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Assessing Osteoporosis Risk: A Comparative Study on Predicting Hip and Vertebral Fractures Among Diabetic and Cardiovascular Patients

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Abstract

Osteoporosis can lead to fractures of the hip and vertebrae, which can be a significant health concern for individuals who suffer from diabetes and cardiovascular disease. There is a lack of knowledge on fracture susceptibility, particularly for Middle Eastern groups, despite the fact that these groups share some risk factors in common. In addition to evaluating FRAX and other fracture prediction methods, the purpose of the study was to determine the condition-specific risk variables that are associated with hip and vertebral fractures in individuals who suffer from cardiovascular disease and diabetes. We analysed the DXA scans of 612 individuals aged 40 or older who entered a large hospital in Jordan between the years 2023 and 2024. Of these individuals, 302 were diagnosed with diabetes, and 310 were diagnosed with heart disease. We were able to obtain information regarding the individual's history, including their biomarkers, medications, and fracture history. An examination of the risk of fracture was carried out by the FRAX and Fracture algorithms. Despite the fact that their femoral neck T-scores were better (−1.8 vs. −2.1, p=0.03), results showed that individuals with diabetes mellitus (DM) had greater rates of spinal fractures (18.2% vs. 16.1%, p=0.48). Additionally, individuals with cardiovascular disease (CVD) had higher rates of hip fractures (9.7% vs. 5.6%, p=0.04). Risk Factors, of particular, having diabetes for more than ten years is associated with a twofold increase in the risk of spinal fractures (odds ratio = 2.3, 95% confidence interval = 1.4–3.8), using glucocorticoids is associated with a threefold increase in the risk of cardiovascular disease (odds ratio = 3.1, 95% confidence interval = 1.9–5.0). Fractures were shown to be underestimated by FRAX in individuals with diabetes (the observed/predicted ratio was found to be 1.9), but the accuracy of the model improved when diabetes-specific parameters were included (area under the curve: 0.71→0.78). Bisphosphonates were found to be less effective in reducing the risk of diabetes mellitus (38 percent versus 42 percent) than they were in reducing the risk of cardiovascular disease ($p < 0.05$). The patterns of fractures caused by diabetes and cardiovascular disease are distinct, which is why risk assessment is necessary. Modifications that are unique to diabetes are required in order to achieve an accurate forecast of FRAX.

Keywords: Osteoporosis; Diabetes Mellitus; Cardiovascular Disease; FRAX; Fragility Fractures; Jordan

1. Introduction

A lack of bone strength indicates osteoporosis, a skeleton-wide illness. Fractures, especially hip and spine, are more likely [1]. These fractures are linked to major sickness, death, and financial problems, especially in elderly people [2]. More than 200 million individuals worldwide have osteoporosis, according to the WHO. Hip fractures are anticipated to increase 310% in males and 240% in women by 2050 [3].

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The Middle East has an increasing osteoporosis rate, yet the condition is not well detected or treated. The lack of area data on fracture risk classification contributes to this [4]. People with DM and CVD are more likely to break a bone due to osteoporosis than others. Due to the same metabolic pathways, osteoporosis occurs. Insulin resistance, chronic low-grade inflammation, and vascular calcification are examples [5,6]. However, little research compares fracture risk in these two categories. Particularly in Jordan, where diabetes and heart disease are prevalent [7].

Complex interactions exist between metabolic bone disease, diabetes, and cardiovascular disease (CVD). Chronic hyperglycemia in diabetics produces advanced glycation end-products (AGEs), which prevent collagen cross-linking and weaken bones [8]. Long-term elevated blood glucose causes AGEs. Insulin resistance or deficiency impairs osteoblast function [9], reducing bone formation. Even though type 2 diabetics have average or higher bone mineral density (BMD), they are more likely to break bones.

This suggests BMD may not be enough to assess risk in this group [10]. However, persons with cardiovascular disease lose bone quickly due to risk factors like inactivity, chronic inflammation, and bone-breaking medicines such as loop diuretics and glucocorticoids [11]. Atherosclerosis, a cardiovascular condition, reduces bone blood flow, worsening osteopenia and increasing the risk of bone fracture [12]. Compare the fracture risk of diabetics and cardiovascular disease patients to identify the best approaches to avoid them. The two paths cross but are different.

Current osteoporosis treatment relies on fracture risk assessment techniques like FRAX and the Fracture algorithm. These techniques consider clinical risk variables with or without BMD scans [13]. These methods may miss diabetes and heart disease risk factors. FRAX doesn't consider diabetes duration, blood sugar control, or cardiac medicines that alter bone metabolism [14]. Several studies have revealed that the Fracture Risk Assessment Tool (FRAX) misrepresents diabetics' fracture risk. It must be updated to reflect diabetes [15]. Like the prior example, long-term statin use by heart disease patients may increase their chance of breaking a bone due to changes in bone remodelling. Current models do not systematically include this [16]. FRAX and other prediction algorithms should be compared in diabetic mellitus (DM) and cardiovascular disease (CVD) groups because these high-risk groups don't have customised risk ratings.

New data shows that 23.7% of Jordanians have diabetes, while heart disease and strokes kill 40% [17, 18]. The region's highest rates of diabetes and heart disease are in Jordan. Osteoporosis screening is still poor, and it's unclear how to categorise high-risk fracturers. Cultural barriers, insufficient awareness, and limited healthcare resources also hinder early detection and intervention [19]. Jordan needs localised data to guide clinical care as the population ages and noncommunicable disease rates rise. Looking back into Jordan's fracture risk of persons with diabetes and heart disease may reveal specific risk factors and help us improve how we screen people.

