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REVIEW ARTICLE

Autologous Platelet-Rich Plasma for the Treatment of Pattern Hair Loss

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Abstract Platelet-rich plasma (PRP) is a solution derived from whole blood that is enriched in the platelet fraction. Platelets serve as a reservoir of growth factors and cytokines. When platelets are activated in vivo, signaling molecules are released into the immediate microenvironment and activate receptors for various pathways. Historically, PRP has been applied to wound beds to promote healing of complex wounds. Over the last decade, it has served as a valuable therapeutic tool in various specialties such as maxillofacial surgery, plastic surgery, orthopedics and sports medicine. Only recently has PRP been utilized for dermatologic purposes, more specifically, for the treatment of male and female pattern hair loss. In this review, we discuss molecular and cellular pathways upregulated by PRP important in hair folliculogenesis, and examine clinical evidence from all previously published studies involving the use of PRP for pattern hair loss.

Key Points

Introduction of platelet-rich plasma (PRP) into the microenvironment of the hair follicle through multiple intradermal injections is increasingly being used as mesotherapy for pattern hair loss.

The use of PRP for the treatment of alopecia is in its nascent stages, but evidence from clinical studies over the last 3 years is promising.

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1 Introduction

Platelet-rich plasma (PRP) was initially used in the 1990s as adjuvant therapy to treat chronic non-healing wounds. In a meta-analysis of PRP use in advanced wound healing, it was determined that PRP significantly enhanced complete healing of chronic wounds and reduced infections in acute wounds compared with standard wound care. Furthermore, topical application of PRP, in addition to standard treatment, shortened healing time and accelerated wound healing velocity compared with standard treatment alone [1].

Over the last decade, PRP's healing and regenerative properties have been utilized in the fields of plastic and reconstructive surgery, oral surgery, dentistry, ophthalmology, and hair transplantation. A systematic review demonstrated a substantial benefit of PRP in increasing the survival rate of fat grafts used for reconstructive plastic surgery and enhancing bone graft regeneration [2]. PRP has been used successfully for avascular osteonecrosis of the jaw from bisphosphonate therapy, mandibular continuity reconstruction and cleft palate surgery [2, 3]. When PRP is applied to the alveolar sockets after tooth extractions, there is observed improvement in soft tissue healing. PRP in the form of eye drops has been recently used by ophthalmologists for the treatment of corneal lesions [4]. PRP has also been shown to significantly increase the yield of transplanted follicular units in hair transplant surgery [5].

Within the last 5 years, the use of PRP has transitioned to the field of medical dermatology for conditions such as leprosy, ulcers secondary to necrobiosis lipoidica, melasma, and, lately, hair loss [6–8]. PRP combined with adipose-derived mesenchymal cells has been used to regenerate vulvar structures in patients with lichen

sclerosis [9]. Budamakuntla et al. recently published a case series of pyoderma gangrenosum treated with PRP [10]. Over the last 3 years, introduction of PRP into the microenvironment of the hair follicle through multiple intradermal injections has been used as mesotherapy for hair loss. The number of manuscripts on this technique is increasing rapidly. This manuscript will review the existing body of knowledge on the use of PRP for pattern hair loss.

2 Methods

Articles were searched using PubMed and EMBASE with the following search terms: "platelet-rich plasma", "platelet-rich plasma gel", "platelet-rich fibrin matrix", "mesotherapy", "androgenetic alopecia", "female pattern hair loss", "male pattern hair loss", "alopecia", and "hair loss". Results were filtered to include only those articles relevant to this review and written in English. References were used to search for more articles that were relevant. A total of 11 clinical studies were found that used PRP as a therapeutic tool for pattern hair loss.

3 Platelet-Rich Plasma

3.1 Function

Platelets in PRP increase the levels of growth factors in the microenvironment by releasing them from intracellular stores. Once platelets are activated, they secrete growth factors such as platelet-derived growth factor (PDGF), transforming growth factor β (TGF-β), vascular endothelial growth factor (VEGF), and insulin-like growth factor (IGF-1) from alpha granules. A list of these growth factors and their functions can be found in Table 1 [11, 12]. Growth factors serve as mitogens for cells such as endothelial cells and fibroblasts, promoting angiogenesis and fibroblast differentiation and proliferation. Growth factors also regulate collagen synthesis and immune cell differentiation and proliferation. Within the first hour after activation, greater than 95 % of pre-synthesized growth factors are released in an initial burst. Platelets continue to synthesize growth factors for another 7 days [13].

