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Review Article

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Updates in Treatment for Androgenetic Alopecia

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ABSTRACT

Androgenetic alopecia (AGA) is a common nonscarring hair loss condition that affects both men and women, often resulting in psychological distress and reduced quality of life. AGA pathogenesis involves genetic predisposition and androgen influence, primarily dihydrotestosterone (DHT), which leads to hair follicle miniaturization and progressive hair thinning. AGA remains challenging to manage due to its chronic progression and the combined influence of genetic and environmental factors. Topical minoxidil and oral finasteride are the most widely used treatments for AGA, addressing follicular miniaturization. However, their reliance on long-term use and potential for side effects or inconvenience has prompted increasing interest in alternative therapies. The mainstream of current AGA treatment can be categorized into androgen-targeting and non-androgen-targeting approaches. Finasteride and dutasteride, both 5-α-reductase inhibitors that reduce DHT levels in hair follicles, are key androgen-targeting treatments, with newer formulations like topical and injectable options emerging alongside traditional oral forms. Topical minoxidil remains central to non-androgen-targeted AGA treatments, though growing evidence supports the efficacy and safety of its low-dose oral form. Additionally, therapies like low-level light therapy, platelet-rich plasma, and exosome treatments are being explored. Recently, therapies targeting the androgen receptor, including small interfering RNA-based approaches, have been developed and are currently in clinical trial stages, offering innovative potential for AGA treatment. This review explores current and emerging treatments for AGA, addressing both androgen-targeted and non-androgen-targeted approaches with an emphasis on their mechanisms, efficacy, and safety. It ultimately aims to provide a comprehensive update on the latest advancements in AGA management.

Keywords: Androgenetic alopecia; Dutasteride; Female androgenetic alopecia; Finasteride; Minoxidil

INTRODUCTION

Androgenetic alopecia (AGA) is a common type of nonscarring hair loss characterized by gradual, patterned hair thinning. Affecting both men and women, it is also referred to as male pattern hair loss (MPHL) or female pattern hair loss (FPHL). The prevalence of AGA increases with age, impacting about 80% of men and nearly 50% of women among Caucasians by age 70[1]. In Korea, the prevalence is slightly lower, with around 50% of men and 25% of women affected by age 70[2]. AGA can significantly impact quality of life, often leading to psychological distress, reduced self-esteem, and social anxiety, particularly as visible hair loss progresses[3]. AGA is a multifactorial condition primarily driven by genetic predisposition and the influence of androgens, particularly dihydrotestosterone

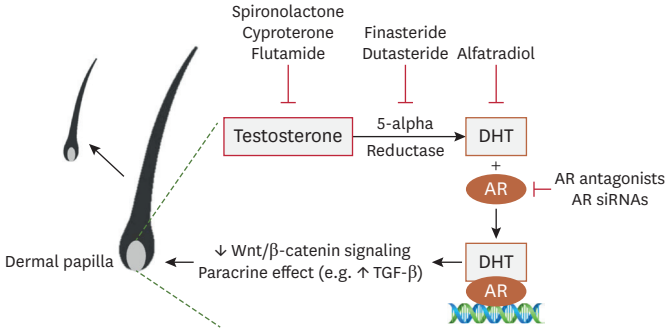

Fig. 1. Pathogenesis of androgenetic alopecia and action mechanisms of androgen-targeted therapies.
DHT: dihydrotestosterone, AR: androgen receptor, siRNA: small interfering RNA.

(DHT) (**Fig. 1**). The condition results from a combination of genetic factors that increase hair follicle sensitivity to androgens, leading to a progressive miniaturization of hair follicles. In individuals with AGA, the enzyme 5-α-reductase converts testosterone into DHT, which binds to androgen receptors (ARs) in hair follicles, particularly in susceptible areas like the frontal and vertex regions. This binding not only directly affects hair follicles but also triggers downstream signaling pathways, including the suppression of the Wnt/β-catenin pathway, which is crucial for hair follicle proliferation and maintenance. Additionally, DHT-AR interaction promotes the release of paracrine factors like transforming growth factor (TGF)-β, which further contribute to follicular miniaturization by shortening the anagen phase of the hair growth cycle[4]. Over time, these processes transform terminal hairs into finer, vellus hairs, leading to visible thinning and the characteristic patterned hair loss of AGA[3,5]. AGA is highly heritable, with genetic factors accounting for about 80% of predisposition and involving polygenic, multifactorial inheritance. Key genetic loci associated with AGA include the AR gene on Xq12, HDAC9 on chromosome 7p21.1, and the PAX1/FOXA2 locus on chromosome 20p11[1,6]. These genetic variations, along with environmental factors, contribute to AGA's diverse expression. In addition, AGA has been linked to other health factors like body mass index, metabolic syndrome, and severe coronavirus disease 2019 outcomes[1]. Despite the high prevalence of AGA, effective management and treatment remain challenging due to its chronic nature and the various issues that can emerge throughout the long course of treatment. Currently, 5-α-reductase inhibitors, topical minoxidil, and low-level light therapy (LLLT) have received approval for the treatment of AGA beside surgery. Among oral 5-α-reductase inhibitors, both finasteride and dutasteride are approved in several countries, including South Korea, while only finasteride has received official approval in the United States. However, not only approved treatments but numerous non-approved treatments have shown to be effective in treating AGA. Moreover, there are active development and clinical trials of new therapies aimed at addressing the limitations of existing treatment options. The present review provides an in-depth analysis of current non-surgical treatments for AGA, examining their mechanisms, efficacy, limitations, and side effects. Additionally, it introduces novel treatments that have recently been developed or are currently under clinical investigation.

ANDROGEN-TARGETED THERAPIES

The main approaches to AGA treatment are largely classified into two main categories: androgen-targeted therapies and non-androgen-targeted therapies (**Table 1**). Androgen-targeted therapy addresses AGA by suppressing the production of DHT, the main factor in its development, or its precursor, testosterone (**Fig. 1**).