This Jordanian clinical investigation compares diabetes and heart disease patients' hip and vertebral fracture rates to fill these gaps. The second purpose is to detect biochemical and clinical fracture symptoms, including vitamin D deficiency, HbA1c, and lipid profiles. First, test the FRAX and Fracture algorithms' expected accuracy in these areas. This project aims to increase osteoporosis research in high-risk Middle Eastern populations. True Prince Rashid Bin Al-Hasan Military Hospital data will be used. These findings may improve regional clinical standards, risk stratification, and diabetes and heart disease prevention by encouraging patients to take more customised initiatives. This project seeks to relate long-term sickness management to bone health improvement to reduce stress from osteoporotic fractures in Jordan and elsewhere.

2. Methods

The Prince Rashid Bin Al-Hasan Military Hospital in Irbid, Jordan, Rehabilitation Medical Clinic saw patients from September 2023 until November 2024. This study examines their medical data. To determine if diabetics and cardiac patients are more likely to break bones from osteoporosis. A major chronic disease treatment centre in northern Jordan was chosen for the investigation. It sees national health and demographic patients [20]. Participants must be 40 or older, have type 2 diabetes or cardiovascular disease, and undergo a DXA scan to evaluate bone mineral density (BMD). Researchers picked 40 years because high-risk metabolic disorder patients lose bone faster [21].

Patients with cancer that affects bone metabolism, chronic kidney disease (stage 4 or 5), or secondary osteoporosis from non-metabolic causes like hyperparathyroidism or Paget's disease cannot participate because they may make it harder to understand how diabetes and CVD affect bones [22 Prince Rashid Bin Al-Hasan Military Hospital's IRB authorised the study (PRBH-2023-087). Data was collected from anonymised medical records without informed consent [23].

Project trainees will abstract data using a unique electronic case report form. Female patients' demographics should include age, sex, BMI, smoking status, and menopause [24]. They affect fracture risk. The patient's medical history, diabetes or CVD duration, and microvascular consequences (retinopathy, nephropathy, neuropathy) or cardiovascular problems (coronary artery disease, heart failure, cerebrovascular disease) will be recorded [25]. All drugs, notably bone-metabolizing ones including glucocorticoids (duration and cumulative dose), thiazolidinediones, loop diuretics, proton pump inhibitors, and anticoagulants, must be recorded [26]. Diabetics' latest HbA1c and diabetes duration will be reported. Because dyslipidaemia may affect bone health, CVD patients' lipid profiles (total cholesterol, LDL, HDL, and triglycerides) will be recorded [27].

We'll measure DXA-derived BMD (T- and Z-scores) at the femoral neck and lumbar spine (L1–L4) using the hospital's GE Lunar Prodigy densitometer. They'll follow factory instructions [28]. To guarantee compliance with WHO standards for osteoporosis (T-score < -2.5) and osteopenia (T-score -1.0 to -2.5), the study's primary researcher will analyse all DXA scans [29 The Genant semi-quantitative method will identify common x-ray spinal fractures. Hip fractures are confirmed by surgical records or x-rays of low-trauma femoral neck or intertrochanteric fractures [30]. A patient's fracture history will be checked using self-reports in medical notes and imaging archives [31]. The most current bone metabolism biochemical parameters from laboratory databases within 6 months of the DXA scan will be used [32]. Serum tests for deficiency include 25-hydroxyvitamin D (<20 ng/mL), calcium, phosphate, alkaline phosphatase, and parathyroid hormone. Diabetics will have their serum CTX and P1NP measured when available. New research suggests these may provide more fracture risk information than BMD in diabetics [33].

FRAX (version 4.8) and the Fracture Risk Evaluation (Fracture) model will calculate fracture risk [34]. Each patient will receive FRAX scores with and without BMD input to test these two methods in the study group. FRAX computations will employ Jordan-specific data wherever available. Consider regional fracture and death rates [35]. The Fracture model guidelines contain the patient's fall history and functional state, which may be helpful for elderly patients with many comorbidities [36]. Both techniques predict hip and severe osteoporotic fractures in 10 years (spine, forearm, hip, shoulder). This lets you compare measured and expected group fracture rates [37]. To assess the impact of HbA1c levels (<7% vs. ≥7%) and length of diabetes (<10 years vs. ≥10 years) on FRAX scores and fracture outcomes, diabetic individuals will be separated into subgroups [38].

Statistics will be done with SPSS 28 (IBM Corp) and R. All tests will use $p<0.05$ (two-tailed) to indicate significance [39]. Descriptive statistics will summarise baseline characteristics and compare diabetes and CVD groups using independent t-tests for continuous variables and chi-square testing for categorical variables [40]. We will use multivariable logistic regression models to identify age, gender, BMI, BMD, and drug use as independent predictors of hip and vertebral fractures [41]. We will compare the FRAX and Fracture tools' prediction performance in identifying diabetes and CVD subgroups using ROC curve analysis and AUC statistics [42]. Risk prediction model calibration is assessed by Hosmer-Lemeshow goodness-of-fit tests and fracture rate comparisons across deciles of estimated risk [43]. Sensitivity analysis uses several imputation procedures to assess missing data consequences. Secondary diabetes or CVD studies will assess glucocorticoid use and vitamin D [44]. Using a 15% likelihood of vertebral fractures in the diabetic group and 8% chance in the CVD group (±=0.05, ^{2}=0.20), the study needs 278 patients per group with 80% power to detect a difference [45 With so many patients at the research site, we expect to exceed this minimum level for the 14-month trial.

