Electrophysiological Pattern of Neuropapathy in Guillain-Barre Syndrome

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Guillain Barre Syndrome is an acute immune mediated polyneuropathy. We conducted a prospective study in Neurology Department, Mayo Hospital, Lahore to evaluate different electrophysiological patterns in Guillain Barre Syndrome. A total of 25 cases were registered over the period of one year and nerve conduction and electromyography was done on each patient. The break down of different patterns of neuropathy was 36% demylinated, 12% axonal and 52% of mixed variety having both demylinating and axonal components. From our study we conclude that pattern of neuropathy in GBS is nearly same as reported in most European and local studies except Chinese endemic cases where axonal form is more frequent.

Key words: GBS, neuropathy, axonal, demylinating

Guillain–Barre syndrome (GBS) is an acute, immune mediated polyneuropathy characterized by progressive motor weakness of more than one limb, areflexia and absence of any other identifiable cause alongwith albuminocytological dissociation in cerebrospinal fluid[1].

Electrodiagnostic studies provide critical diagnostic and prognostic information and typically show "reduction in conduction velocity or conduction blocks in motor nerves "prolonged distal latencies and abnormal F responses in Guillain Barre Syndrome'

Although specific treatment is now available, general care is still of the utmost importance for the GBS patient. Plasma exchange (PE) and more recently intravenous immune globulins (IgIv) have been shown to be effective[2].

On electrophysiological basis, two major forms have been identified i.e. demylinating and axonal. Demylinating form occurs much more frequently than axonal form.

It has been found that axonal form of GBS is more common in Asian countries than reported in western studies[3,4].

Objectives

1- To study the variation in the prevalence of demylinating and axonal forms of Guillain-Barre syndrome in Pakistan, as compared with reported studies.

Material and methods

This prospective study was conducted in Neurology department Mayo hospital, Lahore. This study extended over full one year. All the patients admitted in Neurology department and those admitted in other medical departments of Mayo hospital or elsewhere, and were referred for opinion or nerve conduction studies were included in the study.

A detailed history from the patient or a relative was taken and complete neurological examination was done along with examination of other systems.

Nerve conduction and electromyography was done along with other investigation to confirm the diagnosis and rule out the differential diagnoses.

Inclusion criteria

1- Patients fulfilling the NINCDS criteria for Guillain-Barre syndrome were included in the study.
2- Patients of all age groups were included in the study.
3- Patients of both sexes were included in the study.

Exclusion criteria

Suspected cases of Diphtheria, Porphyria, Hepatits and viral xanthelasma were excluded.

All the data was noted on a Performa designed for the study.

Results

Electrophysiological Pattern

Nerve conduction studies and electromyography was performed on each of the 25 patients. Out of these 9(36%) found to have pure demylinating type of neuropathy and 3(12%) pure axonal neuropathy according to the criteria used (Albers JW, Kelly JJ. Acquired inflammatory polyneuropathies: Clinical and Electrodiagnostic features. Muscles Nerve 1989; 12:435). Majority of the patients 13(52%) had features of both demylinating and axonal neuropathy and were labeled as mixed neuropathy.

Table: Motor nerve conduction studies of all the patients (Conduction studies of only two nerves of each patients is given as required in criteria)

