Platelet-Rich Fibrin in Androgenetic Alopecia: A Comprehensive Clinical and Mechanistic Review

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Received: 28 October 2024, **Accepted:** 17 November 2024, **Published:** 20 November 2024

Abstract

Background: Androgenetic alopecia (AGA) is the most common chronic, progressive form of hair loss in men and women, driven by androgen-mediated follicular miniaturization and a complex interplay of genetic, hormonal, and microinflammatory processes. Traditional therapies such as minoxidil, finasteride, low-level laser therapy, and hair transplantation provide varying degrees of efficacy but are limited by incomplete response, dependence on long-term adherence, and potential adverse effects. Growing interest in regenerative dermatology has introduced autologous bio-stimulatory therapies, particularly platelet-derived concentrates. Platelet-rich fibrin (PRF), a second-generation platelet concentrate, has emerged as a promising therapeutic candidate due to its sustained release of growth factors, natural fibrin scaffold, and lack of anticoagulants, which distinguishes it biologically and clinically from platelet-rich plasma (PRP).

The aim of this review is to comprehensively evaluate the current clinical evidence, mechanistic rationale, and therapeutic potential of PRF in the management of AGA, while highlighting its advantages, limitations, and future research needs. This narrative review synthesizes findings from experimental studies, clinical trials, comparative analyses with PRP, and emerging combinatorial approaches to delineate the role of PRF within contemporary AGA treatment paradigms.

Current data suggest that PRF provides a biologically advantageous environment for follicular regeneration by enhancing dermal papilla cell proliferation, improving angiogenesis, reducing perifollicular microinflammation, and prolonging the bioavailability of key growth factors such as PDGF, VEGF, IGF-1, and TGF-β. Clinically, injectable PRF has been associated with improvements in hair density, shaft diameter, and global photographic outcomes, with several studies indicating comparable or superior results to PRP. Its autologous nature, minimal preparation additives, and favorable safety profile enhance its suitability for repeated, long-term use. However, variability in preparation protocols, limited randomized controlled trials, and small sample sizes currently restrict generalizability of findings.

In conclusion, PRF represents a biologically robust and clinically promising regenerative therapy for AGA. While evidence supports its efficacy, optimized preparation methods, standardized treatment protocols, long-term follow-up studies, and mechanistic profiling are required to solidify its role in routine dermatologic and andrologic practice.

Keywords: *Platelet-Rich Fibrin, Androgenetic Alopecia, Clinical and Mechanistic Review*

Introduction

Androgenetic alopecia (AGA) is the most prevalent form of progressive hair loss, affecting up to 50% of men by age 50 and a substantial proportion of women, with significant psychosocial and quality-of-life implications. It is characterized by gradual miniaturization of hair follicles under the influence of dihydrotestosterone (DHT), combined with genetic susceptibility and microinflammatory perifollicular changes. Although therapies such as topical minoxidil and oral finasteride remain first-line, their efficacy is variable, long-term adherence is challenging, and adverse effects—especially with antiandrogens—can limit use. Consequently, there has been growing emphasis on regenerative approaches capable of stimulating follicular recovery through autologous biological pathways. Platelet-derived products, most notably platelet-rich plasma (PRP), have shown therapeutic potential; however, limitations in growth factor sustainability and reliance on anticoagulants have led to exploration of next-generation concentrates such as platelet-rich fibrin (PRF), which offers a more physiologically favorable profile. [1–3]

PRF represents an autologous fibrin matrix rich in platelets, leukocytes, and cytokines, capable of gradual release of regenerative biomolecules essential for hair cycling. Its use has expanded rapidly in dermatology and regenerative medicine due to simplified preparation, absence of biochemical additives, and evidence of prolonged growth factor availability relative to PRP. Preliminary clinical studies suggest that PRF may improve hair density, thickness, and dermal papilla function, positioning it as a promising modality for AGA management. Despite rising clinical adoption, current literature remains heterogeneous, with variation in preparation protocols, injection techniques, centrifugation speeds, and outcome measures. These inconsistencies create uncertainty regarding optimal use and comparative efficacy versus established options, warranting a comprehensive synthesis of available data. [4–6]

