Evaluation of the effect of tooth vitality on regenerative outcomes in infrabony defects


Abstract

Background, aims. This investigation was designed to evaluate the null hypothesis of no differences in GTR outcomes in infrabony defects at vital and successfully root-canal-treated teeth.

Method: 208 consecutive patients with one infrabony defect each were enrolled. Based on tooth vitality, the treated population was divided at baseline into 2 groups: one with 41 non-vital teeth and the other with 167 vital teeth. The 2 groups were similar in terms of patient and defect characteristics.

Results: A slight unbalance in terms of depth of the infrabony component was observed in the non-vital group compared to the vital group (6.9±2.1 mm versus 6.2±2.3 mm, p=0.08). All defects were treated with GTR therapy. At 1 year, the non-vital and the vital groups showed a clinical attachment level (CAL) gain of 4.9±2.2 mm and of 4.2±2 mm, respectively. The difference was statistically significant (p=0.03). To correct for the baseline unbalance in defect depth, data were expressed as a % of clinical attachment level gains with respect to the original infrabony depth of the defect. % CAL gains were 72.8±42.2% and 73±26.4% for vital and non-vital teeth, respectively: the difference was not statistically significant (p=0.48). Average residual pocket depths were 2.8±1 mm in the vital and 2.8±0.9 mm in the non-vital group. Tooth vitality was assessed at baseline, at 1-year and at follow-up (5.4±2.8 years after surgery): all teeth vital at baseline were still vital at follow-up with the exception of 2 teeth that received endodontic treatment for reconstructive reasons and for caries. At follow-up visit, the difference in CAL with respect to 1-year measurements was 0.9±0.8 mm in the vital group and 0.7±0.8 mm in the non-vital group, indicating stability of the regenerated attachment at the majority of sites.

Conclusions: Data from this study demonstrate that root canal treatment does not negatively affect the healing response of deep infrabony defects treated with GTR therapy; furthermore GTR therapy in deep infrabony defects does not negatively influence tooth vitality.

The clinical outcomes of guided tissue regeneration (GTR) in deep infrabony defects have been tested in many clinical trials (Becker et al. 1988, Handelsman et al. 1991, Cortellini et al. 1993a, 1995, 1996a, b, c, 1998, Laurell et al. 1994, Al-Arrayed et al. 1995, Mattson et al. 1995, Mellado et al. 1995, Tonetti et al. 1996, 1999, Gouldin et al. 1996, Falk et al. 1997, Christgau et al. 1997). They were characterized by a high degree of clinical variability. A number of factors have been proven to directly influence the clinical outcomes of GTR. A group of factors is related to the patient: such as smoking habit (Tonetti et al. 1995, Trombelli et al. 1997), plaque control (Tonetti et al. 1995, 1996a, b) and residual periodontal infection in the mouth (Tonetti et al. 1993, 1996a, b). A 2nd group is related to the site; it comprises defect morphology and in particular defect depth (Tonetti et al. 1993, Trombelli et al. 1997) and defect width (Tonetti et al. 1993, Cortellini & Tonetti 1999). A 3rd group of relevant factors has been traced to variability in the surgical approach: among these, the most important are membrane contamination (Selvig et al. 1992, Nowza-
ri & Slots 1994, Nowzari et al. 1995, De Sanctis et al. 1996a, b), protection of the regenerated tissues (Tonetti et al. 1993, Cortellini et al. 1995a, b) selection of the surgical approach including flap design (Cortellini et al. 1995a, b), and clinician’s experience and surgical skill (Tonetti et al. 1999).

The known factors, however, do not seem to account for all the variability of GTR outcomes. This observation has generated a series of experimental hypotheses. Among these is the possibility that endodontic status may be a relevant factor. This hypothesis has been generated in the context of emerging experimental evidence that root canal treated teeth may respond differently to periodontal therapy (Kornman & Robertson 2000). In particular, animal and human investigations have indicated that the presence of periapical lesions or root canal treatment may decrease the outcomes of periodontal therapy (Morris 1953, 1957, 1960, Prichard 1972, Helländ 1972, Diem et al. 1974, Perlmutter et al. 1987, Chen et al. 1997). An early study in humans, has shown that endodontically treated teeth responded less well to bone grafting procedures carried out to treat deep intrabony defects: 65% of vital teeth resulted in satisfactory bone fills as compared to 33% of root canal treated teeth (Sanders et al. 1983).

