The Clinical Application of Platelet-Rich Fibrin (Prf) and Allograft In Treatment Of Bony Defect - A Case Report

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ABSTRACT: A recent promising innovation in regeneration procedures is the preparation and use of PRF, a concentrated suspension of growth factors found in platelets. Platelet-rich fibrin (PRF) may be considered as a second-generation platelet concentrate widely used to accelerate soft and hard tissue healing because of presence of many growth factors. For complete periodontal regeneration, delivery of growth factors in the local environment holds a great deal in adjunct to bone grafts. This case report present the application of PRF along with bone graft in the treatment of intrabony defect.

KEY WORDS: Platelet-rich fibrin, intrabony defect, bone graft

I. Introduction

Regeneration has been defined as the reproduction or reconstitution of a lost or injured part to restore the architecture and function of the periodontium. ^[1] Periodontal regeneration is a multifactorial process and requires a multi- dependent sequence of biological events, including cell- adhesion, migration, proliferation, and differentiation. ^[2] The goal of periodontal therapy includes not only the arrest of periodontal disease progression, but also the regeneration of structures lost due to disease. ^[3] Growth factors are the vital mediators during this process which can induce the migration, attachment, proliferation and differentiation of periodontal progenitor cells.

Platelet-rich fibrin (PRF) described by Choukroun et al.^[4] is a second generation platelet concentrate which allows one to obtain fibrin membranes enriched with platelets and growth factors, after starting from an anticoagulant-free blood harvest.^[5,6] Enhancement of the regenerative process of human body by utilizing the patient's own blood is a unique concept in dentistry. Post-surgically, blood clots initiate the healing and regeneration of hard and soft tissues. Platelet rich fibrin (PRF) is emerging as a biological revolution in the dental field. Using platelet-rich fibrin, or PRF, is a way to accelerate and enhance the body's natural wound-healing mechanisms. Platelets primarily are involved in wound healing through clot formation and the release of growth factors that initiate and support wound healing.^[7] Many growth factors, such as platelet-derived growth factor (PDGF) and transforming growth factor (TGF-b), are released from PRF.^[5,6,8]

Recently, studies have demonstrated that the PRF membrane has a very significant slow sustained release of key growth factors for at least one week^[5] and up to 28 days,^[9] which means that the membrane stimulates its environment for a significant time during wound healing. The PRF production protocol attempts to accumulate platelets and released cytokines in a fibrin clot. This technology permits to safely harvest and produce a sufficient quantity of platelets from only 8-10 ml of blood drawn from patients in dental office. Surgical sites enhanced with PRF have been shown to heal at rates two to three times that of normal surgical sites. ^[10] PRF is both a healing and interpositional biomaterial. As a healing material, it accelerates wound closure and mucosal healing due to fibrin bandage and growth factor release. As interpositional material, it avoids the early invagination of undesired cells, thereby behaves as a competetive barrier between desired and undesired cells. ^[11]

Following cases describes the healing of a defect which was treated using PRF along with bone graft.

II. Case Report

A 23-year-old female patient was referred to the Department of Periodontology, complains of tooth pain that was dull and excruciating. On periodontal examination and radiographic evaluation, the patient presented with a bony defect extending from distal surface of mandibular right first premolar to mesial surface of mandibular first molar with a probing depth of 8 mm. The patient did not present with pain in relation to number 45 tooth and had no pain on percussion. There was positive electric pulp test response suggesting the concerned tooth was vital. The diagnosis was made to be localized chronic periodontitis. Scaling and root planning of the teeth were performed. [Figure 1] Four weeks following Phase 1 therapy, a periodontal re- evaluation was performed to confirm the suitability of number 45 tooth for periodontal surgical procedure with a bone graft and PRF.

Blood sample was taken on the day of the surgery according to the PRF protocol with an REMI 3000 centrifuge and collection kits. Briefly, 5 ml blood sample was taken from the patient without an anti- coagulant in 10 ml glass test tubes [Figure 2] and immediately centrifuged at 3000 rpm for 10 min. [Figure 3] A fibrin clot was formed in the middle of the tube, whereas the upper part contained cellular plasma, and the bottom part contained red corpuscles. [Figure 4] The fibrin clot was easily separated from the lower part of the centrifuged blood. The PRF clot was gently pressed between two sterile dry gauges to obtain a membrane which was later minced and added to the allogenic graft material (OSSEOMOLD).

