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Case Report

A Case Study on Guillain-Barre Syndrome with Peripheral Sensorimotor Neuropathy

Sneha Sebastian[1], Sherine Justin[1], Athira Thomson[1], Sheik Hajasherief[2]

[1]Pharm D Interns, Department of Pharmacy Practice, Nandha College of Pharmacy, Perundurai Main Road, Erode, Tamil Nadu.
[2]Department of Pharmacy Practice, Nandha College of Pharmacy, Perundurai Main Road, Erode, Tamil Nadu.

Corresponding Author: Sneha Sebastian

ABSTRACT

Guillain-Barre Syndrome (GBS) can be described as the collection of clinical syndromes that includes acute inflammatory polyradiculoneuropathy with diminished reflexes and resultant weakness. A 25 year old male patient suddenly started a feeling of numbness of bilateral hands and sole followed by sudden onset of weakness of bilateral lower limb with difficulty in arising from floor and slowness of walking. On subsequent days the weakness was progressed to both lower and upper extremities and on clinical examination it was found that, distal sensory loss was present, bilateral hip of right was greater(grade 2) than left hip (grade 3).Muscle stretch reflex were absent. On neurological examination, the patient was alert, oriented and intact speech and memory, pupils equally react and cranial nerve was intact. All the laboratory parameters were normal but an elevated protein level of 2.05g/L with increased IgG fraction (25%). Nerve conduction study (NCS) were performed and all 4 limbs showed demyelinating motor sensory polyradiculoneuropathy. Sensory nerve action potentials (SNAP) amplitudes and sensory nerve conduction velocity(SNCV) of both upper and lower limb extremities revealed borderline prolonged distal latencies. Evoked amplitude of compound muscle action potentials (CMAPs) were of reduced amplitude and temporally dispersed reduced conduction velocity, prolonged or absent F-waves reflect proximal spinal roots and distal motor terminals. The patient received a 5-day course of IV immunoglobulins and underwent physiotherapy, speech and language therapy. GBS dependent upon early diagnosis and intervention may be important in the long term prognosis. Medical care for GBS patient is considered crucial for health care team.

Keywords: Guillain-Barre Syndrome, Nerve Conduction Study, SNAP, SNCV, CMAP

INTRODUCTION

Guillain-Barre Syndrome (GBS) or acute idiopathic polyradiculoneuritis is a disorder of anonymous aetiology, involving the peripheral nervous system. [7] This 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 this antibodies leads to activation of T cells and complements, leading to a cascade of inflammation and demyelination. The velocity of nerve conduction and impulse transmission decreases due to demyelination. Clinical manifestations include progressive, symmetrical ascending muscle weakness of limbs, autonomic and brainstem abnormalities, areflexia with or without sensory. [1] The extent of nerve damage varies, as per more damage is seen in the intensely myelinated peripheral nerves, may cause motor and sensory weakness. Nerve conduction studies (NCS) are helpful in the diagnosis of several

peripheral sensorimotor neuropathies, also become more important in diagnosis of demyelinating polyneuropathies such as Guillain-Barre syndrome, chronic inflammatory demyelinating polyneuropathy. [5] GBS is the most common cause of acute muscular paralysis, affecting 0.4–2.4/100 000 people annually. [2]

CASE REPORT

A 25 year old male patient suddenly started a feeling of numbness of bilateral hands and sole followed by sudden onset of weakness of bilateral lower limb with difficulty in arising from floor and slowness of walking. On subsequent days the weakness was progressed to both lower and upper extremities and on clinical examination it was found that distal sensory loss was present, bilateral hip of right was greater(grade 2) than left hip (grade 3).Muscle stretch reflex were absent. On neurological examination, the patient was alert, oriented and intact speech and memory, pupils equally react and cranial nerve was intact.

Laboratory Assessment: All the laboratory parameters were normal and did not reveal any infectious conditions. Rheumatoid factor was negative. A lumbar puncture revealed and support the diagnosis of GBS with normal cell count but an elevated protein level of 2.05g/L with increased IgG fraction(25%) and normal CSF glucose level. Serum infection screen for HIV, Hepatitis B and C,CMV, Epstein-Barr virus were negative.

