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Original Research Article

Electrophysiologic Diagnostic Patterns Analysis in Patients with Guillain Barre Syndrome in Tertiary Care Centre

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Abstract

Introduction: The Guillain-Barre syndrome (GBS) is an acute immune-mediated neuropathy and is recognized clinically by the presence of acute, rapidly progressive flaccid weakness, diminished or absent reflexes and albumin cytological dissociation in cerebrospinal fluid. Each subtype may have a different immunopathogenesis therefore, may have different approach treatments hence identification of these subtypes is important. The aims of the study were to identify the incidence of patients meeting electrophysiological criteria for demyelinating or axonal subtypes based on the well-established Rajabally's criteria.

Materials and Methods: All patients aged clinically diagnosed as GBS and meeting the Brighton diagnostic criteria for diagnosis of GBS were included in the study. Retrospective analysis of clinical and electrophysiological data of 50 Guillaine barre syndrome (GBS) patients of the Thanjavur Medical College, Thanjavur were done.

Results: Age of the patients ranged from 10-70 years with mean age of 37.8 years of age in this study. Flaccid weakness was present in almost all patients. On analysing electrophysiologic pattern of GBS patient's 58 percent showed pure demyelination and 12% of patients showed pure axonal pattern with 14 percent of patients showed either of the patterns.

Conclusion: Our study was showing predominantly demyelinating variant note in 72 percent of patients and F wave abnormalities were noted in 56% of patients. suggesting a slight preponderance of AIDP (acute inflammatory demyelinating polyneuropathy) variety in India. Early detection and characterization of GBS in is useful in timely intervention to reduce morbidity and disability and also useful to assess the prognosis.

Keywords: GBS, Demyelination, Axonal, AIDP, Guillane Barre Syndrome.

Introduction

The Guillain Barre syndrome (GBS) is an acute neurological illness characterized by immunologically mediated polyneuropathy and is clinically recognised by acute rapidly

progressive flaccid weakness, diminished or absent reflexes and in cerebrospinal fluid analysis there will be dissociation in albumin and cell count. Initially GBS was synonymous with Acute Inflammatory Demyelinating Polyneuropathy (AIDP), but in due course many types have been identified viz AMAN variant Acute Motor Axonal Neuropathy, AMSAN variant known as Acute Motor Sensory Axonal Neuropathy and Miller Fisher Syndrome [1,2]. Every subtype has different immunopathogenesis and has different approach to managements and hence identification of these subtypes is important [3,4]. Many Nerve conduction studies (NCS) older criteria are there with good sensitivity for diagnosing GBS [3] of AIDP variant [4] and significant proportion of GBS with axonal variants are being diagnosed according to newer criteria [5,6]. GBS variant diagnosis is highly useful for diagnosing the pathophysiology and also helps to predict the prognosis of GBS patients. NCS in the case of AIDP classically will have decreased conduction velocity [CV], prolonged temporal dispersion [TD] and also in at least 1-2 nerves we can notice conduction blocks [CB]. In AMAN variants we will find reduction in the amplitude of compound muscle action potentials (CMAP).

when nerve conduction is done in clinically diagnosed GBS it may be normal during the initial four days but after one week it will be abnormal. Hence is important to repeat the nerve conduction test if it's normal initially [7,8]. H-reflex abnormality followed by absent or prolonged latency of F-waves, noninvolvement of sural sensory nerve action potential, Distal Motor Latency (DML) of motor nerves may be prolonged, and in demyelinating variants, conducti velocities (CV) will be decreased, and all these findings may occur earlier and helps in early diagnosis and management [9-10]. The latest electrophysiological criteria for classifying GBS into demyelinating and axonal variant is defined by Rajabally *et al*. [11].

