]	25	2	2	2	1	-	2	1
AMAN [0]	-	-	-	-	-	-	-	-
MFS [0]	-	-	-	-	-	-	-	-
Pure Sens [0]	-	-	-	-	-	-	-	-
Unclassified [3]	100	3	3	3	3	-	1	1

* > 3 score in GBS disability grading
  > 5 score in MRC disability grading

Twenty patients in our study needed ventilator support at sometime during the course of hospital stay. Traditionally considered poor prognostic factors not only influence death, but also leads to poor respiratory function requiring ventilatory support. The following table shows the incidence of such factors among the ventilated patients.

Table-15 The poor prognosticators associated with the patients who required ventilatory support

Total	**%**	**Death [10]**	**Autonomic disturbance [13]**	**Bulbar Dysfunction [9]**	**Peak disability reached <8 days [16]**	**Elderly [2]**	**High grade disability at presentation* [19]**	**Diarrhea [7]**	**Tracheostomy [5]**
AIDP [13]	30	4	8	5	10	2	12	4	5
AMSAN [4]	50	2	3	1	3	-	4	2	0
AMAN	-	-	-	-	-	-	-	-	-
MFS	-	-	-	-	-	-	-	-	-
Pure Sens	-	-	-	-	-	-	-	-	-
Unclassfied [3]	10	3	2	3	3	-	1	1	0

* > 3 score in GBS disability grading
  > 5 score in MRC disability grading

In the study, the outcome assessment is based on whether the patients attained independent walking which is a good outcome (≤ 3 in GBS disability score and ≤ 5 in MRC disability scale) or did not attain independent walking which is a poor outcome, at the end of 8 weeks. Those who expired were also included in the poor outcome group. Apart from outcome assessment, ventilator dependence is also a complication related to the severity of illness. Tracheostomy denotes that the patient required prolonged ventilation again adding to the mortality and morbidity. These outcome related events are analyzed on the back ground of poor prognostic factors, which is shown in the Table 16.

Table-16 Outcome related events for patients with poor prognostic factors

		Death	Ventilator dependence	Tracheostomy	Poor Outcome*	Good Outcome**
Bulbar ± Facial Weakness	AIDP [21]	4	10	4	11	10
	AMSAN [2]	1	1	-	2	-
	AMAN [1]	1	-	-	1	-
p = 0.002#	Unclassified [3]	3	3	-	3	-
Autonomic dysfunction	AIDP [17]	4	6	2	8	9
	AMSAN [5]	2	2	-	3	2
	AMAN [1]	-	-	-	1	0
p = 0.029#	Unclassified [2]	2	2	-	2	0
Diarrhea	AIDP [8]	2	4	3	6	2
	AMSAN [2]	1	2	-	2	-
p = 0.165#	AMAN [2]	-	-	-	-	2
	Unclassified [1]	1	1	-	1	-
Peak disability reached within 8 days	AIDP [22]	5	8	2	11	11
	AMSAN [6]	2	4	-	3	3
	AMAN [3]	-	-	-	2	1
p = 0.044#	Unclassified [3]	3	3	-	3	-
Presented with severe form of disability	AIDP [24]	4	12	5	12	12
	AMSAN [8]	2	4	-	4	4
	AMAN [2]	-	-	-	1	1
p = 0.001#	Unclassified [3]	3	3	-	3	-

* > 3 score in GBS disability grading
  > 5 score in MRC disability grading

** ≤ 3 score in GBS disability grading
  ≤ 5 score in MRC disability grading

Significance of influence of prognostic factors for poor outcome

Among the 18 AIDP patients who were treated, 6 were from mild disability group and 12 were from severe disability group. Among the 15 AIDP patients who were treated with injection methyl prednisolone 9 were from severe disability and 6 were from mild disability group. Also 4 patients with severe disability group were treated with plasma exchange and 2 from mild disability group. Four patients with AIDP, who had mild disability were not treated with any specific form of treatment.

Three AMSAN patients of severe disability were treated with IvIg and methyl prednisolone and two by plasma exchange. All patients in the AMSAN group had presented with severe disability.

One AMAN patient of severe disability group was treated each with IvIg and plasma exchange and one with mild disability was treated with methyl prednisolone.

In the group of 3 patients who presented with very acute and severe form of illness, no effective treatment was started before they expired. The following table 17 illustrates the features.

