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Serum ferritin and hair iron content in correlation to hair diameter and density: a case-control study

Arlha Aporia Debinta,[1] Windy Keumala Budianti,[1] Andira Hardjodipuro,[1] Sri Linuwih Menaldi,[1] Dian Kusuma Dewi,[2] Lili Legiawati,[1] Yudo Irawan,[1] Inge Ade Krisanti,[1] Shannaz Nadia Yusharyahya[1]

[1]Department of Dermatology and Venereology, Faculty of Medicine, Universitas Indonesia, Dr. Cipto Mangunkusumo National Central General Hospital, Jakarta; [2]Department of Community Medicine, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

Correspondence: Arlha Aporia Debinta, Department of Dermatology and Venereology, Faculty of Medicine, Universitas Indonesia, Dr. Cipto Mangunkusumo National Central General Hospital, Jl. Pangeran Diponegoro No.71, Jakarta, Indonesia. E-mail: arlha.debinta@gmail.com

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Contributions: Arlha Aporia Debinta: conceptualization, study design, data collection, statistical analysis, interpretation of results, and manuscript drafting; Windy Keumala Budianti, Sri Linuwih Menaldi, Dian Kusuma Dewi, Lili Legiawati, Yudo Irawan: supervision, conceptualization, and critical revision; Andira Hardjodipuro: data collection, statistical analysis, and manuscript writing; Inge Ade Krisanti, Shannaz Nadia Yusharyahya: supervision and critical revision. All the authors read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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Abstract

Androgenetic alopecia (AGA) is the most common non-scarring hair loss disorder in men due to susceptibility to testosterone. AGA causes cosmetic disturbances that affect confidence and quality of life. The correlation between low ferritin serum levels and AGA has been sufficiently proven in women but not in men. Thus, this study evaluated this correlation in Indonesian men. This case-control study included 33 men with AGA and 33 non-alopecia controls. Serum ferritin and hair iron levels were analyzed for differences using the Mann-Whitney U test. Their correlations with hair diameter and density were assessed using the Spearman correlation test. Serum ferritin (AGA *vs.* control, median: 222 *vs.* 232 ng/mL; p=0.758) and total hair iron levels (22.65 *vs.* 39.67 ng/mL; p=0.102) showed no significant differences between the groups. However, in men with milder AGA (grade <4), total hair iron level and hair diameter showed significant weak positive correlations (r=0.312, p=0.033). In the non-alopecia group, serum ferritin and total hair iron levels showed a significant weak negative correlation (r=−0.387, p=0.026). Hair iron levels were positively correlated with hair diameter in men with early AGA, suggesting a potential role in promoting hair thickness and highlighting its promise as a target for adjuvant therapy.

Introduction

Alopecia is a type of hair loss on the scalp that can be experienced by both men and women.[1] The most common type is androgenetic alopecia (AGA), often known as male pattern baldness, characterized by hair miniaturization caused by susceptibility to androgen hormones.[2] According to a study conducted by Legiawati *et al.* at the Dr. Cipto Mangunkusumo Hospital (RSCM) Cosmetic Dermatology Polyclinic (Jakarta, Indonesia) in 2017-2019, 31.2% of patients with hair loss were diagnosed with AGA.[3] In their lifetimes, 50-60% of men experience AGA at the age of 50 years, and 80%, after the age of 70 years.[2] Conditions such as AGA cause appearance disorders that cause reduced self-confidence, psychological stress, and decreased quality of life.[4]

Iron is the primary inorganic component of hemoglobin and other iron-containing enzymes. As such, decreased iron levels could reduce tissue vascularization and hypoxia. Hair follicle matrix cells are rapidly dividing cells in the human body that are particularly sensitive even to a minor decrease in iron availability, resulting in reduced hair growth under various conditions. Apart from the role of genetics and hormones, other factors influence the occurrence of AGA. Iron deficiency is one of the most frequent causes associated with baldness, although its mechanism remains unclear. As the main iron-binding protein that reflects the total iron stores in the body, serum ferritin is used as a marker of iron levels.[5] However, only a few studies have investigated iron status in adult male populations, which is also further associated with other diseases, such as AGA. Iron is an important cofactor of

many enzymes that have functional activity in hair. Thus, it is necessary to evaluate total hair iron levels and their correlation with AGA. This study aimed to evaluate the impacts of serum ferritin and hair iron levels on men with AGA and non-alopecia hair loss.

