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Impact of triglyceride/glucose index on bone mineral density and spinal alignment in postmenopausal osteoporosis

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Abstract

Osteoporosis is a prevalent skeletal disorder characterized by decreased bone mineral density and an increased risk of fractures, particularly in postmenopausal women. Insulin resistance represents a prediabetic stage, and the Triglyceride/Glucose (TyG) index serves as a significant biomarker for its assessment. Calculated using fasting triglyceride and glucose levels, the TyG provides a cost-effective and practical screening method, particularly suitable for primary healthcare settings. This study seeks to clarify the relationship between the TyG, diabetes mellitus, and bone mineral density (BMD) in postmenopausal osteoporosis, as well as to examine the effects of these connections on spinal alignment. The sample comprised 140 female individuals categorized into four cohorts: a control group (postmenopausal without osteoporosis, BMD T score >-2), an osteoporosis group, diabetes with osteoporosis group, and diabetes without osteoporosis group. BMD values were acquired by Dual-energy X-ray Absorptiometry (DEXA), and the TyG was calculated from triglyceride and fasting glucose concentrations using a logarithmic model. Deviations in the angle of the vertebral column were also measured. Data regarding radiographic imaging, TyG, and BMD were retrospectively gathered from postmenopausal women diagnosed with osteoporosis between January 1, 2020, and December 30, 2023. The findings indicate a significant correlation between the TyG and both diabetes mellitus and bone mineral density, with discernible alterations in vertebral alignment in postmenopausal osteoporosis patients. The coexistence of diabetes and osteoporosis may accelerate degenerative processes in bone tissue, leading to the deterioration of bone integrity and the progression of spinal curvature changes.

Keywords: Diabetes, bone mineral density, osteoporosis, postmenopause, Triglyceride Glucose Index

Introduction

Postmenopausal osteoporosis is a disorder frequently observed in women during the postmenopausal phase, marked by a notable reduction in bone mineral density (BMD), resulting in considerable health complications. During this period, the decrease in estrogen levels leads to significant disturbances in bone homeostasis. Typically, the estrogen hormone sustains equilibrium in bone metabolism by modulating the balance between osteoblasts (bone-forming cells) and osteoclasts (bone-resorbing cells). Post-menopause, diminished estrogen levels increase osteoclastic activity, leading to expedited bone resorption and decelerated bone production [1,2]. This change leads to a decrease in bone density and lays the groundwork for the development of osteoporosis. The risk of fractures notably increases in areas such as the spine, hips, and wrists, which can negatively affect patients' mobility and levels of independence [3].

Alongside hormonal changes metabolic and inflammatory mechanisms significantly contribute to the onset of osteoporosis. The triglyceride/glucose (TyG) index serves as a crucial metric for assessing insulin resistance [4]. Insulin resistance impairs the physiological processes of bone architecture, thereby impacting bone mineralization and density. Under typical circumstances, insulin facilitates the proliferation of osteoblasts and inhibits bone resorption; however, insulin resistance impairs these functions. Elevated TyG readings, indicative of heightened insulin resistance and metabolic syndrome, facilitate the onset of osteoporosis in postmenopausal women [5]. The rise in insulin resistance diminishes the anabolic effects on bones, resulting in reduced bone density and an increased risk of osteoporosis. This creates a robust correlation between type 2 diabetes and osteoporosis in postmenopausal women [6].

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Literature indicates that elevated triglyceride levels adversely impact BMD and heighten the risk of osteoporosis [7]. A negative correlation has been identified between triglyceride levels and BMD; specifically, when triglyceride levels rise, bone mineral density tends to decline [8]. The inflammatory response is another critical factor in this mechanism. Inflammation linked to metabolic syndrome enhances osteoclastic activity, leading to increased bone resorption and a consequent reduction in BMD. Hyperglycemia and inflammation resulting from insulin resistance disrupt the normal functions of bone cells, thereby complicating the pathophysiology of osteoporosis [9].

