Physiology



ELECTROPHYSIOLOGICAL STUDY OF GUILLAIN-BARRÉ SYNDROME (GBS) PATIENTS IN AMRAVATI REGION

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ABSTRACT Background: Guillain-Barré Syndrome (GBS) is acute, frequently severe, fulminant polyneuropathy. Electrophysiology has emerged as important tool for early diagnosis and identifying various subtypes of GBS.

Objective: To assess motor and sensory nerve conduction parameters in early course of disease and to classify the patients in various subtypes of GBS using electrodiagnostic (Edx) criteria.

Material and methods: We retrospectively evaluated Edx data of 23 clinically diagnosed cases of GBS presented within 2 weeks of onset of muscle weakness. Nerve conduction studies were done in six motor nerves (Median, ulnar & tibial bilaterally) and six sensory nerves (median, ulnar & sural bilaterally) in each case.

Results: Abnormal F wave was seen in 94.20% nerves tested. Other early nerve conduction findings included prolonged distal motor latencies (DML) in 77.54%, Compound Motor Action Potential (CMAP) reduction in 63.04% and Conduction Velocity (CV) reduction in 34.05% of nerves tested. Abnormal Sensory Nerve Action Potentials (SNAPs) were present in 45.65% of nerves tested. 20 cases (86.96%) were identified as Acute Inflammatory Demyelinating Polyneuropathy (AIDP) while 03 cases (13.04%) had axonal form of disease [Acute Motor Axonal Neuropathy (AMAN) and Acute Motor Sensory Axonal Neuropathy (AMSAN)]

Conclusion: The most affected motor nerve conduction (MNC) parameter was absent/delayed F wave. Increased DML & low CMAPs were other common findings. Reduced CV was less common which can be attributed to early course of the disease. The most common variant of GBS was AIDP in which early Edx marker included abnormal F- wave, increased DML and sensory conduction abnormality showing sural sparing pattern.

KEYWORDS : Guillain-Barre Syndrome, Electrophysiology, Motor nerve conduction, Sensory nerve conduction.

INTRODUCTION-

Guillain-Barré syndrome (GBS) is a fulminant polyradiculo neuropathy that is acute, frequently severe and autoimmune in nature. GBS is the most common cause of acute or subacute generalized paralysis [1]. It is characterised by predominantly motor paralysis with areflexia. History of antecedent infection (respiratory or gastrointestinal) or vaccination may be obtained in 50 – 70 % of cases [2]. The theories suggest an autoimmune mechanism in which the patient's defence system of antibodies and WBC are triggered into damaging the nerve coverings or insulation leading to weakness and abnormal sensation [3].

GBS has an incidence of 0.6-2.4 cases / 100,000 populations [4], but the prevalence & subtype may be variable in different geographic areas [5].

Clinical and laboratory findings, especially cerebrospinal fluid (CSF) analysis have an important role in the diagnosis of GBS. But electrophysiological confirmation of the diagnosis is even more important as the CSF protein level may frequently be normal within the first week [6]. During the acute phase, the disorder can be fatal requiring admission to intensive care unit for mechanical ventilation. Electrophysiological studies have a crucial role, in confirming as well as differentiating the subtypes of GBS. Based on electrophysiological findings, GBS has three major subtypes: acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMSAN).

Edx studies also help in ruling out of some differential diagnosis like myopathic and motor neuron disorders and confirming the neuropathic nature of GBS [7]. Also early diagnosis helps in prompt treatment and impacts its prognosis.

These patients' nerve conduction studies (NCS) show absent or prolonged F waves, prolonged distal motor latency (DML), conduction velocity (CV) slowing, conduction block with or without sensory nerve abnormalities. In axonal form of GBS, NCS shows reduced compound motor action potential (CMAP) without slowing of conduction velocity (CV) [3].

So this study is undertaken to evaluate electrophysiological features in GBS and to identify the subtypes using Edx criteria.

MATERIALAND METHODS:

We retrospectively evaluated Edx data of patients with diagnosis of GBS. Patients of age groups between 6 to 59 years & both gender, referred to our Electrophysiology Lab during 2016 to 2018 were included in the study.

