ORIGINAL ARTICLE

Clinical Profile of Patients with Guillain-Barré Syndrome Admitted to Bangabandhu Sheikh Mujib Medical University

Md. Mahbubur Rahman[1], A.K.M. Anwar Ullah[2]

Abstract:

This retrospective study was done in the Department of Neurology and Intensive Care Unit (ICU) of Bangabandhu Sheikh Mujib Medical University (BSMMU) from January 2004 to June 2005. The objective of the study was to see the different presentation of Guillain-Barré syndrome patients admitted in BSMMU. A total number of 40 patients were enlisted during the study period. Among them, 23 (57.50%) were male and 17 (42.50%) were female. Most of the patients were in second and third decade of life. All the patients had flaccid paralysis in all four limbs with some sensory features without bowel and bladder involvement. Some of the patients developed autonomic features. Thirteen (32.50%) patients developed respiratory failure requiring ICU support. It was concluded that Guillain-Barré syndrome is not uncommon in this country and therefore physicians should be accustomed with different presenting features of the disease.

Introduction:

A leading cause of acute flaccid paralysis of muscles is the acute paralytic neuropathy diagnosed clinically as the Guillain-Barré syndrome (GBS)[1]. Patients with GBS and related variants suffer from an acute onset of autoimmune neuropathy. It occurs year-round at a rate of about one case per million per month. Males and females are equally at risk and adults are more frequently affected than children.

Guillain-Barré syndrome is characterized by symmetrical ascending flaccid paralysis, areflexia and albuminocytological dissociation in cerebrospinal fluid (CSF)[2]. The disease may be difficult to diagnose at the onset because the characteristic changes slow down the nerve conduction and increase in spinal fluid protein may be delayed. Early diagnosis is important as prompt intervention using plasmapheresis or intravenous immunoglobulin (Ig) G can arrest or reverse the disease process.

Patients may initially present with paraesthesia, sensory symptoms with weakness or weakness alone. The fairly symmetrical weakness of the lower limbs ascends proximally over hours to several days to involve the arms, facial and oropharyngeal muscles and in severe cases, respiratory muscles. Its severity varies from mild involvement in which patients are still capable of walking unassisted to quadriplegia. Hyporeflexia or areflexia are invariable features. The progression ends by 1-4 weeks.

Materials and method:

This was a retrospective study done in the Department of Neurology and Intensive Care Unit, Bangabandhu Sheikh Mujib Medical

1. Assistant Professor, Department of Neurology, Holy Family Red Crescent Medical College and Hospital, Dhaka.
2. Professor and Chairman in-charge, Department of Neurology, Bangabandhu Sheikh Mujib Medical University, Dhaka.

University (BSMMU), Dhaka, during the period from January 2004 to June 2005. During this period, a total number of 40 patients were admitted. Patients admitted with acute flaccid type of weakness in all four limbs progressing over days to four weeks were included in the study. Cases with diabetes mellitus, renal and hepatic failure, occupational history of exposure to lead, history of exposure to toxins were not included. Patients with acute intermittent porphyria, with multifocal motor neuropathy (MMN) and amyotrophic lateral sclerosis (ALS) were also not included in this series. Clinical profile of the study population were analyzed which included demographic as well as clinical data.

Results:

All the age categories were almost uniformly distributed. Patients in their second and third decades suffered more than the other age groups (Table-I). There were 23 (57.50%) male and 17 (42.50%) females, with male-female ratio 1:0.74 (Table-II).

Table – I: Age distribution of the study subjects (n=40)

Age (years)	Number of patients	Percentage
<20	05	12.50
20-30	15	37.50
31-40	09	22.50
41-50	08	20.00
>50	03	7.50

Table – II: Sex distribution (n=40)

Sex	Number of patients	Percentage
Male	23	57.50
Female	17	42.50

Cranial nerve involvement was present in 21 (52.50%) patients with seventh cranial nerve, five (12.50%) with ninth and tenth cranial nerves and 14 (35%) had no involvement (Table-III).

Table – III: Cranial nerve involvement (n=40)

Cranial nerve	Number of patients	Percentage
7th	21	52.50
9th + 10th	05	12.50
No involvement	14	35.00

Distribution of motor function showed that nine (22.50%) had wasting of muscle. Muscle tone diminution was seen in all the patients. As muscle power was examined, nine (22.50%) were in grade-0, 19 (47.50%) in grade-1 and 12 (30%) in grade-2. Reflexes were absent in all 40 (100%) cases (Table-IV).

