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Impact of Type 2 Diabetes Mellitus Duration on Sural Nerve Conduction Velocity: A Cross-Sectional Study

Lavkush Maurya[1], Shraddha Singh[1], S C Chaudhary[1], Sudipti Yadav[1], Pradeep Kumar Chauhan[1], Pratibha Kushwaha[1]

[1]Department of Physiology, King George's Medical University, Lucknow, India.

Corresponding Author: Dr. Lavkush Maurya

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ABSTRACT

Objective: To evaluate the nerve conduction velocity (NCV) of the sural nerve in patients with Type 2 Diabetes Mellitus (T2DM) and compare it with healthy controls, focusing on the impact of the disease's duration on NCV.
Methods: This cross-sectional study included 90 T2DM patients divided into two groups based on disease duration (<5 years and >10 years) and 45 healthy controls. Sural nerve NCV was measured using an Octopus NCV/EMG/EP machine. Statistical analysis was performed using SPSS.
Results: The NCV of the sural nerve was significantly lower in T2DM patients compared to controls ($p<0.001$). NCV decreased significantly with increasing duration of diabetes, showing a reduction of approximately 1.80 m/s per year for the left sural nerve and 1.97 m/s per year for the right sural nerve.
Conclusions: Prolonged T2DM is associated with a significant reduction in sural nerve conduction velocity, indicating progressive nerve damage. Early diagnosis and management are crucial to prevent severe neuropathic complications.

Keywords: Nerve Conduction Velocity, Sural Nerve, Type 2 Diabetes Mellitus, Diabetic Neuropathy

INTRODUCTION

Diabetes mellitus, particularly Type 2 diabetes, is a global health concern with significant morbidity and mortality. As of the latest statistics, it affects millions worldwide, imposing a substantial burden on healthcare systems[1]. Among the myriad complications associated with Type 2 diabetes, diabetic neuropathy stands out due to its prevalence and impact on quality of life[2, 3]. Diabetic neuropathy manifests in various forms, with peripheral neuropathy being the most common, affecting the sensory, motor, and autonomic nerves[4].

Nerve conduction studies have emerged as crucial diagnostic tools in assessing diabetic neuropathy[5]. They provide valuable insights into nerve function, particularly measuring the velocity and amplitude of nerve impulses. Among these, the sural nerve—a purely sensory nerve in the lower limb—is frequently studied due to its accessibility and the wealth of information it offers about peripheral nerve health[6].

The velocity at which nerves conduct electrical signals, known as nerve conduction velocity (NCV), is a key indicator of nerve function[7]. In the context of diabetes, elevated

blood sugar levels can lead to biochemical changes in nerves, ultimately slowing down their conduction velocity. This slowdown can be quantitatively assessed through nerve conduction studies, providing a functional measure of nerve impairment[8, 9].

Given the high prevalence of Type 2 diabetes and its potential to cause severe neuropathic complications, there is a pressing need to understand the specific changes in nerve conduction among these patients[10, 11]. This clinical research study aims to find the link between the nerve conduction velocity of the sural nerve in Type 2 diabetic patients, exploring how this parameter varies and what it signifies about the underlying neuropathic condition. By focusing on this aspect, the study hopes to contribute to better diagnostic strategies and potentially pave the way for targeted therapies that could mitigate the progression of neuropathy in diabetic populations[12].

MATERIALS & METHODS

Study Design: This research was conducted using a cross-sectional analytical study design to assess and compare the nerve conduction characteristics among individuals with varying durations of Type 2 Diabetes Mellitus (T2DM) and a healthy control group. The study aimed to identify potential differences in nerve conduction velocity (NCV) as a function of the chronicity of diabetes. The study was conducted within the Physiology department of King George's Medical University, Lucknow, over a one-year period.

