Clinical Characteristics, Laboratory Findings and Functional Outcomes of Guillain-Barre Syndrome: Experience of 50 Cases in Bangladesh

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Abstract

Background: Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy which is one of the most frequent causes of acute flaccid paralysis. GBS has diverse clinical phenotype and functional outcome. **Objective:** The purpose of the present study was to evaluate clinical profiles and outcomes of GBS patients admitted in a tertiary care centre of Bangladesh. **Methodology:** This prospective observational study was conducted in the Department of Neurology from January 2016 to June, 2020 in BIRDEM General Hospital, Dhaka, Bangladesh. Adult patients more than or equal to 18 years of age who had fulfilled the diagnostic criteria of GBS were included in the study. **Results:** Majority of the patients were male (64.0%). The most common presenting symptom at entry was ascending paralysis that occurred in 24 patients (48.0%). Mean age of the study population is 31.5 years ranging from 19 to 60 years. Twenty-four (48.0%) patients had GBS disability score of 4 at entry. On the contrary, 39(78.0%) cases had GBS disability score of 4 at nadir. Diarrhea was reported in 14(28.0%) cases and respiratory tract infection was reported in 9(18.0%) cases. Fifty-six percent of patients had GBS disability score of 0 to 2, 38.0% had 3 to 4 and only 6.0% had 5 to 6. Majority of the patients had Brighton criteria level 1 certainty of diagnosis (62.0%) in this study. GBS variants according to nerve conduction studies were AIDP (54.0%), AMAN (34.0% and AMSAN (12.0%). CSF protein was raised in 72.0% cases. Patients diagnosed with AMAN and AMSAN had worse outcome after 3 months in comparison to AIDP group of patients if we consider GBS disability score. **Conclusion:** GBS has been found more in younger and male population group in this present study where Antecedent events were not found in majority of the patients. AIDP was the commonest variant in our study with comparatively good outcome followed by AMAN. *[Journal of National Institute of Neurosciences Bangladesh, July 2023;9(2):108-115]*

Keywords: Guillain Barre' Syndrome; Acute Inflammatory Demyelinating Polyneuropathy; Acute Motor Axonal Neuropathy; Acute Motor Sensory Axonal Neuropathy; functional outcome of GBS

Introduction

Guillain-Barré Syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy which is monophasic. It affects male slightly more than females irrespective of ages, races and nationalities. The worldwide incidence of GBS ranges from 0.6 to 4.0/100,000 people[1]. GBS is the most frequent cause of acute flaccid paralysis. Overall incidence of GBS is 1.1 to 1.8/100,000 and it was however lower in children at 0.34 to 1.34/100,000[2]. The increased incidence of GBS during winter in some countries is thought to be due to the increased incidence of respiratory tract infections caused by Mycoplasma pneumoniae or Haemophilus influenzae. By contrast, an increase in the incidence of GBS has been observed during summer in northern China and Bangladesh, which is thought to be associated with an increased frequency of preceding diarrhea[3,4]. GBS in Bangladesh is frequently preceded by an enteric infection caused by Campylobacter jejuni[5]. Frequent exposure to enteric pathogens at an early age may

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increase the incidence of GBS. Overall, the crude incidence rate of GBS in children less than 15 years of age varied from 1.5 to 2.5 cases per 100,000 populations per year in the 6 divisions of Bangladesh[6].

