Original Research Article

Comparative evaluation of platelet rich fibrin and advanced platelet rich fibrin in management of periodontal infrabony defect: A clinical radiographic study

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Abstract

Background and objective: The purpose of the study was to clinically and radiographically evaluate and compare Platelet - rich fibrin (PRF) and Advanced Platelet-rich fibrin (A-PRF) in the treatment of human periodontal infrabony defects. **Methods:** A total of 28 patients having infrabony defects were selected for the study. The study sites were randomly divided into group A (PRF) and group B (A-PRF). The clinical parameters like Plaque index, Gingival index, Probing pocket depth and Clinical attachment level were recorded at baseline, 3 months and 6 months. Radiographic evaluation at baseline and 6 months were carried out to evaluate the defect fill, change in alveolar crest height and defect resolution. **Results:** Significant improvements in all clinical parameters were observed in both the groups as compared to baseline. On comparison between the groups statistically significant difference was observed in terms of probing pocket depth and clinical attachment level for A-PRF group. In group A, the mean osseous defect of 8.22 ± 3.75mm at baseline was reduced to 6.23 ± 2.58 mm at 6 months. In group B, the mean osseous defect of 6.22 ± 2.59 mm at baseline was reduced to 5. 07 ± 1.51 mm at 6months. The results being highly significant for both the groups from baseline on intergroup comparison, the mean defect fill and mean defect resolution showed significantly difference in favour of PRF. **Conclusion:** Individually both the groups have shown promising results in the management of periodontal infrabony defects. However, statistically, group A showed better treatment outcome in terms of bone fill and group B showed better results in terms of soft tissue healing.

Key Word: Platelet rich fibrin, infrabony defect, periodontal regeneration, advanced platelet rich fibrin.

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INTRODUCTION

Periodontitis can be defined as "an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or group of the specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession or both".[1] It is one of the most common and widespread diseases affecting mankind in various forms and severities.[2] The etiology is the accumulation of plaque around the surfaces of teeth and gingival margins, initially resulting in gingival inflammation and later spreading to the underlying periodontal tissues. Although periodontitis is an infectious disease of the gingival tissue, changes that occur in the bone are crucial because the destruction of the bone is responsible for tooth loss.[4] The purpose of conventional periodontal therapy, both non-surgical and surgical, is to eliminate the inflammation of the periodontal tissues and to arrest the

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destruction of soft tissue and bone. Over the years, there has been a growing interest in exploring the ability to regenerate the tissues lost due to the disease, and thereby not only arresting the periodontal disease, but also reversing it. Regeneration has been defined as the reproduction or reconstitution of a lost or injured part to restore the architecture and function of the periodontium. The goal of periodontal therapy has always been regeneration of the lost attachment apparatus, in conjunction with shallower probing depths (PD), thus, facilitating periodontal maintenance.[5]New attachment may occur with the formation of new cementum with inserting collagen fibers over a previously contaminated root surface, while regeneration of the periodontal attachment apparatus also includes the formation of new alveolar bone. Several procedures have been suggested for the regenerative therapy of periodontal lesions, including use of autografts, allografts and xenografts[6], Guided tissue regeneration[7], root conditioning methods[8] and the use of growth factors[9] and other molecules which are believed to play a role in growth and differentiation of periodontal cells. Various biomaterials have been developed and based on their endogenous regenerative capacity they were used for regeneration of periodontal tissue but till date no graft material was considered as gold standard. Platelet - rich fibrin (PRF) was developed in France by Choukroun *et al*(2001)[10]. It is a second-generation platelet concentrate, widely used to accelerate hard and soft tissue healing. First biochemical analysis of the PRF indicated that this material consists of an intimate assembly of cytokines, glycemic chains, and structural glycoproteins enmeshed within a slowly polymerized fibrin network. The biologic activity of the fibrin molecule is enough in itself to account for the significant cicatricial capacity of the PRF and the slow polymerization mode confers to the PRF membrane a particularly favourable physiologic architecture to support the healing process.[11] Advanced Platelet Rich Fibrin (A-PRF) is a third-generation product derived from a concentration of platelets and white blood cells. The protective membranes that are produced, release key proteins that stimulate bone and soft tissue growth, accelerating soft tissue and bone healing. The A-PRF membranes contain a large quantity of proteins that aid in increasing cell attachment to soft tissue. With the release of these proteins, healing is more rapid and more effective. The main property of this biomaterial is the slow release of these proteins from the A-PRF material over seven to ten days which accelerates the natural healing phase.[12]To best of our knowledge, no study reported the clinical use of A-PRF for the treatment of periodontal infrabony defects. Thus, the purpose of the present study was to evaluate and compare the clinical and radiographic outcome of Platelet - rich fibrin(PRF) and Advanced Platelet-rich fibrin (A-PRF) in the treatment of human infrabony osseous defect resulting periodontal disease.

