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Exploring Androgenic Alopecia: Mechanisms, Diagnosis, and Therapeutic Advances

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Abstract

Androgenetic alopecia (AGA) is a common form of hair loss, marked by a receding hairline in men and diffuse thinning in women, caused by the enzyme 5-alpha-reductase and dihydrotestosterone (DHT), which leads to hair follicle miniaturization. Several treatments, including medical, surgical, light-based, and nutraceutical options, aim to slow or reverse AGA, but selecting the right therapy can be challenging due to the chronic nature of the condition. FDA-approved treatments like topical minoxidil, oral finasteride, and low-level light therapy (LLLT) are standard, but other options such as oral and topical therapies, hormonal treatments, platelet-rich plasma (PRP), exosome therapies, and hair transplantation are also available. This review evaluates treatment options for AGA, considering effectiveness, side effects, pathophysiology, diagnosis, and management. It highlights recent studies on available and emerging therapies, including combination treatments. While PRP and LLLT show potential.

Keywords - Androgenic alopecia, dihydrotestosterone, minoxidil, finasteride, low-level light therapy, Platelet Rich Plasma, hair follicle.

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1. Introduction

Androgenetic Alopecia (AGA) is nonscarring alopecia, also known as male or female pattern hair loss, and is the most common type of hair loss, affecting more than 40% of adult men and 30% of adult women, respectively[1,2]. It is characterized by a progressive miniaturization of hair follicles with a characteristic pattern of distribution in genetically predisposed men and women. The most typical sign of androgenetic alopecia is excessive hair loss. Hair shaft heterogeneity is caused by the progressive shrinkage of hair follicles. Terminal hair reverts to its less-developed precursors as a result of common hair cycle modifications, such as anagen shortening and telogen extension. Hair can be clinically shortened, which may ultimately result in the total loss of hair shafts inside the hair follicles[3]. As its name suggests, AGA primarily results from the effects of androgenic hormones on the hair follicle (HF). Androgens such as dihydrotestosterone (DHT) and testosterone are potent inducers of HF miniaturization and progression out of the anagen phase, which clinically manifests as gradual hair thinning and shedding. AGA promotes increased activation of androgen receptors, which shrink hair follicles until they can no longer penetrate the epidermis. AGA is the loss of terminal hairs and replacement with short vellus hairs in the temporal, vertex, and mid-frontal sections of the scalp[4].

Progressive hair thinning and shedding on the scalp vertex and frontal hairline are hallmarks of male pattern hair loss (MPHL). Hair thinning and shedding along the middle hairline of the scalp vertex are the hallmarks of female pattern hair loss (FPHL). Because FPHL has higher levels of aromatase, an enzyme that converts testosterone to estrogen. In women, AGA typically affects the crown and bitemporal scalp areas, not the frontal hairline[5].

Minoxidil and finasteride remain the only medical treatments approved by the Food and Drug administration (FDA), and the Lasermax Hair comb is the only device approved by the FDA for treatment by the AGA[6].

1.1 Clinical presentation of androgenetic alopecia in men and women

Male Pattern Hair Loss (MPHL): Progression: Begins with thinning at the temples, forming an M-shaped frontal hairline that recedes over time, often affecting the vertex. Assessment: The Norwood–Hamilton scale classifies MPHL into seven stages, with stage IIIv indicating initial vertex thinning[7].

Female Pattern Hair Loss (FPHL): Types and Classifications: Ludwig Scale: Evaluates FPHL in three stages (I - minimal, II - moderate, III - severe), typically preserving the frontal hairline. Olsen Pattern: Mid-frontal scalp hair loss, often resembling a "Christmas-tree pattern. “Ebling Classification: Five stages, with the latter stages showing more diffuse and frontotemporal recession similar to male pattern baldness. Savin Scale: An eight-level system using computer analysis of hair density, offering frontal and lateral views[3].

