

THE USE OF DEMINERALIZED FREEZE-DRIED BOVINE BONE XENOGRAFT IN REDUCING POST-SURGICAL PERIODONTAL RESSION

Original Article

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ABSTRACT

The aim of this study was to determine the effectiveness of demineralized freeze-dried bone xenograft (DFDBBX) in minimizing post-surgical recession in moderate to advanced adult periodontitis in patients. Nine patients with a total of eighteen intrabony defects were matched for the tooth type, location of defects and periodontal pocket depth (5 to 7mm). Following an initial non-surgical treatment, recession at defects indicated for surgery was measured pre-operatively. Surgical treatment was carried out by split mouth design, where the test sites were assigned DFDBBX and the control sites were subjected to debridement without the use of DFDBBX. Recessions were measured at 3 months, 6 months and 9 months post-operatively. The results showed no statistically significant difference in mean recession at 3, 6 and 9 months post-operatively compared to baseline for both test and control groups. Thus, DFDBBX was ineffective in minimizing recession on patients with moderate to severe periodontitis, as compared to surgical debridement alone.

Key words: periodontal pockets, recession, demineralized freeze-dried xenograft.

INTRODUCTION

Periodontitis is an inflammatory disease of the periodontal tissues, which is characterized by loss of support of the affected teeth, specifically the periodontal ligament (PDL) fibers and the alveolar bone into which they are inserted. Following inflammation, the pathogenic changes that take place includes degradation of gingival connective tissue (CT), destruction of alveolar bone, denudation of the root surface by calculus deposition and apical migration of junctional epithelium (JE). Clinically, these pathogenic changes are seen as pocket formation and gingival recession.

Healing following conventional periodontal therapy be it surgical or non-surgical is by repair and regeneration but repair dominates the healing process. Repair by long junctional epithelium is less

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favorable because it does not completely restore the architecture or function of the lost tissues. On the other hand, regeneration includes reproduction of the lost or injured parts but reconstitutes only a very small portion, only the base of the pocket. Furthermore, regeneration of PDL and alveolar bone is a slower process compared to repair by LJE.

Regeneration of the alveolar bone lost through periodontal disease remains one of the biggest challenges in dentistry. Various efforts have been made to achieve regeneration using the principles of Guided Tissue Regeneration, Guided Bone Regeneration or the use of bone graft. However, bone graft has gained popularity as GTR and GBR were shown to be technique sensitive and highly expensive.

The objectives of periodontal bone graft are to reduce periodontal pocket depth, to achieve gain in clinical attachment level (CAL), to achieve bone fill of the osseous defects and to regenerate new bone, cementum and PDL. Since bone graft functions as a scaffold, it should prevent the apical migration of the soft tissue therefore preventing or minimizing recession. Clinical human and animal studies can provide valuable information on the first 3 objectives and the last objective requires histological analysis for verification.

Historically, autogenous graft is the gold standard in periodontal therapy as it has proven to have regenerative potential. However, recent histological studies have revealed that demineralized freeze-dried bovine bone xenograft (DFDBBX) has regenerative potential as well (1). The DFDBBX was reported to be biocompatible, has osteoconductive potential (2) and its physical properties were similar to human bone mineral in inner surface area,

porosity, crystalline size and calcium-to-phosphorous ratio (3). This investigation was focused on the use of DFDBBX as an alternative graft material in human. It evaluated the effect of DFDBBX in post-surgical recession compared to surgical debridement alone.

MATERIALS AND METHODS

Nine patients (5 females; 4 males) who were diagnosed with moderate to advanced periodontitis participated in the study. They were free from systemic disorders, were non smokers and not on medications or antibiotics in the last 6 months. Explanation was given to each patient regarding procedures involved. Each patient was required to sign a written consent form prior to commencement of treatment.

