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Original Research

Evaluation of role of PRF in treatment of infrabony defects

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ABSTRACT:

Background: Platelet-rich fibrin (PRF) is a second-generation platelet concentrate that allows one to obtain fibrin membranes enriched with platelets and growth factors after starting from an anticoagulant-free blood harvest without any artificial biochemical modification. The present study was conducted to evaluate role of PRF in treatment of infrabony defects. **Materials & Methods:** 40 patients of periodontal IBD of moderate–severe periodontitis of both genders were divided into 2 groups of 20 each. Group I were cases in which autologous PRF was used and in group II scaling and root planning was performed only. Parameters such as PPD, clinical attachment loss, defect depth and gain in clinical attachment was recorded. **Results:** Group I had 8 males and 12 females and group II had 9 males and 11 females. PPD (mm) at baseline, 3 months and 6 months in group I was 8, 6 and 2 and in group II was 8, 7 and 4 respectively. CAL (mm) at baseline, 3 months and 6 months in group I was 0, 5 and 8 and in group II was 10, 7 and 5 respectively. Defect depth (mm) at baseline, 3 months and 6 months in group I was 10, 5 and 4 and in group II was 10, 7 and 6 respectively. The difference was significant (P< 0.05). **Conclusion:** PRF found to be effective in reduction in probing pocket depth, gain in clinical attachment level and significant radiographic defect bone fill in intrabony defects. **Key words:** Platelet-rich fibrin, Periodontitis, intrabony defects

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INTRODUCTION

The ultimate goal of periodontal therapy is to regenerate the lost periodontal tissues caused by periodontitis.¹ Various controlled clinical trials have demonstrated that some of the available grafting procedures may result in periodontal regeneration in intrabony defects, but complete and predictable reconstruction of periodontal tissues is still difficult to obtain. The reason is that periodontal regeneration. The most positive outcome of periodontal regeneration procedures in intrabony defect has been achieved with a combination of bone graft and guided tissue regeneration.²

Treatment of periodontal disease is therefore of utmost importance since epidemic studies have linked periodontitis to a number of systemic diseases including cardiovascular diseases (heart attack/stroke), Alzheimer's, diabetes, obesity, and premature births, among others. It therefore becomes vital to correct the disease as early as possible and halt disease progression and utilize strategies to promote their regeneration.³

Platelet-rich fibrin (PRF), as described by Choukroun et al⁴, is a second-generation platelet concentrate that allows one to obtain fibrin membranes enriched with platelets and growth factors after starting from an anticoagulant-free blood harvest without any artificial biochemical modification. The effect of PRF on human periodontal ligament (PDL) fibroblasts and its application in periodontal intrabony defects (IBD) has been studied, and it was found that PRF increases kinase extracellular signal regulated protein phosphorylation osteoprotegerin in PDL and fibroblasts and the upregulation of alkaline phosphatase activity. IBD have also been found to exhibit pocket reduction and clinical attachment gain after 6 months with bone fill in defects.⁵ The present study was conducted to evaluate role of PRF in treatment of infrabony defects.

MATERIALS & METHODS

The present study comprised of 40 patients of periodontal IBD of moderate–severe periodontitis of both genders. Enrolment of patients in the study was performed after they agreed to participate.

Data such as name, age, gender etc. was recorded. A thorough clinical examination was performed.

RESULTS Table I Distribution of patients

of as clinical attachment loss, defect depth and gain in of clinical attachment was recorded. Results thus obtained were tabulated and analyzed statistically using chi- square test. P value less than 0.05 was . A considered significant. ed.

Patients were divided into 2 groups of 20 each. Group

I were cases in which autologous PRF was used and

in group II scaling and root planning was performed

only. In each patient, a minimum number of two sextants were present, with probing pocket depths (PPD) ≥ 5 mm in at least three teeth. Parameters such

Groups Group I		Group II	
Method	PRF	SRP	
M:F	8:12	9:11	

Table I shows that group I had 8 males and 12 females and group II had 9 males and 11 females.

Table II Comparison of clinical and radiological parameters

Parameters (mm)	Groups	Baseline	3 months	6 months	P value
PPD	Group I	8	6	2	0.01
	Group II	8	7	4	
CAL	Group I	10	6	3	0.01
	Group II	10	7	5	
Gain in clinical	Group I	0	5	8	0.05
attachment	Group II	0	3	5	
Defect depth	Group I	10	5	4	0.04
	Group II	10	7	6	

Table II, graph I shows that PPD (mm) at baseline, 3 months and 6 months in group I was 8, 6 and 2 and in group II was 8, 7 and 4 respectively. CAL (mm) at baseline, 3 months and 6 months in group I was 10, 6 and 3 and in group II was 10, 7 and 5 respectively. Gain in clinical attachment at baseline, 3 months and 6 months in group I was 0, 5 and 8 and in group II was 0, 3 and 5 respectively. Defect depth (mm) at baseline, 3 months and 6 months in group I was 10, 5 and 4 and in group II was 10, 7 and 6 respectively. The difference was significant (P < 0.05).

