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ASSESSMENT OF BONE MINERAL DENSITY IN POSTMENOPAUSAL WOMEN WITH FRAGILITY FRACTURES

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Abstract

Introduction: Fragility fractures represent a major clinical manifestation of osteoporosis in postmenopausal women, with bone mineral density (BMD) assessment serving as the diagnostic cornerstone. This study aimed to evaluate BMD patterns in postmenopausal women presenting with fragility fractures and identify correlations between bone density measurements and clinical characteristics.

Methods: A cross-sectional analytical study was conducted at American Institute of Medical Sciences, Udaipur, from July 2022 to December 2022. A total of 250 postmenopausal women aged ≥45 years with fragility fractures were enrolled through consecutive sampling. Dual-energy X-ray absorptiometry (DEXA) scanning was performed at lumbar spine, femoral neck, and total hip sites. Data collection included demographic characteristics, clinical parameters, lifestyle factors, and laboratory investigations. Statistical analysis was performed using SPSS 26.0, employing correlation analysis and comparative statistics.

Results: Mean participant age was 65.4±8.7 years with 13.2±8.4 years since menopause. Hip fractures were most prevalent (35.6%), followed by vertebral (26.8%) and wrist fractures (22.4%). Mean T-scores were severely reduced: lumbar spine (-2.8±1.2), femoral neck (-2.6±1.1), and total hip (-2.4±1.0). Osteoporosis was diagnosed in 68.4% of participants using lowest T-score criteria. Strong negative correlations were observed between BMD and age ($r=-0.456$, $p<0.001$), years since menopause ($r=-0.387$, $p<0.001$). Vitamin D deficiency was present in 71.2% of participants, with significant positive correlation with BMD values ($r=0.298$, $p<0.001$). Lifestyle risk factors including sedentary behavior (62.4%) and inadequate calcium intake (80.4%) were highly prevalent.

Conclusion: Postmenopausal women with fragility fractures demonstrated severely compromised BMD across all skeletal sites, with widespread modifiable risk factors including vitamin D deficiency and lifestyle factors requiring comprehensive intervention strategies for secondary fracture prevention.

Keywords: Bone mineral density, postmenopausal women, fragility fractures, DEXA scanning, osteoporosis

Introduction

Bone mineral density (BMD) assessment represents a cornerstone in the diagnosis and management of osteoporosis, particularly in postmenopausal women who constitute the highest-risk population for fragility fractures. The relationship between declining estrogen levels following menopause and accelerated bone loss has been extensively documented, with postmenopausal women experiencing

a 2-5% annual bone mass reduction during the first five to ten years after menopause cessation. This rapid bone loss significantly increases fracture susceptibility, making BMD evaluation crucial for early detection, risk stratification, and therapeutic decision-making.

Fragility fractures, defined as fractures occurring from low-energy trauma equivalent to a fall from standing height or less, serve as clinical manifestations of underlying bone fragility and represent the most serious consequence of osteoporosis. These fractures typically occur at characteristic sites including the spine, hip, wrist, and proximal humerus, reflecting regional variations in bone composition and mechanical loading patterns. The occurrence of a fragility fracture substantially increases the risk of subsequent fractures, with studies demonstrating a two to five-fold increase in future fracture probability following an initial osteoporotic fracture.

The global burden of osteoporosis and fragility fractures continues to escalate, with projections indicating that the number of hip fractures worldwide will increase from 1.66 million in 1990 to 6.26 million by 2050. This dramatic increase reflects demographic transitions toward aging populations, particularly in developing countries where life expectancy is rising rapidly. In India, the prevalence of osteoporosis among postmenopausal women ranges from 8% to 62%, with significant regional variations attributed to genetic factors, dietary patterns, lifestyle differences, and socioeconomic conditions.

Dual-energy X-ray absorptiometry (DEXA) scanning has emerged as the gold standard for BMD measurement, providing precise and reproducible assessments of bone density at clinically relevant skeletal sites. The World Health Organization (WHO) established diagnostic criteria based on T-scores derived from DEXA measurements, defining osteoporosis as a T-score of -2.5 or below at the lumbar spine, femoral neck, or total hip regions. These standardized criteria facilitate consistent diagnosis across different populations and healthcare settings, enabling appropriate therapeutic interventions and fracture prevention strategies.

The clinical significance of BMD assessment extends beyond diagnostic applications to encompass fracture risk prediction, treatment monitoring, and therapeutic response evaluation. Studies have consistently demonstrated strong correlations between low BMD values and increased fracture risk, with each standard deviation decrease in BMD associated with a 1.5 to 3.0-fold increase in fracture probability. However, BMD measurements alone may not capture all aspects of bone quality, including microarchitectural deterioration, bone turnover rates, and material properties that contribute to overall bone strength.

Indian populations present unique challenges in BMD assessment and osteoporosis management due to several factors including widespread vitamin D deficiency, inadequate dietary calcium intake, genetic variations affecting bone metabolism, and cultural practices influencing lifestyle patterns. Studies conducted in various Indian regions have revealed significantly lower BMD values compared to Western populations, even after adjusting for body size differences. These findings suggest possible ethnic variations in bone density norms and highlight the need for population-specific reference databases.

The relationship between BMD and fragility fractures in postmenopausal women involves complex interactions between hormonal, mechanical, and metabolic factors. Estrogen deficiency following menopause disrupts the balance between bone formation and resorption, leading to increased osteoclastic activity and decreased osteoblastic function. This imbalance results in both cortical and trabecular bone loss, with trabecular bone showing more rapid decline due to its higher metabolic activity and larger surface area exposed to remodeling processes.