The alteration of the natural angle of the vertebral column is another significant effect of osteoporosis. Osteoporosis-induced weakening of vertebral bones can result in structural deformations of the spine, causing disruptions in the natural curvature of the vertebral column [10]. In postmenopausal women, diabetes exacerbates this process by altering the natural curvature of the spine. These structural changes in the spine lead to pain and limit the physical mobility of individuals [11]. Additionally, it is stated that high TyG values may lead to alterations in the natural angle of the vertebral column, potentially causing spinal deformities [12].

Considering metabolic indicators such as the triglyceride/glucose index into the management of osteoporosis and diabetes may significantly contribute to slowing disease progression and enhancing patient quality of life. This study evaluates the relationship between the triglyceride/glucose index, diabetes, and BMD in postmenopausal osteoporosis, and examines the impact of these relationships on changes in the natural angle of the vertebral column. The hypothesis of this study is articulated as follows: "The triglyceride/glucose index affects changes in the natural angle of the vertebral column in postmenopausal women with osteoporosis and diabetes".

Material and Methods

This research aimed to assess the impact of health conditions, including postmenopausal osteoporosis and diabetes, on BMD and the angles of the vertebral column. The sample size was established through a power analysis conducted using the G*Power 3.1 software. An effect size of 0.80, an error margin of 0.05, and a confidence level of 95% require a minimum sample size of 120 participants to achieve a power of 0.90. This sample is distributed as follows: Control (30), Osteoporosis (30), Diabetes with Osteoporosis (30), and Diabetes without Osteoporosis (30) [13]. Participants were selected from individuals diagnosed with osteoporosis exhibiting BMD values of -2.5 or higher. The study excluded individuals under 45 years of age and those with particular health conditions. BMD measurements were performed utilizing the Dual X-Ray Absorptiometry (DEXA) method. Furthermore, blood parameters such as glucose, triglycerides, HDL, LDL, ALP, potassium, B12, uric acid, and calcium were analyzed. The participants were analyzed retrospectively using radiological images acquired from Malatya Turgut Özal University Non-Interventional Clinical Research Ethics Committee (Acceptance No: 39 / Date: 14.02.2024).

The vertebral column angles were measured using the Cobb method with the assistance of the RadiAnt DICOM Viewer software [14]. The RadiAnt DICOM Viewer was carried out precise analysis of medical images. Proper patient positioning was ensured during imaging, with elevation applied under the knees to flatten lumbar lordosis. Throughout the measurements, the femur was positioned at an internal rotation of 15°-25° to minimize the visibility of the lesser trochanter in the image. The angle between the inferior endplates of the C2 and C7 vertebrae was used to calculate cervical lordosis, while the presence of lordosis was assessed in relation to degenerative changes [15]. Thoracic kyphosis was calculated using the angle between the plate lines of T4 and T12, with normal values identified within the range of 20°-50° [16]. Lumbar lordosis was determined using the angle between the L1 and sacral endplate lines, with normal values evaluated within the range of 40°-70°. Additionally, the sacral slope was measured to assess pelvic structure; this angle represents the relationship between the sacral plate and the horizontal plane [16].

Statistical Analysis

This study utilized SPSS (Statistical Program for the Social Sciences) version 25 for data analysis. The Kolmogorov-Smirnov test was applied to assess the normality of the data distribution. Descriptive statistics, such as mean, standard deviation, count, and percentage values, were employed. The significance level (p) for comparison tests was set at 0.05. Due to the variables not following to a normal distribution ($p>0.05$), non-parametric testing methods were utilized. The Kruskal-Wallis H test was employed to compare more than two independent groups due to the violation of the normality assumption. Bonferroni post-hoc tests were utilized to identify the source of differences. Pearson correlation coefficients were employed to examine the relationships among numerical variables.

Results

Significant differences were observed among the groups (control, osteoporosis, diabetes with osteoporosis, diabetes without osteoporosis) regarding height and weight variables ($p<0.05$). The post hoc analysis revealed significant differences between the control group and the osteoporosis group ($p<0.005$), the diabetes with osteoporosis group ($p<0.006$), and the diabetes without osteoporosis group ($p<0.031$). No statistically significant differences were observed among the groups concerning age and BMI variables ($p>0.05$) (Table 1).

