doi.org/10.58931/cdet.2026.4153

About the Author

Anastasiya Muntyanu, MD, PhD, FRCPC, FAAD

Dr. Anastasiya Muntyanu is a Canadian and US board certified dermatologist, currently practicing in Toronto. She completed her medical school at the University of Ottawa and graduated from the University of Toronto Dermatology Residency Program. She completed her PhD focused on studying environmental triggers of autoimmune and inflammatory skin diseases including psoriasis, systemic sclerosis, and atopic dermatitis. She has over 40 publications in high impact journals and has received numerous awards including from the Canadian Institutes of Health Research award, Canadian Dermatology Association, and the American Dermato-Epidemiology Network. During her residency she was the co-chair of the Canadian Dermatology Association's Resident and Fellow Society and was a resident representative on numerous academic committees for which she received the Resident Leadership Award and the Resident Teaching Award from the Canadian Dermatology Association. Dr. Muntyanu's clinical areas of interest include medical and surgical dermatology with a focus on psoriasis, eczema, systemic sclerosis and morphea, and skin cancer.

Affiliations: Women's College Hospital, Toronto, ON
Princess Margaret Cancer Center, Toronto, ON
St. Joseph's Hospital, Toronto, ON

Androgenetic Alopecia: Pathogenesis, Evaluation, and Management

Anastasiya Muntyanu, MD, PhD, FRCPC, FAAD

Introduction

Androgenetic alopecia (AGA) is a common, non-scarring, alopecia characterized by the progressive transformation of terminal hairs into miniaturized vellus hairs in a patterned distribution. Female AGA—often termed female-pattern hair loss (FPHL)—and male AGA (often termed male-pattern hair loss, MPHL) share similar underlying pathophysiologic, histopathologic, and trichoscopic features; however, they differ in their clinical presentation and patterns of scalp distribution.[1]

AGA represents the most prevalent cause of hair loss in both men and women and can have a significant impact on quality of life. Evidence from a large meta-analysis demonstrates a moderate impairment in health-related quality of life and emotional well-being, underscoring the meaningful psychosocial burden of AGA.[2]

Although patients are often referred for dermatologic evaluation, AGA may also have important endocrine implications. In selected cases—particularly in women or in atypical presentations—it may reflect underlying endocrinologic abnormalities, warranting targeted screening and management.

Epidemiology

The prevalence and severity of AGA increases with age, affecting up to 80% of men and 50% of women over their lifetime.[3] In women, prevalence rises from approximately 12% by age 29 to 25% by age 50, reaching 41%–50% by age 70 and older.[4] In men, a US-based study reported a moderate MPHL in 48% of those aged 18–49 years, including 16% of those aged 18–29 and 53% of those aged 40–49.[5]

Although onset can occur in adolescence, pediatric AGA remains uncommon.

Pathophysiology

AGA is a polygenic, androgen-dependent condition characterized by progressive miniaturization of hair follicles, resulting in the conversion of terminal hairs to fine vellus-like hairs. Established risk factors include genetic predisposition and, in some cases, underlying endocrinologic conditions such as polycystic ovary syndrome (PCOS), congenital adrenal hyperplasia (CAH), and androgen-secreting tumours.

Genetic Factors

AGA has a strong hereditary basis, with polygenic inheritance supported by findings from multiple genome-wide association studies. Variants influencing androgen receptor signalling appear to play a central role, particularly in men. In addition, genetic loci involved in hair follicle development and cycling pathways have been consistently implicated across populations.

Hormonal and Local Mechanisms

Dihydrotestosterone (DHT), produced from testosterone via 5α-reductase, is the key androgen driving AGA. Local DHT levels and 5α-reductase activity are increased in balding scalp compared with non-balding areas, contributing to progressive follicular miniaturization. Rather than acting in isolation, DHT also promotes structural changes around the follicle, including perifollicular fibrosis and reduced vascular support, which may impair nutrient delivery and limit hair growth.[6]

Importantly, most patients—particularly women—have normal circulating androgen levels, indicating that AGA reflects increased local follicular sensitivity to androgens rather than systemic androgen excess.

