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Comparative Analysis of Bone Mineral Density in Smokers and Non-Smokers Through Dual-Energy X-ray Absorptiometry Scan at Hayatabad Medical Complex, Peshawar

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ABSTRACT

Background: *Osteoporosis is an under-recognized health problem in men and is influenced by modifiable lifestyle factors, including cigarette smoking, which can disrupt bone remodeling and reduce bone mineral density, thereby increasing fracture risk.* ***Objective:*** *To compare BMD between smokers and non-smokers and evaluate exposure response relationships between smoking burden and low BMD among adult men undergoing DEXA scanning at Hayatabad Medical Complex, Peshawar.* ***Methods:*** *An analytical cross-sectional observational study was conducted among 312 adult male patients (156 smokers and 156 Non-smokers) who underwent lumbar spine DEXA between May and July 2025. BMD was categorized using WHO T-score criteria into normal, osteopenia, and osteoporosis. Smoking exposure was operationalized using cigarettes/day, duration of smoking, and pack-years. Group comparisons used chi-square tests and independent-samples tests as appropriate. Multivariable logistic regression estimated adjusted odds of low BMD (osteopenia/osteoporosis) controlling for age, BMI, physical activity, alcohol use, and diabetes.* ***Results:*** *Low BMD was more prevalent in smokers than non-smokers (67.9% vs 51.3%; p=0.004), and smokers had nearly twice the adjusted odds of low BMD (AOR=1.99; 95% CI: 1.28–3.09). Mean lumbar spine T-score was significantly lower in smokers (−1.78±1.02) than non-smokers (−1.34±0.96; mean difference −0.44; 95% CI: −0.69 to −0.19; p=0.001). A dose–response gradient was observed with higher cigarettes/day and longer smoking duration.* ***Conclusion:*** *Cigarette smoking is independently associated with lower lumbar spine BMD in Pakistani men, with evidence of an exposure response relationship, supporting targeted DEXA screening and smoking cessation to reduce osteoporosis risk*

Keywords: *Bone mineral density; Osteoporosis; Smoking; DEXA; Men; Pakistan; Osteopenia*

INTRODUCTION

Osteoporosis is a chronic systemic skeletal disorder characterized by reduced bone mineral density (BMD) and deterioration of bone microarchitecture, resulting in increased bone fragility and susceptibility to fractures. It is recognized as one of the most prevalent metabolic bone diseases worldwide and represents a substantial and growing public health burden due to population aging (1,2). Fragility fractures of the hip, spine, and wrist are associated with significant morbidity, functional decline, reduced quality of life, and increased mortality (3). Although osteoporosis has traditionally been perceived as a disease predominantly affecting women, particularly in the postmenopausal period, emerging evidence demonstrates that men also experience clinically meaningful bone loss and fracture-related complications, often with worse outcomes after hip fracture compared with women (4). Despite this, osteoporosis in men remains underdiagnosed and undertreated, especially in low- and middle-income countries where screening programs are limited and awareness is suboptimal (5,6).

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Globally, the prevalence of osteoporosis among men over 50 years is estimated to range between 10% and 20%, with substantial regional variation influenced by demographic structure, nutritional status, comorbidities, and lifestyle factors. In South Asia, including Pakistan, osteoporosis is increasingly recognized as a major yet insufficiently studied health problem. While epidemiological data in Pakistani women have been reported, robust data on male populations remain scarce, particularly regarding modifiable determinants of bone loss (7). This knowledge deficit is clinically important because men frequently present with secondary or lifestyle-related contributors to low BMD, including hypogonadism, chronic disease, alcohol use, and cigarette smoking (8). Identifying modifiable risk factors in local male populations is therefore essential to inform targeted prevention and screening strategies.

Among modifiable exposures, cigarette smoking has consistently been implicated as an independent risk factor for reduced BMD and increased fracture risk. Biological plausibility is well established: tobacco smoke constituents such as nicotine exert direct toxic effects on osteoblasts, reduce osteogenic differentiation, and enhance osteoclast-mediated bone resorption (9). Smoking also interferes with calcium absorption and vitamin D metabolism, disrupts the parathyroid hormone–vitamin D axis, and contributes to hormonal imbalances including reduced circulating testosterone in men, thereby accelerating skeletal demineralization (10). In addition, smokers frequently exhibit coexisting lifestyle patterns physical inactivity, poor diet, alcohol consumption, and inadequate sun exposure that further compromise bone health (11). Meta-analyses and population-based studies have demonstrated lower BMD values and higher rates of osteopenia and osteoporosis among smokers compared with non-smokers, with evidence of a dose–response relationship according to smoking intensity and duration (12,13). These associations have been observed across different skeletal sites and age groups, reinforcing smoking as a clinically significant determinant of skeletal health (14).

