Original article:

Evaluation of nerve conduction abnormalities in type 2 diabetic patients

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Abstract

Introduction: Diabetes mellitus (DM) comprises a group of common metabolic disorders that share the phenotype of hyperglycaemia. Depending on the aetiology of the DM, factors contributing to hyperglycaemia may include reduced insulin secretion, decreased glucose utilization, and increased glucose production. The metabolic deregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems. Diabetic neuropathy is the most common and troublesome complication of diabetes mellitus leading to great morbidity and resulting in a huge economic burden for diabetes care. However, the progression of neuropathy can be reduced by early detection using nerve conduction studies (NCS) and intervention at early stages.

Methods: Forty type 2 diabetes mellitus patients of both sexes in the age group between 35 and 55 were included in the study. Age and sex matched healthy subjects were used as controls. Nerve conduction study (NCS) of median nerve of right upper limb was performed in diabetic cases and healthy controls. Amplitude, latency and nerve conduction velocity were recorded. Results were analysed by student's independent t-test. P- Value <0.05 was considered significant.

Observation and results : The amplitude and conduction velocity were significantly reduced in DM patients compared to healthy controls. Latency of nerve conduction was increased in DM patients compared to healthy controls and was found to be statistically significant.

Conclusion: Nerve conduction study can be used as a tool to assess the involvement and progression of damage to peripheral nerves in DM patients.

Keywords - DM-Diabetes mellitus, NCS – Nerve conduction study

INTRODUCTION

Peripheral nervous system disorders are one of the more frequent long-term complications of diabetes mellitus. Diabetic neuropathy (DN) occurs in approximately 50% of individuals with long-standing type 1 and type 2 DM. It may manifest as polyneuropathy, mononeuropathy, and/or autonomic neuropathy. As with other complications of DM, the neuropathy correlates with the duration of diabetes; both myelinated and unmyelinated nerve fibers are lost. DN is not a single entity but a number of different syndromes ranging from subclinical to clinical manifestation depending on the classes of nerve fibres involved. The main groups of neurological disturbance in DM include.

1) Subclinical neuropathy, determined by abnormalities in electro diagnostic and quantitative sensory testing.

2) Diffuse clinical neuropathy and distal symmetric sensorimotor and autonomic syndromes.

3) Focal syndromes.

ROLE OF ELECTROPHYSIOLOGICAL STUDIES IN DIABETIC NEUROPATHY

Electrophysiological studies are more sensitive than clinical examinations as clinical examinations fail to offer quantitative results and the

electrodiagnostic tests are the least variable non invasive measures of neuropathy. Evaluation of neuropathy is generally undertaken by electrophysiological measurements .According to San Antonio a patient with diabetic neuropathy must have a sign or a symptom and an abnormal electrodiagnostic test. Electrodiagnostic tests have widespread applications and are reliable, reproducible measures of peripheral nervous system function. They are objective measures that are relatively independent of patient effort or cooperation. Nerve Conduction Study (NCS) and needle Electromyography (EMG) are well accepted for the evaluation of diabetic neuropathy. They are sensitive measures, able to detect abnormalities in diabetic patients that may not be clinically apparent. Electrophysiological measures of nerve function have been the mainstay of 'objective' assessment of neurological deficits in diabetic patients. Some form of abnormality can be detected in the majority of patients. Symptoms do not necessarily correlate with the electrophysiological abnormalities.

AIMS AND OBJECTIVES

1. To study the effects of the involvement of peripheral nervous system in non insulin dependent diabetic individuals by doing nerve conduction study and comparing it with controls.
2. To ascertain that nerve conduction study is more sensitive, easily reproducible and reliable than clinical evaluation in assessing diabetic neuropathy.

MATERIALS AND METHODS

The study was conducted in the Institute of Physiology and Experimental Medicine, Madras Medical College, after getting permission from the Institutional Ethical Committee, Madras Medical College, Chennai. Tests were conducted using RMS – EMG MEDICARE SYSTEMS. Type 2 diabetes mellitus patients of both sexes in the age group between 35 and 55, were included in the study. Type 2 Diabetic patients with signs and symptoms of neuropathy were excluded from the study; other exclusion criteria were hypertension, smoking, metabolic abnormalities causing neuropathy and patients on drugs leading to neuropathy. Forty age and sex matched healthy subjects were used as controls. Nerve conduction study of motor and sensory division of right median nerve was done.

