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Original Research

Clinical Evaluation of Combined Porous Hydroxyiapatite and Platelet Rich Plasma in Management of Mandibular Class II Furcation Defects

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Abstract:

Background: The purpose of the present study was to evaluate the role of platelet rich plasma (PRP) in augmenting the effects of porous hydroxyapatite (PHA) in promoting regeneration of furcated defects in mandibular molars Class II.

Materials and Methods: A total of 14 male subjects (age 38-52 years old) were included in this study. All subjects were presented with bilateral mandibular Class II furcation involvement. The split-mouth technique is utilized in an attempt to avoid individual variations. The females were also excluded to avoid their possible hidden hormonal disturbances. Clinical parameters were recorded after the initial phase of scaling and root planning. The baseline clinical measurements included periodontal pocket depth, clinical attachment level, horizontal probing depth, gingival recession and periapical radiographs. In each patient, a unilateral defect site was exposed by modified widman flap, debrided and filled with PRP combined with PHA. 1 month after healing of study site, the contralateral site was managed by M.W. flap only (control site). The same initial clinical measurements were repeated 6 months after surgery.

Results: The results showed that PHA combined with PRP induced a statistically significant improvement in the measured clinical parameters; greater than that of open debridement alone.

Conclusion: It could be concluded that regenerative periodontal surgery using the forementioned combination (PHA and PRP); could be proposed as a modality to enhance treatment outcome of mandibular furcated areas Class II involvement.

Key Words: Furcation, hydroxyiapatite, platelet rich plasma

Introduction

It is well-documented that a considerable number of maxillary and mandibular molars are lost due to furcation involvement.[1] A significant difference between mandibular and maxillary molars is that the mandibular furcation opens to the buccal and lingual directions. Therefore, no matter how advanced the loss bone, it does not damage the interdental bone for the adjacent teeth. The maxillary molars have mesial and distal furcations, and inflammatory lesions can result in the loss of interdental bone on adjacent teeth.[2] The extension of the inflammatory periodontal disease into the furcal area is the main cause of furcation involvement. Moreover, the treatment of multirooted teeth offers a challenge to all practitioners because of the posterior position of these teeth in the dental arch that limits access for diagnosis, therapy, and cleansing by the patient.[3,4]

Different treatment modalities are used in the management of furcation involvement such as; scaling and root planning, gingivectomy, furcaplasty, tunnel preparation, root resection, guided tissue regeneration and osseous potentiating grafting materials. In practice, the decision for one or the other treatment modality depends on the degree of furcation involvement, the morphology of the bont defect strategical importance of the tooth to be treated or the experience of the therapist.[5,6]

Elimination of subgingival plaque and calculus to achieve biologically acceptable root surfaces constitutes an essential component of appropriate periodontal therapy. However, the narrow width of furcation entrances often prevents adequate root instrumentation for this area.[7,8] Furtherly, regenerative techniques seem to have a better prognosis in Grade II than in through and through furcation.[9]

The forementioned presentation of such a problem evoked the interest for a proposed treatment modality. Hence, the purpose of te present study was to evaluate the clinical outcome of using a combination of porous hydroxyapatite (PHA) and platelet rich plasma (PRP) in the management of Class II furcation involvement in mandibular molars. Added to the possible creation of a suitable media for a prospective gain in attachment and periodontal regeneration.

Materials and Methods

About 14 male individuals ranged in age from 38 to 52 years old participated in this study. The all suffered from moderate to severe periodontitis and were free from any systemic disease that contraindicated periodontal surgery. The did not receive any medication for the past 6 months and were included in the screening medical questionnaire by the Cornell Medical Index.[10] Patient who had Class II furcation involvement according to Glickman's classification,[11] in at least two vital

molars bilaterally as determined by clinical examination and periapical radiographs were included in the study (Figure 1).

