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Pathogenetic Rationale for Combined Platelet-Rich Plasma and Low-Level Laser Therapy in Postoperative Hypertrophic Scar Prevention

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Abstract

Platelet-rich plasma (PRP) and low-level laser therapy (LLLT), also known as photobiomodulation therapy, are biologically oriented approaches that may support postoperative wound healing and scar remodeling. This narrative review summarizes the mechanisms of hypertrophic scar formation, PRP effects on dermal fibroblasts and extracellular matrix remodeling, and LLLT-mediated regulation of inflammation, microcirculation and mitochondrial activity. Particular attention is paid to the pathogenetic rationale for combining PRP and LLLT in the early postoperative period. The review emphasizes that this combined approach should be regarded as biologically plausible, but not yet as a standardized method, because clinical protocols, PRP preparation techniques, laser parameters and outcome measures remain heterogeneous.

Keywords. platelet-rich plasma; PRP; low-level laser therapy; LLLT; photobiomodulation; postoperative wound; hypertrophic scar; fibroblast; collagen remodeling; scar prevention.

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1. Introduction

Platelet-rich plasma (PRP) and low-level laser therapy

(LLLT), also referred to as photobiomodulation therapy (PBMT), are two biologically oriented approaches that may influence postoperative wound healing and scar maturation. PRP provides an autologous source of platelet-derived growth factors, cytokines and bioactive mediators involved in chemotaxis, angiogenesis, fibroblast activity and extracellular matrix turnover. In contrast, LLLT acts through non-destructive light–tissue interactions, primarily by modulating mitochondrial activity, local microcirculation, inflammatory responses and cellular metabolism. For postoperative hypertrophic scar prevention, these methods should therefore be discussed not as isolated cosmetic procedures, but as complementary components of a broader pathogenetic strategy aimed at regulating inflammation, preventing prolonged fibroblast activation and supporting more physiological extracellular matrix remodeling [2, 6, 12]. Such an approach is particularly relevant in the early postoperative period, when the wound microenvironment remains biologically active and therapeutic modulation may influence the subsequent quality of scar formation.

Aim of the research

The aim of this review is to analyze the pathogenetic basis for the combined use of PRP and LLLT/PBMT in the prevention of postoperative hypertrophic scars, with emphasis on fibroblast activity, inflammation, collagen remodeling, treatment standardization and clinically relevant outcome assessment.

Materials and methods

A narrative literature review was performed using publications indexed in PubMed, Scopus, Google Scholar and regional scientific sources. Priority was given to recent reviews, clinical recommendations, experimental and clinical studies related to hypertrophic scars, cutaneous wound healing, PRP, LLLT/PBMT and scar assessment. Classical methodological sources were retained when they introduced widely used scar scales or fundamental wound-healing concepts. Publications with uncertain bibliographic verification or indirect relevance to postoperative cutaneous scarring were excluded.

Hypertrophic scar formation as a target for preventive therapy. Hypertrophic scars develop when the normal wound-healing cascade remains biologically active for an excessive period. They remain within the borders of the original wound but are elevated, erythematous, dense and often symptomatic. The main biological drivers are prolonged inflammation, excessive TGF-β/SMAD signaling, persistence of myofibroblasts and imbalance between matrix metalloproteinases and their inhibitors [19, 22]. Pain, pruritus, stiffness and cosmetic dissatisfaction may decrease quality of life and create a need for long-term corrective treatment [20, 26].

Mechanical tension is also a major factor. Wounds located in high-tension areas show stronger activation of fibroblasts and myofibroblasts, which increases extracellular matrix production. Infection, delayed epithelialization, hematoma, seroma and repeated local trauma further prolong the inflammatory phase and increase the risk of abnormal scarring [9, 16]. Therefore, an effective preventive protocol should combine proper surgical closure with biological modulation of the postoperative wound environment [28].

Normal wound healing and scar remodeling. The wound-healing process includes hemostasis, inflammation, proliferation and remodeling. During hemostasis, platelets form a fibrin clot and release growth factors that attract inflammatory and reparative cells. In the inflammatory phase, neutrophils and macrophages clear debris and pathogens. A timely transition from pro-inflammatory to reparative macrophage phenotypes is crucial for proper healing [5, 31]. During proliferation, keratinocytes migrate, endothelial cells form new capillaries and fibroblasts deposit provisional extracellular matrix rich in type III collagen [11].

During remodeling, type III collagen is progressively replaced by type I collagen, collagen fibers become more organized, vascularity decreases and scar tissue gains tensile strength. Pathological scarring occurs when collagen synthesis dominates over degradation, when myofibroblast apoptosis is delayed and when vascular and inflammatory signals remain active [19, 22]. Consequently, outcome assessment in scar-prevention studies should extend beyond early wound closure and include at least 3- to 6-month scar maturation endpoints.