Clinical Presentation

Both MPHL and FPHL are characterized by progressive, non-synchronized follicular miniaturization, resulting in the replacement of terminal hairs with short, thin, vellus hairs and, with longstanding disease, reduced follicular density.

MPHL is characterized by frontotemporal recession and vertex thinning. In contrast, FPHL hair loss predominantly affects the mid-scalp and vertex, with relative preservation of the frontal hairline. Thinning may extend anteriorly along the central part, producing a characteristic “Christmas tree” pattern, with widening of the central part correlating with disease severity. Less commonly, women may exhibit a more extensive pattern with frontotemporal recession and vertex involvement resembling MPHL.[7]

Overlap between these patterns can occur, and a history of increased hair shedding (i.e., telogen effluvium) may precede the diagnosis of FPHL.

Severity Grading

MPHL

The Hamilton and Norwood scale is the most commonly used classification system for grading MPHL (**Figure 1**).

FPHL

In FPHL, the Ludwig classification (three grades) and the Sinclair scale (five stages) are commonly used to assess disease severity (**Figure 2A and Figure 2B**); however, no single grading system has been universally adopted.

Differential Diagnosis

The differential diagnosis includes telogen effluvium (TE), a common cause of diffuse hair shedding that may be triggered by physiological or psychological stress, rapid weight loss, systemic illness, or iron deficiency. TE may coexist with or precede the diagnosis of FPHL. Other considerations include alopecia areata (AA), which can occasionally present with diffuse thinning and mimic early AGA. It is also important to exclude inflammatory causes of hair loss, particularly scarring alopecias, which may present with scalp symptoms (e.g., burning or pruritus) and perifollicular erythema or scaling on dermoscopy.