Sr #	Nerve studied	PML/AMP	DML/AMP	Velocity	F Wave	Pattern
1.	Rt. Median Rt. Com. Peroneal	1.8 ms/ 0.6 mv 11.3 ms/ 0.5 mv	3.8 ms/ 7.5 mv 6.0 ms/ 1.4 mv	60 m/s 58 m/s	30 ms Absent	Axonal
2.	Rt. Median Rt. Com. Peroneal	15.4 ms/ 2.0 mv 28.8 ms/ 150 uv	9.8 ms/ 1.7 mv 16 ms/ 40 uv	46 m/s 27 m/s	Absent Absent	Mixed
3.	Rt. Median Rt. Com. Peroneal	19.4 ms/ 780 uv 29.6 ms/ 210 uv	3.81 ms/ 63 uv 16 ms/ 400 uv	16 m/s 27 m/s	Absent Absent	Mixed
4.	Rt. Median Rt. Com. Peroneal	Absent Absent	Absent Absent	–	–	Axonal
5.	Rt. Median Rt. Com. Peroneal	30 ms/ 500 uv Absent	12 ms/ 350 uv_	16 m/s	146 ms scant.	Mixed
6.	Rt. Median Rt. Com. Peroneal	1.9 ms/ 240 mv 14.4 ms/ 290 uv	3.5 ms/ 220 mv 7.3 ms/ 1.7 mv	55 m/s 42 m/s	Absent Absent	Mixed
7.	Rt. Median Rt. Com. Peroneal	10.2 ms/ 2.4 mv 18.2 ms/ 430 uv	1.9 ms/ 2.5 mv 11.7 ms/100 mv	66 m/s 46 m/s	34 scant. Absent	Mixed
8.	Rt. Median Rt. Com. Peroneal	26 ms/400 uv 15 ms/ 360 uv	15.4 ms/ 600 uv 7.6 ms/ 600 uv	19 m/s 41 m/s	Absent Absent	Mixed
9.	Rt. Median Rt. Com. Peroneal	11.4 ms/ 1.5 mv 17.0 ms/ 100 uv	5.8 ms/ 1.1mv 7.6 ms/ 430 uv	49 m/s 31 m/s	Absent Absent	Mixed
10.	Rt. Median Rt. Com. Peroneal	9.4 ms/ 6.2mv 16 ms/ 0.1mv	3.4 ms/ 12 mv 7.3 ms/ 0.3 mv	48 m/s 40 m/s	36 ms 67 ms	Mixed
11.	Rt. Median Rt. Com. Peroneal	Absent Absent	–	–	–	Axonal
12.	Rt. Median Rt. Com. Peroneal	8.1 ms/ 9.1 mv 16.3 ms/ 0.9 mv	1.1 ms/ 11.0 mv 1.2 ms/ 4.0 mv	54 m/s 33 m/s	34 ms Absent	Demylinating
13.	Rt. Median Rt. Com. Peroneal	10.8 ms/ 1.5 mv 14.0 ms/ 2.2 mv	5.6 ms/ 3.2 mv 7.8 ms/2.8 mv	42 m/s 40 m/s	56 ms Absent	
14.	Rt. Median Rt. Com. Peroneal	19.4 ms/ 473 uv Absent	9.7 ms/ 0.83 mv	25 m/s	Absent Absent	Mixed
15.	Rt. Median Rt. Com. Peroneal	10.5 ms/ 2.3 mv 17.8ms/ 0.6 mv	5.3 ms/ 3.7 mv 13.5 ms/ 1.5 mv	50 m/s 66 m/s	Absent 86 ms	Mixed
16.	Rt. Median Rt. Com. Peroneal	9.5 ms/ 5.6 mv 13.0 ms/ 4.8 mv	4.0 ms/ 6.6 mv 5.7 ms/ 2.7 mv	49 m/s 47 m/s	70 ms scant. 67 ms	Demylinating
17.	Rt. Median Rt. Com. Peroneal	9.1 ms/ 20.5 mv 12.3 ms/ 9.3 mv	4.2 ms/ 2.1 mv 5.4 ms/ 9.3 mv	51 m/s 51 m/s	31-35 ms scant. Absent	Demylinating
18.	Rt. Median Rt. Com. Peroneal	11.3 ms/ 2.8 mv 12.7 ms/ 4.1 mv	1.5 ms/ 2.8 mv 5.6 ms/ 9.9 mv	57 m/s 46 m/s	37 ms scant. Absent	Demylinating
19.	Rt. Median Rt. Com. Peroneal	23.3 ms/ 1.1 mv Absent	16.3 ms/ 1.6 mv	36 m/s	Absent Absent	Demylinating
20.	Rt. Median Rt. Com. Peroneal	10.2 ms/ 1.5 mv 13.7 ms/ 1.3 mv	3.7 ms/ 3.8 mv 9.6 ms/ 1.55 mv	25 m/s 49 m/s	Absent Absent	Mixed
21.	Rt. Median Rt. Com. Peroneal	10.8 ms/ 1.9 mv 15.4 ms/ 3.7 mv	3.8 ms/ 11.7 mv 5.5 ms/ 4.5 mv	41 m/s 31 m/s	49 ms 68-77 ms	Demylinating
22.	Rt. Median Rt. Com. Peroneal	1.6 ms/ 2.5 mv 20.3 ms/ o.5 mv	3.5 ms/ 5.0 mv 10.0 ms/ 0.8 mv	77 m/s 34 m/s	34 scant. 67 scant.	Mixed
23.	Rt. Median Rt. Com. Peroneal	8.2 ms/ 12 mv 14.7 ms/ 610 uv	1.3 ms/ 12 mv 4.2 ms/ 10.0 mv	39 m/s 26 m/s	32 ms 66 ms	Demylinating
24.	Rt. Median Rt. Com. Peroneal	7.1 ms/ 15.0 mv 11.9 ms/ 5.0 mv	3.1 ms/ 15.0 mv 4.7 ms/ 7.6 mv	59 m/s 47 m/s	Absent Absent	Demylinating
25.	Rt. Median Rt. Com. Peroneal	10.0 ms/ 1.7 mv 13.6 ms/ 1.0 mv	5.8 ms/ 3.5 mv 8.2 ms/ 0.4 mv	62 m/s 53 m/s	Absent Absent	Mixed

