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Association Between Smoking and Androgenetic Alopecia: A Cross-Sectional Analysis of Severity and Dose-Response Relationship in Adult Males

Nasira Nasr[1,2], Umaima Afzal[3], Karishma Mushtaq[4]

[1]Department of Dermatology, Poonch Medical College,[2]Department of Dermatology, Combined Military Hospital Rawalakot, [3]Department of Dermatology and Aesthetic Physician, Dr Kashif's Aesthetic Studio, [4 2]Department of Dermatology,Pak Emirates Military Hospital Rawalpindi, Pakistan.

ABSTRACT

Background: Androgenetic alopecia is the most common form of hair loss in men, characterized by progressive thinning of scalp hair. Several factors have been implicated in its pathogenesis, including smoking, which induces oxidative stress that potentially affects the hair follicle health. The study aims to evaluate the association between smoking and the presence and severity of androgenetic alopecia in adult males.

Methods: A cross-sectional study was conducted at the Combined Military Hospital, Rawalakot, for 6 months from July 2024 to December 2024. The inclusion criteria comprised adult males aged 18 to 50 years with a clinical diagnosis of androgenetic alopecia, who were willing to provide informed consent, including both smokers and non-smokers. Data were collected using a structured questionnaire. Smoking status was categorized as current, former, or non-smoker. Clinical assessment of AGA was performed by a dermatologist using the Hamilton-Norwood scale. Statistical analysis was done using SPSS 26, the chi-square test, with significance at $p < 0.05$.

Results: Among the 120 participants, 84 (70%) had androgenetic alopecia. A significant association was observed between smoking status and the presence of AGA ($p < 0.0001$). Furthermore, the severity of AGA was significantly higher among current smokers compared to non-smokers ($p = 0.017$).

Conclusion: There is a substantial correlation between smoking and the degree and presence of androgenetic alopecia. These results underline the significance of quitting smoking at an early age and suggest that smoking may be a modifiable risk factor in the development of AGA.

Keywords: Androgenetic Alopecia, Smoking, Hair loss, Baldness.

Corresponding Author:
Dr Nasira Nasr,
Department of Dermatology,
Poonch Medical College and
Combined Military Hospital Rawalakot, Pakistan.
Email: nasiranasr@gmail.com
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INTRODUCTION

Worldwide, androgenetic alopecia (AGA), also referred to as male-pattern or female-pattern baldness, is the most common form of hair loss that affects both men and women [1, 2]. A genetic predisposition and the influence of androgens, especially dihydrotestosterone (DHT), are the main causes of this condition, which is characterized by increasing hair thinning in a well-defined pattern[3]. Men usually experience hair loss in the temples and crown, but women experience diffuse thinning over the crown while maintaining the frontal hairline[4, 5].

AGA affects a significant portion of the population, with estimates suggesting that up to 80% of men and 50% of women experience some degree of androgenetic alopecia[6,7]. While genetic and hormonal factors remain central in the pathophysiology of AGA, increasing attention has been given to environmental and lifestyle influences that may exacerbate or accelerate the onset of hair loss[8]. Among these, smoking has emerged as a potential risk factor[9,10].

Tobacco smoke contains thousands of chemicals and free radicals that may disrupt microvascular circulation, damage hair follicles through oxidative stress, and interfere with the normal hair growth cycle[11,12]. Several studies have proposed that smoking may contribute to premature graying and hair loss through mechanisms involving vasoconstriction, increased DNA damage, imbalance of protease/antiprotease activity, and reduced oxygen delivery to hair follicle cells[9, 11, 13]. Moreover, smoking may promote the release of pro-inflammatory cytokines that adversely affect the dermal papilla and follicular environment[14].