AIM AND OBJECTIVES

- To evaluate and compare clinical and radiographic outcome of Platelet – rich fibrin (PRF) and Advanced Platelet-rich fibrin (A-PRF) in the treatment of human infrabony osseous defect caused by periodontal disease.
- To determine amount of reduction from baseline osseous defect depth parameters and amount of bone fill in the experimental sites by use of these materials.
- To determine whether the use of Platelet - rich fibrin (PRF) and Advanced Platelet rich fibrin (A-PRF) improve the periodontal status of involved teeth from baseline such as reduction in probing pocket depth gain in clinical attachment level and gingival margin level after a stipulated period of time

METHODS

This study was designed as a prospective randomized controlled clinical trial. The patients were selected from the outpatient department. All participants were informed about the risks and benefits of the procedure and signed informed consent was taken. The two different therapeutics modalities for the treatment of deep intraosseous periodontal defects were compared. Total 30 patients were initially screened for. Finally, 28 patients were selected the study. The selected patients were randomly allocated to either of the experimental groups:

Group I: Open flap debridement, followed by placement of platelet rich fibrin (PRF)

Group II: Open flap debridement, followed by placement of advanced platelet rich fibrin (A-PRF)

Clinical parameters: Plaque Index (PI) **(**Silness and Loe, 1963)[13], Gingival index (Loe and Silness J **1964)**[14]and Clinical Attachment Level (CAL) were recorded at baseline, 3 months and6 months.

Clinical measurements using stent:

1. Fixed reference point (FRP) to gingival margin (GM).
2. Fixed reference point (FRP) to cement enamel junction (CEJ)
3. Fixed reference point (FRP) to base of the pocket (BOP)

All the measurements were standardized using customized acrylic stents with grooves which were prepared on the study model of the patients. The recordings were made using a HU-Friedy UNC 15 probe.

The following calculations were made from the clinical measurements recorded:

I. Probing pocket depth = (FRP to BOP) – (FRP to GM)
II. Clinical attachment level = (FRP to BOP) – (FRP to CEJ)
III. Gingiva margin position = (FRP to CEJ) – (FRP to GM)

Intra –oral periapical radiographs (IOPAR) of all the selected sites were taken using long cone paralleling technique with Extended cone paralleling (XCP) holder at baseline, 3 months and 6 months post-operatively. However, statistical analysis was performed only after final post-operative results i.e. after 6 months.

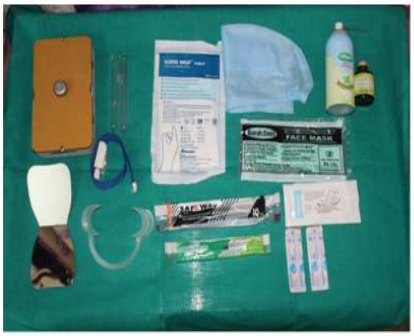


Figure 1: Clinical Armamentarium; **Figure 2:** Centrifugal Machine

Statistical analysis: The above mentioned clinical and radiographic parameters were evaluated at baseline and after 6 months. Indices were evaluated at baseline, 3 months and 6 months.
Formulae Used for Analysis:

RESULTS

The present clinical study was conducted to evaluate and compare the efficacy of platelet rich fibrin and advanced platelet rich fibrin in the treatment of periodontal infrabony defect and their effect on clinical and radiographic parameters. A total of 28 patients were included in the study. The selected patients were randomly allocated to either of the groups:

Group I: Open flap debridement, followed by placement of platelet rich fibrin (PRF)

Group II: Open flap debridement, followed by placement of advanced platelet rich fibrin (A-PRF)

The following clinical parameters were recorded at baseline, 3 and 6 months post-operatively.

