



Research Article

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Comparing the Therapeutic Efficacy of Minoxidil, Dutasteride, and Microneedling in the Treatment of Androgenic Alopecia in Males

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Abstract

Background: Androgenetic alopecia is the most common reason for progressive hair loss in males. Currently a combination of treatment strategies for improving hair regrowth is recommended over a single treatment. **Objective:** To compare the therapeutic effectiveness of only 5% minoxidil (topical), 5% minoxidil (topical) plus microneedling, and 5% minoxidil (topical) plus microneedling and dutasteride (oral) in treating androgenetic alopecia in males. **Methods:** Our study includes 90 males; each group had 30 participants. Group I had 5% minoxidil (topical) alone, Group II had 5% minoxidil (topical) plus weekly microneedling, and Group III had 5% minoxidil (topical), weekly microneedling, and dutasteride (oral) (0.5 mg/day). The patient's evaluation was done at baseline, the 3rd month, and the 6th month through examining clinically, digital dermoscopy, Hamilton–Norwood and Basic and Specific (BASP) classifications, hair density, hair shaft thickness, the Hair Growth Self-Assessment Scale, and patient satisfaction scores. **Results:** The clinical and demographic characteristics at baseline were similar between all the groups. All treatment modalities resulted in significant improvement in hair density, hair shaft thickness, Hamilton–Norwood classification, BASP classification, hair growth self-assessment, and patient satisfaction after six months (all $p < 0.001$). However, combination therapy demonstrated significantly superior efficacy compared with topical minoxidil alone. The greatest improvements were detected in Group III; next came Group II, while Group I showed the least improvement across all efficacy outcomes. **Conclusions:** This multimodal approach represents a therapeutic strategy that is well-tolerated and effective in managing androgenetic alopecia in males.

Keywords: Androgenetic alopecia; BASP classification; Dutasteride; Hair density; Minoxidil; Microneedling.

مقارنة الفعالية العلاجية للمينوكسيديل والدوتاسترايد والوخز الدقيق في علاج الثعلبة الأندروجينية لدى الذكور

الخلاصة

الخلفية: الثعلبة الأندروجينية هي السبب الأكثر شيوعاً لتساقط الشعر التدريجي لدى الذكور. حالياً، يوصى باعتماد استراتيجيات علاجية لتحسين نمو الشعر خلال جلسة واحدة. **الهدف:** مقارنة الفعالية العلاجية لعلاج 5٪ فقط من المينوكسيديل (الموضعي)، 5٪ مينوكسيديل (موضعي) بالإضافة إلى الإبر الدقيقة (الوخز الدقيق)، و 5٪ مينوكسيديل (موضعي) بالإضافة إلى الإبر الدقيقة والدوتاستيريد (الفموي) في علاج الثعلبة الأندروجينية لدى الذكور. **الطرائق:** شملت دراستنا 90 ذكراً؛ كل مجموعة كان بها 30 مشاركاً. المجموعة الأولى استخدمت 5٪ مينوكسيديل (موضعي) فقط، والمجموعة الثانية 5٪ مينوكسيديل (موضعي) بالإضافة إلى الإبر الدقيقة الأسبوعي، والمجموعة الثالثة 5٪ مينوكسيديل (موضعي)، وأبر دقيقة أسبوعي، ودوتاسترايد (عن طريق الفم) (0.5 ملغ/يوم). تم إجراء تقييم المريض في البداية والشهر الثالث والسادس من خلال فحص سريري، وتظليل الجلد الرقمي، وتصنيفات هاميلتون-نورود والتصنيفات الأساسية والمحددة (BASP)، وكثافة الشعر، وسماكة ساق الشعر، ومقياس التقييم الذاتي لنمو الشعر، ودرجات رضا المرضى. **النتائج:** كانت الخصائص السريرية والديموغرافية في الأساس متشابهة بين جميع المجموعات. أدت جميع طرق العلاج إلى تحسين كبير في كثافة الشعر، وسماك ساق الشعر، وتصنيف هاميلتون-نورود، وتصنيف BASP، والتقييم الذاتي لنمو الشعر، ورضا المرضى بعد ستة أشهر (جميعها $p < 0.001$). ومع ذلك، أظهر العلاج المركب فعالية متفوقة بشكل ملحوظ مقارنة بالمينوكسيديل الموضعي وحده. تم رصد أكبر التحسينات في المجموعة الثالثة؛ تلتها المجموعة الثانية، بينما أظهرت المجموعة الأولى أقل تحسن في جميع نتائج الفعالية. **الاستنتاجات:** يمثل هذا النهج متعدد الوسائط استراتيجية علاجية مقبولة وفعالة في علاج الثعلبة الأندروجينية لدى الذكور.

