AJDS LAZHAR Al-Azhar Journal of Dental Science
Vol. 27- No. 2- 225:231- April 2024
Print ISSN 1110-6751 | online ISSN 2682 - 3314
https://ajdsm.journals.ekb.eg
Oral Biology, Medicine & Surgical Sciences Issue *(Oral Biology, Oral Pathology, Oral Periodontology, Medicine, Oral & Maxillofacial Surgery)*

ROLE OF PLATELET-RICH FIBRIN (PRF) IN NON-SURGICAL MANAGEMENT OF PERIODONTAL POCKETS

Khalid M. Alghannam [1*], Mostafa Hosny [2], Mahmoud T. Eldestawy [3]

ABSTRACT

Objective: The present study was performed to evaluate clinically the effect of iPRF in non-surgical treatment of periodontal pockets. **Subjects and method:** Fourteen patients with moderate periodontitis received scaling, root planning (SRP) and oral hygiene regimen. Treatment was received based on split mouth technique, in which each patient were have one side that was treated with SRP only (control group) and the other side was treated with SRP and PRF application (iPRF group). **Results:** Both therapeutic modalities could result in statistically significant improvement of all explored clinical parameters. Control and iPRF groups showed a statistically significant decrease in mean PD and CAL measurements at 1 months followed by an increase at 3 months which was accompanied by an increase in PI and GI then a statistically non-significant change at PD and CAL after 6 months despite the decrease in PI and GI. **Conclusions:** Injection of iPRF with SRP may have a positive effect, comparable clinically to SRP alone, in minimizing PD and CAL of periodontal pockets.

KEY WORDS: Platelet rich fibrin, iPRF, and Periodontal regeneration.

INTRODUCTION

Periodontal diseases are inflammatory disorders elicited by a complex of bacterial species that interact with host tissues and cells causing the release of inflammatory cytokines, chemokines, and mediators. Some of these mediators may aid in the destruction of the tissues surrounding the teeth, including the tooth supporting tissues, periodontal ligament, and alveolar bone. The trigger for the initiation of disease is the presence of complex microbial biofilms that colonize the sulcular regions between the tooth surface and the gingival margin through specific adherence interactions and accumulation due to architectural changes in the sulcus (i.e., attachment loss and pocket formation)[(1)].

The main initiating factor of periodontal disease is bacterial plaque, which induces progressive tissue damage. In the presence of susceptibility to periodontal disease due to systemic conditions, the role of bacterial plaque is debated. It has been considered that periodontal disease cannot be induced without the presence of plaque and suggest that a systemic predisposition simply accelerates the destruction caused by bacterial agents[(2)]. Acquired and genetic factors influence progression and clinical characteristics of the plaque-induced

1. Masters Candidate, Dentist at Ministry of Health.
2. Associate Professor ,department of Oral Medicine, Periodontology, Oral Diagnosis and Oral Radiology, Faculty of Dental Medicine Al-Azhar University
3. Professor, Head of Department of Oral Medicine, Periodontology, Oral Diagnosis and Oral Radiology, Faculty of Dental Medicine Al-Azhar University

• ***Corresponding author*:** dr.khalidelghannam@gmail.com

DOI: *10.21608/AJDSM.2022.121715.1310*

inflammatory periodontal diseases and can modify susceptibility to infection [3].

Periodontal regeneration is a multifactorial process that involves a coordinated sequence of physiologic processes including as cell adhesion, migration, proliferation, and differentiation. Furthermore, periodontal wound repair necessitates interactions among epithelial cells, gingival fibroblasts, periodontal ligament cells, and osteoblasts. Through molecular signals mediated mostly by cytokines and growth factors, the disruption of vasculature during wound healing causes fibrin formation, platelet aggregation, and the release of numerous growth factors into tissues from platelets. The presence of growth factors and cytokines in platelets has been shown to play a function in inflammation and wound healing. Platelets also release fibrin, fibronectin, and vitronectin, which serve as a connective tissue matrix and cell adhesion molecules, allowing for more effective cell movement [4].

