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Guillain Barre Syndrome clinical profile and determination of its prognosis

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ABSTRACT

Introduction: GBS is a potentially life-threatening condition and medical emergency. It is a commonest cause of acute post infectious flaccid paralysis worldwide. GBS can result in serious complications that require immediate assessment and management as hospitalized patients. So in this study we aimed to study the clinical features, outcome and prognosis of GBS affected patients.

Materials and methods: A cross sectional study that included a total of 30 patients confirmed with guillain barre syndrome diagnosis during the study period of one and half year. Details pertaining to study population such as age, sex, personal history, socioeconomic status, past infectious history, relevant family history, seasonal preponderance, comorbidities and physical examination findings were collected. All relevant investigations were performed.

Results: GBS occurs in all age groups with a greater incidence in the 31-40 years age group. Male predominance was observed in the ratio of 4:1. Ascending type of paralysis, facial nerve involvement, autonomic dysfunction are most commonly seen in GBS with a predominant proximal muscle weakness. Respiratory failure occurs in 1/3rd of patients in GBS. Cranial nerve dysfunction occurs in 33.33% of patients in GBS. Rapid progression from onset to peak paralysis, prolonged duration of peak paralysis, need for ventilatory support and severity of paralysis are the factors associated with poor prognosis in GBS. Mortality in GBS is 27%. Delayed onset of recovery from paralysis, requirement of mechanical ventilatory support are significant prognostic factors of outcome in GBS.

Conclusion: Meticulous clinical examination helps to determine the severity of the patient. Early diagnosis and determination of subtypes have an important role for prognosis prediction.

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Observation of ICU patients for features of autonomic dysfunction is important to alleviate serious consequences.

Keywords: Clinical features, Outcome, Guillain-Barre syndrome.

INTRODUCTION

Guillain Barre Syndrome is a rare fatal condition in which immune mediated damage occurs in the peripheral nervous system. It is a potentially life-threatening condition and medical emergency. Exact cause behind Guillain barre syndrome (GBS) is unknown, but the majority of patients triggered by infections in the preceding six weeks.

Guillain Barre syndrome presents in several forms such as 1. acute inflammatory demyelinating polyradiculoneuropathy (AIDP) in which muscle weakness starts in the lower parts of body and causing ascending paralysis, 2.miller fisher syndrome (MFS) in which paralysis starts with the eyes, rare variant with cranial nerve involvement and ataxia predominantly, and 3.acute motor axonal neuropathy (AMAN) and acute motor sensory axonal neuropathy (AMSAN).

Globally GBS is now recognized as the commonest cause of acute post infectious flaccid paralysis [1]. The incidence of GBS is 1-2 cases per 100,000 which is a rare and important neurological emergency. The most common clinical manifestation of GBS is ascending paralysis, which starts in the lower part of the body and typically spreads to the arms. GBS patients can also present with cranial nerve involvement and 25% of patients develop respiratory depression and mechanical ventilation [2].

GBS can affect all age groups, but the risk increases with increasing age and it slightly shows preponderance towards males [3]. GBS may be triggered by most commonly with Campylobacter, Influenza virus, Epstein barr virus, Cytomegalovirus, Zika virus, Hepatitis A, B, C and E, Human immunodeficiency virus, Mycoplasma, surgery, trauma, hodgkin's lymphoma, COVID-19 virus and rarely COVID-19 and influenza vaccinations. GBS usually appears days or weeks after infectious etiology related to respiratory or digestive tract infection [4,5].

Predominant clinical manifestations of GBS are motor, sensory abbreviations in the form of distal paresthesias and numbness. Typically, it begins with fine paresthesia in the toes or fingers, followed within days by motor weakness. Pain is the most common presentation is either bilateral sciatica or aching pain, like the muscle discomfort following exercise, involving the large muscles of the thigh, flanks, or back. Occasional manifestations include objective sensory deficit, joint position, and vibratory sensation impairment, and stocking-glove deficits to pinprick. The common complications of GBS include respiratory failure due to aspiration, pneumonia and embolus, bladder dysfunction, depression, and syndrome of antidiuretic hormone secretion (SIADH) exacerbated by positive pressure ventilation.

Diagnosis of GBS by evaluating the history, presentation and clinical examination plays a vital role to treat the patient at the earliest which helps in recovery. Investigations such as lumbar puncture, nerve conduction studies and excluding other causes by screening infectious etiologies and other nerve pathologies. Nerve conduction abnormalities are the most sensitive and specific laboratory findings in GBS.

