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CORRELATION OF GLYCEMIC CONTROL HBA1C WITH SEVERITY OF DIABETIC PERIPHERAL NEUROPATHY IN TYPE 2 DIABETES MELLITUS PATIENTS

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Abstract

Background: Diabetic peripheral neuropathy (DPN) is a common complication of type 2 diabetes mellitus (T2DM). HbA1c reflects long-term glycemic control and may influence neuropathy severity. ***Methods:*** A cross-sectional study of 100 T2DM patients was conducted. HbA1c levels were measured, and DPN severity was assessed using Neuropathy Disability Score (NDS) and nerve conduction studies (NCS). ***Results:*** DPN was present in 78% of patients. Mean HbA1c increased significantly with neuropathy severity ($p < 0.001$). HbA1c showed positive correlation with NDS ($r = 0.62$) and negative correlation with nerve conduction parameters. ***Conclusion:*** Higher HbA1c levels are significantly associated with greater severity of DPN in T2DM patients.

Keywords: Type 2 diabetes mellitus; HbA1c; Diabetic peripheral neuropathy; Glycemic control; Nerve conduction study

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INTRODUCTION

Diabetic peripheral neuropathy (DPN) is one of the most common microvascular complications of type 2 diabetes mellitus (T2DM), characterized by distal symmetric sensory loss, pain, and impaired protective sensation that increases risk of foot ulceration and amputation. The reported prevalence of DPN among people with diabetes varies by population and diagnostic criteria but large meta-analyses and national cohorts indicate pooled prevalence's roughly in the range of 20–40%, underscoring the public-health burden of this complication [1].

Chronic hyperglycemia is a key driver of microvascular damage in diabetes. Glycated hemoglobin (HbA1c) — an integrated measure of average blood glucose over the preceding 2–3 months -is widely used both clinically and in research as the principal index of glycemic control. Multiple observational studies and cohort analyses have reported associations between higher mean HbA1c (and higher HbA1c variability) and both the presence and the progression of DPN; several studies suggest that mean HbA1c explains a substantial proportion of risk for neuropathic changes independent of diabetes duration [2-3].

Mechanistically, prolonged hyperglycemia promotes a constellation of injurious pathways — increased polyol flux, formation of advanced glycation end products (AGEs), activation of the hexosamine and protein kinase C pathways, mitochondrial dysfunction, and oxidative stress — that impair peripheral nerve structure and function (demyelination, axonal loss, microvascular ischemia). These pathophysiologic links provide biological plausibility for the observed epidemiologic correlation between poor glycemic control (higher HbA1c) and severity of DPN [4].

Despite that link, the strength of association varies across studies and populations; recent work has emphasized not only mean HbA1c but also glycemic variability as

potentially important predictors of neuropathy risk and severity [5]. This nuanced picture motivates further clinically-oriented investigations that correlate HbA1c (mean and variability) with validated measures of DPN severity (symptom scores, clinical examination, and nerve conduction studies) in T2DM cohorts.

OBJECTIVES

To evaluate the correlation between glycemic controls measured by HbA1c and the severity of diabetic peripheral neuropathy in adults with type 2 diabetes mellitus patients

MATERIALS AND METHODS

This analytical cross-sectional study was conducted in the department of medicine in an Indian hospital. Written informed consent was obtained from all participants prior to inclusion.

A total of 100 adult patients diagnosed with type 2 diabetes mellitus (according to ADA criteria) were enrolled.

Inclusion criteria

- Patients age ≥18 years with both gender
- T2DM duration ≥1 year
- Willingness to participate

Exclusion criteria

- Patients age <18 years
- Other causes of peripheral neuropathy (e.g., alcohol abuse, vitamin B12 deficiency, uremia, Guillain–Barré syndrome)
- Recent acute hyperglycemic crises, severe systemic illnesses, or medications known to influence nerve function
- Non willingness to participate

Clinical Data Collection

Demographic data (age, sex), clinical variables (duration of diabetes, body mass index, blood pressure, lipid profile), and treatment details were recorded using a standardized case report form. Comorbid conditions such as hypertension and dyslipidemia were documented.

