RESEARCH PAPER

Median-Nerve Sensory Conduction Study In Diabetes Mellitus Type-2 in Neurologically Asymptomatic Patients in a Tertiary Care Hospital

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Abstract:

Background: Diabetic neuropathy is the most common yet often underdiagnosed complication of type-2 diabetes mellitus (T2DM). Nerve conduction studies (NCS) can identify early, subclinical neuropathic changes even before clinical symptoms become apparent. **Objective:** To evaluate median sensory nerve conduction parameters in asymptomatic patients with T2DM and compare them with non-diabetic controls, and to assess their association with disease duration and glycemic control. **Methods:** This case–control study included 60 participants (30 T2DM patients and 30 age- and sex-matched controls). Median sensory nerve conduction studies were performed using standard electrophysiological techniques. Amplitude, latency, and conduction velocity were assessed and correlated with the duration of diabetes and HbA1c levels. **Results:** Diabetic participants showed significantly prolonged sensory latency and markedly reduced conduction velocity at both finger–wrist and palm–wrist segments compared with controls ($p=<0.001$), while sensory-amplitude remained comparable. No significant correlations were observed between nerve conduction parameters & either duration of diabetes or HbA1c levels ($p>0.05$). **Conclusion:** Asymptomatic individuals with T2DM exhibit early demyelinating changes in median sensory nerves. Median sensory NCS is a sensitive tool for early identification of diabetic neuropathy, supporting its role in routine screening to enable timely preventive interventions.

Keywords: Type 2 Diabetes Mellitus; Diabetic-Neuropathy; Median-Nerve; SNC; Subclinical Neuropathy.

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Introduction: Diabetes mellitus (DM) is a prevalent chronic metabolic disease that leads to progressive pathophysiological alterations in multiple organ systems and continues to pose a major global public health challenge. Its rising prevalence is largely attributed to urbanization, unhealthy lifestyles, physical inactivity, and dietary changes [1]. Worldwide, the prevalence of diabetes rose from 108 million in 1980 to 422 million in 2014, with a more rapid increase observed in low- and middle-income countries. Diabetes is a major cause of blindness, kidney failure, cardiovascular disease, stroke, and lower-limb amputation, and it contributes substantially to premature mortality [2,3]. Despite a global decline in mortality from major non-communicable diseases between 2000 and 2019, diabetes-related deaths continue to pose a substantial burden[4,5]. India bears a disproportionately high burden, with an estimated 77 million adults with type 2 diabetes and nearly 25 million individuals with prediabetes[6]. Alarmingly, over half of affected individuals remain undiagnosed, increasing the risk of preventable complications[7]. Chronic hyperglycemia leads to microvascular and macrovascular damage, including diabetic neuropathy, which affects nearly 50% of patients over the disease course[8]. Diabetic neuropathy results from prolonged nerve injury due to metabolic and ischemic mechanisms, causing symptoms ranging from sensory loss to severe pain and disability[9,10]. Hyperglycemia is believed to increase the susceptibility of peripheral nerves, particularly the median nerve, to hypoxia and infarction, leading to mononeuropathies[11,12]. Although nerve dysfunction can be detected before the onset of overt diabetic polyneuropathy, gaps remain in elucidating the role of nerve conduction studies (NCS) for early detection, especially in asymptomatic type 2 diabetes[13]. This study aims to assess MNC velocity to identify initial neuropathic changes and examine its association with duration of diabetes, thereby contributing to improved early diagnosis and preventive strategies in diabetic neuropathy.

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Material and Methodology:

Study Design, Setting and Period: This case–control study was conducted in the Department of Physiology, School of Medical Sciences and Research (SMS&R), Sharda University, Greater Noida, from April 2024 to October 2025.

Study Population: A total of 60 participants were enrolled, including 30 patients with type 2 diabetes mellitus (T2DM) and 30 age- and sex-matched healthy controls recruited from tertiary care centers and the General Medicine OPD of Sharda Hospital, Greater Noida.

Inclusion and Exclusion Criteria: Participants aged 30–60 years with T2DM (HbA1c >7%) and without clinical evidence of neuropathy were included after obtaining written informed consent. Healthy non-diabetic individuals served as controls. Individuals with clinical neuropathy, coronary artery disease, cerebrovascular disease, other neurological disorders, or unwillingness to participate were excluded.

Sample Size: Sample size was calculated using OpenEpi Version 3 with 95% confidence interval and 80% power. After considering a 20% dropout rate, 30 participants were included in each group.

Data Collection and Blood Sampling: Demographic and clinical details were recorded using a structured case record form. Five milliliters of venous blood were collected aseptically for biochemical analysis, and unsuitable samples were excluded according to standard laboratory protocols.

