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Original Research Article

Correlation of duration of diabetes mellitus with bone mineral density in postmenopausal women

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ABSTRACT

Background: Osteoporosis and type 2 diabetes mellitus commonly coexist in postmenopausal women, both increasing morbidity and fracture risk. Despite often normal or elevated bone mineral density, individuals with T2DM face a paradoxically higher fracture risk. This study aimed to assess the correlation between the duration of T2DM and BMD in postmenopausal women.
Methods: This cross-sectional study was conducted in the Medicine Department of Sir Salimullah Medical College and Mitford Hospital from July 2023 to June 2024. A total of 120 postmenopausal women with T2DM were enrolled according to predefined criteria. Data were analyzed using SPSS 22.0, with a $p<0.05$ considered statistically significant.
Results: Duration of diabetes demonstrated a significant negative correlation with BMD. T-scores showed strong inverse relationships at both the femoral neck ($r=-0.649$, $p<0.001$) and lumbar spine ($r=-0.724$, $p<0.001$). Similarly, Z-scores were negatively correlated at the femoral neck ($r=-0.521$, $p<0.001$) and lumbar spine ($r=-0.540$, $p<0.001$). Comparison of treatment modalities revealed no significant differences: women on oral agents had femoral neck and lumbar spine T-scores of -2.51±0.96 and -3.12±1.50, while those on oral agents plus insulin had scores of -2.94±1.17 and -3.54±1.52, respectively ($p>0.05$).
Conclusions: Longer duration of T2DM is significantly associated with lower BMD in postmenopausal women, particularly at the femoral neck and lumbar spine. Treatment modality did not significantly influence BMD outcomes.

Keywords: Duration, DM, Bone mineral density, Postmenopausal women

INTRODUCTION

Type 2 diabetes mellitus (T2DM) and osteoporosis are two highly prevalent, aging-related conditions that frequently coexist in postmenopausal women. Although dual-energy X-ray absorptiometry (DXA)-derived bone mineral density (BMD) remains the cornerstone of osteoporosis assessment, diabetes introduces a paradox: women with T2DM often present with normal or even higher BMD, yet sustain more fractures than their non-diabetic peers.[1,2] This clinical mismatch suggests that diabetes alters bone quality and fracture resistance through mechanisms that are not fully captured by areal BMD alone, and that patient-level factors-such as duration of hyperglycaemia-may be critical modifiers of skeletal risk.[1-4] Multiple pathophysiologic pathways link chronic diabetes exposure to skeletal deterioration. Persistent hyperglycaemia and oxidative stress accelerate advanced glycation end-product (AGE) accumulation in collagen, reduce osteoblast function, impair osteocyte signaling, and favor low-turnover (adynamic) bone, collectively diminishing material properties despite preserved BMD.[3-5] Microvascular

disease, low-grade inflammation, and diabetes-related sarcopenia and falls risk further amplify fracture susceptibility independent of BMD.[3-5] In postmenopausal women, estrogen deficiency superimposes on these diabetes-specific insults, potentially intensifying the tempo of cortical porosity and trabecular deterioration. Longitudinal data in older women show that, despite higher baseline BMD in those with diabetes, subsequent bone loss at the hip and spine can be faster than in non-diabetic women, consistent with cumulative metabolic injury over time.[10] Observational studies consistently demonstrate that standard BMD-based tools underestimate fracture risk in T2DM. For a given femoral-neck T-score or FRAX probability, fracture rates are higher in people with diabetes, including postmenopausal women, than in those without diabetes.[1,2] Proposed adjustments, such as applying a trabecular bone score (TBS) correction, subtracting 0.5 SD from the BMD input, or adding rheumatoid arthritis as a surrogate risk factor-improve calibration but do not fully resolve the underestimation, again highlighting diabetes-specific skeletal deficits that accumulate with disease chronicity.[2] Against this backdrop, the duration of diabetes emerges as a plausible, clinically accessible proxy for accumulated skeletal toxicity. Cross-sectional and cohort analyses have linked longer diabetes duration with lower hip or femoral-neck BMD, poorer TBS, and higher incident fracture risk, even after accounting for age, BMI, and glycaemic control.[6-9] In Chinese adults with T2DM, a disease duration beyond 10 years was associated with reduced BMD at the femoral neck and total hip, particularly among women, a pattern that aligns with the compounding effects of postmenopausal estrogen deficiency and prolonged hyperglycaemia.[7] Population-based Asian data likewise indicate that each additional diabetes year correlates with decremental femoral-neck BMD, independent of traditional covariates.8 More recently, analyses in elderly men and postmenopausal women have reinforced that longer disease courses are associated with lower BMD and a higher prevalence of diabetes among individuals with low BMD, supporting a bidirectional link between glycometabolic health and skeletal integrity.9 Complementary evidence from large BMD registry cohorts shows that diabetes duration meaningfully augments fracture risk beyond FRAX estimates, underscoring the clinical relevance of chronic exposure even when BMD appears preserved.[2] This study aims to evaluate the correlation between the duration of type 2 diabetes mellitus and bone mineral density in postmenopausal women.

