IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 14, Issue 5 Ver. VI (May. 2015), PP 81-84
www.iosrjournals.org

A case study on Guillain-Barre Syndrome and Peripheral Motor Neuropathy

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Abstract *Guillain-Barre Syndrome (GBS) is a post infectious polyneuropathy involving mainly motor but sometimes may also involve sensory and autonomic nerves. Weakness begins usually in the lower extremities and progressively involves the trunk, upper limbs; complete electrodiagnostic evaluation of patients with suspected Guillain-Barre syndrome requires both motor and sensory conduction studies, F response latency measurements. A 10-year-old boy sudden started felling pins-and-needles sensation, weakness, and numbness in his both upper and lower limb. On neurological examination, his cranial nerve exam was intact. His sensation was intact on both upper and lower extremities. Routine patient's laboratory studies of blood and urine gave normal results and did not reveal any infectious process. CSF elevated protein count of 220 mg/dL, with normal cell count, and an increased IgG fraction (25%). which may suspect the diagnosis of GBS. Nerve Conduction Studies showed motor conduction studies (MNCV) of the both the upper and lower extremities, revealed borderline- prolonged distal latencies, a reduced median and ulnar CMAP amplitude in upper limb ((right and left) along with reduced Common peroneal, posterior tibial in lower limb (right and left) and normal median, ulnar and sural SNAP amplitude recordings in both upper and lower limb. F response latencies were markedly prolonged and were difficult to obtain. The prognosis of GBS is dependent upon early diagnosis and intervention. The care of a GBS patient is challenging for the health care team.*
Key words*: Guillain-Barre Syndrome, Nerve conduction, F-wave, MNCV, SNCV*

I. Introduction

Guillain-Barre Syndrome (GBS) or acute idiopathic polyradiculoneuritis is a disorder of anonymous etiology, involving the peripheral nervous system [1]. It is an immune-mediated peripheral neuropathy characterized by rapidly progressive muscle weakness. The immune response depends on bacterial factors (specificity of lipo-oligosaccharide) and on host factors (immune status). The presence of antibodies leads to activation of T cells and complements, leading to a cascade of inflammation and demyelination. The demyelination decreases the velocity of nerve conduction and slows the impulse transmission along the axons. Clinical features include progressive, symmetrical ascending muscle weakness of limbs, areflexia with or without sensory, autonomic and brainstem abnormalities [2]. The extent of nerve damage varies, as per more damage seen in the intensely myelinated peripheral nerves, may cause motor and sensory weakness. Nerve conduction studies are helpful in the diagnosis of several peripheral neuropathies, also become more important in diagnosis of demyelinating polyneuropathies such as Guillain-Barre syndrome, chronic inflammatory demyelinating polyneuropathy [3].

II. Case report

A 10-year-old boy sudden started felling pins-and-needles sensation, weakness, and numbness in his both upper and lower limb. On subsequent days the weakness was more progressed in upper and lower extremities. His subsequently weakness of his upper and lower extremities and incoordination, was made him unable to do any work, and was hospitalized by his family member. On clinical examination we found that, there was diffuse weakness in all the four extremities (figure 1), distal was greater (grade 3**)** than proximal (grade 4). Muscle tone was decreased. Muscle stretch reflexes were absent. No pathologic reflexes were present. On neurological examination, the patient was alert and oriented with intact speech and memory, pupils equally reactive to light. His cranial nerve exam was intact. His sensation was intact on both upper and lower extremities.

Laboratory Assessment: Routine patient's laboratory studies of blood and urine gave normal results. The patient's laboratory studies did not reveal any infectious process. Rheumatoid factor was negative. Acute

viral titers demonstrated a cytomegalo virus titer of 1: 8, an Epstein-Barr titer of 1:32, and a herpes virus titer of 1:64. Lumbar puncture revealed clear, colourless cerebrospinal fluid (CSF) with CSF glucose was normal(60mg/dl), CSF cultures were sterile, elevated protein count of 220 mg/dL, with normal cell count, and an increased IgG fraction (25%), which may suspect the diagnosis of GBS.

Electrodiagnostic investigation: Nerve conduction study (NCS) was carried out in a quiet room of neurophysiology laboratory at a temperature of 26^0 to 30^0C by using Neuroperfect-2000. Skin temperatures were recorded and maintained above 32°C for all recordings. Nerve conduction studies were performed using standard techniques of supramaxima1 percutaneous stimulation and surface recording. The nerves (median and radian for motor and sensory) in upper limb and the nerves (Common peroneal, posterior tibial for motor and sural for sensory) in lower limb were stimulated sub-cutaneous along their course where they are relatively superficial. The skin resistance was reduced by rubbing with spirit swab; the active electrode was placed over muscle belly and reference electrode over tendon. Amplitudes of compound muscle action potentials (CMAPs) were measured from baseline to negative peak and were reported for stimulation at distal and proximal sites; conduction velocity was measured in the upper and lower limb. Evidence of abnormal temporal dispersion was estimated by comparing proximal and distal CMAP amplitudes, F response latencies were measured as the minimal latency in a series of F responses following distal (wrist or ankle) motor nerve stimulation. Sensory nerve action potential (SNAP) amplitudes were measured peak to peak. Nerve Conduction Studies showed motor conduction studies (MNCV) and sensory conduction studies (SNCV) of the both the upper and lower extremities, revealed borderline- prolonged distal latencies, a reduced median and ulnar CMAP amplitude in upper limb ((right and left) along with reduced common peroneal, posterior tibial in lower limb (right and left) and normal median, ulnar and sural SNAP amplitude recordings in both upper and lower limb (table1, 2; figure 2). Evoked CMAPs were of reduced amplitude and temporally dispersed, with prolonged distal latencies and reduced conduction velocities in nerves tested. F wave studies included F wave conduction velocity and F wave latency. F wave is a late response resulting from antidromic activation of motor neurons involving conduction to and from spinal cord. F wave studies have been established as a valuable tool in clinical neuro-physiology [4]. F response latencies were markedly prolonged in patient (table 3).

