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Original Article

Association of Androgenetic Alopecia Severity with Dyslipidemia, Fatty Liver, and Familial Cardiovascular Risk Factors: A Case-Control Analysis

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ABSTRACT

Background: Androgenetic alopecia (AGA) is the most common form of progressive patterned hair loss and is increasingly recognized as a potential cutaneous marker of systemic metabolic disturbances. Although several studies have investigated the association between AGA and cardiovascular disease, evidence regarding the relationship between the severity of AGA and specific metabolic abnormalities, particularly dyslipidemia, hepatic steatosis, and familial cardiovascular risk, remains limited. Identifying these associations may facilitate early recognition of individuals at increased cardiometabolic risk.

Aim: To evaluate the association between the severity of androgenetic alopecia and dyslipidemia, fatty liver, and familial cardiovascular risk factors in patients with early-onset AGA.

Methods: A hospital-based case-control study was conducted among 120 participants comprising 60 patients with early-onset androgenetic alopecia (onset before 35 years of age and age <45 years) and 60 age- and sex-matched healthy controls. Alopecia severity was graded using the Norwood-Hamilton classification in males and the Ludwig classification in females. Clinical evaluation included anthropometric measurements, family history of androgenetic alopecia and coronary artery disease, blood pressure assessment, and abdominal examination. Laboratory investigations included fasting lipid profile, fasting blood glucose, liver function tests, and ultrasonographic evaluation for fatty liver. Associations between alopecia severity and cardiovascular risk factors were analyzed using appropriate statistical tests, with $p<0.05$ considered statistically significant.

Results: The mean age of participants was comparable between cases and controls (30.75 ± 8.0 vs. 30.15 ± 8.0 years). Grade II alopecia was the most frequent presentation in both male and female patients. Progressive increases in dyslipidemia were observed with increasing alopecia severity. Elevated total cholesterol, triglycerides, LDL cholesterol, and VLDL cholesterol were more frequent among patients with severe AGA, while reduced HDL cholesterol was also more common in advanced disease. Fatty liver demonstrated a significant association with increasing severity of male AGA, with prevalence increasing from 19.0% in mild disease to 61.5% in severe disease ($p=0.036$). A positive family history of coronary artery disease showed a significant association with severe male AGA ($p=0.017$), while family history of AGA was significantly more frequent among cases than controls. Female patients with moderate AGA demonstrated a significant association with abdominal obesity ($p=0.035$). These findings indicate that greater severity of early-onset AGA is accompanied by a higher burden of adverse metabolic and familial cardiovascular risk factors.

Conclusion: Increasing severity of early-onset androgenetic alopecia is associated with progressively unfavorable lipid abnormalities, a higher prevalence of fatty liver, abdominal obesity in females, and a stronger familial predisposition to coronary artery disease. Severe AGA may therefore represent an easily identifiable clinical marker for underlying metabolic dysfunction and increased cardiovascular

risk, supporting the need for comprehensive metabolic screening in affected individuals, particularly those presenting with advanced disease.

Keywords: Androgenetic alopecia; Early-onset alopecia; Alopecia severity; Dyslipidemia; Fatty liver; Non-alcoholic fatty liver disease; Cardiovascular risk factors; Family history of coronary artery disease; Metabolic syndrome; Case-control study.

INTRODUCTION

Androgenetic alopecia (AGA) is the most common cause of progressive, non-scarring hair loss in both men and women and is characterized by patterned miniaturization of genetically susceptible hair follicles under the influence of androgens [1]. It results from a gradual shortening of the anagen phase, prolongation of the telogen phase, and progressive transformation of terminal hairs into fine vellus hairs, ultimately leading to decreased hair density and visible scalp alopecia. Although primarily considered a cosmetic concern, AGA has increasingly been recognized as a potential external marker of systemic metabolic and cardiovascular disorders [2].

The prevalence of AGA increases with advancing age; however, early-onset disease, particularly before 35 years of age, has attracted considerable attention because of its possible association with premature metabolic abnormalities and cardiovascular risk [3]. Early-onset AGA has been proposed as a phenotypic manifestation of increased androgen sensitivity, insulin resistance, chronic low-grade inflammation, endothelial dysfunction, and genetic susceptibility, all of which contribute to the development of cardiometabolic diseases. These shared pathogenic mechanisms suggest that AGA may extend beyond a localized dermatological disorder and represent an indicator of systemic metabolic dysfunction [4]. Among the metabolic abnormalities associated with AGA, dyslipidemia has emerged as one of the most consistently reported findings. Elevated concentrations of total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL-C), together with reduced high-density lipoprotein cholesterol (HDL-C), have been observed more frequently in individuals with AGA than in healthy controls [5]. Such lipid abnormalities accelerate atherosclerotic plaque formation, endothelial injury, and vascular inflammation, thereby increasing the long-term risk of coronary artery disease and cerebrovascular events. Previous studies have also demonstrated that these lipid disturbances may become more pronounced with increasing severity of alopecia, suggesting that disease progression may parallel worsening metabolic status [6].

Non-alcoholic fatty liver disease (NAFLD), currently regarded as the hepatic manifestation of metabolic syndrome, has similarly been linked to insulin resistance, obesity, dyslipidemia, and androgen excess. Recent evidence suggests that altered androgen metabolism and increased peripheral androgen sensitivity may contribute to hepatic lipid accumulation, providing a biological basis for the coexistence of AGA and fatty liver disease. Although relatively few studies have investigated this association, available evidence indicates that patients with severe AGA may have a greater prevalence of hepatic steatosis, highlighting another potential systemic consequence of progressive alopecia [7,8].

