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"Comparative Analysis of Bone Mineral Density and Alveolar Bone Loss in Pre- and Post-Menopausal Women with Chronic Periodontitis: A Cross-Sectional Study"

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Abstract:
Periodontitis is a long-term inflammatory disease that affects the tissues supporting the teeth. It can be worsened by conditions like osteoporosis. Post-menopausal women are more at risk due to a lack of estrogen, which speeds up bone loss and can increase periodontal bone loss. This study aims to evaluate and compare bone mineral density and alveolar bone loss in both pre- and post-menopausal women with chronic periodontitis. The study included ninety-seven women, with 40 pre-menopausal and 57 post-menopausal participants. We measured clinical parameters such as probing pocket depth and clinical attachment loss using Williams and CPI probes. We assessed alveolar bone loss with intraoral periapical radiographs and a Dentech X-ray grid. We determined bone mineral density using the SONOST-2000 ultrasound bone densitometer and analyzed the data with SPSS version 16.0, using the independent t-test.The average T-score in pre-menopausal women was -1.22 ± 0.43, compared to -1.97 ± 0.65 in post-menopausal women. The difference was statistically significant (p = 0.001). Additionally, the average alveolar bone loss was 5.28 ± 2.73 mm in pre-menopausal women and 10.21 ± 4.42 mm in post-menopausal women, showing a significant difference as well (p = 0.001). These findings indicate that menopause, along with the decrease in bone mineral density, significantly raises the risk of alveolar bone loss. The results suggest that estrogen deficiency, increased cytokines, and RANKL-driven osteoclast activation may explain this connection. The study concludes that it is crucial to screen for osteoporosis and periodontitis early in women going through menopause to manage and prevent both systemic and oral health issues effectively.
Keywords: Bone mineral density, Periodontitis, Osteoporosis, Menopause, Alveolar bone loss, Estrogen deficiency, RANKL signaling, Cytokines

1. INTRODUCTION

The oral cavity is a vital part of general health. It plays key roles in chewing, speaking, appearance, and overall well-being. Its structures, which include teeth, gums, periodontal ligaments, alveolar bone, and associated muscles, work together to maintain stability and function. Problems with these structures can harm oral health and also affect the body as a whole, showing the close connection between oral health and overall physiology (1,2).

Periodontitis is a long-lasting inflammatory disease that affects the tissues supporting the teeth. It usually starts with bacterial biofilms in people who are prone to it, leading to gradual damage to the periodontal ligaments and alveolar bone. Clinically, it shows up as pockets around teeth, gum recession, loss of attachment, and eventual tooth loss (3). The causes of periodontitis involve a complicated relationship between bacterial attacks and the body's immune response. Factors like genetics, lifestyle, and other health conditions can influence how severe the disease becomes (4,5). If not treated, periodontitis can lead to tooth loss and increase overall inflammation in the body, highlighting its importance for health (6).

Menopause is a significant biological change. It leads to a drop in estrogen levels, which speeds up the loss of bone mineral and makes women more susceptible to osteoporosis. Osteoporosis is a condition

where bone density decreases and bone structure weakens, raising the risk of fractures (7). Post-menopausal women are especially at risk for osteoporosis and periodontal disease since both issues share common risk factors like age, hormonal changes, smoking, and other health problems (8). The decline in bone density makes the alveolar bone more prone to resorption, worsening the impact of chronic periodontitis (9).

On a molecular level, inflammatory cytokines are key players in the bone remodeling processes seen in both osteoporosis and periodontitis. Interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) promote the activity of cells that break down bone. These cytokines trigger the receptor activator of nuclear factor kappa-B ligand (RANKL), which binds to its receptor RANK on precursor cells, helping them develop into osteoclasts and increase bone resorption (10,11). As a result, the activation of RANKL pathways driven by cytokines connects systemic osteoporosis to the loss of alveolar bone in periodontal disease (12).

