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**Research Article**

# ROLE OF ELECTROPHYSIOLOGICAL STUDY IN EVALUATION AND DIAGNOSIS OF GUILLAIN BARRE SYNDROME

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**A B S T R A C T**

**Objective:**
1. To evaluate electrophysiological features of GBS
2. To know about various GBS variants in studied population

**Materials and Methods:**
50 patients diagnosed as Guillain Barre Syndrome (GBS) fulfilling the criteria as modified by Asbury, admitted in the Medical wards of tertiary hospital, Ahmedabad, From June 2019 to June 2021. It was a prospective observational study.

**Results:**
In this prospective study of 50 patients with GBS (based on Asbury's Criteria), it was found to be commonest in the age group below 50 years and there was a male preponderance. Electrophysiological studies conducted in all patients revealed demyelinating pattern in 15 patients, axonal pattern in 10 patients and mixed pattern in 25 patients.

**Conclusions:**
Mixed pattern is the most common pattern seen on electrophysiological studies. Axonal pattern has better recovery compare to demyelinating and mixed pattern. AIDP was the most common variant in studied population followed by AMAN variant and AMSAN variant respectively.



## INTRODUCTION

Guillain Barre Syndrome (GBS) is an acute inflammatory and autoimmune disorder of the peripheral nervous system triggered usually by a bacterial or viral infection or other antecedent events. It affects 1 to 4 persons/100,000[1] 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 on 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 paresthesia with areflexia. 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

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available at present. Though both have similar outcome measures, many centers prefer the former because of the convenience.

## MATERIALS AND METHODS

50 patients diagnosed as Guillain Barre Syndrome (GBS) fulfilling the criteria as modified by Asbury, admitted in the Medical wards of tertiary hospital, Ahmedabad, From June 2019 to June 2021. It was a prospective observational study.

***Inclusion criteria***

This study consists of patients who presented with features of GBS based on Asbury's criteria which included areflexic motor weakness, with or without cranial nerve dysfunction evolving within period of four weeks and patients with clinical variant of GBS.

***Exclusion criteria***

- Early and prominent bladder and bowel dysfunction
- Marked and persistent asymmetry of symptoms and signs
- Presence of persistent sharp sensory level
- Features of other diseases like myasthenia gravis, botulism, poliomyelitis, porphyria and diphtheria
- Drug or toxin induced acute neuropathy
- Acute exacerbation of CIDP