Patients who underwent nerve conduction studies within 2 weeks of onset of motor weakness were selected for this study. Patients with neuromuscular weakness due to other causes (e.g. Mysthenia Gravis, Botulinism, Poliomylitis, toxic neuropathy, Myopathy etc.), other causes of chronic acquired neuropathy, diabetes mellitus and chronic alcohol abuse were excluded from the study.

Electrophysiological studies were performed on RMS EMG EP Mark-II machine with surface electrodes using standardized techniques and protocol. All tests were performed by the same investigator and under constant room temperature $(30^{\circ}C)$ to shortlist the errors.

Nerves tested were 6 motor (median, ulnar and posterior tibial nerves bilaterally) and 6 sensory (median, ulnar and sural bilaterally). Following parameters were studied-

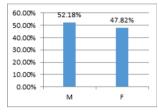
- Distal motor latencies (DML)
- Conduction velocities (CV)
- F wave latencies
- Compound motor action potentials (CMAP)
- Sensory nerve action potentials (SNAPs)

Each variable was compared with normal standards of our laboratory and mean SD of +/- 2.5 was considered abnormal.

RESULT

23 clinically diagnosed patients, in whom Edx study was performed within 2 weeks of onset of symptoms, were included in study. The age of study subjects ranged from 15 years to 65 years with mean age 36.5 years. In our study, there were 12 males &11 females with Male: Female ratio 1.09. (Figure 01)

Figure 01: Gender wise distribution of GBS patients



Total 276 nerves were studied, 138 motor (median, ulnar & posterior tibial bilateraly) & 138 sensory (median, ulnar & sural bilateraly) in 23 patients.

In motor nerve conduction studies, Ulnar nerve was not excitable in 8.7%, Median in 4.35% and Tibial in 6.52% of the nerves.

Motor distal latencies were prolonged in 77.54 % of the nerves tested, CMAP reduction in 63.04%, reduced motor conduction velocities in 34.05 %, F-wave abnormalities in 94.20 % (absent F-waves in 23.91% and prolonged in 70.29 %) of nerves tested.

In sensory nerve conduction studies, abnormal SNAPs were found in 45.65% of nerves tested.

Details of Motor and sensory conduction parameters are given in table 1.

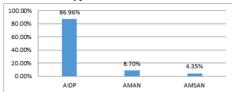
Table 1: Results of nerve conduction studies (NCS) in patients with Guillain-Barre syndrome

NCS Variable				
Motor Nerve Conduction Study				
DML (ms)	Normal n (%)	Prolonged n (%)		
Median	12 (26.09)	34(73.91)		
Ulnar	13 (28.26)	33 (71.74)		
Tibial	6 (13.04)	40(86.96)		
СМАР	Normal	Reduced	NE	
(mv)	n (%)	n (%)		
Median	23 (50.00)	21(45.65)	2 (4.35)	
Ulnar	25 (54.35)	17 (36.96)	4 (8.70)	
Tibial	03 (6.52)	40 (86.96)	3 (6.52)	
CV	Normal	Reduced		
(m/ Sec)	n (%)	n (%)		
Median	29(63.04)	17(36.96)		
Ulnar	33(71.74)	13 (28.26)		
Tibial	29(63.04)	17(36.96)		
F wave latency	Normal	Prolonged	Absent	
(ms)	n (%)	n (%)	n (%)	
Median	03 (6.52)	34(73.91)	09(19.57)	
Ulnar	03 (6.52)	35(76.09)	08 (17.39)	
Tibial	02 (4.35)	40(86.96)	04 (08.70)	
Sensory Nerve Conduction Study				
SNAP	Normal	Reduced	Absent	
(micro V)	n (%)	n (%)	n (%)	
Median	18 (39.13)	24 (52.17)	4 (8.70)	
Ulnar	27 (58.70)	17 (36.96)	2 (4.35)	
Tibial	30 (65.22)	14 (30.43)	2 (4.35)	

CV: conduction velocity; DML: Distal Motor Latency; CMAP: Compound muscle action potential; SNAP: Sensory Nerve Action Potential; NE: Non-Excitable

