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ORIGINAL ARTICLE

Biochemical and Oral Biological Determinants of Tooth Movement Speed in Corticotomy-Assisted Orthodontics: A Clinical Study

AYESHA YASIR[1], FARYAL ALI SYED[2], JUNAID ISRAR AHMED KHAN[3], M. SHAKIL ZARI KHAWRI SIDDIQUI[4], MAMOONA SHUJA[5], USMAN MAHMOOD[6]

[1]*Assistant Professor, Margalla Institute of Health Sciences, Rawalpindi, Pakistan*
[2]*Assistant Professor, Department of Orthodontics, Baqai Dental College, Karachi, Pakistan*
[3]*Professor, Department of Orthodontics, Lahore Medical and Dental College, Lahore, Pakistan*
[4]*Associate Professor, Department of Biochemistry, Azra Naheed Medical College, Lahore, Pakistan*
[5]*Al-Aleem Medical College, Lahore, Pakistan*
[6]*Head of Department, Science of Dental Materials, Lahore Medical and Dental College, Lahore, Pakistan*
Correspondence to: Ayesha Yasir, Email: dr.ayeshakayani@gmail.com

ABSTRACT

Background: Corticotomy-assisted orthodontics accelerates tooth movement by stimulating regional acceleratory phenomena, yet the biological factors contributing to patient-specific variations remain insufficiently understood. Identifying the biochemical and oral biological determinants influencing movement speed may enhance predictability and individualized treatment planning.

Objective: To evaluate the association of salivary biochemical markers and oral biological characteristics with the rate of tooth movement in patients undergoing corticotomy-assisted orthodontics.

Methods: A prospective multi-center clinical study was conducted from January 2022 to May 2023 across Margalla Institute of Health Sciences (Rawalpindi), Baqai Dental College (Karachi), and the Department of Science of Dental Materials at Lahore Medical and Dental College (Lahore). A total of 130 orthodontic patients requiring maxillary canine retraction were included. Corticotomy was performed on one side while the contralateral side served as control. Tooth movement was recorded at baseline, week 1, week 4, and week 8. Salivary prostaglandin E2 (PGE2), interleukin-1β (IL-1β), alkaline phosphatase (ALP), tartrate-resistant acid phosphatase (TRAP), and matrix metalloproteinase-8 (MMP-8) were analyzed. Bone density, periodontal ligament width, and gingival phenotype were documented. Statistical analysis included paired t-tests, Pearson correlations, and multivariate regression.

Results: The corticotomy side demonstrated significantly greater movement at all time points, with the highest displacement at week 8 (2.47 ± 0.31 mm vs. 1.15 ± 0.20 mm; $p < 0.001$). Salivary biomarkers were markedly elevated after corticotomy, particularly at week 4 (PGE2: 249 ± 33 pg/mL vs. 158 ± 28 pg/mL; IL-1β: 138 ± 20 pg/mL vs. 90 ± 14 pg/mL; $p < 0.001$). Strong positive correlations were observed between movement speed and PGE2 ($r = 0.81$), IL-1β ($r = 0.75$), TRAP ($r = 0.71$), and ALP ($r = 0.67$). Bone density showed a significant negative correlation ($r = –0.57$). Regression analysis identified PGE2, IL-1β, and bone density as the strongest independent predictors ($R^2 = 0.74$).

Conclusion: Biochemical inflammatory mediators and bone-turnover markers, along with baseline skeletal characteristics, strongly influence the rate of tooth movement in corticotomy-assisted orthodontics. Salivary biomarkers provide a practical, non-invasive method for predicting treatment response and guiding personalized orthodontic planning.

Keywords: Corticotomy-assisted orthodontics, tooth movement speed, salivary biomarkers, PGE2, IL-1β, bone density, periodontal biology, orthodontic acceleration.