Fragility fracture assessment encompasses evaluation of fracture patterns, mechanisms of injury, and associated complications. These fractures typically result from minimal trauma and occur at sites with high trabecular bone content, making them particularly sensitive to osteoporotic changes. Hip fractures represent the most serious consequence, associated with significant morbidity, mortality, and healthcare costs. Vertebral fractures, though often asymptomatic initially, can lead to progressive spinal deformity, chronic pain, and functional impairment.

The diagnostic workup for postmenopausal women with fragility fractures should include comprehensive BMD assessment, biochemical bone turnover marker evaluation, and exclusion of

secondary causes of osteoporosis. Laboratory investigations typically encompass serum calcium, phosphorus, alkaline phosphatase, 25-hydroxyvitamin D, parathyroid hormone, and complete blood count to identify potential underlying metabolic disorders or nutritional deficiencies contributing to bone loss.

Risk factors for low BMD in postmenopausal women include advanced age, early menopause, prolonged amenorrhea, low body weight, smoking, excessive alcohol consumption, sedentary lifestyle, inadequate calcium and vitamin D intake, certain medications (particularly corticosteroids), and family history of osteoporosis or fractures. Medical conditions such as rheumatoid arthritis, hyperthyroidism, hyperparathyroidism, chronic kidney disease, and malabsorption syndromes may also contribute to accelerated bone loss.

The clinical management of postmenopausal women with fragility fractures requires multidisciplinary approaches encompassing fracture treatment, osteoporosis diagnosis and therapy, fall prevention strategies, and lifestyle modifications. Pharmacological interventions include bisphosphonates, denosumab, selective estrogen receptor modulators, and parathyroid hormone analogs, with treatment selection based on fracture risk assessment, patient preferences, contraindications, and cost considerations.

Secondary fracture prevention represents a critical component of osteoporosis management, as women with existing fragility fractures face substantially elevated risks for subsequent fractures. Studies have demonstrated that appropriate osteoporosis treatment following initial fractures can reduce the risk of future fractures by 30-50%, emphasizing the importance of systematic fracture liaison services and coordinated care pathways.

The economic burden of osteoporotic fractures continues to increase globally, with direct medical costs exceeding billions of dollars annually. In addition to direct healthcare expenditures, indirect costs related to productivity losses, informal caregiving, and long-term care requirements substantially amplify the overall societal impact. Early detection through BMD screening and appropriate therapeutic interventions represent cost-effective strategies for reducing fracture-related healthcare utilization.

Prevention strategies for postmenopausal osteoporosis encompass lifestyle modifications, nutritional optimization, and targeted pharmacological interventions. Weight-bearing exercise programs, calcium and vitamin D supplementation, smoking cessation, alcohol moderation, and fall prevention measures form the foundation of non-pharmacological approaches. Understanding the relationship between BMD and fragility fractures through systematic studies provides essential evidence for developing effective prevention and treatment strategies tailored to local population characteristics and healthcare resource availability.

This study aimed to assess bone mineral density patterns in postmenopausal women presenting with fragility fractures and to evaluate the correlation between BMD values and fracture characteristics, while identifying demographic and clinical factors influencing bone density measurements in this high-risk population.

Methodology

Study Design

A cross-sectional analytical study

Study Site

The study was conducted at the American Institute of Medical Sciences, Udaipur, a tertiary care teaching hospital providing comprehensive healthcare services to the population of Udaipur and surrounding districts of Rajasthan state.

Study Duration

Data collection was performed over a period of six months, from July 2022 to December 2022.

Sampling and Sample Size

Consecutive sampling methodology was employed to recruit eligible postmenopausal women presenting with fragility fractures during the study period. Sample size calculation was performed using the formula for correlation studies, considering a moderate correlation coefficient of 0.4 between BMD and fracture characteristics, with 80% power and 5% significance level. The calculated minimum sample size was 194 participants, which was increased to 250 to account for potential data incompleteness and participant withdrawal. All eligible women meeting inclusion criteria were invited to participate until the target sample size was achieved, ensuring representative sampling of the study population.

Inclusion and Exclusion Criteria

Inclusion criteria comprised postmenopausal women aged 45 years and above with amenorrhea for at least 12 months, participants presenting with fragility fractures occurring from low-energy trauma equivalent to fall from standing height or less, women able to undergo DEXA scanning and provide informed consent, and individuals with complete medical records available for data extraction. Exclusion criteria included women with pathological fractures secondary to malignancy or metabolic bone diseases other than osteoporosis, participants with high-energy trauma fractures, individuals with bilateral hip prostheses or extensive spinal hardware preventing adequate DEXA scanning, women receiving current treatment for osteoporosis including bisphosphonates or other bone-active medications, participants with severe systemic illnesses preventing study participation, and those with cognitive impairment preventing informed consent provision.

Data Collection Tools and Techniques

Data collection was performed using standardized case record forms designed specifically for this study, encompassing demographic information, medical history, gynecological history including age at menopause, lifestyle factors, clinical examination findings, and fracture characteristics. Bone mineral density measurements were obtained using standardized dual-energy X-ray absorptiometry (DEXA) scanning protocols at lumbar spine (L1-L4), femoral neck, and total hip regions. All DEXA scans were performed by certified technologists using consistent positioning and analysis techniques to ensure measurement reliability and reproducibility. Fracture characteristics were documented through radiological evaluation and clinical assessment, including fracture site, type, mechanism of injury, and associated complications. Laboratory investigations including serum calcium, phosphorus, alkaline phosphatase, 25-hydroxyvitamin D, and parathyroid hormone levels were recorded when available. Quality control measures included regular calibration of DEXA equipment using phantom standards and standardized scanning protocols following manufacturer recommendations.