NERVE CONDUCTION STUDY

PRINCIPLES OF MOTOR NERVE CONDUCTION

The motor nerve is stimulated at two points along its course. The pulse is adjusted to record a compound muscle action potential. Typically, the impulse is generated using a bipolar stimulator placed on the surface of the skin over the anatomic course of nerve being tested. The nerve is subjected to supramaximal stimulation keeping the cathode close to the active recording electrode. This prevents the hyperpolarisation effect of anode and anodal conduction block. The surface recording electrodes are used and placed in belly tendon montage, keeping the active electrode close to the motor point and reference to the tendon, ground electrode is placed between the stimulating and recording electrodes. A biphasic action potential with initial negativity is recorded. Surface stimulation of healthy nerve requires a square wave pulse of 0.1 ms duration with an intensity of 5-40 ma (milliamperes). Filter setting for motor nerve conduction study is 5khz-10khz and sweep speed 2-5 ms/division.

The measurement for motor nerve conduction study includes the following 1. Onset latency. 2. Amplitude of compound muscle action potential (CMAP). 3. Duration of compound muscle action potential. 3. Nerve conduction velocity.

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PRINCIPLES OF SENSORY NERVE CONDUCTION

The sensory nerve conduction can be measured orthodromically or antidromically. In orthodromic conduction, a distal portion of the nerve e.g. digital nerve is stimulated and sensory nerve action potential (SNAP) is recorded at a proximal point along the nerve. In antidromic sensory nerve conduction, the nerve is stimulated at a proximal point and nerve action potential is recorded distally.

For orthodromic conduction ,ring electrodes are preferred to stimulate the digital nerve, whereas surface stimulating electrodes are commonly used for antidromic conduction.

The signal enhancement with averaging is proportional to the square root of the number of trials. Change in amplitude =square root of n. n= no of trials, which is kept at 20.

OBSERVATIONS & RESULTS

Forty type 2 diabetic patients and forty healthy controls were included in this study. Nerve conduction tests of median sensory (rt) and median motor (rt) were performed. Latency, amplitude and nerve conduction velocity were measured.

Comparison of latency of sensory division and motor division of median nerve between diabetics and non diabetics.(milliseconds) :

There was a significant increase in the latency of median nerve (sensory and motor division) in the diabetic group compared to the healthy controls

NERVE TESTED	GROUPS	MEAN ±SD	P-VALUE
Median nerve sensory division	Case	22.03+/-4.50	<0.05
	Control	33.96+/-3.95	
Median nerve motor division	Case	3.69+/-1.09	<0.05
	Control	7.70+/-1.22	

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Comparison of amplitude of sensory (µv) and motor division (mv) of median nerve between diabetics and healthy controls

There was a significant decrease in the amplitude of median nerve (sensory and motor division) in the diabetic group compared to the healthy controls.

NERVE TESTED	GROUPS	MEAN ±SD	P-VALUE
Median nerve sensory division	Case	47.92+/-3.73	<0.05
	Control	55.21+/-3.01	
Median nerve motor division	Case	50.11+/-5.00	<0.05
	Control	57.01+/-2.31	

Comparison of NCV of median nerve-sensory and median nerve-motor between diabetics and controls.(meters/sec)

There was a significant decrease in the nerve conduction velocity of median nerve (sensory and motor division) in the diabetic group compared to the healthy controls

NERVE TESTED	GROUPS	MEAN ±SD	P-VALUE
Median nerve- sensory division	Case	4.14 ± 0.96	<0.05
	Control	3.00+/- 0.32	
Median nerve- motor division	Case	4.57+/-1.15	<0.05
	Control	3.56+/-0.28	

