RESEARCH PAPER

Evaluation of Upper Limb Peripheral Nerve Conduction Alterations in Patients with Type 2 Diabetes Mellitus and Their Association with Disease Duration and Glycaemic Status: A Cross-Sectional Observational Study

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ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is a major global health problem associated with several chronic microvascular complications, among which diabetic peripheral neuropathy (DPN) is one of the earliest and most disabling. Although lower-limb neuropathy has been extensively investigated, upper-limb nerve involvement often remains underdiagnosed because it is frequently asymptomatic during the initial stages. Electrophysiological assessment using nerve conduction studies (NCS) offers an objective method for detecting subclinical neuropathy before irreversible nerve damage occurs.

Materials and Methods: A hospital-based cross-sectional observational study was conducted among 64 patients with Type 2 Diabetes Mellitus aged 30–60 years attending a tertiary care teaching hospital. Detailed demographic and clinical data were collected. Fasting blood glucose (FBG), postprandial blood glucose (PPBG), and glycated haemoglobin (HbA1c) were estimated using standardized laboratory techniques. Motor and sensory nerve conduction studies of the median and ulnar nerves were performed using computerized electromyography equipment under standardized laboratory conditions. Correlation between nerve conduction parameters and clinical variables was analysed using Pearson's correlation coefficient.

Results: The study population had a mean age of 48.52 ± 6.84 years and a mean diabetes duration of 8.94 ± 3.42 years. Mean HbA1c was 7.92 ± 0.58%, indicating suboptimal glycaemic control. Median sensory conduction velocity demonstrated the greatest reduction among all evaluated parameters and showed a significant negative correlation with disease duration ($r = –0.349$, $p = 0.005$). Median motor conduction velocity also declined significantly with increasing duration of diabetes ($r = –0.258$, $p = 0.041$). Ulnar nerve parameters were comparatively preserved, and no consistent association was observed between HbA1c and electrophysiological findings.

Conclusion: Upper-limb nerve conduction abnormalities are frequently present in patients with Type 2 Diabetes Mellitus even before clinical manifestations of neuropathy become apparent. Sensory fibres of the median nerve appear to be affected earlier than motor fibres. Disease duration demonstrated a stronger relationship with electrophysiological deterioration than glycaemic indices, emphasizing the importance of periodic nerve conduction studies for early diagnosis of diabetic neuropathy.

Keywords: Type 2 Diabetes Mellitus, Diabetic Peripheral Neuropathy, Median Nerve, Ulnar Nerve, Nerve Conduction Study, Glycaemic Control.

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INTRODUCTION

Diabetes mellitus (DM) is one of the most prevalent chronic metabolic disorders worldwide and has emerged as a major public health concern owing to its rapidly increasing incidence, long-term complications, and substantial socioeconomic burden. Type 2 Diabetes Mellitus (T2DM), which accounts for approximately 90–95% of all diabetes cases, is characterized by insulin resistance, progressive pancreatic β-cell dysfunction, and persistent hyperglycaemia. Chronic elevation of blood glucose initiates multiple metabolic and vascular abnormalities that ultimately damage various organ systems, resulting in microvascular complications such as diabetic retinopathy, nephropathy, and peripheral

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neuropathy, as well as macrovascular cardiovascular disease. These complications significantly reduce quality of life and contribute to increased disability and premature mortality worldwide [1,2].

The global burden of diabetes continues to rise at an alarming rate. According to the **International Diabetes Federation (IDF) Diabetes Atlas, 11th Edition**, approximately **589 million adults aged 20–79 years** were living with diabetes in 2025, representing nearly one in every nine adults globally. This number is projected to increase to **853 million by 2050**, reflecting the continuing impact of urbanization, unhealthy dietary habits, obesity, physical inactivity, and population ageing [3]. Diabetes remains one of the leading causes of cardiovascular disease, chronic kidney disease, blindness, lower-limb amputation, and premature death, making early diagnosis and prevention of complications an important public health priority [4].

India has witnessed one of the fastest increases in diabetes prevalence over the past two decades and currently ranks among the countries with the largest diabetic population worldwide. Recent estimates indicate that nearly **90 million Indian adults** are living with diabetes, while an even larger proportion remains at high risk because of impaired glucose regulation and metabolic syndrome. Rapid socioeconomic transition, increasing obesity, sedentary lifestyles, and changing nutritional patterns have substantially contributed to this growing epidemic. Furthermore, delayed diagnosis and inadequate glycaemic control continue to increase the burden of chronic diabetic complications across both urban and rural populations [3,5].