Nine pairs of intrabony defect sites (a pair of contralateral sites from each patient) were selected and matched for tooth type (molar or premolar), location of defect (mesiobuccal, midbuccal, distobuccal, mesiopalatal, midpalatal and distopalatal) and periodontal pocket of 5 mm or more. These contra-lateral sites were randomly selected for placement of DFDBBX (test sites) or conventional scaling root planing (SRP) only (control sites).

Each patient underwent initial non-surgical treatment prior to the surgical procedures that consisted of oral hygiene instructions, SRP with hand and ultrasonic instruments, and occlusal adjustment if indicated. The patients were reviewed and reassessed 8 weeks following SRP. At this appointment, the intrabony defect sites were indicated for surgery if the periodontal pocket depths remain more or equal to 5 mm.

The clinical parameters assessed were periodontal pocket depth, Visible Plaque Index (4), Gingival Bleeding Index (4) and Mobility index (5). These parameters were recorded at four different time intervals i.e. before initial treatment (baseline), three months, six months and nine months post-surgically.

The study employed a split-mouth technique whereby one side of the mouth was assigned as the test site (DFDBBX) and the contralateral side as the control (SRP only). The surgical procedures for the test sites was the modified Widman technique which consisted of flap reflection, soft tissue debridement, root planing with hand and ultrasonic instruments, decortication of alveolar bone, insertion of DFDBBX and complete flap closure. Similar procedures were carried out for control sites except there was no decortication on the alveolar bone and no DFDBBX was placed in the bone defect. The flaps were replaced and sutured with interrupted sutures.

Post-surgical instructions were verbally given by the clinician to each patient. The patient was advised not to brush the surgical area for two weeks until the sutures were removed, instead, the patient was recommended to rinse with or to place wet gauze soaked in 0.12% chlorohexidine gluconate solution twice daily. Each patient was given medications that consisted of antibiotic (Metronidazole 400mgs twice daily), anti swelling agent (Carilase 250mgs three times a day), and analgesic (Mefenamic acid 250mgs to be taken when required). Additional advice was to place icepack outside the surgical site to prevent swelling. The patient was recalled after 2 weeks, 3 months, 6 months and 9 months. A new orthopantomograph was taken at 6 months post-surgically.

STATISTICAL ANALYSIS

Repeated Measure Analysis was performed on all the parameters to determine the significance of each study. When significance was noted, a post-Hoc analysis was performed to identify the differences. Where there were dissimilar baseline mean values, the data were subjected to another statistical analysis (controlled for baseline) to confirm the results.

In SPSS, the effect size is measured in terms of Eta squared. The value of Eta ranges from 0 to 1. An Eta value more than 0.14 ($\text{Eta} > 0.14$) is accepted as a strong evidence of significance difference. In small sample studies, Eta is chosen as a yardstick to measure the magnitude of the effect.

RESULTS

Table 1. Mean Recession and Standard Deviation (SD) for Both the Test (N=9) And Control (N=9) Groups at 0, 3, 6 and 9 months Post-surgically

Time	Groups	Mean \pm SD (mm)
0 months	Test	1.44 \pm 1.24
	Control	0.33 \pm 0.50
3 months	Test	0.44 \pm 0.73
	Control	1.89 \pm 1.83
6 months	Test	1.33 \pm 0.71
	Control	1.28 \pm 0.75
9 months	Test	0.89 \pm 0.78
	Control	2.00 \pm 1.00

The result showed reduction in mean recession for test group at 3 months, 6 months and 9 months post-surgically compared to baseline. On the other hand, the results showed an increase in mean recession for control groups at 3 months, 6 months and 9 months post-surgically.

The mean value and standard deviation for test group was 1.44 ± 1.24 at baseline, 0.44 ± 0.73 at 3 months, 1.33 ± 0.71 at 6 months and 0.89 ± 0.78 at 9 months post-surgically. The mean value and standard deviation for control group was 0.33 ± 0.50 at baseline, 1.89 ± 1.83 at 3 months, 1.28 ± 0.75 at 6 months and 2.00 ± 1.00 at 9 months post-surgically.