Table 1. Current and emerging treatments for androgenetic alopecia

Treatment category	Androgen-targeted	Non-androgen-targeted
Approved	1. 5-α-reductase inhibitors - Oral finasteride*,† - Oral dutasteride† - Topical finasteride† 2. Topical alfatradiol†	1. Topical minoxidil*,† 2. Low-level light therapy*,†
Non-approved or emerging	1. Systemic injectable 5-α-reductase inhibitors 2. Oral anti-androgens - Spironolactone - Cyproterone acetate - Flutamide 3. AR blocker - Topical AR antagonist - siRNAs targeting AR	1. Low-dose oral minoxidil 2. Platelet-rich plasma 3. Intradermal botulinum toxin 4. Exosome therapy

AR: androgen receptor, siRNA: small interfering RNA.
*Food and Drug Administration-approved (US).
†Approved in countries other than the USA including Korea.

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5-α-reductase inhibitors

1) Oral finasteride

Oral finasteride, Food and Drug Administration (FDA)-approved since 1997 for treating MPHL, is a widely used AGA therapy that inhibits the 5-α-reductase type II enzyme, the most potent isotype for converting testosterone into DHT in hair follicles. By reducing DHT levels in the serum, scalp, and prostate by about 60%–70%, finasteride slows hair loss and frequently promotes regrowth[7]. Standard treatment involves a daily dose of 1 mg, with notable effects typically visible within six months and sustained benefits observed with continued use for 10 years[8,9]. Studies have demonstrated that finasteride is especially effective in the vertex area of the scalp, where it promotes better regrowth compared to the frontal regions[10]. In our retrospective study on 126 Korean male AGA patients, consistent use of 1 mg finasteride over five years halted the progression of hair loss in most patients, with 85.7% showing improvement compared to their condition before treatment[11]. Oral finasteride has demonstrated superior efficacy compared to 5% minoxidil and has shown improved outcomes when used in combination with topical minoxidil compared to monotherapy[12].

Although finasteride is primarily prescribed for male AGA, it is sometimes used off-label in women, especially postmenopausal women or those with hyperandrogenism, where higher doses of 2.5 to 5 mg have been found to be more effective[13,14]. However, strict contraception is necessary due to its teratogenic potential, which poses a risk of fetal abnormalities in male fetuses.

While generally well tolerated, finasteride can cause side effects, primarily sexual, such as reduced libido, erectile dysfunction, and ejaculatory issues. In a meta-analysis examining the adverse sexual effects of 5-α-reductase inhibitors, finasteride showed a relative risk of 1.66 (95% confidence interval [CI], 1.20–2.30)[15]. However, most studies reported these side effects were mild and reversible upon discontinuation, with some cases even noting improvement despite continued use[5,16]. Finasteride also lowers prostate-specific antigen (PSA) levels, potentially masking early signs of prostate cancer, which necessitates regular PSA monitoring, especially in those with a family history of prostate issues[17,18]. Additionally, rare cases of gynecomastia and male breast cancer have been reported, particularly with higher doses, possibly due to an altered estrogen-to-testosterone ratio, and monitoring for breast abnormalities in long-term users is advised[19-21].

2) Oral dutasteride

Dutasteride is a potent option for managing AGA and functions by inhibiting both type 1 and type 2 5-α-reductase enzymes. Specifically, dutasteride is approximately 100 times more effective at inhibiting type 1 and three times more effective at inhibiting type 2[22]. This profile gives it greater potency in reducing DHT levels than finasteride. In addition, dutasteride has a significantly longer half-life (approximately 5 weeks) compared to finasteride (5–6 hours). Clinical studies, including large randomized control trials, have shown that dutasteride can enhance hair growth within three to six months, outperforming finasteride in both hair density and thickness at a daily dose of 0.5 mg[23,24]. Consequently, 0.5 mg oral dutasteride is now approved for male AGA in Japan and South Korea, though it remains off-label in the US and many countries[25]. In a 5-year retrospective study conducted in Korea, 0.5 mg dutasteride led to 89.9% of male AGA patients showing improvement and 93.9% achieving prevention of disease progression, with results comparable to long-term efficacy studies of 1 mg finasteride in male AGA patients[26].

Dutasteride has similar sexual side effects to finasteride, including decreased libido, erectile dysfunction, and ejaculatory issues[27]. In a meta-analysis investigating the adverse sexual effects of 5-α-reductase inhibitors, the relative risk for dutasteride was 1.37 (95% CI, 0.81–2.32)[15]. Although the risk was not statistically significant, this may be attributed to the limited number of studies on dutasteride.

A recent phase 3 clinical trial conducted in South Korea demonstrated that 0.2 mg dutasteride is non-inferior to the 0.5 mg dose in terms of efficacy for treating AGA, while showing a lower incidence of sexual side effects (unpublished data). Based on these findings, the 0.2 mg dutasteride formulation has recently been launched in South Korea.

3) Topical finasteride

Topical finasteride has emerged as a promising alternative for treating AGA, particularly in patients concerned about the systemic side effects associated with oral finasteride. Conventionally, topical formulations of finasteride, including a 0.05% gel and a 0.1% lotion, have shown efficacy in treating male AGA in smaller studies, demonstrating positive hair regrowth effects without significant sexual side effects[1,28,29]. A recent phase III trial demonstrated that a 0.25% finasteride spray significantly increased hair count, achieving efficacy comparable to oral finasteride but with markedly lower systemic exposure and reduced impact on serum DHT levels, thereby minimizing the risk of sexual side effects[30]. Notably, a newer spray formulation using hydroxypropyl chitosan as a vehicle has been developed to reduce passive transfer of finasteride and enhance its penetration via a film-forming mechanism, further improving both its safety and effectiveness.

While effective for male AGA, topical finasteride in combination with minoxidil has also shown potential for FPHL. In a randomized double-blinded study, a topical combination of 0.25% finasteride and 3% minoxidil demonstrated significantly greater efficacy in improving hair diameter compared to minoxidil

solution alone[31]. Reported side effects are typically mild and localized, such as scalp irritation, making topical finasteride a well-tolerated option with a favorable safety profile.