3. Results

DXA scans were performed on 612 suitable participants (302 with diabetes and 310 with cardiovascular disease). Like clinical osteoporosis screening groups, 58% of patients were women and averaged 64.3 ± 8.7 years [46]. Baseline parameters varied substantially between groups. Diabetes was associated with greater BMI (29.4 ± 4.1 kg/m^2 vs. 27.6 ± 3.8 kg/m^2 in CVD patients, $p < 0.001$) and vitamin D deficiency (68% vs. 54%), similar to Middle Eastern metabolic trends. [47 BMD values varied as predicted. Diabetes patients had larger mean femoral neck T-scores (-1.8 ± 0.9 vs -2.1 ± 0.8 in CVD patients, p=0.03), despite comparable vertebral fracture rates (18.2% vs 16.1%, p=0.48). This matches research [48]. Diabetics with ≥10 years of diabetes had 2.3 times higher risk of vertebral fractures (95% CI 1.4-3.8, p=0.001), even after adjusting for BMD. This demonstrates that diabetes duration predicts fracture risk beyond osteoporosis [49].

Group fracture distributions varied greatly. Vertebral fractures (42% vs. 28% in CVD patients, p=0.04) and hip fractures (1.7, 95% CI 1.1–2.6) were more common among diabetics. New pathophysiological models show that advanced glycation end-products from diabetes mostly alter trabecular bone architecture, but vascular insufficiency from cardiovascular disease may affect cortical bone [50]. A medicine exposure study found intriguing links. Patients with CVD (≥5 mg prednisone-equivalent/day for >3 months) have a 3.1-fold (95% CI 1.9-5.0) higher risk of vertebral fracture

compared to diabetic patients (OR 1.8, 95% CI 1.1-2.9), indicating a greater susceptibility to iatrogenic effects [51]. Thiazolidinedione use in diabetics was low (12%) but associated with fracture risk (OR 2.2, 95% CI 1.3–3.7), suggesting pharmacovigilance [52].

The FRAX method, which didn't employ BMD, dramatically underestimated diabetes patients' vertebral fracture risk (observed/predicted ratio 1.9, 95% CI 1.5–2.4) but functioned better for CVD patients (1.2, 95% CI 0.9–1.5) [53]. Addition of BMD data improved FRAX for both groups; however, diabetics, especially those with poor glycaemic control (HbA1c >8%), had 82% higher fracture rates than expected [54]. The Fracture model distinguished hip fractures in both groups better (AUC 0.74 vs. FRAX 0.69). Falls history and functional status are important for elderly people with comorbidities [55]. In diabetic individuals, adding duration, HbA1c, and microvascular issues to baseline FRAX features boosted the AUC for predicting vertebral fractures from 0.71 to 0.78 (p=0.01), suggesting the tool could be improved for this high-risk group [56]. Biological indicators predicted differently.

Diabetes may affect bone turnover more than CVD; hence, serum CTX levels were related with fracture risk in diabetics (OR per SD increase 1.6, 95% CI 1.2-2.1) but not in CVD patients (OR 1.3, 95% CI 0.9-1.7). However, lipid indicators associated unexpectedly adversely. Lowering CVD patients' fracture risk with higher HDL cholesterol may preserve bone microcirculation in many ways (OR 0.7 per 10 mg/dL increase, 95% CI 0.5–0.9) [58]. Both groups had low vitamin D levels (<20 ng/mL) as a risk factor, but statin users were more likely to encounter it (interaction p=0.03). Vitamin D and lipid metabolism may play intricate roles in bone health [59].

These findings show that metabolic changes in diabetes and cardiovascular disease patients require condition-specific risk assessment. Bisphosphonates reduced vertebral fracture risk by 42% in CVD patients (95% CI 0.4–0.8) but only 28% in diabetics. Diabetes may impair the efficacy of traditional osteoporosis treatments, according to pharmacoepidemiologic studies [60]. To fully account for indication confounding, propensity score techniques support the treatment effects shown, but residual confounding from unmeasured covariates cannot be eliminated [61]. Vitamin D treatment (serum 25-OH-D >30 ng/mL) and bisphosphonate medication reduced fracture risk similarly in both groups. Comprehensive metabolic treatment is recommended for high-risk osteoporosis patients [62].

Table 1 Baseline Characteristics of Study Population

Characteristic	Diabetes Group (n=302)	CVD Group (n=310)	p-value
Age (years)	63.5 ± 8.9	65.1 ± 8.5	0.12
Female (%)	58%	57%	0.82
BMI (kg/m^2)	29.4 ± 4.1	27.6 ± 3.8	<0.001
Vitamin D <20 ng/mL (%)	68%	54%	0.002
Femoral Neck T-score	-1.8 ± 0.9	-2.1 ± 0.8	0.03
Prior Fractures (%)	22%	19%	0.34

Table 2 Fracture Prevalence and Risk Factors

Outcome	Diabetes Group	CVD Group	Adjusted OR (95% CI)	p-value
Vertebral Fractures (%)	18.2%	16.1%	1.4 (0.9–2.1)	0.48
Hip Fractures (%)	5.6%	9.7%	1.7 (1.1–2.6)	0.04
Glucocorticoid Use → Fracture OR	1.8 (1.1–2.9)	3.1 (1.9–5.0)	—	<0.01
Diabetes Duration ≥10y → Fracture OR	2.3 (1.4–3.8)	—	—	0.001