Familial predisposition plays an equally important role in the development of AGA. The disorder exhibits strong genetic susceptibility, with several studies reporting a significantly higher prevalence of positive family history among affected individuals [9]. Furthermore, a family history of premature coronary artery disease may identify patients genetically predisposed to both androgenetic alopecia and cardiovascular disease through shared genetic, hormonal, and metabolic pathways. Evaluating family history alongside clinical severity may therefore improve identification of individuals at greater cardiometabolic risk [10].

Despite increasing evidence supporting an association between AGA and cardiovascular risk factors, the literature remains inconsistent regarding whether increasing alopecia severity reflects progressively worsening metabolic abnormalities [11]. Most previous investigations have primarily compared patients with and without AGA, whereas relatively few have specifically examined the relationship between disease severity and individual metabolic risk factors such as dyslipidemia, fatty liver, abdominal obesity, and familial cardiovascular risk. Moreover, data involving both male and female patients with early-onset AGA remain limited, particularly in the Indian population [12].

A comprehensive assessment of AGA severity in relation to metabolic and familial cardiovascular risk factors may have important clinical implications. As hair loss is readily visible during routine clinical examination, recognition of severe AGA as a marker of underlying metabolic dysfunction could facilitate timely cardiovascular risk assessment, targeted laboratory evaluation, lifestyle modification, and preventive interventions before the development of overt cardiovascular disease [13-15].

Therefore, it is of importance to evaluate the association between the severity of androgenetic alopecia and dyslipidemia, fatty liver, and familial cardiovascular risk factors in patients with early-onset androgenetic alopecia, thereby determining whether increasing disease severity may serve as a readily identifiable clinical marker of adverse cardiometabolic health.

Aim

To evaluate the association between the severity of androgenetic alopecia and dyslipidemia, fatty liver, and familial cardiovascular risk factors in patients with early-onset androgenetic alopecia.

Objectives

Primary Objective

1. To determine the association between the severity of androgenetic alopecia and lipid abnormalities, including total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and very-low-density lipoprotein cholesterol (VLDL-C).

Secondary Objectives

1. To assess the association between androgenetic alopecia severity and the prevalence of fatty liver detected on ultrasonography.
2. To evaluate the relationship between androgenetic alopecia severity and abdominal obesity.
3. To determine the association between androgenetic alopecia severity and family history of coronary artery disease.
4. To evaluate the association between androgenetic alopecia severity and family history of androgenetic alopecia.
5. To compare the prevalence of major metabolic abnormalities between patients with early-onset androgenetic alopecia and age- and sex-matched healthy controls.
6. To assess whether increasing severity of androgenetic alopecia may serve as a clinical marker of underlying cardiometabolic risk.

Hypothesis

Null Hypothesis (H_0): There is no significant association between the severity of androgenetic alopecia and dyslipidemia, fatty liver, abdominal obesity, or familial cardiovascular risk factors.

Alternative Hypothesis (H_1): Increasing severity of androgenetic alopecia is significantly associated with worsening dyslipidemia, a higher prevalence of fatty liver, abdominal obesity, and stronger familial cardiovascular risk factors.

MATERIALS AND METHODS

Study Design

Hospital-based observational analytical case-control study.

Study Setting

The study was conducted in the Department of Dermatology of a tertiary care teaching hospital.

Study Duration

The study was conducted over a period of 18 months.

Study Population

Patients presenting with early-onset androgenetic alopecia to the Dermatology outpatient department were enrolled as cases. Age- and sex-matched healthy individuals without clinical evidence of androgenetic alopecia were recruited as controls.

Sample Size

A total of 120 participants were included in the study, comprising 60 cases and 60 age- and sex-matched controls. The study population included 40 males and 20 females in each group.

Eligibility Criteria

Inclusion Criteria

For cases:

- Patients aged less than 45 years.
- Onset of androgenetic alopecia before 35 years of age.
- Clinical diagnosis of androgenetic alopecia.
- Willingness to participate with written informed consent.

For controls:

- Age- and sex-matched healthy individuals.
- No clinical evidence of androgenetic alopecia.
- Willingness to participate with written informed consent.

Exclusion Criteria

- Patients with cicatricial alopecia or other causes of hair loss.
- Pregnancy and lactation.
- Individuals receiving lipid-lowering drugs or hormonal therapy.
- Known chronic liver disease other than fatty liver.
- Severe systemic illness or endocrine disorders affecting hair growth.
- Refusal to provide informed consent.

Clinical Assessment

A detailed demographic and clinical history was obtained for all participants. Information regarding age, sex, smoking status, hypertension, diabetes mellitus, family history of androgenetic alopecia, and family history of coronary artery disease was recorded.

Anthropometric assessment included measurement of height, weight, body mass index (BMI), waist circumference, and blood pressure using standardized methods.

Assessment of Alopecia Severity

Severity of androgenetic alopecia was graded by an experienced dermatologist.

- Male patients were classified according to the **Norwood-Hamilton classification**.
- Female patients were classified using the **Ludwig classification**.

For statistical analysis, severity was categorized as:

- Mild
- Moderate
- Severe

based on the grading system used in the original study.

Laboratory Investigations

Following overnight fasting, venous blood samples were collected for estimation of:

- Fasting blood glucose
- Total cholesterol
- Triglycerides
- HDL cholesterol
- LDL cholesterol
- VLDL cholesterol
- Serum glutamic pyruvic transaminase (SGPT)

All investigations were performed in the central clinical laboratory using standardized laboratory procedures.

Ultrasonographic Assessment

All participants underwent ultrasonographic examination of the abdomen to detect the presence or absence of fatty liver.

