Research Article
J Nepal Soc Perio Oral Implantol. 2021;5(9):66-71.

Submitted: Aug 01, 2021
Accepted: Nov 25, 2021
Published: Dec 03, 2021

Treatment of Periodontal Intrabony Defect by Open Flap Debridement With or Without Platelet-rich Fibrin

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ABSTRACT

Introduction: Periodontitis is a chronic bacterial infection leading to destruction of supporting structures of teeth which may ultimately result in a variety of periodontal intraosseous defects. Goal of periodontal therapy is to regenerate lost periodontal tissue for optimal function and aesthetics. Platelet-rich concentrates are widely being used as regenerative biomaterials as they stimulate and accelerate healing by continuous release of growth factors.

Objective: To evaluate the clinical efficacy of autologous platelet-rich fibrin (PRF) along with open flap debridement (OFD) or OFD alone in the treatment of periodontal intrabony defects.

Methods: This non-randomised clinical study was conducted from June 2015 - May 2016 at Bir Hospital. Patients diagnosed with Chronic Periodontitis were selected using convenience sampling and divided into two groups: Control (OFD only) and Test (OFD+PRF) with 16 in each group. All subjects underwent periodontal examination by a single examiner. The periodontal parameters were assessed and compared between control and test groups at baseline and six months. Data analysis was done using SPSS 22 software program.

Results: The mean PI scores, GI scores, PD reduction, and CAL gain scores improved in both control and test groups at six months compared to baseline. The comparison of mean difference for every parameter measured showed statistically significant difference ($p < 0.05$) at baseline and after six months for both the groups. However, the results were superior in the test group.

Conclusions: The addition of autologous PRF to OFD stimulated a significant improvement in the clinical parameters as compared to OFD alone at six months.

Keywords: Periodontal intrabony defects; periodontal regeneration; platelet-rich fibrin.

INTRODUCTION

Periodontitis is an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or a group of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession, or both. These host responses can result in a variety of intraosseous defects of various architectures.[1]

Regeneration of lost tissues and the maintenance of natural dentition in health and comfortable function is the goal of periodontal therapy. Periodontal regeneration involves the formation of alveolar bone, cementum, and new functional periodontal ligament.[2] Platelet-rich fibrin (PRF) is a second-generation platelet concentrates. From its time of advent, PRF has been widely used in various treatment procedures.[3]

Since, the authors could find no published studies of this kind conducted in the Nepalese population, this study was planned to compare the combined effect of PRF and open flap debridement (OFD) or OFD alone in the treatment of periodontal intrabony defects.

METHODS

A hospital-based prospective longitudinal non-randomised study was carried out in the Periodontology and Oral Implantology (Perio) Unit of the Department of Dental Surgery, National Academy of Medical Sciences (NAMS), Bir Hospital, Mahabouddha, Kathmandu, Nepal from June 2015- May 2016.

Ethical Approval was taken from Institutional Review Board (IRB)-NAMS. The informed written consents were taken from all the participants. Confidentiality was maintained to the utmost. The inclusion criteria included patients

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Citation
Adhikari B, Pradhan S, Shrestha S. Treatment of Periodontal Intrabony Defect by Open Flap Debridement With or Without Platelet-rich Fibrin. J Nepal Soc Perio Oral Implantol. 2021 Jul-Dec;5(10):66-71

aged 25-44 years undergoing periodontal therapy at the study site, with Interproximal probing depth (PD) ≥5 mm following phase I therapy (scaling and root planing, SRP) in vital, all asymptomatic maxillary and mandibular posterior teeth with two- or three-walled bony defects. Whereas the exclusion criteria comprised of patients with present or past systemic illness that are known to affect the outcomes of periodontal therapy, immunocompromised patients, pregnant and lactating females, current and past smokers, patients taking medications that may interfere with wound healing, those allergic to medications, those having unacceptable oral hygiene after the re-evaluation of phase I therapy.

The sample size was calculated by using data from the study of Thorat et al.[4] The formula used was:

n=.

Minimum of 16 subjects in each group: 16 test and 16 controls in altogether 32 patients were enrolled in the studyby nonprobability convenience samplingmethod. The participants were assigned into control and test groups. The test group consisted of patients undergoing OFD for the intrabony periodontal defects followed by the placement of PRF, whereas the control group consisted of patients undergoing OFD only for the intrabony periodontal defects. All subjects underwent periodontal examination by a single examiner and the required information was assessed by means of a Proforma developed for the study.