Glycemic Control Assessment

Blood samples were collected after an overnight fast for laboratory measurement of HbA1c. Where available, historical HbA1c records from the preceding 6–12 months were used to calculate mean HbA1c and/or glycemic variability indices (e.g., coefficient of variation of HbA1c) to capture long-term control. This approach reflects strategies used in studies evaluating both average and variability of HbA1c in relation to DPN outcomes.

Assessment of Diabetic Peripheral Neuropathy (DPN)

Severity of DPN was quantified using a combination of clinical and electrophysiological measures.

1. Clinical Neuropathy Scores:
   - Neuropathy Disability Score (NDS): Assessment of reflexes, vibration, pain (pinprick), and temperature sensations was performed according to standardized scales. Higher scores indicate more severe neuropathy.
2. Nerve Conduction Studies (NCS):
   - Electrophysiological testing was done. Sensory and motor nerve conduction parameters were recorded for bilateral lower limb nerves, including nerve conduction velocity (NCV), latency, and amplitude. Abnormal values were interpreted against laboratory reference standards. NCS provides objective estimation of peripheral nerve function and is considered a gold-standard measure of large fiber neuropathy.
3. Additional Sensory Testing (optional):
   - Quantitative Sensory Testing (QST) and/or Biothesiometry: Vibration Perception Threshold (VPT) testing using a biothesiometer to quantify sensory loss.

Patients were classified into severity categories (e.g., no neuropathy, mild, moderate, and severe DPN) based on predefined NDS and/or NCS criteria, aligning with established clinical grading systems.

Statistical Analysis: Data were captured in Microsoft Excel and analyzed using SPSS version 25 (IBM Corp.). Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) as appropriate. Categorical variables were summarized as percentages. A p-value of <0.05 was considered statistically significant.

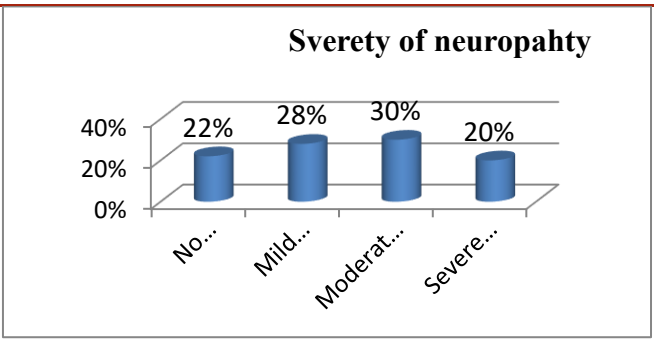
Ethical Considerations: The study adhered to the Declaration of Helsinki principles. Data confidentiality was maintained throughout, and only de-identified data were analyzed for publication.

RESULTS

A total of 100 type 2 diabetes mellitus patients fulfilling the inclusion criteria were enrolled and analysed in this study. The mean age of participants was 56.8 ± 9.4 years, with male predominance (58%). Details description shown in table: 1

DPN was present in 78% of patients, with 50% having moderate-to-severe neuropathy

Graph 1: Distribution of Diabetic Peripheral Neuropathy (DPN) Severity



Mean HbA1c increased significantly with increasing severity of neuropathy ($p < 0.001$)

Table 1: Baseline Demographic and Clinical Characteristics (n = 100)

Variable	Mean ± SD
Age (years)	56.8 ± 9.4
Male	58 (58%)
Female	42 (42%)
Duration of diabetes (years)	8.6 ± 5.1
BMI (kg/m²)	27.4 ± 3.8
Hypertension	62 (62%)
Dyslipidemia	54 (54%)
Mean HbA1c (%)	8.4 ± 1.5