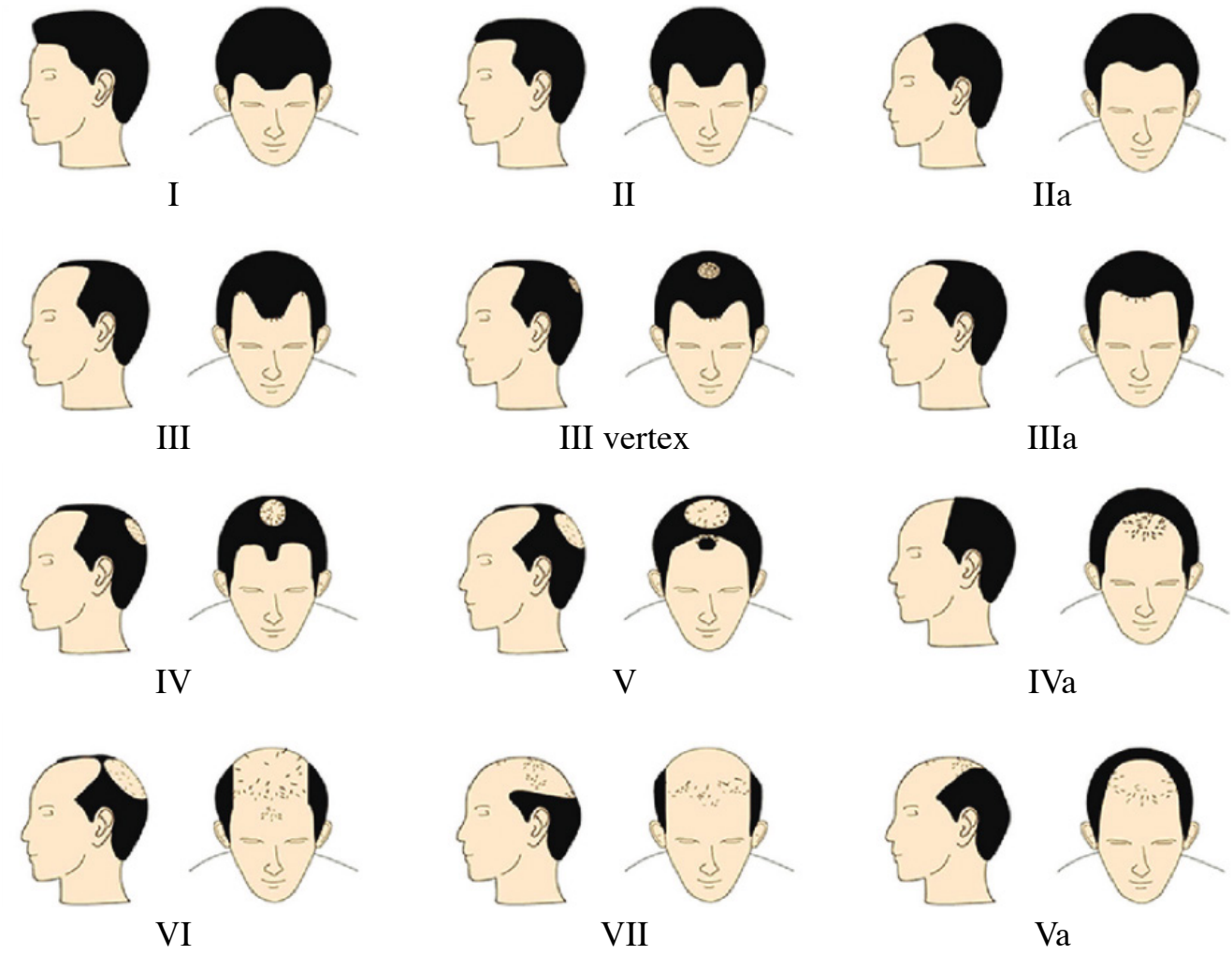

Figure 1. Male pattern baldness: classification and incidence; *adapted from Norwood, O.T.*[8]

These conditions can lead to permanent hair loss and require alternative management strategies.

Endocrine disorders should be considered in the differential diagnosis. Thyroid dysfunction may present with diffuse hair loss, while hyperandrogenic states—including PCOS, CAH, and androgen-secreting tumours—may contribute to or mimic AGA. In women, early-onset or rapidly progressive patterned hair loss, particularly when accompanied by clinical features of androgen excess, should prompt evaluation for hyperandrogenism.

Diagnosis and Investigations

The diagnosis of AGA is primarily clinical, based on patterned thinning, gradual progression, and follicular miniaturization. Trichoscopy can support the diagnosis, demonstrating variation in hair shaft diameter, an increased proportion of single-hair follicular units, and the presence of yellow dots representing follicular openings filled with sebum and keratin. Scalp biopsy is rarely required but may be helpful when the diagnosis is unclear or when scarring alopecia, AA, or chronic TE are suspected.

From an endocrine perspective, the key consideration is whether hair loss reflects local follicular sensitivity to androgens or an underlying systemic disorder. Although AGA is androgen-mediated, most patients have normal circulating androgen levels, and AGA alone is not a reliable marker of hyperandrogenism. Consequently, routine endocrine testing is not required in the absence of clinical features of androgen excess. However, evaluation should be considered in cases of early-onset or rapidly progressive/severe hair loss, or when accompanied by hirsutism, acne, menstrual

Figure 2A. Classification of the types of androgenetic alopecia (common baldness) occurring in the female sex; *adapted from Ludwig E.*[9]