## RESULTS

A total of 50 patients were studied. All patients were hospitalized and the average duration of hospital stay was 17.57 days. 32 patients (64%) were males and 18(36%) were females. The age of patients ranged from 12 to 60 years (Mean age 35.61 years) with the maximum number (34%) of patients in between 21 to 28 yrs age group & in between 29 to 36 (24%) yrs (Table-1). Most number of cases were seen in the months of April to June. However no significant increased incidence in any particular season could be inferred. (Table-1). Twenty three (46%) patients had some antecedent event prior to the development of GBS (Table-1). The most common antecedent illness was Diarrhoea. In patients with a history of preceding illness, the mean duration between onset of GBS and the preceding illness was 9.06 (± 4.21) days. The first symptom of the illness was in the form of motor weakness in 32 (64%) patients and it was sensory in the form of pain, paraesthesia or numbness in the remaining 18(36%) patients. Bulbar weakness was presenting symptom in 1 (2 %) patient and Ataxia in 1 patient. Twenty nine patients (58%) had ascending form of paralysis. Only 3 (6%) patients had descending type of paralysis (Table-1). Sixteen patients (32%) had simultaneous involvement of both proximal and distal muscles. Localised form of onset seen in 2 patients. Twelve patients developed maximal deficit in 1 day, majority of patients (48%) developed peak deficit in 2 days.(Table-1). Eight patients (16%) admitted with respiratory distress, Twenty four patients admitted in bed bound state. During the hospital stay, at peak deficit, Seventeen patients (34%) developed respiratory distress and treated with ventilatory support. Thirty two (64%) patients developed bed bound state during the hospital stay. Three (6%) patients died. The cause of death was respiratory failure following aspiration pneumonia in 1 patient who had rapidly progressed disease. One of the patients, a 18 year old female who had a cardiac arrest, had severe autonomic dysfunction with fluctuating blood pressure and heart rate died on the day of admission itself. Objective sensory loss was elicited in only 7(14%) out of the 50 patients. The sensory deficit was in the form of diminished vibration and joint position sense, which occurred in a glove and stocking distribution. Twenty eight patients had cranial nerve dysfunction. Twenty seven (54%) patients had facial nerve palsy, among which three patients had unilateral facial nerve involvement which progressed to involve contralateral side also in due course. Nine patients had involvement of 9th and 10th cranial nerves. Total external ophthalmoplegia was observed in one patient. This patient also had severe ataxia, areflexia and weakness in the lower limbs. A diagnosis of Miller Fisher variant of GBS was made in them. Seventeen patients had respiratory muscle paralysis and treated with ventilatory support .The development of respiratory distress is monitored by periodic assessment of maximal inspiratory force and expiratory vital capacity, development of neck muscle weakness, by observing single breath count . Patients who were in Grade 4 disability or more were not subjected to standing blood pressure recordings. Only sitting blood pressures were recorded in these patients. In patients who were on ventilator, the spontaneous changes in heart rate and blood pressure were noted. Autonomic dysfunction was detected in 23 (46%) patients (Table.2). One patient who was diagnosed to have Miler-Fisher variant of GBS presented with ataxia. CSF pressure was normal and CSF was clear in all patients. CSF glucose was also normal (approximately half the blood glucose level) in all patients. CSF protein concentration was raised above 45 mg% in 40 (80%) patients at one week. CSF protein level was normal in 10 patients. Three patients had lymphocytic pleocytosis of 20, 30 and 40 cells/cmm. None of the remaining patients had CSF pleocytosis. Nerve conduction studies were conducted in all patients. Fifteen patients were found to have reduced motor conduction velocities consistent with demyelinating neuropathy. Ten patients were found to have decreased amplitude of action potentials consistent with axonal pattern of neuropathy. Twenty five patients had mixed pattern of neuropathy. These patients had both demyelinating features (prolonged distal latency, reduced conduction velocity) as well as axonopathy features (reduction of CMAP amplitude). Motor conduction studies of Median, Ulnar, Peroneal, Tibial nerves were done. Distal latency, distal CMAP amplitude, conduction velocity, H reflex amplitude and latency, F wave latency were assessed. Varying degree of involvement in these nerves was observed, suggesting multi focal nature of the disease. The H reflex was absent in all cases. The electrophysiological study findings were completely normal, with the exception of absent H waves in 2 patients (4%) in the first week of disease onset. Sensory conduction studies of Median, Ulnar, Sural nerves were done. distal latency, SNAP amplitude, conduction velocity were assessed. SNAP abnormalities commonly involved in upper limb nerves in the form of conduction velocity reduction and reduced amplitude. Sural nerve less commonly involved. Preservation of sural nerve SNAP confirms the acquired as well as demyelinating nature of the disease in most of the cases. Aspiration pneumonia is the most common complication in the studied population. Aspiration pneumonia was observed more frequently in patients

admitted with bulbar dysfunction .Septicemia occurred in one diabetic patient .Deep venous thrombosis occurred in one patient after prolonged immobilization for which he was treated with low molecular weight Heparin. Urinary tract infection was noted in one patient. E.Coli was grown on urine culture and treated with appropriate antibiotics. Three patients (6%) died in this study. One patient developed aspiration pneumonia and later died due to septicemia and shock. One patient had fluctuating blood pressure and cardiac arrhythmia. She finally died due to cardiac arrest. One patient died of cardiac arrest while on ventilator. Patients were hospitalized and admitted in various medicine wards. Patients who required ventilatory support were transferred to Intensive medical care unit and respiratory support was given. Average duration of hospital stay was 19.74 days .Maximum duration was 45 days in one patient. Disability after discharge was assessed according to Hughes's scale. Most of the patients (60%) were discharged at Grade 3 (i.e. able to walk with support). Twelve patients (24 %) were discharged at Grade 2.Two patients were discharged at Grade 4. Three patients recovered almost completely; they were discharged at grade 1. Of 50 cases of GBS, acute inflammatory demyelinating neuropathy (AIDP) was the most common subtype forming 39 cases (78%) followed by 7 cases(14%) Acute motor axonal neuropathy (AMAN). Acute Motor Sensory Axonal Neuropathy was observed in 2 (4%) patients. Miller – Fisher variant of GBS was observed in 1 young male patients. One patient presented with Pharyngeal Cervical brachial- variant.