4) Systemic injectable 5-α-reductase inhibitors

Recent advancements in AGA treatment include the development of injectable formulations of 5-α-reductase inhibitors. Promising approaches utilize drug-loaded poly-lactic-co-glycolic acid (PLGA) microspheres or hexagonal liquid crystal phase to achieve controlled, long-term release, maintaining therapeutic drug levels over extended periods with a single injection[32]. In an AGA mouse model, finasteride-loaded PLGA demonstrated the ability to significantly reduce serum DHT levels for several weeks following a single injection and achieved comparable hair regrowth to oral administration[33]. Phase II and III clinical trials have been conducted on finasteride (NCT04945226) or dutasteride (NCT06916793) injectables, exploring the pharmacokinetics and efficacy of monthly or tri-monthly injection regimens. These innovative approaches aim to achieve controlled, long-term release of dutasteride, potentially providing a more effective and convenient treatment option for AGA compared to conventional oral or topical therapies.

Oral anti-androgens

Anti-androgen therapy for AGA primarily targets the reduction of androgen activity. This approach is particularly effective for female AGA, especially in cases where hormonal influences play a significant role.

Spironolactone, a potassium-sparing diuretic, is commonly prescribed off-label for female AGA. It reduces androgen activity by blocking ARs in target tissues and inhibiting testosterone synthesis in the adrenal glands[3]. The typical dosage ranges from 50 to 200 mg per day. Sinclair et al.[34] conducted a single-center, open-label study on 80 women (aged 12–79) with FPHL, treating them with 200 mg of spironolactone daily for 12 months. In addition, a randomized controlled trial found that a combination of 100 mg spironolactone and 2% minoxidil achieved better outcomes than 5 mg finasteride and 2% minoxidil in female AGA patients[35]. Common side effects include electrolyte imbalances, breast tenderness, and irregular menses[36].

Cyproterone acetate blocks ARs and reduces testosterone levels by inhibiting gonadotropin release. It is especially beneficial in women with signs of hyperandrogenism, such as hirsutism or acne, which often accompany AGA[37]. However, cyproterone carries potential side effects, including weight gain, breast tenderness and mood changes, and should be used with caution[29].

Flutamide is a strong nonsteroidal anti-androgen, flutamide binds directly to ARs to prevent testosterone from exerting its effects on hair follicles. While effective in some cases, it is not commonly used for AGA treatment due to its association with liver toxicity, which can limit its safety for long-term use[29].

Topical alfatradiol

Alfatradiol (17α-estradiol) reduces DHT levels by weakly inhibiting 5α-reductase and modulating androgen metabolism, and it has also been reported to stimulate hair follicular matrix cell proliferation[38]. It is commonly applied as a 0.025% lotion twice daily. Research on its effectiveness has yielded mixed results: one study in women reported a decrease in hair count over 6 months, while 2% minoxidil increased it[39]. However, another study found that alfatradiol improved the anagen-to-telogen hair ratio in the frontal scalp, suggesting potential support for hair growth cycles[38]. In a phase IV study conducted in Korea focusing on FPHL, alfatradiol treatment resulted in statistically significant increases in both hair count and hair diameter at 4 and 8 months compared to baseline[38]. In addition, combination therapy with alfatradiol lotion and topical minoxidil has also been suggested as an effective treatment option for FPHL[40]. While alfatradiol is considered safe for use without notable side effects, further studies are needed to clarify its efficacy as a treatment option.

NON-ANDROGEN-TARGETED THERAPIES

Minoxidil

1) Topical minoxidil

Topical minoxidil is the primary treatment for AGA and is also prescribed for other types of hair loss, such as alopecia areata and telogen effluvium[1]. Originally developed as an oral hypertension medication, minoxidil was reformulated for topical use after increased hair growth was observed in patients taking it for blood pressure. The exact mechanism of minoxidil in hair growth remains unclear, but it is hypothesized to enhance scalp blood flow, activating potassium channels and increasing growth factors like vascular endothelial growth factor (VEGF) and prostaglandin E2 to prolong the anagen phase[41]. Additionally, minoxidil promotes dermal papilla cell (DPC) survival by activating extracellular signal-regulated kinase (ERK) and Akt pathways and preventing apoptosis, further supporting hair growth[41,42].

Topical minoxidil is available in various forms, including solutions, foams, and shampoos, with 2%, 3%, and 5% concentrations. The 5% formulation is generally more effective, particularly in MPHL[10], while both 2% and 5% concentrations have demonstrated efficacy for FPHL. Notably, a randomized controlled study found that 5% foam used once daily was noninferior to 2% solution applied twice daily in increasing hair count and width but showed better tolerance[43]. Additionally, participants using 5%

foam reported less interference with hair styling. As a result, the most common regimen involves applying 5% foam once daily for women and twice daily for men, with a minimum recommended treatment period of six months to assess effectiveness. Potential side effects include scalp irritation—such as itching or redness—often due to the propylene glycol in the solution[29]. Foam-based formulations, which are free from propylene glycol, can be an alternative for those with sensitive skin. Additionally, some users experience temporarily increased hair shedding at the beginning of treatment as hair cycles synchronize; this shedding usually subsides with continued use. Although minoxidil is generally considered safe, it is not advised during pregnancy due to insufficient data on its effects on fetal health.

2) Oral minoxidil

Oral minoxidil was initially limited in use for hair loss treatment due to side effects observed at its original doses (10–40 mg) for hypertension, including postural hypotension, fluid retention, tachycardia, pericarditis, and nausea. However, recent data on low-dose applications have demonstrated favorable results in both efficacy and safety, leading to renewed interest in this treatment option[44,45]. Dosing generally ranges from 2.5–5 mg daily for men and 0.25–1.25 mg daily for women. A recently published consensus study indicated that a majority of experts agreed on the most frequently prescribed starting doses of low-dose oral minoxidil (LDOM), which are 2.5 mg/day for men and 1.25 mg/day for women, with subsequent dose adjustments based on efficacy and adverse effects[46]. Studies have shown significant improvements in hair density in both sexes[47,48].

