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Association of glycaemic control with peripheral neuropathy severity among patients with type 2 diabetes mellitus: A cross-sectional observational study.

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ABSTRACT

Background:

To assess the association between glycaemic control and peripheral neuropathy severity among patients with type 2 diabetes mellitus.

Methods:

This hospital-based cross-sectional observational study was conducted in the Department of General Medicine, Government Medical College/Government General Hospital, Nizamabad, Telangana, India, from March to August 2025. A total of 100 eligible patients with type 2 diabetes mellitus were included and analysed. Demographic and clinical variables, fasting blood glucose, postprandial blood glucose, and HbA1c were recorded. Glycaemic control was classified as good (<7.0%), suboptimal (7.0-8.9%), or poor (≥9.0%). Peripheral neuropathy was clinically graded as absent, mild, moderate, or severe. Associations were evaluated using the chi-square test, and correlation analysis assessed the relationship between HbA1c and neuropathy severity.

Results:

The mean age was 52.6 ± 10.8 years, and the mean diabetes duration was 8.7 ± 5.1 years. Good, suboptimal, and poor glycaemic control occurred in 28 (28.0%), 38 (38.0%), and 34 (34.0%) patients, respectively. Peripheral neuropathy was present in 77 (77.0%); 32 (32.0%) had mild, 33 (33.0%) moderate, and 12 (12.0%) severe neuropathy. Neuropathy severity differed significantly across HbA1c categories (χ^2=26.93, p<0.001). Moderate-to-severe neuropathy was more frequent in patients with HbA1c ≥9.0% than in those with HbA1c <9.0% (70.6% versus 31.8%; χ^2=13.63, p<0.001). Mean HbA1c increased from 6.9 ± 0.8% in patients without neuropathy to 10.1 ± 1.1% in those with severe neuropathy (p<0.001), and HbA1c correlated positively with neuropathy severity (r=0.58, p<0.001).

Conclusion:

Poor glycaemic control was significantly associated with greater peripheral neuropathy severity. Higher HbA1c and longer diabetes duration identified patients with an increased neuropathy burden.

Keywords: *Type 2 diabetes mellitus; glycaemic control; HbA1c; diabetic peripheral neuropathy; neuropathy severity*
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INTRODUCTION

Type 2 diabetes mellitus is a major chronic metabolic disorder with a growing clinical and public health burden. Its long-term complications arise from sustained hyperglycaemia, metabolic dysregulation, endothelial dysfunction, oxidative stress, and inflammatory injury. Among these complications, diabetic peripheral neuropathy is one of the most frequent and disabling manifestations. It commonly presents as distal symmetric polyneuropathy, beginning in the feet and progressing proximally in a length-dependent pattern. Patients can remain asymptomatic in early stages, while advanced disease produces numbness,

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burning pain, paraesthesia, loss of protective sensation, gait instability, ulceration, infection, and non-traumatic amputation risk [1-3].

The reported burden of peripheral neuropathy varies widely across clinical settings because of differences in population characteristics, diagnostic tools, diabetes duration, and glycaemic exposure. Reviews and population studies show that neuropathy prevalence among adults with diabetes ranges from low to very high estimates depending on age, duration of disease, and the method used for assessment [4,5]. Indian studies also describe a substantial burden of neuropathy among patients with type 2 diabetes, including newly diagnosed and long-standing disease groups [6-9]. This variation supports the need for local hospital-based data, particularly from tertiary care settings that serve patients with diverse metabolic profiles.

Chronic hyperglycaemia plays a central role in the pathogenesis of diabetic neuropathy. Excess glucose promotes advanced glycation end-products, polyol pathway activation, mitochondrial dysfunction, impaired nerve blood flow, axonal degeneration, and Schwann cell injury. These mechanisms are often accompanied by hypertension, dyslipidaemia, obesity, smoking, and longer diabetes duration, which together increase neuronal vulnerability [3,10]. HbA1c is widely used as an indicator of average glycaemic exposure and remains a practical marker for assessing long-term glycaemic control in routine clinical care. Several studies have linked higher HbA1c levels, HbA1c variability, and prolonged diabetes duration with the presence or severity of diabetic peripheral neuropathy [11-14].

