Original Article

DOI: 10.7860/JCDR/2025/75942.21106

Lifestyle Risk Factors in Male Androgenetic Alopecia: A Cross-sectional Study

Dermatology Section

MAYURESH SHAMSUNDER PAVASKAR[1], SUGANITA[2], DEEPIKA AGARWAL[3], SONAL SACHAN[4], VANDANA YADAV[5]

ABSTRACT

Introduction: Male Androgenetic Alopecia (AGA) is a prevalent form of hair loss affecting a significant portion of the population worldwide. While genetic predisposition is a primary factor, lifestyle factors such as smoking, alcohol consumption, obesity and psychological stress have been increasingly recognised as potential contributors to the progression and severity of male AGA. This study was conducted to identify these risk factors in male AGA utilising various tools like the Depression, Anxiety and Stress Scale (DASS) 21 scale, Body Mass Index (BMI), smoking index and Alcohol Use Disorders Identification Test (AUDIT) scale.

Aim: To identify lifestyle-related risk factors such as smoking, alcohol consumption, obesity and psychological stress in male AGA.

Materials and Methods: A hospital-based observational cross-sectional study was conducted in the dermatology department of a tertiary care facility, involving 100 male patients with AGA aged 18 to 55 years. The study utilised consecutive sampling methods for the enrollment of participants. Each patient underwent a thorough clinical evaluation and the severity of alopecia was determined using the Hamilton-Norwood classification. Detailed questionnaires assessed risk factors including smoking history (using the smoking index), alcohol consumption (using the AUDIT scale), psychological stress levels (using the DASS-21 scale) and obesity was assessed by measuring BMI. Data were analysed using statistical software Statistical Package for the Social Sciences (SPSS) version 26.0 (SPSS Inc., Chicago, IL, USA).

Results: Among the study population, the mean age was 28.78±5.91 years. Family histories included 12 with maternal AGA, 56 with paternal AGA and 32 with no family history. Significant associations were found between AGA severity and several factors, including higher alcohol screening scores (4.71±5.30) (p-value=0.0006) and a greater smoking index (17.31±15.10) (p-value=0.016). The results for BMI (22.33±3.91) (p-value=0.056) and DASS-21 stress score (14.96±5.9677) (p-value=0.351) were not found to be significant with the severity of AGA. Overall, the findings underscore the multifactorial nature of AGA, influenced by lifestyle, genetic predisposition and anthropometric measures, with implications for clinical management and future research.

Conclusion: The study highlights the multifaceted nature of AGA, influenced by lifestyle habits and factors. The findings suggest a strong association between higher alcohol and smoking indices and the severity of AGA. These insights could inform prevention strategies and targeted interventions for individuals affected by AGA.

Keywords: Alcohol consumption, Obesity, Psychological stress, Smoking

INTRODUCTION

Alopecia, particularly AGA, is a frequent concern among patients seen by dermatologists and primary care providers [1]. AGA is the leading cause of hair loss in men, characterised by hereditary, androgen-dependent progressive miniaturisation of scalp hair, manifesting in various patterns [2-4]. Male AGA typically begins around the age of 20 years and affects nearly 50% of men by the age of 50 years [5]. Its aetiopathogenesis is primarily androgen-dependent and modulated via the testosterone metabolite, Dihydrotestosterone (DHT). The expression of hair follicle-related androgen receptors and genetic factors has also been implicated. Earlier onset is associated with more rapid progression than those who develop AGA later in life [6,7].

The diagnosis of AGA is primarily clinical, supported by diagnostic tools such as the hair pull test, trichoscopy and scalp biopsy, to differentiate it from other forms of alopecia [8]. Although the influence of environmental factors on AGA remains unclear, research suggests that individuals with obesity, alcohol consumption, smoking habits, or psychological stress are more susceptible to the condition [9-11].

Smoking potentially plays a role in AGA through multiple mechanisms that work simultaneously, such as vasoconstriction and free radical-induced damage to hair follicles via the formation of Deoxyribonucleic Acid (DNA) adducts. Additionally, free radicals, particularly Reactive Oxygen Species (ROS), can enhance cellular senescence and have detrimental hormonal effects on hair follicles [12]. However, the evidence regarding the role of smoking in AGA remains inconsistent, as some studies have found a positive association while others have not [12-16]. Therefore, the association between smoking and AGA requires further research. Alcohol consumption has also been linked to AGA, potentially due to its impact on trace element levels such as zinc, selenium and copper. These imbalances may contribute to oxidative stress, impaired protein synthesis and disrupted immune responses, which could accelerate AGA [17].

Psychological distress is another important factor in AGA, as hair carries significant symbolic and psychosocial value. Stress is known to cause hair loss by triggering the catagen phase of the hair cycle and promoting inflammatory responses that damage hair follicles [18,19].

