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ORIGINAL RESEARCH

# Consensus Recommendations for the Management of Androgenetic Alopecia in Egypt: A Modified Delphi Study

Sara M Mohy[1,2], Mohamed F Abozeid[1,3], Wael M Seoudy[1,2], Ahmed Elshahid[4], Alaa EA Moubasher[5], Ashraf Hassan[6], Eman Elmorsy[7], Eman RM Hofny[5], Essamelden M Mohamed[8], Hanan Mohammed Saleh[9], Hanan Sabry[10], Khaled Gharib[11], Mohamed Abdelwahed Gaber[12], Mohamed Saad Hegazy[13], Mona A Atwa[14], Nayera Moftah[15], Nermeen Ibrahim Bedair[16], Noha Ezzat Mohammed[17], Samia Esmat[18], Tarek Ameen[19], Tarek Hussein[7], Yasser Mostafa Gohary[20], Mahmoud A Rageh[1,4]

[1]Venus Research Center, The Egyptian Society for Woman Health (VENUS), Cairo, Egypt; [2]Department of Dermatology, Faculty of Medicine, Misr University for Science and Technology, Giza, Egypt; [3]Department of Plastic Surgery, Faculty of Medicine, Cairo University, Giza, Egypt; [4]Department of Dermatology, Faculty of Medicine, Al-Azhar University, Cairo, Egypt; [5]Department of Dermatology, Faculty of Medicine, Assiut University, Assiut, Egypt; [6]Department of Dermatology, Faculty of Medicine, Mansoura University, Mansoura, Egypt; [7]Department of Dermatology, Faculty of Medicine, Alexandria University, Alexandria, Egypt; [8]Department of Dermatology, Faculty of Medicine, Al-Azhar University, Assiut, Egypt; [9]Department of Dermatology, Faculty of Medicine, Ain Shams University, Cairo, Egypt; [10]Department of Dermatology, Faculty of Medicine, Benha University, Benha, Egypt; [11]Department of Dermatology, Faculty of Medicine, Zagazig University, Zagazig, Egypt; [12]Department of Dermatology, Faculty of Medicine, Menoufia University, Menoufia, Egypt; [13]Department of Dermatology, Military Medical Academy, Cairo, Egypt; [14]Department of Dermatology, Faculty of Medicine, Suez Canal University, Ismailia, Egypt; [15]Department of Dermatology, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt; [16]Department of Dermatology, Faculty of Medicine, Helwan University, Cairo, Egypt; [17]Department of Dermatology, Faculty of Medicine, Fayoum University, Fayoum, Egypt; [18]Department of Dermatology, Faculty of Medicine, Cairo University, Giza, Egypt; [19]Department of Dermatology, Faculty of Medicine, Tanta University, Tanta, Egypt; [20]Department of Dermatology, Faculty of Medicine, Beni-Suef University, Beni-Suef, Egypt

Correspondence: Mahmoud A Rageh, Department of Dermatology, Faculty of Medicine, Al-Azhar University, Cairo, Egypt, Email dr.mahmoudrageh@gmail.com



**Background:** Androgenetic alopecia (AGA) is a common dermatologic condition with significant psychological and social impact. Treatment remains challenging due to heterogeneity in patient response and limited long-term efficacy data.

**Objective:** To develop expert consensus guidelines for the diagnosis and management of AGA tailored to the Egyptian population, considering region-specific clinical and systemic factors.

**Methods:** A modified Delphi process was conducted in two rounds. Initially, a structured, evidence-informed questionnaire was distributed to 1,000 practicing dermatologists across Egypt; 723 completed the survey. Responses were analyzed and refined into consensus statements, which were subsequently evaluated by a panel of 20 senior dermatology professors. Statements achieving ≥75% agreement were considered consensus.

**Results:** Twenty-seven consensus statements were established and categorized into seven key areas: diagnosis, minoxidil, antiandrogens, low-level laser therapy, adjuvant treatments, hair transplantation, and counseling/hair aids. These recommendations reflect a synthesis of current evidence and national clinical experience.

