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ORIGINAL RESEARCH

EFFICACY OF LOW DOSE ORAL MINOXIDIL (1.25 MG) AS MONOTHERAPY IN MALE ANDROGENETIC ALOPECIA

Krishnakant Girish Mundhada[1*], **Sanjay Nathmal Agrawal**[2], **Ganesh Ranchoddas Mundhada**[3], **Hitesh Sureshkumar Khatri**[4]

[1] *Junior Resident, Department of Dermatology, Dr P.D.M.M.C Amravati, Maharashtra*
[2] *Professor and HOD, Department of Dermatology, Dr P.D.M.M.C Amravati, Maharashtra*
[3] *Associate Professor, Department of Dermatology, Dr P.D.M.M.C Amravati, Maharashtra*
[4] *Assistant Professor, Department of Dermatology, Dr P.D.M.M.C Amravati, Maharashtra*

*Correspondence: Krishnakant Girish Mundhada <krishna0497mundhada@gmail.com >

ABSTRACT

Androgenetic alopecia (AGA) is the most prevalent form of progressive hair loss in men, characterised by androgen-mediated follicular miniaturisation and significant psychosocial impact. Topical minoxidil, the established first-line therapy, is limited by scalp irritation, poor compliance, and variable absorption. Low-dose oral minoxidil (LDOM) has emerged as a promising alternative formulation, but evidence on the optimal dose and its short-term clinical profile in male AGA remains limited. A prospective, open-label, single-arm observational study was conducted at a tertiary care dermatology unit. Twenty-five adult male patients aged 20–50 years with AGA of Hamilton–Norwood grade ≥ III were enrolled by convenience sampling and treated with oral minoxidil 1.25 mg once daily for 24 weeks. Clinical assessments were performed at baseline, 12 weeks, and 24 weeks, and included global photographic assessment (GPA), trichoscopy, the Global Aesthetic Improvement Scale (GAIS), and a patient satisfaction questionnaire. Adverse events were recorded throughout the study period. At 24 weeks, 80% of patients demonstrated measurable improvement on GPA (mild: 30%, moderate: 40%, marked: 10%). Trichoscopic improvement was observed in 76% of patients, including increased hair density in 70%, reduced hair shaft diameter variability in 60%, and a decrease in the proportion of miniaturised hairs in 50%. On the GAIS, 70% of patients were rated as aesthetically improved. Overall patient satisfaction was 60% at 24 weeks. Adverse events were mild and transient; no cardiovascular or serious adverse effects were recorded. Oral minoxidil 1.25 mg once daily is an effective and well-tolerated monotherapy for male AGA, with progressive clinical and trichoscopic improvement extending through 24 weeks of treatment. These findings support the use of LDOM as a viable oral alternative, particularly for patients intolerant of or non-adherent to topical therapies.

Keywords: Androgenetic alopecia; Low-dose oral minoxidil; Trichoscopy; Hair loss; Global Aesthetic Improvement Scale; Hair density

INTRODUCTION

Androgenetic alopecia (AGA), encompassing both male and female pattern hair loss, is the most common form of progressive alopecia, affecting individuals of all ethnic backgrounds.[1] In men, the condition typically manifests as a predictable bitemporal recession and vertex thinning, classified by the Hamilton–Norwood grading system [1]. Epidemiologically, AGA affects approximately 50% of men by the fifth decade of life and becomes increasingly prevalent with advancing age.[1,2] Beyond its physiological significance, hair loss carries considerable psychosocial consequences: affected individuals frequently report diminished self-esteem, reduced quality of life, and heightened vulnerability to anxiety and depression [2].

The pathogenesis of AGA is mediated through the conversion of testosterone to dihydrotestosterone (DHT) by 5α-reductase enzymes within genetically susceptible hair follicles. DHT binds to androgen receptors, triggering progressive follicular miniaturisation a process characterised by shortening of the anagen phase, reduction in follicular size, and

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replacement of terminal hairs by fine, depigmented vellus-like hairs [1,3]. The result is the clinically recognisable pattern of thinning and recession that defines AGA.

