Case Report

Combined proximal nerve conduction study and F wave analysis confirms diagnosis of unusual unilateral proximal median nerve lesion in a diabetic patient

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Abstract

Diabetic patients usually present with multiple nerve lesions varying in intensity and distributions among them. Nerve conduction studies (NCS) are usually done for measuring the level of block, type of lesion and aid in diagnostic criteria of polyneuropathies. F waves are usually not done in clinical setting but only on demand from the neurophysician. We present a case of uncontrolled diabetic who had features of carpal tunnel syndrome. He had proximal conduction block (median nerve neuropraxia) with normal sensory distal conduction. Further, antidromic action potentials of F wave analysis confirmed a proximal median nerve lesion rather than distal. We hypothesise that F wave analysis may add to the confirmatory diagnosis of proximal nerve lesions to routine NCS.

Key words: Antidromic impulse, carpal tunnel, diabetic polyneuropathy, F wave, proximal median latency, proximal median nerve lesion

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INTRODUCTION

In India, diabetes is growing at an alarming rate so does diabetes-induced polyneuropathy.[1] Sensory, motor, autonomic, mixed, symmetric and asymmetric peripheral neuropathies have been documented earlier.[2] The neuropthathic changes may be attributed to inflammation, glycosylation through polyol pathway, vascular changes in nerves and oxidative stress.[3] The types of nerve damage are neuropraxia (conduction block), axonotemesis, neurogenesis (axon damage with demyelination) and demyelination alone. In diabetics, demyelination (like Guillain Barre syndrome [GBS]) and conduction block (neuropraxia) are common.[4] The neuropathic changes are usually measured by nerve conduction studies (NCS) (sensory nerve action potential, compound motor action potential [CMAP] and F waves).[4]

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We present a case of uncontrolled diabetes with carpal tunnel syndrome, who was diagnosed with normal distal median nerve conduction but diminished proximal conduction. Further, F wave's antidromic latency proved that entrapment is proximal to wrist rather than at distal sites.

CASE REPORT

A 54-year-male, businessman presented with tingling and numbness of right hand to our neurophysiology laboratory. He was referred to our neurophysiology laboratory for the NCS with the possible diagnosis of right hand carpal tunnel syndrome.

On examination, the patient had type II diabetes for past 12 years and on irregular medications such as metformin for past 6 months. His fasting sugar was 146 and postprandial 330 mmol leading to confirmation of uncontrolled diabetes with glucose intolerance. He had tingling, sharp shooting pain occasionally and numbness

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sensation for past 3 months more during night. With the hypothesis of diabetes induced carpal tunnel syndrome, the nerve conduction velocity was done for all the four prominent nerves of body – median, ulnar, radial, tibial, peroneal and sural for delineating polyneuropathy.

Nerve conduction studies were done on ulnar, median and radial nerve sensory points. Ulnar, radial conduction velocities are normal. Median nerve conduction velocity was reduced 50% above wrist and axilla level, whereas the median nerve conduction velocity was normal below wrist and digits in left median nerve compared to right median nerve [Figure 1]. Hence, it is partially confirmed as proximal conduction block of median nerve at the pronator teres (axilla) rather than distal at wrist (carpal tunnel).

F wave analysis was done through motor evoke potentials on median nerve myotome. The F wave antidromic impulses portrayed a proximal conduction rather than distal at carpal tunnel in left median nerve compared to right median nerve [Figure 2]. Thus, the patient who came with diagnosis of carpal tunnel syndrome was reported to the Neurophysician as proximal conduction block (neuropraxia) of right median nerve with confirmation from sensory conduction potentials and F wave analysis.

As we hypothesise diabetic neuropathy, we performed nerve conduction velocity and F wave analysis on sural nerve too. Figure 3 depicts the pattern of sensory nerve conduction velocity of all peripheral nerves. This demonstrated an asymmetrical distal polyneuropathy in liaison with the diagnosis of diabetic peripheral neuropathy with proximal conduction defect of median nerve.