3.2 Pathways

Pathways modulated by PRP in hair folliculogenesis and cycling are only partially understood (Fig. 1). Growth factors in PRP bind to their respective receptor, triggering a complex downstream signaling cascade affecting cellular growth and survival. One important downstream event is the activation of phosphoinositide 3-kinases (PI3Ks), which are important regulators of cellular proliferation and differentiation. PI3Ks phosphorylate phosphatidylinositol 4,5-bisphosphate (PIP2) to phosphatidylinositol 3,4,5-triphosphate (PIP3). PIP3 then acts as a secondary messenger and activates AKT, an anti-apoptotic signaling molecule. AKT, also known as protein kinase B, is a serine and threonine protein kinase that activates downstream signalling pathways leading to cellular growth, metabolism and angiogenesis. Apoptosis or programmed cell death is

Table 1 Select platelet growth factors and their biologic functions [11, 12]

Growth factor	Biologic function relevant to hair folliculogenesis
Platelet-derived growth factor α and β (PDGF-α and -β)	Mitogenic factor for mesenchymal cell differentiation
	Stimulates fibroblast and smooth muscle cell chemotaxis and mitogenesis
	Regulates collagenase secretion and collagen synthesis
	Stimulates macrophage and neutrophil chemotaxis
Transforming growth factor α and β (TGF-α and -β)	Mitogenic factor for mesenchymal cell differentiation
	Regulates endothelial and fibroblast mitogenesis
	Regulates collagenase secretion and collagen synthesis
	Stimulates endothelial chemotaxis and angiogenesis
	Inhibits macrophage and lymphocytes proliferation
Vascular endothelial growth factor (VEGF)	Increases angiogenesis and vessel permeability
	Mitogenic factor for endothelial cell differentiation
Epidermal growth factor (EGF)	Mitogenic factor for mesenchymal cell differentiation
	Stimulates endothelial cell chemotaxis and angiogenesis
	Regulates collagenase secretion
Fibroblast growth factor (FGF)	Mitogenic factor for mesenchymal cell differentiation
Connective tissue growth factor (CTGF)	Promotes platelet adhesion
Insulin-like growth factor-1 (IGF-1)	Stimulates chemotaxis of fibroblasts and stimulates protein synthesis

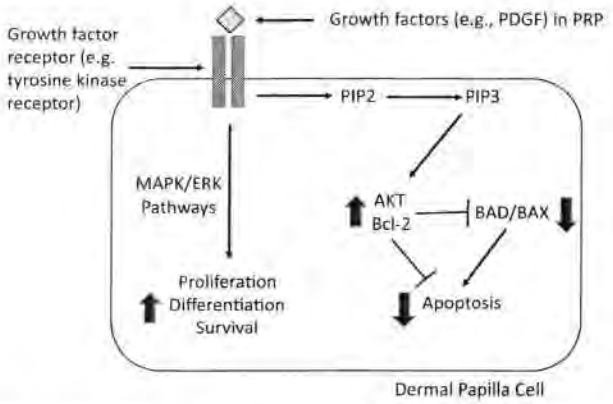

Fig. 1 Pathways modulated by PRP in hair folliculogenesis and cycling. Growth factors in PRP bind to receptors activating the MAPK/ERK pathways leading to proliferation, differentiation, and cellular survival. Furthermore, upon receptor binding, secondary signaling molecule PIP3 activates AKT, leading to inhibition of pro-apoptosis molecules BAD and BAX, leading to an overall decrease in apoptosis of dermal papilla cells. Bcl-2, an anti-apoptosis signaling molecule, which is upregulated in dermal papilla cells treated with PRP, also inhibits BAD and BAX. *BAD* Bcl-2-associated death protein, *BAX* Bcl-2-associated X protein, *ERK* extracellular signal-regulated kinases, *MAPK* mitogen-activated protein kinases, *PDGF* platelet-derived growth factor, *PIP2* phosphatidylinositol 4,5-bisphosphate, *PIP3* phosphatidylinositol 3,4,5-triphosphate, *PRP* platelet-rich plasma

