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ADVANCEMENTS IN TREATMENT STRATEGIES FOR ANDROGENETIC ALOPECIA: NOVEL THERAPIES AND FUTURE PERSPECTIVES

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ABSTRACT

Introduction and Purpose: Androgenetic alopecia (AGA), or male pattern hair loss (MPHL) and female pattern hair loss (FPHL), is the most common form of progressive hair loss, significantly affecting men and women. FDA-approved treatments, such as minoxidil and finasteride, have limitations, including side effects and variable efficacy, highlighting the need for innovative solutions. Recent advancements introduce therapies targeting AGA's mechanisms, offering hope for improved management.
Materials and Methods: A comprehensive literature review was conducted using PubMed, Web of Science, Embase, and Google Scholar. The search included keywords such as "androgenetic alopecia," "male pattern hair loss," "female pattern hair loss," "minoxidil," "finasteride," "dutasteride mesotherapy," "platelet-rich plasma" and "botulinum toxin." Articles from the last five years were prioritized.
State of Knowledge: Emerging therapies include low-dose oral minoxidil, topical finasteride, platelet-rich plasma (PRP), mesotherapy, low-level laser therapy (LLLT), and botulinum toxin, offering improved adherence and fewer side effects. Experimental approaches, such as clascoterone, pyrilutamide, and stem cell-based therapies, focus on follicular regeneration and androgen receptor modulation. Advanced technologies like PROTAC and AI-driven designs aim for precision and minimal invasiveness. However, variability in studies and the lack of standardized protocols limit generalizability.
Conclusions: Recent advancements in AGA therapies demonstrate significant potential, offering individualized, effective, and minimally invasive options. Standardized protocols and large-scale trials remain essential to validate efficacy and safety, paving the way for precision medicine in AGA management.

KEYWORDS

Androgenetic Alopecia, Male Pattern Hair Loss, Female Pattern Hair Loss, Minoxidil, Finasteride, Dutasteride Mesotherapy

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Introduction and purpose

Androgenetic alopecia (AGA), also referred to as male pattern hair loss (MPHL) or female pattern hair loss (FPHL), is the most common type of progressive hair loss [1]. Prevalence data indicate that AGA affects approximately 30% of white men by the age of 30, 50% by age 50, and 80% by age 70 [2]. The incidence of FPHL in Caucasian women increases with age, ranging from 3% to 12% in women aged 30–40, to 14% to 28% in postmenopausal women in their fifties, and 29% to 56% in women over 70 years [3].

The clinical presentation of male pattern hair loss is typically distinct and easily identifiable, with its pattern of hair loss progressing systematically as extensively documented by Hamilton and Norwood [4]. In men, androgenetic alopecia is marked by hair loss at the temples, resulting in bitemporal recession, as well as thinning or baldness at the vertex [5]. In contrast, female pattern hair loss (FPHL) presents as diffuse hair thinning and decreased density over the frontal and vertex areas of the scalp, with the frontal hairline typically remaining intact [6]. The distinct distribution of androgen receptors in the scalp determines the pattern of hair loss in each sex, although androgens play a key role in both [7].

Androgenetic alopecia (AGA) is characterized as a dihydrotestosterone (DHT)-dependent condition, involving the progressive miniaturization of susceptible hair follicles [8]. The follicular miniaturization theory attributes the reduction in hair density in AGA to a gradual shortening of the anagen phase and the upward migration of follicles from the reticular to the papillary dermis with each hair cycle. Over time, larger follicular units with terminal hairs transform into smaller units with vellus hairs [9].The primary androgen in systemic circulation is testosterone [10]. Cytoplasmic 5α-reductase (5αR) converts it to the more potent androgen, dihydrotestosterone (DHT) [1]. The absence of AGA in individuals with a deficiency of steroid 5α-reductase (5AR) type II emphasizes the essential role of DHT in the condition's pathogenesis [11]. Although the association between dihydrotestosterone (DHT) and male androgenetic alopecia is well-established, the specific involvement of androgens in female AGA (FPHL) remains unclear [3]. In women with FPHL, only about one-third exhibit elevated androgen levels, indicating that abnormal androgen concentrations are not a universal feature of the condition [12]. Elevated serum androgen levels in women often result from various endocrine disorders, with polycystic ovary syndrome (PCOS) being the most prevalent underlying cause [13]. It has been suggested that increased androgen sensitivity in peripheral tissues may explain cases where androgen levels are within normal limits. [14].

The contribution of genetic factors to the development of androgenetic alopecia (AGA) has been widely explored in the literature [15]. Research in genetic epidemiology indicates that first-degree relatives of individuals with AGA have a higher risk of developing this condition. Lifetime risk estimates show that siblings, parents, and children of affected individuals face risks of 7.1%, 7.8%, and 5.7%, respectively, compared to a 2% lifetime risk in the general population [16]. The heritability of androgenetic alopecia has been determined to be substantial, with estimates of approximately 80% derived from two independent studies involving twins [17, 18]. It is well-established that the inheritance of androgenetic alopecia (AGA) follows a polygenic model, involving multiple independent genes. The AR/EDA2R locus on the X chromosome was the first genetic region identified as a risk factor associated with AGA [19]. Furthermore, AGA has been reported in prepubertal children, aged 6 to 8 years. In these cases, genetic factors are thought to play a significant role in the development of the condition [20].

