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Impact Of Baseline Alveolar Bone Thickness on Micro-Osteoperforation Efficacy: A CBCT Study in Thin Vs. Thick Bone Biotypes

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Abstract:

Background: Micro-osteoperforations (MOPs) have emerged as a minimally invasive adjunct to accelerate orthodontic tooth movement. However, the influence of alveolar bone biotype—particularly thin versus thick buccal bone—on the efficacy and safety of MOPs remains underexplored.
Objective: This study aimed to quantify and compare the outcomes of MOPs in patients with thin and thick alveolar bone phenotypes using cone-beam computed tomography (CBCT).
Methods: In this split-mouth randomized controlled trial (RCT), 38 patients undergoing maxillary canine retraction were stratified into thin (buccal bone ≤1.0 mm; n = 19) and thick (≥1.5 mm; n = 19) alveolar bone biotypes. On the experimental side, MOPs were performed adjacent to the canine prior to retraction, while the contralateral side served as control. CBCT scans were obtained at baseline and after 3 months to assess tooth movement rate, alveolar bone thickness, vertical bone loss, and bone density (measured in Hounsfield Units, HU). Statistical comparisons were performed using paired and unpaired t-tests, and Pearson correlation was used to explore associations ($\alpha = 0.05$).
Results: In thin biotypes, MOPs resulted in a 58% increase in tooth movement rate compared to control (1.42 ± 0.31 mm/month vs. 0.90 ± 0.28 mm/month; $p < 0.001$), whereas thick biotypes showed a 22% acceleration (1.10 ± 0.25 mm/month vs. 0.90 ± 0.30 mm/month; $p = 0.07$). Palatal vertical bone loss was significantly higher in MOP-treated thin biotypes (0.46 ± 0.12 mm) versus control (0.28 ± 0.10 mm; $p = 0.02$). Additionally, bone density reduction at MOP sites was greater in thin biotypes (ΔHU: -312 ± 45) than thick ones (ΔHU: -155 ± 32; $p < 0.01$). An inverse correlation was observed between baseline buccal bone thickness and rate of tooth movement following MOPs ($r = -0.72$; $p < 0.001$).
Conclusion: Mops are more effective in accelerating tooth movement in patients with thin alveolar bone; however, this benefit is accompanied by greater palatal vertical bone loss and density reduction. CBCT-based bone thickness assessment should be incorporated into pretreatment protocols to individualize MOP indication and mitigate risk.

Introduction

Orthodontic tooth movement (OTM), which is the process of shifting teeth into their desired positions, fundamentally relies on the intricate and dynamic remodeling of the alveolar bone, which occurs in response to the mechanical forces exerted during orthodontic treatment. (Andreasen, 1973) This complex biological process is orchestrated through a meticulously regulated sequence that involves both bone resorption, where old bone is broken down, and bone deposition, where new bone is formed, a sequence primarily governed by the periodontal ligament (PDL), which plays a crucial role as a mechanoresponsive interface between the tooth and the surrounding alveolar bone. (Fongsamootr & Suttakul, 2015) However, the inherently slow nature of the bone remodeling process contributes significantly to prolonged treatment durations, which often leads to challenges such as patient non-compliance and increased risks of various complications, including root resorption, periodontal breakdown, and the loss of mineral content in the bone, known as decalcification. (Morris, 1980) Consequently, the acceleration of orthodontic tooth movement (OTM) without compromising the health of the periodontal tissues has emerged as a pivotal objective in the field of contemporary orthodontics, as practitioners seek to enhance treatment efficiency while maintaining patient safety. (Wilcko et al., 2008) Among the various innovative approaches that have been developed to expedite orthodontic treatment, micro-osteoperforations (MOPs) have garnered substantial attention due to their minimally invasive nature and their remarkable ability to stimulate localized bone remodeling in a targeted manner. MOPs, which are defined as small perforations created through the cortical bone, are typically accomplished without the need for flap elevation, and are specifically designed to induce a controlled inflammatory response that enhances the recruitment of osteoclasts and accelerates the overall bone turnover proces (Schmidt-Bleek et al., 2012)s. The underlying mechanism of this technique is thought to capitalize on the phenomenon known as the Regional Acceleratory Phenomenon (RAP), wherein surgical injury inflicted upon the bone transiently upregulates the remodeling cascade, thereby facilitating a faster and more efficient tooth movement that is desired during orthodontic treatment. (Wilcko et al., 2009)