Table-17 Treatment adopted in GBS subtypes

		IvIg	**Methyl Prednisolone**	**Plasma Exchange**	**Not applicable***	**No Treatment**
AIDP	Mild disability presentation [18]	6	6	2	-	4
	Severe disability presentation [25]	12	9	4	-	-
AMSAN	Mild disability [0]	-	-	-	-	-
	Severe disable [8]	3	3	2	-	-
AMAN	Mild disability [1]	-	1	-	-	-
	Sever disability [2]	1	-	1	-	-
Unclassified	Mild disability [0]	-	-	-	-	-
	Severe disability [3]	-	-	-	3	-

* Not applicable - Patients died on day 1 or 2 before any form of treatments are effective

In the IvIg treated AIDP group of 18 patients, 12 patients had good outcome and 6 had poor outcome. Also in this group one patient expired, 5 needed ventilatory support and one patient required prolonged ventilatory support as evidenced by the need for tracheostomy. In the IvIg treated AMSAN group of 3 patients one had poor outcome and 2 had good outcome. In the IvIg treated single AMAN patient the outcome was poor. In the plasma exchange treated AIDP group of 7 patients 2 had poor and 5 had good outcome.

In the plasma exchange treated AMSAN group of 2 patients 1 each had good and poor outcome. In the single AMAN patient treated with plasma exchange the outcome was good.

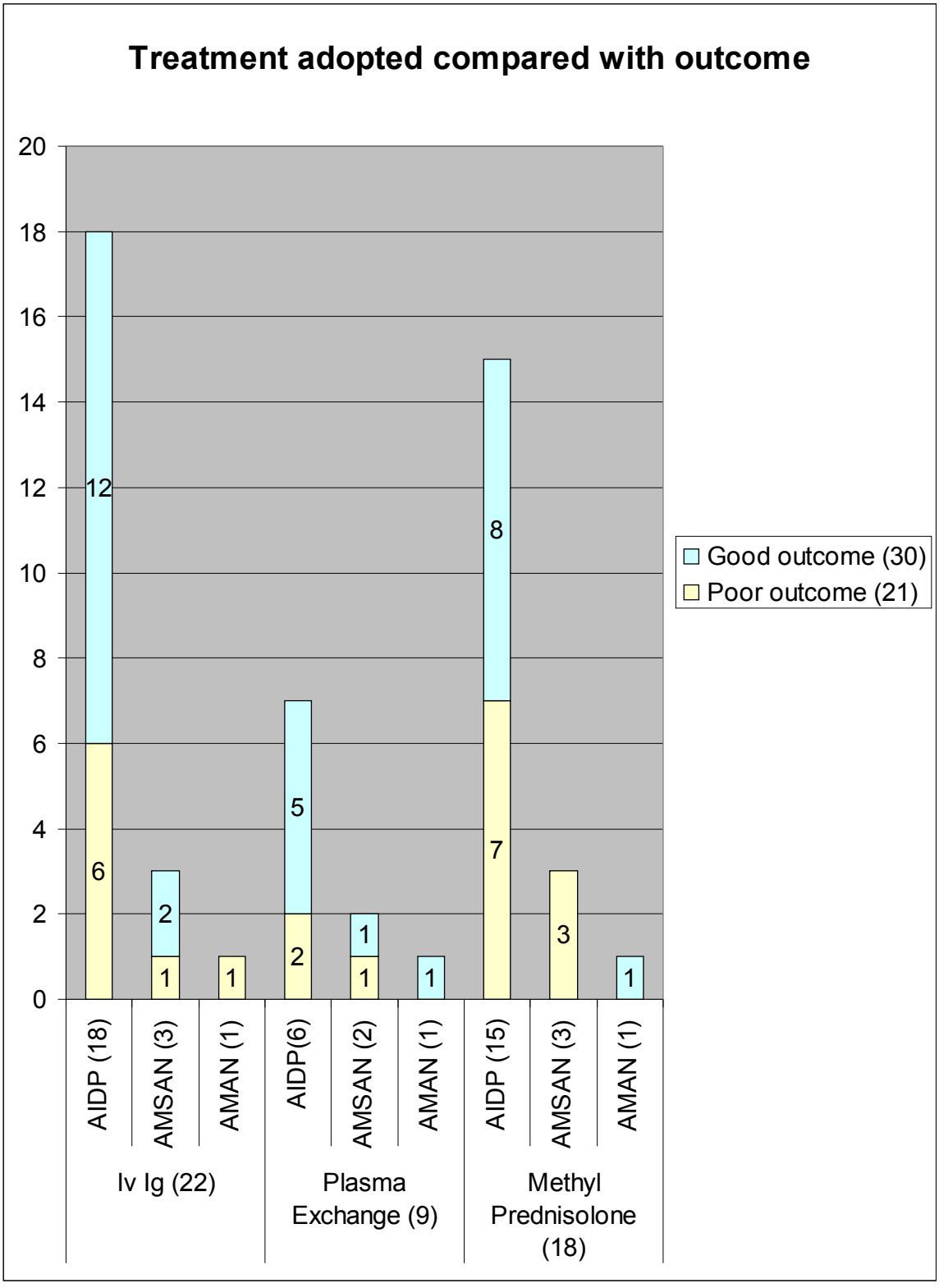
In the methyl prednisolone treated AIDP group of 15 patients, 7 had poor and 8 had good outcome, whereas 3 patients expired and 7 had ventilator support. The need for ventilator support was prolonged in 4 patients who had tracheostomy also. In the methyl prednisolone treated AMSAN group of 3 patients all had poor outcome and one required ventilator support, and death occurred in one patient. In the methyl prednisolone treated single AMAN patient the outcome was good.

