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A study of association of hyperlipidemia and smoking in early onset of Male AGA

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Abstract:

Background: Androgenetic alopecia (AGA) is a common progressive hair loss condition in men, with early-onset cases (<30 years) increasingly linked to systemic risk factors like hyperlipidemia and smoking. This studywas done tofind the association between these modifiable factors and early-onset male AGA.
Method: A hospital-based observational study was conducted at Sri Siddhartha Medical College, Tumkur, over 24 months, involving 75 male patients with early-onset AGA and 75 age-matched controls. Clinical diagnosis was based on the Hamilton-Norwood classification. Smoking history and fasting lipid profiles were documented. Statistical analyses included t-tests, chi-square, and logistic regression.
Results: Participants with AGA had significantly higher levels of total cholesterol, LDL, triglycerides, and lower HDL compared to controls ($p < 0.005$). Severe AGA grades (IV-V+) were more prevalent among smokers and those with hyperlipidemia ($p < 0.05$). Logistic regression showed smoking (OR=3.42, $p<0.001$) and hyperlipidemia (OR=2.18, $p=0.008$) as independent risk factors for early-onset AGA.
Conclusion: Smoking and hyperlipidemia are significantly associated with increased severity and risk of early-onset AGA, showing the need for lifestyle modification and metabolic screening in affected individuals.

Introduction

Androgenetic alopecia (AGA), commonly known as male pattern baldness, is a chronic, progressive condition characterized by hair follicle miniaturization. It affects a significant proportion of men, with early-onset cases (<30 years) raising concerns about potential underlying systemic risk factors. While genetic predisposition plays a primary role, emerging evidence shows that metabolic factors such as hyperlipidemia and smoking may contribute to the pathogenesis and progression of AGA.1

Hyperlipidemia, characterized by increased cholesterol and triglyceride levels, has been linked to microvascular dysfunction and oxidative stress,

potentially accelerating hair follicle aging and miniaturization.2,3 Similarly, smoking has been associated with increased free radical production, DNA damage, and reduced dermal microcirculation, further exacerbating hair loss. Given the increasing prevalence of early-onset AGA and its psychosocial impact, understanding its metabolic and lifestyle-associated risk factors is crucial for early intervention and management.4,5

This study aims to evaluate the association between hyperlipidemia and smoking in men with early-onset AGA, providing insights into potential modifiable risk factors for disease progression.

Method

This hospital-based observational study was conducted at Sri Siddhartha Medical College, Tumkur, over a period of 24 months. A total of 75 male patients with early-onset androgenetic alopecia (AGA) and 75 male without androgenetic alopecia (AGA) as control, aged 18 to 35 years, were included in the study. Patients were diagnosed based on clinical examination using the Hamilton-Norwood classification for AGA.

Institutional Ethics committee permission was obtained before initiation of study. Participants were recruited from the dermatology outpatient department, and written informed consent was obtained prior to enrolment. A detailed history, including the onset and progression of hair loss, smoking habits, and any history of hyperlipidemia or related metabolic disorders, was recorded. Smoking status was classified into non-smokers, current smokers, and former smokers based on self-reported history. Lipid profile assessments, including total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglyceride levels, were conducted after overnight fasting.

Exclusion criteria included individuals with other causes of alopecia (such as alopecia areata, telogen effluvium, or scarring alopecia), those on lipid-lowering medications, patients with systemic illnesses that could affect hair growth, and individuals with a family history of early-onset AGA to minimize genetic predisposition bias.
Statistical analysis was performed using appropriate software, with descriptive statistics presented as means and standard deviations. The correlation between hyperlipidemia, smoking, and AGA severity was assessed using chi-square tests, independent t-tests, and logistic regressionanalysis as applicable. A p-value < 0.05 was considered statistically significant.