DISCUSSION

This is the first case of its kind to depict a proximal lesion of median nerve for a diabetic individual with the working diagnosis of carpal tunnel syndrome using nerve conduction velocity reinforced by F wave analysis patterns.

Diabetic neuropathy, commonest complication of diabetes is underdiagnosed and often overlooked complication in primary care.[5] Neuropathy is still assessed clinically through symptoms and sensation by filaments. Only sophisticated institution and clinics have expertise and equipments for additional neuro-diagnosis. Even with expertise and quality neuro-diagnostic equipments, the proximal conduction blocks are usually missed due to neglect of F waves, which are not uncommon.[5] Western literature has proved that majority of diabetic neuropathy have proximal action potential latency decrease.[6]

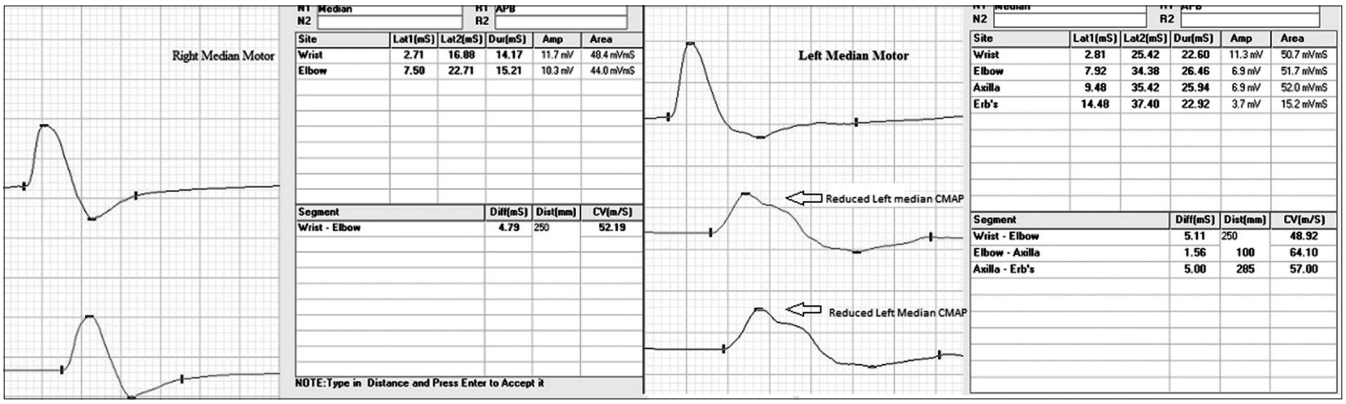


Figure 1: Distal and proximal latencies of median nerve

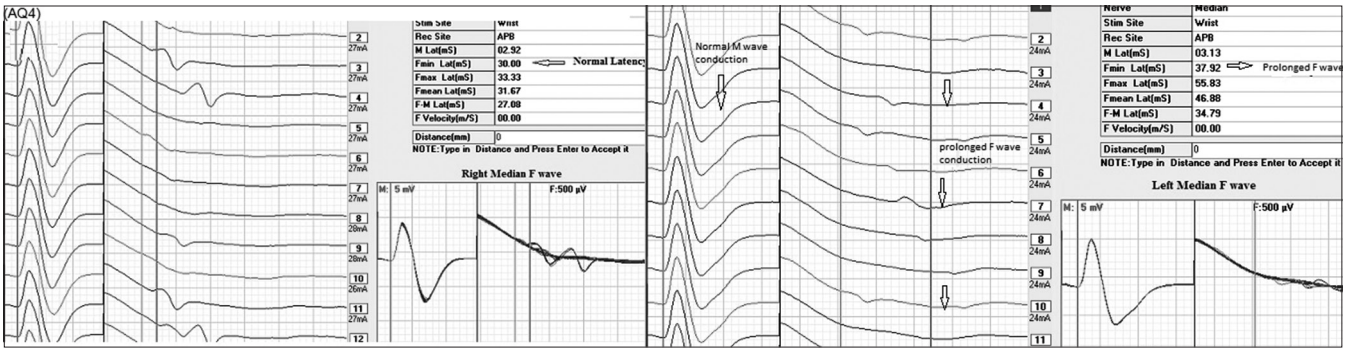
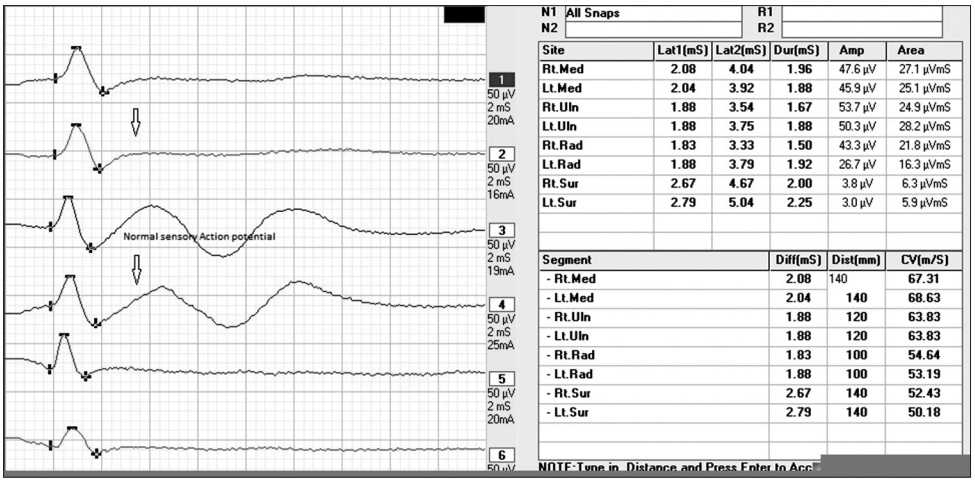


Figure 2: F wave analysis of median nerve (right vs. left)



Site	Lat1(mS)	Lat2(mS)	Dur(mS)	Amp	Area
Rt.Med	2.08	4.04	1.96	47.6 µV	27.1 µVmS
Lt.Med	2.04	3.92	1.88	45.9 µV	25.1 µVmS
Rt.Uln	1.88	3.54	1.67	53.7 µV	24.9 µVmS
Lt.Uln	1.88	3.75	1.88	50.3 µV	28.2 µVmS
Rt.Rad	1.83	3.33	1.50	43.3 µV	21.8 µVmS