**Conclusion:** This consensus provides a regionally relevant, evidence-based framework for AGA management in Egypt. It emphasizes individualized care, multidisciplinary strategies, and the integration of emerging therapies, and may serve as a model for practice in similar healthcare settings.

**Keywords:** androgenetic alopecia, modified Delphi, consensus, hair loss

## Introduction

Androgenetic alopecia (AGA), also known as male or female pattern hair loss, affects approximately 40% of men and 30% of women worldwide. It has a profound psychosocial impact, often leading to diminished self-esteem, social withdrawal, and anxiety. AGA is an androgen-dependent condition characterized by genetic predisposition and increased

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follicular sensitivity to androgens. Its pathogenesis involves the conversion of circulating androgens to dihydrotestosterone (DHT) in hair follicles via increased activity of the 5α-reductase (5AR) enzyme, resulting in follicular miniaturization and progressive hair thinning.[1]

While AGA was historically thought to follow an autosomal dominant inheritance pattern with incomplete penetrance in women, it is now understood to be polygenic, influenced by both genetic and environmental factors—underscoring its multifactorial nature.[2]

A broad spectrum of treatment options exists, including topical and oral medications, hormonal therapies, light-based modalities, bio-injectables, microneedling, and hair transplantation. However, managing AGA remains challenging due to variable treatment responses and the complex nature of its underlying biology. Currently approved treatments—topical minoxidil, oral finasteride, and low-level laser therapy (LLLT)—require long-term adherence to maintain efficacy.[3] Additionally, treatment strategies for AGA vary by gender, with oral finasteride and dutasteride predominantly used in men, while antiandrogen therapies such as spironolactone and cyproterone acetate are often reserved for women with hyperandrogenic profiles. Moreover, treatment safety considerations such as teratogenicity and hormonal regulation differ significantly between male and female patients.[4,5]

Recognizing the significance of region-specific variables such as genetic diversity, environmental influences, and healthcare accessibility, the Venus Research Center initiated this national consensus to establish practical, evidence-based guidelines for AGA management in Egypt. This was achieved through a structured, expert-driven process using a modified Delphi methodology.

## Materials and Methods

This study adhered to the ethical principles of the Declaration of Helsinki and was approved by the Institutional Review Board of Misr University for Science and Technology (Approval No. 2025/0094). A modified Delphi method was used to develop a national consensus on the management of AGA in Egypt.[6]

The initial questionnaire was developed by a core scientific committee of dermatology experts who conducted a comprehensive literature review using PubMed, Embase, and Cochrane databases from January 2019 to June 2024. The review focused on four domains: general principles of AGA, pharmacological management, procedural interventions, and special considerations.

The consensus process involved two sequential rounds. In the first round, a structured, evidence-informed questionnaire (Supplementary Material 1) was distributed via academic and professional networks to 1,000 dermatologists across various regions of Egypt to collect insights into clinical practices and expert opinions regarding AGA. 723 completed the survey (response rate = 723/1,000 = 72.3%). Feedback was thematically analyzed, and non-consensual or ambiguous items were refined, reworded, or eliminated before round two. In the second round, a panel of 20 senior dermatology professors reviewed the aggregated responses and were tasked with refining and evaluating key consensus statements. A three-point Likert scale (Agree, Neutral, Disagree) was used to measure levels of agreement (Figure 1).

This methodology combines broad clinical input with expert validation, ensuring both practical relevance and academic rigor—hallmarks of the modified Delphi technique.

All collected data were analyzed using IBM SPSS Statistics (version 28.0). Descriptive statistics were used to summarize response distributions, with frequencies and percentages calculated for categorical variables. Agreement levels were calculated as the percentage of expert panelists selecting “Agree” on each statement using descriptive statistics. Consensus was defined as ≥75% agreement among expert panelists. Statistical integrity and methodological validity were maintained throughout the process.

