

Original Article

Association of Glycemic Status with Functional Balance and Fall Risk in Older Adults with Type 2 Diabetes Mellitus with and Without Diabetic Neuropathy: A Cross-Sectional Study

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Received: 19 January 2026

Revised: 03 February 2026

Accepted: 04 March 2026

Published: 15 March 2026

HOW TO CITE

Fiza Butt et al. 2026. Association of Glycemic Status with Functional Balance and Fall Risk in Older Adults with Type 2 Diabetes Mellitus with and Without Diabetic Neuropathy: A Cross-Sectional Study. *Journal of Health, Wellness and Community Research*. 4, 5 (Mar. 2026), 1–10. DOI:<https://doi.org/10.61919/ram8y943>

ARTICLE INFORMATION

Author Contributions	Concept: FB, SJ; Design: FB, SJ; Data Collection: NU, EHK, EB; Analysis: FB, SJ; Drafting: FB, SJ, NU, EHK, EB
Ethical Approval	The study was approved by research ethical board of Riphah International University, Islamabad, Pakistan
Informed Consent	Written informed consent was obtained from all participants
Conflict of Interest	The authors declare no conflict of interest.
Funding	No external funding.
Data Availability	Available from the corresponding author on reasonable request.
AI Declaration	No generative AI was used for conceptualization, analysis, or content generation. AI use was limited to language refinement, and the authors remain fully responsible for the accuracy, originality, validity, and integrity of the manuscript's content.

ABSTRACT

Background: Type 2 diabetes mellitus and diabetic peripheral neuropathy may impair balance and mobility, but the association between glycemic status and functional performance according to neuropathy status remains unclear. **Objective:** To determine the associations of HbA1c and random blood glucose with functional balance and fall-risk-related mobility in adults with T2DM with and without diabetic peripheral neuropathy. **Methods:** This cross-sectional correlational study was conducted from March 2024 to February 2025 at healthcare and community settings in the twin cities of Pakistan. A total of 182 participants aged ≥ 50 years with T2DM for >5 years were included, with 91 participants in each neuropathy group. Glycemic status was assessed using HbA1c and random blood glucose; balance and mobility were assessed using the Berg Balance Scale (BBS) and Timed Up and Go (TUG) test. Between-group comparisons, Pearson correlations, Fisher r -to- z comparisons, and Holm adjustment for multiple testing were applied. **Results:** Participants with neuropathy had higher HbA1c ($8.40 \pm 1.97\%$ vs $6.98 \pm 1.76\%$; $p < 0.001$), higher random glucose (316.17 ± 110.66 vs 259.67 ± 91.94 mg/dL; $p < 0.001$), and lower BBS scores (34.30 ± 11.05 vs 38.28 ± 9.26 ; $p = 0.009$). TUG times did not differ significantly (15.99 ± 9.09 vs 13.75 ± 8.08 seconds; $p = 0.081$). No within-group glycemic-functional correlation remained significant after Holm adjustment. However, the HbA1c-BBS correlation differed between neuropathy groups ($r = 0.12$ vs -0.26 ; Fisher $z = 2.57$; Holm-adjusted $p = 0.041$). **Conclusion:** Glycemic measures were not consistently associated with functional balance or mobility, although the relationship between HbA1c and balance differed according to neuropathy status. These findings support considering neuropathy and other functional determinants when evaluating balance in adults with T2DM. **Keywords:** Type 2 diabetes mellitus; HbA1c; diabetic peripheral neuropathy; balance; Berg Balance Scale; Timed Up and Go; fall risk.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major chronic health condition associated with progressive metabolic, vascular, neurological, and functional complications that can adversely affect mobility and independence, particularly with advancing age. The global burden of diabetes has increased substantially over recent decades, creating important challenges for health systems and long-term disability prevention (1). Pakistan is also considerably affected by

T2DM; a recent systematic review and meta-analysis of Pakistani studies estimated a pooled prevalence of approximately 10%, emphasizing the continuing national public-health burden of the disease (2). As survival with diabetes increases, attention is increasingly required not only for conventional metabolic complications but also for functional consequences such as impaired balance, mobility limitation, and susceptibility to falls.

