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Comparative evaluation of autologous platelet-rich fibrin and platelet-rich plasma in the treatment of mandibular degree II furcation defects: a randomized controlled clinical trial

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Background: The treatment of molar furcation defects remains a considerable challenge in clinical practice. The identification of clinical measurements influential to treatment outcomes is critical to optimize the results of surgical periodontal therapy. The present study aimed to explore the clinical and radiographical effectiveness of autologous platelet-rich fibrin (PRF) and autologous platelet-rich plasma (PRP) in the treatment of mandibular degree II furcation defects in subjects with chronic periodontitis.

Material and Methods: Seventy-two mandibular degree II furcation defects were treated with either autologous PRF with open flap debridement (OFD; 24 defects) or autologous PRP with OFD (25), or OFD alone (23). Clinical and radiological parameters such as probing depth, relative vertical clinical attachment level and horizontal clinical attachment level along with gingival marginal level were recorded at baseline and 9 mo postoperatively.

Results: All clinical and radiographic parameters showed statistically significant improvement at both the test sites (PRF with OFD and PRP with OFD) compared to those with OFD alone. Relative vertical clinical attachment level gain was also greater in PRF (2.87 ± 0.85 mm) and PRP (2.71 ± 1.04 mm) sites as compared to control site (1.37 ± 0.58 mm), and relative horizontal clinical attachment level gain was statistically significantly greater in both PRF and PRP than in the control group.

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Conclusions: The use of autologous PRF or PRP were both effective in the treatment of furcation defects with uneventful healing of sites.

Regeneration of the lost attachment apparatus and a return to predisease architecture are the ultimate goals of periodontal therapy. Furcations are frequently not accessible for adequate professional debridement, because their entrance is small for the size of periodontal instruments, and they present with ridges, convexities and concavities that frequently render the defect impossible to instrument effectively (1). The application of a specific treatment method for furcation involvement requires a thorough understanding of tooth anatomy, etiologic factors and the biologic basis for treatment modalities. Regeneration of the periodontium within the furcation defect is considered one of the most challenging aspects of periodontal therapy (2). Molar furcation involvement is one of the most common dentoalveolar sequelae of periodontal disease. It has been reported that molars with periodontitis involving furcation have a higher rate of periodontal breakdown and furcation-involved molars respond less favorably to periodontal therapy than molars without furcation involvement or single-rooted teeth (3–5). This can be explained by anatomy that impedes accessibility for individual oral hygiene (6) and professional root debridement in the molar region (7).

Clinically, successful regeneration at furcation sites is determined as the elimination or reduction of the horizontal and vertical components of the lesion (that is, gain of clinical attachment level and bone fill), but conclusive evidence of true regeneration can only be achieved by histological means (8). Multiple approaches have been used to resolve furcation defects, including autografts (9–12), demineralized freeze-dried bone allografts (10–13), bovine-derived xenografts (10–12,14), barrier membranes (15–18) and combinations of membranes and bone grafts (19,20). Although these regenerative materials are still used today, the introduction of biomimetic agents, such as enamel matrix derivatives (21), platelet-derived growth factor (PDGF) (22–24) and bone morphogenic proteins (21), has given new promise for better outcomes in furcation treatment.

In recent years, there has been a growing interest in the use of platelet concentrates for the treatment of many intraoral clinical conditions, including periodontal defects. A convenient technique to obtain a high concentration of PDGFs is by preparing autologous platelet-rich plasma (PRP) (25). Fundamentally, it involves the sequestration and concentration of platelets in plasma, with subsequent application of this preparation to wound-healing sites. It has been shown that application of PRP to the wound-healing site increases the concentration of platelets (and theoretically of PDGF) by up to 338% (26).

Choukroun's platelet-rich fibrin (PRF), a second-generation platelet concentrate, consists of an intimate assembly of cytokines, glycanic chains and structural glycoproteins enmeshed within a slowly polymerized fibrin network. Beneficial effects of PRF have been studied in various procedures, such as facial plastic surgery (27), a sinus-lift procedure as a sole osteoconductive filling material (28) and multiple gingival recessions treated with a coronally displaced flap (29). PRF has been shown to act as suitable scaffold for culturing human periosteal cells *in vitro*, which may be suitable for bone tissue engineering applications (30).