This mechanistic connection suggests that systemic BMD depletion may influence alveolar bone density, leading to greater susceptibility to periodontal destruction. Several studies have indicated an association between systemic osteoporosis and alveolar bone resorption; however, findings remain inconsistent due to variations in methodology, diagnostic criteria, and study populations (13,14). This highlights a critical research gap, particularly the lack of comparative data on BMD changes between pre- and post-menopausal women with chronic periodontitis.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

This was a descriptive cross-sectional study conducted in the Department of Periodontics and Implantology, Maitri College of Dentistry and Research Center, Anjora, Durg, Chhattisgarh, over a period of six months (January–June 2024). Cross-sectional study designs are widely used in epidemiological research to evaluate associations between systemic conditions and periodontal disease outcomes within a defined population (15).

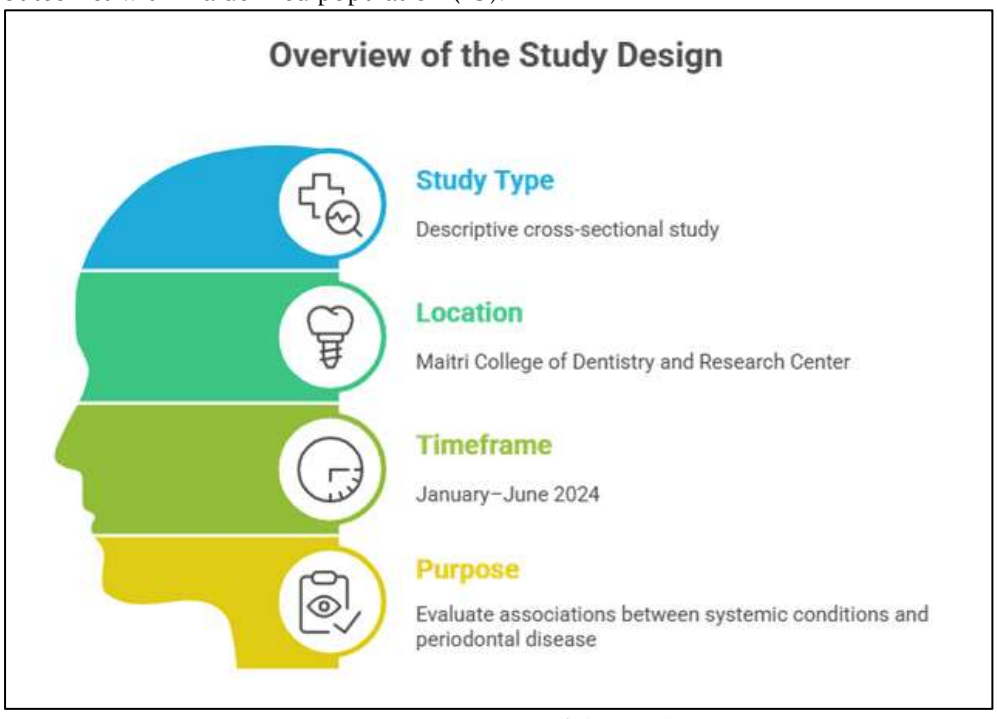

Figure 1 Overview of the Study Design

2.2 Ethical Approval

Prior to initiation, the study protocol was submitted to the Institutional Ethical Committee of Maitri College of Dentistry and Research Center, Anjora, Durg. Ethical clearance was obtained following the Helsinki Declaration of 1975, revised in 2013, to protect participants' rights and welfare (16). Written informed consent was collected from all participants after a detailed explanation of the study procedure.

2.3 Sample Size Estimation

The required sample size was calculated using an effect size (d = 0.6) from a previous study on Clinical Attachment Level (CAL) and Alveolar Bone Loss (ABL) in similar groups. With a 95% confidence interval

and 80% statistical power, the estimated sample size was 90 participants, with 45 in each group. In the end, 97 women who met the criteria were recruited, including 40 pre-menopausal and 57 post-menopausal women. Estimating sample size in cross-sectional dental studies helps achieve proper statistical accuracy and decreases type II errors. (17).