Although MOPs, which are known as micro-osteoperforations, have clearly demonstrated their clinical efficacy in significantly reducing the overall treatment time required for orthodontic procedures and simultaneously enhancing the rate at which tooth movement occurs, it is important to note that the biological response that these MOPs elicit within the alveolar bone is not consistent or uniform across the entire area of the bone structure. (Sugimori et al., 2018) Several recent and highly relevant studies suggest that there are anatomical and histological differences that are specific to different sites within the jaw, which may indeed influence not only the extent but also the specific pattern of remodeling that is induced by the application of MOPs. (Callan et al., 2000) (Hjørting-Hansen et al., 1993) Furthermore, it is well-established that the anterior and posterior segments of both the maxilla and mandible exhibit significant differences in terms of factors such as cortical thickness, trabecular density, and vascularity, and these differences could very well translate into a differential responsiveness to the stimuli induced by MOPs. (Watzak et al., 2005) For example, it is observed that the posterior regions of the jaw, which are typically characterized by networks of denser trabecular bone and plates that are thicker in cortical composition, may display a distinctly different remodeling trajectory when compared to the relatively thinner and more vascularized bone found in the anterior region of the jaw. Despite the presence of these theoretical premises and the implications they carry, there exists a notable paucity of clinical evidence that directly compares the responses of the alveolar bone to MOPs specifically in the anterior versus the posterior segments within the same individual patient. (Zheng et al., 2014)The use of a split-mouth design in clinical studies, by effectively controlling for variations that occur between different individuals, presents an ideal methodology that allows for the isolation of regional differences in bone remodeling that occurs following the application of MOPs. (Zhu et al., 2017)Achieving a better understanding of these variations is of crucial importance, not only for the optimization of intervention strategies that are site-specific but also for minimizing any potential iatrogenic effects that may impact the periodontal support structures, particularly in patients who possess vulnerable phenotypes such as those with thin biotypes or those who have reduced bone volume. (Greenwell & Bissada, 1984)

Therefore, this randomized clinical trial was undertaken to quantify and compare the trabecular bone remodeling response to MOPs in the anterior and posterior maxillary regions using high-resolution cone-beam computed tomography (CBCT). By analyzing pre- and post-intervention changes in bone microarchitecture, this study aims to elucidate whether anatomical location modulates the biological efficacy of MOPs, thereby informing more tailored and biologically sound orthodontic protocols.

Materials and method

This prospective split-mouth randomized controlled trial (RCT) was designed in accordance with the CONSORT guidelines design and was conducted at Sri Rajiv Gandhi College of Dental Science & Hospital, Bangalore, Karnataka, under the auspices of Rajiv Gandhi University of Health Sciences. Institutional Review Board approval was granted prior to commencing the study. The split-mouth design ensured internal control by enabling direct comparison of treated versus control sites within the same individual, thereby minimizing intersubject variability.

The sample size was determined using G*Power 3.1 software. Based on an expected effect size of 0.85, derived from prior data (Solanki et al.), with an alpha value (α) of 0.05 and statistical power (1−β) of 0.80, a minimum of 17 patients per group was required. Considering a 15% anticipated dropout rate, the final enrollment comprised 38 participants: 19 with a thin periodontal biotype and 19 with a thick biotype. The sample size estimation and power analysis were critical to ensure sufficient sensitivity to detect a clinically meaningful difference, thereby avoiding type II errors. The power calculation incorporated key parameters, including the null and alternative hypotheses, the minimum detectable effect size, the acceptable level of significance, and the desired statistical power. G*Power was selected for its user-friendly interface and its compatibility with multiple statistical test types relevant to clinical research.

The study recruited orthodontic patients aged 15 to 30 years who were indicated for maxillary canine retraction. Participants were included if they presented either a thin (buccal bone ≤1.0 mm) or thick (buccal bone ≥1.5 mm) periodontal biotype, as measured at the mid-root level of the maxillary canines using pretreatment cone-beam computed tomography (CBCT). Exclusion criteria comprised the presence of systemic diseases such as diabetes or osteoporosis, periodontal disease (defined as probing pocket depth >4 mm and clinical attachment loss >3 mm), history of prior orthodontic treatment, and tobacco use.

The intervention side for micro-osteoperforations (MOPs) was randomized using a simple coin-toss method. MOPs were performed distal to the maxillary canine on the randomly assigned side using the Propel® device, which generated three perforations with a diameter of 1.5 mm and a depth of 3 mm. This device facilitates minimally invasive cortical bone stimulation to accelerate orthodontic tooth movement by enhancing local bone remodeling. The contralateral side served as a control, receiving a sham procedure involving placement of the Propel® device without activation. Canine retraction on both sides was carried out using nickel-titanium (NiTi) closed-coil springs (3M Unitek), calibrated to deliver a constant force of 150 grams. NiTi springs are widely utilized in orthodontics due to their superelastic properties, allowing for continuous light forces that promote efficient and steady space closure with minimal adjustments.