The majority of the cited studies, however, do not allow a clear conclusion due to the lack of consistent results comparing clinical and animal studies, the small sample size of the experimental populations and the small differences in outcomes among vital and non-vital groups. In addition, no studies have been performed specifically in the GTR field.

The aim of the present study was to evaluate the null hypothesis of no differences in GTR outcomes in intrabony defects at vital and successfully root canal treated teeth. In addition, the possible influence of GTR treatment on tooth vitality was explored.

Material and Methods

Study population and experimental design

208 consecutively treated deep intrabony defects in 208 subjects (mean age 44.3±10.1, range 18 to 76 years, 56.3% females, 70 smokers) were included in this investigation. Inclusion criteria were as follows. (1) Absence of relevant medical conditions. Patients with uncontrolled or poorly controlled diabetes, unstable or life threatening conditions, or requiring antibiotic prophylaxis were excluded. (2) Smoking status. Heavy smokers (more than 20 cigarettes/day) were excluded. (3) Periodontal disease. Only patients with a clinical diagnosis of severe periodontitis were included. (4) Intrabony defect. Presence of at least one tooth with a deep interradicular intrabony defect evident on intra-oral radiographs and associated with clinical attachment level loss and pocket depth of at least 6 mm.

Endodontic evaluation

Pulp vitality was examined with a combination of thermal (cold test) and electric (Vitality Scanner, model 2006, Analytic Technology) stimulation at the buccal and lingual sides of each experimental tooth. Teeth, which did not respond to both thermal and electric stimulation, were considered non-vital.

The quality of the root canal filling and the possible presence of periapical lesions of endodontic origin in the non-vital teeth were evaluated on periapical radiographs. Among the non-vital teeth, one received endodontic treatment for the presence of a periapical lesion associated with pulp necrosis, while five, which displayed improper root canal treatment, were retreated prior to the surgical phase of periodontal therapy. All non-vital teeth, therefore, had clinically acceptable endodontic treatment and root canal filling and did not display periapical radiolucencies at the time of GTR surgery. Among the vital teeth, 3 had to be endodontically treated before GTR for surgical reasons. In fact, the periodontal defects involved the apex of the teeth and the root instrumentation at time of periodontal surgery could have caused pulp necrosis. None of the vital teeth presented with periapical radiolucency.

Patients underwent initial therapy consisting of scaling and root planing, motivation and oral hygiene instructions. Three months after completion of initial therapy, baseline clinical measurements were recorded. Root canal therapy, where necessary, was performed prior to baseline assessment.

According to baseline tooth vitality, 2 subject populations were identified. One included 167 patients with an intrabony defect associated with vital teeth (control group, vital), the other included 41 individuals with defects associated with properly root canal treated teeth with no residual periapical radiolucencies (test group, RCT). 55 patients (33%) in the vital group and 15 (37%) in the non-vital one were smokers (<20 cigarettes/day).

Clinical periodontal measures

The following clinical parameters were evaluated at baseline, 1 year and at the follow-up appointment, after a period ranging 2 to 12-years post GTR treatment.

Full mouth plaque scores (FMPS) were recorded as the percentage of total surfaces (4 aspects per tooth) which revealed the presence of plaque (O’Leary et al. 1972). Bleeding on probing was assessed dichotomously at a force of 0.3 N with a manual pressure sensitive probe (Brodontic probe equipped with a PCP-UNC 15 tip, Hu-friedy); full mouth bleeding scores (FMBS) were then calculated (Cortellini et al. 1993a).

