Some effects of enamel matrix proteins on wound healing in the dento-gingival region


Abstract

Objective: The aim of the present study was to evaluate by clinical means the effect of enamel matrix proteins on the healing of a soft tissue wound produced by periodontal pocket instrumentation.

Material and methods: The study was performed as an intra-individual, longitudinal trial of 3 weeks duration with a double-masked, split-mouth, placebo-controlled and randomized design. The patient material was comprised of 28 subjects with moderately advanced, chronic periodontitis. Each patient presented with 3 sites in each of 2 jaw quadrants with a probing pocket depth (PPD) of ≥5 mm and bleeding following pocket probing (BoP). Baseline examination, including assessments of plaque, gingival inflammation, PPD, BoP and root dentin sensitivity, was carried out one week after oral hygiene instruction and careful self-performed plaque control. All experimental sites were scaled and root planed, and the soft tissue wall of the pocket was curetted to remove the pocket epithelium and adjacent granulation tissue. The site was carefully irrigated with saline. When the bleeding from the pocket had ceased, a 24% EDTA gel was applied in the site and retained for 2 min. This was followed by careful irrigation with saline. Left and right jaw quadrants were then randomized to subgingival application of enamel matrix derivative (Emdogain®) or vehicle-control. All sites were re-examined after 1, 2 and 3 weeks. In addition, a visual analogue scale (VAS) was used to score the degree of post-treatment discomfort. The primary endpoints of treatment success were defined as (i) pocket closure (PPD <4 mm), (ii) no bleeding following pocket probing, (iii) no sign of gingival inflammation (GI score =0) and (iv) low degree of post-treatment discomfort (VAS ≤20). Statistical analyzes of intra-individual differences between the test and control treatments were performed by the use of Wilcoxon signed rank test. For comparison of the proportions of sites reaching the defined endpoints of treatment success, a site-based analysis was performed using 2×2 tables and the Fisher exact test.

Results: The endpoint “GI score =0” was reached at 16% of the sites subjected to application of Emdogain® at 1 week and at 2% of the control sites (p=0.001). At 2 weeks, the corresponding figures were 25% versus 12% (p=0.028). Absence of BoP was at 1 week 57% for the Emdogain® treated sites compared to 35% for the control sites (p=0.003). At 2 weeks, this endpoint was reached in 73% and 59% of the test and control sites, respectively (p=0.051). In terms of the endpoint defined for probing pocket depth, PPD ≤4 mm, no differences between test and control sites were found. At 1 week, the proportion of patients reporting a VAS score ≤20 was significantly higher for the Emdogain® treated quadrants than for controls (p=0.002).

Conclusion: The results indicated that Emdogain® topically applied in instrumented pockets enhance the early healing of periodontal soft tissue wounds.
It has been demonstrated that scaling and root planing are procedures which will (i) effectively reduce/remove subgingival plaque and calculus but also (ii) cause mechanical damage to the gingival epithelium and connective tissue (for review, see Cobb 1996).

In an early experiment by Ramfjord & Kieser (1954), block sections including teeth and adjacent periodontal tissues were obtained from 16 human volunteers with periodontal disease. The tissues were sampled immediately after the teeth had been carefully scaled “for removal of plaques and subgingival calculus”. Following a detailed microscopic examination the authors concluded that routine scaling of teeth will sever the epithelial attachment and “the cut will extend into the adjacent connective tissue attachment” and cause an inflammatory response in the gingiva. Moskow (1962, 1964) confirmed these observations. He sampled gingival biopsies from more than 300 patients undergoing treatment for periodontal disease. The samples were harvested directly after subgingival scaling or curettage as well as at different intervals – from 1 to 150 days – of soft tissue healing. It was observed that subgingival instrumentation (i) produced injury to the sulcus epithelium and adjacent connective tissue, but also that (ii) re-epithelialization of the sulcus occurred and a diminished “inflammatory reaction in the gingival corium” took place within 7 days after instrumentation. A detailed examination of gingival tissue repair following non-surgical periodontal therapy was performed by Bagini et al. (1988). 12 patients with chronic periodontitis participated in the study. Treatment included careful oral hygiene instruction and “quadrant scaling and root planing with hand curettes or ultrasonic scalers”. Healing of the gingival tissues was monitored by gingival fluid measurements. The size and character of the lesions were studied in biopsies sampled prior to therapy and after 5–10 and 30–60 days after subgingival instrumentation. The authors reported that the gingival fluid flow markedly decreased between days 0 and 30. In specimens obtained 5–10 days after treatment early signs of repair could be seen. Further, the number of inflammatory cells in the gingival lesion, compared to baseline values, had decreased. The 30–60 day specimens contained “precisely oriented collagen fiber bundles” and no inflammatory lesion could be observed. This finding was in part confirmed by Boretti et al. (1995) who studied gingival healing following mechanical and ultrasonic debridement. The authors reported that 1 month after treatment, there were significant reductions in clinical signs of gingivitis and a reduced number of crevicular leukocytes.

