Molecular Mechanisms and Targeted Therapeutics in Androgenetic Alopecia: From Signaling Pathways to Regenerative Approaches

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ABSTRACT

Androgenetic alopecia (AGA) is the most common type of hair loss in both sexes, marked by progressive follicular shrinking influenced by genetic, hormonal, metabolic, and environmental variables. This study offers an extensive examination of AGA, addressing its epidemiology, hair biology, pathophysiology, and both existing and emerging treatment modalities. Androgenetic alopecia (AGA) is significantly affected by androgens, especially dihydrotestosterone (DHT), which engages androgen receptors and modifies local growth factor signaling, resulting in hair follicle regression. Genetic susceptibility, disruptions in signaling pathways (WNT/β-catenin, BMP, FGF5), hormonal abnormalities, dietary inadequacies, environmental exposures, and inflammation collectively contribute to the illness. Presently, FDA-sanctioned treatments comprise topical minoxidil and oral finasteride, but novel therapies emphasize stem cell interventions, JAK inhibitors, WNT pathway activators, prostaglandin analogs, and innovative biologics aimed at the molecular causes underlying follicle shrinking. Non-pharmacological therapies, including platelet-rich plasma, microneedling, low-level laser therapy, and hair transplantation, offer supplementary therapeutic advantages. Comprehending the multifaceted etiology of AGA is essential for formulating individualized and efficient treatment options.

Keywords: Androgenetic alopecia, Dihydrotestosterone, Follicular miniaturization, Genetic abnormality, Hormonal imbalances, Signaling pathways.

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Cite this as: Carpenter H, Kumar C, Bhatol J, Vishwakarma N. Molecular Mechanisms and Targeted Therapeutics in Androgenetic Alopecia: From Signaling Pathways to Regenerative Approaches. Premier Journal of Science 2026;20:100278

DOI: https://doi.org/10.70389/PJS.100278

Peer Review:

Received: 26 December 2025

Last revised: 09 March 2026

Accepted: 10 May 2026

Version accepted: 2

Published: 17 May 2026

Ethical approval: N/a

Consent: N/a

Funding: N/a

Conflicts of interest: N/a

Author contribution: Hariom Carpenter – Supervision, Writing – original draft, Writing – review, editing

Guarantor: Hariom Carpenter

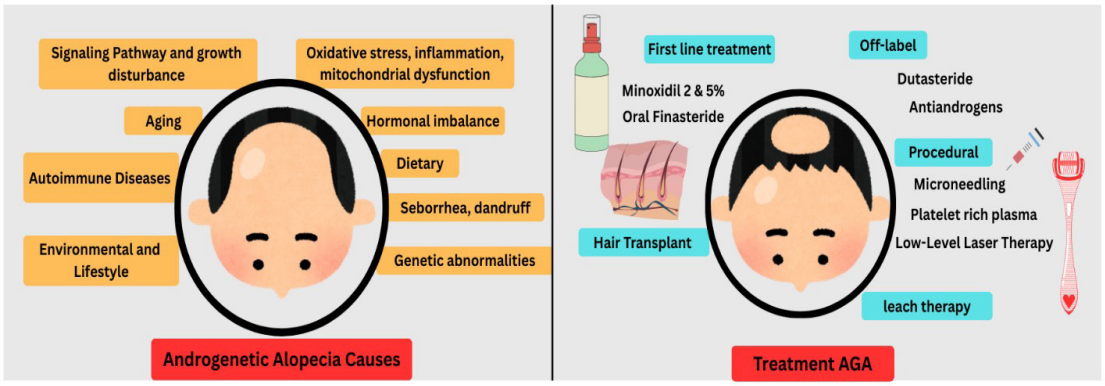
Provenance and peer-review: Unsolicited and externally peer-reviewed

