Edelweiss Applied Science and Technology
ISSN: 2576-8484
Vol. 9, No. 10, 791-800
2025
Publisher: Learning Gate
DOI: 10.55214/2576-8484.v9i10.10534

The osteoimmunology of orthodontic tooth movement: Clinical correlates of inflammatory-mediated crestal bone resorption

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Abstract: While orthodontic treatment is known to stimulate bone remodeling, its effects on the delicate crestal bone between teeth have raised clinical concerns. To explore this, this study examined records from 556 patients who underwent orthodontic care at Riyadh Elm University, Riyadh, Saudi Arabia. The findings showed measurable bone loss after treatment, with notable differences based on the methods used and the patient's age. Mini-screws, often employed for anchorage, correlated with the most significant bone reduction, likely from localized trauma and inflammation during placement. In contrast, clear aligners had the gentlest impact, preserving bone levels more effectively. Older patients (35+ years) faced greater bone loss than younger ones (13–18 years), reflecting natural age-related changes in bone density and healing. Interestingly, gender played no detectable role in these outcomes. These insights stress the value of customized treatment plans, especially for older adults or those with existing periodontal issues. Regular monitoring of bone health before, during, and after orthodontics is crucial to minimizing risks. Approaches like gentler force systems and precise mini-screw protocols could help safeguard bone integrity. Close teamwork between orthodontists and periodontists may further optimize results. The study's retrospective nature and single-center data mean broader conclusions require caution. Future research tracking patients over time could clarify long-term effects, while investigating systemic factors (e.g., osteoporosis) might uncover additional risks. Ultimately, the study underscores a key balance in orthodontics: achieving straighter teeth without compromising the foundation that supports them.

Keywords: *Age-related bone remodeling, Clear aligner biomechanics, Interproximal crestal bone, Mini-screw complications, Orthodontic bone loss.*

1. Introduction

Mechanical loads during orthodontic treatment stimulate cells within the bone tissue, leading to bone resorption on the pressure side and bone deposition on the tension side [1, 2]. The goal of surgically facilitated tooth movement is to stimulate the production of cytokines and other inflammatory markers, which will lead to increased differentiation and maturation of osteoclasts, thereby initiating bone resorption [3, 4]. The application of orthodontic forces initiates a cascade of biological events, including the release of cytokines and growth factors, that mediate bone remodeling and tooth movement [5]. Moreover, the immune system's involvement in orthodontic tooth movement highlights the complexity of the process, where immune cells play a crucial role [6]. Specifically, the interplay between immune-related cytokines and bone remodeling during orthodontic treatment underscores the need for a comprehensive understanding of the underlying mechanisms [6, 7]. Modulation of the inflammatory response has been investigated as a means to accelerate tooth movement and enhance treatment outcomes [8]. The use of mini-screws as temporary anchorage devices has revolutionized orthodontic treatment, providing stable points for force application and enabling complex tooth movements with greater

History: Received: 18 July 2025; Revised: 29 August 2025; Accepted: 4 September 2025; Published: 13 October 2025
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precision. However, the placement of mini-screws can be associated with risks, including damage to adjacent teeth, nerve injury, and inflammation [9-12]. Individual variability in response to orthodontic treatment is increasingly recognized, with genetic and environmental factors influencing the rate and extent of tooth movement [13]. Understanding the genetic factors that influence tooth movement can lead to personalized treatment approaches that optimize outcomes and minimize adverse effects [13-16]. Analyzing the expression of various cytokines revealed significant increases in inflammatory markers, suggesting a potential mechanism for accelerating tooth movement through controlled inflammation [13, 17]. Orthodontic tooth movement involves a complex interplay of mechanical, chemical, and cellular events within the tissues surrounding the teeth [18]. Inflammation plays a crucial role in orthodontic tooth movement, influencing the rate of bone remodeling and the overall treatment outcome [8]. Chemokines, such as monocyte chemoattractant protein-1, are upregulated during orthodontic tooth movement, indicating their involvement in the recruitment of immune cells and bone remodeling [19, 20]. Macrophages also play a crucial role in inflammatory-mediated bone loss [21]. The application of mechanical stress during orthodontic treatment induces the release of chemokines from periodontal ligament cells [22]. Additionally, age-related changes in bone density and metabolism can influence the response to orthodontic treatment, with older patients potentially experiencing slower tooth movement and an increased risk of periodontal complications.

