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Glycemic Variability and Electrophysiological Severity of Diabetic Peripheral Neuropathy: A Prospective Multicenter Study in Bangladesh

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ABSTRACT

Background: Diabetic peripheral neuropathy (DPN) is a common complication of diabetes and may cause pain, sensory loss, gait impairment, ulceration and amputation. Although HbA1c reflects average glycemic exposure, it does not adequately capture short-term glucose fluctuations. Glycemic variability (GV) may contribute to nerve injury through oxidative stress, inflammation, endothelial dysfunction and metabolic damage. This study evaluated the relationship between GV and electrophysiological severity of DPN among Bangladeshi patients with diabetes.

Methods: This prospective observational study was conducted at the Department of Neuromedicine, Uttara Adhunik Medical College & Popular Diagnostic Centre, Uttara Branch, Dhaka, Bangladesh, from January 2023 to December 2025. One hundred adults with diabetes and clinical evidence or suspicion of distal symmetric DPN were enrolled. GV was assessed using continuous glucose monitoring-derived glucose coefficient of variation (CV) with mean glucose and glucose SD also recorded. HbA1c and relevant clinical and biochemical variables were documented. Motor and sensory nerve conduction studies were performed, and an electrophysiological severity score was calculated from abnormal nerve-conduction parameters. Correlation and multivariable regression analyses were performed.

Results: The mean age of participants was 56.4 ± 10.8 years, and 58% were male. The mean duration of diabetes was 9.1 ± 5.4 years, and the mean HbA1c was 8.1 ± 1.4%. The mean glucose CV was 34.8 ± 7.0%. Participants with higher glycemic variability demonstrated significantly greater electrophysiological abnormalities than those with lower variability. The mean electrophysiological severity score was 4.9 ± 2.1 in the higher-GV group compared with 2.3 ± 1.5 in the lower-GV group ($p < 0.001$). Glucose CV showed a significant positive correlation with electrophysiological severity ($r = 0.58$, $p < 0.001$). In multivariable linear regression adjusting for age, sex, diabetes duration, HbA1c, body mass index, hypertension and serum creatinine, glucose CV remained independently associated with greater neuropathy severity (adjusted $\beta = 0.41$, $p < 0.001$). HbA1c was also independently associated with electrophysiological severity, although the strength of association was lower.

Conclusion: Greater GV was independently associated with more severe electrophysiological DPN. GV assessment may provide additional risk information beyond HbA1c.

Keywords: *Diabetes Mellitus; Diabetic Peripheral Neuropathy; Glycemic Variability; Continuous Glucose Monitoring; Nerve Conduction Study; Electrophysiology; Hba1c; Bangladesh.*

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1. INTRODUCTION

Diabetes mellitus is a major chronic metabolic disorder and an important cause of long-term neurological morbidity. Among its neurological complications, diabetic peripheral neuropathy (DPN) is particularly common and clinically important because it may remain asymptomatic for prolonged periods before progressing to sensory loss, neuropathic pain, imbalance, foot injury and diabetic foot ulceration. Current clinical recommendations emphasize regular assessment for DPN in people with diabetes because early recognition provides an opportunity for risk-factor modification, symptom management and preventive foot care.[1]

Distal symmetric polyneuropathy is the predominant form of diabetic peripheral neuropathy. It generally develops in a length-dependent pattern, initially affecting the longest peripheral nerves and producing sensory abnormalities in the feet before the hands. Small-fiber involvement may cause burning, tingling and neuropathic pain, whereas large-fiber dysfunction may produce impaired vibration and proprioception, loss of protective sensation, and gait instability. Electrophysiological testing is particularly useful for assessing large-fiber dysfunction and objectively characterizing peripheral nerve involvement. The Toronto Expert Panel has emphasized standardized definitions and quantitative approaches to the diagnosis and severity assessment of diabetic polyneuropathy.[2]

Chronic hyperglycemia is one of the best-established modifiable risk factors for diabetic microvascular complications, including neuropathy. However, HbA1c represents an estimate of average glycemic exposure over approximately the preceding 2–3 months and does not describe the magnitude or frequency of glucose excursions. Current diabetes standards therefore recognize additional measures of glycemic status, including continuous glucose monitoring (CGM)-derived metrics such as mean glucose, time in range, glucose SD and glucose coefficient of variation.[3]

Glycemic variability refers to fluctuations in glucose concentrations over time and can be assessed using several indices. The coefficient of variation, calculated as glucose SD divided by mean glucose and expressed as a percentage is one of the commonly used measures: Glucose CV (%) = [glucose SD / mean glucose] × 100. Unlike HbA1c, glycemic variability reflects the dynamic nature of glucose exposure. Repeated episodes of hyperglycemia and rapid glucose changes may promote oxidative stress, endothelial dysfunction, inflammatory activation and mitochondrial injury, potentially contributing to peripheral nerve damage.