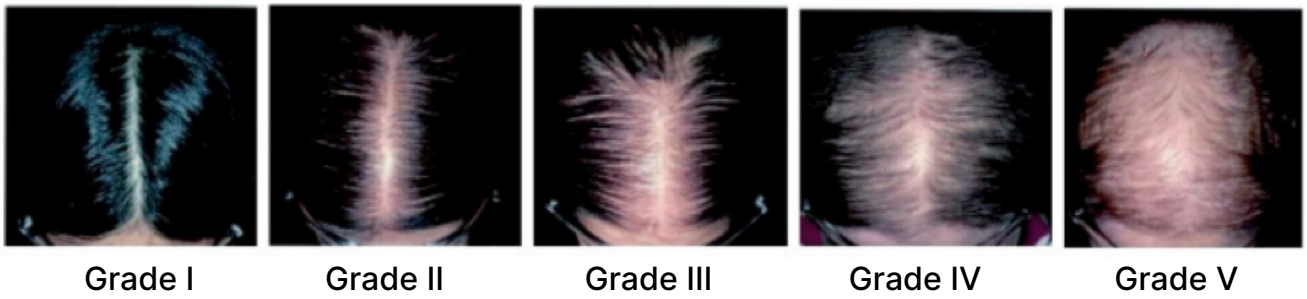

Figure 2B. The reliability of horizontally sectioned scalp biopsies in the diagnosis of chronic diffuse telogen hair loss in women; *adapted from Sinclair R. et al.*[10]

irregularity, infertility, galactorrhea, virilization, or features of thyroid disease. Initial investigations may include total and free testosterone, dehydroepiandrosterone sulfate (DHEA-S), 17-hydroxyprogesterone, and prolactin, with additional testing guided by clinical suspicion. In men with typical MPHL, hormonal testing is not required.

FPHL is relatively common among patients with PCOS, with reported prevalence estimates ranging from 20%–30%.[11] In a cohort study of 254 patients, FPHL was identified in 22% of women with PCOS and was more frequently associated with clinical features of hyperandrogenism, such as acne and hirsutism, compared with those without FPHL (96.3% vs. 70.6%).[12] However, no significant differences were observed between groups in terms of biochemical hyperandrogenism or metabolic parameters. A meta-analysis further demonstrated an increased risk of FPHL in patients with PCOS compared to controls (odds ratio [OR] 4.74, 95% confidence interval [CI] 0.57–39.52).[13] Conversely, the pooled prevalence of PCOS among patients with FPHL was estimated at 32.3% (range 13.6%–59.1%).[13] Overall, the available data are heterogeneous and of modest quality, suggesting that while FPHL frequently coexists with PCOS, it is not a reliable independent marker of endocrine or metabolic severity.

Beyond PCOS, the association between AGA and broader metabolic comorbidities has also been investigated. A meta-analysis demonstrated a significantly higher prevalence of metabolic syndrome among patients with AGA compared to controls (pooled OR 3.46, 95% CI 2.38–5.05; $p<0.001$).[14] The association with metabolic syndrome appears stronger in women, patients with early-onset disease, and individuals of African ethnicity. However, these associations are not consistent across studies. In clinical practice, targeted screening and longitudinal assessment may be considered in selected patients.

Treatments

Treatment options for AGA include topical and systemic therapies, many of which target androgen pathways by reducing androgen production, altering androgen metabolism, or blocking androgen receptor activity. Systemic antiandrogen therapies are contraindicated during pregnancy due to the risk of fetal abnormalities. In patients with an identified underlying endocrine disorder, management should be directed according to established endocrinologic guidelines.

Early diagnosis and initiation of treatment are desirable because treatments are more effective at preventing progression of hair loss than stimulating regrowth.

A Canadian consensus statement has outlined interventions with high levels of agreement (**Table 1**) and proposed a stepwise approach to the management of MPHL and FPHL (**Table 2**).[15]

Topical Minoxidil

Topical minoxidil is approved by Health Canada for the treatment of AGA in both males and females. It promotes hair growth primarily through vasodilation, prolongation of the anagen phase, increases shaft diameter, and activation of follicular signalling pathways (including Wnt/β-catenin). It is typically applied to affected scalp areas as a 5% foam once daily (women) or twice daily (men), or as a 2% solution twice daily. Clinical response is expected after 3–6 months and continued use is required to maintain therapeutic effects.