## Results

All participating dermatologists had a minimum of five years of clinical experience and were actively practicing in Egypt. Following the two-round Delphi process, consensus was reached on 27 statements related to the diagnosis and management of AGA. The full list of consensus statements is provided in Table 1, serving as a practical framework for clinicians involved in the management of AGA. Full item-level distributions are available in Supplementary Table 1.

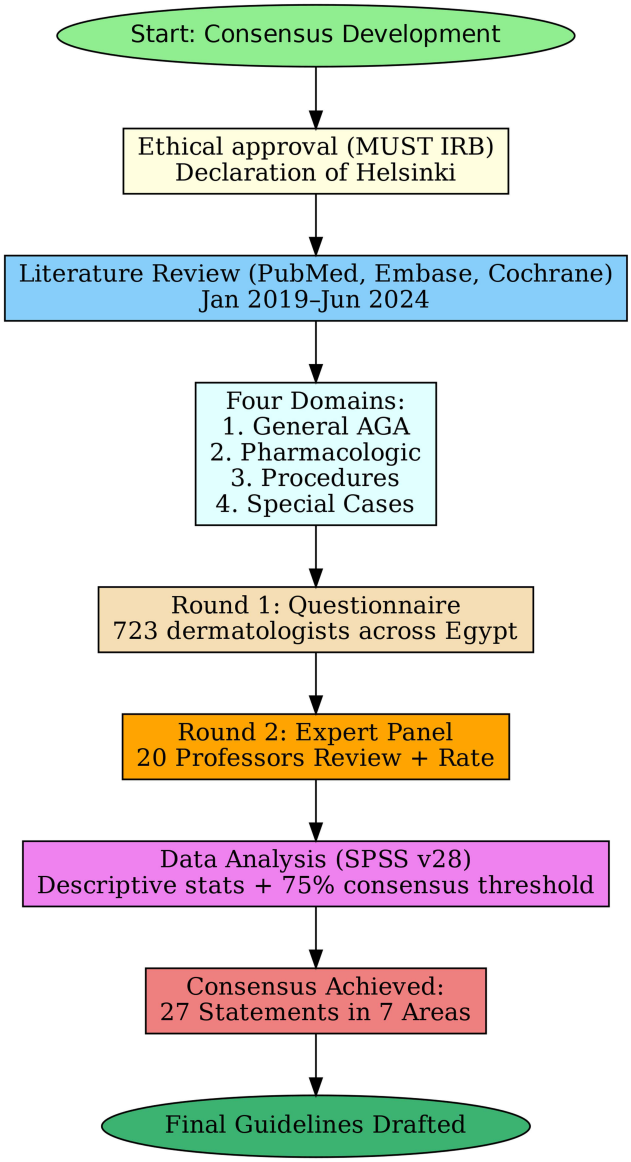


**Figure 1** Flowchart illustrating the modified Delphi methodology used to develop consensus recommendations. The process included ethical approval, literature review, two rounds of expert input, and statistical analysis, culminating in 27 consensus statements across seven clinical domains.

## Discussion and Statements

The expert panel reached consensus on 27 statements, categorized into seven key areas: (1) diagnosis, (2) minoxidil, (3) antiandrogens, (4) low-level laser therapy, (5) adjuvant therapies, (6) hair transplantation, and (7) patient counseling and hair aids. While this work does not introduce new molecular mechanisms or experimental therapies, its primary contribution is clinical and regional: it delivers structured, evidence-informed, and context-specific guidance for AGA in Egypt, integrating real-world practice patterns with expert consensus.