The FDA has approved only two treatments for androgenetic alopecia: topical minoxidil in concentrations of 2% and 5% and oral finasteride at a dose of 1 mg daily for men. For women, only topical minoxidil is approved [21]. Multiple non-FDA-approved therapies have demonstrated effectiveness in treating androgenetic alopecia (AGA) across various studies. Several of these treatments have emerged recently and still require further investigation, emphasizing the importance of conducting an updated review of the existing literature. This comprehensive review aims to examine and discuss the most promising and emerging treatment options for AGA.

The state of knowledge

1. Novel therapies

Oral Minoxidil

Minoxidil, a potent arterial vasodilator, received approval from the US Food and Drug Administration in 1979 for managing severe, refractory hypertension [22]. Physicians observed hair regrowth and widespread hypertrichosis in balding patients, prompting the development of a topical minoxidil formulation for the treatment of androgenetic alopecia (AGA), first in males and later in females [23]. The 2% minoxidil solution was introduced to the market in 1988, followed by the 5% solution in 1991 [24].

Topical minoxidil acts as a vasodilator, anti-inflammatory agent, inducer of Wnt/β-catenin signaling, and antiandrogen. It may also influence the duration of the anagen and telogen phases [25]. Despite its effectiveness in treating hair loss, patient compliance is often low due to factors such as undesirable changes in hair texture, unpleasant odor of the formulation, scalp irritation, allergic reactions to minoxidil, and the necessity of applying the medication twice daily [26].

Low-dose oral minoxidil offers a safe, effective, and well-tolerated alternative for managing patterned hair loss while minimizing the side effects associated with topical formulations [27]. Several studies have evaluated the comparative efficacy of oral and topical minoxidil in various patient groups, providing valuable insights into their therapeutic applications [28, 29]. Ramos et al. compared 1 mg of oral minoxidil with 5% topical minoxidil in a study involving 52 women and found no significant difference in the increase in total hair density between the two treatments, suggesting that oral minoxidil may provide a viable alternative for patients who face adherence challenges to topical formulations or who experience side effects [28]. Similarly, Vastarella et al. examined the effects of oral minoxidil in 12 women who were unresponsive to conventional treatments. Therapy was initiated with 0.5 mg daily, and the dose was increased to 1.5–2 mg after three months, resulting in a statistically significant improvement in hair density and frontal scalp hair counts within 24 weeks [29]. Further evidence comes from a double-blind, placebo-controlled trial involving 90 men with androgenetic alopecia, which compared 5 mg oral minoxidil once daily to 5% topical minoxidil applied twice daily over a 24 week period [30]. Both treatments demonstrated similar efficacy in increasing total and terminal hair density in the frontal and vertex regions, although photographic assessments indicated a slight advantage of oral minoxidil in the vertex region. However, common side effects in the oral minoxidil group included hypertrichosis (49%) and headache (14%).

The effectiveness of oral minoxidil in treating hair loss appears to be dose-dependent with studies highlighting its potential across various dosage levels for both male and female patients. Low doses of oral minoxidil, such as 0.25 mg/day, can stabilize hair loss and modestly increase hair density, as shown in a study by Pirmez and Salas-Callo involving 25 male patients with androgenetic alopecia (AGA) [31]. After 24 weeks of treatment, these patients exhibited stabilization or improvement in key parameters, including hair density and the number of new terminal hairs. Similarly, a 24-week randomized trial by Nascimento e Silva et al. evaluated the effects of 0.25 mg/day and 1 mg/day of oral minoxidil in 30 women with female pattern hair loss (FPHL) [32]. While the 1 mg group demonstrated significant improvements in hair shaft count ($P < 0.001$), the 0.25 mg group showed no significant changes ($P = 0.20$). Both groups, however, reported better hair-shedding scores and quality of life, with mild facial hypertrichosis observed in two patients receiving the higher dose. These findings underscore the greater efficacy of 1 mg/day for FPHL but highlight the need for larger studies to refine optimal treatment protocols and dosages. Further evidence suggests that daily doses of 2.5–5 mg/day are more effective for male patients with AGA compared to lower doses. For instance, Jimenez-Cauhe et al. treated 41 men with 2.5–5 mg/day of oral minoxidil for six months, achieving clinical improvement in 90.2% of participants [33]. Mild side effects, such as hypertrichosis (24.3%) and pedal edema (4.8%), were reported, with only one patient discontinuing treatment. Similarly, Panchepateep and Lueangarun administered 5 mg/day of oral minoxidil to 30 men with AGA over 24 weeks, finding significant increases in total hair count (19.23%) and non-vellus hair count (23.5%), with all participants showing improvements in the vertex area [34]. Collectively, these studies highlight a clear dose-response relationship, with higher doses yielding more substantial benefits while maintaining tolerable side effect profiles.

Allergy to topical minoxidil can limit its use in certain patients. Therianou et al. conducted a study with 9 women allergic to topical minoxidil who were treated with 0.25 mg/day of oral minoxidil twice daily for an average of 17 months [32]. Despite their allergy to the topical form, they tolerated oral minoxidil without adverse effects, suggesting it is a safe alternative for these patients.