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INTRODUCTION

Androgenetic alopecia (AGA), also referred to as male-pattern hair loss, is the most prevalent form of nonscarring alopecia and affects a substantial proportion of men worldwide. Its prevalence increases progressively with age, affecting approximately 30–50% of men by the age of 50 years [1]. Although AGA is not a life-threatening disorder, it has considerable psychosocial consequences, including reduced self-esteem, impaired body image, anxiety, and diminished quality of life. The disease is characterized by progressive miniaturization of genetically susceptible hair follicles under the influence of androgens,

ultimately resulting in the gradual replacement of thick terminal hairs with fine vellus-like hairs and clinically apparent patterned hair loss. Owing to its chronic and progressive nature, AGA remains a major therapeutic challenge despite the availability of several medical and procedural treatment modalities [2]. The AGA pathogenesis is multifactorial and involves a complex interaction between genetic predisposition, androgen metabolism, follicular stem-cell dysfunction, perifollicular microinflammation, and alterations in the hair cycle. Among these mechanisms, dihydrotestosterone (DHT), produced from testosterone through the action of type I and type II 5 α -reductase enzymes, plays a principal role in disease

progression [3]. Increased sensitivity of genetically predisposed hair follicles to DHT shortens the anagen phase, prolongs the telogen phase, and induces progressive follicular miniaturization, ultimately reducing hair density and shaft diameter. Recent advances have also highlighted the contribution of altered Wnt/ β -catenin signaling, vascular dysregulation, oxidative stress, and impaired dermal papilla cell activity to the pathophysiology of AGA, providing several potential therapeutic targets beyond androgen suppression alone [4]. Topical minoxidil remains one of the cornerstone therapies for male AGA and is approved by the United States Food and Drug Administration for this indication. Initially developed as an antihypertensive agent, minoxidil exerts its therapeutic effects after conversion to its active sulfate metabolite by follicular sulfotransferase enzymes. It enhances hair growth through multiple complementary mechanisms, including activation of ATP-sensitive potassium channels, prolongation of the anagen phase, stimulation of dermal papilla cell proliferation, increased expression of vascular endothelial growth factor (VEGF), and activation of prostaglandin synthesis and Wnt/ β -catenin signaling pathways [5]. These biological effects improve perifollicular vascularization, delay follicular regression, and promote the transformation of miniaturized follicles into thicker terminal hairs. Nevertheless, the response to topical minoxidil alone remains variable, and many patients experience only partial hair regrowth, emphasizing the necessity of adjunctive therapeutic strategies to optimize clinical outcomes [2]. Microneedling has recently emerged as a promising adjuvant therapy for androgenetic alopecia because it targets follicular regeneration through mechanisms distinct from conventional pharmacological treatments. The creation of controlled microinjuries within the scalp initiates a physiological wound-healing response characterized by the release of platelet-derived growth factor, transforming growth factor- β , fibroblast growth factor, epidermal growth factor, and vascular endothelial growth factor. In addition, microneedling activates the Wnt/ β -catenin signaling pathway and promotes the proliferation of hair follicle stem cells within the bulge region, thereby facilitating the transition of follicles into the anagen phase [6]. Another important advantage is the transient disruption of the stratum corneum, which enhances transdermal delivery and intrafollicular penetration of topical medications such as minoxidil, providing a strong biological rationale for combined treatment strategies. Recent systematic reviews have consistently demonstrated that combining microneedling with topical therapy significantly improves objective hair growth parameters without increasing treatment-related adverse events [7]. Dutasteride represents another important therapeutic advance in the management of male androgenetic alopecia. Unlike finasteride, which selectively inhibits type II 5 α -reductase, dutasteride inhibits both type I and type II isoenzymes, resulting in more profound suppression of scalp dihydrotestosterone concentrations. This dual inhibitory activity more effectively prevents androgen-

induced follicular miniaturization, prolongs the anagen phase, and promotes the regeneration of terminal hair follicles [8]. Recent evidence has consistently demonstrated that oral dutasteride provides superior improvements in total and terminal hair density compared with several currently available monotherapies, while maintaining an acceptable safety profile in appropriately selected patients. Furthermore, the complementary mechanisms of dutasteride, topical minoxidil, and microneedling provide a strong biological basis for multimodal therapy targeting different stages of the pathogenic cascade of androgenetic alopecia [4]. Growing evidence indicates that combination therapy is more effective than single-agent treatment because it simultaneously enhances follicular regeneration, improves topical drug delivery, and suppresses androgen-mediated follicular damage. Nevertheless, considerable heterogeneity persists among published studies regarding treatment protocols, microneedling techniques, treatment duration, and outcome assessment methods, limiting direct comparison of therapeutic efficacy. Moreover, few prospective comparative studies have evaluated the combined effects of topical 5% minoxidil, weekly microneedling, and oral dutasteride within the same study population using both objective trichoscopic parameters and validated clinical severity scales [9]. Therefore, the present study was designed to compare the therapeutic efficacy of topical 5% minoxidil alone, topical minoxidil combined with weekly microneedling, and the triple combination of topical minoxidil, weekly microneedling, and oral dutasteride in males with androgenetic alopecia, aiming to provide clinically relevant evidence that may help optimize treatment selection and improve outcomes in patients with male androgenetic alopecia.