### Key Area 1: Diagnosis

**Statement 1: All patients with suspected AGA must undergo a comprehensive medical history assessment, encompassing the age of commencement of hair loss, family history; medical conditions such as metabolic syndrome and insulin resistance; use of anabolic substances including whey and isolate protein shakes; and smoking habits**

**Table 1** Summary of Consensus Statements and Agreement (%)

| Key Area | Statement # | Summary Statement | Agreement (%) |
|---|---|---|---|
| Diagnosis | 1 | All patients with suspected AGA must undergo a comprehensive medical history assessment, encompassing the age of commencement of hair loss, family history; medical conditions such as metabolic syndrome and insulin resistance; use of anabolic substances including whey and isolate protein shakes; and smoking habits | 95 |
| | 2 | Clinical examination of the scalp is typically sufficient for diagnosing AGA | 85 |
| | 3 | Complementary assessments, including the pull test and trichoscopy, for evaluating the terminal-to-vellus hair ratio, are useful tools to confirm the diagnosis | 90 |
| | 4 | Basic investigations can be reserved for patients whose history and examination findings suggest a specific disease or deficiency | 90 |
| Minoxidil | 5 | Topical minoxidil solution is administered in a dosage of 1 mL twice daily | 100 |
| | 6 | Minoxidil 5% foam is more effective than 2% solution in female pattern hair loss | 90 |
| | 7 | Higher concentrations of minoxidil >5% have a limited role in therapy | 85 |
| | 8 | Patients on minoxidil should be warned of the possibility of an initial but temporary hair fall, which should not prompt discontinuation of treatment | 95 |
| | 9 | Hypertrichosis is one of the most bothersome side effects of minoxidil but typically resolves within 1–6 months after drug discontinuation | 90 |
| | 10 | Patients with AGA are considered non-responders to minoxidil after six months of continuous use without observable improvement | 90 |
| | 11 | Oral minoxidil in doses starting from 0.25 mg daily is considered, especially for patients with topical minoxidil non-compliance | 85 |
| | 12 | Intradermal minoxidil injection is an alternative for patients with poor compliance with daily topical application | 85 |
| | 13 | AGA in children and adolescents is becoming a more evident phenomenon, and topical minoxidil may be of therapeutic value | 80 |
| Antiandrogens | 14 | Oral finasteride at a daily dosage of 1 mg is a potent modality in males with AGA | 95 |
| | 15 | Oral dutasteride 0.5 mg/day may be used as a line of therapy in male AGA patients | 90 |
| | 16 | Androgen receptor antagonists can play a role in the management of FPHL | 80 |
| Low-Level Laser Therapy | 17 | Low-level laser therapy three times per week causes a decrease in the number of vellus hairs, an increase in terminal hair, and an increase in shaft diameter | 95 |
| Adjuvant Therapies | 18 | Non-minoxidil topical therapies may have a beneficial role | 75 |
| | 19 | Natural oils may be considered in minoxidil non-responders or those who refuse minoxidil therapy | 80 |
| | 20 | Ketoconazole 2% shampoo (2–3 times per week) can be employed as an adjuvant therapy in AGA due to its anti-inflammatory or antiandrogenic properties | 80 |
| | 21 | Nutritional supplements, including amino acids, biotin, zinc, and other micronutrients, have a role as adjuvant therapy in managing AGA or patients who have simultaneous telogen effluvium and AGA | 75 |
| | 22 | Platelet-rich plasma (PRP) sessions for AGA can be done monthly for 3 sessions, then a session every 3–6 months as a maintenance treatment | 90 |
| | 23 | Bio-injectables are promising | 75 |
| | 24 | Microneedling helps boost the therapeutic response to topical treatment | 90 |
| | 25 | Combined therapies with topical minoxidil are recommended for AGA patients | 95 |
| Hair Transplantation | 26 | Hair transplantation is recommended if there is no perceptible improvement or stabilization after 12 months of continuous medical treatment | 95 |
| Counseling/Hair Aids | 27 | In patients with AGA, you may recommend counseling, non-medical aesthetic wigs, hair extensions, and topical powder makeup in late-stage patients or those who refuse any kind of treatment | 90 |

**Note**: Raw counts are provided in Supplementary Table 1.

Male pattern hair loss (MPHL) usually starts between ages 16–30, while female pattern hair loss (FPHL) often appears between 20–30 or postmenopausal at 50–55.[7,8] AGA is hereditary; sons of affected fathers are 5–6 times more likely to experience hair loss, especially if there's also a maternal history.[2]