CBCT scans were obtained using the Carestream CS 8200 3D system with a voxel size of 0.2 mm, a field of view (FOV) of 8×8 cm, and an effective dose of 36.9 mGy·cm². This system provides high-resolution imaging with reduced metal artifacts through CS MAR technology and incorporates a low-dose mode to minimize radiation exposure while preserving diagnostic quality. Imaging was performed at baseline (T0) and at three months post-MOP (T1). All measurements were performed using Dolphin Imaging 11.9 software. The rate of canine retraction was quantified as the linear distance between the distal contact point of the canine and the mesial contact point of the second premolar. Labial and palatal alveolar bone thickness was measured at the mid-root level of the canine, perpendicular to its long axis. Bone density was evaluated in terms of mean Hounsfield units (HU) within a 1 mm radius around the perforation site. Although HU values provide a numerical index of radiodensity, their use in CBCT-based assessments remains controversial due to variability in calibration and susceptibility to noise and scatter. As a result, bone quality is increasingly being defined through structural rather than density-based parameters. Vertical bone loss was measured from the cementoenamel junction (CEJ) to the alveolar crest on both labial and palatal aspects. This parameter is a reliable indicator of periodontal breakdown, with a CEJ–crest distance exceeding 2 mm typically suggestive of bone loss. All radiographic measurements were conducted by two blinded and calibrated examiners, with inter-examiner reliability confirmed by an intraclass correlation coefficient (ICC) greater than 0.91.

Data analysis was performed using SPSS version 28.0. The Shapiro–Wilk test was used to assess data normality. Intragroup comparisons between baseline and post-treatment values were analyzed using paired t-tests, while intergroup comparisons (MOP vs. control) were evaluated with independent t-tests. For datasets

that failed normality assumptions, the Mann–Whitney U test was applied. Correlations between baseline bone thickness and amount of tooth movement were assessed using the Pearson correlation coefficient. A p-value of less than 0.05 was considered statistically significant. SPSS was chosen for its robust data handling capacity and its applicability in performing parametric and non-parametric tests, making it particularly suitable for clinical orthodontic research.

Results

A total of 38 patients completed the study, with 19 participants in each periodontal biotype group. The mean age was 18.2 ± 2.1 years in the thin-biotype group and 17.8 ± 1.9 years in the thick-biotype group. There were no statistically significant differences between the two groups in terms of age, sex distribution, or the magnitude of orthodontic force applied ($p > 0.05$), ensuring a comparable baseline for evaluating intervention effects.

Micro-osteoperforations significantly accelerated the rate of maxillary canine retraction in the thin-biotype group. The mean rate of tooth movement on the MOP side was 1.42 ± 0.31 mm/month compared to 0.90 ± 0.28 mm/month on the control side, reflecting a 58% increase in retraction rate ($p < 0.001$). In contrast, the thick-biotype group exhibited a more modest acceleration, with a mean rate of 1.10 ± 0.25 mm/month on the MOP side versus 0.90 ± 0.30 mm/month on the control side, amounting to a 22% increase that did not reach statistical significance ($p = 0.07$). A direct comparison of the acceleration effect between the thin and thick biotypes revealed a significantly greater response in the thin-biotype group ($p = 0.003$), indicating a phenotype-dependent variation in biological responsiveness to MOPs.

Palatal vertical bone loss was significantly greater at MOP sites in the thin-biotype group compared to their control sides, with a mean loss of 0.46 ± 0.12 mm versus 0.28 ± 0.10 mm, representing a 64% increase ($p = 0.02$). No significant difference in labial vertical bone loss was observed in either biotype ($p > 0.05$). Bone density also decreased more markedly at MOP sites in the thin-biotype group, with a mean reduction of −312 ± 45 Hounsfield units (HU), which was statistically significant ($p < 0.001$). In contrast, the thick-biotype group demonstrated a less pronounced reduction in bone density (−155 ± 32 HU), which did not reach significance ($p = 0.06$). A strong inverse correlation was observed between baseline alveolar bone thickness and the magnitude of MOP-induced tooth movement acceleration ($r = −0.72$, $p < 0.001$), indicating that thinner baseline bone was associated with greater responsiveness to the intervention.

Tooth Movement (Table 1)

Group	MOP Side (mm/month)	Control Side (mm/month)	Acceleration (%)	p-value
Thin biotype	1.42 ± 0.31	0.90 ± 0.28	+58%	<0.001*
Thick biotype	1.10 ± 0.25	0.90 ± 0.30	+22%	0.07

Discussion

This study demonstrates that the efficacy of micro-osteoperforations (MOPs) is profoundly influenced by the underlying alveolar bone phenotype. In patients with a thin alveolar bone biotype (≤1.0 mm), tooth movement was accelerated by 58%—a substantially greater increase than the 22% observed in those with a thick biotype. This confirms our initial hypothesis that thinner bone facilitates greater propagation of the regional acceleratory phenomenon (RAP), likely due to reduced cortical resistance. However, this biomechanical advantage was accompanied by significant palatal bone loss (mean: 0.46 mm), corroborating the findings of Solanki et al., who reported similar patterns of bone loss in extraction-based orthodontic cases. These observations align with Handelman's assertion that the palatal plate of the maxillary alveolar process serves as a critical orthodontic boundary; exceeding this anatomical limit may result in complications such as dehiscences, fenestrations, or gingival recession. In particular, Solanki et al. (2014) reported a significant disparity in vertical bone loss on the palatal aspect compared to the labial aspect following alignment and leveling, further emphasizing the structural vulnerability of the palatal cortex in thin phenotypes.

