PERIODONTAL REGENERATION WITH AND WITHOUT ENDODONTIC TREATMENT: A COMPARATIVE STUDY

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ABSTRACT

It is frequently considered that the endodontic treatment have an effect on the periodontal regeneration. However, contradictory conclusions have been drawn by various investigators, thus it remains doubtful. Considering this, the present study was carried out to evaluate the periodontal regeneration with and without endodontic treatment involving 36 sites, comprising control (n=18, received only open flap debridement) and case sites (n=18, received combined open flap debridement and endodontic therapy) in 12 subjects having at least 2 sites of periodontal defects. Gingival index, probing pocket depth, clinical attachment levels were assessed clinically, while bony defects were assessed radiographically. All the parameters were recorded on day 0 (baseline) and at the end of 3 and 6 months postsurgically. Significant improvement was observed in all the parameters in the case sites compared to that of control sites, except gingival health which showed no difference. This may suggest a positive influence of endodontic treatment on the outcome of periodontal therapy.

KEYWORDS: Regeneration, Probing Pocket Depth, Gingival Index, Endodontic Treatment, Flap

INTRODUCTION

The pulp and periodontium have embryonic, anatomic, and functional interrelationships (Ten Cate, 1994). Anatomically and functionally, they are so closely related that they influence each other during health, function and disease (Mandel et al., 1993). So, infection of one structure may spread to the other. Pulpal disease may cause periodontal changes through lateral canals, accessory canals, accessory and apical foramina. In this case, periodontal infection may initiate at the apical region and progresses coronally to involve the marginal gingiva, referred to as retrograde periodontitis (Simring and Goldberg, 1964). Similarly, pulpitis may also occur as an extension of periodontitis through apical foramen, being started at apical region and extends coronally to involve pulp chamber, referred to as “retrograde pulpitis” (Solomon et al., 1995). The relationship between periodontal and pulpal disease was first described by Simring and Goldberg in 1964. Since then, the term, ‘perio-endo lesion’ has been used to describe such lesions considering the presence of inflammatory products in both the periodontium and the pulpal tissues, though in varying degrees (Simring and Goldberg, 1964).

Periodontal and endodontic diseases have some common symptoms and one disease may mimic the other, clinically or radiographically. Hence, symptoms such as a deep periodontal pocket, swelling of the marginal gingiva, fistulae, tenderness to percussion, increased mobility and angular bone destruction are not exclusively associated with the plaque-associated periodontal disease, but may also be seen in retrograde periodontitis (Simring and Goldberg, 1964). Loss of alveolar bone, either horizontal or vertical, is a common consequence of periodontal disease and results in loss of support and mobility of the involved teeth. In contrast to periodontal disease, the attachment apparatus is lost as a result of
apical extension of pulpal pathosis with no loss of marginal bone in pulpal disease. Again, the clinical attachment loss seen in periodontal disease is rarely regained, while complete regeneration of the periapical bone is routinely achieved after endodontic therapy. Since the clinical and radiographic characteristics of endo-perio lesion regardless of its origin mimic each other, it is very difficult to diagnose the primary lesion, whether of pulpal or periodontal in origin. On the other hand, diagnosis of the primary lesion is important in order to preclude unnecessary and even detrimental treatment (Meng, 1999). However, the sequence in which the endodontic and periodontal treatments are to be performed depends on the identification of the primary etiologic factor. Quick elimination of this is regarded as the key to success in the management of an endo-perio lesion (Brannstrom and Nyborg, 1973).