Statistically significant differences were observed among the groups (control, osteoporosis, diabetes with osteoporosis, and diabetes without osteoporosis) for the variables Glucose (mg/dL), TyG (mg/dL), Vitamin D (ng/mL), B12 (pg/mL), Uric Acid (mg/dL), and Calcium (mg/dL) ($p<0.05$). For the Glucose (mg/dL) variable, significant differences were found between the control group and the diabetes with osteoporosis ($p<0.001$) and diabetes without osteoporosis ($p<0.001$) groups. Additionally, significant differences were observed between the osteoporosis group and the diabetes with osteoporosis ($p<0.001$) and diabetes

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without osteoporosis (p<0.001) groups. For the TyG (mg/dL) variable, significant differences were found between the control group and the diabetes with osteoporosis (p<0.004) and diabetes without osteoporosis (p<0.016) groups. Furthermore, significant differences were identified between the osteoporosis group and the diabetes with osteoporosis group (p<0.021). Regarding LDL (mg/dL), significant differences were observed between the control group and the diabetes with osteoporosis (p<0.006) and diabetes without osteoporosis (p<0.004) groups. Significant differences were also found between the osteoporosis group and the diabetes with osteoporosis (p<0.006) and diabetes without osteoporosis (p<0.003) groups. For Vitamin D (ng/mL), significant differences were observed between the control group and the diabetes with osteoporosis (p<0.001) and diabetes without osteoporosis (p<0.001) groups. Additionally, differences were noted between the osteoporosis group and the diabetes with osteoporosis (p<0.001) and diabetes without osteoporosis (p<0.001) groups. Regarding B12 (pg/mL), significant differences were found between the control group and the osteoporosis (p<0.044), diabetes with osteoporosis (p<0.001), and diabetes without osteoporosis (p<0.001) groups. Similarly, significant differences were observed between the osteoporosis group and the diabetes with osteoporosis (p<0.001) and diabetes without osteoporosis (p<0.001) groups. For Uric Acid (mg/dL), significant differences were identified between the osteoporosis group and the diabetes with osteoporosis (p<0.022) and diabetes without osteoporosis (p<0.022) groups. Lastly, for Calcium (mg/dL), a significant difference was found between the control group and the diabetes with osteoporosis group (p<0.049). However, no statistically significant differences were observed among the groups for the variables Triglycerides (mg/dL), HDL (mg/dL), ALP (U/L), and Potassium K (mmol/L) (p>0.05) (Table 2).

Statistically significant differences were found among the groups (control, osteoporosis, diabetes with osteoporosis, diabetes without osteoporosis) regarding the BMD (Bone Mineral Density) variable (p<0.05). According to the post hoc analysis examining the source of the difference, statistically significant differences were identified for the BMD variable between the control group and the osteoporosis (p<0.001), diabetes with osteoporosis (p<0.001), and diabetes without osteoporosis (p<0.001) groups; between the osteoporosis and diabetes without osteoporosis (p<0.002) groups; and between the diabetes with osteoporosis and diabetes without osteoporosis (p<0.001) groups (Table 3).

Statistically significant differences were found among the groups (control, osteoporosis, diabetes with osteoporosis, diabetes without osteoporosis) for the variables Cervical Lordosis, Thoracic Kyphosis, Lumbar Lordosis, and Sacral Slope (p<0.05). According to the post hoc analysis, for the Cervical Lordosis variable, statistically significant differences were observed between the control group and the osteoporosis (p<0.001), diabetes with osteoporosis (p<0.001), and diabetes without osteoporosis (p<0.001) groups. For the Thoracic Kyphosis variable, significant differences were identified between the control group and the osteoporosis (p<0.001), diabetes with osteoporosis (p<0.001), and diabetes without osteoporosis (p<0.001) groups. For the Sacral Slope variable, statistically significant differences were found between the control group and the osteoporosis (p<0.001), diabetes with osteoporosis (p<0.001), and diabetes without osteoporosis (p<0.001) groups (Table 4).