METHODS

Study design and setting

This retrospective comparative clinical study. Included the records of history taking and follow-up of 90 male patients with clinically diagnosed androgenetic alopecia who attended the Dermatology Outpatient Private Clinic in the Wasit governorate of Iraq during the study period from January 2025 to June 2025. Thirty eligible medical records were randomly selected from each of the three treatment groups using a computer-generated random sequence, resulting in three equal groups of 30 patients. Randomization was applied to record selection rather than treatment allocation; therefore, allocation concealment was not applicable. All the patient's data was collected from the clinical record with no intervention in the follow-up and treatment procedures.

Inclusion criteria

Adult male patients aged between 18 and 50 years with clinically confirmed androgenetic alopecia of mild-moderate-severe severity were considered eligible for

inclusion. All participants had a stable general medical condition and had not received topical or systemic treatment for androgenetic alopecia for an appropriate washout period before enrollment. Patients were also required to provide written informed consent and demonstrate willingness to adhere to the prescribed treatment protocol and scheduled follow-up visits.

Exclusion criteria

Patients were excluded if they had other causes of nonscarring or scarring alopecia, active inflammatory or infectious scalp disorders, previous hair transplantation, hypersensitivity to any of the study medications, or contraindications to oral dutasteride. Individuals receiving medications known to affect hair growth, those with significant systemic illnesses that could interfere with treatment evaluation, or patients who failed to attend scheduled follow-up visits were also excluded. These criteria were adopted to minimize potential confounding factors and ensure that the observed treatment responses were attributable to the investigated therapeutic interventions rather than other underlying conditions.

Treatment protocol

Participants in Group I received topical 5% minoxidil solution as monotherapy. Patients were instructed to apply 1 mL of the solution twice daily to the affected scalp areas throughout the six-month treatment period. Detailed verbal and written instructions regarding the method of application, treatment adherence, and scalp hygiene were provided at enrollment, and compliance was reinforced at each follow-up visit. Participants in Group II received topical 5% minoxidil according to the same regimen in addition to microneedling, which was performed under strict aseptic conditions using a sterile Dr. Pen microneedling device (Guangzhou Ekai Electronic Technology Co., Ltd., Guangzhou, China) equipped with disposable 1.5-mm needles. Eight passes were performed in horizontal, vertical, and diagonal directions over the affected scalp until mild uniform erythema or pinpoint bleeding was achieved. Each microneedling session lasted approximately 20–25 minutes. Topical minoxidil therapy was resumed 24 hours after each microneedling session. Participants in Group III received the same combined regimen as Group II together with oral dutasteride administered at a dose of 0.5 mg once daily throughout the study period. Before treatment initiation, all patients were counseled regarding the expected therapeutic benefits, possible adverse effects, and the importance of treatment adherence. Compliance with both topical and oral medications was evaluated at every follow-up visit through direct patient interviews and medication records review.

Data collection

All the patient's data was collected from the clinical records with no intervention in the follow-up and

treatment procedures. The following describes the treatment protocol routinely followed at the clinic for AGA as documented in the patients' medical records. All participants underwent a comprehensive baseline evaluation before treatment initiation using a standardized case report form. Demographic data, including age, occupation, disease duration, smoking status, family history of androgenetic alopecia, and previous treatment history, were recorded. A complete dermatological examination was subsequently performed to confirm the diagnosis of androgenetic alopecia and exclude other causes of hair loss. Disease severity was clinically evaluated using both the Hamilton–Norwood Classification and the Basic and Specific Classification, which served as standardized grading systems throughout the study. Clinical and dermoscopic assessments were performed at three predefined time points: baseline (before treatment initiation), three months, and six months after initiation of therapy. To minimize interobserver variability, all examinations and measurements were performed by the same dermatologist following a standardized assessment protocol. Objective treatment efficacy was assessed by measuring hair density and hair shaft thickness, which represented the primary outcome measures of the study. Hair density was defined as the number of visible hairs per square centimeter (hairs/cm²), while hair shaft thickness was expressed as the mean diameter of terminal hairs in micrometers (μm). Both parameters were measured using a dermatoscope skin analyzer equipped with digital image analysis software. Measurements were obtained from a standardized scalp area measuring 1 cm², located 10 cm superior and 3 cm anterior to the upper border of the tragus, which was identified at baseline and used consistently throughout the follow-up period. The same magnification, illumination, and image acquisition settings were maintained during all visits to ensure measurement reproducibility and minimize technical variation. These assessment procedures were performed according to the predefined study protocol. Clinical improvement was further evaluated using the Hamilton–Norwood Classification, the most widely accepted grading system for male androgenetic alopecia, which categorizes disease severity into seven progressive stages according to the degree of frontal hairline recession and vertex hair loss. Lower stages indicate milder disease, whereas higher stages represent advanced androgenetic alopecia [10,11]. In addition, disease severity was assessed using the Basic and Specific (BASP) Classification, a standardized classification system that combines four basic patterns (L, M, C, and U) describing the anterior hairline with two specific patterns (F and V) representing frontal and vertex involvement. Owing to its high reproducibility and applicability across different populations, the BASP classification has become a reliable tool for monitoring disease progression and treatment response in clinical studies [12]. Subjective treatment efficacy was assessed using the Hair Growth Self-Assessment Scale, a validated patient-reported outcome measure commonly used in clinical studies of androgenetic alopecia. At each follow-up visit, participants