Although the relationship between hyperglycaemia and neuropathy is biologically plausible, the strength of this association differs across populations and care settings. Hospital-based studies are useful because they identify high-risk clinical groups requiring early intervention. Grading neuropathy severity in relation to glycaemic control also helps clinicians move beyond simple presence-or-absence screening and focus on risk stratification. Therefore, the present study was conducted to assess the association between glycaemic control and peripheral neuropathy severity among patients with type 2 diabetes mellitus. The specific objectives were to describe the glycaemic profile and neuropathy severity distribution, compare neuropathy severity across HbA1c categories, and identify clinical factors associated with moderate-to-severe neuropathy.

METHODOLOGY

Study design and setting

This hospital-based cross-sectional observational study was conducted in the Department of General Medicine at Government Medical College/Government General Hospital, Nizamabad, Telangana, India. The study was carried out from March 2025 to August 2025. The hospital provides outpatient and inpatient services for diabetes, hypertension, metabolic disorders, and related complications. The design assessed glycaemic status and neuropathy severity at a single clinical encounter.

Study population and sampling

The study included 100 patients with type 2 diabetes mellitus attending the medicine outpatient department or admitted under general medicine care. Consecutive eligible patients were enrolled. Inclusion criteria were age 30 years or above, established type 2 diabetes mellitus of at least one year duration, availability of recent glycaemic assessment, and willingness to provide informed consent. Patients with type 1 diabetes mellitus, gestational diabetes, acute critical illness, chronic alcohol dependence, known vitamin B12 deficiency, hypothyroidism, chronic kidney disease with uraemic neuropathy, chemotherapy exposure, neurological disorders unrelated to diabetes, or traumatic peripheral nerve injury were excluded.

Sample size

The sample size was estimated using the single-proportion formula $n=Z^2pq/d^2$. Using an expected diabetic peripheral neuropathy prevalence of 39.3% from a South Indian study [8], 95% confidence level, and 10% absolute precision, the minimum sample size was approximately 92. After allowing for non-response and incomplete records, a final sample size of 100 patients was considered adequate for this observational study.

Data collection and study variables

A structured proforma recorded age, sex, diabetes duration, smoking status, height, weight, body mass index, hypertension, and dyslipidaemia. Fasting blood glucose, postprandial blood glucose, and HbA1c values were obtained from hospital laboratory records. HbA1c was classified as good control (<7.0%), suboptimal control (7.0-8.9%), and poor control (≥9.0%). Peripheral neuropathy was assessed by history of sensory symptoms and bedside neurological examination, including vibration perception, pinprick sensation, ankle reflexes, and protective sensation

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using standard clinical principles recommended for diabetic neuropathy screening [1,2]. Neuropathy severity was graded as absent, mild, moderate, or severe based on symptoms, sensory impairment, reflex involvement, and loss of protective sensation.

Bias control

Selection bias was reduced by enrolling consecutive eligible patients. Measurement bias was minimized through a uniform proforma, predefined definitions, and the same assessment approach. Laboratory values were taken from recent hospital reports to maintain consistency. Patients with alternative causes of peripheral neuropathy were excluded to improve attribution to diabetes-related neuropathy.

Statistical analysis

Data were entered into a spreadsheet and analysed using standard statistical methods. Continuous variables were expressed as mean ± standard deviation. Categorical variables were expressed as frequency and percentage. The association between HbA1c category and neuropathy severity was assessed using the chi-square test. Factors associated with moderate-to-severe neuropathy were also analysed using chi-square statistics. Correlation between HbA1c level and neuropathy severity score was assessed using correlation analysis. A p-value <0.05 was considered statistically significant.

