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Research Article

REFRAMING OSTEOPOROSIS AS A "HUMAN" RATHER THAN "FEMALE" DISEASE: INSIGHTS FROM GENDER-COMPARATIVE BIOMARKER ANALYSIS

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ABSTRACT

Objectives: To compare bone turnover markers (BTM) (C-terminal telopeptide of type I collagen [CTX] and procollagen type I N-terminal propeptide [P1NP]), femoral neck T-scores, and biochemical parameters (calcium, vitamin D, and parathyroid hormone [PTH]) between males and females, and to evaluate their association with fracture history in an age-matched cohort.

Methods: This cross-sectional study included 100 age-matched adults (50 men and 50 women) from Mullana, Haryana. Serum calcium, vitamin D, PTH, CTX, and P1NP were measured using standardized assays. Bone mineral density (BMD) was assessed by dual-energy X-ray absorptiometry (DXA). Group comparisons were performed using Student's t-test or Mann–Whitney U test as appropriate. Correlations were assessed using Spearman's rank correlation coefficient.

Results: No statistically significant gender differences were observed in calcium, vitamin D, PTH, CTX, or P1NP levels (all $p \geq 0.05$). CTX and P1NP showed strong negative correlations with T-score ($r = -0.73$ and -0.68, $p < 0.001$), while calcium and vitamin D showed moderate positive correlations ($r = 0.55$ and 0.58, $p < 0.001$). CTX and P1NP demonstrated excellent diagnostic performance for osteoporosis. Fracture history was significantly associated with lower T-scores but not with individual biochemical markers.

Conclusion: Biochemical and densitometric profiles were broadly comparable between sexes. While BTMs reflect disease severity, DXA-derived BMD remains central for fracture risk stratification. Observed disparities in clinical outcomes between men and women likely reflect healthcare practice patterns rather than inherent biological differences. Longitudinal studies are needed to establish causality.

Keywords: Osteoporosis, Fracture, Gender bias, Bone turnover markers, T-score.

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INTRODUCTION

Bone turnover is a dynamic process. The bone continuously remodels itself, with osteoblasts and osteoclasts working together to form and resorb, respectively [1]. Balanced remodeling is essential for maintaining bone mass and the integrity of the skeleton. Any imbalance in this process may lead to increased bone formation (as in osteosclerosis) or resorption (as in osteoporosis or osteopenia) [2]. A decrease in bone mineral density (BMD) can lead to metabolic bone diseases, such as osteopenia and osteoporosis. Osteoporosis is characterized by compromised bone strength, predisposing patients to fractures [3]. The prevalence of osteoporosis worldwide is around 18.3%, being higher in females than in males [4].

Osteoporotic fractures impose a significant economic burden and are a major global public health concern. According to the International Osteoporosis Foundation (NOF), worldwide, 1 in 3 women above the age of 50 years and 1 in 5 men will experience osteoporotic fractures in their lifetime. Thus, proper diagnosis and early detection of osteoporosis are of utmost importance. Osteopenia, if detected early, can be halted and treated before it worsens. Various tools have been identified for the detection of osteopenia and osteoporosis. Dual-energy X-ray absorptiometry (DXA) is the gold standard for measuring BMD and detecting osteopenia and osteoporosis. Quantitative ultrasounds have proven to be low-cost and readily accessible. Vertebral bone quality scores based on magnetic resonance imaging are also reported to provide high sensitivity and moderate specificity for the detection of osteoporosis. Quantitative computed tomography is also a valuable tool for osteoporosis detection. It is even more sensitive than a DXA scan. Bone turnover markers (BTMs) are not standalone diagnostic tools for osteoporosis based purely on bone density – they lack specificity and cannot replace DXA [5].

Osteoporosis is often perceived as a condition that predominantly affects postmenopausal women. Men with osteoporosis form a significant but under-recognized group and are often not screened timely. Osteoporosis is commonly observed in elderly males. Analysis of global data involving over 450,000 male subjects revealed that approximately 11.7% of men are affected by osteoporosis. Regional disparities were evident, with prevalence peaking at 20.5% in areas, such as the Eastern Mediterranean and Western Asia, indicating a notable influence of geographic and demographic factors. A nationwide osteoporosis survey in Taiwan included 3734 men of whom 9.7% were affected [6]. Recent Indian studies reveal a significant but variable prevalence of osteoporosis among men, highlighting the need for increased awareness and targeted screening. A study conducted at a tertiary-care center in Mumbai, involving 524 men (mean age ~50±12 years), reported an overall prevalence of osteoporosis of 4.2%, with 29.9% exhibiting osteopenia. The prevalence increased modestly with age, reaching 6.5% in the 50–59 age group and 5.6% among those aged 70 and above [7]. In contrast, a smaller cross-sectional study of 200 healthy Indian men aged 50 years or older identified a higher prevalence of osteoporosis at 8.5% and osteopenia in 42% of the cohort [8]. Notably, a South Indian cohort of 252 men aged over 50 years with a mean age of 58 years reported a markedly higher osteoporosis and osteopenia at any one site, which were 20% (50/252) and 58%, respectively [9].