Recently our study evaluated effectiveness of autologous PRF and autologous PRP in the treatment of mandibular degree II furcation defects compared with open flap debridement (OFD) and both PRP and PRF showed significant improvement in clinical parameters compared to OFD independently and implied their role as a regenerative material in the treatment of furcation defects (31,32).

To our knowledge, no study has reported the clinical comparison of the use of autologous PRF and PRP for treatment of mandibular degree II furcation defects. Thus, the purpose of the present study was to investigate and compare the additional efficacy of autologous PRF and PRP with OFD in the treatment of mandibular degree II furcation defects in comparison to OFD alone.

Material and methods

Subject selection

In this 9 mo follow-up, randomized, double blinded, controlled clinical trial, a total of 42 systemically healthy subjects (22 men and 20 women; mean age: 39.4 years) undergoing periodontal therapy at the Department of Periodontics, Government Dental College and Research Institute, Bangalore, India, were selected. The study was conducted from December 2010 to December 2011. The research protocol was initially submitted to the Institutional Ethical Committee and Review Board of the Government Dental College and Research Institute, Bangalore. After ethical approval, all subjects were verbally informed and written informed consent was collected for participation in the study.

The inclusion criteria for the study was the presence of buccal degree II furcation defects (33,34) in endodontically vital, asymptomatic mandibular molars with a radiolucency in the furcation area on an intraoral periapical radiograph with probing depth (PD) $\geq$ 5 mm and horizontal PD $\geq$ 3 mm after Phase I therapy (scaling and root planing). After re-evaluation of Phase I therapy, patients with any of the following were excluded from the study: (i) systemic illness known to affect the outcomes of periodontal

therapy, such as diabetes mellitus, cardiac diseases, insufficient platelet count (< 200,000/mm^3) or immunocompromised (e.g., HIV individuals; patients taking medications, such as corticosteroids or calcium channel blockers, which are known to interfere with periodontal wound healing; (ii) individuals allergic to medications; (iii) pregnant or lactating women; (iv) patients using tobacco in any form; or (v) individuals with unacceptable oral hygiene [plaque index (PI) > 1.5; 35]. In addition, teeth with interproximal intrabony defects (IBD), gingival recession, endodontic (pulpal) involvement or mobility of tooth $\geq$ Grade II (33) were also excluded.

Presurgical therapy

Before surgery, each patient was given careful instructions on proper oral hygiene measures. A full-mouth supra- and subgingival scaling and root planing procedure was performed under local anesthesia. Six to eight wks following phase I therapy, periodontal evaluation was performed to confirm the desired sites for the study. The selected sites were divided randomly (computer-generated tables) into the control and test groups (PRF or PRP). The control group consisted of sites treated with OFD, i.e., conventional flap surgery, whereas the test group sites were treated with OFD (conventional flap surgery) with autologous PRF or PRP.

One operator (PB) performed all the surgeries while another operator (ARP) performed all the clinical and radiographic measurements without knowledge of the groups. Patients were blinded for allocation to a particular group and treatment.

Clinical and radiographic measurements

The clinical parameters recorded before surgical procedures included site-specific PI, sulcus bleeding index (SBI) (36), PD from the gingival margin, relative vertical clinical attachment level (RVCAL) and horizontal clinical attachment level (RHCAL) along with gingival marginal level (GML) from the apical level of customized acrylic stents with grooves to ensure a reproducible placement of a periodontal probe (UNC-15; Hu-Friedy, Chicago, IL, USA) (for vertical measurement) and probe (Nabers probe; Hu-Friedy) (for horizontal measurements).

All bone defects were evaluated at baseline and 9 mo, postoperatively. For the measurement of bone defect, distance from the furcation fornix to base of the defect was considered. Individually customized bite blocks and parallel angle technique were used to obtain standardized radiographs. For assessment, radiographs were obtained with a scanner (Epson Perfection V700, Epson, Bangalore, India) of 6400 DPI by an evaluator who was masked to the surgical procedure performed in patients. The radiographic bone defect depth was measured by a computer-aided software program (Scion Image Corporation, Frederick, MA, USA) as used previously (37).