The most common side effect is hypertrichosis, which occurs in approximately 10%–20% of patients, particularly at higher doses (5 mg), while lower doses yield fewer instances[44]. Hypertrichosis typically appears within the first three months of treatment, primarily affecting the sideburns, temples, arms, upper lip, and chin. It is generally mild and can be managed with hair removal methods (laser, waxing, shaving) or dose reduction, with only a small percentage of patients requiring treatment discontinuation[46,49]. Aside from hypertrichosis, other adverse effects such as lightheadedness (1.7%), fluid retention (1.3%), and tachycardia (0.9%) may rarely occur[44]. These effects are generally dose-dependent and tend to resolve upon discontinuation of the drug. Recent studies have reported that LDOM does not have a significant impact on blood pressure[50,51].

Overall, LDOM presents a promising alternative, particularly for FPHL patients who lack access to or do not benefit from potent 5-α-reductase inhibitors commonly used for MPHL. It is also valuable for MPHL patients who are unable to use 5-α-reductase inhibitors or who have had inadequate responses to these treatments.

LLLT

LLLT is a non-invasive AGA treatment approved by the FDA in 2007 for men and 2011 for women[3]. It uses red or near-infrared light (600–950 nm) to stimulate hair follicles by enhancing mitochondrial activity, increasing ATP production, and promoting nitric oxide release[52-54]. These effects improve scalp blood flow and support follicular growth, potentially prolonging the anagen phase and encouraging hair regrowth.

In clinical studies, LLLT has shown potential in improving AGA. The typical treatment regimen involves 15–20 minute sessions, once a day or three times a week, over a period of at least four to six months, with energy levels between 2 and 10 J/cm^2. Comb-based and helmet-based devices have demonstrated significant results in hair count and thickness in both randomized controlled trials and systematic reviews[55-58]. In our randomized double-blinded study conducted in Korea with 40 male and female AGA patients, 24 weeks of LLLT treatment significantly increased hair density and diameter without any serious adverse[59]. In another randomized study conducted in Korea using a different LLLT device, significant improvements in hair loss were observed in male and female AGA patients after 4 months of treatment[60]. An extension study revealed that continuous treatment for up to one year further enhanced hair density and diameter, with notable benefits observed across mild, moderate, and severe AGA groups (unpublished data).

LLLT is generally well tolerated with minimal side effects, including mild dryness, tenderness, or scalp irritation. While current findings support LLLT as a beneficial adjunct or alternative therapy for AGA, ongoing research aims to refine treatment parameters including light intensity, duration, and frequency, and confirm long-term efficacy to establish standardized protocols for optimal outcomes.

Platelet-rich plasma (PRP)

PRP therapy utilizes concentrated platelets and growth factors to promote healing and regeneration and has recently been applied to hair loss treatment. It stimulates DPC proliferation by enhancing fibroblast growth factor-7, β-catenin, and activating ERK and Akt pathways while promoting angiogenesis via VEGF, supporting active hair growth[61-63]. Studies have shown PRP to be effective, with significant improvements in hair density and thickness observed in both MPHL and FPHL[64,65]. However, individual response rates vary, and the treatment may be less effective in advanced AGA cases where follicles are severely compromised. In a study conducted in Korea using CD34+ cell-containing autologous PRP, two treatments administered at 3-month intervals significantly increased both hair count and thickness compared to baseline[66].

PRP is well-tolerated, with side effects usually limited to temporary scalp discomfort and minor bleeding. The treatment's

limitations, however, include its high cost and the need for regular maintenance sessions, typically requiring an initial series of injections followed by follow-ups every few months. Additionally, there is a lack of standardized protocols in PRP preparation and application, leading to variable results across clinics.

Intradermal botulinum toxin

Botulinum toxin A (BoTX-A) has emerged as a potential treatment for AGA due to its effects on scalp muscle relaxation, which may improve blood flow and reduce the DHT-related suppression of hair follicles[1]. By alleviating scalp tension and inhibiting the release of TGF-β1—a factor influenced by DHT and linked to follicle miniaturization—BoTX-A helps create a healthier environment for hair growth[67]. Studies have shown promising results. In a 24-week trial, BoTX-A injections resulted in notable increases in hair count and visual improvement[67]. Furthermore, BoTX-A injections combined with oral finasteride and minoxidil significantly improved hair density compared to finasteride and minoxidil alone[68,69]. Though these findings support BoTX-A as an effective adjunct to existing AGA treatments, further research is needed to clarify botulinum toxin's role as a treatment for AGA.

Exosomes

Exosomes are rich in growth factors, cytokines, and microRNAs that influence signaling pathways critical to hair follicle development and regeneration[70]. Stem cell-derived exosomes, often referring to the conditioned media obtained from culturing stem cells, can interact with target cells by binding to membrane receptors, activating signaling cascades that promote cell proliferation and differentiation.

In AGA, exosomes show potential for promoting hair follicle regeneration by enhancing DPC proliferation and encouraging follicles to enter the anagen phase[71,72]. Preclinical studies have demonstrated that mesenchymal stem cell-derived exosomes may effectively increase hair growth and follicle activity in animal models of AGA[73]. Early clinical data, though limited, suggest comparable benefits for AGA patients. Studies have shown improvements in hair thickness and density, as well as enhanced photographic assessments, at 12 or 24 weeks following exosome treatment[3,70,74]. However, exosome therapy for AGA still faces several limitations. First, exosomes are currently approved only as cosmetics, restricting their use as injectables or medicinal treatments. Additionally, the lack of standardized application protocols, combined with low production yields and high costs, poses significant challenges. While the initial results are promising, additional research is needed to confirm long-term safety and efficacy, optimize dosing and application methods, and develop comprehensive guidelines before exosome therapy can be widely adopted as a routine AGA treatment.