According to criteria proposed by Ho et al., axonal forms of GBS were distinguished from Acute inflammatory Demylinating Polyneuropathy (AIDP) types [7].Out of the 23 patients, 3 (13.04%) had axonal forms of the disease (2 AMAN and 01 AMSAN) and 20 (86.96%) patients had AIDP. (Figure 2)

Figure 2: Various subtypes of GBS



Sensory nerve conduction in AIDP:

Amongst AIDP patients, median nerve SNAPs were abnormal in 14 (70%) patients: sensory responses were absent in 2 (10%) patients and of reduced amplitude in 12 (60%) patients.

Ulnar nerve SNAPs was abnormal in 9 (45 %) patients: sensory responses were absent in 1 (5 %) patient and of reduced amplitude in 8 (40 %) patients.

Sural nerve SNAPs was reduced in 7 (35 %) patients: The patients with abnormal sural conductions had abnormal median and ulnar sensory conductions in all. Normal or relatively preserved sural SNAP compared with at least two abnormal SNAPs in the upper limb, termed as Sural Sparing [8], was seen in 7 (35 %) patient. while 6 (30 %) patients had normal sensory nerve conduction studies in all nerves tested.

DISCUSSION:

GBS is the most common cause of acute or sub-acute generalized paralysis in practice. GBS occurs in all parts of the world and in all seasons, affecting children and adults of all ages and both sexes [2].

Edx studies are helpful in diagnosis of GBS as well as its classification in various subtypes .In GBS, various electrophysiological parameters are affected like CV, CMAP, DML, F-wave latencies, Conduction block and sensory conduction parameters .Many authors reported that electrophysiological finding vary in early & late course of disease [4,6,9]. But early diagnosis and identifying various subtypes is important as it impacts the prognosis & treatment of GBS.

In present study, where we have evaluated Edx parameters in early course of disease, the most common motor nerve conduction finding seen in all nerves tested was F wave abnormality (94.20%), followed by prolonged DML (77.53%), reduced CMAP (63.04%) and slowing of CV (34.05%). Our findings are in concordance with other authors, who predicted that F wave latency is the most sensitive diagnostic test for early GBS [8, 10, 11].

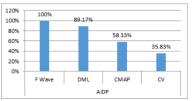
While Alexander M in his study reported CMAP abnormalities in 61.01%, reduced motor CV in 33.9%, F-wave abnormalities in 30.14% of nerves tested, indicating reduced CMAP as most common abnormality [9]. On the other hand, Ropper et al and Shende V et al found DLAT as most common abnormality in AIDP in early GBS [12,13]

The current study also reports the AIDP form to be the most predominant form of GBS in our region. Many Indian studies state similar preponderance of AIDP variety [5,10,14].

While axonal variant was reported as most common subtype (AMAN: 30.4 % and AMSAN: 13.6 %) in a study from south India, where this finding was attributed to more number of children in study group [9].

In AIDP, which is prevalent subtype in our study, F wave abnormality was seen in 100% of patients indicating early predilection for the proximal nerve segments and spinal roots in AIDP [15,16]. Prolonged DML, reduced CMAP & CV were other common abnormalities seen in early GBS (**Figure 3**). Sensory abnormality was seen, but with sural sparing pattern. Similar pattern of sensory conduction was also reported by other authors where they concluded that Sural sparing is also a marker of demyelinating neuropathy [6,17,18.19]

Figure 3: Motor nerve conduction study in AIDP cases



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CONCLUSION:

Edx study is the most useful investigation for confirming diagnosis of clinically suspected cases of GBS and their subtyping according to electrophysiological criteria. In current study AIDP is most common variant of GBS in study population. Abnormalites in F- wave, increased DML and sural sparing pattern in sensory nerves are early electrodiagnostic markers of AIDP.

