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Correlation of HbA1c Levels with Diabetic Peripheral Neuropathy by Nerve Conduction Study – A Retrospective Observational Study

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ABSTRACT

Background

Diabetic peripheral neuropathy (DPN) is a common and disabling complication of type 2 diabetes mellitus (T2DM), often associated with poor glycemic control. While glycated hemoglobin (HbA1c) is a key marker of long-term glycemia, its precise relationship with objective electrophysiological parameters remains variably reported. This study aimed to evaluate the correlation between HbA1c levels and nerve conduction study (NCS) parameters in patients with T2DM and DPN.

Methods

Hospital-based retrospective observational study was conducted in a Tertiary Care Centre at Puducherry from March 1 to May 28, 2025. Seventy patients with confirmed T2DM, aged 30–80 years, who presented with neuropathic symptoms and underwent NCS were included. Patients with type 1 diabetes, thyroid dysfunction, vitamin B12 deficiency, or alcohol abuse were excluded. Clinical data, HbA1c values, and NCS parameters—including motor and sensory conduction velocity, amplitude, and latency—were extracted and analyzed. Pearson correlation was used to assess relationships between HbA1c, diabetes duration, and NCS findings.

Results

The mean age of patients was 59.19 ± 11.7 years; 61.4% were male. The mean duration of diabetes was 9.19 ± 5.53 years and the mean HbA1c was 8.05 ± 1.53%. Axonal neuropathy was the most prevalent pattern (52.9%), followed by mixed (31.4%) and demyelinating (15.7%). A significant negative correlation was found between HbA1c and multiple NCS parameters: motor NCV ($r = -0.28$, $p = 0.0181$), sensory NCV ($r = -0.23$, $p = 0.0485$), motor amplitude ($r = -0.36$, $p = 0.0018$), and sensory amplitude ($r = -0.29$, $p = 0.0144$). Motor and sensory latencies were positively correlated with HbA1c ($r = 0.26$ and $r = 0.27$; $p < 0.05$). No statistically significant correlations were observed between diabetes duration and any NCS parameter.

Conclusion

Elevated HbA1c levels were significantly associated with reduced nerve conduction velocity and amplitude, and increased latency, suggesting a strong link between poor glycemic control and the severity of diabetic peripheral neuropathy. HbA1c may serve as a useful predictor of neuropathic risk, supporting the need for strict glycemic management and early electrophysiological screening in patients with T2DM.

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by sustained hyperglycemia resulting from defects in insulin secretion, insulin action, or both. According to the International Diabetes Federation (IDF), approximately 537 million adults were living with diabetes in 2021, a number projected to rise to 783 million by 2045. Alongside this growing epidemic, the burden of diabetes-related complications continues to escalate, significantly impairing quality of life and increasing healthcare costs globally. Among these complications, diabetic peripheral neuropathy (DPN) is one of the most prevalent and debilitating (1).

DPN affects nearly half of all individuals with diabetes over their lifetime. It is a symmetrical, length-dependent sensorimotor polyneuropathy resulting from chronic hyperglycemia-induced metabolic and vascular insults to peripheral nerves. Clinically, DPN manifests with a wide spectrum of symptoms, including numbness, paresthesia, burning pain, and loss of protective sensation, particularly in the lower extremities. These symptoms not only diminish functional capacity and quality of life but also increase the risk for foot ulceration, infections, and lower limb amputations (2).

Hyperglycemia is widely recognized as the central driver in the pathogenesis of DPN, triggering multiple interrelated biochemical pathways such as the polyol pathway, advanced glycation end product (AGE) formation, oxidative stress, and activation of protein kinase C. These mechanisms collectively

impair neuronal integrity and function. Glycated hemoglobin (HbA1c) reflects the average plasma glucose concentration over the previous 8 to 12 weeks and remains the cornerstone for monitoring long-term glycemic control. Numerous studies have linked poor glycemic control, as indicated by elevated HbA1c levels, with the onset and progression of DPN. However, the strength and nature of this association remain inconsistent across the literature (3).