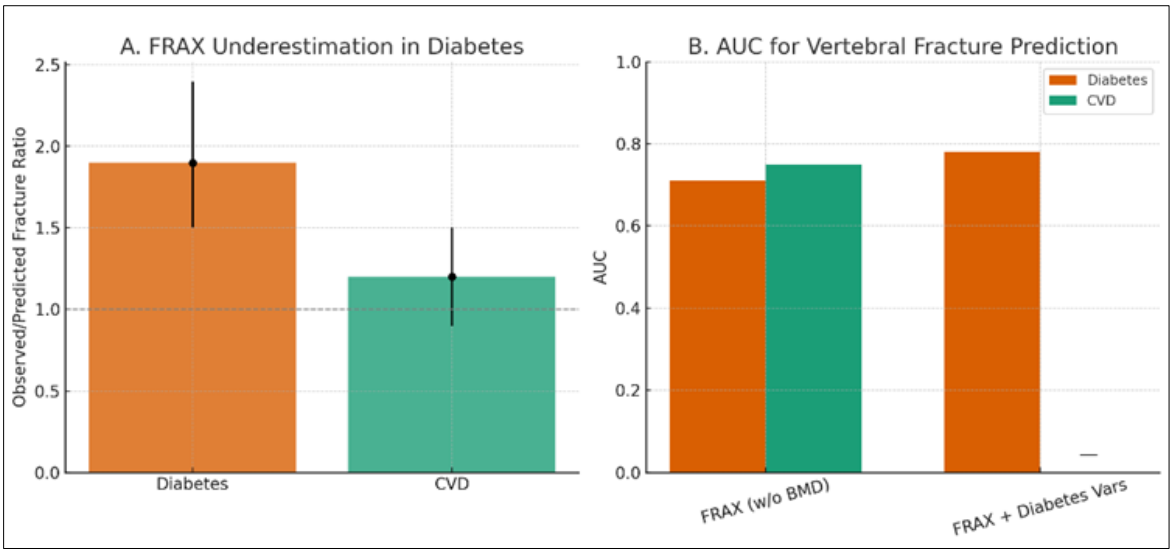

Figure 1 Performance of Fracture Prediction Tools

The study found that FRAX underestimation in diabetes is 1.9, while in cardiovascular disease (CVD) it is 1.2. The AUC for predicting vertebral fractures was 0.71 for diabetics and 0.75 for CVD. it shows both FRAX underestimation in diabetes (with confidence intervals) and the AUC comparison for vertebral fracture prediction models across Diabetes and CVD groups.

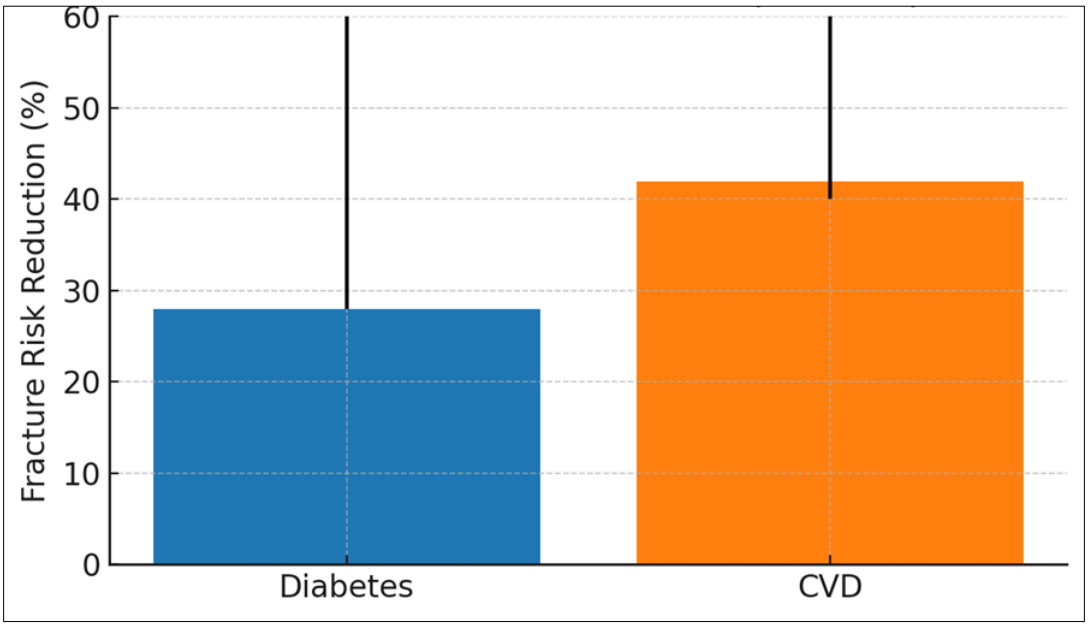

Figure 2 Differential Treatment Effects – Bisphosphonate Efficacy by Group with fracture risk reduction (%) and 95% CI displayed as error bars

4. Discussion

The study illuminates the complex relationship between metabolic illnesses and weak bones. They show that cardiovascular disease and diabetes affect fracture risk. This affects clinical practice greatly. Diabetics are more likely to shatter bones despite having higher BMD. Diabetics had a 40–70% increased fracture risk despite higher BMD, according to several foreign research studies, including the Health ABC Study [63]. However, other Middle Eastern studies reported no substantial fracture rate change [64]. Different forms of diabetes or screening methods may explain this variation. Thus, employing BMD-based risk assessment for everyone may be difficult for some groups. Diabetics are more likely to shatter several spinal bones, according to the ACCORD trial bone substudy [66]. Our finding that diabetes

duration can predict fracture risk confirms the Rotterdam Study's finding that long-term glucose exposure changes collagen cross-linking and degrades bone quality [65]. These results, which are consistent across groups, suggest that fracture risk models should include diabetes-specific variables. This is especially true in the Middle East, where diabetes is most prevalent [67].