regulated by a family of proteins that either activate apoptosis [Bcl-2-associated death protein (BAD) and Bcl-2-associated X protein (BAX)] or inhibit apoptosis (Bcl-2). Activated AKT then phosphorylates BAD, inactivating it, thereby leading to an overall increase in cellular survival and growth. Furthermore, increased expression of Bcl-2 and decreased expression of BAD and BAX have been observed in in vitro cultures of human dermal papilla (DP) cells treated with PRP, leading to the overall inhibition of apoptosis and increased cell survival [14]. Therefore, the net effect of PRP on hair cycling may be the prolongation of the anagen phase through inhibition of apoptosis.

The mitogen-activated protein kinases (MAPK) pathway, otherwise known as the extracellular signal-regulated kinases (ERK) pathway, is upregulated by growth factors in PRP. In one study, phosphorylated or activated ERK was found to be significantly upregulated in human DP cells treated with PRP [14]. In this pathway, growth factors such as epidermal growth factor (EGF) or PDGF bind to their respective transmembrane tyrosine kinase-associated receptors, leading to a complex intracellular signaling cascade. One important downstream target is ERK/MAPK, which, once activated, promotes transcription of genes involved in cellular proliferation, differentiation, and survival.

4 Clinical Use

4.1 Preparation

The first step in preparing PRP involves drawing approximately 15–60 cubic centimeters (cc) of whole blood (WB) on the day of treatment. The addition of an anticoagulant such as citrate dextrose prevents coagulation and premature discharge of platelet granules. In a two-step centrifugation process or "double-spin" method, the WB is centrifuged at a constant rate to form three layers: a bottom layer containing red blood cells (RBCs), a central "buffy layer" containing white blood cells (WBCs), and an uppermost layer containing platelets suspended in plasma. Next, the top layer and the buffy layer (depending on the leukocyte fraction preferred in the final isolate) are transferred and centrifuged in the second spin, resulting in a pellet of platelets. The pellet is reconstituted in plasma to a total volume of 3–5 mL, depending on the desired volume per treatment.

Alternatively, a single centrifugation spin can be used. This can produce higher platelet yields, but may result in more WBC and RBC contaminants. A subsequent leukocyte filtration step can increase platelet purity. Once PRP is processed, some users will add a platelet activator such as calcium gluconate to hasten release of granules containing growth factors 5–30 min before use. If not added, dermal collagen and thrombin are natural activators of platelets and endogenously activate platelets after injection into the scalp.

There is wide variation in the reported protocols for preparing PRP, outlined in Table 2 [15–25]. There are multiple commercially available kits containing centrifuges and other components necessary for the preparation of PRP. The volume of WB drawn differs between reports, as does the number and force of centrifugations and the ultimate mean platelet concentration in PRP compared with WB, called the platelet concentration factor (PCF). At this point in time there is no single method of preparation that is more advantageous. The PCFs according to Table 2 ranged from 3.5 to over 6, with no evident optimal value among the studies. Amable et al. performed optimization studies to establish a reproducible protocol for PRP preparation that resulted in 5.4- to 7.3-fold increases in platelet concentration compared with WB. Their enrichment protocol resulted in a final PRP solution containing 1.4–1.9 million platelets/μL [26]. A study also demonstrated that the optimal platelet concentration for the induction of angiogenesis of human endothelial cells is 1,500,000 platelets/μL, where higher concentrations decreased angiogenesis [27].

