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IP Indian Journal of Clinical and Experimental Dermatology

Journal homepage: www.innovativepublication.com

Original Research Article

Prevalence, severity and associated factor of androgenetic alopecia in the dermatology outpatient clinic: A retrospective study

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ARTICLE INFO

Article history:
Received 15-10-2019
Accepted 07-11-2019
Available online 20-12-2019

Keywords:
Androgenetic alopecia
Prevelance
Severity
Factor

ABSTRACT

Introduction and Objectives: Androgenetic alopecia (AGA) is a patterned hair loss that occurs due to systemic androgen and genetic factors. In both genders, it is the most common cause of hair loss. Individual affected by AGA is suffering from social stigma, psychological distress and emotional misery. The purpose of this study was to investigate the prevalence and severity of androgenetic alopecia in the Indian population. Additionally, we have studied the association of family history, accompanying systemic diseases, body mass index [BMI], alcohol consumption and smoking, menstrual cycle and hyperandrogenemia with androgenetic alopecia in patients who were referred to our outpatient clinic.

Materials and Methods: The study included patients who referred to our clinic from November 2016 to October 2017. A total of 210 patients suffering from AGA were included in the study. To evaluate the factors associated with AGA, a control group (non-AGA group, 50 patients) was organized. The diagnosis of androgenetic alopecia was made upon clinical findings only. Patient's age, gender, weight, height, the pattern of hair loss, the history of other systemic diseases, family history of AGA, concomitant hyperandrogenemia symptoms, alcohol consumption and smoking habits have been reported. Details regarding the menstrual cycle have been recorded in women.

Results: The prevalence of androgenetic alopecia was 79% in men and 21% in women. Type I and II were the most common type in men, and grade I was in women. In men, AGA prevalence (Men: n=166, r =0.728, P =.164) was positively correlated with age but, a statistically significant difference could not be obtained and, severity was positively correlated with age (n=166, r =0.174, P =.025). While, in women, prevalence (n=44, r =0.020, P =.975) and severity (n=44, r =0.090, P =.575) was not correlated with age. In the multivariate model, family history (OR : 16.86, 95% CI : 5.14-55.30, P =.000) was statistically significant covariates positively associated with AGA.

Study Limitations: Although other causes of alopecia are omitted, it is difficult to differentiate Ludwig grade 1 AGA from telogen effluvium based on only clinical features.

Conclusions: AGA prevalence was higher than, the previous studies in Asians and Caucasians. In India, type I and II was the most common type of AGA in males, while grade I was the most common type of AGA in females. Family history seems to be a pivotal risk factor for AGA.

1. Introduction

Androgenetic alopecia (AGA) is the most common type of hair loss occurs in Asian men due to excessive response to androgens, which affects up to 50% of males and females after the onset of puberty. It is characterized by gradual loss of terminal hair of the scalp, with a characteristic pattern in both males and females. In men, this condition is also known as male-pattern hair loss, whereas, in females, it is termed as female-pattern hair loss. Hair loss is most dominant in males over the vertex and frontotemporal regions. In women, the frontal hairline is typically spared with diffuse apical hair loss noted as a broader anterior part of the hair partition. [1–3]

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https://doi.org/10.18231/j.ijced.2019.060

The prevalence of AGA varied with race. White patients are most affected, followed by Asians and African Americans, then Native Americans and Eskimos. Prevalence is in Caucasians considered to be the highest.[4,5]

AGA in men is associated with various medical conditions such as coronary heart disease and prostate enlargement. Additionally, prostate cancer, disorders of insulin resistance (such as diabetes and obesity), and high- blood pressure (hypertension) are also associated with AGA. The disorder is common in females, mainly after the onset of menopause. This hair loss pattern is also associated with an increased risk of polycystic ovary syndrome, characterized by a hormonal imbalance that may contribute to irregular menstruation, acne, hirsutism, and weight gain.[6]

In men, pattern hair loss is assumed to be due to a blend of hereditary qualities and the male androgen hormone dihydrotestosterone. It is caused by androgen-mediated follicular miniaturization, which triggers fine, short, non-pigmented, vellus hair formation. The pathogenesis of female-pattern hair loss has not been well characterized, and in most women, androgens are not expected to play a prominent role.[7] Among researchers, the pathogenesis of androgenic alopecia in females has become a matter of interest in this new era. Female pattern hair loss (FPHL) has risen as the preferred term for androgenetic alopecia in females, inferable from the dubious relationship between androgens and this entity.[8]

Our primary objective of the research was to assess the prevalence and severity of AGA in India. Our secondary objective was to examine the associations between AGA and etiological factors (family history, accompanying systemic diseases, body mass index [BMI], alcohol consumption and smoking, menstrual cycle and hyperandrogenemia) in our outpatient clinic.