The hemostasis phase starts immediately after an injury and overlaps with the inflammatory phase of healing. The inflammatory phase should only last a few days, in which the complement system is activated. Complements are chemotactic factors that attract other leukocytes within 24-48 hours after the injury. One of these cells is the neutrophil that do phagocytosis. Monocytes and lymphocytes enter which become macrophages. These cells remove remaining dead and damaged tissues, also they make more TGF-beta, PDGF, Tumor necrosis factor-alpha (TNF-a) and also cytokines like Il-6, Il-1. All of these growth factors and cytokines from macrophages act to cause the fibroblasts to grow. Then conversion happen from the inflammatory phase to the proliferative phase [5,14].

The proliferation phase is the actual construction phase where the connective tissue is made whole again. The fibrin and fibrin matrix form a scaffold for the granulation tissue that will be converted to the actual tissue needed like ligaments and bone. The fibrin is replaced by collagen on day 2 or 3 after the injury or surgery. More growth factors are released for this process to happen like: vascular endothelial growth factor-A (VEGF-A), fibroblast growth factor 2 (FGF-2), PDGF, TGF-beta. These growth factors also stimulate fibroblasts to make more growth factors that start the process of tissue regeneration [6,14].

Wound remodeling is the last phase of healing and its success relies heavily on how well the other three phases of healing went. It usually starts at week 2 or 3. As the I-PRF steadily released for almost 10 -14 day[7], so it has an enhancing role in the beginning of this phase too. In this phase the tissue matures, weaker collagen type III is replaced by stronger collagen type I. The organization process is regulated by the fibroblasts that secrete proteolytic enzymes that degrades the collagen matrix of the wound bed and allows for the realignment of that collagen into organized networks. The key to real regeneration is the remodeling and re-organization of the extracellular matrix of the damaged tissue[8,14-16].

Using platelets as therapeutic tools is a new approach to improve tissue repair particularly in periodontal wound healing. Choukroun et al[9] have described platelet-rich fibrin (PRF) as a second-generation platelet concentrate which contains platelets and growth factors in the form of fibrin membranes prepared from the patient's own blood without any anticoagulant or other artificial biochemical modifications. Injectable platelet-rich fibrin (I-PRF) is a second generation, fully autologous, blood-derived biomaterial having three-dimensional fibrin meshwork, like that of a PRF clot, while retaining the fluid nature, just like platelet-rich plasma (PRP). Along with platelets and its growth factors, I-PRF predominantly has collagen type-1, lymphocytes along with its growth factors. Preparation of I-PRF is simple and requires minimal instrumentation and materials, making it a cost-effective product [10].

The aim of the present study was to clarify the role of I-PRF in non-surgical treatment of periodontitis.

SUBJECTS AND METHOD

This study had started on 14 patients within the age range between 30 to 45 years old. The selected patients will have moderate periodontitis with periodontal pocket depth ranging from 4 to 6 mm in depth. They were selected from the Outpatient Clinics of the Department of Oral Medicine, Periodontology, Diagnosis and Oral Radiology, Faculty of Dental Medicine (Boys, Cairo) Al-Azhar University code no. 122018. All procedures were told to the patients and signed written consent were requested form them.

Inclusion criteria: Patients having moderate periodontitis according to criteria of Armitage [11], also should be free from any systemic diseases.

Exclusion criteria: Medically compromised patients, patients have class II cavities in the tooth that the pocket is related to and smoking patients.

Patients grouping: Treatment was received based on split mouth technique, in which each patient were have one side that was treated with SRP only (control group) and the other side was treated with SRP and PRF application (I-PRF group).

Patient preparation: All patients were submitted to supragingival and subgingival scaling and root planning (SRP) at the start visit of treatment.

I- PRF preparation:

For preparation of injectable platelet-Rich Fibrin we need a blood sample with no anticoagulant in a 10ml glass or glass-coated plastic tube, then centrifuged using (80-1) Chinese centrifuge at 700 rpm for 3 minutes.

Procedures & application of I-PRF: I-PRF is formed in a liquid form at the upper part of the tube then drawn out using an insulin syringe then injected to fill the pockets under treatment, then periodontal pack (periopack) was used to cover the area to keep the I-PRF in place and allow healing.

Clinical measurements: Pocket Depth (PD), Clinical Attachment Level (CAL), Plaque Index[12] (PI), and Gingival Index[13] (GI) were recorded at the baseline, one month, three months and after six months using graduated periodontal probe.

