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Effects of oral minoxidil on serum VEGF and hair regrowth in androgenetic alopecia

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Abstract

Introduction: Androgenetic alopecia (AGA) is a common hair loss condition characterized by follicular miniaturization. Vascular endothelial growth factor (VEGF) plays a key role in promoting angiogenesis and supporting hair follicle growth. Oral minoxidil has been suggested to upregulate VEGF levels, enhancing hair regrowth.
Methods: This prospective study included 50 participants divided into two groups: oral minoxidil (1 mg/day; $n = 25$) and control ($n = 25$). Serum VEGF levels were measured at baseline and after 12 weeks of treatment. Hair growth parameters, including hair count, diameter, shedding, and pull test results, were assessed systematically.
Results: Baseline VEGF levels were similar between groups ($p = 0.1873$). Post-treatment, VEGF levels increased significantly in the minoxidil group (217.88 ± 22.65 pg/ml vs. 142.81 ± 23.14 pg/ml in the control, $p < 0.0001$). Hair count and diameter improved significantly ($p < 0.0001$ and $p = 0.0040$, respectively), with reductions in shedding and pull test results ($p < 0.0001$). Positive correlations were observed between VEGF and hair count ($r = 0.9965$), whereas shedding showed negative correlations ($r = -0.5374$).
Conclusions: Oral minoxidil significantly enhances VEGF levels, promoting hair growth and reducing shedding. VEGF serves as a promising biomarker for assessing treatment effectiveness and understanding the angiogenic mechanisms involved in AGA.

Keywords: androgenetic alopecia, hair regrowth, vascular endothelial growth factor, oral minoxidil, hair follicle angiogenesis

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Introduction

Androgenetic alopecia (AGA) is a common condition that affects both men and women, with research indicating that nearly 50% of men experience it by the age of 50, often leading to a notable decline in quality of life (1). The pathophysiology of AGA involves a combination of genetic predisposition and androgenic influences, particularly the androgen dihydrotestosterone, which shortens the hair follicle growth phase (2). Although it is not life-threatening, the condition causes significant psychological distress, particularly among women, leading to anxiety, depression, and low self-esteem (3). Recent studies confirm that AGA can lead to a decrease in social functioning and self-confidence, with younger individuals being more affected by these psychological aspects (4). The emotional impact is so profound that it is recommended to integrate psychological support in the management of AGA to improve patient outcomes (5).

Vascular endothelial growth factor (VEGF) plays a central role in promoting angiogenesis, which is essential for hair follicle development and growth. VEGF enhances blood vessel formation around hair follicles, providing the necessary nutrients and oxygen for hair regeneration. Studies have shown that treatments such as minoxidil can upregulate VEGF expression in dermal papilla cells, supporting follicular vascularization and improving hair growth (6). Furthermore, VEGF-induced angiogenesis is crucial in maintaining hair follicle health during the anagen phase, which is stimulated by minoxidil (7). Recent research has highlighted that VEGF's role in hair follicle angiogenesis can be leveraged for therapeutic purposes, making VEGF a promising biomarker for monitoring the effectiveness of hair growth treatments (8). The positive correlation between VEGF levels and improved hair growth emphasizes its potential use in clinical applications targeting hair loss (9). Therefore, VEGF is not only crucial for hair follicle survival, but it also serves as a reliable biomarker for assessing the success of treatments such as minoxidil (10).

The objectives of this study were to explore the effects of oral minoxidil on serum VEGF levels and assess its role in promoting hair regrowth in patients with AGA. The research sought to determine whether changes in VEGF levels during treatment could serve as a predictive marker for therapeutic outcomes, providing insights into the mechanisms by which minoxidil may enhance angiogenesis and stimulate hair follicle activity. In addition, this study aimed to correlate changes in serum VEGF with measurable improvements in hair density, offering a comprehensive understanding of the systemic and localized effects of the therapy.