Ethical considerations

Institutional Ethics Committee approval was obtained from Government Medical College, Nizamabad, Telangana, India, before initiation of the study. Written informed consent was obtained from all participants. Confidentiality was maintained by anonymising patient data during analysis and reporting.

RESULTS

A total of 100 patients with type 2 diabetes mellitus were included in the final analysis. The mean age of the study participants was 52.6 ± 10.8 years. Most patients belonged to the 50-59 years age group, followed by those aged ≥60 years. Males constituted 58 patients (58.0%), while females accounted for 42 patients (42.0%). The mean duration of diabetes mellitus was 8.7 ± 5.1 years. Hypertension and dyslipidaemia were present in 42.0% and 38.0% of patients, respectively. The mean HbA1c level was 8.3 ± 1.6%, indicating suboptimal glycaemic control in a large proportion of the study population (Table 1).

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Table 1: Baseline Clinical Characteristics of the Study Participants (N = 100)

Characteristic	n	%
Age group		
<40 years	14	14.0
40-49 years	24	24.0
50-59 years	36	36.0
≥60 years	26	26.0
Sex		
Male	58	58.0
Female	42	42.0
Duration of diabetes mellitus		
<5 years	22	22.0
5-10 years	42	42.0
>10 years	36	36.0
Body mass index category		
Normal	44	44.0
Overweight	38	38.0
Obese	18	18.0
Hypertension	42	42.0
Dyslipidaemia	38	38.0
Current smoker	24	24.0

Note. BMI = body mass index.

The mean fasting blood glucose level was 164.8 ± 42.6 mg/dL, and the mean postprandial blood glucose level was 236.4 ± 64.8 mg/dL. Based on HbA1c levels, good glycaemic control was observed in 28 patients (28.0%), suboptimal control in 38 patients (38.0%), and poor control in 34 patients (34.0%). Peripheral neuropathy was present in 77 patients (77.0%). Mild neuropathy was observed in 32 patients (32.0%), moderate neuropathy in 33 patients (33.0%), and severe neuropathy in 12 patients (12.0%) (Table 2).

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Table 2: Glycaemic Control and Peripheral Neuropathy Profile

Characteristic	n	%
HbA1c category		
Good control, <7.0%	28	28.0
Suboptimal control, 7.0-8.9%	38	38.0
Poor control, ≥9.0%	34	34.0
Peripheral neuropathy status		
Absent	23	23.0
Present	77	77.0
Peripheral neuropathy severity		
No neuropathy	23	23.0
Mild neuropathy	32	32.0
Moderate neuropathy	33	33.0
Severe neuropathy	12	12.0

Note. HbA1c = glycated haemoglobin.

A clear increase in peripheral neuropathy severity was observed with worsening glycaemic control. Among patients with good glycaemic control, 14 patients (50.0%) had no neuropathy, and none had severe neuropathy. In contrast, among patients with poor glycaemic control, only 2 patients (5.9%) had no neuropathy, while 16 patients (47.1%) had moderate neuropathy and 8 patients (23.5%) had severe neuropathy. The association between HbA1c category and peripheral neuropathy severity was statistically significant (χ^2=26.93, p<0.001) (Table 3).

Table 3: Association Between Glycaemic Control and Peripheral Neuropathy Severity

HbA1c category	No neuropathy	Mild neuropathy	Moderate neuropathy	Severe neuropathy	Total
Good control, <7.0%	14 (50.0)	10 (35.7)	4 (14.3)	0 (0.0)	28
Suboptimal control, 7.0-8.9%	7 (18.4)	14 (36.8)	13 (34.2)	4 (10.5)	38
Poor control, ≥9.0%	2 (5.9)	8 (23.5)	16 (47.1)	8 (23.5)	34
Total	**23**	**32**	**33**	**12**	**100**

Note. Values are n (%), with percentages calculated within each HbA1c category. HbA1c = glycated haemoglobin; $\chi^2(6)=26.93$; p<.001.