Given the widespread prevalence of both AGA and tobacco use globally, especially in countries with high smoking rates, understanding the association between smoking and androgenetic alopecia carries important public health implications. Identifying modifiable risk factors, such as smoking, can provide avenues for preventive strategies and patient counseling. Clarifying whether smoking is significantly associated with the presence or severity of AGA could offer new insights into its pathogenesis and help inform lifestyle interventions in affected individuals. Furthermore, demonstrating this association in a defined population can aid dermatologists and primary care physicians in educating patients about the harmful effects of smoking, not just systemically but also on dermatological health. The present study aimed to evaluate the association between androgenetic alopecia and smoking status among individuals presenting with hair loss complaints.

METHODS

This cross-sectional study was conducted at the Dermatology Outpatient Department (OPD) of Combined Military Hospital, Rawalakot, for 6 months from July 2024 to December 2024.

The study protocol was reviewed and approved by the Institutional Review Board under Approval No:007/SKBZ/CMH, Dated: 1st June 2024. The nature and goal of the study were explained to the participants, and it was made clear that their involvement was entirely voluntary. Throughout the study, individuals' anonymity and confidentiality were rigorously protected.

The sample size was calculated using OpenEpi software (Version 3.01) with a 95% confidence level and 80% power to detect a significant association between smoking and androgenetic alopecia. A previous study reported an OR of 1.77 (95% CI: 1.14–2.76) for moderate or severe AGA for smokers vs. non-smokers. Therefore, we estimated the prevalence of AGA to be approximately 60% among smokers and 40% among non-smokers. With a ratio of exposed (smokers) to unexposed (non-smokers) participants of approximately 1:1, the minimum sample size required would therefore be 120 participants[15]. Using these parameters, the minimum required sample size was estimated to be 120 participants.

The method of non-probability sequential sampling was applied. All patients meeting the inclusion criteria and presenting during the study period were approached and invited to participate until the desired sample size was achieved. The inclusion criteria included adult males aged 18 to 50 years, those diagnosed clinically with androgenetic alopecia (according to the Hamilton-Norwood scale), those willing to provide informed consent, and both smokers and non-smokers. Patients with other types of alopecia (e.g., alopecia areata, telogen effluvium, scarring alopecia), with a history of systemic illnesses affecting hair (thyroid disease, anemia, autoimmune disorders), those on chemotherapy or radiotherapy, and pregnant or lactating women were excluded from the study.

After obtaining ethical approval from the Institutional Review Board (IRB), participants were briefed about the study. All participants provided written informed consent. Trained research assistants conducted in-person interviews with participants using a structured questionnaire to gather data. The questionnaire included demographic data, i-e. age, gender, medical history: Any chronic illness or medication use, smoking history: duration of smoking, number of cigarettes per day, categorized as current smoker, former smoker, or non-smoker, and clinical assessment: severity of AGA was assessed by a

dermatologist using the Hamilton-Norwood scale[16, 17].

AGA was assessed by a dermatologist using the Hamilton-Norwood scale, which classifies the severity of hair loss into seven stages. In addition, visual scalp examination was supplemented by dermoscopy (trichoscopy), allowing for a more detailed evaluation of hair shaft diameter variability, follicular miniaturization, and perifollicular changes[18]. All assessments were conducted in a standardized manner to minimize interobserver variability. The study variables included the independent variables, i-e, smoking status (current smoker, former smoker, non-smoker) and the dependent variable, i-e, the presence and severity of androgenetic alopecia.

Statistical Package for the Social Sciences (SPSS) Version 26 was used to enter and analyze the data. Clinical and demographic features were gathered using descriptive statistics. Continuous variables were reported as mean ± standard deviation (SD); categorical variables were given as frequencies and percentages. The relationship between smoking status and the existence or severity of androgenetic alopecia was evaluated using the chi-square test (or Fisher's exact test, if appropriate). Statistical significance was defined as a p-value of less than 0.05.