GBS is characterized by rapidly progressive ascending weakness that initially affects the limbs and can also affect the cranial and respiratory muscles. The severity of GBS is highly variable, ranging from mild limb weakness to complete paralysis, respiratory failure and even death. There are several variants of GBS which have been defined on the basis of their clinical presentation, including a pure motor variant, sensorimotor variant and Miller Fisher syndrome (MFS). Miller Fisher syndrome (MFS) is characterized by the clinical triad of ophthalmoplegia, ataxia and areflexia. Several subtypes of GBS have also been identified on the basis of electrophysiological features, including acute inflammatory demyelinating polyneuropathy (AIDP) and acute motor axonal neuropathy (AMAN). Patients with AIDP may have the classic sensorimotor variant of GBS, whereas those with AMAN typically have the pure motor variant. In some patients with axonal GBS, both sensory and motor fibres are affected; that is termed acute motor and sensory axonal neuropathy (AMSAN) and is sometimes considered to be a severe variant of AMAN[7-10]. These axonal variants have also been described from other countries. Patients with AMAN have a more rapid progression of weakness to an earlier nadir than in Acute inflammatory demyelinating polyneuropathy (AIDP) resulting in prolonged paralysis and respiratory failure over a few days[11].

GBS is a complex autoimmune disease of especially the proximal peripheral nerves and the nerve roots mediated in AIDP by lymphocytic mononuclear cell infiltration and intense macrophage-associated segmental demyelination. AMAN is characterized by the paucity of lymphocytic infiltration and sparing of the dorsal nerve roots, dorsal root ganglia and peripheral sensory nerves. In cases with AMAN, immunoglobulin G and complement activation products were identified bound to the nodal axolemma of motor fibers. The suspected target auto-antigen is likely GD1a since IgG antibodies to GD1a are detectable in 60.0% of AMAN cases and only 4.0% of AIDP. Molecular mimicry is suggested as the pathogenetic mechanism of AMAN based on the strong association with C. jejuni infection. AMSAN shares many similarities with AMAN although the attack in AMSAN is more severe or longer lasting resulting in more intense and ultimately diffuse Wallerian-like degeneration of both sensory and motor axons[12]. An antecedent infection is noted two to four weeks prior to the onset in most GBS cases. The commonest are upper respiratory infections without any specific organism identified. Known viral precipitants such as Epstein-Barr virus (mononucleosis or hepatitis), and cytomegalovirus (CMV) occur in only 6.0% of cases. C. jejuni enteritis is the most common identifiable antecedent infection and precedes axonal GBS in up to 33.0% of patients[13].

CSF analysis is very important in GBS cases and may reveal albuminocytologic dissociation; that is an elevated protein up to 1,800 mg/dl with 10 or less white cells/in most cases. Half of GBS cases may have a normal CSF protein in the first week but that proportion declines to 10% if the test is repeated a week later. Pleocytosis of 10–20 cells/mm3 is seen in ~5% of cases and should not dissuade one from a diagnosis if the clinical and electrodiagnostic features are otherwise typical. Most MFS cases have albuminocytologic dissociation[14].

Plasma exchange removes antibodies and other potentially injurious factors from the blood stream. It involves connecting the patient's blood circulation to a machine which exchanges the plasma for a substitute solution, usually albumin. Several studies have evaluated plasma exchange for Guillain-Barré syndrome. Plasma exchange is the first and only treatment that has been proven to be superior to supportive treatment alone in Guillain-Barré syndrome. Plasma exchange is regarded as the treatment option in comparison to new treatments, such as intravenous immunoglobulin[15]. The postulated mechanisms of action of IV immunoglobulin (IV IgG) in neuromuscular disorders include interference with co-stimulatory molecules involved in antigen presentation and modulation of autoantibodies, cytokines and adhesion molecules production as well as macrophage Fc receptor[16]. IVIG is a proven effective treatment for GBS. Several clinical factors are associated with the outcome. A low increase of serum IgG levels after a standard IVIG dose appeared to be significantly associated with slower recovery and a worse prognosis. The disease severity at the time of IVIG treatment appears to influence the increase in serum IgG level. The lowest increase in IgG was found in patients with more extensive disability and weakness, as defined by the GBS disability score and the Medical Research Council (MRC) sum score. IVIG is an expensive treatment that may induce (generally minor) side effects and is currently not indicated (proven to be effective) in mildly affected GBS patients. A second IVIG dose potentially seems to be indicated in patients with a poor prognosis[17].