Materials and Methods

Ethical clearance for this study was obtained from the research ethics committee of the Faculty of Medicine, Universitas Indonesia (No. KET-1259/UN2.F1/ETIK/PPM.00.02/2023). The study employed an analytical case-control design to compare two distinct groups of male patients.

Study population, selection, and sample size calculation

The target population was composed of all men diagnosed with AGA in Indonesia who were seeking treatment at the dermatology and venereology outpatient clinic at RSCM. Patients were selected from the accessible population using consecutive sampling based on predefined inclusion and exclusion criteria.

The total group was divided into two main categories: i) case group, consisting of Indonesian men aged 18-59 years who were clinically diagnosed with AGA at a severity level of Hamilton-Norwood classification grade ≥3; ii) control group, including Indonesian men aged 18-59 years who were not clinically diagnosed with AGA but had baldness classified as Hamilton-Norwood grades 1 or 2. Furthermore, the control subjects were required to have no other forms of baldness, as determined by their medical histories and physical examinations.

We used age matching between the case and control groups to ensure comparability. Patients were excluded if they had conditions other than AGA or any conditions or diseases that could affect hair physiology. The following exclusion criteria were based on recent lifestyle and medical interventions: vegetarian (did not consume animal protein) or had taken iron supplementation in the last month. Patients were also excluded if they had applied medication or vitamins that could affect scalp hair growth in the last two weeks, taken drugs that could affect hair physiology in the last three months, taken medication or vitamins that could affect scalp hair growth in the last month, or undergone any procedure that could affect scalp hair growth in the last three months. Finally, patients who had undergone hair dye procedures with bleaching were also excluded.

The final sample size for the study was determined through an *a priori* power calculation to ensure adequate statistical power for the primary endpoints. For the comparison of means between the two nonpaired populations, the calculation was based on a formula using the desired significance level (α=5%, $Z\alpha$=1.96), statistical power of 80% ($Z\beta$=0.84), and differences in means (X_1-X_2) derived from clinical judgment and the literature.[6] For the correlation analysis, the required sample size was

calculated using a separate formula based on the Zα, Zβ, and the minimal correlation coefficient (r) considered meaningful.

Clinical and hair assessments

AGA was diagnosed clinically. The research subjects were evaluated clinically based on the Hamilton-Norwood scale, hair pull test, and trichoscopy. Photos were taken from 7 standardized positions: frontal (1), lateral (2), oblique/temporal (2), vertex (1), and the TrichoScan® test location (1). The primary test location was standardized: 3-5 cm anterior to the upper tip of the tragus and then 10-15 cm superior to that point, with inspections carried out at the same location during each clinic visit.[7]

Hair density and diameter were assessed using a FotoFinder Medicam HD 800 equipped with the TrichoScan® software. This involved clipping the hair in the 1 cm^2 test area. The magnification used in this study was ×20. No hair-dye procedure was performed because most of the patients were Asian men with dark hair. The analysis was automated, which inherently minimized subjective rater bias. However, to ensure complete study integrity, the operator who performed the physical steps was blinded to the subject's clinical diagnosis or treatment status using an anonymous ID number. Furthermore, subsequent data reviewers who performed complex subgroup or statistical analyses received data files stripped of group identifiers. Previous evidence showed that TrichoScan® measurements are highly reproducible, showing excellent intra- and inter-rater reliability (approximately ≥0.962% intraclass correlation coefficient for all parameters).[8]

Separately, a conventional trichoscopy examination was performed using a Heine® Delta 20 Plus dermoscope on a 1 cm^2 area of the frontal scalp on both the left and right sides, with a fixed magnification of ×20. We specifically evaluated hair diameter variability, peripilar signs, empty follicles, and yellow dots. Consistent with the previous protocol, the examiner conducting the physical assessment was blinded to each subject's clinical diagnosis and treatment status, using only an anonymized ID number.

