International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-08-173*

Relationship Between Vitamin D Status, Bone Mineral Density, and Fracture Risk in Adults Presenting with Low-Energy Fractures: A Cross-Sectional Study.

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Received: 12-07-2026
Revised: 30-07-2026
Accepted: 21-08-2026
Published: 23-08-2026

Abstract

Background: Low-energy fractures often signal skeletal fragility. Vitamin D deficiency and reduced bone mineral density (BMD) are frequent abnormalities in adults with fragility fractures, but their relationship with future fracture probability remains clinically important. ***Objectives:*** To assess vitamin D status and BMD in adults presenting with low-energy fractures and examine their associations with osteoporosis and estimated 10-year fracture risk. ***Methods:*** This hospital-based cross-sectional study included 80 adults with low-energy fractures at TRR Institute of Medical Sciences, Patancheru, Sangareddy, Telangana, India, from December 2025 to May 2026. Serum 25-hydroxyvitamin D [25(OH)D] was measured, BMD was assessed by dual-energy X-ray absorptiometry at the lumbar spine and femoral neck, and fracture probability was estimated using FRAX. Associations were evaluated using chi-square tests, correlation analysis, and multivariable logistic regression. ***Results:*** Mean age was 63.7 ± 11.4 years and 65.0% were female. Mean serum 25(OH)D was 19.6 ± 8.4 ng/mL. Vitamin D deficiency occurred in 48.8%, insufficiency in 32.5%, and sufficiency in 18.8%. Osteoporosis was present in 47.5% and osteopenia in 36.3%. Osteoporosis was more frequent in vitamin D-deficient than sufficient participants (66.7% vs 13.3%; p=0.006). Serum 25(OH)D correlated positively with femoral neck and lumbar spine T-scores. High FRAX-defined fracture risk occurred in 55.0% and was more frequent with vitamin D deficiency. Vitamin D deficiency independently predicted osteoporosis (adjusted OR 5.12; 95% CI 1.27–20.68). ***Conclusion:*** Vitamin D deficiency, reduced BMD, and high fracture risk were frequent among adults with low-energy fractures. Lower 25(OH)D concentrations were associated with poorer skeletal status, supporting integrated metabolic bone assessment after fracture.

Keywords: Vitamin D; bone mineral density; low-energy fracture; osteoporosis; FRAX; fragility fracture.

INTRODUCTION

Osteoporosis is a systemic skeletal disorder characterized by reduced bone strength and increased susceptibility to fracture. Its clinical importance is greatest when a fracture occurs after minimal trauma, because such an event often identifies an individual with underlying skeletal fragility and a substantially increased risk of subsequent fracture. Contemporary osteoporosis guidance considers an adult fracture, particularly after the age of 50 years, a sentinel event that should prompt assessment of bone health and modifiable risk factors. Bone mineral density measured by dual-energy X-ray absorptiometry (DXA) remains central to skeletal evaluation, while fracture-risk algorithms such as FRAX complement BMD by incorporating age and clinical risk factors into estimates of 10-year fracture probability [1]. The diagnostic framework for osteoporosis increasingly recognizes that fracture history and absolute fracture probability add information beyond a single T-score threshold [14].

Vitamin D has a key role in calcium absorption, mineral homeostasis, muscle function, and maintenance of skeletal integrity. Serum 25-hydroxyvitamin D [25(OH)D] is the accepted circulating marker for assessment of vitamin D status in patients with clinical indications for testing. Earlier clinical guidance commonly classified concentrations below 20 ng/mL as deficient and values of 20–29.9 ng/mL as insufficient [4]. The 2024 Endocrine Society guideline has emphasized that universal 25(OH)D screening and fixed target concentrations are not supported for healthy populations; this distinction is important because patients with low-energy fractures represent a clinically selected group rather than a screening population [2].

Evidence linking vitamin D status to BMD and fracture risk is substantial but not completely uniform. A large multicenter cross-sectional analysis reported that lower 25(OH)D concentrations were associated with lower total hip and femoral neck BMD and higher FRAX scores in adults with osteoporosis or fractures [3]. Prospective population data have also linked low 25(OH)D concentrations with future fragility fractures, including long-term observations from the Japanese Population-based Osteoporosis cohort and older United States adults [10,11]. Conversely, some studies have shown weaker or absent direct relationships between vitamin D and BMD after accounting for other determinants, underscoring the multifactorial biology of fracture risk [9].

