## Original Research Article

# Early onset androgenetic alopecia in men and associated risk factors: a hospital based study

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### ABSTRACT

**Background:** Androgenetic alopecia (AGA) is the most common form of hair loss affecting up to 80% of the men. It manifests mostly after puberty and is evident by the age of 30. Nowadays the onset of AGA is much earlier and most of them develop AGA in early twenties. Risk factors such as smoking, alcohol consumption and prevalence of AGA in the family are considered to contribute to the early onset. Aims: To estimate the prevalence of and to determine risk factors for AGA in adult males.
**Methods:** A total of 103 patients above 18 years of age attending the dermatology OPD were included in the study. Epidemiological data was collected using a standard questionnaire.
**Results:** In the study population, 68% patients were in the age group 21-30 years. No association was noted between smoking and the age of onset of AGA. An early age of onset was associated in patients with history of alcohol consumption (73.3%). Prevalence of familial AGA was seen in 68% and had a paternal inheritance (62.8%) more than maternal (8.6%). Associated systemic diseases were seen in 12.6% of the patients and hypertension was the most common.
**Conclusions:** AGA is a very common presenting complaint in the younger population. The early onset of AGA itself causes anxiety and apprehension in the patients that further contribute to the hair loss. The presence of a family history in the patients' needs for an early management of the disorder. Environmental factors like smoking and alcohol consumption thought to play a role in the aetiology of the disease.

**Keywords:** Androgenetic alopecia, Smoking, Inheritance, Alcohol

## INTRODUCTION

Androgenetic alopecia (AGA) is the progressive loss of hair usually seen in genetically predisposed males and occasionally in females. It occurs as a result of androgen mediated conversion of terminal hairs into vellus hairs. It commonly begins by 20 years of age and affects nearly 50% of men by the age of 50 years and 50% of women by the age of 60 years.[1,2]

The propensity to develop AGA is polygenic and not Mendelian.[3] In general, sons of fathers who have no alopecia are at a low risk of hair loss themselves. Risk increases in men with a positive maternal grandfather history and even more so in men with a history of paternal alopecia.[4] Environmental factors such as smoking and alcohol intake may play a role in the pathogenesis of AGA. The association between smoking and AGA has been addressed earlier in a few studies with inconsistent results.[5-7]

The first objective of this study was to estimate the age of onset of AGA in the study population. The second objective was to also to explore possible risk factors associated with early onset of AGA.

## METHODS

Male patients attending the Yenepoya Medical College Hospital with complaints of patterned hair loss. This study was conducted from January 2015 to September 2016 from the male patients with AGA at Yenepoya Medical College Hospital, Deralakatte, Mangalore. We analysed 103 patients who attended the outpatient department with complaints of hair loss and were diagnosed with AGA. We excluded patients with other causes of non- scarring alopecia or with precipitating factors such as high fever, emotional stress, surgery or drugs. The patients were evaluated using a questionnaire to ascertain the perceptible duration of hair loss, any family history of hair loss, presence of chronic disease or chronic drug history, and drinking and smoking habits. In addition to classification of AGA, we collected information on age at onset of AGA, duration of hair loss together with smoking and other possible risk factors using a face-to-face questionnaire interview. The patients with an onset of AGA before 35years were classified as early onset.[10]

Smoking status (never or current) and quantum of smoking were documented. In addition to smoking status, we categorized cigarette smoking into 2 groups (current smoker of <20 cigarettes per day, or current smoker of >20 cigarettes per day) for comparison. We also collected information on alcohol consumption. The family history of AGA among first and second-degree relatives and siblings were determined. The past medical history was also obtained on hypertension, diabetes mellitus, ischemic heart diseases and bronchial asthma.

Body mass index was calculated. Blood pressure (BP) was measured and those patients who had systolic BP >140 mmHg and diastolic BP >90 mmHg, it was measured twice with at least a 10 minute interval between measurements. Hypertension was defined as a systolic blood pressure of 140 mmHg or higher and a diastolic blood pressure of 90 mmHg or higher or as taking antihypertensive medication

### Statistical analysis

Collected data was analysed by both descriptive and inferential statistical methods. To summarise the data frequency, percentage, median were used. To measure the significance Chi square test, Fishers exact test and binomial test were used. Analysis was performed using SPSS software version 13. P value less than 0.05 and less than 0.001 were considered significant and highly significant respectively.