compared their current hair growth with baseline and rated the degree of improvement using a five-point ordinal scale ranging from 0 (no improvement) to 4 (76–100% improvement), with higher scores indicating greater perceived hair regrowth [13]. Overall patient satisfaction was evaluated at the end of the six-month follow-up using a standardized four-point satisfaction scale, ranging from 0 (very dissatisfied) to 3 (very satisfied). This scale assessed the participants' overall perception of treatment efficacy, cosmetic improvement, and satisfaction with the therapeutic outcome. Throughout the study, treatment safety and tolerability were carefully monitored. At each follow-up visit, participants were questioned regarding the occurrence of any local or systemic adverse events, and a targeted clinical examination was performed. Local adverse effects, including scalp erythema, pain, burning sensation, pruritus, edema, scaling, folliculitis, and infection, were documented. In patients receiving oral dutasteride, systemic adverse events such as decreased libido, erectile dysfunction, ejaculatory disorders, and other drug-related complications were specifically evaluated. All adverse events were recorded according to their nature, severity, duration, and outcome, and proper management was provided whenever necessary, as mentioned in the case sheet records.

Ethical considerations

The study protocol adhered to the principles of the Declaration of Helsinki for research involving human participants, and written informed consent was obtained from all enrolled patients before participation. A gatekeeper's approval letter was obtained from the recruitment site.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Data were examined for

completeness and normality before analysis. Continuous variables were expressed as mean±SD for normally distributed data or median with interquartile range (IQR) when appropriate, whereas categorical variables were presented as frequencies and percentages. Comparisons of baseline demographic and clinical characteristics among the three treatment groups were performed using one-way analysis of variance (ANOVA) for normally distributed continuous variables or the Kruskal–Walli's test for non-normally distributed variables. Categorical variables were compared using the chi-square test or Fisher's exact test whenever appropriate. Changes within each treatment group over time were evaluated using paired statistical tests according to data distribution, whereas repeated-measures analyses were used to assess treatment effects across different follow-up visits. Post hoc multiple comparison tests with appropriate adjustment for multiple testing were applied whenever significant overall differences were identified. All statistical tests were two-tailed, and a *P* value of <0.05 was considered statistically significant.

RESULTS

A total number of 90 patients were included in our study; the three groups were comparable in terms of age, smoking status, educational level, and family history of androgenetic alopecia, with no statistically significant differences among the groups (*p*= 0.09, 0.80, and 0.70, respectively). The mean age was 38.5 ± 10.5 years in Group I, 36.0 ± 9.0 years in Group II, and 40.0 ± 4.0 years in Group III. Similarly, the distribution of smoking status and educational attainment was comparable across the three groups. The duration of androgenetic alopecia differed significantly among the study groups (*p*= 0.001), with Group III demonstrating the longest disease duration (8.0 ± 1.8 years), followed by Group I (6.0 ± 2.0 years) and Group II (5.5 ± 2.2 years). Furthermore, all enrolled patients (100%) in the three groups reported a positive family history of androgenetic alopecia (Table 1).

Table 1: Demographic and clinical characteristics of the patients studied (n=30 in each group)

Variable	Group I	Group II	Group III	<i>p</i> -value
<i>Age (year)</i>				
mean±SD	38.5±10.5	36±9	40±4	0.09 ^a
Range	18-60	19-58	31-55	
<i>AGA duration (year)</i>				
mean±SD	6±2	5.5±2.2	8±1.8	0.001 ^a
Range	2-11	3-12	5-14	
<i>Smoking status</i>				
Nonsmoker	4(13.3)	6(20)	5(16.7)	0.8 ^b
Current smoker	9(30)	8(26.7)	6(20)	
Former smoker	17(56.7)	16(53.3)	19(63.3)	
<i>Education level</i>				
Primary school	5(16.7)	4(13.3)	4(13.3)	0.7 ^b
High school	12(40)	13(43.3)	13(43.3)	
Bachelor's	11(36.7)	13(43.3)	10(33.3)	
Master's	2(6.7)	0(0.0)	2(6.7)	
Ph.D.	0(0.0)	0(0.0)	1(3.3)	
<i>Family history of AGA</i>				
Yes	30(100)	30(100)	30(100)	-
No	0(0.0)	0(0.0)	0(0.0)	

Values are presented as frequency, percentage, and mean±SD. ^a One Way ANOVA. ^b Chi square test.

According to the vertex hair density, statistically significant differences were observed at baseline and after 3 months of treatment (*p*= 0.001 for both),

whereas no significant difference was detected at 6 months (*p*= 0.60). At baseline, Group I demonstrated the highest median hair density, followed by Group II

and Group III. A similar pattern was observed at the 3-month evaluation. By the sixth month, hair density values became comparable among the three groups, with no significant intergroup differences. Intragroup changes over time showed a statistically significant increase in vertex hair density in all three groups throughout the study period. The absolute change in hair density differed significantly among the groups

($p=0.001$), with the greatest increase recorded in Group III, followed by Group II, while Group I showed the smallest improvement. Likewise, the percentage improvement in hair density demonstrated a statistically significant difference between the three groups ($p=0.001$), with Group III achieving the highest percentage improvement, followed by Group II and Group I (Table 2).