Shorter treatment times benefit both children and adult patients, limiting discomfort and reducing the prevalence of iatrogenic adverse side effects [23]. The duration of orthodontic treatment is an important consideration for patients, with longer treatment times associated with increased risks of complications such as caries, gingivitis, root resorption, and decreased patient compliance [24]. The field of accelerated orthodontics has emerged as a promising approach to reduce treatment time and improve patient outcomes [25]. With the increasing number of adults seeking orthodontic treatment, there is a growing interest in methods to accelerate tooth movement and minimize the overall treatment duration [26].

Piezocision, a minimally invasive surgical technique, has emerged as a promising method for accelerating orthodontic tooth movement. By combining microincisions with localized piezoelectric bone surgery, piezocision aims to reduce trauma and enhance the healing response [27-29]. Corticotomies have garnered increasing interest as an adjunct to orthodontic treatment, driven by a deeper understanding of their effects and more robust evidence-based investigations [30]. The primary rationale for corticotomy-assisted orthodontics is to accelerate tooth movement [31]. Photobiomodulation has demonstrated potential for accelerating tooth movement and reducing pain. By stimulating cellular activity and promoting tissue regeneration, photobiomodulation can enhance the efficiency of orthodontic treatment. The present study investigated the impact of orthodontic treatment on interproximal crest bone levels, emphasizing the importance of a multidisciplinary approach in treatment planning to optimize both functional and aesthetic patient outcomes.

2. Materials and Methods

2.1. Study Design

This retrospective cross-sectional study was conducted to analyze the changes in interproximal crest bone levels following orthodontic treatment, using data from patients treated at Riyadh Elm University, Riyadh, Saudi Arabia. The study was conducted in accordance with the ethical standards of the institutional review board at Riyadh Elm University.

2.2. Study Population

Participants included 556 patients who had completed orthodontic treatment at the university. Eligible participants met the inclusion and exclusion criteria as elaborated in Figure 1.

Edelweiss Applied Science and Technology
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2.3. Data Collection and Classification

Demographic and clinical data were collected, including age, gender, treatment modality, and nationality. Radiographic analysis involved measuring bone levels mesially and distally for Ramfjord teeth (RT) (#16, #21, and #36). The severity of bone level changes was classified as mild, moderate, or severe, based on the percentage change from baseline to post-treatment measurements. According to Ertürk et al. [32], the radiological examinations were classified into 4 groups as shown in Figure 2.

2.4. Radiographic Analysis

Standardized panoramic radiographs were used to assess bone levels. The evaluation of bone loss was conducted visually by examiners, who analyzed the radiographs to measure the distance from the cemento-enamel junction (CEJ) to the alveolar bone crest. The assessment relied on the clinical expertise of two independent examiners, who performed the measurements without the aid of digital software. Discrepancies between the examiners were resolved through discussion to ensure consistency and reliability.

2.5. Statistical Analysis

Data analysis was performed using SPSS software, IBM, USA (version 26.0). Continuous variables, such as bone level changes, were expressed as means ± standard deviations (SD). The Wilcoxon signed-rank test was used to compare pre- and post-treatment bone levels. Differences in bone loss severity across different treatment modalities, age groups, and genders were analyzed using independent samples t-tests and one-way ANOVA, with a significance level set at $\rho < 0.05$.

3. Results

A total of 556 patients met the inclusion criteria and were analyzed in this study. Figure 3 provides a summary of the participants' baseline demographic and treatment characteristics. The average age of the participants was 23.26 years (± 4.39), with females representing a larger proportion of the sample (63.7%, n = 354) compared to males (36.3%, n = 202) (Fig. 3a). The majority of the participants were Saudi nationals (93.7%) (Fig. 3b), and conventional fixed orthodontic treatment was the most common intervention, performed in 68.9% of cases. Other treatment modalities included clear aligners (Invisalign, 1.6%), mini-screws (0.5%), exposure of impacted canines (0.7%), and frenectomy (0.2%), as shown in Figure 3c.

Changes in interproximal alveolar bone levels before and after orthodontic treatment are presented in Table 1. Across all examined tooth sites, there was a statistically significant increase in bone loss following treatment ($\rho < 0.001$). For instance, mean mesial bone loss adjacent to tooth #16 increased from 0.16 mm (± 0.39) pre-treatment to 0.27 mm (± 0.53) post-treatment. Similarly, distal bone loss for the same tooth rose from 0.12 mm (± 0.37) to 0.26 mm (± 0.51). Comparable patterns of significant bone loss were observed at other teeth, including teeth #21 and #36.