Sample collection and laboratory analysis

Six milliliters of venous blood were collected and transferred into a plain storage tube to obtain serum, which was analyzed the same day for ferritin concentration using an enzyme-linked immunosorbent assay. Hair samples were cut as close to the scalp as possible, and each sample weighed 500 mg. The details of the hair washing protocol are as follows:

i) Hair samples were washed three times with a standardized sequence to remove surface contaminants without extracting internal elements. The protocol consisted of immersion and agitation

for 10 min in acetone (a nonpolar solvent), followed by two rinses with deionized water and a final wash in acetone. After washing, the hair samples were dried in an oven at 60°C until a constant weight was achieved.

ii) A precise mass of 500 mg of hair was weighed accurately into a clean digestion vessel. The weighed sample was mixed with 1 mL of concentrated nitric acid (HNO_3; ultrapure grade) and heated at 170°C for 30 min (wet digestion).

iii) After heating, the digestate was quantitatively transferred to a volumetric flask and diluted to a final, specific volume (10 mL) using deionized water. The resulting solution was then centrifuged for 10 min and filtered using a Millex filter before analysis.

iv) The final digestate solution was measured using inductively coupled plasma optical emission spectrometry (ICP-OES) at the Jakarta Regional Health Laboratory. The ICP-OES instrument was calibrated using a minimum of five concentrations of a certified iron standard prepared with matrix-matched blanks containing the same nitric acid concentration as the samples. Quality control was ensured by analyzing certified human hair reference materials alongside the samples. The limit of detection and limit of quantification (LOQ) were determined as 3 and 10 times, respectively, the standard deviations (SDs) of the procedural blank readings. Precision was checked by analyzing every tenth sample in duplicate, and accuracy was confirmed with matrix spike recovery, ensuring that the measured iron concentrations were confidently higher than the LOQ.

Statistical analyses

All data were processed statistically using SPSS® version 25. Categorical data are presented using percentages (counts). Numerical data were tested for normality using the Kolmogorov-Smirnov test. Depending on the distribution, the Mann-Whitney U test was used for group comparisons, and the Spearman correlation test was used to assess relationships between variables. Normally distributed numerical data are presented using the mean ± SD, while abnormally distributed data are presented using the median and interquartile range (IQR). Data are reported in the form of narratives, tables, and graphs, including scatter plots for correlation analysis. The Holm-Bonferroni (HB) method was used to control multiplicity by providing an adjusted p-value or a new alpha level. Multivariate analysis was utilized to identify factors associated with AGA. The primary endpoints of this study were to determine the correlations between serum ferritin levels and AGA and between hair iron levels and AGA in Indonesian men using a correlation coefficient (r) and $p<0.05$ for statistical significance.

Results

Subject characteristics and demographic comparison

A total of 66 men were recruited and equally divided between the case (AGA, n=33) and control (non-alopecia, n=33) groups. The difference in mean age between the AGA (36.97±7.93 years) and control groups (38±9.79 years) was comparable (p=0.640). Most subjects in both groups had a secondary educational level and showed no significant differences in body mass index (BMI), smoking habits, and alcohol consumption between the two groups. Clinical assessment confirmed a statistically significant difference in trichoscopic findings, with the AGA group exhibiting a higher median degree of severity (Hamilton-Norwood grade 4, range 3-5) than the non-alopecia group (grade 1, range 0-1; $p<0.001$). The AGA group showed significantly higher rates of clinical signs of alopecia, including hair diameter diversity (97.0% *vs.* 12.1%, $p<0.001$), peripilar sign (50.0% *vs.* 6.1%, $p<0.001$), empty follicles (97.0% *vs.* 9.1%, $p<0.001$), and yellow dots (84.8% *vs.* 15.2%, $p<0.001$), as shown in Table 1.

Comparison of iron status between the groups

Data for both serum ferritin and total hair iron levels were found to be abnormally distributed (non-normal) using the Kolmogorov-Smirnov test, necessitating the use of the Mann-Whitney U test for group comparison. The median serum ferritin level in the AGA group (222 ng/mL; IQR, 153-406 ng/mL; Figure 1) was slightly lower than that in the control group (232 ng/mL; IQR, 194-305 ng/mL) but not statistically significant (p=0.758; Table 2). Similarly, the median total hair iron level was lower in the AGA group (0.0453 µg/g hair; IQR, 0.0245-0.0969 µg/g) than in the non-alopecia group (0.0793 µg/g hair; IQR, 0.0499-0.1314 µg/g; Figure 2). The difference was also not statistically significant (p=0.102), as mentioned in Table 2.