Pocket Depth was measured as the distance in millimeters from the base of the pocket to the gingival margin.

Clinical attachment level was measured as the distance from the base of the pocket to the cemento-enamel junction (CEJ), which is a more accurate measurement as it depends on fixed point.

PI and GI scores was recorded each time to assess the oral hygiene pledge of the patient.

Clinical procedures

First measurement was taken post SRP and pre injection of I-PRF at the pocket showing PD of 5.5 mm and CAL 4 mm as in (Fig. 1a) then blood sample was taken from the patient and centrifuged at 700 RPM at 3min. then I-PRF was drawn out using an insulin syringe then injected in the pocket then periopack was used to cover the whole tooth and the neighboring teeth to protect the I-PRF from being washed out by saliva, after one week periopack was removed, after one month second measurement was taken showing PD of 5 mm and CAL 2.5 mm which means (Fig. 1b), third measurement was taken after three months post injection showing PD of 5mm and CAL 3.5 mm which means a 1mm relapse (Fig. 1c) (Note the bad hygiene causing bleeding), the fourth measurement was taken after six months post injection showing PD of 5.5 mm and CAL 3.5 mm which shows stable level with little improvement of 0.5 mm from the pre-injection measurement (Fig. 1d)

After SRP first measurement was taken at the control site showing PD of 6 mm and CAL of 4 mm (fig. 2a), second measurement taken after 1 month at the control site showing PD of 5 mm and CAL of 3.5 (fig. 2b) then third measurement taken after 3 months at the control site showing PD of 5 mm and CAL of 4 mm with a 0.5 mm relapse at the CAL (fig. 2c), fourth measurement taken after six months at the control site showing PD of 5 mm and CAL of 4 mm (fig. 2d).

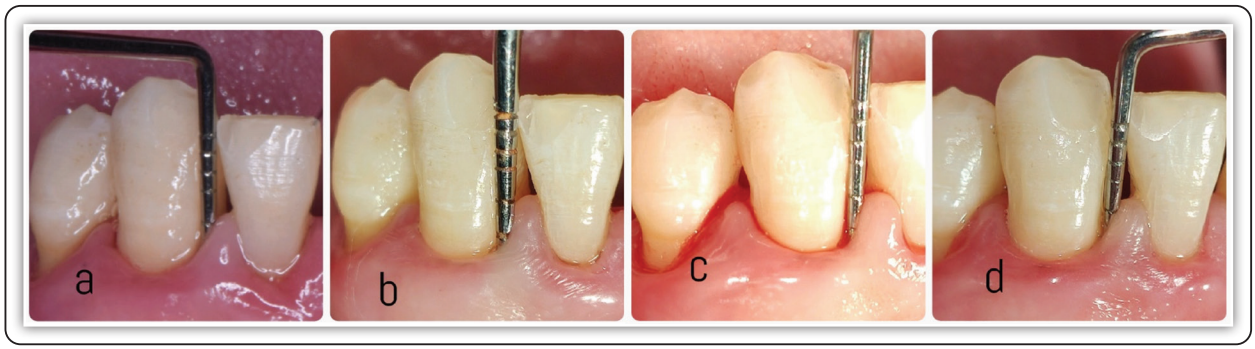

FIG (1) Measurements at the injection side, a)first measurement PD of 5.5 mm and CAL 4 mm, b)after one month measurement PD of 5 mm and CAL 2.5 mm, c)after three months PD of 5 mm and CAL 3.5mm, d)after six months PD of 5.5mm and CAL 3.5 mm

