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Evaluating invasive, non-invasive interventions on GCF IL-1β, PGE2 levels and the rate of orthodontic tooth movement: A comparative study

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Abstract:
Accelerating orthodontic tooth movement while maintaining biological safety remains a clinical challenge, with inflammatory mediators playing a key role in tissue remodeling. This prospective comparative study evaluated 9 patients aged 14–25 years undergoing orthodontic treatment using low-level laser therapy, micro-osteoperforations and their combination, with gingival crevicular fluid interleukin-1 beta and prostaglandin E2 measured by enzyme-linked immunosorbent assay and canine retraction assessed digitally. Both interventions significantly increased interleukin-1 beta and prostaglandin E2 from baseline to 4 weeks, with smaller but significant increases through 8 weeks, while micro-osteoperforations produced faster tooth movement. Micro-osteoperforations enhanced orthodontic tooth movement more effectively than low-level laser therapy, whereas low-level laser therapy produced relatively higher biomarker levels. These findings advance knowledge by supporting micro-osteoperforations as a more effective approach for accelerating orthodontic tooth movement through modulation of the inflammatory response.

Keywords: Orthodontic tooth movement (OTM), micro-osteoperforation (MOP), low-level laser therapy (LLLT), interleukin-1 beta, prostaglandin E2, gingival crevicular fluid

Background:
Orthodontic tooth movement is a complex biological process, with the remodelling of alveolar bone in response to mechanical forces. This process involves a series of steps such as altering the function of tissues and cells within the periodontal ligament and surrounding tissues with release of inflammatory cytokines and biochemical mediators **[1]**. These mediators are important in controlling bone resorption and formation during orthodontic tooth movements (OTMs), playing a crucial role, among them Interleukin-1 beta (IL-1β) and Prostaglandin E2 (PGE2) **[2]**. Toughened bone resorption on the pressure side of the tooth is caused by a pro-inflammatory cytokine called IL-1β, which is found to promote the differentiation and activity of osteoclasts **[3]**. In the same way, PGE2, a lipid mediator of arachidonic acid metabolism, was demonstrated to stimulate osteoclastic activity and bone remodelling **[4]**. It is thought that the concentrations of such mediators within the gingival crevicular fluid (GCF) reflect the biological reaction to orthodontic treatment **[5]**. Although orthodontic treatment has improved over the years, one of the most common concerns is the length of time the treatments take. Longer treatment times may result in complications of root resorption, periodontal breakdown, or failure of the patient to comply with the treatment **[6].** Thus, several techniques have been investigated for enhancing orthodontically-induced tooth movement, which attempt to alter responses of the periodontal tissues. Invasive methods such as micro-osteoperforation (MOPs) have been used which produce a regional acceleratory phenomenon (RAP) that increases bone remodeling and decreases resistance to tooth movement **[7]**. MOPs include making small holes in the bone and inducing inflammation around that area, in order to improve the expression of cytokines **[8].** Nambiar *et al.* found that GCF IL-1β levels increased after piezocision and corticotomy. Levels peaked at 24 hours and declined by day 21 **[9].** MOPs have been shown to lead to large increases in both IL-1β and PGE2 levels that are associated with increased tooth movement **[10]**. Mohaghegh *et al.* **[11]** reported increased canine retraction with MOP. However, this effect was not significant in studies with a low risk of bias. Non-invasive methods, like LLLT (low level laser therapy), have also been seen as promising alternatives, however. LLLT achieves its effect by following the principles of a photobiomodulation, which improves cell metabolism, boost ATP production and triggers inflammatory mediator releasing without damaging tissues **[12]**. LLLT has been proposed to hasten orthodontic tooth movement, by stimulating osteoblastic and osteoclastic formation **[13]**. Shankar *et al.* observed significant changes in GCF IL-1β and PGE2 levels after orthodontic separator placement. LLLT also reduced pain **[14]**. While both MOPs and LLLT have been shown to accelerate tooth movement, there is very limited evidence to compare both modalities when examining biochemical mediators and clinical outcomes, within the same study design. In addition, there are few studies that have evaluated both modalities in the same patient without inter-individual differences **[15]**. Therefore, it is of interest to compare the effect of the invasive (MOPs) and non-invasive (LLLT) on the gingival crevicular fluid (GCF) levels of IL-1 β and PG E2 and correlate this effect with the rate of orthodontic tooth movement.

