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Unveiling Electrophysiological Abnormalities in Guillain Barre Syndrome: A Case Series

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Abstract:

Guillain–Barre syndrome (GBS) is an acute polyneuropathy with a variable degree of weakness that reaches its maximal severity within 4 weeks. The disease is mostly preceded by an infection and generally runs a monophasic course. GBS is subdivided in the Acute Inflammatory Demyelinating Polyneuropathy (AIDP), the most frequent form in the western world; Acute Motor Axonal Neuropathy (AMAN) most frequent in Asia and Japan; and in Miller-Fisher Syndrome (MFS). Additionally overlap syndrome exist GBS-MFS overlap). Electrodiagnostic (EDX) studies are a valid and reliable means of confirming the diagnosis. This case series is a compilation GBS patients and their Nerve Conduction studies' interpretation stating its features.

Introduction

Guillain–Barre syndrome (GBS) is an acute immune-mediated inflammatory demyelinating polyneuropathy characterized by symmetrical limb weakness and areflexia that may be associated with extensive secondary axonal and even anterior horn cell degeneration [1,2,4]. GBS can have different clinical manifestations; hence, the initial symptoms are also varied.

The classical presentation is characterized by an acute monophasic, non-febrile, post-infectious illness manifesting as ascending weakness and areflexia. Sensory, autonomic, and brainstem abnormalities may also be seen [1,2]. The demyelination is seen mainly in motor and sometimes in sensory nerves. GBS is characterized by ascending paralysis (lower limb numbness, paraesthesia, or pain followed by weakness which ascends symmetrically and

gradually progresses throughout 1 to 28 days with maximum severity of weakness by four weeks after the onset [3].

GBS can be broadly divided into two main categories axonal or demyelinating. AIDP is the most common form of GBS in developed countries accounting for 90% of cases and 48.8 to 85.2% in Indian cases [1,2]. Electro-diagnosis plays an important role in early detection and characterization of inflammatory de-myelinating polyradiculopathies [5]
Nerve conduction abnormalities become more prominent during the initial weeks of the disease even if patient's clinical status is improving [6,7].

Nerve conduction studies (NCS) may aid in the clinical diagnosis of GBS by distinguishing between axonal and demyelinating subtypes. Prolonged or missing F-waves are often the first-detected NCS abnormalities, however, additional conduction abnormalities emerge as the illness develops.

The abnormalities discovered on NCS are determined by the GBS subtype **(AIDP, AMAN, or AMSAN)**. NCS reveals demyelination symptoms in AIDP patients, such as delayed distal motor latency, decreased nerve conduction velocity, prolonged F-wave latency, increased temporal dispersion, and conduction block. The sural sensory potential is often retained. Axonal GBS (AMAN or AMSAN) is distinguished by reduced motor and/or sensory amplitudes in the absence of demyelinating characteristics. Sensory-nervous investigations may assist in distinguishing between AMAN and AMSAN.

Acute inflammatory demyelinating polyneuropathy (AIDP) is the predominant subtype in Europe and North America, and the acute motor (sensory) axonal neuropathy (AMAN, AMSAN) subtypes are more frequent in most parts of Asia [8]. We present case series having different presentations with varying severity.

CASE SERIES

Case 1

A 49-year-old housewife presented with paraesthesias in bilateral upper limbs left more than right, for 1 month followed by weakness in both upper and lower limbs. She was able to get up from supine position with some difficulty and had difficulty in walking with impaired balance. She also c/o heaviness of the right side of the face with a deviation of the angle of the mouth to the right. Power in lower limbs was grade 2/5 at the hip, followed by grade 3/5 at the knee and ankle. Power in upper limbs was grade 2/5 at the shoulder with grade 3/5 at elbow and wrist. There was a loss of plantar superficial reflexes. There was a progression of weakness followed by respiratory involvement. The Electrodiagnostic studies were performed to evaluate Bilateral Median, Ulnar, Deep Peroneal, and Tibial Nerves and Bilateral Superficial Peroneal and Sural Nerve Conduction Velocities. NCV studies showed reduced conduction velocities with decreased amplitude of compound motor action potential [CMAP] and prolonged distal motor latency. There existed a conduction block with temporal dispersion in the CMAP. Delayed F waves suggestive of proximal axonal dysfunction were predominant features as seen in GBS.NCV studies were suggestive of demyelinating motor neuropathy with marked axonal loss and conduction block in bilateral upper and lower limbs.