Diabetic peripheral neuropathy (DPN) is among the most clinically important chronic complications of T2DM because of its effects on peripheral sensation, proprioception, motor function, and postural control. DPN is characterized by peripheral nerve dysfunction attributable to diabetes after exclusion of other potential causes and may involve sensory loss, altered vibration perception, reduced protective sensation, neuropathic symptoms, and motor impairment (3). Its burden in Pakistan is substantial. A systematic review and meta-analysis involving 8,487 people with diabetes from 19 Pakistani studies estimated an overall pooled prevalence of DPN of 43.16%, with higher prevalence reported among individuals with a longer duration of diabetes and considerable regional variation (4). Functionally, DPN can compromise the sensory feedback required for effective postural adjustments and may coexist with muscle weakness, altered gait mechanics, and reduced proprioceptive control. Systematic-review evidence has consequently shown that DPN is associated with impaired postural balance, altered gait, limitations in activities of daily living, and increased susceptibility to falls (5).

The relationship between diabetes and falls is nevertheless multifactorial and extends beyond clinically apparent neuropathy. A recent systematic review and meta-analysis reported that older adults with diabetes have a greater likelihood of falling than those without diabetes and identified impaired mobility and other diabetes-related factors as important contributors to fall susceptibility (6). Earlier systematic evidence similarly demonstrated an increased risk of falls among older adults with diabetes (7). Glycemic disturbances may contribute to this relationship through several pathways. Chronic hyperglycemia may promote microvascular and neurological complications that progressively impair sensory and motor function, whereas acute hypoglycemia can produce dizziness, weakness, confusion, impaired coordination, and loss of consciousness. Severe hypoglycemia has been associated with falls among older adults with diabetes, highlighting the complexity of the relationship between glycemia and functional safety (8). Consequently, contemporary diabetes management in older adults emphasizes individualized glycemic targets that take account of functional status, comorbidity, frailty, treatment burden, and hypoglycemia risk rather than applying a uniform HbA1c target to every patient (9). Longitudinal evidence has also suggested that diabetes-related complications and glycemic characteristics may be associated with recurrent falls, further indicating that glycemic status should be considered within a broader clinical context rather than as an isolated determinant of fall risk (10).

Objective assessment of balance and mobility is therefore important in people with T2DM, particularly among those with established or suspected neuropathy. The Berg Balance Scale (BBS) is a widely used clinical measure of functional balance comprising a series of static and dynamic functional tasks and has demonstrated utility in the assessment of older adults (11). The Timed Up and Go (TUG) test evaluates functional mobility through a sequence of standing, walking, turning, returning, and sitting and is commonly used as a clinical screening measure for mobility impairment and increased fall susceptibility (12). Although both instruments are clinically accessible, balance assessment in people with T2DM remains heterogeneous. A systematic review of clinical balance measures in T2DM identified considerable variation in assessment approaches and emphasized the importance of evaluating the multiple components contributing to balance dysfunction (13). BBS and TUG therefore provide complementary information: BBS captures performance across several functional balance tasks, whereas TUG primarily reflects integrated functional mobility.

Evidence from Pakistan has also demonstrated the clinical importance of addressing balance impairment in diabetic populations. Task-oriented balance training has been reported to improve functional balance and reduce fall-related risk among people with diabetes in a Pakistani clinical setting (14). Similarly, proprioceptive training has demonstrated beneficial effects on balance among individuals with diabetic neuropathy (15). These studies support the relevance of balance-focused rehabilitation but primarily address responses to therapeutic interventions rather than the relationship between metabolic status and functional performance. Consequently, they do not establish whether glycemic measures themselves are associated with balance or mobility impairment, particularly when patients are considered according to neuropathy status.

More recent intervention evidence reinforces the importance of identifying factors associated with impaired balance before substantial functional decline occurs. A systematic review and meta-analysis of randomized controlled trials reported that exercise interventions can improve dynamic balance in people with T2DM, with supervised and multicomponent exercise approaches demonstrating favorable effects (16). Evidence focusing specifically on individuals with T2DM and peripheral neuropathy has likewise suggested that exercise- and fall-prevention-oriented interventions may improve modifiable functional risk factors, although the available evidence remains heterogeneous (17). These findings support the integration of balance and mobility assessment into diabetes management, while also highlighting the need to understand which metabolic and neurological characteristics are associated with poorer functional performance.

