Original Research Article

ELECTROMYOGRAPHIC FINDINGS IN GUILLAIN-BARRÉ SYNDROME PATIENTS

ABSTRACT

OBJECTIVE: To determine electromyographic findings in guillain-barré syndrome patients.

SETTING AND DURATION STUDY: This is a descriptive cross sectional study conducted in Neuromedicine departments of tertiary care Hospital JPMC, from1st February 2020 to 30th July 2021

MATERIALS AND METHODS: GBS was diagnosed according to the diagnostic criteria from the National Institute of Neurological Disorders and Stroke (NINDS) from 1990. All patients gave consent to and underwent electromyographic assessment with a Keypoint evoked muscle potential equipment at admission and 2, 3, and 6 months post disease onset. The records of the patients were anonymized and deidentified before analysis.

RESULTS: Age range in this study was from 13 to 70 years with mean age of 36.58±16.0 years . 63% patients were of male gender and 37% patients were females. Frequency of Electromyographic findings were acute demylinating polyneuropathy in 73% cases, acute sensorimotor axonal polyneuropathy 14% cases, acute motor xonal polyneuropathy 12% cases and acute sensory polyneuropathy 1% case.

CONCLUSION: The electromyography plays a role to diagnose GBS but along with this NCV and CSF analysis are also helpful in diagnosis and prognosis prediction.

KEY WORDS : Guillain-barré syndrome, Electromyographic, Autoimmune disorder

INTRODUCTION:

Guillian-Barre syndrome (GBS) is a rare, autoimmune disorder(1) characterized by acute immune-mediated polyneuropathy comprised of rapidly progressive flaccid paralysis which is symmetrical and ascending in nature and a reflexic (2). GBS has been associated with many infections. Respiratory and gastrointestinal illnesses are the two most commonly involved with this disorder. Before the onset of GBS presentation, up to 70% of patients have reported an antecedent illness during 1 to 6 weeks of disease(3). Although post-infectious GBSs have equivocal epidemiological evidence (4). Many bacterias and viruses are linked with GBS including Campylobacter jejuni, Mycoplasma pneumoniae, and Haemophilus influenzae, among viruses are Cytomegalovirus(CMV), influenza, enteroviruses, Ebstein barr virus, herpes simplex virus, hepatitis, human immunodeficiency virus and Zika virus(5,6). It has been noted that molecular mimicry plays a crucial role in the establishment of GBS , particularly the axonal variant. The gangliosides of peripheral nerves show similarities with lipopolysaccharides of campylobacter jejuni. Therefore an immune response generated against infectious agents cross react with nerve sheath (7). Although it is a rare disorder, has an annual incidence of 0.4 to 2 per 100,000 persons, Guillian-Barre Syndrome has a major impact on the health care system. An estimated cost of treatment for a patient with GBS is up to $318,966. The overall cost of medical care for patients with GBS is estimated up to $1.7 billion per year. GBS can affect all ages but incidences are more common in males than in females. It has been estimated that 100,000 patients would contract GBS worldwide annually(4,8). The most common symptom of GBS is ascending paralysis which starts first as symmetrical leg weakness(9,10). In addition, GBS patients develop weakness of extremities, body, and weakness of cranial nerves in just few hours or few days of onset of symtoms(11,12), and the effects on lower limbs are more prominent and dangerous than on upper limbs, which finally causes flaccid paresis and weak or even absent deep tendon reflex. Sometimes in the early stages of GBS absent tendon reflex, dysfunction, and even aphasia develop in pediatrics patients(13,14,15). Guillian-Barre Syndrome consists of the spectrum of immune-mediated polyneuropathy that can be divisible into several subtypes depending upon clinical features and electrophysiological findings, including acute inflammatory demyelinating polyneuropathy(AIDP), acute motor axonal neuropathy(AMAN)(16), and acute motor-sensory axonal neuropathy (AMSAN), and comprises the clinical variant of GBS Miller-Fisher Syndrome(MFS), and Bickerstaff's brainstem encephalitis(8,17). The predominat form of GBS in North America and Europe is acute inflammatory demyelinating axonal polyneuropathy(AIDP). The less prominent form the axonal form accounts for only 5% of patients including acute motor axonal neuropathy(AMAN). Patients with the axonal form of GBS show the worst of symptoms earlier than those with the demyelinating form. However, the recovery rate can be compared between the two(18). Motor sensory axonal neuropathy shows the worst prognosis and progresses to tetraplegia rapidly(19). In Asia, Central and South America axonal forms account for 30-47% of cases(2). At present, clinical

strategies used for the management of GBS are enhanced respiratory management, anti-infective therapy, nutritional care, rehabilitation care, and training, these can immensely improve body dysfunction caused by GBS but the results are quite far away from what is expected (20.21). with emerging medical rehabilitation technology, early rehabilitation is crucial to GBs treatment, and electromyographic biofeedback therapy is extensively used in preventive medicine(22,23).

