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Research Article

Oral Biological and Periodontal Inflammatory Responses Following Endodontic Treatment of Endo-Perio Lesions

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ABSTRACT
Background: Endodontic-periodontal lesions are complex lesions that involve a combination of pulpal infection and periodontal inflammation. Pathological and anatomical connections between the pulp and periodontal tissues make it possible that a pulp infection could help to maintain periodontal tissue destruction and continuing inflammation. A comprehensive assessment of healing after endodontic therapy may be achieved by the evaluation of both clinical parameters and inflammatory markers.
Objective: To evaluate oral biological and periodontal inflammatory responses following endodontic treatment of endo-perio lesions.
Methods: This prospective clinical study included 75 patients with confirmed endodontic-periodontal lesions and was conducted from January 2025 to June 2025 at Women Dental College, Abbottabad. The parameters evaluated at baseline, 3 months and 6 months included probing pocket depth (PPD), clinical attachment loss (CAL), bleeding on probing (BOP), gingival index (GI) and plaque index (PI). Interleukin-1 beta (IL-1β), interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) levels were measured in gingival crevicular fluid samples. Radiographic healing was also assessed as was clinical symptoms. Appropriate statistical tests, repeated measures and comparative were used to analyze data and a p value of < 0.05 was deemed to be statistically significant.
Results: Mean PPD decreased from 6.18 ± 1.27 mm at baseline to 3.29 ± 0.89 mm at six months, while mean CAL improved from 6.74 ± 1.42 mm to 3.91 ± 1.02 mm ($p < 0.001$). BOP declined from 72.4 ± 18.6% to 25.8 ± 13.9%, accompanied by significant improvement in GI and PI. Mean IL-1β decreased from 86.3 ± 22.7 to 43.5 ± 14.9 pg/mL, IL-6 from 41.7 ± 12.6 to 19.6 ± 7.7 pg/mL, and TNF-α from 32.8 ± 9.7 to 16.4 ± 6.2 pg/mL at six months (all $p < 0.001$). Overall treatment success was observed in 64 (85.3%) patients. Greater reductions in inflammatory biomarkers were associated with more pronounced periodontal improvement.
Conclusion: Endodontic treatment of endo-perio lesions was associated with significant improvement in periodontal health, reduction in local inflammatory cytokines, symptom resolution, and favorable radiographic healing. Monitoring IL-1β, IL-6, and TNF-α alongside conventional periodontal parameters may provide additional insight into biological healing following treatment.

Keywords: Endo-Perio Lesion, Endodontic Treatment, Periodontal Inflammation, Il-1β; Il-6, Tnf-A, Probing Pocket Depth, Clinical Attachment Level.

INTRODUCTION

Endodontic-periodontal lesions are more complex lesions encountered in clinical dentistry as they are double and/or multiple disease processes affecting both dental pulp and periodontal tissues. Pulp and periodontium are mutually connected by means of the apical foramen, accessory and lateral canals, dentinal tubules and regions of structural damage. These pathways may allow microorganisms, toxins and inflammatory mediators to pass from the root canal to the surrounding periodontal

tissues. Therefore, disease with a primary focus in the pulp can manifest itself in the periodontal tissues, and more extensive periodontal infection can have a negative effect on the pulp (1-3).

Persistent microbiologic insults and the host inflammatory response seem to be closely associated with the biological basis of endo-perio lesions. The bacterial byproducts in the infected root canals cause inflammation of the soft tissue, periodontal attachment loss and bone resorption. The cytokines most commonly involved in these processes are interleukin-1 beta (IL-1β), interleukin-6 (IL-6), and tumour necrosis factor alpha (TNF-α). Such mediators are involved in leukocyte recruitment, activation of osteoclasts, degradation of connective tissue and amplification of local inflammatory response and they could be used as biological markers of disease activity (4-6).