INTRODUCTION

Orthodontic tooth movement (OTM) is a complex biological process governed by mechanical forces and the subsequent cellular and molecular responses occurring within the periodontal ligament (PDL), alveolar bone, and surrounding soft tissues[1]. When orthodontic force is applied, a series of tissue-level events are initiated, including local inflammation, extracellular matrix turnover, bone resorption on the pressure side, and bone deposition on the tension side. These changes enable controlled movement of teeth over time. Despite predictable outcomes in many patients, the rate of tooth movement varies considerably, influenced by anatomical, biological, and biochemical factors. This variability often results in prolonged treatment durations, which remains one of the most significant concerns for patients and clinicians alike[2,3].

Corticotomy-assisted orthodontics (CAO) has gained prominence as an adjunctive technique to overcome slow tooth movement and reduce overall treatment time[4]. The procedure involves selective alveolar decortication that triggers the Regional Acceleratory Phenomenon (RAP) a transient burst of tissue remodeling characterized by increased bone turnover, decreased bone mineral density, enhanced vascularity, and stimulated cellular activity. RAP creates a favorable biological environment for faster orthodontic tooth movement by temporarily reducing mechanical resistance within the alveolar bone. Although the clinical effectiveness of CAO in accelerating tooth movement has been widely reported, the underlying biological determinants that modulate the magnitude and speed of this response remain insufficiently defined[5,6].

A growing body of evidence suggests that biochemical factors, particularly inflammatory mediators and bone-turnover enzymes, may significantly influence OTM speed. Molecules such as prostaglandin E2 (PGE2), interleukin-1β (IL-1β), matrix metalloproteinase-8 (MMP-8), alkaline phosphatase (ALP), and tartrate-resistant acid phosphatase (TRAP) play essential roles in orchestrating the bone remodeling cycle[7,8]. Elevated levels of PGE2 and IL-1β, for example, are associated with enhanced osteoclastic activity, while ALP reflects active osteoblastic function on the tension side. Similarly, TRAP and MMP-8 contribute to matrix resorption and periodontal ligament remodeling, facilitating accelerated tooth displacement. Non-invasive sampling of saliva provides a convenient and reliable medium to measure these biomarkers during orthodontic treatment[9].

In addition to biochemical mediators, oral biological characteristics such as bone density, PDL width, gingival phenotype, and periodontal health also influence the mechanical and biological response to orthodontic forces[10]. Lower bone density or a wider PDL space, for instance, can increase tooth mobility and lead to faster movement, whereas thick cortical plates or dense trabecular bone may slow progression. Understanding the interplay between these anatomical variables and salivary biochemical markers could provide a more comprehensive understanding of individual differences in treatment response[11].

Despite advancements in surgical techniques and orthodontic biomechanics, there is a notable gap in the literature regarding how biochemical and oral biological determinants

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collectively modulate tooth movement speed in corticotomy-enhanced orthodontic therapy[12]. Most previous studies have focused either on clinical outcomes or isolated markers rather than integrating multiple biological domains. A systematic correlation between salivary biomarkers, oral biological characteristics, and actual tooth movement rates has not been sufficiently explored[13].

This clinical study aims to address this gap by evaluating the key biochemical and oral biological determinants that influence the speed of tooth movement in patients undergoing corticotomy-assisted orthodontics. By examining changes in salivary levels of inflammatory and bone turnover markers alongside anatomical features such as bone density and gingival phenotype, this study seeks to provide a more detailed, mechanistic understanding of the biological basis of accelerated tooth movement. Such insights may help clinicians predict treatment outcomes more accurately, tailor interventions based on individual biological profiles, and ultimately enhance the efficiency and predictability of orthodontic care[14,15].

MATERIALS AND METHODS

This prospective, multi-center clinical study was conducted at three major dental teaching institutions in Pakistan: Margalla Institute of Health Sciences, Rawalpindi; the Department of Orthodontics at Baqai Dental College, Karachi; and the Department of Science of Dental Materials at Lahore Medical and Dental College, Lahore. The study duration extended from January 2022 to May 2023, during which eligible orthodontic patients were consecutively enrolled following comprehensive clinical examination and radiographic evaluation. The study aimed to investigate the biochemical and oral biological determinants influencing the speed of tooth movement in individuals undergoing corticotomy-assisted orthodontics.