AGA is linked to systemic risks like metabolic syndrome and cardiovascular disease. Elevated androgens can promote smooth muscle cell proliferation in blood vessels, increasing the risk of hypertension. Additionally, androgens have been shown to lower HDL cholesterol. Therefore, assessing lipid profiles in AGA patients is crucial to mitigate these risks.[9,10]

Whey protein and branched-chain amino acids may raise testosterone, potentially accelerating hair loss in predisposed individuals.[11] Smoking increases both the risk and severity of AGA, with smokers 1.8 times more likely to develop it.[12]

**Statement 2: Clinical examination of the scalp is typically sufficient for diagnosing AGA**

The diagnosis of AGA typically relies on clinical presentation; however, in instances of uncertainty, trichoscopy may assist clinicians in achieving an accurate diagnosis.[13]

**Statement 3: Complementary assessments, including the pull test and trichoscopy, for evaluating the terminal-to-vellus hair ratio, are useful tools to confirm the diagnosis**

Although not specific to AGA, the pull test helps confirm active shedding. Trichoscopy reveals key AGA features—follicular miniaturization, diameter variability, yellow dots, perifollicular discoloration, and increased vellus hairs—more prominent in the frontal than occipital scalp.[14]

**Statement 4: Basic investigations can be reserved for patients whose history and examination findings suggest a specific disease or deficiency**

Diagnosing AGA in women may require hormonal testing and blood work for vitamin D, iron, B12, TSH, and prolactin. Scalp biopsy is considered when the diagnosis is unclear, and consultations with other specialists may also be necessary for a comprehensive evaluation.[15] In women with signs of hyperandrogenism or menstrual irregularities, evaluation for polycystic ovary syndrome (PCOS) is warranted, including pelvic ultrasound and assessment of serum LH/FSH ratio. Additional endocrine tests, such as serum total and free testosterone, DHEA-S, and 17-hydroxyprogesterone, may be indicated to exclude other androgen-excess disorders such as congenital adrenal hyperplasia or androgen-secreting tumors. Referral to gynecology/endocrinology may be appropriate.[16,17]

## Key Area 2: Minoxidil

**Statement 5: Topical minoxidil solution is administered in a dosage of 1 mL twice daily**

Minoxidil is generally given as a 2% or 5% solution, with 1 mL applied twice daily for individuals aged 18 and older.[1]

**Statement 6: Minoxidil 5% foam is more effective than 2% solution in female pattern hair loss**

Studies indicate that the 5% minoxidil formulation is more effective in treating hair loss in men, while both the 2% and 5% formulations show promising results for women with FPHL. Additionally, foam formulations have demonstrated superior results compared to other forms, like shampoo, spray, or solution.[18,19]

**Statement 7: Higher concentrations of minoxidil >5% have a limited role in therapy**

While some studies suggest that higher concentrations of minoxidil enhance efficacy, others report no additional benefit. Higher concentrations of minoxidil may lead to more side effects, such as hypertrichosis, contact dermatitis, and early telogen shedding. Rare side effects include headaches, breathlessness, palpitations, and tachycardia.[20]

**Statement 8: Patients on minoxidil should be warned of the possibility of an initial but temporary hair fall, which should not prompt discontinuation of treatment**

Minoxidil may initially increase hair shedding as follicles shift to the anagen phase. Telogen effluvium can also occur 6–8 weeks after starting but usually resolves within a few weeks or months with continued use.[21]

**Statement 9: Hypertrichosis is one of the most bothersome side effects of minoxidil but typically resolves within 1–6 months after drug discontinuation**

Minoxidil may cause hypertrichosis, with excess hair growth in non-androgen-dependent areas, often due to accidental application or increased sensitivity of hair follicles.[22,23] It typically resolves within 3–4 months after stopping treatment.[24]

**Statement 10: Patients with AGA are considered non-responders to minoxidil after six months of continuous use without observable improvement**