2. Materials and Methods

In compliance with the Helsinki Declaration, a retrospective observational study was carried out at our private dermatology clinic during the period from November 2016 to October 2017.

2.1. Inclusion criteria

Patients attending the dermatology OPD with patchy or diffuse hair loss, smooth bald surface and no scarring, scaling or inflammation features on the bald area were included in the study. Patients older than 16 years of age who had not received any treatment in the previous 3 months were also included in the study. Patients having AGA only on the scalp were included.

2.2. Exclusion criteria

Our study excluded the following groups:

1. Pregnant women
2. Patients of thyroid and cancer:

2.3. Methods

We have consecutively enrolled 210 patients suffering from AGA according to the predefined inclusion and exclusion criteria. We established a control group (non-AGA group) comprising 50 healthy volunteers deemed to be free of AGA to determine the factors associated with AGA. The diagnosis was made only through clinical examination. Information obtained included patient demographics such as age, gender, weight, height, the pattern of hair loss, the history of other systemic diseases [Hypertension (HT), Diabetes mellitus (DM), Hyperlipidemia (HL)], family history of AGA, alcohol consumption and smoking habits. In women, details regarding the menstrual cycle were reported. Concomitant hyperandrogenaemia symptoms (acne vulgaris, seborrhoea and hirsutism) were also investigated.

BMI (Height/Weight^2 [kg/m^2]) was used to assess the presence of obesity. Patients are classified as follows on the basis of BMI: $<$25 kg/m^2 = normal; 25-30 kg/m^2 = overweight; $\geq$30 kg/m^2 = obese.

The severity of AGA was evaluated using Norwood-Hamilton scale[9,10] in men and Ludwig classification[11] in women.

2.4. Male-pattern baldness (Norwood-Hamilton scale)[9,10]

1. Type I: Minimal hair loss.
2. Type II: Minor recession of the frontotemporal hairline.
3. Type IIIa : The area of recession of the frontotemporal region is almost vertical with the front portion of the ear.
4. Type IIIv : In this type, hair loss is primarily in the vertex with possibly some frontal recession.
5. Type IV: Severe hair loss, especially in the frontal and frontotemporal hair, and significant diffuse hair thinning over the vertex. There is a broad band that separates vertex and the top of hair.
6. Type V: Hair loss at the vertex region is still separated from the frontotemporal region but the division is much less distinct.
7. Type VI: The bridge of hair that once crossed the crown is now been lost with only sparse hair remaining.
8. Type VII: Only a narrow band of hair in a horseshoe shape survives on the sides and back of the scalp.

2.5. Female-pattern AGA (Ludwig classification)[11]

1. Grade I: Perceptible thinning of the hair on the crown with preservation of the frontal hairline
2. Grade II: Pronounced thinning of the hair on the crown
3. Grade III: Total baldness of the hair on the crown

2.6. Statistical analysis

The Statistical Package for Social Sciences (SPSS 21) for Windows program was used for statistical analysis. The prevalence rates and severity were presented as percentages and 95% confidence interval (CI). The Pearson correlation analysis was used to assess the correlation between prevalence and severity of AGA with age in both genders. To assess the association of etiological factors and AGA, univariate and multivariate binary logistic regression analyses were employed. The level of significance level (α) was set at 0.05.

3. Results

3.1. Prevalence and severity of AGA in men and women according to age

The study included 210 patients (166 men, 44 women) with a mean age of onset 30.98±9.27 years. It was found that the prevalence of AGA was 79% (n=166) in men and 21% (n=44) in women. Mean age's were31.80±8.20, 40.76±9.08, 38.05±11.06, 40.89±12.55 in men with AGA, men without AGA, women with AGA and women without AGA, respectively. In men, AGA prevalence (Men: n=166, r =0.728, P =.164) was positively correlated with age but, a statistically significant difference could not be obtained and, severity was positively correlated with age (n=166, r =0.174, P =.025). While, in women, prevalence (n=44, r =0.020, P =.975) and severity (n=44, r =0.090, P =.575) was not correlated with age. In the present study, most of the patients belonged to the third decade of life (21-30 years), suggesting about 52% of the entire study population, followed by the fourth decade of life, which is the second most common age group for AGA (27%). In the AGA, male predominance was higher than female in the second, third, and fourth decades; however, in the following two decades, the prevalence in females was higher (Table 1).

