

Bone Mineral Density in Patients with Type 2 Diabetes Mellitus Patients

Abdulsatar J Mathkhor^{1*}, Abdulnasser H Abdullah²

¹Department of Rheumatology, Basrah Teaching Hospital, Basrah, Iraq;²Department of Rheumatology, Alsader Teaching Hospital, Basrah, Iraq

ABSTRACT

Introduction: It is well-documented that type 1 diabetes mellitus shows decreased Bone Mass Density (BMD). Type 2 Diabetes Mellitus (T2DM) affects bone metabolism, but the relationship between T2DM and BMD remains controversial. This study aimed to evaluate the difference in BMD between diabetic and non-diabetic individuals.

Materials and methods: A total of 150 (50 male and 100 female) diabetic patients and 150 (55 male and 95 females) controls with a mean age of 58 ± 6.5 and 59 ± 7.8 , respectively, were enrolled for the study. They underwent BMD measurement for the lumbar spine and left femoral neck through Dual-energy X-ray Absorptiometry (DXA) for the presence of osteoporosis according to the WHO criteria. The fasting plasma glucose level of all participants was measured.

Results: Of the 150 diabetic patients there were 11(7.33%), and 21 (14.0%) have osteoporosis and osteopenia in the lumbar spine, respectively, compared to 35 (23.33%) and 48(32.0%) in the non-diabetic group. There were 9(6.0%) and 17(11.33%) had osteoporosis and osteopenia in the left femoral neck in the diabetics, respectively, compared to 27(18.66%) and 42(28.0%) in the non-diabetics.

Conclusion: Patients with T2DM have higher BMD levels when compared to non-diabetics.

Keywords: Diabetes mellitus; Bone mineral density; Dual-energy X-ray absorptiometry; Osteoporosis

INTRODUCTION

Diabetes mellitus is a common metabolic condition characterized by persistent hyperglycemia with resultant morbidity and mortality related primarily to its associated microvascular and macrovascular complications. Type 2 Diabetes Mellitus (T2DM) is a highly prevalent disorder worldwide [1]. Diabetes mellitus has evolved as one of the world's most common health problems that affect almost all organ systems, with substantial morbidity and mortality [2]. Musculoskeletal complications related to diabetes are common and can lead to severe morbidity. Consequences following diabetic complications are muscle infarction, neuropathic arthropathy, and Charcot joint [3]. Metabolic derangements can lead to Diffuse Idiopathic Skeletal Hyperostosis (DISH) and osteopenia. Syndromes due to microvascular disease carpal tunnel syndrome, Dupuytren's contracture, flexor synovitis, Adhesive capsulitis, and limited joint mobility. Other complications like sclerodactyly, calcific tendinitis of the shoulder, periarthritis of the shoulder, and reflex sympathetic dystrophy [4,5]. Osteoporosis is defined as the generalized reduction in bone mass that disrupts the microarchitecture of bone and attenuates bone strength, leading to an increased risk of bone fractures [6]. The World Health Organization defines osteoporosis as a Bone Mineral Density (BMD) below 2.5 standard deviations of the mean for young, healthy adults of the same sex, whereas T

scores between -2.5 and -1 standard deviations are defined as osteopenia [7]. Association between T2DM and bone mineral density remains controversial. Because the pathway of the pathogenesis of type 1 differs from that of T2DM, there is no uniform entity of diabetic bone disease. While decreased BMD was observed in patients with type 1 diabetes [8,9], studies on BMD in T2DM showed controversial findings with lower, higher, or even similar results compared to healthy controls [10]. T2DM and osteoporosis share numerous common characteristics; they are both chronic disorders with an important medical burden. Although individuals with type 1 DM showed decreased BMD, those with T2DM often have normal or sometimes slightly elevated BMD compared with healthy age-matched controls [11]. Bone weakness results from reduced bone mineral mass and alterations in the microstructure of bone. Several mechanisms may contribute to increased fractures in T2DM patients. Glucose toxicity, lack of insulin, and other factors affect bone metabolism. Several studies examined the association between T2DM and fracture risk [12,13]. Clinically, assessing the bone microstructure of T2DM patients is difficult because CT or MRI should be used [11]. Therefore determining the BMD is the best approach for now. Because of the controversial results of different studies, this study aimed to evaluate this debatable association between diabetes and osteoporosis.