Probing pocket depth (PPD) and recession of the gingival margin (REC) were recorded to the nearest millimeter with a manual pressure sensitive probe at the deepest location of the interdenital defect. All measurements were taken with a pressure sensitive manual periodontal probe at 0.3 N (Brodontic probe equipped with a PCP-UNC 15 tip, Hu-friedy). Clinical attachment levels (CAL) were calculated as the sum of PPD and REC. Radiographic defect angles were measured on periapical radiographs, as previously described (Tonetti et al. 1993).

Treatment approach

The intrabony defects were treated with GTR therapy (Cortellini et al. 1993a). Membranes (either non-resorbable or bio-resorbable) were positioned following flap elevation, defect debridement and careful root planing. Flaps were sutured to fully cover the barrier membranes. An infection control protocol consisting of a one week course of doxycycline (2/die), and 0.12% chlorhexidine mouth-rinsing three times per day with weekly prophylaxis until resumption of regular oral hygiene procedures was prescribed (Cortellini et al. 1993a). Patients were requested to avoid brushing, flossing and chewing in the treated area for periods spanning 6 to 10 weeks. During this period of time patients were recalled weekly for pro-
Table 1. Baseline patient and defect characteristics at baseline: vital and non-vital teeth. Differences between vital and non-vital teeth was tested with the t-test (α=0.05)

<table>
<thead>
<tr>
<th></th>
<th>Whole (n=208)</th>
<th>Vital (n=167)</th>
<th>Non-vital (n=41)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>FMPS (%)</td>
<td>13.9±5</td>
<td>14.3±5.2</td>
<td>12.3±3.8</td>
<td>NS</td>
</tr>
<tr>
<td>FMBS (%)</td>
<td>11.2±5.2</td>
<td>11.4±5.2</td>
<td>10.0±4.8</td>
<td>NS</td>
</tr>
<tr>
<td>CAL (mm)</td>
<td>10.5±2.5</td>
<td>10.0±2.4</td>
<td>10.8±2.6</td>
<td>NS</td>
</tr>
<tr>
<td>PD (mm)</td>
<td>8.5±2.3</td>
<td>8.4±2.2</td>
<td>8.9±2.4</td>
<td>NS</td>
</tr>
<tr>
<td>Rx-Angle (°)</td>
<td>32±10.6</td>
<td>32.1±10.8</td>
<td>31.3±9.9</td>
<td>NS</td>
</tr>
<tr>
<td>CEJ-BD (mm)</td>
<td>11.9±2.7</td>
<td>11.9±2.7</td>
<td>12.1±2.8</td>
<td>NS</td>
</tr>
<tr>
<td>INFRA (mm)</td>
<td>6.3±2.3</td>
<td>6.2±2.3</td>
<td>6.9±2.1</td>
<td>p=0.08</td>
</tr>
</tbody>
</table>

1. Clinical characterization of selected sites

Defect morphology was characterized intra-surgically in terms of distance between the cemento-enamel junction and the bottom of the defect (CEJ-BD) and total depth of the intrabony component of the defect (INFRA), essentially as previously described (Cortellini et al. 1993a).

Data analysis

Data were expressed as means± standard deviations of 208 defects in 208 patients. No data points were missing. Comparisons between baseline and follow-up data were made using the paired t-test. Clinical attachment level gains (CAL gains) were the primary outcome variable. Percentage fill of the baseline intrabony defects were calculated as: (CAL gains)/INFRA · 100.

Comparisons between the test and control groups were evaluated with 2 sample statistics. Significance was set at 0.05.

Results

Patient and defect characteristics at baseline

The 208 patients presented at baseline with 13.9±5% full mouth plaque score and 11.2±5.2% full mouth bleeding score. Clinical attachment levels and probing pocket depths at the intrabony defect were 10.5±2.5 mm and 8.5±2.3 mm, respectively. The radiographic defect angle was 32±10.6° (range 12° to 70°).

