Original Research

Microneedling with injectable platelet-rich fibrin as an adjunct to nonsurgical periodontal therapy in periodontitis patients: A split-mouth clinical trial

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Abstract:

Aim and Background: Contemporary minimally invasive periodontal approaches focus on conserving tissues and minimizing postoperative discomfort. Microneedling (MN), a technique that induces controlled collagen synthesis, has shown potential in stimulating soft-tissue regeneration and improving tissue volume. When used in addition to injectable platelet-rich fibrin (I-PRF), a biologically rich blood concentrate, the regenerative outcome may be further enhanced. In patients with chronic periodontitis, the current study sought to compare the clinical results of MN with I-PRF versus I-PRF alone after nonsurgical periodontal therapy. **Materials and Methods:** A randomized split-mouth clinical design was adopted, including 120 periodontal sites with Stage II periodontitis. Following initial therapy, the test sites (n = 60) underwent MN followed by I-PRF administration, while contralateral control sites (n = 60) received only I-PRF injections. Clinical parameters – probing pocket depth (PPD), gingival thickness (GT), keratinized tissue width (KTW), and relative attachment level (RAL) – were assessed at baseline, 1 month, and 3 months after treatment. **Results:** All recorded metrics showed a considerable improvement with both treatment regimens (P < 0.001). In the test and control groups, the mean PPD decreased to 3.38 ± 0.42 mm and 3.65 ± 0.57 mm, respectively. Correspondingly, the mean RAL in the test group showed a reduction from 4.91 ± 0.77 mm at baseline to 3.66 ± 0.65 mm after treatment, while the control group exhibited an improvement to 3.87 ± 0.69 mm. After 3 months, the test group demonstrated a mean gain in GT of 0.60 ± 0.31 mm and an increase in KTW of 0.80 ± 0.14 mm, indicating a larger enhancement in periodontal phenotype (P < 0.001). **Conclusion:** MN used adjunctively with I-PRF demonstrated superior outcomes in soft-tissue healing, phenotype improvement, and clinical attachment gain, highlighting its promise as a minimally invasive regenerative modality in nonsurgical periodontal therapy.

Key words:

Collagen induction, injectable platelet-rich fibrin, microneedling, periodontal phenotype, periodontitis

INTRODUCTION

Periodontal disease is a long-standing inflammatory disorder marked by the slow deterioration of the periodontal supporting apparatus, ultimately leading to mobility and eventual tooth loss. Both nonsurgical and surgical periodontal interventions are intended to suppress inflammation, eliminate microbial infection, and arrest disease progression. Complete regeneration of the missing periodontal attachment apparatus remains a major clinical difficulty despite these strategies.[1]

The management of periodontitis primarily relies on nonsurgical periodontal therapy (NSPT), which is widely recognized as the first-line and fundamental mode of intervention for disease control. The success and long-term stability of such therapy are influenced by several biological and anatomical factors, among which the periodontal phenotype – defined by keratinized tissue width (KTW) and gingival thickness (GT) – is essential for preserving periodontal health, function, and esthetics.[2]

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Conventional mucogingival augmentation techniques have been used to enhance soft-tissue dimensions and reinforce the dentogingival complex; however, these methods often involve surgical complexity and patient discomfort. This has led to growing clinical interest in less invasive regenerative modalities.[3]

A recent development in platelet concentrates, injectable platelet-rich fibrin (I-PRF), is made by low-speed centrifugation, which preserves a rich mixture of platelets, leukocytes, and growth factors while maintaining a fluid consistency for a brief working window of approximately 15 min.[4] Its composition allows superior biocompatibility and simple handling during chairside application. Upon activation, I-PRF undergoes fibrin polymerization and promotes the migration of osteoblasts and other periodontal cells essential for biological restoration and regeneration of soft and hard tissues as well as the proliferation of fibroblasts by stimulating the release of important macromolecules including collagen type I and transforming growth factor-beta.[4,5]

Microneedling (MN), a minimally invasive procedure designed to stimulate collagen synthesis through controlled microinjury, has evolved as a minimally invasive approach capable of boosting the regeneration benefits of biomaterials like I-PRF. Microneedling was first described in the dermatologic literature in 1995 for the treatment of atrophic scars. The technique induces controlled micro-injuries that activate cascades of growth-factor release, neocollagenesis, and neoangiogenesis, resulting in improved tissue remodeling. In oral applications, microneedling enhances vascular perfusion and facilitates deeper penetration and sustained retention of bioactive agents such as I-PRF.[4-6]