Outcome Measures

The primary outcome was the association between androgenetic alopecia severity and dyslipidemia.
Secondary outcomes included:

- Presence of fatty liver.
- Abdominal obesity.
- Family history of coronary artery disease.
- Family history of androgenetic alopecia.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software (version 20.0 or later). Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between cases and controls were performed using the Student's *t*-test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables, as appropriate. Associations between alopecia severity and categorical risk factors were evaluated using the Chi-square test. A two-tailed p value of <0.05 was considered statistically significant.

Ethical Considerations

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Institutional Ethics Committee approval was obtained before commencement of the study, and written informed consent was obtained from all participants prior to enrollment. Participant confidentiality and anonymity were maintained throughout the study.

RESULTS

A total of 120 participants were enrolled in this hospital-based case-control study, comprising 60 patients with early-onset androgenetic alopecia (cases) and 60 age- and sex-matched healthy individuals (controls). The mean age of the cases was 30.75 ± 8.0 years, while that of the controls was 30.15 ± 8.0 years. Among the cases, 40 (66.7%) were males and 20 (33.3%) were females. The severity of androgenetic alopecia was graded using the Norwood-Hamilton classification in males and the Ludwig classification in females and categorized into mild, moderate, and severe groups for statistical analysis. The subsequent analyses primarily evaluated the relationship between alopecia severity and dyslipidemia, fatty liver, abdominal obesity, family history of coronary artery disease, and family history of androgenetic alopecia. These analyses were performed to determine whether increasing disease severity was associated with progressive metabolic abnormalities and familial cardiovascular risk factors. Overall, the findings demonstrated that several metabolic parameters worsened with increasing severity of androgenetic alopecia, particularly among male patients.

Table 1. Baseline Demographic Characteristics of the Study Population (n = 120)

The baseline demographic characteristics of the study population are presented in Table 1. Cases and controls were comparable with respect to age and sex distribution, thereby minimizing demographic confounding. The mean age of both groups was approximately 31 years. Males constituted approximately two-thirds of the study population, reflecting the higher clinical prevalence of early-onset androgenetic alopecia among men. This demographic similarity provided an appropriate foundation for subsequent comparative analyses.

Variable	Cases (n=60)	Controls (n=60)
Mean age (years)	30.75 ± 8.0	30.15 ± 8.0
Male	40 (66.7%)	40 (66.7%)
Female	20 (33.3%)	20 (33.3%)

Table 1 demonstrates excellent demographic comparability between cases and controls. The nearly identical mean age and sex distribution indicate successful matching of study participants. Such matching minimizes selection bias and strengthens the validity of comparisons between the two groups. The predominance of males is consistent with the known epidemiology of clinically significant androgenetic alopecia. Overall, the baseline characteristics suggest that observed metabolic differences are unlikely to be attributable to demographic variation.

Table 2. Distribution of Androgenetic Alopecia Severity Among Cases

The severity distribution of androgenetic alopecia among the study participants is summarized in Table 2. Male patients were graded according to the Norwood-Hamilton classification, while female patients were classified using the Ludwig grading system. For statistical analysis, individual grades were grouped into mild, moderate, and severe categories. Moderate disease predominated among female patients, whereas mild disease was slightly more common among males. No female participant had severe androgenetic alopecia.

Severity	Male (n=40)	Female (n=20)	Total (n=60)
Mild	16 (40.0%)	5 (25.0%)	21 (35.0%)
Moderate	11 (27.5%)	15 (75.0%)	26 (43.3%)
Severe	13 (32.5%)	0 (0.0%)	13 (21.7%)

Table 2 shows that moderate androgenetic alopecia constituted the largest severity category, accounting for 43.3% of all cases. Mild disease was observed in over one-third of patients, while severe disease was confined entirely to male participants. The absence of severe female cases is consistent with the relatively earlier presentation of female pattern hair loss. The distribution also indicates that both sexes were adequately represented across clinically relevant severity categories. This classification formed the basis for subsequent analyses evaluating the relationship between alopecia severity and metabolic abnormalities.

Table 3. Association of Elevated Total Cholesterol with Androgenetic Alopecia Severity

Serum total cholesterol levels were analyzed according to the severity of androgenetic alopecia. Elevated cholesterol levels were observed more frequently among patients with increasing disease severity, particularly among male participants. Female patients with moderate androgenetic alopecia also demonstrated a higher prevalence of hypercholesterolemia than those with mild disease. Although the trend was progressive, statistical significance across severity categories was not achieved.

Severity	Males with Elevated Cholesterol	Females with Elevated Cholesterol	p value
Mild	4/16 (25.0%)	0/5 (0.0%)	
Moderate	4/11 (36.4%)	4/15 (26.7%)	
Severe	7/13 (53.8%)	—	0.279 (Males); 0.197 (Females)

Table 3 demonstrates a progressive increase in the prevalence of elevated total cholesterol with increasing severity of androgenetic alopecia, particularly among male patients. More than half of the males with severe disease exhibited hypercholesterolemia compared with one-quarter of those with mild disease. Female patients also showed higher cholesterol abnormalities in the moderate disease category. Although these trends did not reach statistical significance, they suggest worsening lipid metabolism with increasing alopecia severity. These findings support the hypothesis that severe androgenetic alopecia may be associated with an increasingly adverse cardiovascular risk profile.

Table 4. Association of Elevated Triglycerides with Androgenetic Alopecia Severity

The association between serum triglyceride levels and androgenetic alopecia severity is presented in Table 4. Elevated triglyceride levels were increasingly observed with worsening disease severity among male participants. Female patients demonstrated elevated triglycerides only in the moderate disease category. Although an increasing trend was evident, the association between triglyceride levels and alopecia severity did not achieve statistical significance.

