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Study on Clinical Profile of Guillian Barre Syndrome in a Tertiary Care Centre in India

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Abstract:
Guillain-Barre Syndrome is an important cause of acute flaccid paralysis globally, with annual incidence of 1.3-2/1 lakh. Typical clinical features include progressive, symmetrical muscle weakness associated with absent or depressed deep tendon refluxes. Weakness may range from mild weakness to complete paralysis, needing ventilator support. GBS can be diagnosed by established clinical criteria, CSF studies and electrophysiological studies.
Objectives: It is a cross sectional record based study using the copies of discharge cards of GBS patients in Neurology department from Aug 2017 to July 2020.
The patients are enrolled in the study based on definite inclusion and exclusion criteria. Sample size is 40 patients. The data collection tool is a structured proforma. Methodology of data collection record based retrospective analysis. Secondary data of patients with GBS. Particularly variables like age of onset, gender, clinical presentation and classification according to NCS will be done. Data was entered in MS excel & analysied using appropriate software like Epi info. Qualitative data was analysed using proportion. It was tabulated & discussed. There are no ethical issues in the study and confidentiality of data will be maintained.

Key Words
1. GBS - Guillain Barre Syndrome, AIDP – Acute Inflammatory Demylinating Polyneuropathy, AMAN – Acute Motor Axonal Neuropathy, AMSAN – Acute Motor Sensory Axonal Neuropathy, MFS - Miller Fisher Syndrome , LP – Lumbar Puncture, CSF – Cerebro Spinal Fluid, MRC- Medical Research Council

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I. Introduction

Guillain-Barre Syndrome is an important cause of acute flaccid paralysis worldwide. It is an auto immune syndrome characterized by symmetrically progressive demyelinating poly neuropathy. It is characterised by rapidly evolving symmetrical limb weakness, areflexia, absent or mild sensory signs and variable autonomic disturbances[1].

In India, especially Kerala limited information is available regarding the clinical features at the patient level.

II. Background

Guillain-Barre Syndrome is an acute, monophasic symmetrically progressive, ascending demyelinating polyneuropathy. It is a major cause of acute neuromuscular paralysis with an annual incidence of 1.3-2/1 lakh globally. Male to female ratio is 3:2.

History of antecedent viral infection, vaccination or surgery may be obtained in most cases. Most of the patients experience symptoms of viral respiratory or gastrointestinal infections 1-3 weeks prior to the onset of neurological symptoms[2]. Most common agents are Cytomegalo virus and Campylobacter jejuni. Many cranial nerves may be involved, facial, bulbar or ocular

Typical clinical features of GBS are progressive, symmetrical muscle weakness associated with absent or depressed deep tendon refluxes. The weakness may range from mild walking difficulty to complete paralysis of limbs. Motor cranial weakness and life threatening respiratory muscle weakness can also occur[3]. 10-25% of the patients may need venilatory support. GBS can also affect the autonomic nervous system producing dysautonomia

Guillain-Barre Syndrome can be mainly divided into four sub-groups[4]. They are

1. Acute inflammatory demylinating neuropathy (AIDP).
2. Acute Motor Axonal Neuropathy (AMAN)
3. Acute Motor Sensory Axonal Neuropathy (AMSAN)

4. Miller Fisher Syndrome (MFS)

AIDP is frequently seen in Europe and America. AMAN and AMSAN is more common in Asia and South America. The Miller Fisher Syndrome is characterised by opthalmoplegia, ataxia and areflexia constitute 5% of the cases.

GBS is diagnosed by established clinical criteria which is further supported by Albumino cytological dissociation in cerebrospinal fluids and by electrophysiological studies. In AIDP, motor nerve conduction studies revealed prolonged distal latency, reduced motor nerve conduction velocity, conduction block and prolonged or absent F wave in two or more nerves. In AMAN and AMSAN, velocity and latency may be normal but the amplitude will be decreased. In AMSAN in addition to motor conduction sensory, sensory conduction can also be abnormal[5].