The initial phase presurgical included oral hygiene instructions for plaque control added to scaling and root planning; for each participant. 2 months later, the patients were recalled for the baseline presurgical measurements. These included pocket depth, gingival recession, clinical attachment level and horizontal probing depth; which were taken at the mid-buccal surface of the furcation involved molars to the nearest 1.5 mm using a straight periodontal probe. The same measurements were repeated at 6 months after surgery. Moreover, plaque and bleeding indices[12,13] were also recorded at the same intervals as the previous measurements. The same was accomplished as for periapical radiographs using the standardized technique. Modified widman flap was accomplished to expose all furcation areas. After debridement of the defects, the studied sites were filled with porous HA in combination with the PRP. Both are mixed in a volume proportion of 1:1 (Figures 2 and 3). Porous HA "Osteopgraft/ LD-300 low-density hydroxyapatite, creamed corporations, USA." Its granules had a mesh size of 40-60 μ and rounded particle size between 250 μ and 420 μ. The PRP preparation was performed by venipuncture of the antecubital vein to get 8 ml. of blood from each patient. The blood was mixed with 1 ml anticoagulant citrate dextrose A (Red Cross Blood Center, Beijing, China) and centrifuged. The subsequent procedures were accomplished using Camargo *et al.*,[14] and Lekovic *et al.*,[15] techniques; until the coagulated PRP preparation was rendered in a sticky gel consistency. The flaps were repositioned and sutured. Periodontal dressings were used and removed after 1-2 weeks; according to the condition of each case. 1 month must claps between the removal of the dressings and commencement of the surgical procedure on the contralateral sites. Recall appointments were carried out at 1, 2, 3, and 6 months to supervise the patient's oral hygiene.

Measurements of the previous clinical parameters were repeated 6 months postoperatively. A statistical analysis of data was performed by the use of a non-parametric test Paired *t*-test to evaluate the changes over time for clinical index scores, while a 2-way ANOVA was used as a parametric test for presenting differences between groups. Moreover, the design of the present was based on the split-mouth technique.

Results

Healing of surgical sites as for studied and control groups had been accomplished without adverse tissue reaction or post-operative infection. Three cases were excluded from the present study due to neglence of instructed oral hygiene measures or irregular attendance according to the designed schedule. Hence, the results presented are those of eleven individual. Moreover, it must be remembered logically that a written consent was obtained from all participants before sharing this study.

Figure 1: Periapical radio graph illustrating bone loss related to furcation areas of first and second mandibular molars _ Before treatment

Figure 2: Clinical photograph of buccal furcation defects of mandibular molars after debridement of the area

Figure 3: Clinical photograph of the same case after placement of mixture PHA and PRP in furcation defected area.

Changes in pocket depth over 6 months among studied groups showed a statistically significant reduction ($P < 0.001$). The changes in clinical attachment level as for both groups showed almost parallel patterns as that of pocket depth changes (Figure 4). There was a statistically significant gain in clinical attachment level for both groups ($P < 0.001$). Initially, they

were 5.9 ± 1.1 and 5.7 ± 1.2 mm. and after 6 months recorded 4.6 ± 1.3 and 4.2 ± 0.9 mm; for control and grafted sites, respectively. The difference between both sites in the change over time was not statistically significant.

The changes in horizontal probing depth for both groups showed a statistically significant reduction in horizontal probing depth after 6 months; when compared to the baseline measures ($P < 0.001$). The mean reduction reached 1.7 mm

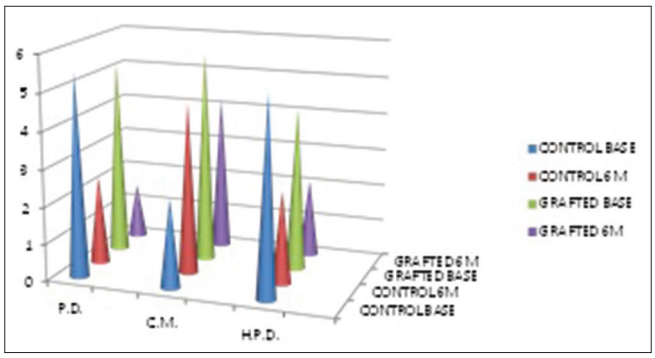

Figure 4: The interrelationship among P.D., CAL and H.P.D_ before and after treatment both groups total cases