Topical minoxidil 2% and 5% solutions remain the primary FDA-approved topical therapy for AGA in men, and their efficacy in promoting hair regrowth and slowing progression is well established [3]. However, topical minoxidil is associated with practical limitations that constrain long-term adherence: scalp irritation, contact dermatitis attributable to propylene glycol in solution formulations, unsatisfactory cosmetic feel, application-site reactions, and the perception that twice-daily application is burdensome [3]. Oral finasteride, a 5α-reductase inhibitor, represents the other established systemic option in male AGA but is contraindicated in men with sexual dysfunction concerns, and is not suitable for women of child-bearing potential [3].

These limitations have driven renewed interest in oral minoxidil, which was originally introduced in the 1970s as an antihypertensive agent and whose serendipitous hair growth-promoting effects were noted during systemic use [4]. At the antihypertensive doses (10–40 mg/day) required for blood pressure control, clinically significant adverse effects including pericardial effusion, tachycardia, and fluid retention precluded its routine use in dermatology [4]. The concept of low-dose oral minoxidil (LDOM), defined generally as doses ≤ 5 mg/day, substantially mitigates this risk while retaining therapeutically meaningful hair growth stimulation.

The pharmacological basis for minoxidil's hair growth effects involves multiple mechanisms [3,5]. Minoxidil is a prodrug that undergoes hepatic and follicular sulphation by sulphotransferase enzymes to yield its active metabolite, minoxidil sulphate. This metabolite acts as a potassium channel opener at the follicular level, inducing cell membrane hyperpolarisation and local vasodilation. Downstream effects include increased expression of vascular endothelial growth factor (VEGF), hepatocyte growth factor (HGF), and prostaglandin E_2, as well as activation of the Wnt/β-catenin signalling pathway all of which promote follicular enlargement, prolong the anagen phase, and stimulate new hair growth [3,5].

A growing body of evidence supports the safety and efficacy of LDOM across a range of alopecia subtypes. A landmark multicentre safety study of 1,404 patients by Vano -Galvan et al. confirmed that LDOM has an acceptable adverse event profile, with hypertrichosis and lightheadedness as the most commonly reported effects and a drug discontinuation rate of only 1.7% [6]. Jimenez-Cauhe et al. documented clinical effectiveness in male AGA using doses of 1.25–5 mg,[7] and Panchaprateep and Lueangarun demonstrated significant improvements in hair count, hair diameter, and global photographic assessment with oral minoxidil 5 mg in a prospective open-label study [8]. Jha et al. specifically evaluated oral minoxidil at the 1.25 mg dose and reported early favourable responses in male AGA, although longer-term and larger-scale data at this precise dose remain limited [9].

Despite these emerging data, evidence specifically evaluating the 1.25 mg dose of oral minoxidil as monotherapy in male AGA over a structured 24-week period using multidimensional outcome measures including trichoscopy, photographic assessment, physician-rated and patient-reported outcomes remains limited in the Indian clinical context. The present study was therefore designed to address this gap, examining the clinical response to LDOM 1.25 mg/day in male patients with Hamilton–Norwood grade III AGA and above at a tertiary care dermatology unit.

MATERIALS AND METHODS

Study Design, Setting, and Ethical Approval: This was a prospective, open-label, single-arm observational study conducted at the dermatology outpatient department of a tertiary care hospital over a 24-week treatment period. The study protocol was approved by the Institutional Ethics Committee (IEC Reference:), and all participants provided written informed consent prior to enrolment.

Study Population: Adult male patients aged 20–50 years presenting to the dermatology outpatient department with clinically confirmed AGA of Hamilton–Norwood grade III or above were considered for enrolment. Patients were recruited by convenience sampling from among those attending the outpatient clinic during the study period.

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Inclusion criteria: (i) male sex; (ii) age 20–50 years; (iii) clinical diagnosis of AGA with Hamilton–Norwood classification grade ≥ III, confirmed by clinical examination and trichoscopy.