Although extensively applied in dermatology for skin rejuvenation and scar management, its application in dentistry has recently gained attention for improving soft-tissue volume and modifying periodontal phenotype. Ozsagir *et al*. (2020)[7] were among the first to report that the combination of MN and I-PRF yielded notable gains in GT in patients presenting with a thin periodontal biotype.[6,8]

This study aimed to evaluate the synergistic potential of MN and I-PRF in promoting GT and increasing the width of keratinized tissue and clinical attachment levels in patients with chronic periodontitis after NSPT; the current randomized split-mouth clinical study was created, building on this new data. The study proposes a biologically driven, minimally invasive therapeutic approach for optimizing soft-tissue outcomes and improving the periodontal phenotype.

MATERIALS AND METHODS

This randomized split-mouth clinical trial was designed to compare the therapeutic outcomes of MN combined with I-PRF versus I-PRF alone in individuals diagnosed with periodontitis. Ethical clearance for the study was obtained from the Institutional Review Board, and the protocol was prospectively registered with the Clinical Trials Registry of India (CTRI/REF/2022/07/056797). All participants provided written informed consent before enrollment. Eligible subjects were recruited from the Department of Periodontics following application of the inclusion and exclusion criteria.

Using a coin toss procedure, 60 patients (120 sites) who satisfied the inclusion and exclusion criteria were chosen and randomized to test and control groups [Figure 1].

Inclusion criteria

1. People with Stage I or II, Grade A or B periodontitis (bone loss/age ratio = 0.25–1.0) who are otherwise healthy
2. Nonsmokers or light smokers (<10 cigarettes/day)
3. Interdental clinical attachment loss (CAL) <4 mm
4. Only the coronal third (less than 30%) shows radiographic horizontal bone loss
5. Maximum probing pocket depth (PPD) ≤5 mm.

Exclusion criteria

1. History of periodontal surgery within the previous 12 months
2. Presence of systemic or local disorders affecting periodontal health
3. Current intake of medications known to alter the gingival condition
4. Pregnancy or lactation
5. History of tobacco chewing or heavy smoking
6. Known carriers of blood-borne infections.

Clinical parameters

Clinical assessments were performed at baseline, 1 month, and 3 months posttreatment. The following parameters were evaluated:

1. Using a UNC-15 periodontal probe, the PPD is measured at six locations per tooth
2. Relative attachment level (RAL): Determined from a customized acrylic stent reference point to the gingival margin, combined with the PPD measurement
3. Plaque Index (PI): Recorded according to the index system described by Silness and Löe.[9]
4. Gingival Index (GI): Evaluated using the gingival assessment method proposed by Löe.[10]
5. KTW: The width of keratinized tissue was determined using a calibrated periodontal probe fitted with a silicone stopper. Measurements were taken from the mucogingival junction to the free gingival margin, corresponding to the lower edge of the silicone marker.[11]
6. GT: GT was assessed using a No. 15 endodontic spreader equipped with a 3-mm silicone stop. The instrument was inserted perpendicularly into the gingiva approximately 1.5 mm apical to the gingival margin until gentle tissue resistance was encountered. The distance between the spreader tip and the silicone stopper represented the GT [Figure 2a and b]
7. Treatment protocol: All eligible participants were provided with comprehensive oral hygiene instructions and motivation to maintain plaque control before the commencement of the study. Baseline clinical parameters were recorded, followed by subgingival debridement using ultrasonic scalers and Gracey curettes under local anesthesia where required.

Following an initial healing phase of 2 weeks, the designated interventions were performed at the test and control sites as described below.