Table 2: Comparison of Vertex hair density among the groups studied (n=30 in each group)

Hair Density (hairs/cm ²)	Group I	Group II	Group III	p-value ^a	p-value ^b
Baseline	84.5(31)	57.5(25)	45.5(6)	0.001	$p1=0.001$ $p2=0.001$ $p3=0.002$
Month 3	84.5(33)	69.5(26)	49.5(13)	0.001	$p1=0.001$ $p2=0.001$ $p3=0.003$
Month 6	90(50)	93.5(25)	85.5(13)	0.6	$p1=0.6$ $p2=0.7$ $p3=0.27$
p-value ^c	0.007	0.001	0.001	-	-
Change	4.5 (-2.5-15.2)	31 (22-37)	41 (38-47)	0.001	$p1=0.001$ $p2=0.001$ $p3=0.001$
Improvement (%)	2.9 (-2.9-13.6)	45 (27-65)	92 (88-95)	0.001	$p1=0.001$ $p2=0.001$ $p3=0.001$

^a Kruskal Wallis test; ^b Mann-Whitney u test; ^c Friedman test.

Regarding the hair thickness, statistically significant differences were observed at baseline and at the 3-month follow-up ($p=0.001$ for both), whereas no statistically significant difference was detected at the 6-month evaluation ($p=0.40$). At baseline, Group I exhibited the highest median hair thickness, followed by Group II and Group III. A similar distribution was maintained at the 3-month mark. By month 6, hair thickness values were comparable among the three groups without significant intergroup differences. With respect to changes in hair thickness over the study

period, a statistically significant improvement was observed in Groups II and III ($p=0.001$ for both), whereas the change in Group I did not reach statistical significance ($p=0.60$). The magnitude of change differed significantly among the three groups ($p=0.001$), with Group III demonstrating the greatest increase in hair thickness, followed by Group II, while Group I showed the least improvement. Similarly, the percentage improvement in hair thickness was significantly higher in Group III compared with Groups II and I ($p=0.001$) (Table 3).

Table 3: Comparison of hair thickness among The groups studied (n=30 in each group)

Hair Thickness (μm)	Group I	Group II	Group III	p-value	p-value
Baseline	56(8)	44(11)	32(5)	0.001	$p1=0.001$ $p2=0.001$ $p3=0.002$
Month 3	59(7)	47(12)	42(9)	0.001	$p1=0.001$ $p2=0.001$ $p3=0.003$
Month 6	61(10)	60(14)	61(7)	0.4	$p1=0.42$ $p2=0.61$ $p3=0.24$
p-value	0.6	0.001	0.001		
Change	2.5 (-2-11)	13 (8-18)	29 (29-32)	0.001	$p1=0.001$ $p2=0.001$ $p3=0.001$
Improvement (%)	3.5 (-3-19)	26 (15-45)	96 (91-100)	0.001	$p1=0.001$ $p2=0.001$ $p3=0.001$

As regards hair growth self-assessment at the 6-month follow-up, a statistically significant difference was observed among the three study groups ($P=0.001$). Half of the patients in Group I (50%) reported no improvement, while 43.3% experienced 1–25% regrowth and only 6.7% reported 26–50% regrowth. In Group II, most patients reported moderate to marked improvement, with 33.3% achieving 26–50%

regrowth, 40% achieving 51–75% regrowth, and 6.7% reporting 76–100% regrowth, whereas 20% reported no improvement. In contrast, all patients (100%) in Group III reported the highest level of improvement, corresponding to 76–100% hair regrowth. In terms of patient satisfaction, statistically significant differences were also identified among the three groups ($p=0.001$). In Group I, 50% of patients reported a satisfaction

score of zero, and the remaining 50% reported a score of 1. Eighty percent of the patients in Group II were satisfied with a score of 2, while twenty percent were not satisfied at all. Conversely, all patients (100%) in

Group III achieved the highest satisfaction score of 3.0, indicating significantly greater patient satisfaction compared with the other treatment groups (Table 4).

Table 4: Hair growth self-assessment and patients' satisfaction at month 6 (n=30 in each group)

Variables	Group I	Group II	Group III	p-value
Self-Assessment Score				
No improvement	15(50)	6(20)	0(0.0)	0.001
1–25%	13(43.3)	0(0.0)	0(0.0)	
26–50%	2(6.7)	10(33.3)	0(0.0)	
51–75%	0 (0.0)	12(40)	0(0.0)	
76–100%	0(0.0)	2(6.7)	30(100)	
Satisfaction Score				
0	15(50)	6(20)	0(0.0)	0.001
1	15(50)	0(0.0)	0(0.0)	
2	0(0.0)	24(80)	0(0.0)	
3	0(0.0)	0(0.0)	30(100)	

Values are presented as frequency and percentage.