Methods

Study design

This prospective interventional study evaluated the effects of oral minoxidil on serum VEGF levels and hair regrowth in patients with AGA. A total of 50 participants were divided into two groups: 25 patients received oral minoxidil, and 25 served as controls. Key parameters, including serum VEGF levels and hair growth outcomes, were systematically assessed over a 12-week period to analyze the systemic and localized effects of oral minoxidil. The study was approved by the Wasit Health Directorate – Institutional Review Board (approval number: WHD/IRB/2023/042).

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Patient enrollment criteria

Fifty participants were recruited based on strict eligibility criteria. Adults 18 to 50 years old with AGA and clinically confirmed hair density reduction were included, provided they were in good health and not undergoing concurrent hair loss treatments.

Exclusion criteria eliminated confounding factors, such as hypersensitivity to minoxidil, systemic illnesses, recent use of hair loss treatments, and scalp conditions such as psoriasis or severe seborrheic dermatitis. In addition, pregnant or breastfeeding individuals, or those with psychiatric conditions or poor medication adherence, were excluded. These criteria ensured a homogenous patient population suitable for evaluating oral minoxidil's therapeutic effects.

Oral minoxidil administration protocol

Participants in the treatment group received 1 mg of oral minoxidil daily, taken consistently each morning with water. The dosage remained steady unless adjusted for tolerability or adverse effects. Regular follow-ups monitored side effects, such as hypertrichosis, and assessed compliance with the dosing regimen.

Systemic effects were evaluated through routine safety assessments, including blood pressure monitoring. This protocol ensured the safe administration of oral minoxidil while allowing thorough evaluation of its effects on VEGF levels and hair regrowth outcomes.

Follow-up period and monitoring schedule

Participants were monitored over a 12-week follow-up period to ensure accurate assessment of treatment outcomes. Clinic visits were scheduled at baseline, 6 weeks, and 12 weeks, during which serum VEGF levels were measured to evaluate the systemic effects of oral minoxidil. Comprehensive hair count assessments were performed using standardized photographic documentation and density evaluation techniques.

Routine safety monitoring included blood pressure measurements at every visit, and patients were encouraged to report symptoms such as hypertrichosis, dizziness, or palpitations. Side effects, tolerability, and patient-reported outcomes were recorded through standardized questionnaires. Regular follow-ups and interim check-ins ensured adherence to the dosing regimen, maintained patient safety, and allowed precise tracking of therapeutic outcomes.

Serum VEGF levels measurement post-treatment

Changes in serum VEGF levels were assessed by collecting blood samples at baseline and after 12 weeks of treatment. Blood was drawn using aseptic techniques, and serum was separated, centrifuged, and stored at −80 °C until analysis. VEGF levels were measured using a high-sensitivity enzyme-linked immunosorbent assay (ELISA).

Duplicate assays were performed to minimize variability, and all analyses were conducted by trained personnel blinded to study groups. Calibration standards and internal controls were used to ensure consistency. VEGF levels were expressed in pg/ml, allowing a clear comparison of pre- and post-treatment values.

Hair count and regrowth assessment techniques

Standardized methods were used to evaluate hair count and regrowth. The strip-based hair count technique involved marking a 1 cm^2 area on the scalp and manually counting hairs under magnification to assess density changes over 12 weeks.

The hair pull test, used to measure active hair shedding, involved gently pulling 40 to 60 hair strands and recording dislodged hairs. A reduction in shedding indicated treatment success. Hair diameter was assessed using a trichometer, which measured changes in hair shaft thickness, reflecting improved follicular health.

Participants also collected shed hairs during a standardized 24-hour activity, such as combing or washing, with counts recorded to assess hair retention. These objective and consistent methods provided quantitative data for evaluating the efficacy of oral minoxidil in promoting hair regrowth and reducing hair loss.

Statistical analysis

Data were analyzed using SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as means, standard deviations, and ranges, and categorical variables were described as frequencies and percentages. A paired *t*-test or the Wilcoxon signed-rank test was employed to evaluate changes in serum VEGF levels and hair count within groups during treatment, depending on normality. An independent *t*-test or the Mann–Whitney *U* test was used to compare outcomes between the minoxidil and control groups when normality assumptions were unmet.