In previous studies, some did not find a link between AGA and BMI [20-22]. However, others indicated that a higher BMI is associated with male AGA [23]. Obesity, a key component of metabolic syndrome, contributes to AGA through insulin resistance and elevated Insulin-like Growth Factor-1 (IGF-1) levels. These factors may stimulate the enzyme 5α-reductase, increasing DHT production and, consequently, the progression of AGA [21,22].

Therefore, the evidence regarding lifestyle-related risk factors is not extensive and requires further validation through research. The aim of this study was to identify lifestyle-related risk factors such as smoking, alcohol consumption, psychological stress and obesity in men with AGA.

MATERIALS AND METHODS

This was a hospital-based observational cross-sectional study conducted in the Department of Dermatology at Hind Institute of Medical Sciences, Barabanki, Uttar Pradesh, India, following the approval of the Institutional Human Ethical Committee (HIMS/IHEC/MD/MS/009/2023). The research focused on male patients aged 18 to 55 years who were attending the Outpatient Department (OPD) for AGA between November 2022 and October 2023. The patients underwent thorough clinical examinations and were evaluated using a detailed questionnaire based on the risk factors under investigation. Written informed consent was obtained from all the study participants.

Inclusion criteria: Male patients aged between 18 and 55 years who had not received treatment for AGA in the last six months were included in the study.

Exclusion criteria: Patients with acute medical or surgical illnesses, cardiovascular disease, chronic diseases, individuals on steroids, individuals with other hair disorders, major psychiatric illnesses, other dermatological conditions and those with a past or present history of chemotherapy or radiotherapy were excluded from the study.

Sample size estimation: Cochran's formula has been used for the estimation of sample size:

$$(n)=\frac{(z_{1-\frac{\alpha}{2}})^{2^*}\,(p)\,(q)}{(d)^2}$$

Z1-α/2=Critical value and a standard value for the corresponding level of confidence;

(At 95% CI or 5% level of significance (type-I error) it is 1.96 and at 99% CI it is 2.58);

P=Expected prevalence or based on previous research (58%);

q=1-p;

d=Margin of error or precision.

$$(n)=\frac{(1.96)^2\,(0.58)\,(1\text{-}0.58)}{(0.1)^2}$$

The prevalence of AGA in males aged 20 to 50 years, as reported by Krupa Shankar D, is 58% [24], with a margin of error of 10%. Therefore, the calculated sample size (n) was 93.58 and the final sample size was set at 100. A consecutive sampling technique was used for sample collection.

Study Procedure

Each patient was thoroughly assessed on clinical grounds for hair loss. A hair pull test was performed on each participant, where the easy removal of more than six hairs during this process was interpreted as positive [25]. The scalp was clinically examined to determine the grading and severity of AGA using the modified Hamilton and Norwood classification [2]. Details such as name, age, gender, weight and height, along with patient particulars, smoking history using the smoking index and history of alcohol intake using the AUDIT scale, were recorded. BMI was calculated by measuring weight and height. DASS-21 was used to measure the psychological stress level.

The smoking index was determined using the formula: Smoking index=(cigarettes per day) CPD×years of tobacco use. The smoking index categories included non smokers, <400, 400-799 and ≥800. The CPD was approximated for current and former smokers [26].

For individuals consuming alcohol, the AUDIT, extensively validated by the World Health Organisation (WHO), was administered in the preferred language of the patient. AUDIT comprises 10 questions about current alcohol use and the frequency of personal drinking behaviours during the last 12 months. It provides a measure of alcohol consumption and dependence on drinking, scored from 0 (never consumed) to 4 (consumed daily) [27].

Obesity was assessed using with BMI (Weight/Height^2 in kg/m^2). Patients were classified based on BMI as follows: <25 kg/m^2=normal; 25-30 kg/m^2=overweight; >30 kg/m^2=obese [28].

The psychological stress level was measured using the seven-item stress subscale of the DASS-21. The DASS-21 questionnaire includes 21 questions across three subscales that assess symptoms of depression (questions 3, 5, 10, 13, 16, 17, 21), anxiety (questions 2, 4, 7, 9, 15, 19, 20) and stress (questions 1, 6, 8, 11, 12, 14, 18). The seven items on the stress scale are graded on a Likert scale from 0 to 3 [29]. Present study focused exclusively on the 7-item stress subscale of the DASS-21 to assess psychological stress, as psychological stress is a crucial factor in present analysis. This subscale comprises seven statements related to feelings of being touchy, agitated, over-reacting to situations, intolerant of any hindrance and finding it hard to wind down and relax. By concentrating solely on the stress subscale, data collection process was streamlined and avoided unnecessary complexity, allowing us to more easily assess other lifestyle risk factors, including alcohol use, smoking and obesity, in relation to Male Androgenetic Alopecia (MAA) [30].