A follow-up study using the sulfotransferase enzymatic activity assay found that after 8 weeks of topical minoxidil treatment, the sulfotransferase enzyme activity in hair stabilized. This suggests that individuals who respond to minoxidil are unlikely to develop resistance to the current dosage, and those who do not respond will not become responders.[25]

**Statement 11: Oral minoxidil in doses starting from 0.25 mg daily is considered, especially for patients with topical minoxidil non-compliance**

Though not approved for alopecia, oral minoxidil may be preferred by patients who find topical application inconvenient or ineffective. Low doses (0.25–1 mg/day, titratable up to 5 mg/day) can reduce local side effects.[26,27]

Patients should be counseled on risks such as hypotension, headache, hypertrichosis, and edema. Blood pressure monitoring is essential, and tapering is advised. It is contraindicated during pregnancy and breastfeeding and should be stopped three months before conception.[28]

**Statement 12: Intradermal minoxidil injection is an alternative for patients with poor compliance with daily topical application**

Intradermal minoxidil is well-tolerated with minimal side effects and no systemic impact. Studies show it increases the terminal-to-vellus hair ratio and anagen hair percentage without notable adverse effects.[29,30] Further research is needed to determine optimal dosing.[31]

**Statement 13: AGA in children and adolescents is becoming a more evident phenomenon, and topical minoxidil may be of therapeutic value**

AGA is often underrecognized in children, resulting in limited data on its trichoscopic patterns and treatment outcomes.[32]

Genetic and hormonal factors play key roles, with 83% of cases reporting family history. Onset typically occurs around adrenarche and may be pre- or postpubertal.[33]

Topical minoxidil has been used safely, but further studies are needed to confirm the efficacy and safety of treatments in pediatric AGA.[34]

## Key Area 3: Antiandrogens

**Statement 14: Oral finasteride at a daily dosage of 1 mg is a potent modality in males with AGA**

Finasteride, a type II 5AR inhibitor approved in 1997 for male AGA, reduces scalp and serum DHT by ~64–68% at 1 mg/day.[35] While effective, it may cause libido loss, erectile dysfunction, or reduced ejaculate volume, which usually resolve after stopping the drug.[36,37]

To minimize side effects, lower or staggered doses (0.2–0.5 mg) may be started and gradually increased.[38] Finasteride is contraindicated in women of childbearing age but may be considered for postmenopausal women with FPHL unresponsive to other treatments.[38]

**Statement 15: Oral dutasteride 0.5 mg/day may be used as a line of therapy in male AGA patients**

Dutasteride, a dual 5AR inhibitor, may be more effective than finasteride for AGA, with faster hair growth observed at a 0.5 mg dose. Despite this, finasteride remains first-line due to FDA approval and wider insurance coverage. Intradermal dutasteride has shown promise with minimal side effects, though the oral form is preferred for its superior DHT suppression and efficacy.[1,3]

**Statement 16: Androgen receptor antagonists can play a role in the management of FPHL**

Androgen receptor antagonists are used in FPHL, especially with signs of hyperandrogenism, though supporting evidence remains limited, and their benefit with minoxidil is unclear.[3]

Spironolactone (25–200 mg/day) blocks aldosterone and androgen receptors. Side effects include fatigue, mastalgia, menstrual changes, and hypotension.[39] Cyproterone inhibits gonadotropins and androgen receptors. Though less effective than minoxidil overall, it may help women with acne or menstrual issues. Side effects include libido changes and weight gain.[40]

Flutamide is rarely used due to hepatotoxicity. Bicalutamide, with lower liver risk, has shown promise and may reduce minoxidil-induced hypertrichosis when combined with oral minoxidil, though more research is needed.[5]

All antiandrogens are contraindicated in pregnancy, requiring effective contraception. Some oral contraceptives with antiandrogenic progestins may also help.[40]

## Key Area 4: Low-Level Laser Therapy

**Statement 17: Low-level laser therapy three times per week causes a decrease in the number of vellus hairs, an increase in terminal hair, and an increase in shaft diameter**

Low-level laser therapy (LLLT), FDA-approved for AGA, promotes hair growth by enhancing scalp circulation, cellular activity, and growth factor production, aiding the shift to the anagen phase. It also reduces inflammation and may lower DHT levels by modulating 5AR activity.[41,42]

## Key Area 5: Adjuvant Therapies

**Statement 18: Non-minoxidil topical therapies may have a beneficial role**

The panel concurred that minoxidil continues to be the cornerstone of treatment for AGA, while acknowledging that other topical formulations might offer additional benefits, though evidence to support this is insufficient.