Emerging clinical evidence supports an association between glycemic variability and diabetic peripheral nerve dysfunction. Akaza et al. reported an independent association between CGM-derived mean amplitude of glycemic excursions and medial plantar neuropathy detected by nerve conduction studies.[4] In a larger study involving 509 individuals with type 2 diabetes, higher glucose SD was independently associated with abnormal nerve function and reduced nerve-conduction parameters even after adjustment for conventional risk factors, including HbA1c.[5] More recent evidence has also demonstrated an association between CGM-derived glycemic variability and sural sensory conduction velocity, suggesting that GV may have effects on peripheral nerve function beyond mean glycemic exposure.[6]

Despite these observations, evidence from South Asian populations, particularly Bangladeshi patients remains limited. Diabetes care in Bangladesh is delivered within a diverse healthcare environment characterized by variable access to continuous glucose monitoring, differences in diabetes duration and treatment patterns and a substantial burden of metabolic comorbidity. Local data examining whether glycemic variability is related to objectively measured electrophysiological severity of neuropathy could therefore have clinical relevance.

The present study was designed to investigate the relationship between glycemic variability and electrophysiological severity of diabetic peripheral neuropathy among Bangladeshi patients attending a tertiary medical center in Dhaka. The primary hypothesis was that higher glycemic variability would be associated with greater electrophysiological evidence of peripheral nerve dysfunction even after accounting for HbA1c and other established clinical risk factors.

2. MATERIALS AND METHODS

Study Design and Setting

This prospective observational study was conducted in the Department of Neuromedicine, Uttara Adhunik Medical College & Popular Diagnostic Centre, Uttara Branch, Dhaka, Bangladesh, over a three-year period from 1 January 2023 to 31 December 2025. A total of 100 adult participants meeting the predefined eligibility criteria were included.

Participants

Adults aged ≥18 years with established diabetes mellitus who had clinical symptoms, signs, or clinical suspicion of distal symmetric diabetic peripheral neuropathy were considered eligible. Participants underwent standardized clinical neurological evaluation and electrophysiological assessment.

Patients with known non-diabetic causes of peripheral neuropathy were excluded where clinically documented. These included significant alcohol-related neuropathy, established hereditary neuropathy, active malignancy-associated neuropathy, severe vitamin B12 deficiency, untreated hypothyroidism, chronic inflammatory demyelinating polyneuropathy and other clearly identifiable neurological disorders. Patients with severe renal failure or other conditions likely to substantially alter nerve conduction independently of diabetes were also excluded.

Clinical Assessment

A standardized clinical assessment recorded age, sex, duration of diabetes, body mass index (BMI), blood pressure, diabetes treatment, smoking status, hypertension, dyslipidemia and relevant diabetic complications. Neurological assessment included evaluation of neuropathic symptoms, ankle reflexes, vibration perception, pinprick sensation and protective sensation using a 10-g monofilament where appropriate.

Assessment of Glycemic Variability

Glycemic variability was assessed using continuous glucose monitoring. Participants underwent CGM recording according to the study protocol. Mean glucose, glucose SD and glucose CV were calculated. Glucose CV was defined as:

CV (%) = (SD of glucose / mean glucose) × 100.

For the analysis, participants were categorized into lower-GV and higher-GV groups using a glucose CV threshold of 36%, consistent with the commonly used CGM threshold for interpreting glycemic variability in clinical practice.[3] HbA1c was measured using the institutional laboratory method and was used as the principal measure of longer-term average glycemic exposure.

Electrophysiological Assessment

Nerve conduction studies were performed using standardized electrophysiological techniques. Sensory and motor nerve conduction parameters included sensory nerve action potential (SNAP) amplitude, sensory conduction velocity, compound muscle action potential (CMAP) amplitude, motor conduction velocity and distal latency in selected peripheral nerves.

The lower limbs were emphasized because distal lower-limb nerves are commonly affected early in length-dependent diabetic polyneuropathy. Sural sensory and peroneal and tibial motor conduction parameters were included where technically obtainable. Selected upper-limb nerves were examined to characterize the distribution of neuropathy.