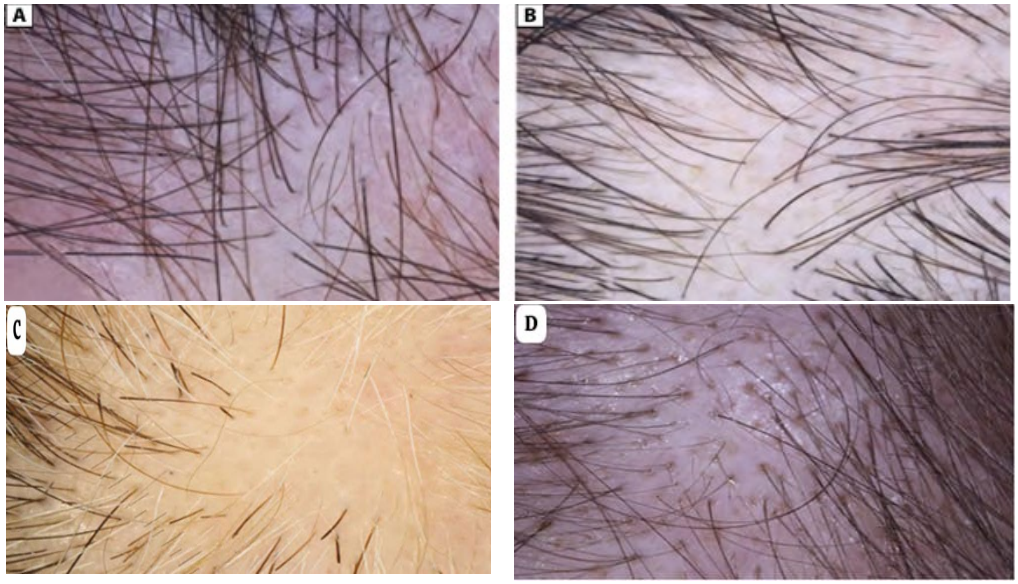

Fig-1. Trichoscopy signs of androgenetic alopecia

(A, B represent hair shaft thickness heterogeneity, C-perifollicular hyperpigmentation, D-yellow dots respectively)[3].

2. Components of the integumentary system

2.1 Skin: The skin consists of two layers: the outer epidermis and the deeper dermis.

The epidermis is a tough, protective layer made of stratified squamous cells, organized into four main layers (stratum corneum, granulosum, spinosum, and basale). In thicker skin, like on palms and soles, there is an additional stratum lucidum layer. Lacking blood supply, the epidermis receives nutrients from the dermis below and regenerates from basal stem cells[8].

The dermis supports the epidermis and is divided into the superficial papillary layer and the deeper reticular layer. The papillary layer contains vascularized loose tissue with dermal papillae, while the reticular layer has dense connective tissue. The dermis also houses blood vessels, nerves, sweat glands, and hair follicles[9].

2.2 Hypodermis: The hypodermis, or subcutaneous tissue, lies between the dermis and underlying organs. Composed of adipose and loose areolar tissue, it connects the skin to deeper structures, provides insulation, and offers cushioning by storing fat[10].

2.3 Associated Glands: Human skin contains three types of exocrine glands: sudoriferous, sebaceous, and mammary glands[11].

- **Sudoriferous (sweat) glands** include eccrine glands, which are spread across the body and secrete fluid to regulate temperature, and apocrine glands, found in the axilla and pubic area, which produce odor-causing, protein-rich sweat[8,11].
- **Sebaceous glands** are part of the pilosebaceous unit, secreting sebum, an oily, lipid-rich substance that forms a protective film, prevents fluid loss, and has antimicrobial properties[11].

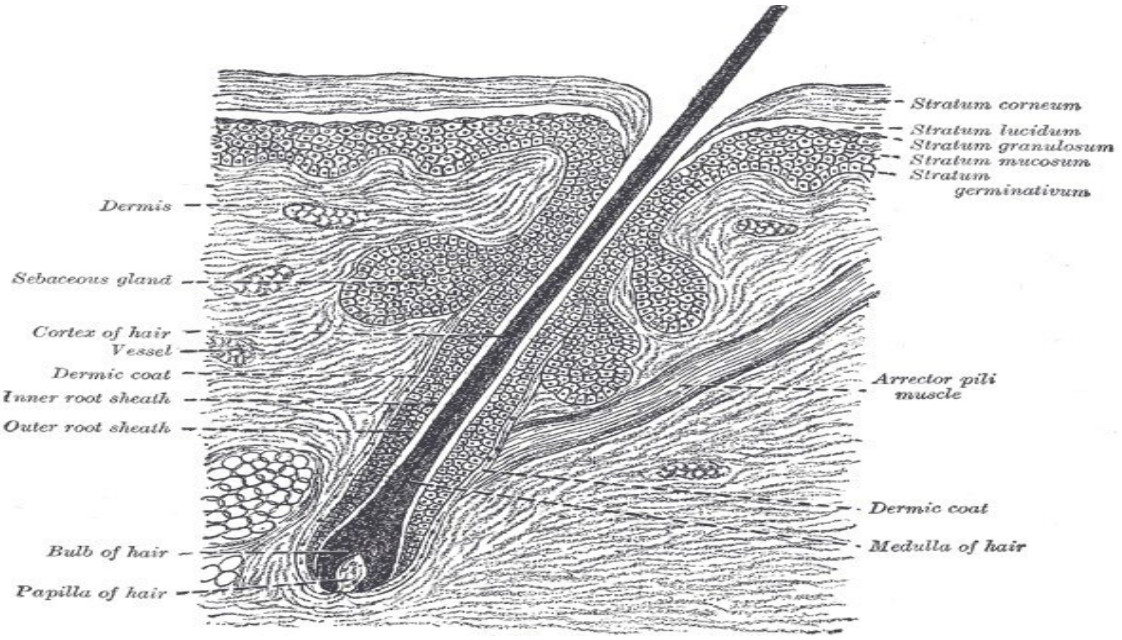

Fig-2. The common Integument, Section of the Skin[10].