Repeated measures analyses were conducted to account for within-subject variations over time, particularly for hair count and serum VEGF levels, to detect longitudinal trends and assess treatment efficacy comprehensively. Pearson's or Spearman's correlation coefficients were calculated to explore relationships between VEGF levels and hair growth parameters (e.g., hair count, hair diameter, shedding, and pull test results).

Statistical significance was set at $p < 0.05$, with 95% confidence intervals provided to ensure precision in estimates. Imputation methods were applied to handle missing data, ensuring the robustness of the findings and minimizing potential bias.

Results

An analysis of demographic characteristics with statistical comparison between the control and minoxidil groups was unremarkable for any of the characteristics measured (Table 1). There was a small discrepancy in age (*p*-value = 0.88) and an even smaller one for body mass index (BMI) (*p*-value = 0.82), indicating no significant difference. Sex distribution was also consistent, with

Table 1 | Statistical comparison of demographic data between control and minoxidil groups.

Parameter	Control ($n = 25$)	Minoxidil ($n = 25$)	*p*-value
Age (years),* mean ± *SD*	40.2 ± 2.5	40.1 ± 3.0	0.88
Sex (male),† *n* (%)	13 (52)	12 (48)	0.95
BMI (kg/m^2),* mean ± *SD*	25.4 ± 1.8	25.3 ± 1.5	0.82
Diabetes,† *n* (%)	6 (24)	5 (20)	0.69
Hypertension,† *n* (%)	9 (36)	10 (40)	0.78

SD = standard deviation, BMI = body mass index.
*Independent samples *t*-test, †chi-squared test; $p < 0.05$ was considered statistically significant.

the ratio of males and females in both groups at parity, and a chi-squared test produced a *p*-value of 0.95, indicating no significant difference between the groups. As with diabetes and hypertension, no great differences between the groups could be observed in the comparative review of patient history. The proportion of patients with a history of diabetes was 20% in the minoxidil group and 24% in the control group ($p = 0.69$). There was a slightly higher history of hypertension in the minoxidil group (40% of those treated vs. 36% of the control group), but the difference was not statistically significant ($p = 0.78$). Comparison of treatment efficacy between the two groups was therefore unconfounded by differences in key demographic variables between the control and minoxidil groups. Moreover, these demographic factors did not show prominent differences, affirming the feasibility of comparing the minoxidil treatment effects on hair growth and on the serum levels of VEGF.

The hair growth parameters of the control group and the group given oral minoxidil are compared in Table 2 (Fig.1). Before the intervention, no statistically significant difference was observed with baseline hair count in a defined scalp area between the two groups. As one would expect, confirming that both groups had identical hair density at the outset of the study should eliminate confusing variables.

Treatment with minoxidil followed by a 12-week follow-up period showed a significant increase in hair count compared to the control group. The robust efficacy of oral minoxidil in inducing a return of hair growth is consistent with this outcome. Its known angiogenic effects improve hair density and may in part account for improved follicular activity and growth cycles.

Furthermore, measurements of hair diameter further supported the advantages of minoxidil. The hairs driven by minoxidil were significantly thicker than those in the control group. The result of this increase in hair caliber is suggestive of a change from fine vellus hairs to thicker terminal hairs, and thus better hair quality and follicular health.

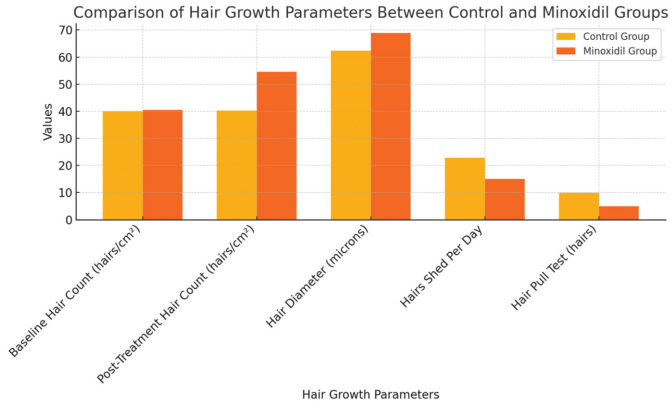

Figure 1 | Comparison of hair growth parameters between control and minoxidil groups.