Probing pocket depth, clinical attachment level, bleeding on probing, probing mobility, percussion response, pulpal testing and radiographic assessment are all used in the traditional clinical evaluation of endo-perio lesions. While these measurements are still necessary for diagnosis and for follow-up, they are primarily used to describe the structural and clinical effects of the disease. Determination of inflammatory markers in GCF or periodontal pocket fluid may be able to give additional information about the biological activity of the lesion and the extent of the resolution of the inflammation after the treatment (7, 8).

Endodontic therapy is successful when the microbial challenge in the root canal system is either controlled or eliminated, reinfection is prevented and periapical and periodontal tissue healing occurs. If there is a significant endodontic involvement in the lesions, proper disinfection of the canal can result in a decrease in intra-lesional inflammation and a positive effect on the surrounding periodontal condition. The extent and course of these biological changes can, however, not be fully reflected by the clinical or radiographic evaluation. It may therefore be useful to follow inflammatory cytokines serially to understand if there is clinical improvement with measurable suppression of local inflammatory activity (9).

Although there is increasing awareness of the relationship between pulpal and periodontal inflammation, little clinical evidence has been available to evaluate both periodontal healing and the alteration in inflammatory biomarkers in the oral cavity after endodontic treatment of endo-periodontal lesions. Previous studies have mainly studied only clinical periodontal parameters, microbiological aspects, or one or a few inflammatory mediators, and fewer studies have examined these aspects in a longitudinal fashion (10, 11).

The present study was therefore designed to evaluate oral biological and periodontal inflammatory responses following endodontic treatment of endo-perio lesions**.** Specifically, it assessed changes in probing pocket depth, clinical attachment level, bleeding on probing, gingival and plaque indices, and the inflammatory biomarkers IL-1β, IL-6, and TNF-α from baseline to follow-up. It also examined clinical symptom resolution, radiographic healing, and the relationship between biomarker reduction and periodontal improvement.

METHODOLOGY

This prospective clinical study was conducted at the Women Dental College, Abbottabad from January 2025 to June 2025. A total of 75 patients diagnosed with endodontic-periodontal (endo-perio) lesions were enrolled in the study. Patients were recruited through consecutive sampling from those presenting to the outpatient dental department during the study period. Before enrollment, each participant underwent a detailed clinical and radiographic assessment to confirm the diagnosis and determine eligibility for inclusion.

The inclusion criteria were for patients (both genders) age 18 years or older who had an endo-perio lesion in a permanent tooth that needed endodontic treatment. Differential diagnosis was made using the clinical periodontal examination, pulpal vitality, percussion, probing of pockets, tooth mobility, and the periapical radiographs. Patients were excluded if they had an uncontrolled systemic condition, a history of recent periodontal or endodontic therapy, current antibiotic or anti-inflammatory therapy, pregnancy, immunocompromise status, or teeth with vertical root fracture, hopeless periodontal prognosis and non-restorable crowns. Those who did not attend the appointments were also not included in the final analysis.

The data collection form was structured and used in order to gather demographic and clinical data at baseline. The variables covered were age, sex, type of tooth, involved arch, presenting symptoms, pain, swelling, sinus tract, and tenderness to percussion and tooth mobility. Next, a thorough examination of the gums was conducted. The pocket depth (PPD)

and clinical attachment level (CAL) were recorded in mm by a calibrated periodontal probe. Bleeding on probing (BOP) was measured as percentage of sites that bled on probing, gingival status was measured using Gingival Index (GI) and plaque status measured using Plaque Index (PI). Periodontal measurements were made with standardised technique to minimise inter-examination variation.

Standardised intraoral periapical radiographs at baseline and follow-up visits were taken for radiographic assessment. The presence and extent of periapical pathology and alveolar bone loss were documented. In case of periapical radiolucency, its grade was classified using the Periapical Index (PAI). The same viewing conditions were used for the evaluation of the radiographs to ensure consistency. The radiographic healing was graded at the last follow-up as complete healing, partial healing, or limited/no healing, depending on the reduction or resolution of the periapical radiolucency and improvement in the surrounding bone architecture.