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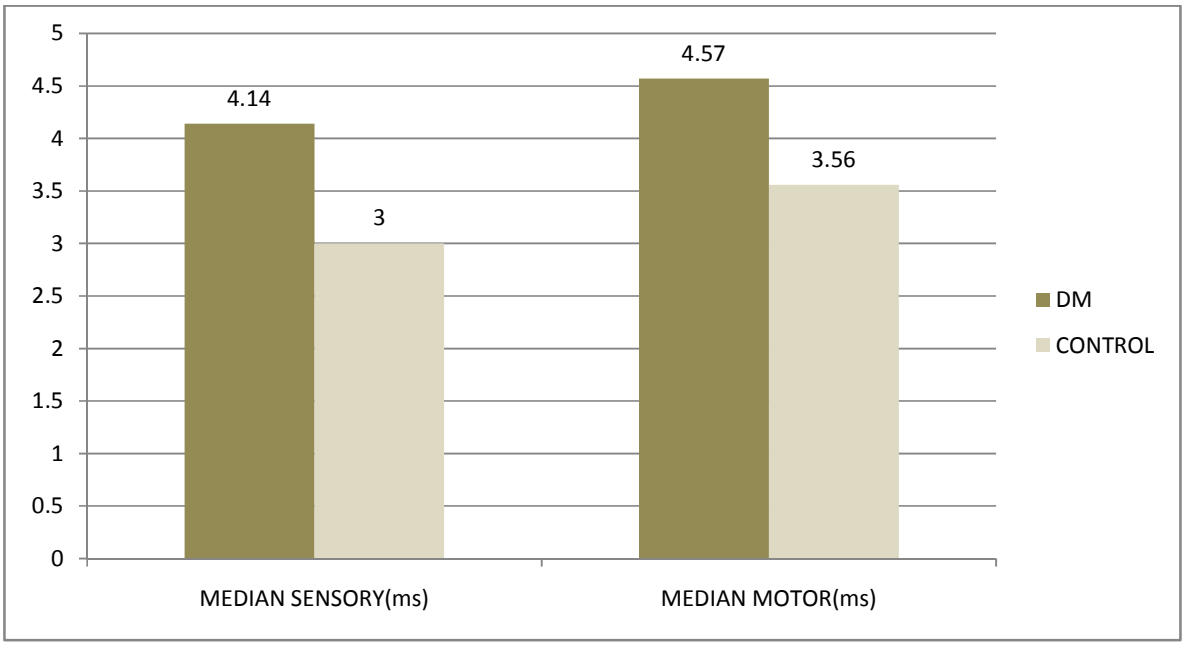
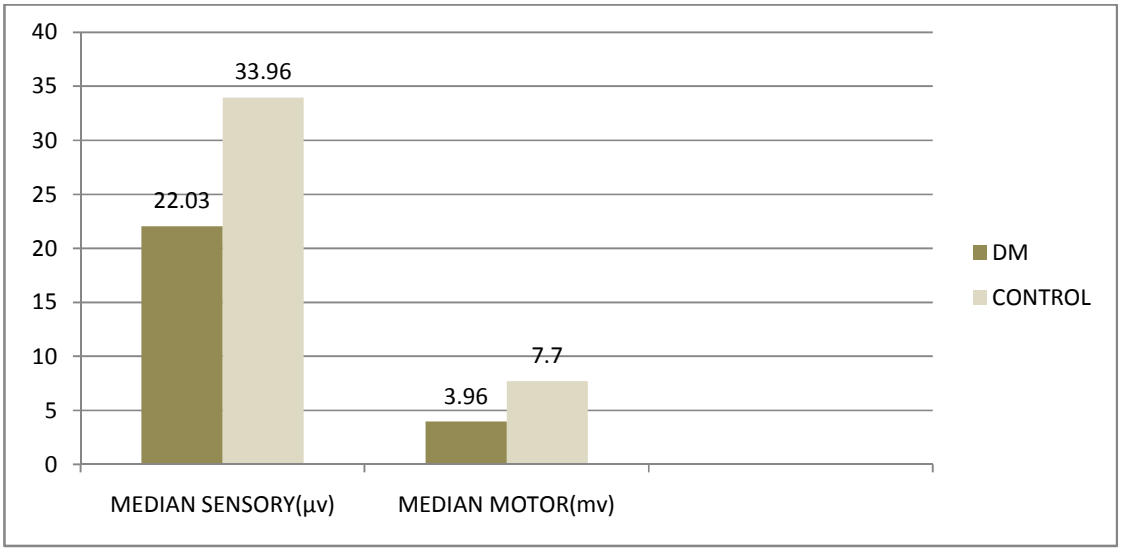
Comparison of latency of sensory division and motor division of median nerve between diabetics and non diabetics.