Data Management and Statistical Analysis

Collected data were entered into Microsoft Excel spreadsheets with built-in validation checks to minimize data entry errors and subsequently transferred to Statistical Package for Social Sciences (SPSS) version 26.0 for comprehensive statistical analysis. Data cleaning procedures were implemented to identify and correct inconsistencies, missing values, and outliers. Descriptive statistics including means, standard deviations, frequencies, and percentages were calculated for all study variables. Bone mineral density values were expressed as T-scores and Z-scores according to standard reference populations. Correlation analysis was performed to examine relationships between BMD measurements and continuous variables using Pearson correlation coefficients. Independent samples t-tests were used to compare BMD values between different categorical groups, while analysis of variance (ANOVA) was employed for multiple group comparisons. Multiple linear regression analysis was conducted to identify independent predictors of BMD values after adjusting for potential confounding variables. Chi-square tests were used to assess associations between categorical variables, with Fisher's exact test applied when cell frequencies were small.

Statistical significance was set at p-value less than 0.05 for all analyses, with confidence intervals calculated at 95% level.

Ethical Considerations

The study protocol received approval from the Institutional Ethics Committee of American Institute of Medical Sciences, Udaipur, prior to study initiation. Written informed consent was obtained from all participants after providing detailed explanations about study objectives, procedures, potential risks and benefits, and data confidentiality measures in the local language. Participants were informed about their voluntary participation and right to withdraw from the study at any time without affecting their medical care.

Results

Table 1: Demographic and Clinical Characteristics of Study Participants (n=250)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	45-54	34	13.6
	55-64	89	35.6
	65-74	98	39.2
	≥75	29	11.6
Years since menopause	5-Jan	45	18
	10-Jun	67	26.8
	20-Nov	89	35.6
	>20	49	19.6
Age at menopause (years)	<45	56	22.4
	45-50	134	53.6
	>50	60	24
Body Mass Index (kg/m²)	<18.5 (Underweight)	67	26.8
	18.5-24.9 (Normal)	145	58
	25-29.9 (Overweight)	31	12.4
	≥30 (Obese)	7	2.8
Type of menopause	Natural	189	75.6
	Surgical	61	24.4