Table 2 | Comparison of hair growth parameters between control and minoxidil groups.

Parameter	Control ($n = 25$) mean ± *SD*	Minoxidil ($n = 25$) mean ± *SD*	*p*-value
Baseline hairs/cm²	40.02 ± 5.74	40.53 ± 4.94	0.7377
Post-treatment hairs/cm²	40.27 ± 5.55	54.58 ± 4.53	0.0000
Hair diameter (microns)	62.38 ± 7.22	68.94 ± 8.09	0.0040
Hairs shed per day	22.87 ± 5.13	15.09 ± 3.78	0.0000
Hair pull test (hairs)	9.90 ± 3.65	5.00 ± 1.90	0.0000

SD = standard deviation.
Independent samples *t*-tests; $p < 0.05$ was considered statistically significant.

The effectiveness of minoxidil in reducing hair loss was demonstrated by a dramatically lower number of hairs shed during a standardized collection period in the minoxidil group. Successful therapy might therefore inhibit shedding through the stabilization of hair follicles in their growth phase.

The hair pull test results showed few dislodged hairs in the minoxidil group, corresponding with improved hair retention. The results of the test show that oral minoxidil not only results in hair regrowth but also stabilizes and strengthens existing natural hair follicles.

Table 3 (Fig. 2) compares the serum VEGF concentration before and after 12 weeks of treatment. Serum VEGF levels were compared between patients treated with oral minoxidil and in the control group. There was no statistically significant difference between groups at baseline VEGF levels prior to treatment initiation. However, after 12 weeks, the VEGF levels in the minoxidil group were significantly higher than in the control group. One explanation is that oral minoxidil increases VEGF, a component contributing to its therapeutic effects in hair regrowth and angiogenesis. The results imply a systemic effect of minoxidil on VEGF expression, suggesting that VEGF is a predictive biomarker for treatment outcome.

Table 4 shows the correlation analysis in hair growth parameters between the control and minoxidil groups. Significantly, the increase in VEGF was positive with regard to post-treatment hair number and hair size.

Figure 3 shows the correlation between VEGF levels and hair growth parameters. The scatter plot also shows the correlation coefficients (r), in which the positive coefficients are marked in blue and the negative coefficients are marked in red.

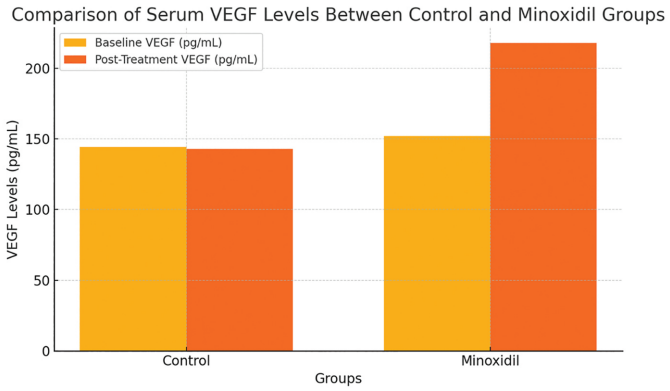

Figure 2 | Comparison of serum vascular endothelial growth factor levels (VEGF) between control and minoxidil groups.

Table 3 | Comparison of serum vascular endothelial growth factor levels between control and minoxidil groups at baseline and 12 weeks of follow-up.

VEGF parameter	Control ($n = 25$) mean ± *SD*	Minoxidil ($n = 25$) mean ± *SD*	*p*-value
Baseline (pg/ml)	144.40 ± 21.04	152.12 ± 19.74	0.1873
At follow-up (pg/ml)	142.81 ± 23.14	217.88 ± 22.65	0.0000

VEGF = vascular endothelial growth factor, *SD* = standard deviation.
Independent samples *t*-test; $p < 0.05$ was considered statistically significant.