The clinical parameters recorded included site-specific Plaque index (PI), Gingival Index (GI), Probing Pocket depth (PPD), Clinical attachment level (CAL). All parameters were evaluated at the baseline, after phase I therapy and six months post-operatively. The PPD measurement was done using a pre-fabricated acrylic stent with grooves to ensure accurate placement and to minimise error caused due to variation in angulation of the periodontal probe. Intrabony defect was evaluated at baseline and after six months. Parallel angle technique was used to obtain standardised radiographs. A review of all the radiographs was performed and the radiographic bone gain was used as the positive reinforcement for bone fill.

For non-surgical therapy, at the initial visit, each patient underwent a full-mouth supra and subgingival SRP and oral hygiene instructions were given. Occlusal interferences on both sides were removed if present. Six-week post SRP, a periodontal evaluation was done to confirm the desired sites for the study. The selected sites were divided randomly into test and control groups. Procedure was performed followed by suturing and periodontal dressing for 7–14 days.

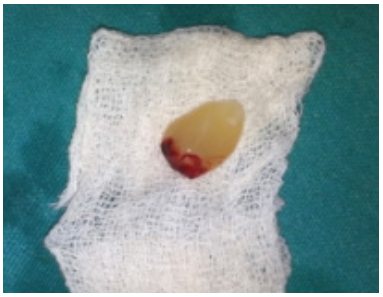
For surgical procedure, lignocaine 1:2,00,000 adrenaline local anaesthesia was administered, buccal and lingual sulcular incisions were made, and mucoperiosteal flaps were reflected. Maximum interproximal soft tissue was preserved to have primary closure. Root planing followed by debridement of the defect were carried out using area-specific curettes (Gracey curettes, Hu-Friedy). No osseous recontouring was done. In the control group the flaps were sutured in their position without placing any regenerative material into the defect but in the test group, PRF of the required size was placed into the defects.

For the preparation of PRF, immediately before surgery, intravenous blood alone (without anticoagulant) was collected by venipuncturing of the antecubital vein in a sterile 10-ml tube and centrifugation was carried out immediately at 3000 rpm for 13 min (Tabletop centrifuge – REMI, R-8C, Mumbai, India). Because of differential densities, it results in the separation of three basic fractions. Topmost layer consisting of acellular platelet poor plasma, middle layer consists of PRF and the bottom layer has the Red Blood Cells (RBCs). A total of 2–3 ml of the top layer was pipetted out with the sterile dropper; the middle layer (PRF) was removed and was used in the defect (Figure 1).

Repositioning of the mucoperiosteal flap was done and the flap was secured using a 3-0 non-absorbable silk suture with interrupted sutures. A periodontal dressing was placed in protection over the surgical site. Medications (Amoxicillin 500 mg, thrice a day for five days; and Ibuprofen 400 mg three times a day as if required) were prescribed. Patients were advised to rinse with chlorhexidine gluconate mouthrinse (0.2%) twice daily for a period of 15 days.

At one week post-operatively, periodontal dressing and

Figure 1: Preparation of platelet-richrich fibrin.



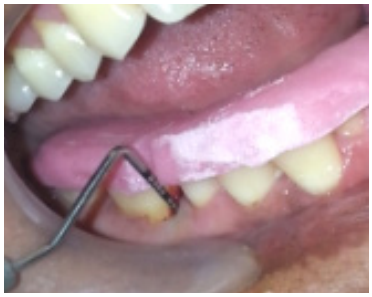


Figure 2: Pocket depth measurement with stent in place.

sutures were removed. Each patient was re-examined weekly up to one month after surgery, no subgingival instrumentation was attempted at any of these appointments. They were then followed at three and six months, and oral hygiene instructions were reinforced at each recall visit. Soft and hard tissue evaluation was performed six months after surgery. Soft tissue measurements were repeated with previously used acrylic stents (Figure 2). For hard tissue reevaluation, second Intraoral Periapical Radiographs (IOPAR) of the same study site was carried out.

Data were analysed using IBM Statistical Package for Social Sciences (SPSS) Statistics for Windows, version 22 (IBM Corp., Armonk, N.Y., USA) software. The change in all periodontal parameters at different time intervals for both test and control groups were compared with help of paired t-test. The comparison of OFD and OFD plus PRF in improvement of periodontal study parameters was done with the help of independent t-test. The level of significance was set at 5%. P value was calculated under the predetermined level of significance (0.05) and Confidence Interval (CI) of 95% was constructed.