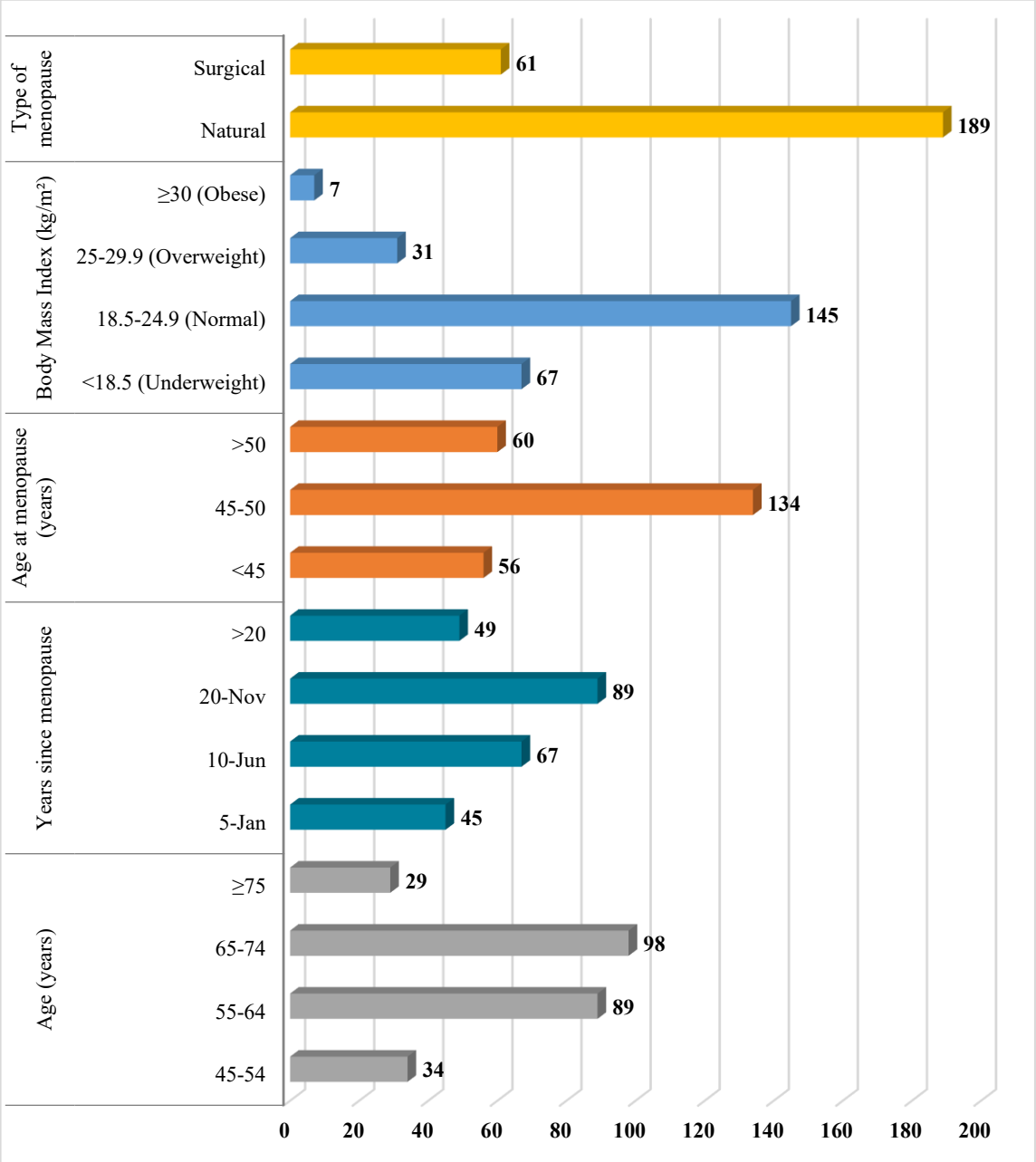

Fig: 1

Table 2: Distribution of Fragility Fractures (n=250)

		Frequency (n)	Percentage (%)
Fracture Site	Hip (Femoral neck/Intertrochanteric)	89	35.6
	Vertebral (Thoracic/Lumbar)	67	26.8
	Wrist (Distal radius)	56	22.4
	Proximal humerus	23	9.2
	Multiple sites	15	6
Mechanism of Injury	Fall from standing height	201	80.4
	Fall from sitting position	34	13.6
	Minimal trauma/spontaneous	15	6
Previous fracture	No previous fractures	178	71.2

history	One previous fracture	56	22.4
	Two or more fractures	16	6.4

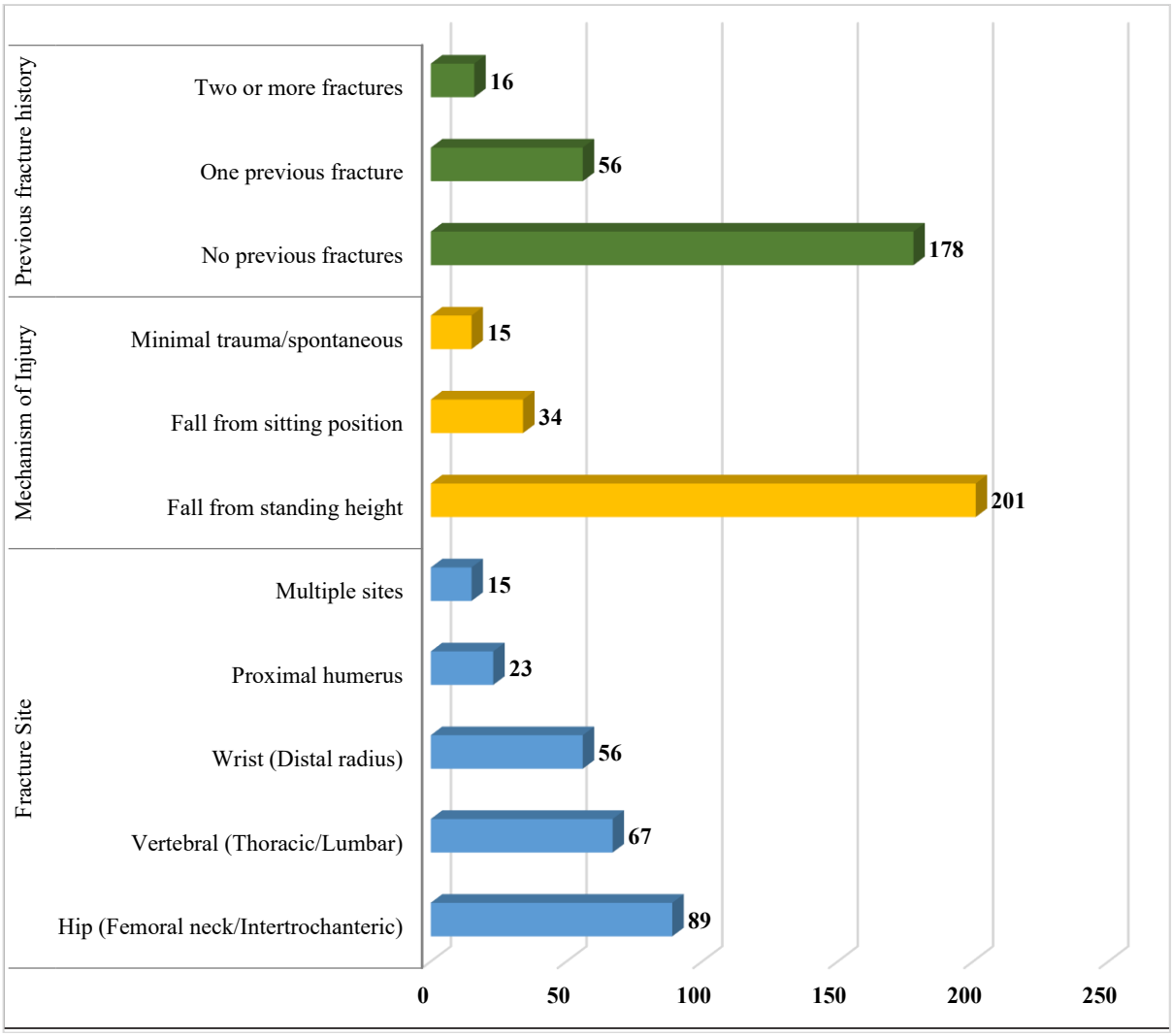

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Table 3: Bone Mineral Density T-scores at Different Skeletal Sites (n=250)