Holm-Bonferroni test

The HB method was used to control multiplicity by providing an adjusted p-value or a new alpha level. The method uses the following formula to obtain the adjusted p-value:

$$\mathrm{HB} = \frac{\text{Target alpha level}}{\mathrm{n} - \text{rank number of pair} + 1},$$

The target alpha level is 0.05, *n* is the number of tests, and the rank number of the pair is the rank of the tests based on their crude p-value and sorted from smallest to largest.

This study evaluated two endpoints, corresponding to two separate tests. Crude p-values were calculated using the Mann-Whitney U test. The results, including crude p-values and ranks, are summarized below (*Supplementary Table 1*):

H_1: Serum ferritin levels in AGA *vs.* non-AGA; crude p=0.758; rank=2;

H_2: Hair iron levels in AGA *vs.* non-AGA; crude p=0.102; rank=1.

HB was calculated as follows:

$$HB_{H1} = \frac{0.05}{2 - 2 + 1}$$

$$HB_{H1} = 0.05$$

$$HB_{H2} = \frac{0.05}{2 - 1 + 1}$$

$$HB_{H2} = 0.025$$

Neither hypothesis reached statistical significance based on the crude p-values. After applying multiplicity correction using the HB method, both hypotheses remained non-significant.

Multivariate analysis of serum ferritin level and AGA status

The differences in serum ferritin levels and age were not statistically significant (*Supplementary Table 2*). BMI and smoking status also showed no significant association with AGA. This model explained only 10.8% of the variance in AGA (r^2=0.108). However, based on the effect size of the odds ratio, individuals with BMIs between 23 and 24.9 kg/m^2 and a history of smoking had a higher likelihood of AGA, which reduced with increasing age (*Supplementary Table 3*). The logistic regression analysis showed that adjustment for each potential confounder did not substantially change the odds ratio for serum ferritin levels (<1%). This indicates the absence of confounders in the analyses of serum ferritin levels and AGA.

Multivariate analysis of hair iron level and AGA status

The multivariable analysis results indicated that the differences in hair iron level and age were not statistically significant (*Supplementary Table 4*). BMI and smoking history were also not significantly associated with the risk of AGA. This model only explained 15.7% of the variance in AGA (r^2=0.157). However, the effect size of the odds ratio indicated that individuals with BMIs between 23 and 24.9 kg/m^2 and a history of smoking have a higher likelihood of AGA, and increasing hair iron levels also increases the likelihood of AGA (*Supplementary Table 5*). However, the likelihood of AGA decreased slightly with increasing age. Logistic regression analysis showed that adjustment

for each potential confounder resulted in minimal changes in the odds ratio for hair iron levels (<1%). This indicates the absence of confounders in the analysis of hair iron levels and AGA.

Correlation analyses

Correlation analyses were performed using the Spearman correlation test. The initial analysis of the overall AGA and non-alopecia groups revealed no statistically significant correlations between serum ferritin level, total hair iron level, and hair physical characteristics (diameter and density; Table 3). Scatter plots further illustrated the wide spread of the data, indicating no clear trend between these variables (Figures 3 and 4).

A further correlation test was performed according to AGA severity, dividing the analysis group into grade ≥4 (n=19) and grade <4 (n=47). This subgroup analysis yielded two statistically significant findings:

i) Hair iron level and diameter (milder AGA): in patients with an AGA severity of grade <4, a statistically significant weak positive correlation was found between the total hair iron level and hair diameter (r=0.312, p=0.033; *Supplementary Table 6*).

ii) Serum ferritin and hair iron (controls): in the non-alopecia population, a statistically significant weak negative correlation was found between serum ferritin and total hair iron levels (r=−0.387, p=0.026; *Supplementary Table 7*).

The correlation between serum ferritin and total hair iron levels in the overall AGA population remained statistically insignificant (r=0.009, p=0.961).

