Original Research

ASSOCIATION BETWEEN HBA1C LEVELS AND SEVERITY OF DIABETIC NEUROPATHY: A HOSPITAL-BASED OBSERVATIONAL STUDY

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ABSTRACT

Background: Diabetic neuropathy is one of the most common microvascular complications of diabetes mellitus and is a major cause of morbidity among affected individuals. Chronic hyperglycemia contributes to nerve damage through multiple metabolic and vascular mechanisms. Glycated hemoglobin (HbA1c) serves as a reliable indicator of long-term glycemic control and may be associated with the severity of diabetic neuropathy.
Aim: To evaluate the association between HbA1c levels and the severity of diabetic neuropathy among patients with type 2 diabetes mellitus.
Materials and Methods: A hospital-based observational cross-sectional study was conducted in the Department of Medicine in collaboration with the Department of Obstetrics and Gynaecology at Nirmala Multispeciality Hospital, Rewari, Haryana, from January 2025 to August 2025. A total of 50 patients with type 2 diabetes mellitus and diabetic neuropathy were enrolled. Demographic and clinical data were collected using a structured proforma. HbA1c levels were measured using standardized laboratory methods. Neuropathy severity was assessed using the Michigan Neuropathy Screening Instrument (MNSI) and categorized as mild, moderate, or severe. Statistical analysis was performed using SPSS version 26.0. Pearson's correlation and one-way ANOVA were applied to assess the association between HbA1c levels and neuropathy severity.
Results: The mean age of the study participants was 56.8 ± 9.4 years, with males accounting for 58% of the study population. The mean duration of diabetes was 8.7 ± 4.1 years, and the overall mean HbA1c level was 8.9 ± 1.7%. Mild, moderate, and severe neuropathy were observed in 32%, 42%, and 26% of patients, respectively. Mean HbA1c levels increased significantly with neuropathy severity, measuring 7.3 ± 0.9% in mild neuropathy, 8.8 ± 1.2% in moderate neuropathy, and 10.2 ± 1.1% in severe neuropathy ($p < 0.001$). A strong positive correlation was observed between HbA1c levels and neuropathy severity scores ($r = 0.71$, $p < 0.001$). Patients with severe neuropathy also had a significantly longer duration of diabetes compared with those having mild or moderate neuropathy ($p < 0.001$).
Conclusion: Higher HbA1c levels are significantly associated with increased severity of diabetic neuropathy among patients with type 2 diabetes mellitus. Poor glycemic control and longer duration of diabetes contribute substantially to neuropathy progression. Regular monitoring of HbA1c and maintenance of optimal glycemic control may help reduce the burden and severity of diabetic neuropathy.

Keywords: Type 2 Diabetes Mellitus, HbA1c, Diabetic Neuropathy, Glycemic Control, Peripheral Neuropathy, Diabetes Complications.

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The global burden of diabetes continues to rise, posing significant challenges to healthcare systems worldwide.
Long-term hyperglycemia contributes to the development of various microvascular and macrovascular complications, among which

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diabetic neuropathy is one of the most common and debilitating conditions [1].

Diabetic neuropathy encompasses a heterogeneous group of nerve disorders caused by diabetes and affects up to 50% of patients with long-standing disease [2]. Distal symmetric polyneuropathy is the most prevalent form and is characterized by progressive sensory loss, paresthesia, pain, and motor dysfunction, predominantly involving the lower extremities [3]. The condition significantly impairs quality of life and is a major risk factor for foot ulceration, infection, and lower-limb amputation [4].

The pathogenesis of diabetic neuropathy is multifactorial and involves chronic hyperglycemia-induced metabolic and vascular abnormalities. Elevated glucose levels promote the formation of advanced glycation end products, oxidative stress, mitochondrial dysfunction, and activation of inflammatory pathways, leading to neuronal damage and impaired nerve conduction [5]. Microvascular insufficiency resulting from endothelial dysfunction further contributes to nerve ischemia and degeneration [6].