Exclusion criteria: (i) concurrent use of any topical or systemic treatment for AGA, including topical minoxidil, finasteride, or dutasteride; (ii) known or clinically documented hypertension or cardiovascular disease; (iii) known bleeding diathesis or coagulopathy; (iv) renal or hepatic impairment; and (vi) unwillingness to attend scheduled follow-up visits or provide informed consent.

Intervention: Eligible participants received oral minoxidil 1.25 mg once daily as monotherapy, dispensed at the baseline visit and at the 12-week follow-up visit. No adjunctive hair growth therapies were permitted during the study period. Participants were counselled on the expected timeline of response, the possibility of an initial telogen shed, and the importance of medication adherence. Clinic visits were scheduled at baseline (week 0), week 12, and week 24.

Outcome Measures: Clinical efficacy was assessed at each scheduled visit using four complementary outcome measures:

Global Photographic Assessment (GPA). Standardised scalp photographs were obtained at baseline, 12 weeks, and 24 weeks under controlled lighting conditions, using a consistent camera-to-scalp distance and patient positioning. Photographs were evaluated independently by two blinded assessors and categorised on a four-point scale: Grade 0 (no improvement), Grade 1 (mild improvement: visible thinning improved but hair loss remains apparent), Grade 2 (moderate improvement: clearly perceptible density gain), and Grade 3 (marked improvement: substantial regrowth with appreciable cosmetic benefit) [3].

Trichoscopy: Trichoscopic evaluation was performed at each visit using a handheld dermoscope (×20 magnification) at a standardised scalp region. Parameters assessed included: hair shaft diameter variability (ratio of thin to thick hairs > 20% classified as significant variability), proportion of miniaturised hairs (< 0.03 mm diameter), perifollicular pigmentation, and hair density expressed as follicular units per cm^2 [10].

Global Aesthetic Improvement Scale (GAIS): At 12 and 24 weeks, the treating physician rated overall aesthetic improvement on a five-category scale: very much improved, much improved, improved, no change, and worse [11].

Patient Satisfaction Score: Patient-reported satisfaction was assessed at 12 and 24 weeks using a four-point Likert scale: very satisfied, satisfied, slightly satisfied, and not satisfied [12].

Safety Assessment: Adverse events were systematically recorded at each visit. Cardiovascular safety was monitored by measuring blood pressure and pulse rate at baseline, 12 weeks, and 24 weeks. Patients were specifically asked about symptoms of fluid retention (oedema), lightheadedness, palpitations, hypertrichosis, and headache at each encounter. Any adverse event requiring drug discontinuation was documented [13].

Statistical Analysis: Data were summarised using descriptive statistics. Categorical variables are presented as frequencies and percentages. Continuous variables are expressed as means ± standard deviations (SD). Changes in outcome proportions between baseline, 12 weeks, and 24 weeks were assessed using the McNemar test for paired proportions. A two-tailed p-value < 0.05 was considered statistically significant.

RESULTS

Baseline Characteristics: Twenty-five male patients completed the 24-week study protocol and were included in the analysis. The mean age was 32.6 ± 7.4 years (range: 21–49 years). On Hamilton–Norwood grading, the majority of participants had grade III (n = 9; 36%) or grade IV (n = 8; 32%) alopecia, with grades V and above comprising the remaining 32% (n = 8). No patient had a previous history of hair loss treatment. Baseline blood pressure and heart rate were within normal limits for all participants.

Global Photographic Assessment: Clinical improvement on GPA was progressive across the study period (Table 2). At 12 weeks, 64% of participants demonstrated some degree of improvement (mild: 28%, moderate: 32%, marked: 4%), with 36% showing no improvement. By 24 weeks, the proportion demonstrating improvement increased to 80% (mild: 30%,

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moderate: 40%, marked: 10%), and 20% showed no improvement. No participant was rated as worse at either time point. The improvement in the proportion of responders between 12 and 24 weeks was statistically significant (p = 0.031).