2.4 Hair:

a) Anatomy of hair - Hair grows from the epidermis but extends its roots deep into the dermis, where it forms the visible hair shaft and the follicle within the skin. The follicle is a cylindrical structure where hair grows, hardening through keratinization as it moves up. Hair is composed of keratin, and only the base of the follicle contains "living" cells, making hair pulling painful[8].

The follicle is nourished by blood vessels and contains sebaceous glands that produce oils for hair softness and elasticity. Arrector pili muscles attach to hair follicles, raising hair in response to cold (goosebumps) to retain warmth. Hair types include thicker, hormone-dependent terminal hairs (in areas like the scalp, axilla, and pubic regions) and fine, androgen-independent vellus hairs elsewhere[8,12].

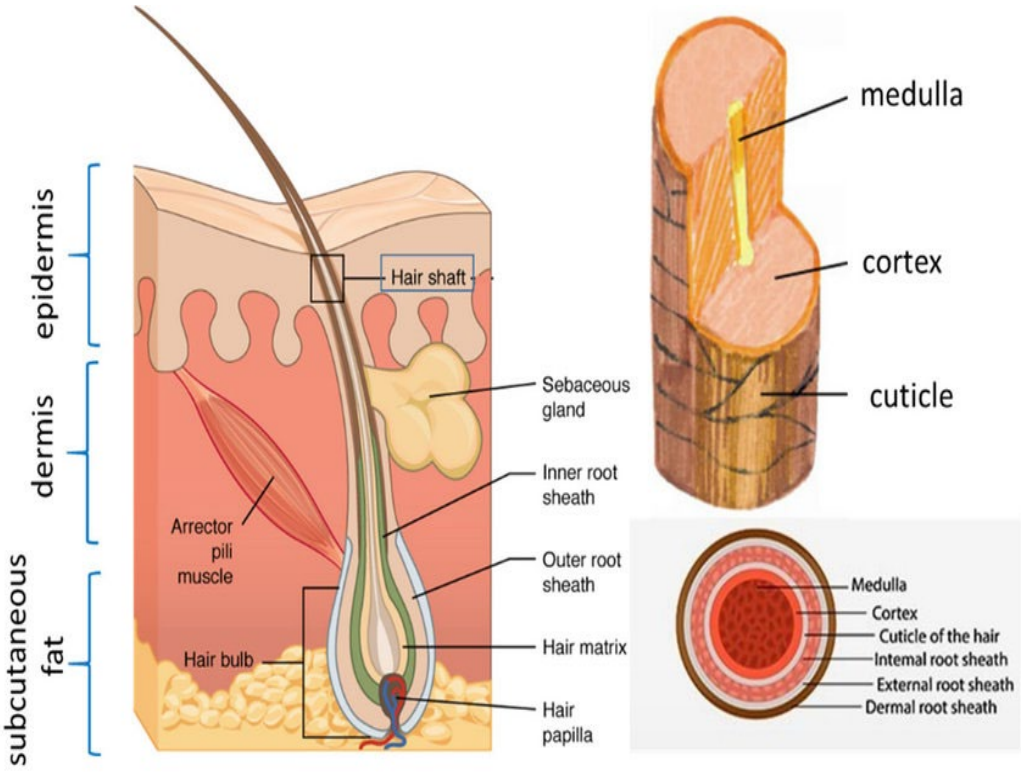

Fig-3. Anatomy of hair[5].

b) Hair growth cycle - Hair growth has three phases:

Anagen (Growth Phase): About 80–90% of scalp hairs are in this active growth phase. Follicles produce new cells that push the hair up the shaft, aided by a "ratcheting" mechanism. Melanocytes in the follicle add melanin, giving hair its color with either dark eumelanin or lighter pheomelanin[13].

Catagen (Transition Phase): In this brief, non-proliferative phase, the follicle stops creating new cells, and the root condenses into a club-shaped bulb[14,15].

Telogen (Resting Phase): This phase involves no cell production, with the hair held in place by a loose connection. Shedding occurs when this connection breaks, restarting the growth cycle. Humans lose about 100 scalp hairs daily as part of this cycle. Hair renewal rates vary, with scalp hair taking around 147 days and eyebrow hair 61 days to renew[15].