Enamel matrix derivative (EMD), applied to the root surface in conjunction with surgical periodontal therapy, may promote periodontal regeneration as demonstrated in both animal experiments and clinical studies (Hammarström et al. 1997, Heijl 1997, Heden et al. 1999). Heden et al. (1999) reported that the initial probing depth, i.e., the dimension of the soft tissue component, influenced the amount of gain of clinical attachment that resulted following surgical treatment and EMD application in intrabony defects. Findings from an in vitro wound healing model demonstrated that EMD not only had an effect on the migration of periodontal ligament cells but also on gingival fibroblasts (Hoang et al. 2000). Proliferation of epithelial cells, however, was not enhanced by EMD (Gestrelius et al. 1997a). Taken together, these observations indicate that EMD, in addition of being a means for periodontal regeneration, may influence soft tissue healing.

The aim of the present model experiment was to examine, by clinical means, the effect of EMD on the healing of soft tissue wounds produced by periodontal pocket instrumentation.

Material and Methods

A total of 28 subjects were recruited among patients referred to the Clinic of Periodontics, Department of Periodontology, Göteborg University, for treatment of moderately advanced chronic periodontitis based on the following inclusion criteria:

- A subject must have 3 flat tooth surfaces (experimental sites) in each of 2 contra-lateral jaw quadrants with a probing pocket depth (PPD) of ≥5 mm and bleeding following pocket probing (BoP+). At least one pair of sites must have a PPD ≥6 mm and the difference in the sum of the PPD between the 2 jaw quadrants must not be greater than 1 mm.
- Experimental teeth must either have a vital pulp (as determined by thermal or electric stimulation) or, if subjected to root canal treatment, be asymptotic and without technical remarks.
- Patients must have an unremarkable medical history.
- Patients must have signed a written informed consent statement and completed a health history questionnaire.

Study design

The study was performed as an intra-individual, longitudinal experiment of 3 weeks duration with a double-masked, split-mouth and randomized placebo-controlled design.

Following a screening examination, all patients were given instruction in proper supragingival plaque control measures and one session of scaling and root planing. The non-surgical instrumentation was carried out on all teeth in the dentition, except those that were selected as experimental teeth (6 teeth per patient). After one week of careful self-performed plaque control, the baseline examination (see below) of the experimental sites was performed.

The experimental teeth were subjected to a single session of comprehensive scaling and root planing under local anesthesia (Xylocain®, AstraZeneca, Södertälje, Sweden). Following the completion of the root instrumentation, the soft tissue wall of the pocket was curetted in an attempt to remove the pocket epithelium. The site was then carefully irrigated with saline. When the bleeding from the pockets had ceased, a 24% EDTA gel (PrefGel®, Biora AB, Malmö, Sweden) was applied in the site by means of a syringe and retained for 2 min. This was followed by careful irrigation with saline. At this interval, the envelope containing the randomization code was opened and the assigned treatment of the site was disclosed. Endogain® (Biora AB, Malmö, Sweden), sterile lyophilized enamel matrix derivative at a concentration 30 mg/ml sterile aqueous solution of propylene glycol alginate (PGA; Biora AB, Malmö, Sweden), was applied in the test sites of the left or the right jaw quadrant according to the randomization code. The vehicle (PGA) alone was used as a placebo-control device in the contra-lateral sites.

The patients were instructed to avoid toothbrushing during the first day after treatment and to use a 0.12% chlorhexidine solution for mouthrinsing, twice
daily for 1 min., during the first week following treatment.