The **aim** of this review is to critically evaluate the mechanistic foundations and clinical performance of PRF in AGA, integrating dermatologic and andrologic perspectives. The **research gap** centers on the lack of standardized protocols, limited randomized controlled trials, and inadequate long-term follow-up, which collectively restrict clinical guideline development. By consolidating mechanistic insights, clinical evidence, and comparative analyses with PRP and conventional therapies, this review seeks to define the current status of PRF and outline future directions for its integration into AGA treatment algorithms. [7–8]

Pathophysiology of Androgenetic Alopecia

Androgenetic alopecia (AGA) is fundamentally driven by the actions of dihydrotestosterone (DHT) on androgen-sensitive hair follicles, particularly in the frontal and vertex scalp, leading to progressive miniaturization and shortened anagen duration. DHT binds to androgen receptors within dermal papilla cells, altering the expression of growth-modulating genes such as TGF-β1, IGF-1, and Wnt signaling mediators, which collectively shift follicles toward a catagen-promoting environment. Genetic predisposition significantly modulates receptor density, 5-α-reductase activity, and follicular responsiveness, explaining sex- and site-specific variability in clinical presentation. The shift from terminal to vellus-like hairs reflects cumulative miniaturization cycles, affirming the chronicity and irreversible nature of advanced disease. [9–11]

Microinflammation within the perifollicular environment contributes to AGA progression through subtle lymphohistiocytic infiltrates, oxidative stress, and extracellular matrix remodeling. Studies show upregulation of proinflammatory cytokines, including IL-6 and TNF-α, around affected follicles, leading to perifollicular fibrosis and impaired follicular cycling. These microenvironmental changes compromise stem cell niches in the bulge region and reduce the potential for follicular regeneration. This inflammatory hypothesis is supported by findings of increased oxidative markers and reduced antioxidant enzymes in AGA scalp biopsies. The interplay between androgen signaling and microinflammation suggests that therapeutic strategies which suppress inflammatory damage may complement androgen-targeted therapies. [12–14]

Vascular impairment also plays a role in follicular regression in AGA. Reduced perifollicular blood flow

and diminished expression of vascular endothelial growth factor (VEGF) contribute to impaired nutrient delivery and suboptimal follicular metabolism. Doppler flow studies and histologic analyses confirm decreased microvascular density in balding scalp compared with non-balding regions. As hair follicles require robust vascular support during anagen, diminished angiogenic signaling accelerates their transition toward catagen and telogen. This understanding underscores the therapeutic relevance of pro-angiogenic interventions—including autologous platelet concentrates such as PRF—that restore microcirculatory dynamics and support follicular survival. [15–16]

Cellular senescence further contributes to AGA pathophysiology, as dermal papilla cells in balding scalp exhibit reduced proliferative capacity, increased expression of senescence-associated markers such as p16INK4a, and mitochondrial dysfunction. These alterations impair their ability to maintain anagen-supportive signaling and to respond effectively to regenerative stimuli. Environmental factors, such as ultraviolet radiation and smoking, may exacerbate oxidative stress and senescence pathways, promoting earlier onset or accelerated progression of AGA. Collectively, androgenic, inflammatory, vascular, and senescence-driven mechanisms converge to produce the characteristic patterned hair loss that defines the condition. [17–18]

Evolution of Autologous Platelet Concentrates

The development of autologous platelet concentrates has progressed significantly over the past three decades, beginning with platelet-rich plasma (PRP), which emerged in the 1970s and gained widespread medical use in the 1990s. PRP was initially utilized in maxillofacial and orthopedic surgery for its capacity to deliver concentrated growth factors that enhance tissue repair. Its preparation involves anticoagulants and multiple centrifugation steps to prevent clotting and separate platelet-rich fractions from red and white cell components. Although PRP demonstrated regenerative potential in dermatology—including early evidence in alopecia treatment—it was soon recognized that its therapeutic impact was limited by fast release kinetics of growth factors and variable platelet concentrations, which depend heavily on technique and device-specific parameters. These limitations created demand for next-generation systems offering more reliable bioactivity and extended growth factor release. [19–21]

