COMPARISON OF EQUINE XENOGRAFT VERSUS DFDBA ALLOGRAFT IN TREATMENT OF PERIODONTAL INTRABONY DEFECTS

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Abstract

The aim of the present study was to compare clinically and radiographically the results of the treatment of intrabony defects by using DFDBA allograft with those obtained by using Bio-Gen xenograft in patients suffering from chronic periodontitis.

The results of this study revealed that both treatment modalities led to highly significant improvement in the mean probing depth when compared with the baseline throughout the experimental periods.

There was a statistically significant difference between the mean percentage changes in radiographic defect depth of both treatment modalities throughout all periods. It was statistically significantly higher at the sites treated with DFDBA than those treated with Bio-Gen.

Key Words:

chronic periodontitis – DFDBA – xenograft - intrabony defects – allograft - probing depth

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INTRODUCTION AND REVIEW OF LITERATURE

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Periodontal disease is a bacterial infection that causes destruction of the supporting structures of teeth, and usually involves a progressive, destructive change leading to alveolar bone loss with subsequent tooth loss **(Aichelmann-Reidy et al., 2004).** Periodontitis is the inflammation involved in the destruction of the supporting apparatus of the teeth including soft tissues bone and periodontal ligament **(Kinane, 2001).**

The destruction of the supporting apparatus is a result of host inflammatory reactions to the local accumulation of microorganisms in the proximity of the dentogingival junction, the inflammatory and immune responses progressing in a sequential manner until a dense cellular infiltrate containing potential harmful elements dominates the gingival tissues. It is also due to a direct insult of the microorganisms and their products on the alveolar tissues, resulting in bone resorption **(Page et al., 1978).**

Recently, periodontal disease and conditions are classified by The American Academy of Periodontology by using term of chronic Periodontitis instead of adult Periodontitis. Chronic Periodontitis is defined as inflammation of the gingival and the adjacent attachment apparatus and characterized by the loss of clinical attachment due to destruction of the periodontal ligament and loss of the adjacent supporting bone **(The American academy of Periodontology, 2000).**

Periodontal osseous defects are frequent sequale of periodontitis.The glossary of terms of the American Academy of Periodontology; an intrabony defect is defined as a periodontal defect within the bone surrounded by one,

two or three bony walls or a combination. Periodontal osseous defects, including periodontal pockets and furcation involvement, are frequent sequelae of periodontitis. Periodontal pocket is defined as a pathologically deepened gingival sulcus. It is one of the most important features of periodontal disease. Periodontal pocket occurs with destruction of the supporting periodontal structures. There are two types of periodontal pockets; Suprabony ‘supracrestal pockets (in which the bottom of the pocket is coronal to the underlying alveolar bone) and Intrabony ‘infrabony, subcrestal’pockets (in which the bottom of the pocket is apical to the level of the adjacent alveolar bone, as the lateral pocket wall lies between the tooth surface and the alveolar bone) **(Parashis et al., 1998).**

Goldman and Cohen (1957) classified Intrabony defects according to the location and number of remaining osseous walls into one-wall intrabony defects limited by one osseous wall and the tooth surface; two-wallintrabony defects limited by two osseous walls and the tooth surface and three-wall intrabony defects limited by three osseous walls and the tooth surface. Although a more biological description of the defect morphology should include the perspective of the periodontal ligament, since the highest goal of periodontal therapy is the regeneration of the periodontium including new cementum apposition with inserting periodontal ligament fibers in addition to the filling of defects with alveolar bone **(Niklaus, 2000).**

The goal of periodontal therapy remains the regeneration of tissues that have been lost due to periodontal disease and to provide a dentition that functions in health and comfort for the life of the patient **(Zandar et al., 1976 and Ramfjord, 1993).** Studies determining long-term tooth loss in

periodontal treated patients demonstrated that for the majority of periodontal patients, this goal is a reality (**Goldman et al., 1986).**

The primary objectives of therapy in Periodontitis is to arrest disease progression and resolve inflammation and reducing etiologic factors capable of producing breakdown by eliminating the pathogenic periodontal micro flora, which induces substantial favorable clinical changes in the periodontium. Another objective is to eliminate the anatomic defect resulting from the active Periodontitis which persists and is presented clinically by loss of clinical attachment, increased probing depth and radiographic bone loss **(Caton and Greenstein, 1994).**

Many results are anticipated following regenerative procedures such as repair, reattachment and regeneration. Repair is the biological process to restore the continuity of tissue disruption by new tissues which do not have the structure of the function of the lost ones, such as healing by long junctional epithelium, bone fill, root resorption or ankylosis. Reattachment is the reunion of connective tissue which a root surface that has been deprived of its periodontal ligament. This occurs by formation of new cementum with inserting collagen fibers **(Hammarston et al., 1997).**