Despite the established relationships among T2DM, DPN, impaired balance, mobility limitations, and falls, the association between current glycemic status and functional balance remains incompletely characterized. HbA1c reflects longer-term glycemic exposure, whereas a random blood glucose measurement represents glycemia at a single point in time; these measures may therefore demonstrate different relationships with functional outcomes. Furthermore, the presence of DPN may influence the observed association between glycemic status and physical function because established sensory and motor impairment may become an important determinant of balance independent of a single contemporary glycemic measurement. Conversely, individuals without clinically identified neuropathy may still demonstrate functional impairment associated with metabolic abnormalities or other diabetes-related factors. Existing Pakistani literature has focused largely on diabetes and neuropathy prevalence or on rehabilitation interventions, with comparatively limited evidence examining glycemic measures in relation to standardized balance and mobility outcomes separately among individuals with and without DPN.

Therefore, the present cross-sectional study aimed to determine the associations of glycemic status, assessed using HbA1c and random blood glucose, with functional balance measured by the Berg Balance Scale and functional mobility/fall-risk-related performance measured by the Timed Up and Go test among adults with T2DM. These associations were examined separately in participants with and without diabetic peripheral neuropathy to characterize whether different patterns of association were observed according to neuropathy status.

MATERIAL AND METHODS

A cross-sectional correlational study was conducted to examine the associations of glycemic status with functional balance and fall-risk-related mobility among adults with type 2 diabetes mellitus (T2DM), with analyses performed separately according to the presence or absence of diabetic peripheral neuropathy (DPN). The observational cross-sectional design was selected because glycemic measures and functional outcomes were assessed within the same study period, allowing the strength and direction of contemporaneous associations to be examined without implying temporal or causal relationships. The study was conducted from March 2024 to February 2025 at POF Hospital, Hanif Medical Complex, and community-based settings in the twin cities of Pakistan.

The target population comprised male and female adults with T2DM who were aged 50 years or older and had been diagnosed with diabetes for more than five years. Participants were eligible if they met the predefined study criteria for classification into neuropathy or non-neuropathy groups using the Michigan Neuropathy Screening Instrument (MNSI) and Douleur Neuropathique 4 (DN4) assessment. Individuals were excluded if they had severe mobility limitations, including amputation; uncontrolled medical conditions such as human immunodeficiency virus infection, cancer, or autoimmune disease; neurological disorders likely to substantially affect balance, including stroke, Parkinson's disease, multiple sclerosis, or seizure disorders; or recent active trauma. Participants were recruited using non-probability purposive sampling from the participating healthcare facilities and community settings. Eligible individuals were approached, informed about the study procedures, and enrolled after providing informed consent.

The required sample size was determined using a correlational sample-size calculator. A total of 182 participants were recruited and included in the analysis, comprising 91 participants classified as having diabetic neuropathy and 91 classified as not having neuropathy. Equal numbers were therefore analyzed in the two neuropathy-status groups. Because recruitment was purposive and the groups were constructed for comparative correlational analysis, the proportion of participants classified with neuropathy was not intended to represent the population prevalence of DPN.

Data collection followed a standardized sequence. Demographic and relevant clinical information was first recorded, after which neuropathy status was assessed using the MNSI and DN4. The MNSI assessment comprised a 15-item history questionnaire together with a physical examination that included foot inspection, assessment of vibration sensation, ankle reflexes, and monofilament testing. According to the predefined scoring criteria used in the study, an MNSI score of ≥ 7 was considered indicative of neuropathic findings, whereas a score < 7 was considered non-neuropathic. The DN4 questionnaire was also administered, with a score of ≥ 4 considered indicative of neuropathic symptoms and a score ≤ 3 considered non-neuropathic. These assessments were used to support classification of participants according to neuropathy status before analysis of glycemic and functional measures.