Material and Methods

This is a descriptive cross sectional study conducted in Neuromedicine departments of tertiary care Hospital JPMC, from1st February 2020 to 30th July 2021. Informed consent was taken from patient's relative.GBS was diagnosed according tothe diagnostic criteria from the National Institute of Neurological Disorders and Stroke (NINDS) from 1990. The records of the patients were anonymized and deidentified before analysis. The study protocol was approved by the local institutional review board at the authors' affiliated institution and patient consent was not required because of the retrospective nature of the study.

Electromyographic assessment

All patients gave consent to and underwent electromyographic assessment with a Keypoint evoked muscle potential equipment at admission and 2, 3, and 6 months post disease onset. Concentric needle electrodes were used to record abnormally evoked resting potential at the abductor pollicis brevis, abductor digiti minimi, vastus medialis, and tibialis anterior muscle. Motor unit action potential was recorded during mild contraction, and cluster type of motor unit action potential was recorded during intense contraction. Motor nerve conduction study was done by stimulating the median nerve, ulnar nerve, the common peroneal nerve, and the motor branch of the tibial nerve to assess CMAPs including onset latency, amplitude, and conduction velocity. Surface electrodes were used to record the mean conduction velocity (MCV) and distal motor latency (DML) of the median nerve, ulnar nerve, common peroneal nerve, and the motor branch of the tibial nerve. Amplitude was measured in negative peak value. Sensory nerves examined included the median nerve, ulnar nerve, and sural nerve. Sensory conduction velocity was recorded, and sensory nerve action potential (SNAP) amplitude was measured in negative wave value. F wave was recorded of

the median nerve and ulnar nerve. Frequency was recorded. Patient's skin temperature was kept 32°C–35°C with an ambient temperature of 24°C–28°C.

RESULTS

Age range in this study was from 13 to 70 years with mean age of 36.58±16.0 years as shown in Table-I. 63% patients were of male gender and 37% patients were females as shown in Table-I. Frequency of Electromyographic findings were acute demylinating polyneuropathy in 73% cases, acute sensorimotor axonal polyneuropathy 14% cases, acute motor xonal polyneuropathy 12% cases and acute sensory polyneuropathy 1% case.

Acute demylinating polyneuropathy found commonly in all age groups while Acute sensorimotor axonal polyneuropathy and Acute motor xonal polyneuropathy were found mostly 3rd and 4th decay of life, While findings were more common in males (Table No.2 and 3).

Table No.1
Patient's characteristic
(n-100)

Variable	Patients	Percentage
Gender (Male to Female ratio 1.7:1)		
• Male	63	63%
• Female	37	37%
Age in years (Means Age 36.58±16.0 years)		
• 10-20 years	21	21%
• 21-30 years	23	23%
• 31-40 years	20	20%
• 41-50 years	15	15%
• 51-60 years	11	11%
• 61-70 years	10	10%
Electromyographic findings		
• Acute Demyelinating Polyneuropathy	73	73%
• Acute Sensorimotor Axonal Polyneuropathy	14	14%
• Acute Motor Xonal Polyneuropathy	12	12%
• Acute Sensory Polyneuropathy	1	1%

Table No.2
ELECTROMYOGRAPHIC FINDINGS ACCORDING TO GENDER (n-100)

ELECTROMYOGRAPHIC FINDINGS	GENDER		P value
	Male	Female	
	Patients(%)	Patients(%)	
• Acute Demylinating Polyneuropathy	47(47%)	26(26%)	0.097
• Acute Sensorimotor Axonal Polyneuropathy	6(6%)	8(8%)	
• Acute Motor Xonal Polyneuropathy	10(10%)	2(2%)	
• Acute Sensory Polyneuropathy	0	1(1%)	

Table No.3
ELECTROMYOGRAPHIC FINDINGS ACCORDING TO AGE (n-100)

ELECTROMYOGRAPHIC FINDINGS	Age in years					
	10-20	20-30	31-40	41-50	51-60	61-70
• Acute Demylinating Polyneuropathy	15	14	14	13	9	8
• Acute Sensorimotor Axonal Polyneuropathy	1	4	4	1	2	2
• Acute Motor Xonal Polyneuropathy	4	5	2	1	0	0
• Acute Sensory Polyneuropathy	1	0	0	0	0	0

DISCUSSION:

Guillian-Barre Syndrome has variable annual incidence worldwide ranging from 0.38 to 2.53 per 100,000, with most studies reporting 1.1 to 1.8 per 100,000 (16). The incidence is higher in adults than in children. Males are 1.5 times more frequently affected than females in all age groups (2,25,26).

In our study, we have evaluated 100 patients: 63% of males (63 patients) and 37% of females (37 patients). The age range in our study was from 13 years to 70 years with a mean age of 36.58±16.0 years. Men were more affected than women, but the mean age in our study was not very high. The incidence of GBS increases with age. This has also been reported in the international GBS outcome study (IGOS) that recruited 925 patients worldwide(24). Guillian-Barre Syndrome also increases with age in North America and Europe(2). In our study, only 10% of patients came under the age of 61-70 years of age and 23% of patients were in the age group of 21-30 years of age.). Parallel to our study, patients from Bangladesh were younger, where the median age was 21 years(24). This discrepant distribution between our study and the international study can be described by the variable demography of the general population, antecedent infections, and treatment.