Participants: The inclusion criteria for the study comprised Group 1 and Group 2 (T2DM Patients), who were diagnosed with Type 2 Diabetes Mellitus as per the American Diabetes Association criteria. Group 1 included patients with a diagnosis of T2DM for less than 5 years, while Group 2 included those with a diagnosis for more than 10 years. Both groups consisted of individuals aged between 20 to 60 years who were willing to provide informed consent and adhere to the study protocols. Group 3 (Control Group) consisted of non-diabetic individuals confirmed by glycated haemoglobin (HbA1c) levels within the normal range. The control group members were age and sex-matched to the T2DM groups and had no history of metabolic, chronic neurological, or musculoskeletal diseases affecting the nervous system. The exclusion criteria included individuals with other types of diabetes, neurological disorders unrelated to diabetes, systemic diseases affecting nerve conduction, or those taking medications known to affect nerve conduction other than those prescribed for diabetes management. Additionally, individuals who were unable or unwilling to comply with the study procedures or provide informed consent were excluded.

Ethical Approval Ethical approval was obtained from the Institutional Ethics Committee of King George's Medical University, Lucknow. Participants were assured of their right to withdraw from the study at any time without affecting their standard of care. The study was conducted in conformance with the principles of the Declaration of Helsinki.

Data Collection Methods: For data collection, the study began with preliminary screening, which included a review of medical histories, physical examinations, and baseline laboratory tests to ensure participants met the study criteria. Baseline data acquisition involved recording demographic and clinical data, such as age, gender, weight, height, and Body Mass Index (BMI). For diabetic subjects, additional information regarding the duration of diabetes, treatment modalities, and any complications or comorbid conditions was also collected.

The measurement of nerve conduction velocity (NCV) in this study was conducted using the Octopus NCV/EMG/EP machine. For electrode placement and skin preparation, the skin overlying the sural nerve was prepared to minimize impedance, and recording electrodes were positioned at specific anatomical landmarks corresponding to the sural nerve pathway. During nerve stimulation, consistent

stimulus intensity sufficient to elicit a maximal response without causing discomfort was used, with the ground electrode placed between the stimulating and recording electrodes to reduce external noise and artifacts. Throughout the recording procedure, participants remained still, and parameters such as stimulus duration, frequency, and filtering settings were standardized. The parameters measured included latency, which is the time between the onset of the stimulus and the onset of the response (ms), amplitude, which is the peak-to-peak measurement of the response (µV), and conduction velocity, which was calculated by dividing the distance between the stimulating and recording electrodes by the latency of the response (m/s).

RESULT

The study included 135 participants: 45 controls (Group 3), 45 T2DM patients with less than 5 years of diabetes (Group 1), and 45 T2DM patients with more than 10 years of diabetes (Group 2). The average age of the participants was 46.36 years, and the Body Mass Index (BMI) averaged 26.64.

A significant reduction in sural nerve conduction velocity (NCV) was observed among T2DM patients compared to healthy controls (Table 1). The NCV in T2DM patients was markedly lower, with a progressive decline observed as the duration of diabetes increased. Linear regression analysis revealed a negative correlation between the duration of diabetes and the NCV of both the left and right sural nerves. Specifically, the NCV decreased by approximately 1.8004 m/s per year for the left sural nerve and 1.9692 m/s per year for the right sural nerve (Table 2, Figure 1), indicating substantial nerve impairment with prolonged diabetes duration.

Statistical analysis using ANOVA demonstrated significant differences in NCV between the three study groups for both the left and right sural nerves (Table 1). The mean NCV was significantly lower in both diabetic groups compared to the control group, with the most pronounced deficits observed in patients with more than 10 years of diabetes.