In the vital group, FMPS and FMBS were 14.3±5.2% and 11.4±5.2%, respectively. CAL was 10±2.4 mm, PPD 8.4±2.2 mm and the radiographic defect angle 32.1±10.8° (min. 12°, max. 70°). The RCT group presented with 12.3±3.8% FMPS, 10±4.8% FMBS, an average CAL of 10.8±2.6 mm, PPD of 8.9±2.4 mm and with a radiographic defect angle of 31.3±9.9° (min. 16°, max. 60°). Differences between the two groups were not statistically significant (Table 1).

Distance from CEJ to the bottom of the defects was 11.9±2.7 mm and the intrabony component of the defects 6.3±2.3 mm in the total population. In the vital group, CEJ-BD was 11.9±2.7 mm, INFRA 6.2±2.3 mm, while in the RCT group CEJ-BD was 12.1±2.8 mm and INFRA 6.9±2.1 mm (Table 1). A slight unbalance in the depth of the intrabony component of the defect between vital and RCT teeth was observed (p=0.08).

1 year outcomes: whole sample

The 208 patients presented at 1 year with FMPS and FMBS of 9.6±3.8% and 7±3.4%, respectively. Improvements in plaque and bleeding scores between baseline and 1 year were highly significant (p<0.001). The intrabony defects treated with GTR resulted in 4.2±2.1 mm of CAL gains (Fig. 1). Differences between baseline (10.5±2.5 mm) and 1 year CAL (6.2±2.2 mm) were statistically highly significant (p<0.0001). The percent clinical attachment level gains were 73±40%. Residual pocket depths were on average 2.8±1 mm. Differences between baseline and 1-year pocket depths were clinically and statistically highly significant (p<0.0001).

1 year outcomes: vital versus RCT

No differences in FMPS and FMBS were found at 1 year between the 2 groups. Clinical attachment level gains in the vital and non-vital group were 4.2±2 mm and 4.9±2.2 mm, respectively (Fig. 1). The difference was statistically significant (p=0.03). The % clinical attachment level gains with respect to the original intrabony defect depth, how-

Fig. 1. The graph shows baseline and 1 year pocket depths (PD, red bars) and recessions (Rec, blue bars) of the 208 experimental teeth. Data are presented for the entire sample and for the vital (167 teeth), and the non-vital (41 teeth) group, separately. The line of the 0 mm represents the cemento-enamel junction (CEJ). The white bars indicate the clinical attachment level (CAL) gains.
Table 2. Endodontic status of experimental teeth at time of patient accrual (accrual), immediately before GTR treatment (baseline), 1 year after GTR treatment (1 year), and at follow up visit (follow-up)

<table>
<thead>
<tr>
<th></th>
<th>Accrual</th>
<th>Baseline</th>
<th>1 year</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>vital teeth</td>
<td>170</td>
<td>167</td>
<td>167</td>
<td>165</td>
</tr>
<tr>
<td>RCT teeth</td>
<td>38</td>
<td>41</td>
<td>41</td>
<td>43</td>
</tr>
</tbody>
</table>

ever, were 72.8±42.2% and 73±26.4% for vital and non-vital teeth: the difference was not statistically significant ($p=0.48$).

Residual pocket depths were on average 2.8±1 mm in the vital and 2.8±0.9 mm in the non-vital group. The difference was not statistically significant. At 1 year, all the teeth belonging to baseline vital group were still vital. One tooth, however, received root canal treatment for reconstructive reasons (Table 2). Fig. 2–9 show 2 representative treated sites.

**Long-term clinical outcomes**

The follow-up evaluation of the treated subjects was performed on average 5.4±2.8 years after GTR therapy. An average CAL loss of 0.8±0.8 mm was detected with respect to 1-year measurements. This indicates a substantial stability of the gained clinical attachment in the 208 sites. Comparing stability of gained attachment in the test and control group no significant difference was observed: the control group lost an average of 0.9±0.8 mm, while in the RCT group lost an average of 0.7±0.8 mm ($p=0.11$).

The endodontic status of each experimental tooth was also assessed at the long-term follow-up appointment. This indicated that no loss of vitality of treated teeth was observed, with the exception of one tooth that had to be endodontically treated for a deep caries lesion 4 years after GTR (Table 2).