2.4 Inclusion and Exclusion Criteria

Inclusion criteria:

- Pre-menopausal women (20–45 years) and post-menopausal women (45–60 years).
- Presence of ≥7 natural teeth.
- Probing pocket depth (PPD) >4 mm and <6 mm.
- Clinical attachment loss (CAL) between 2–5 mm.

Exclusion criteria:

- Antibiotic therapy within the past 6 months.
- Current or previous history of hormone replacement therapy (HRT) or Vitamin D supplementation.
- Bone malignancies.
- Use of contrast agents or participation in nuclear medicine studies within 7 days of bone mineral density (BMD) assessment.

The inclusion and exclusion criteria were framed to eliminate potential confounding factors that could influence systemic bone health and periodontal status (18).

2.5 Data Collection and Study Tools

All participants underwent:

1. Clinical examination with a WHO CPI probe and a Williams probe to check PPD and CAL.
2. Radiographic analysis with intraoral periapical radiographs (IOPA) and a Dentech X-ray grid to measure alveolar crestal bone loss in millimeters.
3. Bone Mineral Density (BMD) assessment using the SONOST-2000 portable ultrasound bone densitometer. This device measured T-score, Z-score, Speed of Sound (SOS), and Broadband Ultrasound Attenuation (BUA). Ultrasound-based densitometry is a dependable, radiation-free method for checking peripheral BMD. It works well for large-scale screening and comparative studies. (19).

2.6 Study Procedure

Participants were divided into two groups based on their menopausal status: pre-menopausal and post-menopausal. A calibrated periodontist conducted standardized periodontal and radiographic examinations for each subject. A trained technician took the BMD measurements, which were then verified by an orthopedic consultant. On average, each evaluation took 15 to 20 minutes per participant.

2.7 Infection Control and Armamentarium

All instruments were sterilized following standard autoclave protocols, while chemical disinfection was performed using Korsolex solution. Disposable gloves, masks, and cotton rolls were used for each participant. The study employed diagnostic armamentarium such as plain mouth mirrors, CPI probes, tweezers, IOPA films, and the SONOST-2000 densitometer.

2.8 Statistical Analysis

Data were compiled using Microsoft Excel 2007 and analyzed with SPSS version 16.0. Descriptive statistics (mean ± SD) were used for continuous variables, and percentages for categorical variables. Between-group comparisons of BMD (T-scores) and alveolar bone loss were performed using independent Student's t-test. A p-value <0.05 was considered statistically significant. Independent t-tests are robust methods for comparing mean values between two groups in clinical dental research (20).

3. RESULTS

3.1 Demographic Data

The demographic characteristics of the study participants are summarized in Tables 1 and 2. The mean age of pre-menopausal women was 34.5 ± 6.6 years, while that of post-menopausal women was 48.6 ± 10.2 years.

Table 1. Descriptive statistics for pre-menopausal women (n = 40)

Variable	N	Minimum	Maximum	Mean	SD
Age (years)	40	24.00	49.00	34.50	6.69
T-score	40	-1.80	0.08	-1.23	0.44
Bone loss (mm)	40	2.00	13.60	5.28	2.73

