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ADJUNCTIVE AUTOLOGOUS PLATELET-RICH PLASMA IN PERIODONTITIS TREATMENT: CLINICAL EVIDENCE, BIOLOGICAL RATIONALE, AND A STEPWISE DECISION FRAMEWORK

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Abstract

Background: Periodontitis is a biofilm-mediated inflammatory disease characterized by progressive loss of periodontal support. Autologous platelet concentrates, including platelet-rich plasma (PRP) and injectable platelet-rich fibrin (i-PRF), have been investigated as adjuncts to conventional therapy, but heterogeneity in preparation complicates interpretation. Objective: To critically assess the biological rationale, clinical effectiveness, and practical limitations of adjunctive platelet-concentrate treatment in periodontitis. Methods: A focused narrative synthesis was conducted using published periodontal guidelines, systematic reviews, and randomized-trial meta-analyses. Evidence was organized around probing pocket depth (PPD), clinical attachment level (CAL), bleeding on probing (BoP), biomaterial characteristics, and the position of adjunctive treatment within staged periodontal care. Results: A published 2025 meta-analysis of ten studies found additional PPD reductions of 0.62 mm (95% CI, 0.22–1.03) and CAL gains of 0.77 mm (95% CI, 0.37–1.18) at 6–12 weeks with autologous platelet concentrates as a class, compared with nonsurgical periodontal therapy alone. At six months, the corresponding improvements were 0.61 and 1.14 mm. However, high heterogeneity and very low certainty of evidence prevent attribution of these estimates to any single injectable PRP protocol. Conclusion: Platelet-derived adjuncts may offer modest clinical benefits in selected patients, but they do not replace biofilm control, risk-factor modification, or periodontal maintenance. Standardized preparation, adequate controls, and longer follow-up are essential before routine adoption.

Keywords: periodontitis; plasmolifting; platelet-rich plasma; injectable platelet-rich fibrin; autologous platelet concentrates; probing pocket depth; clinical attachment level; periodontal regeneration.

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Introduction

Periodontitis is a chronic inflammatory condition initiated and sustained by a dysbiotic subgingival biofilm and modified by the host immune response. Progressive destruction of the periodontal ligament and alveolar bone manifests as clinical attachment loss, periodontal pockets, and radiographic bone changes. The 2018 classification incorporates staging and grading to characterize severity, therapeutic complexity, progression risk, and relevant modifying factors such as smoking and poorly controlled diabetes [1]. Modern periodontal care therefore aims not simply to suppress signs of inflammation but to interrupt the causal disease process and maintain stable function over time.

The European Federation of Periodontology (EFP) S3 guideline recommends a stepwise approach beginning with patient education, effective plaque control, and management of risk factors, followed by supra- and subgingival instrumentation, re-evaluation, and additional treatment where residual disease persists [2]. Autologous platelet preparations have attracted interest because they may release platelet-associated growth factors and provide a supportive environment for wound healing. In clinical discussions, the term “plasmolifting” is often applied to injection-based autologous plasma treatments; however, it does not specify a universally standardized preparation. For scientific reporting, PRP, PRF, and i-PRF must be distinguished according to preparation and final product [3,4].

This article aims to synthesize the available evidence on platelet-derived adjuncts in nonsurgical periodontitis management, identify what the reported clinical effects do—and do not—establish, and propose a clinically responsible evaluation framework for future studies of injection-based PRP protocols.

Literature Review

Autologous platelet concentrates are obtained from a patient’s own blood and differ in centrifugation parameters, cellular composition, fibrin architecture, anticoagulant use, and whether the final material is injected or placed as a clot. Conventional PRP typically involves a platelet-enriched plasma fraction, whereas PRF refers to a fibrin-based concentrate and i-PRF is a fluid formulation used before fibrin polymerization. Consequently, the biological properties of one formulation cannot be assumed to be identical to those of another. Platelet-associated mediators may influence chemotaxis, angiogenesis, fibroblast activity, extracellular-matrix remodeling, and early tissue repair, but biological plausibility alone cannot establish durable periodontal regeneration [3,4].