Discussion

This case-control study aimed at investigating the relationship between iron status and AGA in Indonesian men, a population for which such data are scarce. The subjects' mean age was comparable between the AGA (36.97 years) and the control group (38 years). We observed no significant differences in BMI or smoking habits between the two groups (16 *vs.* 13, p=0.407). Although previous studies have suggested a link between high BMI and more severe alopecia, the limited scope of our retrospective study restricts definitive conclusions on this point.[9,10] We concur that further research is needed to examine the relationship between smoking and AGA, particularly from an oxidative stress perspective. Occupation, educational level, and other lifestyle parameters did not show any significant differences in either group. In this study, considering the almost identical numbers of smoking habits and history (21 *vs.* 25; p=0.522), we agree that further research is needed on the relationship between smoking and AGA loss patterns, especially from the perspective of oxidative stress, as mentioned in previous studies. The findings regarding cigarettes should also be examined

alongside other factors, considering that AGA is influenced by genetic and hormonal factors and that smoking can interact with these factors.[11,12]

The median serum ferritin level was slightly lower in the AGA group (222 ng/mL) than in the control group (232 ng/mL), but not statistically significant (p=0.758). This contrasts with studies in women, where a correlation between low serum ferritin levels and hair disorders, such as telogen effluvium, has been established.[11] Our findings suggest that in the general male AGA population, ferritin levels may not be a definitive indicator of the disorder. Several factors, including the study design and variability in laboratory reference ranges, might have contributed to the discrepant results compared with published reports. Examination with a larger sample size is needed to further clarify the correlation between hair iron levels and the occurrence of hair loss disorders, and to evaluate its validity as a potential initial screening method. Several variables may have contributed to the differences between the results of this study and those reported in previous research, including the study design adopted, variability in the definition of normal serum ferritin levels, and the different reference ranges used by laboratories. The median total hair iron level was lower in the AGA group (0.0453 μg/g hair) than in the control group (0.0793 μg/g hair). Similar to that in ferritin, this difference was not statistically significant (p=0.102). We hypothesized that this nonsignificant result may be due to the inclusion of subjects with mild alopecia (Hamilton-Norwood grade 3). We suggest that examining a larger sample may be necessary to fully determine the correlation of hair iron with the occurrence of hair loss.

The correlation between serum ferritin and total hair iron levels with hair density and diameter in the AGA group was not statistically significant. The mean serum ferritin level in the patients with AGA was significantly lower than that in the control group (23.8 *vs.* 62.3 ng/mL). The mechanism of iron deficiency in the pathogenesis of AGA is due to the limited availability of the ribonucleotide reductase cofactor, an enzyme that restricts the rate of DNA synthesis and therefore affects cell proliferation. In addition, iron deficiency also affects transmembrane transport and cell responses to regulatory molecules, as cells in the hair follicle matrix are one of the fastest-dividing cells in the body.[9] On the basis of the results of this study, whether serum ferritin levels are related to AGA or hair loss in the general male population remains unclear. Optimal hair growth occurs if serum ferritin levels are >70 ng/mL, serum vitamin B12 levels are between 300 and 1000 ng/L, and hemoglobin levels are >13.0 g/dL.[13,14] In this study, serum ferritin levels were analyzed, but their role and other factors were not considered.

The exploratory subgroup analysis yielded two distinct, statistically significant correlations. A crucial finding emerged in the subgroup of men with milder AGA (severity Hamilton-Norwood grade <4), where a statistically significant weak positive correlation was found between total hair iron level and

hair diameter (r=0.312, p=0.033). This result supports the theory that in individuals with a mild hereditary predisposition, low iron stores may reduce their threshold for developing alopecia. Consequently, this correlation suggests that adequate hair iron levels contribute to thicker hair diameters and highlights that these patients might be the most likely to benefit from iron supplementation as an adjuvant therapy. By contrast, the analysis of the non-alopecia population revealed a statistically significant weak negative correlation between serum ferritin and total hair iron levels (r=−0.387, p=0.026). This suggests an inverse relationship between systemic iron stores and the iron content sequestered in the hair shaft in men without hair loss. This specific correlation was not observed in the overall AGA population (r=0.009, p=0.961).

