

Case report**Platelet Rich Fibrin in Tissue Engineering: A boon to Periodontal Regeneration**

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Abstract: The ultimate goal of periodontal therapy is regeneration of tissues destroyed by periodontal disease. Previously, various bone graft materials have been tried for management of osseous defects resulting from the disease process. The recent introduction of Choukroun's Platelet Rich Fibrin (PRF), has revolutionized the field of dentistry. PRF, a rich source of autologous growth factors and cytokines, is an upcoming therapeutic approach in the management of periodontal osseous defects. The purpose of this case report is to present clinical and radiographic results of a wide intrabony periodontal defect treated with PRF.

Key words: Intrabony; Defect; Platelet concentrates; Platelet Rich fibrin; Periodontal; Autograft.

INTRODUCTION:

Periodontitis is an infectious disease that causes destruction of the tooth attachment apparatus. Untreated periodontitis results in progressive attachment loss that may eventually lead to early tooth loss. There are a broad range of treatment options available, but only some may be regarded as truly regenerative procedures. The aim of regenerative periodontal therapy is to restore the structure and function of the periodontium. 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. For many years, research has attempted to use biologically active molecules to achieve periodontal regeneration.¹

Platelet-rich fibrin (PRF), a second generation platelet concentrate has been introduced by Choukroun *et al.* in 2001. PRF is a matrix of autologous fibrin, in which are embedded intrinsically a large quantity of platelet and leukocyte cytokines during centrifugation leading to their progressive release over time (7-11 days), as the network of fibrin disintegrates.² Platelet rich fibrin (PRF) enriched with platelets and growth factors promotes periapical tissue regeneration and healing.³

The PRF clot forms a strong natural fibrin matrix, which concentrates almost all the platelets and growth factors of the blood harvest

and shows a complex architecture as a healing matrix with unique mechanical properties which makes it distinct from other platelet concentrates. PRF is superior to other platelet concentrates like PRP due to its ease and inexpensive method of preparation and also it does not need any addition of exogenous compounds like bovine thrombin and calcium chloride. It is advantageous than autogenous graft also because an autograft requires a second surgical site and procedure.⁴

PRF dwells among a new generation of platelet concentrate that jump-starts the healing process to maximize predictability. It consists of the platelets, cytokines, and the fibrin matrix. Platelets and leukocyte cytokines play an important part in the biology of this biomaterial. Degranulation of platelets entails the release of cytokines able to stimulate cell migration and proliferation within the fibrin matrix, launching the first stages of healing. Fibrin matrix supporting them constitutes the determining element responsible for the real therapeutic potential of PRF. The biologic activity of the fibrin molecule highlights its significant cicatricial capacity.⁵

Periodontists have been experimenting various modalities for regeneration in Periodontal defects with varying degree of success. Periodontal surgery combined with platelet rich fibrin (PRF) has been introduced as a method to promote regeneration of the lost periodontium.⁶ Platelet-rich fibrin (PRF) represents a new step

in the platelet gel therapeutic concept. Clinical data reveal that this biomaterial would be a favorable matrix for the development of a coherent healing without inflammatory excess. It is therefore very important to identify in the PRF clot all the powerful homeostatic regulation molecules capable of controlling postsurgical inflammation.⁷

CASE REPORT:

A 27-year-old female complaining of food lodgment and pain in the upper right maxillary incisor region reported to the Department of Periodontics, Rama Dental College and Research Centre, Kanpur, India. Patient did not give any relevant medical history and there was no systemic condition that could interfere with physiological wound healing. There was no history of dental trauma or orthodontic treatment, and no injurious habit was reported by the patient. On intraoral examination, there was generalized bleeding on probing present but no swelling and no pus exudation was noticed. Pathologic migration was present in 11 & 12 (fig.1). The probing pocket depth (PPD) on the distobuccal aspect of the tooth # 11 was 9 mm. Fremitus test was found to be negative precluding the possibility of trauma from occlusion.



Fig.1: Intrabony Defect

A Periapical radiograph was taken using the standardized techniques, which revealed presence of interproximal intrabony two wall defect (IBD). Blood investigations were done to rule out any infectious disease. & patient consent form was obtained. Keeping all the findings in the mind, a thorough treatment plan was decided, including a series of therapeutic

procedures. Non-surgical periodontal therapy was performed by means of conventional scaling and root planing, using ultrasonic instruments and curettes.

Oral hygiene instructions were given to the patient & patient was motivated for performing effective oral hygiene measures. Patient was kept under observation. Follow-up was done every week to check for oral hygiene maintenance. After 6 weeks PPD & CAL were measured. Surgical periodontal therapy was done 2 weeks after the re-examination of the patient. Before planning for the periodontal surgical procedure, patient's platelet count (3.5 lac/mm³), Hemoglobin (11.5 gm/dl), Bleeding time (2.5 min) and Clotting time (4.5 min) were assessed and found to be within normal limits.