METHODS

This cross-sectional study was conducted in the Department of Medicine at Sir Salimullah Medical College and Mitford Hospital over 12 months from July 2023 to June 2024. A total of 120 postmenopausal women were enrolled, including 60 women with type 2 diabetes mellitus (case group) and 60 age-matched non-diabetic women (control group). Postmenopause was defined as the absence of menstruation for at least 12 consecutive months. Diagnosis of T2DM followed the American Diabetes Association (ADA) criteria, and diabetic participants had been receiving oral antidiabetic drugs (OAD) alone or in combination with insulin for a minimum of one year. Exclusion criteria included women with secondary causes of osteoporosis (such as endocrine disorders or chronic kidney disease), those taking medications that affect bone metabolism (e.g., corticosteroids, bisphosphonates, hormone replacement therapy), women with surgical or premature menopause (<40 years), chronic inflammatory diseases, or malignancies. Data were collected using a structured proforma, documenting socio-demographic characteristics, age at menarche, age at menopause, duration since menopause, and, for diabetic participants, duration of diabetes, glycemic control assessed by HbA1c, and treatment details. Anthropometric measurements were taken to calculate body mass index (BMI). BMD at the femoral neck and lumbar spine was assessed for all participants using DXA, and T-scores and Z-scores were recorded. Statistical analysis was performed using standard software, with continuous variables presented as mean±standard deviation and categorical variables as frequencies and percentages. Comparisons between groups were conducted using unpaired t-tests and chi-square tests, and a $p<0.05$ was considered statistically significant.

RESULTS

Data were expressed as frequency, percentage, and mean (±standard deviation).

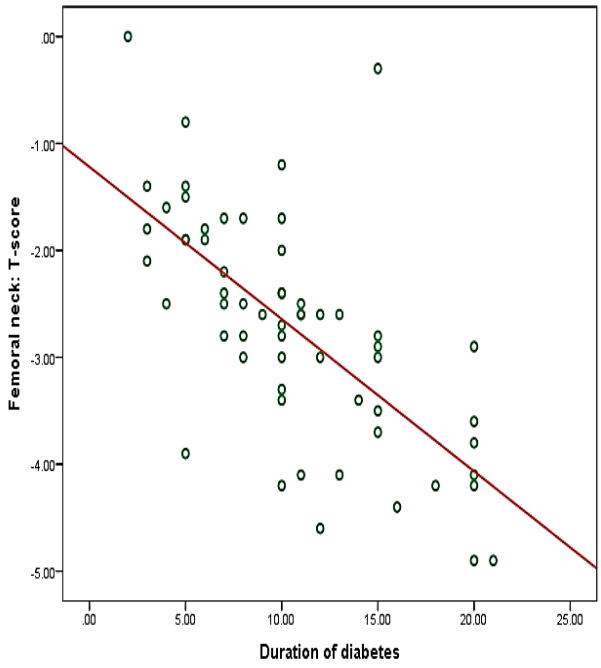

Figure 1: Scatter plot showing correlation of duration of diabetes with bone mineral density (femoral neck: T score) in the case group.

An unpaired t test was done to measure the level of significance.

Table 1 shows the demographic profile of the study subjects. Mean age of the patients was 65.83±8.75 years and 62.17±7.67 years in diabetic and non diabetic postmenopausal patients. There was no significant difference in the age of menarche and age at menopause. But the duration since menopause was significantly higher in diabetic patients than in non-diabetic patients. There was also no significant difference in BMI between the groups.

Table 2 shows the correlation of the duration of DM with bone mineral density in postmenopausal women with diabetes mellitus. There was a significant negative correlation between the duration of DM and with T score and Z score of both the femoral neck and lumbar spine.

An unpaired t test was done to measure the level of significance

Table 3 shows the association of drugs with bone mineral density in postmenopausal women with diabetes mellitus.

There was no association of BMD with the drug in postmenopausal women with diabetes mellitus.