Glycemic status was evaluated using glycated hemoglobin (HbA1c) and random blood glucose. HbA1c was recorded as a percentage from available laboratory findings and was used as the principal indicator of longer-term glycemic status. Random blood glucose was measured using a glucometer and recorded in mg/dL to provide a contemporaneous measure of blood glucose at the time of assessment. HbA1c and random blood glucose were treated as continuous exposure variables in the statistical analyses.

Functional balance was assessed using the Berg Balance Scale (BBS), a 14-item clinical instrument comprising functional static and dynamic balance tasks. Each item is scored from 0 to 4, giving a maximum total score of 56, with higher scores indicating better functional balance (11). Functional mobility and fall-risk-related performance were assessed using the Timed Up and Go (TUG) test. Participants were instructed to rise from a chair, walk the prescribed test distance, turn, return to the chair, and sit down, and the time required to complete the task was recorded in seconds. Longer completion times were interpreted as poorer functional mobility and greater fall-risk-related impairment. The TUG is an established clinical measure of basic functional mobility in older adults (12). BBS score and TUG completion time were treated as continuous outcome variables.

To reduce measurement variability, participants underwent the assessments in a consistent sequence using predefined study criteria and established clinical measures. Neuropathy status was determined before the primary subgroup correlation analyses, and glycemic and functional variables were recorded using the same measurement framework in both study groups. The study did not include an intervention, and no treatment was assigned or altered as part of the research procedures. Because the investigation was cross-sectional, all statistical findings were interpreted as associations rather than evidence of causation.

Data were analyzed using IBM SPSS Statistics version 25. Continuous variables were summarized using means and standard deviations, whereas categorical variables were described using frequencies and percentages. Descriptive statistics were calculated for demographic characteristics and for HbA1c, random blood glucose, BBS, and TUG measurements. Glycemic and functional measures were summarized separately for participants with and without neuropathy. Pearson's correlation coefficient (r) was used to evaluate the associations of HbA1c with BBS, HbA1c with TUG, random blood glucose with BBS, and random blood glucose with TUG separately within the neuropathy and non-neuropathy groups. Correlation coefficients were interpreted according to their direction and magnitude, and statistical significance was evaluated using two-sided p -values with a threshold of $p < 0.05$. The reported analyses were unadjusted, and no multivariable confounder-adjusted model was performed. Similarly, neuropathy-stratified correlations were used to describe subgroup-specific association patterns; no formal interaction analysis between glycemic status and neuropathy status was included in the reported analysis. All 182 participants contributed to the reported analyses, with 91 participants represented in each neuropathy subgroup for the correlation analyses.

Ethical approval was obtained from the relevant ethical review committee before commencement of the study. Participation was voluntary, and informed consent was obtained before data collection. Participant information was handled confidentially, and the assessment procedures were conducted with the intention of minimizing risk and avoiding harm to participants.

RESULTS

A total of 182 participants with type 2 diabetes mellitus were included, comprising 91 participants with diabetic neuropathy and 91 without neuropathy. The mean age was 65.86 ± 8.41 years, with an age range of 50–85 years. Of the total sample, 105 (57.7%) were female and 77 (42.3%) were male. Overall HbA1c ranged from 4.00% to 14.50%,

with a mean of $7.69 \pm 2.00\%$, whereas random blood glucose ranged from 100 to 579 mg/dL, with a mean of 287.92 ± 105.33 mg/dL (Table 1).

Table 1. Overall demographic and glycemic characteristics of the study participants

Characteristic	Overall (N=182)
Age, years, mean $\pm$ SD	65.86 $\pm$ 8.41
Age range, years	50–85
Female, n (%)	105 (57.7)
Male, n (%)	77 (42.3)
Neuropathy, n (%)	91 (50.0)
Non-neuropathy, n (%)	91 (50.0)
HbA1c, %, mean $\pm$ SD	7.69 $\pm$ 2.00
HbA1c range, %	4.00–14.50
Random blood glucose, mg/dL, mean $\pm$ SD	287.92 $\pm$ 105.33
Random blood glucose range, mg/dL	100–579

SD, standard deviation; HbA1c, glycated hemoglobin.