Diabetic peripheral neuropathy (DPN) is one of the most frequent and disabling chronic complications of T2DM, affecting approximately **30–50%** of patients during the course of their illness. The condition develops through a complex interplay of persistent hyperglycaemia, oxidative stress, activation of the polyol pathway, accumulation of advanced glycation end-products (AGEs), mitochondrial dysfunction, endothelial injury, chronic inflammation, and impaired endoneurial blood flow. These pathological mechanisms result in segmental demyelination, axonal degeneration, impaired nerve regeneration, and progressive deterioration of peripheral nerve function. Clinically, patients may experience numbness, paraesthesia, burning sensations, muscle weakness, neuropathic pain, impaired vibration perception, or autonomic dysfunction. However, electrophysiological abnormalities frequently precede clinical symptoms, making early diagnosis challenging without objective investigations [6,7].

Nerve conduction studies (NCS) are regarded as the gold standard electrophysiological technique for evaluating peripheral nerve function and identifying subclinical diabetic neuropathy. Measurement of distal motor latency, compound muscle action potential amplitude, sensory nerve action potential amplitude, and nerve conduction velocity provides quantitative evidence of demyelination and axonal injury. Numerous studies have demonstrated that reduction in nerve conduction velocity represents one of the earliest detectable abnormalities in diabetic neuropathy and may occur long before the appearance of overt neurological manifestations. Consequently, NCS has become an important diagnostic and prognostic tool for evaluating the severity and progression of diabetic peripheral neuropathy [8].

Although diabetic neuropathy has been extensively investigated, the majority of published studies have primarily focused on lower-limb nerves, particularly the sural, tibial, and peroneal nerves because of their direct association with diabetic foot complications. Comparatively limited literature has comprehensively evaluated both motor and sensory conduction characteristics of the **median and ulnar nerves** in the upper limbs. Moreover, existing studies have reported inconsistent findings regarding the relative influence of disease duration and glycaemic control on upper-limb nerve conduction abnormalities. While several investigators have observed a significant association between prolonged duration of diabetes and progressive slowing of nerve conduction velocity, others have suggested that long-term cumulative metabolic exposure rather than isolated HbA1c levels may be a more reliable predictor of peripheral nerve dysfunction [9,10].

Considering the increasing prevalence of diabetes and the need for early recognition of diabetic neuropathy before irreversible nerve damage develops, the present study was undertaken to evaluate motor and sensory nerve conduction parameters of the median and ulnar nerves among patients with Type 2 Diabetes Mellitus and to determine their relationship with disease duration and glycaemic profile. Early identification of electrophysiological abnormalities may facilitate timely therapeutic intervention and contribute to reducing the long-term neurological complications associated with diabetes.

MATERIALS AND METHODS

This hospital-based observational, cross-sectional study was conducted in the **Departments of Physiology and Medicine at Dr. Bhimrao Ramji Ambedkar Government Medical College, Kannauj, Uttar Pradesh, India**, after obtaining approval from the Institutional Ethics Committee. The study was carried out over a period of **12 months from January 2025 to December 2025**. A total of **64 patients** diagnosed with **Type 2 Diabetes Mellitus (T2DM)** were enrolled using a **non-probability consecutive sampling technique** after obtaining written informed consent. Participants of both sexes, aged between **30 and 60 years**, who fulfilled the **American Diabetes Association (ADA) diagnostic criteria** for T2DM and had a disease duration of at least one year were included in the study. Patients with Type 1 Diabetes Mellitus, gestational diabetes mellitus, peripheral neuropathy due to causes other than diabetes, chronic alcohol consumption, current smoking or tobacco use,

vitamin B12 deficiency, chronic kidney disease, chronic liver disease, thyroid dysfunction, autoimmune disorders, peripheral nerve entrapment syndromes, cervical radiculopathy, previous upper-limb trauma or surgery, neuromuscular disorders, cerebrovascular accident, use of medications known to interfere with nerve conduction, or those unwilling to participate were excluded to minimize potential confounding factors.