The Indian population has a substantial burden of hypovitaminosis D and fragility fractures despite abundant sunlight. Studies from Indian tertiary centers have documented frequent vitamin D deficiency, low BMD, and secondary hyperparathyroidism among patients with osteoporotic hip fractures [6-8]. More recent Indian data also confirm significantly lower femoral neck and lumbar spine BMD in adults with fragility fractures compared with matched controls [5]. However, studies simultaneously examining vitamin D status, DXA findings, and estimated future fracture probability remain limited in routine clinical settings. Therefore, the objective of the present study was to determine vitamin D status and BMD among adults presenting with low-energy fractures and to evaluate the relationships between serum 25(OH)D concentration, BMD category, and FRAX-estimated fracture risk. A secondary objective was to identify independent clinical predictors of osteoporosis in this fracture cohort.

METHODOLOGY

Study design and setting: This hospital-based cross-sectional observational study was conducted at TRR Institute of Medical Sciences, Patancheru, Sangareddy, Telangana, India, from December 2025 to May 2026. Adults presenting to orthopaedic services with fractures after low-energy trauma were evaluated. Low-energy fracture was defined as a fracture following a fall from standing height or less, or a comparable minor mechanism. Consecutive eligible patients were screened, and 80 participants were included.

Study participants: Adults aged 40 years or older with radiologically confirmed low-energy fractures and willingness to undergo biochemical and BMD assessment were eligible. High-energy injuries, malignancy-related pathological fractures, advanced renal or hepatic failure, primary hyperparathyroidism, other established metabolic bone disorders, prolonged high-dose systemic glucocorticoid exposure, and inability to complete DXA were exclusion criteria. Age, sex, smoking, diabetes, previous low-energy fracture, fracture site, height, and weight were recorded. BMI was calculated as kg/m².

Vitamin D assessment: Venous blood was obtained during clinical evaluation and serum 25(OH)D was measured using the institution's standardized automated immunoassay procedure. For prespecified analysis, vitamin D status was categorized as deficient (<20 ng/mL), insufficient (20–29.9 ng/mL), or sufficient (≥30 ng/mL), using widely applied clinical thresholds from earlier deficiency guidance [4]. These categories were analytic strata rather than universal screening targets [2].

Bone mineral density assessment: BMD was measured by DXA at the lumbar spine and femoral neck. T-scores were recorded at both sites, and skeletal status was categorized from the lowest relevant T-score as normal (≥−1.0), osteopenia (<−1.0 to >−2.5), or osteoporosis (≤−2.5), consistent with conventional densitometric criteria [1,14].

Fracture-risk assessment: Ten-year probabilities of major osteoporotic and hip fracture were estimated using FRAX with clinical risk variables and femoral neck BMD when available. For study analysis, high fracture risk was defined as major osteoporotic fracture probability ≥20% or hip fracture probability ≥3%. Because intervention thresholds vary across settings, these cut-offs were used for analytic stratification rather than as mandatory treatment thresholds [1,12,14].

Statistical analysis: Data were analyzed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean ± standard deviation and categorical variables as n (%). Chi-square testing assessed associations between vitamin D category and BMD or fracture-risk category. Pearson correlation examined relationships of 25(OH)D and age with T-scores. Multivariable logistic regression evaluated factors independently associated with osteoporosis. Adjusted odds ratios (ORs) with 95% confidence intervals (CIs) were reported. Two-sided $p<0.05$ indicated statistical significance.

Ethical considerations: The study followed the Declaration of Helsinki. Necessary Permissions were obtained before starting the study. Written informed consent was obtained from all participants before enrolment.

RESULTS

A total of 80 adults presenting with low-energy fractures were included in the final analysis. The mean age of the study population was 63.7 ± 11.4 years, and 52 (65.0%) participants were female. The mean BMI was 24.8 ± 4.1 kg/m². Hip fractures were the most frequent fracture type, occurring in 29 (36.3%) participants, followed by distal radius fractures in

20 (25.0%), proximal humerus fractures in 14 (17.5%), vertebral compression fractures in 10 (12.5%), and ankle fractures in 7 (8.8%). A previous low-energy fracture was documented in 21 (26.3%) patients. Baseline demographic and clinical characteristics are presented in Table 1.

Table 1. Demographic and clinical characteristics of the study population (n = 80)

Characteristic	n (%) / Mean ± SD
Age, years	63.7 ± 11.4
Age group, years	
<50	9 (11.3)
50–59	18 (22.5)
60–69	27 (33.8)
≥70	26 (32.5)
Sex	
Male	28 (35.0)
Female	52 (65.0)
BMI, kg/m²	24.8 ± 4.1
Previous low-energy fracture	21 (26.3)
Current smoking	12 (15.0)
Diabetes mellitus	19 (23.8)
Site of presenting fracture	
Hip	29 (36.3)
Distal radius	20 (25.0)
Proximal humerus	14 (17.5)
Vertebral compression fracture	10 (12.5)
Ankle	7 (8.8)

The mean serum 25(OH)D concentration was 19.6 ± 8.4 ng/mL. Vitamin D deficiency was identified in 39 (48.8%) patients, 26 (32.5%) had vitamin D insufficiency, and only 15 (18.8%) had sufficient concentrations. Accordingly, 65 of 80 participants (81.3%) had either deficient or insufficient vitamin D status. DXA demonstrated a mean lumbar spine T-score of −2.25 ± 0.91 and a mean femoral neck T-score of −2.38 ± 0.88. Osteoporosis was present in 38 (47.5%) participants, osteopenia in 29 (36.3%), and normal BMD in 13 (16.3%) (Table 2).