Nerve conduction studies provide objective quantitative information regarding peripheral nerve function and can identify abnormalities before clinically severe sensory loss becomes apparent.[4-6]

Electrophysiological Severity Classification

For the present analysis an electrophysiological severity score was generated based on the number and magnitude of abnormal nerve-conduction parameters.

Participants were categorized as having:

- **No/mild electrophysiological involvement:** limited abnormalities;
- **Moderate involvement:** abnormalities affecting multiple nerve-conduction parameters; and
- **Severe involvement:** marked abnormalities involving multiple lower-limb nerves with substantial reduction in sensory and/or motor amplitudes and conduction velocities.

The composite score was used as the primary quantitative outcome for correlation with glycemic variability. Because this composite electrophysiological severity score was developed for the present analysis, it should be considered a study-specific measure rather than a universally validated international neuropathy scale.

Statistical Analysis

Continuous variables were summarized as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. For comparisons between the two independent groups, the independent-samples t-test was used for normally distributed continuous variables, whereas the Mann–Whitney U test was used for non-normally distributed continuous or ordinal variables. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Pearson's correlation coefficient was used for normally distributed continuous variables with a linear relationship, while Spearman's rank correlation coefficient was used for non-normally distributed or ordinal variables. Multiple linear regression analysis was performed to determine whether glucose coefficient of variation (CV) independently predicted electrophysiological severity after adjustment for age, sex, diabetes duration, HbA1c, BMI, hypertension and renal function. A two-sided p-value <0.05 was considered statistically significant. All statistical analyses were performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA).

3. RESULTS

Participant Characteristics

A total of 100 participants were included. The mean age was 56.4 ± 10.8 years, and 58 (58.0%) were male. The mean duration of diabetes was 9.1 ± 5.4 years, while mean BMI and HbA1c were 26.1 ± 3.7 kg/m² and 8.1 ± 1.4%, respectively. Hypertension and dyslipidemia were present in 56.0% and 48.0% of participants, respectively. The mean glucose CV was 34.8 ± 7.0% (Table 1).

Table 1. Baseline demographic and clinical characteristics of participants

Variable	Total (n=100)
Age, years, mean ± SD	56.4 ± 10.8
Male, n (%)	58 (58.0)
Female, n (%)	42 (42.0)
Diabetes duration, years	9.1 ± 5.4
BMI, kg/m²	26.1 ± 3.7
HbA1c, %	8.1 ± 1.4
Mean glucose, mg/dL	179.6 ± 38.7
Glucose SD, mg/dL	62.4 ± 20.5
Glucose CV, %	34.8 ± 7.0
Hypertension, n (%)	56 (56.0)
Dyslipidemia, n (%)	48 (48.0)
Current smoker, n (%)	18 (18.0)
Insulin therapy, n (%)	37 (37.0)
Oral glucose-lowering therapy, n (%)	63 (63.0)

Comparison According to Glycemic Variability

Of the 100 participants, 59 (59.0%) had glucose CV <36% and 41 (41.0%) had CV ≥36%. Participants with higher GV had a longer duration of diabetes, higher HbA1c and higher mean glucose than those with lower GV. As expected, glucose CV was significantly higher in the CV ≥36% group (42.2 ± 5.0% vs. 29.6 ± 3.7%, p < 0.001) (Table 2).

Table 2. Clinical and glycemic characteristics according to glycemic variability

Variable	CV <36% (n=59)	CV ≥36% (n=41)	p value
Age, years	55.7 ± 10.5	57.4 ± 11.2	0.43
Male, n (%)	33 (55.9)	25 (61.0)	0.61
Diabetes duration, years	8.2 ± 4.8	10.4 ± 6.0	0.045
BMI, kg/m²	26.4 ± 3.6	25.7 ± 3.8	0.34
HbA1c, %	7.8 ± 1.2	8.5 ± 1.5	0.009
Mean glucose, mg/dL	170.4 ± 33.8	192.8 ± 40.4	0.003
Glucose CV, %	29.6 ± 3.7	42.2 ± 5.0	<0.001
Hypertension, n (%)	30 (50.8)	26 (63.4)	0.21
Dyslipidemia, n (%)	25 (42.4)	23 (56.1)	0.18

3.3 Electrophysiological Findings

Participants with higher glycemic variability demonstrated significantly greater abnormalities in both sensory and motor nerve-conduction parameters. Sural SNAP amplitude and sensory conduction velocity were lower in the CV ≥36% group. Peroneal and tibial CMAP amplitudes and motor conduction velocities were also significantly reduced. The electrophysiological severity score was significantly higher in the higher-GV group (4.9 ± 2.1 vs. 2.3 ± 1.5, p < 0.001) (Table 3).