Following enrolment, each participant underwent a detailed clinical evaluation using a predesigned case record form. Demographic characteristics, duration of diabetes, treatment history, associated comorbidities, medication history, and clinical examination findings were documented. Anthropometric measurements including **height, weight, body mass index (BMI), and blood pressure** were recorded using standardized techniques. After an overnight fast of **8–10 hours**, venous blood samples were collected under aseptic precautions for estimation of **fasting blood glucose (FBG), postprandial blood glucose (PPBG), and glycated haemoglobin (HbA1c)**. FBG and PPBG were analysed by the **glucose oxidase–peroxidase (GOD–POD) enzymatic method** using an automated clinical chemistry analyser, while HbA1c was estimated by **High-Performance Liquid Chromatography (HPLC)** standardized according to the **National Glycohemoglobin Standardization Program (NGSP)**.

Motor and sensory nerve conduction studies were performed in the Neurophysiology Laboratory using a **computerized Clarity NCV/EMG four-channel system** under standardized laboratory conditions. The ambient room temperature was maintained between **24°C and 26°C**, and skin temperature was maintained above **32°C** to minimize temperature-related variation in nerve conduction velocity. The **dominant upper limb** was examined in all participants. Motor conduction studies of the **median and ulnar nerves** included measurement of **distal motor latency, compound muscle action potential (CMAP) amplitude, and motor nerve conduction velocity**, whereas sensory conduction studies included assessment of **distal sensory latency, sensory nerve action potential (SNAP) amplitude, and sensory nerve conduction velocity** of the median and ulnar nerves. All electrophysiological recordings were obtained according to the recommendations of the **American Association of Neuromuscular & Electrodiagnostic Medicine (AANEM)** using standardized stimulation and recording techniques.

The primary outcome measure was the assessment of motor and sensory nerve conduction abnormalities of the median and ulnar nerves. Secondary outcome measures included evaluation of the correlation between disease duration and nerve conduction parameters, assessment of the relationship between glycaemic indices (FBG, PPBG, and HbA1c) and electrophysiological findings, and identification of the pattern of upper-limb motor and sensory nerve involvement among patients with T2DM.

Statistical Analysis

Data were entered into **Microsoft Excel** and analysed using **IBM Statistical Package for the Social Sciences (IBM SPSS Statistics), Version 29.0 (IBM Corp., Armonk, NY, USA)**. Confidentiality of participant information was strictly maintained, and all study procedures were conducted in accordance with the ethical principles outlined in the **Declaration of Helsinki (2013 revision)**. Continuous variables were expressed as **mean ± standard deviation (SD)**, whereas categorical variables were summarized as **frequencies and percentages**. The normality of continuous variables was assessed using the **Shapiro–Wilk test**. Differences between groups were analysed using the **Independent Student's t-test** for normally distributed variables and the **Mann–Whitney U test** where appropriate. Associations between categorical variables were evaluated using the **Chi-square test** or **Fisher's exact test**. **Pearson's correlation coefficient** was used to determine the relationship between nerve conduction parameters and duration of diabetes, fasting blood glucose, postprandial blood glucose, and HbA1c levels. A **two-tailed p-value <0.05** was considered statistically significant, whereas **p <0.001** was considered highly statistically significant.

RESULTS

Table 1. Demographic, Clinical, Biochemical and Nerve Conduction Characteristics of Study Participants (N = 64)

Parameter	Mean ± SD
Age (years)	**48.52 ± 6.84**
Duration of T2DM (years)	**8.94 ± 3.42**
Fasting Blood Glucose (mg/dL)	**133.84 ± 15.27**
Postprandial Blood Glucose (mg/dL)	**189.42 ± 22.85**
HbA1c (%)	**7.92 ± 0.58**
Median Motor Distal Latency (ms)	**3.88 ± 0.36**
Median Motor Amplitude (mV)	**8.73 ± 1.84**
Median Motor Conduction Velocity (m/s)	**58.84 ± 4.16**
Median Sensory Distal Latency (ms)	**3.69 ± 0.21**
Median Sensory Amplitude (µV)	**53.41 ± 18.72**
Median Sensory Conduction Velocity (m/s)	**46.13 ± 4.18**
Ulnar Motor Distal Latency (ms)	**3.33 ± 0.37**
Ulnar Motor Amplitude (mV)	**7.36 ± 1.74**

Ulnar Motor Conduction Velocity (m/s)	**63.08 ± 4.08**
Ulnar Sensory Distal Latency (ms)	**3.18 ± 0.30**
Ulnar Sensory Amplitude (μV)	**48.66 ± 17.95**
Ulnar Sensory Conduction Velocity (m/s)	**50.28 ± 4.27**

The study included **64 patients** with Type 2 Diabetes Mellitus having a mean age of **48.52 ± 6.84 years** and an average disease duration of **8.94 ± 3.42 years**. Mean fasting blood glucose, postprandial blood glucose and HbA1c indicated generally poor glycaemic control. Electrophysiological assessment demonstrated greater impairment of **median sensory nerve conduction velocity**, whereas ulnar nerve conduction parameters remained comparatively preserved.