**Table 1** Demographics

**Age and sex distribution:-**

| Sex | 12 – 20 | 21 –28 | 29 –36 | 37-44 | 45-52 | 52-60 |
|---|---|---|---|---|---|---|
| M | 4 | 11 | 8 | 3 | 5 | 1 |
| F | 5 | 6 | 4 | 1 | 1 | 1 |
| Total | 9 | 17 | 12 | 4 | 6 | 2 |

**Seasonal incidence in GBS:-**

| Months | No. of cases | Percentage |
|---|---|---|
| Jan – March | 10 | 20% |
| April – June | 18 | 36% |
| July – September | 12 | 24% |
| Oct-December | 10 | 20% |

**Antecedent events:-**

| Antecedent events | No. of patients | Percentage (%) |
|---|---|---|
| Upper respiratory tract infection | 7 | 14 |
| Diarrhea | 8 | 16 |
| Post vaccination | - | - |
| Lower Respiratory Tract infection | 2 | 4 |
| Fever | 6 | 12 |
| None | 27 | 54 |

**Motor Symptoms:-**

| Motor symptoms | No. of patients | Percentage |
|---|---|---|
| Weakness of UL &LL ( P & D) | 16 | 32 |
| Proximal weakness | 10 | 20 |
| Distal Weakness | 5 | 10 |
| Bulbar weakness | 1 | 2 |
| Total | 32 | 64 |

**Sensory Symptoms:-**

| Sensory symptoms | No. of patients | Percentage |
|---|---|---|
| Paraesthesia | 9 | 18 |
| Pain in back | 3 | 6 |
| Numbness in legs | 5 | 10 |
| Ataxia | **1** | **2** |
| Total | **18** | **36** |

**Mode of onset of GBS:-**

| Mode | No. of patients | Percentage |
|---|---|---|
| Ascending paralysis | 29 | 58 |
| Descending paralysis | 3 | 6 |
| Simultaneous involvement of all 4 limbs | 16 | 32 |
| Localized | 2 | 4 |

**Progression to peak deficit:-**

| DAYS | No. of patients | Percentage |
|---|---|---|
| 1 | 12 | 24 |
| 2 | 24 | 48 |
| 3 | 9 | 18 |
| 4 | 4 | 8 |
| 5 | 1 | 2 |

**Grade of disability (On admission & at peak):-**

| Grade | On admission | | At peak | |
|---|---|---|---|---|
| | No. of patients | Percentage | No. of patients | Percentage |
| 1 | - | - | - | - |
| 2 | 3 | 6 | - | - |
| 3 | 15 | 30 | 1 | 2 |
| 4 | 24 | 48 | 32 | 64 |
| 5 | 8 | 16 | 17 | 34 |
| 6 | - | - | - | - |

**Cranial nerve dysfunction:-**

| Cranial Nerve | Number of patients | Percentage |
|---|---|---|
| VII – Unilateral, Bilateral | **27** | **54** |
| IX, X | **9** | **18** |
| III, IV, VI | **1** | **2** |

**Table 2** Baseline Characteristics

**Autonomic dysfunction:-**

| Autonomic dysfunction | Number of patients | Percentage |
|---|---|---|
| Cardiac Arrhythmia | 5 | 10 |
| Postural Hypotension | 4 | 8 |
| Fluctuating B.P. | 8 | 16 |
| Transient Urinary retention & hesitancy | 6 | 12 |

**CSF Protein level:-**

| CSF Protein (mg %) | No. of patients | Percentage |
|---|---|---|
| < 45 | 10 | 20 |
| 45 – 100 | 22 | 44 |
| 100 – 150 | 15 | 30 |
| >150 | 3 | 6 |

**Nerve conduction studies:-**

| Type | Number of patients | Percentage |
|---|---|---|
| Demyelinating | 15 | 30 |
| Axonal | 10 | 20 |
| Mixed | 25 | 50 |

**Motor conduction abnormalities (Percentage of involved nerves):-**

| Nerve | DL prolongation | Distal amplitude reduction | CV reduction | Conduction block | Inexcitable nerves |
|---|---|---|---|---|---|
| Median | 62 | 80 | 68 | 26 | 8 |
| Ulnar | 68 | 74 | 70 | 18 | 8 |
| Peroneal | 78 | 64 | 74 | 28 | 24 |
| Tibial | 80 | 68 | 78 | 30 | 20 |

