

Original Research Article

A Study on Guillain Barre Syndrome - Clinical Profile and Treatment Outcome

P. Chandrasekaran*

Associate Professor, Department of Neurology, Government Mohan Kumaramangalam Medical College, Salem, Tamil Nadu, India

*Corresponding author email: drpchandrasekaran@yahoo.co.in

| | | |
|--|---|--------------------------------------|
|  | International Archives of Integrated Medicine, Vol. 7, Issue 9, September, 2020. | |
| | Available online at http://iaimjournal.com/ | |
| | ISSN: 2394-0026 (P) | ISSN: 2394-0034 (O) |
| | Received on: 05-08-2020 | Accepted on: 15-08-2020 |
| | Source of support: Nil | Conflict of interest: None declared. |
| How to cite this article: P. Chandrasekaran. A Study on Guillain Barre Syndrome - Clinical Profile and Treatment Outcome. IAIM, 2020; 7(9): 1-11. | | |

Abstract

Background: 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. 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 a large loss of productivity and burdens health care due to its prolonged morbidity. It is a heterogeneous disorder in its type, severity, pathogenesis, and prognosis. GBS is characterized by 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 is Acute Inflammatory Demyelinating polyradiculopathy (AIDP), Acute Motor Sensory Axonal Neuropathy (AMSAN), and Acute Motor Axonal Neuropathy (AMAN) and the rare one are pharynx-cervical Brachial variant, Bilateral foot drop, and bifacial weakness.

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

Materials and methods: This was a prospective study, conducted from August 2018 to December 2019. Those patients who had been admitted with the diagnosis of GBS, in the medical, neuromedical, emergency medical, or intensive medical care unit of the department of neurology, Government Mohan Kumaramangalam Medical College, Salem, Tamil Nadu was included in the study. Data regarding the demographic features like age, sex distribution, and month of occurrence, clinical features like antecedent illness, the involvement of cranial nerves, and autonomic dysfunction were collected.

Results: Uni or bilateral facial nerve involvement was observed in 22 patients and bulbar weakness was observed in 13 patients. 8 patients had both features. Oculomotor weakness was noted in 5 patients who belonged to the Miller Fischer Syndrome group. Autonomic disturbances, which are generally considered bad prognosticators were noted in a total of 27 patients. They were tachy or

bradycardias, heart blocks, and postural hypotension. Among the 63 patients registered, 35 had a typical onset of ascending quadriparesis with areflexia. 4 patients had the onset of their symptoms in the upper limb. In 18 patients, the onset of weakness was simultaneous in all 4 limbs and cranial nerves. The pain was the predominant presenting feature in 5 patients though they had weakness and areflexia. Positive sensory paresthesias like pins and needles sensations were noted in 8 patients.

Conclusion: The mean improvement in the GBS disability scale from admission to the end of the 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.

Key words

Guillain Barre Syndrome (GBS), Autonomic disturbances, Miller Fischer Syndrome.

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 [1]. 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 a large loss of productivity and burdens health care due to its prolonged morbidity. It is a heterogeneous disorder in its type, severity, pathogenesis, and prognosis. GBS is characterized by 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 [2]. Among those which produce weakness, the common one is Acute Inflammatory Demyelinating polyradiculopathy (AIDP), Acute Motor Sensory Axonal Neuropathy (AMSAN), and Acute Motor Axonal Neuropathy (AMAN) and the rare one are pharynx-cervical Brachial variant, Bilateral foot drop, and bifacial weakness [3]. 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 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 normal amplitude. AMAN and AMSAN are characterized by the reduction or absence of amplitude of CMAP and both CMAP and SNAP respectively [4]. 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 [4]. The resemblance of AIDP to experimental autoimmune neuritis suggests pathogenetic mechanisms involving T-cell induced, macrophage associated demyelination. This proposed autoimmune etiology leads to the induction of immunotherapy. Intravenous Immunoglobulin (IVIG) and plasma exchange (PE) are the standard treatment options available at present [5].

Materials and methods

This was a prospective study, conducted from August 2018 to December 2019. Those patients who had been admitted with the diagnosis of GBS, in the medical, neuromedical, emergency medical, or intensive medical care unit of the department of neurology, Government Mohan Kumar Mangalam Medical College, Salem, Tamil Nadu was included in the study. Data regarding the demographic features like age, sex distribution, and month of occurrence, clinical features like antecedent illness, the involvement of cranial nerves, and autonomic dysfunction were collected. The inclusion criteria consisted

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 four weeks. We also included patients who presented with features of GBS subtypes without prominent weakness. The exclusion criteria consisted of the 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. 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, the 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. The cerebrospinal fluid analysis was done whenever possible and relevant. Investigations like liver function tests, thyroid function tests, 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 a disability grade of > 3 in the GBS disability scale and those with progressively increasing weakness, the definite treatment options (IVIG or plasma exchange) were started. Due to non-availability, some patients received only injection methylprednisolone. Patients were followed up throughout their stay in the hospital. Intensive medical care was provided for those patients with an advanced stage of the 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. 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 the prevailing disability score was noted for further analysis.