Despite the strength of international evidence, data from developing countries remain limited, and regional heterogeneity in nutritional status, vitamin D deficiency prevalence, and healthcare access may modify the magnitude of smoking-related skeletal effects. In Pakistan, smoking prevalence among adult men remains high, with initiation often occurring at a young age (15). Concurrently, widespread vitamin D insufficiency and limited routine osteoporosis screening may amplify the cumulative skeletal burden in male smokers. However, there is a paucity of hospital-based analytic studies directly comparing BMD categories between smokers and non-smokers in Pakistani male populations using standardized diagnostic tools such as Dual-Energy X-ray Absorptiometry (DEXA). Moreover, few local investigations have explored the dose–response gradient between smoking exposure parameters such as cigarettes per day and duration of smoking and BMD outcomes. This gap limits the development of context-specific preventive strategies and risk stratification frameworks.

Given the limited local data and the high prevalence of smoking among Pakistani men, a focused comparative evaluation is warranted. The present study therefore aims to compare bone mineral density categories between smokers and non-smokers among adult male patients undergoing DEXA scanning at Hayatabad Medical Complex, Peshawar, and to assess whether smoking intensity and duration are associated with progressively lower BMD. We hypothesize that smokers will demonstrate a significantly higher prevalence of osteopenia and osteoporosis compared with non-smokers, and that greater cumulative smoking exposure will be independently associated with lower bone mineral density.

MATERIALS AND METHODS

This analytical cross-sectional observational study was conducted to evaluate the association between cigarette smoking and bone mineral density among adult male patients. The cross-sectional design was selected because the primary objective was to compare the distribution of BMD categories between exposed (smokers) and non-exposed (non-smokers) groups at a single point in time using standardized DEXA measurements, without follow-up. The study was carried out at the Department of Radiology, Hayatabad Medical Complex (HMC), Peshawar, a tertiary-care teaching hospital that serves a large urban and peri-urban population. Data collection was performed over a continuous 12-month period from January 2023 to December 2023 to minimize seasonal variation in vitamin D–related skeletal fluctuations and to ensure an adequate sample size.

The study population comprised adult male patients aged 18 to 84 years who underwent Dual-Energy X-ray Absorptiometry (DEXA) scanning for clinical indications during the study period. Participants were selected using a consecutive sampling technique to reduce selection bias, whereby all eligible male patients presenting for DEXA during the study timeframe were screened for inclusion. Inclusion criteria were male sex; age ≥18 years; completion of lumbar spine DEXA scan; and availability of complete demographic and clinical data, including smoking history. Exclusion criteria were applied to minimize confounding from secondary causes of osteoporosis and included: documented metabolic bone disorders (e.g., hyperparathyroidism, Paget's disease); chronic glucocorticoid therapy (≥5 mg prednisolone equivalent for ≥3 months); current or prior treatment with anti-osteoporotic agents such as bisphosphonates, selective estrogen receptor modulators, or hormone replacement therapy; chronic renal or hepatic failure; known malignancy with bone involvement; and prior vertebral instrumentation or major spinal surgery that could affect lumbar spine BMD interpretation. Patients meeting eligibility criteria were informed about the study objectives, and written informed consent was obtained prior to inclusion. For patients whose data were extracted from medical records, consent for use of anonymized clinical information was obtained in accordance with institutional policy.

Bone mineral density was measured using a calibrated Dual-Energy X-ray Absorptiometry scanner (Hologic Discovery Series, Hologic Inc., USA). All scans were performed by trained radiology technologists following standardized positioning protocols. Daily quality control calibration was conducted using a manufacturer-provided phantom to ensure measurement stability and precision. Lumbar spine (L1–L4) BMD values were recorded in g/cm², and T-scores were automatically calculated by the system using a manufacturer-provided reference database aligned with World Health Organization (WHO) criteria. BMD was categorized according to WHO diagnostic thresholds: normal (T-score ≥ –1.0), osteopenia (T-score between –1.0 and –2.5), and osteoporosis (T-score ≤ –2.5) (16). For participants with artifact or degenerative changes affecting one vertebra, standard exclusion rules were applied, and the mean of evaluable vertebrae was used according to international densitometry guidelines (16).