Biological assessment involved collection of samples by inserting sterile absorbent paper strips into the periodontal pocket or the gingival crevicular fluid next to the affected tooth, after careful isolation and drying of the area. Any samples that were tainted with saliva/blood were disregarded and re-collected. Samples were collected and placed in sterile tubes and stored under the proper laboratory conditions until analyzed. Selected inflammatory biomarkers including interleukin-1 beta (IL-1β), interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) were quantified using commercially available enzyme-linked immunosorbent assay (ELISA) kits, following the manufacturer's directions. The concentrations of biomarkers were expressed in pg/mL.

The teeth included were treated with nonsurgical root canal therapy at a standard level. Local anesthesia was administered, and an access cavity was prepared. The working length was determined with an electronic apex locator and confirmed radiographically if necessary, after rubber-dam isolation. The root canals were mechanically prepared with copious irrigation with sodium hypochlorite solution and either rotary or hand instrumentation. After proper disinfection of the canals, they were then filled with sterile paper points, dried and obturated with gutta-percha and an appropriate root canal sealer. Access cavity restored to give good coronal seal. Standard postoperative care instructions and oral hygiene instruction were given to patients. The participants were re-evaluated at 3 months and 6 months after treatment. Clinical symptoms and periodontal parameters were re-evaluated with the same methods as at baseline at every follow-up visit. PPD, CAL, BOP, GI, and PI were documented and biological samples were re-collected for measurement of IL-1 β, IL-6, and TNF-α. Other signs recorded included pain, swelling, tenderness to percussion, sinus tract and mobility of the tooth. Radiographic examination was done again at the 6-month visit to evaluate periapical and periodontal healing. The overall treatment success was considered to be when clinical symptoms were resolved or significantly reduced, together with improvement in periodontal parameters and evidence of radiographic healing.

Primary outcomes of the study were changes in PPD, CAL, BOP, IL-1β, IL-6 and TNF-α from baseline to follow-up. Secondary outcomes were change in GI and PI, resolution of clinical symptoms, tooth mobility, radiographic healing, and overall treatment success. The periodontal improvement was also assessed based on the amount of PPD loss and CAL gain. The associations between the decreases in the levels of inflammatory biomarkers and periodontal healing were evaluated to examine biological response in relation to the clinical improvement that occurred after endodontic treatment.

IBM SPSS Statistics version 26 was used to enter and analyze the data. Categorical variables were described as frequencies and percentages and continuous variables were described as mean ± SD or median (IQR), as appropriate. When the variables were normally distributed, repeated-measures analysis of variance tests were used to determine changes at baseline, 3 months, and 6 months between the variables of continuous periodontal measurements and continuous inflammatory variables. When variables did not meet the normality assumption, repeated-measures Friedman tests were used instead. Appropriate post-hoc testing was done after paired comparisons. McNemar's test or Cochran's Q test was used to analyze the changes in clinical findings by categories over time. Independent-samples t-test or Mann-Whitney U test was used to compare continuous variables between treatment-success groups, while chi-square or Fisher's exact test was used for categorical variables. To identify factors independently

associated with successful healing, multivariable logistic regression analysis was performed. Variables considered clinically relevant or showing an association on univariable analysis were entered into the model. Adjusted odds ratios (AORs) with 95% confidence intervals were reported. A **p-value <0.05** was considered statistically significant.

RESULTS

A total of 75 patients with endo-perio lesions completed the study and were included in the final analysis. The participants' mean age was 39.6 ± 10.8 years ranging from 20 to 61 years. Of the total participants, 41 (54.7%) were male and 34 (45.3%) were female. Forty-two patients (56.0%) had mandible teeth while 33 (44.0%) had maxilla teeth. The most common tooth treated was the molars with 37 (49.3%) cases.