PRP composition and methodological standardization. PRP is an autologous blood-derived concentrate containing platelets at a level above baseline. After activation, platelets release PDGF, TGF-β, VEGF, IGF-1, EGF and other mediators involved in chemotaxis, angiogenesis, cell proliferation and matrix synthesis [6]. However, PRP should not be presented as a uniformly beneficial or fully standardized product, because its biological activity depends on platelet concentration, leukocyte content, activation method, injection depth, timing and the inflammatory status of the target tissue [8].

The heterogeneity of PRP preparation is one of the main

barriers to translation. Studies use different centrifugation speeds, centrifugation times, single- or double-spin protocols, anticoagulants, activators and platelet concentration targets. Leukocyte-rich and leukocyte-poor preparations may have different effects on inflammation [6, 8]. In a clinical dissertation protocol, it is therefore necessary to specify the PRP preparation method, final volume, platelet concentration range, injection technique, number of sessions and interval between sessions.

PRP effects on dermal fibroblasts and scar modulation. The most relevant evidence for hypertrophic scar prevention comes from studies involving dermal fibroblasts and cutaneous wound models. Nam and Kim reported that PRP may reduce the expression of connective tissue growth factor and TGF-β1 in hypertrophic-scar fibroblasts, suggesting a possible antifibrotic direction of action [23]. These findings provide a biological rationale for PRP, but they do not prove that PRP alone is sufficient for reliable prevention of postoperative hypertrophic scars.

This distinction is important. PRP contains growth factors that may stimulate repair, but excessive or poorly timed stimulation in a fibrotic microenvironment may theoretically be undesirable. Therefore, PRP should be used not as an empirical injection but as a part of a controlled protocol. For scar prevention, the aim is not maximal fibroblast stimulation; the aim is physiological remodeling, balanced collagen organization and reduction of prolonged inflammation. This is the conceptual point at which LLLT/PBMT becomes particularly relevant.

LLLT/PBMT mechanisms relevant to scar prevention. LLLT/PBMT is a non-ablative and non-destructive light-based treatment, usually using red or near-infrared wavelengths. The main proposed cellular target is mitochondrial cytochrome-c oxidase. Light absorption may increase ATP synthesis, modulate nitric oxide release and regulate reactive oxygen species at signaling levels [7, 12]. These processes can influence microcirculation, cellular metabolism, inflammatory mediator production and reparative cell migration [2, 30].

Unlike ablative and fractional lasers, LLLT does not rely on thermal destruction of scar tissue. This makes it more suitable for early postoperative use, provided that the wound is closed and there are no signs of infection or dehiscence. Experimental and clinical data suggest that LLLT may reduce inflammatory infiltration, support epithelialization, improve local blood flow and influence collagen organization [25, 27]. However, dose selection is crucial because photobiomodulation follows a biphasic dose-response pattern.

For hypertrophic scar prevention, the most relevant LLLT effects are anti-inflammatory action, improvement of microcirculation and modulation of fibroblast behavior. If the inflammatory phase is shortened and tissue oxygenation is improved, the wound may enter the remodeling phase under more physiological conditions. At the same time, LLLT may influence collagen fiber organization and matrix remodeling through changes in fibroblast activity and MMP/TIMP balance [10, 22, 30].

Regional and Russian-language evidence. Russian-language and regional publications are important because they connect the dissertation topic with local surgical practice. Greben and colleagues summarized contemporary mechanisms of low-level laser therapy and emphasized that outcomes depend on wavelength, energy density, exposure time and tissue condition [10]. Preclinical work also supports the need for careful dose selection in low-intensity laser irradiation [25].

Uzbek authors have reported experience with laser and light-based methods in surgical wound management. Hamdamov and co-authors studied laser photodynamic therapy for prevention and treatment of postoperative complications in diabetic foot syndrome [13–15]. Photodynamic therapy is not identical to LLLT, yet these regional works confirm the clinical relevance of controlled light-based technologies for wound inflammation and postoperative complication prevention.

Rationale for combining PRP and LLLT. The combination of PRP and LLLT can be conceptualized as a two-component biological strategy. LLLT may improve tissue microcirculation, mitochondrial metabolism and inflammatory balance, thereby preparing the wound microenvironment. PRP may then provide a concentrated autologous pool of growth factors and signaling molecules. In this model, LLLT does not simply add another procedure; it may improve the biological conditions under which PRP-derived mediators act [6, 7, 12].