Scores from the 7-item stress subscale were summed and multiplied by two to calculate the final score for psychological stress, in accordance with the DASS-21 protocol. The resulting stress scores were then categorised into five levels of severity: mild (0-14), moderate (15-18), severe (19-25), very severe (26-33) and extremely severe (34+) [31].

STATISTICAL ANALYSIS

Data were analysed using statistical software SPSS version 26.0 (SPSS Inc., Chicago, IL, USA). The continuous variables were evaluated using mean (m) and Standard Deviation (SD) values when appropriate. The dichotomous variables were presented as numbers or frequencies and were analysed using the Chi-square test. To compare the means between two or more groups, the Analysis of Variance (ANOVA) test was used. A p-value of <0.05 or 0.001 was regarded as significant.

RESULTS

The study population consisted predominantly of young adults, with a mean age of 28.78 years. A majority of individuals (62%) fell within the 18-28 years age group and they had diverse occupational backgrounds along with relatively low rates of co-morbidities. The demographic characteristics also revealed a balanced distribution of marital status, dietary habits and family history, with 53% of individuals being married, 53% following a non vegetarian diet and 56% reporting a paternal history of the condition. The demographic details of the study participants are presented in [Table/Fig-1].

Parameters		Frequency (%)
Age group (years)	18-28	62.00
	29-38	27.00
	39-49	11.00
Age (years) (Mean±SD)	28.78±5.91	
Occupation	Businessman	15.00
	Constable	9.00
	Dental hygienist	8.00
	Engineer	25.00
	Factory worker	11.00
	Lawyer	7.00
	Student	25.00
Marital status	Yes	53.00
	No	47.00
Co-morbidity	Asthma	3.00
	No	97.00

Diet type	Non vegetarian	53.00
	Vegetarian	47.00
Family history	Maternal	12.00
	Paternal	56.00
	Null	32.00

[Table/Fig-1]: Demographic profile of participants.

Among the study population 64 were smokers, while the remaining 36 were non smokers. Notably, the smoking index scores indicated that the majority of smokers (62%) had a mild smoking habit, with scores below 400, whereas a minimal proportion (2%) had moderate smoking habits, with scores ranging between 400 and 799, as mentioned in [Table/Fig-2].

Smoking	Number %
Yes	64 (64)
No	36 (36)
Smoking index	
<400	62 (62)
400-799	2 (2)
≥800	-

[Table/Fig-2]: Distribution of the smoking details of the enrolled patients.

Within the study population, 44 participants refrained from alcohol consumption. Out of the 56 participants who consumed alcohol, 16 scored within the 1-7 range, 27 scored between 8-14 and a smaller group of 13 participants scored more than 15, as shown in [Table/Fig-3].

Alcohol screening score	Number %
1-7	16 (28.57)
8-14	27 (48.21)
>15	13 (23.21)

[Table/Fig-3]: Alcohol screening score of the enrolled participants.

The average weight was 66.24 kg, with moderate variability indicated by a SD of 12.31 kg. The mean height was 173.43±5.33 cm. The average waist circumference was 92.53±8.95 cm and the average hip circumference was slightly higher at 84.56±9.06 cm. Additionally, the mean BMI among the subjects was 22.33±3.91 kg/m^2, which was within normal limits, as noted in [Table/Fig-4].

BMI	Number %
<25 kg/m^2	52 (52)
25-30 kg/m^2	39 (39)
>30 kg/m^2	9 (9)

[Table/Fig-4]: BMI of enrolled participants.

[Table/Fig-5] represents the association of the risk factors listed below with the severity of male AGA. The mean smoking index was observed to increase gradually from Grade I (9.08±3.99) to Grade VII (32.80±21.43) AGA. Although Grade IV showed a mean smoking index of 12.92±7.94, which was lower compared to Grades II and III, the calculated p-value was 0.016 (F=2.892), indicating a statistically significant association between AGA severity and the smoking index. The mean alcohol screening score was found to increase with the severity of AGA, with Grade I AGA participants showing a mean of 2.8±4.37, which gradually increased with higher grades of AGA. Grade VII had the highest score at 13.2±3.83. These results suggest a strong positive correlation between higher grades of AGA and increased alcohol consumption. The BMI ranged from 21.75±3.28 in Grade I to 20.56±3.18 in Grade VII. The p-values indicate that BMI (p-value=0.056) was statistically insignificant. Thus, the results suggest no statistical association between obesity and the severity of AGA in the enrolled patients.