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GBS is a medical emergency condition that requires immediate assessment and management as hospitalized patients. So in this study we aimed to study the clinical features, outcome and prognosis of GBS affected patients.

The objectives of the study are:

1.To analyze the clinical profile of GBS

2.To study the prognosis in GBS with reference to age, onset of peak, duration of plateau phase, onset of improvement, and autonomic dysfunction features.

MATERIALS AND METHODS:

This study is a cross sectional study that included a total of 30 patients confirmed with guillain barre syndrome diagnosis during the study period of one and half year (January 2021 to June 2022) at the department of General Medicine, Government Medical College, Anantapuram. Informed consent was waived by all study populations. Institutional ethical committee agreed to proceed with this research study.

Among 48 GBS confirmed patients only 30 study populations were further studied and remaining 18 patients were not studied and their prognosis was not evaluated due to drop out from study or not returned to hospital for follow-up. Hence, in this study a total of 30 patients were evaluated.

Inclusion criteria:

i)Patient aged 18 years or more of both sexes diagnosed with GBS

ii)Lower &/or upper limbs weakness of either acute or subacute flaccid paralysis

iii)CSF showing albuminocytological association and electrophysiology revealing features of demyelinating axonal neuropathy and are compatible with a subtype of GBS level >0.45g/L.

Albuminocytological association was defined as CSF with raised protein and total cell count of ≤10/mm3.

Nerve conduction studies were performed on all areflexia or hyporeflexia patients. Motor conduction studies were performed on the median, ulnar and tibial nerves and sensory conduction studies were performed on the median and sural nerves using conventional procedures.

Details pertaining to study population such as age, sex, personal history, socioeconomic status, past infectious history, relevant family history, seasonal preponderance, comorbidities such as diabetes, neoplasia, hypothyroidism, renal failure, physical examination including upper and lower weakness, sensory loss, dysarthria, dysphagia, facial paresis were collected.

CSF analysis was performed for biochemistry and pathological investigations.

Grading of severity of weakness was graded using the medical research council including 5: normal, 4: opposes resistance, 3: opposes gravity, 2: moves joint, 1: flicker and 0: absent [6].

GBS disabling scale defined clinical course, adapted from hughes was graded as 0: healthy state, 1: minor symptoms and capable of running, 2: able to walk 10m or move without assistance but able to run, 3: able to walk 10 m across an open space with help, 4: bedridden or chair bound, 5: respiratory assisted ventilation for at least part of the day, 6: dead [7].

Statistical significance:

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All descriptive variables such as numbers, percentages and frequencies are calculated for categorical (nominal and ordinal) data. The association between qualitative and quantitative variables was analyzed using chi-square and fisher's exact test. The p value <0.05 was considered as statistical significance.

RESULTS:

The age of patients ranged from 13 to 67 years (Mean age 35 years) with the maximum number (46.67%) of patients in the 31- 40 age group. Twenty-four patients were males (80%) and 6 patients were females (20%).

Sixteen (53.3%) patients had some antecedent event prior to the development of GBS. The most common antecedent illness was upper respiratory tract infection (23.3%) followed by diarrhea (13.3%), fever (10%), lower respiratory tract infection (6.67%). In patients with a history of preceding illness, the mean duration between onset of GBS and the preceding illness was15 days (+/-5 days).

The first symptom of the illness was in the form of motor weakness in 11 (36.67%) patients, and it was sensory in the form of pain, paresthesia or numbness in the remaining 19(63.33%) patients. Remaining eleven (36.67%) patients did not have any sensory symptoms throughout the course of their illness (Table 1).

Table 1. Clinical manifestations of various GBS patients

Clinical features	No. of patients	Percentage
Weakness of legs	8	26.67
Weakness of legs and arms	3	10
Weakness of arms alone	0	0
Paraesthesia	12	40
Pain in legs	2	6.67
Pain in back	2	6.67
Numbness in legs	1	3.33
Generalized muscle aches	2	6.67
Type of paralysis		
Ascending paralysis	23	76.67
Descending paralysis	0	0
Simultaneous involvement of all 4 limbs	7	23.33

Twelve patients took up to 1 week to reach maximal weakness, 15 patients took up to 2 weeks, 2 patients took up to 3 weeks and 1 patient took longer than 3 weeks (24 days). The maximum number of admitted patients (40%) reached grade 4 disabilities at peak.

