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OPTIMIZATION OF REPARATIVE OSTEOGENESIS IN THE SURGICAL TREATMENT OF STAGE II–III GENERALIZED PERIODONTITIS USING AUTOLOGOUS PLATELET-RICH FIBRIN AND SERUM VITAMIN D MONITORING

Islomjon Xayrullayevich Telmonov

E-mail: telmonovislomjon@gmail.com | ORCID: 0009-0000-8047-0878

Department of Dentistry, Asia International University, Bukhara, Uzbekistan

ABSTRACT

Background: Regenerative surgery for stage II–III periodontitis with residual intrabony defects aims to recover periodontal attachment and supporting tissues. Autologous platelet-rich fibrin (PRF) provides a fibrin scaffold enriched with platelets and leukocytes, whereas vitamin D status may influence skeletal and periodontal wound healing. Objective: To critically synthesize the evidence for adjunctive PRF in periodontal regenerative surgery and examine the rationale for integrating serum 25-hydroxyvitamin D [25(OH)D] assessment into an individualized perioperative pathway. Methods: A focused narrative synthesis of international clinical guidance, systematic reviews, meta-analyses, and relevant clinical studies was undertaken. Studies of periodontal intrabony defect management, autologous platelet concentrates, and vitamin D–associated surgical outcomes were compared without pooling heterogeneous datasets. Results: European periodontal guidance supports regenerative therapy for appropriately selected deep intrabony defects. Systematic reviews report improved probing-depth reduction and clinical attachment outcomes for PRF added to open-flap debridement in some comparisons, although findings vary by defect morphology, comparator, PRF preparation, and follow-up. An earlier clinical study associated baseline vitamin D deficiency with less favorable periodontal surgical outcomes; however, no conclusion regarding a specific PRF–vitamin D interaction can be drawn. Conclusion: PRF may be considered an adjunct in defect-specific periodontal regenerative management, while serum vitamin D measurement may inform evaluation of suspected deficiency. Prospective trials are required to determine whether vitamin D optimization modifies PRF-associated healing. No original patient-level observations are claimed in this review.

Keywords: stage II–III periodontitis; periodontal regeneration; reparative osteogenesis; platelet-rich fibrin; intrabony defects; vitamin D; 25-hydroxyvitamin D; clinical attachment level.

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INTRODUCTION

Periodontitis is a chronic, biofilm-associated inflammatory disorder characterized by progressive loss of periodontal attachment and alveolar bone. Its contemporary staging framework distinguishes disease severity and the complexity of case management; the stage of a patient does not by itself establish the need for regenerative surgery at every tooth [1]. After behavioral risk-factor control, supragingival and subgingival instrumentation, and reassessment, residual deep pockets with an intrabony component may justify surgical treatment. The principal therapeutic objective is to achieve stable periodontal health while reducing the risk of future tooth loss, rather than merely improving radiographic appearances [2].

Autologous platelet-rich fibrin (PRF) is a second-generation platelet concentrate that may serve as a biologically active fibrin matrix and support early wound stabilization. Reported effects include the local retention and release of mediators associated with tissue repair, but clinical benefit depends on preparation protocol, defect morphology, surgical design, maintenance, and the comparator used [3–5]. Vitamin D is involved in calcium–phosphate homeostasis and immune regulation, creating a biologically plausible link between 25(OH)D status and postsurgical healing. Nevertheless, serum vitamin D status is not a validated surrogate marker of periodontal bone regeneration, and causality should not be inferred from association alone [6–8]. The objective of the present paper is to critically integrate current evidence on PRF-assisted periodontal surgery and vitamin D status into a reproducible clinical research framework for patients with generalized stage II–III periodontitis.

LITERATURE REVIEW

The 2018 consensus classification describes periodontitis using stage, grade, and extent, thereby distinguishing global disease characteristics from tooth-level defect anatomy [1]. The EFP S3 guideline recommends regenerative surgery in teeth with residual deep periodontal pockets associated with intrabony defects of at least 3 mm after appropriate initial therapy. This recommendation primarily derives from trials comparing regenerative approaches with open-flap debridement and does not imply that every PRF formulation is an established equivalent to enamel matrix derivatives or barrier-based regeneration [2]. Furthermore, interproximal tissue preservation, adequate plaque control, patient adherence, and defect-specific biomaterial selection remain central determinants of treatment planning.