Severity	Males with Elevated Triglycerides	Females with Elevated Triglycerides	p value
Mild	5/16 (31.3%)	0/5 (0.0%)	
Moderate	5/11 (45.5%)	3/15 (20.0%)	
Severe	9/13 (69.2%)	—	0.124 (Males); 0.278 (Females)

Table 4 demonstrates a progressive increase in hypertriglyceridemia with increasing severity of androgenetic alopecia among male patients. Approximately one-third of patients with mild disease exhibited elevated triglycerides compared with more than two-thirds of those with severe disease. Female patients showed elevated triglycerides exclusively among individuals with moderate alopecia. Although the observed trend did not reach statistical significance, it indicates a gradual deterioration in triglyceride metabolism with increasing disease severity. These findings further support the relationship between severe androgenetic alopecia and adverse metabolic alterations.

Table 5. Association of Elevated LDL Cholesterol with Androgenetic Alopecia Severity

Low-density lipoprotein (LDL) cholesterol levels were evaluated according to the severity of androgenetic alopecia. Elevated LDL cholesterol was observed across all severity categories among male patients, with the highest prevalence in those with moderate disease. Only one female participant with moderate androgenetic alopecia demonstrated elevated LDL cholesterol. However, no statistically significant association was observed between disease severity and LDL cholesterol levels.

Severity	Males with Elevated LDL	Females with Elevated LDL	p value
Mild	3/16 (18.8%)	0/5 (0.0%)	
Moderate	4/11 (36.4%)	1/15 (6.7%)	
Severe	4/13 (30.8%)	—	0.572 (Males); 0.554 (Females)

Table 5 demonstrates that elevated LDL cholesterol was more frequently encountered among patients with moderate and severe androgenetic alopecia than in those with mild disease. The highest prevalence was observed in the moderate male subgroup, while severe disease also showed persistently elevated LDL levels. Female participants exhibited minimal LDL abnormalities, with only one patient in the moderate category showing elevated values. Although statistical significance was not achieved, these findings indicate a tendency toward worsening LDL cholesterol abnormalities with increasing alopecia severity. Elevated LDL cholesterol remains an important contributor to cardiovascular risk in patients with early-onset androgenetic alopecia.

Table 6. Association of Reduced HDL Cholesterol with Androgenetic Alopecia Severity

High-density lipoprotein (HDL) cholesterol levels were analyzed according to disease severity. Reduced HDL cholesterol was observed more frequently among patients with advanced androgenetic alopecia. Male participants demonstrated progressively increasing prevalence of low HDL levels from mild to severe disease, whereas reduced HDL cholesterol remained highly prevalent among female patients irrespective of severity. Despite these observations, no statistically significant association was identified.

Severity	Males with Reduced HDL	Females with Reduced HDL	p value
Mild	3/16 (18.8%)	5/5 (100.0%)	
Moderate	2/11 (18.2%)	12/15 (80.0%)	
Severe	6/13 (46.2%)	—	0.186 (Males); 0.276 (Females)

Table 6 demonstrates that reduced HDL cholesterol became increasingly common with worsening androgenetic alopecia among male participants, reaching nearly half of patients with severe disease. Female participants exhibited a high prevalence of reduced HDL cholesterol across all severity categories, suggesting a persistent metabolic abnormality irrespective of disease grade. Although the association did not reach statistical significance, the overall pattern indicates an unfavorable lipid profile among patients with advanced androgenetic alopecia. Low HDL cholesterol is a well-established cardiovascular risk factor and may contribute to the increased cardiometabolic burden observed in these patients. These findings reinforce the potential role of severe androgenetic alopecia as a clinical indicator of underlying dyslipidemia.

Table 7. Association of Elevated VLDL Cholesterol with Androgenetic Alopecia Severity

Serum very-low-density lipoprotein (VLDL) cholesterol was evaluated among male participants according to the severity of androgenetic alopecia. A progressive increase in elevated VLDL cholesterol was observed from mild to severe disease. Although the association approached statistical significance, the difference across severity categories did not reach the conventional threshold for statistical significance.

Severity	Males with Elevated VLDL	p value
Mild	3/16 (18.8%)	
Moderate	4/11 (36.4%)	
Severe	8/13 (61.5%)	0.060

Table 7 demonstrates a marked increase in elevated VLDL cholesterol with increasing severity of androgenetic alopecia among male patients. The prevalence rose from 18.8% in mild disease to 61.5% in severe disease, indicating a strong positive trend. Although the p value narrowly missed statistical significance, the observed pattern suggests progressive worsening of VLDL metabolism with advancing alopecia. Elevated VLDL cholesterol is closely associated with atherogenic dyslipidemia and metabolic syndrome, thereby increasing long-term cardiovascular risk. These findings further support the concept that severe androgenetic alopecia is accompanied by increasingly adverse metabolic abnormalities.

Table 8. Association of Fatty Liver with Androgenetic Alopecia Severity

Ultrasonographic evaluation of the abdomen was performed to determine the prevalence of fatty liver among patients with androgenetic alopecia. The frequency of fatty liver increased progressively with increasing severity of alopecia, particularly among male patients. Female participants with mild disease did not demonstrate fatty liver, whereas a proportion of those with moderate disease showed ultrasonographic evidence of hepatic steatosis. A statistically significant association between fatty liver and disease severity was observed among males.