In general, the endo-perio lesions demand for endodontic therapy adjunctive to periodontal therapy, treating one structure and leaving another untreated may not serve the purpose of the periodontal therapy (Goldman and Schilder, 1988; Solomon et al., 1995), which includes the elimination of gingival inflammation, reduction of pocket depth to the level of gingival sulcus, regeneration of the attachment apparatus and prevention of recurrence (Newman et al., 2007). Though both of the periodontal and endodontic therapies are recommended for successful management of combined endo-perio lesions, the influence of the endodontic treatment on periodontal regeneration is controversial (Stallard et al., 2012). Reduced level of cementogenesis (Morris 1957; 1960) and periodontal regeneration (Sanders et al., 1983) were observed in relation to endodontically-treated teeth, suggesting an inhibitory effect of endodontic treatment on periodontal wound healing. In contrast, Dunlap et al. (1981) failed to detect any deleterious effect of endodontic treatment on the periodontal tissues. Hence, the present study was conducted to evaluate the influence of endodontic treatment on periodontal regeneration clinically by comparing pre and postsurgical gingival health, probing pocket depth and clinical attachment level clinically with and without endodontic treatment and alveolar bone height using radiographs.

MATERIALS AND METHODS

The study was carried out on a total number of 12 subjects. They were selected on the basis of probing pocket depth of 5 mm or more with ≥ 3 mm of loss of clinical attachment level and bone loss in relation to more than one tooth. Subjects with known systemic ailments, or having any other oral lesions that influence the periodontal healing were not included in the study. They were explained the entire procedure in details and consent was obtained.

A full mouth clinical examination was carried out on all the subjects. The periodontal health status was recorded using gingival index (Silness and Loe, 1963), Clinical attachment level (CAL) and Probing pocket depth (PPD) on day 0 (baseline) and at the end of 3 and 6 months for both the control and case sites by a single investigator.

Initial scaling and root planing was performed. They were instructed on proper oral hygiene procedures. In the selected subjects, at least 2 sites were considered and the selected sites were divided into two groups: control and case. Control received only open flap debridement, while case received combined open flap debridement and endodontic therapy.

PPD and CAL were measured using UNC-15 probe at distofacial, midfacial and mesiofacial line angles and at centre of lingual surfaces, keeping the probe on vertical grooves prepared on the occlusal stent as a reference point to avoid clinical variations at different time points of measurement. The mean gingival score, PPD and CAL for a tooth were obtained by adding the four values per tooth which was then divided by four. Radiographs were used to assess the bony defects on day 0 (baseline) and at the end of 3 and 6 months postsurgically. To avoid variations in measurements at
different time points, standardization of the intraoral periapical radiographs was done by using paralleling technique. On radiographs, the distance from the cementoenamel junction (CEJ) to the alveolar bone crest and to the base of the defect was measured in horizontal and intrabony defects, respectively, using UNC-15 probe.

**Treatment Protocol**

Subjects selected for the study underwent phase I therapy and an additional endodontic treatment at the case site approximately 1 month before the periodontal surgical procedures.

**Endodontic Treatment**

The teeth of case group were treated endodontically. Access cavity was prepared and Protaper files (Dentsply) and gutta percha (Dentsply) points were used for cleaning, shaping and obturation of the root canals by the crown-down technique. A solution containing 5.25% hypochlorite and 0.9% saline was used as root canal irrigants. Zinc oxide-eugenol was used as root canal sealer. Glass ionomer cement was used for permanent restoration of the teeth.

**Surgical Procedure**

Routine preparation of extraoral site was carried out using Povidone Iodine and then local anaesthesia was instituted. Crevicular incision was given with no. 12 blade and full thickness flaps were reflected to visualize and access the surgical sites. Incision was given with the objective of preserve the gingiva as much as possible so that close approximation of the tissue is attained, thereby exposure of bone after suturing is not there and thus, facilitates healing. Granulation tissue from the inner surface of the flap was removed using Gracey curettes. Subgingival calculus was removed and root planing was performed. The site was irrigated copiously with saline to remove any blood clots and debris. Buccal and lingual flaps were approximated using 3-0 nonresorbable black silk suture by interrupted suturing technique followed by placement of the periodontal dressing.

The patients were instructed to rinse 0.12% chlorhexidine gluconate for 2 weeks. Additionally, a nonsteroidal anti-inflammatory agent was prescribed as needed for pain control. Periodontal dressing and sutures were removed 1 week after surgery and the subjects were instructed to gently brush the area with a soft bristled toothbrush. Oral hygiene was evaluated at each visit and during recall appointments. The patients were seen at 3 and 6 months postsurgically for evaluation. Healing was uneventful. The collected data were analysed statistically using SPSS (SPSS Inc, Chicago) version 16.0 software packages.