Table 1. Baseline demographic and clinical characteristics of study participants (n = 75)

Variable	Frequency (%) / Mean ± SD
Age, years	39.6 ± 10.8
Male	41 (54.7%)
Female	34 (45.3%)
Maxillary teeth	33 (44.0%)
Mandibular teeth	42 (56.0%)
Incisor/canine	12 (16.0%)
Premolar	26 (34.7%)
Molar	37 (49.3%)
Pain at presentation	48 (64.0%)
Tenderness to percussion	45 (60.0%)
Swelling	21 (28.0%)
Sinus tract	17 (22.7%)
Tooth mobility present	28 (37.3%)

All study subjects had signs of periodontal inflammation at baseline. After endodontic treatment, there was a gradual improvement in the periodontal parameters, with mean probing pocket depth of 6.18 ± 1.27 mm and mean clinical attachment level of 6.74 ± 1.42 mm. In the same manner, the mean clinical attachment level (mm) at baseline was 6.74 ± 1.42 mm, which increased to 4.93 ± 1.20 mm at the 3-month follow-up and 3.91 ± 1.02 mm at the 6-month follow-up. These decrease were statistically significant ($p < 0.001$).

Table 2. Changes in periodontal clinical parameters following endodontic treatment

Parameter	Baseline	3 months	6 months	p-value
Probing pocket depth, mm	6.18 ± 1.27	4.37 ± 1.08	3.29 ± 0.89	<0.001
Clinical attachment level, mm	6.74 ± 1.42	4.93 ± 1.20	3.91 ± 1.02	<0.001
Gingival index	2.21 ± 0.47	1.43 ± 0.39	0.91 ± 0.34	<0.001
Plaque index	1.86 ± 0.51	1.28 ± 0.42	0.93 ± 0.37	<0.001
Bleeding on probing, %	72.4 ± 18.6	43.7 ± 16.3	25.8 ± 13.9	<0.001

Bleeding on probing also showed a marked decline during follow-up. The mean proportion of bleeding sites decreased from 72.4 ± 18.6% at baseline to 43.7 ± 16.3% at three months and 25.8 ± 13.9% at six months. Gingival and plaque indices showed significant improvement over the same period, suggesting a decrease in the local periodontal inflammation following the treatment. Concentrations of inflammatory biomarkers also significantly decreased. Baseline mean IL-1β concentration was 86.3 ± 22.7 pg/mL, which declined to 61.8 ± 18.4 pg/mL at three months and 43.5 ± 14.9 pg/mL at six months. Mean IL-6 decreased from 41.7 ± 12.6 pg/mL to 28.9 ± 9.8 pg/mL and 19.6 ± 7.7 pg/mL, respectively. Likewise, TNF-α decreased from 32.8 ± 9.7 pg/mL at baseline to 23.1 ± 7.6 pg/mL at three months and 16.4 ± 6.2 pg/mL at six months. The results of all the biomarker changes were statistically significant ($p<0.001$).

Table 3. Changes in oral inflammatory biomarkers following endodontic treatment

Biomarker	Baseline	3 months	6 months	p-value
IL-1β, pg/mL	86.3 ± 22.7	61.8 ± 18.4	43.5 ± 14.9	<0.001
IL-6, pg/mL	41.7 ± 12.6	28.9 ± 9.8	19.6 ± 7.7	<0.001
TNF-α, pg/mL	32.8 ± 9.7	23.1 ± 7.6	16.4 ± 6.2	<0.001

Treatment also resulted in clinical symptom improvement. Of the 48 patients having pain at baseline, 11 (14.7%) had pain 3 months later, and only 4 (5.3%) at 6 months. The sensitivity to percussion at six months was significantly lower than at baseline (60.0% vs 8.0%, respectively) (P < 0.001). Likewise, the swelling decreased from 28.0% to 2.7% and the percentage of patients with a detectable sinus tract was reduced from 22.7% to 1.3%.

Table 4. Clinical response following endodontic treatment

Clinical finding	Baseline n (%)	3 months n (%)	6 months n (%)	p-value
Pain	48 (64.0%)	11 (14.7%)	4 (5.3%)	<0.001
Tenderness to percussion	45 (60.0%)	13 (17.3%)	6 (8.0%)	<0.001
Swelling	21 (28.0%)	6 (8.0%)	2 (2.7%)	<0.001
Sinus tract	17 (22.7%)	5 (6.7%)	1 (1.3%)	<0.001
Tooth mobility	28 (37.3%)	17 (22.7%)	10 (13.3%)	0.002

At six months, radiographic evaluation showed complete healing in 43 patients (57.3%), partial healing in 24 patients (32.0%) and limited or no improvement in 8 patients (10.7%). Of the 64 patients (85.3%), clinical treatment success (complete resolution of symptoms, periodontal improvement and radiographic improvement) was noted.