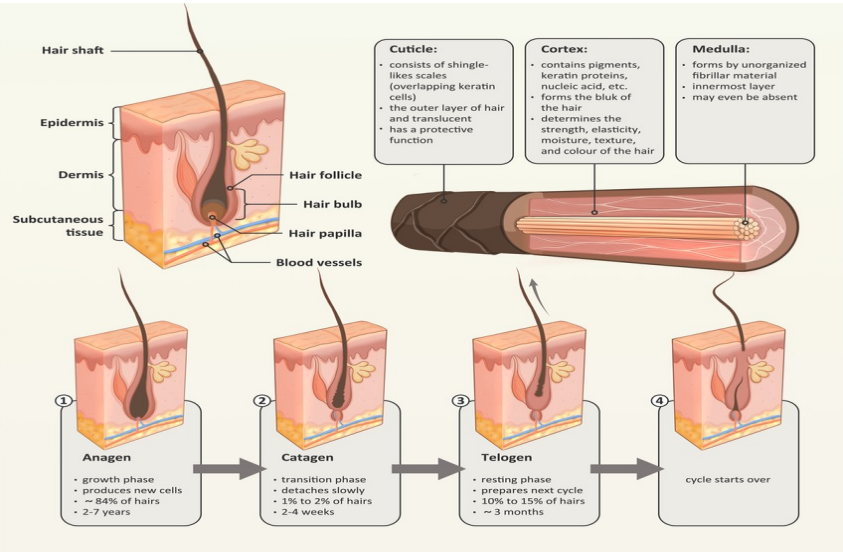

Fig-4. Hair growth cycle [14].

c) Microanatomy

A single hair has three parts: the root (proximal portion within the follicle), the shaft (main part of the hair), and the tip (distal end). Structurally, hair consists of:

Cuticle: Outermost layer with overlapping scales that protect the hair[15].

Cortex: The main body of the hair, containing spindle-shaped cells, pigment granules, and sometimes cortical fusi (small air bubbles found near the root in telogen hairs). Ovoid bodies, irregular, large pigment granules may also appear here[15].

Medulla: The central core, where small disruptions can occasionally appear, resembling tiny "explosions" along the shaft[15].

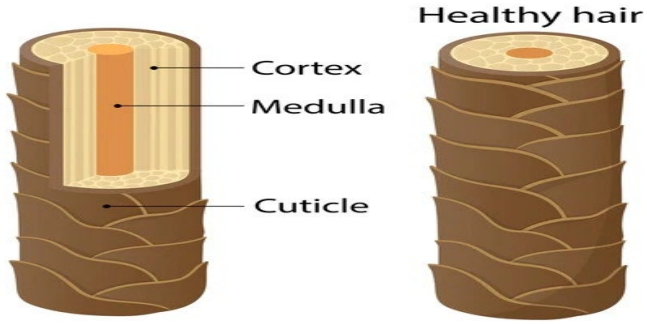

Fig 1.5 Microanatomy of hair[14].

3. Pathophysiology of androgenic alopecia

The pathogenesis of androgenetic alopecia (AGA) is influenced by heredity, androgen sensitivity in scalp hair follicles, and inflammation[16].Genetics play a key role, particularly in male pattern hair loss (MPHL), with over 250 genetic loci linked to susceptibility. MPHL is androgen-dependent, while the role of androgens in female pattern hair loss (FPHL) is less clear but may be stronger in cases of early-onset FPHL. New research highlights additional genetic and signaling pathways, such as the HIF-1 and Wnt pathways, as potential therapeutic targets. Variants in the androgen receptor (AR) gene are also implicated in AGA[17].

Androgens, specifically testosterone and dihydrotestosterone (DHT), contribute to changes in hair growth during puberty and adulthood, and they have different effects on hair follicles depending on location. DHT, converted from testosterone by 5α-reductase type II in the scalp, shortens the hair growth cycle and miniaturizes follicles, resulting in AGA's characteristic thinning in specific scalp areas[3,18]. In women, estrogens can prolong the anagen phase, making AGA more common after menopause when estrogen levels decline[19]. Women also have higher aromatase activity in the scalp, which converts androgens into estrogens, potentially offering further protection. The androgen-sensitive areas of the scalp contain high AR concentrations, with men exhibiting higher AR expression in the frontal area than women, contributing to site-specific hair loss patterns[20].

