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Case Report

Periodontal Regeneration Using Platelet Rich Fibrin, Demineralized Freeze Dried Bone Allograft and a Bio-absorbable Barrier: A 6-Month Follow- Up Case Report

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Abstract

The aim of any invasive procedure is the complete eradication and elimination of the infection and associated necrotic tissue followed by repair and regeneration of the affected tissue. Choukroun's Platelet Rich Fibrin (PRF) is one such material, used by itself and also as an adjunct with grafts. PRF holds on to the growth factors enmeshed in the fibrin network resulting in their sustained release over a period of time that can accelerate the wound healing process. This case report investigates a 6-month follow-up, clinically and radiographically the effectiveness of autologous PRF along with the use of DFDBA (Demineralized freeze dried bone graft) and GTR membrane in the management of intrabony defects.

The following case report has been presented at 2nd International Congress of World Academy of Growth Factors and Stem Cells in Dentistry (WAGro) held during 25 - 27th October 2018.

Keywords: *Intrabony Defects; DFDBA; Platelet Rich Fibrin; Regeneration*

Abbreviations

BMP: Bone Morphogenetic Proteins; CAL: Clinical Attachment Level; DFDBA: Demineralized Freeze Dried Bone Allograft; GTR: Guided Tissue Regeneration; IBD: Intrabony Defects; mm: Millimeters; OPG: Orthopantomogram; OHI-S: Oral Hygiene Index; PRF: Platelet Rich Fibrin; PPD: Probing Pocket Depth; rpm: Rotations Per Minute; TDS: Three Times a Day; UNC: University North Carolina

Introduction

Periodontal disease is defined as a complex, multifactorial disease characterized by the loss of connective tissue attachment with destruction of periodontal tissues. The aim of periodontal therapy is to eliminate inflammatory process, prevent the progression of periodontal disease and also to regenerate the lost periodontal tissues [1].

Access flap surgery is a recognized standard therapy to manage residual pockets after cause-related therapy but conventional open flap debridement falls short of regenerating tissues destroyed by the disease.

Regeneration has been defined as "reproduction or reconstruction of a lost or injured part in such a way that the architecture and function of the lost or injured tissues are completely restored. This takes place by growing precursor cells replacing lost tissue" [2].

Periodontal regeneration requires an orchestrated sequence of biologic events such as cell migration, adherence, growth and differentiation, to have the potential to increase the success and predictability of periodontal regenerative procedures [2].

Platelets regenerative potential was intro¬duced in the 70's, when it was observed that they contain growth factors that are responsible for increase collagen production, cell mitosis, blood vessels growth, recruitment of other cells that migrate to the site of injury, and cell differentiation induction [3].

Periodontal regenerative procedures include soft tissue grafts, bone replacement grafts, root biomodifications, GTR, and combinations thereof for osseous, furcation, and recession defects [4].

Here, we present a 6-month follow-up report of an intra-bony defect assessed with clinical and radiological parameters that was treated with homologous mixture of autologous PRF and DFDBA along with a bio-resorbable GTR membrane.

Case Report

A 44 year old Indian female reported to the Department of Periodontology, Rural Dental College, Loni, Maharashtra, with a chief complaint of bleeding from gums and food lodgment in the upper right back region.

Patient did not give any contributory medical history or presence of any systemic condition that could interfere with physiological wound healing. There was no history of dental trauma or orthodontic treatment, and no adverse habit was reported by the patient.

Upon intra-oral examination; it was noticed that patient had generalized periodontal pockets. PPD ranged from 4 - 8 mm and overall

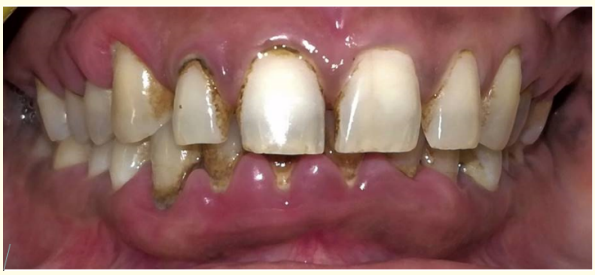

Figure 1: *Intra-oral Frontal View at Presentation.*

OPG revealed bone loss with angular bone defect mesial to #16 (Figure 2). Patient's hematological reports were assessed and found to be within normal limits. On the basis of clinical and radiographic examination, the diagnosis of this case was Generalized Chronic Periodontitis (AAP1999, Consensus Report).

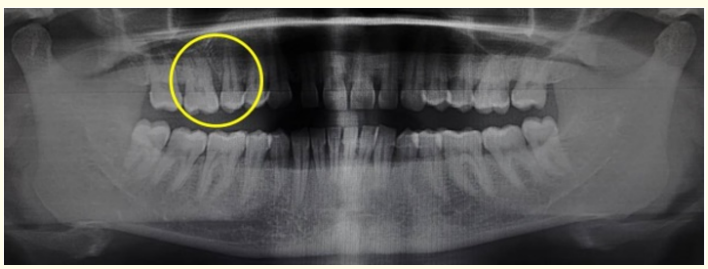

Figure 2: *Orthopantomogram.*

Patient was explained about a comprehensive treatment plan (explained in this paper; as and when required) that was formulated based on the clinical and radiographic examination. After a written consent from the patient, treatment was initiated.