Established treatment of Guillain-Barre Syndrome includes (a) management of severely paralysed patients with ICU care and ventilator support and specific intervention like IVIG and plasma exchange which will help in shortening the course of the disease and aid in rapid resolution[6]. The plasma exchange is recommended for patients with moderate to severe weakness within two weeks of onset of disease. The recommended plasma apharasis scheduled comprises a serious of five exchanges (40-50ml/kg) with a continuous flow machine on alternate days using saline and albumin as a replacement fluid. IVIG is given in the dose of 400mg/kg/day for 5 days is also equally effective in hastening the recovery. But it is costly and side effects are also common.

Objectives

1) To study the clinical presentation, age of onset and gender distribution of patient with Guillain Barre Syndrome admitted in neurology department of a tertiary care hospital in Kerala.

III. Materials And Methods

Study design - Cross sectional observational Record based
Study setting - Tertiary care centre in Kerala, India

Study participants

This is a retrospective study using the copies of discharge cards of Guillain Barre Syndrome admitted in neurology from August 2017 (01/08/2018 to July 2020 (31/07/2020).

Inclusion criteria

All the patients in the study are cases of Guillain-Barre Syndrome diagnosed on clinical features based on Asburys criteria which includes the following paramrters .

1. Acute onset of bilateral and relatively symmetric flacid weakness/paraysis of limbs with the without involvement of respiratory or cranial nerve-innervated muscles.
2. Decreased or absent deep tendon reflexes atleast in the affected limbs, monophasic illness pattern, with maximum weakness reached between 12 hours and 28 days, followed by clinical plateau and subsequent improvement or death.
3. Electrophysiological findings consistent with GBS.
4. Presence of albumin cytological dissociation.
5. Absence of an alternative diagnosis for weakness.

Exclusion criteria

Cases of other lower motor neuron syndromes which may mimic myasthenia like Poliomyelitis, traumatic neuritis, muscle diseases like myopathies are excluded. So also other UMN Syndromes

Study period - 1 month

All cases during the period 01/08/2018 to 31/07/ 2021 will be included and sample size is the 40 patients.

Methodology - Data collection Record based Analysis. Secondary data of patients with GBS were collected using a structured proforma. Data on clinical presentation, age of onset, gender, progression and clinical test performed and diagnostic test employed were collected.

Variables: Include Age of onset Sex, Presentation (clinical presentation) Classification according to the NCS findings

Data Analysis

Data was entered in MS excel & analysed using appropriate software like Epi info. Quantitative data were analysed using proportions. Was tabulated & discussed.