Intraexaminer calibration

Intraexaminer calibration was achieved by two examinations of nine patients, 24 h apart before beginning the study. Calibration was accepted if measurements at baseline and 24 h were similar to 1 mm at the 95% level.

Platelet-rich fibrin preparation

The PRF was prepared following the protocol developed by Choukroun *et al.* (38) and used in our previous study (32). Just before surgery, intravenous blood (by venipuncture of the antecubital vein) was collected in three 10 mL sterile tubes without anticoagulant and immediately centrifuged in a centrifugation machine (R-4C, REMI, Mumbai, India) at (approximately 400 ***g***) for 10 min. Blood centrifugation immediately after collection allows the composition of a structured fibrin clot in the middle of the tube, just between the red corpuscles at the bottom and acellular plasma [platelet-poor plasma (PPP)] at the top. PRF was easily separated from the red corpuscle base [preserving a small red blood cell (RBC) layer] using a sterile tweezers and scissors just after removal of PPP and then transferred on to a sterile compress. A stable fibrin membrane was obtained by squeezing serum out of the PRF clot.

Platelet-rich plasma preparation

The PRP was prepared following the protocol developed by Lekovic *et al.* (25) and used in our previous study (31). On the day of surgery, 20 mL blood was drawn from each patient by venipuncture in the antecubital vein. Blood was collected in sterile plastic test tubes that contained citrate phosphate dextrose-adenine (HL Haemopack; Thiruvanathapuram, Kerala, India) as an anticoagulant in the ratio of 2.8 mL citrate phosphate dextrose-adenine to 20 mL of blood. The blood containing test tubes were shaken gently to enhance complete mixing of blood with anticoagulant. Then it was kept at room temperature for a minimum of 45 min to minimize the complement activity. Later, blood-containing test tubes were centrifuged using a refrigerated centrifugal machine at 400 *g* for 10 min, which resulted in separation of three basic fractions: the bottom RBC, middle PRP and top layer of PPP, because of differential densities. Two to three milliliters of the top layer corresponding to the PPP was aspirated with a pipette and collected in a separate sterile plastic tube. The same aspirated PPP was used to obtain autologous thrombin at the time of application. The PRP was collected in conjunction with the top 1–2 mm of the RBC fraction because the latter is also rich in newly synthesized platelets.

Surgical procedure

Intraoral antisepsis was performed with 0.12% chlorhexidine digluconate rinse and iodine solution was used to carry out extraoral antisepsis. Following administration of local anesthesia, buccal and lingual sulcular incisions were made and

mucoperiosteal flaps were reflected. Care was taken to preserve as much interproximal soft tissue as possible. Meticulous defect debridement and root planing were carried out using ultrasonic instruments (EMS V-Dent; Shantou, Guangdong, China) and area-specific curettes (Gracey; Hu-Friedy). No osseous recontouring was carried out.

Autologous PRF of the required size was filled into the furcation defect and two other parts were used as a membrane to cover furcation in the test group. The mucoperiosteal flaps were repositioned and secured in place using 3-0 non-absorbable black silk surgical sutures (Ethicon; Johnson and Johnson, Somerville, NJ, USA). The interrupted or sling sutures were placed. The surgical area was protected and covered with periodontal dressing (Coe-Pak; GC America, Chicago, IL, USA).

PRP gel was prepared according to the method described by Su *et al.* (39). Beads (5 g) and 10% $CaCl_2$ (0.3 mL) were added to PPP (10 mL) to activate coagulation. The mixture was agitated once a minute for 8–10 min at room temperature. The supernatant containing human thrombin was recovered after an additional 10 min. Human thrombin and PRP, in equal quantities from the same blood donor, were mixed to prepare the gel.

Autologous PRP in the gel form of the required size was packed into the defect in the test group. The mucoperiosteal flaps were repositioned and secured in place using 3-0 non-absorbable black silk surgical suture (Ethicon; Johnson and Johnson). The interrupted or sling sutures were placed. The surgical area was protected and covered with periodontal dressing (Coe-Pak; GC America).

