



A COMPARATIVE STUDY OF MOTOR NERVE CONDUCTION VELOCITY AND DISTAL LATENCY BETWEEN CONTROLLED AND UNCONTROLLED CASES OF TYPE 2 DIABETES MELLITUS PATIENTS IN MUZAFFARNAGAR MEDICAL COLLEGE AND HOSPITAL, MUZAFFARNAGAR

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ABSTRACT

Background: Diabetic neuropathy is one of the most common chronic complications of Type 2 Diabetes Mellitus (T2DM) and may remain asymptomatic during its early stages. Nerve conduction studies provide an objective and sensitive method for detecting subclinical peripheral nerve dysfunction before the onset of clinical manifestations.

Objective: To compare motor nerve conduction velocity (MNCV) and distal latency of the median and ulnar nerves between controlled and uncontrolled patients with T2DM.

Materials and Methods: This hospital-based observational study was conducted in the Departments of Physiology and Medicine at Muzaffarnagar Medical College & Hospital. A total of 140 patients with T2DM aged 40–60 years were enrolled and categorized into controlled (Group A, n=70) and uncontrolled (Group B, n=70) diabetic groups. Patients with neurological disorders, alcohol abuse, smoking, vitamin deficiencies, thyroid disorders, heavy metal exposure, or medications affecting peripheral nerves were excluded. Unilateral motor nerve conduction studies of the median and ulnar nerves were performed using a computerized RMS EMG EP MK II system. Motor nerve conduction velocity and distal latency were recorded. Statistical analysis was performed using SPSS version 17/20, with p value <0.05 considered statistically significant.

Results: The uncontrolled diabetic group demonstrated significantly reduced median and ulnar motor nerve conduction velocities and significantly prolonged distal latencies compared with the controlled diabetic group (p<0.001). These findings indicate early peripheral motor nerve dysfunction associated with poor glycemic control.

Conclusion: Poor glycemic control significantly impairs peripheral motor nerve conduction in patients with T2DM. Motor nerve conduction studies are effective, non-invasive tools for the early detection of subclinical diabetic neuropathy and may facilitate timely intervention to prevent disease progression and improve long-term neurological outcomes.

KEYWORDS: Diabetic Neuropathy-DN, Motor Nerve Conduction Velocity-MNCV, Motor Distal Latency -MDL.

Introduction

Chronic hyperglycemia and disruptions in the metabolism of carbohydrates, fats, and proteins due to deficiencies in insulin production, action, or both are hallmarks of diabetes mellitus[1]. Numerous physiological systems, including the central neural system, peripheral nervous system, musculoskeletal system, autonomic nervous system, and cardiovascular system, are affected by diabetes[2].

Peripheral neuropathy, retinopathy, nephropathy, and vascular problems are common side effects of diabetes[3].

One of the most upsetting long-term consequences of diabetes is diabetic peripheral neuropathy (DPN), which results in severe impairment and a low quality of life[4].

“In both type 1 and type 2 diabetes, the frequency of diabetic neuropathy seems to be correlated with the length and intensity of hyperglycemia[5].

Symptoms of numbness, discomfort, or both, typically symmetrical in distribution and initially felt in the toes, are indicative of peripheral diabetic neuropathy[6].

Signs and symptoms of peripheral nerve dysfunction in a patient with diabetes mellitus (DM) when other causes of peripheral nerve dysfunction have been ruled out are known as diabetic neuropathy (DN), a prevalent condition[7].

The most prevalent type, chronic distal polyneuropathy (also known as diabetic sensorimotor polyneuropathy [DPN]), with a prevalence of about 50%. Its development is a progressive process with a lengthy subclinical stage. Vascular and metabolic abnormalities brought on by long-term hyperglycemia and inadequate glycemic management are believed to be the cause of DPN[7].

Routine NCS include evaluation of motor function of the median, ulnar, peroneal and tibial nerves, and in terms of onset latency, amplitude, and conduction velocity[8].

Neuropathy is one of the earliest and most commonly reported long-term complications, affecting an estimated 50-60 % of patients with type 2 diabetes[1-4].

Peripheral neuropathy is an important late complication of diabetes mellitus. The prevalence of diabetic neuropathy appears to parallel the duration and severity of hyperglycemia in both type I and type 2 diabetes.

Diabetic sensorimotor polyneuropathy is the most common clinical subtype seen in clinical practice. Sensory nerves are affected earlier than motor nerves are. The most common nerve affected in the upper limb is the median nerve, while in the lower limb, it is the sural nerve. Nerve conduction abnormalities exist in the subclinical stages of neuropathy that can be detected in early asymptomatic stage by various electrodiagnostic test[9].

The objectives of the present study was to evaluate motor nerve conduction velocity and distal latency parameters in patients with type 2 DM, with an attempt to analyse

their value in the early detection of subclinical diabetic neuropathy.