Topical Finasteride

Topical finasteride has emerged as an alternative to oral therapy, with studies showing increased hair density at 24 weeks compared to placebo, with efficacy comparable to oral finasteride.[16] It exerts its effect via local inhibition of 5α-reductase, thereby reducing scalp DHT levels. Topical formulations (e.g., 0.25% once daily) are generally well tolerated and are associated with fewer systemic adverse effects, particularly sexual side effects, compared to oral finasteride.

Oral Minoxidil

A 2024 randomized study published in *JAMA Dermatology* demonstrated that oral minoxidil (5 mg daily) was non-inferior to 5% topical minoxidil applied twice daily over 24 weeks, with a comparable safety profile.[17] Although oral therapy showed a trend toward greater improvement—particularly at the vertex—this did not reach statistical significance. In parallel, an international modified Delphi consensus reported 97.7% expert agreement supporting the use of low-dose oral minoxidil in AGA.[18]

Low-dose oral minoxidil is generally well tolerated. The most common adverse effect is hypertrichosis (24% overall), followed by transient shedding, which typically peaks at 4 weeks and resolves by 12 weeks.[18] Cardiovascular-related effects are dose-dependent and include peripheral edema (1.3–16.4%), tachycardia/palpitations (0.9–4%), and orthostatic symptoms (1.7–4.5%).

Oral Spironolactone

Spironolactone is the most commonly used off-label systemic antiandrogen for the treatment of FPHL. It functions as a competitive androgen receptor antagonist and reduces androgen production. Typical dosing ranges from 100–200 mg daily, with treatment durations of at least 6–12 months required to assess efficacy. Available evidence suggests benefit in both stabilizing hair loss and promoting regrowth, with response rates reported between approximately 44% to 74%, and higher responses observed in patients with concomitant hirsutism or acne.[11]

Adverse effects are related to its antiandrogenic and mineralocorticoid activity and may include menstrual irregularities, breast tenderness, fatigue, postural hypotension, and electrolyte disturbances. Spironolactone is contraindicated in pregnancy due to the risk of feminization of a male fetus and should be used with reliable contraception in women of childbearing potential.

Consensus Level	Agreement Supporting the Use of the Intervention
Consensus (>75%)	• Oral dutasteride (100%) • Oral finasteride (100%) • Oral minoxidil (100%) • Platelet-rich plasma (100%) • Topical minoxidil (100%) • Topical finasteride (91%) • Microneedling (82%)
Near consensus (55–74%)	• Ketoconazole shampoo (73%) • Topical products as part of a regimen including shampoos (73%) • Intralesional dutasteride (55%) • Laser (55%) • Aminexil (55%)

Table 1. Final Recommendations for the Management of Androgenic Alopecia; *adapted from Landells I. et al.* ***A Canadian Consensus on Androgenetic Alopecia: Approach and Management***.[15] Please see the full publication for therapies that are not recommended.

Line of Therapy	MPHL	FPHL
First line	• 5% minoxidil foam (BID) • Oral finasteride 1 mg daily	• 5% topical minoxidil
Second line	• Topical finasteride 0.25% daily • Oral minoxidil 1.25–5 mg daily • Oral dutasteride 0.5 mg (2–3 times/week • LLLT • PRP	• Oral spironolactone ± oral minoxidil • Oral finasteride/dutasteride or spironolactone ± oral minoxidil
Third line	• Saw palmetto • Aminexil • Ketoconazole topical • Other adjuncts (topical caffeine, botox, topical cetirizine, mesotherapy with dutasteride)	• Procedural therapies (PRP, laser, mesotherapy)
Other options	—	• Cosmetics (aminexil, nutraceuticals, herbal products)

Table 2. Treatment Guidelines for Androgenic Alopecia (Males and Females); *adapted from Landells I. et al.* ***A Canadian Consensus on Androgenetic Alopecia: Approach and Management***.[15]

Abbreviations: BID: twice daily; **FPHL:** female-pattern hair loss; **LLLT:** low-level laser therapy; **MPHL:** male pattern hair loss; **PRP:** platelet-rich plasma

Oral Finasteride/Dutasteride

Finasteride and dutasteride are 5α-reductase inhibitors that reduce conversion of testosterone to DHT, a key mediator of follicular miniaturization in AGA. Finasteride selectively inhibits type II 5α-reductase and is approved by Health Canada for the treatment of MPHL at 1 mg daily, whereas dutasteride inhibits both type I and type II isoenzymes and is used off-label for AGA.