Table 5. Radiographic and overall treatment outcomes at six months

Outcome	n (%)
Complete radiographic healing	43 (57.3%)
Partial radiographic healing	24 (32.0%)
Limited/no radiographic healing	8 (10.7%)
Overall successful treatment	64 (85.3%)
Unsuccessful/limited response	11 (14.7%)

A decrease in inflammatory markers was correlated with better periodontal healing. Patients that demonstrated a decrease in probing pocket depth of 2 mm or more exhibited greater mean decrease in IL-1β, IL-6 and TNF-α than those with less significant periodontal improvement. This relationship was highest for reduction in IL-1β followed by TNF-α and IL-6.

Table 6. Association between inflammatory biomarker reduction and periodontal improvement at six months

Variable	Patients with ≥2 mm PPD reduction (n = 59)	Patients with <2 mm PPD reduction (n = 16)	p-value
Reduction in IL-1β, pg/mL	46.8 ± 16.4	25.7 ± 12.9	<0.001
Reduction in IL-6, pg/mL	24.5 ± 9.3	13.2 ± 7.6	<0.001
Reduction in TNF-α, pg/mL	18.1 ± 7.4	10.3 ± 6.2	<0.001
CAL improvement, mm	3.08 ± 0.94	1.47 ± 0.68	<0.001

In multivariable analysis, a greater reduction in IL-1β levels and lower residual probing pocket depth were independently associated with successful treatment. Patients demonstrating a substantial IL-1β decline had approximately 2.7-fold higher odds of successful healing, while persistent deep periodontal pockets were associated with a reduced probability of treatment success.

Table 7. Multivariable predictors of successful clinical and radiographic healing

Predictor	Adjusted OR	95% CI	p-value

Greater reduction in IL-1β	2.71	1.26–5.84	0.011
Greater reduction in TNF-α	2.08	1.03–4.21	0.041
Baseline PPD ≥7 mm	0.46	0.22–0.96	0.039
Tooth mobility at baseline	0.52	0.25–1.09	0.083
Improvement in CAL ≥2 mm	2.89	1.31–6.38	0.009

Overall, the findings demonstrated a parallel improvement in periodontal status, clinical symptoms, radiographic healing, and oral inflammatory biomarker levels following endodontic treatment of endo-perio lesions. The decline in IL-1β, IL-6, and TNF-α was particularly evident among patients with greater periodontal pocket reduction and attachment gain, indicating that biological inflammatory responses corresponded closely with clinical healing.