Material and Methods

This hospital-based observational study was conducted in the Department of Physiology in collaboration with the Department of Medicine at Muzaffarnagar Medical College & Hospital, Muzaffarnagar. The study enrolled 140 patients with established Type 2 Diabetes Mellitus (T2DM) aged between 40 and 60 years, diagnosed according to the criteria of the American Diabetes Association. Patients with established diabetic neuropathy, inflammatory demyelinating neuropathies, lumbar or cervical radiculopathies, or any other neurological disorders affecting peripheral nerve conduction were excluded. Additional exclusion criteria included chronic alcoholism, smoking, history of occupational or environmental heavy metal exposure, vitamin B12 or vitamin B6 deficiency, thyroid disorders, and the use of medications known to cause peripheral neuropathy, including isoniazid, anticancer drugs, and antiretroviral therapy.

Written informed consent was obtained from all participants after explaining the purpose and procedure of the study in their vernacular language. A detailed medical and family history was recorded for each participant, followed by a comprehensive general physical and clinical examination. Random blood sugar estimation was performed in all study participants. Patients with clinical signs or symptoms suggestive of autonomic dysfunction or peripheral neuropathy were excluded to ensure that only asymptomatic T2DM patients were evaluated for subclinical peripheral nerve involvement.

Motor nerve conduction studies were performed in all participants using a computerized RMS EMG EP MK II machine with standard surface electrodes under standardized laboratory conditions. Unilateral motor nerve conduction studies of the median and ulnar nerves were carried out in the upper limbs. The parameters recorded included motor distal latency and motor nerve conduction velocity. For motor nerve conduction recording, the active electrode was placed over the motor point of the abductor pollicis brevis muscle, while the reference electrode was positioned approximately 3 cm distal to the active electrode. A ground electrode was placed between the stimulating and recording electrodes to minimize electrical interference. Supramaximal electrical stimulation was delivered at two standard sites: the wrist (S1) and the elbow (S2), and the recorded responses were used to calculate distal latency and conduction velocity according to standard electrophysiological techniques.

All observations were recorded in a predesigned proforma and entered into Microsoft Excel for data management. Statistical analysis was performed using SPSS version 17/20 and Statcal.2 software. Continuous variables were expressed as mean $\pm$ standard deviation, whereas categorical variables were presented as frequencies and percentages.

Comparisons between controlled diabetic patients (group A) and uncontrolled diabetic patients (group B) were performed using appropriate statistical tests based on the distribution of the data. A p-value of less than 0.05 was considered statistically significant. Unpaired t test was applied for the data analysis. The study protocol was approved by the Institutional Ethics Committee of Muzaffarnagar Medical College & Hospital.

Results

Table 1 shows the distal latency of the median nerve and motor nerve conduction velocity are compared between Group A and Group B of Type 2 Diabetes Mellitus in compared to managed diabetic participants, uncontrolled diabetic subjects shows significant increase in distal latency and a significant decrease in median nerve conduction velocity.

Table 1: Comparison of Median Nerve NCV and Distal Latency between Controlled and Uncontrolled cases of Type 2 Diabetes Mellitus

Parameter	Group A (n=70)	Group B (n=70)	p value
Motor NCV (m/s)	58.54 ± 1.71	48.18 ± 1.39	<0.001*
Distal latency (ms)	3.21 ± 0.15	4.82 ± 0.18	<0.001*

Table 2: Comparison of Ulnar Nerve NCV and Distal Latency between Controlled and Uncontrolled cases of Type 2 Diabetes Mellitus

Parameter	Group A (n=70)	Group B (n=70)	p value
Motor NCV (m/s)	57.46 ± 1.51	47.77 ± 1.4	<0.001*
Distal latency (ms)	2.96 ± 0.15	4.46 ± 0.21	<0.001*

Table 2, shows the distal delay of the ulnar nerve and motor nerve conduction velocity are compared between Group A and Group B of Type 2 Diabetes Mellitus in Uncontrolled diabetic patients showed a statistically significant increase in distal latency and a decrease in ulnar nerve conduction velocity, indicating increased peripheral nerve involvement.

Discussion

The present study evaluated motor nerve conduction parameters of the median and ulnar nerves in controlled and uncontrolled patients with Type 2 Diabetes Mellitus (T2DM) to determine the effect of glycemic control on peripheral nerve function. The findings demonstrated a significant reduction in motor nerve conduction velocity (MNCV) and a significant prolongation of distal latency in the uncontrolled diabetic group compared with the controlled diabetic group. These observations indicate that poor glycemic control is

associated with impaired peripheral motor nerve conduction and may reflect early subclinical neuropathic changes even in the absence of overt clinical symptoms.