In men, both agents improve hair density and slow progression. In a randomized trial of 917 men, dutasteride 0.5 mg outperformed finasteride 1 mg at 24 weeks for hair count and photographic outcomes.[19] A meta-analysis similarly reported greater efficacy with dutasteride than finasteride, with no significant differences in sexual adverse events between the two treatments.[20]

Adverse effects may include decreased libido, erectile dysfunction, ejaculatory dysfunction, and mood-related symptoms. As these medications reduce prostate-specific antigen (PSA) levels, baseline PSA assessment and appropriate interpretation of subsequent PSA values should be considered in men undergoing prostate cancer screening. In women, finasteride and dutasteride are not considered first-line and are used off-label, generally in selected postmenopausal women or in premenopausal women only with reliable contraception.

Platelet-Rich Plasma

Platelet-rich plasma (PRP) is a procedural therapy for AGA that involves injection of autologous platelet concentrates into the scalp. Its proposed mechanisms include the release of platelet growth factors that may support follicular function and angiogenesis.

A meta-analysis of randomized controlled trials has shown statistically significant increases in hair density compared to placebo at 3 and 6 months.[21] However, effects on hair thickness are less consistent, and overall study quality is variable. In clinical practice, responses are often heterogeneous. Combination therapy may improve outcomes, with greater benefit when PRP is used alongside topical minoxidil compared to either treatment alone. Overall, PRP may be considered an adjunctive option in selected patients.

Conclusion

AGA is a common form of hair loss that can significantly impact quality of life. Clinical presentation is sex-specific, with characteristic patterns observed in both male- and female-pattern hair loss. Although systemic causes are uncommon, clinicians should remain aware of potential associations with conditions such as PCOS and metabolic syndrome. A range of treatment options is available, with first-line therapies including topical minoxidil and, in men, oral finasteride.

Correspondence

Anastasiya Muntyanu, MD, PhD, FRCPC, FAAD
Email: anastasiya.muntyanu@mail.mcgill.ca

Financial Disclosures

A.M.: None declared.