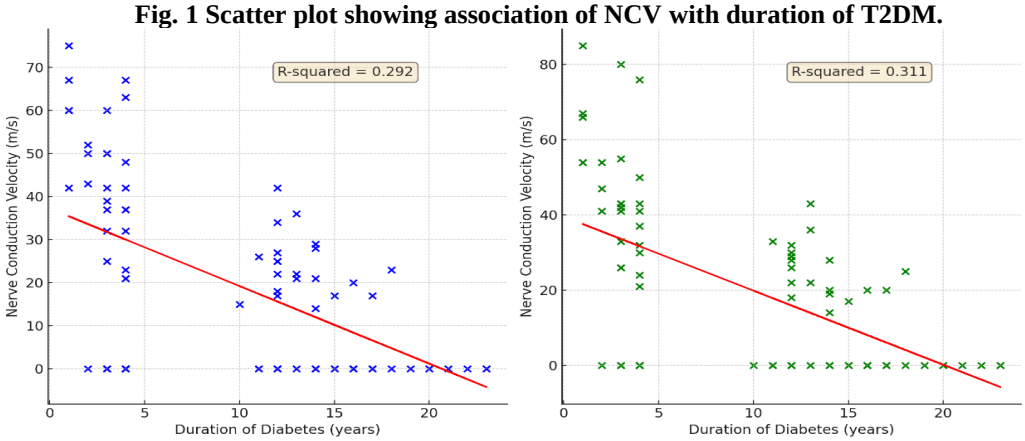
Table 1: ANOVA test results for NCV between all groups

Parameter	df	Mean Square	F	p-value
NCV (m/s) - L	2	13495.980	47.304	<0.001
NCV (m/s) - R	2	16466.696	51.672	<0.001

Table 2: Results of linear regression analysis to find out association between NCV of sural nerve with duration of T2DM

Nerve	Coefficient (Duration of Diabetes)	Standard Error	p-value	Intercept	R-squared
Left Sural	-1.8004	0.311	<0.001	37.2182	0.292
Right Sural	-1.9692	0.326	<0.001	39.5515	0.311

Fig. 1 Scatter plot showing association of NCV with duration of T2DM.



Further comparative analysis between Group 1 (T2DM < 5 years) and Group 2 (T2DM > 10 years) showed significant reductions in NCV, with the latter group exhibiting greater impairments (Table 3). In comparison with the control group, both diabetic groups had significantly lower NCV, but the decline was more severe in those with longer disease duration (Tables 4 and 5, Fig 2).

Table 3: Comparison of Mean NCV Between Patients with T2DM Less Than 5 Years and More Than 10 Years

Nerve	Group	Mean NCV (m/s)	SD NCV (m/s)	T-Statistic	p-value	Interpretation
Left Sural	1	52.48	11.27	6.447	<0.01	Significant difference, higher in Group 1
Left Sural	2	33.94	9.54			

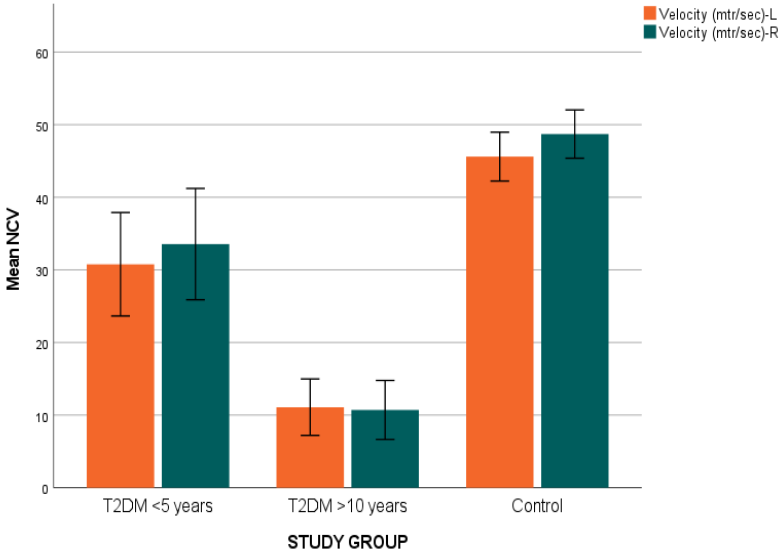
Table 4: Comparison of means between group 1 (T2DM < 5 years) and Group 3 (Control) for NCV Amplitude and Latency