Severity of AGA	BMI	Alcohol screening score	Smoking index	DASS-21
	Mean±SD	Mean±SD	Mean±SD	Mean±SD
G1 (n=25)	21.75±3.28	2.80±4.37	9.08±3.99	18.52± 5.277
G2 (n=17)	21.11±3.19	2.24 ±3.70	14.62±17.60	13.706±5.706
G3 (n=23)	24.22±4.91	5.30±5.71	17.78±11.92	11.652±4.145
G4 (n=17)	22.35±2.70	5.41±3.48	12.92±7.94	14.706±6.257
G5 (n=6)	24.47±5.72	6.17±7.36	26.50±24.11	17.333±6.574
G6 (n=7)	20.47±2.99	6.57±6.50	26.33±9.91	15.857±6.534
G7 (n=5)	20.56±3.18	13.20±3.83	32.80±21.43	13.4±1.356
Total	22.33±3.91	4.71±5.30	17.31±15.10	14.96±5.9677
F, p-value*	2.139, 0.056	4.349, 0.0006	2.892, 0.016	18.205, 0.351

[Table/Fig-5]: Comparison of risk factors with severity of AGA.
p<0.05* statistically significant; p<0.001** statistically highly significant

The distribution of mean DASS-21 scores with SD across different grades of AGA is shown in [Table/Fig-6]. The p-value is 0.351 indicating no significant association between AGA severity and the DASS-21 score.

Severity of AGA	DASS-21 stress score category							
	Mild (0-14) (n=41)		Moderate (15-18) (n=22)		Severe (19-25) (n=24)		Extreme (26-33) (n=13)	
	Count	(%)	Count	(%)	Count	(%)	Count	(%)
G1 (n=25)	7	28.0	3	12.0	10	40.0	5	20.0
G2 (n=17)	9	52.9	5	29.4	2	11.8	1	5.9
G3 (n=23)	10	43.5	6	26.1	4	17.4	3	13.0
G4 (n=17)	7	41.2	4	23.5	5	29.4	1	5.9
G5 (n=6)	2	33.3	2	33.3	0	0.0	2	33.3
G6 (n=7)	3	42.9	0	0.0	3	42.9	1	14.3
G7 (n=5)	3	60.0	2	40.0	0	0.0	0	0.0

[Table/Fig-6]: Comparison of Dass-21 scale with severity of AGA.
Fisher's exact test=18.205, p=0.351

DISCUSSION

The primary goal of this research, which included 100 men aged between 21 and 43 years (mean age of 28.78±5.91 years), was to emphasise the importance of lifestyle factors in the progression of male AGA. In the current study, the largest portion of the sample (62%) fell within the 18-28 years age group. Additionally, 27% were within the 29-38 years age group, while only 11% were in the 39-49 years age group. These results suggest that male AGA primarily impacts younger males, with a noticeable decrease in frequency as age increases. Similar to present study observation, a study conducted by Nargis T et al., identified 68% of AGA cases in the age group 21-30 years. This trend was significant as it highlights the early onset of AGA, which could have considerable social and psychological consequences for younger individuals [12]. This distribution may be attributed to specific genetic and environmental factors contributing to the early onset of AGA, as well as the older patients' lack of perceived necessity for treatment.

The occupational distribution among participants in this study revealed a diverse representation, with engineers and students each comprising 25 individuals. In contrast, dentists and lawyers represented the smaller cohorts, with eight and seven participants, respectively. According to Sawant N et al., no significant difference was found in the occupational activities of the patients involved [32].

The mean duration of hair loss in AGA patients in the current study was 39.96 months, indicating that AGA is a gradually progressing condition that develops over several years. Present findings align with those of Segal AS, who noted that the majority of patients sought treatment within 1-4 years of experiencing hair loss [33]. Gupta S et al., discovered that 50% of the patients experienced hair loss for a period ranging from zero to five years [34]. Similarly,

a Korean study conducted in 2012 demonstrated a comparable duration of disease [35].

Previous studies have identified cardiovascular co-morbidities, endocrine diseases, metabolic diseases and prostate hypertrophy as strongly associated co-morbidities observed in male AGA [36]. In the present study, asthma was the only co-morbidity observed, comprising 3% of the subjects, which suggests that asthma is not a significantly associated finding. This result aligns with another study, indicating that while some AGA patients may have asthma, it does not appear to be a common or strongly associated co-morbidity [12].

In this study, 56 participants had a paternal history of AGA, while 12 reported a maternal history and 32 participants indicated no family history. The higher occurrence of AGA among individuals with a paternal lineage supports the known inheritance patterns of autosomal dominant traits in males and autosomal recessive traits in females. This implies that males with affected fathers are more likely to develop AGA themselves. Additionally, the maternal family history emphasises the potential genetic influence from the maternal side [37]. In line with this, Jang WS et al., discovered that paternal AGA had a more notable impact on AGA expression than maternal influences [38]. Despite AGA being recognised as a polygenic trait, its specific inheritance pattern is still not fully understood. The findings of present study emphasise the critical role of family history in AGA, particularly on the paternal side, in determining the condition's expression.

The determined BMI was within normal limits, as the majority of enrolled patients in the study were in the younger age group. The association between BMI and male AGA was found to be statistically insignificant. The correlation between BMI and male AGA remains uncertain, as previous literature suggested a significant link between higher BMI and male AGA, while newer research found no association in affected male AGA patients, which aligns with the findings of the present study [39-41].