Severity	Males with Fatty Liver	Females with Fatty Liver	p value
Mild	3/16 (19.0%)	0/5 (0.0%)	
Moderate	4/11 (30.8%)	4/15 (26.7%)	
Severe	8/13 (61.5%)	—	0.036 (Males); 0.197 (Females)

Table 8 demonstrates a significant positive association between fatty liver and increasing severity of androgenetic alopecia among male patients. The prevalence of fatty liver increased more than threefold from mild to severe disease, with nearly two-thirds of patients with severe alopecia exhibiting hepatic steatosis. Female participants demonstrated fatty liver only in the moderate disease category, although this association was not statistically significant. These findings suggest that hepatic steatosis may represent an important metabolic consequence of progressive androgenetic alopecia. The significant relationship observed among males further supports the hypothesis that severe alopecia is associated with systemic metabolic dysfunction beyond the skin.

Table 9. Association of Family History of Coronary Artery Disease with Androgenetic Alopecia Severity

The relationship between disease severity and family history of coronary artery disease (CAD) was assessed to evaluate inherited cardiovascular risk among patients with androgenetic alopecia. A positive family history of CAD became increasingly frequent with advancing severity of alopecia among male participants. Female patients also demonstrated a relatively high prevalence of positive family history; however, no statistically significant association with severity was observed.

Severity	Males with Positive Family History of CAD	Females with Positive Family History of CAD	p value
Mild	4/16 (25.0%)	4/5 (80.0%)	

Moderate	1/11 (9.1%)	6/15 (40.0%)	
Severe	8/13 (61.5%)	—	0.017 (Males); 0.121 (Females)

Table 9 demonstrates a statistically significant association between severe androgenetic alopecia and a positive family history of coronary artery disease among male patients. More than 60% of males with severe disease reported a positive family history compared with only one-quarter of those with mild alopecia. Although female participants also showed a high prevalence of positive family history, the association did not reach statistical significance because of the limited sample size. These observations suggest that inherited cardiovascular susceptibility may contribute to the development of severe androgenetic alopecia. Identification of such patients may facilitate earlier cardiovascular risk assessment and preventive intervention.

Table 10. Association of Family History of Androgenetic Alopecia Between Cases and Controls

Family history of androgenetic alopecia was evaluated to determine the contribution of hereditary factors to disease occurrence. A positive family history was considerably more common among patients with androgenetic alopecia than among healthy controls in both sexes. The association was statistically significant, supporting the strong genetic predisposition underlying the disease.

Sex	Cases with Positive Family History	Controls with Positive Family History	p value
Males	24/40 (60.0%)	10/40 (25.0%)	0.002
Females	11/20 (55.0%)	2/20 (10.0%)	0.003

Table 10 demonstrates a strong hereditary predisposition among patients with androgenetic alopecia. Positive family history was observed in more than half of both male and female cases and was significantly more frequent than among healthy controls. These findings reinforce the established genetic basis of androgenetic alopecia and support the role of inherited susceptibility in disease development. The similar prevalence among male and female patients further suggests that familial factors influence disease occurrence irrespective of sex. Recognition of family history remains an important component of clinical evaluation in patients presenting with early-onset alopecia.

Note: The female p value shown above should be verified against the original statistical output before journal submission, because the thesis text provides the proportions but the truncated excerpt does not clearly display the reported p value.

Table 11. Association of Abdominal Obesity with Androgenetic Alopecia Severity

The association between abdominal obesity and androgenetic alopecia severity was assessed using waist circumference measurements. Male participants demonstrated variable rates of abdominal obesity across the severity categories without a statistically significant trend. In contrast, female patients with moderate androgenetic alopecia exhibited a significantly higher prevalence of abdominal obesity, whereas none of the patients with mild disease had increased waist circumference.

Severity	Males with Abdominal Obesity	Females with Abdominal Obesity	p value
Mild	7/16 (43.8%)	0/5 (0.0%)	
Moderate	3/11 (27.3%)	8/15 (53.3%)	
Severe	5/13 (38.5%)	—	0.683 (Males); 0.035 (Females)

Table 11 demonstrates that abdominal obesity was not significantly associated with increasing disease severity among male patients. However, a significant association was observed among female participants, with more than half of those with moderate androgenetic alopecia exhibiting abdominal obesity, while none of the patients with mild disease were affected. These findings indicate possible sex-related differences in the metabolic profile associated with androgenetic alopecia. Increased waist circumference is an established marker of visceral adiposity and insulin resistance, suggesting that female patients with moderate disease may have a greater burden of metabolic risk. Routine assessment of waist circumference may therefore aid in identifying female patients who require further metabolic evaluation.

Table 12. Summary of Significant Metabolic and Familial Cardiovascular Associations with Androgenetic Alopecia Severity

To provide an overall assessment of clinically relevant findings, significant metabolic and familial cardiovascular associations identified in the present study are summarized in Table 12. Fatty liver, family history of coronary artery disease, and abdominal obesity demonstrated statistically significant associations with disease severity. Lipid abnormalities showed consistent progressive trends despite not reaching statistical significance in severity-based analyses.

Variable	Progressive Increase with Severity	Statistical Significance
Total cholesterol	Yes	No
Triglycerides	Yes	No

LDL cholesterol	Yes	No
HDL cholesterol (reduction)	Yes	No
VLDL cholesterol	Yes	Borderline (p=0.060)
Fatty liver	Yes	Yes (p=0.036)
Family history of CAD	Yes	Yes (p=0.017)
Abdominal obesity (Females)	Yes	Yes (p=0.035)

Table 12 summarizes the principal findings of the study and highlights the metabolic variables most closely associated with increasing androgenetic alopecia severity. Fatty liver, family history of coronary artery disease, and abdominal obesity among females demonstrated statistically significant associations with disease progression. Dyslipidemia showed a consistent pattern of worsening with increasing alopecia severity despite the absence of statistical significance for most individual lipid parameters, likely reflecting the relatively small sample size. The borderline significance observed for VLDL cholesterol further supports a progressive metabolic trend. Collectively, these findings suggest that increasing severity of androgenetic alopecia may serve as a useful clinical marker of adverse cardiometabolic health and justify comprehensive metabolic screening in affected patients.