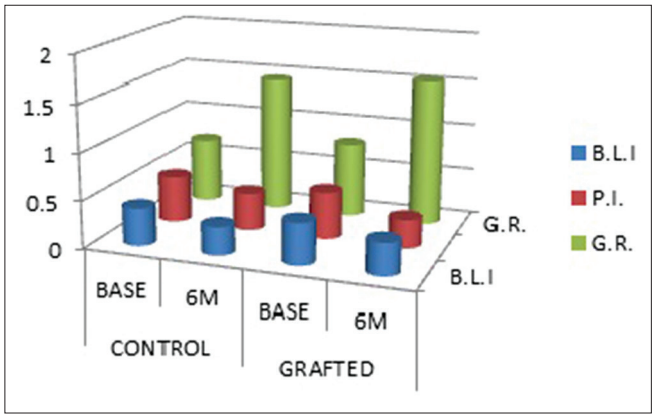

Figure 5: The interplay between the clinical parameters (B.L.I and P.I. scores)_ and Gingival recession_ throughout the versions observation periods _ total cases both groups.

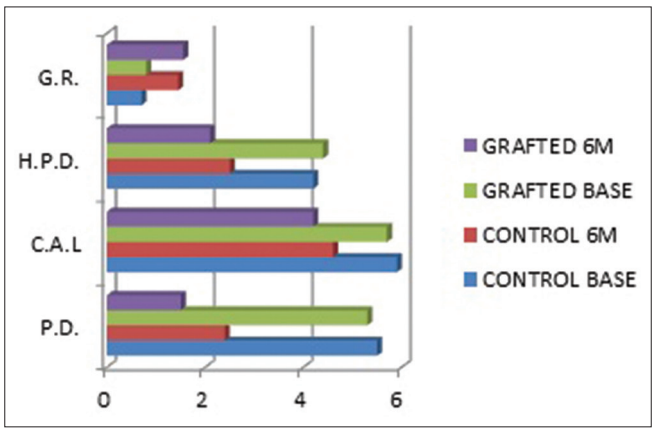

Figure 6: The interrelationship among P.D., CAL ,H.P.D and G.R._ before and after treatment both groups total cases

for control sites and 2.3 mm for grafted sites. A statistically significant difference existed between both groups in the change over time (Figures 4 and 6). Both treatment modalities showed a relative clinical gingival recession at the end of study period (6 months). The mean difference between both sites recorded 0.1 ± 0.39 in gingival recession values. The difference in recession over time in both sites was statistically significant ($P < 0.001$); (Figure 5). In addition to the above-mentioned records, plaque and bleeding index scores revealed no significant changes over time for both studied sites (Figure 5).

The radiographic changes at the end of study period (6 months), showed a relative decrease in graft size and radiopacity (Figure 7); which appeared almost similar to adjacent bone (data not presented). No statistically significant differences could be achieved between both sites; as for change over time.

Discussion

Periodontitis is defined as inflammation involving and destroying the supporting alveolar bone and periodontal ligament. This occurs due to the accumulation of dental plaque in the gingival sulcus which provides an adequate environment for colonization and proliferation of bacteria. The plaque bacteria are capable of initiating the mechanisms of periodontal tissue destruction.[16-18]

The ultimate goal of periodontal therapy included the arrest of progressive periodontal disease and the restoration of those parts of the periodontium that were destroyed by the disease.[19,20]

Furcation involvements represent one of the most demanding therapeutic challenges in periodontics due to their anatomical and geometrical variations. Moreover, teeth with furcation involvements, generally, have a less favorable prognosis than those whose furcation is intact. A convincing management of furcation defects has been an area of indeed interest and research.[21-23]