Table 2 Comparison of protocols for PRP in pattern hair loss

Study	Preparation	Volume of whole blood (mL)	1st centrifugation		2nd centrifugation		Platelet concentration factor[a]
			Force (g)	Time (min)	Force (g)	Time (min)	
Singhal et al. [15]	Not reported	20	1500	6	2500	15	Not reported
Gentile et al. [16]	Cascade-Selphyl-Esforax system (Aesthetic Factors, LLC, Wayne, NJ, USA) with modifications	18	1100	10	–	–	Not reported
	Platelet Rich Lipotransfert system (Corios Soc. Coop, San Giuliano Milanese, Italy) with modifications	60	1200	10	–	–	
Marwah et al. [17]	Not reported						
Khatu et al. [18]	Not reported	20	1500	6	2500	15	Not reported
Cervelli et al. [19]	Cascade-Selphyl-Esforax system	18	1100	10	–	–	Not reported
Gkini et al. [20]	RegenKit BCT-3 (Regenlab, NY, NY)	16	1500	5	–	–	5.8
Kang et al. [21]	SmartPReP2 (Harvest Technologies Corp., Plymouth, MA, USA)	60	Not reported				5.9
Schiavone et al. [22]	Autologous leukocyte-PRP (GPS III Platelet Separation System, Biomet, Warsaw, IN, USA)	60	Not reported				3.5-4
	Autologous plasmatic protein solution (Glo PRP, Glofinn Oy, Glotec Korea)	40	Not reported				4
Sclafani [23]	Selphyl (Aesthetic Factors, Inc., Wayne, NJ, USA)	18	1110	6	–	–	Not reported
Betsi et al. [24]	ACR-C Extra kits (RegenLab SA, Switzerland)	16	1500	5	–	–	Not reported
Takikawa et al. [25]	Nipro Pharma, Osaka, Japan	15	1700	15	3000	5	6.13

The symbol "–" indicates that the protocol used in the respective study does not include a second centrifugation step

ACR-C autologous cellular regeneration-classic, *BCT* blood cell therapy, *g* gravity, *GPS* gravitational platelet separation, *LLC* limited liability company, *PRP* platelet-rich plasma

[a] Ratio of mean platelet concentration in PRP formulation and mean platelet concentration in peripheral blood

A sample protocol of PRP for treatment of pattern hair loss uses the Healeon Medical PRP System (Healeon Medical Inc., Newbury Park, CA, USA). In this single centrifugation spin system 10 cc of peripheral blood is centrifuged for 10 min at 3500 rpm. The PCF of the final PRP ranges from 5 to 8. The volume of PRP injected per treatment is 6 cc over the affected areas on the scalp. Three treatments are recommended 2 months apart for at least 6 months.

4.2 Enhancement

Biologically active molecules and cells have been added to PRP in an attempt to augment its efficacy. Takikawa et al. added microparticles of dalteparin/protamine (D/P MPs) to PRP, which serve as a scaffold for growth factors and release them slowly into the injected areas. While they found no significant difference between PRP with D/P MPs and PRP alone in the mean number of hairs observed after treatment, there they did observe an increase in hair shaft diameter [25]. Kang et al. enriched the CD34+ cells in PRP; however, they did not compare this formulation to PRP, but instead compared it to placental extract. Furthermore, they treated all male patients with 1 mg finasteride in addition to PRP, with no PRP alone control group for comparison [21].

4.3 Injection Technique

PRP is administered into the frontal, parietal or temporal scalps through a series of subcutaneous or intradermal injections in a grid-like pattern. The optimal volume of PRP injected per treatment, the time interval between treatments, and the total number of treatments required is unclear. Table 3 outlines the different dosing regimens that have been used. Total volumes injected per session ranged from 0.8 to 12 cc and have been reported as 0.05–0.1 cc per cm^2 of treated scalp. The time interval between treatments ranged from 2 weeks to 1 month and the number of treatments ranged from 2 to 5.

Table 3 Summary of studies using autologous PRP for the treatment of pattern hair loss