1. Plaque inde
2. Gingival index
3. Probing pocket depth
4. Clinical attachment level

The intra-oral periapical radiograph (IOPAR) were taken of the selected sites using long cone paralleling technique, at baseline and 6 months post-operatively. The radiographic assessment of bone level was done by Digimizer software.

The following parameters were recorded radiographically.

1. Amount of percentage of bone defect
2. Amount and percentage of original defect resolution
3. Change in the level of alveolar crest height

CLINICAL PARAMETERS

Plaque index: (Table 1, 2 and 3 Graph 1)In group A**,** mean plaque score at baseline was 1.50 ± 0.42 which was reduced to 1.03 ± 0.13 at 3 months and 1.0 ± 0.00 at 6 months, showing a mean reduction of 0.47 ± 0.29 at 3 months, 0.50 ± 0.42 at 6 months and 0.03 ± 0.13 when compared between 3 months and 6 months. Whereas in group B, mean plaque score at baseline was 1.62 ± 0.62 which was reduced to 1.12 ± 0.22 at 3 months and 1.00 ± 0.00 at 6 months, showing a mean reduction of 0.50 ± 0.22 at 3 months, 0.62 ± 0.22 at 6 months and 0.12 ± 0.22 when compared between 3 months and 6 months. Thus, it was observed that mean plaque score reduction was statistically significant at 3 and 6 months in both the groups when compared with baseline, but, it was not statistically significant at 6 months when compared with 3 months. The results were also non-significant when compared between both the groups for all the time intervals.

Gingival index: (Table 4, 5 and 6, Graph 3)In group A, mean gingival index score at baseline was 1.27 ± 0.35 which was reduced to 0.93 ± 0.59 at 3 months and 0.50 ±0.49 at 6 months showing a mean reduction of 0.34 ± -0.20 at 3 months, 0.77 ± -0.10 at 6 months and 0.43 ± 0.10 when compared between 3 months and 6 months. Whereas in group B, mean gingival score at baseline was 1.32 ± 0.46 which was reduced to 0.71 ±0.27 at 3 months and 0.38 ± 0.13 at 6 months, showing a mean reduction of 0.61 ± 0.19 at 3 months, 0.94 ± 0.33 at 6 months with a

mean reduction in the gingival score of 0.33 ± 0.14 mm when compared between 3 months and 6 months. Thus, it was observed that gingival score reduction was statistically significant at 3 months and 6 months in both the groups when compared with baseline. Comparison of gingival index score between group A and B, for all time intervals showed non-significant results.

Probing Pocket Depth: (Table 7, 8 and 9)In group A, mean probing pocket depth at baseline was 7.93 ± 1.39 mm which was reduced to 4.20 ± 0.94 mm at 3 months and 3.93 ± 0.96 mm at 6 months, showing a mean reduction of 3.73 ± 0.45 mm at 3 months, 4.00 ± 0.43 mm at 6 months and 0.27± 0.02 mm when compared between 3 months and 6 months. Whereas in group B, mean probing pocket depth at baseline was 7.69 ± 1.03 mm which was reduced to 3.38±0.77 mm at 3 months and 3.31 ± 0.85 mm at 6 months, showing a mean reduction of 3.86 ± 0.26 mm at 3 months, 4.38 ± 0.18 mm at 6 months and 0.07 ± -0.08 mm when compared between 3 months and 6 months. Thus, it was observed that PPD reduction was highly statistically significant after 3 months and 6 months in both the groups when compared with baseline. When doing the intergroup comparison, it was noted that there was a greater reduction of PPD in group B, which was highly significant at both 3 and 6 months.