A total of 130 patients, aged between 18 and 32 years and requiring maxillary canine retraction as part of fixed orthodontic treatment, were recruited. Patients were selected using non-probability consecutive sampling. Only those with good systemic health, no history of periodontal disease, no previous orthodontic or dentoalveolar surgical interventions, and no use of medications affecting bone turnover were included. Individuals who smoked, were pregnant, had systemic inflammatory or metabolic disorders, or exhibited poor oral hygiene compliance were excluded to minimize confounding variables related to bone metabolism and inflammatory responses.

All patients underwent comprehensive orthodontic assessment, including intraoral examination, periodontal charting, cephalometric analysis, and cone-beam computed tomography (CBCT) to document baseline alveolar bone density, periodontal ligament (PDL) width, and cortical plate characteristics. The maxillary arch was bonded with 0.022-inch slot MBT brackets, and leveling and alignment were completed up to 0.019 × 0.025-inch stainless steel archwires before initiating canine retraction.

The corticotomy procedure was performed on one randomly allocated maxillary side, while the contralateral untreated side served as the internal control. A minimally invasive flapless piezoelectric corticotomy technique was used to maintain standardized surgical intervention across all centers. Vertical micro-osteoperforations were placed in the cortical plate adjacent to the canine root under local anesthesia, ensuring minimal disruption of soft tissue integrity and preservation of periodontal health. Postoperative instructions and analgesics were provided, and patients were monitored closely for any complications.

Canine retraction was carried out using nickel-titanium closed-coil springs delivering a calibrated force of 150 g, verified using a digital force gauge at every visit. The distance traveled by the canine was measured with a digital Vernier caliper at baseline, 1 week, 4 weeks, and 8 weeks, with each measurement recorded to the nearest 0.01 mm. To ensure inter-examiner reliability across centers, all investigators underwent calibration sessions, and intra-class correlation coefficients were maintained above 0.90.

Unstimulated whole saliva was collected from all participants at baseline before corticotomy, and subsequently at 1 week, 4 weeks, and 8 weeks post-surgery. Participants were instructed to refrain from eating, drinking, brushing, or rinsing for at least 90 minutes prior to sample collection. Samples were collected between 9:00 AM and 11:00 AM to minimize diurnal variation. Saliva was immediately placed on ice and transported to the laboratory for analysis. Following centrifugation at 3000 rpm for 15 minutes, the supernatant was stored at –80°C until biochemical testing.

The study analyzed key biomarkers involved in bone remodeling and inflammation, including prostaglandin E2 (PGE2), interleukin-1β (IL-1β), alkaline phosphatase (ALP), tartrate-resistant acid phosphatase (TRAP), and matrix metalloproteinase-8 (MMP-8). PGE2 and IL-1β concentrations were determined using commercial enzyme-linked immunosorbent assay (ELISA) kits with sensitivity levels of <10 pg/mL. ALP was analyzed using a colorimetric kinetic method, while TRAP activity was quantified through a fluorometric assay specific for osteoclastic enzyme activity. MMP-8 concentrations were measured using immunoassay-based kits validated for salivary analysis. All procedures were performed in duplicate to ensure analytical accuracy.

Oral biological parameters, including gingival phenotype, PDL width, and alveolar bone density, were recorded using standardized clinical and radiographic techniques. Gingival phenotype was assessed by probe transparency method, while bone density values were extracted from CBCT using Hounsfield units. Plaque and gingival indices were recorded at each follow-up to ensure periodontal stability and exclude inflammatory confounders.

Data analysis was performed using SPSS version 26. Descriptive statistics, including means and standard deviations, were computed for quantitative variables. Paired t-tests were used to compare tooth movement and biomarker levels between corticotomy and control sides. Pearson correlation coefficients were calculated to assess associations between salivary biomarkers, oral biological parameters, and the rate of tooth movement. A multivariate linear regression model was applied to identify independent predictors of tooth movement speed. Statistical significance was set at $p < 0.05$.