Table 2 compares the extent of bone loss across different treatment modalities. While conventional orthodontics was associated with moderate bone loss, cases involving mini-screws exhibited slightly higher levels. For example, mesial bone loss around tooth #16 averaged 0.145 mm (± 0.195) with conventional orthodontics and 0.365 mm (± 1.721) in the mini-screw group. However, these differences did not consistently reach statistical significance at all sites. Clear aligners demonstrated the least amount of bone loss among the modalities studied.

Gender-based comparisons are summarized in Table III. No significant differences in interproximal bone loss were found between male and female participants. For example, distal bone loss adjacent to tooth #21 averaged 0.23 mm (± 0.49) in males and 0.27 mm (± 0.53) in females ($\rho = 0.48$). A similar lack of statistical significance was observed across other sites.

Age, however, was found to have a notable association with bone level changes (Table 4). Younger patients (aged 13–18 years) exhibited less bone loss compared to older age groups. For example, distal

Edelweiss Applied Science and Technology
ISSN: 2576-8484
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bone loss at tooth #36 was 0.17 mm in the youngest group but increased to 0.56 mm in patients aged 35 years and above ($\rho < 0.001$). This trend of increasing bone loss with age was consistent across all measured teeth.

Finally, the impact of secondary treatment modalities on bone loss is detailed in Table 5. Patients who underwent rapid palatal expansion exhibited the least bone loss (0.05 mm ± 0.14), whereas those treated with mini-screws experienced the highest bone loss (0.37 mm ± 1.72). Exposure of impacted canines and frenectomy were associated with moderate bone loss levels.

4. Discussion

The study's findings indicate that orthodontic treatment leads to a statistically significant increase in interproximal crestal bone loss, which corroborates evidence from previous investigations [33]. These changes are more pronounced at specific tooth sites, suggesting that anatomical and biomechanical factors may influence susceptibility to bone remodeling during orthodontic tooth movement. The increased bone loss observed post-orthodontic treatment may be attributed to several factors, including inflammation, altered periodontal ligament stress, and changes in oral hygiene practices during treatment [33]. Furthermore, the use of cone-beam computed tomography to evaluate alveolar bone loss, bone density, and bone thickness showed significant differences between healthy and periodontitis groups [34]. This highlights the need for personalized treatment plans tailored to minimize adverse periodontal outcomes.

The impact of different orthodontic modalities on crestal bone levels reveals clinically relevant variations. Notably, mini-screws were associated with the highest bone loss, while clear aligners exhibited the least impact. The increased bone loss associated with mini-screws might be attributed to localized inflammation and mechanical trauma during placement and removal, which could stimulate osteoclastic activity and bone resorption [35]. In contrast, clear aligners, known for delivering lighter and more controlled forces, appear to have a gentler impact on the periodontium, resulting in less bone remodeling and reduced bone loss. It is crucial to consider that while mini-screws provide effective anchorage control [36], their use necessitates careful monitoring of the adjacent periodontal tissues. The success rate of orthodontic microimplants has been shown to significantly increase with higher cancellous and total bone densities [37]. This underscores the importance of detailed pre-treatment assessments to identify patients at risk for greater bone loss, especially when employing mini-screws or other invasive orthodontic techniques.

The present study's identification of age as a significant predictor of bone loss aligns with established knowledge regarding age-related bone physiology. Older patients (≥35 years) experienced greater bone loss compared to younger individuals (13–18 years), which is consistent with the understanding that bone remodeling dynamics change with age, leading to a net increase in bone resorption [38]. This age-related bone loss may be exacerbated by orthodontic forces, making older individuals more vulnerable to periodontal complications during and after treatment. Age-related systemic conditions, such as osteoporosis, could further increase the risk of oral bone loss [39]. Therefore, a comprehensive evaluation of bone health and careful consideration of treatment mechanics are particularly important in older orthodontic patients.

Gender was not found to be a significant factor in interproximal bone loss following orthodontic treatment, suggesting that the observed bone changes are primarily influenced by mechanical and inflammatory responses to orthodontic forces rather than hormonal or gender-specific factors. This finding contrasts with some studies that report gender-related differences in bone density and periodontal health, which could be attributed to variations in study populations, orthodontic techniques, or measurement methodologies. Although females generally have less medullary bone quantity and connectivity than male patients, the effect of orthodontic treatment on bone loss appears to be equitable across genders [40].