Glycated hemoglobin (HbA1c) is a widely accepted biomarker for assessing long-term glycemic control and reflects average blood glucose concentrations over the preceding two to three months. Several studies have demonstrated that poor glycemic control, as indicated by elevated HbA1c levels, is associated with an increased risk of diabetic complications, including neuropathy [7]. Landmark clinical trials such as the Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study (UKPDS) established that intensive glycemic control significantly reduces the incidence and progression of microvascular complications in diabetic patients [8,9].

Despite advances in diabetes management, diabetic neuropathy remains a major cause of morbidity. Early identification of patients at risk for severe neuropathy is essential for implementing preventive strategies and optimizing treatment outcomes. HbA1c, being a readily available and cost-effective marker, may serve as a useful predictor of neuropathy severity. However, data regarding the relationship between HbA1c levels and the severity of diabetic neuropathy in the Indian population remain limited.

Therefore, the present study was undertaken to evaluate the association between HbA1c levels and the severity of diabetic neuropathy among patients with type 2 diabetes mellitus attending Nirmala Multispeciality Hospital, Rewari, Haryana. The findings may help reinforce the importance of stringent glycemic control in preventing the progression of diabetic neuropathy and improving patient outcomes [10].

Objectives

Primary Objective

To determine the association between HbA1c levels and severity of diabetic neuropathy among patients with type 2 diabetes mellitus.

Secondary Objectives

1. To assess the prevalence of different grades of diabetic neuropathy.
2. To evaluate the correlation between duration of diabetes and neuropathy severity.
3. To identify demographic and clinical factors associated with severe diabetic neuropathy.

MATERIALS AND METHODS

Study Design and Setting

This hospital-based observational cross-sectional study was conducted in the Department of Medicine in collaboration with the Department of Obstetrics and Gynaecology at Nirmala Multispecialty Hospital, Rewari, Haryana, India. The study was carried out over a period of eight months from January 2025 to August 2025.

Study Population

The study included patients diagnosed with type 2 diabetes mellitus who attended the outpatient and inpatient departments during the study period and fulfilled the eligibility criteria. Patients presenting with clinical features suggestive of diabetic peripheral neuropathy were screened and enrolled after obtaining written informed consent.

Sample Size

A total of 50 patients with type 2 diabetes mellitus and diabetic neuropathy were included in the study using a consecutive sampling technique.

Inclusion Criteria

- Patients aged 18 years and above.
- Diagnosed cases of type 2 diabetes mellitus according to American Diabetes Association (ADA) criteria.
- Patients with clinical evidence of diabetic peripheral neuropathy.
- Patients willing to participate and provide informed consent.

Exclusion Criteria

- Patients with type 1 diabetes mellitus.
- Peripheral neuropathy due to causes other than diabetes, including chronic alcohol consumption, vitamin B12 deficiency, hypothyroidism, chronic kidney disease, malignancy, or drug-induced neuropathy.
- Patients with acute diabetic emergencies.
- Pregnant women.
- Patients unwilling to participate in the study.

Data Collection Procedure

After obtaining informed consent, detailed demographic and clinical information was recorded using a structured case record form. Information regarding age, sex, duration of diabetes, smoking status, alcohol consumption, history of

hypertension, and current antidiabetic treatment was collected.

A comprehensive general physical examination and systemic examination were performed for all participants. Height and weight were measured using standardized procedures, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m²).

Assessment of Glycemic Control

Venous blood samples were collected under aseptic precautions. Glycated hemoglobin (HbA1c) levels were measured using standardized high-performance liquid chromatography (HPLC)-based methods in the hospital laboratory. HbA1c values were expressed as percentages and used as an indicator of long-term glycemic control over the preceding two to three months.

For analytical purposes, HbA1c levels were also categorized as:

- Good glycemic control: HbA1c < 7.0%
- Moderate glycemic control: HbA1c 7.0–8.9%
- Poor glycemic control: HbA1c ≥ 9.0%

Assessment of Diabetic Neuropathy

Diabetic peripheral neuropathy was assessed clinically using the Michigan Neuropathy Screening Instrument (MNSI). The assessment included evaluation of neuropathic symptoms and physical examination findings such as vibration perception, ankle reflexes, foot appearance, and monofilament sensation.