Table 4 | Correlation analysis between vascular endothelial growth factor levels and hair growth parameters after treatment.

Parameter	*r*	*p*-value
Post-treatment hairs/cm²	0.9965	0.0001
Hair diameter (microns)	0.3545	0.0115
Hairs shed per day	−0.5374	0.0001
Hair pull test (hairs)	−0.5503	0.0001

Pearson correlation analysis; $p < 0.05$ was considered statistically significant.

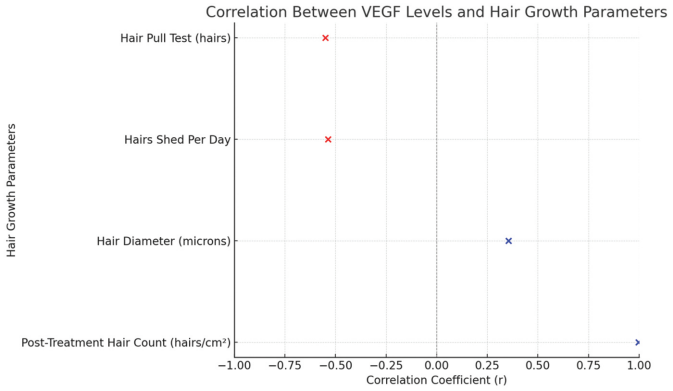

Figure 3 | Correlation between vascular endothelial growth factor levels (VEF) and hair growth parameters.

Discussion

This study evaluated the efficacy of oral minoxidil in increasing serum VEGF levels and promoting hair regrowth in AGA. At baseline, there was no significant difference in serum VEGF levels between the control and minoxidil groups. However, post-treatment, the minoxidil group showed a substantial increase in VEGF levels, from 152.12 ± 19.74 pg/ml at baseline to 217.88 ± 22.65 pg/ml ($p = 0.0000$). This significant elevation suggests that oral minoxidil stimulates angiogenesis, enhancing blood supply to hair follicles and supporting hair growth.

The findings align with the hypothesis that minoxidil exerts its effects by increasing VEGF, which plays a critical role in angiogenesis and the stimulation of hair follicle activity. VEGF promotes blood vessel growth, ensuring the delivery of oxygen and nutrients essential for hair regeneration in patients with AGA.

It is hypothesized that angiogenesis actively supported by VEGF could participate in the regulation of follicular activity and transition to thicker terminal hairs. On the other hand, Yano et al. (11) revealed negative relationships between VEGF and indicators of hair loss. These results indicate that high levels of VEGF in the scalp could be associated with lower hair loss and contain the hair follicle in place. The negative correlations suggest that VEGF may serve to extend the anagen phase of hair growth and thereby decrease hair shedding.

Kuo et al. reported that oral minoxidil improved hair regrowth in cancer survivors. Their study observed substantial increases in hair density, scalp thickness, and color, particularly in the frontal and occipital areas. Although VEGF levels were not directly measured in their study, the improvements in hair growth are consistent with the known angiogenic and hair-stimulating effects of minoxidil. These findings could support the hypothesis that VEGF-mediated mechanisms contribute to minoxidil's efficacy, though further research is needed to confirm this relationship (12).

Similarly, findings by Panchaprateep and Lueangarun corroborate the efficacy of oral minoxidil for treating male AGA. Their open-label study demonstrated significant hair growth improvements in male patients receiving oral minoxidil (5 mg/day). They concluded that systemic minoxidil treatment is more effective than topical applications in promoting hair growth, likely due to increased VEGF levels and enhanced blood supply to hair follicles, consistent with this study's observations (13).

In a comparative study, Gupta et al. found that oral minoxidil (5 mg/day) promoted hair regrowth in AGA. Their findings suggest, that oral minoxidil enhances hair growth through a number of pathways, including the upregulation of VEGF. The authors concluded that angiogenesis and blood supply to hair follicles may play a large role in how minoxidil works for treating hair loss (14).