NOVEL APPROACHES TARGETING THE AR

Recently, directly targeting the AR has gained attention as a promising therapeutic approach for AGA. Research has identified that DHT binding to AR in DPCs is a key driver of hair follicle miniaturization, the hallmark of AGA. These innovative therapies aim to selectively inhibit AR activity in scalp hair follicles, providing effective treatment while reducing systemic side effects typically associated with androgen-targeting therapies.

Pyrilutamide, a topical AR antagonist, has shown promising results in recent phase II clinical trials (https://clinicaltrials.gov/study/NCT05218642). Pyrilutamide has demonstrated efficacy in both MPHL and FPHL patients, significantly increasing hair count in men and improving both hair thickness and count in women. While mild local irritation was reported in some cases, no serious adverse effects were observed, highlighting its safety and efficacy. In addition, GT20029, a proteolysis-targeting chimera compound designed to degrade the AR, has demonstrated significant efficacy in treating MPHL in phase II clinical trials (https://clinicaltrials.gov/study/NCT06692465).

Another advanced approach is small interfering RNA (siRNA) therapy, which utilizes RNA interference to silence AR gene. siRNA is inherently labile, requiring advanced technologies to enhance its stability for therapeutic applications. The development of self-assembled micelle inhibitory RNA has further enhanced this approach by avoiding immune activation and improving delivery to hair follicles[75]. Clinical trials in injection of both low and high doses showed encouraging results, with hair density improvements comparable to standard AGA treatments but with minimal side effects[67]. Another study investigated cell-penetrating asymmetric siRNA, designed to specifically silence AR in targeted cells[76]. It offers distinct advantages over conventional siRNA, including higher selectivity, stability, and efficiency, which enhance its therapeutic potential. In both human scalp tissue models and animal studies, injection of this siRNA reduced AR protein levels, decreased markers associated with follicle regression, and increased hair follicle diameter[76]. These developments suggest that siRNA-based treatments could offer a more focused approach to managing AGA by directly targeting molecular pathways involved in hair follicle miniaturization without the systemic side effects associated with hormonal treatments. Further studies are necessary to refine dosage, delivery methods, and long-term safety to bring these treatments closer to clinical use.

CONCLUSIONS & FUTURE DIRECTIONS

The management of AGA has evolved significantly, expanding from traditional therapies to a range of innovative treatments.

While conventional treatments such as 5-α-reductase inhibitors and minoxidil remain central to AGA management, emerging therapies like PRP, LLLT, botulinum toxin injections, exosome therapy provide valuable alternatives. These options may be particularly beneficial for patients who cannot use or respond poorly to conventional therapies and may also enhance outcomes when combined with traditional treatments. Future directions in AGA treatment should include the development of personalized treatment plans that incorporate a patient's genetic, hormonal, and environmental factors to optimize outcomes. Advancements in delivery systems, such as injectable formulations and targeted siRNA technology, may further improve the efficacy and convenience of AGA therapies. Continued research and clinical trials will be crucial to fully integrate these innovations, ultimately providing safer, more effective options for individuals affected by AGA.

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DATA SHARING STATEMENT

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REFERENCES

1. Devjani S, Ezemma O, Kelley KJ, Stratton E, Senna M. Androgenetic alopecia: therapy update. Drugs 2023;83:701-715. PUBMED | CROSSREF
2. Paik JH, Yoon JB, Sim WY, Kim BS, Kim NI. The prevalence and types of androgenetic alopecia in Korean men and women. Br J Dermatol 2001;145:95-99. PUBMED | CROSSREF
3. Ntshingila S, Oputu O, Arowolo AT, Khumalo NP. Androgenetic alopecia: an update. JAAD Int 2023;13:150-158. PUBMED | CROSSREF
4. Ceruti JM, Leirós GJ, Balañá ME. Androgens and androgen receptor action in skin and hair follicles. Mol Cell Endocrinol 2018;465:122-133. PUBMED | CROSSREF
5. Kaufman KD. Androgens and alopecia. Mol Cell Endocrinol 2002;198:89-95. PUBMED | CROSSREF
6. Tan MH, Li J, Xu HE, Melcher K, Yong EL. Androgen receptor: structure, role in prostate cancer and drug discovery. Acta Pharmacol Sin 2015;36:3-23. PUBMED | CROSSREF
7. Sánchez P, Serrano-Falcón C, Torres JM, Serrano S, Ortega E. 5α-Reductase isozymes and aromatase mRNA levels in plucked hair from young women with female pattern hair loss. Arch Dermatol Res 2018;310:77-83. PUBMED | CROSSREF
8. Gupta AK, Charrette A. The efficacy and safety of 5α-reductase inhibitors in androgenetic alopecia: a network meta-analysis and benefit-risk assessment of finasteride and dutasteride. J Dermatolog Treat 2014;25:156-161. PUBMED | CROSSREF
9. Rossi A, Cantisani C, Scarnò M, Trucchia A, Fortuna MC, Calvieri S. Finasteride, 1 mg daily administration on male androgenetic alopecia in different age groups: 10-year follow-up. Dermatol Ther 2011;24:455-461. PUBMED | CROSSREF
10. Olsen EA, Whiting DA, Savin R, Rodgers A, Johnson-Levonas AO, Round E, et al. Global photographic assessment of men aged 18 to 60 years with male pattern hair loss receiving finasteride 1 mg or placebo. J Am Acad Dermatol 2012;67:379-386. PUBMED | CROSSREF
11. Shin JW, Chung EH, Kim MB, Kim TO, Kim WI, Huh CH. Evaluation of long-term efficacy of finasteride in Korean men with androgenetic alopecia using the basic and specific classification system. J Dermatol 2019;46:139-143. PUBMED | CROSSREF
12. Hu R, Xu F, Sheng Y, Qi S, Han Y, Miao Y, et al. Combined treatment with oral finasteride and topical minoxidil in male androgenetic alopecia: a randomized and comparative study in Chinese patients. Dermatol Ther 2015;28:303-308. PUBMED | CROSSREF
13. Price VH, Roberts JL, Hordinsky M, Olsen EA, Savin R, Bergfeld W, et al. Lack of efficacy of finasteride in postmenopausal women with androgenetic alopecia. J Am Acad Dermatol 2000;43:768-776. PUBMED | CROSSREF
14. Iamsumang W, Leerunyakul K, Suchonwanit P. Finasteride and its potential for the treatment of female pattern hair loss: evidence to date. Drug Des Devel Ther 2020;14:951-959. PUBMED | CROSSREF
15. Lee S, Lee YB, Choe SJ, Lee WS. Adverse sexual effects of treatment with finasteride or dutasteride for male androgenetic alopecia: a systematic review and meta-analysis. Acta Derm Venereol 2019;99:12-17. PUBMED | CROSSREF
16. Irwig MS. Depressive symptoms and suicidal thoughts among former users of finasteride with persistent sexual side effects. J Clin Psychiatry 2012;73:1220-1223. PUBMED | CROSSREF
17. D'Amico AV, Roehrborn CG. Effect of 1 mg/day finasteride on concentrations of serum prostate-specific antigen in men with androgenic alopecia: a randomised controlled trial. Lancet Oncol 2007;8:21-25. PUBMED | CROSSREF
18. Thompson IM, Goodman PJ, Tangen CM, Lucia MS, Miller GJ, Ford LG, et al. The influence of finasteride on the development of prostate cancer. N Engl J Med 2003;349:215-224. PUBMED | CROSSREF
19. Ferrando J, Grimalt R, Alsina M, Bulla F, Manasievska E. Unilateral gynecomastia induced by treatment with 1 mg of oral finasteride. Arch Dermatol 2002;138:543-544. PUBMED | CROSSREF
20. Mansouri P, Farshi S, Safar F. Finasteride-induced gynecomastia. Indian J Dermatol Venereol Leprol 2009;75:309-310. PUBMED | CROSSREF
21. Meijer M, Thygesen LC, Green A, Emneus M, Brasso K, Iversen P, et al. Finasteride treatment and male breast cancer: a register-based cohort study in four Nordic countries. Cancer Med 2018;7:254-260. PUBMED | CROSSREF
22. Clark RV, Hermann DJ, Cunningham GR, Wilson TH, Morrill BB, Hobbs S. Marked suppression of dihydrotestosterone in men with benign prostatic hyperplasia by dutasteride, a dual 5alpha-reductase inhibitor. J Clin Endocrinol Metab 2004;89:2179-2184. PUBMED | CROSSREF
23. Olsen EA, Hordinsky M, Whiting D, Stough D, Hobbs S, Ellis ML, et al. The importance of dual 5alpha-reductase inhibition in the treatment of male pattern hair loss: results of a randomized placebo-