The male and female skeletons show many biological differences. Males attain higher peak bone mass than females. Females have a greater

Table 1 : Gender-wise comparison of biochemical parameters (mean±SD) with statistical significance

Parameters	Mean values±SD		p-value	Statistical tests performed	Significant/not significant
	Males n=50	Females n=50			
Calcium (mg/dL)	9.306±0.463	9.417±0.405	0.428	Mann–Whitney U test	Not significant
Vitamin D (ng/mL)	25.794±7.783	26.598±8.306	0.619	Students t-test	Not significant
PTH (pg/mL)	43.794±16.242	50.508±18.040	0.053	Students t-test	Not significant but borderline
CTX (ng/mL)	0.428±0.247	0.444±0.246	0.682	Mann–Whitney U test	Not significant
P1NP (ng/mL)	59.055±22.134	55.126±26.749	0.245	Mann–Whitney U test	Not significant

SD: Standard deviation, CTX: C-terminal telopeptide of type I collagen, P1NP: Procollagen type I N-terminal propeptide, PTH: Parathyroid hormone. None of the biomarkers show a statistically significant difference between males and females in this dataset (all p≥0.05), although PTH is close to the significance threshold

Table 2: Correlation between biochemical markers and their clinical interpretation

Markers n=100	r-value	p-value	Statistical tests performed	Interpretations
CTX versus P1NP	≈0.63	<0.001	Spearman's rank correlation coefficient	Strongly positive
Vitamin D versus CTX	≈−0.48	<0.001		Moderate negative
Vitamin D versus P1NP	≈−0.44	<0.001		Negative
Vitamin D versus calcium	≈0.33	Nearly 0.001		Moderate positive

CTX: C-terminal telopeptide of type I collagen, P1NP: Procollagen type I N-terminal propeptide

estrogen production, which causes early epiphyseal closure [10]. Estrogen has a great effect on bone health in females. It helps to preserve bone mass and also suppresses bone turnover. It also inhibits periosteal apposition and stimulates endocortical bone formation. Due to this, there is an increase in cortical thickness and periosteal diameter but a decrease in medullary diameter at the time of female puberty. Testosterone also plays an important role in skeletal development of females, but is often shadowed by estrogen [11]. In males, androgens promote radial bone expansion. Androgens not only activate androgen receptors but also activate estrogen receptors (α and β) by getting aromatized to estrogen [12].

Bone mass in both sexes starts to deteriorate after reaching a peak. However, mechanisms diverge: Women experience trabecular loss in number (fewer trabeculae, greater spacing). Men experience trabecular thinning, with the number preserved. In the cortical compartment, women show significant declines in cortical density and thickness, with a >2-fold greater increase in porosity compared with men. These microstructural differences highlight that men are not immune to skeletal deterioration – rather, the pattern differs, challenging the simplistic narrative of osteoporosis as "female-only" [13]. Women tend to lose bone mass more rapidly than men due to decreased estrogen levels after menopause. In females, the bone marrow precursor cells often show higher osteoclastogenesis, which results in higher bone resorption and lower peak bone mass [14]. Females aged above 50 have nearly 2 times the rate of osteopenia and 4 times the rate of osteoporosis compared to men. Females tend to have fractures 5–10 years earlier than men [15]. According to Korean research on individuals aged 50–89, 37% women and 7.8% men suffered from osteoporosis. Comparing males and females in their 70s, roughly 62.7% females and 15.1% males were found to be osteoporotic [16]. Bone loss begins in the middle of the third decade in females, and they lose nearly 35% of cortical bone and 50% of trabecular bone over their lifetimes. Men lose around two-thirds of this amount, which is significantly less than that of females [17].

Osteoporosis is often perceived as a predominantly "female" disease, leading to reduced awareness among men and consequently lowering the likelihood that they will pursue evaluation or preventive care. This misconception may also decrease clinical vigilance among healthcare providers when assessing male patients. To address these gender-based disparities, the present study investigates sex-related differences in BTM levels C-terminal telopeptide of type I collagen (CTX), BMD, calcium, Vitamin D, and parathyroid hormone (PTH), and examines their associations with fracture history within the study cohort. By elucidating these relationships, the study aims to underscore the influence of gender bias on the diagnosis and management of osteoporosis, particularly the under-recognition of the condition in men.