Statistical analysis

THE mean and standard deviation was calculated for certain variables, which follow a normal distribution. The association of two categorical variables was evaluated by chi-square tests. The significance of the association of certain factors like the treatment adopted and poor prognosticators with the outcome variables like death, ventilator need, tracheostomy, and bedridden state were measured by stepwise logistic regression analysis. Statistical significance was considered when the p-value was < 0.05. The mean improvement of the disability score was calculated for each of the treatment modalities.

Results

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 (**Table – 1**).

Uni or bilateral facial nerve involvement was observed in 22 patients and bulbar weakness was observed in 13 patients. 8 patients had both features. Oculomotor weakness was noted in 5 patients who belonged to the Miller Fischer Syndrome group (**Table – 2**).

Autonomic disturbances, which are generally considered bad prognosticators were noted in a total of 27 patients. They were tachy or bradycardias, heart blocks, and postural hypotension (**Table – 3**).

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 | - | - |

Table – 2: Cranial nerve involvement.

| Total of 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 | - |

Table – 3: Autonomic dysfunction.

| Total (23) | Tachycardia (17) | Bradycardia (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 | - | - |

Table – 4: Mode of presentation.

| | Typical Ascending Type Quadriparesis | Upper limb onset | Simultaneous Onset In Limbs and Cr Nv | Pain is a 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 | - | - |

Among the 63 patients registered, 35 had a typical onset of ascending quadriparesis with areflexia. 4 patients had the onset of their symptoms in the upper limb. In 18 patients, the onset of weakness was simultaneous in all 4

limbs and cranial nerves. The pain was the predominant presenting feature in 5 patients though they had weakness and areflexia. Positive sensory paresthesias like pins and needles sensations were noted in 8 patients (**Table – 4**).

Table – 5: CSF results.

| Total (35) | Done in I Week (11) | | Done in II weeks (24) | |
|--------------|---------------------|----------|-----------------------|----------|
| | Normal | Abnormal | Normal | Abnormal |
| AIDP | 8 | 1 | 8 | 7 |
| AMSAN | - | - | 3 | 1 |
| AMAN | - | - | 1 | 1 |
| MFS | 2 | - | 2 | 1 |
| Pure Sens | - | - | - | - |
| Unclassified | - | - | - | - |

Table – 6: Peak disability after onset of symptoms (in days).

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

Table – 7: 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 |
| Unclassified (0) | - | - | - | - | - |

Table – 8: Treatments given for various subtypes of GBS.

| | I 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 |

CSF analysis was done in 11 patients in the first week and in 24 patients during the 2nd week and 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 – 5**).

Table – 9: Outcome related events for patients with poor prognostic factors.

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

* > 3 score in GBS disability grading

** ≤ 3 score in GBS disability grading

> 5 scores in MRC disability grading

≤ 5 scores in MRC disability grading #

Significance of influence of prognostic factors for poor outcome.

Table – 10: Comparison of treatment options with clinical outcome at 8 weeks among GBS subtypes who presented with severe disability.

| | | Death | Ventilator | Tracheo stormy | 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) | - | - | - | - | - |

Table – 11: Mean improvement in disability scores for various treatment options.

| Disability Scale | Treatment Option | Total Numbers | Mean Improvement |
|--|--------------------|---------------|------------------|
| GBS disability scale p = 0.002 [#] | I | 22 | 1.09 |
| | Plasma Exchange | 9 | 0.67 |
| | Methylprednisolone | 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 |
| | Methylprednisolone | 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 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 – 6**).

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 persistence or prolongation of minimal latency was noted in 48 patients. Distal latency prolongation was noted in 39 patients (**Table – 7**).

In our study IVIG was given to 18 patients, plasma exchange was given for 9 patients and injection methylprednisolone was given for 25 patients. No specific treatment was provided for 4 patients who presented with a 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 were initiated (**Table – 8**).