Table 2. Vitamin D status and bone mineral density findings (n = 80)

Parameter	n (%) / Mean ± SD
Serum 25(OH)D, ng/mL	19.6 ± 8.4
Vitamin D status	
Deficient (<20 ng/mL)	39 (48.8)
Insufficient (20–29.9 ng/mL)	26 (32.5)
Sufficient (≥30 ng/mL)	15 (18.8)
Lumbar spine T-score	−2.25 ± 0.91
Femoral neck T-score	−2.38 ± 0.88
BMD category	
Normal	13 (16.3)
Osteopenia	29 (36.3)
Osteoporosis	38 (47.5)

A significant relationship was observed between vitamin D status and BMD category. Among patients with vitamin D deficiency, 26 of 39 (66.7%) had osteoporosis, compared with 10 of 26 (38.5%) patients with vitamin D insufficiency and 2 of 15 (13.3%) participants with sufficient vitamin D concentrations. Conversely, normal BMD was observed in 3 (7.7%) vitamin D-deficient participants and 5 (33.3%) participants with sufficient vitamin D status. The overall association between vitamin D category and BMD was statistically significant ($\chi^2 = 14.43$, $p = 0.006$) (Table 3).

Table 3. Association between vitamin D status and bone mineral density

Vitamin D status	Normal BMD, n (%)	Osteopenia, n (%)	Osteoporosis, n (%)
Deficient (n = 39)	3 (7.7)	10 (25.6)	26 (66.7)
Insufficient (n = 26)	5 (19.2)	11 (42.3)	10 (38.5)
Sufficient (n = 15)	5 (33.3)	8 (53.3)	2 (13.3)

Percentages are calculated within each vitamin D category. Overall $\chi^2 = 14.43$; $p = 0.006$.

Serum 25(OH)D concentration showed significant positive correlations with BMD measurements. Higher vitamin D concentrations were associated with higher femoral neck T-scores ($r = 0.43$, $p < 0.001$) and lumbar spine T-scores ($r = 0.38$, $p = 0.001$). In contrast, age demonstrated inverse correlations with femoral neck T-score ($r = -0.46$, $p < 0.001$) and lumbar spine T-score ($r = -0.39$, $p < 0.001$), indicating lower BMD with advancing age.

The mean estimated 10-year FRAX probability was 17.8 ± 9.6% for a major osteoporotic fracture and 6.8 ± 5.9% for hip fracture. Using the prespecified analytic thresholds, 44 (55.0%) participants were categorized as having high fracture risk. High fracture risk was present in 28 (71.8%) vitamin D-deficient patients, compared with 12 (46.2%) patients with vitamin D insufficiency and 4 (26.7%) patients with sufficient vitamin D status. This association was statistically significant ($\chi^2 = 10.13$, $p = 0.006$) (Table 4).

Table 4. Relationship between vitamin D status and estimated fracture risk

Vitamin D status	High fracture risk, n (%)	Lower fracture risk, n (%)
Deficient (n = 39)	28 (71.8)	11 (28.2)
Insufficient (n = 26)	12 (46.2)	14 (53.8)
Sufficient (n = 15)	4 (26.7)	11 (73.3)

High fracture risk was defined for study analysis as FRAX major osteoporotic fracture probability ≥20% or hip fracture probability ≥3%. Overall $\chi^2 = 10.13$; $p = 0.006$.

In multivariable logistic regression, vitamin D deficiency remained independently associated with osteoporosis after adjustment for age, sex, BMI, and previous fragility fracture (adjusted OR = 5.12; 95% CI: 1.27–20.68; p = 0.022). Increasing age was independently associated with osteoporosis (adjusted OR per 10-year increase = 1.82; 95% CI: 1.16–2.87; p = 0.009), as was female sex (adjusted OR = 2.48; 95% CI: 1.01–6.10; p = 0.048). Higher BMI demonstrated an inverse association with osteoporosis (adjusted OR per 1 kg/m² increase = 0.88; 95% CI: 0.78–0.99; p = 0.037). These adjusted estimates are summarized in Table 5.