Table 2: Mean HbA1c According to Severity of DPN

DPN Severity	Mean HbA1c (%) ± SD
No Neuropathy	6.9 ± 0.7
Mild	7.8 ± 0.9
Moderate	8.9 ± 1.1
Severe	10.2 ± 1.3
p-value (ANOVA)	< 0.001

HbA1c showed a strong positive correlation with Neuropathy Disability Score ($r = 0.62$, $p < 0.001$) and significant negative correlation with nerve conduction parameters

Table 4: Correlation between HbA1c and Neuropathy Parameters

Variable	Correlation Coefficient (r)	p-value
HbA1c vs NDS Score	+0.62	<0.001
HbA1c vs Peroneal NCV	-0.58	<0.001
HbA1c vs Sural Nerve Amplitude	-0.49	<0.001

Positive correlation with clinical severity score; negative correlation with nerve conduction velocity and amplitude
On multivariate regression, HbA1c was the strongest independent predictor of neuropathy severity

Table 4: Multivariate Linear Regression Analysis for Predictors of Neuropathy Severity

Variable	β Coefficient	Standard Error	p-value
HbA1c (%)	0.52	0.08	<0.001
Duration of Diabetes	0.31	0.09	0.002
Age	0.18	0.07	0.041
BMI	0.09	0.05	0.210

(Model adjusted $R^2 = 0.54$)

DISCUSSION

We observed a significant positive correlation between higher HbA1c levels and increased severity of diabetic peripheral neuropathy (DPN) as assessed by clinical scores and electrophysiological parameters. Patients with more severe neuropathy had progressively higher mean HbA1c values ($p < 0.001$), and HbA1c was an independent predictor of neuropathy severity in multivariate analysis.

Our results align with other studies performed by Shinde C, et al [6] and Hunaifi et al [8] that reported a significant relationship between HbA1c and the severity of DPN measured by neuropathy disability scores (NDS) and nerve conduction abnormalities, suggesting that poor glycemic control is associated with more severe neuropathy. Observational data indicate that elevated HbA1c is associated with decreased nerve conduction velocity and amplitude.

Longitudinal real-world evidence also supports these associations with Nozawa et al [1] and Feldman EL, et al [7], reported that higher 3-year mean HbA1c levels were significantly associated with the development or progression of DPN in a large cohort of type 2 diabetic patients, with an adjusted odds ratio suggesting meaningful clinical relevance of chronic hyperglycemia. This supports the pathophysiologic concept that chronic glycemic exposure accelerates microvascular and metabolic injury to peripheral nerves through oxidative stress, advanced glycation end-product formation, and impaired blood flow, mechanisms observed in neuropathy research.

Beyond absolute HbA1c levels, glycemic variability has emerged as an important factor related to neuropathy severity. Several studies have shown that not only mean HbA1c but also variability in HbA1c over time is associated with peripheral nerve impairment. Su et al [4] and Nozawa K, et al [10] demonstrated that increased HbA1c variability was independently linked to the presence and severity of DPN in type 2 diabetic patients, suggesting that fluctuating glycemic patterns may exert additional stress on nerve tissues beyond that conferred by sustained hyperglycemia alone. Lee SS and colleagues similarly found that HbA1c variability together with chronic glycemic impairment was strongly associated with composite scores of nerve conduction abnormalities, supporting the notion that both aspects of glycemic control contribute to neuropathic severity [9].

Our findings mirror these observations, indicating that the relationship between poor glycemic control and DPN severity is well supported across different populations and study designs. Moreover, meta-analytic evidence suggests that glycemic variability assessed by continuous glucose monitoring metrics is associated with an increased risk of developing DPN, reinforcing the clinical relevance of monitoring both average glucose levels and glycemic fluctuations [11].

In contrast to some earlier reports in type 1 diabetes where glycemic control showed a less consistent effect on neuropathy progression, evidence in T2DM has increasingly pointed to a meaningful correlation between HbA1c and peripheral nerve dysfunction [12]. The heterogeneity in measures and populations across studies highlights the complexity of neuropathy development and the need for multifaceted glycemic assessment.