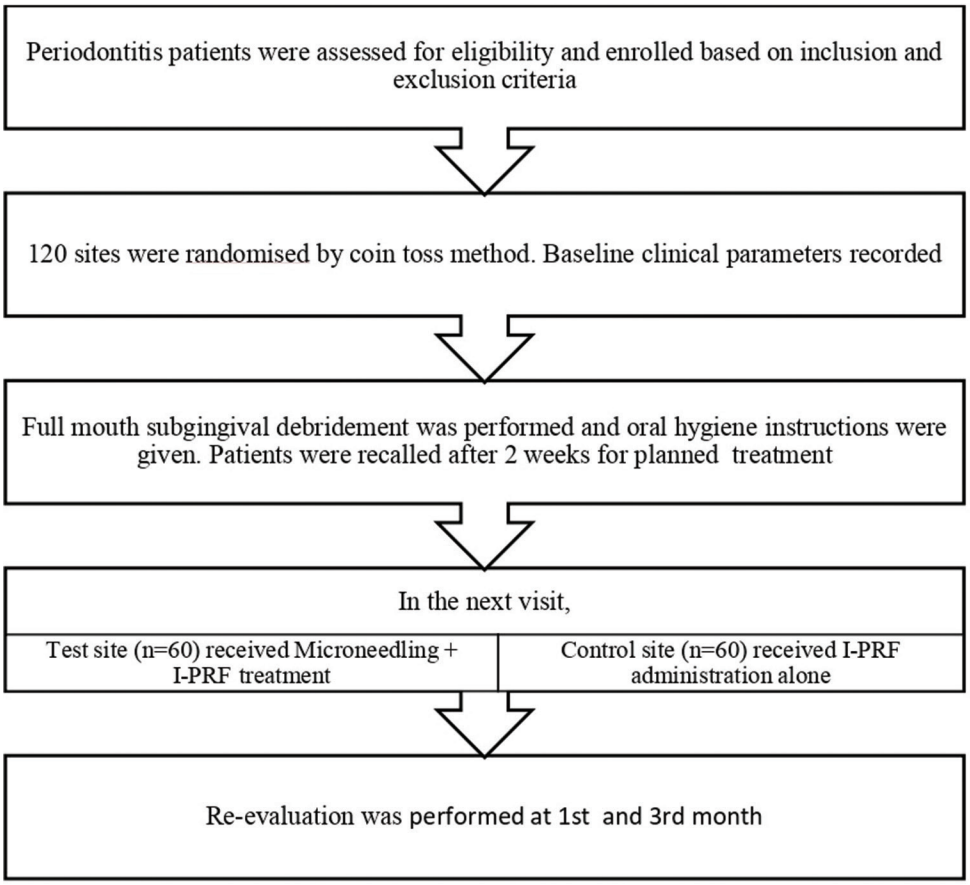

Figure 1: Study design flowchart showing participant enrollment, randomization, interventions, and follow-up schedule. I-PRF: Injectable platelet-rich fibrin

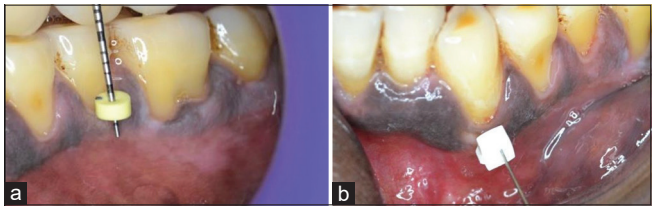

Figure 2: Measurement of gingival parameters (a) keratinized tissue width; (b) gingival tissue thickness

Injectable platelet-rich fibrin preparation

Venous blood samples were drawn into two sterile 5 ml tubes without the use of any anticoagulant. The collected samples were immediately centrifuged at 700 rpm (approximately 60 g) for 3 min to obtain the I-PRF fraction. The upper yellow fluid layer obtained postcentrifugation was carefully aspirated as I-PRF. This was immediately administered at the control sites using insulin syringes. Injections were given subgingivally at four standardized locations – buccal, lingual, mesial, and distal – and additionally along the mucogingival junction. Visible blanching of the tissue was considered a clinical indicator of proper platelet concentrate delivery.

Microneedling procedure (test sites)

For the test sites, MN was performed using Sujok microneedles (0.18 mm × 7 mm) inserted perpendicularly into the demarcated gingival area up to the periosteal level. Multiple microchannels were created as distance from the gingival edge to the pocket base on both buccal/labial and lingual/palatal surfaces, adjusted according to tissue area. The procedure was continued until pinpoint bleeding appeared, signifying adequate stimulation. Immediately afterward, I-PRF was injected subgingivally following the same protocol applied to the control group [Figure 3a-c].

All individuals received standard postoperative instructions. All parameters were clinically reassessed at 1 and 3 months after the operation [Figure 4].

RESULTS

All statistical analyses were performed using IBM SPSS Statistics software, version 25.0 (IBM Corp., Armonk, NY, USA). Changes in clinical parameters – namely, PI, GI, KTW, GT, PPD, and RAL – were evaluated over different time intervals using the Friedman test. Intergroup and intragroup comparisons were carried out through independent *t*-tests and repeated-measures ANOVA, respectively. $P < 0.005$ was considered indicative of statistical significance.