Global Photographic Assessment: Clinical improvement on GPA was progressive across the study period (Table 2). At 12 weeks, 64% of participants demonstrated some degree of improvement (mild: 28%, moderate: 32%, marked: 4%), with 36% showing no improvement. By 24 weeks, the proportion demonstrating improvement increased to 80% (mild: 30%, moderate: 40%, marked: 10%), and 20% showed no improvement. No participant was rated as worse at either time point. The improvement in the proportion of responders between 12 and 24 weeks was statistically significant (p = 0.031).

Trichoscopic Findings: Trichoscopic evaluation demonstrated progressive improvement in multiple follicular parameters from baseline to 24 weeks (Table 3). The most frequently observed improvements were an increase in hair density (70% of participants), a reduction in hair shaft diameter variability (60%), and a decrease in the proportion of miniaturised hairs (50%). Improvement in perifollicular pigmentation, reflecting enhanced follicular health and anagen activity, was observed in 40% of participants. The mean hair density increased from 62.4 ± 8.3 follicular units/cm² at baseline to 74.1 ± 9.6 follicular units/cm² at 24 weeks (mean increase: 11.7 FU/cm²; p = 0.003). These findings collectively indicate reversal of follicular miniaturisation and improved hair cycling.

Global Aesthetic Improvement Scale (GAIS): Physician-rated global aesthetic improvement on the GAIS showed consistent gains between 12 and 24 weeks (Table 4). At 12 weeks, 68% of participants were rated as improved or better (much improved: 12%, improved: 56%); 32% showed no change. By 24 weeks, 70% were rated as improved or better, with an increase in the proportion rated as much improved (30% vs. 12% at 12 weeks). No participant was rated as worse at either time point.