The following table 18 shows the details.

Table-18 Comparison of treatment adopted with clinical outcome at 8 weeks among GBS subtypes

		Death [7]	**Ventilator Support [16]**	**Tracheostomy [5]**	**Poor outcome [21]**	**Good outcome [30]**
Iv Ig [22] p = 0.410#	AIDP [18]	1	5	1	6	12
	AMSAN [3]	-	-	-	1	2
	AMAN [1]	-	-	-	1	-
Plasma Exchange [9] p = 0.687#	AIDP [6]	1	1	-	2	5
	AMSAN [2]	1	2	-	1	1
	AMAN [1]	-	-	-	-	1
Methyl Prednisolone [18] p = 0.134#	AIDP [15]	3	7	4	7	8
	AMSAN [3]	1	1	-	3	-
	AMAN (1)	-	-	-	-	1

Significance of influence of treatment options for outcome


Treatment adopted compared with outcome
20
18
16
14
12
10
8
6
4
2
0
12
6
2
1
1
5
2
1
1
1
8
7
3
1
AIDP (18)
AMSAN (3)
AMAN (1)
AIDP(6)
AMSAN (2)
AMAN (1)
AIDP (15)
AMSAN (3)
AMAN (1)
Iv Ig (22)
Plasma Exchange (9)
Methyl Prednisolone (18)
Good outcome (30)
Poor outcome (21)

