REVIEW ARTICLE

Platelet rich fibrin: New treatment modality in Grade II furcation defects

Dhruv Mehta¹, Neeraj Deshpande², Deepak Dave², Bhavesh Modi², Ashit Bharwani²

¹Department of Periodontology, K. M. Shah Dental College and Hospital, Vadodara, Gujarat, India, ²Department of Periodontology, K. M. Shah Dental College and Hospital, Sumandeep Vidyapeeth, Piparia, Wagholi, Vadodara, Gujarat, 391760 India

Abstract

Platelet rich fibrin (PRF) is an autologous healing biomaterial, which incorporates a matrix of autologous fibrin, leukocytes, platelets and growth factors, harvested from a simple blood sample. Various growth factors present in PRF are well-known source of cytokines, usable for clinical applications. Grade II furcation lesion is essentially a cul-de-sac with a definite horizontal component and involvement of the interradicular bone without a through-and-through ability to probe. Furcation defects represent a formidable problem in the treatment of periodontal disease, which is related to the complex and irregular anatomy of furcation. The early Grade II furcation requires surgical management. Surgery permits access for root debridement, odontoplasty, osseous recontouring, and periodontal regeneration. Regeneration of the previously destroyed periodontal attachment tissues is biologically possible, and the regeneration has become the goal of therapy since the 1990s. Regenerative attempts such as bone grafts, guided tissue regeneration, application of growth factors, and enamel matrix derivatives are currently used for periodontal regeneration in the treatment of Grade II furcation involvements which under favorable conditions, can induce roughly 60-70% regeneration of the bone lesion's height or volume, with concomitant improvement in the clinical conditions. This review will help in understanding regenerative property of PRF in furcation defects.

Keywords
Grade II furcation, periodontal regeneration, platelet concentrate, plateletrich fibrin

Correspondence
Dr. Dhruv Mehta, Department of Periodontics, K M Shah Dental College & Hospital, Piparia, Vadodara - 391 760, Gujarat, India. Phone: +91-8866131076, E-mail:dhruvmehta511@gmail.com
Received 31 March 2016; Accepted 5 July 2016
doi: 10.15713/ins.jodm.7

Introduction

Periodontitis is a chronic destructive inflammatory disease of the supporting tissue of the teeth resulting in pocket formation, loss of attachment, and alveolar bone loss. A furcation involvement is bone resorption and attachment loss in the interradicular space that results from plaque associated periodontal diseases. According to Glickman (1953), Grade II furcation can affect one or more of the furcation of the same tooth. The furcation lesion is essentially a cul-de-sac with a definite horizontal component. The furcation defects represent a formidable problem in the treatment of periodontal disease, which is related to the complex and irregular anatomy of furcation. The early Grade II furcation requires surgical management. Surgery permits access for root debridement, odontoplasty, osseous recontouring, and periodontal regeneration. Regenerative attempts such as bone grafts, guided tissue regeneration, application of growth factors, and enamel matrix derivatives are currently used for periodontal regeneration in the treatment of Grade II furcation involvements.

In periodontal, regeneration platelet concentrate also plays an important role. The first generation incorporates the plateletrich plasma (PRP), while the second generation involves the plateletrich fibrin (PRF). The second generation platelet concentrate was introduced by Dohan et al. (France) in 2001[1] can be defined as an autologous healing biomaterial, incorporating in a matrix of autologous fibrin most leukocytes, platelets and growth factors harvested from a simple blood sample. The platelet growth factors are a well-known source of cytokines, usable for clinical applications.

PRF Second Generation Platelet Concentrate

The PRF protocol

A blood sample is taken without anticoagulant in 10 ml tube which is immediately centrifuged in a table centrifuge at 3000 rpm approximately 400 g for 10 min.[1] The absence of anticoagulant implies the activation in a few minutes of most
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1. Platelets of the blood sample in contact with the glass tube walls and the release of the coagulation cascades. Fibrinogen is initially concentrated in the high part of the tube before the circulating thrombin transforms it into fibrin. A fibrin clot is then obtained in the middle of the tube, just between the red corpuscles at the bottom and a cellular plasma at the top. Quick handling is the only way to obtain a clinically usable PRF clot.