**RESULTS**

**Changes in Gingival Index (GI)**

The mean value of GI in control group was found to be 2.00 (range 1.75 - 2.00) on day 0. It is reduced to 1.50 (range 1.25 - 1.50) on day 90 from baseline and is further reduced to 1.25 (range 1.00 - 1.25) on day 180, which is 25% and 37.5%, respectively. The mean difference in GI on day 90 and 180 was found to be 0.25 (range 0.19 - 0.25), which is 12.5%.

The mean value of GI in case group was found to be 2.00 (range 1.75-2.00) on day 0, 1.50 (range 1.44 - 1.56) on day 90 and 1.00 (range 1.00 - 1.31) on day 180. Thus, in case group, GI is reduced to 1.50 (range 1.44-1.56) on day 90 from baseline and is further reduced to 1.00 (range 1.00 - 1.31) on day 180, which is 25% and 44%, respectively. The mean difference in GI on day 90 and 180 was found to be 0.50 (range 0.00-0.50), which is 25%. This is depicted in Table 1.
The reduction in GI within the group was found to be very highly significant statistically on day 180 both for the control and case group (\(p < 0.001\)). However, the change in GI between case and control group was found to be not significant on each day of evaluation (\(p > 0.05\)). The changes in the mean GI for both the groups are shown in Figure 1.

Figure 1: Changes in Gingival Index

Note: That GI in both Control and Case Groups is Reduced on Day 90 and 180 Compared to Day 0 (Baseline) and More Reduction is Seen in Case Group

| Table 1: Intra and Intergroup Comparison of Gingival Index |
|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| Groups                        | Day 0 Mean (Range) | Day 90 Mean (Range) | Day 180 Mean (Range) | Day 0 Vs 90 | Day 0 Vs 180 | Day 90 Vs 180 |
| Control                      | 2.00 (1.75-2.00)  | 1.50 (1.25 - 1.50) | 1.25 (1.00 - 1.25)  | 0.50** (0.50 - 0.75) | 0.75 (0.75 - 1.00) | 0.25*** (0.19 - 0.25) |
| Case                         | 2.00 (1.75 2.00)  | 1.50 (1.44 1.56)  | 1.00 (1.00 - 1.31)  | 0.50** (0.44 - 0.56) | 0.88 (0.69 - 1.00) | 0.50*** (0.00 - 0.50) |

**= not significant (\(p > 0.05\))

***= very highly significant (\(p < 0.001\))

Changes in Probing Pocket Depth (PPD) (in Mm)

The mean value of PPD in the control group was found to be 4.77 ± 1.03 (range 3.74 - 5.80) mm on day 0 which is reduced by 1.23 ± 0.33 (0.89 - 1.55) mm on day 90, 1.73 ± 0.53 (range 1.20 - 2.26) mm on day 180, which are of 25.58% and 36.27%, respectively. The difference on day 90 and 180 was 0.51 ± 0.37 (range 0.14 - 0.88) mm (10.69%). Similarly, the mean value of PPD in the case group was found to be 5.54 ± 1.14 (range 4.40 - 6.68) mm on day 0 which is reduced by 1.63 ± 0.50 (range 1.13 - 2.13) mm, 2.67 ± 0.72 (range 1.95 - 3.39) mm, on day 90 and 180, respectively and are of 29.42% and 48.19%. The difference on day 90 and 180 was 1.04 ± 0.39 (range 0.65 - 1.43) mm (18.77%), as shown in Table 2

Thus, the reduction in PPD in the case group appears to be more in comparison to the control. Compared to the baseline, the reduction in PPD was found to be highly significant statistically (\(p = 0.008\)) on day 90 and very highly significant (\(p < 0.001\)) on day 180 in both the groups. Again, the change in between day 180 and 90 was found to be very highly significant statistically (\(p < 0.001\)). Intergroup comparison showed a significantly higher reduction in PPD in the case group (\(p \leq 0.05\)) than that of the control on each day of evaluation. The changes in the mean PPD for both the groups are shown in Figure 2.
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Note: That PPD in both Case and Control Groups is reduced on Day 90 and 180 compared to Day 0 (Baseline) and more reduction is seen in Case Group.