Table 2. Distribution of Demographic and Clinical Characteristics (N = 64)

Parameter	Frequency (n)	Percentage (%)
Sex		
Male	36	56.25
Female	28	43.75
HbA1c Category		
Good Control (<7%)	8	12.50
Poor Control (≥7%)	56	87.50
Duration of Diabetes		
<5 years	12	18.75
5–10 years	29	45.31
>10 years	23	35.94
Total	**64**	**100.00**

Male participants constituted **56.25%** of the study population. Poor glycaemic control (HbA1c ≥7%) was observed in **87.50%** of patients. Nearly half of the participants had diabetes for **5–10 years**, while more than one-third had disease duration exceeding ten years.

Table 3. Median Nerve Motor and Sensory Conduction Parameters

Parameter	Mean ± SD	Minimum	Maximum
Motor Distal Latency (ms)	**3.88 ± 0.36**	3.18	4.55
Motor Amplitude (mV)	**8.73 ± 1.84**	4.92	12.08
Motor Conduction Velocity (m/s)	**58.84 ± 4.16**	49.84	65.22
Sensory Distal Latency (ms)	**3.69 ± 0.21**	3.14	4.08
Sensory Amplitude (μV)	**53.41 ± 18.72**	24.12	90.42
Sensory Conduction Velocity (m/s)	**46.13 ± 4.18**	38.46	53.12

Median nerve evaluation revealed mild prolongation of distal latency with reduction in conduction velocity, particularly affecting sensory fibres. Sensory conduction velocity demonstrated the greatest electrophysiological abnormality, suggesting early sensory nerve involvement in diabetic peripheral neuropathy.

Table 4. Ulnar Nerve Motor and Sensory Conduction Parameters

Parameter	Mean ± SD	Minimum	Maximum
Motor Distal Latency (ms)	**3.33 ± 0.37**	2.68	3.94
Motor Amplitude (mV)	**7.36 ± 1.74**	3.82	10.14
Motor Conduction Velocity (m/s)	**63.08 ± 4.08**	54.36	70.28
Sensory Distal Latency (ms)	**3.18 ± 0.30**	2.48	3.82
Sensory Amplitude (μV)	**48.66 ± 17.95**	14.72	79.84
Sensory Conduction Velocity (m/s)	**50.28 ± 4.27**	42.38	58.26

Compared with the median nerve, ulnar nerve conduction parameters were relatively preserved. Motor conduction velocity remained within near-normal limits, while only mild reduction in sensory conduction velocity was observed.

Table 5. Correlation Between Duration of Type 2 Diabetes Mellitus and Median Nerve Conduction Parameters

Parameter	r value	p value
Median Motor Distal Latency	**0.281**	**0.028***
Median Motor Amplitude	**−0.142**	0.263
Median Motor Conduction Velocity	**−0.258**	**0.041***
Median Sensory Distal Latency	0.066	0.601
Median Sensory Amplitude	−0.081	0.523
Median Sensory Conduction Velocity	**−0.349**	**0.005**

* p <0.05 statistically significant

Disease duration demonstrated a significant positive correlation with median motor distal latency and a significant negative correlation with both motor and sensory conduction velocities. Median sensory conduction velocity exhibited the strongest association with duration of diabetes, indicating progressive sensory nerve dysfunction with increasing disease chronicity.

Table 6. Correlation Between Duration of Type 2 Diabetes Mellitus and Ulnar Nerve Conduction Parameters

Parameter	r value	p value
Ulnar Motor Distal Latency	0.084	0.512
Ulnar Motor Amplitude	−0.153	0.228
Ulnar Motor Conduction Velocity	−0.171	0.181
Ulnar Sensory Distal Latency	**0.248**	**0.047***
Ulnar Sensory Amplitude	−0.098	0.441
Ulnar Sensory Conduction Velocity	−0.137	0.282

* p <0.05 statistically significant

Only ulnar sensory distal latency showed a statistically significant association with disease duration. Other motor and sensory parameters demonstrated weak, non-significant correlations, suggesting that the ulnar nerve remains relatively resistant to electrophysiological deterioration compared with the median nerve.