Assessments
The baseline examination of the experimental sites included the following parameters:
- **Oral hygiene status** – presence/absence of plaque in the area of the soft tissue margin.
- **Gingival condition** – according to the criteria of the gingival index system (Löe 1967).
- **Probing pocket depth** – the distance from the gingival margin to the bottom of the probable pocket assessed with the use of a manual periodontal probe (UNC no. 15; tip diameter of 0.45 mm) to the nearest whole mm.
- **Bleeding on probing (BoP)** – presence (+) or absence (−) of bleeding within 15 seconds following pocket probing.
- **Dentine hypersensitivity (RDS)** – recorded (+/−) following air-blast stimuli (60 psi, 22°C) produced by a dental syringe and directed to the root surface for 1 s. The syringe was kept perpendicular to and 2–3 mm from the root surface. During testing the examiner’s gloved fingers shielded the neighbouring teeth.

Re-examinations were performed 1, 2 and 3 weeks following treatment. All examinations were carried out by a dental hygienist blinded with respect to the design of the trial and to treatment assignments.

**Degree of post-treatment discomfort** was scored – for each jaw quadrant – by the patient at the 1- and 3-week follow-up examinations. A 100 mm Visual Analogue Scale (VAS), labelled at the two extremes with “no discomfort” at the zero point and with “severe discomfort” at the 100 mm extreme was used. Data from the VAS scorings were recorded by measuring (mm) the distance between the zero point and the sign marked by the patient on the scale.

Data analysis
The primary endpoints of treatment success were defined as (i) pocket closure (PPD < 4 mm), (ii) no bleeding following pocket probing (BoP−), and (iii)

### Table 1. Baseline characteristics for test (Emdogain®) and control (PGA gel) sites

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control (PGA gel)</th>
<th>Test (Emdogain)</th>
</tr>
</thead>
<tbody>
<tr>
<td>sum-plaque score</td>
<td>0.6 (1.10)</td>
<td>0.8 (1.12)</td>
</tr>
<tr>
<td>sum-gingival index (GI)</td>
<td>4.0 (0.88)</td>
<td>4.1 (1.02)</td>
</tr>
<tr>
<td>probing Pocket depth (PPD)</td>
<td>5.9 (0.53)</td>
<td>5.9 (0.60)</td>
</tr>
<tr>
<td>5–6 mm</td>
<td>82% (69)</td>
<td>82% (69)</td>
</tr>
<tr>
<td>7–8 mm</td>
<td>18% (15)</td>
<td>16% (13)</td>
</tr>
<tr>
<td>≥9 mm</td>
<td>−</td>
<td>2% (2)</td>
</tr>
<tr>
<td>bleeding on probing (BoP+)</td>
<td>100% (64)</td>
<td>100% (64)</td>
</tr>
<tr>
<td>root dentin sensitivity</td>
<td>18% (15)</td>
<td>19% (16)</td>
</tr>
</tbody>
</table>

Individual mean value (SD) for sum-plaque scores (maximum value=3) and sum-GI (maximum value=9), probing pocket depth (SD) and percentage (number of sites) of various PPD categories, BoP+ and root dentin sensitivity.
no signs of gingival inflammation (GI score = 0). An additional variable considered was (iv) low degree of post-treatment discomfort (VAS ≤ 20). For the comparison of the proportions of sites reaching the defined endpoints of treatment success, a site-based analysis was performed using 2×2 tables and the Fisher exact test (2-tailed).

The sum of scores obtained from the various examination time intervals

Table 2. Proportion of sites (%) reaching the endpoint of treatment success of no signs of gingival inflammation (GI score = 0) at the various examination time intervals

<table>
<thead>
<tr>
<th></th>
<th>Control (PGA gel) (%)</th>
<th>Test (Emdogain) (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>baseline</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>1 week</td>
<td>2</td>
<td>16</td>
<td>0.001</td>
</tr>
<tr>
<td>2 weeks</td>
<td>12</td>
<td>25</td>
<td>0.028</td>
</tr>
<tr>
<td>3 weeks</td>
<td>36</td>
<td>40</td>
<td>0.662</td>
</tr>
</tbody>
</table>

* The Fisher exact test.

Table 3. Proportion of sites (%) reaching the endpoint of treatment success of no bleeding on probing at the various examination time intervals

<table>
<thead>
<tr>
<th></th>
<th>Control (PGA gel) (%)</th>
<th>Test (Emdogain) (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>baseline</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>1 week</td>
<td>35</td>
<td>57</td>
<td>0.003</td>
</tr>
<tr>
<td>2 weeks</td>
<td>59</td>
<td>73</td>
<td>0.051</td>
</tr>
<tr>
<td>3 weeks</td>
<td>74</td>
<td>75</td>
<td>&gt;0.999</td>
</tr>
</tbody>
</table>

* The Fisher exact test.