Topical tretinoin may promote hair growth, especially when combined with minoxidil, showing synergistic effects in vitro.[43] Its enhancement of minoxidil absorption may result from reduced skin barrier function or increased sulfonation, as tretinoin has been shown to stimulate sulfotransferase activity.[44,45]

Aminexil may reduce root aging by countering fibrosis and collagen stiffening. While nanocarrier formulation showed enhanced hair growth in chemotherapy-induced alopecia in rats, its efficacy remains lower than minoxidil in other studies.[46,47]

Karaca and Alkpolat reported that redensyl, capixyl, and procapil (RCP) led to 2.5 times more hair growth than 5% minoxidil after 24 weeks, though the study lacked objective evaluation methods.[48]

**Statement 19: Natural oils may be considered in minoxidil non-responders or those who refuse minoxidil therapy**

Given their effectiveness, high safety profile, and minimal side effects, these natural oils present a viable and potentially beneficial option for managing hair loss.

Rosemary oil may promote hair growth by improving scalp circulation and modulating prostaglandins. In a 6-month trial, it was as effective as 2% minoxidil, with less scalp itching.[49]

Pumpkin seed oil may aid hair growth by inhibiting 5AR and reducing IL-6. In a trial, men taking 400 mg daily saw a 40% hair count increase vs 10% with placebo.[50]

**Statement 20: Ketoconazole 2% shampoo (2–3 times per week) can be employed as an adjuvant therapy in AGA due to its anti-inflammatory or antiandrogenic properties**

Ketoconazole has antifungal, anti-inflammatory, and antiandrogenic effects, including inhibition of testosterone synthesis and reduction of DHT, making it a potential AGA treatment. Studies have shown improvements in hair shaft diameter with applications ranging from twice weekly to daily, with positive results even at 2–3 times per week. It is generally well-tolerated and associated with minimal side effects.[3,51]

**Statement 21: Nutritional supplements, including amino acids, biotin, zinc, and other micronutrients, have a role as adjuvant therapy in managing AGA or patients who have simultaneous telogen effluvium and AGA**

The role of oral supplements in hair loss remains debated. In telogen effluvium, nutrients like L-cystine and B-complex vitamins may improve the anagen rate, but their benefit in AGA is unclear.[52]

Iron is vital for hair follicle cell division, and deficiency may impair growth. Treatment is recommended when ferritin ≤30 µg/L.[53] Low ferritin and vitamin D have been linked to telogen effluvium and FPHL, with vitamin D playing a role in anagen initiation and follicle cycling.[54]

Supplements like saw palmetto and marine proteins may offer antiandrogenic and anti-inflammatory effects but require further study.[52]

**Statement 22: Platelet-rich plasma (PRP) sessions for AGA can be done monthly for 3 sessions, then a session every 3–6 months as a maintenance treatment**

PRP promotes hair regrowth by delivering growth factors like platelet-derived growth factor, vascular endothelial growth factor (VEGF), fibroblast growth factor, and others, influencing cellular proliferation, differentiation, and angiogenesis. PRP also activates pathways such as Wnt/β-catenin to stimulate follicle regeneration.[55] However, variability in preparation, protocols, and outcome measures leads to mixed results, with some studies showing improved diameter but inconsistent density gains.[56]