Ten patients (33.33%) developed respiratory paralysis and required mechanical ventilation. Eight (26.66%) patients died. The cause of death was aspiration pneumonia in 1 patient and cardiac arrest while on mechanical ventilation in other 7 patients. Later these 7 members had severe autonomic dysfunction with fluctuating blood pressure and heart rate.

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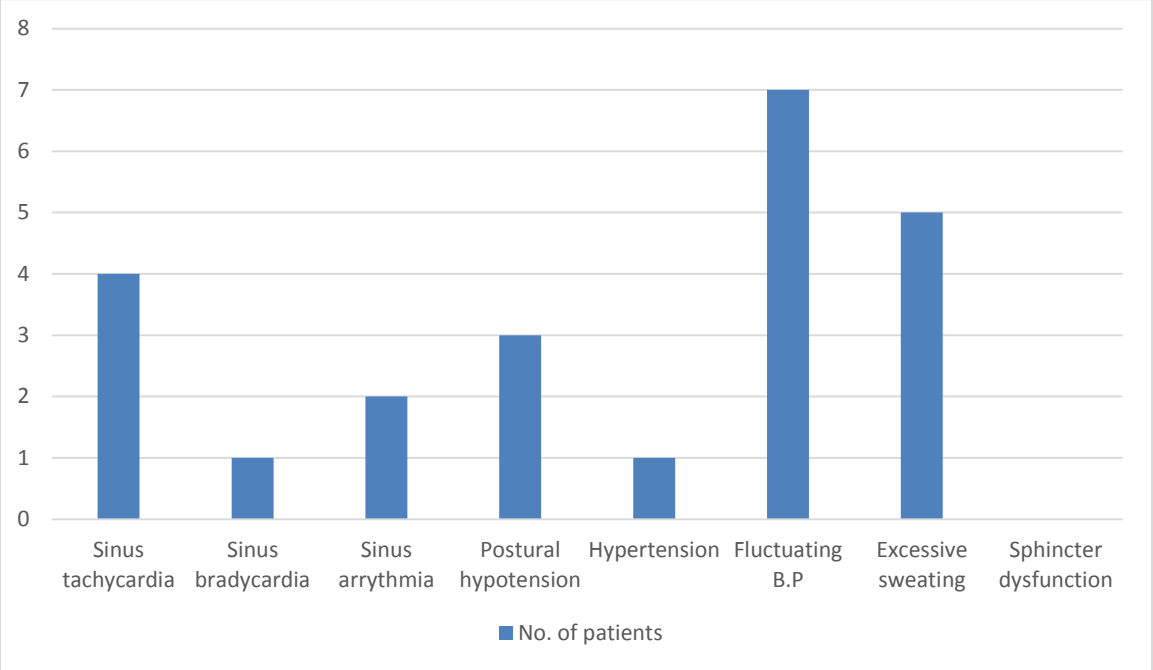
Ten patients (33.33%) had cranial nerve dysfunction. Seven patients (23.2%) had facial nerve palsy, among which 5 (16.6%) were bilateral. Three patients (10%) were involved in 9th & 10th cranial nerves.

Autonomic dysfunction was detected in 13 (43.33%) patients. Patients had fluctuating blood pressure, excessive sweating and sinus tachycardia followed by sinus bradycardia unresponsive to atropine. They finally died due to cardiac arrest (Fig 1).

Fig 1. Autonomic dysfunction features of GBS patients



Nerve conduction studies were conducted in all patients. Twenty patients (66.6%) were found to have reduced motor conduction velocities consistent with demyelinating neuropathy. Five patients (16.6%) were found to have decreased amplitude of action potentials consistent with axonal pattern of neuropathy. Three patients (10%) had mixed patterns of neuropathy. The remaining 2 patients (6.66%) had normal conduction studies.

33.33% patients had urinary tract infections, 26.67% patients developed aspiration pneumonia and 6.66% patients had septicemia. Eight patients (13.33%) died in this study. All eight patients developed respiratory failure and required assisted mechanical ventilation. One patient developed aspiration pneumonia and later died due to septicemia and shock. The other 7 patients died while on ventilation due to cardiac arrest (Fig 2).