Rajabally criteria:

The demyelinating variant features of one of the below in two nerves like 1. conduction velocity will be less than seventy percent of lower limit of normal (LLN) 2. distal motor latency prolonged greater than 150 % of the upper limit of normal (ULN) 3. Presence of conduction block in two nerves other than tibial nerve with amplitude ratio of Proximal to distal CMAP less than 0.7, with an added parameter in one more nerve 4. F wave latency prolonged more than 120 percent of upper limit of normal. Note when distal CMAP amplitude is less than fifty percent, f wave latency should be prolonged by greater than 150% of normal, and when distal CMAP amplitude is greater or equal to twenty percent of LLN, the absence of F-wave in 2 nerves with an extra parameter in one more nerve.

For diagnosing AMAN variant: the above features may be present in only one nerve. One demyelinating feature can be present in one nerve when distal CMAP amplitude is less than ten percent of LLN, and at least the presence of one of the below: (1) Distal CMAP amplitude is less than eighty percent in minimal 2 nerves. (2) Absence of F wave in two nerves when the distal CMAP amplitude is greater than or equal to 20% of LLN, without any features of demyelination.

(3) Presence of conduction block in two nerves other than tibial nerve with amplitude ratio of Proximal to distal CMAP less than 0.7, (4) F wave latency prolonged more than 120 percent of the upper limit of normal. Note when distal CMAP amplitude is less than fifty percent, f-wave latency should be prolonged by greater than 150% of normal, and when distal CMAP amplitude is greater or equal to twenty percent of LLN, absence of F-wave in One nerve or the Presence of conduction block in one lower limb nerves

other than tibial nerve with amplitude ratio of Proximal to distal CMAP less than 0.7 with distal CMAP less than 80 percent of LLN in one other nerve.

We retrospectively analysed the NCS of fifty clinically diagnosed with classical features of GBS and compared it with other world statistics. The main aims of the study were to identify the incidence of patients meeting electrodiagnostic criteria fitting to demyelinating or axonal variants based on the Rajabally's criteria [11] and important electrophysiological parameters were analysed.

Materials and Methods

Inclusion criteria:

All patients clinically diagnosed as GBS and meeting the Brighton diagnostic criteria for diagnosis involving clinical, lab and electrophysiological parameters were included in the study [12].

Exclusion criteria:

Patients With already diagnosed Peripheral Neuropathies, Hypokalemia and other clinical differential diagnosis like Transverse Myelitis, Peripheral Neuropathy risk factors like alcoholic nutritional peripheral neuropathy, vasculitic neuropathy in connective tissue, and rheumatological diseases, chronic renal failure, and nutrition-related neuropathies were excluded from the study.

Patients with the clinical diagnosis of GBS admitted and managed as an inpatient in the Neurology department of the Thanjavur medical college, Thanjavur were taken for analysis. The period of study was from January 2021 and December 2022. Patients presented with clinical features suggestive of GBS based on the Brighton Criteria and their nerve conduction studies were analysed. Approval by the ethical committee was obtained for this retrospective study at the Thanjavur medical college. Nerve conduction reports were scrutinised and those patients with abnormal reports like pure demyelinating, a pure axonal, mixed combination of demyelinating and axonl or inexcitable were tabulated. Inexcitable NCS is considered a variant of severe axonal GBS [11]. NCS parameters observed are CMAP Conduction velocity (CV), duration of Distal motor latency (DML) normal or prolonged, Amplitude of CMAP, the latency of F wave (F) and its presence or absence, and presence of Sural sensory SNAP and its amplitude.

Results

Totally 50 GBS patients were studied. Among them, 26 were males and 24 were females. GBS patients with age from 10 to 70 years with mean age of 37.8 were analysed in this study. Flaccid weakness was present in almost all patients.

Table 1: Patient's parameters

S.No	Parameters	No of patients
1	Male	26
2	Females	24
3	Age range	10-70years
4	Areflexia	46/50
5	Weakness	50/50
6	Preceding infection	29/50
7	Sensory loss	4/50
8	Respiratory failure	18/50

72% of patients showed demyelination patterns, 26% of patients showed axonal patterns while 58 percent showed pure demyelination and 12% of patients showed a pure axonal pattern with 14 percent of patients showing either of the patterns. Sensory loss is not a predominant feature and it was present only in 4% of patients.