Correspondence to: Abdulsatar J Mathkhor, Department of Rheumatology, Basrah Teaching Hospital, Basrah, Iraq, Tel: +9647812601037; E-mailamathkhoor@ yahoo.co.uk

Received: 12-Dec-2022, Manuscript No. RCR-22-20768; Editor assigned: 15-Dec-2022, PreQC No. RCR-22-20768 (PQ); Reviewed: 30-Dec-2022, QC No. RCR-22-20768; Revised: 06-Jan-2023, Manuscript No. RCR-22- 20768 (R); Published: 13-Jan-2023, DOI: 10.35841/2161-1149.22.13.322

Citation: Mathkhor AJ, Abdullah H (2023) Bone Mineral Density in Patients with Type 2 Diabetes Mellitus Patients. Rheumatology (Sunnyvale). 13: 322

Copyright: © 2023 Mathkhor AJ, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

MATERIALS AND METHODS

A cross-sectional study was carried out at the Department of Rheumatology and Rheumatology Outpatient Unit in Basrah Teaching Hospital from December 2020 to July 2022. A total of 150 (50 male and 100 female) diabetic patients and 150 (55 male and 95 females) controls with a mean age of 58 ± 6.5 and 59 \pm 7.8, respectively, were enrolled in the study. BMI was calculated for all participants, and the adopted BMI ranges were: normal (18.5-24.9), overweight (25-29.9), and obese (\geq 30) [14]. All participants underwent BMD measurement for the lumbar spine and left femoral neck through Dual-energy X-ray Absorptiometry (DXA) for the presence of osteoporosis according to the WHO criteria. BMD (as g/cm²) was measured at the lumbar spine (L1-L4) and hip (femoral neck) by DXA machine in the Rheumatology unit in Basra Teaching Hospital. The T score (comparison with normal, young subjects of the same sex) was based on the reference values in the DXA machine provided by the manufacturer (GE Lunar Prodigy Pro). The definition of osteoporosis according to WHO guidelines were (T score ≤ -2.5 SD) and (T score ≥ -1.0 SD) defined as normal BMD) [15]. All patients and controls were subjected to detailed clinical evaluation and necessary blood investigations. Patients with chronic kidney disease, malabsorption syndromes, malignant diseases, chronic pancreatitis or pancreatotomy, primary hyperparathyroidism, thyroid disorder, Paget's disease, and inflammatory disorders such as rheumatoid arthritis, systemic lupus erythematosus, postmenopausal women or those with history of hysterectomy, patients treated with steroids, immunosuppressants, anticonvulsants, calcium, and Vitamin D supplements, and bed-ridden patients were excluded from the study.

Statistical analysis

SPSS software version 25.0 was used for data analysis. Percentages and mean were used to present the data in tables. In addition, a comparison of study groups was carried out using a chi-square test for categorical data and Student's t-test for continuous data. A P-value of <0.05 was considered statistically significant.

RESULTS

The demographic distributions of patients are shown in Table 1. Of the 150 diabetic patients, there were 50 (33.33%) and 100 (66.67%) men and women, respectively, with mean age and disease duration of 58 \pm 6.5 years12 \pm 5.8 years, respectively. Of the 150 non-diabetic individuals, there were 55 (36.66%) and 95 (63.34%) men and women, respectively, with a mean age of 59 ± 7.8 years. Of the 150 diabetic patients, there were 11(7.33%), and 21 (14.0%) had osteoporosis and osteopenia in the lumbar spine, respectively, compared to 35 (23.33%) and 48 (32.0%) in the non-diabetic group the difference was statistically significant (p=0,022). There were 9 (6.0%) and 17 (11.33%) had osteoporosis and osteopenia in the left femoral neck in the diabetics, respectively, compared to 27 (18.66%) and 42 (28.0%) in the non-diabetics the difference was statistically significant (p=0,022). Osteoporosis and osteopenia were more pronounced in women than men, as shown in Tables 2 and 3. Table 4 shows the statistically significant differences in BMI between diabetics and non-diabetics (p=0.022). There is no significant difference in BMI in the lumbar spine and femoral neck according to the disease duration in diabetic patients (p>0.05), as shown in Table 5.