4. Diagnosis of androgenetic alopecia

The diagnosis of androgenic alopecia (AGA) primarily involves a detailed clinical history and physical examination, often supported by diagnostic tools such as trichoscopy and the hair pull test. Baseline global photographs are valuable for monitoring treatment progress[7]. In certain cases, particularly in women, further investigations like scalp biopsies or laboratory tests may be required to rule out underlying causes. A comprehensive history should include demographic factors such as age, sex, onset and progression of hair loss, along with family history, lifestyle habits (e.g., smoking, sun exposure), diet, hormonal disorders, and medication use, as these factors may influence AGA. The physical assessment should examine the scalp, facial and body hair, and nails, noting any signs of erythema, scaling, inflammation, or scarring alopecia. While AGA typically presents with a smooth, glossy scalp free of atrophy, it can sometimes coexist with conditions such as seborrheic dermatitis [21].

AGA presents with miniaturized scalp hair distributed in a gender-specific pattern: men generally experience receding hairlines and vertex thinning, while women often have diffuse thinning over the crown with preserved frontal hairlines. Diagnostic tools for AGA include the hair pull test, used to gauge active hair loss, which is often negative in AGA except during intense telogen effluvium episodes[22].

I. Trichoscopy, a non-invasive method, reveals AGA's characteristic hair shaft thickness variation, with additional findings like perifollicular discoloration, yellow dots, vellus hairs, and a lower terminal-to-vellus hair ratio. Advanced trichoscopy, such as videodermatoscopy, provides enhanced magnification and photographic documentation, allowing for detailed tracking of treatment progress[23] .

II. Scalp biopsies, particularly helpful for scarring alopecia, may clarify AGA in ambiguous cases. Biopsies involve two 4-6 mm cylindrical punches, one for vertical and one for horizontal sections, to evaluate follicle depth, follicular units, and miniaturization. In AGA-affected areas, biopsies reveal increased miniaturized follicles with a telogen-to-vellus hair[24].

5. Treatment of androgenetic alopecia

5.1 Topical therapies

- **Topical minoxidil therapy**: Minoxidil, initially developed as an oral hypertension medication, was repurposed after its hair growth side effects were discovered. It works as a prodrug, converting into minoxidil sulfate within hair follicles, where it promotes vasodilation, reduces inflammation, enhances the Wnt/β-catenin pathway, and increases vascular endothelial growth factor (VEGF) levels to support follicle health. Available in 2% and 5% solutions, as well as a 5% foam for men and both concentrations for women, minoxidil is effective for treating hair loss. However, side effects can include dermatitis, initial shedding, hypertrichosis, and rare headaches. Compliance may be challenging due to these side effects and the need for daily use[3,6,25] .
- **Topical finasteride -** Topical finasteride targets DHT production directly in hair follicles, minimizing systemic exposure and reducing teratogenic risks compared to oral finasteride. Side effects are generally mild, with skin irritation and pruritus at the application site being the most common. Notably, no sexual side effects have been reported[26] .
- **Topical spironolactone-** It inhibits androgens at hair follicles with minimal systemic effects. Combination with 5% minoxidil enhanced outcomes[27] .

5.2 Oral therapies

- **Oral minoxidil -** Originally developed as an antihypertensive, low-dose oral minoxidil (0.25–5 mg/day) is now used for treating androgenetic alopecia (AGA). Its liver metabolism increases bioavailability, offering benefits to those who don't respond to topical minoxidil. Side effects include hypertrichosis (55.4%), hair shedding (32%), and pedal edema (6%), with 8% of users discontinuing due to side effects or lack of efficacy[6,25].
- **Oral finasteride -** Finasteride, an FDA-approved 5α-reductase inhibitor (1997), works by blocking the conversion of testosterone to DHT, reducing scalp DHT levels to prevent hair

follicle miniaturization. Side effects can include sexual dysfunction (1–40%), muscle atrophy, and depression. There is also a potential increased risk of high-grade prostate cancer, so patients should be informed and monitored regularly[28].

- **Oral dutasteride_-** Dutasteride inhibits both type I and II 5α-reductase enzymes, reducing DHT, and was originally used for BPH treatment. Side effects include decreased libido, impotence, ejaculatory issues, and gynecomastia, with about 10% experiencing erectile dysfunction. Its high side effect rate (53%) makes long-term use challenging[29] .
- **Oral spironolactone for FPHL -** Spironolactone is a potassium-sparing diuretic with anti-androgen effects, reducing androgens that affect hair follicles. Side effects include hypotension, hyperkalemia, and occasional urticaria, with a starting dose of 50 mg daily[30].