Niemczyk and colleagues reviewed PRP and injectable PRF in nonsurgical periodontitis therapy and emphasized the emerging but heterogeneous clinical evidence [3]. A subsequent systematic review and meta-analysis by Lipovec and colleagues quantitatively synthesized ten studies from a larger evidence base and found statistically favorable pooled results for PPD and CAL following adjunctive autologous platelet concentrates [4]. Importantly, the pooled analysis included more than one concentrate type, while between-study heterogeneity was high and certainty of evidence was very low. Thus, the average effect provides a hypothesis-generating estimate for the wider intervention class; it does

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not validate a specific commercial plasmolifting regimen or demonstrate histological regeneration of periodontal ligament, cementum, or alveolar bone.

The EFP guideline positions successful plaque and calculus control, risk modification, periodontal reassessment, and supportive care at the center of treatment [2]. This sequence matters when evaluating biological adjuncts: a reduction in pocket depth may reflect resolution of inflammation and soft-tissue shrinkage, whereas true regeneration requires evidence of new functional attachment. Similarly, improved bleeding scores can reflect effective plaque control irrespective of the platelet product. Comparator selection, examiner calibration, baseline pocket depth, smoking status, glycemic control, and follow-up duration therefore substantially influence the interpretation of small additional improvements [1–4].

Materials and Methods

This manuscript is a focused narrative evidence synthesis, not a report of a newly conducted clinical trial. Its evidence base comprises the 2018 periodontal classification framework, the 2020 EFP S3 treatment guideline, the 2024 systematic review of PRP and i-PRF, and the 2025 meta-analysis of autologous platelet concentrates as adjuncts to nonsurgical periodontal therapy [1–4]. Studies were interpreted according to intervention identity, comparators, endpoints, follow-up duration, and methodological limitations. No unpublished patient measurements, author-generated treatment outcomes, or statistical tests were introduced.

The prespecified interpretive outcomes were PPD reduction, CAL gain, and BoP reduction. Effect estimates and 95% confidence intervals were extracted from the published meta-analysis rather than independently recalculated. The figures were newly prepared for this manuscript from reported aggregate estimates; they are not images of original patients or reproductions of published figures. Because the available data concern multiple platelet-concentrate formulations, the synthesis does not pool or extrapolate a distinct effect size for plasmolifting injections alone. A clinical decision pathway was constructed as an educational synthesis of the EFP treatment sequence and evidence limitations.

Results

The strongest quantitative synthesis identified for this focused review included ten papers in the meta-analysis, with 270 participants in platelet-concentrate groups and 230 in comparator groups. For a single adjunctive application at 6–12 weeks, the pooled between-group advantage was 0.62 mm for PPD reduction (95% CI, 0.22–1.03) and 0.77 mm for CAL gain (95% CI, 0.37–1.18). At six months, the advantages were 0.61 mm (95% CI, 0.09–1.13) and 1.14 mm (95% CI, 0.34–1.94), respectively. BoP reduction was not significantly different at 6–12 weeks. These values represent published aggregate results for the autologous platelet-concentrate category and not findings from patients treated by the present author [4].

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Table 1. Published pooled incremental clinical effects of autologous platelet concentrates versus nonsurgical periodontal therapy alone [4].

Outcome	Follow-up	Additional benefit (mm)	95% CI (mm)
PPD reduction	6–12 weeks	0.62	0.22–1.03
CAL gain	6–12 weeks	0.77	0.37–1.18
PPD reduction	6 months	0.61	0.09–1.13
CAL gain	6 months	1.14	0.34–1.94