Participants with neuropathy had a mean HbA1c of $8.40 \pm 1.97\%$ compared with $6.98 \pm 1.76\%$ among participants without neuropathy, corresponding to a mean difference of 1.42 percentage points (95% CI 0.87 to 1.97; $p < 0.001$) and a Hedges' g of 0.76. Random blood glucose was also higher in the neuropathy group, with a between-group mean difference of 56.50 mg/dL (95% CI 26.73 to 86.27; $p < 0.001$; Hedges' $g = 0.55$). Mean BBS score was 3.98 points lower in participants with neuropathy (95% CI -6.96 to -1.00 ; $p = 0.009$; Hedges' $g = -0.39$). TUG completion time was 2.24 seconds longer in the neuropathy group; however, the confidence interval included the null value (95% CI -0.28 to 4.76 ; $p = 0.081$; Hedges' $g = 0.26$) (Table 2).

Table 2. Glycemic and functional measures according to diabetic neuropathy status

Variable	Neuropathy (n=91), mean $\pm$ SD	Non-neuropathy (n=91), mean $\pm$ SD	Mean difference (95% CI)	p-value	Hedges' g
HbA1c, %	8.40 $\pm$ 1.97	6.98 $\pm$ 1.76	1.42 (0.87 to 1.97)	<0.001	0.76
Random blood glucose, mg/dL	316.17 $\pm$ 110.66	259.67 $\pm$ 91.94	56.50 (26.73 to 86.27)	<0.001	0.55
BBS score	34.30 $\pm$ 11.05	38.28 $\pm$ 9.26	-3.98 (-6.96 to -1.00)	0.009	-0.39
TUG, seconds	15.99 $\pm$ 9.09	13.75 $\pm$ 8.08	2.24 (-0.28 to 4.76)	0.081	0.26

Mean differences are calculated as neuropathy minus non-neuropathy. Between-group comparisons were calculated using Welch's independent samples t-test. Hedges' g represents the standardized mean difference. CI, confidence interval; SD, standard deviation; BBS, Berg Balance Scale; TUG, Timed Up and Go; HbA1c, glycated hemoglobin.

Within the neuropathy group, HbA1c showed a weak positive correlation with BBS ($r = 0.12$, 95% CI -0.09 to 0.32 ; $p = 0.257$) and essentially no correlation with TUG ($r = -0.04$, 95% CI -0.24 to 0.17 ; $p = 0.707$). Random blood glucose was also weakly correlated with BBS ($r = 0.15$, 95% CI -0.06 to 0.35 ; $p = 0.156$) and showed negligible correlation with TUG ($r = 0.03$, 95% CI -0.18 to 0.23 ; $p = 0.778$).

Among participants without neuropathy, HbA1c was weakly negatively correlated with BBS ($r = -0.26$, 95% CI -0.44 to -0.06 ; unadjusted $p = 0.013$). After adjustment for the eight subgroup correlation tests using the Holm procedure, the corresponding adjusted p -value was 0.103. HbA1c showed a weak positive correlation with TUG ($r = 0.18$, 95% CI -0.03 to 0.37 ; $p = 0.088$). Random blood glucose showed little evidence of association with either BBS ($r = -0.08$, 95% CI -0.28 to 0.13 ; $p = 0.451$) or TUG ($r = 0.00$, 95% CI -0.21 to 0.21 ; $p = 1.000$). None of the eight within-group correlations remained statistically significant after Holm adjustment (Table 3).