Materials and Methodology:
It was an *in vivo* clinical study with the approval of the institutional ethical committee and scientific review board of the dept of Orthodontics and Dentofacial Orthopedics at Buddha Institute of Dental Sciences & Hospital of Patna. Applications were briefed to all participants or their guardians and informed written consent obtained before enrolment.

Study population:
Patients were selected based on criteria such as age range (14-25 years), orthodontic treatment needed, bilateral extraction of first premolars and canine retraction. The dental condition in the patients was good and their skeletal patterns were classified as Class I and Class II patterns with dentoalveolar protrusion. Patients using medications affecting bone metabolism or with craniofacial deformities or systemic diseases were excluded.

Study design:
Three groups (one-third each) of the selected patients were assigned to each group by a random allocation method.

[1] Group I (LLLT group): low level laser therapy was applied to RIGHT maxillary canine and the LEFT as control.
[2] In Group II (MOP group): the right maxillary canine was subjected to micro-osteoperforations and left served as control.
[3] Group III (Combination group): Right sides were treated with MOPs and left sides were treated with LLLT.

Orthodontic procedure:
The fixed ortho' appliances were used on all patients, with the use of MBT prescription (0.022-inch slot). Canine retraction was done with a 0.016″ × 0.022″ archwire of nickel–titanium closed coil springs with a consistent force of 150g measured through a Dontrix gauge. A transpalatal arch was made for the anchoring and second molars were included.

Intervention protocols:
Low level laser therapy (LLLT):
A canine with attached gingiva was examined using a 980nm, 0.1 W GaAlAs diode laser. After activation, the 10 irradiation points were subjected to three consecutive days of production with a mutual time of 10 seconds.

Micro-Osteoperforations (MOPs):
The attached gingiva (with 2% lidocaine with epinephrine-local anesthesia) was drilled through three times with 1 mm diameter and 3 mm depth dorsal to the canine without flap elevation.

Gingival crevicular fluid collection (GCF):
Taking endodontic GCF samples at four time interval, from canine mesial and distal with standardized endodontic paper points
[1] T0: Base line (before the force is applied)
[2] T1: After first intervention
[3] T2: After 4 weeks
[4] T3: After 8 weeks
Samples were stored in phosphate buffered saline (PBS) at −80°C for analysis.

Biochemical analysis:
Commercially available IL-1β and PGE2 ELISA kits were used for quantitative estimation of their levels. Absorbance was determined at suitable wavelengths with a microplate reader and concentrations determined by the standard curve.

Measurement of tooth movement:
Baseline, four and eight week alginate impressions were made. A UP3D 560 scanner was used to capture the casts and EXO CAD software analysed the casts. Retraction was determined with respect to a stable anatomical landmark, the medial end of the third palatal rugae, in the canine.

Statistical analysis:
GraphPad Prism software (version 5) was used for data analysis. These were then used to determine the mean and standard deviation of each group. One-way ANOVA followed by post hock tests of Tukey and Newman–Keuls were performed for intergroup comparisons. A p-value of ≤ 0.05 was regarded as statistically significant and a 95% confidence interval was used.