Etiotrophic phase

1. Oral hygiene instructions and motivation of the patient in performing effective oral hygiene measures.
2. Non-surgical periodontal therapy by means of conventional scaling and root planning, using curettes and ultrasonic instruments, with coronoplasty to remove trauma from occlusion and usage of chlorhexidine mouthwash 0.2% twice daily for 7 days.
3. Recall after every week and re-examination of the patient after 4 weeks following non-surgical periodontal therapy.

Follow-up after etiotrophic phase

Upon follow up it was noticed that the clinical parameters after the non-surgical periodontal therapy, at the end of 4 weeks, it was found that with respect to #14 - #17; bleeding on probing was present, PPD and CAL on the mesial and distal surfaces (measured using UNC-15 Probe) were 8 mm and 5 mm respectively was noticed with respect to #16 (Figure 3). Hence, surgical periodontal therapy was scheduled.

Figure 3: *Interproximal PPD.*

Surgical phase

1. Localized periodontal flap therapy with #14 - #17 (Combination of Kirkland and Modified Widman Flap surgery was carried out on the distobuccal aspect of #16). Patient performed pre-procedural mouthrinse with 0.2% Chlorhexidine digluconate and was scrubbed with Betadine solution (extra-oral asepsis) prior to commencing surgery.
2. Following local anesthesia administration, buccal and palatal full thickness mucoperiosteal flaps were reflected.
3. Thorough debridement was done (Figure 4) and PRF was prepared (Figure 5) by collecting 10 ml of intravenous blood in a sterile tube without anticoagulant and immediately centrifuged at a speed of 1600 rpm for 12 minutes. A structured fibrin clot in the middle of the tube, just between the red corpus¬cles at the bottom and acellular plasma (Platelet-poor plasma) at the top was obtained and after the removal of PPP and PRF was then transferred onto a sterile dapen dish that was mixed with DFDBA bone graft until a homogenous mixture was obtained.
4. After pre-suturing of the flaps, placement of PRF+ DFDBA Bone Graft in the 3-wall intrabony defect (IBD) in the mesial aspect of #16 and GTR membrane (Healiguide) was placed (Figure 6).
5. Periodontal dressing and sutures were placed (Figure 7).
6. After 10 days, dressing was removed and the surgical site was gently cleaned and irrigated using saline and betadine during suture removal.

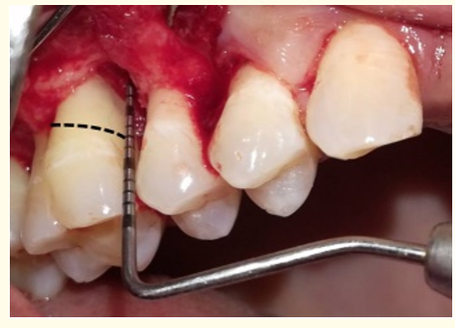

Figure 4: *Intrabony Defect noticed upon surgical exploration.*

Figure 5: *Preparation of PRF.*

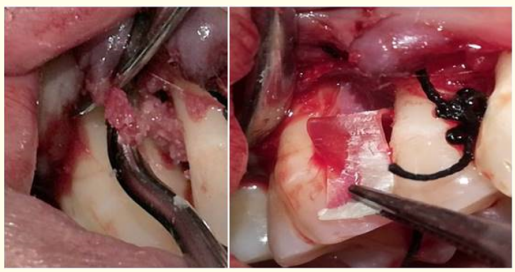

Figure 6: *PRF + DFDBA + Healiguide placement at IBD site.*

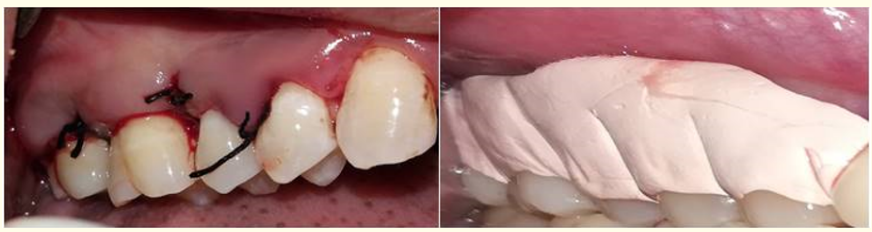

Figure 7: *Sutures and periodontal dressing in place.*

Post-operative care

1. Antibiotics and analgesics were prescribed:
    a. Antibiotics: Amoxycillin 500 mg TDS for 5 days.
    b. Analgesics: A combination of Paracetamol (500 mg) and Aceclofenac (100 mg) for 3 days.
2. Chlorhexidine digluconate 0.2% concentration was advised to be used twice daily in 1:1 ratio.
3. Patient was advised to avoid chewing and brushing at the surgical site for 10 days.
4. After 10 days, upon suture removal, oral hygiene instructions were reinforced.

Patient was examined weekly up to 1 month after surgery. Regular follow up till 6 months post- surgery as part of maintenance phase was followed. No periodontal probing and sub-gingival instrumentation was attempted during this period.