The present study demonstrated that upper-limb peripheral neuropathy was common among patients with Type 2 Diabetes Mellitus despite the absence of overt neurological symptoms. Electrophysiological abnormalities predominantly affected the **median sensory nerve**, whereas the ulnar nerve remained comparatively preserved. Disease duration showed stronger correlations with nerve conduction abnormalities than glycaemic indices, indicating that chronic metabolic exposure may play a more important role in the progression of diabetic neuropathy than isolated measurements of glycaemic control.

DISCUSSION

The present study included 64 patients with Type 2 Diabetes Mellitus, with a mean age of **48.52 ± 6.84 years**, mean disease duration of **8.94 ± 3.42 years**, fasting blood glucose of **133.84 ± 15.27 mg/dL**, postprandial blood glucose of **189.42 ± 22.85 mg/dL**, and HbA1c of **7.92 ± 0.58%**, indicating generally suboptimal glycaemic control. Electrophysiological evaluation demonstrated greater impairment of median sensory nerve conduction compared with motor conduction, whereas ulnar nerve conduction parameters were relatively preserved. The mean median sensory conduction velocity (**46.13 ± 4.18 m/s**) was considerably lower than the corresponding median motor conduction velocity (**58.84 ± 4.16 m/s**) as well as the ulnar motor (**63.08 ± 4.08 m/s**) and sensory (**50.28 ± 4.27 m/s**) conduction velocities, suggesting preferential involvement of median sensory fibres during the early stages of diabetic neuropathy. These findings are in agreement with **Hamid et al. (2021)**, who reported significant deterioration of nerve conduction parameters with increasing disease duration in patients with T2DM despite studying an older population (mean age **57.71 ± 12.20 years**) with longer disease duration (**14.67 ± 9.24 years**) [11]. Likewise, **Zaidi and Samad (2025)** evaluated 139 diabetic patients with a mean age of **62.20 ± 12.03 years** and reported nerve conduction slowing in **54.7%** of participants, with significant association between nerve dysfunction and diabetes duration (**p = 0.019**) [12]. Collectively, these observations suggest that electrophysiological abnormalities may develop relatively early during the course of diabetes and progressively worsen with prolonged metabolic exposure.

The present study demonstrated a slight male predominance, with **36 (56.25%) males** and **28 (43.75%) females**. Poor glycaemic control was observed in the majority of participants, as **56 patients (87.50%)** had HbA1c ≥7%, while only **8 (12.50%)** achieved good glycaemic control. Nearly half of the study population (**45.31%**) had diabetes for **5–10 years**, whereas **35.94%** had disease duration exceeding ten years, indicating a predominance of moderately longstanding diabetes. Similar demographic characteristics were reported by **Zaidi and Samad (2025)**, who observed male predominance (**60.4%**) together with poor glycaemic control in **75.5%** of patients and clinically detectable neuropathy in **80.6%** [12]. Furthermore, **Nisar et al. (2015)** demonstrated that increasing duration of diabetes and higher HbA1c levels were independently associated with diabetic peripheral neuropathy [13]. In addition, **Nozawa et al. (2022)** reported that long-term mean HbA1c rather than isolated HbA1c measurements showed a stronger association with the development and progression of diabetic neuropathy [14]. These findings support the observation that prolonged diabetes and persistent hyperglycaemia considerably increase the risk of peripheral nerve dysfunction.

The present study demonstrated mild prolongation of median motor distal latency (**3.88 ± 0.36 ms**) together with reduced motor conduction velocity (**58.84 ± 4.16 m/s**) and more pronounced slowing of median sensory conduction velocity (**46.13 ± 4.18 m/s**). Despite reduced conduction velocity, sensory amplitude remained relatively preserved (**53.41 ± 18.72 µV**), suggesting predominantly demyelinating rather than advanced axonal changes. When compared with healthy reference values reported by **Singh et al. (2017)**, who documented motor distal latencies of **2.90 ± 0.16 ms** in males and **2.60 ± 0.43 ms** in females together with motor conduction velocities exceeding **59 m/s**, the present findings indicate significant electrophysiological slowing among diabetic patients [15].

Similarly, **Sepat and Wasnik (2020)** observed significant sensory nerve involvement involving both the median and ulnar nerves in patients with Type 2 Diabetes Mellitus and concluded that sensory abnormalities frequently precede severe motor dysfunction [8]. Therefore, the present findings reinforce the concept that sensory conduction velocity represents one of the earliest electrophysiological markers of diabetic peripheral neuropathy.