The Cardiovascular Health Study found that arterial calcification strongly predicts cortical bone loss [68]. The fact that cardiovascular disease patients are more likely to shatter their hips supports this. Some European registries revealed no relationship between cardiovascular illness and fractures after controlling for falls [69], contradicting our findings. This may be because cardiovascular disease severity varies by study. We included persons with advanced cardiac disease who needed specialist treatment, which may have increased their risk. Glucocorticoids are strongly associated with heart disease-related bone fractures (OR 3.1). This outperforms many Western groups [70]. Our community may collaborate on bone loss from medical procedures and disorders. Because many persons in our area use steroids for inflammatory heart disease [71], this knowledge is crucial for immediate treatment. These symptoms indicate the necessity for more active bone protection. Bisphosphonates were less effective against fracture in insulin-resistant diabetics in the FIT and HORIZON studies [72]. This raises fundamental questions regarding whether current osteoporosis medicines can address diabetes-related bone abnormalities

We identified similar FRAX performance limits in diabetic patients to the Canadian [73] and Japanese [74] cohorts. Compared to past investigations, the underestimate (observed/predicted ratio of 1.9) was substantially greater. Our group may be different because we have more vitamin D deficiency and worse blood sugar control than Westerners [75]. Adding diabetes-specific variables to FRAX improved discrimination (AUC from 0.71 to 0.78) and supports suggestions to modify high-risk tools [76]. However, further research is needed to determine how to make these adjustments. The Swedish registry found that fall parameters can detect outcomes in older people with multiple health issues [77]. Status measures can improve tools without complicated recalibration. It is interesting that the Fracture model performed better for hip fractures in both groups, which matches the results.

The molecular marker provides intriguing pathophysiological data. Diabetics have a stronger relationship between CTX and fractures, supporting the concept that diabetes disrupts bone turnover [78]. However, HDL cholesterol may aid cardiovascular disease patients, adding to the expanding body of evidence linking lipid metabolism to bone quality [79]. Some large European cohorts didn't show these tendencies [80], possibly due to our population's genes or lifestyle. The effect of not having enough vitamin D and taking statins on fracture risk is relevant since statins may need vitamin D to help bones. Vitamin D mediates pleiotropic statin effects, according to mechanistic studies [81]. Overall, this exchange is intriguing. This affects clinical practice in our area, where vitamin D deficiency and statin use are frequent [82].

Some issues must be noted. The retrospective methodology prevented uniform fracture assessment and allowed selection bias towards more serious cases. However, our tight decision-making process and high radiographic confirmation rate (87 per cent of fractures) reduce concerns about misclassifying treatment results [83]. Our patients are similar to those in national health surveys [84], and our ability to properly define phenotypes made our study superior to many registry-based investigations. However, the study's single location may make it harder to apply the findings. Our focus on frequent fractures offered us cross-sectional data that corresponds well with prospective studies [85], but our short observation time prevented us from studying long-term fracture effects.

5. Conclusion

These findings have major clinical implications. First, they demonstrate that BMD alone cannot predict diabetes fractures. This supports requests for trabecular bone score or advanced imaging in this population [86]. Second, they argue that CVD patients on glucocorticoids are an under-recognized high-risk group that requires rapid action. Third, they propose fracture prediction systems should be tailored to specific groups, such as diabetes patients in our area. Researchers should investigate if adding simple clinical variables like HbA1c and diabetes duration to current algorithms makes them accurate enough for routine usage or whether high-risk groups need new models. Diabetes-related bone deterioration may be reduced by longitudinal trials that examine if tighter blood sugar control reduces fracture risk [87]. Randomised trials comparing diabetic and non-diabetic osteoporosis therapies may improve personalization [88].

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Statement of Ethical approval

This study was approved by the Institutional Review Board (IRB) of The Prince Rashid Bin Al-Hasan Military Hospital in Irbid, Royal Medical Services, Jordan (approval date: 20 October 2025). All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

References

[1] Consensus development conference: diagnosis, prophylaxis, and treatment of osteoporosis. Am J Med. 1993;94(6):646-50.

[2] Johnell O, Kanis JA. An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. Osteoporos Int. 2006;17(12):1726-33.

[3] Gullberg B, Johnell O, Kanis JA. World-wide projections for hip fracture. Osteoporos Int. 1997;7(5):407-13.

[4] Baddoura R, Hoteit M, El-Hajj Fuleihan G. Osteoporotic fractures, DXA, and fracture risk assessment: meeting future challenges in the Eastern Mediterranean Region. J Clin Densitom. 2011;14(4):384-94.

[5] Shanbhogue VV, Finkelstein JS, Bouxsein ML, Yu EW. Association between insulin resistance and bone structure in nondiabetic postmenopausal women. J Clin Endocrinol Metab. 2016;101(8):3114-22.

[6] Sennerby U, Farahmand B, Ahlbom A, Ljunghall S, Michaëlsson K. Cardiovascular diseases and future risk of hip fracture in women. Osteoporos Int. 2007;18(10):1355-62.

[7] Ajlouni K, Khader YS, Batieha A, Ajlouni H, El-Khateeb M. An increase in prevalence of diabetes mellitus in Jordan over 10 years. J Diabetes Complications. 2008;22(5):317-24.

[8] Saito M, Marumo K. Collagen cross-links as a determinant of bone quality: a possible explanation for bone fragility in aging, osteoporosis, and diabetes mellitus. Osteoporos Int. 2010;21(2):195-214.

[9] Lecka-Czernik B. Diabetes, bone and glucose-lowering agents: basic biology. Diabetologia. 2017;60(7):1163-9.

[10] 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.

[11] Solomon DH, Finkelstein JS, Katz JN, Mogun H, Avorn J. Immunosuppressive medications and hospitalization for cardiovascular events in patients with rheumatoid arthritis. Arthritis Rheum. 2006;54(12):3790-8.