## RESULTS

### Age distribution and age of onset

Among the patients attended with AGA, 68% to the age group 21-30 years followed by 20% in the age group 31-40 years, 7% in the age group 41-50 years and 1% in above 50 years of age (Figure 1). Out of the 103 patients, 51 (49.5%) patients had hair loss between 19- 23 years of age. The median age of onset was 23 years. Among the 103 patients, 63 (61.2%) patients presented within 1-4 years of onset of hair loss.
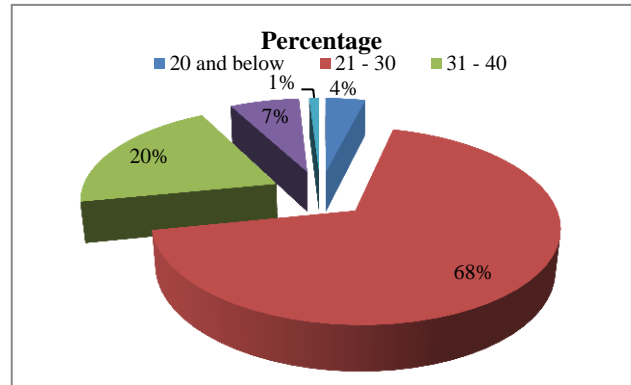


**Figure 1: Prevalence of AGA based on the age group.**

### Risk factors associated with AGA

*Smoking and alcohol history*

Forty-one (39.8%) patients among the 103 gave a history of smoking. Among the 41 smokers, 22 smoked less than 20 cigarettes/day and had onset of AGA before 35 years of age and was statistically significant with a p value of 0.014. Among the patients who smoked more than 20 cigarettes per day, 13 patients had onset of AGA before 35 years and 6 patients had onset after 35years of age and was statistically insignificant. However, no statistically significant associations were found for intensity of smoking. Out of the 15 patients with history of alcohol consumption, 11 (73.3%) had an early onset of AGA and p value was statistically significant <0.001 (Figure 2).
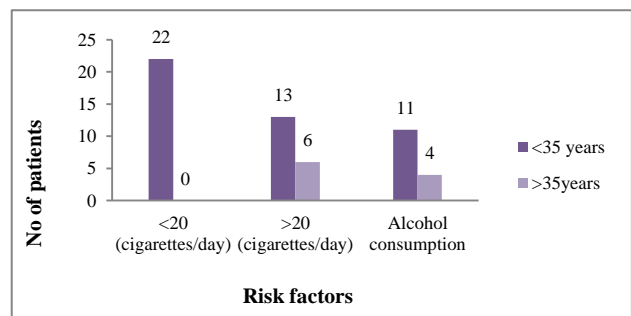


**Figure 2: Distribution of cases based on the smoking and alcohol consumption with the age of onset of hair loss.**

*Family history*

A positive family history for AGA was present for 70 (68%) of the 103 male patients. Patients with a family history, 44 (62.8%) patients had a similar history among their father and paternal first degree relatives with a highly significant p value of 0.000145 (Figure 3). In addition, the age of onset of AGA was significantly lower for those with a family history (69.8%) than for those without a family history of AGA with a statistically significant with a p value <0.05.
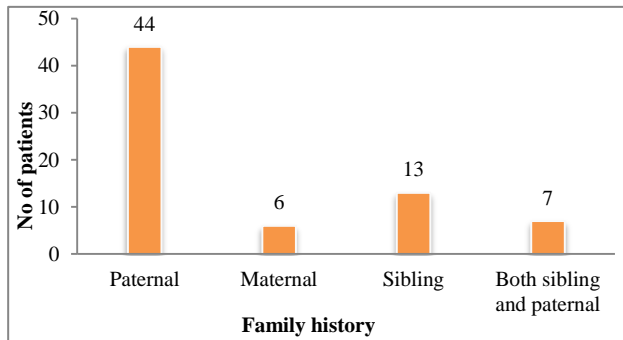


**Figure 3: Distribution of cases based on the prevalence of AGA in the family.**

**Table 1: Association between family history, hypertension, body mass index and age of onset of AGA.**

| Risk factors | Age of onset | | p value |
|---|---|---|---|
| | <35 years(n) | >35years(n) | |
| **Family history** | | | |
| Yes | 67 (69.8%) | 3(42.9%) | p<0.05 |
| No | 29(30.2%) | 4(57.1%) | |
| **HTN** | | | |
| Yes | 6(6.3%) | 4(57.1%) | p =0.37 |
| No | 90(93.7%) | 3(42.9%) | |
| **BMI (kg/m$^2$)** | | | |
| <25 | 70 (72.9%) | 5(71.4%) | p<0.05 |
| >25 | 26 (27.1%) | 2 (28.6%) | |

*Body mass index (BMI)*

Twenty eight (27.2%) patients among the 103 patients were overweight with BMI >25 kg/m$^2$. However, it was noticed that 70 patients had BMI <25 kg/m$^2$ and onset of AGA before 35 years.