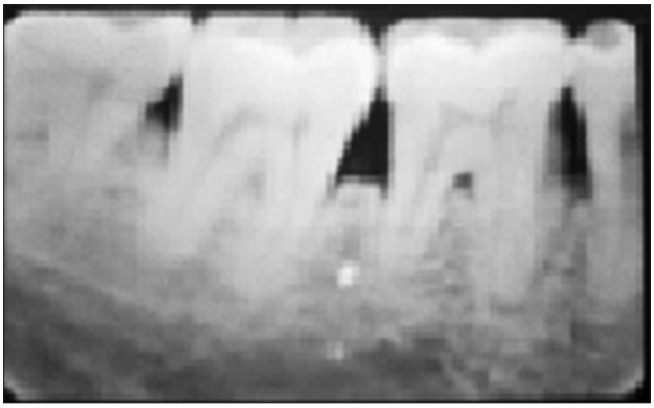

Figure 7: Postoperative periapical radiograph showing an almost relative filling of the furcated boney defect (six months).

In the presented study, the split-mouth technique is utilized to avoid as possible; the individual variations, which could render accuracy and standardization of the obtained results. All patients completed the scheduled study periods, as evidenced by their regular attendance. No reluctant cases were reported during the entire follow-up periods.

The parameters used in assessing the periodontal condition are those commonly used in clinical trials.[13] These reversible indices had proved to be useful means of screening the periodontal condition. Moreover, they also provide the possibility of selecting specified areas or teeth when large numbers are examined, or utilizing all areas of all teeth in the examination of a small sample. Furtherly, they also combine the degree of inflammation with the assumed main etiological factors; dental plaque.

The aim of the present study focused on the possible role of PRP in augmenting the effects of PHA in promoting regeneration of furcated bony defects in humans. PHA was selected to act as filler and/or scaffold for PRP since PHA is an alloplast that offers the advantages of unlimited quantity, no need for the additional surgical site, and no potential for disease transmission.[24]

Evaluation of the treatment modality after 6 months in this study; although considered too short time to fully evaluate the effect of periodontal therapy. Yet, this time frame seems to be the standard for this type of research whether natural materials, synthetic materials, or surgical debridement is the therapy provided.[25-27] Moreover, this period was chosen also, because the soft tissue changes are usually completed within 6 months following treatment[28] a finding which is comparable to that performed by other investigators.[24,29-31]

Several alloplastic materials have been utilized in an attempt to improve clinical conditions and promote regeneration of bony defects accompanying periodontal diseases.

Clinical results with synthetic graft materials are more or less similar to results obtained with autogenous or allogenic materials; both in direct comparative studies and in cross-sectional comparisons of similar evaluations and studies. The choice of material then becomes based more on availability, cost, morbidity, and ease of handling than on clinical superiority.[6,9,15]

PHA alloplast was used successfully in the management of furcations and intraosseous defects where clinical studies showed pocket reduction, attachment gain and bone level improvement.[19,21,22,29,31]

The application of PRP in clinical periodontal regeneration community was found more effective than open flap debridement alone; as it can contribute to the improvement of clinical outcome of grafted sites (PRP + PHA). The biological effects of PRP could be achieved via secretion of growth factors; which is initiated by the clotting process of blood when PRP is activated by thrombin.[19,21,22,29,31] The secreted growth factors could immediately bind to their transmembrane receptors on adult mesenchymal stem-cells, osteoblasts, fibroblasts, endothelial cells with subsequent cellular proliferation, matrix formation, osteoid production, and collagen synthesis through cellular message transforming. Moreover, PRP also contains 3 plasma proteins known to act as cell adhesion molecules for osteoconduction and as a matrix for bone end connective tissue, namely, fibrinogen, fibronectin, and vitronectin.[32-34] Furtherly, once PRP preparation is coagulated, it assumes a sticky consistency due to its high fibrin content; a finding which could serve as a hemostatic agent, and stabilizes the graft material and the blood clot.[14] In addition to the forementioned data, PRP is an autogenous preparation and is inherently safe and free from concerns over transmissible diseases. Its preparation is also a simple and rapid procedure; with a consistency that could improve the handling properties of the graft material, and may be helpful to shorten the operating time.[15]