Results

Table 1: Baseline Characteristics of Study Participants

Characteristic	Mean ± SD / n (%)
Age (years)	26.4 ± 4.1
BMI (kg/m²)	24.1 ± 3.2
Family History of AGA	22 (29.3%)
Current Smokers	35 (46.7%)
Former Smokers	15 (20.0%)
Non-Smokers	25 (33.3%)
Hyperlipidemia	38 (50.7%)

The study was done among 75 male participants with early-onset androgenetic alopecia (AGA), with a mean age of 26.4 ± 4.1 years. The mean BMI of the participants was 24.1 ± 3.2 kg/m², indicating that most individuals were in the normal to overweight category. A positive family history of AGA was reported in 22 participants (29.3%), showing a genetic predisposition in some cases. Regarding smoking status, 35 participants (46.7%) were current smokers, 15 (20.0%) were former

smokers, and 25 (33.3%) were non-smokers. Hyperlipidemia was seen in 38 participants (50.7%), showing a high prevalence of lipid abnormalities in the study population

Table 2: Lipid Profile in Participants with and without AGA

Lipid Parameter	AGA (n=75)	Control (n=75)	p-value
Total Cholesterol (mg/dL)	205.3 ± 32.1	178.6 ± 28.4	**0.002**
LDL (mg/dL)	132.5 ± 28.3	108.4 ± 24.7	**0.001**
HDL (mg/dL)	42.1 ± 6.5	48.6 ± 5.9	**0.003**
Triglycerides (mg/dL)	178.7 ± 34.2	150.9 ± 30.8	**0.004**

The lipid profile analysis showed significant differences between AGA patients and the control group. The mean total cholesterol level was 205.3 ± 32.1 mg/dL in AGA patients, which was significantly higher than 178.6 ± 28.4 mg/dL in controls (p = 0.002). Similarly, LDL levels were increased in AGA patients (132.5 ± 28.3 mg/dL) compared to controls (108.4 ± 24.7 mg/dL, p = 0.001). Conversely, HDL levels were lower in AGA patients (42.1 ± 6.5 mg/dL) compared to controls (48.6 ± 5.9 mg/dL, p = 0.003). Triglyceride levels were also significantly higher in the AGA group (178.7 ± 34.2 mg/dL) compared to controls (150.9 ± 30.8 mg/dL, p = 0.004). These findings show a strong association between dyslipidemia and early-onset AGA.

Table 3: Smoking Status and AGA Severity (Hamilton-Norwood Classification)

AGA Grade	Non-Smokers (n=25)	Smokers (n=50)	p-value
Grade II	12 (48.0%)	8 (16.0%)	**0.02**
Grade III	9 (36.0%)	15 (30.0%)	**0.15**
Grade IV	4 (16.0%)	18 (36.0%)	**0.03**
Grade V+	0 (0%)	9 (18.0%)	**0.01**

Among non-smokers, the majority had mild AGA (Grade II-III), with 12 (48.0%) having Grade II and 9 (36.0%) having Grade III. In contrast, smokers had a higher proportion of severe AGA, with 18 (36.0%) having Grade IV and 9 (18.0%) having Grade V+, compared to 4 (16.0%) and 0 (0%), respectively, in non-smokers. The differences in severity were statistically significant for Grade II (p = 0.02), Grade IV (p = 0.03), and Grade V+ (p = 0.01), showing that smoking may contribute to more advanced AGA progression.

Table 4: Correlation Between Hyperlipidemia and AGA Severity

AGA Severity	Hyperlipidemia (n=38)	Normal Lipids (n=37)	p-value
Mild (Grade II–III)	15 (39.5%)	22 (59.5%)	**0.04**
Moderate (Grade IV)	14 (36.8%)	10 (27.0%)	**0.12**
Severe (Grade V+)	9 (23.7%)	5 (13.5%)	**0.03**

Among participants with mild AGA (Grade II–III), 39.5% had hyperlipidemia, while the remaining 59.5% had normal lipid levels (p = 0.04). However, in the moderate AGA (Grade IV) group, hyperlipidemia was present in 36.8% compared to 27.0% in the normal lipid group, though the difference was not statistically significant (p = 0.12). In the severe AGA (Grade V+) group, 23.7% of participants had hyperlipidemia compared to only 13.5% in those with normal lipid levels, with a significant p-value of 0.03. These findings indicate that hyperlipidemia is more prevalent in severe AGA cases.