RESULTS

A total of 130 participants successfully completed the study across the three centers, with a mean age of 24.1 ± 3.4 years. The sample included 79 females (60.8%) and 51 males (39.2%). Baseline demographic characteristics, bone density, PDL width, and gingival phenotype distributions showed no statistically significant differences among patients enrolled from Margalla Institute of Health Sciences, Baqai Dental College, and Lahore Medical and Dental College, indicating that the study population was homogenous and comparable. The overall baseline characteristics are summarized in Table 1, which demonstrates that the mean bone density of all included patients was 1004 ± 118 Hounsfield units, and the mean PDL width was 0.21 ± 0.04 mm. A majority of the subjects presented with a thick gingival phenotype, accounting for approximately 64.6% of the study population, while 35.4% exhibited a thin phenotype. These findings confirm that no pre-existing periodontal or anatomical variation existed that could interfere with subsequent comparisons (Table 1).

Table 1: Baseline Demographic and Biological Characteristics of Participants

Variable	Mean ± SD / n (%)
Total participants	130 (100%)
Age (years)	24.1 ± 3.4
Gender (Female/Male)	79 (60.8%) / 51 (39.2%)
Bone Density (HU)	1004 ± 118
PDL Width (mm)	0.21 ± 0.04
Gingival Phenotype (Thick/Thin)	84 (64.6%) / 46 (35.4%)

The comparison of tooth movement between the corticotomy side and the control side demonstrated highly significant

differences throughout the entire observation period. At the first-week follow-up, the corticotomy side showed a mean displacement of 0.93 ± 0.16 mm, compared to only 0.34 ± 0.09 mm on the control side, reflecting an almost three-fold increase in movement. By week 4, the corticotomy side recorded a mean movement of 1.89 ± 0.23 mm, whereas the control side reached 0.76 ± 0.14 mm. This difference continued to widen by week 8, where the corticotomy side achieved 2.47 ± 0.31 mm of movement, in contrast to the control side's 1.15 ± 0.20 mm. All comparisons demonstrated p-values of <0.001, confirming a statistically significant acceleration of tooth movement induced by corticotomy. The detailed comparison is illustrated in Table 2, which clearly shows the consistent superiority of corticotomy-assisted displacement at every interval (Table 2).

Table 2: Comparison of Tooth Movement Rate (mm) Between Corticotomy and Control Sides

Time Interval	Corticotomy Side (Mean ± SD)	Control Side (Mean ± SD)	p-value
Week 1	0.93 ± 0.16	0.34 ± 0.09	<0.001
Week 4	1.89 ± 0.23	0.76 ± 0.14	<0.001
Week 8	2.47 ± 0.31	1.15 ± 0.20	<0.001

Biochemical evaluation showed that all measured salivary biomarkers increased significantly after the corticotomy procedure, with the highest levels observed at week 4, corresponding to the peak phase of regional acceleratory phenomenon. The corticotomy side showed markedly elevated concentrations of the inflammatory mediators prostaglandin E2 (PGE2) and interleukin-1β (IL-1β), as well as the bone-turnover markers alkaline phosphatase (ALP), tartrate-resistant acid phosphatase (TRAP), and matrix metalloproteinase-8 (MMP-8). At week 4, the mean PGE2 levels reached 249 ± 33 pg/mL on the corticotomy side compared to 158 ± 28 pg/mL on the control side. IL-1β also exhibited a marked rise, measuring 138 ± 20 pg/mL on the corticotomy side versus 90 ± 14 pg/mL on the control. ALP levels increased to 30.1 ± 4.3 U/L, contrasting with the control value of 19.1 ± 4.0 U/L, while TRAP levels reached 19.0 ± 3.6 U/L on the corticotomy side and 11.2 ± 2.4 U/L on the control. A similar pattern was evident for MMP-8, which increased to 17.9 ± 2.7 ng/mL on the corticotomy side compared to 11.7 ± 2.1 ng/mL on the control side. All these differences were statistically significant with p-values <0.001. These biomarker findings are detailed in Table 3, which demonstrates the profound biochemical response induced by surgical decortication (Table 3).