Study	*N* (M/F)	Formulation	Number of treatments	Interval between treatments	Major results	Adverse reactions
Singhal et al. [15]	10 (8/2)	Autologous PRP, 8–12 cc per treatment	4	2 weeks	10/10 patients treated with PRP had improvement on global images compared with no improvement in control subjects 65 % positive hair pull reduction in treated group vs. no reduction in control group	Mild headache
Gentile et al. [16]	23 (23/0)	Autologous PRP, 0.1 cc/cm^2 per treatment	3	4 weeks	Significant increase in mean hair count, total hair density and terminal hair density compared with baseline Increase in epidermal thickness and increase in number of hair follicles in PRP-treated skin compared with baseline ($p < 0.05$) Increase in Ki67+ basal keratinocytes in epidermis and hair follicular bulge cells compared with baseline ($p < 0.05$) Increase in small blood vessels around hair follicles in treated skin compared with baseline ($p < 0.05$) 4 patients reported progressive hair loss that was more evident 16 months after the last treatment	None observed
Cervelli et al. [19]	10 (10/0)	Autologous PRP, 0.1 cc/cm^2 per treatment	3	4 weeks	Statistically significant increase in mean hair count and hair density compared with baseline at 3 months, with an increase of 18.0 hairs in a target area ($p < 0.0001$) Increase in epidermal thickness and number of hair follicles compared with baseline at 3 months ($p < 0.05$) Increase in Ki67+ basal keratinocytes of epidermis and follicular bulge cells compared with baseline ($p < 0.05$) Slight increase of blood vessels around hair follicles in PRP-treated skin ($p < 0.05$)	None observed
Gkini et al. [20]	20 (18/2)	Autologous PRP activated with calcium gluconate, 0.05–0.1 cc/cm^2	3	3 weeks, booster at 6 months	Hair density (hair/cm^2) increased compared with the onset of therapy at every time point ($p < 0.001$) Highest hair density peaked at 3 months then declined at 6 and 12 months High patient satisfaction (7.1 on a scale 1–10) 85 % reported improvement of hair quality and thickness	Mild pain Scalp sensitivity

Table 3 continued

Study	*N* (M/F)	Formulation	Number of treatments	Interval between treatments	Major results	Adverse reactions
Kang et al. [21]	26 (15/11)	Autologous PRP-containing CD34+ cells, 0.05–0.1 cc/cm^2, 1 mg of finasteride was initiated in M patients	2	3 months	Increase in mean number of hairs, hair thickness and mean two-point scores in both cohorts compared with baseline values at 3 and 6 months ($p < 0.0001$) CD34+ PRP cohort had higher degree of improvement than placental extract treatment in hair thickness ($p = 0.027$) and two-point score ($p = 0.023$), but not in hair count ($p > 0.05$)	Pain Transient erythema and edema Folliculitis in placental extract treatment group
Schiavone et al. [22]	64 (42/22)	(A) Autologous leukocyte-PRP; (B) autologous plasmatic protein solution	4 injections of A, followed 3 months later with 4 injections of B	3 months	Observed improvement in 62/64 patients by evaluator 1 and 64/64 patients by evaluator 2	None observed
Sclafani [23]	15 (9/6)	Autologous PRFM or PRFM activated by $CaCl_2$ 0.1 mL of PRFM per 5–8 mm	3	4 weeks	Hair density indices increased at 2 ($p = 0.0031$) and 3 ($p = 0.0277$) months after the initial treatment compared with baseline Hair density index decreased at 6 months and was not statistically different than baseline ($p = 0.0606$)	None observed
Khatu et al. [18]	11 (11/0)	Autologous PRP, 2–3 cc	4	2 weeks	Average gain of 22.09 follicular units per cm^2 7 out of 10 on patient satisfaction score	Minimal pain and redness Pinpoint bleeding
Marwah et al. [17]	10 (10/0)	Autologous PRP, unknown volume	6	1 week	Improvement in global pictures in 2/10 patients All patients were satisfied with treatment	None observed
Betsi et al. [24]	42 (34/8)	Autologous PRP, 8–12 cc	5	2-month treatment period	Hair pull test after last injection was negative (averaging 3 hairs) in all subjects, which represented a statistically significant decrease from pretreatment values ($p < 0.01$) Subjective increase in hair volume and quality in global pictures High patient satisfaction (7.0 on a scale 1–10)	Drowsiness Sensible [sic] scalp
Takikawa et al. [25]	26 (16/10)	Autologous PRP mixed with D/P MPs designated as PRP&D/P MPs, 3 mL of PRP&D/P MP or PRP	5	Treatment at 0, 2, 4, 6 and 9 weeks	No significant difference between PRP&D/P MPs and PRP-injected groups in terms of mean number of hairs observed at 12 weeks Increase in mean cross-section of hairs in PRP&D/P MPs and PRP groups vs. placebo ($p < 0.01$) Observed thicker epithelium, proliferation of collagen fibers and fibroblasts and greater numbers of blood vessels around hair follicles in PRP&D/P MPs and PRP groups vs. placebo (no statistic)	Temporary pain

cc Cubic centimeters, *D/P MPs* dalteparin/protamine microparticles, *F* female, *M* male, *PRFM* platelet-rich fibrin matrix, *PRP* platelet-rich plasma