Table 1.1



Comparison of amplitude of sensory and motor division of median nerve between diabetics and healthy controls

Table 1.2



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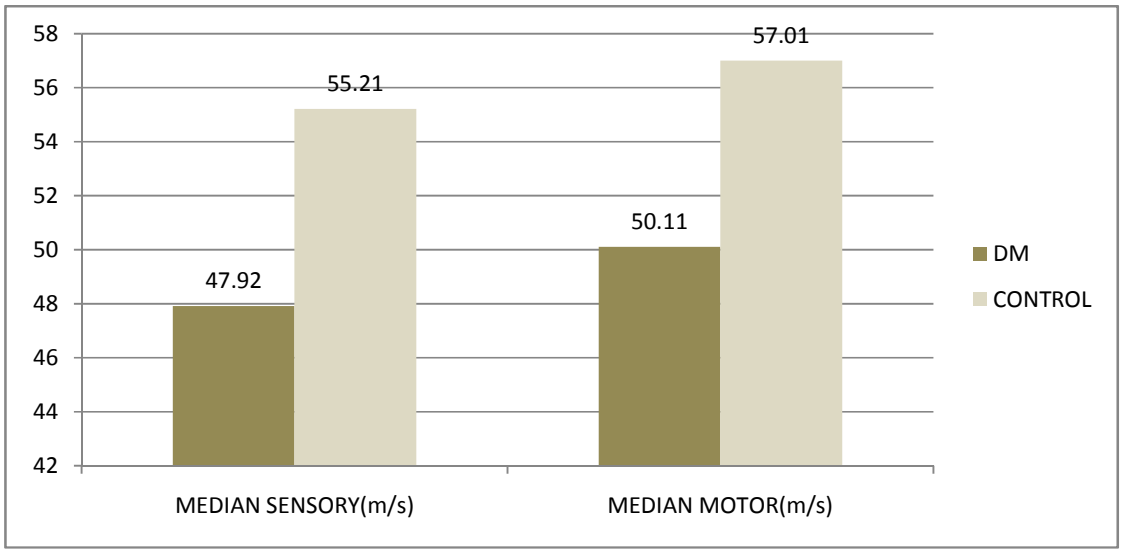
Comparison of NCV of median nerve-sensory and median nerve-motor between diabetics and controls.

Table 1.3



DISCUSSION

Nerve conduction studies are simple, sensitive and objective technique for evaluating impulse conduction along the peripheral nerves. The present study deals with the abnormalities in nerve conduction study in non insulin dependent diabetes mellitus patients. In our study nerve conduction parameters of sensory and motor component of median nerve were studied unilaterally (right side). In several clinical trials, nerve conduction studies were often used, and were shown to be symmetrical in patients with diabetic sensory and sensorimotor polyneuropathy, thus justifying unilateral evaluation. In our study a statistically significant difference was noted between the study group and control group in latencies of all the nerves tested viz., sensory and motor division of median nerve, we also observed a significant decrease in the amplitude and nerve conduction velocity in diabetics when compared with controls.

Both latency and nerve conduction velocity depend on an intact, myelinated nerve as myelin and the saltatory conduction are essential for fast action potential propagation in normal subjects. In contrast, the amplitude of the waveform depends primarily on number of axons functioning within the nerve. Slowing of conduction velocity or prolongation of latency usually implies demyelinating injury, while loss of amplitude usually correlates with axonal loss or dysfunction.

Diabetic neuropathy is the most common and troublesome complication of diabetes mellitus leading to great morbidity and resulting in a huge economic burden for diabetes care. However, the progression of neuropathy can be reduced by early detection and intervention. In a prospective study of over 4400 diabetic out patients, Pirart J et al reported an overall 12% prevalence rate of diabetic neuropathy in patients with newly diagnosed diabetes and the incidence of neuropathy increased with the duration of diabetes and after 25 years of diabetes, over 50% of patients had DN. In another study it was reported prevalence of neuropathy was 5% in the 20 to 29 year old group and increased with age, reaching 44.2% in patients between 70 to 79 years of age. Prevalence is typically higher if ascertainment is based on electrophysiological measurements, but lowers if it is based on

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subjective symptoms and physical findings only. This is the reason DM patients with clinically established diabetic neuropathy were excluded from the study to find out the efficacy of nerve conduction studies in finding out the damage caused to peripheral nerves.