PRF Collection:

During surgery, intravenous blood (by venipuncturing of the antecubital vein) was collected in a 10-ml sterile tube without anticoagulant and immediately centrifuged in centrifugation machine at 3,000 revolutions per minute for 10 minutes. Blood centrifugation immediately after collection allows the composition of a structured fibrin clot in the middle of the tube, just between the red corpuscles at the bottom and acellular plasma (Platelet-poor plasma) at the top. PRF was easily separated from red corpuscles base (fig.2) [preserving a small red blood cell (RBC) layer] using a sterile tweezers and scissors, transferred onto a sterile dappen dish (fig. 3).

Surgical Procedure:

Following administration of local anaesthesia, buccal and lingual sulcular incisions were made and mucoperiosteal flaps were reflected. Care was taken to preserve as much inter-proximal soft tissue as possible. Meticulous defect debridement and root planing were carried out using hand instruments, and area specific curettes (fig.1).

No osseous recontouring was carried out. PRF was mixed with bone graft hydroxyapatite crystals to form a paste & was filled into the intrabony defect (fig.4). The mucoperiosteal flaps were repositioned and secured in place



Fig. 2: Harvesting the PRF membrane from the vacutainer tube after centrifugation



Fig. 3: Obtained PRF

using 3-0 non-absorbable black silk surgical suture. The simple interrupted sutures were placed. The surgical area was protected and covered with periodontal dressing.



Fig 4: Defect Filled With PRF

Postoperative Care:

The Suitable antibiotics and analgesics (amoxicillin 500 mg two times per day for 5 days and diclomol two times per day) were prescribed, along with chlorhexidine digluconate rinses (0.2%) twice daily for 2 weeks. Periodontal dressing and sutures were removed 1 weeks post-operatively. Surgical wounds were

gently cleansed with povidine iodine and patient was instructed for gentle brushing with a soft toothbrush.

Follow-up:

Follow up was done 1 week after the suture removal. On clinical examination satisfactory healing of gingival tissue & no adverse reactions were observed & patient was reinforced for proper oral hygiene measures. No subgingival instrumentation was attempted. After 1 month healing was found to be good (fig.5).



Fig 5: After 1 month

Discussion:

The goal of periodontal therapy includes not only the arrest of periodontal disease progression, but also the regeneration of structures lost due to disease. Recently, the use of growth factors in periodontal regeneration has shown promising results. Growth factors are a class of natural biologic mediators that regulate key cellular events in tissue regeneration including cell proliferation, chemotaxis, differentiation, and matrix synthesis via binding to specific cell surface receptors.³

These growth factors are involved in wound healing & are postulated as promoters of tissue regeneration.⁸ PRF could improve the periodontal osseous defect healing, as PRF can up regulate phosphorylated extracellular signal regulated protein kinase expression and suppress the osteoclastogenesis by promoting secretion of osteoprotegerin (OPG) in osteoblasts cultures.⁹ Recently, studies have demonstrated that the PRF membrane has a very significant slow-sustained release of key growth factors for at least 7days and up to 28 days, which means that the PRF membrane stimulates its environment

for a significant time during remodeling. The properties of this natural fibrin biomaterial thus offer great potential during wound healing. It has been clearly demonstrated that fibrin matrix leads directly to angiogenesis. Fibrin constitutes a natural support to immunity and reduces inflammatory process.¹⁰

Several studies performed to evaluate the efficacy of PRF in treating periodontal defects have demonstrated favourable results. Joseph *et al*, Thorat *et al* and Sharma *et al* reported higher gains in clinical attachment levels and radiographic bone fill in intrabony defects when utilizing PRF with open flap debridement. Some authors reported higher gains in vertical clinical attachment levels in mandibular grade II furcation defects treated with PRF and OFD. In the present case PRF was combined with hydroxyapatite bone graft material. Some authors observed that hydroxyapatite when added to PRF, increased its regenerative effects when treating three wall intrabony defects.¹¹

Conclusion: After surgical procedure the soft tissue healing was good. As the radiographic changes in bone defect treatment appears after 6 months to 1 year, patient is still under follow up. PRF by Choukroun's technique is a simple and inexpensive technique, and the systematic use of this biomaterial for periodontal regeneration seems a very promising option IN Tissue engineering. This case report demonstrates that the use of PRF, as the sole grafting material in periodontal osseous defects, is an effective modality in promoting a clinical resolution. The practical aspect of PRF use in periodontal osseous defects may be clinically relevant. Because PRF preparation utilizes the patient's own blood, the risk of human to human/animal disease transmission is virtually eliminated, making it a safe treatment modality.

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How to cite this article:. Roopa DA, Gupta I, Chauhan SS, Singh S. Platelet Rich Fibrin in Tissue Engineering: A boon to Periodontal Regeneration. J Dent Res Updates 2014 Dec;1(1):50-54

Sources of support: Nil

Conflict of Interest: None declared