The mean age of AGA onset was 29.64±8.27 in men and 36.07±11.03 in women.

Men: Type I and II (n=38/166, 23 %, n=39/166, 23%, respectively) was the most common type, whereas IIIa (n=6/166, 4%) was the least common type. Type II was the most common type in men between 11-20 years of age, whereas type IV, V and VI did not identify in this age group. Type I was the most common type in men between 21-30 years of age, while type II was the most common type in men between 31- 40 years of age. Type IIIv was the most prevalent among the 41-50 years of age and type I, IIIa, VI were absent in this age group. Type IV was the most common type in men over 50 years of age, whereas the prevalence of type II, IIIV and V were similar (Table 2).

Women: The most common type was grade I (28/44, 64%) in women and, the least common type was grade III (3/44, 7%). AGA prevalence was found to be 63% among women over 50 years of age, 86% among women aged 21-30 years and no prevalence among women aged 11-20 years. Prevalence of AGA among women aged group 31-40 and 41-50, respectively, was 83% and 91%. The most common type in women over 50 years of age was grade II and grade III, in the rest of the women grade I was the most common type (Table 3).

3.2. Factors affecting AGA

In total, 260 patients including AGA group (n=210) and non-AGA group (n=50) were studied to assess the factor associated with AGA. In the univariate model, age at onset (21-30 years, *OR* :7.27, 95% *CI* : 2.40-1.92, *P* =.000; 31-40years, *OR* :3.45, 95% *CI* :1.14-10.44, *P* =.00 8) and family history (Present, *OR* :15.33, 95% *CI* :5.33-44.15, *P* =.000) were statistically significant covariates positively associated with the AGA, whereas age at onset (41-50 years, *OR* : 0.94, 95% *CI* : 0.30-2.92, *P* =.029) were statistically significant covariates negatively associated with the AGA. Subsequent multivariate analysis showed that age groups, 21-30 years (*O*R :33.89, 95% *CI* :5.14-223.35, *P* =.000) and 31-40 years (*O*R : 11.68, 95% *CI* :1.90-71.97, *P* =.008) were independently positively associated with AGA. The family history was statistically significant covariates positively associated with AGA (*O*R :16.86, 95% *CI* :5.14-55.30, *P* =.000). Additionally, age at onset (11-20 and 41-50), gender, concomitant hyperandrogenemia symptoms(acne vulgaris, seborrhea, hirsutism, gynecomastia), BMI(<25,25-30), personal history of systemic disease (HT, DM, HT and DM), alcohol consumption and smoking habits and menstrual cycle (regular, irregular, menopause) were not associated with AGA with p-values of .998, .553,1.00,.862,.999,.999,1.00,1.00,1.00,.069,.091,.999,.496, .143,1.00,1.00,1.00 respectively (Table 4).

4. Discussion

Various outcomes have been documented regarding the prevalence of AGA, especially in men based on ethnic gatherings regarding. According to Hamilton's 1951 study, by the age of 30 years, the mean prevalence was 30%, 40% in mid-forties and this rate ascends to 50% by the age of 50 years in Caucasian men.[11] Studies from the United States, Italy, Norway, and Australia showed similar findings to those reported in studies by Hamilton.[12–15] Based on findings, the prevalence rate has risen with increased age in these ethnic gatherings. These rates were 28%, 6% and 3% respectively in our study, reflecting contradictory findings from the previously mentioned studies. In the Indian context, Krupa Shankar et al[16] firstly conducted a population-based study of 1005 subjects showing 74% and 26% AGA prevalence, respectively, in males aged 30-40 and 41-50 years. Our findings are somewhat higher in men (79%) and slightly lower in women (21%) that go

Table 1: Prevalence of AGA according to age wise and sex wise distribution CI: Confidence interval.