GBS has diverse clinical phenotype. Majority of

large-scale studies on Guillain-Barré syndrome (GBS) have been conducted in developed countries. Some studies on clinical profile, incidence and outcome are done in Bangladesh also. This present study was undertaken to evaluate clinical profile and outcome of GBS patients admitted in a tertiary care centre of Bangladesh.

Methodology

Study Design: This prospective observational study was conducted in the department of neurology, BIRDEM general Hospital from January 2016 to June, 2020 for a period of four and an half year. The adult patients more than or equal to 18 years of age who fulfilled the diagnostic criteria of GBS admitted in the neurology department were included in the study. Patients with previous trauma leading to paresis, previous neuromuscular weakness, periodic paralysis, transverse myelitis, hypokalemic paralysis, acute-onset chronic inflammatory demyelinating polyneuropathy, spinal disc herniation, vasculitis, previous episode of Guillain-Barre´ syndrome were excluded from the study.

Study Procedure: The data regarding the epidemiology, clinical profile, laboratory values, electrodiagnostic finding, treatment received and outcome of the patients with GBS were recorded. Diagnosis of Guillain-Barre syndrome was assessed by Brighton criteria and classified into different levels of certainty ranging from level 1 to level 4. Clinical course was described by using the Guillain-Barre´ syndrome disability scale, a widely accepted scale of disability for patients with Guillain-Barre´ syndrome ranging from 0 (normal) to 6 (death)[18]. Weakness was expressed using the Medical Research Council (MRC) sum score of six bilateral muscles in arms and legs, ranging from 0 (tetraparalytic) to 60 (normal strength). Nadir was defined as the highest Guillain-Barre´ syndrome disability score or the lowest MRC sum score excluding small fluctuations of less than five points within the margins of the inter-observer variations[18-19]. All necessary data were collected to classify these patients according to the Brighton criteria. Data from nerve conduction studies were used to classify patients in electrophysiological subgroups, including demyelinating polyneuropathy, axonal polyneuropathy or combined. CSF count and protein concentration were determined by routine diagnostic methods. The normal value for CSF protein concentration was 0.15 to 0.45 g/l. Subjects in this study were classified according to Brighton criteria[18]. The primary outcome of the study was the clinicoepidemiological profile and functional

outcome of patients with Guillain- Barre syndrome. Functional outcome of the patients was assessed by Guillain-Barre´ syndrome disability scale[18].

Statistical Analysis: Data were filled into MS Excel 2010 and analyzed by SPSS 20 version. For descriptive analysis frequency, percentage, mean, median, standard deviation, and interquartile range were calculated and presented in tabular form whereas for inferential statistics independent t-test was applied as per need to find out the difference between groups. The considered values were statistically significant at a 95% confidence interval if P was less than 0.05.

Ethical Clearance: The study was approved by the local ethical committee of BIRDEM and all patients gave written informed consent.

Results

In this observational study, among 50 patients with GBS, the majority of the patients were male (64.0%). The most common presenting symptom at entry was ascending paralysis that occurred in 24 patients (48.0%). The other common symptoms presented were sensory disturbances in 13(26.0%) patients, respiratory failure in 9(18.0%) patients, cranial nerve involvement in 9(18.0%) patients and dysphagia in 8(16.0%) patients. Mean age of the study population was 31.5 years ranging from 19 to 60 years. However, 28(56.0%) patients were from urban population and 24(48.0%) patients had GBS disability score of 4 at entry. On the contrary, 39(78.0%) had GBS disability score of 4 at nadir. Furthermore, 13(26.0%) patients had respiratory failure and 14(28.0%) patients had cranial nerve involvement at nadir. Diarrhea was reported in 14(28.0%) and respiratory tract infection was reported in 9(18.0%) cases. However, the antecedent event among 54.0% cases were unknown (Table 1).