Clinical implications of these findings underscore the importance of personalized treatment planning in orthodontics. Comprehensive periodontal assessments should be conducted before, during, and after orthodontic treatment to monitor bone levels and identify early signs of periodontal complications.

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Orthodontic treatment should be individualized to minimize potential adverse effects on the periodontium, especially in older patients or those with pre-existing periodontal conditions. Collaboration between orthodontists and periodontists is essential to address complex cases and ensure optimal periodontal health. Moreover, employing techniques that minimize trauma to the periodontal tissues, such as light-force orthodontics and careful management of mini-screws, may help reduce the risk of iatrogenic bone loss. The interplay between pathogenic plaque and the host response significantly influences oral bone loss. Oral hygiene instructions should be reinforced throughout orthodontic treatment, and adjunctive measures such as antimicrobial mouth rinses may be considered to reduce inflammation and prevent further bone loss.

The study has some limitations that should be considered when interpreting the results. The study's retrospective cross-sectional design limits the ability to establish causal relationships between orthodontic treatment and bone loss. Longitudinal studies are needed to track bone level changes over time and to evaluate the long-term impact of different treatment modalities. Additionally, the sample population was limited to patients treated at a single institution, which may limit the generalizability of the findings. Future research should explore the impact of systemic factors, such as osteoporosis, on orthodontic-related bone loss and investigate the effectiveness of preventive strategies in minimizing adverse periodontal outcomes [38, 41].

Bone loss in the oral cavity can arise from several factors, including infections, systemic conditions, and local alterations in the host response. Periodontitis, characterized by the resorption of alveolar bone and soft tissue attachment loss, can lead to continued alveolar bone loss, tooth mobility, abscesses, and ultimately, tooth loss [42]. Inflammation plays a pivotal role in the pathogenesis of periodontitis, where the interaction between pathogenic plaque and the host immune response leads to the destruction of periodontal tissues [43]. Inflammation, hereditary factors, hormones, aging and lifestyle impact bone loss [44]. In managing periodontal disease and other conditions characterized by bone loss, approaches that inhibit bone resorption and promote alveolar bone regeneration are particularly valuable [44]. Therapies such as medication, exercise, and anti-inflammatory treatments all help combat bone loss [44].

5. Conclusion

Orthodontic treatment is associated with statistically significant increases in interproximal bone loss. Treatment modality and patient age significantly influence the magnitude of bone loss. Personalized treatment planning, considering patient-specific risk factors and employing techniques to minimize periodontal trauma, is essential to optimize outcomes and minimize adverse effects. Further prospective studies are warranted to better understand the long-term impact of orthodontic treatment on periodontal health and to evaluate the effectiveness of preventive strategies in minimizing bone loss.

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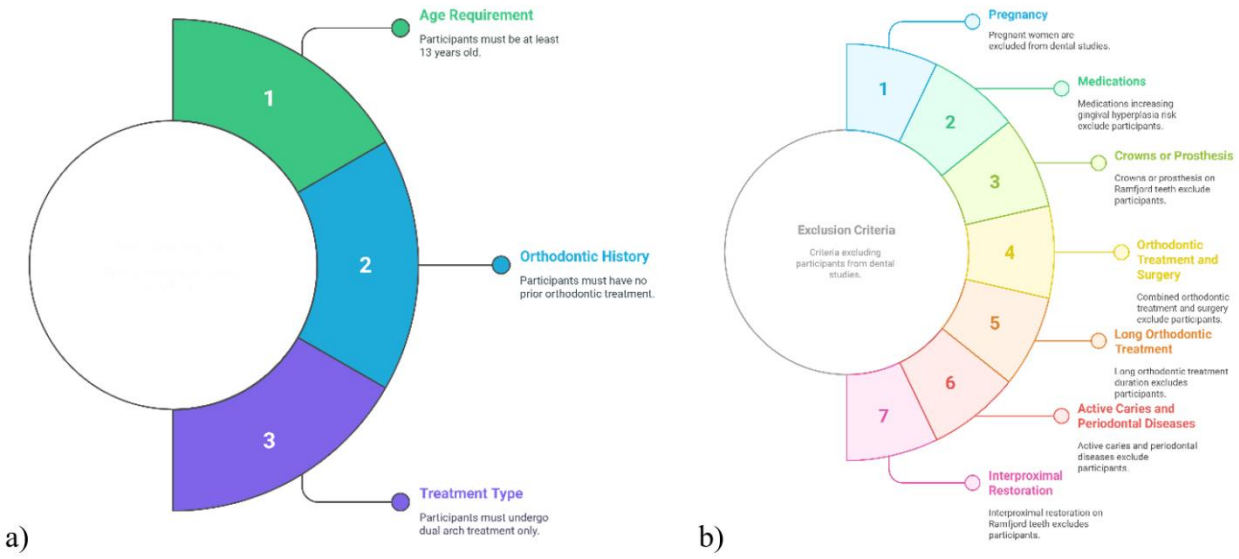