Age grade	No. with disorder (Men/women)	Prevalence % (95% CI)		
		All(n=210)	Men(n=166)	Women(n=44)
Total	210(166/40)	0	79(73-85)	21(15-27)
11-20	15(15/0)	7(3-10)	90(85-94)	0
21-30	109(90/19)	52(45-59)	54(46-61)	43(28-58)
31-40	56(46/10)	27(21-33)	28(21-35)	23(10-35)
41-50	20 (10/10)	9(5-13)	6(2-10)	23(10-35)
51-60	10(5/5)	5(2-8)	3(0.4-6)	11(2-20)

¥ The severity of AGA was evaluated with Ludwig classification in women.

Table 2: Prevalence of severity of AGA in men according to age groups

Severity Grade	Age grade				
	11-20 n(%) (n=15)	21-30 n(%) (n=99)	31-40 n(%) (n=57)	41-50 n(%) (n=26)	51-60 n(%) (n=10)
Non-AGA(n=50)	0	9(9%)	11(20%)	16(61%)	5(50%)
Male(n=166) Male	15(100%)	90(91%)	46(81%)	10(38%)	5(50%)
AGA type I Male	6(40%)	26(26%)	6(10%)	0	0
AGA type II Male	7(46%)	20(21%)	10(18%)	1(4%)	1(10%)
AGA type IIIa Male	1(7%)	3(3%) 15(15%)	2(4%)	0	0
AGA type IIIv Male	1(7%)	6(6%)	9(16%)	5(19%)	1(10%)
AGA type IV Male	0	9(9%)	6(10%)	3(12%)	2(20%)
AGA type V Male	0		7(12%)	1(4%)	1(10%)
AGA type VI	0		6(10%)	0	0

¥ The severity of AGA was evaluated with Norwood-Hamilton scale in men.

Table 3: Prevalence of severity of AGA in women according to age groups

Severity Grade	Age grade				
	11-20 n(%) (n=0)	21-30 n(%) (n=22)	31-40 n(%) (n=12)	41-50 n(%) (n=11)	51-60 n(%) (n=8)
Non-AGA(n=50)	0	3(13%)	2(17%)	1(9%)	3(38%)
Female(n=44)	0	19(86%)	10(83%)	10(91%)	5(63%)
Grade I	0	14(64%)	6(50%)	7(64%)	1(12%)
Grade II	0	5(23%)	3(25%)	3(27%)	2(25%)
Grade III	0	0	1(8%)	0	2(25%)

¥ The severity of AGA was evaluated with Ludwig classification in women.

against previously reported verdicts by Krupa Shankar et al. Findings obtained from the previous study and the current study in the Indian population, prevalence based on age-wise distribution follow a different path compared to other ethnic groups. This contrast might be due to variations in genetic and environmental factors among different ethnic group. Interestingly, the age-specific prevalence of AGA in Indians was also inconsistent with findings from other Asians, such as Japanese, Korean, Taiwanese and Chinese, reported by Lee WS and Lee HJ.[17] Future studies with a large sample size will be needed for better clarification of this affair.

The prevalence also decreased in women with increased age and the rates were 43%, 23%, 23%, 11% for the age group 21-30, 31-40, 41-50 and 51-60 years, respectively. Birch et al reported that, in England, the prevalence of female type AGA in under the age of 50 years was 6 %, whereas, in women over the age of 50 years it was 52.6%.[18] Nevertheless, in our study, the prevalence of female type AGA in women younger than 50 years and older than 50 years was found to be 89% and 11%, respectively.

Salman et al[19] reported that in Caucasian men, the prevalence of AGA was 50% and, in Caucasian women, it was 19%. Khumalo et al[20] stated that the prevalence of AGA was 14.6% in African men and 3.5% in African women, much lower than in Caucasians. In Koreans, the prevalence of AGA was 14.1% in men and 5.6% in women,[21] which was much lower than in Caucasians and close to predominance in Africans. The study reported from Thailand found that there was 38.52% prevalence of AGA in Thai people, almost close to that of Caucasians.[22] Besides, studies performed in Shanghai and Taiwan found that the prevalence of AGA in Chinese individuals was like that in Koreans.[23,24] In our study, AGA prevalence was found to be 67.1% of men and 23.9% in women, much higher than in Caucasians.