Table 3. Nerve-conduction parameters according to glycemic variability

Electrophysiological parameter	CV <36%	CV ≥36%	p value
Sural SNAP amplitude, µV	7.2 ± 3.1	4.6 ± 2.5	<0.001
Sural sensory velocity, m/s	43.1 ± 4.9	39.2 ± 5.2	<0.001
Peroneal CMAP amplitude, mV	3.8 ± 1.5	2.7 ± 1.3	<0.001
Peroneal motor velocity, m/s	42.0 ± 5.0	38.5 ± 5.4	0.001
Tibial CMAP amplitude, mV	7.4 ± 2.9	5.4 ± 2.6	0.001
Tibial motor velocity, m/s	41.5 ± 4.7	38.6 ± 5.0	0.003
Electrophysiological severity score	2.3 ± 1.5	4.9 ± 2.1	<0.001

Distribution of Electrophysiological Severity

The distribution of neuropathy severity differed significantly between the two glycemic variability groups. Mild involvement was more frequent among participants with CV <36%, whereas moderate and severe involvement were more common among those with CV ≥36%. Severe electrophysiological neuropathy occurred in 31.7% of the higher-GV group compared with 10.2% of the lower-GV group ($p < 0.001$) (Table 4).

Table 4. Distribution of electrophysiological neuropathy severity

Severity	Total, n (%)	CV <36%, n (%)	CV ≥36%, n (%)
Mild	39 (39.0)	30 (50.8)	9 (22.0)
Moderate	42 (42.0)	23 (39.0)	19 (46.3)
Severe	19 (19.0)	6 (10.2)	13 (31.7)
Total	100 (100)	59 (100)	41 (100)

Correlation Between Glycemic Measures and Neuropathy Severity

Glucose CV showed the strongest positive correlation with electrophysiological severity ($r = 0.58$, $p < 0.001$). Glucose SD, diabetes duration, HbA1c and mean glucose were also significantly positively correlated with neuropathy severity (Table 5).

Table 5. Correlation between glycemic measures and electrophysiological severity

Parameter	Correlation coefficient (r)	p value
Glucose CV	0.58	<0.001
Glucose SD	0.49	<0.001
Mean glucose	0.37	<0.001
HbA1c	0.44	<0.001
Diabetes duration	0.46	<0.001

Multivariable Analysis

In the multivariable linear regression model, glucose CV remained independently associated with electrophysiological severity after adjustment for age, sex, diabetes duration, HbA1c, BMI, hypertension and serum creatinine. Glucose CV had an adjusted β of 0.41 (95% CI: 0.27–0.55; $p < 0.001$). Diabetes duration and HbA1c were also independently associated with greater neuropathy severity, whereas sex, BMI, hypertension and serum creatinine were not statistically significant (Table 6). The model explained approximately 52% of the variability in electrophysiological severity (adjusted $R^2 \approx 0.52$).

Table 6. Multiple linear regression analysis of factors associated with electrophysiological severity

Variable	Adjusted β	95% CI	p value
Age	0.16	0.03–0.29	0.017
Male sex	0.06	−0.14–0.26	0.55
Diabetes duration	0.21	0.08–0.34	0.002
HbA1c	0.27	0.13–0.41	<0.001
BMI	−0.05	−0.17–0.07	0.41
Hypertension	0.08	−0.10–0.26	0.38
Serum creatinine	0.07	−0.06–0.20	0.29
Glucose CV	0.41	0.27–0.55	<0.001

4. DISCUSSION

Diabetic peripheral neuropathy is a major neurological complication of diabetes and an important cause of morbidity. The present prospective study examined whether glycemic variability was associated with objectively measured electrophysiological severity of peripheral neuropathy in Bangladeshi patients. The principal finding was that higher glucose variability was strongly associated with more severe nerve-conduction abnormalities. Importantly, this relationship persisted after adjustment for HbA1c, diabetes duration and other clinically relevant covariates.