RESULTS

Out of total 32 patients enrolled in the study, male:female ratio was 17:15 encompassing 53% and 47% male and female respectively. Mean age of the study participants was 37 years. Three-fourths (75%) of the patients were between 35 to 44 years of age reflecting the occurrence of the disease in the older age group.

The mean Plaque Index and Gingival Index scores for both control and test groups show statistically significant results both at baseline and six months follow-up (Figure 3 and 4 respectively).

There were statistically significant mean pocket depth scores (P <0.001) at the test and control group both at baseline and six months follow-up, however the test sites showed a greater reduction in mean pocket depth as compared to the control sites (Table 1).

There were statistically significant mean clinical attachment loss scores (P <0.001) at the test and control groups both at baseline and six months follow-up, however mean gain in clinical attachment level is higher in the test group as compared to the control (Table 2).

The mean pocket depth reduction in the control group was less as compared to the test group. Similarly, the mean clinical attachment level gain in the test group was higher as compared to the control group (Figure 5).

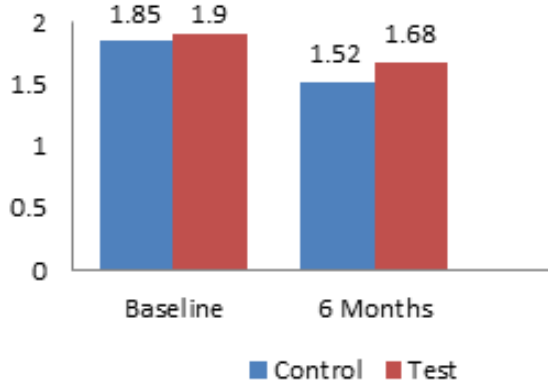


Figure 3: Mean plaque index scores for both control and test groups (n=32).

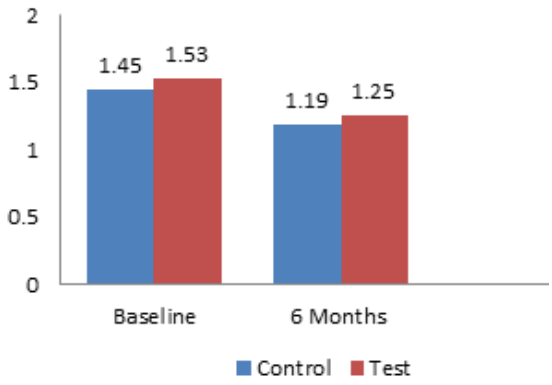


Figure 4: Mean gingival index scores for both control and test groups (n=32).

Table 1: Mean pocket depth scores (in millimeters) for both control and test groups.

Pocket Depth	Group	Mean ± SD	P value
Baseline	Control (n=16)	7.187 ± 1.797	<0.001
Six months		3.687 ± 0.873	<0.001
Baseline	Test (n=16)	8.312 ± 1.778	<0.001
Six months		3.625 ± 1.204	<0.001

Table 2: Mean clinical attachment loss scores (in millimeters) for both control and test groups.

Clinical Attachment Loss	Group	Mean ± SD	P value
Baseline	Control (n=16)	8.187 ± 2.228	<0.001
Six months		6.062 ± 1.526	<0.001
Baseline	Test (n=16)	9.312 ± 1.740	<0.001
Six months		5.625 ± 1.500	<0.001

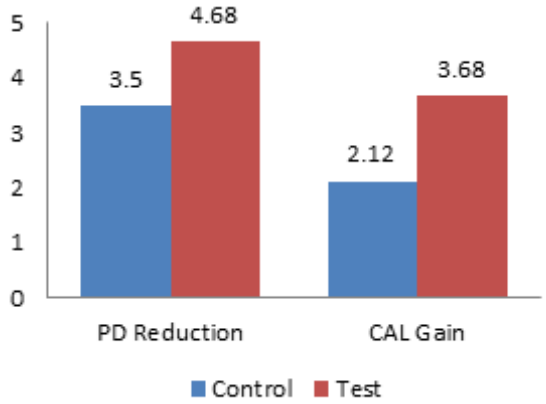


Figure 5: Mean pocket depth reduction and clinical attachment level gain (in millimeters) in control and test groups after six months (n=32).