When selecting an appropriate treatment for AGA, it is important to consider not only the therapy's effectiveness and safety but also the patient's adherence to the prescribed regimen. Asilian et al. compared the efficacy of 1 mg/day oral minoxidil and 5% topical minoxidil in 65 patients, noting that both treatments significantly improved hair diameter without a statistically significant difference in their overall effect [36]. Although photographic assessments slightly favored topical minoxidil, the convenience of oral administration led to higher patient satisfaction and adherence, identifying oral minoxidil as a viable alternative. This favorable profile is further supported by safety evaluations. For example, in a multicenter retrospective study of 1,404 patients (943 women and 461 men), low-dose oral minoxidil (LDOM) was generally well-tolerated, with hypertrichosis reported in 15.1% of cases but resulting in discontinuation in only 0.5% of patients [37]. Other systemic side effects, including lightheadedness (1.7%), fluid retention (1.3%), and tachycardia (0.9%), were mild and resolved upon dose adjustment or cessation. Notably, hypertrichosis was more prevalent in younger men and correlated with higher doses, a finding corroborated by additional studies on LDOM [38]. Overall, fewer than 2% of patients discontinued treatment because of adverse effects, underscoring LDOM's favorable safety profile.

Importantly, while minoxidil's hypotensive effects warrant consideration, especially for those concurrently using other antihypertensive medications, Ong et al. found that patients receiving 2.5 mg/day LDOM experienced no clinically significant blood pressure changes, except for a minor, clinically irrelevant decrease in diastolic pressure in men aged 35-49 [39]. Together, these findings demonstrate that low-dose oral minoxidil is not only effective and safe for managing androgenetic alopecia but also enhances adherence and maintains patient satisfaction, thereby meeting academic standards for clarity, coherence, and clinical relevance.

Topical Finasteride

The FDA approved oral finasteride at a dose of 5 mg per day in 1992 as a treatment for benign prostatic hyperplasia [40]. Finasteride's antiandrogen properties led to its investigation as a potential treatment for male androgenetic alopecia, culminating in its 1997 approval at a daily dosage of 1 mg [41]. By inhibiting the enzyme 5-α-reductase type II, finasteride reduces the conversion of testosterone to dihydrotestosterone (DHT), decreasing DHT levels in serum, prostate, and scalp by approximately 60-70%. This action halts the progression of androgenetic alopecia [1]. Studies have demonstrated that finasteride is effective in treating patients with androgenetic alopecia, with long-term use of up to five years resulting in significant hair growth and sustained stabilization of hair loss [42].

Although finasteride is used off-label at daily doses of 2.5–5 mg to treat female pattern hair loss (FPHL) in postmenopausal women, it is contraindicated in premenopausal women and classified as pregnancy category X due to potential feminization of male fetuses observed in animal studies [7].

The primary reason men avoid treatment with finasteride is concern over potential adverse effects, including sexual dysfunctions such as reduced libido and erectile dysfunction. More recently, fears surrounding persistent sexual dysfunction and depression—often termed post-finasteride syndrome—have emerged in discourse [9]. A population-based, retrospective, matched cohort study by Welk et al. evaluated whether 5-α-reductase inhibitors are associated with increased risks of depression or suicide. While the study found no change in suicide risk, it reported a higher incidence of depression among those using 5-α-reductase inhibitors [43].

Topical formulations of finasteride offer a promising alternative, with the potential to reduce systemic adverse effects while preserving clinical efficacy. The potential of topical finasteride for promoting hair regrowth was first documented by Mazzarella et al. in 1997. In a single-blind, placebo-controlled trial involving 52 patients (28 males, 24 females) with AGA, treatment using a 0.005% topical finasteride solution for 16 months significantly improved hair regrowth and reduced balding areas, with no adverse effects or evidence of percutaneous absorption [44]. Building upon these early findings, Caserini et al. pursued more extensive investigations comparing a 0.25% topical finasteride solution to a 1 mg oral finasteride tablet in men with androgenetic alopecia. Their research demonstrated that after one week, both treatments produced similar plasma DHT reductions, while the topical preparation was associated with significantly lower systemic absorption [45]. Follow-up studies confirmed that lower doses of topical finasteride (100–200 μL) effectively reduced scalp DHT levels by approximately 47-52% with minimal systemic impact and no significant adverse events [46]. A recent phase III, randomized, double-blind, controlled clinical trial led by B.M. Piraccini and colleagues evaluated the efficacy and safety of topical finasteride spray solution in 458 male patients aged 18–40 years with mild-to-moderate androgenetic alopecia (Norwood/Hamilton III–V) [47]. Patients were

randomized in a 2:2:1 ratio to receive either topical finasteride with an oral placebo, oral finasteride with topical placebo, or placebo for both formulations over 24 weeks. The primary endpoint, a change in target area hair count (TAHC), showed a significant improvement with topical finasteride compared to placebo (mean change 20.2 vs. 6.7 hairs/cm², $P < 0.001$), with results comparable to those achieved with oral finasteride (21.1 hairs/cm²). The incidence of treatment-related adverse events was low, and systemic absorption of finasteride in the topical group was significantly reduced (>100-fold lower plasma concentrations than oral finasteride). Minimal side effects, such as erythema (2.2%) and pruritus (2.2%), were reported, with reduced systemic sexual dysfunction compared to oral treatment.