There was a baseline difference in mean recession value i.e. the control group was lower than the test group. Except for the baseline, test of equality of error variances among the test and control groups at 3, 6 and 9 months did not show any significant deviation from homogeneity ($p > 0.05$). Although the baseline value was not the same, the test of between-subjects effect showed that there was no significant difference in the mean between the control and treatment groups over baseline, 3, 6 and 9 months post-surgically ($p > 0.05$; $\eta^2 < 0.14$). Thus, there was no difference between the test and control groups means.

The test of within-subjects was carried out to determine the effect of time and time by group interaction on the test and control groups. As for time, the test showed there was no significant difference at baseline, 3, 6 and 9 months periods ($p > 0.05$; $\eta^2 < 0.14$). However, the test for time by group interaction was significant with a p-value of 0.001 and η^2 value was 0.294 ($p < 0.05$; $\eta^2 > 0.14$). Thus, there was a strong evidence of time by group interaction.

Since there was a baseline difference, the data from both test and control groups were subjected again to statistical analysis, controlled for baseline measurement. The value adjusted for baseline recession mean value was 0.89mm.

The test of equality of error variance also showed no significant deviation from homogeneity between the test and control groups ($p > 0.05$). The test between-subjects effect showed that the test and control groups were not significantly different at 3, 6 and 9 months post-surgically, when controlled for baseline ($p > 0.05$).

The test of within-subjects was carried out to determine the effect of time as well as time by group interaction on mean recession. As for time, there was no statistical significant difference in mean recession over 3, 6 and 9 months post-surgically ($p > 0.05$; $\eta^2 < 0.14$). Similarly, there was no statistically significant difference in time by group interaction ($p > 0.05$; $\eta^2 < 0.14$). Thus, there was no strong evidence of any difference between the means over time, when controlled for baseline values. Once controlled for the baseline values, even the time by group interaction effect was not significant.

In conclusion, it was observed that the time by group interaction was not a real difference. It could be caused by the difference of mean values at baseline.

DISCUSSION

This study was carried out to determine the effectiveness of DFDBBX in reducing recession. An initial hypothesis was made that DFDBBX would effectively minimize recession.

In general, the results showed that there was recession reduction in test group and recession increase in the control group, compared to baseline. Despite dissimilar recession values at baseline, the statistical analysis did not show any significant group mean difference between test and control groups at baseline, 3, 6 and 9 months post-surgically ($p > 0.05$; $\eta^2 < 0.14$). In other words, the test group did not show greater recession reduction as compared to the control group. The result was confirmed by repeating statistical analysis to the data, controlled for baseline (adjusted mean baseline value was 0.89mm). Again, the statistical analysis showed that DFDBBX did not effectively reduce recession.

As for time effect, both test and control groups showed no significant differences when compared at baseline, 3, 6 and 9 months time periods ($p > 0.05$; $\eta^2 < 0.14$). No significant difference was obtained when data was subjected to another statistical analysis, controlled for baseline measurement (the adjusted mean value was 0.89 mm).

The reduction in recession could be attributed to the placement of DFDBBX in intrabony defects in the test group. The osteoconductive potential of the DFDBBX passively acted as scaffold and provided mechanical support to the flaps thus preventing the flaps from shrinking.

Interestingly, there was fluctuation in recession reduction post-surgically in the test group. The marked recession reduction at 3 months compared to baseline was because after filling up the intrabony defects with DFDBBX, every attempt was made to suture the flaps at the ACJ. However, active wound healing including contraction of wound, shrinkage of gingival connective tissues and migration of the epithelial tissue during the first few weeks as well as DFDBBX resorption in later weeks could have caused some additional recession. Thus the mean recession at 6 months was higher than at 3 months. Samsuddin et al (6) have shown in their study that DFDBBX started to resorb as early as one month post-surgically.