controlled study of dutasteride versus finasteride. J Am Acad Dermatol 2006;55:1014-1023. PUBMED | CROSSREF

24. Gupta AK, Venkataraman M, Talukder M, Bamimore MA. Relative efficacy of minoxidil and the 5-α reductase inhibitors in androgenetic alopecia treatment of male patients: a network meta-analysis. JAMA Dermatol 2022;158:266-274. PUBMED | CROSSREF
25. Gupta AK, Talukder M, Williams G. Comparison of oral minoxidil, finasteride, and dutasteride for treating androgenetic alopecia. J Dermatolog Treat 2022;33:2946-2962. PUBMED | CROSSREF
26. Choi S, Kwon SH, Sim WY, Lew BL. Long-term efficacy and safety of dutasteride 0.5 mg in Korean men with androgenetic alopecia: 5-year data demonstrating clinical improvement with sustained efficacy. J Dermatol 2024;51:684-690. PUBMED | CROSSREF
27. Herz-Ruelas ME, Álvarez-Villalobos NA, Millán-Alanís JM, de León-Gutiérrez H, Ocampo-Garza SS, Gómez-Flores M, et al. Efficacy of intralesional and oral dutasteride in the treatment of androgenetic alopecia: a systematic review. Skin Appendage Disord 2020;6:338-345. PUBMED | CROSSREF
28. Hajheydari Z, Akbari J, Saeedi M, Shokoohi L. Comparing the therapeutic effects of finasteride gel and tablet in treatment of the androgenetic alopecia. Indian J Dermatol Venereol Leprol 2009;75:47-51. PUBMED | CROSSREF
29. Kelly Y, Blanco A, Tosti A. Androgenetic alopecia: an update of treatment options. Drugs 2016;76:1349-1364. PUBMED | CROSSREF
30. Piraccini BM, Blume-Peytavi U, Scarci F, Jansat JM, Falqués M, Otero R, et al. Efficacy and safety of topical finasteride spray solution for male androgenetic alopecia: a phase III, randomized, controlled clinical trial. J Eur Acad Dermatol Venereol 2022;36:286-294. PUBMED | CROSSREF
31. Suchonwanit P, Iamsumang W, Rojhirunsakool S. Efficacy of topical combination of 0.25% finasteride and 3% minoxidil versus 3% minoxidil solution in female pattern hair loss: a randomized, double-blind, controlled study. Am J Clin Dermatol 2019;20:147-153. PUBMED | CROSSREF
32. Kim H, Ryu C, Lee M, Lee KR, Kim J. A phase I, open-label, sequential, single-dose clinical trial to evaluate the pharmacokinetic, pharmacodynamic, and safety of IVL3001, a finasteride-based novel long-acting injection for androgenetic alopecia. Adv Ther 2024;41:2936-2952. PUBMED | CROSSREF
33. Kim JH, Na J, Bak DH, Lee BC, Lee E, Choi MJ, et al. Development of finasteride polymer microspheres for systemic application in androgenic alopecia. Int J Mol Med 2019;43:2409-2419. PUBMED | CROSSREF
34. Sinclair R, Wewerinke M, Jolley D. Treatment of female pattern hair loss with oral antiandrogens. Br J Dermatol 2005;152:466-473. PUBMED | CROSSREF
35. Sadeghzadeh Bazargan A, Tavana Z, Dehghani A, Jafarzadeh A, Tabavar A, Alavi Rad E, et al. The efficacy of the combination of topical minoxidil and oral spironolactone compared with the combination of topical minoxidil and oral finasteride in women with androgenic alopecia, female and male hair loss patterns: a blinded randomized clinical trial. J Cosmet Dermatol 2024;23:543-551. PUBMED | CROSSREF
36. Atanaskova Mesinkovska N, Bergfeld WF. Hair: what is new in diagnosis and management? Female pattern hair loss update: diagnosis and treatment. Dermatol Clin 2013;31:119-127. PUBMED | CROSSREF
37. Vexiau P, Chaspoux C, Boudou P, Fiet J, Jouanique C, Hardy N, et al. Effects of minoxidil 2% vs. cyproterone acetate treatment on female androgenetic alopecia: a controlled, 12-month randomized trial. Br J Dermatol 2002;146:992-999. PUBMED | CROSSREF
38. Kim JH, Lee SY, Lee HJ, Yoon NY, Lee WS. The efficacy and safety of 17α-estradiol (Ell-Cranell® alpha 0.025%) solution on female pattern hair loss: single center, open-label, non-comparative, phase IV study. Ann Dermatol 2012;24:295-305. PUBMED | CROSSREF