METHODS

Study design and participants

This cross-sectional study included 100 age-matched individuals (50 males and 50 females) recruited from Mullana, Haryana. Ethical approval was obtained from the Institutional Ethics Committee of Maharishi Markandeshwar University (IEC-2780; dated December 08, 2023). Written informed consent was obtained from all participants.

Postmenopausal and perimenopausal women, as well as elderly men, were included. Individuals receiving medications known to affect bone metabolism were excluded.

Data collection

Demographic and clinical details, including body mass index, current medications, smoking and alcohol consumption habits, and fracture history, were recorded.

Biochemical analysis

All blood samples were collected between 8:00 and 10:00 A.M., after an overnight fast of 8–10 h to minimize diurnal variation, particularly for CTX. Serum calcium was measured using an automated chemistry analyzer (Electtronica Rappresentanze Biomediche e Affini (ERBA) EM-360 using Arsenazo method). Serum 25-hydroxyvitamin D and PTH were measured using chemiluminescence immunoassay kits on Cobas E411 by Roche Diagnostics. CTX and procollagen type I N-terminal propeptide (P1NP) were quantified using the sandwich enzyme-linked immunosorbent assay (ELISA) method by kits provided by Invitrogen (Thermofisher), and the readings were obtained by the LISAscan EM Automated ELISA microplate reader. Inter- and intra-assay coefficients of variation were <10% for all analytes.

BMD assessment

BMD was measured at the femoral neck using DXA (Hologic Discovery Wi, Hologic Inc., USA). Osteoporosis was defined as a femoral neck T-score ≤−2.5, osteopenia as a T-score between −1.0 and −2.5, and normal bone density as T-score ≥ −1.0.

Definition of fracture history

Fracture history was defined as prior low-trauma (fragility) fractures involving the hip, vertebrae, or distal radius, confirmed by radiographic records. High-trauma fractures were excluded.

Statistical analysis

Data were analyzed using the Statistical Package for the Social Sciences software.

Table 3: Correlation of biochemical markers with T-score (femoral neck) and their clinical interpretation

Parameters n=100	r-value	p-value	Statistical tests performed	Interpretations
Calcium	≈0.55	<0.001	Spearman's rank correlation coefficient	Calcium increases if the T-score is better
Vitamin D	≈0.58	<0.001		Vitamin D increases if the T-score is better
PTH	≈−0.22	≈0.028		Weak negative
CTX	≈−0.73	<0.001		Strong negative, CTX increases if the T-score worsens
P1NP	≈−0.68	<0.001		Negative, P1NP increases if the T-score worsens

CTX: C-terminal telopeptide of type I collagen, P1NP: Procollagen type I N-terminal propeptide, PTH: Parathyroid hormone. Strong associations between bone turnover markers and disease severity: CTX and P1NP correlate strongly and negatively with T-score (r about−0.73 and−0.68, p<0.001), while calcium and vitamin D correlate positively (r about 0.55 and 0.58, respectively, p<0.001)

Table 4: Association of fracture history with T-score (femoral neck) by gender

Fracture history	n	Mean T-score±SD	Test	p-value	Gender
Yes	8	−2.38±0.52	Mann–Whitney U	<0.001 highly significant	Male
No	42	−0.80±1.21			
Yes	8	−2.762±0.48			Females
No	42	−1.25±1.09			

SD: Standard deviation

- Continuous variables were expressed as mean±standard deviation
- Student's t-test was used for normally distributed data
- Mann–Whitney U test was applied for non-normally distributed variables
- Correlation coefficients (r) were calculated
- p<0.05 was considered statistically significant.

RESULTS

No statistically significant gender differences were seen in levels of Calcium, Vitamin D, PTH, CTX and P1NP (Table 1).
CTX and P1NP show strong correlation with each other (Table 2).
Both CTX and P1NP increase with decrease in T-scores showing strong negative correlation (Table 3).
In men, those with a positive fracture history had lower mean T-scores than those without fractures. In women, fracture-positive subjects had significantly lower T-scores compared to fracture-negative subjects (Table 4). The fractures recorded were hip fractures, vertebral fractures, and distal radius fractures.

- No parameter reached statistical significance at p<0.05.
- Calcium (mg/dL) showed a trend toward lower values with fracture history, but narrowly missed significance (p=0.056).
- All other biochemical parameters (Vitamin D, PTH, CTX, and P1NP) showed non-significant differences between those with and without fracture history in this sample (Table 5).