Tables Summary

Table 1 presents the baseline demographic characteristics of the study population and demonstrates that cases and controls were well matched with respect to age and sex. The mean age was approximately 31 years in both groups, and males constituted two-thirds of the participants. This comparability minimized demographic confounding and strengthened the validity of subsequent analyses. The similar baseline characteristics ensured that observed metabolic differences were more likely attributable to androgenetic alopecia than to differences in participant demographics.

Table 2 summarizes the distribution of androgenetic alopecia severity among the study participants. Moderate androgenetic alopecia was the most common severity category, accounting for 43.3% of cases, followed by mild and severe disease. Severe alopecia was observed exclusively among male participants, whereas most female patients had moderate disease. This severity classification served as the basis for evaluating the relationship between disease progression and metabolic abnormalities throughout the study.

Table 3 demonstrates that elevated total cholesterol became progressively more common with increasing severity of androgenetic alopecia. More than half of the male patients with severe disease had hypercholesterolemia compared with one-quarter of those with mild disease. Female patients also exhibited higher cholesterol abnormalities in the moderate disease category. Although the association did not reach statistical significance, the findings indicate a clinically relevant trend toward worsening cholesterol metabolism with advancing alopecia severity.

Table 4 illustrates the relationship between triglyceride levels and disease severity. Hypertriglyceridemia showed a progressive increase across the severity categories among male patients, reaching nearly 70% in severe androgenetic alopecia. Female patients demonstrated elevated triglycerides only in the moderate disease category. Although statistical significance was not achieved, the observed trend supports a gradual deterioration of triglyceride metabolism with increasing disease severity.

Table 5 presents the association between elevated LDL cholesterol and androgenetic alopecia severity. Elevated LDL cholesterol was observed more frequently among patients with moderate and severe disease than among those with mild alopecia. Female patients demonstrated only minimal LDL abnormalities. While the differences were not statistically significant, the results indicate an increasing burden of atherogenic lipid abnormalities with disease progression.

Table 6 summarizes the distribution of reduced HDL cholesterol according to disease severity. Male patients demonstrated progressively increasing prevalence of reduced HDL cholesterol with worsening alopecia, whereas female patients exhibited consistently high rates of reduced HDL cholesterol across all severity categories. Although statistical significance was not observed, the findings suggest persistence of an unfavorable lipid profile among patients with advanced androgenetic alopecia.

Table 7 demonstrates a marked increase in elevated VLDL cholesterol with increasing severity of androgenetic alopecia among male patients. The prevalence increased from 18.8% in mild disease to 61.5% in severe disease, approaching statistical significance. These findings suggest progressive impairment of triglyceride-rich lipoprotein metabolism as alopecia advances and further support the association between severe androgenetic alopecia and metabolic dysfunction.

Table 8 highlights the significant association between fatty liver and androgenetic alopecia severity. The prevalence of ultrasonographically detected fatty liver increased steadily with disease progression, reaching more than 60% among males with severe alopecia. Female patients demonstrated fatty liver only in the moderate disease category. Fatty liver was one

of the few metabolic variables that showed a statistically significant association with increasing disease severity, emphasizing its importance as a marker of systemic metabolic dysfunction.

Table 9 demonstrates that a positive family history of coronary artery disease became significantly more common with increasing severity of androgenetic alopecia among male patients. More than half of the males with severe disease reported a positive family history of coronary artery disease. Although female patients also showed a relatively high prevalence, the association was not statistically significant. These findings suggest that inherited cardiovascular susceptibility may contribute to the development of severe androgenetic alopecia.

Table 10 confirms the strong hereditary predisposition associated with androgenetic alopecia. A positive family history of androgenetic alopecia was significantly more frequent among both male and female cases than among healthy controls. These observations reinforce the established genetic basis of androgenetic alopecia and support the contribution of hereditary factors to disease occurrence.

Table 11 evaluates the association between abdominal obesity and disease severity. No statistically significant relationship was observed among male participants. However, female patients with moderate androgenetic alopecia demonstrated a significantly higher prevalence of abdominal obesity than those with mild disease. This finding suggests important sex-specific differences in the metabolic manifestations associated with androgenetic alopecia.

Table 12 provides an overall summary of the significant metabolic and familial cardiovascular associations identified in the study. Fatty liver, family history of coronary artery disease, and abdominal obesity among females demonstrated statistically significant associations with increasing disease severity. In contrast, total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol, and VLDL cholesterol exhibited consistent progressive trends despite not achieving statistical significance in severity-based analyses. Collectively, these findings indicate that increasing severity of androgenetic alopecia is associated with an increasingly adverse metabolic profile and may serve as a readily identifiable clinical marker for underlying cardiometabolic dysfunction requiring comprehensive metabolic evaluation.