Electrophysiological measures of nerve function have been the mainstay of 'objective' assessment of neurological deficits in diabetic patients. Some form of abnormality can be detected in the majority of patients. Symptoms do not necessarily correlate with the electrophysiological abnormalities 40. The recording of a nerve action potential of normal latency, amplitude, and wave form requires synchronous conduction in the large myelinated fibres. In diabetic neuropathy, the sensory nerve potentials are characterized by reduced amplitude, a polyphasic shape and an increased latency of the initial peak. These alterations can also appear, though to a lesser degree, when a careful clinical examination of the nervous system is negative.

CONCLUSION

From this study it can be concluded that the peripheral nervous system is involved in diabetes mellitus as evidenced by abnormal nerve conduction parameters. In this study it was found that DM patients with no clinical evidence of neuropathy had abnormal nerve conduction findings. This study suggests that periodic evaluation of diabetic individuals to such tests will help in monitoring the progress of neuropathy.

REFERNCES

1. Dennis L. Kasper, Eugene Braunwald, Anthony S. Fauci, Stephen L. Hauser,Dan L. Longo, J. Larry Jameson, and Kurt J. Isselbacher, Eds.Harrison's Principles Of Internal Medicine Sixteenth Edition, 2005.
2. McCarty D, Zimmet P. Diabetes 1994 to 2010, Global Estimates and Projections.Melbourne: International Diabetes Institute; 1994
3. Dyck PJ, O'Brien PC. Meaningful degrees of prevention or improvement of nerve conduction in controlled clinical trials of diabetic neuropathy. Diabetes Care 1989; 12:649-52.
4. A.I.Vinik, A.Mehrabyan. Type 2 Diabetes Mellitus. Med Clin N Am 88 (2004) 947-999.
5. Graham WA, Goldstein R, Keith M , Nesathurai S. Serial nerve conduction studies of the tail of rhesus monkey (Macaca malatta) and potential implications for interpretation of human neurophysiological studies. Ann Indian Acad Neurol 2007; 10:252-4
6. Vinik AI, Holland MT, Le Beua JM, Liuzzi FJ, Satnsberry KB, Colen LB. Diabetic neuropathies. Diabetic care 1992; 15(12): 1926-1951.10. Soliman E.Diabetic neuropathy. e Medicine Journal 2002;3.
7. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2007; 30(S1):S42-7
8. American Clinical Neurophysiology Society. Guidelines on short-latency auditory evoked potentials.
9. Boulton AJM. Peripheral neuropathy and the diabetic foot. The Foot 1992; 2: 67-72.
10. Malik RA, Veves A, Fernando DJS, Masson EA, Schady W, Sharma AK, Lye RJ, Boulton AJM. Non invasive nerve function tests; do they reflect the extent of nerve fibre and microvascular damage in early human diabetic neuropathy? Diabetes 1991; 40: suppl 554A
11. UK Misra, J Kalita In: History of Clinical Neurophysiology. Clinical Neurophysiology, 1st Ed 2005, Elsevier, New Delhi 1-7.
12. Perkins BA, Ngo M, Bril V. Symmetry of nerve conduction studies in different stages of diabetic polyneuropathy. Muscle Nerve 2002; 25:212-217. By demonstrating that diabetic polyneuropathy is electro diagnostically symmetrical, this paper provides support for the performance of unilateral studies in treatment trials.

13. Thomas H Brannagan, Louis H Weimer, and Norman Lator. Acquired neuropathis. In: Merritt's neurology, 11th edition, Lewis P. Rowland, Philadelphia: 761
14. .Pirart J: Diabetes mellitus and its degenerative complications; A prospective study of 4400 patients observed between 1947 and 1973. Diabetes Care 1978; 1: 168.
15. Young MJ, Boulton AJM, Macleod AF,et al: A multicentre study of the prevalence of diabetic peripheral neuropathy in the United Kingdom hospital clinic population. Diabetologia 1993; 36: 150
16. Malik RA, Veves A, Fernando DJS, Masson EA, Schady W, Sharma AK, Lye RJ, Boulton AJM. Non invasive nerve function tests; do they reflect the extent of nerve fibre and microvascular damage in early human diabetic neuropathy? Diabetes 1991; 40: suppl 554A
17. Dawson, G. D. (1956). The relative excitability and conduction velocity of sensory and motor nerve fibres in man Journal of Physiology, 131, 436-451.
18. Downie, A. W., and Newell, D. J. (1961). Sensory nerve conduction in patients with diabetes mellitus and controls. Neurology (Minneap.), 11, 876-882.

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