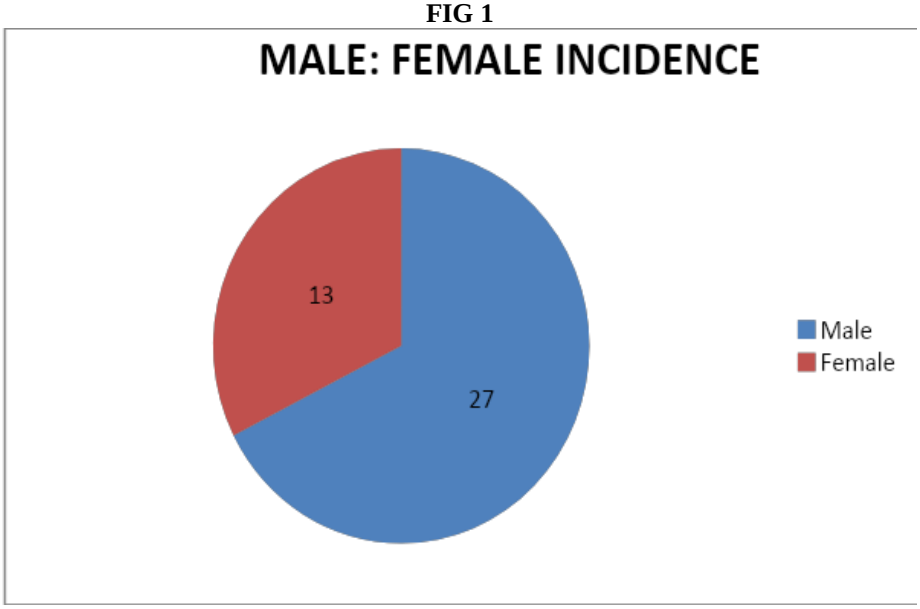
Ethical consideration

This is a record based study. It was started only after getting the approval from the research and ethical committee. Confidentiality of data will

IV. Results :

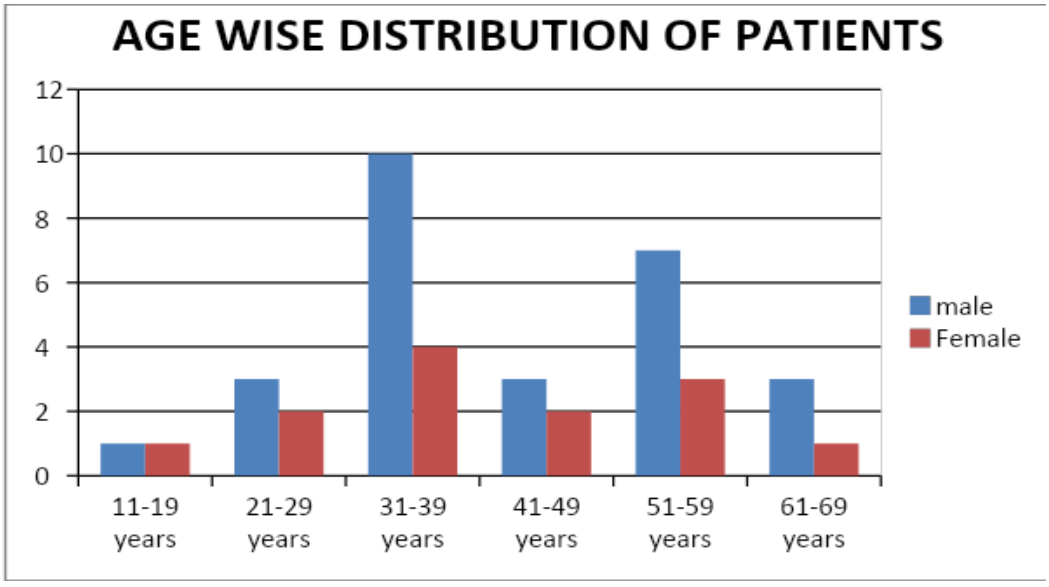
There were 40 patients out of which 27 (67.5%) were males 13 (32.5%) females. Illustrations given in figure 1

FIG 1



The number of patients in various age groups were as follows : 10-20 group two patients, 21-30 five patients, 3-40 fourteen patients, 41 -50 five patients, 50-60 ten patients and 60-70 four patients. Illustrations given in figure 2