An intrasulcular incision was made on labial and lingual aspect of the tooth number 43, 44, 45, 46. [Figure 5] A full thickness flap was raised, and inner surface of the flap was curetted to remove the granulation tissue. Root surfaces were thoroughly planed using hand instruments and ultrasonic scalers. The right mandibular second premolar demonstrated a crater type intrabony defect extending lingually from mesial surface of mandibular right second premolar to mesial surface of mandibular right first molar, after removing granulation tissue thoroughly, the intrabony defect was found to extend in lingual and apical aspect. [Figure 6]

Briefly, minced PRF was mixed with allograft (OSSEOMOLD) and was applied to the defect walls and root surfaces [Figure 7]. The allograft with PRF was then condensed using amalgam condensers. [Figure 8] The flap were repositioned to their presurgical levels and sutured with silk utilizing an interrupted technique. [Figure 9] Periodontal dressing was given to the patient. [Figure 10]

After the surgery, the patient was prescribed systemic antibiotics (amoxicyllin 500 mg tid, 5 days), nonsteroidal antiinflammatory drug (ibuprofen 600 mg tid, 5 days) and 0.12% chlorhexidine rinse (twice a day for 4 weeks). Sutures were removed after 7 days. Clinical healing was normal with neither infectious episodes nor untoward clinical symptoms. [Figure 11,12]

In this case report, the reduction in pocket depth and gain in clinical attachment were found after 6 months of follow- up.

III. Discussion

The four critical factors that influence bone regeneration after the periapical surgery are primary wound closure, angiogenesis as a blood supply and source of undifferentiated mesenchymal cells, space maintenance, and stability of the wound (PASS principle).^[12] Alloograft has shown positive results with respect to periodontal regeneration in periapical defects.^[13] It has been reported that combination of Allograft with PRF resulted in greater pocket depth reduction, gain in clinical attachment and defect fill than PRF used alone.^[14] For this reason, we chose allograft, as that it could enhance the effects of PRF by maintaining the space for tissue regeneration to occur, as well as by exerting an osteoconductive effect in the bony defect area. The intended role of the minced PRF in the intrabony defect was to deliver the growth factors in the early phase of healing.

Platelet rich fibrin by Choukroun's technique is prepared naturally without addition of thrombin, and it is hypothesized that PRF has a natural fibrin framework and can protectgrowth factors from proteolysis.^[15] Thus, growth factors can keep their activity for a relatively longer period and stimulate tissue regeneration effectively. The main characteristics of PRF compared with other platelet concentrates, including PRP, are that it does not require any anti- clotting agent.^[16] The naturally forming PRF clot has a dense and complex three- dimensional architecture and this type of clot concentrates not only platelet, but also leukocytes. PRF is simpler and less expensive to prepare, as well as being less risky to the patients. Owing to its dense fibrin matrix, PRF takes longer to be resorbed by the host, which results in slower and sustained release of platelet and leukocyte derived growth factors in to the wound area.^[17,18]

In this case report, the reduction in pocket depth and gain in clinical attachment were found after 6 months of follow- up. These are the important clinical outcomes for any periodontal regenerative procedures.

Furthermore, many growth factors are released from PRF as PDGF, TGF and has slower and sustained release up to 7 days^[19] and up to 28 days,^[20] which means PRF stimulates its environment for a significant time during remodeling. Moreover, PRF increase cell attachment, proliferation, and collagen related protein expression of human osteoblasts.^[21] PRF also enhances protein kinase- like endoplasmic reticulum kinase, OPG and ALP expression, which benefits periodontal regeneration by influencing human PDL fibroblasts.^[22]Since

the surface of PRF membrane is smoother, it can cause superior proliferation of human periosteal cells thereby enhancing bone regeneration.^[23]

IV. Conclusion

According to the results obtained in this case report, the positive clinical impact of additional application of PRF with allogenic graft material in treatment of periodontal intrabony defect is based on:

- Reduction in probing pocket depth
- Gain in clinical attachment level

To conclude, PRF membrane has been used as a barrier membrane over a large bony defect to maintain a confined space for the purpose of guided tissue regeneration. On the basis of the results obtained in our case report, we hypothesize that the use of PRF in conjunction with HA crystals might have accelerated the resorption of the graft crystals and would have induced the rapid rate of bone formation.

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Figure 1

Figure 2



Figure 3

Figure 4



Figure 5



Figure 6



Figure 7



Figure 8



Figure 9



Figure 10



Figure 11



Figure 12