The results of the present study showed that, PRP combined with PHA resulted in statistically significant greater reduction in probing depth and statistically significant greater gain in clinical attachment level than open debridement alone, i.e., PRP augmented the beneficial effect of PHA in the treatment of bony defects. These results are in accordance to other clinical trials which revealed that PRP can augment the results of bone replacement grafts and barrier membranes in the treatment of infrabony defects, furcation defects and around dental implants.[21,24,26,29]

The radiographic findings are a more or less parallel to those of clinical results, and furtherly, in accordance to those who reported that growth factors obtained by adding PRP to bone graft showed radiographic maturation rate 1.62 times than that of grafts without PRP.[35] Moreover, it has been found that compared grafted to ungrafted furcations have shown a little positive response to surgical debridement alone. In other words, both sides showed an almost comparable improvement in most clinical aspects except for a more significant improvement in horizontal probing in favor of grafted side. These findings are almost in agreement with the observations of other investigators;[24,25,28,30] who attributed the reduction in horizontal probing depth to the resorption of graft particles in bony defects. This could occur via solution-mediated resorption as a consequence of the PH of surrounding media. The lowered PH level is usually accompanied by the presence of inflammation; a fact which could explain the forementioned findings.[19,27,30]

Conclusion

It could be concluded that a combination of PRP and PHA were clinically useful in the management of Class II

furcation defects; compared to surgical sites alone. Moreover, regenerative periodontal surgery using the forementioned combination could be proposed as a modality; to enhance treatment outcome in furcated bony defects.