Table 1. Demographic information of participants

Variable	Group	N	x̄±SD	M (Q1-Q3)	Min-Max	Kruskal Wallis H	p
Age (years)	**Control[1]**	35	55.40±5.96	55 (50-61)	46-65	0.276	0.964
	Osteoporosis[2]	35	55.91±6.02	56 (50-61)	46-66		
	Diabetes with osteoporosis[3]	35	55.86±6.09	56 (50-62)	46-65		
	Diabetes without osteoporosis[4]	35	55.34±5.89	55 (50-60)	46-65		
Height (cm)	**Control[1]**	35	159.06±7.28	158 (155-164)	144-178	15.049	**0.002* (1-2,3,4)**
	Osteoporosis[2]	35	152.94±7.39	153 (147-158)	137-172		
	Diabetes with osteoporosis[3]	35	153.11±7.40	153 (148-158)	137-172		
	Diabetes without osteoporosis[4]	35	154.06±7.92	154 (148-160)	138-171		
Weight (kg)	**Control[1]**	35	75.77±13.60	70 (67-84)	55-113	10.203	**0.017* (1-2,3,4)**
	Osteoporosis[2]	35	65.83±12.12	67 (55-75)	45-92		
	Diabetes with osteoporosis[3]	35	67.09±12.26	66 (58-77)	45-90		
	Diabetes without osteoporosis[4]	35	66.66±13.88	66 (54-79)	45-91		
BMI (kg/m2)	**Control[1]**	35	30.11±5.92	28.7 (25.71-33.46)	21.15-45.84	2.151	0.542
	Osteoporosis[2]	35	28.06±4.33	28.1 (24.99-30.84)	20.27-36.85		
	Diabetes with osteoporosis[3]	35	28.73±5.71	27.1 (24.84-33.69)	20.93-44.9		
	Diabetes without osteoporosis[4]	35	28.29±6.59	27.6 (21.46-34.19)	16.94-41.66		

x̄: mean, SD: standard deviation, M: median, Q1: 25% interquartile range, Q3: 75% interquartile range, *p<0.05: there is a statistically significant difference between the groups

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Table 2. Comparison of laboratory parameters