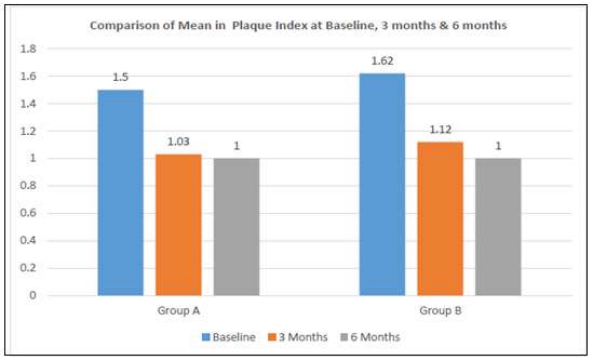
Clinical Attachment Level: (Table 10, 11 and12)In group A, mean CAL level at baseline was 8.80 ± 1.21 mm which was reduced to 5.20 ± 0.86 mm at 3 months and 5.13 ± 0.92 mm at 6 months, showing a mean reduction of 3.60 ± 0.35 mm at 3 months, 3.67 ± 0.29 mm at 6 months and 0.07 ± 0.06mm from 3 months to 6 months. Whereas in group B, mean CAL at baseline was 8.00 ± 1.53 mm which was reduced to 4.46 ±1.20 mm at 3 months and 4.46 ± 1.27 mm at 6 months, showing a mean reduction of 3.54 ± 0.33 mm at 3 months, 3.54 ± 0.26 mm at 6 months with a mean reduction in the clinical attachment level of 0.00 ± 0.27 mm when compared between 3 months and 6 months. Thus, it was observed that the scores which were obtained after 3 months and 6 months were statistically significant in both the groups when compared with baseline, But, it is not statistically significant at 6 months when compared with 3 months (P=1.00). When doing the intergroup comparison, it was noted that there was a significantly greater gain in attachment in group B at both 3 and 6 months time interval.

RADIOGRAPHIC EVALUATION

Mean defect fill:(Table 13, 14 and 15)In group A, mean distance from the CEJ to the base of the defect at baseline was 8.22 ± 3.75 mm which was reduced to 6.23 ± 2.58 at 6 months post-operatively, showing a mean defect fill of 1.99 ± 1.17 mm, whereas in group B, mean distance from the CEJ to the base of the defect at baseline was 6.22 ± 2.59 mm which was reduced to 5.07 ± 1.51mm at 6 months, showing a mean defect fill of 1.15 ± 1.08 mm. It was observed that in both the groups, the scores which were obtained at 6 months were statistically highly significant when compared with that of the baseline. When intergroup comparision was done it was noted that there was a greater gain in CEJ to the base of the defect in group A at baseline to 6 months.

Mean Defect Resolution: (Table 16,17 and 18)In group A, mean distance from the alveolar crest to the base of the defect at baseline was 6.27 ± 2.27 mm which was reduced to 4.13 ± 1.48 at 6 months postoperatively, showing a mean defect resolution of 2.14 ± 0.79 mm at 6 months postoperatively, whereas in group B,mean distance from the alveolar crest to the base of the defect at baseline was 4.76 ± 1.52 mm which was reduced to 3.77 ± 0.77 mm at 6months post-operatively, showing a mean defect resolution 0.99 ± 0.75 mm at 6 months post-operatively. It was observed that in both the **groups** the scores which was obtained for 6 months is highly significant when compared with that of the baseline. When intergroup comparision was done it was noted that there was a greater gain in alveolar crest to the base of the defect in group A at baseline to 6 months.

Change in alveolar crest Height: (Table 19, 20 and 21)In group A, mean distance from CEJ to the alveolar crest at baseline was 1.95± 3.25 mm which was reduced to 2.11 ± 2.59 at 6 months post-operatively, showing a mean gain in alveolar crest height of -0.16 ± 0.66 mm at 6 months post-operatively, whereas in group B mean distance from CEJ to the alveolar crest at baseline was 1.45± 1.42 mm which was reduced to 1.36 ± 1.47 mm at 6 months post-operatively, showing a mean gain in alveolar crest height of 0.99 ±- 0.05 mm at 6 months. Comparision between the two groups post-operatively revealed statistically insignificant differences(P=0.2343)



Graph 1: Intragroup comparison of Mean Plaque index in Group A and Group B at Baseline, 3 Months and 6 Months