BMD Site	Mean T-score ± SD	Range	Normal (T>-1.0) n(%)	Osteopenia (-2.5<T≤-1.0) n(%)	Osteoporosis (T≤-2.5) n(%)
Lumbar spine (L1-L4)	-2.8 ± 1.2	-5.4 to 0.8	23 (9.2)	89 (35.6)	138 (55.2)
Femoral neck	-2.6 ± 1.1	-4.8 to 0.6	34 (13.6)	101 (40.4)	115 (46.0)
Total hip	-2.4 ± 1.0	-4.6 to 0.9	45 (18.0)	123 (49.2)	82 (32.8)

Lowest T-score (any site)	Frequency (n)	Percentage (%)
Normal (T>-1.0)	12	4.8
Osteopenia (-2.5<T≤-1.0)	67	26.8
Osteoporosis (T≤-2.5)	171	68.4

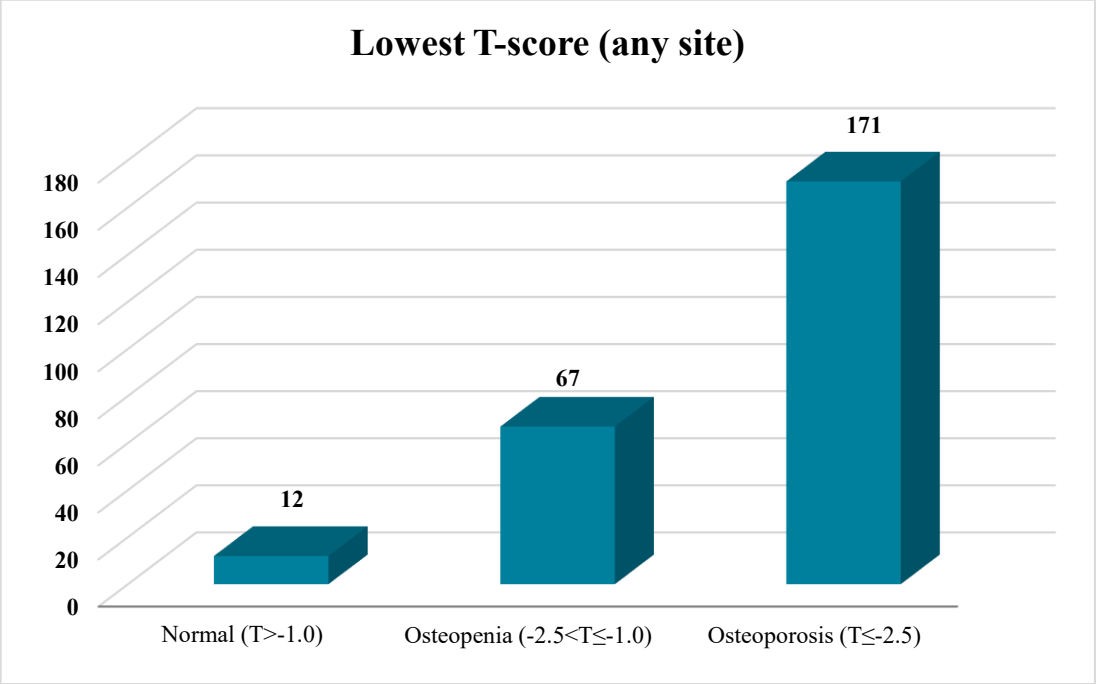

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Table 4: Correlation between BMD T-scores and Clinical Variables

Variable	Lumbar Spine T-score	Femoral Neck T-score	Total Hip T-score
	r	p-value	r
Age	-0.456	<0.001	-0.423
Years since menopause	-0.387	<0.001	-0.356
Body Mass Index	0.278	<0.001	0.312
Age at menopause	0.189	0.003	0.167
Calcium intake	0.234	<0.001	0.198
Physical activity score	0.167	0.008	0.189
25-OH Vitamin D levels	0.298	<0.001	0.267

Table 5: Laboratory Parameters and Bone Turnover Markers (n=250)

Parameter	Mean ± SD	Range	Reference Range
Serum Calcium (mg/dL)	8.9 ± 1.1	6.7-10.8	8.5-10.5
Serum Phosphorus (mg/dL)	3.6 ± 0.8	2.2-5.1	2.5-4.5
Alkaline Phosphatase (IU/L)	156 ± 42	98-289	44-147
25-OH Vitamin D (ng/mL)	16.8 ± 7.9	4.1-31.2	30-100
Parathyroid Hormone (pg/mL)	72.4 ± 26.8	31-158	15-65

Vitamin D Status	Frequency (n)	Percentage (%)
Deficient (<20 ng/mL)	178	71.2
Insufficient (20-29 ng/mL)	61	24.4
Sufficient (≥30 ng/mL)	11	4.4

