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Bone Mineral Density and Vertebral Fracture Frequency in a Group of Type 1 Diabetic Patients

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What is already known on this topic?

- *People with type 1 diabetes mellitus (T1DM) are known to have an increased risk of bone fragility and fractures, even when their bone mineral density appears normal.*
- *The reasons for this are not fully understood and may involve changes in bone quality and metabolism rather than just bone density.*

What this study adds to this topic?

- *This retrospective analysis demonstrates that vertebral fractures are significantly more frequent in adults with T1DM compared to healthy controls.*
- *This study highlights that altered bone turnover and lower serum phosphate may contribute to fragility beyond bone density.*

Abstract

Objective: This study aimed to evaluate the relationship between vertebral fracture and various biological factors in patients with type 1 diabetes mellitus (T1DM).

Methods: The study included 61 patients diagnosed with T1DM who were evaluated in the Diabetes Outpatient Clinic between 2017 and 2019, and 49 healthy individuals forming the control group. Demographic and clinical characteristics (age, gender, hypertension, hyperlipidemia, obesity), biochemical parameters (fasting blood glucose, HbA1c, calcium, phosphate, parathyroid hormone, 25-OH vitamin D, C-terminal telopeptide, osteocalcin, alkaline phosphatase), and bone-related variables (L1–L4 and femoral neck bone mineral density [BMD], vertebral fracture status) were comprehensively collected during outpatient visits. Vertebral fractures were assessed by the Genant semi-quantitative method using lateral vertebral fracture assessment images obtained by dual-energy X-ray absorptiometry. Data were analyzed using non-parametric statistical tests and expressed as median (interquartile range). Logistic regression analysis adjusted for age, sex, body mass index (BMI), and phosphate levels was performed.

Results: Vertebral fracture prevalence was significantly higher in T1DM patients (67.2%) compared with controls (26.5%, $P < .001$). Patients had significantly lower body weight and BMI and higher rates of hyperlipidemia. Femoral neck BMD was significantly lower in the T1DM group (0.967 vs. 1.193 g/cm^2, $P < .001$). Among diabetic patients, those with vertebral fractures had significantly lower serum phosphate levels than those without fractures (3.20 vs. 3.85 mg/dL, $P = .008$). No significant differences were observed in bone turnover markers or BMD values between patients with and without fractures.

Conclusion: Type 1 diabetes mellitus patients showed a markedly higher prevalence of vertebral fractures and lower femoral neck BMD compared with healthy controls. Lower phosphate levels were observed in patients with fractures, suggesting a potential role of phosphate metabolism in diabetic bone fragility.

Keywords: Bone mineral density, phosphate, type 1 diabetes, vertebral fracture

Introduction

Osteoporosis is a major public health concern characterized by decreased bone strength and increased fracture risk.[1,2] It leads to pain, functional impairment, reduced quality of life, and increased mortality.[2]

The incidence of low bone mineral density (BMD) in patients with type 1 diabetes mellitus (T1DM) is increasing, particularly in younger adults, and has been linked to an elevated risk of fractures.[3] The underlying mechanisms remain unclear; however, several factors may contribute to skeletal fragility in T1DM, including deficits in BMD, bone geometry, bone microarchitecture, and biomechanical properties.[4-6]

Meta-analyses have demonstrated a 4- to 7-fold higher risk of hip fracture among patients with T1DM compared with controls.[7,8] Despite variations in study populations and age groups, this elevated risk appears consistent across the lifespan. In a population-based cohort from Scotland, Hothersall et al[9] reported a markedly increased hip fracture incidence in both men and women with T1DM aged 20-49 years compared to age-matched controls.

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Collectively, these findings highlight that young to middle-aged adults with T1DM are as susceptible to fractures as postmenopausal individuals, underscoring the need to investigate non-density factors influencing bone strength.

In this study, the aim was to evaluate the relationship between vertebral fracture frequency and biochemical as well as clinical parameters in patients with T1DM.

Methods

This cross-sectional study included 61 T1DM patients and 49 healthy control individuals. Inclusion criteria for T1DM patients were age between 18 and 65 years, and a T1DM diagnosis for at least 5 years under insulin therapy. Exclusion criteria included other diseases that could be a cause of secondary osteoporosis or the use of medications affecting bone metabolism. Subjects with renal dysfunction, malabsorption syndromes, or vitamin D supplementation were also excluded.