The enhanced response observed in thin alveolar bone can be attributed to its reduced cortical thickness, which likely promotes rapid diffusion of signaling molecules—such as RANKL—and facilitates the recruitment of osteoclasts. The regional acceleratory phenomenon (RAP), triggered by surgical stimuli such as MOPs, initiates

a cascade of cellular and tissue-level remodeling responses. (Hong et al., 2003) At the cellular level, RAP is characterized by activation of basic multicellular units (BMUs) and expansion of bone remodeling spaces. Initially, woven bone is formed, which is later remodeled into lamellar bone. (Martínez-Reina et al., 2009)MOPs are believed to amplify this process by stimulating cytokine release and promoting a localized inflammatory environment. In orthodontic contexts, tooth movement is driven by bone turnover, particularly through osteoclastic activity on the pressure side of the periodontal ligament. The application of orthodontic force constricts blood vessels within the ligament, triggering release of pro-inflammatory mediators including chemokines and cytokines. These mediators facilitate differentiation of osteoclast precursors into multinucleated bone-resorbing cells. Central to this process is the RANK/RANKL/OPG signaling axis, which governs osteoclastogenesis. RANKL binds to its receptor RANK to promote osteoclast differentiation, while osteoprotegerin (OPG) acts as a decoy receptor that neutralizes RANKL's activity, thus preserving bone mass. The observed inverse correlation ($r = -0.72$) between baseline alveolar bone thickness and MOP-induced acceleration highlights the biological interplay between cortical bone architecture and inflammatory responsiveness. In contrast, patients with thicker bone may exhibit dampened RAP activity due to reduced cytokine penetration and higher trabecular density, which may buffer against both acceleration and alveolar height loss. The greater palatal bone loss observed in the thin biotype suggests that the combined impact of retraction mechanics and MOP-induced remodeling may pose compounded risks in areas where bony support is inherently limited.

These findings underscore the critical importance of individualized treatment planning in orthodontics. Cone-beam computed tomography (CBCT) screening is essential for identifying patients with thin alveolar bone, allowing clinicians to tailor MOP application more precisely. Specifically, pretreatment evaluation of alveolar thickness enables both the optimization of tooth movement efficiency and the minimization of unintended consequences such as iatrogenic bone loss or cortical perforation. In patients with pre-existing palatal deficiencies—such as those frequently encountered in Class II division 2 malocclusions—MOPs may be contraindicated or should be applied with extreme caution. Furthermore, the implementation of personalized protocols based on biotype, such as modulating the magnitude of retraction force or reducing MOP frequency in susceptible individuals, may improve clinical outcomes while preserving periodontal integrity.

While this study provides valuable insights into phenotype-specific responses to MOPs, certain limitations should be acknowledged. The short-term follow-up period (3 months) restricts interpretation of long-term stability and tissue remodeling. Additionally, the single-center design may limit generalizability. Future research should explore the molecular dynamics underlying MOP efficacy, particularly by investigating the RANKL/OPG ratio as a potential biomarker for predicting treatment response. Given the pivotal role of this system in regulating bone resorption, understanding its fluctuations in response to MOPs could yield novel strategies for individualized modulation of orthodontic mechanics. Another promising avenue is the use of CBCT-guided customization of MOP depth. Although specific clinical protocols are not yet established, emerging interest in 3D-printed guide plates for precise MOP placement suggests a potential method for maximizing efficiency while minimizing adverse effects. Finally, long-term studies evaluating post-treatment stability, relapse rates, and periodontal outcomes will be essential to fully validate the safety and sustainability of MOP-enhanced orthodontic strategies.

Conclusion

Micro-osteoperforations (MOPs) accelerate orthodontic tooth movement 2.6 times more effectively in patients with thin alveolar bone (≤ 1.0 mm) compared to those with thick bone phenotypes. However, this biomechanical advantage is accompanied by a significant increase in palatal vertical bone loss, underscoring a critical trade-off between treatment efficiency and periodontal risk. These findings highlight the indispensable role of pre-treatment CBCT evaluation in identifying alveolar bone phenotype, enabling clinicians to tailor MOP application safely and effectively. Moving forward, the development of phenotype-specific MOP protocols is essential to enhance clinical outcomes while minimizing the risk of iatrogenic bone loss, particularly in high-risk biotypes.

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