The mean with SD of MRC score at entry was 38.3 ± 6.8 and at nadir was 33.5 ± 5.3. The mean with SD of GBS disability score at entry was 3.68 ± 0.65 and at nadir was 4.14 ± 0.45 (Table 2).

Severity of limb weakness at entry and nadir expressed as MRC score was shown in figure I. Mean with SD of MRC score at entry was 38.3 ± 6.8 and at nadir was 33.5 ± 5.3.

Duration of the progressive phase was shown in figure II. Time of evolution to maximal weakness was between 6 to 10 days in 44.0% of patients and more than 10 days in only 18.0% of the patients. Majority of the patients (82%) reached the nadir of weakness within 10 days in this study.

Table 1: Description of the patients with Guillain-Barre syndrome (n=50)

Variables	Frequency	Percent
Gender		
• Male	32	64.0
• Female	18	36.0
Age of Study Population	31.5 (19 to 60)	
Residence		
• Urban	28	56.0
• Rural	22	44.0
Diabetes mellitus		34.0
Symptoms of antecedent infection		
• Respiratory tract infection	9	18.0
• Diarrhoea	14	28.0
Neurological symptoms at entry		
GBS disability score		
3	21	42.0
4	24	48.0
5	5	10.0
Ascending paralysis	24	48.0
Sensory disturbance	13	26.0
Dysphagia	8	16.0
Bladder involvement	4	8.0
Respiratory failure	9	18.0
Cranial nerve involvement	9	18.0
Neurological symptoms at nadir		
• Respiratory failure	13	26.0
• Cranial nerve involvement	14	28.0
Treatment		
• I/V IgG	15	30.0
• Physiotherapy	17	34.0
• Plasmapheresis	18	36.0
GBS disability score after 3 months		
• 0 to 2	28	56.0
• 3 to 4	19	38.0
• 5 to 6	3	6.0

Table 2: Neurological Symptoms, MRC Score and GBS Disability Score at Entry and Nadir

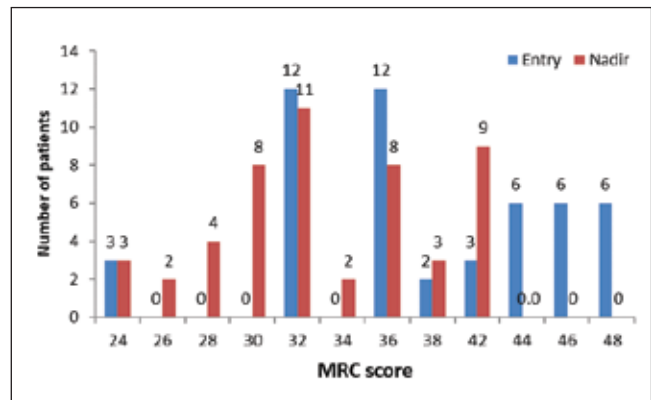| Variables	At entry	At nadir
GBS disability score		
3	21 (42.0%)	2 (4.0%)
4	24 (48.0%)	39 (78.0%)
5	5 (10.0%)	9 (18.0%)
GBS disability score (mean±SD)	3.68 ± 0.65	4.14 ± 0.45
MRC score (mean±SD)	38.3 ± 6.8	33.5 ± 5.3
Ascending paralysis	24 (48.0%)	-
Sensory disturbance	13 (26.0%)	-
Dysphagia	8 (16.0%)	-
Bladder involvement	4 (8.0)	
Respiratory failure	9 (18.0)	13 (26.0)
Cranial nerve involvement	9 (18.0)	14 (28.0)