Both the test and control groups exhibited a significant reduction in plaque accumulation and gingival inflammation over time ($P < 0.001$). Improvements observed between baseline, 1 month, and 3 months reflected effective plaque control and a sustained decrease in gingival inflammation following therapy.

Intergroup comparison [Table 1]:
Independent *t*-test analysis revealed that the test group (MN + I-PRF) demonstrated greater gains in both keratinized tissue width (KTW) and gingival thickness (GT) compared to the control group (I-PRF alone).
KTW gain: 1.0 mm at 1 month and 0.8 mm at 3 months
GT gain: 0.9 mm at 1 month and 0.6 mm at 3 months

These findings indicate superior soft-tissue enhancement in the MN-treated sites [Table 1].

Intragroup comparison [Table 2]:
Both KTW and GT showed statistically significant improvements within each treatment group from baseline to 1-month and 3-month intervals.

Test group:
Mean KTW increased to 2.87 ± 0.57 mm at 1 month and 2.66 ± 0.52 mm at 3 months from baseline ($P < 0.001$).

GT improved from 1.84 ± 0.73 mm at baseline to 2.71 ± 0.40 mm at 1 month and 2.41 ± 0.44 mm at 3 months.

Control group:
Mean GT increased to 2.50 ± 0.41 mm at 1 month and 2.20 ± 0.47 mm at 3 months ($P < 0.001$ across intervals).

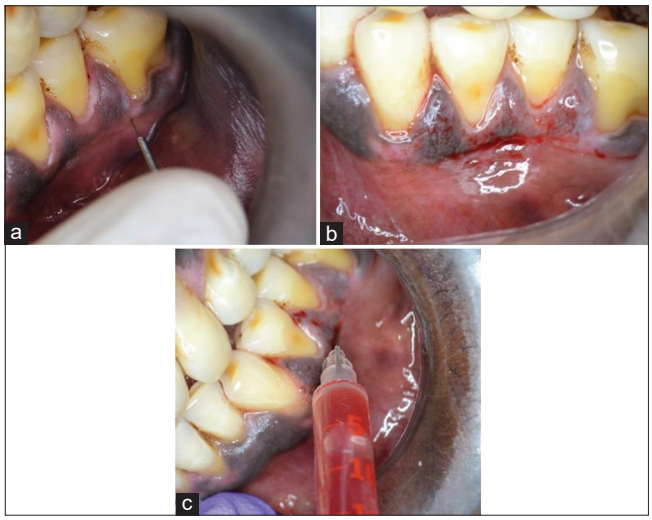

Figure 3: Clinical procedure of microneedling and injectable platelet-rich fibrin (I-PRF) application: (a) microneedling performed in the test site; (b) pinpoint bleeding indicating completion; and (c) subgingival injection of I-PRF

These intragroup changes confirm progressive soft-tissue improvement in both groups over time [Table 2].

Both intervention approaches produced a statistically significant decrease in PPD throughout the study period ($P < 0.001$).

1. In the test group, the mean PPD reduced from 4.63 ± 0.81 mm at baseline to 3.65 ± 0.57 mm after 3 months
2. Similarly, the control group exhibited a decline in mean PPD, reaching 4.08 ± 0.64 mm at the 3-month evaluation.

The combination therapy group (MN + I-PRF) exhibited a greater reduction in PPD, with mean differences of 1.3 ± 0.4 mm at 1 month and 1.0 ± 0.2 mm at 3 months compared to the control group. These differences were statistically significant ($P = 0.009$ and $P = 0.001$, respectively). Likewise, both treatment groups demonstrated a notable improvement in RAL over the evaluation period. In the test group, the mean RAL improved from 4.91 ± 0.77 mm at baseline to 3.66 ± 0.65 mm at 1 month and 3.87 ± 0.69 mm at 3 months ($P < 0.001$).

The control group also showed improvement, though the extent of attachment gain was comparatively lower.

Overall, both intragroup and intergroup analyses confirmed that the combination therapy achieved superior outcomes in GT, KTW, and clinical attachment gain relative to I-PRF alone [Tables 1 and 2 and Figures 3 and 4].