Fifty-six individuals acknowledged the use of alcohol, while 44 refrained from its consumption. This was consistent with previous studies that have shown a higher prevalence of alcohol use among male AGA patients [12]. To provide a more accurate assessment of alcohol consumption patterns, the AUDIT scale was employed, which is superior to previous methods that relied on binary responses. The AUDIT scale is an internationally recognised tool for identifying alcohol-related issues and offers a thorough evaluation of the quantity, duration and frequency of alcohol intake [27]. As such, the versatility and reliability of the AUDIT scale established a valid association between alcohol consumption and male AGA risk in our study.

The results revealed that 16 participants scored within the 1-7 range, indicating low to moderate alcohol consumption; 27 participants scored between 8-14, suggesting moderate to high intake, with a smaller group of 13 participants scoring more than 15, indicative of potential alcohol-related issues. Notably, a clear association emerged between the severity of AGA and alcohol intake, as evidenced by the progressive increase in AUDIT scores from a mean of 2.8 in Grade I to 13.2 in Grade VII, with a statistically significant result (p-value=0.0006). These findings underscore the importance of evaluating alcohol use in AGA patients, particularly those exhibiting more severe conditions, as part of more comprehensive patient care.

Present study corroborates the findings of Nargis T et al., which reinforce the association between increased alcohol consumption and the severity of AGA, while Danesh-Shakiba M et al., found no significant link between alcohol consumption and AGA severity [12,16]. This possible association can be explained by alcohol-induced metabolic alterations disrupting the body's macro- and microelement homeostasis, thereby influencing hair growth processes [17].

The comparison of findings in this study with existing literature emphasises a strong connection between smoking and hair loss, shedding light on multiple potential ways in which smoking exacerbates AGA. Present study data, which indicate that 64% of the participants were smokers, closely correspond with a recent meta-analysis conducted by Gupta AK et al., which recognised smoking as a significant risk factor for male AGA [42]. Participants designated as Grade VII in present study demonstrated a notable rise in their average smoking index of 32.80±21.43, suggesting significant smoking exposure compared to lower grades. The findings of this study indicate an association between higher smoking indices and increased severity of male AGA, consistent with Fortes C et al.,'s discovery of a correlation between smoking severity and AGA in men [10]. The possible mechanisms related to smoking as a risk factor, along with its association with greater disease severity in male AGA, as noted by several studies, include oxidative damage to the microvasculature of the dermal hair papilla and genotoxic effects on hair follicles [43]. Additionally, although present study data did not establish a clear association between smoking status and the onset of AGA, it highlights the multifaceted nature of this relationship, pointing to the need for further investigation to fully understand the underlying pathways involved.

This research evaluated the levels of psychological stress in patients with different degrees of AGA using the DASS-21 scale. However, statistically significant findings were not obtained (p-value=0.351), which was consistent with the conclusions drawn by Lewinsohn PM et al., and Huang C et al., [44,45]. Nevertheless, the results of this study differ from a recent systematic review by Aukerman EL and Jafferany M, which indicated an association between psychological stress and AGA [46]. The inconsistencies in these results might be attributed to diverse cultural values, individual differences in coping mechanisms for psychological stress, satisfaction with one's external appearance, healthcare-seeking behaviour and the availability of affordable treatment options among study participants in various regions around the globe. Nonetheless, further research is necessary to discern the role of psychological stress in male AGA.

Limitation(s)

The cross-sectional design of the study limits the ability to establish causal relationships between predictor variables and the severity of AGA. The study's reliance on a specific patient population or recruitment from a particular clinical setting may introduce sampling bias and limit the generalisability of the findings to broader populations. Future studies should aim for more diverse and representative samples. The reliance on self-reported data for variables such as alcohol consumption and smoking may introduce recall bias and underreporting. Utilising objective measures or corroborating self-reports with biochemical assays could enhance data accuracy. Furthermore, the study's findings may not apply to populations with different demographic characteristics or cultural backgrounds. Therefore, replication of the study in diverse populations is essential to confirm the generalisability of the results. Despite statistical adjustments, the presence of unmeasured or residual confounding variables could influence the observed associations. Future studies should consider additional confounders and employ robust statistical methods to address potential biases.

CONCLUSION(S)

The presence of a greater percentage of younger participants in the current study, which assessed the lifestyle characteristics among 100 adult men with AGA, enabled the early identification of lifestyle risk factors that might potentially slow disease progression as well as minimise its severity. The key findings of this study were the associations between alcohol consumption and cigarette smoking with male AGA and its severity, among the various lifestyle risk factors assessed in male AGA. The evaluation of lifestyle risk factors

supports the idea that multifaceted mechanisms are involved in male AGA. Modifying daily habits to avoid or limit these risk factors may help slow down AGA progression and aid in the development of new interventions in future studies.