CONCLUSION

In patients with T2DM, poor glycemic control as reflected by higher HbA1c levels is significantly associated with greater severity of diabetic peripheral neuropathy. HbA1c is an independent predictor of neuropathy severity, supporting its role not only as a marker of average glucose exposure but also as a prognostic indicator for diabetic nerve injury. These findings reinforce the importance of optimized glycemic management to mitigate neuropathic complications in T2DM, and they suggest that strategies to reduce both mean HbA1c and glycemic variability may enhance nerve health and reduce the burden of neuropathy.

REFERENCES

1. Casadei, G.; Filippini, M.; Brognara, L. Glycated Hemoglobin (HbA1c) as a Biomarker for Diabetic Foot Peripheral Neuropathy. Diseases 2021, 9, 16. https://doi.org/10.3390/ diseases9010016
2. González P, Lozano P, Ros G, Solano F. Hyperglycemia and Oxidative Stress: An Integral, Updated and Critical Overview of Their Metabolic Interconnections. Int J Mol Sci. 2023 May 27; 24(11):9352. doi: 10.3390/ijms24119352. PMID: 37298303; PMCID: PMC10253853.
3. Hamid WS, Ahmed HS, Osman MA, Babiker R. Nerve conduction and its correlations with duration of diabetes mellitus and glycosylated haemoglobin in type 2 diabetes mellitus (T2DM). Journal of Endocrinology, Metabolism and Diabetes in South Africa. 2021; 26(2):46-51.
4. Su, Jb., Zhao, Lh., Zhang, Xl. et al. HbA1c variability and diabetic peripheral neuropathy in type 2 diabetic patients. Cardiovasc Diabetol 17, 47 (2018). https://doi.org/10.1186/ s12933-018-0693-0
5. Sun J, Wang Y, Zhang X, Zhu S, He H. Prevalence of peripheral neuropathy in patients with diabetes: A systematic review and meta-analysis. Prim Care Diabetes. 2020 Oct; 14(5):435-444. doi: 10.1016/j.pcd.2019.12.005. Epub 2020 Jan 6. PMID: 31917119.
6. Dr. Chandrakant Shinde, *Sharda Shinde, Swati bhusari, Dr. Anshuja Dongre, Correlation Study between Development of Diabetic Peripheral Neuropathy, Level of Glycosylated Hemoglobin and

Duration of Diabetic Mellitus, Int. J. Pharm. Sci. Rev. Res., ISSN: 0976 – 044X, 84(12) – December 2024; Article No. 03, Pages: 23-27

7. Feldman EL, Callaghan BC, Pop-Busui R, Zochodne DW, Wright DE, Bennett DL, et al. Diabetic neuropathy. Nat Rev Dis Primers. 2019;5(1):41.
8. Hunaifi I, et al. Correlation between HbA1c and neuropathy disability score in type 2 diabetes mellitus patients. Acta Med Indones. 2018; 50(2):107-113.
9. Lee SS, Han HS, Kim HJ, et al. The relationship between glycemic control and severity of diabetic polyneuropathy assessed by nerve conduction studies. Ann Rehabil Med. 2016;40(5):832-840.
10. Nozawa K, et al. Association of mean HbA1c levels with development and progression of diabetic neuropathy in type 2 diabetes: A real-world study. J Diabetes Investig. 2022;13(6):1043-1051.
11. Singarayar, E. K., Josephrajan, T. A., & Jayabalan, P. (2025). Relationship between blood glucose levels and diabetic neuropathy examination score in patients with diabetes mellitus. International Journal of Research in Medical Sciences, 13(4), 1577–1581. https://doi.org/10.18203/2320-6012.ijrms20250984
12. Yang Y, et al. Glycemic variability and risk of diabetic peripheral neuropathy: A systematic review and meta-analysis. Diabetes Res Clin Pract. 2023; 198:110616.