Both the autologous PRF and PRP were prepared just before placement in the defect and were not stored, as the success of this techniques entirely depends on the speed of blood collection and transfer to the centrifuge.

Postoperative care

Suitable antibiotics and analgesics (amoxicillin 500 mg four times per day for 5 d and ibuprofen 800 mg three times per day) were prescribed along with chlorhexidine digluconate rinses (0.12%) twice daily for 2 wk. Periodontal dressing and sutures were removed 2 wk postoperatively. Surgical wounds were gently cleansed with 0.12% of chlorhexidine digluconate and subjects were instructed for gentle brushing with a soft toothbrush. Each patient was reinstructed for proper oral hygiene measures at 8 wk postoperatively and examined weekly up to 1 mo after surgery and then at 3 and 9 mo. No subgingival instrumentation was attempted at any of these appointments.

Postsurgical measurements

Soft and hard tissue evaluation was performed 9 mo after surgery. Soft tissue measurements were repeated with previously used acrylic stents. For hard tissue re-evaluation, a second intraoral periapical radiograph of the same study site was carried out and bone defect measurement was reassessed at 9 mo.

Primary and secondary outcome measures

The primary outcome of the study was complete clinical closure of the defect and bone defect fill. The secondary outcomes included RVCAL, RHCAL, PD, GML, PI and SBI.

Statistical analysis

The data were analyzed using statistical software (SPSS version 10.5, SPSS, Chicago, IL, USA). Power calculations were performed before the study was initiated. To achieve 85% power and detect mean differences of the clinical parameters between groups, 20 sites per group were required. The results were averaged (mean standard deviation) for each clinical and radiographical parameter at baseline and 9 mo modified SBI and PI were expressed as absolute and relative counts and comparison was performed using a chi-squared test. Normality assumption of the data was tested using Shapiro–Wilk's W test. The difference between each pair of measurements was then calculated (baseline 9 mo). Paired t-test was applied to assess the statistical significance between time points within each group for clinical and radiographic parameters. ANOVA and *post hoc* Tukey's test were used for intergroup comparison of clinical and radiological parameters. The mean intra-examiner standard deviation of differences in repeated PD measurements and RVCAL and RHCAL measurements were obtained using single passes of measurements with the UNC-15 probe (correlation coefficients between duplicate measurements; $r = 0.95$).

Results

Thirty-seven (72 sites) of 42 subjects completed the study (Fig. 1). All treated cases showed uneventful wound healing. A statistically significant reduction in the PI and modified SBI was observed in all three groups at 9 mo postoperatively ($p \leq 0.001$; Table 1).

Mean values for clinical and radiological parameters at baseline and 9 mo are reported in Table 2 while mean changes in the parameters are reported in Table 3. Both PRF and PRP sites presented with a significantly greater PD reduction (4.29 ± 1.04 mm, 3.92 ± 0.93 mm respectively) than control site (1.58 ± 1.02 mm) at 9 mo postoperatively ($p \leq 0.05$). RVCAL gain was also greater in the PRF (2.87 ± 0.85 mm) and PRP (2.71 ± 1.04 mm) sites as compared to control site (1.37 ± 0.58 mm), also RHCAL gain was statistically significantly greater in both PRF and PRP than the control group (Table 3). PRF ($44.01 \pm 9.98\%$) and PRP ($42.83 \pm 11.15\%$) presented with a significantly greater IBD fill than the control sites ($2.78 \pm 0.68\%$) at 9 mo ($p < 0.001$) (Table 3). There was no significant difference between PRP and PRF groups for any of the clinical or radiological parameters at the end of 9 mo.