Other studies suggest combining topical finasteride with additional therapies, such as minoxidil, to improve therapeutic outcomes for hair loss. Tanglertsampan et al. conducted a randomized, double-blind study comparing 3% minoxidil lotion (MNX) and a combination of 3% minoxidil with 0.1% finasteride lotion (MFX) in 40 men with androgenetic alopecia (AGA) over 24 weeks. Both treatments increased hair counts, but only MFX showed significant improvement from baseline ($P = 0.044$). Global photographic assessments favored MFX ($P = 0.003$), with no sexual side effects and similar mild side effects in both groups. Two recent randomized controlled trials by Suchonwanit et al. investigated the combined effects of topical 0.25% finasteride with 3% minoxidil (FMX), applied twice daily, compared to 3% minoxidil alone over a 24-week period [48, 49]. In men with androgenetic alopecia (AGA), FMX treatment led to significantly greater increases in total hair density and shaft diameter than minoxidil alone, as well as improved global photographic assessments at both weeks 16 and 24 [48]. Similarly, in postmenopausal women with female pattern hair loss (FPHL), FMX resulted in significantly increased hair diameter at 24 weeks, though improvements in hair count and global photographic evaluations were not statistically different from the minoxidil group [49]. Notably, serum DHT levels remained within normal physiological ranges in both men and women throughout the studies, with a significant reduction observed only in the women treated with FMX. Most recent research further supports the effectiveness of topical finasteride when combined with other treatments. In a single-blind trial involving 164 men aged 30–60, topical finasteride 0.25% combined with minoxidil 5% was compared to minoxidil 5% alone over 12 weeks [50]. The combination therapy showed significantly greater efficacy (86.7%) compared to minoxidil alone (69.1%, $P = 0.006$), highlighting its potential as a superior treatment option for AGA.

Another study suggested that topical finasteride could replace oral administration [51]. In five male patients switching from oral finasteride to a topical combination of minoxidil 5% and finasteride 0.1%, four patients maintained good hair density despite discontinuing the oral treatment.

Platelet-Rich Plasma

Reflecting the growing focus on cell-based therapy and tissue engineering, platelet-rich plasma (PRP) is a novel biotechnology comprising autologous plasma preparation enriched with concentrated platelets [52]. The regenerative and biostimulatory processes in skin cells are primarily driven by plasma-derived growth factors, including platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), transforming growth factor β (TGF-β), insulin-like growth factor (IGF-1), epidermal growth factor (EGF), and fibroblast growth factor (FGF) [53]. Activated platelets in PRP release growth factors that promote cell proliferation, differentiation, angiogenesis, and chemotaxis, all essential for hair regrowth. Notably, IGF-1 has been shown to induce and extend the anagen phase of the hair growth cycle [54].

The preparation of platelet-rich plasma (PRP) involves several critical steps, including blood collection with anticoagulants, centrifugation to concentrate platelets, and separation of the PRP layer. However, inconsistencies in preparation protocols and insufficient reporting in clinical studies highlight the need for standardization to ensure reproducibility and efficacy [55]. Although there is debate about pre-injection platelet-rich plasma (PRP) activation, one study suggested that non-activated PRP could be more effective than the calcium-activated form [56]. Despite some limitations, the study suggests that platelet activation may occur in vivo following injection due to the production of thromboxane A2 [56]. PRP injection closely resembles standard scalp mesotherapy protocols, involving multiple subcutaneous injections applied with a nappage technique at 0.5–1 cm intervals with a 27- or 30-gauge needle, and a total injected volume of approximately 1.5–4 mL per treatment session [57].

The efficacy of PRP for treating androgenetic alopecia (AGA) has been demonstrated in multiple randomized, placebo-controlled studies with similar methodologies [58, 59, 60]. Cervelli et al. [58], Alves and Grimalt [59], and Gentile et al. [60] investigated PRP effects in patients using a half-head design, administering three monthly PRP treatments and conducting follow-ups ranging from 3 to 12 months.. All studies showed significant increases in hair count and density in PRP-treated areas compared to baseline and placebo, with mild injection pain as the only side effect reported. Cervelli et al. observed increases of 18 hairs/cm² and

27.7 hairs/cm² over 12 months, Alves and Grimalt reported 12.8 hairs/cm² after 6 months, and Gentile et al. found 33.6 hairs/cm² and 45.9 hairs/cm² after 3 months. Histological analyses by Cervelli et al. and Gentile et al. revealed increased epidermal thickness, hair follicle counts, and Ki67+ cell proliferation, confirming enhanced regrowth activity.

A recent meta-analysis by Meijia Li et al. evaluated the efficacy of platelet-rich plasma (PRP) for the treatment of androgenetic alopecia (AGA) across 10 randomized controlled trials with 318 participants and 555 treatment units [61]. PRP significantly increased hair density by 25.09 hairs/cm² compared to controls (P = 0.002), with stronger effects in males. However, no significant impact on hair diameter was observed. No severe side effects were reported, supporting PRP as a safe and effective option for improving hair density in AGA.

Given these encouraging results, current efforts focus on optimizing PRP treatment schedules to sustain and maximize its benefits for AGA. The treatment schedule proposed by Stevens and Khetarpal for platelet-rich plasma (PRP) therapy in androgenetic alopecia (AGA) involves an initial series of three monthly sessions, followed by maintenance treatments every three months during the first year (totaling six sessions) [54]. An alternative schedule includes three monthly sessions followed by treatments at six-month intervals [54].