In addition, Feldman et al. performed a systematic review and Bayesian network meta-analysis of hair regrowth treatment, including oral minoxidil, and they found that oral minoxidil significantly contributes to hair regrowth, confirming that the mechanisms are likely angiogenic. The conclusions of this study also fit with the authors' suggestion that minoxidil supplementation worked by increasing VEGF levels, which led to increases in hair density and regrowth (15).

Meephansan et al. also investigated minoxidil's effects on hair growth and found that minoxidil level significantly increased VEGF levels in skin, which indicated that VEGF is crucial to minoxidil-mediated hair regrowth. In line with this study, which showed increased VEGF induction following minoxidil treatment, the team's findings are consistent with VEGF induction induced through minoxidil treatment (16).

The overall trends of this study's findings are in line with a recent study by Dhurat et al. that focused on caffeine and minoxidil as a therapy for AGA (17). The minoxidil group significantly improved hair count, hair diameter, and hair retention compared to the control group, and the minoxidil group demonstrated significantly superior values in all parameters measured. These data demonstrate that oral minoxidil presumably induces hair regrowth via its known vasodilatory and angiogenic effects to promote follicular activity.

This is further supported by a recent review of the clinical benefits of oral minoxidil, particularly its ability to improve hair density in patients with AGA. Ramos highlighted systemic effects of minoxidil, demonstrating improvements in both male and female patients treated with low-dose oral regimens. Consistent with the findings of this study, Ramos reported positive outcomes in hair growth following oral minoxidil treatment. However, it was previously identified that hypertrichosis was a common side effect, underscoring that while oral minoxidil is an effective therapy, it is associated with side effects, including unwanted hair growth in non-target areas (18).

A study by Asilian et al. compared low-dose oral minoxidil (1 mg/day) to topical minoxidil in patients with AGA. Hair diameter significantly improved with both treatments, but hair density improved less dramatically in the oral minoxidil group than in the group using the topical. Their findings thus contrast with the increasing hair density observed in this study, indicating that oral minoxidil at higher doses can be as effective or even more effective than topical treatment in some patient populations. At the same time, Asilian et al. noted that both treatment routes are generally well tolerated, and both also have relatively high patient satisfaction rates (19).

A further review by Villani et al. of oral minoxidil for various forms of alopecia concluded that oral minoxidil is a well-tolerated alternative for patients with AGA, particularly for those that experience problems with topical formulations due to poor adherence or side effects. Moreover, topical minoxidil remains the gold standard, although oral minoxidil is a good alternative, with oral doses from 0.25 mg to 5 mg per day providing good efficacy without major side effects. This result is consistent with the conclusion of this study that oral minoxidil is effective for reducing hair shedding while promoting regrowth and being relatively safe for long-term use (20).

Yin et al. performed a retrospective analysis of low-dose oral

minoxidil for AGA and found that it increased hair density and thickness significantly, confirming the results seen in this study. A cohort analysis of 60 patients demonstrated significant improvement of hair density, lending further weight to the assumption that oral minoxidil is a clinically effective treatment for AGA, particularly if dosed properly. Moreover, low dose-oral minoxidil may also serve as a second-line option for cases where initial treatments have failed.

In a more recent randomized controlled trial comparing oral spironolactone with topical minoxidil to minoxidil monotherapy for female pattern hair loss, Liang et al. report a positive effect with oral spironolactone plus topical minoxidil compared to topical minoxidil alone. The combination treatment showed the most significant increase in hair density, but oral minoxidil alone was efficacious, with the appearance of considerable hair density and diameter, particularly at those sites where topical minoxidil had previously failed. These demonstrate the versatility of oral minoxidil as an alternative or adjunct to topical treatments and further prove the broad therapeutic potential of oral minoxidil for AGA (22).