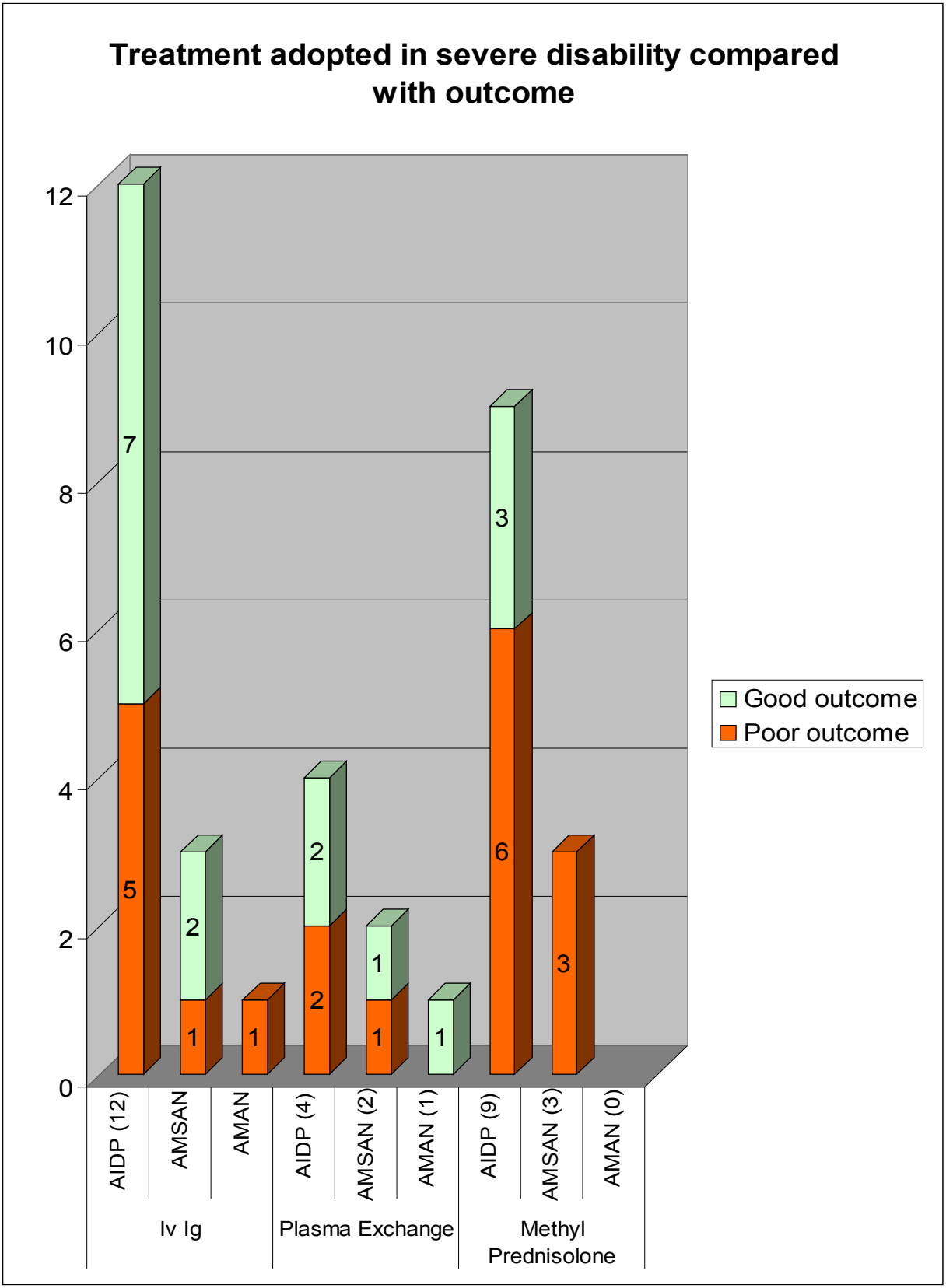
In the IvIg treated AIDP patients (12) who presented with severe disability the outcome was poor in 5 patients and good in 7 patients. Ventilator was needed in 4 patients and one had prolonged ventilatory support. In the IvIg treated AMSAN patients who presented with severe disability the outcome was poor for one and good for two. In the IvIg treated single AMAN patient who presented with severe disability the outcome was poor.

In the plasma exchange treated 4 AIDP patients who presented with severe disability 2 had poor and 2 had good outcome. One patient had expired in this group after a ventilator support. In the plasma exchange treated 2 AMSAN patients who presented with severe disability one each had poor and good out come. In the plasma exchange treated single AMAN patient with severe disability the outcome was good.

In the methyl prednisolone treated 9 AIDP patients who presented with severe disability the outcome was good for 3 and poor for 6 patients. In the methyl prednisolone treated 3 AMSAN patients who presented with severe disability the outcome was poor for all 3 patients.

Table-19 Comparison of treatment options with clinical outcome at 8 weeks among GBS subtypes who presented with severe disability

		Death	**Ventilator**	**Tracheo stomy**	**Poor outcome**	**Good outcome**
Iv Ig	AIDP [12]	-	4	1	5	7
	AMSAN	-	1	-	1	2
	AMAN	-	-	-	1	-
Plasma Exchange	AIDP [4]	1	1	-	2	2
	AMSAN [2]	1	1	-	1	1
	AMAN [1]	-	-	-	-	1
Methyl Prednisolone	AIDP [9]	3	6	3	6	3
	AMSAN [3]	1	1	-	3	-
	AMAN [0]	-	-	-	-	-


Treatment adopted in severe disability compared with outcome
12
10
8
6
4
2
0
7
5
2
1
1
2
2
1
1
3
6
3
Good outcome
Poor outcome
AIDP (12)
AMSAN
AMAN
AIDP (4)
AMSAN (2)
AMAN (1)
AIDP (9)
AMSAN (3)
AMAN (0)
Iv Ig
Plasma Exchange
Methyl Prednisolone

The mean improvement in GBS disability scores from admission to the end of 8 weeks was calculated for all GBS patients which is shown in the table 20 below.