No predefined lower body mass index (BMI) cutoff was applied. However, none of the included participants had clinically significant underweight requiring exclusion.

The control group consisted of individuals without diabetes who attended routine outpatient clinics. Comorbid conditions such as hypertension and obesity were recorded and are presented in Table 1.

Vertebral fractures were assessed using lateral vertebral fracture assessment (VFA) images obtained via dual-energy X-ray absorptiometry (DXA). The presence and severity of fractures in the thoracic (T4–T12) and lumbar (L1–L4) vertebrae were determined by an expert radiologist using the Genant semi-quantitative method. All images were evaluated by a single experienced endocrinologist.

Bone mineral density measurements were performed in all participants using a DXA device at the L1–L4 lumbar spine and femoral neck regions. Results were recorded in g/cm^2 and as T-scores and *Z*-scores. The DXA device was calibrated daily according to manufacturer recommendations.

Demographic data, T1DM duration, insulin dose, and comorbidities were recorded. Biochemical parameters included fasting plasma glucose (FPG), HbA1c, calcium, phosphate, parathyroid hormone (PTH), 25-hydroxy vitamin D (25-OH Vit D), bone turnover markers (C-terminal telopeptide [CTX], osteocalcin, alkaline phosphatase [ALP]), and routine tests. Serum calcium levels were measured as total calcium and were not corrected for albumin. Bone turnover markers (osteocalcin, CTX, and ALP) were measured and reported with their respective units, and reference ranges to enhance interpretability. Fasting plasma glucose was analyzed using an enzymatic UV test (hexokinase method), and HbA1c via high-performance liquid chromatography.

The study was approved by the Marmara University Faculty of Medicine Clinical Research Ethics Committee (Approval No.: 09.2019.878; Date: October 04, 2019) and was conducted in accordance with the ethical principles of the Declaration of Helsinki. Due to the retrospective design of the study and the use of anonymized data, the requirement for individual informed consent was waived by the ethics committee.

Statistical analyses were performed using SPSS v24.0 (IBM SPSS Corp.; Armonk, NY, USA). Normality of continuous variables was tested using the Shapiro–Wilk test. Continuous variables were expressed as median (interquartile range, IQR). Group comparisons were performed using the Mann–Whitney *U*-test and the chi-square or Fisher's exact test. Correlations were evaluated using Spearman's rank correlation analysis. Multivariate logistic regression analyses were adjusted for age, sex, BMI, and phosphate levels. Post-hoc power analysis based on the observed difference in

Table 1. Demographic and Laboratory Characteristics of Study Groups

Variables	Patient Group (n = 61)	Control Group (n = 49)	*P*
Female [n (%)]	32 (52.5)	29 (59.2)	.481
Diabetes duration (years)	13.18 (11)	–	–
Vertebral fracture [n (%)]	41 (67.2)	13 (26.5)	**<.001**
Age (years)	27.0 (17)	32.0 (12)	**.022**
Body weight (kg)	66.0 (15.1)	79.0 (21.8)	**<.001**
BMI (kg/m^2)	23.55 (6.20)	26.14 (7.16)	**.003**
BMD – Femur neck (g/cm^2)	0.967 (0.243)	1.193 (0.188)	**<.001**
BMD – L1-L4 (g/cm^2)	1.116 (0.223)	1.062 (0.261)	.113
T-score – Femur neck	0.10 (1.7)	0.60 (1.5)	.106
T-score – L1-L4	−0.20 (1.95)	0.65 (1.80)	.063
Z-score – Femur neck	0.30 (1.6)	0.25 (1.7)	.963
Z-score – L1-L4	0.10 (2.0)	0.35 (1.7)	.078
Hypertension [n (%)]	7 (11.5)	3 (6.3)	.508
Hyperlipidemia [n (%)]	13 (21.3)	0 (0)	**.001**
Stroke (CVA) [n (%)]	1 (1.6)	0 (0)	1.000
Obesity [n (%)]	2 (3.3)	14 (28.6)	**<.001**
Coronary artery disease [n (%)]	1 (1.6)	0 (0)	1.000
Menopause status [n (%)]	3 (7.7)	0 (0.0)	.255
Fasting glucose (FBG)	181 [144]	90 [18]	**<.001**
HbA1c (%)	8.5 [3.1]	5.2 [0.5]	**<.001**
Calcium (Ca, mg/dL)	9.9 [0.6]	9.7 [0.6]	.100
Phosphate (P, mg/dL)	3.5 [1.0]	3.3 [0.9]	.242
PTH (pg/mL)	30.7 [23.7]	39.5 [34.7]	**.009**
25-OH Vitamin D (ng/mL)	20.5 [17.8]	13.1 [13.3]	**.001**
CTX (ng/mL)	0.37 [0.27]	0.27 [0.16]	**.001**
Osteocalcin (ng/mL)	4.54 [5.13]	13.8 [14.9]	**<.001**
ALP (U/L)	73.0 [36]	59.0 [26]	**.001**
FSH (mIU/mL)	5.53 [4.17]	5.09 [4.97]	.481
LH (mIU/mL)	6.44 [5.08]	5.59 [4.65]	**.045**
TSH (µIU/mL)	1.80 [1.47]	1.45 [1.11]	**.038**
Creatinine (mg/dL)	0.72 [0.28]	0.68 [0.24]	.570
ALT (U/L)	14.0 [8]	20.0 [15]	**<.001**
AST (U/L)	17.0 [7]	19.0 [8]	**.001**