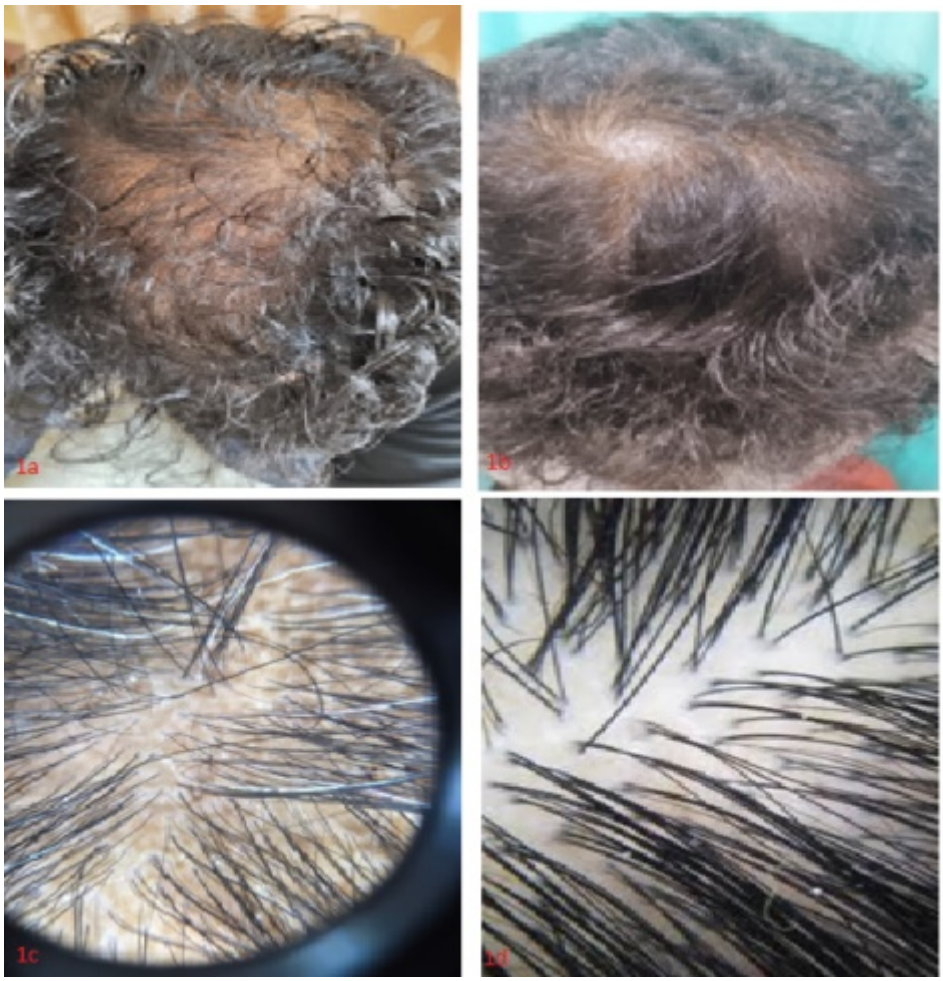

Figure 1. *Photograph of pre and post (1a Before treatment, 1b: Ater 24 weeks follow-up, 1c: Trichoscopuc photo before, 1d: After treatment)*

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Table 1. Baseline Characteristics of Study Participants (n = 25)

Characteristic	Value
Age, mean ± SD (years)	32.6 ± 7.4
Age range (years)	21–49
Hamilton–Norwood Grade, n (%)	
Grade III	9 (36.0%)
Grade IV	8 (32.0%)
Grade V	5 (20.0%)
Grade VI	2 (8.0%)
Grade VII	1 (4.0%)
Mean duration of hair loss (years)	4.2 ± 2.8
Mean baseline hair density (FU/cm²)	62.4 ± 8.3
Prior AGA treatment, n (%)	0 (0%)

Note: SD, standard deviation; FU, follicular units; AGA, androgenetic alopecia.

Table 2. Global Photographic Assessment (GPA) Results at 12 and 24 Weeks (n = 25)

GPA Grade	12 Weeks, n (%)	24 Weeks, n (%)
Grade 0: No improvement	9 (36.0)	5 (20.0)
Grade 1: Mild improvement	7 (28.0)	7 (28.0)
Grade 2: Moderate improvement	8 (32.0)	10 (40.0)
Grade 3: Marked improvement	1 (4.0)	3 (12.0)
Total with any improvement (Grades 1–3)	16 (64.0)	20 (80.0)

Note: p = 0.031 for change in proportion of responders between 12 and 24 weeks (McNemar test).

Table 3. Trichoscopic Parameters at Baseline, 12 Weeks, and 24 Weeks

Trichoscopic Parameter	Baseline	12 Weeks	24 Weeks	p-value*
Mean hair density (FU/cm²), mean ± SD	62.4 ± 8.3	68.3 ± 8.9	74.1 ± 9.6	0.003
Hair shaft diameter variability improved, n (%)		11 (44.0)	15 (60.0)	0.041
Miniaturised hair proportion reduced, n (%)		9 (36.0)	12 (48.0)	0.083
Hair density increased, n (%)		14 (56.0)	18 (72.0)	0.031
Perifollicular pigmentation improved, n (%)		7 (28.0)	10 (40.0)	0.219

Note: *McNemar test for paired proportions (12 vs. 24 weeks) for categorical variables; paired t-test for mean hair density. FU, follicular units; SD, standard deviation. , not applicable at baseline.

Table 4. Global Aesthetic Improvement Scale (GAIS) Ratings at 12 and 24 Weeks (n = 25)

GAIS Category	12 Weeks, n (%)	24 Weeks, n (%)
Very much improved	0 (0.0)	1 (4.0)
Much improved	3 (12.0)	7 (28.0)
Improved	14 (56.0)	10 (40.0)
No change	8 (32.0)	7 (28.0)
Worse	0 (0.0)	0 (0.0)
Total improved (any category)	17 (68.0)	18 (72.0)

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Patient Satisfaction

Patient-reported satisfaction scores at 12 and 24 weeks are summarised in Table 5. At 12 weeks, 64% of patients reported being satisfied or very satisfied with their treatment outcome; this proportion was 60% at 24 weeks. The slight decline in overall satisfaction at 24 weeks, despite improvement in objective and physician-rated parameters, may reflect patients' own expectations of more pronounced regrowth and the inherently gradual nature of hair growth response. Dissatisfaction (not satisfied) was reported by 20% of participants at each time point.