Table 4. Proportion of sites (%) reaching the endpoint of treatment success of probing pocket depth ≤ 4 mm at the various examination time intervals

<table>
<thead>
<tr>
<th></th>
<th>Control (PGA gel) (%)</th>
<th>Test (Emdogain) (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>baseline</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>1 week</td>
<td>20</td>
<td>23</td>
<td>0.731</td>
</tr>
<tr>
<td>2 weeks</td>
<td>43</td>
<td>49</td>
<td>0.478</td>
</tr>
<tr>
<td>3 weeks</td>
<td>61</td>
<td>6</td>
<td>&gt;0.999</td>
</tr>
</tbody>
</table>

* The Fisher exact test.

Table 5. Number of subjects (%) reporting a VAS score ≤ 20 at the various time intervals

<table>
<thead>
<tr>
<th></th>
<th>Control (PGA gel) (%)</th>
<th>Test (Emdogain) (%)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 week</td>
<td>31</td>
<td>54</td>
<td>0.002</td>
</tr>
<tr>
<td>3 weeks</td>
<td>54</td>
<td>65</td>
<td>0.149</td>
</tr>
</tbody>
</table>

* The Fisher exact test.

Results

All 28 patients completed the trial. Each subject contributed with 6 sites for the study, 3 test and 3 control sites and, hence, a total of 168 periodontal sites were included in the final analysis.

Data describing the baseline characteristics of the experimental sites are given in Table 1. There were no significant differences between the test and control groups regarding the descriptive variables.

The mean values, on the subject level, for the sum of plaque and GI scores, respectively, as well as the mean PPD at the various follow-up intervals are presented in Figs. 1–3. All three variables showed reduced values, i.e. an improvement over the 3-week study period in both the test and the control group. The plaque score was improved at 1 week post-treatment most likely as the result of the chlorhexidine regimen, and was maintained at a low level throughout the study. The mean PPD decreased from an average of 5.9 mm (S.D. 0.53) at baseline to 4.4 mm (0.69) at 3 weeks in the control group and from 5.9 mm (0.60) to 4.3 mm (0.61) in the test group. A statistically significant difference between the test and the control group was found for the mean sum-GI score at 1 week; 2.6 for the test group versus 3.0 for the control group (p = 0.011). The same magnitude of difference, although not statistically significant (p=0.065), was observed also at 2 weeks.

The proportion of teeth demonstrating root dentin sensitivity (RDS) increased in both groups following treatment. Hence, in the control group the proportion of teeth with RDS had increased from 18% (baseline) to 37% one week post-treatment, and in the test group from 19% to 35%. The increased frequency of RDS remained unchanged in both groups at the 3-week re-examination.

The results of the site-based analyses with respect to the defined primary endpoints of treatment success are presented in Tables 2–5. The endpoint “GI score = 0” was reached at 16% of the Emdogain® sites at 1 week and at 2% of the control sites (p=0.001). At 2 weeks post-treatment the corresponding figures were 25% and 12% (p = 0.028). No statistically significant difference between the treatment groups was found at 3 weeks post-treatment.

The frequency of “BoP−” (Table 3) was at 1 week 57% for the Emdogain® treated sites compared to 35% for the control sites (p = 0.003). At 2 weeks “BoP−” was reached in 73% and 59% for test and control sites, respectively (p = 0.051). There was no difference with respect to “BoP−” between the groups at the final examination.

In terms of the endpoint pocket closure, “PPD ≤ 4 mm”, no differences were found between the test and control groups at any of the 3 examination intervals (Table 4). At the final examination 61% of the sites in both groups had reached this endpoint of treatment success.

Table 5 describes the data from the patients’ VAS scorings of post-treatment discomfort. At 1 week, the proportion of patients reporting only minimal post-treatment discomfort (VAS score ≤ 20) was significantly higher for the Emdogain® treated quadrants than for controls (54% versus 31%; p = 0.002), while no differences were found at the re-examination at 3 weeks.

Discussion

The results of the present clinical model experiment demonstrated that Emdogain® (EMD), topically applied in instrumented pocket sites, improved the early events in the healing of a periodontal soft tissue wound. Hence, EMD application resulted, after 1 and 2 weeks, in significantly higher frequencies of sites without (i) clinical signs of inflammation and (ii) bleeding following pocket probing, compared to control sites. In addition, the patients reported significantly less post-treatment discomfort at sites subjected to EMD application.