39. Blume-Peytavi U, Kunte C, Krisp A, Garcia Bartels N, Ellwanger U, Hoffmann R. Comparison of the efficacy and safety of topical minoxidil and topical alfatradiol in the treatment of androgenetic alopecia in women. J Dtsch Dermatol Ges 2007;5:391-395. PUBMED | CROSSREF
40. Choe SJ, Lee S, Choi J, Lee WS. Therapeutic efficacy of a combination therapy of topical 17α-estradiol and topical minoxidil on female pattern hair loss: a noncomparative, retrospective evaluation. Ann Dermatol 2017;29:276-282. PUBMED | CROSSREF
41. Messenger AG, Rundegren J. Minoxidil: mechanisms of action on hair growth. Br J Dermatol 2004;150:186-194. PUBMED | CROSSREF
42. Han JH, Kwon OS, Chung JH, Cho KH, Eun HC, Kim KH. Effect of minoxidil on proliferation and apoptosis in dermal papilla cells of human hair follicle. J Dermatol Sci 2004;34:91-98. PUBMED | CROSSREF
43. Blume-Peytavi U, Hillmann K, Dietz E, Canfield D, Garcia Bartels N. A randomized, single-blind trial of 5% minoxidil foam once daily versus 2% minoxidil solution twice daily in the treatment of androgenetic alopecia in women. J Am Acad Dermatol 2011;65:1126-1134.e2. PUBMED | CROSSREF
44. Vañó-Galván S, Pirmez R, Hermosa-Gelbard A, Moreno-Arrones ÓM, Saceda-Corralo D, Rodrigues-Barata R, et al. Safety of low-dose oral minoxidil for hair loss: a multicenter study of 1404 patients. J Am Acad Dermatol 2021;84:1644-1651. PUBMED | CROSSREF
45. Ramos PM, Sinclair RD, Kasprzak M, Miot HA. Minoxidil 1 mg oral versus minoxidil 5% topical solution for the treatment of female-pattern hair loss: a randomized clinical trial. J Am Acad Dermatol 2020;82:252-253. PUBMED | CROSSREF
46. Akiska YM, Mirmirani P, Roseborough I, Mathes E, Bhutani T, Ambrosy A, et al. Low-dose oral minoxidil initiation for patients with hair loss: an international modified Delphi consensus statement. JAMA Dermatol 2025;161:87-95. PUBMED | CROSSREF
47. Vastarella M, Cantelli M, Patrì A, Annunziata MC, Nappa P, Fabbrocini G. Efficacy and safety of oral minoxidil in female androgenetic alopecia. Dermatol Ther 2020;33:e14234. PUBMED | CROSSREF
48. Panchaprateep R, Lueangarun S. Efficacy and safety of oral minoxidil 5 mg once daily in the treatment of male patients with androgenetic alopecia: an open-label and global photographic assessment. Dermatol Ther (Heidelb) 2020;10:1345-1357. PUBMED | CROSSREF
49. Jimenez-Cauhe J, Saceda-Corralo D, Rodrigues-Barata R, Hermosa-Gelbard A, Moreno-Arrones OM, Gil-Redondo R, et al. Characterization and management of hypertrichosis induced by low-dose oral minoxidil in the treatment of hair loss. J Am Acad Dermatol 2021;84:222-223. PUBMED | CROSSREF
50. Müller Ramos P, Melo DF, Bhoyrul B, Sanabria BD, Jimenez-Cauhe J, Saceda-Corralo D, et al. Prospective evaluation of blood pressure, heart rate, and side effects in hypertensive patients using low-dose oral minoxidil for hair loss. J Am Acad Dermatol 2024;91:1011-1012. PUBMED | CROSSREF
51. Chen M, Weichert M, Guo W, Ruzic R, Briley J. Low-dose oral minoxidil does not significantly affect blood pressure: a systematic review and meta-analysis. J Am Acad Dermatol 2025;92:554-555. PUBMED | CROSSREF
52. Mester E, Szende B, Gärtner P. The effect of laser beams on the growth of hair in mice. Radiobiol Radiother (Berl) 1968;9:621-626. PUBMED
53. Jampa-Ngern S, Viravaidya-Pasuwat K, Suvanasuthi S, Khantachawana A. Effect of laser diode light irradiation on growth capability of human hair follicle dermal papilla cells. Annu Int Conf IEEE Eng Med Biol Soc 2017;2017:3592-3595. PUBMED | CROSSREF

54. Kalia S, Lui H. Utilizing electromagnetic radiation for hair growth: a critical review of phototrichogenesis. Dermatol Clin 2013;31:193-200. PUBMED | CROSSREF