In this study, the multivariate analysis revealed no significant association between smoking status or BMI and hair iron or hair ferritin levels after adjusting for potential confounders. This finding suggests that smoking and adiposity may not independently influence iron deposition in hair. Hair iron and ferritin levels likely reflect long-term mineral accumulation and may be less sensitive to lifestyle-related variations than serum iron markers. In addition, iron homeostasis is tightly regulated through absorption and storage mechanisms, which may compensate for the potential effects of smoking-related oxidative stress or BMI-associated inflammation. Dietary iron intake, bioavailability, and individual absorption capacity may therefore play a more dominant role in determining hair iron status.[10,14] Furthermore, the limited variability in smoking exposure or BMI in the study population might have reduced the ability to detect small effect sizes. Overall, these results indicate that smoking and BMI are not major determinants of hair-based iron biomarkers in this population.

Limitations

The hair collection protocol presented a significant limitation because the substantial hair mass required (500 mg) to measure total hair iron level led to the exclusion of many patients with severe AGA who could not provide an adequate sample. This requirement likely resulted in a selection bias, skewing the AGA group toward subjects with mild-to-moderate disease (Hamilton-Norwood grade ≥3). Consequently, the findings are less generalizable to the entire male AGA population, particularly those with advanced hair loss.

Further limitations stem from uncontrolled variables and potential confounders. The analysis focused solely on serum ferritin and hair iron levels, neglecting the role of other critical factors known to affect hair growth, such as vitamin B12, which is considered optimal for hair growth along with ferritin.[13] Similarly, the potential impacts of other interrelated micronutrients were not considered. Regarding lifestyle and environmental factors, while some variables, such as smoking and BMI, were

measured, the study did not fully control for or analyze the complex interactions of oxidative stress with the genetic and hormonal factors underlying AGA. Lastly, although a tendency toward overweight or obesity was noted in the AGA group, the limited parameters evaluated hindered a deeper analysis of the relationship between BMI and alopecia severity due to the retrospective nature of the study.

Conclusions

Overall, this case-control study found that the differences in the mean levels of serum ferritin ($p=0.758$) and total hair iron ($p=0.102$) were not statistically significant between the AGA and non-alopecia groups. However, the subgroup analysis revealed a significant weak positive correlation between total hair iron level and hair diameter in the men with milder AGA ($r=0.312$, $p=0.033$), which suggests that maintaining adequate hair iron levels contributes to thicker hair and supports the use of iron supplementation as a potential adjuvant therapy in early AGA. Conversely, a weak negative correlation between serum ferritin and total hair iron levels was found only in the non-alopecia controls ($r=-0.387$, $p=0.026$). Despite these correlational findings, the conclusions derived from this study are limited by methodological constraints, including selection bias due to the exclusion of patients with severe AGA. Future prospective studies must address these limitations to confirm the role of iron status in male AGA.

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Table 1. Sociodemographic characteristics of subjects.

Characteristic	AGA n=33	Control n=33	p-value
Age (years), mean (SD)	36.97 (7.93)	38 (9.79)	0.640*
Occupation, n (%)			
Private sector	2 (6.1)	0 (0.0)	0.049
Civil servant/State-owned enterprise employees	25 (75.8)	19 (57.6)	
Others	6 (18.2)	14 (42.4)	
Level of education, n (%)			
Secondary	24 (72.7)	23 (69.7)	1.000
Higher	9 (27.3)	10 (30.3)	
BMI, n (%)			
Underweight	5 (15.2)	9 (27.3)	0.407
Normal	12 (36.4)	11 (33.3)	
Overweight	12 (36.4)	7 (21.2)	
Obesity	4 (12.1)	6 (18.2)	
Smoking, n (%)			
Yes	14 (42.4)	18 (54.5)	0.522
History of smoking	7 (21.2)	7 (21.2)	
No	12 (36.4)	8 (24.2)	
Alcohol, n (%)			
History of consumption	1 (3.0)	1 (3.0)	1.000
No	32 (97.0)	32 (97.0)	
AGA degree of severity, median (range)	4 (3-5)	1 (0-1)	<0.001^
Hair density (cm^2), mean (SD)	180.84 (51.70)	191.20 (49.71)	0.410
Hair diameter (mm^2), median (range)	0.04 (0.03-0.04)	0.07 (0.06-0.08)	<0.001*
Hair diameter diversity, n (%)			
Yes	32 (97.0)	4 (12.1)	<0.001
No	1 (3.0)	29 (87.9)	
Peripilar sign, n (%)			
Yes	16 (50.0)	2 (6.1)	<0.001
No	16 (50.0)	31 (93.9)	
Empty follicle, n (%)			
Yes	32 (97)	3 (9.1)	<0.001
No	1 (3)	30 (90.9)	
Yellow dots, n (%)			
Yes	28 (84.8)	5 (15.2)	<0.001
No	5 (15.2)	28 (84.8)	