REFERENCES

[1] Phillips TG, Slomiany WP. Hair loss: Common causes and treatment. Am Fam Physician. 2017;96(6):371-78. PMID: 28925637.

[2] Sehgal VN, Kak R. Male pattern androgenetic alopecia in an Indian context: A perspective study. J Eur Acad Dermatol Venereol. 2007;21:473-79. Doi: 10.1111/j.1468-3083.2006.01920.x. PMID: 17373973.

[3] Trüeb RM. Molecular mechanisms of androgenetic alopecia. Exp Gerontol. 2002;37(8-9):981-90. Doi: 10.1016/s0531-5565(02)00093-1. PMID: 12213548.

[4] Hoffmann R, Happle R. Current understanding of androgenetic alopecia. Part I: Etiopathogenesis. Eur J Dermatol. 2000;10(4):319-27. PMID: 10846263.

[5] Sinclair RD, Dawber RP. Androgenetic alopecia in men and women. Clin Dermatol. 2001;19(2):167-78. PMID: 11397596.

[6] Ellis JA, Sinclair R. Androgenetic alopecia: Pathogenesis and potential for therapy. Expert Rev Mol Med. 2002;4(22):01-11. Doi: 10.1017/S1462399402005112. PMID: 14585162.

[7] Vinay K, Bhattachajee R. Clinical and metabolic characteristics of males with early-onset androgenetic alopecia. Indian J Dermatol Venereol Leprol. 2023;89(4):530-35. Doi: 10.25259/IJDVL_949_2021. PMID: 36688892.

[8] Kaliyadan F, Nambiar A, Vijayaraghavan S. Androgenetic alopecia: An update. Indian J Dermatol Venereol Leprol. 2013;79(5):613-25. Doi: 10.4103/0378-6323.116730. PMID: 23974579.

[9] Su LH, Chen THH. Association of androgenetic alopecia with metabolic syndrome in men: A community-based survey. Br J Dermatol. 2010;163(2):371-77. Doi: 10.1111/j.1365-2133.2010.09816.x. Epub 2010 Apr 23. PMID: 20426781.

[10] Fortes C, Mastroeni S, Mannooranparampil TJ. The combination of overweight and smoking increases the severity of androgenetic alopecia. Int J Dermatol. 2017;56(8):862-67. Doi: 10.1111/ijd.13652. Epub 2017 May 29. PMID: 28555720.

[11] Sinikumpu SP, Jokelainen J. Association between psychosocial distress, sexual disorders, self-esteem and quality of life with male androgenetic alopecia: A population-based study with men at age 46. BMJ Open. 2021;11(12):e049855. Doi: 10.1136/bmjopen-2021-049855. PMID: 39192534; PMCID: PMC8719200.

[12] Nargis T, Bejai V, Pinto M, Shenoy MM. Early onset androgenetic alopecia in men and associated risk factors: A hospital-based study. Int J Res Dermatol. 2017;3(2):267. Doi: 10.18203/issn.2455-4529.IntJResDermatol20172209.

[13] Kavadya Y, Mysore V. Role of smoking in androgenetic alopecia: A systematic review. Int J Trichology. 2022;14(2):41-48. Doi: 10.4103/ijt.ijt_59_21. Epub 2022 Apr 4. PMID: 35531482; PMCID: PMC9069908.

[14] Salem AS, Ibrahim HS, Abdelasis HH, Elsaie ML. Implications of cigarette smoking on early-onset androgenetic alopecia: A cross-sectional study. J Cosmet Dermatol. 2021;20:1318-24. Doi: 10.1111/jocd.13727.

[15] Park SY, Oh SS, Lee WS. Relationship between androgenetic alopecia and cardiovascular risk factors according to BASP classification in Koreans. J Dermatol. 2016;43:1293-300. Doi: 10.1111/1346-8138.13355.

[16] Danesh-Shakiba M, Poorolajal J, Aliresaei P. Androgenetic alopecia: Relationship to anthropometric indices, blood pressure and life-style habits. Clin Cosmet Investig Dermatol. 2020;13:137-43.

[17] Osturk P, Kurutas E, Ataseven A, Dokur N, Gumusalan Y, Gorur A, et al. BMI and levels of sinc, copper in hair, serum and urine of Turkish male patients with androgenetic alopecia. J Trace Elem Med Biol Organ Soc Miner Trace Elem GMS. 2014;28(3):266-70. Doi: 10.1016/j.jtemb.2014.03.003. Epub 2014 Mar 16. PMID: 24746780.

[18] Hadshiew IM, Foitsik K, Arck PC, Paus R. Burden of hair loss: Stress and the underestimated psychosocial impact of telogen effluvium and androgenetic alopecia. J Invest Dermatol. 2004;123(3):455-57. Doi: 10.1111/j.0022-202X.2004.23237.x. PMID: 15304082.