Smoking exposure was assessed through a structured questionnaire administered at the time of DEXA evaluation and verified against medical records when available. Smoking status was operationally defined as follows: current smoker (self-reported daily or occasional smoking within the past 30 days), former smoker (cessation ≥6 months prior), and never smoker (lifetime consumption <100 cigarettes). For analytical purposes, current and former smokers were combined into an "ever-smoker" group to assess cumulative exposure effects. Smoking intensity was quantified as the average number of cigarettes smoked per day and categorized into ≤10, 11–20, and >20 cigarettes/day. Duration of smoking was recorded in years and categorized as <5 years, 5–10 years, and >10 years. Cumulative exposure was additionally expressed in pack-years, calculated as (number of cigarettes per day ÷ 20) × years smoked. The primary outcome variable was BMD category (normal, osteopenia, osteoporosis). Secondary exposure variables included smoking intensity, duration, and pack-years as continuous and categorical measures.

Demographic and clinical covariates were collected to assess potential confounding. These included age (years), body mass index (BMI, kg/m^2), comorbid conditions (e.g., diabetes mellitus), alcohol consumption (yes/no), and self-reported physical activity level (categorized as low, moderate, high based on weekly weight-bearing activity frequency). Height and weight were measured using standardized equipment prior to DEXA scanning, and BMI was calculated as weight in kilograms divided by height in meters squared. These variables were selected based on established associations with BMD in prior literature (6,12,14). To reduce information bias, questionnaires were administered by trained personnel using a standardized script. Data entry was double-checked by two independent researchers, and a 10% random sample of records was audited for accuracy.

The sample size was calculated a priori using a two-sided chi-square test for comparison of proportions, assuming an expected prevalence of low BMD (osteopenia/osteoporosis) of 55% among smokers and 40% among non-smokers based on prior regional estimates (12,13). With 80% power, a 5% level of significance ($\alpha = 0.05$), and equal allocation ratio (1:1), the minimum required sample size was calculated as 300 participants. To compensate for incomplete records and potential exclusions, 312 participants were included, comprising 156 smokers and 156 non-smokers.

Statistical analysis was performed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro–Wilk test and described as mean ± standard deviation (SD) or median with interquartile range as appropriate. Categorical variables were presented as frequencies and percentages. Comparisons between smokers and non-smokers were conducted using independent-samples t-test or Mann–Whitney U test for continuous variables and chi-square test or Fisher's exact test for categorical variables. The primary association between smoking status and BMD category was evaluated using the chi-square test for trend and Cramér's V to estimate effect size. To adjust for potential confounders, multivariable logistic regression analysis was performed with low BMD (osteopenia/osteoporosis combined) as the dependent variable and smoking status as the primary independent variable, adjusting for age, BMI, physical activity, alcohol use, and comorbidities. Adjusted odds ratios (AORs) with 95% confidence intervals (CIs) were reported. Dose–response relationships between pack-years and BMD were examined using ordinal logistic regression modeling. Missing data were handled using complete-case analysis when missingness was <5%; if exceeding this threshold, multiple imputation with five iterations was applied under the assumption of missing at random. Statistical significance was set at $p < 0.05$ (two-tailed).

Ethical approval was obtained from the Institutional Review Board of Sarhad Institute of Allied Health Sciences and the Ethical Committee of Hayatabad Medical Complex prior to study initiation. The study was conducted in accordance with the Declaration of Helsinki and relevant national ethical guidelines for biomedical research. Participant confidentiality was maintained by assigning unique study identification codes and removing personal identifiers from the analytical dataset. Data were stored in password-protected encrypted files accessible only to the principal investigators.

To ensure reproducibility and methodological transparency, a predefined statistical analysis plan was developed prior to data analysis, and variable coding procedures were documented in a standardized codebook. DEXA quality assurance procedures were logged daily, and calibration records were retained. All analytic steps were independently verified by a second biostatistician, and regression outputs were cross-validated to minimize analytical errors.