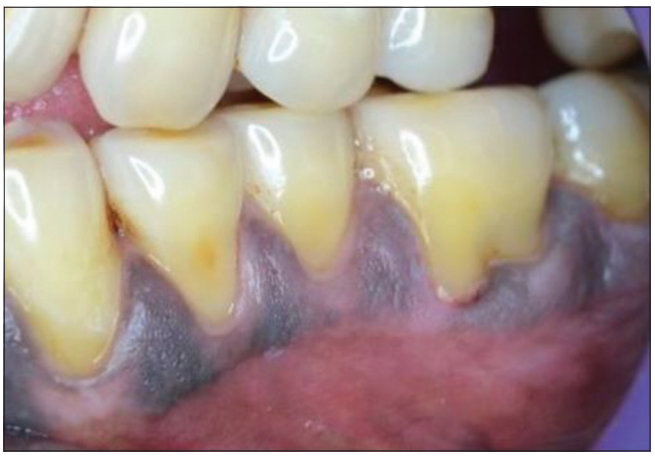

Figure 4: Three-month postoperative view showing improvement in gingival thickness and keratinized tissue width at the treated site

Table 1: Intergroup comparison of keratinized tissue width, gingival thickness, pocket depth, and relative attachment level between microneedling + injectable platelet-rich fibrin and injectable platelet-rich fibrin alone at different time points

Time interval	Intergroup comparison							
	KTW		GT		PD		RAL	
	Mean±SD	*P*	Mean±SD	*P*	Mean±SD	*P*	Mean±SD	*P*
Baseline MN + I-PRF	1.807±0.663	0.44	1.84±0.725	0.417	4.628±0.805	0.000*	4.905±0.771	0.000*
I-PRF	1.868±0.605		1.744±0.65		4.617±0.777		4.715±0.531	
1 month MN + I-PRF	2.865±0.568	0.001*	2.717±0.402	0.014*	3.388±0.414	0.009*	3.657±0.653	0.000*
I-PRF	2.647±0.517		2.5±0.419		3.615±0.551		3.81±0.64	
3 months MN + I-PRF	2.663±0.524	0.000*	2.41±0.439	0.000*	3.65±0.571	0.001*	3.867±0.693	0.000*
I-PRF	2.333±0.507		2.197±0.465		4.08±0.636		4.118±0.606	

P<0.05 considered statistically significant. Intergroup comparison performed using Independent *t*-test. KTW – Keratinized tissue width; GT – Gingival thickness; PD – Probing depth; RAL – Relative attachment level; MN – Microneedling; I-PRF – Injectable platelet-rich fibrin; SD – Standard deviation; *P* – Probability value

Table 2: Intragroup comparison of mean keratinized tissue width, gingival thickness, pocket depth, and relative attachment level at baseline, 1 month, and 3 months in microneedling + injectable platelet-rich fibrin and injectable platelet-rich fibrin alone groups

Time intervals	KTW, mean±SD	GT, mean±SD	PD, mean±SD	RAL, mean±SD	*P*
MN + I-PRF					
Baseline	1.807±0.6634	1.84±0.7255	4.628±0.8053	4.905±0.7713	0.000*
1 month	2.865±0.5689	2.717±0.4026	3.388±0.4147	3.657±0.6537	
3 months	2.663±0.5249	2.41±0.4397	3.65±0.5716	3.867±0.6935	
I-PRF					
Baseline	1.868±0.6052	1.744±0.6509	4.617±0.7779	4.715±0.5316	0.000*
1 month	2.647±0.5178	2.5±0.4194	3.615±0.5517	3.81±0.6402	
3 months	2.333±0.5074	2.197±0.4657	4.08±0.6365	4.118±0.6064	

P<0.05 considered statistically significant. KTW – Keratinized tissue width; GT – Gingival thickness; PD – Probing depth; RAL – Relative attachment level; MN – Microneedling; I-PRF – Injectable platelet-rich fibrin; SD – Standard deviation; *P* – Probability value

DISCUSSION

This is the first clinical study that we are aware of that evaluates the adjunctive use of MN therapy in conjunction with I-PRF among patients with chronic periodontitis. The present findings demonstrate that combining MN with I-PRF significantly enhanced the periodontal phenotype and yielded superior improvements in clinical attachment level (CAL) compared with I-PRF alone.