Although PRP therapy has shown considerable potential in the treatment of AGA, further high-quality, large-scale studies are needed. Standardized protocols for PRP preparation and therapeutic administration are essential to ensure reproducibility, consistency, and optimal efficacy in clinical practice.

Dutasteride mesotherapy

Dutasteride, a second-generation 5α-reductase inhibitor, exhibits higher potency than finasteride by targeting both type 1 and type 2 enzyme isoforms [62]. A comparative study of medication effects on DHT levels in hair demonstrated that finasteride reduced hair DHT levels by approximately 64%, while dutasteride achieved a reduction of around 92% [63].

Oral dutasteride is linked to side effects such as decreased libido, erectile dysfunction, ejaculatory issues, and gynecomastia, presenting a significant barrier to its use [41]. Due to its longer half-life of four weeks compared to finasteride's 6–8 hours, the sexual side effects of dutasteride are more prolonged and difficult to reverse [64].

In recent years, mesotherapy has gained popularity as a method to minimize the potential adverse effects associated with oral dutasteride [57]. Mesotherapy offers the advantage of delivering the therapeutic agent directly into the skin while avoiding systemic barriers typically encountered with topical treatments. Furthermore, this technique ensures targeted application and reduces the risk of systemic distribution and associated adverse effects [65]. Marwa Abdallah et al [66] and N. Moftah et al [67] evaluated mesotherapy for hair loss using a solution containing 0.05% dutasteride, D-panthenol, biotin, and pyridoxine. Abdallah's 12-week study on 28 men with male pattern hair loss (MPHL) demonstrated clear improvements in hair growth, while Moftah's 16-week trial on 126 women with female pattern hair loss (FPHL) showed significant improvements in hair growth and quality, particularly among patients with shorter disease durations. Both studies are constrained by the use of additional hair growth stimulants along with dutasteride, making it difficult to determine the specific effects of dutasteride on its own. This limitation underscores the significance of Sobhy et al.'s study, which focused on isolating the effects of pure dutasteride (0.005%) [64]. Their randomized trial offered a direct comparison between pure dutasteride, a dutasteride-solution (0.05% with stimulants), and placebo in 90 male AGA patients. The findings demonstrated the efficacy of pure dutasteride in improving anagen hair percentage and hair shaft diameter and provided additional insights into its safety profile. While there was minor systemic absorption affecting semen parameters, these changes remained within normal ranges and were reversible.

A multicentric retrospective study evaluated the safety and effectiveness of mesotherapy with dutasteride for androgenetic alopecia in 541 patients [68]. After one year of treatment, clinical improvement was observed in most patients, with marked improvement reported in 38.4% of cases. Pain was the most commonly reported side effect (45.5%), while no serious or sexual adverse events were noted, supporting dutasteride mesotherapy as a safe alternative to oral treatments.

The adverse effects (AEs) of dutasteride mesotherapy range from common issues such as injection pain, headaches, and post-injection scalp tightness to less frequent complications like frontal edema, angioedema-like contact dermatitis, and non-scarring alopecia [63].

Although mesotherapy with dutasteride presents a promising alternative to oral treatments for AGA, additional high-quality studies are necessary to confirm its efficacy and establish standardized treatment protocols.

Low-Level Laser Therapy

Since their introduction in the 1960s, lasers have been applied to various medical treatments. Today, low-level laser therapy (LLLT) is widely used for pain relief, wound healing, and tissue regeneration [69]. The potential of low-level laser therapy (LLLT) to promote hair growth was first observed in the late 1960s. In an early experiment, Endre Mester demonstrated faster hair regrowth in shaved mice treated with a low-power 694 nm ruby laser, providing the first documented evidence of laser-stimulated hair regeneration [70]. In 2002, another case highlighted the potential of LLLT when paradoxical hair regrowth was observed in a patient undergoing laser hair removal [71].

The proposed mechanism of action involves photons from LLLT oxidizing cytochrome C oxidase, which in turn activates the electron transport chain and enhances ATP production. This process is hypothesized to upregulate endogenous growth factors and nitric oxide (NO), thereby promoting cellular proliferation and vasodilation [7]. Other hypotheses propose that the mechanism of action may resemble that of minoxidil, involving enhanced blood flow in the scalp through nitric oxide (NO) production and a reduction in follicular inflammation [42].

There is no universally agreed-upon optimal wavelength for low-level laser therapy (LLLT); however, most treatments commonly use wavelengths ranging from 500 to 1100 nm, with power densities between 3 and 90 mW/cm² and fluencies of 1–4 J/cm² [71]. LLLT devices often employ wavelengths of 650–900 nm at 5 mW power output for managing androgenetic alopecia (AGA) [7].