References

1. Alessandrini A, Bruni F, Piraccini BM, Starace M. Common causes of hair loss - clinical manifestations, trichoscopy and therapy. J Eur Acad Dermatol Venereol. 2021;35(3):629–640. doi:10.1111/jdv.17079
2. Huang C-H, Fu Y, Chi C-C. Health-related quality of life, depression, and self-esteem in patients with androgenetic alopecia: a systematic review and meta-analysis. JAMA Dermatol. 2021;157(8):963–970. doi:10.1001/jamadermatol.2021.2196
3. Shin JW, Huh CH. Updates in treatment for androgenetic alopecia. Ann Dermatol. 2025;37(6):327–335. doi:10.5021/ad.25.042
4. McMullen E, Aldien AS, Sibbald C, Donovan J. Female pattern hair loss. CMAJ. 2026;198(8):E295. doi:10.1503/cmaj.251058
5. Rhodes T, Girman CJ, Savin RC, Kaufman KD, Guo S, Lilly FR, et al. Prevalence of male pattern hair loss in 18-49 year old men. Dermatol Surg. 1998;24(12):1330–1332. doi:10.1111/j.1524-4725.1998.tb00009.x
6. English RS, Jr. A hypothetical pathogenesis model for androgenic alopecia: clarifying the dihydrotestosterone paradox and rate-limiting recovery factors. Med Hypotheses. 2018;111:73–81. doi:10.1016/j.mehy.2017.12.027
7. Fabbrocini G, Cantelli M, Masarà A, Annunziata MC, Marasca C, Cacciapuoti S. Female pattern hair loss: a clinical, pathophysiologic, and therapeutic review. Int J Womens Dermatol. 2018;4(4):203–211. doi:10.1016/j.ijwd.2018.05.001
8. Norwood OT. Male pattern baldness: classification and incidence. South Med J. 1975;68(11):1359–1365. doi:10.1097/00007611-197511000-00009
9. Ludwig E. Classification of the types of androgenetic alopecia (common baldness) occurring in the female sex. Br J Dermatol. 1977;97(3):247–254. doi:10.1111/j.1365-2133.1977.tb15179.x
10. Sinclair R, Jolley D, Mallari R, Magee J. The reliability of horizontally sectioned scalp biopsies in the diagnosis of chronic diffuse telogen hair loss in women. J Am Acad Dermatol. 2004;51(2):189–199. doi:10.1016/s0190-9622(03)00045-8
11. Carmina E, Azziz R, Bergfeld W, Escobar-Morreale HF, Futterweit W, Huddleston H, et al. Female pattern hair loss and androgen excess: a report from the Multidisciplinary Androgen Excess and PCOS Committee. J Clin Endocrinol Metab. 2019;104(7):2875–2891. doi:10.1210/jc.2018-02548
12. Quinn M, Shinkai K, Pasch L, Kuzmich L, Cedars M, Huddleston H. Prevalence of androgenic alopecia in patients with polycystic ovary syndrome and characterization of associated clinical and biochemical features. Fertil Steril. 2014;101(4):1129–1134. doi:10.1016/j.fertnstert.2014.01.003
13. Rayner DG, McMullen E, Mundle K, Pham M, Sibbald C, Donovan J. Bi-directional association between female pattern hair loss and polycystic ovary syndrome: A systematic review and meta-analysis. JAAD Int. 2025;23:24–26. doi:10.1016/j.jdin.2025.08.004
14. Qiu Y, Zhou X, Fu S, Luo S, Li Y. Systematic review and meta-analysis of the association between metabolic syndrome and androgenetic alopecia. Acta Derm Venereol. 2022;102:adv00645. doi:10.2340/actadv.v101.1012
15. Landells I, Chow E, Gupta AK, Jasso Olivares JC, Thuraisingam T, Ahluwalia R, et al. A Canadian Consensus on androgenetic alopecia: approach and management. J Cutan Med Surg. 2025;29(5_suppl):5s–14s. doi:10.1177/12034754251368849
16. 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(2):286–294. doi:10.1111/jdv.17738
17. Penha MA, Miot HA, Kasprzak M, Müller Ramos P. Oral minoxidil vs topical minoxidil for male androgenetic alopecia: a randomized clinical trial. JAMA Dermatol. 2024;160(6):600–605. doi:10.1001/jamadermatol.2024.0284
18. 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(1):87–95. doi:10.1001/jamadermatol.2024.4593
19. Gubelin Harcha W, Barboza Martínez J, Tsai TF, Katsuoka K, Kawashima M, Tsuboi R, et al. A randomized, active- and placebo-controlled study of the efficacy and safety of different doses of dutasteride versus placebo and finasteride in the treatment of male subjects with androgenetic alopecia. J Am Acad Dermatol. 2014;70(3):489–498.e3. doi:10.1016/j.jaad.2013.10.049
20. Zhou Z, Song S, Gao Z, Wu J, Ma J, Cui Y. The efficacy and safety of dutasteride compared with finasteride in treating men with androgenetic alopecia: a systematic review and meta-analysis. Clin Interv Aging. 2019;14:399–406. doi:10.2147/cia.S192435
21. Zhang X, Ji Y, Zhou M, Zhou X, Xie Y, Zeng X, et al. Platelet-rich plasma for androgenetic alopecia: a systematic review and meta-analysis of randomized controlled trials. J Cutan Med Surg. 2023;27(5):504–508. doi:10.1177/12034754231191461