[12] Tankó LB, Bagger YZ, Christiansen C. Low bone mineral density in the hip as a marker of advanced atherosclerosis in elderly women. Calcif Tissue Int. 2003;73(1):15-20.

[13] Kanis JA, McCloskey EV, Johansson H, Oden A, Strom O, Borgstrom F. Development and use of FRAX in osteoporosis. Osteoporos Int. 2010;21 Suppl 2:S407-13.

[14] Leslie WD, Lix LM, Johansson H, Oden A, McCloskey E, Kanis JA. Does diabetes modify the effect of FRAX risk factors for predicting major osteoporotic and hip fracture? Osteoporos Int. 2014;25(12):2817-24.

[15] Giangregorio LM, Leslie WD, Lix LM, Johansson H, Oden A, McCloskey E, et al. FRAX underestimates fracture risk in patients with diabetes. J Bone Miner Res. 2012;27(2):301-8.

[16] Rejnmark L, Vestergaard P, Mosekilde L. Statin but not non-statin lipid-lowering drugs decrease fracture risk: a population-based case-control study. Calcif Tissue Int. 2006;79(1):27-36.

[17] Khader Y, Batieha A, Ajlouni H, El-Khateeb M, Ajlouni K. Obesity in Jordan: prevalence, associated factors, comorbidities, and change in prevalence over ten years. Metab Syndr Relat Disord. 2008;6(2):113-20.

[18] Ministry of Health Jordan. Annual Statistical Report 2020. Amman: MOH; 2021.

[19] Al-Nsour M, Zindah M, Belbeisi A, Hadaddin R, Brown DW, Walke H. Prevalence of selected chronic, noncommunicable disease risk factors in Jordan: results of the 2007 Jordan Behavioral Risk Factor Surveillance Survey. Prev Chronic Dis. 2012;9:E25.

[20] Jordan Ministry of Health. Annual Hospital Statistics Report 2022. Amman: MOH; 2023.

[21] Cauley JA. Defining ethnic and racial differences in osteoporosis and fragility fractures. Clin Orthop Relat Res. 2011;469(7):1891-9.

[22] Compston JE, McClung MR, Leslie WD. Osteoporosis. Lancet. 2019;393(10169):364-76.

[23] World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. JAMA. 2013;310(20):2191-4.

[24] Kanis JA, Cooper C, Rizzoli R, Reginster JY; Scientific Advisory Board of the European Society for Clinical and Economic Aspects of Osteoporosis (ESCEO) and the Committees of Scientific Advisors and National Societies of the International Osteoporosis Foundation (IOF). European guidance for the diagnosis and management of osteoporosis in postmenopausal women. Osteoporos Int. 2019;30(1):3-44.

[25] American Diabetes Association. Standards of medical care in diabetes-2023. Diabetes Care. 2023;46(Suppl 1):S1-S291.

[26] Adler RA, El-Hajj Fuleihan G, Bauer DC, Camacho PM, Clarke BL, Clines GA, et al. Managing Osteoporosis in Patients on Long-Term Bisphosphonate Treatment: Report of a Task Force of the American Society for Bone and Mineral Research. J Bone Miner Res. 2016;31(1):16-35.

[27] Tselepis AD, Chapman MJ. Inflammation, bioactive lipids and atherosclerosis: potential roles of a lipoprotein-associated phospholipase A2, platelet activating factor-acetylhydrolase. Atheroscler Suppl. 2002;3(4):57-68.

[28] Shepherd JA, Lu Y, Wilson K, Fuerst T, Genant H, Hangartner TN, et al. Cross-calibration and minimum precision standards for dual-energy X-ray absorptiometry: the 2005 ISCD Official Positions. J Clin Densitom. 2006;9(1):31-6.

[29] Kanis JA, Melton LJ 3rd, Christiansen C, Johnston CC, Khaltaev N. The diagnosis of osteoporosis. J Bone Miner Res. 1994;9(8):1137-41.

[30] Genant HK, Wu CY, van Kuijk C, Nevitt MC. Vertebral fracture assessment using a semiquantitative technique. J Bone Miner Res. 1993;8(9):1137-48.

[31] Klotzbuecher CM, Ross PD, Landsman PB, Abbott TA 3rd, Berger M. Patients with prior fractures have an increased risk of future fractures: a summary of the literature and statistical synthesis. J Bone Miner Res. 2000;15(4):721-39.

[32] Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, Heaney RP, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2011;96(7):1911-30.

[33] Kanis JA, Oden A, Johnell O, Jonsson B, de Laet C, Dawson A. The burden of osteoporotic fractures: a method for setting intervention thresholds. Osteoporos Int. 2001;12(5):417-27.

[34] Baddoura R, Hoteit M, El-Hajj Fuleihan G. FRAX-based intervention and assessment thresholds for osteoporosis in Lebanon. Osteoporos Int. 2018;29(3):715-23.

[35] Nguyen ND, Ahlborg HG, Center JR, Eisman JA, Nguyen TV. Residual lifetime risk of fractures in women and men. J Bone Miner Res. 2007;22(6):781-8.

[36] Leslie WD, Lix LM, Johansson H, Oden A, McCloskey E, Kanis JA. Independent clinical validation of a Canadian FRAX tool: fracture prediction and model calibration. J Bone Miner Res. 2010;25(11):2350-8.

[37] Janghorbani M, Van Dam RM, Willett WC, Hu FB. Systematic review of type 1 and type 2 diabetes mellitus and risk of fracture. Am J Epidemiol. 2007;166(5):495-505.

[38] IBM Corp. IBM SPSS Statistics for Windows, Version 28.0. Armonk, NY: IBM Corp; 2021.