2. **Structure of PRF**

   The PRF clot composed of two main parts observable with the naked eye: A fibrin yellow portion, constituting the main body, and a red portion located at the end of the clot (full of red blood cells [RBCs]). Between these two areas, a whitishe layer called the “buffy coat.” At the junction between the red and yellow parts of the PRF clot, the scanning electron microscopic examination showed leukocytes that clearly appeared as spherical structures with irregular surface. Platelet aggregates appeared very clearly along the fibrin strands. Beyond theuffy coat base, two distinguished different areas: The first area is composed of thick fibrin strands and a few scattered RBCs (probably from contamination during clot handling). The fibrin network appeared to be mature. The second area corresponded to the platelet veins. This area contained platelets and fibrin that formed large and dense clusters due to extensive aggregation and clotting. This aggregate formed a solid and thick mesh. Therefore, platelets seemed to be highly activated during the PRF-preparation protocol. At a low magnification, the PRF membrane surface showed the print of the gauze threads. Fibrin is physiologic glue, therefore, the compression of the fibrin clot to the platelet veins. This area contained platelets and fibrin that formed large and dense clusters due to extensive aggregation and clotting. This aggregate formed a solid and thick mesh. Therefore, platelets seemed to be highly activated during the PRF-preparation protocol. At a low magnification, the PRF membrane surface showed the print of the gauze threads. Fibrin is physiologic glue, therefore, the compression of the fibrin clot into a membrane provided a very compact matrix. In the fibrin, one end of the membranes is clearly organized in parallel strands that appeared very thick and dense [Figure 1].

3. **Properties of PRF**

   1. PRF composition indicates that this biomaterial consists of an intimate assembly of cytokines, glycanic chains, structural glycoproteins enmeshed within a slowly polymerized fibrin network. These biochemical components have well-known synergetic effects on healing processes.
   2. PRF is not only a platelet concentrate but also an immune node able to stimulate defense mechanisms. It is likely that the significant inflammatory regulation noted on surgical sites treated with PRF is the outcome of retro control effects from cytokines trapped in the fibrin network and released during the remodeling of this initial matrix.
   3. Role of fibrin matrix of PRF:
      - Natural guide of angiogenesis
      - Natural support to immunity
      - Fibrin matrix guides the coverage of injured tissues, affecting the metabolism of epithelial cells and fibroblasts.
   4. Cytokines present in the PRF are platelet-derived growth factor (PDGF), vascular endothelial growth factor, insulin growth factor-1, fibroblast growth factor, epidermal growth factor, and transforming growth factor (TGF).

4. **Clinical Implications of PRF**

   - In sinus lift procedures, socket preservation, filling of the cystic cavity
   - PRF membrane has been used for gingival recession coverage with coronally advanced or lateral pedicle flap for multiple and single recession, respectively. PRF acts both as healing and interpositional biomaterial
   - In the treatment of combined periodontic-endodontic lesion/furcation defect and as a scaffold for human periosteal cells in vitro, which finds application in the field of tissue engineering.

5. **Limitations of PRF Technology**

   1. Only a limited volume of PRF can be used. Because it is obtained from an autologous blood sample, the quantities produced are low
   2. PRF tissue banks are unfeasible. The fibrin matrix contains all the circulating immune cells and all the highly antigenic plasmatic molecules. That is, why PRF membranes are totally specific to the donor and can not constitute an allogeneic graft tissue.

6. **Evidence for the role of PRF in periodontal regeneration**

   According to Choukroun et al., PRF was initially used in implant surgery to enhance the healing properties of the bone. PRF can promote the healing of osseous defects by the following mechanisms. According to Chang et al., PRF promotes the expression of phosphorylated extracellular signal-regulated protein kinase (p-ERK) and stimulates the production of osteoprotegerin (OPG), which in turn causes proliferation of osteoblasts. Another study by Huang et al. reported that PRF stimulates the osteogenic differentiation of the human dental pulp cells by upregulating OPG and alkaline phosphatase expression. PRF also releases growth factors, such as PDGF and TGF, which promote periodontal regeneration. Tsai et al. in a study reported that PRF stimulates cell proliferation in a specific manner. PRF induces cell proliferation of osteoblasts, periodontal ligament cells and growth factors during a 3-day culture period and suppressed oral epithelial cell growth. These cell type-specific actions may be beneficial for periodontal regeneration. Diss et al. in a 1 year prospective study on osteotome sinus floor elevation using Choukroun’s PRF grafting material clearly demonstrated that fibrin matrix of PRF directly promotes angiogenesis. PRF when used as a membrane for guided tissue regeneration as a grafting material creates an improved space making effect which facilitates cell events that
are favorable for periodontal regeneration leading to mineralized tissue formation. PRF is having an inherent osteoconductive and/or osteoinductive property which is beneficial for regeneration of the bone. Sharma and Pradeep conducted the study for the treatment of mandibular degree II furcation defects with PRF and showed a significant improvement in pocket depth reduction, gain in clinical attachment level and bone fill in test group when compared to controls. The effect of PRF on human periodontal ligament fibroblasts and application in periodontal infrabony defects was studied by Chang and Zhao and reported that PRF was found to increase ERK phosphorylation and OPG in periodontal ligament fibroblasts and upregulation of alkaline phosphatase activity. Furthermore, infrabony defects exhibited pocket reduction and clinical attachment gain after 6 months with bone fill in defects. The systematic appraisal of current evidence of PRF in inflammatory bowel diseases has confirmed the benefits and advantages of using PRF alone compared to open flap debridement.