Table 1. Comparative mechanisms of PRP and LLLT/PBMT relevant to postoperative scar prevention

Target process	PRP contribution	LLLT/PBMT contribution	Clinical relevance
Inflammation	Modulates cytokines and growth-factor signaling	Supports anti-inflammatory photobiomodulation	May reduce prolonged inflammatory stimulation of fibrosis
Microcirculation	Provides angiogenic mediators such as VEGF	May improve perfusion through NO-related mechanisms	Improves oxygenation and early tissue recovery
Fibroblast activity	May regulate CTGF/TGF-β-related responses in scar fibroblasts	Modulates cellular metabolism and fibroblast behavior	Aims to avoid excessive matrix deposition
Collagen remodeling	Influences extracellular matrix synthesis and organization	May affect collagen fiber organization and MMP/TIMP balance	Targets scar height, density and elasticity
Protocol control	Requires standardized preparation and injection technique	Requires defined wavelength, energy density and treatment schedule	Improves reproducibility and comparability of outcomes

The expected effect of combined therapy in scar prevention is not merely faster epithelialization. A clinically meaningful result would be a lower incidence of hypertrophic scars, lower Vancouver Scar Scale and POSAS scores, reduced scar thickness on ultrasound, lower stiffness on elastography, less erythema and vascular activity on

dermoscopy, and reduced pain or itching [4, 29]. Therefore, the study endpoints should reflect scar quality and maturation rather than only early wound closure.

Preclinical and translational relevance. Preclinical models remain valuable because they allow histological and molecular assessment that cannot be routinely performed in human patients. Skin wound models can evaluate inflammatory infiltration, capillary density, collagen fiber orientation, type I/type III collagen ratio and expression of TGF-β-related markers. These data support biological plausibility, but they must be translated cautiously because animal wound healing differs from human postoperative scarring [25, 31].

For the planned dissertation, the strongest translational pathway is to align experimental mechanisms with standardized clinical observation. If preclinical data suggest more organized collagen and lower inflammatory activity, clinical endpoints should assess the same processes indirectly: scar height, stiffness, vascularity, pigmentation, itching, pain and objective thickness.

Clinical protocol implications. A rational clinical protocol should include patients with comparable surgical wounds, preferably in one anatomical region or within a limited group of operations. The intervention should begin only after exclusion of infection, hematoma, seroma or wound dehiscence. LLLT parameters must be recorded in detail: wavelength, output power, energy density, exposure time, mode, treated area and number of sessions. PRP parameters must also be described: blood volume, centrifugation protocol, platelet concentration, activation status, injection depth, total volume and injection spacing.

Follow-up should be long enough to capture scar maturation. The first 10–14 days mainly reflect wound closure and early inflammation. Scar outcomes should be assessed at 30, 60, 90 and 180 days, with 360-day follow-up if feasible. The primary outcome may be the incidence of hypertrophic scar formation at 6 months. Secondary outcomes may include VSS, POSAS, ultrasound thickness, dermoscopic vascularity, elastographic stiffness, pain, itching and patient satisfaction.

Table 2. Recommended clinical endpoints for evaluation of postoperative scar prevention

Endpoint	Assessment method	Recommended timing
Hypertrophic scar incidence	Clinical examination using predefined diagnostic criteria	90 and 180 days
Scar appearance	Vancouver Scar Scale and standardized photography	30, 60, 90 and 180 days
Patient-reported symptoms	POSAS patient component, pain and itching scores	Every follow-up visit
Scar thickness	High-frequency ultrasound	90 and 180 days
Vascular activity	Dermoscopy or standardized clinical imaging	30, 60 and 90 days
Scar stiffness	Elastography when available	90 and 180 days
Safety	Infection, dehiscence, hematoma, seroma and adverse local reactions	Throughout follow-up

Limitations of existing evidence. Despite promising mechanisms, the existing literature has several limitations. PRP studies remain heterogeneous and often use different preparations under the same term. LLLT studies vary in wavelength, energy density, exposure time and treatment schedule. Many scar-related laser studies focus on already formed scars rather than early postoperative prevention. Combination protocols involving PRP and LLLT for hypertrophic scar prevention remain insufficiently standardized, and randomized controlled clinical studies with objective scar assessment remain limited [6, 8, 30].

Therefore, the dissertation should avoid overstating the proven efficacy of the combined method. A stronger scientific position is to state that PRP and LLLT have complementary mechanisms and that their combined use is biologically plausible, but requires controlled clinical validation. This formulation creates a clear research gap and justifies the planned experimental-clinical design.

Conclusion

PRP and LLLT/PBMT are promising biological approaches for postoperative wound healing and scar modulation. PRP supplies autologous platelet-derived mediators, while LLLT may regulate inflammation, microcirculation, mitochondrial activity and extracellular matrix remodeling. Their combination is pathogenetically plausible for prevention of hypertrophic scars, but it should not be presented as a proven standard therapy without controlled clinical validation. The most appropriate dissertation focus is therefore a targeted investigation of combined PRP and LLLT therapy using standardized preparation, clearly defined laser parameters, sufficient follow-up and objective scar-quality endpoints.

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