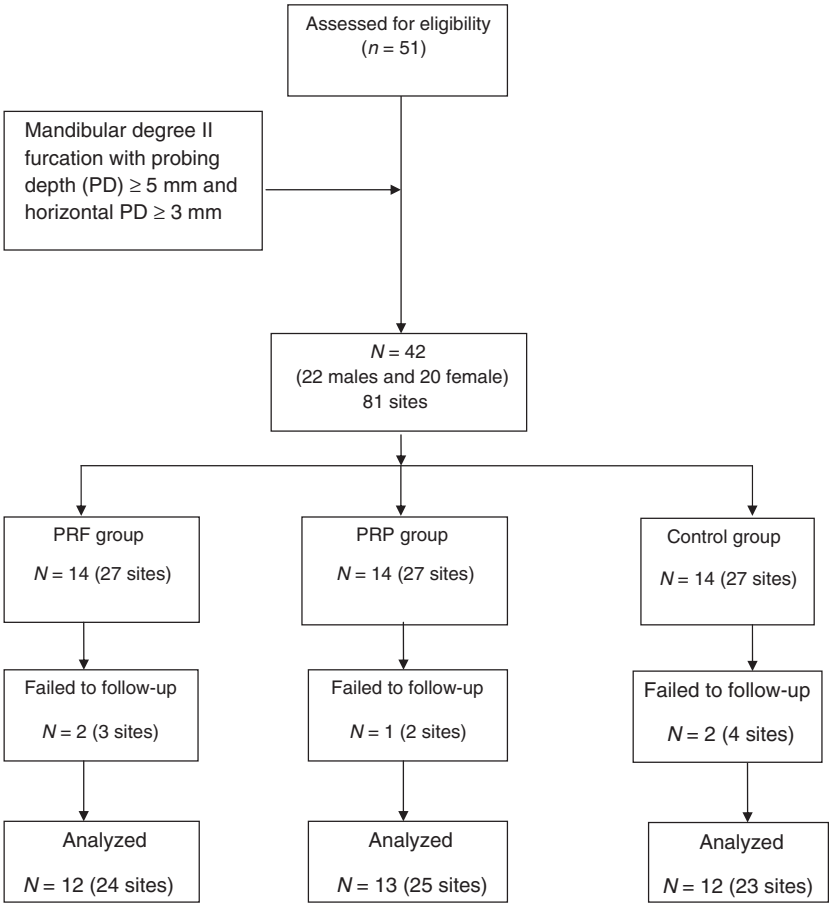

Fig. 1. Study flow chart.

Table 1. Bleeding on probing and plaque index in PRF, PRP and control group at baseline and 9 mo

		BOP index score				PI Score			
Group	Visit	0–0.5	0.6–1.0	1.1–1.5	*p* value	0–0.5	0.6–1.0	1.1–1.5	*p* value
PRF	Baseline	0 (0)	11 (45.83)	13 (54.17)	< 0.001	0 (0)	10 (41.67)	14 (58.33)	< 0.001
n (%)	9 mo	19 (79.17)	5 (20.83)	0 (0)		22 (91.67)	2 (8.33)	0 (0)	
PRP	Baseline	0 (0)	12 (50)	12 (50)	< 0.001	0 (0)	10 41.67	14 (58.33)	< 0.001
n (%)	9 mo	20 (83.33)	4 (16.67)	0 (0)		21 (87.50)	3 (12.50)	0 (0)	
Control	Baseline	0 (0)	10 (41.67)	14 (58.33)	< 0.001	0 (0)	11 (45.83)	13 (54.17)	< 0.001
n (%)	9 mo	20 (83.33)	4 (16.67)	0 (0)		22 (91.67)	2 (8.33)	0 (0)	

BOP, bleeding on probing; PI, plaque index; PRF, platelet-rich fibrin; PRP, platelet-rich plasma.

Discussion

In this study, 37 patients were treated by conventional surgical (OFD) or autologous PRF and autologous PRP techniques. The treatment protocol emphasized the principles of careful soft tissue handling, wound stability and infection control. Controls were treated by OFD only, whereas in the experimental group sites were treated with OFD with autologous PRF or PRP. The results demonstrated that the autologous PRF or PRP resulted in significantly more PD reduction, RHAL and RVCAL gains, and furcation closure rates than traditional open flap instrumentation.