DISCUSSION

Reduction in PD and gain in CAL are the major clinical outcomes measured to determine the success of any periodontal treatment. In the present study, a significant reduction in PD and CAL gain were found in both groups when compared with baseline and six months. The mean probing pocket depth values for control and test groups at baseline were 7.18 ± 1.79 mm and 8.31 ± 1.77 mm respectively. In follow-up visit at six months, the mean probing pocket depth reduced to 3.68 ± 0.87 mm and 3.62 ± 1.20 mm for control and test groups respectively. There was a statistically significant (P <0.001) pocket depth reduction in both control and test groups after six months of the procedure; however, the test sites showed a greater reduction in mean pocket depth as compared to the control sites. Thus, OFD with PRF in the treatment of periodontal intrabony defects produce significant reduction in pocket depth as compared to OFD alone. These findings are consistent with the findings observed by Thorat et al. and Ajwani et al.[4,5]

The mean clinical attachment loss was 8.18 ± 2.22 mm and 9.31 ± 1.70 mm for control and test groups respectively at baseline which decreased to 6.06 ± 1.52 mm and 5.62 ± 1.50 mm respectively in six months follow-up visits. The gain in clinical attachment level is also statistically significant (P <0.001) with mean gain in clinical attachment level being higher in the test group as compared to the control groups. Thus, OFD with PRF in the treatment of periodontal intrabony defects produce significant gain in clinical attachment levels as compared to OFD alone. These findings are also consistent with the findings observed by Thorat et al. and Ajwani et al.[4,5]

It has been reported that the CAL gain after conventional or regenerative periodontal treatment was dependent on the initial PD; that is, deeper the initial PD, the greater the PD reduction and the CAL gain.[6] This is significant considering that the baseline levels of probing pocket depth and clinical attachment levels in the present study were comparable to studies that used Platelet-rich plasma (PRP).[7,8] Markou et al. reported a mean improvement in PD of 3.92 ± 1.1 mm, CAL of 3.08 ± 0.95 mm1year after periodontal surgery.[8] Ilgenli et al. reported a mean improvement in PD of 2.1 ± 0.5 mm, CAL of 1.5 ± 0.7 mm 18 months after surgery.[7] In contrast, the present study reports a mean change in PD of 4.68 ± 1.40 mm, CAL of 3.68 ± 0.79 mm six months after surgery in the test group.

The treatment protocol emphasised the principles of careful soft tissue handling, wound stability, and infection control. In assessing the success of these treatment methods, complete closure of the defect is desirable. Therapeutic results can be measured by PD and CAL, bone regeneration, and evidence of histologic periodontal regeneration. Although histological evaluation is the most accurate method of evaluation, surgical closure of the defect and improvements in PD and CAL serve as suitable and practical outcome measures.[9]

The uneventful healing in the patients is in agreement with several studies.[10,11] Thus it supports the excellent properties of autologous PRF to enhance periodontal wound healing. Plaque, infection and smoking are the important factors that have been shown to significantly influence the outcomes of regenerative periodontal surgery.[12,13] Because the present study excludes smokers and only includes patients who were able to maintain acceptable oral hygiene, it may be assumed that the careful patient selection was also responsible for the positive outcomes obtained in both groups.

Only three and two-wall intrabony defects were included because the number of remaining bony walls was found to be correlated positively with regeneration potential in

grafting procedures.[14] Space maintenance is provided by the defect walls to minimise a membrane collapse and/or to provide the protection and retention of grafts.[15]

In the present study platelet-rich fibrin was used rather than the more extensively studied platelet-rich plasma as it offers several advantages like ease of preparation, no biochemical handling of blood or use of any gelling agent like calcium chloride and no risks associated with the use of bovine thrombin. As it is a completely autologous material it is highly cost-effective.[16]

The success of this technique entirely depends on the speed of blood collection and immediate centrifugation.[3] In order to obtain a clinically usable platelet-rich fibrin clot, in the present study a chair side centrifuge was used and it was ensured that the freshly drawn blood was immediately transferred to the centrifuge without any delay to prevent dehydration.