Table 1: Mean Index in Plaque Reduction in Group A

Time Interval	Mean± SD	Difference from Baseline	Significance (p)*
Baseline	1.50± 0.42	-	-
3 Months	1.03±0.13	0.47±0.29	t = 2.144, p = 0.0004 (HS)
6 Months	1.00±0.00	0.50±0.42	t = 2.144, p = 0.0004 (HS)
3 Months, 6 Months		0.03±0.13	t = 2.144, p = 0.334 (NS)

Note: HS- Highly Significant

Table 2: Mean reduction in plaque index in Group B

Time Interval	Mean± SD	Difference from Baseline	Significance (p)*
Baseline	1.62± 0.62	-	-
3 Months	1.12±0.22	0.50±0.22	t = 2.178, p = 0.0003
6 Months	1.00±0.00	0.62±0.22	t = 2.177, p = 0.0004
3 Months, 6 Months	-	0.12±0.22	t = 2.177, p = 0.082

Table 3: Comparison of mean reduction in plaque for Group A and Group B

Time Interval	Group A (Mean± SD)	Group B (Mean± SD)	Difference (Mean ± SD)	Significance
Baseline	1.50± 0.42	1.62±0.62	(-0.12) ± (-0.20)	t = 2.144, p = 0.6073,
3 Months	1.03±0.13	1.12±0.22	(-0.09) ± (-0.09)	t = 2.144, p = 0.6337,
6 Months	1.00±0.00	1.00±0.00	0.00 ± 0.00	t = 2.144, p = 0.1643,

Table 4: Mean reduction in gingival index in group A

Time Interval	Mean± SD	Difference from Baseline	Significance (p)*
Baseline	1.27 ± 0.39	-	-
3 Months	0.93 ± 0.59	0.34 ± (-0.20)	t = 2.145, p = 0.110
6 Months	0.50 ± 0.49	0.77 ± (-0.10)	t = 2.145, p = 0.0003 (HS)
3 Months, 6 Months	-	0.43 ± 0.10	t = 2.145, p = 0.0074 (HS)

Table 5: Mean reduction in gingival index in Group B

Time Interval	Mean± SD	Difference from Baseline	Significance (p)*
Baseline	1.32 ± 0.46	-	-
3 Months	0.71 ± 0.27	0.61 ± 0.19	t = 2.178, p = 0.001
6 Months	0.38 ± 0.13	0.94 ± 0.33	t = 2.177, p = 0.000016 (HS)
3 Months, 6 Months	-	0.33 ± 0.14	t = 2.177, p = 0.00064 (HS)

Table 6: Comparison of mean reduction in gingival index for group A and Group B

Time Interval	Group A (Mean± SD)	Group B (Mean± SD)	Difference (Mean ± SD)	Significance
Baseline	1.27 ± 0.39	1.32 ± 0.46	(-0.05) ± (-0.07)	t = 2.144, p = 0.3506,
3 Months	0.93 ± 0.59	0.71 ± 0.27	0.22 ± 0.32	t = 2.144, p = 0.1387,
6 Months	0.50 ± 0.49	0.38 ± 0.13	0.12 ± 0.36	t = 2.144, p = 0.323,

Table 7: Mean reduction in probing pocket depth in Group A

Time Interval	Mean ± SD	Difference from Baseline	Significance(p*)
Baseline	7.69 ± 1.03	-	-
3 months	3.38 ± 0.77	3.86 ± 0.26	t=2.177, p=<0.001
6 months	3.31 ± 0.85	4.38 ± 0.18	t=2.178, p=<0.001
3 months, 6 months	-	0.07 ± (-0.08)	t=2.178, p=0.5844

Table 8: Mean reduction in probing pocket depth in Group B

Time Interval	Mean± SD	Difference from Baseline	Significance (p)*
Baseline	7.93 ± 1.39	-	-
3 Months	4.20 ± 0.94	3.73 ± 0.45	t = 2.144, p = < 0.01
6 Months	3.93 ± 0.96	4.00 ± 0.43	t = 2.145, p = <0.01
3 Months, 6 Months	-	0.27 ± 0.02	t = 2.145, p = 0.0405