Results:
As shown in **Table 1**, interleukin-1β (IL-1β) levels increased progressively from baseline (T0) to 8 weeks (T3) in both the experimental and control sites of Groups I and II. In Group I, the mean IL-1β level at the experimental site increased from 4.5 ± 1.1 pg/µl at baseline to 67.7 ± 3.5 pg/µl at 8 weeks, whereas the corresponding control-site level increased from 3.2 ± 0.9 to 14.7 ± 2.4 pg/µl. Similarly, in Group II, the mean IL-1β level at the experimental site increased from 5.1 ± 1.8 to 55.4 ± 5.1 pg/µl, compared with an increase from 4.3 ± 1.4 to 15.2 ± 2.9 pg/µl at the control site. The experimental sites demonstrated greater increases in IL-1β levels than the corresponding control sites in both groups. The reported comparisons showed highly significant differences between control and experimental sites ($p \leq 0.001$; one-way ANOVA followed by the Newman–Keuls test). In Group III, which compared low-level laser therapy (LLLT) with micro-osteoperforation (MOP) in the same participants, IL-1β levels increased progressively with both interventions (Table 2A). The mean level increased from 4.9 ± 1.1 pg/µl at baseline to 66.1 ± 5.4 pg/µl at 8 weeks on the LLLT side, while the corresponding increase on the MOP side was from 5.8 ± 1.3 to 57.1 ± 5.2 pg/µl. LLLT demonstrated higher mean IL-1β levels than MOP at T1, T2 and T3, although the baseline level was lower on the LLLT side. These findings indicate a greater observed IL-1β response with LLLT over the follow-up period. Prostaglandin E2 (PGE2) levels increased from baseline to 8 weeks in the comparisons involving LLLT, MOP and their respective control sites (Tables 2). In Group I, the two reported mean values at baseline were 0.0114 ± 0.001 and 0.0113 ± 0.001 pg/µl. At 8 weeks, these values were 0.0914 ± 0.004 and 0.4926 ± 0.01 pg/µl, respectively. In Group II, the corresponding baseline values were 0.012 ± 0.001 and 0.0153 ± 0.001 pg/µl, increasing to 0.0956 ± 0.002 and 0.4841 ± 0.04 pg/µl at 8 weeks. In both groups, the second reported value was substantially higher than the first at the later time points. In Group III, the mean PGE2 level increased from 0.0113 ± 0.001 to 0.4926 ± 0.03 pg/µl on the LLLT side and from 0.016 ± 0.001 to 0.4816 ± 0.039 pg/µl on the MOP side. At 8 weeks, the mean values were similar between the two interventions. Although the supplied results indicate a statistically significant increase in PGE2 following both interventions compared with their respective controls ($p \leq 0.001$), the difference between LLLT and MOP in Group III was reported as statistically nonsignificant ($p > 0.05$). Thus, both interventions were associated with increased PGE2 levels, without a statistically significant difference between the two modalities in the direct comparison. As presented in **Table 3**, greater canine retraction was reported on the intervention sides than on the corresponding control sides across all three group comparisons. In Group I, total canine retraction from T0 to T2 was 2.3 ± 0.5 mm on the LLLT side, compared with 0.9 ± 0.2 mm on the control side. During T0–T1, retraction was 1.0 ± 0.2 mm with LLLT and 0.4 ± 0.1 mm on the control side; during T1–T2, the corresponding values were 1.3 ± 0.6 and 0.5 ± 0.1 mm. In Group

II, total canine retraction was 3.8 ± 0.9 mm on the intervention side and 1.8 ± 0.5 mm on the control side. Retraction during T0–T1 was 1.8 ± 0.3 mm and 1.0 ± 0.4 mm, respectively, while during T1–T2 it was 1.1 ± 0.4 mm and 0.7 ± 0.1 mm. For Group III, the reported total retraction values were 2.2 ± 0.4 mm and 4.3 ± 0.8 mm for the two intervention sides. The corresponding interval-specific values were 0.8 ± 0.1 and 1.3 ± 0.2 mm during T0–T1 and 1.3 ± 0.3 and 2.6 ± 0.6 mm during T1–T2. The supplied results indicate statistically significant differences in canine retraction ($p \leq 0.05$). Overall, the tabulated findings show greater total retraction on the intervention sides than on the corresponding control sides in Groups I and II, while the direct comparison in Group III requires confirmation of the treatment labels before determining which modality produced greater retraction.

Table 1: Combined comparison of interleukin-1β (pg/µl) at control and experimental sites (lllt & mop) at different time intervals

Time Interval	Group I Control (LLLT) Mean ± SD	Group I Experimental (LLLT) Mean ± SD	Group II Control (MOP) Mean ± SD	Group II Experimental (MOP) Mean ± SD
T0 (Baseline)	3.2 ± 0.9	4.5 ± 1.1	4.3 ± 1.4	5.1 ± 1.8
T1	8.4 ± 1.8	25.1 ± 3.6	10.4 ± 2.3	16.6 ± 2.7
T2 (4 weeks)	12.3 ± 2.1	56.3 ± 2.8	11.7 ± 3.1	39.3 ± 4.5
T3 (8 weeks)	14.7 ± 2.4	67.7 ± 3.5	15.2 ± 2.9	55.4 ± 5.1

Table 2: Combined comparison of il-1β and prostaglandin e2 (pg/µl) at different time intervals

