

Early Guillain Barre Syndrome Electrodiagnostic Findings.

Geetanjali Sharma

Corresponding author

Geetanjali Sharma,
Department of Physiology, University of Health Sciences,
Haryana, India.

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Abstract

Methods : A de-myelinating polyradiculoneuropathy with an acute immune mediator, Guillain Barre Syndrome (GBS). This study was done to analyse the electro-physiological abnormalities in the first week of GBS in this area because early diagnosis favours a positive outcome after treatment.

Basic procedures : The study was conducted to identify 65 clinically diagnosed patients with GBS who reported muscle weakness within a week using electro-physiological tests between 2010 and 2012.

Discussion : Electro-physiological studies play an important role in the early detection; characterization & treatment of GBS because timely intervention reduces morbidity and disability. Increased DML, absent F- wave, decreased median with normal Sural SCV (sensory conduction velocity) is diagnostic of early GBS.

Keywords

Peripheral neuropathic weakness; Electro-diagnosis; Early signs.

Introduction

De-myelinating polyradiculoneuropathy with auto-immune involvement is GBS. Both men and women are equally vulnerable. Areflexia with or without sensory, autonomic, and brainstem abnormalities are among the clinical signs, as are gradual, symmetrical ascending muscle weakening of

more than two limbs (Table 1). When neurological symptoms first appear, there is no fever and leg muscles are more weak than arms. Affected cranial nerves may have an impact on swallowing, eye movements, face muscles, and the airway [1]. Typically, tingling and numbness in the feet are the first symptoms [2].

The histo-pathological characteristics of 50 fatal cases of GBS were reported by Haymaker and Kernohan in 1949. Within the first week of the illness, proximal nerve edoema and myelin sheath degradation were the first symptoms [3].

An essential component of electrodiagnosis is De-myelinating polyradiculopathies that are inflammatory are early identified and described [4].

Even while the patients' clinical status is improving in the early weeks of the disease, nerve conduction abnormalities become more noticeable [5,6]. Early signs of nerve conduction include irregular or nonexistent F waves with low CNAPs, an irregular upper extremity sensory nerve action potential paired with a normal sural response, and many indirect discharges [4,7,8].

Material and Methods

Ethics committee approval: Because the patients were submitted by our Institute's Department of Medicine for electro-diagnostic evaluation, there was no ethical committee approval issue during this study (Table 2).

The goal of the current study was to speed up the confirmation of clinically diagnosed GBS cases that were transferred to the Department of Physiology for nerve conduction during the first week of sickness. These 65 subjects (42 men and 23 women) were studied with the RMS EMG EP Mark-II Chandigarh on a range of ages from 6 to 70. Rapidly progressing limb weakness with or without distal limb paresthesias and diminished deep tendon reflexes were necessary for the clinical diagnosis of GBS.

Four main groupings of parameters were taken into consideration:

1. Research on motor conduction The median, ulnar, tibial, and peroneal conduction velocities, amplitudes, and their distal motor latencies were measured in motor conduction investigations.
2. Medial and sural nerve conduction velocities were measured as part of sensory conduction experiments.
3. F wave studies. F wave studies comprised F wave delay and F wave conduction velocity. The antidromic activation of motor neurons, which involves conduction to and from the

spinal cord, causes the late response known as the F wave. F wave studies are a useful tool in clinical neurophysiology, as has been proven [9].

In GBS, where demyelination may impact the proximal portion of the nerve and even the roots, which cannot be examined by normal nerve conduction investigations, prominent slowing of F waves has been described [10].

Discussion

In order to diagnose and distinguish the demyelinating type of GBS, which responds to treatment and has a better prognosis, electro-diagnostic investigations are useful [13]. Prolonged distal motor latencies, prolonged/absent F wave latencies, primarily in the lower limbs, slow motor conduction velocities/conduction block with absent F wave, and abnormal upper extremity sensory nerve action potentials as compared to the sural nerve are electrophysiological hallmarks of early demyelination. The most accurate diagnostic test for early GBS is the F wave. In our investigation, proximal conduction block and decreased motor conduction velocity were mostly observed in the lower limbs. The aforementioned findings concur with those made by Gordon, Jun Kimura, and Kuwahara [4,10,14]. In a research conducted by Ropper et al. on 41 GBS patients, 16 individuals reported abnormalities of compound muscle action potentials, such as dispersion, delayed latency, low amplitude, conduction velocity slowing, conduction block, or aberrant F-waves [15]. The same outcomes were cited by Clouston et al. [16].

Prolonged F waves and prolonged distal motor latencies indicate early preference for proximal spinal root and distal motor terminal involvement. upper body SNAPs, especially those of the median nerve, can be impacted earlier and more severely than those of the sural nerve. This observation has a multifactorial interpretation. Sites more prone to entrapment include the median nerve in Carpal Tunnel. Secondary axonal degeneration and conduction obstruction can cause decreased SNAP amplitudes [17]. Both the upper and lower limbs' terminal segments had the largest conduction block, with the lower limb having the larger block. These results supported Brown's [18] findings. He linked it to a related blood-nerve barrier deficit.

The myelin sheath is damaged when conduction velocity decreases; cellular and immunological processes both play significant roles in this. Lymphocytic infiltration characterises the earliest inflammatory lesions; later macrophages start to proliferate. The perivascular oedema, mononuclear cell accumulation, paranodal & less frequently segmental demyelination, and paranodal & segmental demyelination are the peripheral nerve alterations [19].