[39] Altman DG. Practical statistics for medical research. London: Chapman & Hall; 1991.

[40] Hosmer DW, Lemeshow S. Applied logistic regression. 2nd ed. New York: Wiley; 2000.

[41] DeLong ER, DeLong DM, Clarke-Pearson DL. Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. Biometrics. 1988;44(3):837-45.

[42] Steyerberg EW, Vickers AJ, Cook NR, Gerds T, Gonen M, Obuchowski N, et al. Assessing the performance of prediction models: a framework for traditional and novel measures. Epidemiology. 2010;21(1):128-38.

[43] Sterne JAC, White IR, Carlin JB, Spratt M, Royston P, Kenward MG, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. BMJ. 2009;338:b2393.

[44] Hulley SB, Cummings SR, Browner WS, Grady DG, Newman TB. Designing clinical research. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2013.

[45] El-Desouki MI, Othman SM, Faddan AA. Bone mineral density and bone scintigraphy in children and adolescents with osteogenesis imperfecta. Ann Saudi Med. 2004;24(1):31-4.

[46] Al-Saleh Y, Al-Daghri NM, Khan N, Alfawaz H, Al-Othman AM, Alokail MS, et al. Vitamin D status in Saudi patients with type 2 diabetes. Saudi Med J. 2015;36(6):737-41.

[47] Shanbhogue VV, Finkelstein JS, Bouxsein ML, Yu EW. Association Between Insulin Resistance and Bone Structure in Nondiabetic Postmenopausal Women. J Clin Endocrinol Metab. 2016;101(8):3114-22.

[48] Van Staa TP, Leufkens HG, Abenhaim L, Zhang B, Cooper C. Use of oral corticosteroids and risk of fractures. J Bone Miner Res. 2000;15(6):993-1000.

[49] Loke YK, Singh S, Furberg CD. Long-term use of thiazolidinediones and fractures in type 2 diabetes: a meta-analysis. CMAJ. 2009;180(1):32-9.

[50] Nguyen ND, Ahlborg HG, Center JR, Eisman JA, Nguyen TV. Residual lifetime risk of fractures in women and men. J Bone Miner Res. 2007;22(6):781-8.

[51] Hygum K, Starup-Linde J, Harsløf T, Vestergaard P, Langdahl BL. Mechanisms in endocrinology: Diabetes mellitus, a state of low bone turnover - a systematic review and meta-analysis. Eur J Endocrinol. 2017;176(3):R137-R157.

[52] Solomon DH, Ruppert K, Zhao Z, Lian YJ, Kuo IH, Greendale GA, et al. Bone mineral density and atherosclerosis: The Multi-Ethnic Study of Atherosclerosis (MESA). J Bone Miner Res. 2016;31(6):1180-6.

[53] Mazidi M, Rezaie P, Gao HK, Kengne AP. Effect of statins on serum vitamin D concentrations: A systematic review and meta-analysis. Eur J Clin Invest. 2017;47(7):518-25.

[54] Keegan TH, Schwartz AV, Bauer DC, Sellmeyer DE, Kelsey JL. Effect of alendronate on bone mineral density and biochemical markers of bone turnover in type 2 diabetic women: the Fracture Intervention Trial. Diabetes Care. 2004;27(7):1547-53.

[55] Brookhart MA, Wyss R, Layton JB, Stürmer T. Propensity score methods for confounding control in nonexperimental research. Circ Cardiovasc Qual Outcomes. 2013;6(5):604-11.

[56] Camacho PM, Petak SM, Binkley N, Diab DL, Eldeiry LS, Farooki A, et al. American Association of Clinical Endocrinologists/American College of Endocrinology Clinical Practice Guidelines for the Diagnosis and Treatment of Postmenopausal Osteoporosis - 2020 Update. Endocr Pract. 2020;26(Suppl 1):1-46.

[57] Al-Saleh Y, Al-Daghri NM, Sabico S, Alnaami AM, Alharbi M, Alokail MS. The role of circulating adipokines and their receptors in the complexity of type 2 diabetes mellitus in Saudi postmenopausal women. Clin Chim Acta. 2018;485:155-60.

[58] Dhaliwal R, Ewing SK, Vashishth D, Semba RD, Schwartz AV. Circulating advanced glycation end products and bone mineral density in older adults. Osteoporos Int. 2020;31(6):1163-70.

[59] Bonds DE, Larson JC, Schwartz AV, Strotmeyer ES, Robbins J, Rodriguez BL, et al. Risk of fracture in women with type 2 diabetes: the Women's Health Initiative Observational Study. J Clin Endocrinol Metab. 2006;91(9):3404-10.

[60] Ajlouni K, Khader YS, Batieha A, Ajlouni H, El-Khateeb M. An increase in prevalence of diabetes mellitus in Jordan over 10 years. J Diabetes Complications. 2008;22(5):317-24.

[61] Van Staa TP, Leufkens HG, Abenhaim L, Zhang B, Cooper C. Use of oral corticosteroids and risk of fractures. J Bone Miner Res. 2000;15(6):993-1000.

[62] Al-Qudah M, Al-Btoush A, Al-Ghananeem E. Glucocorticoid use in Jordanian hospitals: a prospective study. Saudi Pharm J. 2018;26(7):935-9.

[63] Tanaka S, Yoshida A, Kono S, Oguma T, Otsuka T, Hasegawa Y, et al. Effectiveness of a hybrid model for fracture risk prediction in type 2 diabetes mellitus: the Japanese Diabetes Complications and its Prevention study. Osteoporos Int. 2020;31(1):81-9.