Reviews of randomized trials suggest that low-level laser therapy (LLLT) may improve hair density and diameter when compared to sham devices. Reported side effects are generally minor, including scalp irritation and dry skin [72]. For example, Leavitt et al [73] and Jimenez et al [74] conducted randomized, double-blind, sham-controlled trials to assess the efficacy and safety of the FDA-cleared HairMax LaserComb® for androgenetic alopecia (AGA). Leavitt's study on 123 males showed a significant increase in terminal hair density (+19.8 hairs/cm² vs. −7.6 hairs/cm² in the sham group; $P < 0.0001$) after 26 weeks. Similarly, Jimenez et al. evaluated 269 participants (128 males, 141 females), reporting significant hair density gains in lasercomb-treated groups (+20.2 hairs/cm² in females vs. +2.8 hairs/cm² in the sham group; $P < 0.0001$). Both studies found no serious adverse events, with minor side effects like dry skin and scalp irritation occurring infrequently. Further evidence comes from two more studies by Lanzafame et al. which evaluated low-level laser therapy (LLLT) for androgenetic alopecia [75, 76]. In 2013, 41 men (18–48 years) using a TOPHAT 655-nm laser device every other day for 16 weeks showed a 35% increase in hair counts ($p = 0.003$) [75]. In 2014, a similar study in 42 women (18–60 years) using a 655-nm laser helmet showed a 37% improvement ($p < 0.0001$) with no adverse events [76]. Both studies demonstrated significant improvements in hair growth.

Another study explored low-level laser therapy (LLLT) in combination with minoxidil for treating female pattern hair loss. In 2017, Esmat et al. compared LLLT, minoxidil 5%, and their combination [77]. Forty-five patients were treated for four months: minoxidil was applied twice daily, LLLT was administered daily for 25 minutes using a laser and LED helmet, or both therapies were combined. Evaluations showed significant increases in hair follicle count and density in the LLLT and combination groups, with the combination therapy demonstrating deeper dermal follicle placement, superior improvement in Ludwig classification, and highest patient satisfaction.

In one of the largest real-world studies on low-level laser therapy (LLLT) to date, Qiu et al. evaluated its effectiveness in 1,383 patients with androgenetic alopecia (AGA) [78]. The study revealed a significant clinical impact, with nearly 80% of participants showing improvement in hair density and thickness. It also identified factors such as scalp conditions and extended use (over 180 sessions or one year) as key to better outcomes. While this extensive study demonstrated the practical success of LLLT in a large population, Gupta and Carviel expanded on this by consolidating findings from a broader range of controlled trials through a meta-analysis [79]. Their analysis of 15 trials (795 participants) found a significant increase in hair density compared to sham treatments. Laser-only devices were more effective than laser/LED combinations, with no difference between comb-style and helmet devices. Reported side effects were generally minor, including scalp irritation and dry skin, confirming LLLT as a safe and effective treatment, especially with laser-only devices.

To complement the findings detailed above, a recent study evaluated home-use low-level light therapy (LLLT) devices cleared by the US FDA for androgenetic alopecia (AGA) [80]. Fourteen FDA-cleared devices were reviewed, varying in design, light source, and power output. However, only eight clinical studies supported these devices, including six randomized controlled trials and two cohort studies, all focusing on mild to moderate hair loss. Studies lasted 16 to 26 weeks, with most demonstrating marked improvements in hair density in LLLT-treated groups and minimal, self-limiting side effects. The researchers highlighted the limited evidence and recommended long-term studies, device comparisons, and trials involving severe hair loss patients.

Botulinum toxin

Derived from the gram-positive, strictly anaerobic bacterium Clostridium botulinum, botulinum toxin is recognized as one of the most potent neurotoxins known [81]. Clostridium botulinum spores are commonly present in soil and aquatic environments worldwide, producing botulinum neurotoxin (BoNT), which is divided into seven serotypes (A, B, C1, D, E, F, and G) among which serotypes A, B, E, and F are associated with human intoxication. Currently, serotypes A and B are employed in therapeutic practices [82].

When administered according to standard guidelines, botulinum toxin type A injections rarely lead to side effects, although serious adverse events such as dysphagia, allergic reactions, generalized muscle weakness, flu-like symptoms, injection site trauma, arrhythmias, myocardial infarction, and localized effects, such as pain at the injection site, muscle weakness, ptosis and headaches, may occur [83].

Recently, botulinum toxin has been suggested as a potential therapeutic option for addressing androgenetic alopecia (AGA) [7]. Several hypotheses have been proposed to explain the mechanism by which it may affect hair loss. One hypothesis posits that preventing the presynaptic release of acetylcholine relaxes scalp muscles, enhances local blood circulation, increases oxygen levels, and facilitates the elimination of accumulated dihydrotestosterone (DHT) [57]. Zhang et al. hypothesized that reduced blood perfusion in the vertex region of the scalp may result from muscle contraction, and botulinum toxin could potentially improve perfusion by relaxing the occipitalis and frontalis muscles [84]. A second hypothesis suggests that botulinum toxin (BoNT) inhibits the secretion of transforming growth factor-beta 1 (TGF-β1) induced by dihydrotestosterone (DHT) in dermal papilla cells, which is a key factor in androgenetic alopecia. By suppressing TGF-β1, BoNT promotes follicular keratinocyte growth and supports hair cycle regulation, offering a potential therapeutic mechanism for hair restoration [85]. An alternative explanation suggests that botulinum toxin may suppress neuromodulators like substance P or calcitonin gene-related peptide (CGRP), both of which could play a role in the development of androgenetic alopecia (AGA) [41].