Table 4: Univariate and multivariate (enter the method of analysis) binary logistic regression analyses showing factors associated with AGA

Characteristics	AGA group (n=210)	Non-AGA group (n=50)	Crude OR (95% CI)	*P* value	Adjusted OR (95% CI)	*P* value
Age at onset in	30.98±9.27	0	1292379874(0.00-HN)	.998	7065533012	.998
years (mean±SD)	15(7%)	12(24%)	7.27(2.40-1.92)	.000***	(0.00-HN)	.000***
11-20	109(52%)	13(26%)	3.45(1.14-10.44)		33.89 (5.14-223.35)	
21-30	56(27%)	17(34%)	0.94(0.30-2.92)	.008**	11.68 (1.90 -71.97)	.008**
31-40	20(9%)	8(16%)	Reference	.029* -	1.74 (0.29-10.57)	
41-50	10(5%)				Reference	.553 -
51-60						
Gender (%)	44(21%)	9(18%)	0.83(0.37-1.83)	.642 -	0.00	1.00 -
Female	166(79%)	41(82%)	Reference		Reference	
Male						
Family history of	120(57%)	4(8%)	15.33(5.33-44.15)	.000***	16.86(5.14-55.30)	.000***
AGA (%)	90(43%)	46(92%)	Reference	-		-
Present						
Absent						
CHS	28(13%)	3(6%)	2.708(0.79-9.30)	.114	0.87(0.19-4.08)	.862
Acne vulgaris	12(6%)	0	468687145.8(0.00-HN)	.999	148823082.9(0.00-	.999
Seborrhea	7(3%)	0		.999	HN)	.999
Hirsutism	1(0.5%)	0	468687145.8(0.00-HN)	1.00	1117384357(0.00-	1.00
Gynecomastia	162(76%)	47(94%)		.644	HN)	1.00
Absent			468687145.8(0.00-HN)		609942284.1(0.00-	
					HN)	
			Reference		Reference	
BMI	171(81%)	34(60%)	8124099235(0.00-HN)	1	1182255621 (0.00-	1.00
<25	39(19%)	16(32%)	4199826739(0.00-HN)	1	HN)	1.00
25-30	0	0	Reference	.180	1056773592(0.00-	.975
≥30					HN)	
					Reference	
History of other	26(12%)	3(6%)	2.02(0.58-7.02)	.267	4.90(0.88-27.18)	.069
systemic disease	11(5%)	5(10%)	0.60(0.18-2.03)	.418	4.53(0.79-26.04)	.091
HT	2(1%)	0	1.3	.999	398278327.0(0.00-	.999
DM	0	0	-	-	HN)	-
HT and DM	173(82%)	42(84%)	Reference	.729	-	.166
HL					Reference	
No systemic disease						
Alcohol	17(8 %)	5(10%)	0.79(0.28-2.26)	.664	1.69(0.37-7.65)	.496
consumption	193(92%)	45(90%)	Reference	-	Reference	-
Alcoholics						
Non-Alcoholics						
Smoking habit	35(17%)	3(6%)	0.30	.067	3.01(0.69-13.12)	.143
Smoker	175(83%)	47(94%)		-	Reference	-
Non-smoker						
Menstrual cycle in	26(12%)	4(8%)	1.59(0.58-4.82)	.408	0.00	1.00
women	9(4%)	2(4%)	1.10(0.23-5.30)	.901	0.00	1.00
Regular	9(4%)	3(6%)	0.66(0.17-2.58)	.545	0.00	1.00
Irregular	166(79%)	41(82%)	Reference	.766	Reference	-
Menopause						
Absent(men)						

¥ OR, odds ratios differ significantly: * P <.05,**P <.01,***P <.001 from 1.0. CI: Confidence interval; AGA: Androgenetic alopecia; CHS: Concomitant hyperandrogenemia symptoms; BMI: Body mass index; HN: Highly negative; HT: Hypertension; DM: Diabetes mellitus.

In the United States, Rhodes et al[25] conducted a study on European-American men showing predominance for frontal baldness (Type A variant in Norwood-Hamilton classification) in 12% and a Type III or worse pattern in 16% of males aged 18-29 years old, which gradually increased to 53% in those aged 40-49 years old. Type I (31%), followed by Type II (26%) and Type V or worse (20%), was most commonly reported by DeMuro-Mercon et al[26] from a study of 20-50 year old Norwegian men. Besides, the most common type in the Korean population was type VI over 70 years of age and type IIIv around 30-70 years of age.[21] As per reported data, type II (27.27%) followed by type I(22.12%) and then type III (21.78%) was the most common pattern of alopecia in Indians.[5] In our study, the most common type was type IV beyond 50 years age, which opposed to the outcome of the Korean population and recently reported Indian reports; type IIIv between the third decade to the six-decade was almost like to Korean people, however, argued to the findings of the Indian population. Female pattern AGA was seen in 12 men (2.9%) and 13 men (1.8%) within the study reported from Turkey and Taiwan.[27] Numerous studies, however, have reported a substantial quantity of female pattern AGA in men.[12,21,23] Such results suggest integrating female pattern AGA into the category of male pattern AGA. Nonetheless, female pattern AGA was not observed in men in the presented study.