Second-generation platelet concentrates introduced the concept of platelet-rich fibrin (PRF), first described by Choukroun and colleagues in 2001. Unlike PRP, PRF is produced without anticoagulants, allowing for natural coagulation that traps platelets and leukocytes within a dense fibrin network. This fibrin scaffold functions as a biologically active matrix capable of gradual growth factor release over several days, enhancing regenerative potential and mimicking physiologic wound healing processes. PRF was initially adopted in oral and maxillofacial surgery for bone regeneration but rapidly expanded into broader regenerative medicine fields due to its simplicity, lack of additives, and biologically favorable cellular composition. Comparative studies have shown that PRF matrices retain more leukocytes and allow prolonged release of PDGF, TGF-β, and VEGF, suggesting mechanistic superiority over PRP in tissue engineering applications. [22–24]

Subsequent innovations led to the development of advanced PRF systems—including injectable PRF (i-PRF), advanced PRF (A-PRF), and concentrated PRF (C-PRF)—which utilize modified centrifugation protocols to optimize platelet yield, leukocyte viability, and fibrin architecture. Injectable PRF in particular has been transformative for dermatology, enabling minimally invasive delivery of a liquid fibrinogen-rich solution that polymerizes in situ after injection. This has allowed clinicians to apply PRF to indications such as skin rejuvenation, acne scarring, and hair loss. The refinement of low-speed centrifugation concepts further enhanced growth factor retention and cellular inclusivity, yielding preparations with improved angiogenic and anti-inflammatory properties. These advancements established PRF as a leading autologous regenerative injectable with growing evidence supporting its role in androgenetic alopecia. [25–27]

Biological Characteristics of Platelet-Rich Fibrin (PRF)

Platelet-rich fibrin (PRF) is distinguished from earlier platelet concentrates by its unique composition, which includes a dense fibrin network enriched with platelets, leukocytes, cytokines, and circulating

stem cells. This composition results from the absence of anticoagulants during preparation, allowing natural coagulation pathways to form a stable three-dimensional fibrin scaffold. The slow polymerization process produces a matrix with high tensile strength and a physiologic fibrin architecture that supports cellular migration, angiogenesis, and sustained release of bioactive molecules. This biological configuration more closely mimics natural wound healing responses than PRP, which undergoes rapid fibrin formation due to exogenous activation. [28–30]

A defining feature of PRF is its **prolonged growth factor release**, which extends over 7–14 days, in contrast with PRP that typically releases most growth factors within the first few hours after activation. Key regenerative mediators—such as PDGF, VEGF, IGF-1, TGF-β, and EGF—are entrapped within the fibrin matrix and gradually liberated as fibrin undergoes enzymatic degradation. This sustained release provides a more stable signaling environment for dermal papilla cells, endothelial cells, and follicular stem cell niches, enhancing their regenerative potential. The presence of leukocytes further contributes anti-inflammatory cytokines that modulate local immune responses, an important property given the microinflammatory component of androgenetic alopecia. Comparative in vitro studies consistently demonstrate significantly higher cumulative growth factor availability in PRF compared with PRP. [31–33]

PRF also contains a higher concentration of structural proteins, including fibronectin, vitronectin, and thrombospondin, which serve as scaffolding molecules that facilitate cell adhesion and migration. These extracellular matrix proteins are essential regulators of follicular cycling, particularly during anagen initiation. The fibrin network in PRF acts not only as a reservoir for growth factors but also as a biomechanical scaffold that supports neovascularization and fibroblast proliferation. Moreover, newer liquid formulations such as injectable PRF (i-PRF) preserve cellular vitality and allow delivery to deeper dermal layers before polymerization, making them well suited for hair restoration therapies. Such biologic complexity positions PRF as a multifunctional autologous biomaterial capable of addressing multiple pathogenic pathways in AGA simultaneously. [34–36]