Case 2

A 13-year female child fully vaccinated presented with bilateral upper and lower limb weakness 15 days post-fall. She complained of difficulty in walking and impaired balance which was progressive. She also stated that she had poor gripping of objects in bilateral

upper limbs. Power was grade 2/5 at the hip, grade 3/5 at the knee and grade 2/5 at the ankle in the lower limbs whereas grade 3/5 in the upper limbs. She was started with IvIgG. Nerve conduction studies revealed reduced amplitude of CMAP with conduction block and temporal dispersion in bilateral Deep Peroneal and Left Tibial nerves of the lower limb and bilateral Median and Ulnar nerves of the upper limb. Also, the sensory nerve conduction velocities were affected including the Sural nerves exhibiting prolonged latencies suggestive of predominant demyelination. Absent F wave findings were seen in the left Median nerve whereas prolonged F waves were seen in the right Median nerve suggesting axonal dysfunction as in GBS. Prolonged Sural nerve latency with decreased conduction velocity bilaterally suggests an acute motor axonal neuropathy [AMAN] variant of GBS.

Case 3

A 26-year-old male was brought with complaints of weakness in both upper limbs and lower limbs for 2 months. He complained of paraesthesias in the right upper limb. He had difficulty standing, walking maintaining balance. The severity of the weakness had progressed post fever 5days back. There were no signs of respiratory failure. However patient had a history of Hepatitis for which he was on medications. On examination patient had hypotonia in all four limbs. Wasting of both upper limbs and lower limbs was noted predominantly on bilateral thenar, hypothenar, and palmar arches. Mild sensory involvement was noted in Ulnar nerve distribution. Power was ⅖ in the intrinsics of hand whereas the toe and digital extensors were found to be grade 2/5.Moderate tightness of bilateral calf muscles was present and ulnar claw hand position was evident. Motor Nerve conduction studies of the upper and lower limbs showed decreased amplitude of CMAP with conduction block and temporal dispersion with predominant axonal degeneration.A sensory nerve conduction study of the Right Ulnar nerve revealed prolonged latency suggestive of predominant demyelination. The above findings were suggestive of axonal and demyelinating sensorimotor polyneuropathy. Neurological manifestations of hepatitis B may take the form of Guillain-Barré syndrome [9].

Cases	Age/Sex	Nerve	Latency	Amplitude	Conduction velocity	NCV Report
1	49/F Motor	Left Median	21.67 ms	0.5 mv	46.31 m/s	Reduced conduction velocities with decreased amplitude of compound motor action potential [CMAP] and prolonged distal motor latency. There existed conduction block with temporal dispersion in the CMAP. Delayed F waves suggestive of proximal axonal dysfunction were predominant features as seen in GBS. NCV studies were suggestive of demyelinating motor neuropathy with marked axonal loss and conduction block in bilateral upper and lower limbs .
		Right Median	17.5 ms	0.7 mV	52.00 m/s	
		Left Ulnar	6.46 ms	1.5 mV	54.28 m/s	
		Right Ulnar	5.63 ms	0.6 mV	46.97 m/s	
		Left Tibial	8.96 ms	28.2 µV	26.81 m/s	
		Right Tibial	8.96 ms	164.3 µV	27.53 m/s	
		Left Deep peroneal	22.71 ms	0.4 mV	30.67 m/s	
		Right Deep peroneal	17.08 ms	1.4 mV	52.63 m/s	
	Sensory	Left Median	3.13 ms	12.5 µV	41.67 m/s	
		Right Median	6.46 ms	2.3 µV	21.67 m/s	
		Left Ulnar	3.38 ms	41.9 µV	29.59 m/s	
		Right Ulnar	3.04 ms	3.1 µV	39.47 m/s	
		Left Sural	6.00 ms	8.1 µV	35.00 m/s	
		Right Sural	8.63 ms	11.0 µV	24.36 m/s	
		Left Superficial Peroneal	4.79 ms	7.4 µV	22.96 m/s	