Table 9: Comparison of mean reduction in Probing Pocket depth for Group A and Group B

Time Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	Difference (Mean ± SD)	Significance
Baseline	7.93 ± 1.39	7.69 ± 1.03	0.54 ± 0.36	t=2.144, p=0.123
3 months	4.20 ± 0.94	3.38 ± 0.77	0.82 ± 0.17	t=2.144, p=0.0012
6 months	3.93 ± 0.96	3.31 ± 0.85	0.62 ± 0.11	t=2.144, p=0.00607

Table 10: Mean reduction in clinical attachment level in group A

Time Interval	Mean ± SD	Difference from Baseline	Significance (p*)
Baseline	8.80 ± 1.21	-	-

3 months	5.20 ± 0.86	3.60 ± 0.35	t=2.144, p=0.581
6 months	5.13 ± 0.92	3.67 ± 0.29	t=2.145, p=<0.01
3 months, 6 months	-	0.07 ± 0.06	t=2.145, p=0.5816

Table 11: Mean reduction in clinical attachment level in group B

Time Interval	Mean ± SD	Difference from Baseline	Significance (p*)
Baseline	8.00 ± 1.53	-	-
3 months	4.46 ± 1.20	3.54 ± 0.33	t=2.178, p=<0.001
6 months	4.46 ± 1.27	3.54 ± 0.26	t=2.177, p=<0.001
3 months, 6 months	-	0.00 ± 0.27	t=2.177, p=1.00

Table 12: Comparison of Mean Reduction in clinical Attachment level for Group A and Group B

Time Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	Difference (Mean ± SD)	Significance
Baseline	8.80 ± 1.21	8.00 ± 1.53	0.80 ± (-0.32)	t=2.145, p=0.335
3 months	5.20 ± 0.86	4.46 ± 1.20	0.75 ± (-0.34)	t=2.144, p=0.031
6 months	5.13 ± 0.92	4.46 ± 1.27	0.67 ± (-0.35)	t=2.144, p=0.0415

Table 13: Comparison of Mean defect fill CEJ to BD in group A and group B

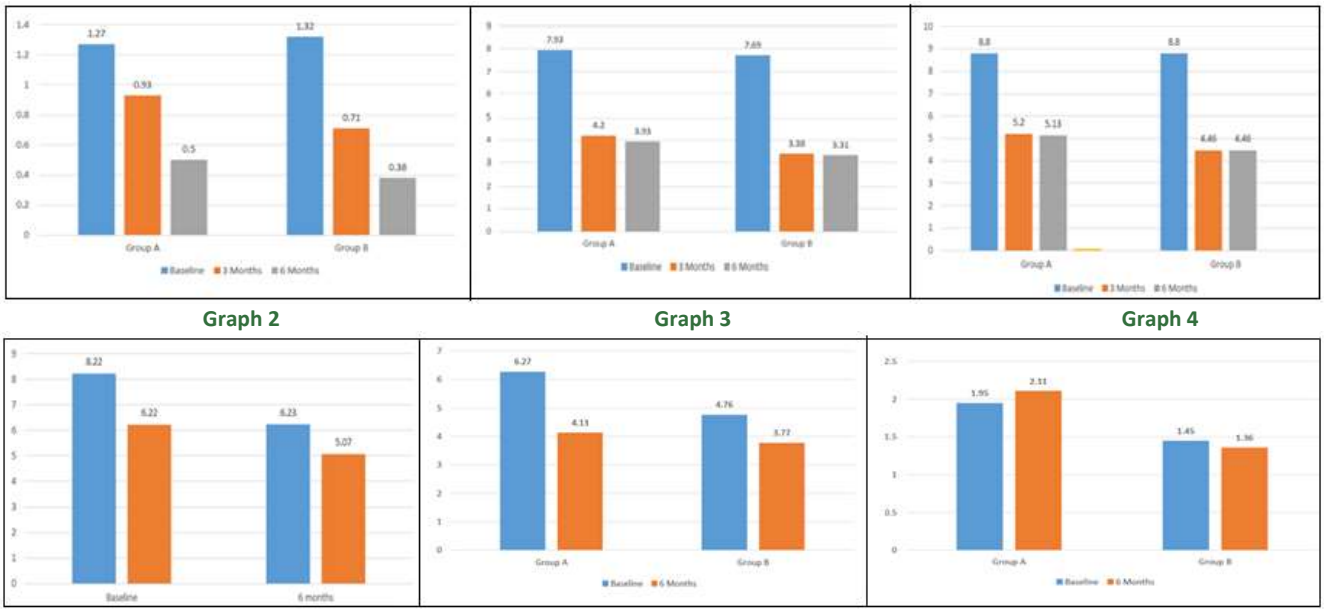
Time Interval	Baseline	6 Months	Difference	Significance
Group A	8.22 ± 3.75	6.23 ± 2.58	1.99 ± 1.17	t=2.145, p=0.00018
Group B	6.22 ± 2.59	5.07 ± 1.51	1.15 ± 1.08	t=2.144, p=0.01468
Difference A and B	2.00 ± 1.16	0.96 ± 1.07	0.84 ± 0.09	t=2.045, p=0.000009