4.4 Efficacy

Methods used to measure the efficacy of treatment with PRP for alopecia varied between the studies reviewed. Efficacy was evaluated using both subjective and objective measures, including assessing for improvement on global pictures, hair pull test before and after treatment, mean hair count or hair density before and after treatment, and patient satisfaction surveys. A summary of the efficacy of PRP in pattern hair loss in the 13 studies reviewed can be found in Table 3 [15–25].

Evidence suggests that treating hair loss at earlier stages with PRP may be more beneficial than treating at later stages. In the majority of studies, the degree of hair loss was classified using the Norwood-Hamilton scale for men and the Ludwig scale for women. Generally, more male subjects were included in the studies than females; 196 (76 %) were male and 61 (24 %) were female. Unfortunately, efficacy of treatment was not reported for each gender, making differences between male and female pattern baldness difficult to discern. Betsi et al. found less improvement in hair regrowth in patients with marked alopecia, or stages VI–VII of the Norwood-Hamilton scale [24]. Although these authors did not report efficacy data per the Norwood-Hamilton scale, they generally found that improvement was more obvious for patients who had alopecia for less than 2 years and earlier stages of alopecia. Furthermore, Gkini et al. found that male patients with stage II–III alopecia had increased hair density and improvement on global pictures compared with patients with more advanced alopecia [20]. Despite evidence to support treating male patients at earlier stages of alopecia than treating at later stages, one study did not find that the Norwood-Hamilton scale was predictive of success [23].

The majority of the clinical studies presented here have not followed the patient's clinical course for longer than 6 months. However, several studies observed a declining effect with long-term follow-up after the last treatment. Gkini et al. found the highest density of hair at 3 months after treatment, but noted that hair density declined at 6 and 12 months after the last treatment. They proposed a "booster" treatment at 6 months [20]. Sclafani determined that hair density indices increased at 2 and 3 months after treatment compared with baseline, but declined at 6 months to a value statistically insignificant from baseline indices [23]. Gentile et al. followed patients 12 months after their last treatment and noted that four out of 20 male patients reported progressive hair loss, which was more evident at 16 months [16]. Betsi et al. recommended a series of five sessions every 3 weeks for a total of 15 weeks, repeated twice a year, to maintain results [24]. Results from a representative patient can be seen in Fig. 2.

Although histologic data is limited, evidence does suggest that PRP increases epidermal proliferation, stimulates growth of follicular bulge cells and induces angiogenesis, although it is unclear how these are interrelated. Cervelli et al. performed a 3-mm punch biopsy of the scalp on male patients at baseline and 2 months after the last PRP treatment. They observed an increase in the epidermal thickness and an increase in the number of hair follicles and blood vessels around hair follicles with treatment compared with baseline [19]. Cervelli et al. observed an increase in expression of Ki67, a nuclear protein that serves as a marker for proliferation, in basal keratinocytes and follicular bulge cells in PRP-treated samples. The significance of increased Ki67 expression in basal keratinocytes is unclear; however, increased expression in the follicular bulge cells could signify hair folliculogenesis. Takikawa et al. performed scalp biopsies at baseline and after treatment with PRP alone or PRP and D/P MPs and observed a thicker epidermis, an increase in collagen fibers, an increased number of fibroblasts and an increase in blood vessels around hair follicles after treatment, although no statistics were reported [25]. Gentile et al. observed increased