**Discussion**

The effect of the endodontic conditions on periodontal therapy has been studied in humans and animals. Several lines of evidence support the hypothesis that endodontic pathology and/or root canal treatment may have possible negative effects on repair of periodontal...

Fig. 2. Clinical photograph showing a deep intrabony defect located at the distal aspect of a vital upper central incisor. The tip of the probe is near to the root apex.

Fig. 3. Radiograph showing the baseline radiolucency at the distal aspect of the upper central incisor. No periapical radiolucency is present.

Fig. 5. Radiograph taken 7 years after GTR treatment. Note the presence of cortical bone. The tooth is still vital. No periapical radiolucency is evident.
Fig. 6. A deep distal intrabony defect and a buccal dehiscence affect a second lower non-vital bicuspid.

Fig. 7. A bioresorbable barrier membrane positioned to cover the defect.

Fig. 8. The baseline radiograph shows the defect and the root canal filling. No periapical radiolucency is present.

Fig. 9. The 1-year radiograph shows the complete resolution of the intrabony component of the defect.

lesions (Pritchard 1972, Ehnevid et al. 1993a, b, Jansson et al. 1993a, b, 1995b). This investigation addressed the impact of successful root canal treatment on the outcomes of periodontal regeneration.

The observed 1-year and long-term clinical outcomes did not support the mentioned experimental hypothesis. In facts, the regenerative treatment of 208 consecutively treated intrabony defects resulted in similar clinical attachment level gains and pocket depth reductions in vital and non-vital teeth (Fig. 1). The comparison was made between two groups of patients/defects (a group with vital teeth associated with a deep intrabony defect and the other with successfully root canal treated ones associated with a deep intrabony defect). The two groups were comparable at baseline (Table 1). The depth of the intrabony component of the defects, however, was slightly deeper in the RCT group ($p=0.08$). The fact that the intrabony component of the defect had a tendency to be deeper in the RCT group is noteworthy (Tonetti et al. 1993, 1996a, b).

Previous investigations have suggested that periodontitis may progress at a faster rate at sites with endodontic pathology or RCT (Jansson et al. 1993a, b, 1995a, b, Jansson & Ehnevid 1998). Deeper defects may therefore suggest that such mechanisms may have played a role in the apical spread of periodontitis in the test group.

All patients had good oral hygiene and good control of periodontitis as indicated by low full-mouth bleeding scores. All teeth in the RCT group did not show signs or symptoms of persistent endodontic pathology, including residual periapical radio-lucences. Care should therefore be exerted in extrapolating the results of this investigation to clinical situations with sub-optimal control of the endodontic pathology. As expected from previous investigations (for review, see Cortellini & Tonetti (2000)), the 208 defects treated with GTR, gained consistent amounts of clinical attachment ($4.4\pm2.1$ mm) at 1 year (Fig. 1). The 41 non-vital teeth gained $4.9\pm2.2$ mm, while the vital teeth gained $4.2\pm2$ mm. The difference was statistically significant ($p=0.03$). It is important to underline, however, that a slight unbalance existed between the two groups in terms of baseline defect depth. As previously shown, defect depth affects the healing outcome (Tonetti et al. 1993, 1996a, b). In the presence of a slight unbalance in defect depth, expression of data as % of the theoretically possible CAL gain repre-
sents a more robust estimate of the regenerative potential (Cortellini et al. 1998). Pocket depth reduction was also essentially identical in the 2 groups. Therefore data were expressed as % of CAL gain with respect to the baseline depth of the intrabony defect. The % CAL gains were almost identical in the two groups: 72.8±2.2% in the vital and 73±26.4% in the RCT group. In both the test and the control groups, the treated sites healed with shallow pockets of less than 3 mm, on average (Fig. 1). These data indicate that GTR therapy performed at intrabony defects associated with successful RCT has similar outcomes with respect to vital teeth.