Data availability statement: N/a

Introduction

Alopecia is a disorder of hair loss in which the scalp or other areas of the body experience partial or total hair loss. Numerous factors, including genetic predisposition, autoimmune illnesses, hormonal imbalances, stress, environmental factors, dietary deficiencies, and the effects of certain drugs, can contribute to this issue, which can be either temporary or permanent. The two primary types of alopecia are scarring (where hair follicles are permanently destroyed) and non-scarring (where hair follicles remain). Alopecia areata (circular bald spots caused by autoimmune illnesses), telogen effluvium (excessive hair loss following stress or sickness), traction alopecia (due to tightly knotted hairstyles), and androgenic alopecia (male/female pattern baldness) are among the non-scarring kinds. Conditions like lichen planopilaris and folliculitis decalvans, which permanently harm hair follicles, are included in scarring alopecia. While some forms of alopecia can only be managed symptom-wise, others can be cured.[1,2] Androgenetic alopecia (AGA), often known as male pattern baldness, is the most frequent cause of baldness in men. It is mostly brought on by heredity and the effects of androgen hormones, dihydrotestosterone (DHT). Despite being a normal biological process, it has been demonstrated to cause men to age, lose confidence, and experience mental stress. People spend crores of rupees on hair treatment because it is directly associated with social and sexual attractiveness. The value of hair and how to heal baldness are frequently mentioned in history, including in Aristotelian ideas and ancient Egyptian writings. Hamilton's 1942 study, which demonstrated that AGA happens in men whose hair roots are genetically sensitive to DHT, marked the beginning of modern scientific understanding. AGA can still be triggered by androgen hormone, even if the body's levels are normal.[3]

The highest rates of AGA were found in women aged 60–69 (29.32%) and men aged 20–39 (29.37%).[4] According to a study, early balding in young Indian



Graphical Abstract: Androgenetic alopecia causes and their treatment

men may indicate metabolic issues, which include elevated blood pressure (136/91) and blood sugar (114), with 50% of men and 20% to 30% of women experiencing this condition by the age of 50.[5] AGA affects 30%–50% of males under 50 and 80% of Caucasian men, making it increasingly prevalent as people age. Older women have comparable rates.[6] Androgenetic alopecia is the most common reason for loss of hair in both men and women, but the pattern is different. It may thin at the top of the head and cause baldness in men. It begins at the temples and develops an "M" shaped hairline. Complete baldness is uncommon in women, although the hairline does not recede and the middle parting widens as the top of the head thins. It is connected to diabetes, high blood pressure, obesity, and heart disease, and prostate issues in males, and to hormonal abnormalities, including Polycystic Ovary Syndrome (PCOS) in women.[7] AGA was discovered in 58% of men in India between the ages of 30 and 50, compared to a substantially lower prevalence in China (21.3%) and Korea (14.1%). To diagnose AGA, a thorough patient history (family history, hormonal issues, lifestyle) and clinical examination (stretch test, trichoscopy) are crucial. AGA is identified by variations in hair diameter (HDD > 20%) and yellow spots in trichoscopy. Women may require PCOS and hormonal testing (prolactin, dehydroepiandrosterone sulfate). Only in exceptional circumstances are investigations such as phototrichograms and scalp biopsies conducted. To monitor the course of treatment, global photography is helpful.[8] The limitations of existing medications are addressed by antibody-based medicines (HMI-115, Tocilizumab, Anti-CXCL12, and Anti-DKK1), which provide a next-generation strategy to treating AGA. These novel medicines might soon be available to patients thanks to the ongoing research.[9] The first human clinical study of Eirion Therapeutics' topical product ET-02 has been successfully concluded. By healing the damage to the stem cells in the hair follicles, this treatment promotes the growth of natural hair again.[10]

Epidemiology of AGA

AGA has serious social and psychological repercussions and is one of the most prevalent causes of hair loss in India. According to studies, more than half of Indian males suffer from the condition; one estimate places the frequency among men between the ages of 30 and 50 at roughly 58%. As men age, the severity of hair loss increases. 79% of cases in a large outpatient sample were male, and type I or type II patterns of loss were the most common presentation.[11,12] Further investigation revealed that Ludwig grades I and II are the most common patterns among Indian women, with women accounting for 21% of cases and most frequently experiencing grade I hair loss. Although there is a substantial correlation between AGA severity and age in men, there is no discernible age association in the frequency of AGA in women.[13] Family history is significant; according to one study, those who have a family history of AGA are over 17 times more likely to be impacted.[14] In the Indian population, AGA is associated with a number of medical disorders in addition to hereditary risk. It has been discovered that men with AGA are more likely to develop metabolic syndrome, which is characterized by traits including lower high-density lipoprotein (HDL) cholesterol, a greater waist circumference, and higher triglycerides. Additionally, compared to healthy controls, vitamin D deficiency has been documented at noticeably greater rates in people with AGA. The psychological toll is significant, and research indicates that the primary determinant of quality of life is the extent of hair loss rather than cultural customs like donning a cap. All things considered, AGA is a very common ailment in India that affects both men and women. It is connected to systemic metabolic health, psychological wellness, and aging, in addition to heredity.[15,16]