**Statement 23: Bio-injectables are promising**

Bio-injectables such as stem cells, exosomes, and growth factors offer promise for AGA, especially in women and older adults.[57] Stem cells from hair follicles and fat tissue have improved hair density and thickness, likely due to the responsiveness of resting follicles in older patients.[58]

Exosomes promote follicle growth and anagen transition, with early studies showing increased density and minimal side effects.[59]

Botulinum toxin may also help by inhibiting TGF-β1, with some reports noting hair count improvement.[60]

Variability in study designs, stem cell sources, treatment protocols, and evaluation methods limit the ability to draw definitive conclusions. Inconsistent control groups and a lack of comparisons with established treatments weaken the evidence base.[57]

**Statement 24: Microneedling helps boost the therapeutic response to topical treatment**

Microneedling (MN) promotes hair growth by enhancing topical absorption, stimulating follicular stem cell regeneration, and activating the Wnt/β-catenin pathway, which boosts growth factors like VEGF.[61,62] A meta-analysis showed that MN combined with minoxidil or other treatments significantly improved hair density and thickness without notable side effects.[63]

**Statement 25: Combined therapies with topical minoxidil are recommended for AGA patients**

Combining minoxidil with microneedling or LLLT offers better outcomes with minimal side effects. Finasteride is effective but requires monitoring. For those avoiding oral drugs, topical finasteride or intradermal dutasteride can be effective alternatives with lower systemic exposure.[64,65] Tailored approaches based on patient preferences and tolerance are essential for sustained results.

## Key Area 6: Hair Transplantation

**Statement 26: Hair transplantation is recommended if there is no perceptible improvement or stabilization after 12 months of continuous medical treatment**

Hair loss typically becomes noticeable after approximately 50% of native hair is lost, a threshold often used to determine candidacy for hair transplantation. Hair transplantation is optimally performed after the age of 25. Suitable candidates include AGA patients with sufficient donor hair, especially men at Norwood III to V and women at Ludwig stage II.[66]

## Key Area 7: Counseling/Hair Aids

**Statement 27: In patients with AGA, you may recommend counseling, non-medical aesthetic wigs, hair extensions, and topical powder makeup in late-stage patients or those who refuse any kind of treatment**

Given the psychological effects of AGA, effective patient counseling is a crucial component of treatment. The previously mentioned non-medical aesthetic hair aids contribute to enhancing appearance in AGA. The use of synthetic hair for prosthetic implantation is no longer recommended, owing to the high incidence of adverse effects.[67]

Taken together, these statements outline a patient-centered framework; to facilitate uptake, implementation pathways may include incorporation into national CME activities, society-endorsed practice tools, and dermatology residency curricula. Several regional consensus efforts in hair disorders have emphasized similar multimodal approaches; our statements align with these themes while addressing region-specific factors such as healthcare access, cost, and patient preferences.[68–71] Prospective multicenter audits and registry-based tracking could evaluate uptake and real-world outcomes.

This study has several limitations. Beyond the inherent subjectivity of Delphi processes and potential selection bias in expert recruitment, the use of a 3-point scale may limit granularity relative to 7- or 9-point formats. We did not measure patient-level outcomes or cost-effectiveness; these should be addressed in future implementation studies. Finally, the national focus may limit generalizability to settings with different epidemiology or resource availability.

# Conclusion

This consensus offers structured, evidence-based guidelines derived from the clinical experience of Egyptian dermatologists, emphasizing individualized care, multimodal therapy, and continued research into emerging treatments. By addressing region-specific considerations, this work offers a practical framework for AGA care in Egypt and may serve as a model for similar contexts globally.

# Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

# Ethics Approval and Informed Consent

The study was done in accordance with the Declaration of Helsinki after approval of the ethical committee of the Faculty of Medicine, Misr University for Science and Technology (Approval No. 2025/0094).

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## Author Contributions

All authors made a significant contribution to this study, whether that is in the conception, study design, execution, acquisition of data, analysis, and interpretation, or all these areas; took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

## Consent for Publication

No consent needed as no patients were recruited.

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## Disclosure

The authors report no conflicts of interest in this work.

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