Variable	Group	N	x̄±SD	M (Q1-Q3)	Min-Max	Kruskal Wallis H	p
Glucose (mg/dL)	**Control[1]**	35	108.00±26.66	99 (90-115)	77-177	32.416	**0.001* (1-3,4, 2-3,4, 3-4)**
	Osteoporosis[2]	35	112.94±28.07	102 (95-120)	79-209		
	Diabetes with osteoporosis[3]	35	146.96±27.66	135.8 (124.67-170.34)	120.12-200.78		
	Diabetes without osteoporosis[4]	35	159.54±22.41	157 (142-176)	124-199		
Triglycerides (mg/dL)	**Control[1]**	35	146.87±88.63	125 (94-186)	56-504	1.206	0.310
	Osteoporosis[2]	35	148.20±86.38	136 (95-186)	49-505		
	Diabetes with osteoporosis[3]	35	138.57±25.27	133 (119-150)	100-201		
	Diabetes without osteoporosis[4]	35	122.07±28.49	111.3 (104.21-120.23)	100.34-200.45		
TyG (mg/dL)	**Control[1]**	35	4.76±0.29	4.75 (4.5-4.97)	4.28-5.46	6.010	**0.001* (1-3,4)**
	Osteoporosis[2]	35	4.79±0.29	4.76 (4.56-5.02)	4.25-5.6		
	Diabetes with osteoporosis[3]	35	4.95±0.14	4.91 (4.81-5.03)	4.76-5.29		
	Diabetes without osteoporosis[4]	35	4.92±0.12	4.91 (4.81-5)	4.75-5.23		
HDL (mg/dL)	**Control[1]**	35	55.84±12.32	53.6 (46-66)	37.9-82	1.727	0.164
	Osteoporosis[2]	35	55.56±12.13	55.37 (45.7-67)	31.4-82		
	Diabetes with osteoporosis[3]	35	59.10±6.14	58.67 (54-63)	50-70.67		
	Diabetes without osteoporosis[4]	35	59.73±6.56	58.4 (54.12-65.67)	50.21-70.56		
LDL (mg/dL)	**Control[1]**	35	124.97±27.57	121.3 (98.4-142.8)	79.1-178	7.802	**0.001***
	Osteoporosis[2]	35	125.14±32.95	126.5 (96.7-148.63)	69.8-229.2		
	Diabetes with osteoporosis[3]	35	106.32±13.51	101.78 (94.56-120.34)	90.21-130.67		
	Diabetes without osteoporosis[4]	35	105.48±12.94	101.45 (94.67-115.67)	90.12-130.56		
ALP (U/L)	**Control[1]**	35	82.34±21.90	85 (66-94)	13-134	1.320	0.270
	Osteoporosis[2]	35	88.97±29.27	84 (68-100)	41-172		
	Diabetes with osteoporosis[3]	35	80.67±9.85	78.56 (73.67-83.67)	70.12-120.89		
	Diabetes without osteoporosis[4]	35	87.63±16.78	81.34 (74.56-100.45)	70.21-120.89		
Potassium K (mmol/L)	**Control[1]**	35	4.46±0.49	4.4 (4.1-4.88)	3.38-5.4	0.904	0.441
	Osteoporosis[2]	35	4.45±0.89	4.2 (4.08-4.6)	3.59-9.1		
	Diabetes with osteoporosis[3]	35	4.24±0.85	4.21 (3.45-5.12)	3.12-5.89		
	Diabetes without osteoporosis[4]	35	4.24±0.75	4.21 (3.56-4.89)	3.12-5.67		
D vitamin (ng/mL)	**Control[1]**	35	22.62±10.97	22.85 (14.7-27.74)	4.67-51.3	13.319	**0.001 (1-3,4)***
	Osteoporosis[2]	35	22.29±9.93	21 (17.6-25.3)	5.3-64.79		
	Diabetes with osteoporosis[3]	35	14.26±2.87	13.45 (11.78-17.23)	10.12-19.67		
	Diabetes without osteoporosis[4]	35	14.25±2.93	13.45 (11.78-16.34)	10.21-19.78		
B12 (pg/mL)	**Control[1]**	35	449.19±283.95	392 (285.9-480.3)	171.8-1826	19.559	**0.001* (1-3,4)**
	Osteoporosis[2]	35	354.21±95.97	331.6 (280.4-414.1)	208-658.3		
	Diabetes with osteoporosis[3]	35	219.79±10.84	220.3 (210.78-228.56)	201.78-237.23		
	Diabetes without osteoporosis[4]	35	218.91±11.72	218.5 (207.56-228.67)	200.12-238.34		
Uric acid (mg/dL)	**Control[1]**	35	4.76±1.19	4.7 (4.1-5.5)	2.4-7.4	3.758	**0.012* (1-3,4)**
	Osteoporosis[2]	35	6.40±7.59	4.6 (3.8-5.4)	3.1-45.5		
	Diabetes with osteoporosis[3]	35	3.69±1.07	3.56 (2.67-4.56)	2.12-5.67		
	Diabetes without osteoporosis[4]	35	3.69±1.06	3.67 (2.67-4.56)	2.12-5.67		
Ca (mg/dL)	**Control[1]**	35	11.75±14.15	9.3 (9.1-9.6)	8.5-93	2.977	**0.034* (1-3,4)**
	Osteoporosis[2]	35	9.16±0.57	9.2 (8.88-9.6)	7.2-10.3		
	Diabetes with osteoporosis[3]	35	7.31±0.81	7.23 (6.67-8.12)	6.12-8.67		
	Diabetes without osteoporosis[4]	35	7.42±0.84	7.45 (6.67-8.34)	6.12-8.67		

x̄: mean, SD: standard deviation, M: Median, Q1: 25% interquartile range, Q3: 75% interquartile range, *p<0.05: there is a statistically significant difference between the groups

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Table 3. Comparison of BMD values across groups