Values are given as median (IQR). Reference ranges: CTX, 0.035-0.836 ng/mL; Osteocalcin, 3-13 ng/mL.
ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase.; BMD, bone mineral density; BMI, body mass index; CTX, C-terminal telopeptide; FSH, follicle-stimulating hormone; LH, luteinizing hormone; PTH, parathyroid hormone; TSH, thyroid-stimulating hormone.

vertebral fracture prevalence (67.2% vs. 26.5%) indicated a power exceeding 95% (α = 0.05).

Results

The study included 61 patients with T1DM and 49 healthy controls. The prevalence of vertebral fracture was significantly higher among patients with T1DM compared to controls (67.2% vs. 26.5%, *P* < .001) (Table 1).

Regarding demographic characteristics, the T1DM group was significantly younger (median age: 27.0 vs. 32.0 years, *P* = .022) and had lower body weight and BMI (*P* < .01). In biochemical assessments, FPG (181 vs. 90 mg/dL, *P* < .001) and HbA1c (8.5% vs. 5.2%, *P* < .001) were markedly higher in the T1DM group compared with controls.

Bone measurements revealed that femoral neck BMD was significantly lower in patients with T1DM (0.967 vs. 1.193 g/cm^2, *P* < .001). No significant difference was observed in lumbar spine BMD values (*P* > .05). Additionally, bone turnover markers showed significant alterations in patients with T1DM. Serum CTX levels were significantly higher (0.37 vs. 0.27 ng/mL, *P* = .001), whereas osteocalcin levels were markedly lower (4.54 vs. 13.8 ng/mL, *P* < .001) compared to controls.

When T1DM patients were stratified according to vertebral fracture status, 41 patients were identified with fractures and 20 without. There were no significant differences between these subgroups regarding age, sex, BMI, diabetes duration, insulin dose, or glycemic control (fasting glucose and HbA1c) (Table 2). According to the Genant semi-quantitative grading system, 3 patients had grade 2 vertebral fractures, while the remaining 38 had grade 1 fractures.

However, serum phosphate levels were significantly lower in patients with fractures than in those without (3.20 vs. 3.85 mg/dL, *P* = .008). There were no significant differences in BMD, *T*-scores, or *Z*-scores at either the femoral neck or lumbar spine (*P* > .05).

Correlation analyses demonstrated a significant negative correlation between vertebral fracture presence and serum phosphate levels (*r* = −0.324, *P* = .01). No significant correlations were observed between fracture presence and HbA1c, duration of diabetes, or other biochemical parameters (*P* > .05).

The logistic regression model, including serum phosphorus as the independent variable, was not statistically significant (*P* = .231). Phosphorus was not identified as an independent predictor of vertebral fracture (odds ratio [OR] = 1.402; 95% CI: 0.806-2.438).

Multivariate logistic regression did not reveal additional independent predictors of vertebral fracture after adjustment for age, sex, BMI, and phosphate levels.