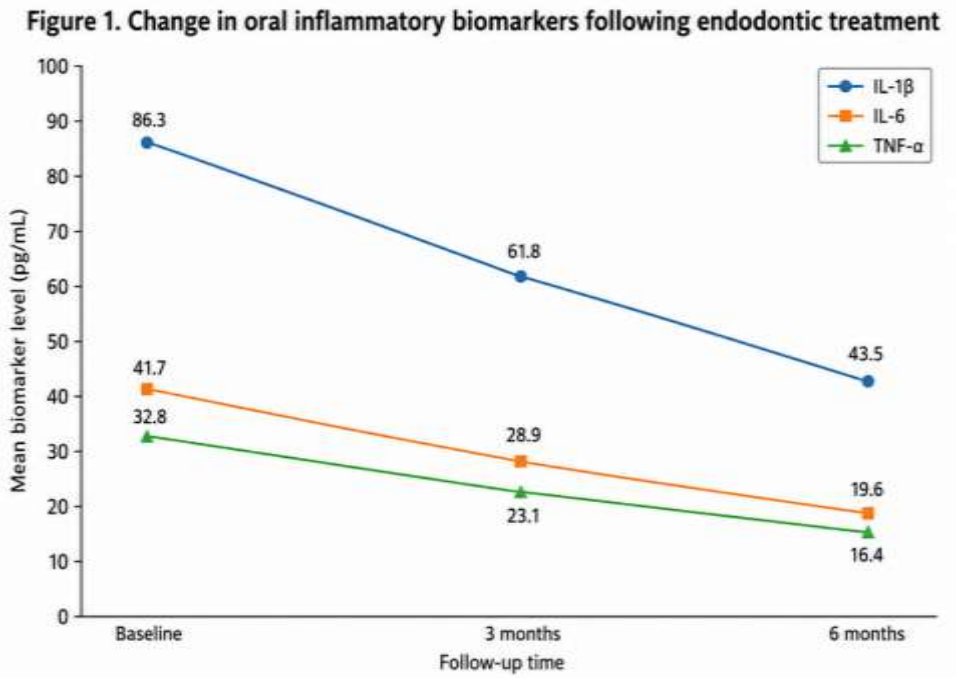

Figure 1. Change in oral inflammatory biomarkers following endodontic treatment of endo-perio lesions.

Line graph showing the mean levels of IL-1β, IL-6, and TNF-α at baseline, 3 months, and 6 months after endodontic treatment. A progressive decline in all three inflammatory biomarkers was observed over the follow-up period, indicating a favorable biological and periodontal inflammatory response after treatment.

DISCUSSION

The present study demonstrated substantial improvement in periodontal clinical parameters and oral inflammatory responses following endodontic treatment of endo-perio lesions. Over the six-month follow-up period, probing pocket depth (PPD), clinical attachment level (CAL), bleeding on probing (BOP), gingival index (GI), and plaque index (PI) all improved significantly. At the same time, concentrations of the pro-inflammatory cytokines IL-1β, IL-6, and TNF-α progressively decreased. Clinical symptoms, including pain, tenderness to percussion, swelling, sinus tract formation, and tooth mobility, also became less frequent, while the majority of treated teeth showed complete or partial radiographic healing. Taken together, these findings suggest that elimination of the endodontic infectious source may have favorable effects not only on periapical tissues but also on the associated periodontal inflammatory environment (12-15).

The present study indicated that there was a biological relationship between pulpal infection and periodontal inflammation, in that the decrease in pocket depth and increase in clinical attachment were demonstrated. Endodontic-periodontal lesions are those where the root canal system communicates with the periodontal tissues through the apical foramen, accessory canals, dentinal tubules or regions of structural damage. As a result, both compartments can be impacted by

microorganisms and inflammatory products. Current recommendations still stress the need for proper diagnosis and consideration of treating the endodontic problem in a combined lesion where it is a factor. Similarly, when studies were performed on PECLs, those that were treated with root canal treatment alone showed improvement in probing depth, bleeding index, plaque index, and gingival index, while those that were treated with both root canal and periodontal therapy showed greater improvement in these parameters. These observations are congruent with the clinical improvements observed in our patients in the periodontal parameters (16, 17).

Progressive decreases in IL-1β, IL-6, and TNF-α were an important finding in the present investigation. These cytokines have important roles as mediators of tissue destruction during inflammatory processes and may mediate osteoclastic activity leading to alveolar or periapical bone resorption. Increased levels of pro-inflammatory cytokines, such as tumour necrosis factor (TNF-α) and interleukin (IL-1β), have been detected in gingival crevicular fluid from teeth with apical periodontitis, which suggests that these molecules may be objective markers of endodontic inflammatory activity. Similarly, a clinical study of combined endodontic-periodontal lesions found that chemomechanical preparation successfully decreased microbial components, lipopolysaccharide concentrations, IL-1β and TNF-α in both the periodontal and root canal regions. The results therefore confirmed the hypothesis that the elimination of the root canal microbial load will reduce the inflammatory stimulus to the surrounding periodontal tissues (18).