Table-20 Mean improvement in disability scores for various treatment options

Disability Scale	Treatment Option	Total Numbers	Mean Improvement
GBS disability scale $p = 0.002^{\#}$	IvIg	22	1.09
	Plasma Exchange	9	0.67
	Methyl prednisolone	19	0.00
	No Treatment	4	1.00
	Not Applicable	3	-1.00
MRC disability scale $p = 0.000^{\#}$	IvIg	22	2.50
	Plasma Exchange	9	1.33
	Methyl prednisolone	19	0.42
	No Treatment	4	2.00
	Not Applicable	3	-1.67

[#] Significance of influence of treatment options on mean improvement in disability scores

The mean improvement in GBS disability scores from admission to the end of 8 weeks was calculated for AIDP subtype of GBS patients which is shown in the table 21 below.

Table-21 Mean improvement in disability scores for AIDP patients with various treatment options

Disability Scale	**Treatment Option**	**Total Numbers**	**Mean Improvement**
GBS disability scale $p = 0.116^{\#}$	IvIg	18	1.06
	Plasma Exchange	6	0.67
	Methyl prednisolone	15	0.13
	No Treatment	4	1.00
MRC disability scale $p = 0.043^{\#}$	IvIg	18	2.39
	Plasma Exchange	6	1.33
	Methyl prednisolone	15	0.53
	No Treatment	4	2.00

[#] Significance of influence of treatment options on mean improvement in disability scores

DISCUSSION

DISCUSSION

In the present study totally 63 patients were enrolled from January 2006 to December 2007. Patients who got initially admitted in the emergency medical ward, were subsequently transferred to medical/neuromedical ward or to the intensive medical care unit according to the severity of illness and its progression.

Among the total 63 patients registered, most patients were noted in the 20 to 40 year age group (50.8%). Only 3.2% of patients were elderly (>60 years). The number of patients represented by <20 years age group may not reflect the true incidence in our study, because the total health care of pediatric age group patients is provided by the Institute of Child Health, a separate hospital attached to Madras Medical College. Most surveys show a slight peak in late adolescence and young adult. Some studies show a peak also in the elderly age group. Dowling et al[89] has reported a bimodal peak in the age specific incidence. Whereas in our study no such phenomenon has been observed.

Several studies have shown a slight male[5,6] preponderance for GBS. In the present study also there is a male preponderance (55.6% vs 44.4%) with a male : female ratio of 1.25 : 1. A similar preponderance is noted in the subtypes of GBS also.

In our study, GBS is noted in all months of a year with a slight increase in the months from June to October, which is the end of summer and the beginning of rainy season. Several studies have shown such a seasonal[90] clustering.

Several studies had established that the prior infection may be a precipitating event for GBS, and it can occur in about 60-70% of cases. [12,77,91,92] Whereas the antecedent illness according to Ashok et al[93] was 49% and Sunder et al[94] was 52%. We have noted 46% (29 patients) of patients with antecedent illness which include diarrhea, respiratory infection, pregnancy, chicken pox and surgery. 54% of our patients did not have any specific preceding illness. Each 20.6% of patients had respiratory infection or diarrhea as preceding illness.

Typical pattern of ascending type areflexic quadriparesis was noted in 47.6% of patients and simultaneous onset of illness in all 4 limbs ± cranial nerve involvement was noted in 27% of patients. Pain as a predominant feature in addition to weakness was observed in 5 patients (8%) of cases. In this group, 2 patients landed up in Rheumatology Department with a provisional diagnosis of inflammatory myopathy and was later transferred to Neurology ward.

Cranial nerve involvement in the form of either facial or bulbar weakness was noted in 27 patients (42.7%) and 8 patients had both facial and bulbar weakness. Sundar et al[94] showed facial involvement in 39% of cases and bulbar involvement in 43% of cases. Kaida et al[95] showed facial involvement in 77% of cases and bulbar involvement in 65% of cases. Our study shows facial nerve involvement in 34.9% of cases and bulbar involvement in 20.6% of cases. Opthalmoparesis as a feature was noted only in MFS.

Autonomic dysfunction in GBS ranged from isolated tachy or bardycardia without hemodynamic disturbance to severe form of illness like Sudden Cardiac Death. The incidence varied in several studies ranging from

17% by Ashok et al[93] to 33% by Sundar et al[94]. In the present study autonomic dysfunction was noted in 42.9% of patients. In the course of illness 5 patients had severe postural hypotension and all these patients had poor outcome in the form of death. Hence we believe severe postural hypotension may be a predictor of poor prognosis.