**Table 2: Intra and Intergroup Comparison of Probing Pocket Depth (Mm)**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Day 0 Mean±Sd (Range)</th>
<th>Day 90 Mean±Sd (Range)</th>
<th>Day 180 Mean±Sd (Range)</th>
<th>Day 0 Vs 90 p value</th>
<th>Day 0 Vs 180 p value</th>
<th>Day 90 Vs 180 p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>4.77±1.03 (3.74-5.80)</td>
<td>3.54±0.94 (2.60-4.48)</td>
<td>3.04±0.71 (2.33-3.75)</td>
<td>1.23±0.33 (0.89-1.55)</td>
<td>1.73±0.53 (1.20-2.26)</td>
<td>0.51±0.37 (0.14-0.88)</td>
</tr>
<tr>
<td>Case</td>
<td>5.54±1.14 (4.40-6.68)</td>
<td>3.92±0.78 (3.14-4.70)</td>
<td>2.88±0.49 (2.39-3.37)</td>
<td>1.63±0.50 (1.13-2.13)</td>
<td>2.67±0.72 (1.95-3.39)</td>
<td>1.04±0.39 (0.65-1.43)</td>
</tr>
</tbody>
</table>

SD= standard deviation

*** = highly significant (p < 0.01)

**** = very highly significant (p < 0.001)

**Changes in Clinical Attachment Level (CAL) (in mm)**

The mean CAL in control group A was found to be 7.72±1.97 (range 5.75-9.69) mm on day 0. It is gained by 1.67±0.69 (range 0.98-2.36) mm and 2.67±1.14 (range 1.53-3.81) mm on day 90 and day 180, respectively. Similarly, the mean CAL in the case group was found to be 9.39±2.00 (range 7.39-11.39) mm on day 0. It is gained by 2.61±0.92 (range 1.69-3.53) mm and 4.50±1.42 (range 3.08-5.92) mm on day 90 and 180, respectively, as shown in Table 3. Thus, the gain in CAL is recorded as 21.63% on day 90 and 34.59% on day 180 in control group, while it is calculated as 27.80% and 47.92% on day 90 and 180, respectively for the case group.

Thus, the gain in CAL is more in the case group and found to be significantly higher for the case group as compared to the control on each day of evaluation (p ≤ 0.05). The mean changes in CAL are graphically represented in Figure 3.
Changes in Alveolar Bone Height

The mean alveolar bone height in the control group was found to be 8.22 ± 1.70 (range 6.52 - 9.92) for day 0. The defect is reduced by 0.89 ± 0.68 (range 0.21 – 1.57) and 1.44 ± 1.04 (range 0.40 - 2.48) on day 90 and 180, respectively, and is calculated as 10.83% and 17.52%. As shown in Table 4, the mean alveolar bone height in the case group was found to be 10.00 ± 2.47 (range 7.53 - 12.47) on day 0. The defect is reduced by 1.56 ± 0.86 (range 0.70 – 2.42) and 2.94 ± 1.00 (range 1.94 – 3.94) on day 90 and 180, respectively, and is calculated as 15.60% and 29.40%.

Thus, reduction in the bony defect is more in case group and found to be significantly higher in the case group as compared to the control on each day of evaluation (p ≤ 0.05). The mean changes in alveolar bone height are graphically represented in Figure 4.