Table 14: Comparison of Mean Reduction Alveolar crest for group A and Group B

Time Interval	Baseline	6 Months	Difference	Significance
Group A	6.27 ± 2.27	4.13 ± 1.48	2.14 ± 0.79	t=2.145, p=0.00013
Group B	4.76 ± 1.52	3.77 ± 0.77	0.99 ± 0.75	t=2.144, p=0.01967
Difference A and B	1.51 ± 0.75	0.36 ± 0.71	1.15 ± 0.04	t=2.045, p=0.000012

Table 15: Comparison of mean reduction CEJ to AC for Group A and Group B

Time Interval	Baseline	6 Months	Difference	Significance
Group A	1.95 ± 3.25	2.11 ± 2.59	(-0.16) ± 0.66	t=2.145, p=0.576
Group B	1.45 ± 1.42	1.36 ± 1.47	0.09 ± (-0.05)	t=2.145, p=0.5264
Difference A and B	1.50 ± 1.83	0.75 ± 1.12	(-0.25) ± 0.71	t=2.045, p=0.7834



Graph 2 Graph 3 Graph 4

Graph 5 Graph 6 Graph 7

Graph 2: Intragroup comparison of mean in gingival index in Group A and Group B at baseline, 3 Month and 6 months; **Graph 3:** Intragroup comparison of mean Probing Pocket depth in Group A and Group B at baseline ,3 months and 6 months; **Graph 4:** Intragroup comparison of mean Clinical attachment Level (CAL) in Group A and Group B at baseline, 3 months and 6 Month; **Graph 5:** Intragroup comparison of mean Defect fill in Group A and Group B at baseline and 6 Month; **Graph 6:** Intragroup comparison of mean Alveolar crest in group A and Group B at baseline and 6 Month; **Graph 7:** Intragroup comparison of mean CEJ to alveolar crest heigh in groups A and B and baseline and 6 month