Table – IV: Status of motor function (n=40)

Parameters	Number of patients	Percentage
Motor function		
Wasting of muscles	09	22.50
Tone of muscles diminished	40	100.00
Power of muscle diminished:		
Grade 0	09	22.50
Grade 1	19	47.50
Grade 2	12	30.00
Reflexes		
Absent	40	100.00

Evaluation of sensory function showed paraesthesia/pain in 23 (57.50%) cases. All 40 (100%) cases demonstrated intact sensory function like pain, touch and temperature sense. Position sense was lost in five (12.50%), however, vibration sense was significantly lost in eight (22.50%) cases (Table-V).

Table – V: Status of sensory function (n=40)

Parameters	Number of patients	Percentage
Paresthesia/pain	23	57.50
Pain, touch, temperature sensation retained	40	100.00
Position sense lost	05	12.50
Vibration sense lost	09	22.50

Evaluation of autonomic function showed that 17 (42.50%) had tachycardia, three (7.50%) bradycardia, two (50%) hypertension, two (50%) hypotension and three (7.50%) trophic changes (Table-VI).

Table – VI: Distribution of autonomic function (n=40)

Parameters	Number of patients	Percentage
Pulse:		
Tachycardia	17	42.50
Bradycardia	03	7.50
Normal	20	50.00
Blood pressure:		
Hypertensive	02	5.00
Hypotensive	02	5.00
Normal	36	90.00
Trophic change:		
Present	03	7.50

Respiratory function involvement was present in 13 (32.50%) cases (Table-VII).

Table – VII: Respiratory function involvement (n=40)

Involvement	Number of patients	Percentage
Present	13	32.50
Absent	27	67.50

Discussion:

This was a retrospective study which included 40 patients. Guillain-Barré syndrome occurs in any age group. In this study most of the patients were in second and third decades of life. It coincides with the study of Haque et al.[3] in which most patients were in the third decade of life. In the study 57.50% subjects were male and rest were female (ratio 1:0.74). It conforms with a study in which males appeared to be commonly affected[4].

In this study, 26 (65%) patients had cranial nerve involvement, of which 52.50% had facial nerve involvement. This is consistent with the study of Asbury and Cornblath[5], who showed involvement of facial nerve in about 45-70% patients. Almost all patients showed weakness, hypotonia, hypo- or areflexia. These findings are more or less similar to the studies by Ho et al[1] and Ropper et al[6].

Ropper et al[6] showed that 95% of patients with GBS had developed weakness in legs. The clinical signs (wasting, hypotonia, hyporeflexia, decreased power) found in this study revealed similarity with another study[7]. Nearly 55% complained of paraesthesia/pain before the development of weakness. This finding is consistent with that of Winer et al[8], who showed that 80% GBS patients had sensory symptoms. Position sense was lost in 15% cases and vibration sense was significantly lost in 25% cases. None had impairment of pain, touch, temperature sense.

Regarding autonomic involvement, there was involvement of pulse and trophic changes. Blood pressure involvement was also seen in patients. These findings are consistent with those of Bosch and Smith[9]. In this study, 13 (32.50%) patients had respiratory function involvement, which is consistent with the finding of Bosch and Smith[9].

References:

1. Ho TW, Mishee B, Lie Y, Gao CY, Bornblath DR, Griffin JW, et al. Guillain-Barré syndrome in northern China: relationship to *Campylobacter jejuni* infection and antiglycolipid antibodies. Brain 1995; 118: 597-605.
2. Guillain G, Barre JA, Strohl A. Sur un syndrome de radienlo-nevrite avec hyperalbuminose du liquide cephalora-chidien sans reaction cellulaire: remarques sur les caracters clinique at graphiques des reflexes turdinex. Bull Soc Med Hosp Paris 1916; 40:1462-70.
3. Haque MA, Haque S, Rahman MA, Hossain S, Gosh PK, Begum T, et al. Presentation of GBS - a prospective study in BIRDEM Hospital. Bangladesh J Neurosci 1998; 14: 35-40.
4. Sebevuratne U. Guillain-Barré syndrome. Postgrad Med J 2000; 76: 774-82.
5. Asbury AK, Cornblath DR. Assessment of current diagnostic criteria for Guillain-Barré syndrome. Ann Neurol 1990; 27(Suppl.): 21-4.
6. Ropper AH. The Guillain-Barré syndrome. N Engl J Med 1992; 326: 1130-6.
7. Gordon PH, Wilbourn AJ. Early electrodiagnostic findings in Guillain-Barré syndrome Arch Neurol 2001; 58: 913-7.
8. Winer JB, Hughes RAC, Osmond C. A prospective study of acute idiopathic neuropathy. I. Clinical featuers and their prognostic value. J Neurol Neurosurg Psychiat 1998; 51: 605-12.
9. Bosch EP, Smith BE. Disorders of peripheral nerves. In: Bradley WG, Daroff RB, Enichee GM, Marsden ED ((editors). Neurology in Clinical Practice, Third edition, Vol. II. Oxford: Buttrworth-Heinmann, 2000. pp-2045-130.