It is well known that the rapidity of attaining peak disability is a poor prognostic sign. In our study 7 patients (11%) attained peak disability within 2 days, 29 patients (46%) attained peak disability in 3-8 days, 20 patients (32%) attained peak disability in 15-28 days. Totally 37 patients (58.7%) presented with peak disability within 8 days which is a high risk group for poor outcome.[96]

Nerve conduction studies were done in all except for 8 patients, because of early death or difficulty to mobilize. Prolongation of distal latency was the commonest abnormality noted (97%) in GBS patients with AIDP subtype. F-wave abnormality in the form of impersistance or prolongation of minimum latency was the next commonest abnormality noted (87.2%) in GBS as a whole and in its sub type AIDP (92.7%).

In the present study, nerve conduction study was not done on many patients in the initial few days or weeks, due to difficulty in mobilizing the sick patients from imcu or medical wards to the Neurology department where the facility is available. Hence electrophysiological parameters are not used to assess or predict the prognosis in a particular patient in this study. But they are helpful to categorize GBS subtypes and to monitor the progress.

Out of the total 63 patients, AIDP formed the bulk (68.3%) and AMSAN constituted 12.7% of cases. AMAN was noted in 4.8% and MFS in 7.9% patients. Three patients presented in an acute and severe form of illness which bulbar weakness and autonomic dysfunction on whom no specific investigation or treatment was effectively initiated except for the respiratory support and symptomatic treatment. All the three patients expired within one or two days and hence this group increases the overall mortality percentage of GBS patients. This type of fulminant course was noted in 5 to 20 % of GBS cases by Alan R. Berger.[76]

In our study IvIg was administered to 22 patients (34.9%), plasma exchange was given to 9 patients (14.3%) and injection methyl prednisolone

was given to 25 patients (39.6%). As already noted 3 patients with fulminant form of illness were not able to receive either of these treatment modality and were also not grouped in any of the GBS subtypes (unclassified in our study).

Respiratory muscle weakness, necessitating ventilatory support in GBS is an important cause for mortality and morbidity. Apart from periodically assessing the motor power of limbs, patient's adequacy of respiratory function was done traditionally by Single Breath Count (SBC), cough reflex and neck muscle weakness. Apart from this, the peak flow rate at one second in PEF meter was used in the study to objectively assess and document the respiratory adequacy. Three attempts were given and the average score was noted. The PEFR of >500 correlated with SBC of >30 indicating adequate power of respiratory muscles. The PEFR of <100 correlated with SBC<10 indicating an impending ventilatory dependence. The PEFR of 100-300 and 300-500 correlated with SBC of 10-20 and 20-30. Watchful expectancy has to be done for those with SBC of 10-20 (PEFR 100-300). All the 4 parameters (SBC, PEFR, Cough reflex, neck weakness) used in the study adequately predicted the need for ventilatory need.

38 patients were admitted in our study with admission disability score of more than 3 in GBS disability scale and >5 in MRC disability scale. They constituted 60.3% of the total patients registered. Good outcome is considered when the patient is able to walk which is a score of ≤ 3 in Hughe's GBS disability scale and ≤5 in MRC disability scale. Poor outcome is considered when the patient chair or bed bound, ventilator or expired, which is a score of >3 in GBS disability scale and >5 in MRC disability scale. In our study at the end of 8 weeks, 24 patients had GBS disability score of >3 (MRC disability score >5) which contributed 38.1% of the total admitted patients.

Totally 19 patients needed ventilatory support which constituted 31.7% of the total admission. In various studies a similar incidence is noted.[98,99] Lawn et al[97] noted that 53% of patients needed ventilatory support, whereas Kalitha et al[102] noted only in 10% of patients. In the AIDP subtype 27.9% of patients needed ventilatory support whereas in the AMSAN type the need was for 50% cases.

Totally 10 deaths are recorded in our study which constituted 15.9% of the total admission. Even in well equipped centers with aggressive ICU care, the mortality is noted to be around 5-10%[76] and 4-15%.[100,101]

Totally 5 patients required tracheostomy for the need of prolonged ventilation which constituted 7.9% of the total admission.