Table 2. Clinical and Bone Characteristics by Vertebral Fracture Status

Variable	Type 1 Diabetic Patients with Vertebra Fracture (n = 41)	Type 1 Diabetic Patients Without Vertebra Fracture (n = 20)	*P*
Sex (Female) [n (%)]	21 (51.2)	11 (55.0)	1.000
Diabetes duration (years)	13.27 (10)	13 (12.5)	.622
Menopause status [n (%)]	1 (4.2)	2 (13.3)	.547
Diabetic retinopathy [n (%)]	9 (22.0)	2 (10.5)	.476
Albuminuria [n (%)]	12 (29.3)	6 (30.0)	1.000
Obesity [n (%)]	1 (2.4)	1 (5.0)	1.000
PPI use [n (%)]	1 (2.4)	0 (0.0)	1.000
Age (years)	30.0 (20)	24.0 (10)	.150
Total insulin dose (U/day)	53.02 (37.0)	59.0 (18.5)	.242
Body weight (kg)	66.75 (16.5)	66.05 (15.7)	.628
BMI (kg/m^2)	24.19 (6.54)	23.20 (5.39)	.390
Fasting glucose (mg/dL)	181.0 [146.0]	182.5 [134.0]	.919
HbA1c (%)	8.65 [3.2]	8.05 [2.9]	.818
Calcium (mg/dL)	9.80 [0.7]	9.95 [0.7]	.396
Phosphate (mg/dL)	3.20 [1.00]	3.85 [0.65]	**.008**
PTH (pg/mL)	31.00 [28.30]	30.04 [18.04]	.631
25(OH) Vitamin D (ng/mL)	21.42 [20.78]	19.46 [14.64]	.406
CTX (ng/mL)	0.34 [0.25]	0.39 [0.38]	.638
Osteocalcin (ng/mL)	4.01 [5.46]	6.42 [4.89]	.326
ALP (U/L)	73.00 [33.00]	76.50 [41.00]	.755
TSH (μIU/mL)	1.80 [1.43]	1.73 [1.82]	.747
Creatinine (mg/dL)	0.72 [0.27]	0.71 [0.34]	.914
BMD – Femoral neck	0.961 [0.274]	1.063 [0.291]	.267
BMD – L1–L4	1.098 [0.237]	1.134 [0.307]	.509
T-score – Femoral neck	0.000 [1.7]	0.700 [1.7]	.333
T-score – L1–L4	−0.300 [1.95]	−0.100 [2.1]	.839
Z-score – Femoral neck	0.300 [1.5]	0.800 [1.6]	.315
Z-score – L1–L4	−0.100 [2.0]	0.800 [2.1]	.792

Reference ranges: CTX, 0.035-0.836 ng/mL; Osteocalcin, 3-13 ng/mL. ALP, alkaline phosphatase; BMD, bone mineral density; BMI, body mass index; CTX, C-terminal telopeptide; PPI, proton pump inhibitor; PTH, parathyroid hormone; TSH, thyroid-stimulating hormone.

Discussion

In this study, BMD, vertebral fracture frequency, and biochemical parameters were evaluated in patients with T1DM. These findings confirmed that patients with T1DM had significantly lower femoral neck BMD compared with healthy controls, while lumbar spine BMD was not significantly different. The prevalence of vertebral fractures was markedly higher in the T1DM group. These results support the notion that bone fragility in T1DM appears to result from both quantitative and qualitative skeletal alterations, emphasizing the importance of assessing bone quality beyond DXA-derived BMD.[2,3,7,10]

The current findings indicate that vertebral fractures may occur even in patients with only mild or no reduction in lumbar BMD, suggesting that microarchitectural changes contribute to skeletal fragility beyond mineral density.[10,11]

The mechanisms underlying bone fragility in T1DM are multifactorial. Insulin deficiency and chronic hyperglycemia can impair osteoblast differentiation, reduce bone formation, and increase bone resorption.[7,8,11] In this cohort, serum osteocalcin levels were significantly lower in diabetic patients, while CTX and alkaline

phosphatase levels were elevated. This pattern indicates a shift toward increased bone resorption and impaired bone formation, consistent with previous studies describing altered bone turnover in diabetes.[5,7,10,12]