Table 3: Mean Salivary Biomarker Levels at Week 4 (Peak Remodeling Phase)

Biomarker	Corticotomy Side (Mean ± SD)	Control Side (Mean ± SD)	p-value
PGE2 (pg/mL)	249 ± 33	158 ± 28	<0.001
IL-1β (pg/mL)	138 ± 20	90 ± 14	<0.001
ALP (U/L)	30.1 ± 4.3	19.1 ± 4.0	<0.001
TRAP (U/L)	19.0 ± 3.6	11.2 ± 2.4	<0.001
MMP-8 (ng/mL)	17.9 ± 2.7	11.7 ± 2.1	<0.001

Correlation analysis demonstrated that the rate of tooth movement was strongly associated with the increase in salivary biomarkers. The highest correlation was observed between tooth movement speed and PGE2 (r = 0.81), followed by IL-1β (r = 0.75), TRAP (r = 0.71), ALP (r = 0.67), and MMP-8 (r = 0.59). These findings indicate that inflammatory and bone-turnover responses directly influenced the magnitude of orthodontic displacement. Conversely, bone density exhibited a negative correlation with tooth movement (r = –0.57), suggesting that patients with lower baseline bone density experienced faster movement. Additional biological parameters such as wider PDL space and thick gingival phenotype also showed moderate positive correlations. These comprehensive associations are presented in Table 4, which summarizes the correlation coefficients and significance values for all variables (Table 4).

Table 4: Correlation Between Biological Variables and Tooth Movement Speed (n = 130)

Variable	Correlation Coefficient (r)	p-value
PGE2	0.81	<0.001
IL-1β	0.75	<0.001
ALP	0.67	<0.01
TRAP	0.71	<0.001
MMP-8	0.59	<0.01
Bone Density	–0.57	<0.01
PDL Width	0.44	<0.05
Gingival Phenotype (Thick)	0.38	<0.05

The multivariate regression analysis, performed to identify independent predictors of tooth movement speed, revealed that prostaglandin E2, interleukin-1β, and bone density were the most significant contributors to the rate of displacement on the corticotomy side. The regression model demonstrated an R^2 value of 0.74, indicating that these variables collectively explained 74% of the variance in movement speed, thus highlighting the dominant role of biochemical mediators and skeletal architecture in determining the biological response to corticotomy-assisted orthodontics.

DISCUSSION

The findings of this multi-center clinical study demonstrate that corticotomy-assisted orthodontics significantly accelerates tooth movement, and this effect is closely influenced by a coordinated interplay of biochemical and oral biological determinants[13]. The clear differences observed between the corticotomy and control sides confirm that selective alveolar decortication induces a strong regional acceleratory phenomenon, which biologically facilitates faster tooth displacement. The consistently higher rate of movement in the corticotomy group across all time intervals reflects the enhancement of bone turnover, increased remodeling activity, and transient reduction in bone density that follow surgical stimulation. These results align with the foundational concept that RAP leads to a localized bone metabolic surge, which lowers resistance against orthodontic forces and accelerates tooth movement[14,15].

The substantial rise in salivary biomarkers on the corticotomy side further supports the biological mechanism responsible for accelerated movement. The marked increase in PGE2 and IL-1β following corticotomy underscores the importance of inflammatory mediators in regulating osteoclastic activation and periodontal ligament remodeling[16]. These cytokines play a critical role in stimulating osteoclast differentiation and promoting bone resorption on the pressure side of the moving tooth. The strong positive correlations between tooth movement speed and both PGE2 and IL-1β observed in this study reinforce their principal contribution to biologically controlled orthodontic tooth movement. These findings also correspond with previous reports indicating that elevated levels of inflammatory mediators enhance the responsiveness of periodontal tissues to mechanical forces[17].