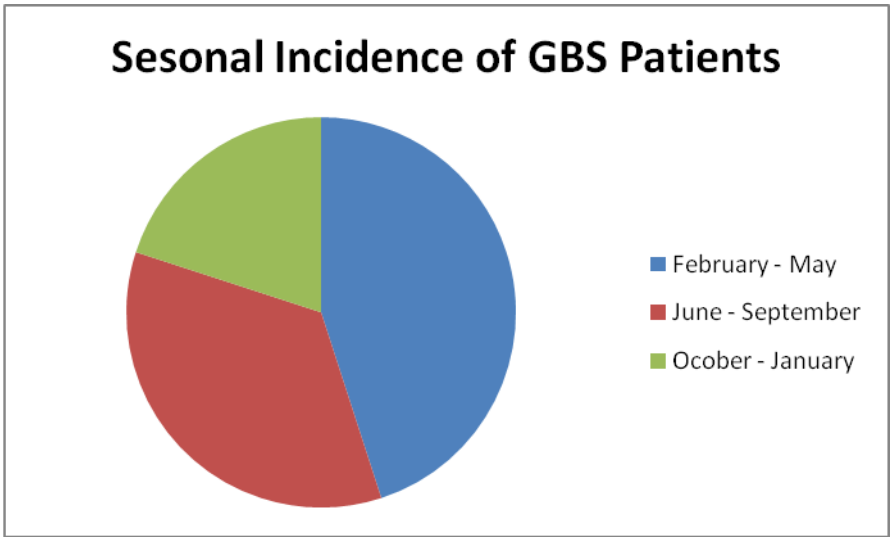
FIG 2



For categorizing the patients, the calendar year was divided into 3, S1 season :cold season, consisting of the months October, November, December and January. S2 season :Summer season, consisting of the months February ,March, April and may. S2 season :Rainy season , consisting of the months June, July, august and September.

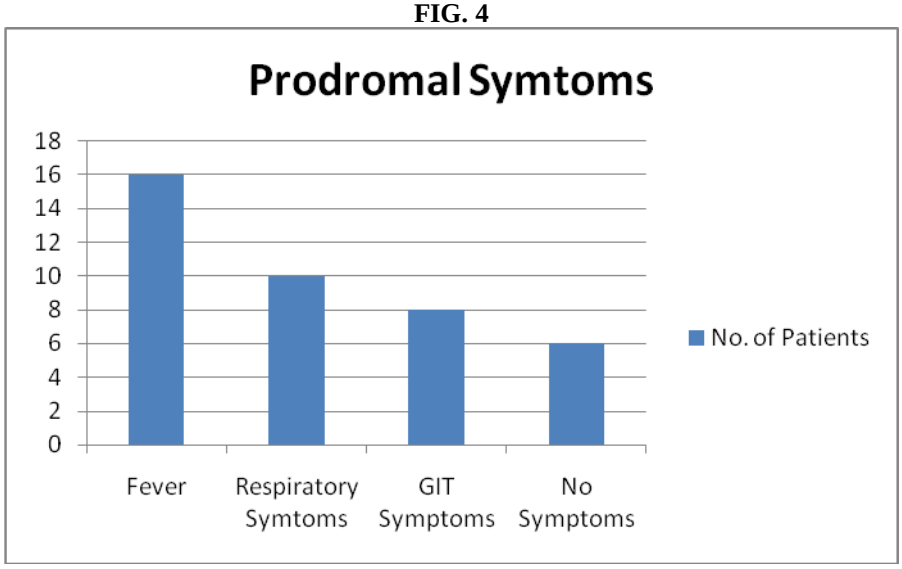
Maximum number of patients were seen in the quarter February – May in which there were 18 patients, 14 patients were seen in the quarter June –September and 8 patients in the quarter October – January. Illustrations given in figure 3

FIG 3



In our study 34 patients gave a history of proceeding illness of these. 16 patients (40%) had a history of fever with viral flue like symptoms. 10 patients (25%) had respiratory symptoms, 8 patients (20%) had gastrointestinal symptoms like loose tools and vomiting. In 6 patients (15%) no history of any such infections was found. Illustrations given in figure 4