Table 2: Rajabally's criteria parameters

Parameters	Patients	Percentage %
Pure demyelination	29	58
Pure axonal	6	12
Axonal+demyelination	7	14
Inexcitable	8	16

Table 3

Pure Demyelination Variant	29	58%
Pure Axonal Variant	6	12%
Axonal+Demyelinating Variant	7	14%
Sural Sparing	21	42%
Prolonged F Wave	8	16%
Absent F Wave	20	40%

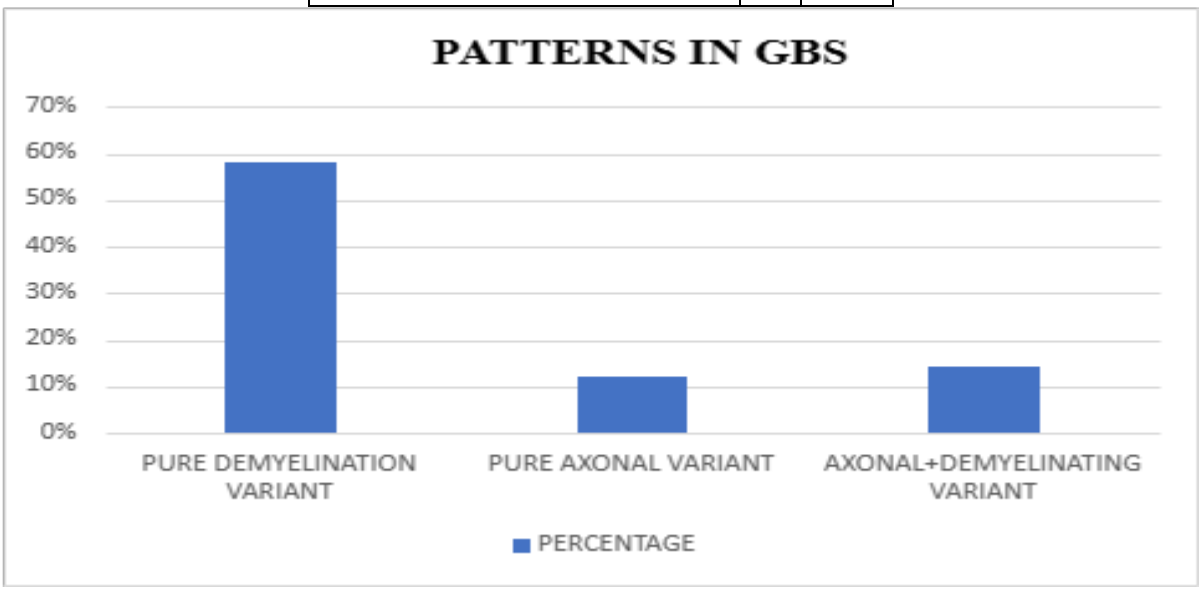

Figure 1: Patterns in GBS

Discussion

To diagnose, classify the subtypes of GBS and predict the prognosis electro-diagnostic studies are helpful [13,14]. Demyelination hallmarks include prolonged latency of distal CMAPs, increased latency of F wave, reduced conduction velocities, conduction block, and abnormal SNAPs in upper limbs in contrast to sural nerve SNAPs. In this study, CMAP CV has decreased in 36 (72%) patients, Prolonged DML was noted in 36 (72%) and in at least one nerve, and conduction block was noticed in 20 (40%) patients.

GBS patients admitted within a week of onset of symptoms conducted by Ropper *et al*. in forty-one patients, CMAP abnormalities like pound muscle action potentials including temporal dispersion, prolonged DML, reduction of CMAP amplitude, reduced conduction velocity and other findings were noticed in 16 patients [15]. Clouston *et al* also showed similar results [16]. Abnormally prolongation of distal motor latency & F waves abnormality denotes early involvement of motor spinal roots and distal motor nerves which helps in early diagnosis of GBS. Earlier and more severe abnormality of SNAP in upper limbs especially in the median nerve than those of the lower limbs (sural nerve). Sites with the high possibility of entrapment are prone to involve i.e. median nerve in Carpal Tunnel. Conduction block and secondary axonal degeneration are the reason behind reduced SNAP amplitudes [17]. The terminal segment of lower limbs were showing maximal conduction block than upper limbs. Brown *et al* study also showed similar results [18]. A possible explanation given for the conduction block by the study was a relative deficiency of the neurovascular barrier. Damage to sheaths of myelin is the reason for conduction velocity abnormality. Inflammation with early lymphocytic infiltrate followed by macrophages infiltrate later, paranodal and less commonly segmental demyelination of peripheral nerves with significant perivascular edema, and accumulation of mononuclear cells were hallmark features noted in peripheral nerve [19]. Our study is comparable with the Sharma *et al* study conducted at the university of health science Haryana, India [20]