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 histologic evaluation is most accurate, surgical closure of the furcation defect and improvements in PD and CALs serve as suitable and practical outcome measures (40). The current study assesses clinical and

Table 2. Clinical and radiographic parameters in test and control groups at baseline and 9 mo

		PRF		PRP		OFD	
Parameter	Visits	Mean ± SD	*p* value	Mean ± SD	*p* value	Mean ± SD	*p* value
PD	Baseline	7.29 ± 0.95	< 0.001	7.17 ± 1.01	< 0.001	6.87 ± 0.90	< 0.001
	9 mo	3.0 ± 0.51		3.25 ± 0.68		5.29 ± 0.99	
RVCAL	Baseline	7.42 ± 0.78	< 0.001	7.08 ± 0.72	< 0.001	7.32 ± 0.80	< 0.001
	9 mo	4.54 ± 0.51		4.38 ± 0.71		5.92 ± 0.70	
RHCAL	Baseline	8.17 ± 0.82	< 0.001	8.08 ± 0.65	< 0.001	7.96 ± 0.86	< 0.001
	9 mo	5.42 ± 0.72		5.58 ± 0.72		6.87 ± 0.85	
GML (mm)	Baseline	1.58 ± 0.50	0.295	1.54 ± 0.51	0.747	1.62 ± 0.49	0.05
	9 mo	1.41 ± 0.50		1.50 ± 0.51		1.87 ± 0.34	
Bone defect depth (mm)	Baseline	4.18 ± 0.27	< 0.001	4.09 ± 0.30	< 0.001	3.89 ± 0.24	< 0.001
	9 mo	2.32 ± 0.35		2.33 ± 0.43		3.78 ± 0.23	

GML, gingival marginal level; OFD, open flap debridement; PD, probing depth; PRF, platelet-rich fibrin; PRP, platelet-rich plasma; RHCAL, relative horizontal clinical attachment level; RVCAL, relative vertical clinical attachment level.

Table 3. Mean changes in the clinical and radiographic parameters over 9 mo period between the groups

	PRF	PRP	OFD	*p* value	
Mean PD change (mm)	4.29 ± 1.04	3.92 ± 0.93	1.58 ± 1.02	PRF vs. PRP	0.399
				PRF vs. OFD	< 0.001*
				PRP vs. OFD	< 0.001*
Mean RVCAL (mm)	2.87 ± 0.85	2.71 ± 1.04	1.37 ± 0.58	PRF vs. PRP	0.774
				PRF vs. OFD	< 0.001*
				PRP vs. OFD	< 0.001*
Mean RHCAL (mm)	2.75 ± 0.94	2.5 ± 0.83	1.08 ± 0.50	PRF vs. PRP	0.514
				PRF vs. OFD	< 0.001*
				PRP vs. OFD	< 0.001*
Mean gingival marginal level (mm)	0.17 ± 0.76	0.04 ± 0.62	-0.25 ± 0.61	PRF vs. PRP	0.794
				PRF vs. OFD	0.085
				PRP vs. OFD	0.291
Mean bone defect depth (mm)	1.85 ± 0.49	1.77 ± 0.52	0.11 ± 0.03	PRF vs. PRP	0.747
				PRF vs. OFD	< 0.001*
				PRP vs. OFD	< 0.001*
Bone defect fill (%)	44.01 ± 9.98	42.83 ± 11.15	2.78 ± 0.68	PRF vs. PRP	0.886
				PRF vs. OFD	< 0.001*
				PRP vs. OFD	< 0.001*

OFD, open flap debridement; PD, probing depth; PRF, platelet-rich fibrin; PRP, platelet-rich plasma; RHCAL, relative horizontal clinical attachment level; RVCAL, relative vertical clinical attachment level. *denotes statistically significant.

radiographic parameters to evaluate the effect of autologous PRF and autologous PRP on soft and hard tissue. The uneventful healing in patients is in agreement with previous studies (31,32), thus supporting the excellent properties of autologous PRF and PRP to enhance periodontal wound healing.

It has been found that PRF consists of a fibrin matrix polymerized in a tetramolecular structure; the incorporation of platelets, leukocyte, and cytokines; and circulating stem cells (41). Slow fibrin polymerization during PRF processing leads to the intrinsic incorporation of platelet cytokines and glycanic chains in the fibrin meshes. In addition, PRF slows down the blood activation process, which could induce an increased leukocyte degranulation and cytokine from proinflammatory mediators, such as interleukin (IL)-1β, IL-6, and tumor necrosis factor-α, to anti-inflammatory cytokines, such as IL-4. It is also found that PRF organizes as a dense fibrin scaffold with a high number of leukocytes concentrated in one part of the clot (42), with a specific slow release of growth factors (e.g., transforming growth factor-1β, PDGF-AB, and vascular endothelial growth factor) and glycoproteins (e.g., thrombospondin-1) during < 7 d (43).