AGA, androgenetic alopecia; BMI, body mass index; SD, standard deviation; categorical variables were analyzed using the Chi-square test; *independent *t*-test; ^Mann-Whitney U test.

Figure 1. Boxplot of serum ferritin levels in both test groups.

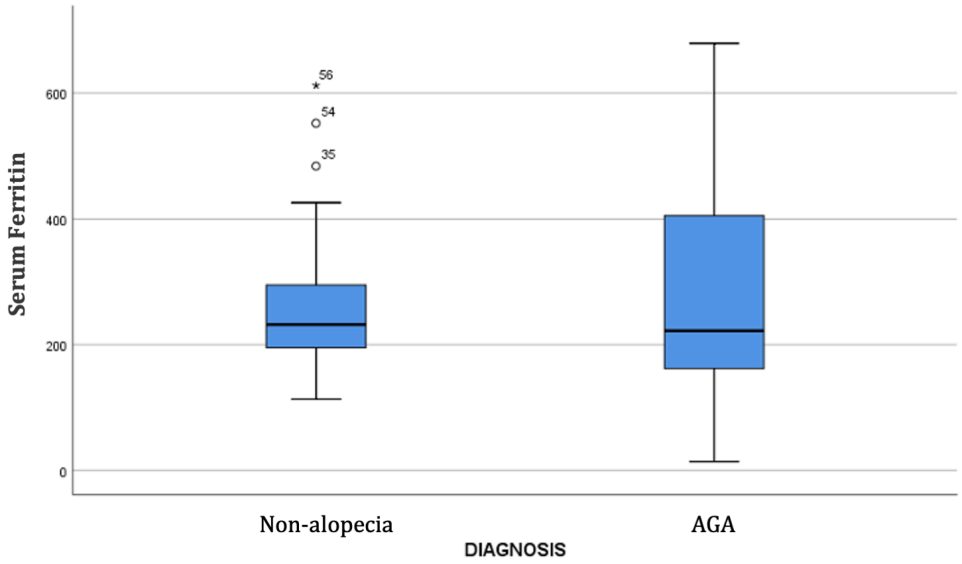

Figure 2. Boxplot of total hair iron content in both test groups.

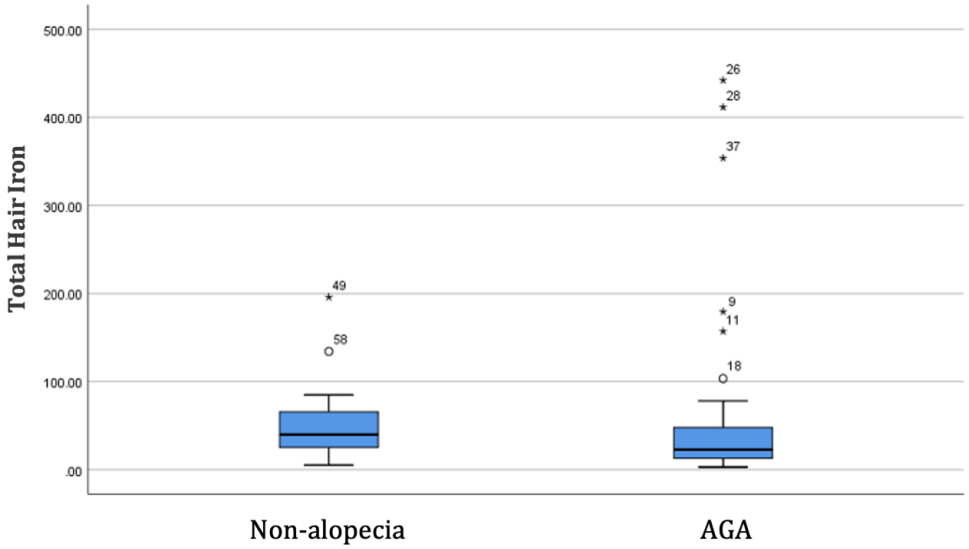

Table 2. Comparison of serum ferritin and hair total iron in the two groups.