Table 1: Demographic data for both diabetics and non-diabetics.

	Diabetics	Non-diabetics	P value
Total No.	150 (100%)	150 (100%)	
Men	50 (33.33%)	55 (36.66%)	
Women	100 (66. 67%)	95 (63.34%)	
Age (mean ± SD)	58 ± 6.5	59 ± 7.8	>0.05
BMI (mean ± SD)	27 ± 3.7	22 ± 4.1	<0.05
Disease duration	12 ± 5.8		

OPEN OACCESS Freely available online

 Table 2: Osteoporosis and osteopenia in the lumbar spine in diabetics and non-diabetics.

	Diabetics	Non- diabetics	P value
Total	150 (100%)	150 (100%)	
Osteoporosis (total)	11 (7.33%)	35 (23.33%)	0.022
Male	3 (2.0%)	25 (16.66%)	
Female	8 (5.33%)	10 (6.67%)	
Osteopenia (total)	21 (14.0%)	48 (32.0%)	0.022
Male	7 (4.66%)	10 (6.67%)	
Female	21 (9.34%)	38 (25.33%)	

Table 3: Osteoporosis and osteopenia in femoral neck diabetics and nondiabetics.

	Diabetics	Non- diabetics	P value
Total	150 (100%)	150 (100%)	
Osteoporosis (total)	9 (6.0%)	27 (18.66%)	
Male	2 (1.33%)	7 (4.66%)	0.023
Female	7 (4.67%)	20 14.0%)	
Osteopenia (total)	17 (11.33%)	42 (28.0%)	
Male	4 (2.66%)	32 (21.33%)	0.023
Female	13 (8.67%)	10 (6.67%)	

 Table 4: BMD for lumbar spine and femoral neck in both study groups.

	Lumbar spine	Femoral neck
Diabetics	-1.4 ± 0.22	-1.5 ± 0.24
Non-diabetics	-2.3 ± 0.12	- 2.2 ± 0.17
P value	0.022	0.022

 Table 5: BMD for lumbar spine and femoral neck according to disease duration in diabetic patients

Disease duration	≥ 5 years	<5 years	P value
BMD of the lumbar spine	-1.4 ± 0.22	-1.3 ± 0.92	>0.05
BMD of femoral neck	-1.5 ± 0.24	-1.4 ± 0.84	>0.05

DISCUSSION

Osteoporosis is a common and prevalent metabolic bone disease, and its occurrence in diabetic patients further increases the burden of the disease. In this study, a significant difference in BMD was observed between diabetic patients and non-diabetic individuals. Diabetic patients have a higher BMD than the control group in both the lumbar spine and femoral neck. The association between BMD and T2DM is still an obscure and debatable issue. However, the findings of this study are consistent with those of previous studies conducted by Ma L et al., Vestergaard P et al., Leidig-Bruckner G et al., and Raj et al. in that DM patients have higher BMDs [16-19]. In contrast to our study, Shivank Prakash et al. Yaturu and Dutta N et al. found that diabetic patients were associated with lower BMD [20-22]. In this study, our diabetic patients have a higher BMI than the control group. Diabetes could influence bone through several mechanisms, some of which may have contradictory effects. Obesity, widespread in T2DM, is strongly associated with higher BMD, probably through mechanical loading and hormonal factors, including insulin, estrogen, and leptin [23-25]. The adipose tissue produces different adipokines that have an essential role in regulating bone remodeling. Diabetic patients have higher concentrations of plasma leptin than healthy controls [26]. Leptin stimulates the growth of bone by stimulating osteoblast proliferation and differentiation [27]. Hyperinsulinemia may promote bone formation [28]. However, low insulin levels and the progression of T2DM may

OPEN OACCESS Freely available online

Mathkhor AJ, et al.