Hair and Its Growth Cycle

Hair comprises a complicated structure with two primary components: the living hair follicle situated beneath the skin and the rich in keratin hair shaft that is visible above the skin. The follicle comprises three components: the infundibulum (the superior segment housing sebaceous glands), the isthmus (the intermediate segment containing stem cells), and the hair bulb (the inferior segment containing matrix cells). The outermost layer of hair comprises three layers: the outer protective cuticle, the middle cortex that imparts strength, and the inner medulla found in coarse hair. The dermal papilla governs hair growth and synthesizes the pigment melanin. This complete structure imparts hair its form, hue, resilience, and growing capacity.[17,18]

Hair growth is an ongoing cyclical process with four primary phases. The initial phase is anagen (growth phase), which endures for 2–6 years; during this period, hair proliferates fast, with 85%–90% of scalp hair in this phase. The second phase, catagen (transition phase), endures for approximately two weeks, during which hair growth ceases and the follicle begins to diminish. The third phase, telogen (resting stage), endures for 2–3 months during which hair growth ceases entirely. During the final phase, exogen, hair spontaneously sheds, thus initiating the cycle anew. The entire process is regulated by proteins such as Wingless-type/Int-1 (WNT), Bone Morphogenetic Protein (BMP), Fibroblast Growth Factor 5 (FGF5), and different hormones. The hair growth cycle persists throughout life, with its rate influenced by age, nutrition, hormonal levels, and various other factors.[19]

Pathophysiology

Genetic anomalies, hormonal influences, metabolic processes, micronutrient levels, micro-inflammation, and the effects of cranial bone prominences on scalp blood circulation collectively contribute to androgenetic alopecia (AGA). It is marked by progressive shrinkage caused by alterations in the hair cycle, ultimately leading to vellus hair due to the elongation of the anagen phase and the extension of the telogen phase.[17]

DOI: https://doi.org/10.70389/PJS.100278 | Premier Journal of Science 2026;20:100278

Signaling Pathway Disturbance

The regulation of the hair cycle relies on a careful equilibrium between activating and suppressive signaling pathways, with WNT/β-catenin, BMP, and FGF5 being central components. The WNT/β-catenin pathway starts the anagen phase by stabilizing β-catenin, allowing it to enter the nucleus and trigger T-cell factor/lymphoid enhancer factor (TCF/LEF)-dependent transcription, which encourages keratinocyte proliferation and follicle regeneration. In AGA, DHT increases the levels of Dickkopf-1 (DKK1), a strong WNT inhibitor, which disrupts β-catenin signaling and hinders stem cell activation, resulting in follicular miniaturization.[20,21] In contrast, BMP signaling, mainly through BMP2 and BMP4, acts as a negative regulator by activating Small Mothers Against Decapentaplegic (SMAD) proteins that enforce stem cell quiescence in the bulge region. Excessive BMP activity in the balding scalp further suppresses WNT activity, preventing effective regeneration and contributing to reduced hair thickness.[22] Complementing this, FGF5 functions as a molecular timer that terminates anagen by binding to FGFR1 and activating mitogen-activated protein kinase/extracellular signal-regulated kinase (MAPK/ERK) signaling, thereby inducing catagen. In AGA, enhanced or premature FGF5 signaling shortens anagen duration, producing progressively thinner and shorter hairs.[23] Together, these disturbances suppressed WNT activity and upregulated BMP signaling, and overactive FGF5 pathways interact with androgen signaling in dermal papilla cells to create the characteristic pattern of progressive follicle miniaturization seen in AGA.[24]

Genetic Abnormalities

Genetics play a major role in AGA, which is now understood to follow a polygenic pattern, influenced by many genes rather than simple dominant inheritance. Strong associations have been found on the X chromosome, particularly in the androgen receptor (AR) gene and the Ectodysplasin A2 Receptor (EDA2R) gene, with variants such as the AR-E211 A allele reducing the risk of hair loss. Other important genes include WNT10A on chromosome 2q35, which is crucial for hair follicle development, and loci on chromosomes 1p36.22, 2q37, 7p21.1, 7q11.22, 17q21.31, 18q21.1, and 20p11.[25] Mutations in the APCDD1 gene, a WNT signaling inhibitor, have also been linked to baldness, indicating that androgen-independent pathways contribute alongside androgen-related pathways.[26] In women, the X-chromosome AR/EDA2R locus and the aromatase (CYP19A1) gene have been associated with early-onset female pattern hair loss, highlighting differences in hormonal and genetic interactions between men and women. These findings, confirmed by recent genome-wide studies, emphasize the complex genetic architecture of AGA and are important for developing personalized diagnostic and therapeutic methodologies.[27,28]