*Systemic diseases*

Among the 103 patients, 10 (9.7%) patients had hypertension and were on treatment (Figure 4). Out of the 10 patients with HTN, 6 patients gave history of onset of hair loss before 35 years of age. However, statistical significance could not be determined on the age of onset because of the small number of the patients affected with HTN.
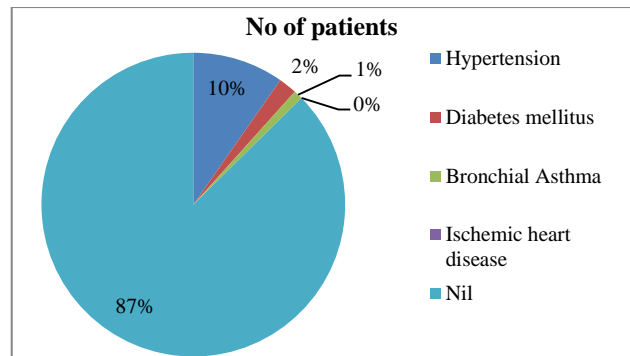


**Figure 4: Prevalence of systemic diseases among the patients.**

**DISCUSSION**

AGA is caused by genetic factors, but the mode of inheritance remains unclear. Osborn reported that AGA is an autosomal dominant disorder in men and an autosomal recessive disorder inwomen.[11] However, the strong concordance of AGA seen between fathers and sons is in consistent with a simple Mendelian trait, and a polygenic mode of inheritance.[3]

Studies have reported a gradual increase in AGA with age. Wang et al. estimated that prevalence rates in Caucasian populations are around 30% for men in their 30s, 40% for men in their 40s and 50% for men in their 50s.[12] In our study, 68% prevalence of AGA was seen in the age group 21-30 years. This is in agreement with Norwood's study on 1000 men with AGA, in which he reported the maximum prevalence of AGA in the age group 18-29 years.[13] This distribution may be due to certain genetic and environmental factors leading to early onset of AGA and also the fact that older patients did not feel the need to treat AGA.

Earlier studies reported that onset of AGA occurred along with puberty.[14] In a study on the Indian population Sehgal et al observed that in 50% of study population onset of hair loss was between the ages of 20 and 24.[15] Our study is in concordance with the previous study as most of our patients belonged to the age group 19-23 years and the median age of onset was 23 years. This early onset of AGA may be due to stressors like competitive structure of the society or due to lifestyle modifications which has to be further studied.

The duration of hair loss is a very important factor in deciding the treatment for the patient. Sehgal et al in his study observed that most of the patients presented within 1-4 years of onset of hair loss.[15] We saw a similar pattern in our study where 61.2% patients presented within 1-4 years duration of hair loss. This may be due to the fact that patients are concerned about hair loss only during the early years and gradually accepting their fate with the increasing age.

Smoking is associated with increased levels of serum androgens[8] and we therefore hypothesized an increased risk of AGA associated with smoking because of the reported association between serum testosterone and AGA.[9] Su et al in his study demonstrated a positive relation between AGA and smoking but he did not quantify the cigarettes. The mechanisms by which smoking causes hair loss may be multifactorial: it may be deleterious to the microvasculature of the dermal hair papilla, as a genotoxicants may damage DNA of the hair follicle or may cause an imbalance in the follicular protease or antiprotease system. Smoking may induce oxidative stress leading to release of pro-inflammatory cytokines that, in turn, results in follicular microinflammation and fibrosis or may yield a relative hypoestrogenic state by increased hydroxylation of estradiol and inhibition of aromatase.[6] Severi et al observed that smoking was not associated with onset of AGA.[7] In our study, 41 patients gave a history of smoking, we observed that most of our patients (62.9%) were less than years of age and smoked less than 20 cigarettes per day. Thus our findings did not show an association of onset of AGA with smoking status, nor did we find an association with either quantity of cigarettes smoked.