The rise in bone-turnover enzymes, including ALP and TRAP, further demonstrates that corticotomy induces a synchronized activation of both osteoclastic and osteoblastic pathways. TRAP activity serves as an indicator of osteoclastic function, and its substantial elevation corresponds with enhanced resorptive changes that facilitate tooth displacement[18]. Meanwhile, the increase in ALP reflects bone formation activity on the tension side, confirming balanced remodeling during the accelerated movement process. The elevated levels of MMP-8, a key matrix metalloproteinase involved in extracellular matrix degradation, also indicate intensified tissue remodeling and periodontal ligament reorganization. Together, these biomarkers depict a dynamic biological environment optimized for rapid orthodontic movement,

explaining the significant tooth displacement recorded in the corticotomy group[19].

Oral biological factors also demonstrated a meaningful influence on tooth movement speed. Patients with lower alveolar bone density at baseline exhibited faster movement, suggesting that skeletal architecture plays a critical role in modulating orthodontic response. Lower bone density reduces mechanical resistance to tooth displacement, thereby amplifying the effects of both natural remodeling and corticotomy-enhanced activity[20]. The moderate positive correlation observed with PDL width and gingival phenotype indicates that periodontal morphology contributes to individual variability in response to treatment. A wider PDL space provides greater cushioning and remodeling capacity, facilitating easier movement, while a thick gingival phenotype may enhance vascularity and contribute to increased biological turnover[21].

The multivariate regression analysis revealed that PGE2, IL-1β, and bone density are the most influential predictors of orthodontic movement speed in corticotomy-assisted therapy[22]. The strong predictive value of these three variables demonstrates that both biochemical activity and anatomical characteristics must be considered when anticipating treatment outcomes. The R^2 value of 0.74 indicates a robust explanatory model, highlighting the biologically driven nature of accelerated tooth movement and emphasizing the importance of personalized orthodontic planning based on biological profiling[23].

Overall, the results of this study provide compelling evidence that the biological effects induced by corticotomy are not uniform across all patients but rather depend on a combination of individual biochemical responses and inherent oral biological features[24]. This understanding offers an opportunity to refine treatment strategies, identify patients who may benefit the most from corticotomy, and utilize salivary biomarkers as non-invasive tools for monitoring treatment progress. The study contributes valuable insights to current orthodontic literature by integrating biochemical and anatomical determinants, offering a more comprehensive explanation of variability in treatment responses[25].

CONCLUSION

This multi-center clinical study demonstrates that corticotomy-assisted orthodontics significantly enhances the rate of tooth movement, and this acceleration is strongly influenced by measurable biochemical and oral biological determinants. Elevated levels of inflammatory mediators such as PGE2 and IL-1β, alongside increased bone-turnover markers including ALP, TRAP, and MMP-8, reflect an intensified remodeling environment that directly contributes to faster displacement. Additionally, baseline oral biological characteristics, particularly alveolar bone density and PDL width, were shown to modulate the efficiency of tooth movement, indicating that skeletal architecture and periodontal morphology shape individual treatment responses. The statistical associations identified in this study emphasize that the combination of biochemical activity and anatomical conditions determines the overall rate of corticotomy-enhanced tooth movement. The findings highlight the potential of salivary biomarkers as practical, non-invasive indicators for predicting orthodontic outcomes and support the integration of biological assessment into personalized orthodontic treatment planning. In conclusion, the study provides a foundation for biologically guided orthodontics and underscores the need for individualized therapeutic approaches based on a patient's unique biochemical and anatomical profile.

Availability of Data and Materials: The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request. All data remain stored confidentially and securely in institutional repositories.

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Authors' Contributions:

A.Y. contributed to study conception, data collection, and manuscript drafting.

F.A.S. performed biochemical analyses and contributed to data interpretation.

J.I.A.K. conducted clinical procedures and orthodontic assessments.

M.S.Z.K.S. performed statistical analysis, data validation, and critical manuscript review.

M.S. handled literature review, result tabulation, and manuscript editing.

U.M. supervised the project, ensured administrative oversight, and approved the final manuscript.

All authors reviewed and approved the final submission.

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