Compared with the median nerve, the ulnar nerve demonstrated relatively preserved electrophysiological function. Mean ulnar motor conduction velocity (**63.08 ± 4.08 m/s**) remained higher than median motor conduction velocity, while ulnar sensory conduction velocity (**50.28 ± 4.27 m/s**) also exceeded the corresponding median sensory value. Healthy reference data reported by **Owolabi et al. (2022)** showed a mean ulnar sensory conduction velocity of **55.22 ± 5.67 m/s**, indicating that although the present values were lower than those observed in healthy individuals, they remained within the lower end of the reported physiological range [16]. Nevertheless, relative preservation should not be interpreted as absence of diabetic involvement. **Gündüz et al. (2020)** demonstrated electrophysiological evidence of ulnar neuropathy in **36 of 82** patients with T2DM despite clinical suspicion in only three individuals, highlighting the high prevalence of subclinical ulnar nerve dysfunction [17]. The present findings therefore suggest that the ulnar nerve is affected less severely than the median nerve but may still undergo gradual electrophysiological deterioration with disease progression.

Duration of diabetes demonstrated a significant positive correlation with median motor distal latency (**r = 0.281, p = 0.028**) and significant negative correlations with median motor conduction velocity (**r = −0.258, p = 0.041**) and median sensory conduction velocity (**r = −0.349, p = 0.005**). In contrast, median motor amplitude, sensory distal latency and sensory amplitude showed no statistically significant association with disease duration. The strongest correlation observed for median sensory conduction velocity suggests that sensory fibres may be more susceptible to cumulative metabolic injury than motor fibres. Similar findings were reported by **Hamid et al. (2021)**, who demonstrated progressive deterioration of nerve conduction parameters with increasing duration of diabetes [11]. Likewise, **Zaidi and Samad (2025)** found a significant association between diabetes duration and nerve conduction slowing (**p = 0.019**) [12]. The preservation of nerve amplitudes despite slowing of conduction velocity further supports the hypothesis that demyelination precedes substantial axonal degeneration during the early stages of diabetic peripheral neuropathy.

Among the evaluated ulnar nerve parameters, only sensory distal latency showed a statistically significant positive correlation with disease duration (**r = 0.248, p = 0.047**), whereas motor distal latency, motor amplitude, motor conduction velocity, sensory amplitude and sensory conduction velocity did not demonstrate significant associations. These findings indicate that subtle sensory abnormalities may develop before clinically important motor involvement becomes evident. Comparable observations were reported by **Sepat and Wasnik (2020)**, who identified sensory abnormalities involving both the median and ulnar nerves, although median nerve involvement was considerably more pronounced [8]. Similarly, **Gündüz et al. (2020)** reported frequent electrophysiological evidence of clinically silent ulnar neuropathy among patients with T2DM [17]. Therefore, the present findings suggest that although the ulnar nerve remains relatively resistant to diabetic injury compared with the median nerve, progressive sensory dysfunction may still occur with increasing disease duration.

LIMITATIONS

The cross-sectional design prevents determination of temporal or causal relationships. The sample was drawn from a single tertiary-care institution and may not represent the wider diabetic population. A healthy control group was not included; therefore, nerve conduction values could not be directly compared with age- and sex-matched non-diabetic individuals. Only the dominant upper limb was evaluated, and lower-limb nerves were not assessed. A single HbA1c measurement may not accurately reflect long-term glycaemic exposure or variability. Potential confounders such as BMI, dyslipidaemia, hypertension, renal function and medication use were not incorporated into multivariable analysis. Small-fibre neuropathy cannot be excluded because conventional NCS predominantly evaluates large myelinated fibres.

CONCLUSION

Patients with Type 2 Diabetes Mellitus demonstrated measurable upper-limb nerve conduction abnormalities characterized by predominant sensory dysfunction and greater involvement of the median nerve than the ulnar nerve. Median sensory conduction velocity was the most sensitive electrophysiological parameter and showed the strongest deterioration with increasing disease duration. Relative preservation of response amplitudes suggests that conduction slowing and demyelinating changes may precede extensive axonal loss.

Disease duration was more consistently associated with electrophysiological abnormalities than a single HbA1c measurement. However, evidence from longitudinal studies indicates that long-term mean HbA1c and glycaemic variability remain important contributors to neuropathy. Upper-limb nerve conduction studies may therefore assist in detecting subclinical diabetic neuropathy, particularly among patients with longstanding diabetes or persistently inadequate metabolic control.

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