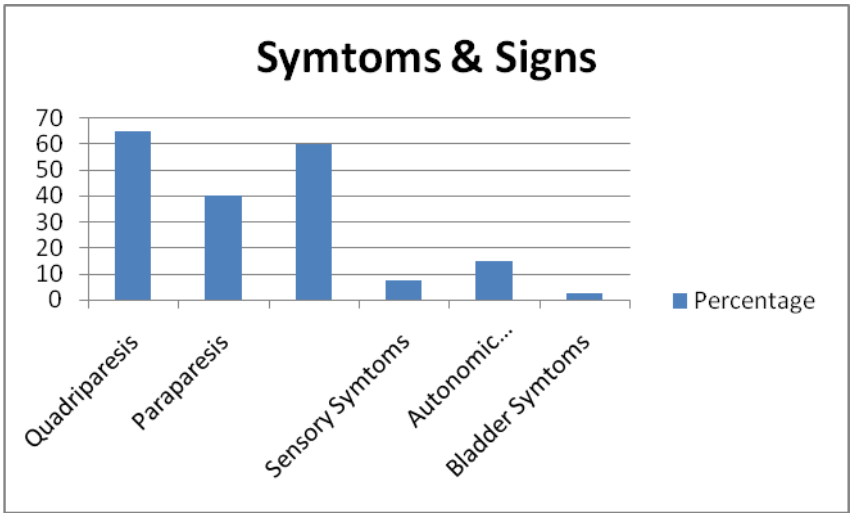
FIG. 4



Majority of the patients motor weakness was maximum within 3-5 days of the illness. The progression of weakness ranged from 1-12 days. In 4 (10%) patients the weakness reached a maximum peak in less than 3 days, 30 (75%) patients suffered maximum motor weakness between 3-5 days, 3 patients (7.5%) between 6-10 days and 3 patients (7.5%) between 10-12 days.

Symptoms: Quadriparesis was present in 26 patients (65%), paraparesis 16 patients (40%), cranial nerve involvement in 24patients (60%). Sensory symptoms were present in 3 patients (7.5%), bladder symptoms in one patient (2.5%). Hypotonia was present in 90% of patients areflexia was present in all patients. Sensory involvement was found in 3 patients, bladder involvement in one patient and autonomic dysfunction was present in 6 patients (15%). Illustrations given in figure 5.

FIG. 5



Ascending type of paralysis was present in 28 (70%) patients. Illness started as a rubbery feeling and symmetrical weakness which started on the lower limbs and ascended symmetrically and then involved the upper limbs. In 8 patients (20%) there was a descending type of paralysis which started on the upper limbs and then descended down to the lower limbs. In 4 patients (10%) there was simultaneous involvement of both upper and lower limbs. Respiratory muscle involvement was present in 8 patients (20%). Sensory abnormalities were present in 3 patients (7.5), autonomic dysfunction was present in 6 patients (15%) and bladder involvement

Table 1

Type of Paralysis	**No. of Patients**
Ascending Type	28 (70%)
Descending Type	8 (20%)
Simultaneous of involvement of all limbs	4 (10%)

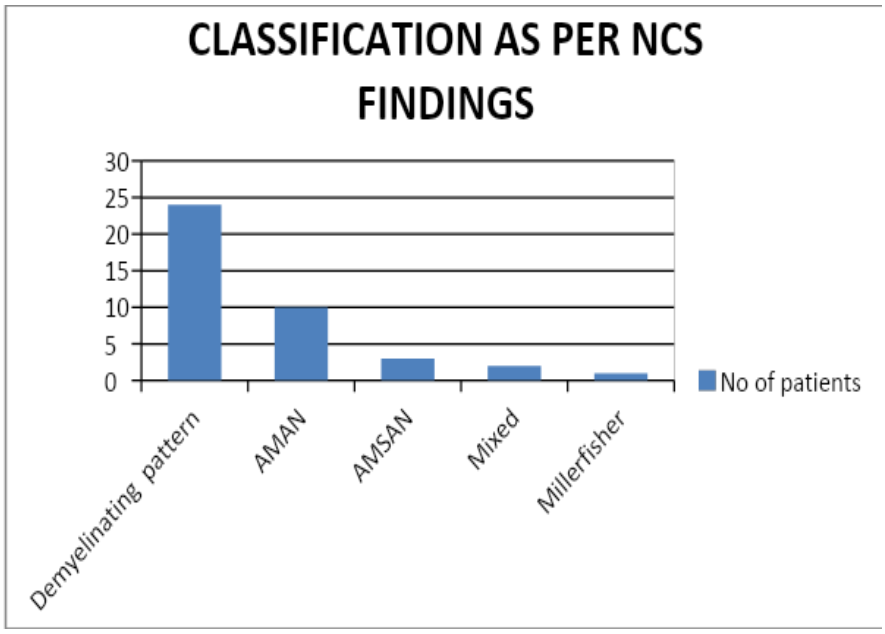
24 (60%) patients who had cranial nerve deficits. Out of the 24 patients, 20 (83.3%) had bilateral LMN facial paralysis. 3 (12.5%) had involvement of the 9^{th}, 10^{th}, and 11^{th} cranial nerves. 1 (4.1%) patient had involvement of 3^{rd}, 4^{th}, and 6^{th} cranial nerve and ataxia also.