REFERENCES:

- Ropper AH, Brown RH, Adams and Victor's Principles of Neurology, 8th ed. New York: 1 McGraw-Hill; 2005.
- John J & Kannan A. Clinical profile of GuillainBarresyndrome in a tertiary care center. 2.
- John J & Kalman A. Chinkar Johne of Guntambaresyndrome in a certary care center. Int J Res Med Sci. 2014 May; 2(2):445-447. Dhadke SV, Dhadke VN, Bangar SS, Korade MB.Clinical Profile of GuillainBarre Syndrome. J Assoc Physicians India. 2013 Mar;61(3):168-72. 3 4
- Sudulagunta SR, Sodalagunta MB, Sepehrar M, Khorram H, Bangalore Raja SK, Kothandapani S. Guillain-Barré syndrome: clinical profile and management. Ger Med
- Kalita J, Misra UK, Das M. Neurophysiological criteria in the diagnosis of different clinical types of Guillain-Barre syndrome. J Neurol Neurosurg Psychiatry 2008; 79(3): 5. 289-93
- Baraba R, Sruk A, Sragalj L, Butković-Soldo S, Bielen I. Electrophysiological findings 6. in early Guillain-Barré syndrome. Acta Clin Croat. 2011 Jun; 50(2):201-7. Ho TW, Mishu B, Li CY, Gao CY, Cornblath DR, Griffin JW, et al. Guillain-Barre
- 7 syndrome in orthern China. Relationship to Campylobacter jejuni infection and anti-glycolipid antibodies. Brain 1995;118:597-605. Yadegari S, Nafissi S, Kazemi N. Comparison of electrophysiological findings in axonal
- 8.
- and demyclinating Guillain-Barre syndrome. Iran J Neurol. 2014;13(3):138-43. Alexander M, Prabhakar A T, Aaron S, Thomas M, Mathew V, Patil A K. Utility of neurophysiological criteria in Guillain-Barre' syndrome: Subtype spectrum from a tertiary referral hospital in India. Neurol India. 2011 Sep-Oct;59(5):722-6. 9.
- 10
- 12.
- tertiary referral hospital in India. Neurol India. 2011 Sep-Oct;59(5):722-6.
 Sharma G, Sood S, Sharma S. Early Electrodiagnostic Findings of GuillainBarre Syndrome. J Neurol Neurophysiol 2013, 4: 142.
 Winer JB. GuillainBarré syndrome. J Clin Pathol:Mol Pathol 2001;54:381–385.
 Ropper AH, Wijdicks EF, Shahani BT. Electrodiagnostic abnormalities in 113 consecutive patients with Guillain-Barré syndrome. Arch Neurol. 1990; 47: 881-887.
 Shende V, Pawar S, Jiwane T, Chaudhari A R, Shende A. Study of motor nerve conduction parameters in guillain-barre syndrome patients of Centernpary Medical Research 2016; 3(3):859-861.
 Gupta D, Nair M, Baheti NN, Sarma PS, Kuruvilla A, et al. (2008) DiplomateAmerican Board (2008) Electrodiagnostic and clinical aspects of Guillain-Barré syndrome: an analysis of 142 cases. L (Din Neuromuscul Dis 10: 42-51. 13
- 14 analysis of 142 cases. J Clin Neuromuscul Dis 10: 42-51. Amato AA. Guillian Barre syndrome and related disorders. Rev Mex Neuroci 2005;
- 15. 6(5): 455-469 Rabinstein AA, Guillain-Barré Syndrome. The Open General and Internal Medicine 16.
- Journal, 2007, 1, 13-22
- Kuwabara S, Ogawara K, Misawa S, Mizobuchi K, Sung JY, Kitano Y, Mori M, Hattori T. Sensory nerve conduction in demyelinating and axonal Guillain-Barré syndromes. 17 Eur. Neurol.2004; 51:196-198.
- Hadden RD, Cornblath DR, Hughes RA, et al. Electrophysiological classification of 18 Guillain-Barré syndrome: clinical associations and outcome. Ann Neurol 1998; 44: 780-788
- Taly AB, Veerendrakumar M, Das KB, Gupta SK, Suresh TG, Rao S, Nagaraja D, 19. Swamy HS.Sensory dysfunction in GB syndrome: a clinical and electrophysiological study of 100 patients. Electromyogr Clin Neurophysiol. 1997 Jan-Feb; 37(1): 49-54.

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