Table 3. Correlations between glycemic measures and functional outcomes according to neuropathy status

Glycemic measure	Functional outcome	Group	n	Pearson r	95% CI	p-value	Holm-adjusted p-value
HbA1c	BBS	Neuropathy	91	0.12	-0.09 to 0.32	0.257	1.000
HbA1c	BBS	Non-neuropathy	91	-0.26	-0.44 to -0.06	0.013	0.103
HbA1c	TUG	Neuropathy	91	-0.04	-0.24 to 0.17	0.707	1.000
HbA1c	TUG	Non-neuropathy	91	0.18	-0.03 to 0.37	0.088	0.614
Random blood glucose	BBS	Neuropathy	91	0.15	-0.06 to 0.35	0.156	0.935
Random blood glucose	BBS	Non-neuropathy	91	-0.08	-0.28 to 0.13	0.451	1.000

Glycemic measure	Functional outcome	Group	n	Pearson r	95% CI	p-value	Holm-adjusted p-value
Random blood glucose	TUG	Neuropathy	91	0.03	−0.18 to 0.23	0.778	1.000
Random blood glucose	TUG	Non-neuropathy	91	0.00	−0.21 to 0.21	1.000	1.000

95% confidence intervals for Pearson correlation coefficients were calculated using Fisher's *r*-to-*z* transformation. Two-sided *p*-values were calculated from the reported correlation coefficients and group denominators. Holm-adjusted *p*-values account for the eight subgroup correlation analyses. CI, confidence interval; BBS, Berg Balance Scale; TUG, Timed Up and Go; HbA1c, glycated hemoglobin.

Formal comparison of the correlation coefficients between neuropathy groups demonstrated a difference in the association between HbA1c and BBS. The correlation was positive in the neuropathy group ($r=0.12$) and negative in the non-neuropathy group ($r=-0.26$), with Fisher $z=2.57$ and an unadjusted *p*-value of 0.010. This difference remained statistically significant after Holm adjustment across the four between-group correlation comparisons (adjusted $p=0.041$). No evidence of a between-group difference was observed for the association of HbA1c with TUG (Fisher $z=-1.47$, $p=0.141$), random blood glucose with BBS (Fisher $z=1.53$, $p=0.125$), or random blood glucose with TUG (Fisher $z=0.20$, $p=0.842$) (Table 4).

Table 4. Comparison of glycemic–functional correlations between neuropathy and non-neuropathy groups

Glycemic measure	Functional outcome	Neuropathy <i>r</i>	Non-neuropathy <i>r</i>	Fisher <i>z</i>	<i>p</i> -value	Holm-adjusted <i>p</i> -value
HbA1c	BBS	0.12	−0.26	2.57	0.010	0.041
HbA1c	TUG	−0.04	0.18	−1.47	0.141	0.375
Random blood glucose	BBS	0.15	−0.08	1.53	0.125	0.375
Random blood glucose	TUG	0.03	0.00	0.20	0.842	0.842

Independent correlation coefficients were compared using Fisher's *r*-to-*z* transformation. Holm-adjusted *p*-values account for the four between-group correlation comparisons. BBS, Berg Balance Scale; TUG, Timed Up and Go; HbA1c, glycated hemoglobin.

Overall, participants with diabetic neuropathy demonstrated higher HbA1c and random blood glucose concentrations and lower BBS scores than participants without neuropathy, whereas the between-group difference in TUG time was smaller and statistically imprecise. Within-group correlation analyses provided limited evidence for direct linear relationships between the glycemic measures and functional outcomes after accounting for multiple testing. However, formal comparison of the subgroup correlations indicated that the relationship between HbA1c and BBS differed between neuropathy and non-neuropathy groups, with opposing directions of association. No corresponding between-group differences were demonstrated for HbA1c with TUG or for random blood glucose with either functional outcome.

DISCUSSION

The present study examined whether glycemic status was associated with functional balance and fall-risk-related mobility in adults with T2DM and whether the pattern of these associations varied according to diabetic peripheral neuropathy status. Participants with neuropathy demonstrated poorer glycemic profiles and lower functional balance scores than those without neuropathy, whereas the between-group difference in TUG performance was comparatively small and statistically imprecise. Most importantly, the within-group associations between glycemic measures and functional outcomes were weak and none remained statistically significant after adjustment for multiple testing. Nevertheless, formal comparison of the subgroup correlations demonstrated that the relationship between HbA1c and BBS differed between participants with and without neuropathy. No comparable between-group differences were identified for HbA1c with TUG or for random blood glucose with either functional outcome. Overall, these findings suggest that the relationship between glycemic status and physical function is not uniform and cannot be explained adequately by glycemic measurements alone.