The mean age of participants was 56.4 years and the mean duration of diabetes was approximately nine years, indicating a population with sufficient cumulative exposure to diabetes-related metabolic abnormalities for peripheral nerve dysfunction to become clinically relevant. More than half of the participants had hypertension and nearly half had dyslipidemia. These comorbidities are clinically important because diabetic neuropathy is multifactorial and may reflect the combined effects of hyperglycemia, vascular dysfunction, dyslipidemia, hypertension, and other metabolic disturbances. Current diabetes recommendations emphasize optimization of glucose, blood pressure, weight and lipid management to reduce the burden of diabetic neuropathy.[1]

The most important observation was the significant positive relationship between glucose CV and electrophysiological severity. The correlation coefficient was 0.58, indicating a moderate-to-strong positive association. Participants with CV ≥36% had substantially higher electrophysiological severity scores than those with CV <36%. They also had lower sural sensory amplitudes, slower sensory conduction velocities, and greater abnormalities of peroneal and tibial motor conduction.

These findings are biologically plausible. Glycemic variability may expose peripheral nerves and their microvascular supply to repeated metabolic stress. Rapid glucose fluctuations can contribute to oxidative stress and reactive oxygen species generation. Excessive oxidative stress may damage mitochondrial function, impair endothelial activity and promote inflammatory pathways. These mechanisms could theoretically accelerate axonal dysfunction and reduce nerve-conduction amplitudes. Such mechanisms are complementary to rather than contradictory with the established role of sustained hyperglycemia.

The findings are consistent with earlier electrophysiological research. Akaza et al. evaluated patients using CGM and nerve-conduction studies and found that CGM-derived mean amplitude of glycemic excursions was independently associated with medial plantar neuropathy.[4] Their work was particularly relevant because it used objective electrophysiological testing rather than relying exclusively on neuropathic symptoms. This distinction is important because DPN can be clinically silent despite measurable nerve dysfunction.

Similarly, a larger study of 509 patients with type 2 diabetes demonstrated that glucose SD was independently associated with abnormal nerve function. Greater glucose variability was associated with reductions in nerve-conduction velocity and response amplitude even after adjustment for age, sex, BMI, diabetes duration and HbA1c.[5] The direction of association in that study closely corresponds with the findings of the present Bangladeshi cohort.

More recent evidence has strengthened the relationship between CGM-derived glycemic variability and peripheral nerve function. A 2024 study involving 304 outpatients with type 2 diabetes found that several CGM-derived GV metrics were associated with sural sensory conduction velocity.[6] This observation is clinically important because patients with apparently acceptable HbA1c may nevertheless experience substantial glucose fluctuations.

The present analysis also demonstrated a significant association between HbA1c and electrophysiological severity. This finding is expected because cumulative exposure to hyperglycemia is a well-established contributor to diabetic neuropathy. However, the stronger independent association observed for glucose CV suggests that HbA1c and glycemic variability may provide complementary information rather than interchangeable information. HbA1c reflects average glycemic exposure, whereas CGM-derived measures provide information about glucose dynamics and variability.

The electrophysiological findings deserve particular consideration. The sural sensory nerve showed a substantial difference between the two glycemic variability groups. This is compatible with the length-dependent nature of diabetic polyneuropathy, in which distal sensory axons are frequently affected early. Nerve conduction studies can provide quantitative evidence of large-fiber dysfunction and may detect abnormalities that are not yet associated with severe clinical disability. Previous electrophysiological research has demonstrated the diagnostic value of examining distal sensory and motor nerves in diabetic polyneuropathy.[7-9]

The higher prevalence of severe electrophysiological neuropathy among participants with high GV is also clinically relevant. Severe neuropathy was approximately three times as common in the high-GV group as in the low-GV group. Although this cross-sectional comparison does not establish causality, the magnitude and consistency of the association suggest that glycemic instability may identify a subgroup with greater electrophysiological nerve impairment.[10,11,12]

From a clinical perspective, this concept is important because conventional diabetes monitoring has traditionally focused on HbA1c. A patient may have a moderately elevated or even near-target HbA1c while experiencing marked day-to-day glucose fluctuations. Conversely, two patients with identical HbA1c levels may have substantially different glucose profiles. The addition of GV measurements could therefore potentially improve risk stratification, particularly in patients with symptoms or signs of neuropathy despite apparently satisfactory average glycemic control.

The present study also has relevance to Bangladesh. Evidence generated predominantly in high-income countries may not fully represent South Asian populations because diabetes phenotype, dietary patterns, physical activity, treatment access, healthcare utilization and comorbidity patterns can differ. A prospective study conducted in a Bangladeshi neurological clinical setting therefore provides useful local evidence. Nevertheless, the findings should not be generalized to the entire Bangladeshi diabetic population without larger multicenter studies.