Hormonal Imbalance

Hormones, particularly androgens, are central to AGA. Testosterone is converted to DHT by 5α-reductase enzymes (types I and II) in dermal papilla cells. DHT binds to androgen receptors (AR), especially in vertex and frontal follicles, and initiates transcription of genes promoting catagen induction, keratinocyte apoptosis, and hair follicle miniaturization.[29] DHT also alters the local balance of growth factors, upregulating inhibitors like transforming growth factor-beta (TGF-β) and Dickkopf-related protein 1 (DKK1) while downregulating promoters, including vascular endothelial growth factor (VEGF) and insulin-like growth factor 1 (IGF-1). This imbalance reduces follicular proliferation and vascular support, progressively transforming terminal hairs into thin vellus hairs. Enhanced expression of 5α-reductase and AR in susceptible follicles further amplifies DHT's effects, explaining the regional pattern of hair loss in AGA.[30,31] Estrogens may confer protection against hair loss by counteracting androgen effects, while melatonin can promote hair growth and reduce DHT's impact. On the other hand, prolactin and parathyroid hormone can trigger early hair cycle changes and worsen baldness. Stress-related hormones like corticotropin-releasing hormone (CRH) are linked to scalp inflammation and premature hair regression. Together, these findings highlight that AGA is not caused by a single factor but by a network of hormones and genes, offering new directions for developing treatments beyond the commonly used finasteride and minoxidil.[32,33]

Dietary Factors

Nutrition significantly impacts hair follicle health. Deficiencies in iron, zinc, biotin, protein, vitamin D, and essential fatty acids can impair keratinocyte proliferation, follicular matrix formation, and hair shaft quality. Conversely, diets high in sugar and fat may promote systemic inflammation, insulin resistance, and hormonal imbalances, indirectly exacerbating hair follicle miniaturization. These nutritional imbalances can accelerate AGA progression by weakening follicular resilience and reducing the regenerative potential of dermal papilla and bulge stem cells.[34,35]

Environmental Factors

Environmental exposures and lifestyle factors can contribute to follicular damage. Smoking, alcohol, UV radiation, and environmental pollutants generate oxidative stress and free radicals, damaging hair follicle cells.[36] Chronic psychological stress elevates cortisol levels, disrupting the hair cycle and promoting premature entry into catagen. Additionally, stress can modify the immune and inflammatory milieu around the follicle, further impairing growth. These factors act synergistically with genetic and hormonal predispositions to accelerate hair thinning.[37]

Other Factors

Other contributors include inflammation, oxidative stress, microbiome changes, and follicular aging. Perifollicular lymphocytic infiltration, mast cell activation, and cytokines like IL-6 create a pro-inflammatory environment that damages follicular cells.[38] Oxidative

stress and mitochondrial dysfunction reduce dermal papilla cell proliferation and stem cell potential. Scalp conditions, such as seborrhea, dandruff, and microbial imbalances, can further exacerbate local inflammation. Finally, aging-related follicular senescence reduces the duration of anagen and diminishes the regenerative capacity of stem cells, making hair miniaturization more pronounced over time.[39]

Treatment of Androgenetic Alopecia

AGA is multifaceted and includes pharmacological, non-pharmacological, surgical, and cosmetic approaches. The only FDA-approved drugs for AGA are topical minoxidil and oral finasteride. Minoxidil, offered in 2% formulations for women and 5% formulations for men, extends the anagen phase, enlarges follicular size, and improves scalp blood circulation.[40,41] Oral finasteride (1 mg/day) inhibits type II 5α-reductase, reducing DHT levels by about 60%–70% and slowing follicular miniaturization. Dutasteride, a dual type I and II 5α-reductase inhibitor, is more potent than finasteride and has shown superior efficacy in clinical trials; although not FDA-approved for AGA, it is used off-label in several countries.[42] Topical finasteride, designed to reduce systemic side effects, is gaining popularity in clinical practice. In women, especially those with hyperandrogenism, antiandrogen medications such as spironolactone, cyproterone acetate, and flutamide are administered, either individually or in conjunction with oral contraceptives. Additionally, ketoconazole 2% shampoo is used as an adjuvant due to its antiandrogenic and anti-inflammatory properties (Figure 1).[43,44]