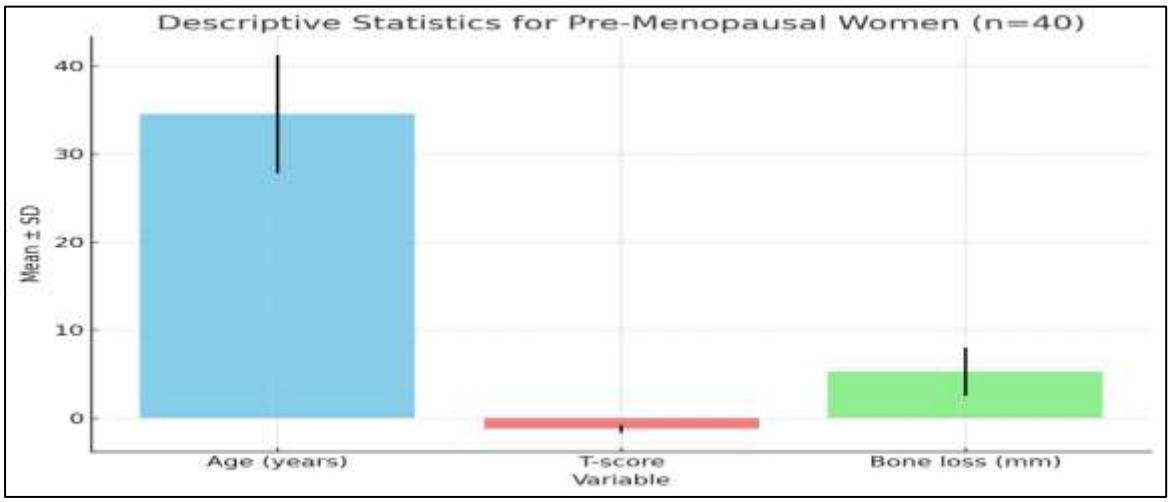

Figure 2 Descriptive Statistics for Pre-Menopausal Women (n=40)

Table 2. Descriptive statistics for post-menopausal women (n = 57)

Variable	N	Minimum	Maximum	Mean	SD
Age (years)	57	25.00	75.00	48.67	10.27
T-score	57	-3.20	-0.70	-1.97	0.66
Bone loss (mm)	57	1.50	19.00	10.21	4.42

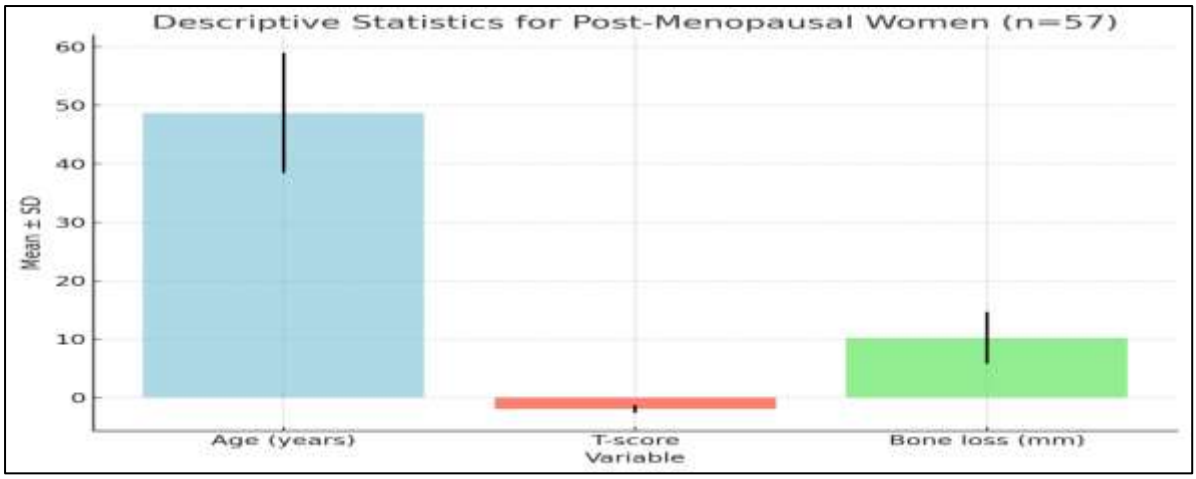

Figure 3 Descriptive Statistics for Post-Menopausal Women (n=57)

3.2 Bone Mineral Density

The comparison of bone mineral density (BMD) between pre- and post-menopausal women is summarized in Table 3. The mean T-score among pre-menopausal women was **-1.22 ± 0.43**, whereas in post-menopausal women it was **-1.97 ± 0.65**. The mean difference of **0.74** was found to be statistically significant (**p = 0.001**). This indicates that post-menopausal women exhibited a greater degree of bone density reduction compared to pre-menopausal women.