Figure I: Severity of Limb Weakness at Entry and Nadir Expressed as MRC Score (n=50).
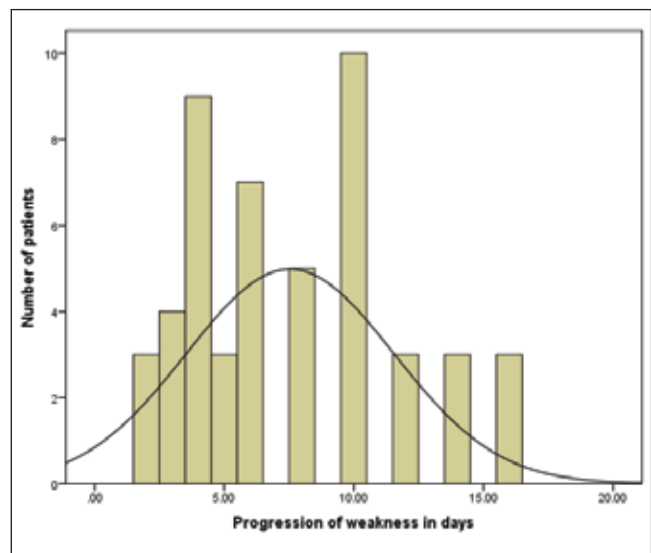

Figure II: Duration of the progressive phase defined as the number of days between onset of Limb Weakness and Reaching Nadir (n=50)

The time of evolution to maximal weakness was between 6 to 10 days in 44.0% patients and more than 10 days in only 18.0% patients (Table 3).

Table 3: Time of evolution to maximal weakness in study population with GBS (n=50)

Time to maximal weakness (days)	Frequency	Percent
<3	3	6.0
3 to 5	16	32.0
6 to 10	22	44.0
More than 10	9	18.0
Total	**50**	**100.0**

GBS variants according to nerve conduction studies were AIDP (54%), AMAN (34% and AMSAN (12.0%). By considering the CSF study, 94.0% patients had less

than 5 cells. CSF protein was raised in 72.0% cases (Table 4).

Table 4: CSF finding, protein and cell count of the patients with Guillain-Barre syndrome

Lab Parameters	Frequency	Percent
Cell Count		
• 0 to 5	47	94.0
• More than 5	3	6.0
Nerve conduction study	31.5 (19 to 60)	
AIDP	27	54.0
AMAN	17	34.0
AMSAN	6	12.0
CSF Protein		
• Normal	14	28.0
• Raised	36	72.0

The majority of the patients had Brighton criteria level 1 certainty of diagnosis (62.0%) (Figure III).
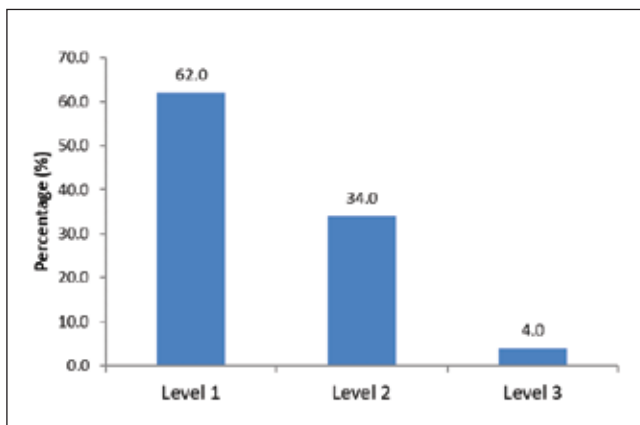
Figure III: Brighton criteria level of diagnostic certainty of diagnosis of Guillain-Barre syndrome (n=50)

About 34.0% cases of study population had level 2 diagnosis certainty. Again 36.0% cases of the patients received plasmapheresis and 30% of the patients received I/V immunoglobulin. There was significant statistical difference between AIDP, AMAN and AMSAN groups of patients if we consider GBS disability score and MRC score at entry and at nadir (Table 5).

MRC score at entry was significantly lower at entry and nadir in AMAN and AMSAN group of patients. Patients diagnosed with AMAN and AMSAN had worse outcome after 3 months in comparison to AIDP group of patients if we consider GBS disability score (Table 6).