55. Leavitt M, Charles G, Heyman E, Michaels D. HairMax LaserComb laser phototherapy device in the treatment of male androgenetic alopecia: a randomized, double-blind, sham device-controlled, multicentre trial. Clin Drug Investig 2009;29:283-292. PUBMED | CROSSREF

56. Jimenez JJ, Wikramanayake TC, Bergfeld W, Hordinsky M, Hickman JG, Hamblin MR, et al. Efficacy and safety of a low-level laser device in the treatment of male and female pattern hair loss: a multicenter, randomized, sham device-controlled, double-blind study. Am J Clin Dermatol 2014;15:115-127. PUBMED | CROSSREF

57. Pillai JK, Mysore V. Role of low-level light therapy (LLLT) in androgenetic alopecia. J Cutan Aesthet Surg 2021;14:385-391. PUBMED | CROSSREF

58. Lueangarun S, Visutjindaporn P, Parcharoen Y, Jamparuang P, Tempark T. A systematic review and meta-analysis of randomized controlled trials of united states food and drug administration-approved, home-use, low-level light/laser therapy devices for pattern hair loss: device design and technology. J Clin Aesthet Dermatol 2021;14:E64-E75. PUBMED

59. Kim H, Choi JW, Kim JY, Shin JW, Lee SJ, Huh CH. Low-level light therapy for androgenetic alopecia: a 24-week, randomized, double-blind, sham device-controlled multicenter trial. Dermatol Surg 2013;39:1177-1183. PUBMED | CROSSREF

60. Kim JWKY, Chang YY, Hong SH, Shin JW, Na JI, Huh CH. Low-level laser therapy with novel array of light source and individualized program for treatment of androgenetic alopecia: a 16-week, randomized, double-blind, sham device-controlled study. Med Lasers 2020;9:150-158. CROSSREF

61. Li ZJ, Choi HI, Choi DK, Sohn KC, Im M, Seo YJ, et al. Autologous platelet-rich plasma: a potential therapeutic tool for promoting hair growth. Dermatol Surg 2012;38:1040-1046. PUBMED | CROSSREF

62. Gupta AK, Cole J, Deutsch DP, Everts PA, Niedbalski RP, Panchaprateep R, et al. Platelet-rich plasma as a treatment for androgenetic alopecia. Dermatol Surg 2019;45:1262-1273. PUBMED | CROSSREF

63. Cheng H, Zhang J, Li J, Jia M, Wang Y, Shen H. Platelet-rich plasma stimulates angiogenesis in mice which may promote hair growth. Eur J Med Res 2017;22:39. PUBMED | CROSSREF

64. Paichitrojjana A, Paichitrojjana A. Platelet rich plasma and its use in hair regrowth: a review. Drug Des Devel Ther 2022;16:635-645. PUBMED | CROSSREF

65. Tosti A, Rigopoulos D. Platelet-rich plasma in the treatment of hair disorders. Skin Appendage Disord 2018;4:I. PUBMED | CROSSREF

66. Kang JS, Zheng Z, Choi MJ, Lee SH, Kim DY, Cho SB. The effect of CD34+ cell-containing autologous platelet-rich plasma injection on pattern hair loss: a preliminary study. J Eur Acad Dermatol Venereol 2014;28:72-79. PUBMED | CROSSREF

67. Shon U, Kim MH, Lee DY, Kim SH, Park BC. The effect of intradermal botulinum toxin on androgenetic alopecia and its possible mechanism. J Am Acad Dermatol 2020;83:1838-1839. PUBMED | CROSSREF

68. Zhou Y, Yu S, Zhao J, Feng X, Zhang M, Zhao Z. Effectiveness and safety of botulinum toxin type a in the treatment of androgenetic alopecia. BioMed Res Int 2020;2020:1501893. PUBMED | CROSSREF

69. Tian K, Gao S, Jia Z, Xu W, Li K, Wu L. A study of combination unilateral subcutaneous botulinum toxin a treatment for androgenetic alopecia. J Cosmet Dermatol 2022;21:5584-5590. PUBMED | CROSSREF

70. Ersan M, Ozer E, Akin O, Tasli PN, Sahin F. Effectiveness of exosome treatment in androgenetic alopecia: outcomes of a prospective study. Aesthetic Plast Surg 2024;48:4262-4271. PUBMED | CROSSREF

71. Gupta AK, Wang T, Rapaport JA. Systematic review of exosome treatment in hair restoration: preliminary evidence, safety, and future directions. J Cosmet Dermatol 2023;22:2424-2433. PUBMED | CROSSREF

72. Kim H, Jang Y, Kim EH, Jang H, Cho H, Han G, et al. Potential of colostrum-derived exosomes for promoting hair regeneration through the transition from telogen to anagen phase. Front Cell Dev Biol 2022;10:815205. PUBMED | CROSSREF

73. Kost Y, Muskat A, Mhaimeed N, Nazarian RS, Kobets K. Exosome therapy in hair regeneration: a literature review of the evidence, challenges, and future opportunities. J Cosmet Dermatol 2022;21:3226-3231. PUBMED | CROSSREF

74. Lee E, Choi MS, Cho BS, Won YJ, Lee JH, Kim SR, et al. The efficacy of adipose stem cell-derived exosomes in hair regeneration based on a preclinical and clinical study. Int J Dermatol 2024;63:1212-1220. PUBMED | CROSSREF

75. Yun SI, Lee SK, Goh EA, Kwon OS, Choi W, Kim J, et al. Weekly treatment with SAMiRNA targeting the androgen receptor ameliorates androgenetic alopecia. Sci Rep 2022;12:1607. PUBMED | CROSSREF

76. Moon IJ, Yoon HK, Kim D, Choi ME, Han SH, Park JH, et al. Efficacy of asymmetric siRNA targeting androgen receptors for the treatment of androgenetic alopecia. Mol Pharm 2023;20:128-135. PUBMED | CROSSREF

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