Figure 1.
Inclusion and exclusion criteria of the participants.

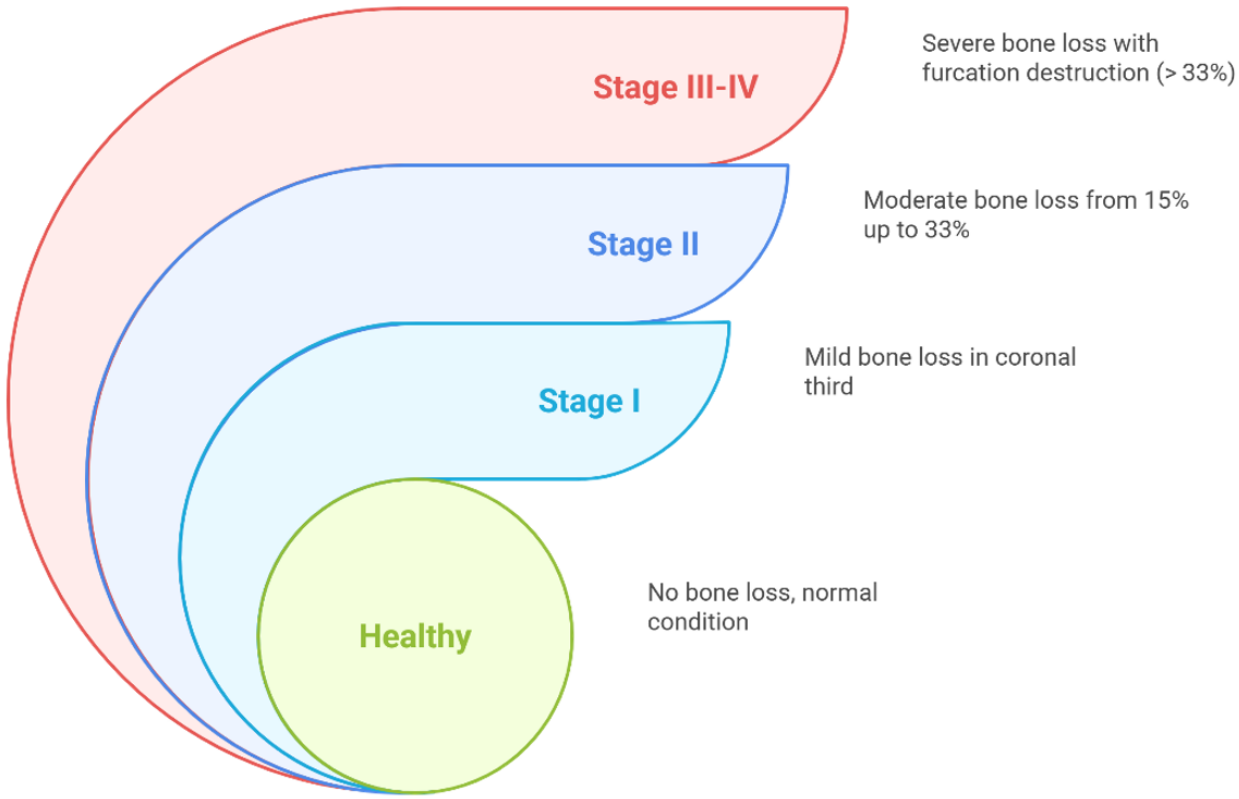

Figure 2.
Periodontal disease radiographic stages classification.
Source: Ertürk et al. [32].