Table 4

Ncs Parameters	**Our Study**	**Sharma *Et al***
CV reduced	72%	76.92%
DML increased	72%	76.92%
CB present	40%	15.38%
CMAP reduced	26%	67.6%
F wave absent	40%	76.92%
F wave latency prolonged	16%	23.07%
Sural SNAP spared	14%	61.29%
Demyelinating variant	72%	92.3%
Axonal type	26%	7.69%

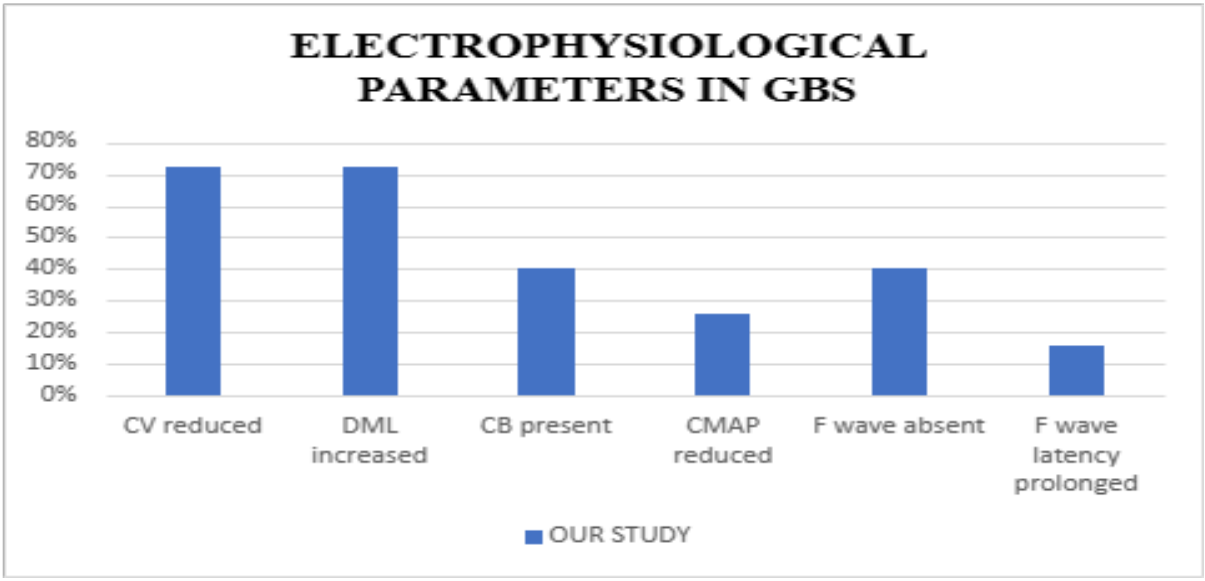

Figure 2

Conclusion

To conclude, among analysing electrophysiological patterns of GBS, 58% of patients showed a pure demyelination pattern, 12% of patients showed a pure axonal pattern and 14 % showed a mixed pattern. Our study was showed a predominantly demyelinating variant similar to Gupta *et al*. and Meena *et al*. [21,22]. F wave abnormalities were noted in 56% of patients. These studies were suggesting a mildly increased incidence of demyelinating AIDP variant in India. Early suspicion and nerve conduction studies of GBS and their classification are useful in timely intervention to reduce morbidity and disability and also useful to assess the prognosis.

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