5.3 Hormonal therapies

- **Oral spironolactone for FPHL-** Originally used for cardiovascular issues, spironolactone is effective in treating FPHL due to its antiandrogen effects, blocking testosterone and androgen receptors. Common side effects include electrolyte imbalances, renal issues, and hypotension, though it is generally well tolerated[6,25] **.**
- **Flutamide and bicalutamide for FPHL_-** Flutamide (250 mg/day) is effective for resistant FPHL but carries a risk of liver toxicity (4% discontinuation) and common side effects like hot flashes and potential warfarin interactions. Bicalutamide is a safer alternative, especially for PCOS-related hirsutism, with 95% adherence and mild side effects like elevated liver enzymes and edema. Seven of 316 bicalutamide users discontinued due to hot flashes and warfarin interaction[6] **.**
- **Cyproterone acetate-** Cyproterone acetate inhibits gonadotropin secretion, 5-alpha-reductase, and androgen receptors, making it effective for AGA and acne in women, particularly those with hyperandrogenism or elevated BMI. Side effects include weight gain, breast tenderness, and decreased libido. It has high patient compliance due to oral administration, though it is not available in the U. S[31].

5.4 Light therapies

- **Therapy Low-Level Laser Therapy (LLLT) for AGA-** Low-level laser therapy (600–1100 nm) stimulates hair regrowth by promoting the anagen phase and preventing early transition to catagen. The Lasermax Hair comb, approved by the FDA in 2018 for AGA, has minimal side effects, such as temporary shedding, pruritus, tenderness, and acne. It is a low-cost, effective adjunct to other treatments[1,25,32].
- **Light--emitting diode devices-** red and orange light therapy (620–660 nm) promote blood flow, reduce inflammation, and inhibit DHT to aid hair regrowth. It is safe for most patients,

with minimal side effects, though caution is advised for those with dysplastic scalp lesions. The non-invasive, at-home treatment requires just 10 minutes daily, with mobile app tracking to improve compliance[33].

5.5 Injectables

- **Platelet-rich plasma treatment**– Platelet-rich plasma (PRP), derived from a patient's blood, contains growth factors (PDGF, VEGF, TGFβ, IGF) that promote hair follicle growth and reduce DHT's negative effects. It has a low risk of adverse effects, such as mild scalp sensitivity and scaling, but is not suitable for individuals with certain health conditions[3,6,25].
- **Exosomes–** SC-Exosomes, containing growth factors like VEGF and IGF-1, stimulate dermal papilla cell proliferation, promoting hair growth by converting hair follicles from telogen to anagen and increasing hair density and thickness. Therapies like ExoFlo and ExoCel are under exploration, though further research is needed. Side effects are minimal, with minor injection site pain resolving within 24–48 hours. The in-office procedure requires monthly sessions for optimal results[34]**.**

5.6 Adjuvant therapy

- **Microneedling-** Microneedling uses micron-sized needles to create microinjuries, promoting hair growth via Wnt/β-catenin signaling and VEGF upregulation, while potentially enhancing drug delivery. Combined with minoxidil, it has been shown to significantly increase hair count and density, especially in refractory AGA cases. Side effects include pain, bruising, folliculitis, erythema, and pruritus, with minimal serious complications. However, patient compliance may be hindered by cost and discomfort[35].

5.6 Supplements & OTC treatments

Used as monotherapy or adjuvant therapy for conditions like photoprotection, vitiligo, and melasma. Limited clinical evidence, but good tolerability in trials. Can be used alone or with other treatments.

- **Oral nutraceutical supplement containing synergen complex-** This supplement, containing saw palmetto, ashwagandha, curcumin, collagen, and biotin, has been shown in studies to improve hair growth, reduce shedding, and enhance hair quality. Trials report a significant increase in terminal and vellus hairs with no adverse events, making it particularly effective for women aged 40-65 with hair thinning. The recommended dosage is 4 capsules daily[36].
- **Marine complex supplement-** This supplement, containing shark/mollusk extracellular matrix, vitamin C, horsetail, and flaxseed extract, has been shown to increase hair growth, density, and reduce shedding in AGA patients. Clinical trials report a 38% increase in non-vellus hair in males, with similar results in females. Side effects are mild, including potential

arthralgia, bloating, and nausea, but no adverse events were noted. The recommended dosage is 2 oral tablets daily, with results typically seen after several months[37].

- **Serenoa repens -** SR inhibits 5-alpha-reductase and has been shown to improve hair growth in AGA, with a 60% improvement in one study and a 35% increase in hair density with topical use. In a comparative study, SR resulted in 38% hair growth, compared to 68% with finasteride after 24 months. Side effects are minimal, primarily gastric discomfort, and it can lower PSA levels, potentially affecting prostate cancer detection. The oral regimen is taken once daily[38].
- **Plant--based oils: rosemary oil, tea tree oil, pumpkin seed oil, coconut oil, castor oil, amla oil -** Rosemary oil improves hair count similar to minoxidil, pumpkin seed oil increases hair count by 40%, and tea tree oil aids in inflammation and scalp health. Side effects are minimal, typically scalp irritation. These oils are easy to apply topically at home but may interfere with hair styling due to their greasy texture. They are not FDA-regulated[18].
- **Ketoconazole**- Shows improvements in hair shaft diameter and anagen phase; effective as an adjuvant or alternative treatment for AGA. No significant side effects. Can be used once daily, twice daily, or 2–3 times per week. Further studies needed to confirm its full effectiveness[32].