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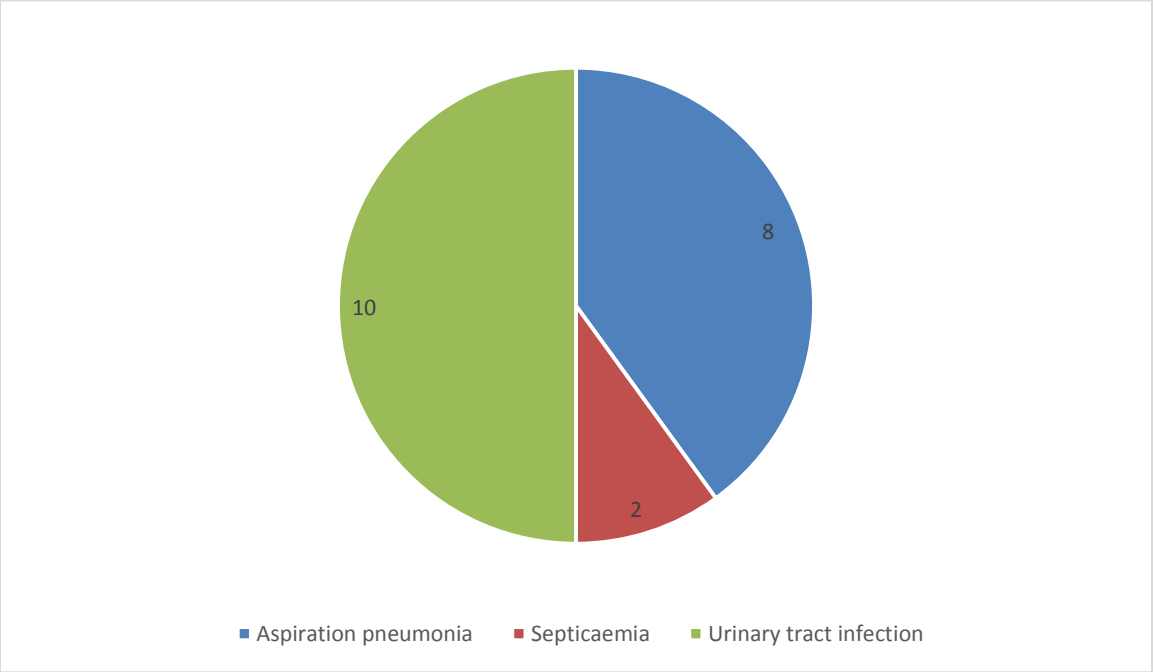
Fig 2. Complications observed among study population



A 'Good Outcome' group which had a disability grade of 3 or less at the end of 3 months and a 'Poor Outcome' group which had a disability grade of greater than 3 at the end of 3 months. Eighteen (81.81%) patients were found to have a good outcome while 4 (18.19%) had a poor outcome. In relation to age the p-value calculated was 0.91 which was not significant when it measured against outcome of patients.

Patients were grouped into two categories: a) 40 years and below and b) above 40 years of age. Outcome was then determined in each of these groups (Table 2).

Table 2. Age in relation to outcome of GBS patients

Age in years	No. of patients	Good outcome number	Bad outcome number
≤40	16	13 (81.25%)	3 (18.7%)
>40	6	5 (83.33%)	1 (16.67%)

Time taken from onset of GBS to peak deficit - The outcome in patients who attained peak paralysis in 1 week was compared with those who attained peak paralysis after 1 week. Six patients had peak paralysis within the first week and the remaining 16 patients attained peak paralysis only after 1 week. Fifteen patients in the delayed peak deficit group and 3 patients in the early peak deficit group had a good outcome. Three patients in the former group and one patient in the latter had a poor outcome.

Duration of Plateau Phase – Outcome in patients with a peak paralysis period (Plateau phase) of 1 week or less, was compared with outcome in those with a peak paralysis period of more than 1 week. Sixteen patients had a peak paralysis period of up to 1 week, while the remaining 6 patients had a plateau phase of more than 1 week. Fifteen patients in the former group and 3

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patients in the latter group, had a good outcome. Only 1 patient in the former group had a poor outcome, whereas 3 patients in the latter group had a poor outcome.

Duration from onset of GBS to recovery - The outcome of patients with duration from the onset of GBS to onset of recovery of up to 3 weeks was compared with outcome of those who took more than 3 weeks to recover. Eighteen patients started recovering within 3 weeks of onset while 4 patients took longer than 3 weeks. Sixteen patients in the former group and 2 in the latter group had a good outcome. Two patients in the former and 2 in the latter group had a poor outcome (Table 3).

On assessment of statistical significance of onset of peak paralysis, peak paralysis period showed significance in relation to duration of critical time.