		Right Superficial Peroneal	7.17 ms	16.4 µV	15.34 m/s	
2	13/F Motor	Left Median	2.50 ms	1.3 mv	18.76 m/s	Reduced amplitude of CMAP with conduction block and temporal dispersion in bilateral Deep Peroneal and Left Tibial nerves and bilateral Median and Ulnar nerves .Decreased sensory nerve conduction velocities affecting the Sural nerves exhibiting prolonged latencies suggestive of predominant demyelination.Absent F wave findings were seen in left Median nerve whereas prolonged F waves were seen in right Median nerve suggesting axonal dysfunction as in GBS.Prolonged Sural nerve latency with decreased conduction velocity bilaterally suggests acute motor axonal neuropathy [AMAN] variant of GBS.
		Right Median	12.29 ms	1.1 mV	17.46 m/s	
		Left Ulnar	2.19 ms	3.8 mV	45.33 m/s	
		Right Ulnar	1.25 ms	1.9 mV	36.73 m/s	
		Left Tibial	3.44 ms	0.3 mV	43.22 m/s	
		Right Tibial	1.25 ms	129.1 µV	50 m/s	
		Left Deep peroneal	1.25 ms	0.6 mV	41.39 m/s	
		Right Deep peroneal	1.88 ms	194.8 µV	28 m/s	
	Sensory	Left Median	2.67 ms	110.4 µV	48.69 m/s	
		Right Median	4.21 ms	128.7 µV	30.88 m/s	
		Left Ulnar	3.58 ms	52.8 µV	30.73 m/s	
		Right Ulnar	5.13 ms	52.4 µV	21.48 m/s	
		Left Sural	4.63 ms	219.7 µV	25.97 m/s	
		Right Sural	8.25 ms	113.5 µV	14.55 m/s	
3	26/M Motor	Left Median	3.02 ms	13.2 mV	44.15 m/s	Motor Nerve conduction studies of upper and lower limb showed decreased amplitude of CMAP with conduction block and temporal dispersion with predominant axonal degeneration.Sensory nerve conduction study of Right Ulnar nerve revealed prolonged latency suggestive of predominant demyelination. The above findings were suggestive of axonal and demyelinating sensorimotor polyneuropathy. Neurological manifestations in hepatitis B may take the form of Guillain-Barré syndrome.
		Right Median	8.23 ms	17.1 mV	46.07 m/s	
		Left Ulnar	couldnot be performed			
		Right Ulnar	7.19 ms	1.7 mV	49.90 m/s	
		Left Deep peroneal	3.54 ms	1.0 mV	28.04 m/s	
		Right Deep peroneal	7.81 ms	56.3 µV	32.69 m/s	
	Sensory	Right Ulnar	3.29 ms	9.6 µV	36.47 m/s	
		Right Superficial Peroneal	2.17 ms	52.8 µV	64.52 m/s	

Discussion:

Nerve conduction studies are diagnostic tests that can measure the conduction speed of your nerve impulses. NCV can identify nerve damage. Nerve conduction studies (NCSs) are an essential tool in the evaluation of the peripheral nervous system[18]. Electro-diagnostic studies are helpful in diagnosis and demarcating the demyelinating variety of GBS which responds to treatment and has a better prognosis [10]. Electro-physiological hallmarks of early demyelination include prolonged distal motor latencies, prolonged/absent F wave latencies mainly in the lower limbs, slow motor conduction velocities/conduction block with absent F wave, and abnormal upper extremity sensory nerve action potential [11]

F wave is the most sensitive diagnostic test for early GBS. In our study, motor conduction velocity was decreased and proximal conduction block was noticed mainly in the lower limbs. The findings of our study are synchronous with findings of Gordon, Jun Kimura, and Kuwabara [5,12,13]. In a study done by Ropper et al. abnormalities of compound muscle action potentials including dispersion, delayed latency, low amplitude, slowing of conduction velocity, conduction block, or abnormal F-waves were evident[14]. Similar results have been found in studies done by Clouston et al.[15] .Prolonged distal motor latencies & prolonged or absent F waves reflect an early predilection for involvement of proximal spinal roots and distal motor terminals. The conduction block was maximal in the terminal segment in the upper and lower limbs, more so in the lower limb in our study. These findings were

concurrent with those of studies by Brown [16]. A decrease in conduction velocity is damage to the myelin sheath; both cellular and immune mechanisms play important roles in it.
In pediatric patients, acute motor axonal neuropathy (AMAN) was the most common finding in our study. This was similar to the study by Kalra et al[5] who also showed that AMAN may not always indicate a bad prognosis compared with AIDP (Acute Inflammatory Demyelinating Polyneuropathy) There are two patterns of recovery seen in patients with AMAN— firstly the rapidly recovering conduction block at the nodes of Ranvier having low CMAP (compound muscle action potential) with a good prognosis and secondly the inexcitable nerves with more protracted recovery in those with extensive axonal degeneration having a poorer prognosis. In our present study, the 13-year-old recovered rapidly with intravenous Immunoglobulins[IvIgG]. Alexander et al. compared the electrophysiological findings among adults and children and showed that AMAN is more common in children than adults [17].

CONCLUSION

Electro-diagnosis plays an important role in the early detection and differentiation of inflammatory demyelinating poly-radiculopathy in the first week of symptomology. The presentation of GBS can vary from mere weakness of limbs to complete flaccid paralysis. Symmetrical lower limb weakness was the most common complaint seen in our patients. Evidence of conduction block and temporal dispersion are hallmarks of multifocal demyelination. Guillain-Barre syndrome requires both motor and sensory conduction studies performed on nerves in upper and lower extremities, and F wave latency measurements.

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