cause reductions in BMD. High fasting insulin levels play an essential role in the development of DM, mainly resulting in higher bone mass. In advanced and complicated T2DM, elevated insulin levels have a deleterious effect. As the resistance to insulin increases, the fasting insulin levels are inversely related to BMD, and this correlation becomes highly significant as the degree of insulin resistance increases [29]. In advanced T2DM requiring insulin, pancreatic & cell function certainly decreases. However, the exact timing of this phenomenon remains to be determined. In this study, we observed no significant association between BMD and disease duration in diabetic patients, a result that is contradictory to the findings of Miso Jang et al., who found a significant correlation between disease duration and low BMD [30]. In this study, even osteoporosis and osteopenia were less prevalent in the diabetic group than in the controls, but it was observed to be higher in women than in men. Gender has a crucial effect on BMD in T2DM. Barrett-Connor [31] observed that older diabetic women had higher BMD levels at all sites. Studies also suggested that obesity and hyperinsulinemia can decrease bone turnover in diabetic women [26], so the impact of estrogen deficiency on bone mass is attenuated after menopause [19].

CONCLUSION

One limitation of this study is the low number of participants, and large prospective studies are needed to establish the mechanisms underlying this association and, most importantly, the relationship with fracture risk, the most adverse consequence of osteoporosis. In this study, we found BMI was higher in type 2 diabetic patients than in non-diabetic healthy controls. Osteopenia and osteoporosis were less prevalent in diabetic patients than in non-diabetic healthy controls, and BMD was higher in diabetics than non-diabetics in both the lumbar spine and femoral neck. Disease duration was not correlated to BMD in diabetic patients.

ETHICAL CONSIDERATION

Written consent was obtained from all participants prior to their involvement. The study was conducted in accordance with the principles of the Declaration of Helsinki.

AUTHOR'S CONTRIBUTIONS

AM: Conceptualization, Methodology, Software, Data curation, Writing the Original draft, and preparing the final manuscript.

AA: Visualization, Investigation, Software, and Validation of the final manuscript.

AM and AA: Writing-Reviewing and Editing, approval of the final manuscript.

ACKNOWLEDGMENT

We kindly appreciate the role of all participants in the study.

Data availability

Data available on request

Funding disclosure

No funding was received for this manuscript.

Conflicts of interest

The authors declare that there is no conflict of interest.

REFERENCES

- 1. ZMisra R. Diabetes and musculoskeletal manifestations. J Indian Rheumatol Assoc. 2003; 11:23.
- Asokan AG, Jaganathan J, Philip R, Soman RR, Sebastian ST, Pullishery F. Evaluation of bone mineral density among type 2 diabetes mellitus patients in South Karnataka. J Nat Sci Biol Med. 2017; 8(1):94.