III. Discussion

GBS is an auto-immune mediated de-myelinating polyradiculo-neuropathy. It is a nonfamilial inflammatory demyelinating disease of peripheral nerve that may be associated with extensive secondary axonal and even anterior horn cell degeneration [5]. Antecedent events are common and include infections (viral, mycoplasmal, and chlamydial), immunization, malignant disease, and surgery. The etiology of Guillain-Barri Syndrome has not been established, but immunologic mechanisms almost certainly are involved [6]. Relative deficiency of the blood nerve barrier and decrease in conduction velocity may damage to the myelin sheath; both cellular and immune mechanisms play important roles in it [7]. Weakness was more prominent in leg muscles as compared to arms; there was absence of fever at the onset of neural symptoms [2].

Prominent slowing of F waves was present which may due to demyelination, which may affect the proximal segment of nerve and even the roots which cannot be assessed by routine nerve conduction studies [8]. In my patient, abnormalities of compound muscle action potentials including, prolonged motor latencies, low amplitude, slowing of conduction velocity, conduction block or prolonged or absent F waves reflect early predilection for involvement of proximal spinal roots and distal motor terminals [9-11]. The conduction block was maximal in the terminal segment of the upper and lower limbs [7]

IV. Conclusion

The prognosis of GBS is dependent upon early diagnosis and intervention. The results were in line with the electro-diagnostic criteria for early diagnosis of GBS. Electro-diagnostic techniques play an important role in the early detection and characterization of inflammatory demyelinating poly-radiculopathy in the first week of symptomology and assume importance in treatment of this syndrome because timely intervention reduces morbidity and disability.

The care of a GBS patient is challenging for the health care team and the caregivers because some symptoms can be devastating. GBS is a life event with a potentially long-lasting influence on physical and psychosocial well-being, transforming a healthy, independent person into a critically ill and physically helpless person. "The acute progression of motor weakness and fatigue makes a profound effect on the patient's healthy life.

Acknowledgement

The authors gratefully acknowledge the people's University for their financial support and Senior Laboratory Technician, Mr. Kamta Prasad Ahirwar for taking reading during nerve conduction study of the patient.

Conflict of interest

The authors declared no conflict of interest.

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TABLE 1: MNCV (Upper and Lower limb)

NERVE	Rec – Stim Site	Distance (mm)	Latency difference(ms)	NCV(m/s)
Rt. CPN	EDB- ANKLE	78	5.38	14.49
	EDB-FIB.HEAD	410	9.84	41.66
Lt. CPN	EDB- ANKLE	80	4.95	16.16
	EDB-FIB.HEAD	420	9.60	43.75
Rt. PTN	Abd. Halls- ANKLE	105	7.45	14.09
	Abd. Halls- POP. FOSSA	425	11.23	37.84
Lt. PTN	Abd. Halls- ANKLE	100	5.50	20.00
	Abd. Halls- POP. FOSSA	430	9.80	43.87
Rt. Median	APB-WRIST	80	2.80	28.57
	APB-ELBOW	250	6.52	38.34
Lt. Median	APB-WRIST	75	3.30	22.72
	APB-ELBOW	240	7.12	33.70
Rt. ULNAR	ADM-WRIST	70	2.88	24.31
	ADM-ELBOW	245	5.87	41.33
Lt. ULNAR	ADM-WRIST	80	2.75	25.45
	ADM-ELBOW	240	5.63	42.62

TABLE 2: SNCV (Upper and Lower limb)

NERVE	Rec – Stim Site	Distance (mm)	Latency difference (ms)	NCV (m/s)
Rt. SURAL	Laterals Malls-MID CALF	165	2.55	64.70
Lt. SURAL	Laterals Malls- MID CALF	175	2.80	62.50
Rt. Median	2nd Digit -WRIST	130	1.90	68.42
Lt. Median	2nd Digit- WRIST	135	1.80	75.00
Rt. ULNAR	5th Digit - WRIST	130	1.95	66.66
Lt. ULNAR	5th Digit - WRIST	125	1.80	69.44

TABLE 3: F WAVE (Upper and Lower limb)

NERVE	Distance (mm)	Latency difference (ms)	Velocity (m/s)
Rt. CPN	90	35.13	2.5
Lt. CPN	80	34.15	2.34
Rt. PTN	110	35.50	3.09
Lt. PTN	105	32.26	3.25
Rt. Median	80	27.50	2.90
Lt. Median	70	24.63	2.84
Rt. ULNAR	70	29.50	2.37
Lt. ULNAR	80	25.63	3.12

Figure 1

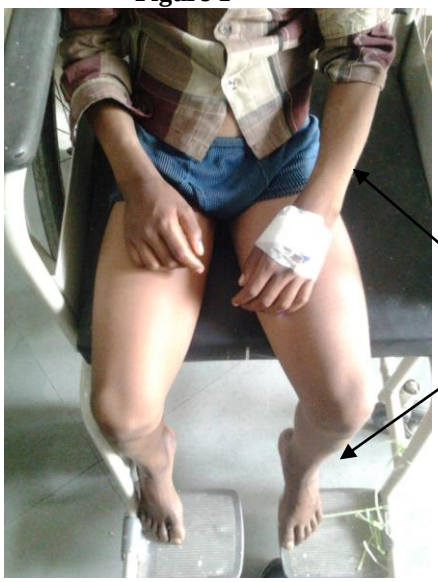

Figure 2