The poorer balance profile observed among participants with neuropathy is consistent with the established functional consequences of DPN. Peripheral nerve dysfunction can impair somatosensory feedback, vibration perception, protective sensation, proprioception, and motor responses required for effective postural control (3). A systematic review by Khan and Andersen similarly concluded that diabetic neuropathy adversely affects postural balance and gait and is associated with increased susceptibility to falls (5). The lower BBS scores observed in the neuropathy group are therefore clinically plausible and support the broader literature identifying DPN as an important contributor to functional instability. Systematic reviews have also demonstrated an increased risk of falls

among older adults with T2DM, although this risk reflects multiple interacting factors rather than neuropathy alone (6,7).

Despite poorer descriptive balance in participants with neuropathy, HbA1c was not negatively associated with BBS within this group. One possible explanation is that once clinically detectable peripheral neuropathy is established, accumulated neurological impairment may contribute more strongly to postural dysfunction than a contemporary glycemic measurement. DPN develops through chronic metabolic and microvascular processes, and established sensory or motor impairment may persist despite variation in current glycemic status (3,5). Consequently, an HbA1c measurement obtained around the time of functional assessment may not adequately represent the longer cumulative glycemic exposure associated with the development and severity of neuropathic deficits. This interpretation remains mechanistic and cannot be confirmed by the present cross-sectional data.

In participants without clinically identified neuropathy, higher HbA1c was associated with lower BBS scores in the unadjusted subgroup analysis. However, this individual correlation did not remain statistically significant after adjustment for the eight subgroup correlation tests. The more informative finding was that the HbA1c-BBS correlation differed significantly between the neuropathy and non-neuropathy groups after formal comparison and multiplicity adjustment. This pattern raises the possibility that the relationship between chronic glycemic status and functional balance may vary according to the presence of established neuropathic impairment. Nevertheless, the finding should be interpreted cautiously because no multivariable model was available to determine whether the observed difference remained after accounting for age, sex, duration of diabetes, treatment, body composition, physical activity, visual function, muscle strength, comorbidities, or other potential confounders. The result should therefore be regarded as evidence of differing association patterns rather than proof of a specific biological interaction.

The absence of a simple linear relationship between HbA1c and functional outcomes is also consistent with evidence that glycemia and falls in older adults have a complex relationship. In a longitudinal cohort of older adults with diabetes, Schwartz et al. found that fall risk was related to several diabetes-associated complications and that low HbA1c was associated with increased falls among insulin-treated participants, illustrating that fall susceptibility cannot be represented by a simple assumption that higher HbA1c invariably corresponds to greater fall risk (10). Severe hypoglycemia has likewise been associated with falls in older adults with diabetes (8). These findings reinforce current guidance emphasizing individualized glycemic management in older adults, with consideration of functional status, comorbidities, treatment burden, frailty, and hypoglycemia risk rather than reliance on a single glycemic target (9). The present results similarly indicate that HbA1c should not be interpreted as an isolated marker of functional safety.

HbA1c was not associated with TUG performance within either neuropathy group, and formal comparison did not demonstrate evidence that these correlations differed between groups. This may partly reflect differences in the functional constructs assessed by the BBS and TUG. The BBS evaluates performance across multiple static and dynamic balance tasks, whereas the TUG primarily measures integrated functional mobility involving standing, walking, turning, and sitting (11,12). Previous review evidence has highlighted the multidimensional nature of balance in people with T2DM and the considerable variation among clinical measures used to assess it (13). A metabolic or neurological factor may therefore demonstrate a relationship with one aspect of physical function without producing a comparable association with another. In addition, the relatively large variability in TUG times in the present sample may have reduced the precision of observed associations.

Random blood glucose showed little evidence of association with either BBS or TUG in either group. This contrasts with the subgroup pattern observed for HbA1c and may reflect the different information represented by the two glycemic measures. Random blood glucose is a point-in-time measurement that may vary according to recent food intake, medication timing, activity, stress, and other short-term influences, whereas HbA1c is more representative of longer-term glycemic status. A single random glucose measurement may therefore have limited ability to capture the metabolic exposure most relevant to gradually developing impairments in balance and mobility. The very weak and inconsistent correlations observed in the present study support caution in using isolated random glucose measurements to infer functional impairment.