Table 3. Mean comparison of T-scores between pre- and post-menopausal women

Groups	N	Mean T-score	SD	Mean Difference	p-value
Pre-menopausal	40	-1.22	0.43		
Post-menopausal	57	-1.97	0.65	0.74	0.001 (s)

Statistical test: Independent Student's t-test; (p < 0.05 – significant).

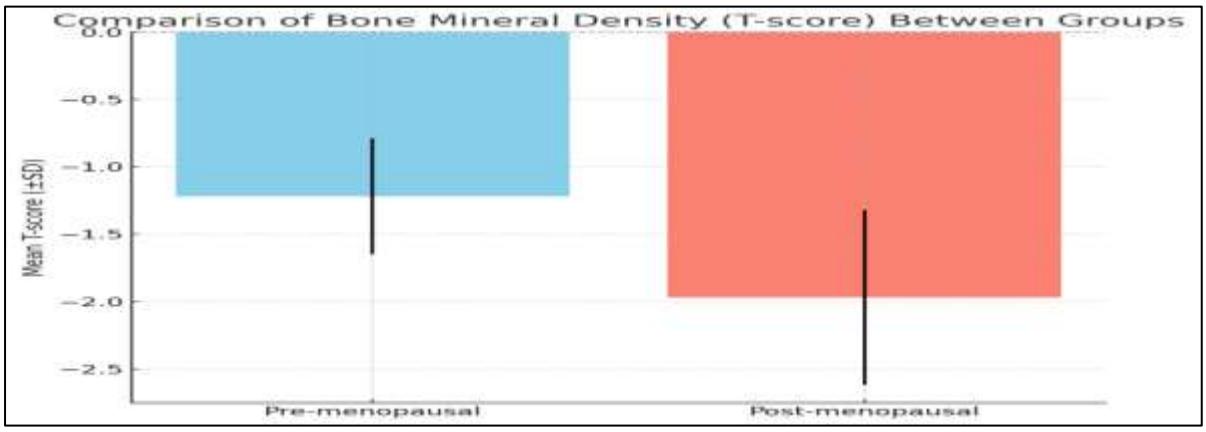

Figure 4 Comparison of Bone Mineral Density

3.3 Alveolar Bone Loss

The comparison of alveolar bone loss between pre- and post-menopausal women is presented in Table 4. The mean alveolar bone loss in pre-menopausal women was **5.28 ± 2.73 mm**, while in post-menopausal women it was significantly higher at **10.21 ± 4.42 mm**. The mean difference of **-4.92 mm** was found to be statistically significant (**p = 0.001**), indicating that post-menopausal women experience greater periodontal bone resorption compared to pre-menopausal women.

Table 4. Mean comparison of alveolar bone loss between groups

Groups	N	Mean Bone Loss (mm)	SD	Mean Difference	p-value
Pre-menopausal	40	5.28	2.73		
Post-menopausal	57	10.21	4.42	-4.92	0.001 (s)

Statistical test: Independent Student's t-test; (p < 0.05 – significant).