Several large-scale trials, including the Diabetes Control and Complications Trial (DCCT) and the UK Prospective Diabetes Study (UKPDS), have demonstrated that intensive glycemic control reduces the risk of developing neuropathic complications in both type 1 and type 2 diabetes. Yet, the relationship between HbA1c and the severity or electrophysiological characteristics of DPN is not always linear or consistent. Some studies suggest a clear dose-response relationship, while others fail to observe a statistically significant correlation. These discrepancies may be attributable to differences in study design, population characteristics, duration of diabetes, comorbid conditions, and methods used for neuropathy assessment (4).

Nerve conduction studies (NCS) represent the gold standard for the objective evaluation of peripheral nerve function. They are capable of detecting subclinical neuropathy and quantifying the severity of nerve impairment through parameters such as conduction velocity, amplitude, and latency of both motor and sensory nerves. Unlike symptom-based scales or clinical examination, which are subject to patient reporting and inter-examiner variability, NCS provide reproducible, quantifiable, and pathophysiologically grounded data on peripheral nerve integrity. Despite their diagnostic value, NCS are underutilized in routine diabetes care and research, and their integration with metabolic indices such as HbA1c remains limited in clinical practice (5).

In this context, our study seeks to address the gap in the literature by exploring the correlation between HbA1c levels and nerve conduction parameters in individuals with diabetes diagnosed with DPN. By using a retrospective observational design, we aim to capture real-world data reflective of routine clinical practice. Understanding this relationship is crucial for several reasons. Firstly, it may validate the role of HbA1c not only as a glycemic control marker but also as a potential predictor of neuropathic risk. Secondly, it could help stratify patients at higher risk for neuropathy who may benefit from earlier electrophysiological screening and targeted interventions. Lastly, elucidating this correlation may provide further impetus to integrate nerve conduction assessments into the standard management protocols for diabetes, particularly in those with persistently elevated HbA1c levels (6).

Through this study, we aim to determine whether higher HbA1c levels are associated with worsening nerve conduction parameters, thereby reinforcing the importance of strict glycemic control in preventing or mitigating diabetic neuropathy. The findings may also contribute to refining the clinical decision-making process regarding screening frequency and therapeutic prioritization in patients with suboptimal glycemic control.

METHODOLOGY

Study Design and Setting

This study was designed as a hospital-based retrospective observational study conducted in a Tertiary Care Centre at Puducherry (IEC Ref No: 54/SVMCH/IEC-Cert/June.25). The study aimed to analyze the correlation between glycated hemoglobin (HbA1c) levels and diabetic peripheral neuropathy using nerve conduction study (NCS) parameters.

Study Period and Sample Size

Retrospective data were collected over a period of three months, from March 1, 2025, to May 28, 2025. The sample size was estimated using the formula:

$$n = z^2pq/d^2,$$

where z represents the confidence interval (95%), p is the estimated prevalence, $q = 1-p$, and d is the margin of error (7%). Based on this calculation, a minimum sample size of 70 was determined to be adequate for analysis (7).

Sampling Technique

A purposive sampling method was employed to select eligible cases from the medical records. Only those fulfilling the predefined inclusion and exclusion criteria were included in the study (8).

Inclusion Criteria

Patients were included if they met the following criteria:

1. Had a confirmed diagnosis of type 2 diabetes mellitus based on medical history and clinical assessment.
2. Were between 30 and 80 years of age.
3. Presented with clinical symptoms suggestive of diabetic neuropathy such as pain, tingling, numbness, or weakness in the extremities.
4. Had a documented duration of diabetes of at least one year.

Exclusion Criteria

The following patients were excluded:

1. Individuals diagnosed with type 1 diabetes mellitus.
2. Patients with coexisting thyroid disorders, vitamin B12 deficiency, or chronic alcohol use.
3. Those with incomplete or unreliable medical records or in whom nerve conduction studies had not been performed.