Among the 10 patients who had expired autonomic disturbance and high grade disability at presentation were noted in 9 patients and all the patients who expired reached their peak disability within 8 days of onset of symptoms. Bulbar dysfunction noted in 7 out of 10 patients and diarrhea was noted in only 4 patients.

Among the 20 patients who were ventilated autonomic disturbance was noted in 13 patients, in 16 patients peak disability was reached before 8 days and high grade disability at presentation was noted in 17 patients, whereas diarrhea was noted in only 8 patients.

Totally 27 patients (47.4%) had cranial nerve involvement and the rest did not. When the cranial nerve involvement was present, good outcome was noted in 17.5% of patients and poor outcome was noted in 29.8% patients. Whereas in patients without cranial nerve involvement good outcome was noted in 40.4% of patients and poor outcome was noted only in 12.3% of patients. The value is statistically significant (P = 0.002).

Totally 26 patients (45.6%) had autonomic disturbance. When autonomic dysfunction was present 19.3% of patient had good outcome and 26.3% had poor outcome. Whereas in the absence of autonomic dysfunction good outcome was noted in 15.8% of patients. The value is statistically significant (P = 0.029).

Totally 29 patients had antecedent illness and diarrhea was noted in 13 patients (22.8%). In the patients with diarrhea, good outcome was present in 7% of patients and poor outcome was present in 15.8% of patients. The value is not statistically significant (P = 0.165).

Totally 34 patients attained peak disability ≤ 8 days for this group of patients, good outcome was noted in 28.1% of patients and poor outcome was noted in 31.6% of patients. Whereas for those patients who had not attained peak disability in ≤ 8 days the good outcome was noted in 29.8% of patients and poor outcome was noted in only 10.5% of patients. The value is statistically significant (P = 0.044).

Totally 20 patients were admitted with ≤ 3 GBS disability score (≤ 5 MRC disability score) and 37 patients with > 3 GBS disability score (> 5 MRC disability score) and 37 patients with > 3 GBS disability score (> 5 MRC disability score). At the end of 8 weeks for those patients who were with ≤ 3 GBS disability scale, good outcome was noted in 29.8% of patients and poor outcome was noted in 38.6% of patients. Though good outcome appears to be similar for both groups, they differ profoundly in terms of poor outcome. The value is statistically significant (P = 0.001).

Among the 22 patients who were treated with IvIg 14 patients (24.6%), had good outcome and 8 patients (14.0%) has poor outcome. Among the 9 patients who were treated with plasma exchange, 6 patients had good outcome and 3 had poor outcome. Among the 19 patients, who were treated with injection methyl prednisolone, 8 patients had good outcome and 11 patients had poor outcome. The values obtained are not statistically significant (P = 0.076).

Among the IvIg treated 18 AIDP patients, 1 required tracheostomy (5%) and among the methyl prednisolone treated 15 AIDP patients 4 required tracheostomy (26%).

Among the IvIg treated 18 AIDP patients, 5 required ventilatory support (27%) and among the methyl prednisolone treated group 7 required ventilatory support (46%).

Among the IvIg treated 18 AIDP patients, 1 patient expired and among the methyl prednisolone treated 15 AIDP patients, 3 had expired (20%).

The IvIg treated AIDP patients clearly showed less percentage of death, need for ventilatory support and tracheostomy when compared with methyl prednisolone treated patients, though the values are statistically not significant. The other subtypes and variables are very small and hence cannot be compared.

Among the AIDP patients, who presented with severe disability at admission[25], 12 were treated with IvIg, 4 with plasma exchange and 9 with methyl prednisolone.

The outcome was good for 7 patients (58.3%) and poor for 5 patients (41.6%) in the IvIg treated group. The outcome was good for 2 patients and poor for 2 patients in the plasma exchange treated group. The outcome was good for 3 patients (33%) and poor for 6 patients (66%).

Among the AMSAN patients, who presented with severe disability at admission, 3 each were treated with IvIg and methyl prednisolone and 2 patients were treated with plasma exchange. The outcome was good for 2 and poor for 1 patient, treated with IvIg, whereas all 3 patients had poor outcome in methyl prednisolone treated group. Plasma exchange resulted in 1 good and 1 poor outcome.

Even in the group of patients who presented with severe disability (> 3 in GBS disability score) at admission, among the AIDP patients the outcome appears to be better for those treated with IvIg compared to methyl prednisolone. Among the AMSAN patients also, the outcome is poor for the methyl prednisolone treated 3 patients. The values are statistically not significant.