Parameter	t	df	p-value	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference
Latency (ms) - L	-2.155	88	0.034	-0.6156	0.2856	-1.1831 to -0.0480
Amplitude - L	-0.951	88	0.344	-1.1956	1.2574	-3.6945 to 1.3033
Velocity (m/s) - L	-3.791	88	<0.001	-14.8333	3.9123	-22.6082 to -7.0584

Table 5: Comparison of means between group 2 (T2DM >10 years) and Group 3 (Control) for NCV Amplitude and Latency

Parameter	t	df	p-value	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference
Latency (ms) - L	-0.639	88	0.525	-0.2911	0.4559	-1.1971 to 0.6149
Amplitude - L	-7.249	88	<0.001	-5.887	0.8123	-7.501 to -4.273
Velocity (m/s) - L	-9.004	88	<0.001	-34.522	3.8352	-42.138 to -26.906

Fig. 2 Bar chart showing mean NCV in left and right sural nerve amongst all groups.



In summary, the results indicate a clear and progressive decline in sural nerve conduction velocity associated with the duration of Type 2 Diabetes Mellitus. The findings emphasise the critical impact of prolonged diabetes on peripheral nerve function and highlight the importance of early intervention and regular monitoring to mitigate neuropathic complications.

DISCUSSION

The findings of this study offer substantial insights into the sural nerve conduction velocities (NCV) in individuals with Type 2 Diabetes Mellitus (T2DM) and serve to validate the hypothesis posited at the outset. The hypothesis conjectured that there would be a significant decrease in NCV as the duration of T2DM increases, implying that prolonged exposure to diabetes could detrimentally affect nerve function.

Analysing the data, a remarkable trend became evident: patients with T2DM for more than 10 years (Group 2) showed a significantly reduced NCV compared to those with T2DM for less than 5 years (Group 1) and the non-diabetic control group (Group 3). This pattern was consistent in both left and right sural nerves, as evidenced by the ANOVA results which indicated significant differences in mean NCV across the groups for both the left and right sural nerves.

Additionally, the t-test comparisons further substantiated these findings, revealing a pronounced decrease in NCV in the long-standing diabetes group compared to controls. These findings were significant for velocity on both sides, with a marked reduction in nerve conduction velocity for both left ($t(88) = -13.530$, $p < .001$) and right sural nerves ($t(88) = -14.593$, $p < .001$) in the T2DM group with a duration of more than 10 years.

The consistency and reproducibility of these results align with previously documented research indicating the progressive nature of diabetic neuropathy and its impact on NCV as diabetes duration extends[13, 14, 15]. Besides, the comparative analysis demonstrated that the velocity reductions were notably significant, while latency and amplitude measures exhibited variability and were less consistently different between the groups, suggesting that velocity might be a more sensitive indicator of early diabetic neuropathic changes.

These findings underline the importance of early and sustained management of T2DM to potentially mitigate the decline in nerve function, echoing the sentiments of established literature which posits that better metabolic control is associated with preserved nerve function. Considering the study's objectives, the results offer a quantifiable correlation between NCV and T2DM duration, which holds significant potential for influencing clinical diagnostic and treatment strategies for diabetic neuropathy.

The research findings offer a compelling narrative confirming the hypothesized relationship between the chronicity of Type 2 Diabetes Mellitus (T2DM) and the reduction in nerve conduction velocity (NCV) of the sural nerve. The data distinctly demonstrate that individuals with a T2DM duration exceeding 10 years exhibited significantly lower NCV compared to those with a shorter duration of the disease and the non-diabetic control group. This aligns with previous studies that have observed a progressive decline in NCV with the increasing duration of diabetes, indicative of advancing diabetic neuropathy.