In addition, it was suggested that PRP contains high concentrations of several growth factors such as PDGF and transforming growth factor-β, which may strongly modulate the regeneration process. Data from *in vitro* studies have shown that PRP stimulates the proliferation of periodontal ligament and osteoblastic cells while, at the same time, epithelial cell proliferation is inhibited (44). It was also speculated that due to its fibrinogen content, PRP reacts with thrombin and induces fibrin clot formation, which in turn is capable of upregulating collagen synthesis in the extracellular matrix and provides a favorable scaffold for cellular

migration and adhesion (45). The fibrin component of PRP gel not only works as a hemostatic agent aiding in stabilization of the graft material and blood clot, but also adheres to the root surface and may impede the apical migration of epithelial cells and connective tissue cells from the flap (46), and thus PRP may exert guided tissue regeneration-like effect (may act as a barrier to cover bone and periodontal ligament, temporarily separating them from the gingival epithelium) in the treated defects.

To our knowledge, there are no studies reporting the comparison of autologous PRF or autologous PRP in the treatment of furcation defects. Therefore, a direct comparison with other studies is not possible. PD reduction, RVCAL and RHAL gains, GML change, IBD depth reduction and percentage IBD fill found in the current study are similar to the mean changes found in a previous study on PRF in furcation defects; however, for PRP slightly better results were obtained as compared to a previous study (31,32). In the present study, a significant reduction in PD and RVCAL and RHAL gain were found in all three groups when compared with baseline and 9 mo. However, there was more PD reduction (4.29 ± 1.04) in the PRF-treated and PRP-treated groups (3.92 ± 0.93) compared with the subjects treated with conventional periodontal flap surgery alone (1.58 ± 1.02). The present study also reflects that the percentage of IBD fill in the PRF group ($44.01 \pm 9.98\%$) and PRP group ($42.83 \pm 11.15\%$) is higher than the conventionally treated subjects, supporting the significance and advantage of various growth factors present in PRF and PRP that may accelerate soft and hard tissue healing (38,47). Although, there was no significant difference between the PRP and PRF group for any clinical or radiological parameter, there are many advantages of using PRF, a second-generation platelet concentrate, over PRP. First, PRF can be squeezed to form a membrane and can be used as fibrin bandage serving as a matrix to accelerate the healing of wound edges (48,49). Second, PRF differs from other commercially available PRP systems in that it does not use bovine thrombin or other exogenous activators in the preparation process. The PRF preparation process creates a gel-like matrix that contains high concentrations of non-activated, functional, intact platelets, contained within a fibrin matrix, which release a relatively constant concentration of growth factors over a period of 7 d (50). Third, the chair-side preparation of PRF is quite easy and fast, and simplified processing with no artificial biochemical modification as in PRP, which takes up more time (51). Fourth, this produces an inexpensive autologous fibrin membrane in approximately 1 min and, hence, no cost for membrane and bone graft making periodontal regeneration techniques affordable to patients.

Conclusion

To our knowledge, this study is the first prospective, randomized, double-blinded and controlled pivotal clinical trial reported to date assessing and comparing two promising periodontal regeneration techniques. The study demonstrated that in the use of autologous PRF or PRP, both were effective in the treatment of furcation defects with uneventful healing of sites. In addition, there was no significant difference between the two. Treatment with autologous PRF or PRP stimulated a significant reduction in PD and increase in the RVCAL and RHAL gain, and bone fill as compared to OFD at 9 mo. As PRF and PRP are autologous preparations from a patient's own blood, it decreases the cost of regeneration therapy and is less time consuming for both the surgeon and patient. Again placement of PRF does not require a skill and it is less technique sensitive than guided tissue regeneration and bone graft placement. However, long-term, randomized, controlled clinical trials will be needed to know its effect over other treatment modalities.

Conflict of interest

The authors report no potential conflict of interests.

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