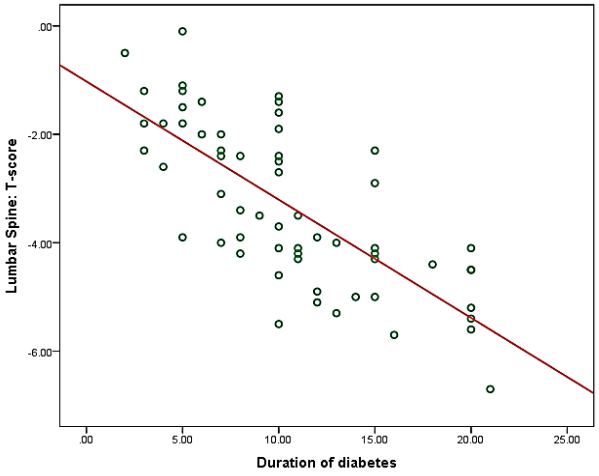

Figure 2: Scatter plot showing correlation of duration of diabetes with bone mineral density (Lumber: T score) in the case group.

Table 1: Demographic profile of the study subjects (n=120).

Demographics	Diabetic (n=60) N (%)	Non diabetic (n=60) N (%)	P value
Age (in years)			
50-59	8 (13.3)	19 (31.7)	
60-69	32 (53.3)	32 (53.3)	
≥70	20 (33.3)	9 (15.0)	
Mean±SD	65.83±8.75	62.17±7.67	0.016
Age of menarche (in years)	14.78±0.69	14.63±0.64	0.219
Age at menopause (in years)	46.90±3.39	46.95±3.04	0.932
Duration since menopause (in years)	19.70±7.82	15.15±8.27	0.002
Body mass index (kg/m^2)	24.60±4.87	25.26±3.40	0.397

Table 2: Correlation of duration of DM with BMD in case group (n=60).

Parameters	R value	P value
T score		
Femoral neck	-0.649	<0.001
Lumbar spine	-0.724	<0.001
Z score		
Femoral neck	-0.521	<0.001
Lumbar spine	-0.540	<0.001

Table 3: Association of treatment modality BMD in case group (n=60).

Parameters	Drugs		P value
	OAD (n=31) [Mean±SD]	OAD+insulin (n=29) [Mean±SD]	
T-score			
Femoral neck	-2.51±0.96	-2.94±1.17	0.129
Lumbar spine	-3.12±1.50	-3.54±1.52	0.287
Z-Score			

Continued.

Parameters	Drugs		P value
	OAD (n=31) [Mean±SD]	OAD+insulin (n=29) [Mean±SD]	
Femoral neck	-0.96±0.81	-1.37±1.16	0.116
Lumbar spine	-1.53 ± 1.06	-1.70 ± 1.45	0.594

DISCUSSION

In this study, the mean age of participants was 65.83±8.75 years in diabetic postmenopausal women and 62.17±7.67 years in non-diabetic postmenopausal women. In comparison, Anaforoglu et al reported mean ages of 61.9±8.6 years and 60.1±9.3 years in diabetic and non-diabetic postmenopausal women, respectively.[11] Thus, the mean age in our study population was slightly higher than that observed in similar research. The mean age at menarche was 14.78±0.69 years in the diabetic group and 14.63±0.64 years in the non-diabetic group, closely resembling the findings of Hadzibegovic et al.[12] The mean age at menopause in our study was 46.90±3.39 years among diabetic women and 46.95±3.04 years among non-diabetic women, which is consistent with the results reported by both Anaforoglu et al and Hadzibegovic et al.[11,12] Regarding the duration since menopause, our study recorded 19.70±7.82 years in the diabetic group compared with 15.15±8.27 years in the non-diabetic group. Other studies have also documented a longer postmenopausal duration in diabetic women compared to non-diabetic counterparts, aligning with our findings.[11,13] The present study revealed a significant negative correlation between the duration of DM and BMD in postmenopausal women. Duration of DM showed strong inverse relationships with both T scores (femoral neck: r=-0.649, p<0.001; lumbar spine: r=-0.724, p<0.001) and Z scores (femoral neck: r=-0.521, p<0.001; lumbar spine: r=-0.540, p<0.001). Comparable results were reported in earlier studies. Jang et al. observed that women with >10 years of diabetes had significantly lower femoral neck BMD (0.701±0.12 g/cm²) compared to those with <5 years duration (0.754±0.11 g/cm²).[8] In our study, although absolute BMD values were not presented, the stronger negative correlations (up to -0.724 at lumbar spine) suggest a greater influence of disease duration on skeletal deterioration. Similarly, Guo et al reported lower femoral neck BMD in Chinese women with long-standing diabetes (0.64±0.09 g/cm²) compared with those with shorter disease duration (0.72±0.08 g/cm², p<0.05), which closely parallels the decline demonstrated in our results.[7] Luo et al also confirmed that postmenopausal women with diabetes of >10 years' duration had higher rates of low BMD (42.5%) than those with <10 years (29.4%).[9] Together with our data, this supports the concept that cumulative metabolic insult from prolonged diabetes substantially accelerates bone loss. The mechanisms underlying this relationship are well recognized. Chronic hyperglycemia induces AGEs, which impair collagen cross-linking, reduce bone toughness, and lead to low bone turnover.[3] Hofbauer et al highlighted that diabetes-related bone fragility is largely independent of BMD, yet prolonged disease duration amplifies the structural and microarchitectural damage, making duration a clinically relevant predictor of skeletal risk.[5] When evaluating treatment modality, our study found no statistically significant association between glucose-lowering therapy and BMD. Women on oral antidiabetic drugs (OAD) alone had a mean femoral neck T score of -2.510.96, compared to -2.94±1.17 in those on OAD plus insulin (p=0.129). At the lumbar spine, T scores were -3.12±1.50 versus -3.54±1.52 (p=0.287). Although insulin-treated patients had numerically lower BMD, these differences were not significant, suggesting that treatment type alone does not determine bone density. Previous studies have shown mixed findings. Viégas et al reported that insulin-treated postmenopausal women had lower lumbar spine T-scores (-2.8±0.9) compared with those on OADs (-2.3±0.8, p<0.05), a difference not observed in our COHORT.[6] This discrepancy may be explained by differences in sample size, duration of therapy, or degree of metabolic control. Gilbert and Pratley noted that insulin has direct anabolic effects on bone but is often prescribed in patients with longer-standing disease, making it difficult to distinguish treatment effect from disease severity.[11] Starup-Linde and Vestergaard further reported that bone turnover markers are lower in insulin users, reflecting suppressed remodeling, which may predispose to fragility despite similar BMD.[12]