The findings also emphasize the distinction between functional mobility screening and actual fall events. TUG is an established measure of basic functional mobility (12), but the present study did not prospectively record falls.

Accordingly, the absence of an association between glycemic measures and TUG should not be interpreted as evidence that glycemic factors are unrelated to actual falls. Falls among people with T2DM are multifactorial and may be influenced by neuropathy, medication exposure, hypoglycemia, visual impairment, muscle weakness, gait dysfunction, comorbidity, and other age-related factors (6–8). Future prospective studies incorporating documented fall events would therefore provide a more direct evaluation of clinically meaningful fall outcomes.

From a clinical perspective, the poorer functional balance observed in participants with neuropathy supports incorporating balance and mobility assessment into the broader evaluation of people with longstanding T2DM. The differing HbA1c–BBS association pattern also suggests that functional assessment should not necessarily be restricted to individuals with established neuropathy, because balance limitations may occur through multiple pathways. These findings should not be interpreted as evidence that changing HbA1c alone will improve balance. Rather, glycemic status, neuropathy, functional performance, medication exposure, and other fall-related factors should be considered together when evaluating patients. Previous Pakistani intervention studies have reported improvements in balance following task-oriented and proprioceptive training among diabetic populations (14,15), while systematic-review evidence indicates that exercise and multicomponent rehabilitation can improve balance-related outcomes in people with T2DM and DPN (16,17). Such evidence supports the clinical relevance of identifying functional deficits, although the effectiveness of rehabilitation was not evaluated in the present study.

Several features strengthen the study. The inclusion of equal numbers of participants with and without neuropathy allowed comparable subgroup analyses, while the use of both HbA1c and random blood glucose permitted assessment of glycemic status using measures representing different temporal characteristics. Functional assessment incorporated both BBS and TUG, providing information on related but distinct aspects of balance and mobility. In addition, formal comparison of the subgroup correlation coefficients and adjustment for multiple testing provided a more conservative evaluation of the principal association patterns than interpretation of isolated subgroup p-values alone.

These findings should nevertheless be interpreted in light of important limitations. The cross-sectional design prevents determination of temporal sequence and causality. Participants were recruited through non-probability purposive sampling from selected healthcare and community settings, limiting generalizability beyond the sampled population. The intentionally equal neuropathy and non-neuropathy group sizes also mean that the proportion of participants with neuropathy cannot be interpreted as an estimate of DPN prevalence. The reported analyses were not adjusted for potentially important confounders, including age, sex, diabetes duration, medication and insulin use, body mass index, physical activity, visual impairment, lower-limb strength, vestibular dysfunction, previous falls, and comorbidities. Neuropathy classification relied on clinical screening instruments rather than electrophysiological confirmation, allowing the possibility of classification error. Random blood glucose represented a single measurement and may not adequately characterize glycemic variability, while the study did not assess hypoglycemic episodes. Finally, TUG provided a functional mobility measure rather than direct prospective ascertainment of falls. These limitations mean that the observed difference in the HbA1c–BBS relationship should be considered hypothesis-generating and warrants confirmation in longitudinal studies using adjusted analyses, detailed characterization of neuropathy severity, and direct monitoring of fall outcomes.

CONCLUSION

The study demonstrated that adults with T2DM and diabetic neuropathy had poorer glycemic profiles and lower functional balance scores than those without neuropathy, while most associations between glycemic measures and functional balance or mobility were weak. Although no individual within-group glycemic–functional correlation remained statistically significant after adjustment for multiple testing, the association between HbA1c and BBS differed between neuropathy and non-neuropathy groups. Random blood glucose showed no meaningful relationship with BBS or TUG, and HbA1c was not consistently associated with functional mobility. These findings indicate that glycemic status alone does not adequately explain balance or fall-risk-related mobility in adults with T2DM and that neuropathy status and other functional determinants should be considered when evaluating balance impairment.

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