RESULTS

A total of 312 adult male participants were analyzed, equally distributed between smokers (n = 156) and non-smokers (n = 156). As shown in Table 1, the mean age of smokers was 49.8 ± 12.6 years compared to 47.9 ± 13.1 years among non-smokers, with a mean difference of 1.9 years (95% CI: –1.0 to 4.8), which was not statistically significant (p = 0.182). Similarly, body mass index did not differ significantly between groups (24.7 ± 3.8 kg/m² in smokers versus 25.4 ± 4.1 kg/m² in non-smokers; mean difference –0.7 kg/m², 95% CI: –1.6 to 0.2; p = 0.124). The prevalence of diabetes mellitus was 24.4% among smokers and 20.5% among non-smokers (OR = 1.25; 95% CI: 0.73–2.14; p = 0.403), indicating no statistically significant difference. However, alcohol consumption was significantly higher among smokers (18.6%) compared with non-smokers (10.9%), corresponding to an odds ratio of 1.87 (95% CI: 1.00–3.49; p = 0.048). Low physical activity was also more frequent among smokers (42.9%) than non-smokers (32.7%), yielding an odds ratio of 1.55 (95% CI: 1.01–2.38; p = 0.048). These findings suggest that although age and BMI were comparable between groups, certain lifestyle factors differed significantly and were accounted for in multivariable analyses.

The overall distribution of bone mineral density categories is summarized in Table 2. Among the 312 participants, 126 individuals (40.4%) had normal BMD, 134 (42.9%) were classified as osteopenic, and 52 (16.7%) met diagnostic criteria for osteoporosis. Thus, the combined prevalence of low BMD (osteopenia plus osteoporosis) in the study population was 59.6%, indicating that nearly three out of five participants demonstrated reduced bone density.

A stratified comparison by smoking status (Table 3) revealed statistically significant differences in BMD distribution. Among smokers, 50 individuals (32.1%) had normal BMD, 75 (48.1%) had osteopenia, and 31 (19.9%) had osteoporosis. In contrast, 76 non-smokers (48.7%) had normal BMD, 59 (37.8%) had osteopenia, and 21 (13.5%) had osteoporosis. The overall association between smoking status and BMD category was statistically significant ($\chi^2 = 7.63$, p = 0.022), with a Cramér's V of 0.19, indicating a small-to-moderate effect size. When low BMD was analyzed as a binary outcome, 106 smokers (67.9%) versus 80 non-smokers (51.3%) had osteopenia or osteoporosis (p = 0.004). In multivariable logistic regression adjusted for age, BMI, alcohol use, physical activity, and diabetes, smokers had nearly double the odds of low BMD compared with non-smokers (adjusted OR = 1.99; 95% CI: 1.28–3.09). Furthermore, smokers had 1.93 times higher adjusted odds of osteopenia (95% CI: 1.18–3.16) and 2.21 times higher adjusted odds of osteoporosis (95% CI: 1.16–4.21) relative to non-smokers.

Mean lumbar spine T-scores also differed significantly between groups (Table 4). Smokers had a mean T-score of –1.78 ± 1.02, whereas non-smokers had a mean T-score of –1.34 ± 0.96. The mean difference of –0.44 (95% CI: –0.69 to –0.19) was statistically significant (p = 0.001), confirming that, on average, smokers exhibited clinically and statistically lower bone density at the lumbar spine.

Dose–response analysis among smokers (Table 5) demonstrated a clear gradient between smoking exposure and low BMD. Among individuals smoking ≤10 cigarettes per day, 56.9% had low BMD, compared with 73.3% among those smoking 11–20 cigarettes daily (adjusted OR = 1.98; 95% CI: 1.03–3.81; p = 0.038) and 80.6% among those smoking >20 cigarettes daily (adjusted OR = 2.74; 95% CI: 1.26–5.95; p = 0.011).