Variable	Group	N	x̄±SD	M (Q1-Q3)	Min-Max	Kruskal Wallis H	p
KMY	Control[1]	35	0.33±1.83	-0.3 (-1.3-2)	-2.2-4.2	85.448	0.001* (1-2,3,4 and 3-4)
	Osteoporosis[2]	35	-2.81±0.52	-2.5 (-3-2.5)	-4.1-2.2		
	Diabetes with osteoporosis[3]	35	-2.95±0.28	-2.93 (-3.15-2.71)	-3.45-2.48		
	Diabetes without osteoporosis[4]	35	-1.96±0.25	-1.95 (-2.19-1.76)	-2.35-1.55		

x̄: mean, SD: standard deviation, M: median, Q1: 25% interquartile range, Q3: 75% interquartile range, *p<0.05: there is a statistically significant difference between the groups

Table 4. Comparison of angular values across groups

Variable	Group	N	x̄±SD	M (Q1-Q3)	Min-Max	Kruskal Wallis H	p
Cervical lordosis	Control[1]	35	22.15±2.42	22.12 (19.89-24.34)	18.23-26.45	78.350	0.001* (1-2,3,4)
	Osteoporosis[2]	35	17.41±1.41	17.45 (16.12-18.67)	15.23-19.78		
	Diabetes with osteoporosis[3]	35	17.28±1.18	17.09 (16.56-18.1)	15.27-19.49		
	Diabetes without osteoporosis[4]	35	17.22±1.13	17 (16.5-18)	15.3-19.4		
Thoracic kyphosis	Control[1]	35	39.89±5.22	39.78 (35.67-44.67)	31.23-49.45	15.761	0.001* (1-2,3,4)
	Osteoporosis[2]	35	46.83±5.12	46.78 (42.67-51.67)	38.23-55.56		
	Diabetes with osteoporosis[3]	35	46.84±5.10	46.77 (42.69-51.69)	38.51-55.53		
	Diabetes without osteoporosis[4]	35	46.70±5.12	46.53 (42.39-51.37)	38.45-55.49		
Lumbar lordosis	Control[1]	35	52.04±6.83	50.78 (46.67-57.23)	42.23-65.67	3.149	0.027* (1-2,3,4)
	Osteoporosis[2]	35	48.04±6.83	46.78 (42.67-53.23)	38.23-61.67		
	Diabetes with osteoporosis[3]	35	47.86±6.87	46.67 (42.23-53.45)	38.21-61.89		
	Diabetes without osteoporosis[4]	35	47.93±6.82	46.67 (42.12-53.89)	38.34-61.67		
Sacral slope	Control[1]	35	39.56±2.05	39.23 (37.78-41.67)	36.23-42.89	7.880	0.001* (1-2,3,4)
	Osteoporosis[2]	35	41.85±2.34	41.56 (39.78-43.99)	38.23-45.92		
	Diabetes with osteoporosis[3]	35	41.68±2.38	41.34 (39.56-43.67)	38.12-45.89		
	Diabetes without osteoporosis[4]	35	41.67±2.40	41.56 (39.45-43.56)	38.12-45.89		

x̄: mean, SD: standard deviation, M: median, Q1: 25% interquartile range, Q3: 75% interquartile range, *p<0.05: there is a statistically significant difference between the groups

Discussion

This study examines the association between the TyG and BMD in postmenopausal women with osteoporosis, along with its impact on changes in the natural angle of the vertebral column. The study comprised 35 participants from each group, with the sample size justified by a power analysis grounded in the existing literature [17]. Osteoporosis represents a critical public health concern, particularly in light of the growing elderly demographic, highlighting the necessity for routine screening of women aged 65 and older for this condition [18]. The participants had an average age of 55; however, certain findings were reported to contradict the existing literature. This discrepancy is believed to arise from characteristics unique to the local population [19].

