Section: Dermatology

Original Research Article

ASSOCIATION BETWEEN CIGARETTE SMOKING AND SEVERITY OF ANDROGENETIC ALOPECIA: A CROSS-SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Androgenetic alopecia (AGA) is the most common form of hair loss affecting both men and women and is characterized by progressive thinning of scalp hair due to follicular miniaturization. Although genetic predisposition and androgen activity are well-established factors in its pathogenesis, environmental and lifestyle factors such as cigarette smoking have also been suggested to influence the onset and severity of the condition. Smoking may impair hair follicle health through mechanisms including microvascular damage, oxidative stress, and inflammatory changes. However, evidence regarding the association between smoking and androgenetic alopecia remains limited in many populations. This study was designed to evaluate the association between cigarette smoking and the severity of androgenetic alopecia among patients attending a dermatology outpatient department.

Materials and Methods: This cross-sectional study included 148 patients clinically diagnosed with androgenetic alopecia who attended the Dermatology Department of Maheshwara Medical College and Hospital, Isnapur, Telangana, between January 2024 and December 2024. Detailed clinical history and examination were performed for all participants. Information regarding smoking habits, duration, and intensity was recorded, and the Smoking Index (SI) was calculated. The severity of androgenetic alopecia was assessed using the Hamilton–Norwood classification. Data were analysed using descriptive statistics and appropriate statistical tests to determine the association between smoking exposure and alopecia severity.

Results: The majority of participants belonged to the 20-30-year age group. Approximately 77% of patients reported a history of smoking, with moderate smokers forming the largest subgroup. Grade II androgenetic alopecia was the most frequently observed stage. Increased smoking exposure was associated with greater severity of alopecia.

Conclusion: The findings suggest a significant relationship between cigarette smoking and androgenetic alopecia severity. Smoking may represent a modifiable lifestyle factor influencing the progression of hair loss, highlighting the importance of smoking cessation counselling in patients with androgenetic alopecia.

Keywords: Androgenetic alopecia, Smoking, Smoking index, Male pattern baldness, Hair loss, Hamilton-Norwood classification.

INTRODUCTION

Androgenetic alopecia (AGA) is the most common form of hair loss affecting both men and women worldwide. It is characterized by progressive thinning of scalp hair due to gradual miniaturization of hair follicles in genetically predisposed individuals. Clinically, AGA presents as patterned hair loss involving the frontal, temporal, and vertex regions of the scalp in men, while women typically demonstrate diffuse thinning over the crown with preservation of the frontal hairline. The prevalence of

androgenetic alopecia increases with age, and epidemiological studies indicate that nearly half of men develop some degree of patterned hair loss by the age of 50 years.[1]

The pathogenesis of androgenetic alopecia is multifactorial and involves a complex interaction between genetic susceptibility, hormonal influence, and environmental factors. Dihydrotestosterone (DHT), a potent androgen formed from testosterone by the enzyme 5-alpha reductase, plays a central role in the miniaturization of susceptible hair follicles. Increased sensitivity of hair follicles to androgens leads to shortening of the anagen phase of the hair cycle and progressive reduction in hair shaft diameter.[2] Although genetic and hormonal mechanisms are well recognized, increasing attention has been directed toward lifestyle and environmental factors that may contribute to the onset and progression of androgenetic alopecia.

Among these factors, cigarette smoking has been proposed as a potential risk factor for hair loss. Tobacco smoke contains numerous toxic chemicals that can affect microcirculation, oxidative stress pathways, and inflammatory responses in the skin. Several studies have suggested that smoking may impair blood flow to the dermal papilla of hair follicles, resulting in reduced oxygen and nutrient supply required for normal hair growth.[3] Furthermore, smoking has been associated with increased production of reactive oxygen species and inflammatory mediators that may damage hair follicle cells and accelerate follicular aging.[4]

Previous epidemiological studies have reported a higher prevalence of moderate to severe androgenetic alopecia among smokers compared with non-smokers. A population-based study conducted among Asian men demonstrated that smoking intensity and duration were significantly associated with the severity of male pattern baldness.[5] Similarly, other investigations have indicated that heavy smokers are more likely to develop early onset and more advanced stages of androgenetic alopecia.[6] Despite these observations, the relationship between smoking and hair loss remains incompletely understood, and the available evidence varies across different populations.

Understanding the role of modifiable lifestyle factors in androgenetic alopecia is important for developing preventive strategies and improving patient counselling. Therefore, the present study was undertaken to evaluate the association between cigarette smoking and androgenetic alopecia among patients attending a dermatology outpatient department. The study also aims to assess the severity of hair loss in relation to smoking exposure and to contribute further evidence to the growing literature on lifestyle influences in androgenetic alopecia.