Electrodiagnostic Investigation: Nerve conduction study (NCS) were performed and all 4 limbs showed demyelinating motor sensory polyradiculoneuropathy. The nerves radian for motor and sensory amplitude of compound muscle action potential (CMAPs) were measured from baseline to negative peak. Conduction velocity was measured in the upper and lower limb. F-response latencies were measured following distal (wrist/ankle) motor nerve stimulation. Sensory nerve action potentials (SNAP) amplitudes and sensory nerve conduction velocity (SNCV) of both upper and lower limb extremities, revealed borderline prolonged distal latencies. Evoked CMAPs were of reduced amplitude and temporally dispersed with reduced conduction velocities in nerves tested. F-nerve studies include both F-wave conduction velocity and F- wave latency. F-wave resulting from the antidromic activation of motor neurons involving the conduction to and from spinal cord, it is considered as valuable tool in clinical neurophysiology [4] (Table:1).

Treatment: The patient received a 5-day course of IV Immunoglobulin and underwent physiotherapy, speech and language therapy.

Table 1: Nerve Conduction Studies, Upper and Lower Limbs

Nerve	Amplitude (Reference normal	Latency (Reference normal	Conduction velocity (Reference normal	F-wave (Reference normal
Left-median sensory	17.3µV(>20µV)	4.4ms(<3.5ms)	49m/s(>50m/s)	-
Left ulnar sensory	6.0µV(>10µV)	5.1ms(<3.7ms)	40m/s(>50m/s)	-
Right-median motor				
Wrist	10.6mV	5.2ms(<4.4ms)	48m/s(>49m/s)	Absent(<29ms)
Antecubital fossa	2.0mV(>4mv)	8.3ms		
Left median motor				
wrist	10.9Mv	5.1ms(<4.4ms)	42m/s(>49m/s)	35ms small and impersistent(<29ms)
Antecubital fossa	2.0mV(>4mv)	8.8ms		
Right ulnar motor				
Wrist	0.Mv	5.5ms(<3.5ms)	41m/s(>49m/s)	Absent(<29ms)
Elbow	0.9mV(>6mv)	8.3ms		
Left ulnar motor				
Wrist	5mV	5.4ms(<4.4ms)	48m/s(>49m/s)	Absent(<29ms)
Elbow	2mV(>6mv)	6.5ms		
Left peroneal motor				
Ankle	2mV	6.4ms(<6ms)	42m/s(>46m/s)	
Knee	0.6mV(>2mV)	9.7ms		
Left-tibial motor	2.5mV(>3mV)	6.6ms(<6ms)		52.5ms(<50ms)
Left-sural (sensory)	0.7µV(>2µV)	45ms(<42ms)	48m/s	

DISCUSSION

GBS is defined as an auto-immune mediated polyradiculoneuropathy. It may be associated with non-familial inflammatory demyelinating axonal and anterior horn cell degeneration. GBS is considered to be post infectious targeting peripheral nerves. Antecedent bacterial or viral illness prior to the onset of neurologic symptoms [1] Muscle and legs weakness was more as compared to arms and there was absence of fever at the onset of neural symptoms. [3]

Slowing of F waves was associated with demyelination; affect the proximal segment of nerve roots. In my patient abnormalities of muscle action potential including, low amplitude, prolonged latency, slowing of conduction velocity, prolonged or absent F-waves reflect proximal spinal roots and distal motor terminals. [6] The conduction block was maximal in the terminal segment of upper and lower limbs. [1]

CONCLUSION

GBS dependent upon early diagnosis and intervention may be important in the long term prognosis. Medical care for GBS patient is considered crucial for health care team. Electro-diagnostic techniques play an important role in early detection and characterization of inflammatory polyradiculoneuropathy and assume the importance of treatment and timely intervention reduces morbidity and disability.

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