Severi et al observed that consumption of alcoholic beverages more than once a month on average was associated with a significantly increased risk of AGA.[7] In our study, 14.6% patients gave history of alcohol intake and among the 15 patients, 73.3% patients had onset of AGA before 35 years of age and hence the p value of <0.001 was statistically significant. Though there was an early age of onset with patients with history of alcohol consumption but most of the patients had early onset of AGA invariably of consuming alcohol or not. So a further study with a duration and quantity alcohol consumed in a larger study population needed to the association of alcohol with AGA.

AGA is considered a genetically predisposed condition but the mode of inheritance has not been well-described. In an Indian study of 150 subjects by Sehgal et al, positive family histories were found in 127 (85%) of subjects, paternal in 101 (67%), maternal in 7 (5%), both in 19 (13%) and 23 (15%) patients no family history could be elicited. In our study, 70 (68%) patients gave a positive family history among the first and second degree relatives, paternal in 44 (62.8%), maternal in 6 (8.6%), sibling in 13 (18.6%) and both paternal and sibling in 7 (10%). We also observed that 67 (69.8%) patients with a positive family history had onset of AGA below 35 years of age. This implies that the patients with a positive family history among the first or second degree relatives have a higher chance of developing early onset AGA. This suggests that patients with early onset AGA should receive early advice to prevent further deterioration. These results suggest that AGA expression is influenced by familial AGA prevalence and, particularly, that the

prevalence of paternal AGA has more effect on AGA expression than maternal AGA.

Previous studies have mentioned on the effect of BMI on the development of AGA but with inconsistent results.[16,17] Su et al in a study on policemen observed that higher BMI values were significantly associated with onset of AGA in younger policemen.[6] In our study, 28 (27.2%) patients had BMI more than 25 kg/m$^2$. Among the patients with early onset of AGA, 26 (27.1%) had BMI >25 kg/m$^2$ which was statistically significant but clinically was not significant as most of the patients with BMI <25 kg/m$^2$ also had onset of AGA before 35 years. Thus, concluding the fact that BMI does not play a role in the early onset of AGA.

The most common systemic disease associated with AGA was hypertension. Yeo et al. in his study reported that hypertension (14.8%), dyslipidemia and diabetes were commonly associated with AGA.[18] In our study, 10 (9.7%) were hypertensive, 2 (2%) were diabetic, 1 had bronchial asthma and none of them had history of dyslipidemia or ischemic heart disease.

Many studies have mentioned an association between hypertension (HTN) and AGA. The proposed explanations for the association of HTN and AGA are as follows: (a) the high level of circulating androgens which bind to mineralocorticoid receptors might be responsible for the observed difference in blood pressure, (b) hyperaldosteronism; itself may directly participate in the development of alopecia.[19] The severity, early onset, and longevity of AGA are related to subclinical vascular damage in adult untreated men with newly diagnosed essential hypertension. In addition, hypertensive patients with severe and early AGA onset seem to be exposed to an increased cardiovascular risk.[20] Blood pressure screening in patients with AGA is essential to facilitate the early diagnosis of unknown HTN and to initiate an appropriate treatment. El Esawy et al in her study observed that patients with AGA had higher blood pressure values when compared to controls, thus reporting AGA as an early marker in hypertension.[20]We observed that 10 (9.7%) patients were hypertensive and were on treatment and all others had blood pressure below 140/90 mmHg. Sixty percent of hypertensive patients had onset of AGA before 35 years of age but when compared onset of AGA before 35 years only 6.3% had hypertension. Our study was not in concordance with the previous studies.

### Limitations

Small size of the study population. A case control study in a community based survey would give a better idea about the effect of environmental risk factors and lifestyle on early onset of AGA. There is also need to quantify the amount and duration of alcohol intake and smoking to accurately comment on their effect on the early onset of hair loss.

## CONCLUSION

In our study, we noticed the prevalence of AGA in the younger age group thus signifying its importance in diagnosing it and the need for early treatment. There was no association between risk factors such as smoking, increased BMI and systemic diseases with early onset of AGA. A positive correlation was noted between family history and alcohol consumption with early onset of AGA. The early onset of AGA in the study population necessitates the need for identifying the potential risk factors in the aetiology of AGA. Identification and elimination of the potential risk factor could delay the progression of the AGA. Risk factors such as smoking, alcohol consumption and increased body mass index whether still considered to be a potential risk needs further evaluation in a large scale population with the help of case-control study by proper methods of quantification. Studies needed to be done based on the assessment of the dietary habits and also lifestyle of an individual that could play a role in the aetiology of AGA.

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