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**Table 3: Intra and Intergroup Comparison of Clinical Attachment Level**

<table>
<thead>
<tr>
<th>Groups</th>
<th>Day 0 Mean ± Sd (Range)</th>
<th>Day 90 Mean ± Sd (Range)</th>
<th>Day 180 Mean ± Sd (Range)</th>
<th>Day 0 Vs 90 p value (Unpaired t-test)</th>
<th>Day 90 Vs 180 p value (Unpaired t-test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>7.72 ± 1.97 (5.75 – 9.69)</td>
<td>6.06 ± 1.77 (4.29 – 7.83)</td>
<td>5.06 ± 1.35 (3.71 – 6.641)</td>
<td>1.67 ± 0.69 (0.98 – 2.36)</td>
<td>2.67 ± 1.14 (1.53 – 3.81)</td>
</tr>
<tr>
<td>Case</td>
<td>9.39 ± 2.00 (7.39 – 11.39)</td>
<td>6.78 ± 1.38 (4.95 – 8.61)</td>
<td>4.89 ± 1.18 (3.71 – 6.07)</td>
<td>2.61 ± 0.92 (1.69 – 3.53)</td>
<td>4.50 ± 1.42 (3.08 – 5.92)</td>
</tr>
</tbody>
</table>

SD= standard deviation

**= highly significant (p < 0.01)

***= very highly significant (p < 0.001)

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**Note:** The Gain in CAL in both Case and Control Groups is on Day 90 and 180 to Day 0 (Baseline) and More Reduction is Seen in Case Group.

Figure 3: Mean Change in CAL (Mm)

Figure 4: Mean Change in Alveolar Bone Height (Mm)
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Note: The Gain in Bone Height in both Case and Control Groups is On Day 90 and 180 Compared to Day 0 (Baseline) and More Reduction in Defect is Seen in Case Group

Table 4: Intra and Intergroup Comparison of the Depth of the Bone Defect

<table>
<thead>
<tr>
<th>Groups</th>
<th>Day 0 Mean ± Sd (Range)</th>
<th>Day 90 Mean ± Sd (Range)</th>
<th>Day 180 Mean ± Sd (Range)</th>
<th>Day 0 Vs 90 (Range)</th>
<th>Day 0 Vs 180 (Range)</th>
<th>Day 90 Vs 180 (Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>8.22 ± 1.70 (6.52 – 9.92)</td>
<td>7.33 ± 1.53 (5.80 – 8.86)</td>
<td>6.78 ± 1.52 (5.26 – 8.30)</td>
<td>0.89 ± 0.68 (0.21 – 1.57)</td>
<td>1.44 ± 1.04 (0.40 – 2.48)</td>
<td>0.56 ± 0.78 (0.22 – 1.34)</td>
</tr>
<tr>
<td>Case</td>
<td>10.0 ± 2.47 (7.53–12.47)</td>
<td>8.44 ± 2.28 (6.16-10.72)</td>
<td>7.06 ± 1.96 (5.10 – 9.02)</td>
<td>1.56 ± 0.86 (0.70 – 2.42)</td>
<td>2.94 ± 1.00 (1.94 – 3.94)</td>
<td>1.39 ± 0.70 (0.69 – 2.09)</td>
</tr>
</tbody>
</table>

*p* value (Unpaired t-test): 0.014 0.000 0.002

SD= standard deviation

**= highly significant (*p* < 0.01)

*** = very highly significant (*p* < 0.001)

DISCUSSIONS

Management of endo-perio lesions has been a challenge to the clinician since historical times. Diagnosis of an endo-perio lesion has been difficult since the earlier times. Though clinical symptoms disappear in most of the endo-perio lesions following successful endodontic therapy (Dunlap *et al*., 1981; Rotstein and Simon, 2006), it becomes essential to correct the periodontal defect simultaneously in these cases to prevent recurrence, and to improve the functional status of the tooth (Jaoui *et al*., 1995). Again, in some clinical situations it is often impossible to predictably differentiate endodontic from periodontal origin. For this reason, it was deemed logical to perform a combination of endodontic and periodontal treatment (Didilescu *et al*., 2008). Few studies have also suggested to perform the endodontic therapy first followed by evaluation of the radiograph on recall and periodontal correction is indicated only then, if healing failed (Vakalis *et al*., 2005, Rotstein and Simon, 2006). Another few studies have suggested that endodontic treatment unfavourably affects the periodontal regeneration (Lima *et al*., 1997; Miyashita *et al*., 1998). Hence, the effect of endodontic treatment on periodontal regeneration remains controversial till date. So the present study was carried out in 12 subjects with moderate to severe periodontitis.