RESULTS

The study consisted of 120 male participants (100%), with a mean age of 36.9 ± 7.5 years. The participants were grouped into three age categories: the majority (42.5%) were between 31 and 40 years of age, followed by 34.1% in the 41–50 years age group, and 23.3% between 18 and 30 years. In terms of medical background, 16.6% of participants reported having a chronic illness, while 48.0% were on some form of medication at the time of the study. Regarding smoking habits, 43.3% of the participants were identified as current smokers, 7.5% as former smokers, and 49.1% as non-smokers. Among the current smokers (n = 52), the duration of smoking varied, with 30.0% smoking for 1–5 years, 42.3% for 6–10 years, and 26.9% for more than 10 years. The daily cigarette consumption among current smokers showed that 50.0% smoked 5–10 cigarettes per day, 26.9% smoked fewer than 5, and 23.0% smoked more than 10 cigarettes daily. Clinical assessment revealed that 70% of participants were found to have androgenetic alopecia (AGA), while 30% showed no signs of AGA. This high prevalence of AGA highlights its potential association with lifestyle factors such as smoking, which is further explored in subsequent analysis shown in **Table 1; Figure 1.**

Table 1: Demographic and Clinical Characteristics of Participants (n = 120)

Variable	Frequency (n)	Percentage (%)
Male	120	(100%)
Age Group (years)		
18–30	28	23.3%
31–40	51	42.5%
41–50	41	34.1%
Age Mean ± SD	36.9 ± 7.5 years	
Medical Condition		
Chronic Illness (any)	20	16.6%
Current Medication Use	25	48.0%
Smoking Status		
Current Smoker	52	43.3%
Former Smoker	9	7.5%
Non-Smoker	59	49.1
Duration of Smoking in current smokers (Years)		
1–5 years	16	30.0%
6–10 years	22	42.3%
>10 years	14	26.9%
Cigarettes per Day		
<5 cigarettes	14	26.9%
5–10 cigarettes	26	50.0%
>10 cigarettes	12	23.0%
Androgenetic Alopecia		
Present	84	70%
Absent	36	30%

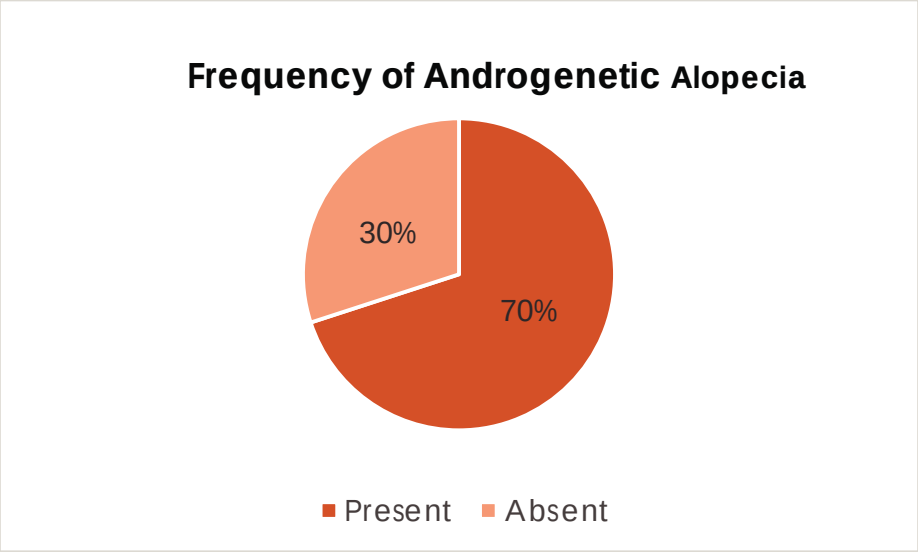

Figure 1: A Pie Chart Showing the Frequency of Androgenic Alopecia Among Study Participants

Table 2: Association between smoking status and AGA by Hamilton-Norwood Scale (n=84)

Smoking Status	Mild AGA (Grade I–II)	Moderate AGA (Grade III–IV)	Severe AGA (Grade V–VII)
Current Smoker	10 (11.9%)	17 (20.2%)	25 (29.7%)
Former Smoker	1 (1.1%)	5 (5.9%)	3 (3.5%)
Non-Smoker	22 (26.1%)	25 (29.7%)	12 (14.2%)