Data Collection Procedures

Case sheets of eligible diabetic patients were obtained from the Medical Records Department (MRD). Clinical data, including demographic details such as age and sex, medical history, comorbidities, and physical examination findings, were extracted and reviewed. Specific attention was paid to symptoms and signs suggestive of peripheral neuropathy (9).

Nerve conduction study reports were examined to collect data on electrophysiological parameters, including latency, amplitude, and nerve conduction velocity for both motor and sensory nerves. Additionally, laboratory data were reviewed to obtain HbA1c levels and the documented duration of diabetes (10).

All data were recorded on a predesigned and pretested proforma to ensure uniformity and minimize transcription errors. Confidentiality of patient information was maintained at all stages of the study, and the data were stored securely and will be retained for a period of three years.

STATISTICAL ANALYSIS

Data were entered into Microsoft Excel and subsequently analyzed using SPSS software version 23.0. Categorical variables were expressed as frequencies and percentages, while continuous variables were summarized using means and standard deviations.(11) The association between HbA1c levels and nerve conduction parameters was evaluated using appropriate statistical tests, with significance determined at a p-value < 0.05.

RESULTS

Demographic and Clinical Characteristics

A total of 70 patients with confirmed type 2 diabetes mellitus (T2DM) were included in this retrospective observational study. The mean age of the study population was 59.19 ± 11.7 years, with the majority falling within the 50–70-year age range. There was a male predominance, with 43 males (61.4%) and 27 females (38.6%). The mean duration of diabetes was 9.19 ± 5.53 years, with the minimum being 1 year and the maximum 25 years.(Table 1)

The mean HbA1c level among participants was 8.05 ± 1.53%, indicating poor overall glycemic control in the population studied. Based on HbA1c levels, patients were categorized into three groups: good control (<7%) – 12 patients (17.1%), moderate control (7–9%) – 36 patients (51.4%), and poor control (>9%) – 22 patients (31.4%). Most patients also had comorbid conditions such as hypertension and dyslipidemia, although these were not directly analyzed in relation to neuropathy outcomes.(Table 2)

Distribution of Neuropathy Patterns

Neuropathy patterns were classified based on NCS findings into axonal, demyelinating, and mixed types. Among the 70 patients, axonal neuropathy was the most common pattern, observed in 37 patients (52.9%). Mixed-type neuropathy was seen in 22 patients (31.4%), and demyelinating neuropathy in 11 patients (15.7%). These findings align with the common pathophysiological mechanisms in diabetic neuropathy, where axonal damage predominates due to chronic hyperglycemia and oxidative stress.(Figure 1)

Nerve Conduction Abnormalities

Nerve conduction studies revealed substantial abnormalities among participants. The mean motor nerve conduction velocity (Motor NCV) was 41.13 ± 5.12 m/s, and the sensory NCV was 38.00 ± 4.23 m/s. Motor nerve amplitude averaged 6.22 ± 1.44 mV, while sensory nerve amplitude averaged 12.35 ± 3.50 μV. Motor latency was 4.45 ± 0.54 ms, and sensory latency was 3.28 ± 0.41 ms.

Based on standard clinical thresholds, 44.3% of patients had abnormal Motor NCV (<40 m/s), and 22.9% had abnormal Sensory NCV (<35 m/s). These values reflect a high burden of diabetic peripheral neuropathy among the studied population.(Table 3)

Correlation of HbA1c with Nerve Conduction Parameters

To evaluate the primary objective of this study, correlation analysis was performed between HbA1c levels and NCS parameters. A significant negative correlation was observed between HbA1c and Motor NCV (r = -0.28, p = 0.0181), indicating that higher HbA1c levels were associated with slower motor nerve conduction. A similar inverse relationship was found for Sensory NCV (r = -0.23, p = 0.0485). (Figure 2)