These results are not in agreement with a previous investigation that retrospectively examined the outcomes of bone grafting in intrabony defects at endodontically obturated and nonobturated teeth (Sanders et al. 1983). In that study, the primary outcomes that were evaluated were frequencies of bone fill of the intrabony defect. Authors reported satisfactory bone fill in a larger number of nonobturated teeth as compared to obturated teeth. Differences in design, treatment and definition of the experimental groups may explain the divergent observations of the two studies. In their study, Sanders evaluated the endodontic status using the presence or absence of a root canal filling as the discriminating factor. No information was gathered with regard to pulp status of nonobturated teeth or about the absence of sign and symptoms in the root canal treated teeth. In the present investigation, none of the RCT teeth presented signs and/or symptoms of persistent endodontic or periapical pathology. Such difference may in itself explain the inconsistency of the two studies. Furthermore, the primary outcome variable was different: clinical attachment level gain with reference to the initial depth of the intrabony component in this study, and classes of bone fill of the intrabony defect in the previous investigation. Lastly, possible differences in the employed regenerative principles, i.e., grafting versus GTR, should also be considered.

The clinical results obtained with GTR were maintained in the vital and RCT teeth over an average follow-up period of 5.4±2.8 years. In fact, in both groups the differences between the 1-year measurements and measurements made at follow-up visits in terms of clinical attachment levels, were less than 1 mm of attachment loss. This was consistent with previous observations of stability of the clinical attachment gained with GTR in patients with good oral hygiene, enrolled in a supportive periodontal care program (Cortellini et al. 1994, 1996a, b, c, 1999). This observation is interesting with reference to previous reports that have indicated a threefold amplification of progression of periodontitis (Jansson et al. 1995b).

From an endodontic point of view, the GTR treatment did not affect negatively the vitality of teeth in the short- and long-term period. In fact, the 167 teeth that were vital at baseline were still vital at 1 year. One tooth was endodontically treated for prosthodontic reasons at 1 year, while a second tooth received a root canal treatment after 4 years for a deep caries.

A total of 165 teeth were still vital after 5.4±2.8 years, on average (Table 2). Given the fact that bone loss on average extended almost 12 mm apical to the cemento-enamel junction, the apical and middle portions of these roots were carefully scaled and planed during the surgical procedure. These observations indicate that, in spite of the high prevalence of lateral canals in the apical and middle third of the roots (De Deus 1975), instrumentation of these areas did not result in pathological changes of the pulp leading to loss of vitality (Berghenoltz & Lindhe 1978, Ryan et al. 1984, Adriaens et al. 1988). These observations are in agreement with the concept that scaling and root planing procedures do not represent a threat to vitality of the pulp (Bergenholtz & Lindhe 1978, Hattler & Listgarten 1983, Nilveus & Selvig 1983, Bergenholtz & Nyman 1984). In this context, prophylactic root canal treatment does not seem to be a rationale approach in cases of deep periodontal defects with no extension of periodontal breakdown to the periapical area.

In summary, three-conclusions can be drawn from the data presented in this study. (I) Previously performed and successful root canal treatment did not negatively affect the healing response of deep intrabony defects treated with GTR therapy. (II) No differences in the stability of the regeneratived clinical attachment were observed comparing vital and root canal treated teeth. (III) Regenerative therapy involving instrumentation of the apical portion of the root surface associated with deep intrabony defects was not associated with loss of tooth vitality.

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Zusammenfassung

Zum Einfluss der Zahnhärtigkeit auf die Ergebnisse regenerativer Therapie bei intraalveolären Defekten