The chi-square statistic is 11.93; The p-value is 0.017; The result is significant at p < 0.05

Among the 84 participants diagnosed with AGA, a detailed comparison of AGA severity using the Hamilton-Norwood Scale revealed a statistically significant association with smoking status (χ^2 = 11.93, p = 0.017), indicating that smoking may influence the progression or severity of AGA. Among current smokers, 11.9% had mild AGA (Grade I–II), 20.2% had moderate AGA (Grade III–IV), and a notable 29.7% presented with severe AGA (Grade V–VII). This suggests a trend toward greater severity of hair loss among active smokers. In the former smoker group, 1.1% had mild, 5.9% had moderate, and 3.5% had severe AGA. Although the number is smaller, it may indicate a residual or delayed effect of past smoking on hair loss. In contrast, among non-smokers, 26.1% had mild AGA, 29.7% had moderate AGA, and only 14.2% had severe AGA, demonstrating a relatively lower proportion of severe cases compared to current smokers. The significant p-value of 0.017 indicates that the association between smoking status and AGA severity is statistically significant and not due to random chance. These findings support the hypothesis that smoking may contribute to the progression of androgenetic alopecia, possibly through vascular or oxidative stress-related mechanisms shown in **Table 2.**

Table 3: Association Between Smoking Status and Androgenetic Alopecia (n = 120)

Smoking Status	AGA Present (n = 84)	AGA Absent (n = 36)
Current Smoker	52 (43.3%)	68 (56.6%)
Former Smoker	9 (7.5%)	111 (92.5%)
Non-Smoker	59 (49.1%)	61 50.8%)

The chi-square statistic is 54.975; The p-value is < 0.0001; The result is significant at p <0 .05.

Out of the total 120 participants, a strong and statistically significant association was found between smoking status and the presence of androgenetic alopecia, with a chi-square value of 54.975 and a p-value of < 0.0001, indicating a highly significant result (p < 0.05). Of the 84 individuals with AGA, 49.1% were non-smokers, 7.5% were past smokers, and a sizable majority (43.3%) were current smokers. In comparison, only 56.6% of the 36 people without AGA were smokers at the time of the study, 92.5% were ex-smokers, and 50.8% were not smokers shown in **Table 3.**

Table 4: Association Between Quantity of Cigarettes Per Day and Androgenetic Alopecia in Current Smokers

Cigarettes Per Day	AGA Present (n=52)	AGA Absent (n=68)
<5 cigarettes	14 (23.8%)	41 (31.1%)
5–10	26 (19.3%)	20 (26.0%)
>10 cigarettes	12 (8.2%)	7 (10.7%)

The chi-square statistic is 13.4589; The p-value is 0.001; The result is significant at p < 0.05.

Among current smokers, the association between the quantity of cigarettes smoked per day and the presence of androgenetic alopecia (AGA) was statistically significant ($\chi^2 = 13.46$, $p = 0.001$). Of the 52 current smokers who had AGA, 23.8% smoked fewer than 5 cigarettes per day, 50.0% smoked 5–10 cigarettes daily, and 26.1% smoked more than 10 cigarettes daily. In comparison, among the 68 current smokers without AGA, 31.1% smoked fewer than 5 cigarettes per day, 26.0% smoked 5–10, and 10.7% smoked more than 10 cigarettes per day. According to the findings, there was a dose-response association between the prevalence of AGA and the daily cigarette consumption shown in **Table 4.**

DISCUSSION

The present cross-sectional study assessed the association between smoking status and androgenetic alopecia (AGA) in a sample of 120 male participants. Our findings demonstrate a statistically significant relationship between smoking and both the presence and severity of AGA. Specifically, 70% of the participants had AGA, with a higher prevalence and severity observed among current smokers. The chi-square analysis confirmed a strong association between smoking status and AGA presence ($p < 0.0001$), as well as with the severity of AGA as assessed by the Hamilton-Norwood Scale ($p = 0.017$).