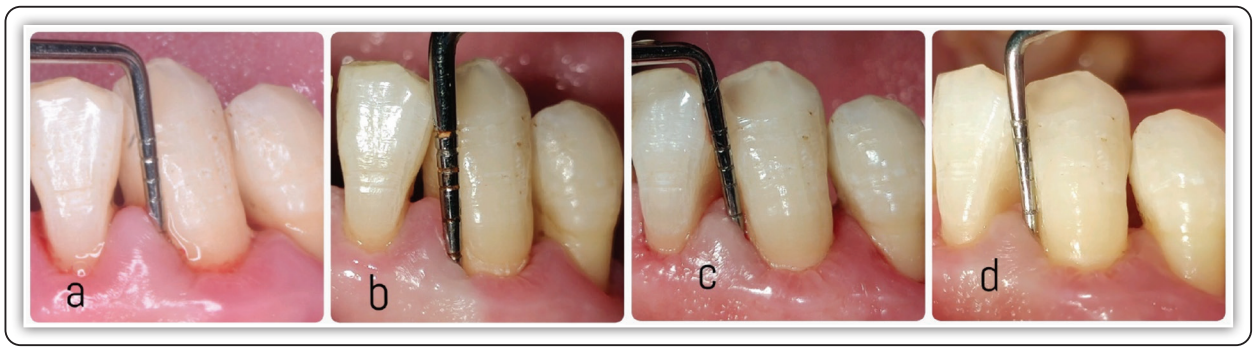

FIG (2) Measurements at the control side, a)first measurement PD of 6 mm and CAL 4 mm, b) after one month measurement PD of 5mm and CAL 3.5 mm, c) after three months PD of 5mm and CAL 4mm, d)after six months PD of 5 mm and CAL 4 mm.

RESULTS

Clinical findings (Table 1):

Comparison between the two studied groups according to PD

Preoperative, 1, 3, and 6 months: there was a statistically non-significant difference in mean PD in the two groups (p=0.661, 0.805, 0.696, and 0.325 respectively). Change from pre to 1, 3, and 6 months: there was a statistically non-significant difference in mean PD in the two groups (p=0.605, 0.605, and 0.190 respectively).

Comparison between the two studied groups according to CAL

At 1 months: there was a statistically a significant difference in mean CAL in the two groups (p=0.011*). I-PRF group showed a lower CAL than control group. Preoperative, 3, and 6 months: there was a statistically non-significant difference in mean CAL in the two groups (p=0.058, 0.545, and 0.891 respectively). Change from pre to 1, 3, and 6 months: there was a statistically non-significant difference in mean CAL in the two groups (p=0.436, 0.297, and 0.258 respectively).

TABLE (1) Comparison between the two studied groups according to PD and CAL

	Control	I-PRF	Test of Sig.	p
PD (mm)				
Pre	5.17 ± 0.83	5.33 ± 0.75	t=0.447	0.661
1 month	3.83 ± 0.83	3.72 ± 1.03	t=0.251	0.805
3 months	4.17 ± 0.75	4.39 ± 1.50	t=0.399	0.696
6 months	4.22 ± 0.67	4.83 ± 1.68	t=1.016	0.325

	Control	I-PRF	Test of Sig.	p
Change from pre to				
1 month	1.33 ± 0.94	1.61 ± 0.74	U=34.5	0.605
3 months	1.0 ±0.94	0.94 ± 1.47	U=35.0	0.605
6 months	0.94 ± 0.68	0.50 ± 1.56	U=25.0	0.190
CAL (mm)				
Pre	5.89 ± 0.60	4.94 ± 1.21	t=2.097	0.058
1 month	4.50 ± 0.75	3.39 ± 0.89	t=2.857*	0.011*
3 months	4.11 ± 0.96	3.78 ± 1.30	t=0.618	0.545
6 months	4.67 ± 0.66	4.61 ± 0.99	t=0.140	0.891
Change from pre to				
1 month	1.39 ± 0.55	1.56 ± 0.58	U=31.0	0.436
3 months	1.78 ± 0.94	1.17 ± 1.60	U=28.50	0.297
6 months	1.22 ± 0.79	0.33 ± 1.44	U=27.0	0.258

Data was expressed using Mean ± SD.

t: Student t-test U: Mann Whitney test

p: p value for comparing between the two studied groups

DISCUSSION

Periodontal regeneration is used to refer to the complete replacement of damaged tissue with new fully functional tissue.it involves a coordinated sequence of physiologic processes including as cell adhesion, migration, proliferation, and differentiation. Furthermore, periodontal wound healing necessitates interactions among epithelial cells, gingival fibroblasts, periodontal ligament cells, and osteoblasts[4]. Platelets are magical fragments of cells in the blood stream with awesome healing capacity the platelets in a clot secrete growth factors that are integral to the next steps in healing**.** they release Transforming growth factor Alpha (TGF-alpha), Transforming growth factor Beta (TGF-beta), Insulin growth factor (IGF), Platelet derived growth factor (PDGF), Interleukin-1 (IL-1). PDGF recruits fibroblasts so collagen begins to be deposited beginning the healing process[14].