The mean GI (1.43) and PI (1.55) in this study were like those reported by Bhansali[12] who also saw similar results after traditional NSPT. Previous studies have confirmed the therapeutic benefits of I-PRF as a scaling and root planing (SRP) adjuvant. I-PRF application was shown to significantly promote soft-tissue healing and reduce periodontal inflammation in studies by Albonni *et al*. and Vučković *et al*.[13,14] In line with these findings, the present investigation demonstrated statistically significant improvements in gingival margin level (GML) and PPD between groups ($P = 0.040$ and $P = 0.006$, respectively), as well as a significant decrease in mean CAL in the test group, from 1.97 ± 0.75 mm to 1.07 ± 0.44 mm.

The control group also demonstrated favorable changes in periodontal parameters. The RAL decreased from 4.72 ± 0.53 mm at baseline to 3.81 ± 0.64 mm at 1 month and 4.12 ± 0.61 mm at 3 months. Likewise, PPD reduced from 4.62 ± 0.78 mm to 4.08 ± 0.64 mm at 3 months ($P < 0.001$), reaffirming the clinical efficacy of SRP as a standalone nonsurgical approach.

The outcomes of this investigation agree with the findings of Kavi *et al*. which suggested that GT increased by 0.54 ± 0.085 mm after receiving mucogingival therapy with I-PRF assistance.[15] The regeneration potential of I-PRF in soft-tissue augmentation was confirmed in the current investigation when the test group's GT improved by 0.46 mm after 3 months. Histologic examinations by Hou *et al*.[16] have shown that MN stimulates neocollagenesis and neoangiogenesis, supporting its role as a minimally invasive, cost-effective modality for soft-tissue regeneration.[16,17]

A substantial body of dermatological research has consistently highlighted MN as an effective technique for promoting collagen remodeling and skin rejuvenation in treating pigmentation problems, acne scarring, and skin laxity, as reported by Alster and Graham[17] and Ramaut *et al*.[18] The biological mechanism underlying its effectiveness is attributed to controlled microinjury, which initiates a cascade of wound-healing processes involving fibroblast activation, collagen synthesis, and extracellular matrix remodeling. When applied to periodontal tissues, these processes may explain the improved healing response and tissue augmentation achieved in the MN + I-PRF group in the current study.[19,20]

The conceptual framework of this study draws upon the work of Ozsagir *et al*.[7], who first reported that the combination of MN with I-PRF led to a significant increase in GT among individuals presenting with a thin periodontal biotype.[7] Their results – demonstrating an increase in GT from 0.40 ± 0.14 mm to 0.66 ± 0.12 mm – are consistent with the findings of the present investigation, reinforcing the concept that MN can function as an effective, nonsurgical adjunct for soft-tissue enhancement.

In the current study, KTW increased to 2.87 ± 0.57 mm and 2.66 ± 0.52 mm at 1 and 3 months, respectively. In contrast, after a baseline of 1.84 ± 0.73 mm, GT increased to 2.72 ± 0.71 mm at 1 month and 2.41 ± 0.44 mm at 3 months. There was statistical significance in these improvements ($P < 0.001$).

A minor decrease in KTW and GT values between 1 and 3 months was noted; however, the difference did not reach statistical significance. This subtle reduction may reflect a phase of tissue remodeling or minor regression during the maturation phase of soft-tissue healing.

These observations highlight the potential of MN combined with I-PRF as a promising biologically driven approach for enhancing soft-tissue parameters. Nonetheless, they also emphasize the necessity for long-term longitudinal studies to validate the durability and stability of these clinical outcomes over extended follow-up periods.

Limitations and future directions

This study has a few limitations that merit consideration. The sample size was modest, and the follow-up period of 3 months may not adequately capture the long-term stability of treatment outcomes. Moreover, the absence of radiographic evaluation and histologic examination restricted the assessment to clinical parameters alone. Another limitation was the nonstandardized monitoring of plaque control, which could have influenced soft-tissue healing dynamics.

Future studies should include larger cohorts, extended observation periods, and integrated radiographic and histologic

analyses to validate the long-term regenerative potential of MN combined with I-PRF. In addition, incorporating biochemical markers and advanced imaging modalities may provide deeper insight into the cellular and molecular mechanisms underlying periodontal tissue repair and regeneration.

CONCLUSION

The combination of I-PRF and MN was demonstrated to be a useful supplement to NSPT, within the parameters of this study. The synergistic approach accelerated wound healing, improved the periodontal phenotype, and achieved greater clinical attachment gain compared with I-PRF alone. This minimally invasive, biologically driven technique holds potential as an effective therapeutic option for patients with periodontitis, particularly in cases requiring soft-tissue enhancement and phenotype correction.

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Conflicts of interest
There are no conflicts of interest.

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