5.8 Combination therapy

- **Combination Topical Minoxidil and Topical Finasteride-** Combination improves hair density and diameter more than minoxidil alone. No significant adverse effects; preferred over oral finasteride due to fewer side effects. Plasma DHT levels slightly decreased (5%) with combination treatment[39].
- **Combination Topical Minoxidil with LLLT-** Some studies show improved follicle count and patient satisfaction with combination therapy, but results are mixed.
  Side Effects: No increase in adverse effects[40].
- **Combination Treatment with PRP, Minoxidil, LLLT, and Microneedling-** PRP combined with minoxidil, LLLT, or microneedling shows superior results compared to monotherapy, improving hair density and growth. No increased risk of adverse effects[41].
- **Combination of finasteride and dutasteride in the treatment of FPHL-** Off-label for female pattern hair loss (FPHL), typically after other treatments fail. Finasteride (1-5 mg daily, commonly 2.5 mg); Dutasteride (0.5 mg daily). Teratogenic, avoid during pregnancy; contraception required for 1 month after finasteride and 6 months after dutasteride. Used when hyperandrogenism isn't due to endogenous factors[25].

5.9 Hair transplantation - Used for advanced AGA when medical treatments fail; high graft survival rate (>90%); natural results. Methods like Ellipse: Strip of skin removal, minimal bleeding, linear scar. No linear scar, small pinpoint scars; robotic FUE offers precision and reduced

error, but is costly. Side effects involve Anesthesia reactions, bleeding, pain, edema, potential scarring (FUE)[6].

5.10 Anti androgen therapy - Treats hyperandrogenism-related conditions (acne, hirsutism, FPHL). Spironolactone: Most used for FPHL; dose: 50-200 mg/day**.** hyperkalemia, hypotension, frequent urination, menstrual issues. Avoid in pregnancy (teratogenic) are observed as side effects. Cyproterone Acetate (CPA): Declining use due to CNS meningioma risk. Flutamide: Rarely used orally; effective as a 2% topical solution with minoxidil. Oral Contraceptives: Recommended for PCOS patients with AGA; provides anti-androgenic effects. Contraception: Essential for women on teratogenic drugs (spironolactone, finasteride, flutamide)[3].

5.11 Stem cell therapy - Stem cell therapy activates Wnt signaling to promote hair follicle growth and differentiation. Adipocyte-derived stem cells can achieve up to 20% hair growth in 8 weeks, while Autonomous Cellular Micrograft's (ACM), using scalp-derived cells, show increased hair density and thickness over 6 months in clinical studies. However, this treatment is experimental, costly, invasive, and its long-term safety is unknown, with potential risks like graft-versus-host disease (GVHD)[25].

5.12 Botulinum toxin- Reduces scalp muscle tone, possibly enhancing blood flow and DHT removal from hair follicles; may inhibit neuromodulators related to hair loss. Generally mild—headache, pain, redness; rare effects include madarosis, facial alopecia, and hairline regression at high doses[42] .

5.13 Supplements and dietary options- May inhibit 5-alpha reductase, boost IGF-1, reduce oxidative stress, and improve follicle health. Examples: Saw palmetto, pumpkin seed oil, Forti5; antioxidants like vitamins E and C for women; protein-based supplements; probiotics (e.g., kimchi). May stabilize hair loss and promote growth; small-scale studies show increased density and thickness. Side Effects: Minimal, but supplements are not FDA-approved; limited research and small sample sizes[22].

Table 1- Drugs used in the treatment of androgenic alopecia

Drug	Formulations	Efficacy	Side-effects	Mechanism	Reference
Minoxidil	Topical-spray and oil, Solid effervescent, Nanostructured lipi carrier, foam	5% solutio effective	Dermatitis, initial shedding, hypertrichosis and rare headaches	Prodrug converts t minoxidil sulfate i follicles, promotin vasodilation, reducing inflammation.	6,32,37,38
	Oral-tablet	0.25–5mg	Hypertrichosis, hai shedding	-	
Finasteride	Topical	0.25%and 1%	Mild skin irritation and pruritus	Reduces DHT in follicles, minimizing systemic exposure and teratogenic risks	12,24,38,40
	Oral tablet (film coated), capsule	1 mg/day	Sexual dysfunction muscle-atrophy, depression, prostat cancer	5α-reductase inhibitor	
Dutasteride	Oral tablet	0.5mg/day	Decreased libido, impotence, ejaculatory issues, and gynecomastia, Erectile dysfunctio	Blocks both type I and II 5α-reductas enzymes, reducing DHT	35
Flutamide	Hormonal therapy	250 mg/day	Liver toxicity, hot flashes, potential Interaction with warfarin	-	11