Time Interval	LLLT Side Mean ± SD	MOP Side Mean ± SD
A. Interleukin-1β (Group III: LLLT vs MOP in Same Subjects)		
T0 (Baseline)	4.9 ± 1.1	5.8 ± 1.3
T1	24.1 ± 2.7	17.4 ± 3.3
T2 (4 weeks)	52.3 ± 5.1	37.8 ± 4.4
T3 (8 weeks)	66.1 ± 5.4	57.1 ± 5.2
B. Prostaglandin E2 (Group I: LLLT vs Control)		
T0 (Baseline)	0.0114 ± 0.001	0.0113 ± 0.001
T1	0.0251 ± 0.003	0.1159 ± 0.01
T2 (4 weeks)	0.0653 ± 0.004	0.4712 ± 0.01
T3 (8 weeks)	0.0914 ± 0.004	0.4926 ± 0.01
C. Prostaglandin E2 (Group II: MOP vs Control)		
T0 (Baseline)	0.012 ± 0.001	0.0153 ± 0.001
T1	0.0247 ± 0.002	0.1204 ± 0.002
T2 (4 weeks)	0.0669 ± 0.003	0.4038 ± 0.02
T3 (8 weeks)	0.0956 ± 0.002	0.4841 ± 0.04
D. Prostaglandin E2 (Group III: LLLT vs MOP in Same Subjects)		
T0 (Baseline)	0.0113 ± 0.001	0.016 ± 0.001
T1	0.1159 ± 0.02	0.125 ± 0.02
T2 (4 weeks)	0.4212 ± 0.04	0.4107 ± 0.03
T3 (8 weeks)	0.4926 ± 0.03	0.4816 ± 0.039

Statistical Significance: Significant difference ($p \leq 0.05$)

Table 3: Combined Comparison of Canine Retraction (mm) at Different Time Intervals

Time Interval	Control Mean ± SD (mm)	LLLT Mean ± SD (mm)
A. Group I (LLLT vs Control)		
T0–T1	0.4 ± 0.1	1.0 ± 0.2
T1–T2	0.5 ± 0.1	1.3 ± 0.6
Total (T0–T2)	0.9 ± 0.2	2.3 ± 0.5
T0–T1	1.0 ± 0.4	1.8 ± 0.3
T1–T2	0.7 ± 0.1	1.1 ± 0.4
Total (T0–T2)	1.8 ± 0.5	3.8 ± 0.9
T0–T1	0.8 ± 0.1	1.3 ± 0.2
T1–T2	1.3 ± 0.3	2.6 ± 0.6
Total (T0–T2)	2.2 ± 0.4	4.3 ± 0.8

Statistical Significance: Significant ($p \leq 0.05$)

Discussion:

Orthodontic tooth movement (OTM) is a biomechanically driven process with the remodeling of alveolar bone regulated by the biology. A series of inflammatory events which are triggered by cytokines, chemokines and prostaglandins released from the periodontal ligament (PDL) and gingival crevicular fluid (GCF) mediate this remodeling process. These mediators include Interleukin-1beta (IL-1β) and Prostaglandin E2 (PGE2) which is both believed to be important factors in the severity of the inflammatory reaction and the speed of tooth movement **[2]**. The present study has compared and studied the effects of both invasive (micro-osteoperforations, MOP) and non-invasive (low-level laser therapy, LLLT) methods on these mediators and correlation them with clinical tooth movement. The results obtained in the present study showed a gradual and statistically significant rise in the level of IL-1 β from baseline (T0) to 8 weeks (T3), in both control and experimental group. The rise was however significant in experimental groups suggesting that both LLLT and MOP, significantly promotes the biological mechanisms associated with orthodontic tooth movements. This finding coincides with previous research, which showed that IL-1β is an important molecule for osteoclast activation and bone resorption in OTM **[4]**. With LT group I, a significant and persistent rise in IL-1β levels was obtained, peaking at T3. The increase at T1 was over five times 'as much' and rose further afterwards. Together this indicates that LLLT is highly stimulatory by photobiomodulation on the release of cytokines. The mechanism of action of LLLT is hyperactivity of mitochondria, ATP synthesis and modulation of ROS, ultimately affecting the cellular signalling and release of inflammatory mediators **[6]**. In the same manner, Jain *et al.*, noted increased levels of IL-1β after laser irradiation and linked with a faster tooth movement **[7]**. However, Group II (MOP) also showed a significant rise of IL-1β in comparison to the control but to some extent as compared to the LLLT group at corresponding time intervals. In the area of the injury induced by MOP, the bone turnover and the release of cytokines are increased due to the regional acceleratory phenomenon (RAP) that is triggered by this loading device. Elevated level of IL-1β after MOP in this study is in line with the results of previous studies conducted by Alikhani *et al.*, which it demonstrated that the planoplex type of micro-osteoperforations leads to increase the level of inflammatory mediator **[8]**. Interestingly, in group III, both interventions were applied in the same patient; the concentration of Il-1B was significantly higher on the LLLT side than on the MOP side ($p \leq 0.05$) was found. Thus it is believed that LLLT could result in a stronger biochemical reaction compared to MOP. It should be emphasized, however, not the rate of tooth movement is completely dependent on the biological response; mechanical and structural factors also have a significant influence **[11]**. A review of the levels of PGE2 in the evaluation also supports the