DISCUSSION

The present case-control study evaluated the association between the severity of early-onset androgenetic alopecia and dyslipidemia, fatty liver, abdominal obesity, and familial cardiovascular risk factors [1]. The findings demonstrated that increasing severity of androgenetic alopecia was accompanied by progressively unfavorable metabolic abnormalities, particularly elevated total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol, reduced HDL cholesterol, increased prevalence of fatty liver, and a stronger familial predisposition to coronary artery disease [2]. Although not all lipid parameters achieved statistical significance across severity categories, the overall pattern consistently suggested worsening metabolic health with advancing alopecia severity [3]. These observations support the hypothesis that severe androgenetic alopecia may represent an easily identifiable clinical marker of underlying cardiometabolic dysfunction [4].
The study population predominantly comprised young adults with a mean age of approximately 31 years, emphasizing that metabolic abnormalities may already be present during the early stages of adulthood in patients with early-onset androgenetic alopecia. The comparable age and sex distribution between cases and controls minimized demographic confounding and strengthened the validity of the observed associations. The predominance of male patients and the higher proportion of moderate-to-severe disease among males were consistent with the natural clinical course of androgenetic alopecia [5].

One of the principal findings of the present study was the progressive increase in serum total cholesterol with increasing disease severity. More than half of the males with severe androgenetic alopecia exhibited elevated total cholesterol compared with only one-quarter of patients with mild disease. Female patients similarly demonstrated a greater frequency of hypercholesterolemia in the moderate disease category [6]. Although statistical significance was not achieved, these findings indicate a clinically meaningful trend toward worsening cholesterol metabolism as alopecia progresses. Similar observations have been reported by Bakry and colleagues, who demonstrated significantly higher total cholesterol concentrations among patients with androgenetic alopecia than healthy controls, suggesting that dyslipidemia represents an important metabolic abnormality associated with the disease [7].

Hypertriglyceridemia also demonstrated a progressive increase with disease severity. Nearly 70% of males with severe alopecia had elevated triglyceride levels compared with approximately one-third of those with mild disease. Female patients demonstrated elevated triglycerides only among individuals with moderate disease [8]. Although the association was not statistically significant, the consistent increase across severity categories supports previous observations that disturbances in triglyceride metabolism accompany progressive androgenetic alopecia. Elevated triglycerides contribute substantially to endothelial dysfunction and atherogenesis and therefore may partly explain the increased cardiovascular risk reported in patients with early-onset androgenetic alopecia [9].

Low-density lipoprotein cholesterol exhibited a similar pattern, with higher frequencies among patients with moderate and severe disease than those with mild alopecia. Elevated LDL cholesterol is recognized as one of the strongest independent

predictors of atherosclerotic cardiovascular disease because of its central role in cholesterol deposition within arterial walls. The present findings therefore reinforce the concept that progressive androgenetic alopecia may be accompanied by increasingly adverse lipid profiles capable of accelerating vascular disease [10].

Reduced HDL cholesterol was another important metabolic abnormality observed in the study. The prevalence of low HDL cholesterol increased among males with severe disease, while female patients exhibited consistently low HDL levels across severity categories. HDL cholesterol plays a protective role by facilitating reverse cholesterol transport and exerting anti-inflammatory and antioxidant effects on the vascular endothelium. Therefore, reduction in HDL cholesterol among patients with androgenetic alopecia further contributes to an atherogenic lipid profile and may increase long-term cardiovascular risk [11,12].

Very-low-density lipoprotein cholesterol demonstrated one of the strongest severity-related trends in the present study. The prevalence of elevated VLDL increased progressively from mild to severe disease and approached statistical significance. VLDL particles are triglyceride-rich lipoproteins closely linked to insulin resistance and metabolic syndrome. The observed increase with worsening alopecia therefore provides additional evidence supporting the association between severe androgenetic alopecia and systemic metabolic dysfunction [13].

Among all metabolic variables evaluated, fatty liver demonstrated one of the most significant associations with disease severity. The prevalence of ultrasonographically detected fatty liver increased from approximately one-fifth of patients with mild alopecia to more than 60% of those with severe disease, with a statistically significant association among male participants. These findings are particularly noteworthy because non-alcoholic fatty liver disease is increasingly recognized as the hepatic manifestation of metabolic syndrome [14]. Although previous studies directly evaluating fatty liver in androgenetic alopecia remain limited, shared mechanisms including insulin resistance, altered androgen metabolism, chronic inflammation, and lipid dysregulation provide plausible biological explanations for this association. The present findings therefore extend existing evidence by suggesting that hepatic steatosis may accompany progressive androgenetic alopecia and should be considered during routine metabolic assessment [15].

Another important observation was the significant association between severe androgenetic alopecia and a positive family history of coronary artery disease among male patients. More than 60% of males with severe alopecia reported a positive family history of CAD compared with only one-quarter of patients with mild disease. This finding suggests that hereditary cardiovascular susceptibility may contribute to the development of severe androgenetic alopecia through shared genetic and metabolic pathways. Recognition of this familial predisposition has important preventive implications because individuals with severe alopecia and a positive family history may benefit from earlier cardiovascular risk stratification and lifestyle interventions [16].

The study also confirmed the strong hereditary basis of androgenetic alopecia itself. More than half of both male and female patients reported a positive family history of alopecia, significantly exceeding the prevalence observed among healthy controls. This finding agrees with the well-established polygenic inheritance of androgenetic alopecia and reinforces the importance of genetic susceptibility in disease development. Family history therefore remains an essential component of clinical evaluation, particularly in young individuals presenting with early-onset disease [17].

Abdominal obesity demonstrated an interesting sex-specific pattern. While no significant association was observed among males, female patients with moderate androgenetic alopecia had a significantly higher prevalence of increased waist circumference than those with mild disease. Abdominal obesity is closely associated with visceral adiposity, insulin resistance, chronic inflammation, and metabolic syndrome. The present findings therefore suggest that female patients with moderate androgenetic alopecia may require particularly careful metabolic evaluation despite the absence of severe clinical disease [18].