Parameter	AGA n=33	Control n=33	p-value
Ferritin serum, median (IQR)	222 (153-406)	232 (194-305)	0.758
Total hair iron, median (IQR)	22.65 (12.26-48.48)	39.67 (24.95-65.72)	0.102

IQR, interquartile range.

Table 3. Correlation analysis of serum ferritin levels and total hair iron with hair diameter and density in the AGA and control groups.

Variable	Hair diameter		Hair density	
	r	p	r	p
AGA				
Serum ferritin	−0.044	0.808	−0.182	0.310
Total hair iron	−0.160	0.374	0.057	0.753
Control				
Serum ferritin	0.043	0.811	−0.269	0.130
Total hair iron	−0.055	0.761	0.269	0.130

AGA, androgenetic alopecia.

Figure 3. Correlation of serum ferritin levels with hair diameter and density.

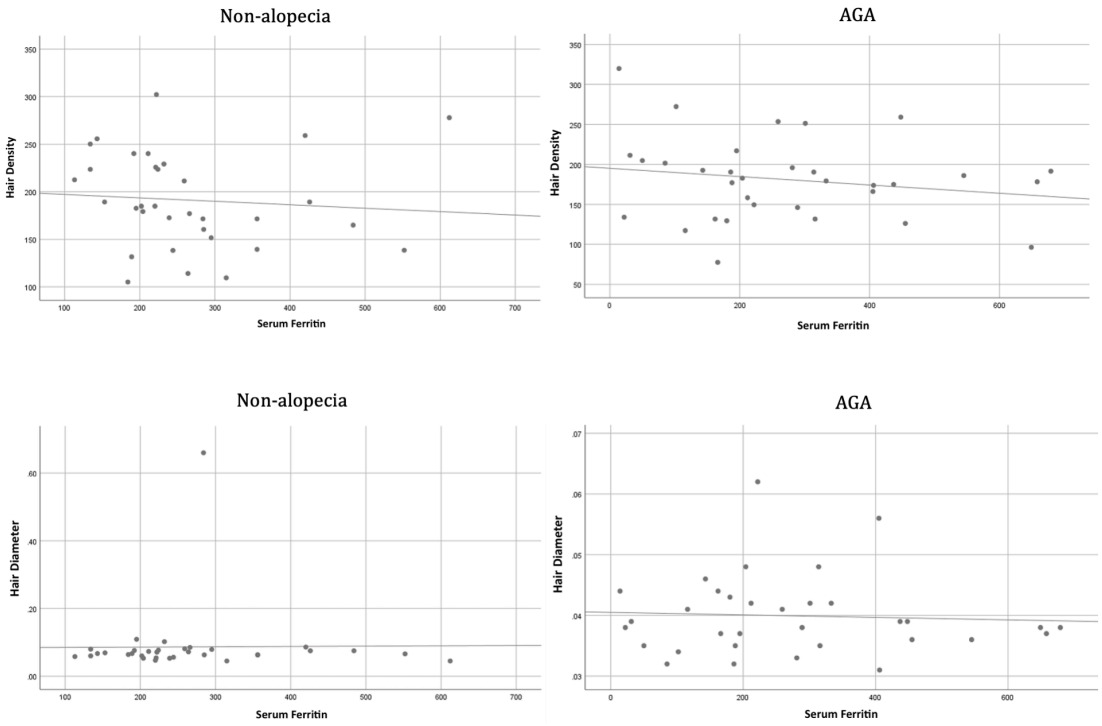

Figure 4. Correlation of hair total iron content with hair density and diameter.