Based on MNSI scores, neuropathy severity was categorized as:

- Mild neuropathy: Score 2-4
- Moderate neuropathy: Score 5-7
- Severe neuropathy: Score ≥8

Patients were subsequently classified into mild, moderate, and severe neuropathy groups for comparison.

Study Variables

Primary Outcome Variable

- Association between HbA1c levels and severity of diabetic neuropathy.

Secondary Variables

- Age
- Gender
- Duration of diabetes mellitus
- Body mass index (BMI)
- Hypertension status
- Smoking history
- Neuropathy severity score

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) version 26.0. Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. Differences in mean HbA1c levels among neuropathy severity groups were assessed using one-way Analysis of Variance (ANOVA). Pearson's correlation coefficient was used to evaluate the relationship between HbA1c levels and neuropathy severity scores. The Chi-square test was used to analyze associations between categorical variables wherever applicable. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 50 patients with type 2 diabetes mellitus and clinically diagnosed diabetic neuropathy were included in the study. The mean age of the participants was 56.8 ± 9.4 years, with the majority belonging to the age group of 51–60 years. Of the total participants, 29 (58%) were males and 21 (42%) were females, resulting in a male-to-female ratio of 1.38:1. The mean duration of diabetes was 8.7 ± 4.1 years. Hypertension was present in 31 (62%) patients, while the overall mean body mass index (BMI) was 27.4 ± 3.2 kg/m². The mean HbA1c level among all study participants was 8.9 ± 1.7%.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants (n = 50)

Variable	Value
Age (years), Mean ± SD	56.8 ± 9.4
Male, n (%)	29 (58.0)
Female, n (%)	21 (42.0)
Duration of Diabetes (years), Mean ± SD	8.7 ± 4.1
BMI (kg/m²), Mean ± SD	27.4 ± 3.2
Hypertension, n (%)	31 (62.0)
HbA1c (%), Mean ± SD	8.9 ± 1.7

Assessment of neuropathy severity revealed that moderate neuropathy was the most common category, observed in 21 (42%) patients. Mild neuropathy was present in 16 (32%) patients, whereas severe neuropathy was identified in 13 (26%) patients. These findings indicate that nearly two-thirds of the study population had moderate-to-severe neuropathic involvement, highlighting the substantial burden of neuropathy among patients with poorly controlled diabetes.

Table 2: Distribution of Patients According to Severity of Diabetic Neuropathy

Neuropathy Severity	Number (n)	Percentage (%)
Mild	16	32.0
Moderate	21	42.0
Severe	13	26.0
Total	**50**	**100.0**

Comparison of HbA1c levels across different neuropathy severity groups demonstrated a progressive increase in mean HbA1c values with increasing neuropathy severity. Patients with mild neuropathy had a mean HbA1c level of 7.3 ± 0.9%, whereas those with moderate neuropathy had a mean HbA1c level of 8.8 ± 1.2%. The highest mean HbA1c level was observed among patients with severe neuropathy (10.2 ± 1.1%). Analysis of variance (ANOVA) showed a statistically significant difference in HbA1c levels among the three groups ($p < 0.001$), suggesting a strong association between poor glycemic control and increasing neuropathy severity.

Table 3: Comparison of HbA1c Levels According to Neuropathy Severity

Neuropathy Severity	Mean HbA1c (%)	SD
Mild	7.3	0.9
Moderate	8.8	1.2
Severe	10.2	1.1
ANOVA p-value	**<0.001**	

Further analysis was performed to examine the relationship between HbA1c levels and neuropathy severity scores. Pearson's correlation analysis demonstrated a strong positive correlation between HbA1c and neuropathy severity score ($r = 0.71$, $p < 0.001$). This finding indicates that increasing HbA1c levels are associated with worsening neuropathic manifestations. The correlation remained statistically significant, emphasizing the importance of glycemic control in preventing progression of diabetic nerve damage.