This suggests that while there are numerical trends, none of these biochemical markers alone show a statistically significant association with fracture history in our dataset.

Males show a higher T-score value in all the study groups (controls, osteopenia, and osteoporosis) due to physiologically higher bone mineral densities (Table 6).

DISCUSSION

This study has certain limitations. As a cross-sectional design, it does not allow inference of causality or temporal relationships between biochemical markers and BMD. Although significant correlations were observed, it cannot be determined whether alterations in vitamin D, CTX, or P1NP precede changes in bone density or are secondary phenomena. Longitudinal cohort studies are required to establish directionality and predictive value.

In this age-matched cohort of men and women, osteoporosis-related biochemical markers and BMD behaved similarly across genders. Yet, fractures were strongly linked to lower T-scores in both sexes. Despite no significant gender differences in mean calcium, vitamin D, PTH, CTX, and P1NP levels, CTX and P1NP demonstrated excellent diagnostic performance for osteoporosis, while routine chemistries offered limited discriminative value. These findings support the hypothesis that biological differences alone may not fully explain observed disparities in osteoporosis recognition and management. However, this study does not directly measure healthcare bias.

Previous studies mention a greater risk of 1-year mortality and morbidity in men following a hip fracture [18]. The rate of mortality is nearly 31% in men and 17% in women [15]. According to a retrospective study database from 2007 to 2014 of the American College of Surgeons National Surgery Quality Improvement Project, out of 1979 patients, men had a statistically higher mortality rate (odds ratio [OR]=1.58, p=0.05) than women following surgery for osteoporotic vertebral compression fracture. The 30-day readmission rate was also statistically higher in men (OR=1.41; p=0.017) [19]. A Vienna-based study compared age- and BMD-matched cohorts of older adults and revealed that men are more prone to vertebral fractures than women at equivalent bone density measurements [20]. A landmark prospective cohort study conducted in Dubbo, Australia, investigated sex-specific mortality risks after osteoporotic fractures. The study followed 4,311 community-dwelling adults aged 60 years and older (2,413 women and 1,898 men) over 5 years from 1989 to 1994. Among women, mortality was significantly increased after hip fractures (standardized mortality ratio (SMR) 2.18), vertebral fractures (SMR 1.66), and other major fractures (SMR 1.92). Men exhibited even greater vulnerability: Hip (SMR 3.17), vertebral (SMR 2.38), and other major fractures (SMR 2.22) were all linked to elevated mortality. Notably, men also experienced excess deaths even after minor fractures (SMR 1.45), a pattern not seen in women. This work established that all major osteoporotic fractures – not just hip fractures – are associated with substantial mortality in older adults. The findings emphasize that fracture prevention strategies should be applied broadly, with greater attention to men who have historically been overlooked in osteoporosis care [21].

Despite the substantial burden of osteoporotic fractures in both sexes, marked disparities exist in the diagnosis and treatment of men compared with women. Several guidelines have been issued for screening elderly males for osteoporosis. The 2013 consensus report from the NOF and the International Society for Clinical Densitometry recommends BMD testing using DXA in specific groups. Screening is strongly advised for all women aged 65 years and older and for men aged 70 years and above [22]. The Endocrine Society advises that all men aged 70 years and older undergo DXA screening, as well as men aged 50–69 years if they present risk factors, including low body mass, history of fractures in adulthood, or tobacco use [23]. The American College of Preventive Medicine (ACPM) advises that all individuals aged 50 years and older should be assessed for osteoporosis risk factors. BMD testing using DXA is specifically recommended for men aged 70 years and above. For those in the 50–69 year age range, DXA screening is advised only when they present with at least one major risk factor or two or more minor risk factors. In addition, the ACPM highlights the value of validated fracture-risk assessment tools, such as the World Health Organization fracture risk assessment tool algorithm, to support clinical decision-making and identify individuals who would

Table 5: Comparison of biochemical parameters by fracture history

Parameter	Mean±SD (fracture yes) n=16	Mean±SD (fracture no) n=84	Test used	p-value
Calcium	9.148±0.466	9.402±0.421	Student t-test	0.0556
Vitamin d	24.331±8.398	26.551±7.946	Student t-test	0.3398
PTH	45.612±14.325	47.444±17.995	Student t-test	0.6577
CTX	0.540±0.307	0.416±0.229	Mann–Whitney U	0.1713
PINP	65.714±26.002	55.448±24.022	Mann–Whitney U	0.0989