**Sensory conduction abnormalities (Percentage of involved nerves):-**

| Nerve | CV reduction | Reduced amplitude | Absent SNAP |
|---|---|---|---|
| Median | 24 | 18 | 34 |
| Ulnar | 28 | 26 | 22 |
| Sural | 18 | 18 | 16 |

**Complications:-**

| Complications | Number of patients | Percentage |
|---|---|---|
| Aspiration pneumonia | 8 | 16 |
| Septicemia | 1 | 2 |
| Urinary tract infection | 1 | 2 |
| DVT | 1 | 2 |

**Disability on discharge:-**

| Grade | No. of Patients | Percentage |
|---|---|---|
| 1 | 3 | 6 |
| 2 | 12 | 24 |
| 3 | 30 | 60 |
| 4 | 2 | 4 |
| 5 | - | - |
| 6 | 3 | 6 |

**GB syndrome variants:-**

| Variants | No. of patients | Percentage |
|---|---|---|
| AIDP | 39 | 78 |
| AMAN | 7 | 14 |
| AMSAN | 2 | 4 |
| Miller-Fisher variant | 1 | 2 |
| Pharyngeal Cervical brachial variant | 1 | 2 |

## DISCUSSION

A total of 50 patients were included in this prospective study. The maximum number of patients was in between 21 to 28 years age group (34%) and Mean age was 35.61 years. Kaplan *et al*[1] reviewed 2575 cases and found the peak incidence to be between 50 and 60 years of age. Similarly Peter C. Dowling[7] also reported peak incidence between 16 to 25 years age group. In Thanakan *et al*[3] series however, the mean age of study group was only 28 years. There is a male preponderance in our study which is in conformity with the report by Robert M. *et al*.[8] However, Peter C. Dowlin's[7] study Showed an equal incidence in males and females. No seasonal variation in incidence of GBS could be inferred from this study in conformity with the majority of studies in literature[8]. However a few studies have noted a seasonal clustering of cases. Kaur *et al*[9] reported an increased incidence in summer and autumn. Peter C. Dowling[7] also noted an increase in summer. Twenty three (46%) of our patients had a definite antecedent event prior to onset of illness. Winer *et al*[5] reported 52% of GBS patients experience symptoms of viral respiratory or gastrointestinal infections. Ropper *et al* also reported a high incidence of 73%. Zhahirul Islam *et al* showed 69% had antecedent illness of which 37% of cases[12]. The interval between prodromal illness and onset of GBS is most frequently from 1-3 weeks. Occasionally it is as long as 6 weeks. Kaur *et al*[9] reported a mean interval of 9.2 days. In our study there is a mean interval of 9.06 (± 4.21) days between the prodrome and the onset of GBS. Ascending paralysis was noted in 58% (29 patients) and descending paralysis in 6% (3 patients), while 32 % (16 patients) had simultaneous involvement of all four limbs. A study Winer *et al*[5] showed ascending paralysis in 66% and involvement of four limbs simultaneously in 34% cases. A metaanalysis of large series by Allan H. Ropper[2] showed ascending paralysis in 60%, descending paralysis in 20% and involvement of all four limbs simultaneously in 20% cases. The first symptom of illness was motor in the form of flaccid paralysis in 64% cases and 36% had sensory symptoms. Allan H. Ropper[2] in his metaanalysis reported 85% incidence of paraesthesia. In a study by Winer *et al*[5] 75% patients had paraesthesia. All patients had involvement of the legs and involvement of limbs was symmetric in all cases. None of the patients had involvement of hands alone, which is inconformity to the observation of Winer *et al*[5]. Respiratory failure was present in 34% of our patients. Allan H.Ropper2 in his meta analysis showed that 10% of patients have respiratory failure. Winer *et al*5 noted a 23% incidence of respiratory failure. Thirty two (64%) patients reached grade IV (bedridden state). In the study by Winer *et al*[5] noted 88% bedridden cases. This is in contrast to the report by RDM Hadden *et al*[4] who said 40% patients become bedbound. Overall, about 50% of patients with GBS reach maximal weakness by 1 week, 70% by 2 weeks, and 80% by 3 weeks in the course of illness5. In this study 24% of patients reached peak deficit within 1 day of onset of illness, 90% by 3 days. 56% of our patients had cranial nerve dysfunction.20% of patients had involvement of multiple cranial nerves. This is in conformity with the 50% incidence reported by Winer *et al*[5] and 60% in Allan H. Ropper's2 meta analysis. Kaur *et al*[9] reported an incidence of 41% in her study from North India. Autonomic dysfunction is reported to occur in up to 50% of GBS patients P.Hachenecker *et al*[10] noted dysfunction in 69% of their patients. NK Singh *et al*[6] documented 67% incidence. In this study autonomic dysfunction occurred in 46% of patients. Cardiac arrhythmia occurred in 10% of cases, postural hypotension in 8% of cases, Fluctuating Blood pressure was noted in 16% of patients. Transient sphincteric dysfunction in the form of urinary retention and hesitancy was seen in six (12%) patients. Allan H.Ropper's meta analysis reported 15% incidence of transient bladder disturbances in GBS patients NK Singh *et al*6 observed sphincter disturbance in 20% of patients. CSF protein was raised above 45 mg% in 40(80%) patients. Winer *et al*[5] reported raised CSF protein in 80% patients while 90% was reported in Allan H.Ropper's2 meta analysis.CSF pleocytosis was seen in three patients. CSF mononuclear cell counts of up to 50 per cmm may be seen in GBS and does not rule out diagnosis of GBS. Electrophysiological studies were conducted in all patients and 15 of them showed demyelinating pattern, 10 of them showed axonal pattern, 25 patients mixed pattern. Two patients initially had prolongation of f latency and absent H wave as the only feature and rest of the conduction were normal, on repeat conduction after one week showed demyelinating pattern. Many authors have found a proportion of patients to have normal nerve conduction and also involvement of nerves in varying severity. The population varies form 9% to 20%11 and is higher in the first few weeks of illness. This multi focal involvement has been explained as due to The patchy nature of pathology of GBS which means that studies confined to one or two nerves may miss abnormal findings. Maximum conduction velocities may conceal abnormalities since conduction can occur normally in some fibres while being partially blocked in some others.