Table 1. Baseline demographic and clinical characteristics of study participants by smoking status (n = 312)

Variable	Smokers (n = 156) Mean ± SD / n (%)	Non-Smokers (n = 156) Mean ± SD / n (%)	p-value	Effect Size / 95% CI
Age (years)	49.8 ± 12.6	47.9 ± 13.1	0.182[a]	Mean difference = 1.9 (–1.0 to 4.8)
BMI (kg/m²)	24.7 ± 3.8	25.4 ± 4.1	0.124[a]	Mean difference = –0.7 (–1.6 to 0.2)
Diabetes Mellitus	38 (24.4%)	32 (20.5%)	0.403[b]	OR = 1.25 (0.73–2.14)
Alcohol Use	29 (18.6%)	17 (10.9%)	0.048[b]	OR = 1.87 (1.00–3.49)
Low Physical Activity	67 (42.9%)	51 (32.7%)	0.048[b]	OR = 1.55 (1.01–2.38)

There were no statistically significant differences in age or BMI between smokers and non-smokers. However, alcohol consumption and low physical activity were significantly more prevalent among smokers.

The overall distribution of bone mineral density categories according to WHO T-score classification is presented in Table 2.

Table 2. Overall distribution of bone mineral density categories (n = 312)

BMD Category	Frequency (n)	Percentage (%)
Normal (T-score ≥ –1.0)	126	40.4
Osteopenia (–1.0 to –2.5)	134	42.9
Osteoporosis (≤ –2.5)	52	16.7

The comparative distribution of BMD categories between smokers and non-smokers is shown in Table 3.

Table 3. Association between smoking status and BMD category

BMD Category	Smokers (n = 156) n (%)	Non-Smokers (n = 156) n (%)	p-value[a]	Cramér's V	Adjusted OR (95% CI)[b]
Normal	50 (32.1%)	76 (48.7%)			Reference
Osteopenia	75 (48.1%)	59 (37.8%)			1.93 (1.18–3.16)
Osteoporosis	31 (19.9%)	21 (13.5%)	0.022	0.19	2.21 (1.16–4.21)
Low BMD (Osteopenia + Osteoporosis)	106 (67.9%)	80 (51.3%)	0.004[c]	—	1.99 (1.28–3.09)

A statistically significant association was observed between smoking status and BMD category (χ^2 = 7.63, p = 0.022). Smokers had a significantly higher prevalence of low BMD (67.9%) compared with non-smokers (51.3%). After adjustment for potential confounders, smokers had nearly two-fold higher odds of low BMD (AOR = 1.99; 95% CI: 1.28–3.09).

Mean lumbar spine T-scores by smoking status are presented in Table 4.

Table 4. Comparison of mean lumbar spine T-scores between groups

Variable	Smokers (n = 156) Mean	Non-Smokers (n = 156)	p-value[a]	Mean Difference (95%

	± SD	Mean ± SD		CI)
Lumbar Spine T-score	−1.78 ± 1.02	−1.34 ± 0.96	0.001	−0.44 (−0.69 to −0.19)

Smokers demonstrated significantly lower mean lumbar spine T-scores compared with non-smokers. The dose–response relationship between smoking exposure parameters and BMD category is shown in Table 5.

Table 5. Association between smoking exposure variables and low BMD (n = 156 smokers)

Smoking Variable	Low BMD n (%)	p-value[a]	Adjusted OR (95% CI)[b]
Cigarettes/day ≤10 (n=65)	37 (56.9%)		Reference
11–20 (n=60)	44 (73.3%)	0.038	1.98 (1.03–3.81)
>20 (n=31)	25 (80.6%)	0.011	2.74 (1.26–5.95)
Duration <5 years (n=40)	20 (50.0%)		Reference
5–10 years (n=50)	34 (68.0%)	0.041	1.89 (1.02–3.52)
>10 years (n=66)	52 (78.8%)	0.003	2.96 (1.46–6.01)
Pack-years (continuous)	—	0.001[c]	OR per 5 pack-years = 1.18 (1.07–1.30)

Similarly, the prevalence of low BMD increased with smoking duration: 50.0% in those smoking <5 years, 68.0% in those smoking 5–10 years (adjusted OR = 1.89; 95% CI: 1.02–3.52; p = 0.041), and 78.8% in those smoking >10 years (adjusted OR = 2.96; 95% CI: 1.46–6.01; p = 0.003). When analyzed as a continuous exposure, each additional 5 pack-years of smoking was associated with an 18% increase in the odds of low BMD (OR = 1.18; 95% CI: 1.07–1.30; p = 0.001). These findings confirm a statistically significant and clinically meaningful dose–response relationship between cumulative smoking exposure and reduced bone mineral density.