Clinical attachment level (CAL) is one of the best ways to assess the periodontal damage as it measures the distance between cementoenamel junction to base of the periodontal pocket (Armitage 2004). Probing pocket depth is used as an important parameter for evaluating the success of periodontal therapy by comparing the post treatment PPD value to that of prior to therapy. In this study, both of these parameters are applied. Alveolar bone height was measured on the radiographs, as it is a noninvasive procedure and thereby, no severance of the attachment in contrast to the other option referred to as surgical re-entry. The accuracy and precision of the radiographic assessment at various times was maintained by using a paralleling kit. Endodontic treatment was performed by the same investigator in all the case sites to avoid any discrepancy between the procedure and technique.

There are different opinions in the literature regarding the time point for assessment of the healing response to surgical periodontal therapy. Badersten *et al*., (1981) reported that most of the healing in periodontal wound occurs in the first 4-5 months of therapy in periodontal pocket of 4-7 mm depth, while little changes occur during rest of the 13-months.
of observation period. Again, early bone formation is noted as early as two months postoperatively and mature calcification is radiographically visible at six months (Becker and Becker 1999). Considering this, in the present study the response to therapy was evaluated on day 90 and 180.

GI was found to be reduced significantly in both the control and the case groups (p<0.001). The reduction in GI after open debridement is in support of various investigators (Tsao et al., 2006; Slotte et al., 2012; Miranda et al., 2013). However, the changes in GI between the case and control group was found to be not significant on each day of evaluation (p > 0.05). This is in support with the findings of Lindskog et al., (1983) and Perlmutter et al., (1987).

The PPD was found to be reduced significantly in both the case and the control groups. The reduction in PPD in the case group was observed to be significantly higher than that in the control group (p ≤ 0.05) on each day of evaluation. This was in accordance with the findings of several investigators (Vakalis et al., 2005 and Greenstein et al., 2009).

The gain in CAL was seen in both the control and case groups, however, the gain in CAL in the case group compared to the control was found to be highly significant statistically both on day 90 and 180 (p ≤0.001). This was in support with the findings of Jaoui et al., (1995), Vakalis et al., (2005), Lin et al., (2008) and Kwon et al., (2013).

The gain in alveolar bone height was seen to be statistically significant in both the control and case groups (p ≤ 0.05). Again, the gain in alveolar bone height in the case group compared to the control was found to be highly significant statistically on day 90 and 180 (p < 0.01). This supports the findings of Tsao et al., (2006), and Miranda et al., (2013).

However, the finding of the present study is not supporting the finding of Timmerman and Weijden (2006) and Greenstein et al., (2009). This may be due to the variations in technique of endodontic treatment, variations in food habits or oral hygiene maintenance regime of the subjects who have participated in the present study from the subjects included in the studies carried out by them. These factors are referred to have influence on the outcome of periodontal therapy (Lima et al., 1997 and Miranda et al., 2013). It is noted that there are some variables like initial defect depth, individual healing potential, differences in oral hygiene maintenance, which may influence the results and are difficult to control. There are certain shortcomings in the present study, such as small sample size, follow up of short duration, etc. Considering these limitations, the present study could provide only limited informations, thereby it is not sufficient to assess the long term effects and prognosis of the treatment. Hence, long term studies with a larger sample size are recommended to determine the effect of endodontic treatment on periodontal regeneration.

CONCLUSIONS

From the observations made in the present study, we may draw conclusions that combined endodontic and periodontal therapies have an additional advantage in the outcome of periodontal therapy in the management of endo-perio lesions, being endodontic treatment performed prior to the open flap debridement.

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