The underlying mechanisms driving these outcomes can be attributed to the long-term effects of hyperglycaemia on peripheral nerves. Chronic hyperglycaemia in diabetes is known to lead to glycation of nerve proteins, reduced nerve blood flow due to microangiopathy, and direct neuronal damage through the accumulation of sorbitol and fructose in nerve cells. Additionally, oxidative stress and inflammatory pathways are significantly involved in diabetic neuropathy pathogenesis. Studies have suggested that inflammatory markers, such as TNF-α and IL-6, are elevated in individuals with T2DM and correlate with NCV reduction, substantiating the inflammation theory in neuropathy development[14].

Comparatively, our study's results are coherent with the findings by Hussain *et al.* (2014), who reported significant electrophysiological changes with the duration of the disease, noting NCV reductions in lower limbs even in patients with shorter disease duration but normal

upper limb NCV[13]. This indicates that the lower limbs are possibly more susceptible to diabetic changes earlier in the disease course, which may be due to the longer nerve fibres being more vulnerable to metabolic insults.

The fact that the study results corroborated the hypothesis may not only emphasize the importance of early and proactive management of T2DM to prevent or delay the onset of neuropathy but also suggest that sural NCV could serve as a sensitive early biomarker for the detection of neuropathy in diabetic patients. Our findings add weight to the argument for regular screening and monitoring of NCV in T2DM patients as part of routine clinical care to identify early signs of neuropathy before significant clinical symptoms appear.

While the results substantiate existing knowledge, they also highlight the necessity for ongoing research into the specific biological mechanisms at play and how they may be interrupted or mitigated. The potential for novel therapies targeting the molecular pathways implicated in the pathogenesis of diabetic neuropathy, such as antioxidants and anti-inflammatory agents, is an area ripe for further exploration.

The findings from this study are poised to have significant implications for the clinical management of diabetic neuropathy and future research in this field. They provide valuable insights into the progression of diabetic neuropathy in relation to the duration of Type 2 Diabetes Mellitus (T2DM), highlighting the importance of early diagnosis and targeted intervention to mitigate the condition's debilitating effects.

Clinically, the results suggest that regular monitoring of nerve conduction velocities in patients with T2DM could be a critical factor in the early identification and treatment of diabetic neuropathy. This is particularly relevant given that current research supports the efficacy of glucose control in halting the progression of diabetic neuropathy in type 1 diabetes, with more modest effects in type 2 diabetes[10]. The study results align with this perspective, indicating that stringent glycaemic control from the onset of T2DM could significantly reduce the risk of developing neuropathy.

The research emphasizes the need for intensified multifactorial intervention in T2DM management, especially for patients with a longer duration of the disease. These interventions should target hyperglycaemia, hypertension, dyslipidaemia, and microalbuminuria to slow the progression of neuropathy and other related complications[16].

The study's implications extend to theoretical contributions as well. It strengthens the theory that diabetic neuropathy's progression is closely associated with the duration of T2DM, suggesting that metabolic memory and the long-term effects of hyperglycaemia play critical roles in the development of neuropathy[17]. The results also provide evidence for the prevailing theory that the lower extremities are affected earlier in the course of T2DM due to the vulnerability of longer nerve fibres, a perspective supported by existing literature[13].

This study contributes to the evolving narrative on the clinical and theoretical understanding of diabetic neuropathy. It corroborates the notion that early and sustained management of T2DM, encompassing glycaemic control and comprehensive risk factor management, is crucial for preventing the onset and progression of diabetic neuropathy. Further research is essential to develop more targeted therapies that address the underlying pathophysiological mechanisms of this condition.

CONCLUSION

This study confirms that prolonged T2DM is associated with significant reductions in sural nerve conduction velocity, indicating progressive peripheral nerve damage. Early diagnosis and effective management of diabetes are crucial to prevent severe neuropathic complications. Regular monitoring of NCV can aid in the early detection and timely intervention of diabetic

neuropathy, ultimately improving patient outcomes and quality of life[18, 19].

Declaration by Authors

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