Lt.Rad	1.88	3.79	1.92	26.7 µV	16.3 µVmS
Rt.Sur	2.67	4.67	2.00	3.8 µV	6.3 µVmS
Lt.Sur	2.79	5.04	2.25	3.0 µV	5.9 µVmS

Segment	Diff(mS)	Dist(mm)	CV(m/S)
- Rt.Med	2.08	140	67.31
- Lt.Med	2.04	140	68.63
- Rt.Uln	1.88	120	63.83
- Lt.Uln	1.88	120	63.83
- Rt.Rad	1.83	100	54.64
- Lt.Rad	1.88	100	53.19
- Rt.Sur	2.67	140	52.43
- Lt.Sur	2.79	140	50.18

Figure 3: Figure showing sensory nerve conduction velocity of all the peripheral nerves

In our study, we confirmed a proximal conduction defect through proximal conduction velocity or proximal CMAP and F waves. CMAP is used to measure the conduction velocity in sensory fibres of median and F waves for antidromic motor conduction velocity.[5] In our suspected case of carpal tunnel syndrome, both the studies yielded a similar diagnosis of proximal conduction block of median (axilla to forearm) rather at periphery (carpal tunnel). Asymmetrical demyelinating polyneuropathies are not uncommon in diabetes mimicking GBS.[7] In our patient too, asymmetrical distributions of conduction blocks and latencies of peripheral nerves were shown.

Hence this report may depict that a diabetic individual presenting symptoms of carpal tunnel syndrome may have proximal conduction blocks, which may be further confirmed with F wave analysis.

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