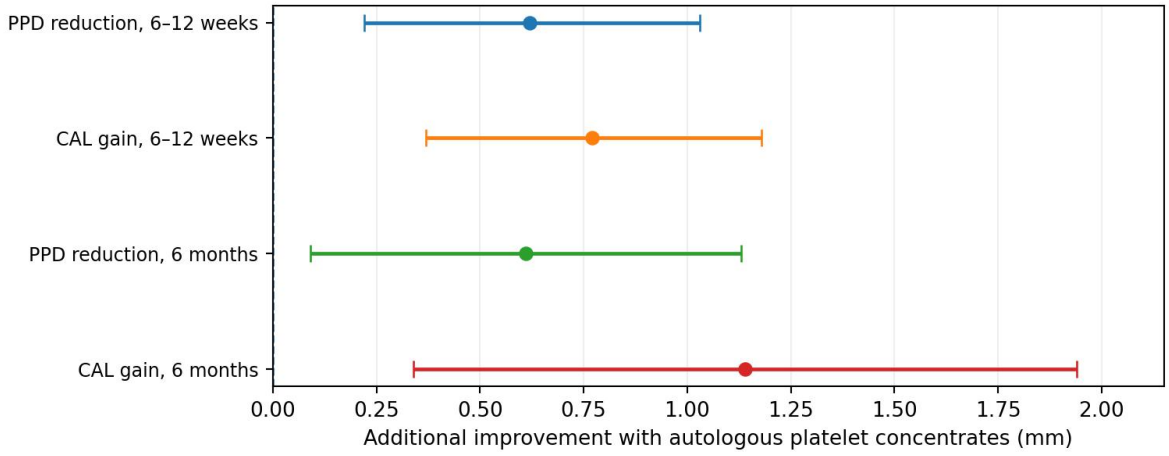


Figure 1. Additional improvements reported in the meta-analysis by Lipovec et al. [4]; dots indicate pooled mean differences expressed as benefit magnitude and whiskers indicate 95% confidence intervals. Mixed platelet formulations were included; estimates are not plasmolifting-specific.

This evidence profile supports a possible incremental improvement in clinical periodontal indices but does not resolve product-specific effectiveness. The meta-analysis reported high statistical heterogeneity and very low certainty for the assessed outcomes. Furthermore, the evidence does not establish that injection-based PRP achieves better long-term tooth retention, definite hard-tissue regeneration, or superiority over high-quality conventional treatment in all periodontal phenotypes [4].

Table 2. Comparison of interventions relevant to the term plasmolifting in periodontal care.

Intervention	Preparation / application	Interpretive limitation
PRP	Autologous platelet-enriched plasma; activation and cellular composition vary	Injection protocols and platelet dose are not interchangeable
PRF	Fibrin matrix with entrapped platelets; frequently applied locally	Clinical evidence for PRF cannot be assigned to PRP

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Injectable PRF	Fluid fraction before fibrin polymerization; locally injected	Preparation speed and timing influence properties
Standard nonsurgical care	Biofilm removal, oral hygiene and risk control	Essential reference treatment; never replaced by platelet adjuncts