Edelweiss Applied Science and Technology
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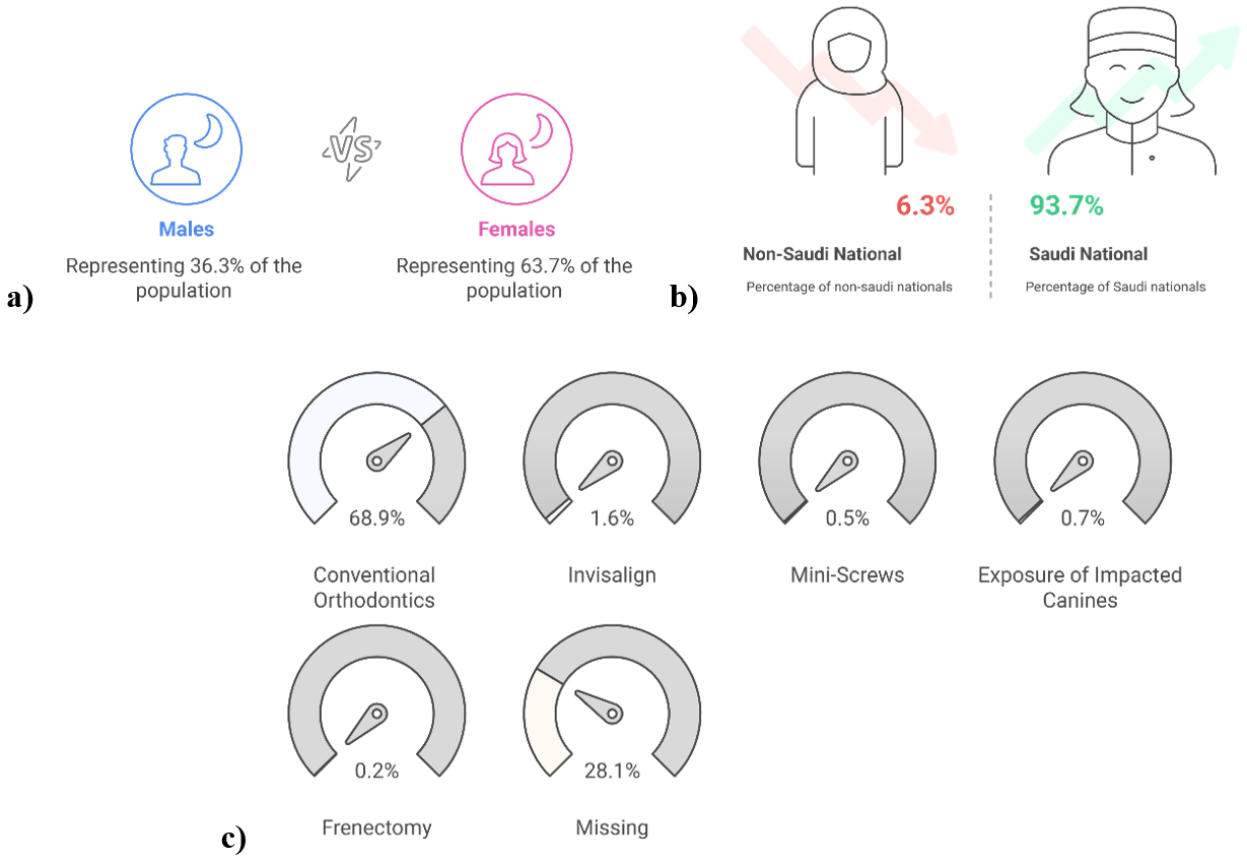

Figure 3.
Baseline Demographic and Treatment Characteristics of Study Participants. (a) gender distribution of the participants; (b) Saudi and non-Saudi individuals in the study; & type of therapeutic approach for orthodontic treatment mentioned in the files.

Table 1.
Changes in interproximal bone levels before and after orthodontic treatment.

Tooth (FDI Numbering System)	Time		ρ-Value
	Before Treatment Mean (mm) ±SD	After Treatment Mean (mm) ±SD	
#16 Mesial	0.16 ± 0.39	0.27 ± 0.53	**< 0.001**
#16 Distal	0.12 ± 0.37	0.26 ± 0.51	**< 0.001**
#21 Mesial	0.12 ± 0.33	0.22 ±0.48	**< 0.001**
#21 Distal	0.11 ± 0.31	0.26 ±0.52	**< 0.001**
#36 Mesial	0.09 ± 0.28	0.17 ± 0.44	**< 0.001**

Note: * Wilcoxon signed-rank tests for statistical analysis.

Table 2.
Bone loss across different orthodontic treatment modalities.