Dohan et al.[15] shows that IPRF contains more GFs than PRF, which is six to seven times more loaded with GFs than PRP. In addition, those GFs are released steadily within 12-14 days. This process is enabled due to the fact that after a short period of time, approximately 15 minutes, I-PRF is transformed into a matrix scaffold [16]. This scaffold was proved to have a direct impact on the ability of human gingival fibroblasts to migrate, proliferate, release additional GFs and periodontal ligament cell growth, as well as to increase the differentiation of osteoblasts[17]. By preventing the down-growth of junctional epithelium to the root surfaces and suppressing its interference between the root and soft tissue, a new attachment on root surfaces can be formed. Furthermore, antimicrobial and anti-inflammatory effects of PRF have also been described [18].

The obtained results demonstrated that Preoperative, 1, 3, and 6 months: there was a statistically non-significant difference in mean PI, GI and PD in the two groups. Both therapeutic modalities could result in statistically significant improvement of all explored clinical parameters. Control and I-PRF groups showed a statistically significant decrease in mean PI and GI measurements at 1 months followed by non-significant increase at 3 months and a statistically significant decrease again after 6 months. The positive clinical outcomes of the control group after three months correspond with the previous findings concerning clinical efficacy of SRP in treatment of Periodontitis. This indicates that in subjects with Periodontitis, SRP was successful in reducing PD and improving CAL[19].

Vučković et al.[20] investigated whether there are differences in therapeutic effect between initial treatments of chronic periodontitis [scaling and root planning (SRP)] alone and SRP in conjunction with injectable platelet-rich fibrin (I-PRF) application, comparing clinical parameters after three months. Compared to baseline, both treatment modalities

demonstrated an improvement in investigated clinical parameters. Initial periodontal therapy in conjunction with injectable platelet-rich fibrin proved to display significant improvement in all clinical parameters compared to initial periodontal therapy alone.

In agreement with our results, Albonni et al. [21], clarified the clinical efficacy of using I-PRF as an adjunctive sub gingival irrigation to scaling and root planing (SRP) in the treatment of periodontitis. Statistically significant decreases in PI, PD, CAL for test and control groups between pretreatment and 3 months post treatment were noted in both test and control groups. For inter-group comparisons, there was no statistically significant difference in all clinical indices. In this study, both groups were clinically effective as nonsurgical periodontal treatments, without any clinical benefits of using I-PRF.

The reduction matches the previous systematic reviews on SRP with different adjuncts, showing that a one-month therapy leads to the CAL value was 1.56 mm. Our results representing better outcome compared to the control group with only 1.39 mm reduction. CAL gain during SRP with I-PRF was far higher when compared to SRP alone. The greater clinical value of CAL gain may be due to more rapid wound healing, less short-term gingival inflammation, and sustained reduction of perio-pathogenic bacteria[22].

In summary in the current study, I-PRF contains more GFs, that are released steadily within 10-14 days only and the results can be attributed to the short periods of I- PRF. since it has number of short time growth factors which are responsible for tissue regeneration and non-sustained reduction of perio-pathogenic bacteria.

CONCLUSION

Within limits of our study, injection of I-PRF after SRP has a positive clinical effect compared to SRP alone in decreasing PD and CAL.