Mechanisms of Action of PRF in Hair Follicle Regeneration

One of the primary mechanisms through which PRF contributes to hair follicle regeneration is its ability to stimulate dermal papilla cell (DPC) proliferation and prolong anagen duration. The sustained release of key growth factors—including PDGF, VEGF, IGF-1, and FGF-7—plays a central role in activating intracellular pathways that support follicular survival and cycling. VEGF enhances perifollicular angiogenesis, ensuring a richer microvascular environment essential for active anagen follicles, while IGF-1 promotes DPC proliferation and reduces apoptosis. In vitro experiments have shown that PRF-conditioned media significantly upregulate β-catenin and Wnt signaling, pathways critical for hair follicle activation and regeneration. These molecular effects mirror the pathways suppressed in AGA, making PRF a biologically targeted therapy. [37–39]

PRF also exerts potent anti-inflammatory actions that counteract the perifollicular microinflammation observed in AGA. Leukocytes embedded within the PRF matrix release regulatory cytokines such as IL-4, IL-10, and TGF-β, which modulate inflammatory cascades and help restore homeostasis within the follicular microenvironment. This attenuation of inflammation may prevent progressive perifollicular fibrosis, a contributing factor in long-term follicular miniaturization. Additionally, PRF has demonstrated an ability to reduce levels of reactive oxygen species and oxidative stress markers in treated tissues. Given that oxidative injury plays a role in stem cell depletion in AGA, these antioxidant effects enhance the potential for follicular recovery and improved regenerative response. [40–42]

Another crucial mechanism is PRF's ability to enhance angiogenesis and extracellular matrix (ECM) remodeling. The fibrin scaffold created during PRF formation mimics physiologic wound healing, supporting fibroblast migration, collagen synthesis, and neovascularization. Angiogenesis is indispensable for robust anagen maintenance, and several studies highlight the increased expression of VEGF and CD31-positive microvessels following PRF application. ECM proteins contained within PRF, such as fibronectin and vitronectin, further facilitate hair follicle anchorage and structural integrity within the dermis. This combination of growth factor signaling, improved vascularity, and ECM

remodeling creates a supportive niche that promotes the regeneration of miniaturized follicles. [43–44] Finally, the cellular components of PRF—including leukocytes, platelets, and circulating progenitor cells—contribute synergistically to tissue regeneration. Circulating CD34+ progenitor cells present in PRF have been shown to enhance angiogenesis and tissue repair, potentially augmenting follicular revitalization. Furthermore, the slow polymerization process of PRF preserves cell viability and improves the homing capacity of regenerative cells once injected into scalp tissue. This cellular synergy allows PRF to work across multiple levels of AGA pathophysiology, addressing inflammation, vascular dysfunction, follicular signaling deficits, and ECM remodeling. Such multi-targeted action distinguishes PRF from single-mechanism pharmacologic therapies and underlies its emerging role in regenerative hair medicine. [45–46]

Clinical Evidence for PRF in Androgenetic Alopecia

Clinical studies evaluating platelet-rich fibrin (PRF) in androgenetic alopecia (AGA) have grown rapidly in recent years, demonstrating encouraging results in both male and female patients. Early pilot studies using injectable PRF (i-PRF) showed improvements in hair density, thickness, and overall scalp coverage within 3–4 months of treatment. For example, Sclafani and colleagues reported that PRF injections led to measurable increases in hair caliber and patient satisfaction, suggesting a biologic advantage over PRP due to prolonged growth factor availability. These observational findings provided the foundation for more structured clinical trials and positioned PRF as a viable regenerative option for pattern hair loss. [47–48]