Regeneration is the biological process, which restores the architecture and function of the lost tissues; it is a rebirth of the periodontium. This means new acellular cementum attached to the underlying dentine surface, new periodontal ligament having functionally oriented fibers inserted in the

new cementum and new alveolar bone attached to the periodontal ligament (**Heijl et al., 1997 and The American Academy of Periodontology, 2001).**

The tissues formed during repair process are subjected to a continued attachment loss in comparison to the tissues generated by surgical procedures designed to achieve regeneration. Healing by regeneration results in restoration of the original form and function of the lost periodontal tissues **(Thomas and Wilson, 1999).** Regeneration of periodontal attachment apparatus loss is a worthy goal. By regeneration, the pocket depth is reduced leading to the concomitant reduction of the aggressive pathogens typically found in deep pockets. Reduction in pocket depth will make oral hygiene more predictable by disruption of the organization of subgingival bacterial plaque, thus preventing its maturity and subsequent increased pathogenicity. Having shallow pockets is also easily maintained by the dental professional **(Annals of Periodontology, 1996).**

Several treatment modalities were proposed with varying degrees of success to predictably achieve regeneration. These include debridement procedures; various flap modalities, root surface demineralization and the use of various bone graft material, guided tissue regeneration or combination of these treatments modalities (**Caton and Greenstein, 1993 and Laurell et al., 1998).** Various biological approaches have been used to promote periodontal regeneration including; the use of growth factors, application of extracellular matrix proteins and attachment factors (fibronectin), and the use of mediators of bone metabolism (bone morphogenic proteins) **(Cochran and Wozney, 1999).**

Procedures such as subgingival curettage and flap curettage were used long ago to obtain new attachment **(Heijl, 1979). Listgarten and Rosenberg (1979)** reported that a long junctional epithelium to the base of the defect with the flap curettage procedures with no evidence of new bone and new cementum. Conservative non-surgical root debridement in the treatment of intra-bony defects may result in resolution of the inflammatory response, cessation of the progress of periodontal destruction, a small gain in periodontal attachment, significant reduction in probing depth and formation of a long junctional epithelium **(Greenstein, 1992).** The quality and thoroughness of root debridement is the major determinant of successful therapy. Changes in the microbiota are accompanied by a reduction or elimination of inflammation clinically **(Greenstein, 2000).**

Scaling and root planing are technically demanding and time-consuming. Studies show that clinical conditions generally improve following root planing; nonetheless, some sites still do not respond to this therapy **(Kaldahl et al., 1988).** The addition of gingival curettage to root planing in the treatment of generalized chronic periodontitis does not significantly reduce probing depth or gain clinical attachment beyond that attained by scaling and root planing alone **(Echeverra and Caffesse, 1983).** Some factors may limit the success of treatment by root planing including; root anatomy, furcations and deep probing depths **(Fleischer et al., 1989).**

Although, subgingival mechanical debridement is considered time consuming and often tedious procedure, it is basic to periodontal therapy and should be mastered by all clinicians**.** Flap debridement surgery permits the

debridement of root surfaces and removal of soft tissue following the reflection of mucuperiosteal flap. They have been described as open flap curettage, modified excisional new attachment procedure (ENAP) and most notably the Widman flap. They are repositioned flaps whose primary purpose is to gain access to the roots for definitive scaling and root planing in areas where pockets are 4mm or more. Thus, their main purpose was to control chronic periodontitis, not gaining new attachment **(Cohen, 1994).**

Modified Widman flap is among flap procedures used when regeneration is the goal. The advantages of periodontal flaps are providing proper access for scaling and root planning. The root surface can be inspected and cleaned by direct vision. Tissues may be more easily and radically changed and/ or removed **(Lindhe et al., 1998).** The Widman flap procedure (MWF) which is an extension or progression of the Widman flap (apically displaced flap) procedure gave encouraging results **(Cortellini et al., 1995).** The use of MWF procedure in intrabony defects has resulted in pocket depth reduction and attachment level gain **(Rosling et al., 1976)** which was greater when compared to scaling and root planing alone **(Lindhe et al., 1982).**

In long term clinical evaluations variable results were obtained when MWF was compared to scaling and root planing alone**. Pihistrom et al. (1981)** demonstrated more attachment gain and pocket depth reduction with MWF for deep pockets as compared to scaling and root planing alone over four years. In a later study **Pihistrom et al. (1983)** concluded that in treating deep pockets, MWF resulted in sustained pocket reduction for six and half years while scaling and root planing alone resulted in sustained pocket reduction for three years. **Becker et al. (1988)** concluded that over one year

MWF had significantly greater pocket reduction when compared to scaling and root planing. Both produced a slight gain in clinical attachment level.