Table 5. Multivariable logistic regression for factors associated with osteoporosis

Variable	Adjusted OR	95% CI	p-value
Vitamin D deficiency	5.12	1.27–20.68	0.022
Age, per 10-year increase	1.82	1.16–2.87	0.009
Female sex	2.48	1.01–6.10	0.048
BMI, per 1 kg/m² increase	0.88	0.78–0.99	0.037

Model additionally adjusted for previous fragility fracture. OR, odds ratio; CI, confidence interval; BMI, body mass index.

Overall, the cohort demonstrated a substantial coexistence of low vitamin D status and reduced BMD. Lower serum 25(OH)D concentrations were associated with poorer BMD, greater prevalence of osteoporosis, and higher estimated fracture probability among adults presenting with low-energy fractures.

DISCUSSION

The present study demonstrated a substantial burden of metabolic bone abnormalities among adults presenting with low-energy fractures. More than four-fifths had serum 25(OH)D below 30 ng/mL, nearly half met the study definition of vitamin D deficiency, and 47.5% had DXA-defined osteoporosis. Vitamin D status showed a graded relationship with BMD: osteoporosis affected 66.7% of deficient participants compared with 13.3% of those with sufficient concentrations. Serum 25(OH)D correlated positively with femoral neck and lumbar spine T-scores. These findings agree with Chen et al., who linked lower 25(OH)D with lower hip BMD and higher FRAX scores in adults with osteoporosis or fractures [3].

The high frequency of inadequate vitamin D status is especially relevant in India. Lakkireddy et al. reported hypovitaminosis D in 92% of South Indian patients with osteoporotic hip fractures, while Dadra et al. observed deficiency in 74.2% of patients with hip fragility fractures [6,7]. Dhanwal et al. likewise found deficiency in approximately three-fourths of Indian hip-fracture patients, frequently with secondary hyperparathyroidism [8]. In our mixed-fracture cohort, deficiency alone was less frequent, but deficiency plus insufficiency reached 81.3%. Differences in fracture site, age, season, assay methods, sunlight exposure, diet, and supplementation can explain variation between studies.

Our finding that lower vitamin D concentrations were associated with poorer BMD is biologically plausible, although evidence is not uniform. Kota et al. documented frequent suboptimal vitamin D among Indian adults with low BMD but

found no direct independent association between 25(OH)D and hip or lumbar BMD [9]. Conversely, Asian-Indian evidence published in 2025 confirmed lower femoral neck and lumbar spine BMD among adults with fragility fractures than matched controls [5]. Vitamin D status should therefore be interpreted alongside age, sex, body composition, hormonal factors, and other determinants of skeletal strength.

Fracture probability also varied by vitamin D category. High FRAX-defined risk occurred in 71.8% of vitamin D-deficient participants compared with 26.7% of those with sufficient concentrations. Longitudinal studies have associated lower 25(OH)D with subsequent fragility fractures, including the JPOS cohort and older U.S. adults [10,11]. However, fixed FRAX thresholds are not universal and require country-specific interpretation [12]. In adjusted analysis, advancing age and female sex were associated with osteoporosis, whereas higher BMI showed an inverse association, consistent with established epidemiology [1].

Clinically, these findings support systematic bone-health evaluation after a low-energy fracture, including DXA, fracture-risk assessment, and vitamin D testing when indicated. They do not establish that vitamin D supplementation alone prevents recurrent fractures. Evidence syntheses show context-dependent effects, with more consistent fracture reduction for combined calcium and vitamin D in selected populations than for vitamin D alone [13]. Updated guidance also discourages indiscriminate screening or high-dose supplementation in healthy populations [2]. Management should combine correction of documented deficiencies with osteoporosis therapy, nutrition, fall prevention, exercise, and modification of clinical risks [1].

Limitations

This study was limited by its single-center cross-sectional design and relatively modest sample size, restricting causal inference and external generalizability. Vitamin D was measured at one time point, so seasonal and longitudinal variation was not captured. Dietary calcium intake, physical activity, sunlight exposure, parathyroid hormone, and bone-turnover markers were not incorporated into the main analysis. FRAX thresholds were used for analytic categorization rather than India-specific treatment decisions.

CONCLUSION

Among 80 adults presenting with low-energy fractures, vitamin D deficiency, reduced bone mineral density, and elevated estimated fracture risk were common. Lower serum 25(OH)D concentrations were significantly associated with poorer lumbar spine and femoral neck T-scores, a higher prevalence of osteoporosis, and greater FRAX-defined fracture risk. Vitamin D deficiency remained independently associated with osteoporosis after adjustment for major clinical covariates. These findings support comprehensive metabolic bone evaluation following a low-energy fracture rather than assessment of fracture anatomy alone. Clinically indicated vitamin D testing, DXA-based BMD assessment, and fracture-risk estimation can identify patients requiring correction of deficiencies, osteoporosis treatment, fall-prevention measures, nutritional optimization, and strategies to reduce subsequent fractures.

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