Discussion
In this present study, the clinical, electrophysiological and laboratory features in adult patients with Guillain-Barré´ syndrome was examined. Among 50 patients with GBS, the majority of the patients were male (64.0%). The most common presenting symptom at entry was ascending paralysis that occurred in 24(48.0%) patients. The other common symptoms presented were sensory disturbances in 13(26.0%) patients, respiratory failure in 9(18%) patients cranial nerve involvement in 9(18.0%) patients and dysphagia in 8 patients (16.0%). Mean age of the study population is 31.5 years ranging from 19 to 60 years. Another study shows that, among 31 cases their patient's mean age was 17 years (SD-12). The common symptoms were ascending paralysis in 29(93.5%) patients, sensory disturbance in 7(22.6%) patients, and respiratory failure in 5(16.1%) patients. The most common antecedent event was respiratory tract infection (29.0%) followed by surgery (9.7%)[20-21].
The prevalence of ascending paralysis in some other studies showed 100% in India and 83.33% in china[22].

Table 5: GBS disability score and MRC score at entry and at nadir of the patients with Guillain-Barre syndrome according to nerve conduction study findings

Variables	AMAN	AMSAN	AIDP	P Value
GBS disability score at entry	3.47±0.80	4.00±0.00	3.74±0.59	0.182
GBS disability score at nadir	4.29±0.47	4.37±0.00	4.07±0.47	0.213
MRC score at entry	35.76±7.90	34.00±2.19	40.96±5.80	0.010
MRC score at nadir	31.65±5.44	31.00±1.10	35.26±5.30	0.039

Table 6: GBS disability score after 3 months of the patients with Guillain-Barre syndrome

Outcome after 3 months	AMAN	AMSAN	AIDP	P Value
0 to 2	5 (29.4%)	0 (0.0%)	23 (85.2%)	
3 to 4	9 (52.9%)	6 (100.0%)	4 (14.8%)	0.001
5 to 6	3 (17.6%)	0 (0.0%)	0 (0.0%)	

In this study, twenty-four (48%) patients had GBS disability score of 4 at entry. On the contrary, 39(78%) had GBS disability score of 4 at nadir. Thirteen (26.0%) patients had respiratory failure, 14 patients (28%) had cranial nerve involvement at nadir. Another study showed that, (61.0%) patients had GBS disability score of 4 at entry, 10.0% of their patients needed ventilator support[23]. In this study, diarrhea was reported in history in 14(28.0%) and respiratory tract infection was reported in 9(18.0%) cases as antecedent event. However, the antecedent event among 54.0% was unknown. Diarrhea was reported in 24.0% and respiratory tract infection was reported in 38.0% cases in another study[23]. It has been found that, mean with SD of MRC score at entry was 38.3 ± 6.8 and at nadir was 33.5±5.3. Mean with SD of GBS disability score at entry was 3.68 ± 0.65 and at nadir was 4.14 ± 0.45 in this study. Severity of weakness (MRC sum score) was found to be 44 at entry and 39 at nadir in another study[23]. It has been found that 56.0% had GBS disability score of 0 to 2, 38.0% had 3 to 4 and only 6.0% had 5 to 6 after 3 months. Time of evolution to maximal weakness was between 6 to 10 days in 44.0% of patients and more than 10days in only 18.0% of the patients. Majority of the patients (82%) reached the nadir of weakness within 10 days in this study. In a study in Netherlands, 97.0% of patients their reached the nadir of their disease within 4 weeks[24].

If we consider the CSF study, 94.0% patients had less than 5 cells and CSF protein was raised in 72.0% cases in this study. CSF protein was found to be more than 64.0% cases and cell count was less than 5 in 85.0% of cases in other study. In all 455 patients in that study, where CSF was examined, the cell count was less than 50 cells/mL, confirming the specificity of this finding[23]. CSF examination may be useful in cases of clinical uncertainty about the diagnosis, especially to exclude other causes associated with CSF pleocytosis, such as infectious polyradiculitis and acute poliomyelitis[24]. In another study, CSF analysis was performed in 123(78.8%) patients, at a median of 5 (IQR, 2-14) days after symptom onset, and "cytoalbuminological dissociation" was observed in 85 (69.1%) patients[25].