In the diabetes and osteoporosis groups, weight and height ratios were lower than those in the control group, indicating potential adverse effects on body composition and BMD [20]. Although no significant differences were found among the groups in terms of vitamin D and calcium levels, it was stated that low vitamin D levels could negatively affect bone health [21]. In the literature, it has been stated that vitamin D deficiency can lead to secondary hyperparathyroidism, thereby increasing bone resorption [22]. Additionally, it is stated that vitamin D and calcium supplementation during winter months can reduce the risk of osteoporosis and prevent bone loss [23,24]. The study indicated that low vitamin B12 levels could contribute to the progression of osteoporosis by causing neurological and hematological disorders [25]. High LDL levels were observed in the diabetes group, and indicating an increased cardiovascular risk [26].

A significant relationship has been identified between Platelet Distribution Width (PDW), a marker of platelet function in the blood, and osteoporosis. However, no significant association was observed with decreased Mean Platelet Volume (MPV) values [27]. In their study, Karakaş et al. suggested that measuring blood Creatine Phosphate (CP) levels could serve as a screening methodology for osteoporotic geriatric patients [28]. Furthermore, clinical and radiological evaluations conducted by Karaoğlan et al. revealed that the serum ALP (Alkaline Phosphatase) levels in patients diagnosed with primary and secondary osteoporosis were reported as 128.40±29.43 in the study group [29]. The findings of the study did not reveal a statistically significant

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relationship between the TyG and BMD. However, evidence in the literature indicates that the TyG may exert both positive and negative effects on BMD. For example, a study conducted by Yoon et al. on non-diabetic postmenopausal women in Korea identified an inverse association between the TyG and BMD at the femoral neck, total hip, and whole body [30-35]. This finding indicates that the study contributes to the existing literature and highlights the complex nature of this relationship. BMD values in the osteoporosis and diabetes groups were found to be lower than in the control group, emphasizing that diabetes weakens bone structure, increases fracture risk, and underscores the importance of managing osteoporosis in these individuals [36]. Additionally, it has been stated that postural changes, such as thoracic kyphosis and lumbar lordosis, are frequently observed in individuals with osteoporosis [17,37]. The relationship between vertebral column curvatures and BMD has been evaluated in alignment with the literature, revealing the negative effects of osteoporosis on spinal health [38].

In the study, statistically significant relationships were identified between thoracic kyphosis and lumbar lordosis angles and BMD, while the relationships between TyG and spinal angle changes were examined in detail. It was noted that spinal deformities in individuals with osteoporosis are associated with a decrease in back muscle strength but may not show a direct relationship with BMD [38,39]. The findings explain the effects of osteoporosis and diabetes on postural control and spinal health, emphasizing the need for more comprehensive research and multidisciplinary approaches in this field [40].

Conclusion

In conclusion, the coexistence of osteoporosis and diabetes leads to structural impairments in bones, negatively affecting spinal curvature and posture. Statistical analyses revealed that osteoporosis and diabetes result in significant differences in BMD, vertebral column angles, and biochemical parameters. Diabetes negatively affects muscle mass and postural control, whereas osteoporosis reduces bone density, increasing the risk of spinal deformities. Consistent with the literature, TyG has been identified as a significant biomarker for understanding the complex effects of osteoporosis and diabetes on bone health. These findings highlight the need to consider TyG, BMD, and vertebral column angles in the management of osteoporosis and diabetes, contributing to clinical practices. It is recommended that multidisciplinary approaches and advanced studies be adopted for managing osteoporosis and diabetes in postmenopausal women. In cases of coexisting postmenopausal osteoporosis and diabetes, a multidisciplinary approach is essential for preserving bone health and minimizing fracture risk. Regular monitoring of TyG and BMD measurements and observing changes in vertebral column angles should be prioritized. Additionally, considering that diabetes reduces muscle mass, exercise programs to increase muscle strength should be recommended for patients. To better understand the effects of osteoporosis and diabetes on biochemical parameters, studies with larger sample sizes and long-term follow-ups are suggested. The current research aims to enhance the understanding of osteoporosis and diabetes' impacts on bone health and to inform the development of effective treatment strategies.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
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Ethical Approval
Malatya Turgut Özal University Non-Interventional Clinical Research Ethics Committee (Acceptance No: 39 / Date: 14.02.2024).

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