Table 2

Cranial Nerve involvement	No. of Patients
7^{th} Cranial Nerve B/L	20
9^{th}, 10^{th} , 11^{th}	3
3^{rd}. 4^{th}, 6^{th}	1

Based on the neurophysiological findings patients were divided into four groups. The important parameters that were looked included the latency, amplitude, velocity, presence the conduction block and F wave latency of the motor and sensory action potentials of the different nerves. As per these parameters, 24 patients (60%) had the demylinating pattern, 10 patients (25%) belonged to the AMAN group, 3 patients (7.5%) belonged to the AMSAN group, 2 patients (5%) had mixed demylinating and axonal pattern and one patient (2.5%) belonged to the Miller fissure syndrome. Illustrations given in figure 6.

FIG. 6



Lumbar puncture and CSF studies were done in all patients. CSF protein ranged from 50-300mg% self ranged from 0-10 albuminocytological dissociation was noted in 34 patients (85%).
Patients who had no contraindications to plasmapheresis were subjected to plasma exchange therapy. Others who could not be subjected to plasmapheresis were treated IVIG. Some of the patients who were initially treated with plasmapheresis showed no improvement. They were subsequently given IVIG. The improvement was assessed by the MRC sum scores. Majority of the patients (75%) who had low MRC scores on admission showed an improvement of MRC score after 4 weeks of treatment. Other patients required prolonged supportive treatments and rehabilitation measures for 6 months.

V. Discussion

There were 40 patients out of which 27 were males 13 females. There is a male preponderance in our study, with a male to Female ratio of 2.07:1. Manju et al (70%)[7] and Robert M et al[8] have also described a similar male preponderance in their studies. The peak incidence of patients was found between 31-39 age group in our study and another lesser peak between 50-59. Peter C, Dowling JP also has reported 2 peaks of incidents in his study[9]. Rees et al has also described a bimodal distribution of age group in patients with 2 peaks[10]

Maximum number of patients seen in summer season in our study with peak incidence in the month of May. This was followed by the rainy season with maximum incidence in the month of June. Peter C Dowling et al has reported an increase incidence of patients in the summer season[9]. Kaur et al has reported an increase incidence of GBS patients in summer and autumn[11]. It is probably because infection like viral fever, respiratory tract infections occur mostly in summer and rainy season.

History of antecedent infections in our study was 85%. Ropper et al has reported antecedent infections in 73% of his patients[8]. Arvind Vyas et al has described in his study that 23% of his patients had respiratory infections and 26% had gastroenteritis and 20% had fever without any identifiable cause. Although stool culture, blood culture and other relevant investigations were done. We could not isolate any organism in out study[12]. 36 (90%) out of the 40 patients developed neurologically illness within four weeks of the antecedents infections while 2 patients developed the illness within 6 weeks. Such an interval has been mentioned in 95% of the patients by Chirag et al[3].

Majority of the patients motor weakness was maximum within 3-5 days of the illness. The progression of weakness ranged from 1-12 days. In 4 (10%) patients the weakness reached a maximum peak in less than 3 days, 30 (75%) patients suffered maximum motor weakness between 3-5 days, 3 patients (7.5%) between 6-10 days and 3 patients (7.5%) between 10-12 days. In 85% of the patients, weakness progressed and reached maximum deficit within 5 days. Such a rapid evolution has been described by Md. Rashedul et al [13].