The results of this study are interesting in linking serum VEGF levels with major hair growth parameters, including hair count, hair diameter, and hair loss metrics (hair shedding and hair pull test). As shown in Table 4, there are positive correlations between VEGF levels and the hair health measure of post-treatment hair count and diameter. However, a negative correlation was seen with parameters of hair loss (number of hairs shed per day and the hair pull test result). As with angiogenic paracrine signals, if VEGF plays a role in promoting hair growth and stability, it would presumably promote follicular activity and aid in the switch of hair follicles from fine temporary hairs to thicker terminal hairs and reduced shedding. Consistent with minoxidil's therapeutic effects of VEGF in AGA, these findings suggest that VEGF mediates these effects of minoxidil in AGA.

Several key trends emerge when these findings are compared to more recent studies. A study by Zhang et al. demonstrated that VEGF can protect hair follicle stem cells against apoptosis by androgen treatment through the PI3K/Akt pathway. According to this study, VEGF also protects hair follicles from damage and promotes the initiation of hair regeneration in AGA (23). In addition, a study by Andjelkov et al. showed that VEGF in the adipose-derived stem cell secretome is important for promoting hair growth and for the elevation of VEGF in areas with active hair growth, and it demonstrated the potential of using VEGF for the treatment of AGA (24).

VEGF is critically involved in tumor angiogenesis, and targeting VEGF in combination with other modalities has been shown to improve outcomes in oncology. While the role of VEGF in hair follicles is an area of interest, further research is needed to determine if the mechanisms of anti-VEGF therapies in non-small cell lung cancer (NSCLC) and other tumors share similarities with its effects in hair biology (25).

Interestingly, while the role of VEGF in promoting hair growth is well-documented in these studies, work by Soyama et al. has suggested that excessive inhibition of VEGF might have negative prognostic implications in cancer treatment (26). This suggests a more complex relationship: while VEGF is often associated with promoting hair growth, imbalances in its levels—whether too low or too high—might contribute to unintended effects.

Additionally, the findings of this study align with those of Pramanik et al., who showed that VEGF could serve as a biomarker for treatment response rather than directly indicating hair growth. This supports the idea that biomarkers like VEGF are valuable for assessing the effectiveness of a therapy (27).

Conclusions

This article summarizes the therapeutic potential of oral minoxidil in treating AGA based on the systemic effects of its VEGF-dependent antioxidative/antiangiogenic effects. It was found that VEGF levels were associated with increased hair count and diameter, and decreased hair loss. The results suggest that oral minoxidil may not only enhance follicular vasculature but also contributes to stabilizing hair follicles, leading to improved regrowth and reduced shedding. These findings align with the clinical potential of oral minoxidil for AGA and suggest that VEGF could serve as a useful marker for both diagnosis and monitoring treatment response.

Although this study provides valuable insights into the effects of oral minoxidil on serum VEGF levels and hair regrowth in AGA, several limitations should be acknowledged: 1) Absence of a placebo-controlled group: the control group did not receive a placebo intervention, which may have introduced inadvertent bias in the findings. Future studies should incorporate a placebo group to better isolate the therapeutic effects of oral minoxidil. 2) Short follow-up duration: the 12-week follow-up period may not fully capture the long-term effects of oral minoxidil on hair regrowth and serum VEGF levels. Extended observation periods are recommended to better evaluate sustained outcomes and potential late-emerging side effects. 3) Small sample size: with only 50 participants divided into two groups, the study's sample size was limited. Larger multicenter studies are needed to improve the generalizability and statistical power of the findings. 4) Self-reported adherence: participant compliance with the daily dosing regimen was assessed through self-reporting, which may have introduced recall bias. Objective methods of monitoring adherence, such as electronic pill dispensers, should be considered in future research. 5) Restricted population scope: the study included only adults 18 to 50 years old with AGA. The findings may not be generalizable to older individuals or patients with other types of alopecia. Future studies should explore a broader range of demographics and conditions.

By addressing these limitations, future research can build upon the findings of this study to provide a more comprehensive understanding of the therapeutic potential and mechanisms of oral minoxidil.

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