At 9 months post-surgically, there was less recession compared to 6 months. This could be due to an increase in host bone formation and deposition at the defect sites. Although most of the DFDBBX would have resorbed by 6 months, its osteoconductive and osteoinductive potential would have stimulated the host cells to lay down more bone. Therefore, by 9 months, more new bone has been

deposited thus reducing recession. The reduction in recession at 9 months could also be explained by the "creeping phenomenon" (7). As more bone is being deposited filling up the defect sites, the soft tissues are moved upward and downwards as the case maybe depending on the location of the teeth. This reduces the distance between the gingival margin and ACJ.

For the control group, the marked increase in recession at 3 months post-surgically was due to the fact that there was no xenograft in situ to act as soft tissue support and as a scaffold for bone formation. Therefore, it was unable to prevent the apical migration of epithelium from taking place thus causing further recession compared to baseline. The amount of recession was shown to maximize immediately post-surgically and only expected to average out later. This was due to active wound healing process which includes contraction of wound, shrinkage of gingival connective tissues and migration of the epithelial tissues (8).

The mean recession values at 6 months post-surgically were reduced compared to at 3 months post-surgically. This finding was consistent with other clinical trials that documented marked recession as immediate response to surgery and subsequently more plateau mean recession values for up to 6 months to 1 year following surgery. This was probably due to the fact that the wound healing has matured and stabilized (8, 9).

At 9 months post-surgically, the recession value was the highest, which can be explained by the fact that since there was no additional osteoconductive or inductive potential that could stimulate the host cells to lay down new bone. It has to rely primarily on the normal bone metabolism mechanism that normally takes longer time.

The tendency for patients to improve following treatment irrespective of any interventions is called Hawthorne effects (10). In this study, both test and control groups received surgical and non-surgical treatment. The main difference in this study was the use of DFDBBX for the test group. Therefore, even without intervention (bone graft) the same result can be achieved.

CONCLUSION

The conclusion that can be drawn from this study was DFDBBX was ineffective in minimizing periodontal recession in moderate to severe CIPD, as compared to surgical debridement alone. These conclusions were based on the 9 months follow up.

REFERENCES

1. Mellonig JT. Human histological evaluation of a bovine-derived bone xenograft in The Treatment Of Periodontal Osseous Defects. *Int J Periodont Restorat Dent* 2000; 20: 19-29.
2. Camelo M, Schenk RK, Rasperini G, Nevins M. Clinical, radiographical and histological evaluation of human periodontal defects treated with Bio-Oss and Bio-Gide. *Int J Periodont Restorat Dent* 1998; 18: 321-31.
3. Valdhe G, Mongiorgi R, Ferrieri P, Corvo G, Cattaneo V, Tartaro G. Scanning electron microscopy and microanalysis applied to the study of biomaterials for dental use. *Minerva Stomatol* 1995; 44: 55-68.
4. Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. *Int Dent J* 1975; 25: 229.
5. Miller SC. *Textbook of Periodontia*, 3rd ed. Philadelphia, The Blakeston Co. 1950.
6. Samsuddin AR, Meor Kamal, Afifi Abu Bakar. The use of lyophilized bovine bone xenograft in mandibular reconstructive surgery – an animal experimental surgery. The 7th International Conference On Tissue Banking APASTB. Abst: 126, 1998.
7. Genco RJ, Goldman H, Cohen DW. Pathogenesis and host response in periodontal disease. In: *Contemporary Periodontics*. The CV Mosby Company 1990; pp 184-93.
8. Kadhal W, Kalkwarf K, Patil K, Bates Jr. R. Evaluation of four modalities of periodontal therapy. Mean probing depth, probing attachment level and recession changes. *J Periodontol* 1998; 59: 783-93.
9. Becker W, Becker BE, Ochsenein C, Kerry G, Caffesse R, Morrison EC, Prichard J. A longitudinal study comparing scaling, osseous surgery and modified Widman procedures. Results after one year. *J Periodontol* 1988; 59: 351-65.
10. Jeffcoat MK. Principles and pitfalls of clinical trial design. *J Periodontol* 1992; 63: 1045-51.