Bicalutamide	Hormonal therapy	-	-	Hot flashes, elevated liver enzymes, edema	[23]
Cyproterone acetate	Hormonal therapy	-	Weight gain, breast tenderness, decrease libido	Inhibits gonadotropin secretion,5-alpha-reductase and androgen receptors	[17]
Spironolactone	Topical	1% daily	-	Inhibits androgens at follicles With minimal systemic effects.	[43]
	Oral tablet	100–200mg daily	Hypotension, hyperkalemia, occasional urticaria	Potassium-sparing diuretic with anti-androgen effect	
	Hormonal therapy	100–200mg daily	Electrolyte imbalance, renal function issues, hypotension	Blocking testosterone and androgen receptors	

Table 2- Drugs associated with the exacerbation or triggering of androgenetic alopecia.

Category	Drug class	Clinical indication(s)	Mechanism of action	Route of administration
Drugs that promote androgenic activity	Testosterone	Hypogonadism and associated symptoms, delayed puberty, testicular failure, gender transition therapy	Supplementation of endogenous androgen	Intramuscular injection, topical (gel, patch), subdermal implant (pellet)

	Anabolic androgenic steroids (danazol, trenbolone, oxymetholone, etc.)	Hypogonadism and associated symptoms, pathologic muscle atrophy	Potent androgen agonists	Intramuscular injection, oral, topical (cream, gel, or patch), subdermal implant (pellet)
	Progestins (levonorgestrel, norgestrel)	Contraception, endometriosis	Synthetic progesterone	Oral (OCP), intrauterine device (IUD), subdermal implant
	Human chorionic gonadotropin (HCG)	Hypogonadism, and associated symptoms, male or female infertility,	Supplementation of endogenous gonadotropin	Subdermal or intramuscular injection
Drugs that inhibit estrogenic activity	Aromatase inhibitors (anastrozole, exemestane, letrozole)	Estrogen-receptor-positive breast cancer	Inhibition of aromatase enzyme	Oral
	Selective estrogen receptor modulators (tamoxifen, raloxifene, toremifene)	Estrogen-receptor-positive breast cancer, osteoporosis, menopausal symptoms, infertility	Agonist or antagonize estrogen receptors	Oral

6. New upcoming therapies or ongoing research

- **Clascoterone -** Clascoterone, a topical androgen receptor antagonist, was FDA-approved in August 2020 for treating hormonal acne. It works by inhibiting androgen receptors in the skin, mimicking the effects of finasteride. Its mechanism involves blocking DHT's effect on hair miniaturization and inflammation in the dermal papillae. Clascoterone has shown potential for treating androgenetic alopecia (AGA). A 6-month study revealed significant improvement in hair loss in patients using 7.5% Clascoterone twice daily compared to placebo[44].
- **Prostaglandin Analog** - Latanoprost, a prostaglandin analog originally used for glaucoma, was found to stimulate hair growth by prolonging the anagen phase. A study involving 16 men with mild AGA showed increased hair density with daily application of latanoprost 0.1%. However, the study was limited to mild cases of AGA[6].
- **Other Emerging Treatments - Topical Cetirizine:** Preliminary results suggest that cetirizine may be effective in treating AGA. **Scalp Threading with PDO Threads:** This technique may increase hair density by stimulating hair follicles. **Cacumen Platycladi Extract:** Derived from Platycladus orientalis, this water extract has shown effects similar to finasteride by activating the Akt/GSK3β/β-catenin signaling pathway, promoting hair growth. Other naturally derived substances are also being explored for their potential benefits in hair restoration[44]

7. Conclusion

Managing androgenetic alopecia (AGA) is complex but promising treatment options are available, with combination therapies showing potential to become standard care. Topical minoxidil is effective but challenging for long-term compliance, which may improve when combined with Low-Level Laser Therapy (LLLT). Oral minoxidil offers a convenient alternative, while oral finasteride is effective but can have adherence-limiting side effects. Other options include topical finasteride, PRP, and microneedling, which enhance therapies like minoxidil and LLLT.

As a chronic condition, AGA requires tailored, often lifelong treatment, with options selected based on patient-specific factors. While FDA-approved treatments are limited, other hormonal and surgical treatments are also available. Dermatologists play a crucial role in navigating treatment choices. Additionally, a comprehensive understanding of hormonal and drug influences is important, as certain medications can worsen AGA in predisposed patients. High-quality research and standardized methodologies are essential to better compare treatments and optimize patient care.

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