Concerning the Norwood–Hamilton classification, statistically significant differences were observed among the three study groups at both baseline and the 6-month follow-up ($p=0.001$ for both). At baseline, Group I patients were classified as grade IV, whereas Group II patients were equally distributed between grades V and VI, and all patients in Group III were classified as grade VII. At the end of the study, all patients in Group III had improved to grade III, while Group II patients were predominantly classified as grades III and IV. In Group I, patients were equally distributed between grades III and IV at the 6-month evaluation. The intragroup analysis revealed

significant improvement in the Norwood–Hamilton grade in all three treatment groups over the study period ($p=0.001$ for each group). The improvement score differed significantly among the groups ($p=0.001$), with Group III demonstrating the greatest improvement, followed by Group II and Group I. Regarding the degree of improvement, half of the patients in Group I improved by one grade, while the remaining half showed no improvement. In Group II, 80% of patients improved by two grades and 20% showed no improvement. All patients in Group III achieved an improvement of four grades (Table 5).

Table 5: Norwood-Hamilton scale of the patients studied (n=30 in each group)

Variable Median (IQR)/ n(%)	Group I	Group II	Group III	p-value
Baseline grade	4(0.0)	5.5(1)	7(0)	0.001
Month 6 grade	3.5(1)	4(1)	3(0)	0.001
Improvement score	0.5(1)	2(0)	4(0)	0.001
p-value	0.001	0.001	0.001	
<i>Improvement</i>				
No improvement	15(50)	6(20)	0(0.0)	0.001
One level	15(50)	0(0.0)	0 (0.0)	
Two level	0(0.0)	24(80)	0(0.0)	
Four level	0(0.0)	0(0.0)	30(100)	

As regards the BASP classification, statistically significant differences were observed among the three study groups in the distribution of the basic pattern at baseline and at the 6-month follow-up ($p=0.001$ for both). At baseline, all patients in Group I were classified as M2/C2, while Group II patients were equally distributed between M3/C3 and U1 patterns. All patients in Group III were classified as U2. At the end of the study, Group III patients had all improved to the M1/C1 pattern, whereas Group II patients were predominantly classified as M1/C1 and M2/C2. In Group I, patients were equally distributed between M1/C1 and M2/C2 patterns. The BASP-specific pattern showed statistically significant differences among the three groups at both baseline and the 6-month assessment ($P=0.001$ for both). Initially, all patients in Group I exhibited the F1/V2 pattern, whereas Group II patients were equally distributed between F2/V3 and F2 patterns, and all patients in Group III presented with the F3 pattern. After six

months of treatment, all patients in Group III had improved to the V1 pattern, while most patients in Groups I and II demonstrated V1 or F1/V2 patterns, with only a small proportion of Group II remaining in the F2/V3 or F2 categories. The overall BASP classification showed a statistically significant difference among the three treatment groups at baseline and after 6 months of follow-up ($P=0.001$ for both). At baseline, Group I patients were uniformly classified as M2/C2–F1/V2, Group II patients were divided equally between M3/C3–F2/V3 and U1–F2, while all Group III patients were classified as U2–F3. At the 6-month evaluation, all patients in Group III had improved to the M1/C1–V1 classification. In Group II, most patients were classified as M1/C1–V1 or M2/C2–F1/V2, whereas Group I patients were equally distributed between M1/C1–V1 and M2/C2–F1/V2 (Table 6). The significant baseline difference in disease duration among the groups may represent a possible confounding factor.

Table 6: BASP Classification of the patients studied (n=30 in each group)

Pattern	Group I	Group II	Group III	p-value
<i>BASP basic pattern at baseline</i>				
M1 or C1	0(0.0)	0(0.0)	0(0.0)	0.001
M2 or C2	30(100)	0(0.0)	0(0.0)	
M3 or C3	0(0.0)	15(50)	0(0.0)	
U1	0(0.0)	15(50)	0(0.0)	
U2	0(0.0)	0(0.0)	30(100)	
<i>BASP basic pattern at month 6</i>				
M1 or C1	15(50)	13(43.3)	30(100)	0.001
M2 or C2	15(50)	11(36.7)	0(0.0)	
M3 or C3	0(0.0)	2(6.7)	0(0.0)	
U1	0(0.0)	4(13.3)	0(0.0)	
U2	0(0.0)	0(0.0)	0(0.0)	
<i>BASP specific pattern at baseline</i>				
V1	0(0.0)	0(0.0)	0(0.0)	0.001
F1 / V2	30(100)	0(0.0)	0(0.0)	
F2 / V3	0(0.0)	15(50)	0(0.0)	
F2	0(0.0)	15(50)	0(0.0)	
F3	0(0.0)	0(0.0)	30(100)	
<i>BASP specific pattern at month 6</i>				
V1	15(50)	13(43.3)	30(100)	0.001
F1 / V2	15(50)	11(36.7)	0(0.0)	
F2 / V3	0(0.0)	2(6.7)	0(0.0)	
F2	0(0.0)	4(13.3)	0(0.0)	
F3	0(0.0)	0(0.0)	0(0.0)	
<i>BASP Classification final type at baseline</i>				
M1 or C1 / V1	0(0.0)	0(0.0)	0(0.0)	0.001
M2 or C2 / F1 V2	30(100)	0(0.0)	0(0.0)	
M3 or C3 / F2 V3	0(0.0)	15(50)	0(0.0)	
U1 / F2	0(0.0)	15(50)	0(0.0)	
U2 / F3	0(0.0)	0(0.0)	30(100)	
<i>BASP Classification final type month 6</i>				
M1 or C1 / V1	15(50)	13(43.3)	30(100)	0.001
M2 or C2 / F1 V2	15(50)	11(36.7)	0(0.0)	
M3 or C3 / F2 V3	0(0.0)	2(6.7)	0(0.0)	
U1 / F2	0(0.0)	4(13.3)	0(0.0)	
U2 / F3	0(0.0)	0(0.0)	0(0.0)	