Because the electro-diagnosis was made soon after the onset of symptoms in 30 of the 65 individuals, EMG tests were performed on them. Demyelinating type of neuropathy was the most common form of GBS (83.33%) in our series, according to EMG investigations. This agreed with the findings of Yakoob et al. [20].

Conclusion

Guillain Barre Syndrome is thought to affect between 1.1 and 1.8 million people worldwide each year [21]. As a result, this illness accounts for a sizable portion of demyelinating polyneuropathy cases globally. According to Gupta et al. and Meena et al. [22,23], there may be a little overrepresentation of the AIDP (acute inflammatory demyelinating polyneuropathy) variant in India. In our investigation, specifically in the state of Haryana, AIDP was the most prevalent type. The outcomes matched the electro-diagnostic criteria for a preliminary diagnosis of GBS that have been described in the literature. Due to the fact that prompt management lowers morbidity and disability, electro-diagnostic techniques are crucial in the early detection and characterisation of inflammatory demyelinating polyradiculopathy in the first week of symptomology.

References

1. Hauser SL, Asbury AK (2009) Guillain-Barre Syndrome & other immunemediated neuropathies: 2667-2671.
2. Amato AA (2005) Guillain Barre syndrome & related disorders. *Rev Mex Neuroci* 6: 455- 469.
3. Haymaker W, Kernohan JW (1949) The Landry-Guillain-Barré syndrome;a clinicopathologic report of 50 fatal cases and a critique of the literature.*Medicine (Baltimore)* 28: 59-141.
4. Gordon PH, Wilbourn AJ (2001) Early electrodiagnostic findings in GuillainBarré syndrome. *Arch Neurol* 58: 913-917.
5. Albers JW (1989) AAEM Case report #4- Guillain Barre Syndrome 12: 705-711.
6. McLeod JG (1995) Investigation of peripheral neuropathy. *J Neurol Neurosurg Psychiatry* 58: 274-283.
7. Roth G, Magistris MR (1999) Indirect discharges as an early nerve conduction abnormality in the Guillain-Barré

- syndrome. *Eur Neurol* 42: 83-89.
8. Aminoff MJ, Greenberg DA, Simon RP (2005) In *Clinical Neurology*. 6th ed. McGraw-Hill Medical, New York.
 9. Fisher MA (2002) H reflexes and F wave fundamentals normal and abnormal patterns. *Neurol Clin N Am* 20: 339.
 10. Kimura J, Butzer JF (1975) F-wave conduction velocity in Guillain-Barré syndrome. Assessment of nerve segment between axilla and spinal cord. *Arch Neurol* 32: 524-529.
 11. Meulstee J, Van Der Meche FG (1995) Electrodiagnostic criteria for polyneuropathy and demyelination: application in 135 patients with Guillain-Barré syndrome. Dutch Guillain-Barré Study Group. *J Neurol Neuro Surg Psychiatry* 59: 482-486.
 12. Mishra VA, Kalita J (2006) *Clinical Neurophysiology*, (2nd edn). Elsevier Health Sciences, Gurgaon: 235.
 13. Nadir ZK, Narullah M (1998) Electrodiagnostic study of 40 cases presenting as Guillain Barre Syndrome. *Pak J Neurol* 4: 50-54.
 14. Kuwabara S, Ogawara K, Mizobuchi K, Koga M, Mori M, et al. (2000) Isolated absence of F waves and proximal axonal dysfunction in Guillain-Barré syndrome with antiganglioside antibodies. *J Neurol Neurosurg Psychiatry* 68:191-195.
 15. Ropper AH, Wijdicks EF, Shahani BT (1990) Electrodiagnostic abnormalities in 113 consecutive patients with Guillain-Barré syndrome. *Arch Neurol* 47: 881-887.
 16. Clouston PD, Kiers L, Zuniga G, Cros D (1994) Quantitative analysis of the compound muscle action potential in early acute inflammatory demyelinating polyneuropathy. *Electroencephalogr Clin Neurophysiol* 93: 245-254.
 17. Amato AA, Dumitru D (2002) Acquired neuropathies. In: Dumitru D, Amato AA, Zwarts MJ. Editor. *Electrodiagnostic medicine*, (2nd edn). Philadelphia: Hanley & Belfus, Inc: 937-1041.
 18. Brown WF, Snow R (1991) Patterns and severity of conduction abnormalities in Guillain-Barré syndrome. *J Neurol Neurosurg Psychiatry* 54: 768-774.
 19. Ramachandran TS, Lorenzo NY (2011) Acute Inflammatory Demyelinating Polyradiculoneuropathy.
 20. Yakoob MY, Rahman A, Jamil B, Syed NA (2005) Characteristics of patients with Guillain Barre Syndrome at a tertiary care centre in Pakistan, 1995-2003. *J Pak Med Assoc* 55: 493-496.
 21. McGrogan A, Madle GC, Seaman HE, de Vries CS (2009) The epidemiology of Guillain-Barré syndrome worldwide. A systematic literature review. *Neuroepidemiology* 32: 150-163.
 22. Gupta D, Nair M, Baheti NN, Sarma PS, Kuruvilla A, et al. (2008) Diplomat American Board (2008) Electrodiagnostic and clinical aspects of Guillain-Barré syndrome: an analysis of 142 cases. *J Clin Neuromuscul Dis* 10: 42-51.
 23. Meena AK, Khadilkar SV, Murthy JM (2011) Treatment guidelines for Guillain-Barré Syndrome. *Ann Indian Acad Neurol*.