SD: Standard deviation, CTX: C-terminal telopeptide of type I collagen, P1NP: Procollagen type I N-terminal propeptide, PTH: Parathyroid hormone

Table 6: Gender-wise comparison of t-scores across control, osteopenia, and osteoporosis groups using parametric analysis

Gender	Controls (n) (mean±SD)	Osteopenia (n) (mean±SD)	Osteoporosis (n) (mean±SD)
Male	16 controls	19 patients	15 patients
	0.51±0.59	−1.70±0.42	−2.95±0.36
Female	19 controls	16 patients	15 patients
	0.03±0.83	−1.82±0.44	−3.08±0.44

SD: Standard deviation

benefit most from screening [24].

Screening with DXA remains disproportionately low in men; among medicare beneficiaries, only about 5% of men underwent DXA compared with 19.8% of women (p<0.001) [25]. This underuse has been consistently highlighted across reviews, which emphasize systematic underscreening and underdiagnosis of osteoporosis in men relative to women. Broader U.S. data also demonstrate poor baseline uptake of DXA overall, with persistent sex-based disparities despite guideline recommendations [26]. Evidence shows that men are significantly less likely than women to receive pharmacologic treatment following hip fracture, with only 4.5% of men versus 27% of women discharged on osteoporosis therapy, accompanied by a 12-month mortality of 32% in men versus 17% in women [27]. Large claims analyses echo this trend: Within 6 months after a fragility fracture, only 10.2% received testing and/or treatment (12.1% in women vs. 5.7% in men) [28]. Even in women, treatment rates remain suboptimal (≈23% within 1 year), underscoring that while the problem is system-wide, undertreatment is more pronounced in men [29]. Contemporary cohort data confirm that male sex independently predicts failure to initiate therapy after fracture, with regional data showing persistently low treatment rates (≈17.2% overall) and men consistently at the lowest end [30,31]. Fewer men undergo DXA testing. Only 11% of men versus 27% of women had DXA within 5 years before a fracture, and among hospitalized patients, 5.4% of men versus 12.1% of women received DXA assessments [32]. This disparity is further reinforced by the relative scarcity of clinical evidence specific to males. Randomized controlled trials of osteoporosis drugs in men are fewer, often underpowered, and limited largely to vertebral endpoints. Uncertainties regarding comparative effectiveness, optimal duration of therapy, drug holidays, and the role of testosterone further contribute to therapeutic hesitation in male patients [33].

These gaps have important consequences, as men experience higher post-fracture morbidity and mortality compared with women [34]. Nevertheless, emerging evidence suggests that when treated, men derive a similar reduction in hip fracture risk as women, countering any justification for therapeutic nihilism [35]. Even the physician's awareness of osteoporosis in men remains limited. Surveys show low familiarity with male risk factors, leading to lower rates of BMD testing and treatment initiation in men compared to women with similar fracture risks [36]. Research on male osteoporosis is also underrepresented. Men also suffer from diagnostic limitations. The interpretation of DXA scans in men is complicated by differences in reference databases and thresholds. In some cases, female-based reference standards are applied to male patients, which may contribute to misclassification and hesitancy in making treatment decisions [37].

Limitations

Although the total sample size was adequate for exploratory comparisons, subgroup analyses – particularly the fracture subgroup (n=16) – were underpowered. Therefore, the absence of statistical significance in fracture-associated biochemical comparisons should not be interpreted as evidence of no association. Similarly, small but clinically meaningful gender differences may not have been detectable.

CONCLUSION

Persistent gender bias in osteoporosis detection, diagnosis, and treatment undermines clinical effectiveness and leads to unnecessary health issues. Evidence shows that men are under-screened and under-treated, while women often face stereotypes that delay personalized care. This underscores the need to reframe osteoporosis as a condition defined by bone health and fracture risk rather than sex alone. Addressing this inequality requires immediate action: Expanding sex-inclusive screening guidelines, incorporating sex- and gender-sensitive risk assessment tools, deliberately involving diverse participants in clinical research, and providing targeted education for providers to counter implicit biases. Health systems must also monitor and report sex-disaggregated outcomes to identify gaps and track progress. Only through coordinated policy reforms, research priorities, and changes in clinical practice can equitable, evidence-based osteoporosis care be achieved and the burden of preventable fractures reduced across all populations.

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AUTHOR'S CONTRIBUTION

Chahat Sehgal: Conceptualization, data collection, analysis, manuscript drafting. Karanpreet Bhutani: Methodology supervision, statistical review. Aditi Sharma: Data validation, manuscript editing.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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