Lastly it is likely that proximal conduction blocks occur commonly in GBS that distal motor conduction would be unaffected15 Three patients died in this study. One patient developed aspiration pneumonia and later died due to Septicemia and shock. One patient had fluctuating blood pressure and cardiac arrhythmia and died due to cardiac arrest. One patient died due to cardiac arrest while on ventilator. Case fatality in this study was 6%. Mortality in GBS varies from 1.3% to 13% in different series with a mean of about 6% Winer *et al*[5] reported 13% mortality in his study of 100 patients. NK Singh *et al*[6] noted 8% mortality. Of all GB syndrome variants AIDP subtype predominates which was demonstrated in various studies. In this study 39 patients (78%) diagnosed to have AIDP, 7 patients (14%) diagnosed to have AMAN, 2 patients (4%) diagnosed to have AMSAN. Other variants like Miller- Fisher variant was observed in 1 patients and pharyngeal cervical brachial variant was observed in 1 patient. AIDP is the predominant subtype in United states and Europe (up to 90%) and axonal subtype predominates in china (70% AMAN, 25%AIDP,5% others)13 Hadden *et al*[14] noted 71% AIDP,24% AMAN, 4% AMSAN, 1% Miller Fisher subtypes in his study. Zhahirul Islam[12] *et al* showed AIDP in 82% cases, AMAN in 15% cases, AMSAN in 2% cases and MFS IN 1 % cases. Gupta *et al*[7]1noted AIDP in 70% cases, AMAN in 20% cases, Miller Fisher variant in 5 % cases in India.

**Comparison of age group in different study:-**

| | Present study | Kaplan *et al* | Peter C Dowling *et al* |
|---|---|---|---|
| Max No of Patients | 21 to 28 year | 50-60 year | 16-25 year |

**Comparison of sex distribution in different study:-**

| | Present study | Robert m etal | Peter c dowling |
|---|---|---|---|
| Male | 64% | 60% | 50% |
| Female | 36% | 40% | 50% |

**Comparison of antecedent illness prior to GBS in different study:-**

| | Present study | Winter *et al* | Allan H Rooper *et al* | Zhahirul islam *et al* |
|---|---|---|---|---|
| Antecedent illness | 46% | 52% | 73% | 69% |