Our findings are consistent with several previous studies that also reported a significant link between tobacco use and androgenetic alopecia. In a seminal case-control study in the UK, it was observed that smokers had twice the risk of developing AGA compared to non-smokers, particularly in younger age groups[19]. Similarly, a large-scale study conducted in Taiwan involving over 740 men found a dose-dependent relationship between cigarette consumption and the severity of AGA, supporting the theory that both the duration and intensity of smoking contribute to follicular damage15. Another study by Pradigo et al. (2019 from Indonesia also reported that smokers had an increased risk of developing mild to moderate AGA[20].

The possible pathophysiological mechanisms linking smoking to hair loss have been widely discussed in the literature. Smoking induces vasoconstriction, reduces blood supply to the scalp, and introduces oxidative stress by releasing free radicals[21]. This results in damage to the hair follicles, premature aging of the scalp tissue, and alteration of the normal hair growth cycle. Additionally, nicotine and other toxins in cigarette smoke may impair the microvascular supply to dermal papilla cells, leading to miniaturization of hair follicles, a hallmark of AGA[22].

Our study found that severe grades of AGA (Grade V–VII) were most prevalent among current smokers (29.7%), in contrast to non-smokers, who showed a higher proportion of mild and moderate forms. These results align with a study that proposed that chronic exposure to smoke accelerates the aging process of scalp tissues and enhances perifollicular inflammation, both of which can exacerbate the severity of androgenetic alopecia[10].

Interestingly, former smokers in our study also showed moderate to severe AGA, although at a lower frequency compared to current smokers. This suggests that past exposure may have a lingering effect, but cessation of smoking may help slow further progression. However, none of the studies suggested that quitting smoking may improve hair growth after cessation of smoking, as reported by a systematic review[9]. Another meta-analysis conducted in Canada and a study by Salem et al., 2021 found a significant association between smoking and AGA[23, 24].

Our findings highlight a significant dose-dependent relationship between cigarette consumption and the presence of androgenetic alopecia (AGA), consistent with earlier research studies that emphasized that higher levels of oxidative stress and microvascular compromise from heavy smoking can exacerbate follicular miniaturization[22, 25]. The progressive increase in AGA prevalence among individuals smoking more than 10 cigarettes daily in our study supports these conclusions. The observed pattern aligns with the concept that not only

smoking status but also the quantity of tobacco exposure plays a pivotal role in the pathogenesis of AGA, reinforcing the need for targeted counseling on smoking reduction and cessation in at-risk populations.

Despite its strengths, including a standardized clinical assessment by dermatologists using the Hamilton-Norwood Scale and a clearly defined smoking history, our study also has limitations. The cross-sectional design limits the ability to establish causality. Additionally, self-reported smoking history may introduce recall bias, and other confounding variables such as diet, stress, and family history of AGA were not controlled for in this analysis.

CONCLUSION

This study demonstrates a significant association between smoking and the presence as well as the severity of androgenetic alopecia (AGA) in males. Current smokers were found to have a higher prevalence and more severe grades of AGA compared to non-smokers and former smokers. These findings support the hypothesis that smoking is a modifiable risk factor contributing to the progression of AGA, likely through mechanisms involving oxidative stress, vascular impairment, and follicular damage. Early identification and smoking cessation may play a vital role in preventing or minimizing hair loss, highlighting the need for integrating smoking history into dermatological assessments of patients presenting with hair loss.

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CONFLICT OF INTEREST

None

ETHICAL APPROVAL

The study was approved by the Ethical Review Committee of the Combined Military Hospital, Rawalakot, under Approval No:007/SKBZ/CMH, Dated: 1st June 2024.

PATIENT CONSENT

Informed consent was obtained from all patients involved in the study.

AUTHORS' CONTRIBUTIONS

NN: contributed to the study design, data acquisition, and interpretation. **UA:** was involved in data interpretation. **KM** contributed to drafting the manuscript and reviewing it for important intellectual content. All authors reviewed and approved the final version of the manuscript.

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