Table 5: Logistic Regression Analysis of Risk Factors for Early-Onset AGA

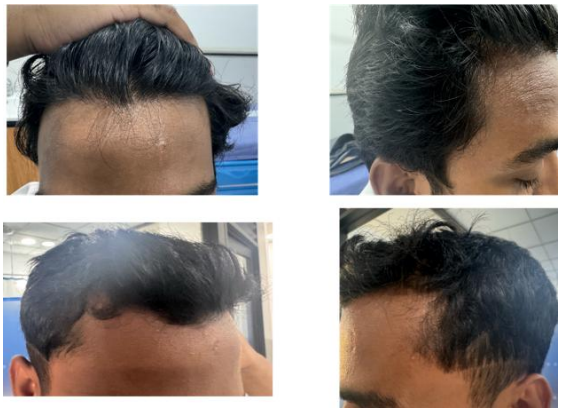
Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
Hyperlipidemia	2.18	1.24 – 3.89	**0.008**
Smoking	3.42	1.76 – 5.23	**<0.001**
Family History	1.91	0.96 – 3.21	0.07
BMI >25	1.56	0.84 – 2.85	0.09

Hyperlipidemia was associated with an increased risk of AGA (OR = 2.18, 95% CI: 1.24–3.89, p = 0.008), confirming a significant association. Smoking showed the strongest association with early-onset AGA (OR = 3.42, 95% CI: 1.76–5.23, p < 0.001), indicating that smokers were more than three times more likely to develop AGA than non-smokers. A positive family history of AGA showed a borderline association (OR = 1.91, p = 0.07), while BMI > 25 did not show a statistically significant association (OR = 1.56, p = 0.09). These findings that hyperlipidemia and smoking are independent risk factors for early-onset AGA, whereas family history and BMI may play a contributory but less significant role.

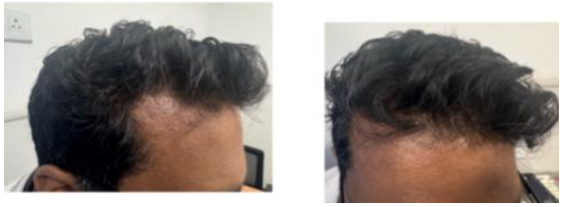
Discussion

Our study evaluated the association between hyperlipidemia and smoking in the early onset of male androgenetic alopecia (AGA), showing importance of the metabolic and environmental factors influencing this condition. The mean age of our participants was 26.4 ± 4.1 years, similar to previous studies that predominantly focused on early-onset AGA cases in men aged 18-30 years (Ekmekci et al., 2011[6]; Kaya Erdogan et al., 2016[7]; Ertas et al., 2015 [8]). The consistency in the age distribution across studies reinforces the notion that AGA manifests in early adulthood, showing the need for early intervention strategies.

Grade 1

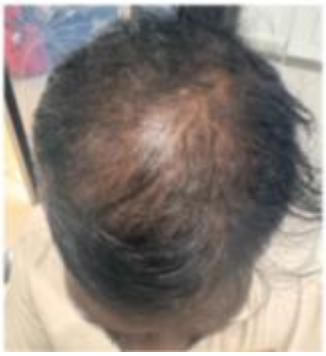


Grade 2



Grade 3

Grade 4



In our study, the mean BMI of participants was 24.1 ± 3.2 kg/m², which falls within the normal range but trends toward the higher end. Previous studies, such as those by Ertas et al. (2015)[8] and Hirsso et al. (2007)[9], have reported a higher BMI among AGA patients compared to controls. This aligns with findings from Wang et al. (2024)[10], which demonstrated a higher prevalence of metabolic syndrome among AGA patients, showing a possible link between AGA and metabolic disturbances.
Family history was present in 29.3% of our participants, which is in line with prior studies showing the genetic predisposition of AGA (Kaya Erdogan et al., 2016[7]; Tosti et al., 2005[11]). The hereditary nature of AGA is well-documented, with research showing that genetic factors contribute significantly to disease onset and progression.

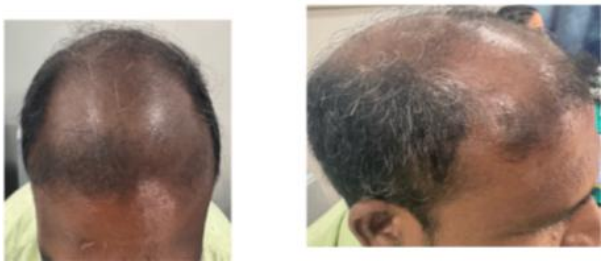
Smoking was a significant factor in our study, with 46.7% of participants being current smokers and 20.0% being former smokers. This supports previous studies showing a positive association between smoking and AGA severity. Su and Chen (2007)[12] reported an increased risk of moderate to severe AGA among smokers (OR: 1.77, 95% CI: 1.14-2.76), with the risk further elevated in heavy smokers consuming over 20 cigarettes per day (OR: 2.34, 95% CI: 1.19-4.59). These findings, similar to our findings, show that smoking may accelerate AGA progression, possibly through mechanisms involving oxidative stress and microvascular impairment.