Non-pharmacological treatments are increasingly popular, including low-level laser therapy (LLLT), which is FDA-cleared for home use and has been shown to stimulate mitochondrial activity in follicle cells.[45] Platelet-rich plasma (PRP) injections, which deliver growth factors like VEGF, FGF, and IGF-1 directly to the scalp, are widely used and have shown improvement in hair density and thickness. Microneedling, alone or combined with minoxidil or PRP, enhances drug penetration and activates wound-healing pathways that favor follicular regeneration.[46]

In advanced situations, hair transplantation is the gold standard, with methods such as follicular unit transplantation (FUT) and follicular unit extraction (FUE), providing enduring cosmetic treatments. Cosmetic options, such as hair fibers, sprays, scalp micropigmentation, wigs, and extensions, provide psychological relief and improve quality of life.[47,48]

In recent years, investigational therapies have emerged. Prostaglandin analogs, such as latanoprost and bimatoprost, have been tested for promoting hair growth. Wnt/β-catenin pathway activators, like valproic acid and SM04554, aim to stimulate follicular stem cells.[49] JAK inhibitors (ruxolitinib, tofacitinib, baricitinib), already successful in alopecia areata, are being explored for AGA to overcome stem cell dormancy.[50,51] Stem cell-based therapies, including adipose-derived stem cell-conditioned media and follicular cell injections, are in clinical trials, while growth factor-based peptides and bioengineered follicle regeneration hold promise for the future.[52,53] Despite these advancements, minoxidil, finasteride, and hair transplantation remain the cornerstones of current AGA management, while PRP, microneedling, and LLLT serve as effective adjuvants.[54]

Novel therapies aim to address molecular, cellular, and regenerative mechanisms underlying follicle miniaturization, offering the potential for more sustained and targeted outcomes. All the emerging and upcoming therapies are summarized in Table 1.

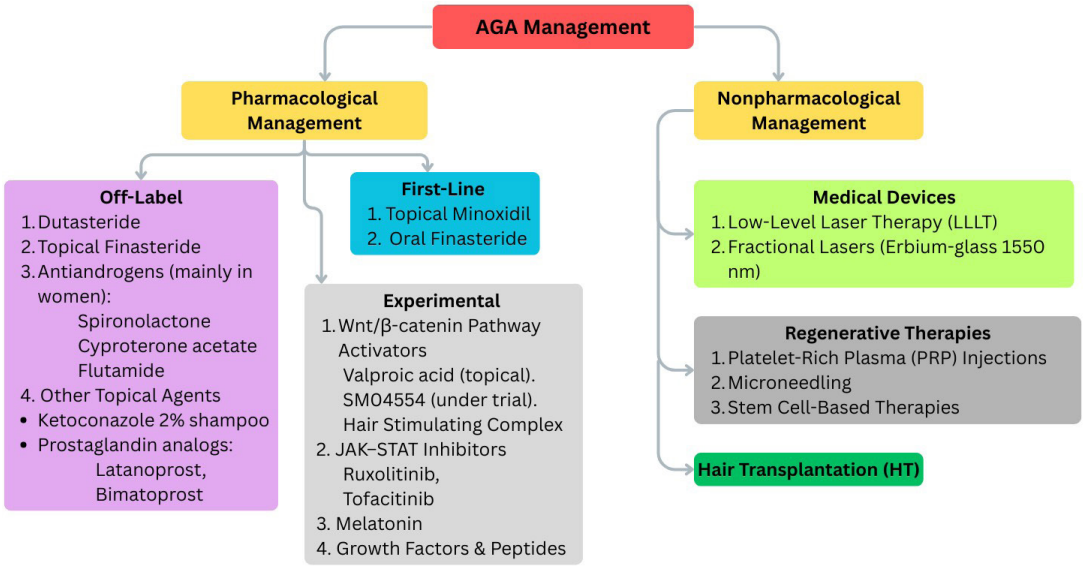


Fig 1 | Androgenetic alopecia management via pharmacological and non-pharmacological approaches

DOI: https://doi.org/10.70389/PJS.100278 | Premier Journal of Science 2026;20:100278