In the present study, the median nerve showed significantly lower conduction velocity and higher distal latency in uncontrolled diabetic patients than in controlled patients. Similar findings have been reported by several investigators who observed that worsening glycemic control is associated with progressive deterioration in motor nerve conduction parameters. These studies suggest that the median nerve is particularly susceptible to chronic metabolic injury because of its superficial course and increased vulnerability to ischemic and metabolic changes. Early electrophysiological abnormalities detected in the median nerve may therefore serve as useful indicators of impending diabetic neuropathy.

Comparable changes were also observed in the ulnar nerve, where uncontrolled diabetic patients demonstrated significantly reduced conduction velocity and prolonged distal latency. The involvement of both median and ulnar nerves supports the concept that diabetic neuropathy is a diffuse and symmetrical process affecting multiple peripheral nerves simultaneously. Although clinical manifestations may be absent during the early stages, electrophysiological abnormalities can already be detected, emphasizing the importance of routine nerve conduction studies in high-risk diabetic patients. The statistically significant differences observed between the two groups indicate that deterioration of peripheral nerve function parallels poor metabolic control.

The findings of the present study are consistent with previous reports demonstrating reduced motor nerve conduction velocity and prolonged distal latency among patients with poorly controlled T2DM. Several authors have shown that higher glycated hemoglobin levels and longer duration of diabetes are associated with worsening electrophysiological parameters, supporting the role of chronic hyperglycemia in the development and progression of diabetic neuropathy. Early identification of these abnormalities provides an opportunity for timely intervention through strict glycemic control and lifestyle modification, thereby reducing the risk of irreversible nerve damage and subsequent disability.

A limitation of the present study is its hospital-based observational design, which may limit the generalizability of the findings to the wider diabetic population. Furthermore, only motor conduction parameters of the median and ulnar nerves were evaluated, whereas sensory nerve conduction studies and long-term follow-up could provide additional information regarding the progression of diabetic neuropathy. Nevertheless, the study highlights the clinical utility of nerve conduction studies as a reliable, non-invasive, and objective method for the early detection of peripheral nerve dysfunction in patients with T2DM. Overall, the findings emphasize that maintaining adequate glycemic control plays a crucial role in preserving peripheral nerve function and delaying the onset and progression of diabetic neuropathy.

Conclusion

The present study demonstrates that poor glycemic control in patients with Type 2 Diabetes Mellitus is associated with significant impairment of peripheral motor nerve function, as evidenced by reduced motor nerve conduction velocity and prolonged distal latency of the median and ulnar nerves. These electrophysiological abnormalities indicate the presence of early subclinical peripheral nerve involvement even in the absence of overt clinical symptoms. Motor nerve conduction studies provide a sensitive, objective, and non-invasive method for detecting diabetic neuropathy at an early stage, allowing timely intervention before irreversible nerve damage occurs. The findings emphasize the importance of maintaining optimal glycemic control to preserve peripheral nerve function and reduce the risk of progression to symptomatic diabetic neuropathy. Routine nerve conduction assessment in patients with Type 2 Diabetes Mellitus, particularly those with poor glycemic control, may facilitate early diagnosis, appropriate therapeutic management, and improved long-term neurological outcomes, thereby enhancing the overall quality of life of affected individuals.

Conflict of Interest

None

References

1. Lindsay KW, Bone I, Callander R. Neurology and Neurosurgery Illustrated. 3rd ed. Edinburgh: Churchill Livingstone; 1997. p. 525.
2. Partanen J, Niskanen L, Lehtinen J, Mervaala E, Siitonen O, Uusitupa M. Natural history of peripheral neuropathy in patients with non-insulin-dependent diabetes mellitus. *N Engl J Med*. 1995;333:89–94.
3. Levitt NS, Stansberry KB, Wychanek S, et al. Natural progression of autonomic neuropathy and autonomic function tests in IDDM. *Diabetes Care*. 1996;19:751–754.
4. Tkac I, Bril V. Glycaemic control is related to the electrophysiologic severity of diabetic peripheral sensorimotor polyneuropathy. *Diabetes Care*. 1998;21:1749–1752.
5. Chandalia HB, Krishnaswamy PR. Glycated hemoglobin. *Curr Sci*. 2002;83:1522–1532.
6. Perkins BA, Bril V. Diagnosis and management of diabetic neuropathy. *Curr Diab Rep*. 2002;2:495–500.
7. Sadikot SM, Nigam A, Das S, et al. Prevalence of diabetes in India study (PODIS). *Diabetes Res Clin Pract*. 2004;66:301–307.
8. Boulton AJ, Vinik AI, Arezzo JC, et al. Diabetic neuropathies: ADA statement. *Diabetes Care*. 2005;28:956–962.
9. Joshi SR, Parikh RM. India—diabetes capital of the world. *J Assoc Physicians India*. 2007;55:323–324.