Table 4: Correlation Between HbA1c Levels and Neuropathy Severity Score

Variable	Correlation Coefficient (r)	p-value
HbA1c vs Neuropathy Severity Score	0.71	<0.001

The association between duration of diabetes and neuropathy severity was also evaluated. Patients with severe neuropathy had a longer mean duration of diabetes (12.1 ± 3.2 years) compared to those with moderate (8.4 ± 2.7 years) and mild neuropathy (5.6 ± 2.1 years). The difference was statistically significant ($p < 0.001$), indicating that prolonged exposure to hyperglycemia contributes to progressive nerve damage and worsening neuropathic symptoms.

Table 5: Duration of Diabetes According to Neuropathy Severity

Neuropathy Severity	Mean Duration of Diabetes (Years)	SD
Mild	5.6	2.1
Moderate	8.4	2.7
Severe	12.1	3.2
ANOVA p-value	**<0.001**	

Overall, the study findings demonstrated a significant association between elevated HbA1c levels and increasing severity of diabetic neuropathy. Patients with poor glycemic control exhibited more severe neuropathic manifestations, while longer duration of diabetes further contributed to disease progression. These results underscore the importance of maintaining optimal glycemic control to reduce the risk and severity of diabetic neuropathy.

DISCUSSION

Diabetic neuropathy is among the most prevalent and disabling chronic complications of diabetes mellitus, contributing significantly to morbidity, reduced quality of life, foot ulceration, and lower-limb amputation [1,4,17]. The present study evaluated the association between HbA1c levels and the severity of diabetic neuropathy among patients with type 2 diabetes mellitus and demonstrated a strong positive relationship between poor glycemic control and worsening neuropathic manifestations. The findings indicate that patients with higher HbA1c levels are more likely to develop severe neuropathy, emphasizing the critical role of sustained glycemic control in preventing disease progression.

In the present study, the mean HbA1c level among all participants was 8.9 ± 1.7%, reflecting suboptimal glycemic control in a substantial proportion of patients. Furthermore, HbA1c values increased progressively with neuropathy severity, from 7.3% in patients with mild neuropathy to 10.2% in those with severe neuropathy. These findings are consistent with previous studies that have identified chronic hyperglycemia as a major determinant of diabetic neuropathy development and progression [1,7,15]. Dyck et al. reported that the prevalence and severity of neuropathy increase significantly among patients with poor metabolic control [7], while Ang et al. highlighted the importance of maintaining optimal HbA1c levels to minimize the risk of neuropathic complications [15]. The strong positive correlation observed between HbA1c levels and neuropathy severity scores ($r = 0.71$, $p < 0.001$) further supports the role of long-term glycemic exposure in nerve damage. Similar observations have been reported by Callaghan et al., who demonstrated that worsening glycemic control is associated with increased neuropathic symptoms and functional impairment [3]. Bansal et al. also identified elevated HbA1c as an independent predictor of peripheral diabetic neuropathy among patients with type 2 diabetes mellitus [14]. These findings suggest that HbA1c may serve not only as a marker of glycemic status but also as a useful predictor of neuropathy severity.

The pathophysiological mechanisms linking hyperglycemia to neuropathy are multifactorial. Chronic elevation of blood glucose leads to increased oxidative stress, activation of the polyol pathway, accumulation of advanced glycation end products, mitochondrial dysfunction, and inflammatory responses that damage peripheral nerves [5,6]. Feldman et al. described the central role of oxidative injury and metabolic dysregulation in diabetic nerve degeneration [5], while Vinik et al. emphasized the contribution of vascular insufficiency and endothelial dysfunction to progressive neuronal damage [6]. More recent evidence has shown that inflammation and impaired nerve repair mechanisms also contribute substantially to neuropathy progression [18,19].