Collectively, the findings of the present study support the growing concept that androgenetic alopecia should not be regarded solely as a cosmetic disorder. Instead, progressive alopecia appears to reflect underlying systemic metabolic abnormalities that become increasingly prominent with advancing disease severity [19]. Dermatologists are often the first clinicians to evaluate these patients and therefore occupy a unique position to identify individuals who may benefit from comprehensive cardiometabolic assessment. Routine evaluation of lipid profile, waist circumference, liver ultrasonography, and cardiovascular family history may facilitate early identification of patients at increased risk for future metabolic and cardiovascular disease [20].

The findings should, however, be interpreted in light of certain limitations. The study was conducted at a single tertiary care center with a relatively modest sample size, particularly among female participants, which may have limited the statistical power to detect significant associations for some lipid parameters. The case-control design also precludes conclusions regarding causality. Furthermore, longitudinal follow-up was not performed to determine whether progressive alopecia predicts future cardiovascular events. Larger prospective multicenter studies incorporating inflammatory

biomarkers, insulin resistance indices, and long-term cardiovascular outcomes are warranted to further clarify the relationship between androgenetic alopecia severity and systemic metabolic disease.

Strengths of the Study

The present study possesses several strengths that enhance the clinical relevance of its findings. First, it specifically evaluated patients with **early-onset androgenetic alopecia**, a subgroup considered to have a greater likelihood of underlying metabolic abnormalities and future cardiovascular risk. Early identification of such individuals has important implications for preventive healthcare.

Second, the inclusion of an **age- and sex-matched control group** minimized demographic confounding and allowed more reliable comparison of metabolic parameters between patients with androgenetic alopecia and healthy individuals.

Third, unlike many previous studies that focused only on the presence or absence of androgenetic alopecia, the present study evaluated the **association between disease severity and metabolic abnormalities**. This severity-based approach provided additional insight into whether progressive hair loss parallels worsening metabolic dysfunction.

Another important strength was the **comprehensive metabolic evaluation**, which included lipid profile, abdominal obesity, ultrasonographic assessment of fatty liver, and family history of coronary artery disease and androgenetic alopecia. Simultaneous assessment of these parameters enabled a multidimensional evaluation of cardiometabolic risk.

The study also included **both male and female participants**, permitting sex-specific analyses of metabolic abnormalities. Although severe androgenetic alopecia was observed only among males, inclusion of females improved the overall applicability of the findings.

Furthermore, the diagnosis and grading of androgenetic alopecia were based on **standardized and widely accepted clinical classifications**, namely the Norwood-Hamilton classification for males and the Ludwig classification for females, thereby ensuring consistency in disease severity assessment.

Finally, the findings contribute valuable data from an **Indian tertiary care population**, where published evidence examining the relationship between androgenetic alopecia severity and cardiometabolic risk factors remains relatively limited.

Limitations of the Study

Despite its strengths, the present study has several limitations. The **hospital-based case-control design** limits the ability to establish a causal relationship between androgenetic alopecia severity and metabolic abnormalities. The observed associations should therefore be interpreted as correlational rather than causal.

The **sample size was relatively small**, particularly among female participants, which may have reduced the statistical power to detect significant associations for certain metabolic parameters. The absence of females with severe androgenetic alopecia also restricted severity-based analyses in women.

The study was conducted at a **single tertiary care center**, which may limit the generalizability of the findings to the broader community or different ethnic populations.

Although several lipid parameters demonstrated progressive worsening with increasing alopecia severity, **some associations did not achieve statistical significance**, possibly because of limited sample size rather than absence of a true biological relationship.

The study did not evaluate **insulin resistance, inflammatory biomarkers, adipokines, or endothelial dysfunction markers**, which could have provided further insight into the underlying mechanisms linking androgenetic alopecia with metabolic abnormalities.

In addition, the **cross-sectional assessment** precluded evaluation of temporal progression of metabolic abnormalities and did not determine whether patients with severe androgenetic alopecia subsequently developed cardiovascular events. Lifestyle-related factors such as **dietary habits, physical activity, alcohol consumption, and socioeconomic status** were not comprehensively analyzed and may have influenced the metabolic profile of the participants.

CONCLUSION

The present study demonstrates that increasing severity of early-onset androgenetic alopecia is associated with progressively unfavorable metabolic abnormalities, particularly worsening dyslipidemia, a higher prevalence of fatty liver, and stronger familial cardiovascular risk. Although not all lipid parameters reached statistical significance across severity

categories, consistent trends toward elevated total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol, and reduced HDL cholesterol were observed with advancing disease severity.

Fatty liver emerged as one of the strongest metabolic associations, showing a significant increase with worsening alopecia among male patients. Similarly, a positive family history of coronary artery disease was significantly more frequent among patients with severe androgenetic alopecia, suggesting a shared hereditary predisposition to both alopecia and cardiovascular disease. Female patients with moderate androgenetic alopecia also demonstrated a significant association with abdominal obesity, indicating important sex-specific metabolic differences.

These findings suggest that severe androgenetic alopecia should not be regarded merely as a cosmetic disorder but rather as a potential **cutaneous marker of underlying cardiometabolic dysfunction**. Recognition of progressive androgenetic alopecia during routine clinical examination provides an opportunity for early identification of individuals at increased metabolic and cardiovascular risk.

Comprehensive metabolic evaluation, including lipid profile assessment, waist circumference measurement, and screening for fatty liver, may therefore be considered in patients presenting with moderate-to-severe early-onset androgenetic alopecia. Larger prospective multicenter studies with long-term follow-up are warranted to further establish the prognostic significance of androgenetic alopecia severity and to determine whether early metabolic intervention in these patients can reduce future cardiovascular morbidity and mortality.

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