Table 1 | Current and emerging therapies for androgenetic alopecia: Mechanisms and clinical status

Category	Therapies	Mechanism/Notes	References
FDA-approved	Topical minoxidil	Prolongs anagen and increases follicle size and blood flow.	[55]
	Oral finasteride	5α-reductase inhibitor; reduces DHT by ~60%–70%.	[56]
Off-label	Dutasteride (oral/topical)	More potent dual 5α-reductase inhibitor.	[57]
	Oral minoxidil (low-dose)	Systemic vasodilator, stimulates follicles.	[58]
	Topical finasteride	Reduces systemic side effects; gaining use.	[59]
	Antiandrogens (spironolactone, cyproterone, flutamide, bicalutamide)	Block androgen receptors, esp. in women.	[60]
	Ketoconazole shampoo	Antiandrogenic & anti-inflammatory adjuvant.	[61]
Procedural/Device	PRP injections	Growth factors (VEGF, IGF-1, FGF) stimulate follicles.	[62]
	Microneedling (±PRP/minoxidil)	Enhances penetration & stem cell activation.	[59]
	Low-level laser therapy (LLLT)	Photobiostimulation enhances ATP in the dermal papilla.	[63]
	Hair transplant (FUE/FUT)	Permanent surgical replacement.	[64]
Emerging/Experimental	Stem cell therapy & exosomes	Regenerative medicine activates Wnt signaling.	[57]
	Antibody-based therapies (HMI-115, Tocilizumab, anti-CXCL12, anti-DKK1)	Novel biologics targeting inflammatory & Wnt/DKK1 pathways.	[65]
	Botulinum toxin	Reduces scalp tension, improves vascularity.	[66]
	Prostaglandin analogues (Bimatoprost, Latanoprost)	Stimulate prostaglandin F2α → anagen prolongation.	[67]
	FGF-5 inhibitors (peptides)	Block catagen-inducing FGF-5 → longer anagen.	[68]
	Prostaglandin receptor modulators (PGD2 antagonists, Setipiprant)	Block inhibitory PGD2 signaling.	[67]
	Wnt/β-catenin activators (e.g., valproic acid, SM04554, WAY-316606)	Stimulate follicle stem cells.	[67]
In clinical trials	Pyrilutamide (KX-826)	Topical AR antagonist; Phase III in China/US.	[69]
	SM04554 (Samumed)	Wnt pathway small molecule, ↑ density in Phase II.	[67]
	Clascoterone (CB-03-01)	Topical AR blocker; early trials promising.	[67]
	JAK inhibitors (tofacitinib, ruxolitinib, baricitinib)	Target JAK-STAT, under study in AGA (success in alopecia areata).	[70]
	ET-02 (Eirion therapeutics)	Topical stem cell-repair agent; completed Phase I.	[10]
	Raloxifene (SERM)	Under study: estrogen receptor modulation.	[69]
	Hair follicle bioengineering/organoids	Proof-of-concept: regenerates follicles for transplant.	[70]

Conclusion

Genetic predisposition, hormonal effects, metabolic variables, environmental exposures, and alterations in the local follicular microenvironment all interact to cause AGA, a complex disorder. New therapeutic methods are being made possible by developments in molecular biology and regenerative medicine, even while conventional treatments like minoxidil, finasteride, and hair transplantation are still the cornerstone of care. Targeting the underlying mechanisms of follicular shrinkage and stem cell dormancy, emerging interventions like stem cell-based therapies, JAK inhibitors, WNT pathway modulators, and prostaglandin analogs hold promise for more long-lasting and individualized results. To maximize hair regrowth and enhance patients' psychosocial well-being, early diagnosis, suitable treatment, and a comprehensive assessment of lifestyle, diet, and systemic health are crucial.

Future Perspective

In order to improve treatment efficacy, precision medicine techniques that combine genetic profiling, biomarker discovery, and customized therapy selection are probably going to be the main focus of future AGA research. For extreme situations, advanced regenerative techniques including organoid transplantation and bioengineered hair follicles may provide long-term fixes. Furthermore, combination treatments that target several pathways at once, hormonal, inflammatory, and stem cell, may have synergistic effects. The development of novel, non-invasive, and long-lasting treatments will be made easier by ongoing research into the molecular and cellular mechanisms behind hair follicle shrinking. This could revolutionize the management of AGA from symptomatic control to actual hair regeneration.

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