Additionally, motor amplitude (r = -0.36, p = 0.0018) and sensory amplitude (r = -0.29, p = 0.0144) showed significant negative correlations with HbA1c, suggesting progressive axonal degeneration in patients with poor glycemic control. Conversely, motor latency (r = 0.26, p = 0.0258) and sensory latency (r = 0.27, p = 0.0212) were positively correlated with HbA1c, reflecting delayed nerve conduction associated with increased glycemic burden.(Figure 3, Table 4)

These findings are supported by existing literature, where chronic hyperglycemia has been shown to result in demyelination, impaired axonal transport, and reduced conduction velocities. Similar correlations were reported by Shaji et al. (2023) and Sharma et al. (2018), who found significant associations between high HbA1c and reduced NCS performance.

Correlation of Duration of Diabetes with NCS Parameters

To address the secondary objective, the relationship between duration of diabetes and nerve conduction parameters was evaluated. While longer diabetes duration tended to be associated with reduced conduction velocities and amplitudes, these correlations were not statistically significant in this study.

Specifically, the correlation between duration of diabetes and Motor NCV was weak and non-significant (r = -0.04, p = 0.7136). Sensory NCV showed a slightly stronger but still non-significant correlation (r = 0.20, p = 0.0892). No meaningful correlations were observed between diabetes duration and amplitude or latency parameters.(Figure 4, Table 5)

This suggests that while duration of diabetes may contribute to nerve dysfunction, glycemic control (as indicated by HbA1c) plays a more dominant and statistically evident role in the development and progression of diabetic peripheral neuropathy. The lack of significance with duration in this cohort could be attributed to relatively limited variation in disease duration or confounding comorbidities not accounted for in this analysis.

DISCUSSION

This retrospective observational study aimed to investigate the correlation between glycated haemoglobin (HbA1c) levels and nerve conduction parameters among patients with type 2 diabetes mellitus (T2DM), and to examine the role of diabetes duration in the development of diabetic peripheral neuropathy (DPN). Our findings reveal significant correlations between poor glycemic control and impaired nerve conduction, supporting the hypothesis that chronic hyperglycemia is a major contributor to neuropathic changes in diabetes (12).

In the present study, a significant proportion of patients demonstrated abnormal nerve conduction findings, with 44.3% showing reduced motor nerve conduction velocity (NCV) and 22.9% with reduced sensory NCV. The most common neuropathy pattern identified was axonal neuropathy (52.9%), consistent with the typical neuropathological changes seen in DPN, which initially affect the longest nerves and gradually lead to axonal loss (13).

We found statistically significant negative relationships between nerve amplitudes and HbA1c levels and both motor and sensory NCV. Lower amplitudes and slower conduction velocities were seen in patients with increased HbA1c levels, suggesting both axonal degradation and demyelination. Additionally, latency metrics, which indicate delayed nerve signal transmission, showed a favorable correlation with HbA1c. These results concur with a number of published research. Shaji et al. (2023) reported similar inverse associations between HbA1c and nerve conduction parameters in T2DM patients, while Sharma et al. (2018) found that even moderately elevated HbA1c levels could impair sensory NCV.(14,6).

Conversely, our analysis did not reveal significant correlations between duration of diabetes and nerve conduction parameters. Although longer disease duration has been traditionally associated with higher risk of neuropathy, this association was not statistically significant in our cohort. This may be due to the relatively narrow distribution of diabetes duration in the study population, or the stronger impact of current glycemic control compared to historical exposure. Some previous studies have also reported similar results, emphasizing that present glycemic control (HbA1c) may be a better predictor of neuropathic damage than disease duration alone.(15)

Additionally, the use of nerve conduction studies (NCS) as an objective diagnostic tool in this study adds robustness to our findings. NCS remains the gold standard in diagnosing and classifying the type and severity of peripheral neuropathy, and our study reinforces its role in early identification of subclinical nerve dysfunction in diabetic patients (16).