Among the 18 AIDP patients who were treated, 6 were from mild disability group and 12 were from severe disability groups. Among the 15 AIDP patients who were treated with injection methylprednisolone, 9 were from severe

disability and 6 were from mild disability groups. Also, 4 patients with severe disability groups were treated with plasma exchange, and 2 from mild disability groups. Four patients with AIDP, who had a mild disability were not treated with any specific form of treatment. Three AMSAN patients of severe disability were treated with IVIG and methylprednisolone and two by plasma exchange. All patients in the AMSAN group had presented with a severe disability. One AMAN patient of severe disability group was treated each with IVIG and plasma exchange and one with a mild disability was treated with methylprednisolone. In the group of 3 patients who presented with a very acute and severe form of illness, no effective treatment was started before they expired (**Table – 9**).

The mean improvement in GBS disability scores from admission to the end of 8 weeks was calculated for all GBS patients (**Table – 10**).

The mean improvement in GBS disability scores from admission to the end of 8 weeks was calculated for AIDP subtype of GBS patients (**Table – 11**).

Discussion

Among the total 63 patients registered, most patients were noted in the 20 to 40 year age group (50.8%). Only 3.2% of the 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 Government Mohan Kumar Mangalam Medical College, Salem, Tamil Nadu. Most surveys show a slight peak in late adolescence and young adult. Some studies show a peak also in the elderly age group. 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. We have noted 46% (29 patients) of patients with an antecedent illness which includes diarrhea, respiratory infection, pregnancy, chickenpox, and surgery. 54% of our patients did not have any specific preceding illness. Each 20.6% of patients had respiratory infection or diarrhea as a preceding illness. A typical pattern of ascending type areflexic quadriparesis was noted in 47.6% of patients and the simultaneous onset of illness in all 4 limbs \pm cranial nerve involvement was noted in 27% of patients [7]. Pain as a predominant feature in addition to weakness was observed in 5 patients (8%) of cases. 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. Our study shows facial nerve involvement in 34.9% of cases and bulbar involvement in 20.6% of cases. 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 a poor outcome in the form of death. Hence we believe severe postural hypotension may be a predictor of poor prognosis [8]. 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 outcomes. 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 persistence or prolongation of minimum latency was the next commonest abnormality noted (87.2%) in GBS as a whole and its subtype AIDP (92.7%) [9]. 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 him 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 [10]. AMAN was noted in 4.8% and MFS in 7.9% of 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 [11]. All the three patients expired within one or two days and hence this group increases the overall mortality percentage of GBS patients. In our study IVIG was administered to 22 patients (34.9%), plasma exchange was given to 9 patients (14.3%) and injection methylprednisolone was given to 25 patients (39.6%). As already noted 3 patients with a fulminant form of illness were not able to receive either of this 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 [12]. Apart from periodically assessing the motor power of limbs, the 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 [13]. 38 patients were admitted in our study with an admission disability score of more than 3 in the GBS disability scale and >5 in the MRC disability scale. They constituted 60.3% of the total patients registered. A good outcome is considered when the patient can walk which is a score of ≤ 3 in Hughe's GBS disability scale and ≤ 5 in the MRC disability scale. Poor outcome is considered when the patient chair or bed bound, ventilator, or expired, which is a score of >3 in the GBS disability scale and >5 in MRC disability scale. In our study at the end of 8 weeks, 24 patients had a GBS disability score of >3 (MRC disability score >5) which contributed 38.1% of the total admitted patients [14]. 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 [15]. 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 [16]. Totally 27 patients (47.4%) had cranial nerve involvement and the rest did not. When the cranial nerve involvement was present, a good outcome was noted in 17.5% of patients and poor outcome was noted in 29.8% of 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 ($P = 0.002$) [17]. Totally 26 patients (45.6%) had autonomic disturbance.

When autonomic dysfunction was present 19.3% of patients had a good outcome and 26.3% had a 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 an antecedent illness and diarrhea was noted in 13 patients (22.8%) [18]. In the patients with diarrhea, the 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, the good outcome was noted in 28.1% of patients and poor outcome was noted in 31.6% of patients [19]. 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 ($P = 0.044$). Among the 22 patients who were treated with IVIG 14 patients (24.6%), had a good outcome and 8 patients (14.0%) has a poor outcome. Among the 9 patients who were treated with plasma exchange, 6 patients had a good outcome and 3 had a poor outcome. Among the 19 patients, who were treated with injection methylprednisolone, 8 patients had a good outcome and 11 patients had a 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%) [20].

Conclusion

The prolonged morbidity of the illness evidenced by the need for tracheostomy is more for those treated with methylprednisolone 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 a single breath count. Autonomic dysfunction, bulbar weakness, rapidity of onset of illness, severe grade

disability, and diarrhea are significantly correlating with poor outcomes. 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.