Evidence concerning PRF is encouraging but heterogeneous. A 2021 systematic review and meta-analysis evaluated randomized comparisons of PRF-based protocols for periodontal intrabony defects [3]. A subsequent network meta-analysis assessed 32 trials and identified favorable clinical outcomes for several PRF-containing combinations, while noting variability in intervention protocols [4]. A comprehensive synthesis published in Periodontology 2000 reported that PRF combined with open-flap debridement improved probing depth, clinical attachment, and radiographic bone-fill outcomes relative to debridement alone in relevant comparisons; however, superiority was not consistently demonstrated against other established regenerative materials [5]. These observations

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support selective adjunctive use and further standardization, not an unconditional claim of histologically confirmed new bone formation.

Interest in vitamin D arises from its involvement in mineral metabolism, immune signaling, and the regulation of inflammatory responses. Bashutski and colleagues studied 40 patients undergoing periodontal surgery within a trial involving teriparatide or placebo and reported less favorable clinical attachment gain and pocket-depth reduction in vitamin D–deficient placebo participants [6]. Because that study did not evaluate PRF, its findings support an association between vitamin D status and postsurgical prognosis but cannot establish a direct benefit from combining PRF with vitamin D monitoring. A later systematic review likewise suggested that deficiency may adversely affect periodontal treatment outcomes, while emphasizing the small evidence base [7]. A 2023 meta-analysis examining vitamin D and periodontitis found heterogeneous associations and limited effects across different periodontal endpoints [8]. The distinction between testing, clinical correction of documented deficiency, and unproven supplementation solely to enhance PRF-related healing is therefore essential.

MATERIALS AND METHODS

This article was developed as a focused narrative, evidence-based synthesis with an embedded prospective-study framework. The sources comprised the consensus classification of periodontitis, the European Federation of Periodontology treatment guideline, meta-analyses of platelet-rich fibrin for intrabony defects, and clinical research or reviews on serum 25(OH)D in periodontal therapy [1–8]. Findings were organized by population, intervention, comparison, outcomes, and relevance to the proposed combined approach. Formal study selection counts, exhaustive search documentation, risk-of-bias scoring, and quantitative meta-analysis were not performed; accordingly, this work is not presented as a systematic review and does not report newly recruited patients.

For a future ethics-approved prospective trial, the target population would comprise adults with generalized stage II or III periodontitis and at least one residual intrabony defect suitable for regenerative surgery after standardized non-surgical periodontal treatment. Disease classification would follow contemporary staging and grading criteria, with tooth-level defect depth, width, wall configuration, and mobility documented separately. Baseline assessments would include plaque control, bleeding on probing, probing pocket depth (PPD), clinical attachment level (CAL), standardized periapical radiography (and CBCT only when clinically justified), and serum 25(OH)D where deficiency is suspected or clinical risk justifies assessment. Important confounders include smoking, diabetes, body mass index, metabolic bone disease, relevant medicines, and oral hygiene adherence.

A controlled design could compare defect-specific regenerative surgery with PRF versus an appropriate standard surgical comparator, with prespecified stratification by baseline 25(OH)D status rather than experimentally inducing vitamin D deficiency. Vitamin D deficiency, where established, should be managed according to applicable medical guidance independent of trial assignment. PRF preparation must record tube material, centrifuge model, relative centrifugal force, rotor geometry, spin duration, time to

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application, and membrane or clot configuration; reporting only revolutions per minute is insufficient for reproducibility. Clinical outcomes should be assessed by calibrated examiners blinded where feasible at baseline and 6 and 12 months. CAL gain would serve as a defensible primary clinical outcome; PPD reduction, radiographic defect-depth changes, postoperative morbidity, and adverse events should be secondary outcomes. Radiographic fill would not be equated with histological evidence of new bone, cementum, and periodontal ligament.

A prospectively registered statistical plan should define one principal comparison, a clinically meaningful effect size, and a sample-size calculation. Mixed-effects regression or another appropriate model would account for repeated measures and clustering of defects within participants, while vitamin D status and major covariates would be entered as prespecified variables. Interaction between PRF allocation and baseline 25(OH)D could be exploratory unless adequately powered. Reporting should include effect estimates, 95% confidence intervals, exact P values, missing-data methods, and adverse-event frequencies. Institutional ethics approval and informed consent are necessary before any patient recruitment.

RESULTS

Because the source materials provide no clinical dataset collected by the named author, the results below report findings of published studies and a proposed measurement framework only. The EFP guideline synthesizing 22 randomized trials found an average adjunctive clinical attachment benefit of 1.34 mm (95% confidence interval [CI] 0.95–1.73) and an average pocket-depth reduction benefit of 1.20 mm (95% CI 0.85–1.55) for regenerative interventions compared with open-flap debridement; these numbers concern the broader category of periodontal regenerative procedures and must not be attributed specifically to PRF [2]. PRF meta-analyses suggest benefit for selected comparisons against debridement alone, but the effect size is not uniform across studies, formulations, or other active comparators [3–5].