involvement of inflammatory mediators in orthodontic tooth movement. The PGE2 level significantly increased in both Group I and Group II experimental doses as compared to control doses in both the groups, reaching their highest values at T2 and T3. PGE2 is known to stimulate osteoclastic activity and bone resorption, which will allow tooth movement **[13]**. Thus, the greatly increased level of PGE2 in both the LLLT and the MOP group are affirmatory of the two treatments being effective for stimulating bone remodeling processes. The comparison between LLLT and MOP in Group III showed that there was no statistically significant difference for levels of PGE2 ($p > 0.05$), however. Both modalities seem to provide an equivalent effect on PGE2 activity release. The present results are consistent with previous work that showed that numerous cues such as mechanical stress, laser illumination and surgical injuries trigger PGE2 production as a common downstream pathway **[16].** The clinical results (canine retraction) showed a different pattern, although LLLT showed a better The data revealed that both LLLT and MOP were effective in the acceleration of tooth movement in contrast to its control groups. MOP did go beyond LLLT though, in terms of the amount of dogging of the doll carts. In group I, the total retraction of the dog was 2.3 mm, whereas, for the control group, the total retraction was 0.9 mm showing significant raise in canine retraction. This is in line with the previous research performed by Markovic *et al.* who showed a higher cellular activity and bone remodeling with LLLT as a result of enhanced tooth movement **[17]**. For in Group II, MOP led to a canine movement of 3.8 mm as compared to 1.8 mm in the control group, which shows stronger effect than the effect of LLLT. One potential explanation for the increase in tooth movement with MOP is that it causes an induction of RAP, temporarily decreasing bone density, which activates more bone remodeling activity, allowing for more rapid tooth movement **[9, 18].** Rao *et al.* reported faster mandibular anterior decrowding with piezocision than with MOP. This suggests that treatment effects may vary by surgical technique and outcome **[19]**. Moreover, the patients in Group III (with both interventions) showed a significantly higher degree of canine retraction in the MOP group (4.3 mm) than in the LLLT group (2.2 mm). These findings indicate the strong clinical effectiveness of the MOP approach compared to LLLT even though certain inflammatory mediators were measured in greater density in the LLLT treatment. This seeming paradox indicates that biochemical markers like IL-1β and PGE2 are important markers of biological activity, but the overall rate of tooth motion depends on other factors like bone density, mechanical resistance and magnitude of surgical stimulation **[20]**. The nature of cytokine release was also observed in this study to be another point of interest. The level of both IL-1β and PGE2 increased progressively over the 8 weeks. This significant increase was seen during the first 4 weeks immediately after beginning orthodontic therapy, a time of active inflammation from tooth movement. The concentrations of IL-1β and PGE2 also rose between 4-8 weeks after initiation, but not to the extent it had been in the first 4-week period. This indicates that inflammatory activation is decreasing, not that the protein in question is being down regulated, meaning that it is slowing down, not stopping, in its biological response. It follows the pattern of the inflammatory response found in nature; after a peak period, remission takes place as tissue remodelling proceeds to normal levels **[21]**. This means it has important clinical implications. LLLT is a non-invasive and patient-friendly with good biochemical stimulation effect, while MOP is better for acceleration of teeth movement clinically. So treatment choice should depend on the patient's preference, clinical needs and site-specific invasiveness and treatment efficacy. Castillo-Montaño *et al.* **[22]** found variable evidence for invasive and non-invasive acceleration methods. Differences in study design and outcomes limit direct comparisons. But there are a few drawbacks to the study, namely its limited number of participants and limited length of time, which could limit the overall conclusions drawn. Further validation of these findings and long-term effects of the interventions are recommended to be investigated in future studies with larger numbers, longer follow-up periods and other biomarkers.

Conclusion:

Both low-level laser therapy and micro-osteoperforations significantly enhanced inflammatory mediator release and accelerated orthodontic tooth movement. Micro-osteoperforations achieved faster canine retraction, whereas low-level laser therapy elicited a stronger biochemical response, particularly in interleukin-1 beta expression. Thus, data support micro-osteoperforations as a more effective approach for accelerating tooth movement while highlighting low-level laser therapy as a less invasive alternative with favourable biological effects.

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