Table 3. Critical time in relation to outcome of GBS patients

Critical time	No. of patients	Good outcome number (%)	Bad outcome number (%)
Onset of peak paralysis			
Up to 1 week	6	3 (50%)	3 (50%)
>1 week	16	15 (93.7%)	1 (6.25%)
Peak paralysis period (Plateau)			
Up to 1 week	16	15 (93.7%)	1 (6.25%)
>1 week	6	3 (50%)	3 (50%)
Onset of recovery			
Up to 3 weeks	18	16 (88.8%)	2 (11.12%)
>3 weeks	4	2 (50%)	2 (50%)

The outcome at the end of 3 months was correlated with the severity of paralysis (MRC grading) at plateau period. Four patients had the power of 0 – 1, among whom 1 patient had a good outcome and 3 had a poor outcome. Eighteen patients had power of grade 2 – 4, among whom 17 had a good outcome and 1 had a poor outcome. On applying Fisher's test, the difference in outcome in the two groups is found to be significant regarding the severity of paralysis and outcome (p=0.006).

The presence or absence of objective sensory loss was also compared with respect to outcome. All 3 patients who had sensory loss had a good outcome. Among the remaining 19 patients with no sensory loss, 15 had a good outcome and 4 had a poor outcome.

In 3 patients with evidence of bulbar paralysis, 1 had a good outcome and 2 had a poor outcome. In patients without bulbar paralysis, 17 and 2 patients had a good and poor outcome respectively.

Evidence of autonomic dysfunction was seen in 13 patients and 10 of them had a favorable outcome at 3 months. In patients without any autonomic dysfunction 8 out of 9 had a good outcome (Table 4).

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Table 4. Other possible prognostic neurological signs

Neurological sign	Number of patients	Good outcome number (%)	Bad outcome number (%)
Severity of paralysis			
Power grade 0-1	4	1 (25)	3 (75)
Power grade 2-4	18	17 (88.9)	1 (11.1)
Sensory loss			
Present	3	3(100)	0
Absent	19	15 (78.94)	4(21.06)
Sphincter dysfunction			
Present	0	0	0
Absent	22	18 (81.82)	4 (18.18)
Bulbar paralysis			
Present	3	1(33.33)	2(66.67)
Absent	19	17 (89.47)	2 (10.53)
Autonomic dysfunction			
Present	13	10(76.93)	3(23.07
Absent	9	8 (88.88)	1 (11.12)

DISCUSSION:

Guillain Barre Syndrome is an acute onset immune mediated peripheral neuropathy which is characterized by rapidly progressive motor weakness involving the spinal roots, the peripheral nerves and occasionally the cranial nerves. All patients with suspected GBS should be hospitalized for vigilant monitoring due to the high risk of respiratory failure and need for intubation and mechanical ventilation. Baseline spirometry, including FVC and oximetry, should be obtained. The cornerstone of treatment is that of meticulous general medical support.

A very mild case with only distal paresthesia and mild limb weakness may not need treatment, but it is advisable to wait approximately two weeks before concluding that there will be no further progression. Patients with vital capacities that are rapidly declining or below 18 ml/kg or those with cardiovascular dysautonomia are appropriate candidates for observation in an ICU.

The most important advances in treatment of GBS have been positive pressure ventilation and intensive respiratory and medical management in the ICU, both introduced during the European Poliomyelitis Epidemic of the 1950s. These have allowed patients with complications of immobilization and respiratory failure to survive and recover from paralysis. Mortality rate of GBS remains high and about 20% of patients admitted may be facing a long-term disability despite effective management regimens such as intravenous immunoglobulin and PE [8].

A long run retrospective study from 1986-2001 noted among 23 childhood patients 18 had acute inflammatory demyelinating polyradiculoneuropathy (AIDP), 2 had miller fisher syndrome, and 1 patient had axonal forms, and 2 patients were unclassified [9]. Behnaz Ansari et al [10] reported based on electrophysiologic and clinical findings majority of the study population had

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acute motor and sensory axonal neuropathy (AMSAN) (24%), followed by 13.6% patients with acute motor axonal neuropathy (AMAN), 12.6% patients with AIDP and remaining 2.1% acute polyradiculopathy.

In this prospective study of 30 patients with GBS (based on Asbury's Criteria), it was found to be commonest in the 31-40 years age group and there was a male preponderance. A study from Oman on GBS epidemiology and clinical features observed the age-specific incidence in the age group of 1-4 years and 5-9 years was 1.26 and 0.24 per 100,000 children respectively [11]. Male predominance seen in GBS patients and number of cases more in young adults and the elderly patients [12]. Behnaz Ansari et al [10] noted that out of 388 GBS patients, males were 241 (62.1%) and females were 147 (37.9%). The mean age of the patients was 42.78±21.34. 53.6% patients did not have any preceding infection, 21.4% patients had upper respiratory tract infections, 8% patients had diarrhea and vomiting, 3.6% patients presented with fever and remaining 13.4% patients manifested with other infections.