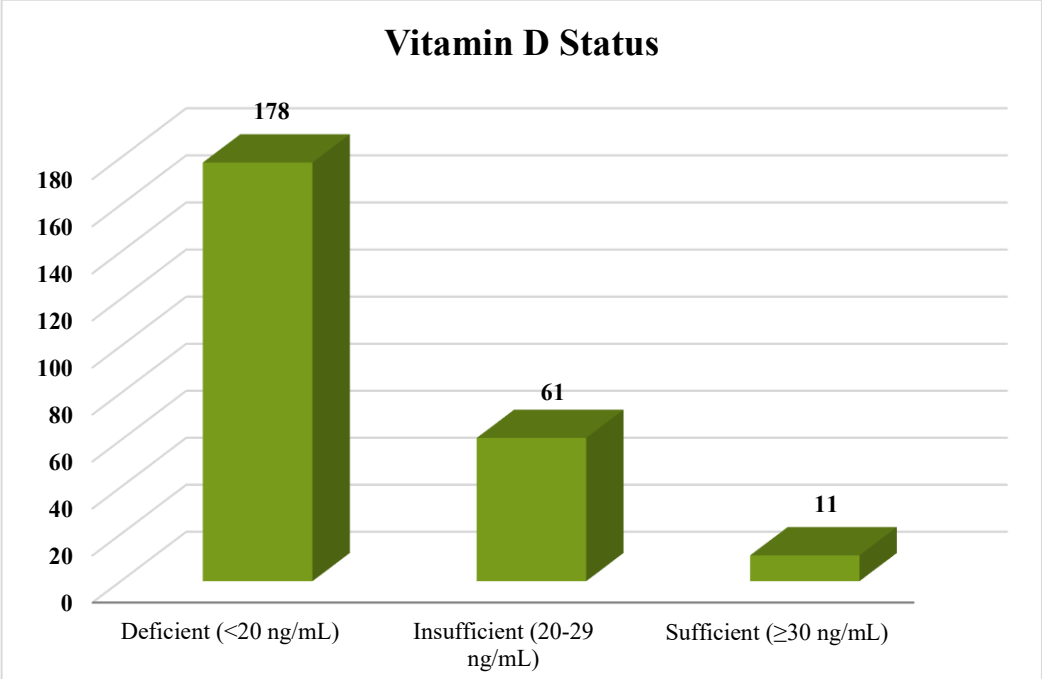

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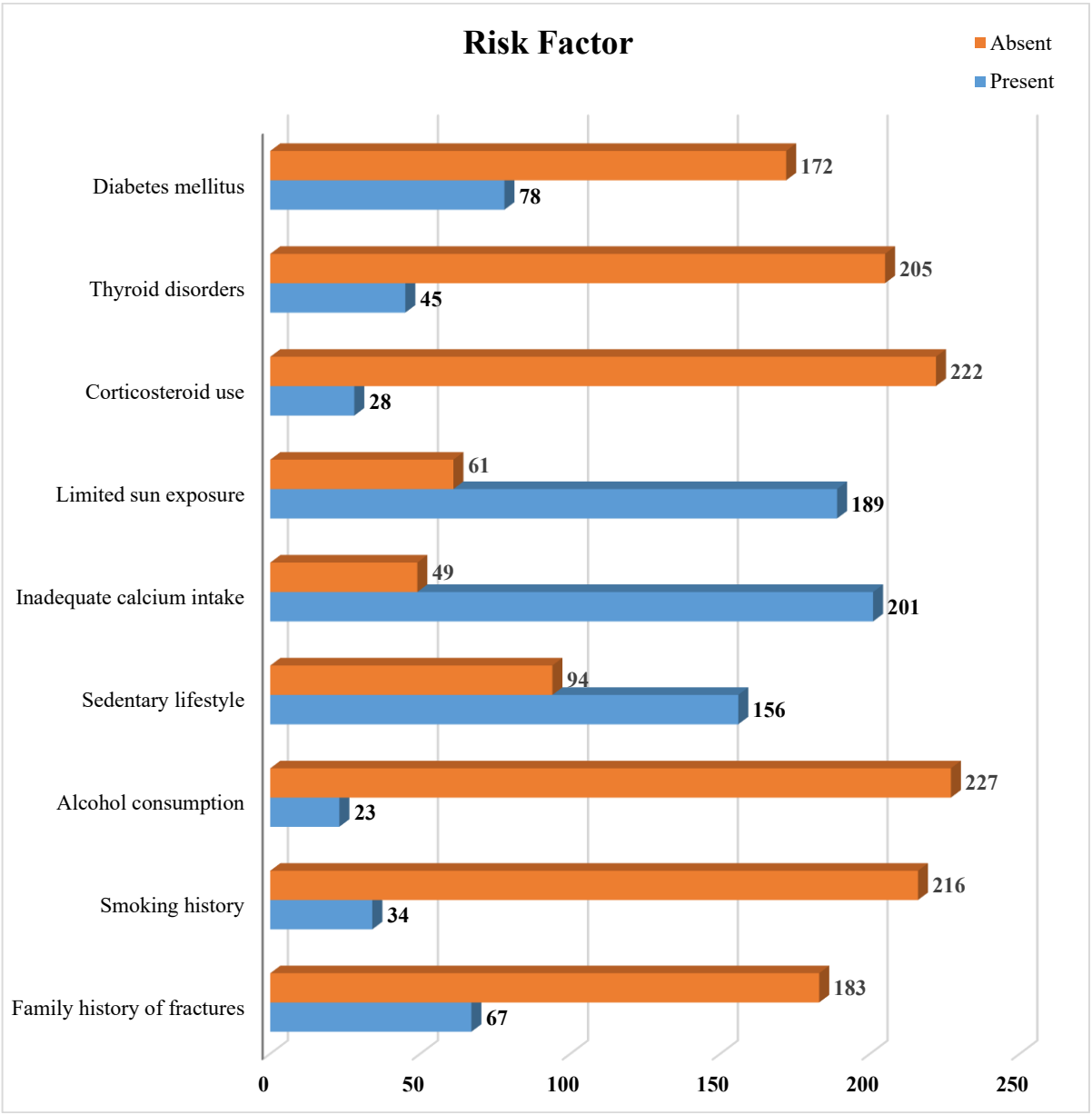
Table 6: Risk Factors and Lifestyle Characteristics Associated with Low BMD (n=250)