References

1. Olive J M, Castillo C, Garcia Castro R, de Quadros CA. Epidemiologic study of Guillain-Barre syndrome in children <15 years of age in Latin America. *J Infect Dis.*, 1997; 175 (suppl): S160-64.
2. Giovannoni G, Hartung H P. The immunopathogenesis of multiple sclerosis and Guillain-Barre syndrome. *Curr Opin Neurol.*, 1996; 9: 165-77.
3. Asbury AK, Amason BGW, Karp HR, McFarlin DF. Criteria for diagnosis of Guillain-Barre syndrome. *Ann Neurol.*, 1998; 3: 565-66.
4. Asbury AK, Comblath DR. Assessment of current diagnostic criteria for Guillain-Barre syndrome. *Ann Neurol*, 1990; 27 (suppl): S21-24.
5. Jiang GX, Cheng Qi, Link H, de Pedro-Cuesta J. Epidemiological features of Guillain-Barre syndrome in Sweden, 1978-93. *J Neurol Neurosurg Psychiatry*, 1997; 62: 447-53.
6. Beghi E, Kurland LT, Mulder DW, Wiederholt WC. Guillain-Barre syndrome: clinico-epidemiologic features and effectiveness of the influenza vaccine. *Arch Neurol*, 1985; 42: 1053-57.
7. Govoni V, Granieri E, Casetta I, et al. The incidence of Guillain-Barre syndrome in Ferrara, Italy: is the disease increasing? *J Neurol Sci.*, 1996; 137: 62-68.
8. Kaplan JE, Schonberger LB, Hurwitz ES, Katona P. Guillain-Barre syndrome in the United States, 1978-1981: additional observations from the national surveillance system. *Neurology*, 1983; 33: 633-37.
9. Visser LH, van der Meche FGA, Meulstee J, et al. Cytomegalovirus infection and Guillain-Barre syndrome: the clinical, electrophysiologic, and prognostic features. *Neurology*, 1996; 47: 668-73.
10. Prevots DR, Sutter RW. Assessment of Guillain-Barre syndrome mortality and morbidity in the United States: implications for acute flaccid paralysis surveillance. *J Infect Dis.*, 1997; 175 (suppl): 151-55.
11. Winer JB, Hughes RAC, Anderson MJ, et al. A prospective study of acute idiopathic neuropathy: II antecedent events. *J Neurol Neurosurg Psychiatry*, 1988; 51: 613-18.
12. Rees JR, Soudain SE, Gregson NA, Hughes RAC. *Campylobacter jejuni* infection and Guillain-Barre syndrome. *N Engl J Med.*, 1995; 333: 1374-79.
13. Jacobs BC, van Doorn PA, Schmitz PIM, et al. *Campylobacter jejuni* infections and anti-GMT antibodies in Guillain-Barre syndrome. *Ann Neurol.*, 1996; 40: 181-87.
14. Mishu B, Ilyas AA, Koski CL, et al. Serologic evidence of previous *Campylobacter jejuni* infection in patients with the Guillain-Barre syndrome. *Ann Intern Med.*, 1993; 118: 947-53.
15. Kuroki S, Saida T, Nukina M, et al. *Campylobacter jejuni* strains from patients with Guillain-Barre syndrome belong mostly to Penner serogroup 19 and contain (3-N- acetylglucosamine residues. *Ann Neurol.*, 1993; 33: 243-47.
16. Fisher M. An unusual variant of acute idiopathic polyneuritis (syndrome of ophthalmoplegia, ataxia, and areflexia). *N Engl J Med.*, 1956; 255: 57-65.
17. Jacobs BC, Endtz Hugh, Van der Meche

- FGA, et al. Serum anti-GQ1b IgG antibodies recognize surface epitopes on *Campylobacter jejuni* from patients with Miller Fisher syndrome. *Ann Neurol.*, 1995; 37: 260-64.
18. Walsh FS, Cronin M, Koblar S, et al. Association between glycoconjugate antibodies and *Campylobacter* infection in patients with Guillain-Barre syndrome. *J Neuroimmunol.*, 1991; 34: 43-51.
19. Rees JH, Gregson NA, Hughes RAC. Anti-ganglioside GMI antibodies in Guillain- Barre syndrome and their relationship to *Campylobacter jejuni* infection. *Ann Neurol.*, 1995; 38: 809-16.
20. Chiba A, Kusunoki S, Shimizu T, Kanazawa I. Serum IgG antibody to ganglioside GQ1b is a possible marker of Miller Fisher syndrome. *AnnNeurol.*, 1992; 31: 677-79.