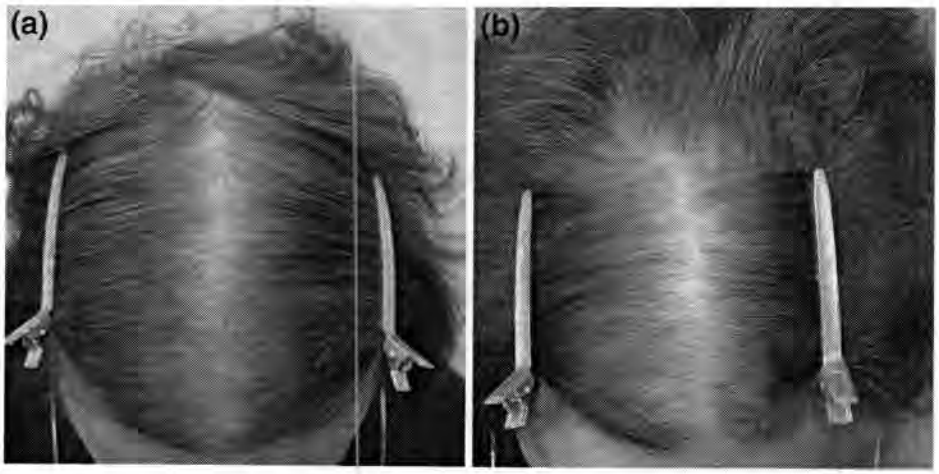

Fig. 2 A 76-year-old woman with female pattern hair loss pre-treatment (**a**) and 6 months later, following three treatments with platelet-rich plasma (**b**)

epidermal thickness and an increased number of hair follicles in treated areas compared with controls. They also observed an increase in Ki67+ basal keratinocytes and follicular bulge cells in treated scalp compared with baseline ($p < 0.05$) [16].

4.5 Side Effects

All reported side effects in the reviewed studies were mild and temporary. The most commonly reported side effect was short-lived pain after injection. Some studies pretreated the scalp with injections of lidocaine, although one study mentioned that patients requested this be discontinued because of increased discomfort [21, 22, 24]. There were no reports of bacterial, viral, or mycobacterial infections, folliculitis, panniculitis, hematoma or seroma formation, increased hair loss such as telogen effluvium, changes in nerve sensation or scarring.

4.6 Patient Satisfaction

Four studies reported on patient satisfaction with PRP treatment of pattern hair loss [16–18, 24]. Three of these reported a satisfaction rate of 7 on a 10-point scale [16, 18, 24]. However, satisfaction did not always correlate with efficacy. Marwah et al. reported that only two out of ten patients had improvement on review of global photographs, yet all patients were satisfied with their treatment and results [17].

4.7 Feasibility

The greatest expense in using PRP as treatment for hair loss is the cost to generate the PRP itself. Most clinicians use a commercially available kit, which includes centrifugation tubes, syringes, phlebotomy butterfly needles and treatment needles. Prices per kit range from US$100.00 to US$250.00. Kits that include a centrifuge cost more. Additional costs include those related to the specialized ancillary staff and training required for phlebotomy and preparation of PRP. If standardization of platelet concentration is desired, an automated or manual cell counter and a light microscope are necessary. Costs can be decreased by purchasing a standard centrifuge separately from the kit. Further cost savings can be achieved by purchasing bulk quantities of sterile centrifuge tubes, vacuumed venous blood collection tubes, phlebotomy kits and 27- or 30-gauge needles.

Numerous obstacles must be overcome before the widespread implementation of PRP for hair loss. A major obstacle is the lack of evidence from randomized clinical trials, without which a standardized PRP preparation and treatment protocol cannot be readily established. More studies are needed to further elucidate the efficacy and safety of this treatment modality before the Food and Drug Administration (FDA) will grant approval of PRP for hair loss. A major obstacle for patients to undergo this treatment is the lack of insurance coverage and cost. Without FDA approval and proven efficacy, insurance coverage is unlikely.

5 Conclusions

The use of PRP for the treatment of alopecia is in its nascent stages, but evidence from clinical studies over the last 3 years is promising. There do not appear to be any safety issues, and side effects are minimal. However, preparation protocols, dosage, injection technique, and treatment schedules vary. While injections of PRP do appear to be beneficial, assessment of improvement has not been standardized. Additional randomized clinical trials with larger cohorts and objective measures of efficacy are needed. Such studies would provide insight into optimal PCF and frequency of injections, as well as the duration of treatment necessary for sustainable results.

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Compliance with Ethical Standards

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Conflict of interest Babu Singh and Lynne J. Goldberg have no conflicts of interest to declare.

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