Graph I Comparison of clinical and radiological parameters



DISCUSSION

Periodontitis is a disease of the periodontium characterized by the irreversible loss of connective

tissue attachment and supporting alveolar bone.⁶ The goal of any periodontal therapy is to control the active inflammation to the arrest of disease progression and

the reconstruction of structures lost to the periodontal disease, where appropriate.⁷ Although the effectiveness of scaling and root planning (SRP) or surgical access for root planing, as well as regular maintenance care in moderate to severe periodontal disease cases has been well established, the efficacy is judged based on the ability of the therapy to improve osseous lesions.⁸

Since PRF was first launched more than two decades ago in regenerative medicine, its use has gained widespread acceptance across many fields of medicine including for periodontal regeneration where nearly 40 randomized clinical trials (RCTs) have investigated its regenerative potential. One of the advantages of PRF is that following centrifugation, it forms a fibrin-dense clot with host platelets and leukocytes being entrapped favoring a more extended release of growth factors over time.⁹ The present study was conducted to evaluate role of PRF in treatment of infrabony defects.

In present study, group I had 8 males and 12 females and group II had 9 males and 11 females. Chatterjee et al¹⁰ compared the effectiveness of open flap debridement (OFD) alone and OFD with either autologous platelet-rich fibrin (PRF) or titanium PRF (TPRF) in the treatment of intrabony defects (IBD). Thirty-eight patients with 90 periodontal IBD of moderate-severe periodontitis were selected and assigned to the OFD alone group (group I), the OFD with autologous PRF group (group II), or the OFD with TPRF group (group III). In each patient, a minimum number of two sextants were present, with probing pocket depths (PPD) ≥ 5 mm in at least three teeth. At 9 months' postoperatively, upon comparing the PPD reduction, defect depth reduction, and clinical attachment level gains, it was noted that groups II and III showed statistically-significant improvements compared with group I, but no statistically-significant difference was noted between groups II and III.

We found that PPD (mm) at baseline, 3 months and 6 months in group I was 8, 6 and 2 and in group II was 8, 7 and 4 respectively. CAL (mm) at baseline, 3 months and 6 months in group I was 10, 6 and 3 and in group II was 10, 7 and 5 respectively. Gain in clinical attachment at baseline, 3 months and 6 months in group I was 0, 5 and 8 and in group II was 0, 3 and 5 respectively. Defect depth (mm) at baseline, 3 months and 6 months in group I was 10, 5 and 4 and in group II was 10, 7 and 6 respectively. Miron et al11 compared the treatment outcomes of periodontal intrabony defects by using platelet-rich fibrin (PRF) with other commonly utilized modalities. Studies were classified into 10 categories as follows: (1) open flap debridement (OFD) alone versus OFD/PRF; (2) OFD/bone graft (OFD/BG) versus OFD/PRF; (3) OFD/BG versus OFD/BG/PRF; (4-6) OFD/barrier membrane (BM), OFD/PRP, or OFD/enamel matrix derivative (EMD) versus OFD/PRF; (7) OFD/EMD versus OFD/EMD/ PRF;

(8-10) OFD/PRF versus OFD/PRF/metformin, OFD/PRF/bisphosphonates, or OFD/PRF/statins. Weighted means and forest plots were calculated for probing depth (PD), clinical attachment level (CAL), and radiographic bone fill (RBF). Results From 551 articles identified, 27 RCTs were included. The use of OFD/PRF statistically significantly reduced PD and improved CAL and RBF when compared to OFD. No clinically significant differences were reported when OFD/BG was compared to OFD/PRF. The addition of PRF to OFD/BG led to significant improvements in CAL and RBF. No differences were reported between any of the following groups (OFD/BM, OFD/PRP, and OFD/EMD) when compared to OFD/PRF. No improvements were also reported when PRF was added to OFD/EMD. The addition of all three of the following biomolecules (metformin, bisphosphonates, and statins) to OFD/PRF led to statistically significant improvements of PD, CAL, and RBF.

Despite of the fact that PRF is a denser and firmer agent than other biological preparations, such as PRP and enamel matrix derivative (EMD), it is still non-rigid to a degree that its space maintaining ability in periodontal defects is non-ideal.¹² It has been reported that the combination of a mineralized, rigid bone mineral, with a semi fluid, non-rigid agent, such as EMD, significantly enhanced the clinical outcome of intrabony defects than treated without the addition of bone mineral.

CONCLUSION

Authors found that PRF found to be effective in reduction in probing pocket depth, gain in clinical attachment level and significant radiographic defect bone fill in intrabony defects.

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