Platelet-rich fibrin alone was used in the test group and not as an adjunct to other regenerative approaches like bone replacement grafts or guided tissue regeneration in the present study. There have been conflicting reports regarding the use of platelet concentrates along with bone replacement grafts. Ilgenli et al. claim a superior clinical effectiveness for the combination, however Markou et al. claim no added advantages for the combination.[7,8] The recent metaanalysis by Fabbro et al. states that combination of platelet concentrate with guided tissue regeneration (GTR) masks the true effectiveness of platelet concentrates.[17]

The improvement in clinical parameters and better bone fill in the test group are suggestive of the effectiveness of platelet-rich fibrin in regenerative periodontal therapy. These results may be attributed to the contents of the PRF clot namely fibrin, platelets, leukocytes, growth factors, and cytokines. The fibrin matrix supporting the PRF clot constitutes the determining element responsible for the therapeutic potential of platelet-rich fibrin.[18] The fibrin matrix plays important role in four highly specific aspects of healing: angiogenesis, immune control, harnessing the circulating stem cells, and wound protection by epithelial cover.[18] The angiogenesis property of fibrin matrix is explained by the 3-dimensional structure of the fibrin gel and by the simultaneous action of cytokines trapped in the meshes. During hemostasis and healing, the fibrin clot traps the circulating stem cells and allows the vascular and tissue restoration.[19]

In the present study we observed a significant radiographic bone fill in the test group. Direct interactions between fibrin and osseous cells during healing are insufficiently documented.[18] On the other hand, numerous animal studies deal with the fibrin effect on osseous healing. The results are contradictory; osseous healing is either improved or remains unchanged.[20] Growth factors contained in the platelet-rich fibrin clot could have contributed to the radiographic bone fill observed in the present study. Platelet-derived growth factor (PDGF) has been shown to have a significant regenerative impact on periodontal ligament cells and osteoblasts.[21] It has also been reported that PRF induces a significant and continuous stimulation of proliferation in all cell types of the periodontium except epithelial cells. Platelet-rich fibrin stimulates human bone mesenchymal cell proliferation and differentiation.[22]

Results in the test group of the current study were compared to those studies using platelet-rich plasma alone as periodontal regenerative approach.[7,8] Current results are in accordance with these studies in terms of changes in clinical attachment levels and probing depths. However, the magnitude of change in CAL and PD at reevaluation is much higher in the present study as compared to studies using PRP alone.

The reason for the improved results with platelet-rich fibrin may be attributed to the difference in structure between PRP and PRF as explained by Dohan et al. and their growth factor content.[3] Platelet-rich plasma uses a bovine thrombin and calcium chloride resulting in sudden fibrin polymerization. Platelet-rich fibrin has the characteristic of polymerising naturally and slowly under physiologic concentrations of autologous thrombin. This difference in polymerisation results in two different biochemical architectures for the resulting product: Condensed tetra molecular or bilateral junctions in PRP and connected trimolecular or equilateral junctions in PRF.

There were limitations in this study like the sample size was very small and the clinical parameters were assessed only for a short duration of time. Other limitation was the inability to measure the amount of bone gain due to lack of proper equipment to standardise the IOPAR and also the unavailability of grids to calibrate the radiographs, so, though IOPAR was taken at baseline and after six months of follow up, the radiographic bone fill was taken only as a positive reinforcement for the effectiveness of our treatment and the procedure we used. Similarly, subjective bias, though unintentional could have impacted the measurement of periodontal parameters like PD, CAL, PI, and GI.

CONCLUSION

The addition of autologous PRF to OFD stimulated a significant improvement in the clinical parameters compared to OFD alone at six months. However, long-term,

multicentre histological studies are warranted to determine the precise effects of PRF on bone regeneration. The authors recommend that the use of PRF can be further promoted in the clinical practice.

ACKNOWLEDGEMENTS

The authors acknowledge the support received from Assistant Prof. Dr. Ranjita Shrestha Gorkhali, Dr. Sushil KC, and Dr. Sujaya Gupta.

Conflict of Interest: None.