MATERIALS AND METHODS

This hospital based cross-sectional observational study was conducted in the Department of Dermatology at Maheshwara Medical College and Hospital, Isnapur, Telangana from January 2024 to December 2024. The study was carried out to assess the relationship between cigarette smoking and the occurrence as well as severity of androgenetic alopecia among patients attending the dermatology outpatient department. A total of 148 patients presenting with clinical features suggestive of androgenetic alopecia at the dermatology OPD during the study period were screened for eligibility. Written informed consent was obtained from all the study participants and study protocol was reviewed and approved by the Institutional Ethics Committee.

Inclusion Criteria

Patients clinically diagnosed with androgenetic alopecia, aged ≥18 years, and willing to participate in the study.

Exclusion Criteria

Patients with other forms of alopecia such as alopecia areata, telogen effluvium, traction alopecia, or scarring alopecia, Individuals with dermatological or systemic disorders known to affect hair growth, under treatment for hair loss prior to participation and not willing to provide informed consent.

All participants were undergone a detailed clinical assessment using a structured proforma. Information recorded included demographic details, clinical history, and lifestyle factors. The parameters including age of onset of hair loss, duration and progression of hair loss, family history of androgenetic alopecia, smoking history including number of cigarettes smoked per day and duration of smoking, alcohol consumption, associated comorbid conditions such as diabetes mellitus, hypertension, bronchial asthma, or cardiovascular diseases.

All participants underwent a thorough scalp examination. The pattern and severity of hair loss were assessed clinically and graded according to the Hamilton-Norwood classification system, which is commonly used to evaluate the stages of androgenetic alopecia. This classification enabled categorization of patients based on the extent and distribution of hair loss.

Smoking exposure was quantified using the Smoking Index (SI). The smoking index was calculated using the following formula:

Smoking Index = Number of cigarettes smoked per day × Duration of smoking in years

Based on the smoking index, participants were categorized into different groups such as light, moderate, or heavy smokers.

Statistical Analysis: The collected data was extracted to Microsoft Excel sheet and analysed using SPSS v.26.0. Descriptive statistics were used to summarize demographic and clinical characteristics. The association between smoking habits and the severity of androgenetic alopecia was evaluated using

appropriate statistical tests. A p<0.05 was considered as statistically significant.

RESULTS

Majority participants belonged to 20-30 years of age indicating that androgenetic alopecia commonly presents during early adulthood.

Table 1: Demographic and clinical profile of Study Participants (n = 148)

Parameters	Total no of patients (n=148)	
	Frequency	Percentage (%)
Age (In years)		
20–30	66	44.6%
31–40	36	24.3%
41–50	24	16.2%
51–60	16	10.8%
>60	6	4.1%
Gender		
Male	102	68.91%
Female	46	31.08%
Occupation		
Daily wage labour	32	21.62%
Industrial workers	58	39.18%
Housewives	15	10.13%
Agricultural workers	23	15.54%
Unemployed	20	13.51%
Duration of androgenetic alopecia		
Less than 1 year	22	14.9%
1-5 years	64	43.2%
6-10 years	38	25.7%
Above 10 years	24	16.2%
Family history		
Positive	86	58.1%
Negative	62	41.9%

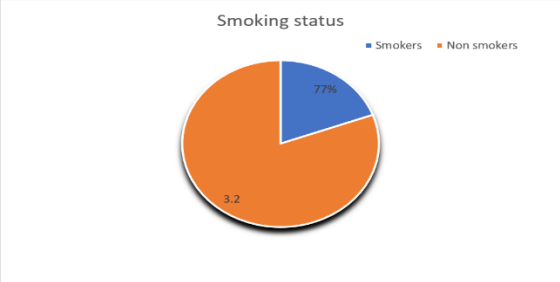

Figure 1: Smoking status of study participants.

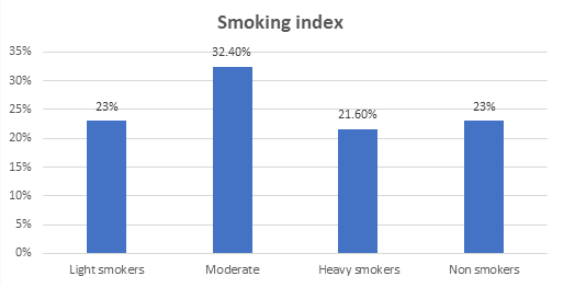

Figure 2: Distribution of cases based on smoking index.

Table 2: Associated comorbidities in study participants.

Comorbidity	No of patients	Percentage (%)
Diabetes Mellitus	14	9.5%
Hypertension	12	8.1%
Bronchial Asthma	3	2.0%
Coronary Artery Disease	1	0.7%
No comorbidity	118	79.7%

Table 3: Severity of androgenetic alopecia as per Hamilton-Norwood classification.

Grade	Number of Patients	Percentage (%)
Grade I	32	21.6
Grade II	41	27.7
Grade III	28	18.9
Grade IV	22	14.9
Grade V	15	10.1
Grade VI	10	6.8

Table 4: Association between smoking index and androgenetic alopecia severity.