Table 5. Patient Satisfaction Scores at 12 and 24 Weeks (n = 25)

Satisfaction Category	12 Weeks, n (%)	24 Weeks, n (%)
Very satisfied	4 (16.0)	3 (12.0)
Satisfied	12 (48.0)	12 (48.0)
Slightly satisfied	4 (16.0)	5 (20.0)
Not satisfied	5 (20.0)	5 (20.0)
Overall satisfaction (Very + Satisfied)	16 (64.0)	15 (60.0)

Safety and Adverse Events: All adverse events recorded during the study period were mild and self-limiting (Table 6). The most commonly reported adverse effect was mild body hair increase (hypertrichosis), reported by 4 patients (16%), predominantly affecting the forearms and lower limbs. Mild lightheadedness was reported by 2 patients (8%), occurring predominantly within the first four weeks and resolving without dose modification. No cases of fluid retention, tachycardia, periorbital oedema, headache, pericardial effusion, or clinically significant blood pressure change were observed at any time point. No participant discontinued treatment due to adverse effects.

Table 6. Adverse Events Recorded During the 24-Week Study Period (n = 25)

Adverse Event	n (%)	Severity	Management / Outcome
Hypertrichosis (body hair increase)	4 (16.0)	Mild	Managed with depilation; treatment continued
Lightheadedness / dizziness	2 (8.0)	Mild	Resolved spontaneously within 4 weeks
Fluid retention / oedema	0 (0.0)		
Tachycardia or palpitations	0 (0.0)		
Periorbital oedema	0 (0.0)		
Headache	0 (0.0)		
Drug discontinuation due to AE	0 (0.0)		

Note: AE, adverse event, not applicable.

DISCUSSION

The results of this study demonstrate that oral minoxidil 1.25 mg once daily, administered as monotherapy over 24 weeks, produces clinically meaningful and progressive improvement in hair density and global appearance in male patients with AGA of Hamilton–Norwood grade III and above. The 80% objective response rate on GPA at 24 weeks, the 72% improvement rate on the GAIS, and the trichoscopic documentation of increased follicular density and reduced miniaturisation are collectively consistent with the efficacy reported in the existing literature on LDOM at comparable and higher dose levels.

The progressive, time-dependent pattern of improvement observed across all outcome parameters in the present study is biologically plausible. Minoxidil's principal mechanism prolongation of the anagen phase requires one or more full hair cycling periods before its effects become clinically manifest; visible improvement in hair density typically emerges over several months of continuous treatment [3,5]. The incremental gains between the 12-week and 24-week assessments in GPA, trichoscopic density, and GAIS ratings in this study reflect this expected time course, and suggest that 24 weeks may represent the minimum duration necessary to adequately evaluate treatment response.

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The molecular pharmacology of minoxidil's hair growth effects is now increasingly well characterised. As a prodrug, minoxidil requires sulphation by scalp sulphotransferase enzymes to generate the pharmacologically active minoxidil sulphate.[3,5] This active metabolite opens ATP-sensitive potassium channels in follicular smooth muscle and vascular endothelium, resulting in hyperpolarisation, local vasodilation, and an increase in perifollicular blood flow.[3,5] Additionally, minoxidil upregulates vascular endothelial growth factor (VEGF), hepatocyte growth factor (HGF), and prostaglandin E_2 all of which promote follicular cell survival and proliferation. Activation of the Wnt/β-catenin signalling cascade, a key regulator of follicular cycling, has also been implicated in minoxidil's anagen-promoting effects [5].

The efficacy observed in the present study is comparable with findings from published studies using oral minoxidil in male AGA. Jimenez-Cauhe et al. reported a mean hair density increase of 14.2 FU/cm² in male AGA patients treated with 2.5–5 mg oral minoxidil over 24 weeks,[7] while Panchaprateep and Lueangarun, using 5 mg oral minoxidil in 30 male AGA patients over 24 weeks, documented an increase of approximately 35 hairs/cm² a substantially greater quantitative gain reflecting the higher dose employed [8]. The mean hair density increment of 11.7 FU/cm² observed in the present study with the 1.25 mg dose falls within a plausible dose-response range, suggesting that the 1.25 mg dose, while producing a more modest magnitude of hair density gain than higher doses, does so with a markedly superior safety profile [6,7].