Nerve Conduction Study: Median sensory nerve conduction studies were performed using standard surface electrode techniques. Sensory latency, amplitude, and conduction velocity were recorded following supramaximal stimulation.

Ethical Considerations: Ethical approval was obtained from the Institutional Ethics Research Committee, SMS&R, Sharda University. Written informed consent was obtained from all participants.

Statistical Analysis: Data were analyzed using SPSS version 22. Quantitative variables were expressed as mean ± SD. Student's t-test, Chi-square test, and Pearson correlation analysis were used where appropriate. A p-value <0.05 was considered statistically significant.

Results:

Table 1. Baseline demographic & metabolic profile (T2DM vs control):

Variable	Diabetic mean±SD	Control mean±SD	*p* value
Age(years)	53.83± 7.46	44.93± 8.80	0.0000
Height (cm)	164.87±5.35	160.87±7.29	0.019
Weight (kg)	79.53±8.11	71.73±13.12	0.008
BMI (kg/m²)	29.22±2.12	27.70±4.93	0.128
Fasting glucose (mg/dL)	134.14±25.79	89.73±4.93	0.000
Random glucose (mg/dL)	228.37±22.98	119.47±10.28	0.000
HbA1c (%)	7.39±0.30	5.50±0.28	0.000

Table 1 shows that diabetic participants were significantly older and had higher height and weight than controls ($p<0.05$). BMI was higher in diabetics but not statistically significant ($p=0.128$). Fasting glucose, random glucose, and HbA1c levels were significantly elevated in the diabetic group (all $p<0.001$), indicating poor glycemic control.

Table 2. Gender distribution by group:

Group	Female n (%)	Male n (%)
Diabetic	20 (66.7%)	10 (33.3%)
Non-diabetic	13 (43.3%)	17 (56.7%)

Table 2 shows that females were more common in the diabetic group, while males predominated in controls; however, the difference was not statistically significant ($\chi^2=2.42$, $p=0.12$), indicating comparable gender distribution between groups.

Table 3. Median sensory nerve conduction parameters (dominant hand):

Variable	Diabetic mean ±SD	Control mean ±SD	*p* value
Mean latency (ms)	3.43±0.35	3.11±0.27	0.00026
Mean amplitude (μV)	17.04±5.53	17.15±2.54	0.918
Mean conduction velocity (m/s)	38.97±3.98	55.10±2.90	3.5e-24

Table 3 shows significantly prolonged sensory latency and markedly reduced conduction velocity in diabetic participants compared to controls ($p<0.001$), indicating impaired nerve conduction. Sensory amplitude did not differ significantly ($p=0.918$), suggesting relative preservation of axonal integrity and early demyelinating neuropathic changes.

Table 4. Median sensory NCS (finger–wrist) by group:

Variable	Diabetic(n=30)	Non-diabetic(n=30)	t-statistic	*p*-value
Latency (ms)	3.487 ± 0.331	3.113 ± 0.252	4.930	0.0000

Amplitude (μV) (peak-to-peak or baseline-to-peak)	15.940 ± 6.910	17.000 ± 2.586	-0.787	0.4364
Conduction velocity (m/s)	38.967 ± 3.978	55.100 ± 2.905	-17.939	0.0000

Table 4 shows significantly prolonged latency and reduced conduction velocity in the finger–wrist segment among diabetic participants compared to controls ($p<0.001$), indicating delayed nerve conduction. Sensory amplitude did not differ significantly ($p=0.436$), suggesting preserved axonal function.

Table 5. Median sensory NCS (palm–wrist) by group:

Variable	Diabetic\n (mean ± SD)	Non-diabetic\n (mean ± SD)	t-statistic	*p* value
Latency (ms).1	3.375 ± 0.575 (n=30)	3.110 ± 0.312 (n=30)	2.220	0.0315
Amplitude (μV).1	18.130 ± 6.014 (n=30)	17.300 ± 2.575 (n=30)	0.695	0.4912
Conduction velocity (m/s).1	39.310 ± 3.752 (n=29)	54.767 ± 3.070 (n=30)	-17.284	0.0000

Table 5 shows significantly prolonged latency and reduced conduction velocity in the palm–wrist segment among diabetic participants compared to controls ($p<0.05$ and $p<0.001$, respectively). Sensory amplitude remained comparable ($p=0.491$), indicating preserved axonal integrity and diffuse slowing of nerve conduction characteristic of diabetic neuropathy.