A retrospective study on 27 children diagnosed with GBS diagnosis noted group 1 children had complete or partial recovery extended beyond 2 months from onset of the disease and group 2 children recovered fully within 2 months from onset of the disease [13]. A multicenter retrospective study [14] in Saudi Arabia reported that GBS patients were presented with upper respiratory tract infection (39.1%), diarrhea (27.8%), weakness (98.7%), sensory symptoms (64.1%), facial diplegia (43.1%), oropharyngeal weakness (33.8%), ophthalmoplegia (12.4%) and 26.3% needed mechanical ventilation. Death of one patient was caused by septicemia.

Areflexic symmetric motor paralysis was the presenting feature of all the patients with the majority showing an ascending type of paralysis (76.66%) in this study. Pi-Lien Hung et al [9] observed limb weakness is the most common clinical manifestation, with various degrees of motor weakness. The predominant cranial palsy manifestation was bulbar involvement, it was 30% of the episodes. Sensor symptoms were observed in 61% of the episodes. Limb weakness was the most common symptom noted in various studies [15,16].

33.33% of patients in this study had cranial nerve dysfunction. This is in conformity with the 50% incidence reported by Winer et al [17] and 60% in Allan H Ropper's [18] meta analysis. Kaur et al [19] reported an incidence of 41% in her study from North India.

Case fatality in this study was 26.66%. Mortality in GBS varies from 1.3% to 13% in different series with a mean of about 6%. Winer et al [17] reported 13% mortality in his study of 100 patients. NK Singh et al [20] noted 8% mortality.

In this study, as predictors of poor prognosis, we found in this study there was a rapid progression to peak paralysis (within 7 days), a prolonged duration of peak paralysis (greater than 7 days), need for ventilation and severity of peak paralysis, axonal and mixed pattern of nerve conduction study results, all significantly affected outcome of patients.

Ammache Z [13] et al study documented that clinical features and electrophysiological features were assessed statistically which showed clinical features including maximum disability score at presentation, intubation and cranial nerve involvement were considered as poor outcome and electrophysiologic feature such as conduction block was considered as good outcome. The

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relation between maximum disability score at presentation and the probability of incubation was significant.

In this study were the severity of muscle weakness at peak, presence of objective sensory loss, cranial nerve paralysis and autonomic dysfunction. However, none of the above parameters were found to be statistically significant. The study by NK Singh et al [20] shows similar results with severity of paralysis at peak, adversely affecting outcome in the patients. However, his study showed that the presence of bulbar paralysis is also associated with a poor prognosis. In our study, although out of 10 patients with cranial nerve paralysis, three had a bad outcome, the association with outcome is not found to be significant.

A study stated that after a follow-up of 1 year or more, 20 patients recovered, 3 had residual and no fatalities occurred. Clinical manifestations of GBS among children noted shorter recovery time when compared to adult patients. Accurate and prompt management helps to recover patients with a predictive of good outcome [14]. Using the MRC-SS score categorization the outcome of GBS patients was analyzed, 25% of patients showed full recovery, 61.3% showed good recovery but with minor residual weakness and 13.7% had particle recovery with significant persistent weakness. There were no patients with limited or no recovery [21].

Conclusion:

GBS occurs in all age groups with a greater incidence in the 31-40 years age group. Male predominance was observed in the ratio of 4:1. Ascending type of paralysis, facial nerve involvement, autonomic dysfunction are most commonly seen in GBS with a predominant proximal muscle weakness. Respiratory failure occurs in 1/3rd of patients in GBS. Cranial nerve dysfunction occurs in 33.33% of patients in GBS. Rapid progression from onset to peak paralysis, prolonged duration of peak paralysis, need for ventilatory support and severity of paralysis are the factors associated with poor prognosis in GBS. Mortality in GBS is 27%. Delayed onset of recovery from paralysis, requirement of mechanical ventilatory support are significant prognostic factors of outcome in GBS. Meticulous clinical examination helps to determine the severity of the patient. Early diagnosis and determination of subtypes have an important role for prognosis prediction. Observation of ICU patients for features of autonomic dysfunction is important to alleviate serious consequences.

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