PML Proximal motor latency DML Distal motor latency
Amp Amplitude M/s Meters per second
Ms Milli second Mv Milli volt
Uv Microvolt

Discussion

The management and functional outcome of patients suffering from Guillain Barre Syndrome depends upon the electrophysiological pattern of neuropathy in this disease. Therefore, it is important to know the type of neuropathy to predict reliably the prognosis and manage appropriately. In addition the association of a disease with antecedent or concurrent events may provide important clues to its etiology.

N.Z. Khan et al did electrophysiological studies in 40 cases of Guillain-Barre Syndrome and found that 33/40 (82.5%) were having a demylinating pattern, 5(12.5%) were having pure axonal pattern while remaining 3 (7.5%) studies showed normal pattern[5].

We did nerve conduction and electromyographic studies in each of our patients after one week of onset of symptoms and the pattern was as follows:-

The maximum number of patients 13/25(52%) were having features of both demylinating and axonal pathology according to the criteria already mentioned, followed by 9/25(36%) patients with demylinating pattern. Three out of 25(12%) showed pure axonal pattern. As is shown above, the nerve conduction findings in our study reveals quite high percentage of mixed type of neuropathy as compared with series of N.Z. Khan et al while axonal type is similar. Higher percentage of mixed type of neuropathy might be related to higher incidence of diarrhea (indirectly to C. jejuni infection) as opposed to other study where respiratory tract infection was leading preceding event.

J.S. Katz et al in a series of 167 patients reported 82(49%) patients having AIDP, 32(19%) with Fischer syndrome, 6(4%) axonal pattern and 47(28%) with inconclusive pattern[6]. Similar pattern has also been reported by Emilia-Romagana study group[7].

Exceptionally high percentage of axonal pattern has been reported in studies from China and Japan. G. Sobu et al in a study from Japan reported 30% incidence of axonal pathology in autopsied cases with macrophage invasion rather than lymphocytes while in Chinese endemic cases of GBS axonal pathology has been reported in upto 50% of patients which is higher than any other reported study[8]. Angelika F. Hahn in a review reported that AIDP is the most prevalent form of sporadic GBS in western countries and accounts for 80-90% cases. Acute motor axonal neuropathy (Axonal form of GBS) have been observed worldwide and represent 10-20% of cases in contemporary prospective studies[9].

It is obvious from above discussion that pattern of neuropathy in cases of GBS is quite similar to that reported from Pakistan and most of the studies from western countries. In contrast to those from northern china and some parts of Japan where endemic GBS cases show higher incidence of axonal pathology making upto 50% of cases.

Conclusion

From our study we conclude that electrophysiological study shows similar pattern of neuropathy as compared with local and western studies except Chinese and Japanese endemic GBS patients where higher axonal pathology is reported.

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