Limitations of the study

The study was conducted in a single hospital with a small sample size. So, the results may not represent the whole community.

CONCLUSION

The study found that longer duration of diabetes mellitus is significantly associated with lower bone mineral density in postmenopausal women, particularly at the femoral neck and lumbar spine, while treatment modality showed no significant effect.

Recommendations

It is recommended that postmenopausal women with long-standing diabetes mellitus be routinely evaluated for osteoporosis through timely bone mineral density screening, particularly at the femoral neck and lumbar spine. Preventive measures, including lifestyle modification, adequate calcium and vitamin D intake, weight-bearing exercise, and fall-prevention strategies, should be emphasized.

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Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Schwartz AV, Vittinghoff E, Bauer DC, Hillier TA, Strotmeyer ES, Ensrud KE, et al. Association of BMD and FRAX score with risk of fracture in older adults with type 2 diabetes. JAMA. 2011;305(21):2184-92.
2. Leslie WD, Rubin MR, Schwartz AV, Kanis JA. Type 2 diabetes and bone. J Bone Miner Res. 2012;27(11):2231-7.
3. Shanbhogue VV, Mitchell DM, Rosen CJ, Bouxsein ML. Type 2 diabetes and the skeleton: new insights into sweet bones. Lancet Diabet Endocrinol. 2016;4(2):159-73.
4. Napoli N, Chandran M, Pierroz DD, Abrahamsen B, Schwartz AV, Ferrari SL, et al. Mechanisms of diabetes mellitus-induced bone fragility. Nat Rev Endocrinol. 2017;13(4):208-19.
5. Hofbauer LC, Busse B, Eastell R, Ferrari S, Frost M, Müller R, et al. Bone fragility in diabetes: novel concepts and clinical implications. Lancet Diabet Endocrinol. 2022;10(3):207-20.
6. Viégas M, Costa C, Lopes A, Griz L, Medeiro MA, Bandeira F. Prevalence of osteoporosis and vertebral fractures in postmenopausal women with type 2 diabetes mellitus and their relationship with duration of the disease and chronic complications. J Diabet Complicat. 2011;25(4):216-21.
7. Guo L, Gao Z, Ge H. Effects of serum 25-hydroxyvitaminD level on decreased bone mineral density at femoral neck and total hip in Chinese type 2 diabetes. PLoS One. 2017;12(11):e0188894.
8. Jang M, Kim H, Lea S, Oh S, Kim JS, Oh B. Effect of duration of diabetes on bone mineral density: a population study on East Asian males. BMC Endocr Disord. 2018;18(1):61.
9. Luo W, Li X, Zhou Y, Xu D, Qiao Y. Correlation between bone mineral density and type 2 diabetes mellitus in elderly men and postmenopausal women. Sci Rep. 2024;14(1):15078.
10. Schwartz AV, Ewing SK, Porzig AM, McCulloch CE, Resnick HE, Hillier TA, et al. Diabetes and change in bone mineral density at the hip, calcaneus, spine, and radius in older women. Front Endocrinol. 2013;30;4:62.
11. Gilbert MP, Pratley RE. The impact of diabetes and diabetes medications on bone health. Endocr Rev. 2015;36(2):194-213.
12. Starup-Linde J, Vestergaard P. Biochemical bone turnover markers in diabetes mellitus-a systematic review. Bone. 2016;82:69-78.

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