Randomized controlled trials have begun to solidify the evidence base for PRF in AGA. Gentile and Garcovich conducted one of the first comparative studies, demonstrating that both PRP and PRF improved hair density, but PRF produced significantly greater increases in hair count and shaft thickness at 3 and 6 months. The authors attributed this superiority to PRF's fibrin architecture and slow-release kinetics, which sustain follicular stimulation over a longer period. The study also noted that PRF required fewer treatment sessions to achieve comparable or superior outcomes, highlighting potential advantages in patient compliance and cost-effectiveness. Similar findings were reported in split-scalp studies where PRF outperformed PRP on global photography and trichoscopy. [49–50]

Emerging clinical trials have evaluated advanced PRF formulations, such as A-PRF and i-PRF, with consistent demonstration of improved anagen/telogen ratios and increased follicular activity on trichoscopic analysis. A study by El-Sheikh et al. showed that i-PRF significantly increased hair density and anagen hair percentage after three monthly sessions in male AGA patients, with no major adverse effects. These results suggest that low-speed centrifugation techniques, which preserve leukocytes and progenitor cells, may enhance therapeutic outcomes. Collectively, the literature indicates that PRF is effective across multiple AGA severities and may be particularly beneficial in early to moderate disease stages where miniaturized follicles remain viable. [51–52]

In addition to quantitative improvements, PRF therapy demonstrates a favorable safety profile. Across multiple clinical reports, adverse events have been limited to transient erythema, mild edema, and injection-site tenderness, with no reports of scarring, infection, or paradoxical shedding beyond expected telogen effluvium. Patients generally report high satisfaction levels, attributed to the autologous nature of PRF and its lack of chemical additives. These safety characteristics make PRF suitable for repeated sessions and long-term maintenance therapy, an important consideration in chronic conditions such as AGA. However, the current evidence remains limited by small sample sizes, short follow-up durations, and heterogeneity in preparation protocols, underscoring the need for large-scale, standardized trials. [53–54]

Conclusion

Platelet-rich fibrin (PRF) has emerged as a significant advancement in the field of regenerative dermatology and andrology, offering a biologically sophisticated and clinically promising approach for the management of androgenetic alopecia (AGA). Its unique composition—featuring a natural fibrin matrix enriched with platelets, leukocytes, and progenitor cells—provides prolonged and

physiologically regulated release of growth factors, setting it apart from earlier platelet concentrates such as PRP. By targeting multiple pathogenic mechanisms simultaneously, including follicular miniaturization, perifollicular microinflammation, vascular compromise, oxidative stress, and extracellular matrix deterioration, PRF addresses the multifaceted nature of AGA more comprehensively than conventional pharmacologic treatments.

Clinical evidence to date supports the efficacy of PRF in improving hair density, thickness, anagen/telogen ratio, and global appearance in both male and female pattern hair loss. Its favorable safety profile, minimal invasiveness, and autologous origin enhance patient acceptability and make it suitable for long-term use. Additionally, emerging data suggest that PRF may outperform PRP in both short- and mid-term outcomes, likely due to its superior biologic properties and sustained release kinetics. This advantage has positioned PRF as a preferred option among regenerative injectables, particularly for patients seeking non-pharmacologic therapies or wishing to augment standard treatments.

However, despite its promise, important gaps remain. Variability in preparation protocols, centrifugation speeds, and injection techniques limits comparability across studies and hinders the development of universal clinical guidelines. Moreover, most available studies feature small cohorts and short follow-up durations, underscoring the need for large-scale randomized controlled trials to validate long-term efficacy and optimal treatment regimens. Future research should also explore combination therapies, biomarkers of response, and the molecular effects of different PRF formulations to refine patient selection and maximize therapeutic benefit.

In summary, PRF represents a meaningful step forward in biological hair restoration for AGA, combining mechanistic plausibility with encouraging clinical outcomes. With continued investigation and protocol standardization, PRF has the potential to become an integral component of evidence-based, multimodal AGA management in dermatology and andrology.

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