Smoking Category	Mild (I-II)	Moderate (III-IV)	Severe (V-VI)	p-value
Non-smokers	22	9	3	0.001
Light smokers	18	11	5	
Moderate smokers	16	21	11	
Heavy smokers	8	13	11	

DISCUSSION

Androgenetic alopecia (AGA) is the most common cause of progressive hair loss and is influenced by both genetic and environmental factors. The present study evaluated the association between smoking habits and the clinical severity of androgenetic alopecia among 148 patients attending a dermatology outpatient department. The findings demonstrated that a large proportion of patients with AGA were smokers and that increased smoking exposure was associated with more severe grades of alopecia. These findings support the growing body of literature indicating that smoking may play a contributory role in the development and progression of androgenetic alopecia.

In the present study, the majority of participants were between 20-30 years of age. This observation is consistent with earlier epidemiological studies which have reported that androgenetic alopecia often begins in early adulthood and progresses gradually with advancing age. Genetic predisposition and hormonal factors are known to influence the onset of AGA; however, environmental influences such as lifestyle habits may also contribute to disease progression. Previous studies have shown that individuals with early onset AGA may experience more rapid progression compared with those who develop the condition later in life.

A notable finding of the current study was the high prevalence of smoking among patients with androgenetic alopecia. Approximately three-fourths of the participants reported a history of smoking. Similar observations have been reported in earlier investigations. A population-based study conducted in Taiwan found a significant association between smoking and androgenetic alopecia among Asian men, with smokers showing a higher prevalence of moderate to severe hair loss compared with non-smokers.[5] These findings suggest that tobacco exposure may be an important modifiable risk factor in individuals susceptible to patterned hair loss.

In addition to the prevalence of smoking, the present study also demonstrated that increasing smoking exposure was associated with greater severity of alopecia. Participants categorized as moderate or heavy smokers were more likely to present with advanced stages of AGA compared with non-smokers. These observations are in agreement with the findings of Fortes et al., who reported that smoking, particularly when combined with other metabolic risk factors such as increased body mass index, was associated with a significantly higher likelihood of moderate to severe androgenetic alopecia.[6] Similarly, a recent meta-analysis reported that individuals who smoked ten or more cigarettes per day had a significantly higher risk of developing AGA compared with those who smoked fewer cigarettes or did not smoke at all.[7]

The biological mechanisms linking smoking with androgenetic alopecia have been discussed in several studies. Cigarette smoke contains numerous toxic substances that can affect hair follicle physiology. Smoking is known to cause vasoconstriction and impaired microcirculation, which may reduce blood flow to the dermal papilla of the hair follicle. This reduction in nutrient and oxygen supply can negatively influence hair growth and follicular health. In addition, tobacco smoke generates reactive oxygen species that lead to oxidative stress and cellular damage within the hair follicle. These mechanisms may contribute to follicular miniaturization, which is the hallmark pathological feature of androgenetic alopecia.[8]

Another proposed mechanism involves the hormonal effects of smoking. Tobacco exposure may alter the balance of androgens and estrogens by increasing the metabolism of estrogen and reducing aromatase activity. Such hormonal alterations may enhance androgen-mediated follicular miniaturization, thereby accelerating the progression of androgenetic alopecia. Moreover, cigarette smoke has been shown to induce inflammatory processes in the scalp, resulting in micro-inflammation and fibrosis around the hair follicles. These pathological changes may further disrupt the hair growth cycle and promote premature transition of follicles from the anagen phase to the telogen phase.[9]

The results of the present study are also supported by recent systematic reviews examining the relationship between smoking and hair disorders. Evidence from multiple observational studies indicates that smoking is associated with a higher prevalence of hair loss and other hair abnormalities such as premature hair greying.[4] Furthermore, some investigators have suggested that nicotine and its metabolites may directly influence hair follicle biology by accelerating follicular aging and altering the hair growth cycle.

Despite these findings, the relationship between smoking and androgenetic alopecia remains somewhat controversial. Certain studies have reported only a modest association or failed to demonstrate a clear relationship between smoking intensity and disease severity.[10,11] Differences in study design, sample size, population characteristics, and assessment of smoking exposure may account for these variations. Therefore, further large-scale prospective studies are needed to clarify the causal role of smoking in the development and progression of androgenetic alopecia.

The present study also observed a positive family history of alopecia in more than half of the participants, highlighting the strong genetic component of the disease. AGA is known to be a polygenic disorder influenced by androgen receptor sensitivity and hormonal factors. Lifestyle influences such as smoking may act as additional triggers that accelerate the manifestation or progression of genetically predisposed hair loss.

CONCLUSION

This study highlights a significant association between cigarette smoking and androgenetic alopecia. A higher prevalence of smoking was observed among patients with androgenetic alopecia, and increased smoking exposure was associated with greater severity of hair loss. These findings suggest that smoking may act as an important environmental factor influencing the progression of androgenetic alopecia in genetically predisposed individuals. Early identification of modifiable lifestyle factors such as smoking may help in preventing or slowing disease progression. Dermatologists should consider incorporating smoking history assessment and counselling for smoking cessation as part of the comprehensive management of patients presenting with androgenetic alopecia.

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