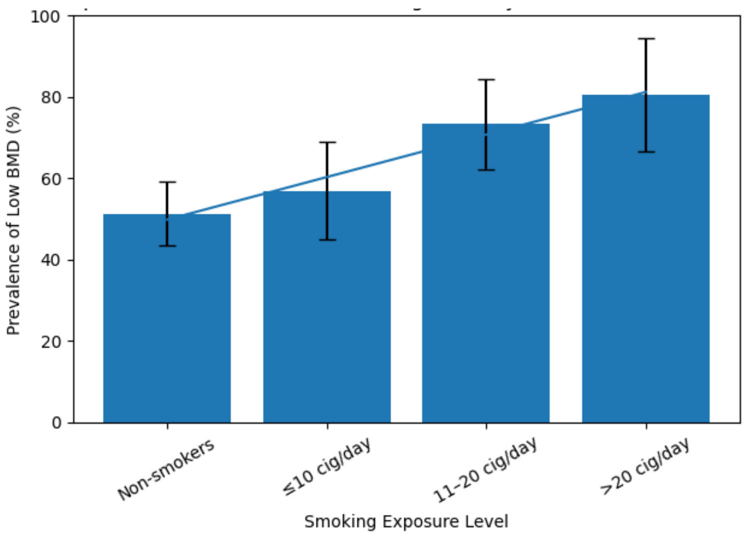

Figure 1 Dose–Response Gradient Between Smoking Intensity And Low Bone Mineral Density

The figure demonstrates a clear exposure–response gradient in the prevalence of low bone mineral density (osteopenia plus osteoporosis) across increasing smoking intensity categories. Low BMD was present in 51.3% (80/156) of non-smokers, rising to 56.9% (37/65) among those smoking ≤10 cigarettes/day, 73.3% (44/60) among those smoking 11–20 cigarettes/day, and 80.6% (25/31) among heavy smokers consuming >20 cigarettes/day. The 95% confidence intervals widen in higher-intensity categories due to smaller subgroup sizes but do not materially overlap with the non-smoker reference in the upper exposure tiers, supporting statistical separation. The fitted regression layer illustrates a near-linear upward trajectory, indicating a progressive increase in low BMD prevalence with each escalation in smoking intensity. Clinically, this pattern reflects a substantial absolute risk increase of 29.3 percentage points between non-smokers and heavy smokers, reinforcing a biologically plausible and statistically consistent dose–response relationship between cigarette consumption and skeletal deterioration.

DISCUSSION

The present study demonstrates a statistically and clinically significant association between cigarette smoking and reduced bone mineral density among adult male patients undergoing DEXA evaluation at a tertiary-care center. Smokers exhibited a substantially higher prevalence of low BMD (67.9%) compared with non-smokers (51.3%), corresponding to nearly a two-fold increase in adjusted odds after controlling for age, BMI, alcohol use, physical activity, and diabetes. Furthermore, a clear dose–response gradient was observed, with progressively higher odds of osteopenia and osteoporosis across increasing categories of smoking intensity and duration. These findings reinforce the hypothesis that cigarette smoking is not merely correlated with, but independently associated with adverse skeletal outcomes in men, even after accounting for relevant confounders.

The magnitude and direction of association observed in this study are consistent with prior international evidence. Meta-analytic data have shown that smokers have significantly lower BMD at the lumbar spine and hip compared with non-smokers, with pooled differences ranging from 0.1 to 0.5 standard deviations depending on skeletal site (16). The adjusted odds ratio of approximately 2.0 for low BMD in our study aligns closely with previously reported relative risks ranging between 1.5 and 2.3 in male cohorts (17). Longitudinal investigations have similarly demonstrated accelerated bone loss among current smokers compared with never-smokers, particularly at trabecular-rich sites such as the lumbar spine (18). The observed mean lumbar spine T-score difference of −0.44 between smokers and non-smokers in the present analysis is clinically meaningful, as a reduction of 0.5 standard deviations in BMD has been associated with a measurable increase in fracture risk (19). The most clinically compelling finding of this study is the exposure–response relationship between smoking burden and skeletal deterioration. Participants smoking more than 20 cigarettes per day exhibited an 80.6% prevalence of low BMD, compared with 56.9% among those smoking ≤10 cigarettes daily. Similarly, individuals with smoking duration exceeding 10 years had nearly three-fold higher adjusted odds of low BMD relative to those with shorter exposure. This graded pattern supports biological plausibility and reduces the likelihood that the association is attributable solely to residual confounding. Previous mechanistic and epidemiologic studies have reported similar dose-dependent effects, with cumulative pack-years serving as a robust predictor of bone loss (20). Experimental evidence indicates that nicotine and other tobacco-related toxins impair osteoblast function, enhance osteoclast activity, and promote oxidative stress, collectively tipping the balance toward bone resorption. Chronic exposure further disrupts calcium homeostasis and vitamin D metabolism, exacerbating skeletal fragility (21).