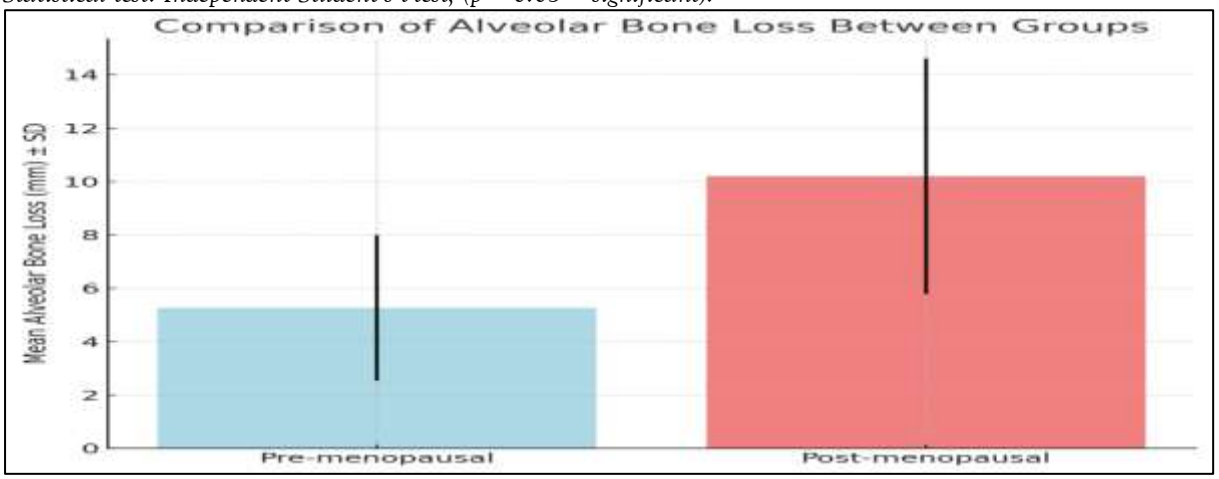

Figure 5 Comparison of Alveolar Bone Loss Between Groups

4. DISCUSSION

The present study highlights a significant correlation between systemic osteoporosis and periodontal bone loss, emphasizing the interdependence of skeletal health and oral health. Reduced bone mineral density, as observed in the post-menopausal women of this study, was strongly associated with increased alveolar bone resorption. This finding reinforces the concept that osteoporosis, a systemic skeletal disease characterized by decreased bone density and structural deterioration, can have direct implications on periodontal integrity. Since alveolar bone serves as the foundation for tooth stability, systemic reductions in mineral density leave it more vulnerable to inflammatory insults, resulting in greater loss of periodontal support (21). A key factor in this relationship is the role of estrogen deficiency in post-menopausal women. Estrogen controls bone turnover by reducing osteoclast activity and supporting bone formation. After menopause, estrogen levels drop significantly, leading to an imbalance between bone resorption and bone deposition. This change in hormones not only makes the skeletal system weaker but also increases the risk of periodontal bone loss. In this study, post-menopausal women demonstrated significantly lower T-scores and higher alveolar bone loss than their pre-menopausal counterparts, suggesting that estrogen

deficiency accelerates the destructive interaction between microbial plaque and host immune response within the periodontium (22). The findings are consistent with earlier studies. Tezal et al. (2000) reported that post-menopausal women with osteoporosis exhibited significantly higher rates of alveolar bone resorption compared to healthy controls, establishing a direct link between systemic BMD and periodontal disease severity (23). Similarly, Wactawski-Wende et al. (2005) observed that reduced BMD was correlated with decreased alveolar crestal height in post-menopausal women, reinforcing the association between systemic osteoporosis and oral ore loss (24). Suresh et al. (2010) extended these observations in an Indian population, confirming that women with osteoporosis were more likely to develop moderate to severe periodontitis (25). More recently, Chethana et al. (2021) showed that women with osteoporosis had much higher levels of periodontal bone loss compared to those with normal BMD. This supports our findings. Together, these results strengthen the evidence for a link between osteoporosis and periodontitis. The biological mechanism behind this connection is largely driven by pro-inflammatory cytokines and pathways that regulate bone. Periodontitis leads to an increase in cytokines like interleukin-1, interleukin-6, and tumor necrosis factor-alpha, which encourage the differentiation and activation of osteoclasts. (27). These cytokines stimulate the receptor activator of nuclear factor kappa-B ligand (RANKL), a critical regulator of osteoclastogenesis. Elevated RANKL levels enhance osteoclast-mediated resorption of alveolar bone in periodontal disease. Estrogen deficiency further amplifies these pathways by increasing cytokine expression, thereby intensifying the destructive effects of chronic inflammation on both systemic and alveolar bone (28). This synergistic action provides a strong biological rationale for the observed clinical association between osteoporosis and periodontitis.