- Carnevale V, Romagnoli E, D'Erasmo L, D'Erasmo E. Bone damage in type 2 diabetes mellitus. Nutr Metab Cardiovasc Dis. 2014;24(11):1151-1157.
- Mcneil JD. Musculoskeletal manifestations of diabetes mellitus. Br J Sports Med. 2003;37:30-35.
- Fauci B, Kasper H, Longo J. Loscalzo. Harrison's: Priciples of Internal Medicine. 7th Edition. The McGrawHill Companies Inc. 2008:158.
- Mohammed JQ, Mathkhor AJ, Abed AH. Osteoporosis in Psoriasis and Psoriatic Arthritis Patients. Middle East J Fam Med. 2020; 7(10):38.
- Dreiher J, Weitzman D, Cohen AD. Psoriasis and osteoporosis: A sexspecific association? J Invest Dermatol. 2009;129(7):1643-1649.
- 8. Saller A, Maggi S, Romanato G, Tonin P, Crepaldi G. Diabetes and osteoporosis. Aging Clin Exp Res. 2008; 20(4):280-289.
- 9. van Daele PL, Stolk RP, Burger H, Algra D, Grobbee DE, Hofman A, et al. Bone density in non-insulin-dependent diabetes mellitus: the Rotterdam Study. Ann Intern Med. 1995;122(6):409-414.
- Kahn A, Gibbons R, Perkins S, Gazit D. Age-Related Bone Loss: A Hypothesis and Initial Assessment in Mice. Clin Orthop Relat Res. 1995; 313:69-75.
- Napoli N, Chandran M, Pierroz DD, Abrahamsen B, Schwartz AV, Ferrari SL. Mechanisms of diabetes mellitus-induced bone fragility. Nat Rev Endocrinol. 2017; 13(4):208-219.
- Yamaguchi T, Sugimoto T. Bone metabolism and fracture risk in type 2 diabetes mellitus. Bonekey Rep. 2011:1107160591.
- 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):2184-2192.
- Ser TR. Physical status: The use and interpretation of anthropometry. Report of a WHO Expert Committee. World Health Organ Tech Rep Ser. 1995; 854:1-452.
- 15. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis. Report of a WHO Study Group. World Health Organ Tech Rep Ser. 1994;843:1-129.
- Ma L, Oei L, Jiang L, Estrada K, Chen H, Wang Z, et al. Association between bone mineral density and type 2 diabetes mellitus: A metaanalysis of observational studies. Eur J Epidemiol. 2012;27(5):319-332.
- Vestergaard P. Discrepancies in bone mineral density and fracture risk in patients with type 1 and type 2 diabetes—a meta-analysis. Osteoporos Int. 2007;18(4):427-444.
- Leidig-Bruckner G, Grobholz S, Bruckner T, Scheidt-Nave C, Nawroth P, Schneider JG. Prevalence and determinants of osteoporosis in patients with type 1 and type 2 diabetes mellitus. BMC Endocr Disord. 2014;14(1):1-3.
- Raj S, Baiju SJ, Vijayan R, Rajan GV. Association between Bone Mineral Density and Type 2 Diabetes Mellitus-An Original. BJR. 2014;(2):63-67.
- Prakash S, Jatti R, Ghagane S, Jali SM, Jali MV. Prevalence of osteoporosis in type 2 diabetes mellitus patients using dual energy X-ray absorptiometry (DEXA) scan. Int J Osteoporos Metab Disord. 2017;10(2):10-16.
- 21. Yaturu S. Diabetes and skeletal health. J Diabetes. 2009;1(4):246-254.
- 22. Dutta N, Saikia AM, Saikia AM, Das AK. Status of bone mineral density in adult population using calcaneal ultrasound bone densitometer: A study from Assam, India. Indian J Basic Appl Med Res. 2015;4:150-158.
- 23. Wakasugi M, Wakao R, Tawata M, Gan N, Koizumi K, Onaya T. Bone mineral density measured by dual energy x-ray absorptiometry in patients with non-insulin-dependent diabetes mellitus. Bone. 1993;14(1):29-33.
- 24. Reid IR, Evans MC, Cooper GJ, Ames RW, Stapleton JO. Circulating insulin levels are related to bone density in normal postmenopausal

Mathkhor AJ, et al.

women. Am J Physiol. 1993;265(4):E655-E659.

- 25. Paul RG, Bailey AJ. Glycation of collagen: the basis of its central role in the late complications of ageing and diabetes. Int J Biochem Cell Biol. 1996;28(12):1297-1310.
- 26. Kawai M, Rosen CJ. Insulin-like growth factor-I and bone: lessons from mice and men. Pediatr Nephrol. 2009;24(7):1277-1285.
- Wasnich RD, Benfante RJ, Yano K, Heilbrun L, Vogel JM. Thiazide effect on the mineral content of bone. N Engl J Med. 1983;309(6):344-347.
- 28. Yamagishi S, Nakamura K, Inoue H. Possible participation of advanced

glycation end products in the pathogenesis of osteoporosis in diabetic patients. Med Hypotheses. 2005;65(6):1013-1015.

- 29. Shin D, Kim S, Kim KH, Lee K, Park SM. Association between insulin resistance and bone mass in men. J Clin Endocrinol Metab. 2014 Mar 1;99(3):988-995.
- 30. 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):1-9.
- Pun KK, Lau P, Ho PW. The characterization, regulation, and function of insulin receptors on osteoblast-like clonal osteosarcoma cell line. J Bone Miner Res. 1989;4(6):853-862.