Values are presented as frequency and percentage.

DISCUSSION

Recently, increasing interest has been directed toward combination therapeutic strategies of AGA, particularly the addition of microneedling and oral dutasteride, to enhance hair regrowth through complementary mechanisms, including stimulation of follicular regeneration, improved transdermal drug delivery, and inhibition of androgen-mediated follicular miniaturization [14]. Recent clinical studies and systematic reviews have demonstrated that combination therapies generally provide superior efficacy compared with monotherapy; however, the optimal treatment approach remains an area of ongoing investigation. In terms of vertex hair density, all treatment groups showed significant improvement after 6 months; however, the greatest increase was observed in the group treated with topical minoxidil, weekly microneedling, and oral dutasteride, followed by the minoxidil plus microneedling group, whereas topical minoxidil alone achieved the least improvement. The enhanced efficacy of combination therapy may be explained by the complementary mechanisms of the three interventions. Minoxidil promotes anagen prolongation and follicular proliferation; microneedling stimulates growth factor release and enhances transdermal drug delivery, while dutasteride suppresses androgen-mediated follicular miniaturization through dual inhibition of type I and

type II 5 α -reductase enzymes, resulting in greater improvement in hair density [15]. Our findings are consistent with [9,16], who reported that microneedling combined with topical therapy significantly improves hair density compared with minoxidil alone. Similar results were demonstrated by [13,17], who observed greater increases in hair count and dermoscopic hair density with combination therapy. Furthermore, the superior response observed in the triple-therapy group is supported by [15,18], who demonstrated that oral dutasteride provides greater improvement in hair count than conventional medical therapies. Variations in microneedling protocols, treatment duration, patient characteristics, and outcome assessment methods [19] may account for the minor differences among published studies. Nevertheless, the present findings further support the superiority of multimodal therapy for improving hair density in male androgenetic alopecia. Regarding hair thickness, the present study demonstrated significant improvement in all treatment groups after 6 months, with the greatest increase observed in patients treated with topical minoxidil, weekly microneedling, and oral dutasteride, followed by those receiving minoxidil plus microneedling, while topical minoxidil alone produced the smallest improvement. Although significant differences were observed among the groups during the early treatment period, comparable hair thickness values at the end of follow-up indicate that all treatment modalities were effective, with combination

therapy providing the greatest clinical benefit. Minoxidil promotes dermal papilla cell activity and prolongs the anagen phase, microneedling enhances follicular regeneration through growth factor release and improves transdermal delivery of topical minoxidil, whereas dutasteride suppresses androgen-mediated follicular miniaturization through dual inhibition of type I and type II 5 α -reductase enzymes [13,20,21]. Our findings are consistent with those of [16,22], who demonstrated that microneedling combined with topical therapy significantly improves hair thickness compared with minoxidil monotherapy. Similar observations were reported by [13], who showed progressive conversion of miniaturized hairs into thicker terminal hairs following combined microneedling and topical minoxidil. Likewise [23], it found superior objective hair growth with dutasteride-containing combination therapy compared with topical minoxidil alone. These findings are further supported by the network meta-analysis of [24] and the meta-analysis by [25], both of which concluded that combination therapy consistently provides greater improvement in objective hair growth parameters, including hair thickness, than monotherapy. With respect to hair growth self-assessment and patient satisfaction, significant differences were observed among the three treatment groups after 6 months. Patients receiving topical minoxidil alone reported the lowest perceived improvement and satisfaction, whereas the addition of microneedling significantly enhanced both outcomes. The highest self-assessment and satisfaction scores were achieved in patients receiving the triple combination of topical minoxidil, weekly microneedling, and oral dutasteride, indicating that subjective patient perception closely paralleled the objective improvements in hair density and hair thickness. These findings are consistent with [13], who reported significantly greater patient satisfaction and perceived hair regrowth with microneedling plus topical minoxidil than with minoxidil alone. Likewise, the available evidence regarding combination therapy significantly improved both objective efficacy and patient satisfaction compared with monotherapy, supporting the present findings [16]. Regarding the Norwood–Hamilton classification, all treatment groups demonstrated significant improvement in clinical disease severity after 6 months, although the degree of improvement varied considerably among the treatment modalities. Patients who received topical minoxidil, weekly microneedling, and oral dutasteride experienced the most significant decrease in disease severity. The second most significant decrease was seen in the minoxidil plus microneedling group, and the least significant decrease was seen in the topical minoxidil group. These findings indicate that multimodal therapy is more effective in reversing the clinical manifestations of androgenetic alopecia than topical monotherapy. The greater improvement observed with combination therapy is biologically plausible, as minoxidil stimulates follicular activity, microneedling enhances follicular regeneration and drug delivery, and dutasteride suppresses androgen-mediated follicular miniaturization, collectively