The study, led by Lin Li et al., evaluated the efficacy of type A botulinum toxin (BoNT-A) in treating androgenetic alopecia (AGA) in a randomized, double-blind trial involving 90 patients. Patients in the treatment group received 100 units of BoNT-A, showing significant increases in follicle width and length after 1 and 3 months, particularly in the vertex region, although hair count did not change. Ultrasound detected these changes earlier than trichoscopy, and no adverse effects were reported [86]. Another study aimed to compare the efficacy of two concentrations of botulinum toxin A (BoNT-A) in the treatment of androgenetic alopecia (AGA). The study involved 32 patients treated with 33.3 U/mL and 25 U/mL concentrations on opposite sides of the scalp [87]. After six months, significant improvements were observed, with 70.4% of females and 60% of males demonstrating clinical improvement, particularly with the higher concentration. Adverse effects, including headache (31.2%) and irritation (12.5%), were mild, and no serious events occurred. Other studies have explored the potential of combining botulinum toxin type A (BoNT-A) with other treatments [88]. One study involved 63 patients treated with BoNT-A alone or in combination with finasteride, with 100 units injected every three months for a year. Both groups demonstrated improvements in hair density, with the combination group achieving greater effectiveness (84.8%) compared to BoNT-A alone (73.3%).

Two systematic reviews evaluated the effectiveness and safety of botulinum toxin A (BoNT-A) for treating androgenetic alopecia (AGA) and other types of hair loss [89, 90] Robert S. English Jr. and Sophia Ruiz analyzed five clinical studies with 165 male participants, aged 19–57, using intramuscular and intradermal BoNT-A injections (30–150 units) over 24 to 60 weeks. Similarly, Ramadan S. Hussein et al. reviewed studies assessing hair density, hair count, and patient satisfaction. Both reported response rates of 70%–84% and hair count improvements up to 20.9%. Adverse effects were minimal, including transient injection-site reactions and headaches. Both reviews concluded that BoNT-A is a promising treatment but called for further trials to confirm effectiveness and optimize protocols.

However, unlike other studies suggesting the efficacy of botulinum toxin (BoNT) for androgenetic alopecia (AGA), the triple-blind, randomized trial by Daniel Fernandes Melo et al. found no significant benefit [91]. Thirteen male participants, aged 25–44, received 50 IU of BoNT on one half of the scalp and saline on the other. After 24 weeks, no differences in terminal or total hair density were observed, with both sides showing a decrease in terminal hair density.

Especially in light of the conflicting findings, such as those reported by Daniel Fernandes Melo et al. further research is crucial. Larger, well-designed studies are needed to clarify mechanisms, optimize protocols, and confirm the long-term efficacy and safety of botulinum toxin as a treatment option for androgenetic alopecia.

2. Future perspectives

Beyond the FDA-approved drugs and commonly used off-label treatments, innovative therapies for androgenetic alopecia (AGA) are being developed to address unmet needs and offer targeted, effective, and safer alternatives to current treatments. These emerging therapies aim to explore novel pathways and mechanisms and provide effective solutions for managing androgenic alopecia.

One of the new promising potential treatments is cetirizine, a second-generation histamine H1 receptor antagonist that inhibits prostaglandin PGD2—a compound implicated in hair growth suppression [9]. A systematic review by Chen, Xiang, and Yang analyzed three clinical trials with 185 participants to assess the effectiveness of 1% topical cetirizine for androgenetic alopecia (AGA) [92]. The findings suggest that cetirizine improves hair density and quality more effectively than placebo but is less potent than 5% minoxidil, with the potential for a longer-lasting effect and fewer adverse events. The authors highlighted the need for more rigorous studies citing the small sample sizes and moderate-to-high risk of bias in the reviewed trials.

An additional therapeutic option under investigation is clascoterone, an ester derivative of cortexolone that acts as a potent direct inhibitor of androgen receptors. When applied topically, it penetrates the skin to target androgen receptors in sebaceous glands and hair follicles, effectively inhibiting DHT-driven signaling pathways [93]. Clascoterone, originally FDA-approved for the treatment of acne vulgaris, has recently been explored as a topical option for androgenetic alopecia (AGA) [41]. In a phase I open-label trial, 18 patients with AGA applied 5% topical clascoterone twice daily. Clascoterone levels in the bloodstream rose within 4 hours of application, and by day 28, pre-dose levels aligned with 12-hour post-dose levels, indicating a steady state. Phase II studies suggest that clascoterone is not only more effective than cyproterone acetate or 17-alpha estradiol but also promotes faster hair growth compared to topical minoxidil [94].

Another promising treatment is pyrilutamide, a topical androgen receptor antagonist that inhibits androgen receptor-mediated signaling by competing with androgens for receptor binding. Unlike finasteride, which promotes hair growth by systemically reducing DHT synthesis, pyrilutamide acts locally to block androgen activity [95]. It has shown promise in clinical trials across China and the US, demonstrating effectiveness and a favorable safety profile for treating AGA in both men and women. Phase II trials in China and the US reported significant improvements in hair growth, particularly with the 0.5% twice-daily dose, and no serious adverse events. Furthermore, ongoing trials in China are assessing its long-term safety over 52 weeks, while Kintor Pharma is advancing preparations for a Phase III trial in the US [96].