References

1. Hart TC, Kornman KS. Genetic factors in the pathogenesis of periodontitis. Periodontol 2000 1997;14:202-15.
2. De Leonardis D, Garg AK, Pedrazzoli V, Pecora GE. Clinical evaluation of the treatment of Class II furcation involvements with bioabsorbable barriers alone or associated with demineralized freeze-dried bone allografts. J Periodontol 1999;70(1):8-12.
3. Eickholz P, Hausmann E. Evidence for healing of class II and III furcations after GTR therapy: Digital subtraction and clinical measurements. J Periodontol 1997;68(7):636-44.
4. Hou GL, Tsai CC. Types and dimensions of root trunk correlating with diagnosis of molar furcation involvements. J Clin Periodontol 1997;24(2):129-35.
5. Meijer GJ, de Bruijn JD, Koole R, van Blitterswijk CA. Cell-based bone tissue engineering. PLoS Med 2007;4(2):e9.
6. Khang D, Carpenter J, Chun YM. Nanotechnology for regenerative medicine. Biomed Microdevices 2008;64:31-6.
7. Kolenbrander PE, Palmer RJ Jr., Rickard AH, Jakubovics NS, Chalmers NI, Diaz PI. Bacterial interactions and successions during plaque development. Periodontol 2000-2006;42:47- 79.
8. van Winkelhoff AJ, Winkel EG. Microbiological diagnostics in periodontics: Biological significance and clinical validity. Periodontol 2000 2005;39:40-52.
9. Giannoudis PV, Pountos I. Tissue regeneration. The past, the present and the future. Injury 2005;36 Suppl 4:S2-5.
10. Abramson JH. The cornell medical index as an epidemiological tool. Am J Public Health Nations Health 1966;56(2):287-98.
11. Glickman I. Clinical Periodontology, 3rd ed. Philadelphia: W.B. Saunders; 1953.
12. Silness J, Löe H. Periodontal disease in pregnancy 3. Response to local treatment. Acta Odontol Scand 1966;24(6):747-59.
13. Löe H. The gingival index, the plaque index and the retention index systems. J Periodontol 1967;38(6):610-6.
14. Camargo PM, Lekovic V, Weinlaender M, Vasilic N, Madzarevic M, Kenney EB. Platelet-rich plasma and bovine porous bone mineral combined with guided tissue regeneration in the treatment of intrabony defects in humans. J Periodontal Res 2002;37(4):300-6.
15. Lekovic V, Camargo PM, Weinlaender M, Vasilic N, Aleksic Z, Kenney EB. Effectiveness of a combination of platelet-rich plasma, bovine porous bone mineral and guided tissue regeneration in the treatment of mandibular grade II molar furcations in humans. J Clin Periodontol 2003;30(8):746-51.
16. Goodson JM, Palys MD, Carpino E, Regan EO, Sweeney M, Socransky SS. Microbiological changes associated with dental prophylaxis. J Am Dent Assoc 2004;135(11):1559-64.
17. Herrera D, Roldán S, Sanz M. The periodontal abscess: A review. J Clin Periodontol 2000;27(6):377-86.
18. Haffajee AD, Socransky SS. Microbial etiological agents of destructive periodontal diseases. Periodontol 2000 1994;5:78-111.
19. Langer R, Vacanti C. Bone formation by autoinuction. Science 2008;150:893-9.
20. Massagué J. The transforming growth factor-beta family. Annu Rev Cell Biol 1990;6:597-641.
21. Burg KJ, Porter S, Kellam JF. Biomaterial developments for bone tissue engineering. Biomaterials 2000;21(23):2347-59.
22. Jin QM, Anusaksathien O, Webb SA, Rutherford RB, Giannobile WV. Gene therapy of bone morphogenetic protein for periodontal tissue engineering. J Periodontol 2003;74(2):202-13.
23. Asiyanbola B, Soboyejo W. For the surgeon: An introduction to nanotechnology. J Surg Educ 2008;65(2):155-61.
24. Zohar R, Tenenbaum HC. How predictable are periodontal regenerative procedures? J Can Dent Assoc 2005;71(9):675-80.
25. Heitz-Mayfield LJ. How effective is surgical therapy compared with nonsurgical debridement? Periodontol 2000 2005;37:72-87.
26. Christgau M, Moder D, Hiller KA, Dada A, Schmitz G, Schmalz G. Growth factors and cytokines in autologous platelet concentrate and their correlation to periodontal regeneration outcomes. J Clin Periodontol 2006;33(11):837-45.
27. Ouyang XY, Qiao J. Effect of platelet-rich plasma in the treatment of periodontal intrabony defects in humans. Chin Med J (Engl) 2006;119(18):1511-21.
28. Jeffcoat M. What is clinical significance? J Clin Periodontol 2002;29 Suppl 2:30-2.
29. Drosse I, Volkmer E, Capana R. Tissue engineering for bone defects healing. Injury 2008;39(2):15-20.
30. Soucacos PN, Johnson EO, Eabis G. An update on recent advance in bone regeneration. Injury 2008;39(2):15-20.
31. Okuda K, Tai H, Tanabe K, Suzuki H, Sato T, Kawase T, *et al.* Platelet-rich plasma combined with a porous hydroxyapatite graft for the treatment of intrabony periodontal defects in humans: A comparative controlled clinical study. J Periodontol 2005;76(6):890-8.
32. Sánchez AR, Sheridan PJ, Kupp LI. Is platelet-rich plasma the perfect enhancement factor? A current review. Int J Oral Maxillofac Implants 2003;18(1):93-103.
33. Philippart P, Brasseur M, Hoyaux D, Pochet R. Human recombinant tissue factor, platelet-rich plasma, and tetracycilne induce a high-quality human bone graft: A 5-year survey. Int J Oral Maxillofac Implants 2003;18(3):411-6.
34. Borzini P, Mazzucco L. Platelet gels and releasates. Curr Opin Hematol 2005;12(6):473-9.
35. Marx RE. Platelet rich plasma: What is platelet rich plasma and what in not platelet rich plasma? Implant Dent 2001;10:225.