The combination of low osteocalcin and elevated CTX suggests a net catabolic state within the bone microenvironment. Such alterations may predispose diabetic individuals to fragility fractures even when BMD is within normal limits.[11,13]

Another notable finding was the significantly lower phosphate levels observed in patients with vertebral fractures. This observation supports a potential role for phosphate metabolism in maintaining bone integrity.[12] Among diabetic patients, lower serum phosphate levels were significantly associated with vertebral fractures, indicating that phosphate imbalance may exacerbate skeletal fragility in T1DM.[12] Although serum phosphorus levels were correlated with vertebral fracture occurrence, logistic regression analysis revealed that phosphorus was not an independent predictor of fracture risk when controlling for age, sex, and BMI. This suggests that while phosphate imbalance may contribute to skeletal fragility, its predictive value diminishes when other covariates are taken into account. Phosphate imbalance may result from increased urinary phosphate loss due to glycosuria or altered fibroblast growth factor 23 activity, both of which have been implicated in diabetes-related bone fragility.[12,14]

Although glycemic control was poorer in the diabetic group, no direct correlation between HbA1c levels and fracture occurrence was observed, emphasizing that glucose metrics alone may underestimate skeletal risk in patients with T1DM. This suggests that metabolic control alone does not fully capture the complexity of bone health in T1DM, emphasizing the need for multifactorial assessment.[13,14] The higher 25-OH vitamin D levels observed in T1DM patients may reflect seasonal variation or supplementation rather than intrinsic metabolic effects.

Overall, the current data demonstrate that T1DM patients exhibit decreased femoral BMD, altered bone turnover, lower phosphate levels, and a higher prevalence of vertebral fractures.[1-3,7,10-13] In line with recent reviews on diabetic bone fragility, these results reinforce the concept that bone quality and remodeling dysregulation are central in diabetes-associated skeletal risk.[13,14]

This study's main strength lies in its comprehensive evaluation of bone health in patients with T1DM, integrating VFA with biochemical and densitometric parameters in a well-defined cohort. The use of VFA via DXA allowed detection of mild and asymptomatic fractures that might otherwise remain unrecognized.

Several limitations should be acknowledged. The retrospective and cross-sectional design limits causal inference and precludes strict age matching between groups; however, age and sex were accounted for in multivariate analyses. The relatively small sample size may have reduced the power to detect subtle subgroup differences, particularly in sex-specific and fracture severity analyses. In addition, the lack of detailed bone microarchitectural assessment and incomplete lifestyle data (including smoking, alcohol consumption, neuropathy, autonomic dysfunction, and fall history) may restrict interpretation of the underlying mechanisms of skeletal fragility.

Bone-specific alkaline phosphatase could not be assessed due to the retrospective design; however, the absence of biochemical evidence of cholestasis supports the interpretation that elevated total alkaline phosphatase levels are more likely related to altered bone turnover rather than hepatic pathology.

Total hip BMD measurements were not available in this study; therefore, femoral neck BMD was used as a surrogate for hip assessment. Future studies, including total hip measurements, may provide a more comprehensive evaluation of hip fracture risk in T1DM.

Despite these limitations, the findings underscore the potential contribution of phosphate imbalance and dysregulated bone turnover to increased vertebral fracture risk in individuals with T1DM.

In patients with T1DM, vertebral fractures were significantly more frequent despite only modest reductions in BMD. This finding indicates that bone fragility in T1DM extends beyond bone density and involves metabolic and microarchitectural alterations. Lower phosphate levels and increased bone turnover markers were independently associated with fracture presence, suggesting that these biochemical factors contribute to skeletal vulnerability. Early identification and management of phosphate imbalance and abnormal bone turnover may help reduce fracture risk in this population, thereby improving long-term skeletal outcomes in T1DM.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Artificial Intelligence Usage Statement: The authors declared that no Artificial Intelligence tool was used in the preparation of the manuscript.

Ethics Committee Approval: The study protocol was approved by the Marmara University Faculty of Medicine Clinical Research Ethics Committee (Approval No.: 09.2019.878; Date: October 04, 2019).

Informed Consent: Due to the retrospective design of the study and the use of anonymized data, the requirement for individual informed consent was waived by the ethics committee.

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