Table 6. Correlation of diabetes duration with NCS parameters (diabetic group only):

Outcome variable	Pearson r	*p* value
Latency (ms)	-0.133	0.4848
Latency (ms).1	0.269	0.1501
Conduction velocity (m/s)	-0.096	0.6155
Conduction velocity (m/s).1	-0.259	0.1742

Table 6 shows no statistically significant correlation between the predictor variable and nerve conduction parameters ($p>0.05$). Latency and conduction velocity demonstrated weak, non-significant correlations, indicating no meaningful linear association.

Table 7. Correlation (Pearson r) between duration/HbA1c and conduction (T2DM only):

Predictor	Outcome	r	*p* value
Duration (years)	Mean latency (ms)	0.157	0.407
Duration (years)	Mean conduction velocity (m/s)	-0.096	0.615
Duration (years)	Mean amplitude (μV)	-0.136	0.475
HbA1c (%)	Mean latency (ms)	0.144	0.447
HbA1c (%)	Mean conduction velocity (m/s)	0.047	0.807
HbA1c (%)	Mean amplitude (μV)	-0.100	0.597

Table 7 presents the correlation analysis, which revealed no significant linear association between diabetes duration or glycemic control & median sensory nerve-conduction parameters. Duration of diabetes showed weak, non-significant correlations with sensory latency ($r=0.157$, $p=0.407$), conduction velocity($r=–0.096$, $p=0.615$), and amplitude($r=–0.136$, $p=0.475$). Similarly, HbA1c levels demonstrated very weak and non-significant correlations with latency($r=0.144$, $p=0.447$), conduction velocity($r=0.047$, $p=0.807$), and amplitude($r=–0.100$, $p=0.597$).

Discussion

The present study demonstrated significant median sensory nerve conduction abnormalities in asymptomatic patients with type 2 diabetes mellitus (T2DM). Diabetic participants showed prolonged sensory latency and significantly reduced conduction velocity compared to controls, while sensory amplitude remained relatively preserved. These findings indicate early electrophysiological impairment suggestive of subclinical diabetic neuropathy.

Diabetes mellitus continues to be a major global health challenge with steadily increasing incidence and mortality worldwide [2,3]. India carries a particularly high burden of T2DM, with a large proportion of patients remaining undiagnosed or inadequately controlled [7,8]. Chronic hyperglycemia contributes to microvascular and metabolic alterations leading to complications such as diabetic neuropathy [4,9]. Previous studies have reported that diabetic neuropathy may develop even before the appearance of overt clinical symptoms [10].

In the present study, diabetic participants demonstrated significantly prolonged sensory latency and markedly reduced conduction velocity. Similar findings were reported by Sepat and Wasnik [1], who observed reduced sensory nerve conduction velocity in median, ulnar, and radial nerves among T2DM patients. Garg et

al. [12] also reported significant slowing of median nerve conduction velocity in neurologically asymptomatic diabetic individuals, supporting the usefulness of nerve conduction studies in early detection of neuropathy.

The reduction in conduction velocity observed in our study likely reflects early demyelinating changes caused by chronic metabolic and ischemic injury to peripheral nerves [4]. Bertora et al. [16] similarly demonstrated subclinical neuropathy in diabetic patients through abnormalities in sensory and motor nerve conduction velocity distribution. These findings suggest that electrophysiological abnormalities may precede clinically detectable neuropathy.

In contrast, sensory amplitude in the present study did not differ significantly between diabetic and control groups, indicating relative preservation of axonal integrity. This suggests that demyelination may occur earlier than axonal degeneration during the progression of diabetic neuropathy.

The present study also demonstrated similar abnormalities in both finger–wrist and palm–wrist segments, indicating diffuse nerve involvement. Such findings further support the presence of generalized peripheral nerve dysfunction in diabetes rather than localized pathology.

No significant association was observed between nerve conduction parameters and duration of diabetes or HbA1c levels in our study. Although some studies have identified duration of diabetes and poor glycemic control as predictors of neuropathy severity [13–15], the lack of significant correlation in the present study may be related to the relatively small sample size and variability in disease progression among individuals.

Previous studies have emphasized the high prevalence of subclinical neuropathy among diabetic patients. Lu et al. [14] and Pfannkuche et al. [15] reported diabetic peripheral neuropathy as one of the most frequent complications of T2DM. Similarly, Govindasamy et al. [17] identified significant nerve conduction abnormalities in asymptomatic diabetic patients, highlighting the importance of electrophysiological screening for early diagnosis.

Conclusion: Patients with T2DM exhibit significant median sensory nerve conduction abnormalities even in the absence of clinical neuropathy. Prolonged sensory latency and reduced conduction velocity indicate early demyelinating changes, while preserved sensory amplitude suggests relative axonal integrity. No significant association was observed with diabetes duration or HbA1c levels. Median sensory nerve conduction studies are sensitive tools for early detection of subclinical diabetic neuropathy and may help enable timely preventive interventions.

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