[19] Frith H, Jankowski GS. Psychosocial impact of androgenetic alopecia on men: A systematic review and meta-analysis. Psychol Health Med. 2023;29(4):822-42. Available from: https://doi.org/10.1080/13548506.2023.2242049.

[20] Matilainen V, Laakso M, Hirsso P, Koskela P, Rajala U, Keinänen-Kiukaanniemi S, et al. Hair loss, insulin resistance and heredity in middle-aged women. A population-based study. J Cardiovasc Risk. 2003;10(3):227-31. Doi: 10.1097/01.hjr.0000070200.72977.c6. PMID: 12775957.

[21] Acibucu F, Kayatas M, Candan F. The association of insulin resistance and metabolic syndrome in early androgenetic alopecia. Singapore Med J. 2010;51(12):931-36. PMID: 21221497.

[22] Arias-Santiago S, Gutiérres-Salmerón MT, Castellote-Caballero L, Buendía-Eisman A, Naranjo-Sintes R. Androgenetic alopecia and cardiovasculer risk factors in men and women: A comparative study. J Am Acad Dermatol. 2010;63:420-29. Doi: 10.1016/j.jaad.2009.10.018.

[23] Yang CC, Hsieh FN, Lin LY, Hsu CK, Sheu HM, Chen W. Higher body mass index is associated with greater severity of alopecia in men with male-pattern androgenetic alopecia in Taiwan: A cross-sectional study. J Am Acad Dermatol. 2014;70(2):297-302.e1. Doi: 10.1016/j.jaad.2013.09.036. Epub 2013 Nov 1. PMID: 24184140.

[24] Krupa Shankar D, Chakravarthi M, Shilpakar R. Male androgenetic alopecia: Population-based study in 1,005 subjects. Int J Trichology. 2009;1(2):131-33. Doi: 10.4103/0974-7753.58556. PMID: 20927235; PMCID: PMC2938575.

[25] Blume-Peytavi U, Blumeyer A, Tosti A, Finner A, Marmol V, Trakatelli M, et al. S1 guideline for diagnostic evaluation in androgenetic alopecia in men, women and adolescents. Br J Dermatol. 2011;164:05-15. Doi: 10.1111/j.1365-2133.2010.10011.x. Epub 2010 Dec 8. PMID: 20795997.

[26] Nagata N, Niikura R, Shimbo T, Kishida Y, Sekine K, Tanaka S, et al. Alcohol and smoking affect risk of uncomplicated colonic diverticulosis in Japan. PLoS One. 2013;8(12):e81137. Doi: 10.1371/journal.pone.0081137. PMID: 24339905; PMCID: PMC3858234.

[27] Mahajan VK, Dhattarwal N, Chauhan PS, Mehta KS, Sharma R, Sharma A, et al. The association of alcohol use disorder and chronic plaque psoriasis: Results of a pilot study. Indian Dermatol Online J. 2020;12(1):128-33. Doi: 10.4103/idoj.IDOJ_226_20. PMID: 33768034; PMCID: PMC7982022.

[28] Sakhiya J, Sakhiya D, Modi M, Gandhi S, Daruwala F. Prevalence, severity and associated factor of androgenetic alopecia in the dermatology outpatient clinic: A retrospective study. IP Indian J Clin Exp Dermatol. 2019;5(4):280-87.

[29] Ali AM, Alkhamees AA, Hori H, Kim Y, Kunugi H. The Depression Anxiety Stress Scale 21: Development and validation of the Depression Anxiety Stress Scale 8-Item in psychiatric patients and the general public for Easier Mental Health Measurement in a Post COVID-19 World. Int J Environ Res Public Health. 2021;18(19):10142. Doi: 10.3390/ijerph181910142. PMID: 34639443; PMCID: PMC8507889.

[30] Soliman MM. Depressive, anxiety, stress, and insomnia symptoms in patients with psoriasis: A cross-sectional study. Postepy Dermatol Alergol. 2021;38(3):510-19. Doi: 10.5114/ada.2020.98726. Epub 2020 Sep 18. PMID: 34377136; PMCID: PMC8330873.

[31] Lovibond SH, Lovibond PF. Manual for the depression anxiety & stress scales. (2nd Ed.) Sydney: Psychology Foundation. 1995.

[32] Sawant N, Chikhalkar S, Mehta V, Ravi M, Madke B, Khopkar U. Androgenetic alopecia: Quality-of-life and associated lifestyle patterns. Int J Trichology. 2010;2(2):81-85. Doi: 10.4103/0974-7753.77510. PMID: 21712908; PMCID: PMC3107963.

[33] Segal AS. Alopecia associated with atorvastatin. Am J Dermatol. 2002;113(2):171. Doi: 10.1016/s0002-9343(02)01135-x. PMID: 12133763.

[34] Gupta S, Goyal I, Mahendra A. Quality of life assessment in patients with androgenetic alopecia. Int J Trichology. 2019;11(4):147-52. Doi: 10.4103/ijt.ijt_6_19. PMID: 31523105; PMCID: PMC6706984.