However, several limitations must be acknowledged. This was a single-center retrospective study with a limited sample size, which may affect generalizability. Confounding factors such as medication adherence, lifestyle factors, and coexisting metabolic conditions (e.g., dyslipidemia, hypertension) were not controlled for, which could influence nerve function independently of HbA1c.(17)

CONCLUSION

Our study demonstrates that poor glycemic control, as reflected by elevated HbA1c levels, is significantly associated with impaired nerve conduction parameters in patients with type 2 diabetes mellitus. HbA1c showed a stronger correlation with NCS abnormalities than diabetes duration, suggesting that current metabolic control plays a more decisive role in the progression of diabetic peripheral neuropathy.

These findings highlight the importance of tight glycemic control in preventing or mitigating diabetic neuropathy. Routine nerve conduction studies should be considered in patients with poor glycemic control, even in the absence of overt neurological symptoms, to enable early detection and intervention.

Further prospective, multicentric studies with larger sample sizes and inclusion of other metabolic parameters are recommended to validate and expand upon these findings.

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- **TABLES AND FIGURES:**
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- **Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (N = 70)**

Parameter	Value
Age (years)	59.19 ± 11.7
Duration of Diabetes (years)	9.19 ± 5.53
HbA1c (%)	8.05 ± 1.53
Fasting Blood Sugar (mg/dL)	157.26 ± 33.59
Postprandial Blood Sugar (mg/dL)	221.43 ± 45.22
Motor NCV (m/s)	41.13 ± 5.12
Sensory NCV (m/s)	38.00 ± 4.23
Motor Amplitude (mV)	6.22 ± 1.44
Sensory Amplitude (μV)	12.35 ± 3.50
Motor Latency (ms)	4.45 ± 0.54
Sensory Latency (ms)	3.28 ± 0.41
F-Wave Latency (ms)	29.55 ± 2.78
Gender, n (%)	Male: 43 (61.4%), Female: 27 (38.6%)
Neuropathy Pattern, n (%)	Axonal: 37 (52.9%), Mixed: 22 (31.4%), Demyelinating: 11 (15.7%)

- **Table 2: Distribution of Glycemic Control Categories (N = 70)**

Glycemic Control Category	Number of Patients (n)	Percentage (%)
Good (<7%)	12	17.1%
Moderate (7–9%)	36	51.4%
Poor (>9%)	22	31.4%

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- **Table 3: Nerve Conduction Abnormalities Detected (N = 70)**

Parameter	Number of Patients (n)	Percentage (%)
Motor NCV Abnormal (<40 m/s)	31	44.3%
Sensory NCV Abnormal (<35 m/s)	16	22.9%

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- **Table 4: Correlation of HbA1c with Nerve Conduction Study (NCS) Parameters**

NCS Parameter vs HbA1c	Pearson r	p-value	Significance
Motor NCV vs HbA1c	-0.28	0.0181	Significant
Sensory NCV vs HbA1c	-0.23	0.0485	Significant
Motor Amplitude vs HbA1c	-0.36	0.0018	Significant
Sensory Amplitude vs HbA1c	-0.29	0.0144	Significant
Motor Latency vs HbA1c	0.26	0.0258	Significant
Sensory Latency vs HbA1c	0.27	0.0212	Significant

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- **Table 5: Correlation of Diabetes Duration with Nerve Conduction Study (NCS) Parameters**

NCS Parameter vs Duration	Pearson r	p-value	Significance
Motor NCV vs Duration	-0.04	0.7136	Not Significant
Sensory NCV vs Duration	0.20	0.0892	Not Significant
Motor Amplitude vs Duration	-0.12	0.3268	Not Significant
Sensory Amplitude vs Duration	-0.01	0.9174	Not Significant
Motor Latency vs Duration	0.01	0.9612	Not Significant

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- Figure 1: Distribution of Neuropathy Patterns