The reduction in IL-6 noted in our study is also consistent with periodontal treatment and combined endodontic-periodontal treatment studies. In previous clinical studies, in which combined periodontal-endodontic lesions were present, levels of both IL-6 and IL-1β were reduced and periodontal indices were improved after root canal therapy and basic periodontal therapy. A meta-analysis of inflammatory responses after periodontal treatment also revealed that there was a significant decrease in serum and GCF IL-6 after periodontal treatment. Likewise, studies of gingival crevicular fluid biomarkers found that IL-6, IL-1β, and TNF-α tend to be lower during healing, especially at the intermediate (1- to 3-month) and 3-month time points. The progressive reduction in cytokine levels over the course of our study (baseline to three months to six months) would therefore seem to be biologically compatible with progressive resolution of local inflammatory activity (19, 20).

Another key aspect of treatment response was symptom resolution and radiographic healing. Pain, tenderness to percussion, swelling, and sinus tract were relatively common at baseline, but decreased significantly during follow-up. 57.3% of patients had complete radiographic healing and 32.0% had partial healing by 6 months of treatment. The results of this study are consistent with the established inflammatory etiology of apical periodontitis where immune activation by microorganisms leads to bone resorption and destruction of the periapical tissues. Studies further suggest that the treatment of endodontic infection can also decrease levels of circulating inflammatory mediators such as IL-1β, further highlighting the broader biologic effects of endodontic infection control. It is possible, in our study, that the clinical and radiographic improvement is the result of microbial clearance, decrease in inflammatory signaling and subsequent repair of the periodontal and periapical tissues (20).

Another significant factor of treatment response was clinical symptom resolution and radiographical healing. Baseline, pain, tenderness to percussion, sinus tract formation and swelling were relatively common but decreased significantly with follow-up. At 6 months, 57.3% of patients had complete healing, and 32.0% had partial healing. The results are consistent with the known inflammatory pathogenesis of apical periodontitis, in which the presence of microorganisms results in immune activation, bone loss, and destruction of periapical tissues. Further studies have shown that successful endodontic therapy can also lower the levels of other inflammatory mediators, such as IL-1β, in the blood stream, as part of its broader biological effects on controlling endodontic infection. Therefore, the clinical and radiographic improvement observed in the present study may be attributed to the elimination of microorganisms, decreased inflammatory signaling and repair of periodontal and periapical tissues (10).

Several limitations should be considered when interpreting these findings. The study was carried out in one center and the sample size was relatively small (75 patients), limiting the generalizability of the findings. Follow-up period of six months was enough to show the

early and intermediate clinical and biological improvement but cannot be seen as a long-term tooth survival or recurrence. Furthermore, the present study assessed three of the major inflammatory biomarkers, whereas other inflammatory mediators that play a role in periodontal and periapical inflammation were not evaluated. Additionally, smoking, systemic inflammatory status, oral hygiene behavior, lesion classification, root morphology, and microbiological composition may be factors that affect the response to treatment. Therefore, further multicenter studies using a larger patient population, longer follow-up periods, microbiological evaluation with more comprehensive panels of inflammatory and regenerative investigations are warranted. These studies can be useful for determining if biomarker monitoring can become a standard part of predicting healing and prognosis for patients with endodontic-periodontal lesions.

CONCLUSION

Endodontic treatment of endo-perio lesions was associated with significant improvement in both periodontal clinical status and oral inflammatory responses. Significant reductions in probing pocket depth, clinical attachment loss, bleeding on probing, gingival inflammation, and plaque accumulation were accompanied by progressive decreases in IL-1β, IL-6, and TNF-α over six months. Clinical symptoms also resolved substantially, and the majority of treated teeth demonstrated complete or partial radiographic healing.

Greater reductions in inflammatory biomarkers, particularly IL-1β, were associated with more pronounced periodontal improvement and a higher likelihood of successful healing. These findings emphasize the biological interrelationship between pulpal and periodontal inflammation and suggest that controlling the endodontic infectious source may contribute substantially to periodontal and periapical recovery. Measurement of inflammatory biomarkers alongside conventional clinical and radiographic parameters may provide additional information for assessing treatment response and prognosis in endo-perio lesions.

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