The common GBS variants according to nerve conduction studies were AIDP (54%), AMAN (34.0% and AMSAN (12.0%) in this study. Routine nerve electrophysiology was performed in 440 patients in another study. In almost all patients the findings were compatible with the presence of a neuropathy. The predominant subtype was acute inflammatory demyelinating polyneuropathy (48.0%)[23]. Another study showed that, demyelinating neuropathy was more common that axonal variety[26]. There was significant statistical difference between AIDP, AMAN and AMSAN groups of patients if we consider GBS disability score and MRC score at entry and at nadir. MRC score at entry was significantly lower at entry and nadir in AMAN and AMSAN group of patients. In this study, it has been found that, patients diagnosed with AMAN and AMSAN had worse outcome after 3 months in comparison to AIDP group of patients if GBS disability score is considered. Another study revealed that, patients with AMAN had a more rapid progression of weakness to an earlier nadir than in AIDP resulting in prolonged paralysis and respiratory failure over a few days[27].

At present there are no definite agreed-upon diagnostic electrophysiological criteria for the diagnosis of Guillain-Barré syndrome. All current electrophysiological criteria focus on the discrimination between axonal and demyelinating subtypes of Guillain-Barré syndrome. The subtyping of Guillain-Barré syndrome is complex as the electrophysiology examination requires high standards and skills; various classification systems have been developed; and patients with axonal variants may initially show features usually attributed to demyelination, such as conduction blocks and prolonged distal motor latency[28]. The diagnostic value of electrophysiology may be improved by serial measurements and more sensitive techniques and by developing criteria both for Guillain-Barré syndrome in general and optimizing the criteria for the various subtypes of Guillain-Barré syndrome[29]. Majority of the patients had Brighton criteria level 1 certainty of diagnosis (62.0%) in this study. 34.0% of study population had level 2 diagnosis certainty. Another study showed that, patients had Brighton criteria level 1 certainty of diagnosis (61.0%), level 2(33.0%) in their study[23].

Early and accurate recognition of Guillain-Barré syndrome may be challenging in such a clinically heterogeneous disorder, especially when there are also alternative diagnoses possible. As such, it will be important to emphasize careful documentation of clinical features of suspected cases of Guillain-Barré syndrome to physicians. Additional investigations may play a crucial role in the diagnosis of Guillain-Barré syndrome. It would be helpful if electrophysiological criteria were developed that could support the

diagnosis of Guillain-Barre´ syndrome in general, instead of discriminating between the variant subtypes of Guillain-Barre´ syndrome[29]. Guidelines for the diagnostic work-up, documentation and management of Guillain-Barre´ syndrome in clinical practice are therefore most needed. In this study, it has been tried to see the demographic profile, patient characteristics and functional outcome in Bangladesh perspective. It is one of the few studies of the country to deliver data regarding clinical-epidemiological profile and outcome of the GBS. Further studies are required to clarify these issues which will be helpful for the clinicians and researchers in managing the patients. Further multicentre prospective studies among patients with GBS to determine their long-term prognosis and interventional studies to assess and compare the effectiveness of therapeutics in GBS are recommended. There are some limitations of this study. This was single centered study observational study with small sample size. Long-term prognosis of GBS could not be assessed.

Conclusion

GBS was found more in younger and male population group in our study. Antecedent events were not found in majority of the patients. Majority of the patients had Brighton criteria level 1 certainty of diagnosis and had good functional outcome. AIDP is the commonest variant in our study with comparatively good outcome followed by AMAN. Early diagnosis and initiation of treatment is vital for good functional outcome.

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