However, conflicting evidence has been reported in the literature. Some studies have failed to demonstrate a strong relationship between systemic bone mineral density and periodontal destruction. Variability in study design, diagnostic criteria, population differences, and measurement methods may account for such discrepancies. For instance, the use of dual-energy X-ray absorptiometry (DEXA) versus ultrasound-based BMD assessment, as well as differences in periodontal disease classification, can influence outcomes. Moreover, other systemic or lifestyle factors such as nutrition, calcium intake, smoking, and socioeconomic status may act as confounding variables, making it difficult to isolate the direct effect of osteoporosis on periodontal health (29).From a clinical perspective, the present findings underscore the importance of early screening of post-menopausal women for both osteoporosis and periodontal disease. Since both conditions share common inflammatory mechanisms and risk factors, integrated screening programs could facilitate earlier diagnosis and management. Dentists and physicians should adopt a collaborative approach, recognizing periodontitis not merely as an oral disease but also as a potential indicator of underlying systemic skeletal fragility. Timely interventions such as periodontal therapy, lifestyle modification, and systemic bone health management could help reduce the burden of tooth loss and skeletal fractures in this vulnerable population (30). Despite the valuable insights gained, this study has certain limitations. Its cross-sectional design prevents the establishment of a causal relationship between osteoporosis and periodontal bone loss. A longitudinal follow-up would be necessary to determine whether reduced BMD directly predicts progression of periodontitis. Additionally, while clinical and radiographic assessments were employed, the study did not include biomarker analysis of inflammatory mediators such as interleukin-1, interleukin-6, and TNF-α, which could have provided deeper mechanistic evidence of the cytokine-mediated link between systemic and alveolar bone loss. Future research integrating biochemical markers, larger sample sizes, and longitudinal designs will be critical to further elucidate this multifactorial relationship (31).

5. CONCLUSION:

The findings of the present study demonstrate that post-menopausal women with chronic periodontitis exhibit significantly lower bone mineral density and greater alveolar bone loss compared to their pre-menopausal counterparts. This confirms that systemic osteoporosis and periodontal bone resorption are interlinked conditions, with menopause acting as a critical factor that accelerates both systemic and oral skeletal deterioration. Estrogen deficiency, through its influence on bone turnover and inflammatory pathways, appears to play a pivotal role in enhancing periodontal vulnerability during the post-menopausal phase. From a biological perspective, the study supports the hypothesis that cytokine-mediated osteoclast activation, particularly via IL-6, TNF-α, and RANKL signaling pathways, underlies the shared mechanism of bone loss in osteoporosis and periodontitis. These molecular interactions not

only explain the observed clinical outcomes but also highlight the potential for targeted interventions that address both systemic and oral bone health simultaneously.

Clinically, the results underscore the importance of early screening and integrated management strategies for post-menopausal women. Periodontal assessment should be considered a valuable adjunctive tool in identifying women at risk of osteoporosis, while bone density evaluation may provide insights into their susceptibility to advanced periodontitis. Preventive measures such as lifestyle modification, nutritional supplementation, and timely periodontal therapy can help mitigate the dual burden of tooth loss and skeletal fractures. Although the cross-sectional nature of this study limits causal inference, the evidence presented reinforces the need for multidisciplinary collaboration between dental professionals and medical practitioners. Future longitudinal and biomarker-based studies are recommended to further clarify the mechanistic links and to establish evidence-based guidelines for the joint management of osteoporosis and periodontitis.

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