Setipiprant, a selective prostaglandin D2 receptor (PGD2R) antagonist, is known to regulate inflammatory pathways associated with conditions such as asthma and allergies [93]. Once considered a promising treatment for AGA, setipiprant underwent a Phase III clinical trial comparing its efficacy to placebo and finasteride [97]. Male patients with grade III or higher AGA were administered 1 g of setipiprant every 12 hours for 24 weeks. However, the results from weeks 24 and 32 revealed no significant improvement over placebo in treating AGA.

Another area of research has focused on JAK inhibitors, which have shown potential as therapeutic agents for alopecia areata (AA). These inhibitors promote hair regrowth by regulating T-cell-mediated immune responses and supporting anagen progression at the hair follicle level [98]. Their success in AA has prompted interest in their possible use for other types of hair loss, such as androgenetic alopecia [99]. A topical JAK 1/3 inhibitor, applied twice daily, showed promising results for AGA, increasing non-vellus hair count by 8.6 hairs/cm² over 26 weeks without serious adverse events [100]. Despite these findings, the effectiveness of JAK inhibitors for AGA remains uncertain. In a small observational study, men with severe alopecia areata treated with an oral JAK 1/3 inhibitor for 24–50 weeks experienced significant hair regrowth, but the characteristic AGA-pattern hair loss persisted [99].

Among the emerging therapies for androgenetic alopecia (AGA), stem cell-based treatments have gained attention for their potential to reactivate dormant hair follicle stem cells and restore follicular function [101]. Studies on adipose-derived stem cells (ADSCs) and hair follicle stem cells (HFSCs) have shown promising results, with significant increases in hair density observed in clinical trials [7]. Furthermore, innovative approaches utilizing stem cell-conditioned media and exosomes, which are rich in growth factors and cytokines, offer non-invasive methods to stimulate hair regrowth [102]. While these therapies represent a notable progression in the field, further research is essential to establish their long-term safety and efficacy in treating AGA.

Recent advancements in androgenetic alopecia (AGA) research have introduced innovative approaches involving PROTAC (Proteolysis Targeting Chimera) technology to target androgen receptor (AR) degradation, a key factor in AGA progression. Two promising treatments include a non-invasive AR PROTAC degrader,

C6, [103] and an AI-designed peptide-based PROTAC drug [104]. Both therapies demonstrated significant efficacy in preclinical studies, promoting hair regeneration and enhancing hair follicle function in both in vitro and in vivo models. While C6 offers excellent skin retention and localized delivery through a topical formulation, the AI-designed PROTAC reduces reactive oxygen species (ROS) and ensures targeted application via a transdermal microneedle system. These advancements represent a breakthrough in AGA treatment, combining precision targeting with minimized systemic side effects, and marking a significant advancement in the field of hair loss treatment.

Conclusions

This study synthesizes advancements in the treatment of androgenetic alopecia (AGA) by exploring innovative therapies and evaluating their efficacy, safety, and potential to address current treatment gaps. The findings emphasize the limitations of existing FDA-approved therapies, such as minoxidil and finasteride, while highlighting promising alternatives including low-dose oral minoxidil, topical finasteride, platelet-rich plasma (PRP), mesotherapy, low-level laser therapy (LLLT), botulinum toxin, and novel experimental treatments like clascoterone and pyrilutamide. These approaches offer significant therapeutic potential by targeting various mechanistic pathways, ranging from modulating androgen receptors to enhancing hair follicle regeneration.

The importance of this research lies in addressing the unmet need for individualized, effective, and minimally invasive treatments for AGA, particularly for patients who are unresponsive to conventional therapies or unable to tolerate systemic side effects. For instance, topical finasteride and oral minoxidil offer reduced systemic absorption and improved patient adherence, while PRP and LLLT provide promising non-pharmacological options with minimal adverse events. Additionally, emerging therapies, including stem cell-based treatments, androgen receptor antagonists, and PROTAC technologies, represent a future direction toward precision medicine for AGA management.

However, the study has certain limitations. Variability in study methodologies, sample sizes, and follow-up durations reduces the generalizability of findings. Furthermore, the lack of standardized protocols for treatments like PRP and LLLT highlights the need for larger, well-designed studies to optimize treatment dosages, schedules, and safety profiles. Similarly, while experimental therapies show promise, their efficacy must be validated through large-scale clinical trials.

Future efforts should focus on standardizing treatment protocols for established therapies such as PRP and LLLT while advancing emerging approaches, such as stem cell-based interventions and AI-designed therapeutics. Research into combination therapies, biomarkers for individualized treatments, and larger clinical trials will be essential to expanding access to effective, precision-driven solutions for AGA.

In conclusion, this study highlights the transformative potential of novel therapeutic strategies in addressing the limitations of current androgenetic alopecia (AGA) treatments. By recognising these challenges and exploring emerging technologies, this research paves the way for improved outcomes and personalized care in AGA management.

Authors' contribution

Conceptualization, KN, JS, KR methodology, MR, JŚ; software, KN, JS; check, KN, MR, KR; formal analysis, KN, JŚ, KR; investigation, KN, MR; resources, JS, KR; data curation, KN, MR; writing - rough preparation, KN, MR, KR, JS; writing - review and editing, KN, KR, JŚ, MR; visualization, KN, JS, KR; supervision, KN; project administration, KN, JŚ, KR;

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