In the vitamin D trial reported by Bashutski et al., placebo-group patients with deficiency had a CAL change of −0.43 mm compared with 0.92 mm among vitamin D–sufficient participants; corresponding PPD reductions were 0.43 and 1.83 mm, respectively [6]. These are published subgroup results from a study with a different intervention and should be interpreted as contextual evidence rather than quantitative predictions for PRF-treated patients. No validated cutoff for a vitamin D–dependent PRF response, causal interaction effect, or universally effective supplementation regimen can be inferred from the available studies.

Table 1 compares evidence sources and their limits, while Table 2 summarizes the variables required to test the proposed approach. Figure 1 illustrates the published vitamin D subgroup observations, and Figure 2 provides a conceptual diagnostic and treatment pathway, not empirical study outcomes.

Evidence source	Main reported finding	Interpretive limitation

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EFP S3 guideline [2]	Regenerative treatment recommended for selected deep residual intrabony defects.	Pooled benefit not PRF-specific.
PRF meta-analyses [3–5]	Several PRF-assisted strategies improve PPD/CAL relative to selected comparators.	Protocol and comparator heterogeneity; bone fill ≠ histologic regeneration.
Vitamin D surgical study [6]	Baseline deficiency associated with less favorable outcomes in a subgroup.	Different co-intervention; cannot show PRF × vitamin D effect.
Vitamin D synthesis [7–8]	Associations between low 25(OH)D and outcomes remain plausible.	Evidence heterogeneous; monitoring itself is not treatment.

Table 1. Summary of external evidence relevant to the proposed combined strategy.

Domain	Suggested measure	Timing / interpretation
Periodontal severity	Stage and grade; PPD, CAL, BOP, plaque	Baseline; 6 and 12 months
Bone defect	Standardized defect depth/width and radiographic fill	Pre-surgery and follow-up; calibrated imaging
Systemic status	Serum 25(OH)D, medical risk factors	Baseline; retest only if clinically indicated
PRF reproducibility	RCF, time, centrifuge/tubes, clot placement	At surgery
Healing and safety	Wound closure, pain, infection, adverse events	Early and late follow-up

Table 2. Proposed measurement framework (not observed patient data).

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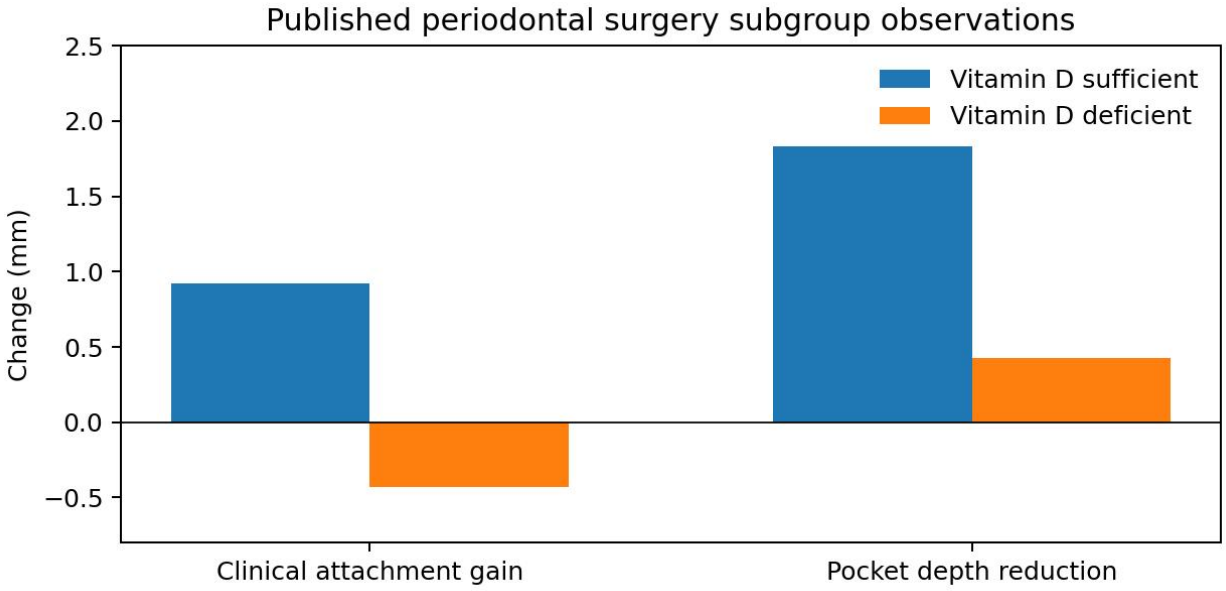


Figure 1. Published subgroup mean changes after periodontal surgery reported by Bashutski et al. (2011) [6]. Neither group was treated with PRF as part of this analysis; bars are illustrative, not pooled effects.