**Comparison of time interval between prodromal illness and onset of GBS in different study:-**

| | Present study | Kaur *et al* |
|---|---|---|
| Time interval between prodromal illness and GBS | 9.06 days | 9.2 days |

**Comparison of mode of onset of paralysis in GBS:-**

| | Present study | Allan H Rooper *et al* | Winter *et al* |
|---|---|---|---|
| Ascending paralysis | 58% | 60% | 66% |
| Descending paralysis | 6% | 8% | 0% |
| Quadriparesis | 32% | 32% | 34% |

**Comparison of first symptom of GBS in different study:-**

| | Present study | Winter *et al* | Allan H Ropper *et al* |
|---|---|---|---|
| Motor | 64% | 25% | 15% |
| Sensory | 36% | 75% | 85% |

**Comparison of respiratory failure in different study:-**

| | Present study | Allan H Ropper *et al* | Winter *et al* |
|---|---|---|---|
| Respiratory failure (Grade 5) | 34% | 10% | 23% |

**Comparison of bed bound state in different study:-**

| | Present study | Hadden *et al* | Allan h ropper *et al* |
|---|---|---|---|
| Bed bound state (Grade 4) | 64% | 40% | 88 |

**Comparison of cranial nerve involvement in different study:-**

| | Present study | Winter *et al* | Allan h roPper | Kaur *et al* |
|---|---|---|---|---|
| Cranial nerve involvement (Most common-seventh nerve) | 56% | 50% | 60% | 41% |

**Comparison of autonomic dysfunction in different study:-**

| | Present study | N K singh *et al* | P. Hachenecker |
|---|---|---|---|
| Autonomic dysfunction | 46% | 67% | 69% |

**Comparison of CSF proein elevation in different study:-**

| CSF protein (mg%) | Present study | Winter *et al* | Allan h ropper |
|---|---|---|---|
| <45 | 20% | 20% | 10% |
| >45 | 80% | 80% | 90% |

**Comparison of case fatality in different study:-**

| | Present study | Winter *et al* | N k singh *et al* |
|---|---|---|---|
| Case fatality | 6% | 13% | 8% |

**Comparison GBS variant in different study:-**

| GBS variant | Present study | Hadden *et al* | Zhahirul *et al* | Gupta *et al* |
|---|---|---|---|---|
| AIDP | 78% | 71% | 82% | 70% |
| AMAN | 14% | 24% | 15% | 20% |
| AMSAN | 4% | 4% | 2% | 5% |
| MILLER FISCHER | 2% | 1% | 1% | 5% |

**Abbrevations**

**GBS-** Guillain barre syndrome; **AIDP-** Acute Inflammatory Demyelinating Polyradiculoneuropathy; **AMAN-** Acute Motor Axonal Neuropathy; **AMSAN-**Acute Motor Sensory Axonal Neuropathy; **MFS-** Miller Fisher syndrome; **CIDP-**Chronic Inflammatory Demyelinating Polyradiculoneuropathy; **CSF-**Cerebro spinal fluid; **NINCDS-** National Institute of Neurological and Communicative Disorders and Stroke; **SNAP-** Sensory Nerve Action Potential; **CMAP-**Compound Muscle Action Potential; **IVIG-**Intravenous Immunoglobulin; **EMG-** Electromyography ; **NCV-**Nerve Conduction Velocity; **MNC-**Motor Nerve Conduction; **SNC-**Sensory Nerve Conduction; **CV-**Conduction velocity; **QSART-**Quantitative Sudomotor Axon Reflex Test; **MMSE-**Mini Mental State Examination; **CVS-**Cardiovascular system; **RS-**Respiratory system; **P/A-** Per Abdomen; **DL-** Distal latency; **P & D-** Proximal And Distal.

## CONCLUSION

GBS occurs in all age groups with a greater incidence in the age group below 50 years. However age did not have any correlation with prognosis. Mixed pattern is the most common pattern seen on electrophysiological studies. Axonal pattern has better recovery compare to demyelinating and mixed pattern. AIDP was the most common variant in studied population followed by AMAN variant and AMSAN variant respectively.

**Consent:-**

Informed consent was taken as per the standard procedures in the institution.

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**Conflicts of interest:-**

There are no conflicts of interest.

**Ethical clearance:-**

Obtained from the ethical committee of the institution.

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