REFERENCES

1. Zander HA, Polson AM, Heijl LC. Goals of periodontal therapy. J Periodontol. 1976;47(5):261-6.
2. Wang HL, Pappert TD, Castelli WA, Chiego DJ, Jr., Shyr Y, Smith BA. The effect of platelet-derived growth factor on the cellular response of the periodontium: an autoradiographic study on dogs. J Periodontol. 1994;65(5):429-36.
3. Dohan DM, Choukroun J, Diss A, Dohan SL, Dohan AJ, Mouhyi J, et al. Platelet-rich fibrin (PRF): a second-generation platelet concentrate. Part I: technological concepts and evolution. Oral Surg, Oral Med, Oral Pathol, Oral Radiol, Endod. 2006;101(3):e37-44.
4. Thorat M, Pradeep AR, Pallavi B. Clinical effect of autologous platelet-rich fibrin in the treatment of intra-bony defects: a controlled clinical trial. J Clin Periodontol. 2011;38(10):925-32.
5. Ajwani H, Shetty S, Gopalakrishnan D, Kathariya R, Kulloli A, Dolas RS, et al. Comparative evaluation of platelet-rich fibrin biomaterial and open flap debridement in the treatment of two and three wall intrabony defects. J Int Oral Health. 2015;7(4):32-7.
6. 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.
7. Ilgenli T, Dundar N, Kal BI. Demineralized freeze-dried bone allograft and platelet-rich plasma vs platelet-rich plasma alone in infrabony defects: a clinical and radiographic evaluation. Clin Oral Investig. 2007;11(1):51-9.
8. Markou N, Pepelassi E, Vavouraki H, Stamatakis HC, Nikolopoulos G, Vrotsos I, et al. Treatment of periodontal endosseous defects with platelet-rich plasma alone or in combination with demineralized freeze-dried bone allograft: a comparative clinical trial. J Periodontol. 2009;80(12):1911-9.
9. Machtei EE. Outcome variables for the study of periodontal regeneration. Ann Periodontol. 1997;2(1):229-39.
10. Pradeep AR, Shetty SK, Garg G, Pai S. Clinical effectiveness of autologous platelet-rich plasma and Peptide-enhanced bone graft in the treatment of intrabony defects. J Periodontol. 2009;80(1):62-71.
11. Pradeep AR, Rao NS, Agarwal E, Bajaj P, Kumari M, Naik SB. Comparative evaluation of autologous platelet-rich fibrin and platelet-rich plasma in the treatment of 3-wall intrabony defects in chronic periodontitis: a randomized controlled clinical trial. J Periodontol. 2012;83(12):1499-507.
12. Trombelli L, Kim CK, Zimmerman GJ, Wikesjo UM. Retrospective analysis of factors related to clinical outcome of guided tissue regeneration procedures in intrabony defects. J Clin Periodontol. 1997;24(6):366-71.
13. Tonetti MS, Prato GP, Cortellini P. Factors affecting the healing response of intrabony defects following guided tissue regeneration and access flap surgery. J Clin Periodontol. 1996;23(6):548-56.
14. Schallhorn RG, Hiatt WH, Boyce W. Iliac transplants in periodontal therapy. J Periodontol. 1970;41(10):566-80.
15. Blumenthal NM, Alves ME, Al-Huwais S, Hofbauer AM, Koperski RD. Defect-determined regenerative options for treating periodontal intrabony defects in baboons. J Periodontol. 2003;74(1):10-24.
16. Marx RE. Platelet-rich plasma: evidence to support its use. J Oral Maxillofac Surg. 2004;62(4):489-96.
17. Del Fabbro M, Bortolin M, Taschieri S, Weinstein R. Is platelet concentrate advantageous for the surgical treatment of periodontal diseases? A systematic review and meta-analysis. J Periodontol. 2011;82(8):1100-11.
18. Choukroun J, Diss A, Simonpieri A, Girard MO, Schoeffler C, Dohan SL, et al. Platelet-rich fibrin (PRF): a second-generation platelet concentrate. Part IV: clinical effects on tissue healing. Oral Surg, Oral Med, Oral Pathol, Oral Radiol, Endod. 2006;101(3):56-60.
19. Feng X, Clark RA, Galanakis D, Tonnesen MG. Fibrin and collagen differentially regulate human dermal microvascular endothelial cell integrins: stabilization of alphav/beta3 mRNA by fibrin1. J Investig Dermatol. 1999;113(6):913-9.
20. Soffer E, Ouhayoun JP, Anagnostou F. Fibrin sealants and platelet preparations in bone and periodontal healing. Oral Surg, Oral Med, Oral Pathol, Oral Radiol, Endod. 2003;95(5):521-8.
21. Ramseier CA, Abramson ZR, Jin Q, Giannobile WV. Gene therapeutics for periodontal regenerative medicine. Dent Clin North Am. 2006;50(2):245-63, ix.
22. Dohan Ehrenfest DM, Doglioli P, de Peppo GM, Del Corso M, Charrier JB. Choukroun's platelet-rich fibrin (PRF) stimulates in vitro proliferation and differentiation of human oral bone mesenchymal stem cell in a dose-dependent way. Arch Oral Biol. 2010;55(3):185-94.