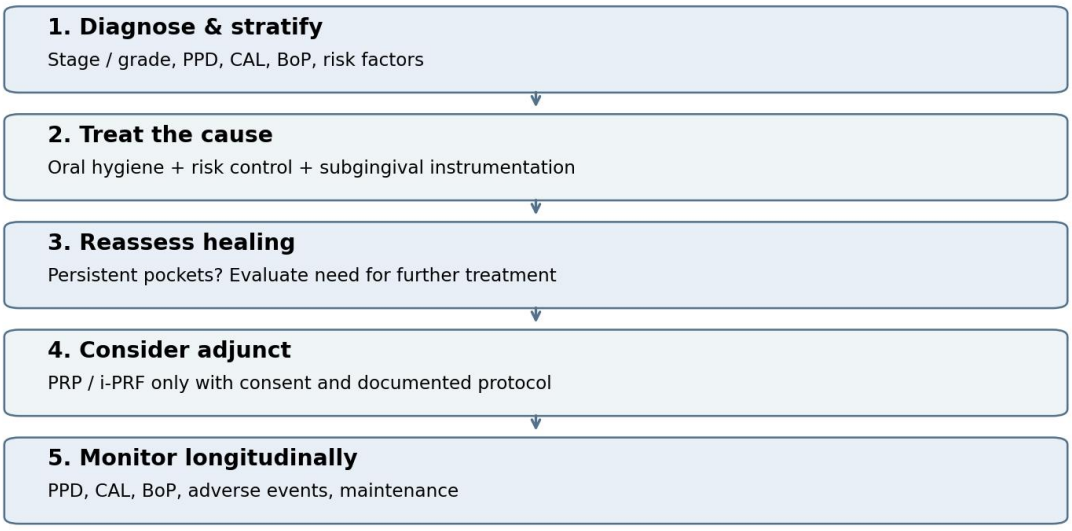


Figure 2. Proposed evidence-informed clinical evaluation pathway. This conceptual scheme is not a tested treatment algorithm and does not imply guideline endorsement of routine PRP injections.

Discussion

The synthesis suggests that adjunctive autologous platelet concentrates may improve some short-term clinical outcomes after nonsurgical periodontal treatment. Yet the clinically meaningful question is whether a reproducible injection-based PRP protocol offers additional benefit over careful debridement, excellent plaque control, risk-factor management, and maintenance. The currently available pooled evidence cannot answer that question conclusively because the source studies differ in concentrate type, number of applications, processing conditions, periodontal disease characteristics, and outcome measurement. The pooled point estimates are therefore best interpreted as possible incremental effects rather than guaranteed gains for each patient [3,4].

The distinction between the endpoints is important. PPD reduction can follow the resolution of edema and inflammation without new attachment formation; CAL gain is a more informative clinical indicator but is not direct histological evidence of periodontal regeneration. BoP assesses inflammatory response, yet pooled BoP estimates were inconclusive. These findings favor cautious integration of platelet preparations as

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investigational adjuncts, particularly when patients have residual disease after conventional therapy or when a predefined trial protocol calls for standardized local biologic treatment. They do not justify abandoning the EFP stepwise approach [2].

For a future original clinical study, a parallel-group randomized design comparing conventional nonsurgical treatment plus standardized injectable PRP with the same treatment plus an appropriate comparator would be preferable. Enrollment should follow internationally accepted staging and grading criteria, with recorded smoking status, diabetes control, initial PPD/CAL/BoP, oral hygiene, and relevant medication use. The investigational product must be characterized by blood collection volume, centrifugation protocol, device, anticoagulant, cellular composition if available, injection sites, number of sessions, and adverse-event surveillance. The primary endpoint might be CAL gain at six or twelve months, with PPD, BoP, patient-reported discomfort, and rescue treatment as secondary endpoints. Sample size, randomization, examiner blinding, ethical approval, and trial registration must be specified prospectively rather than inferred retrospectively.

Biological interventions should also be assessed for practical risks and cost. Autologous origin reduces certain compatibility concerns but does not eliminate risks associated with venipuncture, contamination during handling, local injection, pain, or inappropriate use in patients with relevant contraindications. Research reports should distinguish routine clinical observations from regulated procedures and describe product handling in sufficient detail for external replication. A demonstrated short-term change in a surrogate endpoint is not, by itself, evidence of a reduction in tooth loss or need for surgery.

The principal limitation of this article is that it synthesizes a selected evidence base rather than conducting a new systematic search or primary trial. The major quantitative results derive from a pooled analysis with high heterogeneity and very low certainty, and the platelet formulation used in the contributing trials was not uniform. Consequently, causality and comparative effectiveness of a specific plasmolifting protocol remain uncertain. These limitations should guide interpretation and the design of subsequent clinical research rather than be obscured by an overly favorable narrative.

Conclusion

Autologous platelet-derived adjuncts are biologically plausible options for supporting periodontal healing, and published pooled analyses suggest modest additional improvements in PPD and CAL after nonsurgical therapy. Nevertheless, the existing evidence is heterogeneous, has very low certainty, and cannot validate plasmolifting as a stand-alone or universally indicated treatment for periodontitis. Core treatment must remain centered on biofilm control, risk-factor modification, reassessment, and supportive periodontal care. Future research should evaluate clearly characterized injectable PRP preparations in prospectively registered, adequately powered trials with clinically meaningful follow-up and transparent adverse-event reporting.

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Declarations

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