Re-examination after 6 months showed a dramatic decrease in PPD (mesial and distal PPD measured 3 mm with William's Graduated Probe) with no sign of bleeding on probing (Figure 8) and a significant radiographic bone formation mesial to #16 (Figure 9).

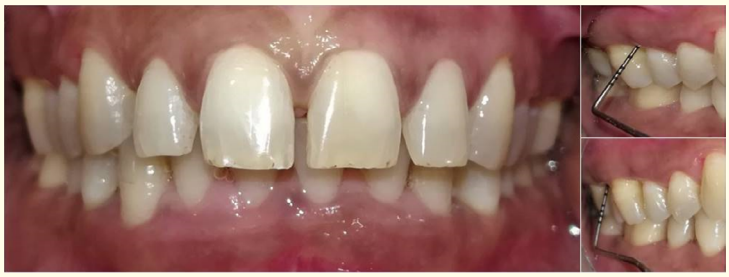

Figure 8: *6-Months Post-Operative Intra-oral View.*

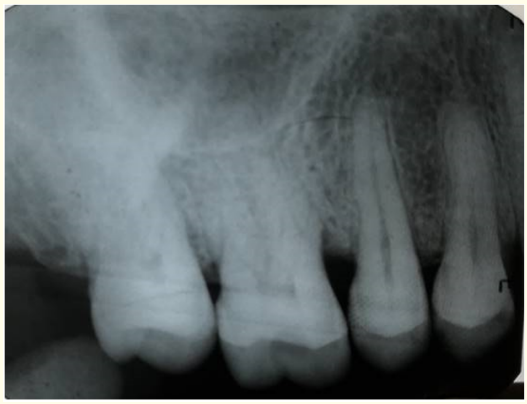

Figure 9: *6-Months Post-Operative IOPA.*

Discussion

Periodontal destruction occurs in an episodic, intermittent manner, with periods of inactivity or quiescence. The destructive periods leads to loss of collagen and alveolar bone with deepening of the periodontal pocket [5].

Page and Schroeder [5], postulated a range of effectiveness of about 1.5 - 2.5 mm within which bacterial plaque can induce loss of bone. Interproximal angular defect can appear only in spaces that are wider than 2.5 mm because narrower spaces would be destroyed entirely.

Neilson*., et al.* [6] reported vertical defects that were detected radiographically have been reported to appear most commonly on the interproximal surfaces. However, three wall defects are more frequently found on the mesial surfaces of upper and lower molar and Papapanou*., et al.* [7] found that the frequency of IBD increases with age and more commonly in the mesial than distal tooth surfaces (ratio 1.6: 1). Similarly in our case, intrabony defect was noticed with maxillary first molar affecting its mesial aspect.

Various biomaterials have been introduced and used in different combinations for regeneration of lost periodontal structures, especially the lost alveolar bone, which is a matter of prime concern in the clinical management of periodontal disease as bone destruction is primarily responsible for tooth loss.

The present case report evaluated the clinical efficacy of PRF + DFDBA along with GTR membrane which served the purpose of a barrier to prevent the reduction in PPD clinically and complete healing of IBD radiographically.

PRF was first described by Choukroun., *et al.* in 2004 in France. It is referred to as a second generation platelet concentrate as the natural concentrate is produced without any anticoagulants. PRF consists of an intimate assembly of cytokines, glycanic chains, and structural glycoproteins enmeshed within a slowly polymerized fibrin network. This biologic activity of fibrin molecule and the slow polymerization mode confers to the PRF membrane a particularly favorable physiological architecture to support the healing process [4].

Concept of GTR introduced by Melcher, utilizes placement of a physical barrier between the gingival flap and the defect before flap repositioning and suturing prevents gingival epithelium and connective tissue (undesirable cells) from contacting the space created by the barrier. It also facilitates repopulation of the defect by regenerative cells [8].

DFDBA is known to have an osteogenic potential that is manifested by exposing BMPs which presumably have the ability to induce host cells to differentiate into osteoblast [4]. DFDBA is available in various particle sizes. For our case we used particle size < 500 microns DFDBA.

Dohan., *et al*. [9] reported that the interleukin (IL) 1β, IL- 6, TNF-α, IL -4, vascular endothelial growth factor (VEGF) in the PRF clot play a crucial role in balancing the tissue homeostasis, whereas the healing cytokines IL- 4 and VEGF inhibit inflammatory signal pathways thereby support and coordinate the neovascularization which may be the reason for uneventful healing.

Mellonig., *et al.* [10] carried out a study to evaluate DFDBA in human periodontal defects. Defects evaluated after 6 months postoperatively showed 64.7% of bone-fill in the sites which were treated with DFDBA compared to just open flap debridement.

Conclusion

PRF when used as a membrane or as a grafting material facilitates cell events that are favorable for periodontal regeneration. PRF acts as a biological connector between bone particles and a combination of PRF and DFDBA demonstrated better clinical results.

Declaration of Patient Consent

The authors certify that the patient had given consent for images and other clinical information to be reported in the journal. Patient understands that efforts will be made not to reveal identity.

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