Hyperlipidemia was present in 50.7% of AGA patients in our study, showing its potential role in AGA pathogenesis. Studies by Arias-Santiago et al. (2010)[13] and Ertas et al. (2015)[8] reported significantly elevated lipid profiles in AGA patients, with higher total cholesterol, LDL, and triglyceride levels. Similarly, our lipid profile data show significantly higher total cholesterol (205.3 ± 32.1 mg/dL vs. 178.6 ± 28.4 mg/dL, p=0.002), LDL (132.5 ± 28.3 mg/dL vs. 108.4 ± 24.7 mg/dL, p=0.001), and triglycerides (178.7 ± 34.2 mg/dL vs. 150.9 ± 30.8 mg/dL, p=0.004) in AGA patients compared to controls. Conversely, HDL levels were lower in AGA patients (42.1 ± 6.5 mg/dL vs. 48.6 ± 5.9 mg/dL, p=0.003), supporting previous findings by Kim et al. (2016)[14], which showed an abnormal lipid profile in AGA patients.

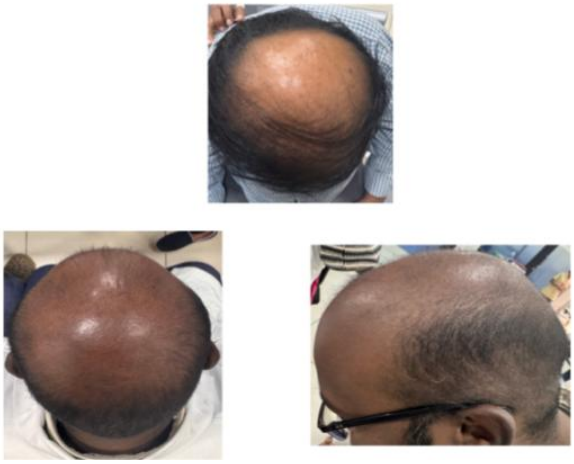
Smoking was also associated with increased AGA severity in our study, with a higher proportion of severe AGA cases (Grade IV-V) among smokers (54%) compared to non-smokers (16%). This is similar to findings by Fortes et al. (2017)[15], who reported a significantly increased risk of moderate-to-severe AGA among smokers (OR: 6.72; 95% CI: 2.57-17.6). Additionally, logistic regression analysis in our study showed that smoking (OR: 3.42; 95% CI: 1.76-5.23, p<0.001) and

hyperlipidemia (OR: 2.18; 95% CI: 1.24-3.89, p=0.008) were significant risk factors for early-onset AGA, showing their potential etiological roles.

Grade 5



Grade 6



Our findings show a strong association between hyperlipidemia, smoking, and early-onset AGA, showing the potential metabolic and lifestyle-related risk factors contributing to AGA progression. These results show the importance of lifestyle modifications, including smoking cessation and lipid management, in individuals at risk for early-onset AGA.

Conclusion

The present study shows a significant association between hyperlipidemia, smoking, and the early onset of androgenetic alopecia (AGA). Participants with AGA had higher total cholesterol, LDL, and triglyceride levels, while HDL levels were lower compared to controls, showing a strong link between dyslipidemia and AGA. Additionally, smoking was associated with greater AGA severity, with a higher proportion of smokers had advanced grades of hair loss. Logistic regression analysis confirmed that hyperlipidemia (OR = 2.18, p = 0.008) and smoking (OR = 3.42, p < 0.001) were independent risk factors for early-onset AGA. While a positive family history showed a borderline significance, BMI is not found as a significant predictor. These findings show that lipid abnormalities and smoking play a crucial role in the progression of AGA, reinforcing the need for early screening and lifestyle modifications in individuals at risk.

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