resulting in improved scalp coverage and clinical staging. Our findings agree with Gupta and Charrette [26], who identified oral dutasteride as the most effective medical therapy for achieving global clinical improvement in androgenetic alopecia. Similarly, it has been reported that adding microneedling to topical therapy significantly enhanced investigator-assessed clinical outcomes compared with topical treatment alone, consistent with the greater improvement in Norwood–Hamilton classification observed in the present study [16]. With respect to the BASP classification, all treatment groups demonstrated significant improvement after 6 months; however, the magnitude of improvement differed among the treatment modalities. The greatest reduction in disease severity was observed in patients treated with topical minoxidil, weekly microneedling, and oral dutasteride, followed by the minoxidil plus microneedling group, whereas topical minoxidil alone produced the least improvement. These findings indicate that multimodal therapy provides superior clinical improvement compared with monotherapy. The enhanced improvement in BASP classification is likely attributable to the complementary mechanisms of the investigated treatments. Minoxidil promotes anagen prolongation and follicular proliferation, microneedling enhances follicular regeneration and transdermal drug delivery, and dutasteride suppresses androgen-mediated follicular miniaturization through dual inhibition of type I and type II 5 α -reductase enzymes, resulting in improved frontal hairline and vertex scalp coverage. Our findings are consistent with [12], who introduced the BASP classification as a reliable tool for monitoring disease severity and treatment response in pattern hair loss. Similarly, other investigators reported that combination therapies targeting multiple pathogenic mechanisms produce greater overall clinical improvement than monotherapy [27]. In addition, it has been demonstrated that dutasteride-based therapy provides superior global clinical improvement compared with topical therapy alone [26], supporting the marked reduction in BASP severity observed in the triple-therapy group. Differences in disease duration at baseline ($p=0.001$) could have affected the observed therapeutic responses and should therefore be considered as a confounding factor. Although follicular miniaturization in AGA may be partially reversible and treatment may promote anagen activity and hair regrowth, the marked improvement from Norwood grade VII to grade III within six months represents an unusually large clinical response and even its positive result; however, the small sample size limits its generalizability, and we think that confirmation in larger studies is warranted. The marked clinical improvement from Norwood grade VII to grade III within six months may potentially be explained by several biological mechanisms. In advanced AGA, a proportion of follicles may remain miniaturized rather than being completely destroyed, potentially allowing partial restoration of follicular activity when responsive follicles are exposed to effective therapy. Treatment-induced prolongation of the anagen phase and

recruitment of follicles from telogen-kenogen into anagen may contribute to increased hair density and shaft diameter. In addition, microneedling-induced wound-healing responses may promote regenerative signaling and angiogenic pathways, including VEGF-related signaling, potentially enhancing the perifollicular microenvironment and supporting hair regrowth.

Study Limitations

Several limitations should be acknowledged. The relatively small sample size design may limit the generalizability of the results. In addition, the six-month follow-up was insufficient to evaluate the long-term durability of treatment response, and dermoscopic assessment was not supplemented by advanced quantitative techniques such as phototrichogram analysis. Furthermore, quality-of-life outcomes were not assessed using validated instruments such as the Dermatology Life Quality Index or Hairdex. Despite these limitations, the consistent improvement across objective and subjective outcomes supports the superiority of combining topical minoxidil, weekly microneedling, and oral dutasteride over topical minoxidil monotherapy for the treatment of male androgenetic alopecia. Given the retrospective observational design, the primary analyses were intended to provide unadjusted comparisons of the observed treatment groups rather than adjusted estimates of treatment effects. We have therefore retained the original analysis and explicitly acknowledged disease duration as a potential confounding factor in our study. Accordingly, the between-group findings should be interpreted as unadjusted associations. Our marked improvement may potentially be related to the persistence of viable miniaturized follicles and treatment-induced stimulation of follicular activity, including prolongation of the anagen phase and activation of regenerative signaling pathways, but our positive findings are limited to the small sample size.

Conclusion

All treatment modalities significantly improved androgenetic alopecia after six months. However, the combination of topical 5% minoxidil, weekly microneedling, and oral dutasteride achieved the greatest improvements in hair density, hair thickness, clinical severity, and patient satisfaction. These findings suggest that multimodal therapy is a more effective treatment option than topical minoxidil monotherapy for male androgenetic alopecia.

Conflict of interests

The authors declared no conflict of interest.

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Data sharing statement

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

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