The intent of the current study was not to evaluate per se the benefit of Em-
dogain® – topically applied in the dento-gingival region – as an adjunct to non-surgical periodontal therapy. Rather subgingival treatment measures were used to produce a soft tissue wound, the early healing of which was monitored by clinical methods. For this purpose periodontal sites were selected which were BoP+ and had a PPD of ≥5 mm. About 80% of the sites had a PPD of 5–6 mm while about 20% were ≥7 mm in depth. In other words, all sites utilized in the study exhibited signs of inflammation, i.e., harbored a lesion in the gingiva, and had a pocket epithelium that was at least 5 mm long. The model included the following features: (i) scaling and root planing to remove subgingival plaque and calculus, i.e. the infection from the root surface, (ii) excision of epithelium and granulation tissue from the pocket wall to remove the chronic lesion and to produce a fresh soft tissue wound, (iii) monitoring by clinical means the process of wound closure during an early 3-week period of healing, and (iv) identifying when certain endpoints of treatment success were reached; “GI=0” and “BoP−” indicating complete resolution of inflammation and “PPD ≤=4 mm” demonstrating pocket closure. Further, in order to properly identify a possible effect of Emdogain® on the resolution of the wound a double-blinded, placebo-controlled and intra-individually randomized design of the study was used.

The examinations performed during a 3-week period after root surface debridement and soft tissue curettage revealed that the mechanically produced soft tissue wound exhibited gradually reducing signs of inflammation. This was evidenced by lower Gingival Index scores and BoP+ values (Fig. 2) and confirms data previously reported (Ramfjord & Kiester 1954, Moskov 1962, 1964, Biagini et al. 1988, Boretti et al. 1995). The most important observation made in the current model study was, however, that there was a significantly larger proportion of test than control sites that after 1 and 2 weeks of healing had reached the predetermined endpoint of success in terms of resolution of inflammation (Tables 2, 3). A further analysis of the “GI=0” and “BoP−” values indicated that the EMD treated sites, in fact, reached these endpoints 1 week earlier than the control sites. The validity of this observation is in some way supported by the data describing patients’ post-treatment discomfort (Table 5). Thus, while only 31% of the control quadrants reached a “VAS score ≤=20” at week 1, 54% the test quadrants fulfilled this for the patient important criterion of treatment success. At the 3-week examination, however, no difference could be observed between the test and control quadrants regarding any of the endpoint parameters studied.

The observations made in the current model are supported by data from in vitro findings by Gestrelius et al. (1997b). They reported that Emdogain® applied on an instrumented and conditioned root surface may remain active in this location for a period not exceeding 1–2 weeks. Based on the above findings it is therefore suggested that Emdogain® during the first 2 weeks after non-surgical therapy may influence and enhance the healing of a soft tissue wound in the dento-gingival region.

The biological mechanisms behind this effect of EMD on the early phase of healing is presently not understood. In this context, however, the following observations may be of guidance. The most important events in the healing of a gingival wound include the formation of a de novo epithelial lining (Moskov 1962, 1964), the resolution of the inflammatory lesion and connective tissue repair (Moskov 1964, Biagini et al. 1988). These events seem to occur during the first 2 weeks after instrumentation, i.e., in a period when the enamel matrix proteins are present at the site (Gestrelius et al. 1997b). However, it is not likely that the enhanced healing is related to an early epithelialization of the soft tissue wall, since epithelial cells may not be influenced by EMD (Gestrelius et al. 1997a). On the other hand, Van der Pauw et al. (2000) demonstrated that EMD stimulates human gingival fibroblasts to release transforming growth factor β1 (TGFβ1), a mediator which has been suggested to influence the early phase of mucosal healing. The findings by Van der Pauw et al. (2000) was supported by data presented by Huang et al. (2000). They performed an in vitro wound healing experiment, and reported that EMD appeared to stimulate gingival fibroblasts and increase their rate of migration. Further studies are needed, however, to elucidate the mechanisms behind the influence of EMD on soft tissue wound healing.

**Zusammenfassung**

**Auswirkungen des Schmelzmatrixproteins auf**

**die Wundheilung der dentotägivalen Region**

**Zielsetzung:** Klinische Untersuchung der Wirkung von Schmelzmatrixprotein (SMP) auf die Heilung der durch subgingivale Instrumentierung verursachten Wunde.