The mean HbA1c level increased progressively with increasing neuropathy severity. Patients without neuropathy had a mean HbA1c of 6.9 ± 0.8%, while those with mild, moderate, and severe neuropathy had mean HbA1c values of 7.8 ± 1.1%, 8.9 ± 1.2%, and 10.1 ± 1.1%, respectively. This difference was statistically significant (p<0.001). A positive correlation was observed between HbA1c level and neuropathy severity score (r=0.58, p<0.001), indicating that poorer glycaemic control was associated with greater neuropathy burden.

Moderate-to-severe neuropathy was more frequent among patients aged ≥60 years, those with diabetes duration >10 years, poor glycaemic control, hypertension, and dyslipidaemia. Poor glycaemic control showed the strongest

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association, with moderate-to-severe neuropathy present in 70.6% of patients with HbA1c ≥9.0% compared with 31.8% of those with HbA1c <9.0% (χ^2=13.63, p<0.001). Longer duration of diabetes mellitus was also significantly associated with moderate-to-severe neuropathy (χ^2=13.58, p<0.001) (Table 4).

Table 4: Factors Associated with Moderate-to-Severe Peripheral Neuropathy

Variable	Moderate/severe neuropathy present	Moderate/severe neuropathy absent	χ^2 value	p-value
Age ≥60 years	17/26 (65.4)	9/26 (34.6)	5.90	.015
Diabetes duration >10 years	25/36 (69.4)	11/36 (30.6)	13.58	< .001
HbA1c ≥9.0%	24/34 (70.6)	10/34 (29.4)	13.63	< .001
Hypertension	26/42 (61.9)	16/42 (38.1)	8.36	.004
Dyslipidaemia	23/38 (60.5)	15/38 (39.5)	5.97	.015
BMI ≥25 kg/m²	30/56 (53.6)	26/56 (46.4)	3.78	.052

Note. Values are n/N (%). BMI = body mass index; HbA1c = glycated haemoglobin.

Overall, the findings demonstrated a significant graded association between poor glycaemic control and increasing severity of peripheral neuropathy among patients with type 2 diabetes mellitus. Patients with higher HbA1c values and longer duration of diabetes had a greater burden of moderate-to-severe neuropathy.

DISCUSSION

The present study demonstrated a significant graded association between glycaemic control and peripheral neuropathy severity among patients with type 2 diabetes mellitus. Peripheral neuropathy was present in 77.0% of participants, and moderate-to-severe neuropathy accounted for 45.0% of the sample. The burden observed in this hospital-based cohort was higher than several community-based and population-based reports, including the Chennai Urban Rural Epidemiology Study and other Indian studies that reported lower prevalence estimates [6-8]. This difference is clinically understandable because tertiary care settings commonly receive patients with longer diabetes duration, higher metabolic burden, and established complications.

Poor glycaemic control showed the strongest association with moderate-to-severe neuropathy. Patients with HbA1c ≥9.0% had a substantially greater frequency of moderate-to-severe neuropathy than those with HbA1c <9.0%. Mean HbA1c increased stepwise from patients without neuropathy to those with severe neuropathy, and the positive correlation between HbA1c and neuropathy severity supported a dose-response pattern. These findings are consistent with studies showing that higher HbA1c levels and long-term glycaemic exposure are linked to diabetic peripheral neuropathy in type 2 diabetes [11,14]. HbA1c variability has also been associated with neuropathy risk and severity, suggesting that both chronic hyperglycaemia and unstable glycaemic exposure contribute to nerve injury [12,13].

Longer duration of diabetes was significantly associated with moderate-to-severe neuropathy in the present study. This result agrees with previous reports showing that duration of type 2 diabetes is an important determinant of peripheral neuropathy [7,9]. Chronic exposure to hyperglycaemia promotes metabolic and microvascular injury through advanced glycation, oxidative stress, polyol pathway activity, and impaired endoneurial blood flow [3,10]. These mechanisms produce progressive sensory fibre dysfunction and loss of protective sensation. Therefore, patients with longer disease duration require periodic foot and neurological assessment even when symptoms are mild or absent.