In our study, the prevalence of GBS increased with age, for both males and females. GBS in our patients affected a broad range of ages. Corresponding age distribution has been found in a previous study (16,4). The frequency of males for GBS in our study was more than females. Male to female ratio in International GBS outcome study (IGOS) is 1.5:1(24). Such male to female ratio has also been reported in another study(2,34). Therefore, male gender and increasing age are non-modifiable risk factors for developing GBS worldwide. Polyradiculopathy patients have more mean age than any other form and acute motor axonal polyneuropathy has less mean age than any other form. In our study, Acute demyelinating polyneuropathy was found commonly in all age groups while Acute sensorimotor axonal polyneuropathy and Acute motor axonal polyneuropathy were found mostly 3rd and 4th decay of life. This shows a relative proportion to a study where the mean age of patients with acute motor axonal polyneuropathy (2nd decay of life) and acute sensorimotor axonal polyneuropathy is lower than acute demyelinating polyneuropathy(35). The predisposition of acute motor axonal polyneuropathy in young age group specifically younger than 40 was also

observed in another study(36,37). However, in the Northern China epidemic, acute motor axonal polyneuropathy was mostly reported in children(38).

Guillian barre syndrome is a group of heterogeneous syndrome having many different subtypes (27). In our study, Acute demyelinating polyneuropathy was predominantly reported electrophysiological subtype, accounting for 73% of the GBS cases. The other prevalent electrophysiological subtypes came under second and third position were Acute sensorimotor axonal polyneuropathy and acute motor axonal neuropathy, accounting for 14% and 12% of the cases, respectively. Acute sensory axonal polyneuropathy was the least one, accounting for only 1% of the cases. The parallel correspondent is observed in a cohort study in Oman in which 44 patients were included. They had relative proportion of electrophysiological subtypes as our study, accounting 52% for acute demyelinating polyneuropathy, 30% for acute motor axonal polyneuropathy, and 14% for acute sensorimotor axonal polyneuropathy (28). One more study from Kuwait, comparatively older study also showed an increased proportion of Acute demyelinating polyneuropathy of about 68%, and decreased proportion of other axonal electrophysiological subtypes(15%) (29). There are two latest retrospective studies from Northern and Southern China, which have reported different frequencies of electrophysiological subtypes of GBS. The study conducted in Northern China reported acute motor axonal polyneuropathy as a predominant subtype accounting for 55.8% and acute demyelinating polyneuropathy was relatively less frequent(21.2%) (30). In contrast to Southern China study, a higher proportion of Acute demyelinating polyneuropathy of about 49.0% was reported as compared to a lower proportion of Acute motor axonal polyneuropathy of about 18.8% (14). The corresponding frequencies of GBS subtypes of our study are relatively comparable to Southern China study (14). However, the proportion of acute motor axonal polyneuropathy reported in North America and Europe (3.0%) is still lower than the proportion reported in China and South Arabia (31). The geographical variance in the prevalence of acute motor axonal polyneuropathy and acute demyelinating polyneuropathy may be affected by certain environmental factors, variance in the frequencies, and types of antecedent infections, and genetic polymorphism of Compylobacter jejuni strains. The relative resemblance between our study and the study of Southern China (14) is a representation of the two different ethnic groups, proclaiming against a role of human genetic polymorphism to affect Guillain-Barre Syndrome subtype. Poor recovery trend has been noted in acute motor axonal polyneuropathy subtype as compared to acute demyelinating polyneuropathy in Europe, America, and Bangladesh(32). In a study in

Notherneast China, more severe symptoms had been observed in acute motor axonal polyneuropathy than in acute demyelinating polyneuropathy at admission, but the prognosis between the two was almost similar (33). This demonstrates that the severity of the disease is also varying among different regions just like electrophysiological subtypes. Although the impact of electrophysiological subtypes on prognosis is still under debate, as there is a slow and incomplete recovery in axonal GBS because of degeneration of axon, or faster because of conduction block transient recovery, and it may also depend upon criteria of subtypes(34,39).

The region-to-region variation in frequencies of clinical and electrphysiological subtypes of GBS can be partly explained by variation in local exposure to infections. The one suggested mechanism is that infection generates an immune response which then cross-reacts with peripheral nerve(molecular mimicry), which then damages the myelin sheath and axons. Campylobacter infection is the most predominant microorganism causing GBS(40).

CONCLUSION:

The study demonstrated frequency, sex distribution, and age distribution of Guillian Barre Syndrome similar to other studies. In our study, the most predominant type of GBS was acute demyelinating polyneuropathy. Acute motor axonal and acute sensorimotor axonal polyneuropathy were in the second and third distribution. Mean age was lower in our study with males predominant. Electromyography plays a role to diagnose GBS but along with this NCV and CSF analysis are also helpful in diagnosis and prognosis prediction.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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