**Ergebnisse:** Das Erfolgskriterium “GI=0” war nach 1 Woche bei 16% der Test- und 8% der Kontrollstellen erreicht (p<0.001). Nach 2 Wochen lagen die Proportionen für Test und Kontrolle bei 25% bzw. 12% (p=0.028). Kein BOP war nach 1 Woche bei 57% der Test- und bei 35% der Kontrollstellen zu beobachten (p=0.003), nach 2 Wochen lagen die Werte bei 73% bzw. 59% (p=0.051). Hinsichtlich des Kriteriums ST ≤=4 mm konnten keine Unterschiede zwischen Test und Kontrolle gefunden werden. 1 Woche nach Instrumentierung war der Anteil der Patienten in der Testgruppe, die eine VAS ≤=20 angaben, höher als in der Kontrollgruppe (p=0.002). 3 Wochen nach Therapie wiesen beide Gruppen hinsichtlich keines der Erfolgskriterien mehr statistisch signifikante Unterschiede auf.

**Schlussfolgerungen:** Die topische subgingivale Applikation von SMP in instrumentierte parodontale Taschen könnte die frühe Wundheilung des Weichgewebes begünstigen.
Résumé
Quelques effets des protéines de la matrice amélaire sur la guérison de la région gingivodentaire
But: Le but de l’étude présente a été d’évaluer cliniquement l’effet des protéines de la matrice amélaire (Emdogain®) sur la guérison des tissus mous produits par l’instrumentation de la poche parodontale.
Matériels et méthodes: Cette étude a été effectuée en tant qu’essai longitudinal intra-individuel de 3 semaines avec un modèle en double aveugle, par bouche divisée, au hasard et contrôlé par placebo. 28 sujets avec parodontopathie chronique modérément avancée ont participé à cette étude. Chaque patient présentait 3 sites dans 2 quadrants avec une profondeur au sondage (PPD) ≥ 5 mm et un saignement au sondage (BoP). L’examen initial comprenant la prise des indices de plaque, d’inflammation gingivale, de PPD de BoP et de la sensibilité dentinaire a été effectué une semaine après l’instruction en hygiène buccale et le contrôle de plaque dentaire réalisé par la personne elle-même. Tous les sites expérimentaux ont été détartrés et surfacés, et la paroi de tissu mou de la poche a été curée pour enlever l’épithélium de la poche et le tissu de granulation adjacent. Ce site a été irrigué avec du sérum physiologique. Lorsque le saignement de la poche avait cessé, un gel d’EDTA 24% a été appliqué dans le site et est resté en situ pendant 2 min. Ensuite une nouvelle irrigation avec un sérum physiologique a été prodiguée. Les quadrants gauches et droits étaient ensuite distribués au hasard pour l’application sous-gingivale du dérivé de la matrice amélaire (Emdogain®) ou en tant que véhicule contrôlé. Tous les sites ont été ré-examinés après 1, 2 et 3 semaines. De plus une échelle analogue de vision (VAS) a été utilisée pour mesurer le degré d’inconfort post-traumatique. Les points principaux du succès du traitement étaient définis comme suit: (1) fermeture de la poche (PPD ≤ 4 mm), (2) absence de saignement au sondage, (3) aucun signe d’inflammation gingivale (GI ≤ 0) et (4) un faible degré d’inconfort post-traumatique (VS ≤ 20). Les analyses statistiques des différences intra-individuelles entre les traitements tests et contrôles ont été effectuées à l’aide du test par Wilcoxon Signed Rank. Pour la comparaison des proportions de sites atteignant le succès souhaité, une analyse basée sur les sites a été effectuée en utilisant des tables 2×2 et le test exact de Fisher.
Résultats: Le but GI=0 a été atteint dans 16% des sites avec Emdogain® après 1 semaine seulement et dans 2% des sites contrôles (p=0.001). A 2 semaines, les figures correspondantes étaient 25% versus 12% (p=0.028). L’absence de BoP à 1 semaine atteignait 57% des sites traités par Emdogain® contre 35% pour les contrôles (p=0.003). A 2 semaines, ce but était atteint dans respectivement 73% et 59% des sites tests et contrôles (p=0.051). En terme de PPD>4 mm, aucune différence n’a été trouvée entre les sites. A 1 semaine, la proportion de patients qui avaient un VAS ≤ 20 était significativement plus importante dans le groupe traité par Emdogain® que chez les contrôle (p=0.002).
Conclusion: Les résultats ont indiqué que l’Emdogain® placé localement dans des poches nettoyées peut augmenter la guérison précoce des tissus mous parodontaux.

References


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