Among the 12 AIDP patients, who presented with severe disability at admission and treated with IvIg, 1 out of 12 required tracheostomy (8%) and there was no death in this group.

Among the 9 AIDP patients, who presented with severe disability at admission and treated with injection methyl prednisolone, 6 out of 9 required tracheostomy (33%) and there were 3 deaths in this group (33%).

Among the AIDP patients who presented with severe disability at admission itself, the need for ventilatory support with or without tracheostomy and the death are more in methyl prednisolone treated group than in the IvIg treated group.

The other variables and subtypes are too small to compare.

The mean improvement in GBS disability scores from admission to the end of 8th week was calculated for each treatment modality. For IvIg it is 1.09, for plasma exchange it is 0.67 and for methyl prednisolone it is 0.00. The values are statistically significant (P = 0.02).

Similarly the mean improvement in MRC disability scores from admission to the end of 8th week was calculated. For IvIg it is 2.05, for plasma exchange it is 1.33 and for methyl prednisolone it is 0.42. The values are statistically significant (P = 0.00).

For AIDP subtype of GBS patients, the mean improvement in GBS disability scores from admission to the end of 8th week was calculated. For IvIg it is 1.06, for plasma exchange it is 0.67 and for methyl prednisolone it is 0.13. The values are statistically not significant.

Similarly for AIDP patients, the mean improvement in MRC disability scores from admission to the end of 8th week was calculated. For IvIg it is 2.39, for plasma exchange it is 1.33 and for methyl prednisolone it is 0.53. The values are statistically significant (P = 0.0).

CONCLUSION

CONCLUSION

- A significant proportion of patients present with peak disability within 8 days of onset of illness to whom definite treatment options are to be made available to enhance a good and early recovery, because this is the group associated with poor outcome.

- The mean improvement in GBS disability scale from admission to the end of 8th week is more for IvIg treated patients when compared to methyl prednisolone treated group, which is statistically significant. It is also applied well to the AIDP subtype of GBS.

- Though statistically not significant in this study, injection methyl predisolone is associated with high percentage of poor outcome when compared to IvIg and plasma exchange.

- The prolonged morbidity of the illness evidenced by the need for tracheostomy is more for those treated with methyl prednisolone when compared to other definite treatment options.

- Peak expiratory flow rate can also be used as an objective measure to assess the respiratory function, which is handy, and it correlates with the standard assessment like single breath count.

- Autonomic dysfunction, bulbar weakness, rapidity of onset of illness, severe grade disability and diarrhea are significantly correlating with poor outcome.

- Postural hypotension was noted in all patients who had expired and it needs further analysis, as a specific prognosticating parameter in patients having autonomic dysfunction.

- A high index of suspicion is needed to diagnose GBS types like those who present with pain as the predominant feature.

- A small but significant group of patients present with a fulminant form of disease for whom the prognosis is the worst in spite of good supportive measures and imcu care.

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GBS - STUDY PROFORMA

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Name : **Occupation:**
Age/Sex : DOA:
Address : IMCU:

Clinical contributes;

History : Mode of onset: Assc pain

Details of progression Autonomic symptoms

Peak of weakness: Resp. distress

Sensory symptoms: Post H/O similar illness

Antecede nil illness: URI/ AGE / Surgery/ Others:

Examination

General Exam:

Cranial nerve:

SMS: Tone:
Bulk:
Power:
Reflexes: Superficial:

DTR:

Sensory system:

Cerebellum

Gait:

ANS:

GBS disability grading:

Modified Hughes:

MRC:

Laboratory contributes:

CBC:

Biochemical Parameters:

Radiology:

Electro physiology:

CSF:

Others:

Therapeutic Contributes:

Mode of Primary Treatment:

Others: Ventilatory support:

Secondary complication:

Outcome measurement:

4 weeks:

8 weeks:

Follow up details:

INSTITUTE OF NEUROLOGY
MADRAS MEDICAL COLLEGE, CHENNAI-3

NERVE CONDUCTION STUDY

Name: **Unit:** **Date**
Age/sex: **MIN NO:**

MOTOR NERVE CONDUCTION STUDY

NERVE	LATENCY milli sec		AMPLITUDE MV		NCV M/S	F WAVE milli sec

SENSORY NERVE CONDUCTION STUDY

NERVE	LATENCY	AMPLITUDE	VELOCITY

GBS - MASTER CHART