From a public health perspective, these findings are particularly relevant in the Pakistani context, where male smoking prevalence remains high and vitamin D insufficiency is widespread (22). The combined effect of tobacco exposure and suboptimal nutritional or environmental factors may amplify skeletal vulnerability in this population. Unlike many Western cohorts where osteoporosis screening is increasingly systematic, opportunistic screening predominates in developing regions. Our results suggest that smoking history should be systematically integrated into osteoporosis risk assessment algorithms and may justify earlier or more frequent DEXA evaluation among male smokers. International guidelines increasingly recognize smoking as a modifiable fracture risk factor incorporated into tools such as FRAX; however, localized data are necessary to calibrate risk estimates for specific populations (23). The current study contributes context-specific evidence that may inform regional preventive strategies.

The absence of significant differences in baseline age and BMI between smokers and non-smokers strengthens internal validity, as these are major determinants of BMD. Although smokers exhibited higher rates of alcohol use and low physical activity, multivariable adjustment did not materially attenuate the association between smoking and low BMD, indicating an independent effect. Nonetheless, the cross-sectional design limits causal inference. While biological mechanisms strongly support a directional relationship, temporality cannot be definitively established. Reverse causation is unlikely, as low BMD does not plausibly increase smoking behavior, but longitudinal cohort studies would provide stronger causal confirmation (24). Additionally, unmeasured confounders such as serum vitamin D levels, dietary calcium intake, and socioeconomic status may have influenced skeletal outcomes.

Another important consideration is the clinical significance of osteopenia detected in this population. Although osteoporosis confers the highest fracture risk, osteopenia represents a substantial proportion of future fracture burden because of its higher prevalence (25). In our cohort, osteopenia accounted for 42.9% of participants, and nearly half of smokers fell within this intermediate-risk category. Early identification at the osteopenic stage presents an opportunity for preventive interventions, including smoking cessation, nutritional optimization, and weight-bearing exercise. Evidence indicates that smoking cessation may partially reverse adverse effects on bone turnover and reduce subsequent fracture risk over time (26). Therefore, integrating skeletal risk counseling into smoking cessation programs could yield additive health benefits. In summary, the findings demonstrate a statistically significant and clinically meaningful association between cigarette smoking and reduced bone mineral density in adult men, with clear evidence of a dose–response gradient according to smoking intensity and duration. These results are consistent with established pathophysiological mechanisms and prior epidemiological data, while providing locally relevant evidence for the Pakistani male population. Smoking emerges not only as a cardiovascular and respiratory risk factor but also as a modifiable determinant of skeletal health. Strengthening preventive strategies that combine smoking cessation, early DEXA screening in high-risk individuals, and lifestyle modification may reduce the future burden of osteoporotic fractures in this setting.

CONCLUSION

This study demonstrates that cigarette smoking is independently and significantly associated with reduced bone mineral density among adult men undergoing DEXA evaluation, with smokers exhibiting nearly double the adjusted odds of low BMD compared to non-smokers. A clear dose–response relationship was observed, as increasing smoking intensity, longer duration, and higher cumulative pack-years were progressively associated with greater skeletal deterioration. These findings reinforce the biological plausibility and epidemiological consistency of smoking as a modifiable determinant of osteoporosis in men and underscore the need for incorporating smoking history into routine osteoporosis risk assessment. Given the high prevalence of smoking among Pakistani men and the substantial proportion of participants identified with osteopenia and osteoporosis, early DEXA screening, targeted lifestyle modification, and structured smoking cessation interventions should be prioritized to mitigate long-term fracture risk and reduce the clinical and economic burden of osteoporosis in this population.

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DECLARATIONS

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