Paik et al[21] reported that Ludwig grade I was the most commonly recognized type in women yo unger than 60 years and, grade I and II more common in women with age more than 60 years. In contrast, in any case, they did not identify grade III. In our investigation, as per Ludwig classification, grade I was the most well-known type in women younger than 50 years, and grade II and grade III was the most frequent in those older than 50 years. It has been found that the prevalence and types of AGA vary among races and provinces.

In this study, we found that the prevalence and severity of AGA were positively correlated with increased age in men, while in women both were not correlated with age. Conversely, Salman et al[19] reported a correlation between the severity and prevalence of AGA with age. This result might be attributed to the large discrepancy in sample size between these two studies.

AGA is considered a genetically predisposed disorder. Throughout Korea, 48.5% of men and 45.2% of women had a family history of baldness. In our study, 61% of men and 41% of women had a positive family history. Such statistics are higher for men than those published in Koreans and Shanghai (men 55.8%). For women, this was lower than the figures reported in Koreans[21] and, higher than those reported in Shanghai (32.4%).[24]

In Turkey, 78.28% of men and 71.02% of women had a family history of baldness, which was the highest compared to all previously reported figures.[19] The explanation behind the variety in family ancestry in various investigations is obscure. One conceivable reason might be the contrast in hereditary factors between study populations. Notably, our study also demonstrated that those with a family history were 15.33 times more likely to perceive AGA as a problem than a person without AGA.

The study conducted by Severy et al[12] reported no significant difference between the presence of acne and AGA. Salman et al[19] hypothesised that the incidence of seborrhea and acne among women with AGA was significantly higher than among women without AGA. In this study, the frequency of previously mentioned concomitant hyperandrogenemia symptoms in women with AGA was higher than in women without AGA. In particular, seborrhea and gynecomastia were positively associated with AGA. Acne and hirsutism were negatively associated with AGA, although, there was no statistically significant difference was obtained.

Menstruation was negatively associated with AGA in the present study, but it was not statistically significant. We observed that postmenopausal women had a higher frequency of AGA than premenopausal women. The higher number of women with increased age in AGA relative to those without AGA may explain this finding.

In our study, in the AGA group, 81% were normal weight and, 19% were overweight. There was no obesity patient. Here, we note BMI were more likely to be associated with AGA, yet, no significant *P* values were obtained. Reports were obtained in contrast to other similar published studies.[19,23,28,29] An association between AGA and BMI was accounted for in only one of the four previous case-control studies.[30–33]

Studies reported by Ellis et al,[28] Ahouansou et al[34] and Aslan[35] revealed, increased hypertension rate in AGA patients. Conversely, in other studies, there were no significant differences in terms of risk factors of CVD between men with and without AGA.[36] In our study, hypertension and diabetes mellitus was positively associated with AGA but, statistically significant difference s could not be obtained.Studies on the association between metabolic syndromes, DM and AGA have reported contradictory results.[23,36–38]

Controversy, results were reported between alcohol consumption/smoking and AGA in two studies.[12,23] We concluded that alcohol consumption and smoking positively correlated with AGA as no significant difference was observed.

5. Limitations

The study is limited due to smaller sample size and retrospective nature. Although other causes of alopecia were excluded, differentiation of Ludwig grade I from telogen effluvium without further tests and biopsy is difficult.

6. Conclusions

In our study, the prevalence of AGA was higher than, the previous studies in Asians and Caucasians. This could be due to our research's outpatient clinic-based design. According to our findings, in men, the most common type of AGA was type I and II, whereas, in women, it was grade I. Family history appears to be a pivotal risk factor for AGA.

7. Source of Funding

None.

8. Conflict of Interest

None.

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Cite this article: 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. *Indian J Clin Exp Dermatol* 2019;5(4):280-287.