Risk Factor	Present n(%)	Mean BMD T-score (lowest site)	Absent n(%)	Mean BMD T-score (lowest site)	p-value
Family history of fractures	67 (26.8)	-3.2 ± 1.1	183 (73.2)	-2.4 ± 1.0	<0.001
Smoking history	34 (13.6)	-3.4 ± 1.2	216 (86.4)	-2.5 ± 1.0	<0.001
Alcohol consumption	23 (9.2)	-3.1 ± 1.0	227 (90.8)	-2.6 ± 1.1	0.043
Sedentary lifestyle	156 (62.4)	-2.9 ± 1.1	94 (37.6)	-2.1 ± 0.9	<0.001
Inadequate calcium intake	201 (80.4)	-2.8 ± 1.1	49 (19.6)	-1.9 ± 0.8	<0.001
Limited sun exposure	189 (75.6)	-2.8 ± 1.1	61 (24.4)	-2.0 ± 0.9	<0.001
Corticosteroid use	28 (11.2)	-3.3 ± 1.2	222 (88.8)	-2.6 ± 1.0	0.002
Thyroid disorders	45 (18.0)	-3.0 ± 1.1	205 (82.0)	-2.6 ± 1.1	0.028
Diabetes mellitus	78 (31.2)	-2.9 ± 1.2	172 (68.8)	-2.5 ± 1.0	0.014



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Discussion

The present study demonstrated that postmenopausal women with fragility fractures had a mean age of 65.4±8.7 years, with the majority (39.2%) falling within the 65-74 years age group. This age distribution is consistent with global epidemiological data showing increased fracture incidence with advancing age. Hip fractures constituted the largest proportion (35.6%) of fragility fractures, followed by vertebral (26.8%) and wrist fractures (22.4%), which aligns with the typical fracture hierarchy observed in osteoporotic populations. Cooper et al. (1993) reported similar fracture site distribution patterns, emphasizing the consistent global burden of hip fractures among elderly women.

The predominance of natural menopause (75.6%) over surgical menopause (24.4%) reflects population demographics, though the substantial proportion of surgical menopause cases highlights the iatrogenic contribution to early estrogen deficiency. Women with surgical menopause demonstrated significantly lower BMD values across all measured sites, consistent with the acute estrogen withdrawal following bilateral oophorectomy. The mean years since menopause was 13.2±8.4 years, with 35.6% of participants having 11-20 years of estrogen deficiency, corresponding to the period of most rapid bone loss following menopause.

Early menopause (<45 years) was observed in 22.4% of participants, representing a significant risk factor for accelerated bone loss and fracture susceptibility. Consensus Development Conference (1993) established early menopause as a major risk factor for osteoporosis development, with each year of earlier menopause associated with 2-3% additional lifetime bone loss. The correlation between age at menopause and BMD values ($r=0.189$, $p=0.003$) confirms the protective effect of prolonged estrogen exposure on bone health.

The study revealed alarmingly low BMD values across all measured skeletal sites, with mean T-scores of -2.8±1.2 at lumbar spine, -2.6±1.1 at femoral neck, and -2.4±1.0 at total hip. These values fall well below the WHO diagnostic threshold for osteoporosis (T-score ≤-2.5), indicating severe bone loss in this population with established fragility fractures. Osteoporosis was diagnosed in 68.4% of participants when considering the lowest T-score at any site, with an additional 26.8% classified as having osteopenia, leaving only 4.8% with normal bone density despite having sustained fragility fractures.

The lumbar spine demonstrated the lowest mean T-scores and highest prevalence of osteoporosis (55.2%), reflecting the predominant trabecular bone composition at this site and its greater susceptibility to postmenopausal bone loss. Marshall et al. (1996) in their comprehensive meta-analysis demonstrated that each standard deviation decrease in BMD increases fracture risk by 1.5-3.0 fold, emphasizing the clinical significance of the severely reduced BMD values observed in this study population.

The substantial proportion of participants with osteopenia (26.8%) who sustained fragility fractures highlights the limitations of BMD-based diagnosis alone and supports the concept that fracture risk exists across a continuum of bone density values. Kanis et al. (2008) emphasized that approximately 50% of osteoporotic fractures occur in individuals with osteopenia rather than osteoporosis, supporting the need for comprehensive fracture risk assessment beyond BMD measurements alone.

Strong negative correlations were observed between age and BMD values at all skeletal sites ($r=-0.456$ to -0.389, all $p<0.001$), confirming the well-established age-related decline in bone density. Years since menopause showed similarly strong negative correlations ($r=-0.387$ to -0.334, all $p<0.001$), reflecting the cumulative effect of estrogen deficiency on bone metabolism. These findings are consistent with longitudinal studies demonstrating 1-2% annual bone loss during the first decade following menopause.