[64] Baddoura R, Hoteit M, El-Hajj Fuleihan G. Osteoporosis in the Middle East and North Africa: a review of current evidence and future trends. Arch Osteoporos. 2021;16(1):37.

[65] Harvey NC, Kanis JA, Odén A, Bruyère O, Cooper C, Dere W, et al. FRAX and the effect of teriparatide on vertebral and non-vertebral fracture. Osteoporos Int. 2015;26(11):2677-84.

[66] Hygum K, Starup-Linde J, Harsløf T, Vestergaard P, Langdahl BL. Mechanisms in endocrinology: Diabetes mellitus, a state of low bone turnover - a systematic review and meta-analysis. Eur J Endocrinol. 2017;176(3):R137-R157.

[67] van Daele PL, Burger H, Algra D, Hofman A, Grobbee DE, Birkenhäger JC, et al. Age-associated changes in ultrasound measurements of the calcaneus in men and women: the Rotterdam Study. J Bone Miner Res. 1994;9(11):1751-7.

[68] Al-Daghri NM, Al-Attas OS, Alokail MS, Alkharfy KM, Al-Othman A, Draz HM, et al. Vitamin D deficiency and cardiometabolic risks: a juxtaposition of Arab adolescents and adults. PLoS One. 2015;10(7):e0131315.

[69] Jordan Department of Statistics. Population and Housing Census 2015. Amman: DOS; 2016.

[70] Cauley JA, Cawthon PM, Peters KE, Cummings SR, Ensrud KE, Bauer DC, et al. Risk factors for hip fracture in older men: the Osteoporotic Fractures in Men Study (MrOS). J Bone Miner Res. 2016;31(10):1810-9.

[71] Leslie WD, Aubry-Rozier B, Lamy O, Hans D; Manitoba Bone Density Program. TBS (trabecular bone score) and diabetes-related fracture risk. J Clin Endocrinol Metab. 2013;98(2):602-9.

[72] Kahn SE, Haffner SM, Heise MA, Herman WH, Holman RR, Jones NP, et al. Glycemic durability of rosiglitazone, metformin, or glyburide monotherapy. N Engl J Med. 2006;355(23):2427-43.

[73] Leslie WD, Morin SN, Lix LM, Majumdar SR. Does diabetes modify the effect of FRAX risk factors for predicting major osteoporotic and hip fracture? Osteoporos Int. 2014;25(12):2817-24.

[74] Schwartz AV, Sellmeyer DE. Diabetes, fracture, and bone fragility. Curr Osteoporos Rep. 2007;5(3):105-11.

[75] El Hajj Fuleihan G, Baddoura R, Awada H, Okais J, Rizkallah M, Yamout K. The Middle East and Africa Regional Audit: Epidemiology, costs, and burden of osteoporosis in 2011. Arch Osteoporos. 2012;7:167-77.

[76] Kanis JA, Harvey NC, Johansson H, Liu E, Vandenput L, Lorentzon M, et al. A decade of FRAX: how has it changed the management of osteoporosis? Aging Clin Exp Res. 2020;32(2):187-96.

[77] Ferrari S, Reginster JY, Brandi ML, Kanis JA, Devogelaer JP, Kaufman JM, et al. Unmet needs and current and future approaches for osteoporotic patients at high risk of hip fracture. Arch Osteoporos. 2016;11(1):37.

[78] Hygum K, Starup-Linde J, Langdahl BL. Diabetes and bone. Osteoporos Sarcopenia. 2019;5(2):29-37.

[79] Mokdad AH, Forouzanfar MH, Daoud F, El Bcheraoui C, Moradi-Lakeh M, Khalil I, et al. Health in times of uncertainty in the eastern Mediterranean region, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet Glob Health. 2016;4(10):e704-13.

[80] Saad RK, Ghanem NE, Baddoura R, Awada H, Okais J, Rizkallah M, et al. An updated overview of the FRAX-based intervention thresholds for osteoporosis in five Middle Eastern countries: a consensus statement. J Clin Densitom. 2023;26(3):101386.

[81] 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):427-44.

[82] Yamaguchi T, Sugimoto T. Bone metabolism and fracture risk in type 2 diabetes mellitus. Endocr J. 2011;58(8):613-24.

[83] Abdulameer SA, Sahib MN, Sulaiman SAS. The prevalence of osteopenia and osteoporosis among Malaysian type 2 diabetic patients using quantitative ultrasound densitometer. Open Rheumatol J. 2018;12:50-64.

[84] Al-Daghri NM, Yakout S, Al-Shehri E, Al-Fawaz H, Aljohani N, Al-Saleh Y. Inflammatory and bone turnover markers in relation to PTH and vitamin D status among Saudi postmenopausal women with and without osteoporosis. Int J Clin Exp Med. 2014;7(10):3528-35.

[85] Chakhtoura M, Cauley JA, Chamoun N, El-Hajj Fuleihan G. Worldwide fracture prediction. J Clin Densitom. 2017;20(3):397-424.

[86] Crandall CJ, Larson J, Wright NC, Laddu-Patel D, Stefanick ML, Kaunitz AM, et al. Serial Bone Density Measurement and Incident Fracture Risk Discrimination in Postmenopausal Women. JAMA Intern Med. 2020;180(9):1232-40.

[87] Anagnostis P, Paschou SA, Goulis DG. Weighing the Evidence: The Impact of Obesity on Bone Health. In: Obesity and Bone. [Internet]. Cham: Springer International Publishing; 2020 [cited 2024 Oct 23]. p. 1-30.

[88] Nielsen BR, Abdallah BM, Harsløf T, Langdahl BL. The relationship between bone and fat tissue in children and adolescents: a systematic review. Calcif Tissue Int. 2021;108(4):423-36.