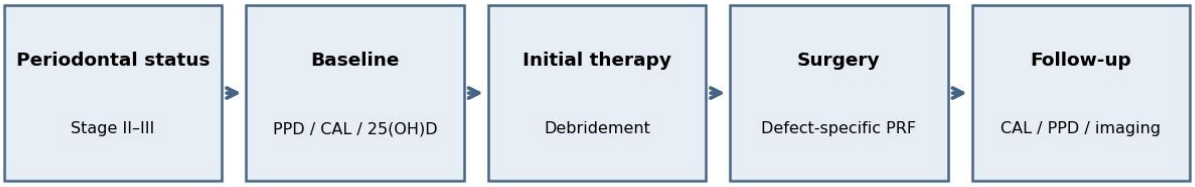


Figure 2. Conceptual sequence for evaluation and follow-up of PRF-assisted periodontal regenerative treatment with clinically indicated vitamin D assessment (proposed framework, not a tested algorithm).

DISCUSSION

The available evidence supports a staged and defect-specific approach to periodontal regeneration. Inflammation and plaque control should be established first, followed by reassessment of persistent sites and selection of an appropriate flap and regenerative technique. PRF is attractive because it is autologous and can be prepared chairside, yet its performance is dependent on procedural standardization and the surrounding surgical

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environment. Favorable effects on CAL or radiographic defect reduction should be interpreted as clinical or radiographic outcomes, not direct proof of reparative osteogenesis at the histological level [2–5].

The rationale for serum vitamin D monitoring is distinct from the rationale for PRF. Serum 25(OH)D can help identify suspected deficiency in patients with relevant medical risk factors, while vitamin D biology offers a plausible contribution to mineral metabolism and inflammatory regulation. Nonetheless, an association between low 25(OH)D and poor surgical healing may be confounded by age, nutrition, body mass, smoking, diabetes, and other factors. The existing literature does not establish that routine screening of every periodontal surgical patient improves outcomes, nor does it prove that supplementation enhances the regenerative effect of PRF in patients without deficiency [6–8]. A rigorous trial would therefore separate baseline status, medical treatment of deficiency, and the effect of PRF.

Potential sources of heterogeneity require particular attention. PRF protocols encompass different cell compositions, relative centrifugal forces, and fibrin architectures; combining these under one intervention label can obscure differences. Similarly, two-wall and three-wall defects, initial PPD and CAL, and the degree of wound stability influence regenerative potential. Multiple defects within one person are not statistically independent. Prospective analysis should avoid overstating small changes, document patient-centered outcomes, and specify whether the principal goal is clinical attachment gain, radiographic fill, or another endpoint. If direct evidence of true new bone formation is sought, ethical and clinically appropriate histological investigation would be required, which is generally impractical in routine human periodontal surgery.

This synthesis is limited by its narrative rather than systematic methodology and by the absence of author-generated clinical data. It nonetheless identifies a testable hypothesis: baseline 25(OH)D status may modify the clinical response to standardized PRF-assisted regenerative surgery in selected stage II–III periodontitis patients. The hypothesis should be evaluated without assuming that low vitamin D causes poor healing or that its pharmacologic correction necessarily changes outcomes. The proposed framework may aid protocol development and transparent reporting, but it is not a validated treatment algorithm.

CONCLUSION

Adjunctive autologous PRF is a promising component of selected periodontal regenerative procedures, with published evidence of improvements in some clinical outcomes relative to open-flap debridement alone. Periodontal stage II–III classification must be supplemented by tooth-level assessment before selecting a surgical regenerative intervention. Vitamin D status may be clinically relevant to postsurgical healing, but an independent or synergistic benefit from combining PRF with vitamin D monitoring has not been demonstrated. A prospective, standardized, adequately powered investigation with calibrated clinical measurements, transparent PRF preparation, defined vitamin D assessment, and long-term follow-up is needed to establish the effectiveness of the combined approach.

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