[35] Han SH, Byun JW, Lee WS, Kang H, Kye YC, Kim KH, et al. Quality of life assessment in male patients with androgenetic alopecia: Result of a prospective, multicenter study. Ann Dermatol. 2012;24(3):311-18. Doi: 10.5021/ad.2012.24.3.311. Epub 2012 Jul 25. PMID: 22879715; PMCID: PMC3412240.

[36] Chen S, Xie X, Zhang G, Zhang Y. Comorbidities in androgenetic alopecia: A comprehensive review. Dermatol Ther (Heidelb). 2022;12(10):2233-47. Available from: https://doi.org/10.1007/s13555-022-00799-7.

[37] Anastassakis K. Hormonal and genetic etiology of male androgenetic alopecia. In: Androgenetic alopecia from A to S. Springer, Cham. 2022. Available from: https://doi.org/10.1007/978-3-030-76111-0_11.

[38] Jang WS, Son IP, Yeo IK, Park KY, Li K, Kim BJ, et al. The annual changes of clinical manifestation of androgenetic alopecia clinic in Korean males and females: A outpatient-based study. Ann Dermatol. 2013;25(2):181.

[39] Hirsso P, Rajala U, Hiltunen L, Jokelainen J, Keinänen-Kiukaanniemi S, Näyhä S, et al. Obesity and low-grade-inflammation among young finnish men with early-onset alopecia. Dermatology. 2007;214:125-29.

[40] Gopinath H, Upadya GM. Metabolic syndrome in androgenic alopecia. Indian J Dermatol Venereol Leprol. 2016;82(4):404-08. Doi: 10.4103/0378-6323.174421. PMID: 27279298.

[41] Vora RV, Kota RKSK, Singhal RR, Anjaneyan G. Clinical profile of androgenic alopecia and its association with cardiovascular risk factors. Indian J Dermatol. 2019;64(1):19-22. Doi: 10.4103/ijd.IJD_526_16. PMID: 30745630; PMCID. PMC6340244.

[42] Gupta AK, Bamimore MA, Talukder M. A meta-analysis study on the association between smoking and male pattern hair loss. J Cosmet Dermatol. 2024;23(4):1446-51. Doi: 10.1111/jocd.16132. Epub 2024 Jan 4. PMID: 38174368.

[43] Su LH, Chen TH. Association of androgenetic alopecia with smoking and its prevalence among Asian men: A community-based survey. Arch Dermatol. 2007;143(11):1401-06.

[44] Lewinsohn PM, Seeley JR, Roberts RE, Allen NB. Center for Epidemiologic Studies Depression Scale (CES-D) as a screening instrument for depression among community-residing older adults. Psychol Aging. 1997;12(2):277-87. Doi: 10.1037/0882-7974.12.2.277.

[45] Huang C, Fu Y, Chi C. Health-related quality of life, depression, and self-esteem in patients with androgenetic alopecia: A systematic review and meta-analysis. JAMA Dermatol. 2021;157(8):963-70. Doi: 10.1001/jamadermatol.2021.2196.

[46] Aukerman EL, Jafferany M. The psychological consequences of androgenetic alopecia: A systematic review. J Cosmet Dermatol. 2022;22(1):89-95. Available from: https://doi.org/10.1111/jocd.14983.

PARTICULARS OF CONTRIBUTORS:
1. Postgraduate Student, Department of Dermatology, Venereology and Leprosy, Hind Institute of Medical Sciences, Barabanki, Uttar Pradesh, India.
2. Associate Professor, Department of Dermatology, Venereology and Leprosy, Hind Institute of Medical Sciences, Barabanki, Uttar Pradesh, India.
3. Associate Professor, Department of Dermatology, Venereology and Leprosy, Hind Institute of Medical Sciences, Barabanki, Uttar Pradesh, India.
4. Assistant Professor, Department of Dermatology, Venereology and Leprosy, Hind Institute of Medical Sciences, Barabanki, Uttar Pradesh, India.
5. Assistant Professor, Department of Dermatology, Venereology and Leprosy, Hind Institute of Medical Sciences, Barabanki, Uttar Pradesh, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Sonal Sachan,
Assistant Professor, Department of Dermatology, Venereology and Leprosy,
Hind Institute of Medical Sciences, Safedabad,
Barabanki-225001, Uttar Pradesh, India.
E-mail: sonalsachan900@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Oct 03, 2024
- Manual Googling: Jan 24, 2025
- iThenticate Software: Feb 13, 2025 (10%)

ETYMOLOGY: Author Origin

EMENDATIONS: 8

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Oct 01, 2024**
Date of Peer Review: **Nov 21, 2024**
Date of Acceptance: **Feb 15, 2025**
Date of Publishing: **Jun 01, 2025**