Mean Bone Loss ±SD (mm) by various treatment modalities					
Tooth (FDI Numbering System)	Conventional Orthodontics	Mini-Screws	Exposure of Impacted Canines	Clear Aligners (Invisalign)	ρ Value
#16 Mesial	0.15 ±0.20	0.37 ±1.72	0.26 ±0.51	0.10 ±0.15	0.058
#16 Distal	0.11 ± 0.16	0.24 ± 0.48	0.15 ± 0.34	0.09 ±0.12	0.034
#21 Mesial	0.12 ± 0.16	0.32 ± 1.63	0.23 ± 0.68	0.09 ± 0.15	0.021
#21 Distal	0.10 ± 0.14	0.30 ± 1.59	0.210 ± 0.61	0.07 ± 0.11	0.012
#36 Mesial	0.08 ± 0.11	0.27 ± 1.53	0.20 ± 0.71	0.06 ± 0.10	0.003

Edelweiss Applied Science and Technology
ISSN: 2576-8484
Vol. 9, No. 10: 791-800, 2025
DOI: 10.55214/2576-8484.v9i10.10534

Table 3.
Gender-based comparisons of bone loss using independent samples t-tests.

Tooth (FDI Numbering System)	Before Treatment Bone Height Loss (mm± SD)			After Treatment Bone Height Loss (mm± SD)		
	Male	Female	ρ value	Male	Female	ρ value
# 16 Mesial	0.15 ± 0.35	0.16 ± 0.40	0.95	0.26 ± 0.51	0.27± 0.54	0.88
# 16 Distal	0.09± 0.34	0.14± 0.38	0.42	0.28± 0.55	0.25± 0.50	0.76
# 21 Mesial	0.11± 0.31	0.13± 0.34	0.65	0.22± 0.45	0.23± 0.49	0.90
# 21 Distal	0.08± 0.27	0.12± 0.33	0.33	0.23± 0.49	0.27± 0.53	0.48
# 36 Mesial	0.06± 0.24	0.08± 0.27	0.68	0.19± 0.46	0.15± 0.43	0.61

Table 4.
Age-related bone loss before and after orthodontic treatment.

Tooth (FDI Numbering System)	Before Treatment Bone Height Loss (mm± SD)				After Treatment Bone Height Loss (mm± SD)			
	13–18	19–25	≥35	ρ Value	13–18	19–25	≥35	ρ Value
# 16 Mesial	0.09 ±0.28	0.16 ± 0.36	0.33± 0.49	**< 0.001**	0.17± 0.44	0.27±0.53	0.5± 0.71	**< 0.001**
# 16 Distal	0.08± 0.26	0.12± 0.37	0.29± 0.44	**< 0.001**	0.16± 0.44	0.26±0.51	0.56±0.69	**< 0.001**
# 21 Mesial	0.11± 0.32	0.12± 0.33	0.25± 0.42	**< 0.001**	0.22± 0.48	0.25±0.52	0.5± 0.71	**< 0.001**
# 36 Mesial	0.08± 0.28	0.09± 0.29	0.26± 0.53	**< 0.001**	0.17± 0.44	0.18±0.46	0.56±0.71	**< 0.001**

Table 5.
Effects of secondary treatment modalities on bone loss.

Tooth (FDI Numbering System)	Effect of Treatment Modality Bone Loss Mean (mm) ±SD				
	Rapid Palatal Expansion	Exposure of Impacted Canines	Mini-Screws	Frenectomy	ρ Value
#16 Mesial	0.05 ± 0.14	0.26 ± 0.51	0.37± 1.72	0.27± 0.41	< 0.001
#16 Distal	0.09± 0.19	0.15± 0.34	0.24± 0.48	0.29± 0.51	< 0.001
#21 Mesial	0.07± 0.14	0.21± 0.61	0.32± 1.63	0.27± 0.48	< 0.001
#21 Distal	0.09± 0.20	0.21± 0.61	0.3± 1.59	0.29± 0.51	< 0.001
#36 Mesial	0.06± 0.1	0.2± 0.71	0.27± 1.53	0.27± 0.41	< 0.001

Transparency:

The authors confirm that the manuscript is an honest, accurate, and transparent account of the study; that no vital features of the study have been omitted; and that any discrepancies from the study as planned have been explained. This study followed all ethical practices during writing.

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ISSN: 2576-8484
Vol. 9, No. 10: 791-800, 2025
DOI: 10.55214/2576-8484.v9i10.10534