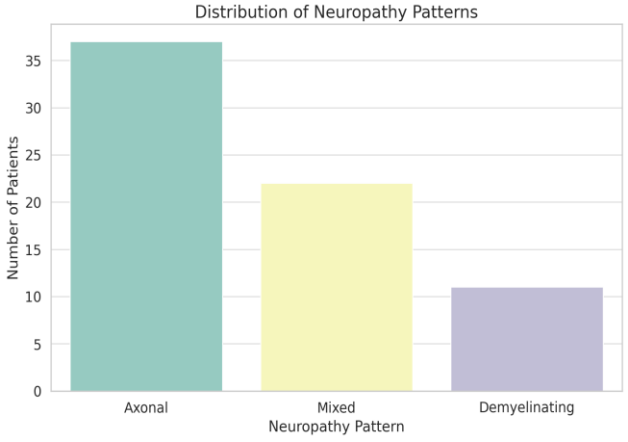

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Figure 2: Correlation of HbA1c with Motor Nerve Conduction Velocity

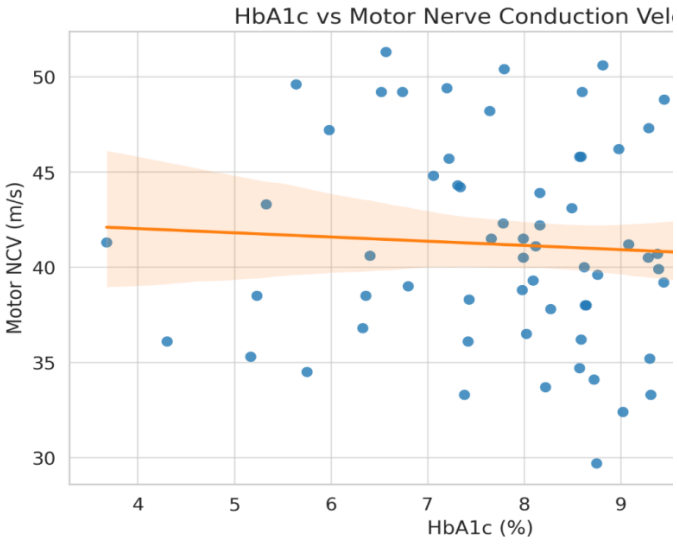

Figure 3: Motor Nerve Conduction Velocity by Glycemic Control Category

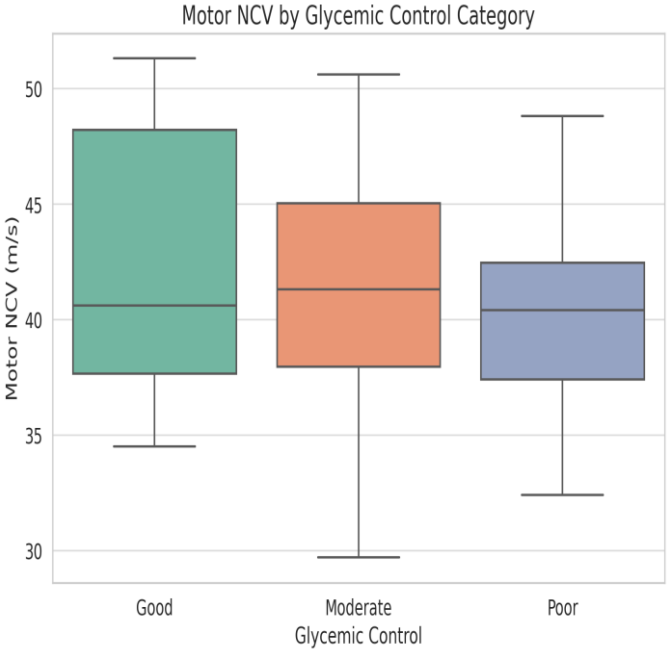

Figure 4: Duration of Diabetes vs Sensory NCV