Various treatment of Grade II furcation

Molars with furcation involved, caused by periodontitis, have a higher rate of periodontal breakdown and respond less favorably to periodontal therapy than molars without furcation involvement or single-rooted teeth. This can be explained by an anatomy that impedes accessibility for individual oral hygiene in the molar region and professional root debridement.

Multiple approaches have been used to resolve furcation defect including autografts, demineralized freeze-dried bone allografts, bovine-derived xenografts, barrier membranes, and combinations of membranes and bone grafts. Although these regenerative materials are still used today, the introduction of bio-mimetic agents such as enamel matrix derivatives, PRP, PDGF, and bone morphogenetic proteins has given new promise for better outcomes in furcation treatment.

Method to use PRF in Grade II furcation

During presurgical therapy, each patient has to be given careful instructions on proper oral hygiene measures. A full-mouth supragingival and subgingival scaling and root planing procedure have to be performed under local anesthesia. 6-8 weeks after phase I therapy, a periodontal evaluation has to be performed to check the suitable site for surgery. The clinical parameters [Figures 2-4] and radiographic parameters [Figure 5] have to be recorded before the surgical procedure and have to be evaluated at baseline and 9 months postoperatively. For the measurement of bone defect, distance from the furcation fornix to the base of the defect can be considered. Individual bite blocks and parallel angle technique can be used to obtain radiographs.

Before surgical procedure, intraoral antiseptics has to be performed with 0.12% chlorhexidine digluconate rinse and an iodine solution can be used to carry out extraoral antiseptics. After administration of local anesthesia, buccal and lingual sulcular incisions have to be made and mucoperiosteal flaps have to be reflected [Figure 6]. Care has to be taken to preserve as much interproximal soft tissue as possible. Meticulous defect debridement and root planing have to be carried out using ultrasonic instruments and area-specific curettes. Autologous PRF of the required size can be filled into the furcation defect, and two other parts can be used as a membrane to cover furcation [Figure 7]. The mucoperiosteal flaps have to be repositioned and secured in place using surgical suture [Figure 8]. The surgical

Figure 1: (a) Prepared platelet-rich fibrin (PRF) after centrifugation. (b) PRF plug

Figure 2: Pre-operative

Figure 3: Vertical probing

Figure 4: Prepared platelet-rich fibrin (PRF) after centrifugation

Figure 5: PRF plug

Figure 6: Mucoperiosteal flaps

Figure 7: Membrane to cover furcation

Figure 8: Surgical suture
area can be protected and covered with a periodontal dressing [Figure 9].

Postoperatively suitable antibiotics and analgesics (500 mg amoxicillin, 4 times per day for 5 days, and 800 mg ibuprofen, 3 times per day) have to be prescribed, along with chlorhexidine digluconate rinses (0.12%) twice daily for 2 weeks.
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Review of Literature

Sharma and Pradeep (2011)\(^{[5]}\) stated that PRF has been shown to be an effective modality of therapy in the regenerative treatment of degree II mandibular furcation. Within the limitations of this study, there was a greater reduction in PD, and more RVCAL gain with significant bone defect filled with PRF treatment in degree II furcation defects.

Thorat et al. (2011)\(^{[11]}\) investigated the clinical and radiological effectiveness of autologous PRF in the treatment of intrabony defects of chronic periodontitis patients and reported a greater reduction in pocket depth, more gain in clinical attachment level and greater intrabony defect fill at sites treated with PRF than those treated with open flap debridement alone.

Chang and Zhao (2011).\(^{[11]}\) The effect of PRF on human periodontal ligament fibroblasts and application in periodontal intrabony defects was studied by Chang et al. and reported that PRF was found to increase ERK phosphorylation and OPG in periodontal ligament fibroblasts and upregulation of alkaline phosphatase activity. Furthermore, intrabony defects exhibited pocket reduction and clinical attachment gain after 6 months with bone fill in defects.

Sánchez et al. (2003)\(^{[5]}\) stated that the PRF when used as a membrane for guided tissue regeneration as a grafting material creates an improved space making effect which facilitates cell events that are favorable for periodontal regeneration leading to mineralized tissue formation. PRF is having an inherent osteoconductive and/or osteoinductive property which is beneficial for regeneration of the bone.

Conclusion

Many of the study PRF has shown to have a greater reduction in PD, and more relative vertical clinical attachment level and relative horizontal clinical attachment gain with significant bone defect fill. PRF has been shown to be an effective modality of therapy in the regenerative treatment of degree II mandibular furcation also.

References


How to cite this article: Mehta D, Deshpande N, Dave D, Modi B, Bharwani A. Platelet rich fibrin: New treatment modality in Grade II furcation defects. J Oral Dis Market 2017;1:15-19.