Ascending type of paralysis was present in 28 (70%) patients. Illness started as a rubbery feeling and symmetrical weakness which started on the lower limbs and ascended symmetrically and then involved the upper limbs. In 8 patients (20%) there was a descending type of paralysis which started on the upper limbs and then descended down to the lower limbs. In 4 patients (10%) there was simultaneous involvement of both upper

and lower limbs. Manju et al (ref.7) has described the frequency of the evolution of the different patterns as 70% (ascending), 4% (descending) and 26% (mixed) respectively in his study. In another study by Allen H Ropper et al the frequencies of these paralysis patterns were 60%, 20% and 20% respectively[8]. Respiratory muscle involvement was present in 8 patients (20%). All these patients required ventilator support.2 of the patients developed Pneumonia and sepsis. They expired in spite of treatment. Rajendra Singh Jain et al has described respiratory failure in 12.2% of his patients[1] and Chirag et al has noted a frequency of 28% incidents in his study.

24 (60%) patients who had cranial nerve deficits. Arvind et al and Emilia et al in their studies have noted the frequency of cranial nerve involvement in 50 and 55% of cases of GBS in their studies[12,13]. Out of the 24 patients, 20 (83.3%) had bilateral LMN facial paralysis. 3 (12.5%) had involvement of the 9th, 10th, and 11th cranial nerves. The incidence of bulbar involvement was 7.5 in the study by Juby et al [2] (4.1%). 1 patient had involvement of 3rd, 4th, and 6th cranial nerve and ataxia also.

Sensory abnormalities were present in 3 patients (7.5%), autonomic dysfunction was present in 6 patients (15%). Autonomic symptoms presented in the form of variations in pulse , orthostatic hypotension , cardiac arrhythmias , sweating abnormalities , gastro intestinal dysfunction etc. Chirag et al[3] in his study has found the incidence of sensory abnormalities and autonomic dysfunction as 15% and 39.7% respectively in his study.

Albuminocytological dissociation was observed in 85% of our patient. In GBS inflammation of the radicles a d nerves. This finding is a hallmark of GBS[14] As a result of inflammation, there is disruption of blood- CSF barrier. Anitha Bhargava et al has described that 80% of the patients had albuminocytological dissociation[15].

Based on the neurophysiological findings patients were divided into four groups. The important parameters that were looked included the latency, amplitude, velocity, presence the conduction block and F wave latency of the motor and sensory action potentials of the different nerves. As per these parameters 24 patients (60%) had the demylinating pattern, 10 patients (25%) belonged to the AMAN group, 3 patients (7.5%) belonged to the AMSAN group, 2 patients (5%) had mixed demylinating and axonal pattern and 1 patient (2.5%) belonged to the Miller fissure syndrome. Juby etal in his study has reported 57.6% incidence of demylination. 3% axonal and 12, 1% mixed, and 6.1% MFS incidence in the NCS study[2]. Chirag et al[3] in his study categorised his patients depending on NCS parameters and 57% of his patients had demylinating pattern while AMAN and AMSAN characteristic observed in 15% and 8% patients respectively.

VI. Conclusion :

Guillian Barre Syndrome is more common in males. It is common in the age group between 31-39 years. But another smaller peak incidence was seen in 51-59 age group. Usually a history of preceding infections like viral fever, respiratory tract infections or GIT infections was noticed. Ascending type of weakness was the most common presentation. Bilateral facial nerve involvement was present in majority of the cases. Areflexia was the most common sign and it was present in all the cases. Albuminocytological dissociation was an important finding in our study group. Nerve conduction studies are very much useful in the diagnosis of GBS and differentiating them into different patterns. Acute inflammatory demyelinating poly neuropathy was the predominant pattern observed in our group.

Conflicts of Interest

There are no conflicts of interest

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