Jha et al. specifically evaluated oral minoxidil at 1.25 mg in male AGA and reported subjective and photographic improvement in the majority of patients, supporting the utility of this dose as a clinically effective starting regimen [9]. The findings of the present study complement and extend this evidence by incorporating trichoscopic quantification, physician-rated global assessment, and structured patient satisfaction reporting alongside photographic evaluation providing a more complete multidimensional characterisation of clinical response at this dose.

The safety profile observed in this study is reassuring and consistent with the established literature on LDOM. Hypertrichosis the most commonly reported adverse effect of oral minoxidil across published series was observed in 16% of participants in the present study, a rate closely concordant with the 15.1% reported by Vañó-Galván et al. in their multicentre safety cohort of 1,404 patients [6]. Hypertrichosis at the 1.25 mg dose was confined to body hair and was mild in all affected individuals, causing no treatment discontinuation. The 8% incidence of mild, transient lightheadedness is consistent with the vasodilatory mechanism of action and resolves spontaneously, as reported in other series.[6,7] No cardiovascular adverse events, fluid retention, pericardial effusion, or clinically significant haemodynamic changes were observed, which is of clinical relevance given that cardiovascular safety at low doses remains a concern that leads some practitioners to withhold LDOM [6].

The theoretical and practical advantages of the oral route over topical administration merit consideration. Topical minoxidil requires application to the entire affected scalp surface, which is technically difficult and cosmetically unappealing in patients with extensive hair loss, and may result in non-uniform coverage [3,8]. The compliance literature consistently demonstrates that once-daily oral administration improves medication adherence compared with twice-daily topical regimens, a factor directly relevant to the chronic, indefinite nature of AGA treatment.[8] Furthermore, LDOM bypasses topical contact sensitisation, scalp irritation, and vehicle-related adverse effects issues that drive discontinuation of topical formulations [3].

The patient satisfaction rate of 60% at 24 weeks, while positive, was lower than the objective response rate of 80% a discordance that warrants comment. This pattern is well recognised in AGA trials and reflects the gap between the expectations patients carry into treatment typically substantial regrowth within a few months and the reality of a slow, incremental response [14] Comprehensive pre-treatment counselling regarding the timeline of expected response, the initial telogen shed phenomenon, and the importance of sustained use is therefore a critical component of clinical management in order to optimise retention and satisfaction.

CONCLUSION

This prospective study demonstrates that oral minoxidil 1.25 mg once daily is an effective and well-tolerated monotherapy for male AGA, producing progressive and clinically meaningful improvements in hair density, follicular miniaturisation, global appearance, and patient satisfaction over 24 weeks. The favourable safety profile with no serious adverse events and

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no treatment discontinuation supports the clinical utility of this dose as an initial oral treatment strategy, particularly for patients who are intolerant of or poorly adherent to topical minoxidil. Randomised controlled trials with larger sample sizes, objective trichometric analysis, and longer follow-up are warranted to further define the dose-efficacy relationship and long-term outcomes of LDOM in male AGA.

Limitations: The small sample size of 25 patients limits the statistical power and generalisability of findings. The absence of a control arm or comparator group (e.g., topical minoxidil or higher-dose oral minoxidil) prevents direct comparative efficacy conclusions. The open-label design introduces the potential for placebo effect and assessment bias, despite the use of blinded photographic evaluation. Anthropometric and haematological monitoring was limited, and the study did not include baseline or follow-up hormonal profiling. The 24-week duration, while sufficient to demonstrate initial response, does not address long-term efficacy, maintenance of response, or late-emerging adverse effects. A randomised controlled trial with larger sample sizes, objective phototrichogram or TrichoScan analysis, and a longer follow-up period would provide stronger evidence to support these preliminary findings.

DECLARATIONS

Conflicts of Interest: The authors declare no conflicts of interest.

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