Hypertension and dyslipidaemia were also significantly associated with moderate-to-severe neuropathy. This finding is relevant because diabetic neuropathy is not driven by glycaemia alone. Vascular risk factors, obesity, smoking, and lipid abnormalities contribute to microvascular dysfunction and neural ischemia [2,4]. In the present study, BMI ≥25 kg/m² showed a borderline association, indicating that excess adiposity could still contribute to neuropathy risk through insulin resistance and inflammatory pathways. These observations support a comprehensive risk-reduction approach rather than isolated glucose management.

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Generalizability

The findings are most applicable to adult patients with type 2 diabetes attending tertiary care medicine services in similar Indian hospital settings. Because the study was conducted in a government teaching hospital, the results reflect routine clinical profiles seen in public-sector care. Extrapolation to community populations, newly diagnosed diabetes groups, and primary-care settings should be cautious because neuropathy burden, glycaemic status, and comorbidity patterns differ across these populations.

Conclusion

This cross-sectional observational study showed a significant graded association between glycaemic control and peripheral neuropathy severity among patients with type 2 diabetes mellitus. Poor glycaemic control, reflected by HbA1c ≥9.0%, was strongly associated with moderate-to-severe neuropathy. Mean HbA1c increased progressively with neuropathy severity, and HbA1c showed a positive correlation with neuropathy score. Longer diabetes duration, older age, hypertension, and dyslipidaemia further increased neuropathy burden. These findings highlight the need for routine neuropathy screening, regular HbA1c monitoring, and integrated management of metabolic and vascular risk factors in patients with type 2 diabetes. Early identification supports timely foot care, counselling, prevention of disabling complications, and routine follow-up in resource-limited clinical settings.

Limitations

The cross-sectional design limited temporal interpretation between glycaemic control and neuropathy severity. The study was conducted in a single tertiary care hospital with a sample size of 100, restricting wider representativeness. Neuropathy was assessed clinically without routine nerve conduction studies. Glycaemic control was based on available HbA1c values rather than repeated longitudinal measurements. Only current clinical records were used for associated variables.

Recommendations

All patients with type 2 diabetes should undergo periodic peripheral neuropathy screening using a structured symptom review and bedside foot examination. Patients with HbA1c ≥9.0%, diabetes duration above 10 years, hypertension, dyslipidaemia, obesity, or smoking history require closer follow-up. Diabetes clinics should integrate HbA1c monitoring, blood pressure control, lipid management, weight reduction, smoking cessation, foot-care education, and early referral for advanced neuropathy. Hospitals should maintain standard diabetic foot assessment records. Future multicentre prospective studies with nerve conduction evaluation and longitudinal HbA1c tracking are required to confirm risk patterns and guide prevention strategies in Indian populations.

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Abbreviations

BMI: Body mass index
DPN: Diabetic peripheral neuropathy
FBG: Fasting blood glucose
HbA1c: Glycated haemoglobin
IEC: Institutional Ethics Committee
PPBG: Postprandial blood glucose
SPSS: Statistical Package for the Social Sciences
T2DM: Type 2 diabetes mellitus

Source of funding

The study has no funding.

Conflict of interest

The authors declare no conflict of interest.

Author contributions

RD-Concept and design of the study, results interpretation, review of literature, and preparing the first draft of the manuscript. Statistical analysis and interpretation, revision of the manuscript. **MSK** -Concept and design of the study, results interpretation, review of literature, and preparing the first draft of the manuscript, revision of the manuscript. **BI** -Review of literature and preparing the first draft of the manuscript. Statistical analysis and interpretation.

Data availability

De-identified data underlying the findings of this study are available upon reasonable request.

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