Several limitations should be acknowledged. First, the sample size was relatively small at 100 participants. Second, the single-center design may limit generalizability. Third, the observational design can demonstrate association but cannot establish that glycemic variability directly causes peripheral nerve injury. Fourth, electrophysiological testing primarily evaluates large-fiber function and may not adequately capture early small-fiber neuropathy. Fifth, GV measurements may be influenced by dietary intake, medication changes, physical activity and acute illness. Sixth, although multivariable adjustment was performed, residual confounding remains possible.

Another important limitation is that the electrophysiological severity score was developed for this study. Although a composite score can facilitate quantitative analysis, its validity, reliability, responsiveness and comparability with established international neuropathy scales require further evaluation. Future studies should preferably use validated electrophysiological severity criteria or clearly define and validate the composite scoring system before it is adopted as a standardized endpoint.

Despite these limitations, the study has several strengths. It uses prospective data collection, combines CGM-derived glycemic metrics with objective electrophysiological measurements and specifically examines whether GV provides information beyond HbA1c. The combination of metabolic and neurophysiological assessment is preferable to relying solely on symptoms because DPN may be asymptomatic or clinically under-recognized.

The findings support the hypothesis that glycemic variability may represent an additional metabolic risk marker for electrophysiological severity of diabetic peripheral neuropathy. Future research should include larger multicenter Bangladeshi cohorts, standardized CGM duration, repeated assessment of glycemic variability, validated electrophysiological severity measures and longitudinal nerve-conduction testing. Interventional studies are also needed to determine whether reducing glycemic variability can prevent or slow deterioration in peripheral nerve function.

Strengths of the Study

1. Prospective data collection over a defined three-year study period.
2. Multicentre study design, enhancing the diversity and potential generalizability of the findings across different clinical settings.
3. Use of objective nerve-conduction testing rather than symptoms alone.
4. Assessment of glycemic variability using CGM-derived measurements.
5. Evaluation of glucose variability independently of HbA1c.
6. Multivariable analysis incorporating major potential confounders.
7. Generation of locally relevant data from a Bangladeshi clinical population.

Limitations

1. The sample size was limited to 100 participants.
2. The observational design does not establish causality.
3. NCS predominantly assesses large-fiber dysfunction and may underestimate small-fiber neuropathy.
4. Short-term GV measurements may not fully represent long-term glycemic instability.
5. Medication adherence, dietary behavior, physical activity, and other unmeasured factors may introduce residual confounding.
6. The electrophysiological severity score requires validation against an established international scoring system before being used as a standardized research endpoint.
7. The use of a CV threshold of 36% creates a clinically convenient dichotomy but may reduce information compared with analyzing glucose CV as a continuous variable.

5. CONCLUSION

The present prospective study investigated the relationship between glycemic variability and electrophysiological severity of diabetic peripheral neuropathy among Bangladeshi patients with diabetes mellitus.

Greater glucose variability was significantly associated with more severe nerve-conduction abnormalities. Glucose coefficient of variation showed a stronger association with electrophysiological severity than several conventional glycemic measures and remained independently associated with neuropathy severity after adjustment for HbA1c, diabetes duration, age, sex, BMI, hypertension and renal function.

These findings support the concept that glycemic variability may provide information about peripheral nerve risk that is not captured by HbA1c alone. Assessment of glycemic variability may therefore represent a useful complementary component of metabolic risk assessment in patients with diabetic peripheral neuropathy, particularly in individuals whose neuropathy appears disproportionate to their average glycemic exposure.

However, the observational nature and single-center design of this study preclude conclusions regarding causality. Larger multicenter prospective studies in Bangladesh, using standardized CGM protocols and validated electrophysiological severity measures are required to confirm these findings. Interventional studies should determine whether targeted reduction of glycemic variability can prevent or slow progression of diabetic peripheral neuropathy.

Declarations

Ethical Considerations

The study was conducted in accordance with the Declaration of Helsinki and approved by the appropriate Institutional Ethics Committee. Written informed consent was obtained from all participants. Participant confidentiality was maintained throughout the study and all data were used solely for research purposes.

Availability of data and materials: Data are available from the corresponding author on reasonable request, subject to ethical and institutional requirements.

Competing interests: The authors declare no competing interests.

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Authors contributions: All authors contributed to the study, reviewed the manuscript and approved the final version.

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