The present study also demonstrated that patients with severe neuropathy had a significantly longer duration of diabetes than those with mild or moderate disease. This observation is in agreement with earlier studies that identified disease duration as one of the strongest predictors of diabetic neuropathy [2,7]. Tesfaye et al. reported that both prolonged diabetes duration and associated vascular risk factors contribute significantly to neuropathy development [12]. Continuous exposure to hyperglycemia over many years leads to cumulative neuronal injury, explaining the higher frequency of severe neuropathy among patients with longstanding diabetes.

In addition to hyperglycemia, several studies have highlighted the role of other risk factors in neuropathy progression. Hypertension, obesity, dyslipidemia, smoking, and metabolic syndrome have all been implicated in accelerating nerve damage among diabetic individuals [12,14]. The relatively high prevalence of hypertension observed in the present study may have contributed to the severity of neuropathic manifestations, although this association was not specifically evaluated.

The predominance of moderate and severe neuropathy in the present study is comparable to findings from previous epidemiological investigations. Population-based studies have reported a substantial burden of neuropathic symptoms among patients with type 2 diabetes, particularly among those with poor glycemic control and prolonged disease duration [11,13]. Abbott et al. reported that painful diabetic neuropathy affects a significant proportion of diabetic patients and remains underrecognized despite its adverse impact on daily functioning and quality of life [11]. Similarly, Ziegler et al. observed increased neuropathic symptoms among individuals with impaired glucose metabolism and diabetes, highlighting the close relationship between metabolic control and nerve dysfunction [13].

The findings of the present study are further supported by evidence from major clinical trials. The Diabetes Control and Complications Trial (DCCT) demonstrated that intensive glycemic control significantly reduced the incidence and progression of diabetic neuropathy compared with conventional treatment [8]. Long-term follow-up through the Epidemiology of Diabetes Interventions and Complications (EDIC) study confirmed that the benefits of intensive glycemic control persist for many years, even after glycemic differences between treatment groups diminish [20]. Similarly, the United Kingdom Prospective Diabetes Study (UKPDS) established that tighter glycemic control reduces the risk of microvascular complications in patients with type 2 diabetes mellitus [9]. These landmark studies strongly support the association observed in the present investigation.

From a clinical perspective, the results emphasize the importance of routine HbA1c monitoring and early neuropathy screening among diabetic patients. Current guidelines recommend regular assessment of glycemic status and evaluation for microvascular complications to facilitate timely intervention [10,16]. Since HbA1c testing is inexpensive, standardized, and widely available, it may serve as a valuable tool for identifying individuals at increased risk of severe neuropathy. Early optimization of glycemic control, along with management of associated cardiovascular risk factors, may help delay neuropathy progression and improve patient outcomes [1,16,18].

The present study has certain limitations. The relatively small sample size and single-center design may limit the generalizability of the findings. Additionally, the cross-sectional nature of the study precludes establishing a definitive causal relationship between HbA1c levels and neuropathy severity. Nerve conduction studies and other objective neurophysiological assessments were not routinely performed, which may have provided additional insights regarding neuropathy severity. Nevertheless, the study provides valuable evidence regarding the relationship between glycemic control and diabetic neuropathy in a real-world clinical setting.

Overall, the findings of the present study reinforce the growing body of evidence demonstrating that poor glycemic control is strongly associated with increased severity of diabetic neuropathy. Elevated HbA1c levels, longer duration of diabetes, and the presence of associated vascular risk factors collectively contribute to neuropathy progression. Therefore, maintaining optimal glycemic control remains the cornerstone of preventing or delaying neuropathic complications and improving the long-term health outcomes of patients with type 2 diabetes mellitus [1,8,9,10].

Limitations

1. Small sample size.
2. Single-center study.
3. Cross-sectional design limits causal inference.
4. Nerve conduction studies were not performed in all patients.

CONCLUSION

HbA1c levels are significantly associated with the severity of diabetic neuropathy among patients with type 2 diabetes mellitus. Poor glycemic control is linked to more severe neuropathic manifestations. Regular monitoring of HbA1c and early intervention to achieve optimal glycemic targets may help reduce the burden of diabetic neuropathy and improve patient outcomes.