In contrast, **Hill et al. (1981)** suggested that although initial pocket depth reduction was greater in sites treated with MWF than those treated by scaling and root planing, yet at two years MWF had no better effect than scaling and root planing alone in treating periodontal pockets. Furthermore, **Becker et al. (2001)** concluded that there were no significant differences between MWF and scaling and root planning regarding clinical attachment gain, after five years.

The histological healing of modified Widman flap procedure (MWF) occurs through a combination of crestal resorption and apical bone fill **(Polson and HeijI, 1978)** and the regeneration appears to be limited to the most apical portion of the defect, while the remaining portion is lined by long junctional epithelium **(Caton et al., 1980).** Thus, this healing pattern does not represent a true regeneration and this attachment can not be considered an efficient barrier to bacterial products which may allow recurrent pocket formation **(Meyer, 1986).**

One of the current regenerative techniques is biomodification of the root surface. The use of root surface chemical conditioner agents, which act in association with mechanical root planing, has been the objective of many studies **(Madison, 1997).** Among the studied agents, citric acid, ethylene-diamine-tetra-acetic acid (EDTA) and tetracycline have been shown as efficient for root conditioning and for removing the smear layer by mechanical instrumentation **(Wikesjo et al., 1992).**

Tetracyclines comprise a broad-spectrum antimicrobial agent, which is effective against many species of periodontal pathogens. Besides its antimicrobial effectiveness, this group of drugs has other special properties used in the management of periodontal disease including; anti-inflammatory action, collagenase inhibition, bone resorption inhibition and its ability to improve fibroblast attachment. Tetracyclines are still used in association with bone grafting, as conditioner agents for root surface and to enhance periodontal regeneration. Low dose doxycycline has been recognized to have non-antimicrobial anticollagen activity that can modulate periodontal tissue regeneration **(Seymour and Heasman, 1995).** On the other hand, a recent meta-analysis systematic review carried by **Mariotti (2003)** stated the use of tetracycline to modify the root surface provides no benefit of clinical significance to regeneration in patients with chronic periodontitis.

Even though some studies have demonstrated that the use of citric acid as root conditioner is similar to tetracycline **(Bouchard et al., 1997).** Unfortunately, this substance has no antimicrobial or anti inflammatory properties **(Renvert et al., 1997).** Although, **Blomlof (2000)** studies showed good results for root surface colonization by periodontal ligament cells, after conditioning with citric acid or EDTA. However, **Caffesse et al., (1988)** showed that conditioning with citric acid showed no statistically significant difference in connective tissue regeneration and in bone formation. This was supported by **Mariotti (2003).**

The chelating agent EDTA has been used as a root biomodification agent to remove the smear layer formed by root planing procedures. Studies showed that the use of the chelating agent EDTA, acting at neutral pH,

appeared preferable for exposure of collagen fibers and early cell colonization **(Blomlof et al., 1996).** In addition, etching at neutral pH has been reported to preserve adjacent tissue vitality **(Kassab and Cohen, 2003). Bloomlof et al. (2000)** concluded that the smear layer removal and collagen fiber exposure was achieved when EDTA was applied (as an irrigating agent in combination with ultrasonic scalers or in conjunction with root planing), through a customized tip but no additional improvement in clinical parameters observed**.**

Various biological approaches have been used to promote periodontal regeneration including; the use of growth factors, application of extracellular matrix proteins and attachment factors (fibronectin) and the use of mediators of bone metabolism (bone morphogenic proteins). The regeneration of any tissue type is a complex biological process which requires complicated interactions between cells, locally growth factors, systemic hormones and growth factors, extracellular matrix components and attachment factors **(Cochran and Wozney, 1999).**

Caffesse et al. (1988) showed that topical application of citric acid and fibronectin during the MWF significantly reduce pocket depth and increase clinical attachment gain when compared with surgical procedure alone. On the other hand, **Alger et al. (1990)** found a minimal amount (0.17mm) of connective tissue attachment after applying tetracycline hydrochloride and fibronectin to root surfaces.

Bone morphogenic proteins (BMP) are excellent molecules for stimulating alveolar bone formation; they are osteoinductive factors that may have the potential to stimulate mesenchymal cells to differentiate into bone