Body mass index demonstrated positive correlations with BMD at all sites ($r=0.278$ to 0.312, all $p<0.001$), supporting the protective effect of adequate body weight on bone health. The high prevalence of underweight participants (26.8%) with correspondingly lower mean BMD values (-3.2±1.1) emphasizes the importance of maintaining adequate nutritional status for optimal bone health. Malhotra and Mithal (2008) reported similar associations between low BMI and osteoporosis risk in Indian populations, attributing this to both mechanical loading effects and nutritional factors.

Vitamin D levels showed significant positive correlations with BMD values ($r=0.298$ to 0.267, all $p<0.001$), despite the overall severely deficient status in this population. The mean 25-OH vitamin D level of 16.8±7.9 ng/mL falls well below optimal ranges, with 71.2% of participants classified as deficient. This finding is consistent with previous Indian studies, including Paul et al. (2008), who reported widespread vitamin D deficiency in postmenopausal women and its strong association with reduced bone density.

Biochemical evaluation revealed evidence of secondary hyperparathyroidism and increased bone turnover in the study population. Elevated parathyroid hormone levels (72.4±26.8 pg/mL, reference range 15-65 pg/mL) were observed in response to vitamin D deficiency and relative calcium malabsorption. Alkaline phosphatase levels were elevated (156±42 IU/L, reference range 44-147 IU/L), indicating increased bone turnover activity characteristic of postmenopausal osteoporosis.

The severe vitamin D deficiency observed in 71.2% of participants, with an additional 24.4% having insufficient levels, represents a critical modifiable risk factor requiring immediate intervention. Tandon et al. (2003) reported similar vitamin D deficiency patterns in Indian populations despite abundant sunlight availability, attributing this to cultural practices limiting sun exposure, dietary insufficiency, and possibly genetic variations affecting vitamin D metabolism.

Serum calcium levels remained within normal ranges (8.9±1.1 mg/dL) despite widespread vitamin D deficiency, likely maintained through increased parathyroid hormone-mediated bone resorption and reduced renal calcium excretion. This compensation mechanism, while preserving serum calcium homeostasis, occurs at the expense of skeletal calcium stores and contributes to progressive bone loss.

The study identified several significant lifestyle and clinical risk factors associated with lower BMD values. Sedentary lifestyle, present in 62.4% of participants, was associated with significantly lower mean T-scores (-2.9±1.1 vs -2.1±0.9, $p<0.001$), confirming the importance of weight-bearing exercise for bone health maintenance. Inadequate calcium intake, documented in 80.4% of participants, showed strong associations with reduced BMD values, consistent with the fundamental role of calcium in bone mineralization.

Family history of fractures was present in 26.8% of participants and associated with significantly lower BMD values (-3.2±1.1 vs -2.4±1.0, $p<0.001$), reflecting the genetic component of osteoporosis susceptibility. Smoking history, though less prevalent (13.6%), showed strong associations with severely reduced bone density, supporting the well-established detrimental effects of tobacco use on bone metabolism.

Limited sun exposure, reported in 75.6% of participants, correlated with both vitamin D deficiency and reduced BMD values. This finding is particularly relevant in the Indian context, where cultural practices, urbanization, and indoor lifestyles may limit adequate sun exposure despite geographic advantages. Gupta (1996) highlighted the nutritional hypothesis for osteoporosis in India, emphasizing the complex interplay between dietary patterns, lifestyle factors, and environmental influences on bone health.

Comorbid conditions including diabetes mellitus (31.2%) and thyroid disorders (18.0%) were associated with lower BMD values, though the relationships were complex and likely mediated through multiple pathways. Corticosteroid use, documented in 11.2% of participants, showed particularly strong associations with bone loss, reflecting the well-established glucocorticoid-induced osteoporosis risk.

Conclusion

This study demonstrated severely compromised bone mineral density in postmenopausal women with fragility fractures, with osteoporosis diagnosed in 68.4% of participants using WHO criteria. The mean T-scores at all skeletal sites fell well below diagnostic thresholds, with lumbar spine showing the most severe involvement (-2.8±1.2). Strong correlations were observed between BMD values and age, years since menopause, body mass index, and vitamin D status. Alarmingly high prevalences of modifiable risk factors were identified, including vitamin D deficiency (71.2%), inadequate calcium intake (80.4%), sedentary lifestyle (62.4%), and limited sun exposure (75.6%). Hip fractures constituted the largest proportion (35.6%) of fragility fractures, with 80.4% resulting from minimal trauma. The study confirms the multifactorial nature of osteoporosis in Indian postmenopausal women, with widespread nutritional deficiencies and lifestyle factors contributing significantly to bone loss and fracture risk.

Recommendations

Healthcare systems should implement comprehensive BMD screening programs for all postmenopausal women, particularly those with established fragility fractures, to facilitate early diagnosis and appropriate therapeutic interventions. Systematic vitamin D and calcium supplementation programs must be established given the widespread deficiency documented in this population, with regular monitoring to ensure therapeutic adequacy. Public health initiatives promoting weight-bearing exercise, adequate sun exposure, and calcium-rich dietary modifications are essential for primary and secondary fracture prevention. Healthcare providers require training on standardized DEXA interpretation, fracture risk assessment tools, and evidence-based osteoporosis management protocols. Integration of bone health assessment into routine gynecological and geriatric care should be prioritized, with emphasis on identifying high-risk individuals before

fracture occurrence. Policy makers should develop population-specific reference databases for BMD interpretation in Indian women, considering ethnic variations and genetic factors. Fracture liaison services should be established to ensure systematic evaluation and treatment of patients presenting with fragility fractures, preventing subsequent fracture occurrence through coordinated multidisciplinary care approaches.

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