REFERENCES

1. Smalley JW, Birss AJ, Withnall R, Silver J. Interactions of Porphyromonas gingivalis with oxyhaemoglobin and deoxyhaemoglobin. Biochem J. 2002;362(2):239-45.
2. Sllamniku-Dalipi Z, Dragidella F, Disha M, Meqa K, Disha S. ISPUB. com. Internet Journal of Dental Science. 2014;13(1).
3. Russo C, Palaia G, Loskutova E, Libotte F, Kornblit R, Gaimari G, et al., editors. Photodynamic therapy in non-surgical treatment of chronic periodontitis: short term randomized clinical trial study. Sixth International Conference on Lasers in Medicine; 2016: International Society for Optics and Photonics.
4. Harrison P. Platelet function analysis. Blood Rev. 2005; 19:111–23.
5. Coger V, Million N, Rehbock C, Sures B, Nachev M, Barcikowski S, Wistuba N, Strauß S, Vogt PM. Tissue concentrations of zinc, iron, copper, and magnesium during the phases of full thickness wound healing in a rodent model. Biological trace element research. 2019 Sep;191(1):167-76.
6. Bowden LG, Byrne HM, Maini PK, Moulton DE. A morphoelastic model for dermal wound closure. Biomechanics and modeling in mechanobiology. 2016 Jun;15(3):663-81.
7. Shashank B, Bhushan M. Injectable Platelet-Rich Fibrin (PRF): The newest biomaterial and its use in various dermatological conditions in our practice: A case series. J Cosmet Dermatol. 2021 May;20(5):1421-1426.
8. Ninan N, Thomas S, Grohens Y. Wound healing in urology. Adv Drug Deliv Rev. 2015 Mar;82-83:93-105.
9. Choukroun J, Dohan D, Diss A, Dohan S, Dohan A, Mouhyi J, et al. Platelet-rich fibrin (PRF): a second-generation platelet concentrate. Part I: technological concepts and evolution. Endod. 2006; 101:37-44.
10. Shashank B, Bhushan M. Injectable Platelet-Rich Fibrin (PRF): The newest biomaterial and its use in various dermatological conditions in our practice: A case series. J Cosmet Dermatol. 2021 May;20(5):1421-1426.
11. Armitage G. The complete periodontal examination. Periodontol 2000. 2004; 34:22-37.
12. Silness J, Löe H. Correlation between oral hygeine and periodontal condition. Acta Odontol Scand. 1964; 22:295-87.
13. Löe H, Silness J. Periodontal disease in pregnancy. Acta Odontol Scand. 1963; 70:196-10.
14. Almadani YH, Vorstenbosch J, Davison P, Murphy A. Wound healing: A comprehensive review. InSeminars in Plastic Surgery 2021 Jul 15. Thieme Medical Publishers, Inc.

15. Dohan Ehrenfest DM, de Peppo GM, Doglioli P, Sammartino G. Slow release of growth factors and thrombospondin-1 in Choukroun's platelet-rich fibrin (PRF): a gold standard to achieve for all surgical platelet concentrates technologies. Growth Factors. 2009;27(1):63–9.
16. Miron RJ, Fujioka-Kobayashi M, Hernandez M, Zhang Y, Ghanaati S, Choukroun J. Injectable platelet rich fibrin (i-PRF): opportunities in regenerative dentistry? Clin Oral Investig. 2017;21(8):2619–27.
17. Kour P, Pudakalkatti PS, Vas AM, Das S, Padmanabhan S. Comparative evaluation of antimicrobial efficacy of plateletrich plasma, platelet-rich fibrin, and injectable platelet-rich fibrin on the standard strains of porphyromonas gingivalis and aggregatibacter actinomycetemcomitans. Contemp Clin Dent. 2018;9(2): S325–30.
18. Badade PS, Mahale SA, Panjwani AA, Vaidya PD, Warang AD. Antimicrobial effect of platelet-rich plasma and platelet-rich fibrin. Indian J Dent Res. 2016;27(3):300–4.
19. Van der Weijden GA, Timmerman MF. A systematic review on the clinical efficacy of subgingival debridement in the treatment of chronic periodontitis. J Clin Periodontol. 2002;29 Suppl 3:55–71; discussion 90–1.
20. Vučković, Mila, et al. "The effect of injectable platelet-rich fibrin use in the initial treatment of chronic periodontitis." Srpski arhiv za celokupno lekarstvo 148.5-6 (2020): 280-285.
21. Albonni H, El Abdelah AAAD, Al Hamwi MOMS, Al Hamoui WB, Sawaf H. Clinical effectiveness of a topical subgingival application of injectable platelet-rich fibrin as adjunctive therapy to scaling and root planing: a double-blind, split-mouth, randomized, prospective, comparative controlled trial. Quintessence Int. 2021 Jul 20;52(8):676-685.
22. Dastoor SF, Travan S, Neiva RF, Rayburn LA, Giannobile WV, Wang HL. Effect of adjunctive systemic azithromycin with periodontal surgery in the treatment of chronic periodontitis in smokers: a pilot study. J Periodontol. 2007;78(10):1887–96.