Ergebnisse: Beide Versuchsgruppen waren hinsichtlich Patienten- und Defektkaracteristik gleich. Ein geringes Ungleichegewicht bestand zwischen beiden Gruppen hinsichtlich der Tiefe der intraalveolären Komponente (Mittelwert±SD: avital: 6.9±2.1 mm versus vital: 6.2±2.3 mm; p=0.08). 1 Jahr postoperativ wurde bei den avitalen Zähnen ein klinischer Attachmentgewinn (CAL) von 4.9±2.2 mm und bei den vitalen Zähnen von 4.2±0.00 mm beobachtet (p<0.03). Nach Korrektur der Ungleichheit der Gruppen hinsichtlich Tiefe der Knochentasche zu Studienbeginn durch Berechnung des relativen Attachmentgewinns in % der Knochendefekte ergaben sich folgende Ergebnisse: %CAL avital 73.0±26.4%, vital 72.8±42.2%; p=0.48. Die mittleren postoperativen Sondierungsstiefen (ST) lagen bei den avitalen Zähnen bei 2.8±0.9 mm und bei den vitalen Zähnen bei 2.8±1.0 mm. Über den gesamten Nachuntersuchungszeitraum (5.4±2.8 Jahre postoperativ) blieben alle Zähne, die zu Studienbeginn vital getestet worden waren vital mit der Ausnahme von 2 Zähnen, die aus prothetischen Gründen bzw. wegen Karies wurzelkanalbehandelt werden mußten. Bei den Nachkontrollen hatte sich der CAL in der avitalen Gruppe um -0.7±0.8 mm und in der vitalen Gruppe um 0.9±0.8 mm gegenüber den Ergebnissen 1 Jahr postoperativ verändert, was auf Stabilität des regenerier-ten Attachments in der Mehrzahl der Zähne hinweist.
Résumé
Evaluation de l’effet de la vitalité dentaire sur la régénération des lésions intraosseuses
Cette étude a été effectuée pour évaluer l’hypothèse nulle soutenant qu’il n’y avait aucune différence dans la guérison par régénération tissulaire guidée (GTR) dans les lésions intraosseuses des dents vitales ou traitées endodontiquement avec succès. 208 patients ayant une lésion intraosseuse ont été inclus dans cette étude. Sur base de la vitalité dentaire, la population traitée a été divisée lors de l’examen initial en 2 groupes: le premier comptait 41 dents dévitalisées, l’autre 167 dents vivantes. Les 2 groupes étaient semblables tant ou point de vue des caractéristiques et du patient que de la lésion. Une légère différence dans les termes de profondeur de la lésion était observée lors d’une comparaison entre les 2 groupes: dents dévitalisées 6,9±2,1 mm versus dents vivantes 6,2±2,3 mm (p=0,08). Toutes les lésions ont été traitées par GTR. Après une année, les deux groupes présentaient un gain de niveau d’attache (CAL) de respectivement 4,9±2,2 mm et 4,2±2,0 mm (p=0,03). Afin de corriger la différence due à la profondeur de la lésion lors de l’examen initial, les données ont été exprimées en γ de gain de niveau d’attache clinique vis-à-vis de la profondeur initiale de la lésion. Le % de gain CAL était respectivement de 73±26% et 73±42% (p=0,48). La moyenne de profondeur de poches résiduelle était respectivement de 2,8±0,9 mm et 2,8±1,0 mm. La vitalité dentaire a été mesurée lors de l’examen initial, à un an et lors du rappel (5,4±2,8 années après la chirurgie). Les dents vivantes lors de l’examen initial le demeuraient lors des réexamin à l’exception de deux dents qui avaient reçu un traitement endodontique pour cause de reconstruction ou de lésions carieuses. Lors de la visite de rappel, la différence de CAL vis-à-vis des mesures de la première année était de respectivement de –0,7±0,8 mm et de 0,9±0,8 mm montrant ainsi la stabilité de l’attache régénérée dans la plupart des sites. Les données de cette étude démontrent donc que le traitement endodontique n’influence pas négativement la réponse à la guérison des lésions intraosseuses profondes traitées par GTR. De plus, le traitement GTR n’agit pas de manière négative sur la vitalité dentaire au niveau des lésions intraosseuses profondes.

Schlussfolgerungen: Die Ergebnisse dieser Studie zeigen, dass eine Wurzelkanalbehandlung die Ergebnisse der GTR-Therapie tiefer intraalveolärer Defekte nicht negativ beeinflusst. Darüber hinaus beeinflusst auch die GTR-Therapie tiefer Knochenstücke die Zahnnormalität nicht negativ.

References


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