DISCUSSION

Since the dawn of civilization, mankind has witnessed/ suffered from oral diseases, chiefly periodontitis and dental caries. Periodontitis disrupts the harmony between the various parts of the periodontium which finally leads to tooth mortality. Reconstruction of the lost periodontal structures as a consequence of periodontal diseases has been an evasive goal more than a century (Carranza and Kenney,1991).[15]Various regenerative modalities have been investigated for the management ofinfrabony periodontal defects e.g. bone grafts (BG) and substitutes, guided tissueregeneration (GTR), growth factors, enamel matrix derivatives (EMD) and combinedapproaches.105Polypeptide growth factors (PGFs) revealed a potential application in woundhealing by promoting periodontal regeneration via cell proliferation, angiogenesis,chemotaxis and differentiation. Autologous blood concentrates constitute a safe andconvenient approach to deliver high concentrations of PGFs to periodontal surgical wounds.[16,17] PRF is a second-generation platelet concentrate, widely used to accelerate soft and hard tissue healing.The use of this platelet and immune concentrate during bone grafting offers the following advantages. First, the fibrin clot plays an important mechanical role, with the PRF membrane maintaining and protecting the grafted biomaterials and PRF fragments serving as biological connectors between bone particles.4 Second, the integration of this fibrin network into the regenerative site facilitates cellular migration, particularly for endothelial cells necessary for the neoangiogenesis, vascularization, and survival of the graft. Third, the platelet cytokines (PDGF, TGF-b, IGF-1) are gradually released as the fibrin matrix is resorbed, thus creating a perpetual process of healing. Lastly, the presence of leukocytes and cytokines in the fibrin network can play a significant role in the self-regulation of inflammatory and infectious phenomena within the grafted material; PRF is also a supportive matrix for bone morphogenetic protein. Advance Platelet rich fibrin (A-PRF) is a relatively new concept for cell-based tissue engineering by mean of inflammatory cells. A-PRF is a third-generation platelet concentrate derived from the platelet concentration of platelets and leukocytes. Growth factor release from A-PRF are PDGFAA, PDGF-AB, PDGFBB, TGF-β, VEGF, EGF and IFG. Protein release from A-PRF is significantly high when compare with PRP and PRF. The subsequent significant increase in total protein release may therefore present additional advantage for clinical use. A-PRF contain more living progenitor cells and platelets when compared with PRF.A-PRF releases more growth factors, platelets andneutrophilic granulocytes than PRF, thus, it may be hypothesized that these cellscontributed to the slight increase in total growth factor accumulation after

10 days period. However, only a few studies have been reported to evaluate the influence of APRF on bone healing. In A-PRF, neutrophilic granulocytes are present in the distal part of the clot. Neutrophilic granulocytes contribute to monocyte differentiation into macrophages. Accordingly, a higher presence of these cells might be able to influence the differentiation of host macrophages within the clot. Thus, A-PRF might influence bone and soft tissue regeneration, especially through the presence of monocytes/macrophages and their growth factors. Complex tissue engineering concepts needs to be evaluated in terms of their clinical applicability. Thus, the overall goal was to establish a method that could ideally be completed within a short time span before or during intended regenerative surgical procedures. The present study revealed that A-PRF has better effect on soft tissue regeneration. The present study suggested that PRF showed better treatment outcome when compared with A-PRF in context of osseous healing and regeneration. As the soft tissue healing and hard tissue healing occur in different phases of wound healing, we can concluded from this study that A-PRF secretes more growth factors compared to LPRF which promote fibroblast proliferation leading to better soft tissue healing where as PRF owing to its better organization and denser fibrin network might support the osseous healing better. One of the important criteria to measure the periodontal regeneration is the histological examination. The ethical concern was the main reason not to include the histological aspect in the present study. Due to certain limitation, clinical and radio graphical evaluation was done in this study to evaluate the bone regeneration in infrabony defects. However, a long-term, multicenter randomized controlled clinical trial is needed to determine the clinical and radiographic effects of A-PRF on bone regeneration.

SUMMARY AND CONCLUSION

The present study was conducted to evaluate and compare the efficacy of Platelet rich fibrin (PRF) and Advanced platelet rich fibrin (A-PRF) in the treatment of periodontal infrabony defects. A total of 28 patients with infrabony defects were selected randomly and were divided into group A (PRF) and group B (A-PRF). Ethical clearance was obtained from the ethical committee. Informed consents were also obtained from the study subjects. The clinical parameters like Plaque Index, Gingival Index, Probing Pocket Depth and Clinical Attachment Level, were recorded at baseline, 3 months and 6 months. Radiographic parameters were also recorded at baseline and 6 months post-operatively, on standardized intra-oral periapical radiographs. The radiographic interpretation was done with the help of Digimizer softwere. All the pre and post operatively clinical and radiographic measurements were statistically analyzed. The following conclusion was drawn from present study: It was observed that both the groups showed the potential of enhancing the periodontal healing and filling of the defect. But, statistically, the PRF group was found to be better in terms of defect fill and defect resolution; whereas the A-PRF seems to supports soft tissue healing better. A long-term, multicenter randomized controlled clinical trial is needed to determine the clinical and radiographic effects of PRF as well A-PRF on bone and soft tissue and the mechanism behind it.

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