REFERENCES

1. Pop-Busui R, Boulton AJM, Feldman EL, Bril V, Freeman R, Malik RA, et al. Diabetic neuropathy: a position statement by the American Diabetes Association. Diabetes Care. 2017;40(1):136-54.
2. Tesfaye S, Boulton AJM, Dyck PJ, Freeman R, Horowitz M, Kempler P, et al. Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. Diabetes Care. 2010;33(10):2285-93.
3. Callaghan BC, Cheng HT, Stables CL, Smith AL, Feldman EL. Diabetic neuropathy: clinical manifestations and current treatments. Lancet Neurol. 2012;11(6):521-34.
4. Boulton AJM, Vinik AI, Arezzo JC, Bril V, Feldman EL, Freeman R, et al. Diabetic neuropathies: a statement by the American Diabetes Association. Diabetes Care. 2005;28(4):956-62.
5. 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.
6. Vinik AI, Casellini C, Nevoret ML. Diabetic neuropathy. Endocrinol Metab Clin North Am. 2013;42(4):747-87.
7. Dyck PJ, Kratz KM, Karnes JL, Litchy WJ, Klein R, Pach JM, et al. The prevalence by staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: the Rochester Diabetic Neuropathy Study. Neurology. 1993;43(4):817-24.
8. The Diabetes Control and Complications Trial Research Group. The effect of intensive diabetes therapy on the development and progression of neuropathy. Ann Intern Med. 1995;122(8):561-8.
9. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet. 1998;352(9131):837-53.
10. American Diabetes Association Professional Practice Committee. Standards of Care in Diabetes—2025. Diabetes Care. 2025;48(Suppl 1):S1-S350.
11. Abbott CA, Malik RA, van Ross ER, Kulkarni J, Boulton AJM. Prevalence and characteristics of painful diabetic neuropathy in a large community-based diabetic population in the United Kingdom. Diabetes Care. 2011;34(10):2220-4.
12. Tesfaye S, Chaturvedi N, Eaton SEM, Ward JD, Manes C, Ionescu-Tîrgovişte C, et al. Vascular risk factors and diabetic neuropathy. N Engl J Med. 2005;352(4):341-50.
13. Ziegler D, Rathmann W, Dickhaus T, Meisinger C, Mielck A; KORA Study Group. Neuropathic pain in diabetes, prediabetes and normal glucose tolerance: the MONICA/KORA Augsburg Surveys. Pain Med. 2009;10(2):393-400.
14. Bansal D, Gudala K, Muthyala H, Esam HP, Nayakallu R, Bhansali A. Prevalence and risk factors of development of peripheral diabetic neuropathy in type 2 diabetes mellitus in a tertiary care setting. J Diabetes Investig. 2014;5(6):714-21.

15. Ang L, Jaiswal M, Martin C, Pop-Busui R. Glucose control and diabetic neuropathy: lessons from recent large clinical trials. Curr Diab Rep. 2014;14(9):528.
16. American Diabetes Association. Microvascular complications and foot care: Standards of Medical Care in Diabetes. Diabetes Care. 2024;47(Suppl 1):S244-S259.
17. Hicks CW, Selvin E. Epidemiology of peripheral neuropathy and lower extremity disease in diabetes. Curr Diab Rep. 2019;19(10):86.
18. Sloan G, Selvarajah D, Tesfaye S. Pathogenesis, diagnosis and clinical management of diabetic sensorimotor peripheral neuropathy. Nat Rev Endocrinol. 2021;17(7):400-20.
19. Said G. Diabetic neuropathy-a review. Nat Clin Pract Neurol. 2007;3(6):331-40.
20. Albers JW, Herman WH, Pop-Busui R, Feldman EL, Martin CL, Cleary PA, et al. Effect of prior intensive insulin treatment during the Diabetes Control and Complications Trial on peripheral neuropathy in type 1 diabetes during the Epidemiology of Diabetes Interventions and Complications study. Diabetes Care. 2010;33(5):1090-6.

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