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Original Research Article

Androgenetic alopecia: evaluation of hormonal profile and its systemic implications

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ABSTRACT

Background: Androgenetic Alopecia is a hereditary androgen-dependent disorder characterized by a gradual conversion of terminal hair into miniaturized hair with typical bitemporal recession and balding vertex and is considered the most common type of baldness characterized by progressive hair loss. This study evaluated the hormonal profile in males with androgenetic alopecia. This study evaluated the hormonal profile of early androgenetic alopecia in males.
Methods: This prospective study included 84 patients attending the outpatient Department of Dermatology. Forty-four cases and 40 controls were included in the study. The study had 44 male patients presenting with complaints of grade ≥ 3 androgenetic alopecia in the age group 19-35 years, whereas 40 age and sex-matched patients attending Dermatology OPD for unrelated complaints with no history of hair loss or clinical examination suggestive of androgenetic alopecia were included in the control group. After a detailed history, and examination, testosterone, LH, FSH, Prolactin, and SHBG were estimated.
Results: The mean age of onset was found to be 24.29±3.28 years. Positive family history was seen in 65.90% of patients. The mean testosterone, LH, FSH, prolactin, SHBG and free androgen index in cases versus controls was 6.44±2.58 versus 3.32±1.53 ng/ml, 8.01±2.64 IU/l versus 3.01±1.16 IU/l, 3.82±1.33 IU/l versus 5.07±1.27 IU/l, 15.50±5.11 ng/ml versus 9.84±3.91 ng/ml, 12.72±2.63 nmol/l versus 29.18±4.90 nmol/l and 51.03±21.78 versus 11.40±4.66 respectively. LH/FSH ratio was 2.17±0.54 versus 0.63±0.27. These parameters had p values <0.05 and were statistically significant.
Conclusions: Our study concluded that serum testosterone, prolactin, LH, LH/FSH, and FAI are increased whereas serum FSH and SHBG are decreased in cases of androgenetic alopecia compared to controls.

Keywords: Androgenetic alopecia, Hormonal profile, PCOD

INTRODUCTION

Hair is an appendage of skin with no vital function in humans. But they still have a role in social and sexual communication and also affect the psychological functions of a human being. Hair is the keratinized product of hair follicles and is of the following types: prenatal i.e., lanugo hair, and postnatal hair i.e., vellus and terminal hair.

The term androgenetic alopecia was coined by dermatologist from New York, Norman Orentreich in the year 1960.[1] It is a hereditary androgen-dependent disorder characterized by a gradual conversion of terminal hair into miniaturized hair, with typical bitemporal recession and balding vertex and is considered to be the most common type of baldness characterized by progressive hair loss.[2-4] Androgenetic alopecia (AGA) is a multifactorial disorder.

It is caused by interactions between several genes and environmental factors. It is thought to have polygenic inheritance based on the finding that baldness risk increases with the number of affected family members, and the high frequency of baldness is seen in the fathers of balding men. Locus at chromosome 20p11.22, harboring risk alleles has a 7-fold increased odd of androgenic alopecia.[5] Genetic variability at the AR/EDA2R locus is thought to enhance the effect of androgens by increasing the number of androgen receptors in affected scalp tissue.

Based on Norwood Hamilton classification male androgenetic alopecia is graded into 7 types (Table 1). But Norwood Hamilton classification has its own drawbacks and its reproducibility is unsatisfactory.[6] Khumalo et al to simplify it further gave a few modifications and called it as adapted Norwood Hamilton classification.[7] Additional designations of the original one were removed and replaced by A (anterior) and V (vertex) to indicate the main site of hair loss. The newest classification is known as basic and specific classification (BASP) given by Lee et al.[8] The basic types define the shape of the anterior hairline using the letters L, M, C, and U which correspond to how the hairline looks when viewed from above. L, M, C, and U designate the anterior hairline as linear, resembling the letter M, as letter C and horseshoe shape respectively. The specific type represents the general thinning of the scalp. These types are divided based on location, whether it is frontal (F) or vertex (V) and further divided into three subtypes i.e., V1, V2, V3 each. Reproducibility of Norwood, Norwood-Hamilton, adapted Norwood-Hamilton, BASP is 98%, 39.9-49.9%, 93%, and 83% respectively.[8] The most practical classification is the Norwood-Hamilton classification.[6]

On the scalp, there is a hierarchy of sensitivity i.e. the most anterior of the hairs at the temples are sensitive and so are lost first, whereas those on the occipital area are resistant to the effects of androgens. Subsequent hair miniaturizing occurs in a highly ordered fashion from the frontotemporal area or from the vertex to produce patterned baldness. The major circulating androgen is testosterone, but in most body sites except axillae and pubic region, the effect on hair growth is mediated by dihydrotestosterone. The tissue effects of androgens are mediated through binding to the intracellular nuclear hormone receptor. Androgens are the main regulator of human hair growth and the androgens promote the gradual transformation of large terminal scalp follicles to tiny vellus ones causing androgenetic alopecia.

The body androgens stimulate hair growth in the axilla and pubic area of both sexes and hair on the face, upper pubic diamond, and chest in men.[9-12] They have the opposite effect on specific scalp areas, often in the same individual, which ultimately ends in balding.[13] Androstenedione, an endogenous weak androgenic steroid in nature is a major metabolite in the hair roots of patients with androgenic alopecia.[14] Also, elevated levels of E2 (estrogen) are found due to increased peripheral conversion of androgen to estrogen. Luteinizing hormone (LH) also contributes to the process of androgenetic alopecia. Increased LH levels lead to increased testosterone, ultimately leading to male pattern baldness. At the same time, follicle-stimulating hormone (FSH) stimulates Sertoli cells to release testosterone. Prolactin a hormone released from the posterior pituitary also plays a role by acting via supra renal release of androgens and also by modulating the activity of 5α-reductase.

Recently with further introspection into the complex multifactorial pathogenesis of androgenetic alopecia, the influence at the gene level, and the altered hormonal level, similarities with female PCOS have been proposed. The hormone profile of women with polycystic ovarian syndrome (PCOS) includes elevated levels of testosterone, dehydroepiandrosterone sulfate (DHEAS), and luteinizing hormone (LH) with altered LH/FSH ratio, hyperinsulinemia due to increased insulin resistance (IR), and decreased sex hormone binding globulin (SHBG).[15-17]

PCOS in females initially is diagnosed based on features of hyperandrogenaemia or infertility but later it can lead the patient into serious complications like diabetes mellitus, heart diseases, altered lipid profile, hypertension, and ultimately stroke. These complications can be detrimental to the life of the patient and considering the autosomal dominant nature of PCOS in females at the genetic level, it has been suggested that the same can be expressed in males as a premature AGA.[17] Therefore, the patients of AGA may develop complications similar to those of PCOS females. Also, it is proposed that androgenetic alopecia of Norwood Hamilton grade 3 has been found to be a marker for insulin resistance.[18]

Therefore, the aim of our study was to study the hormonal profile of men with early androgenetic alopecia.

METHODS

This prospective study was conducted on a total of 84 patients attending the outpatient Department of Dermatology, Jawaharlal Nehru Medical College, AMU between November 2018 to October 2020 after receiving ethical clearance from the institutional ethics committee, D. No 947/FM.

The sample size was calculated to be 31 based on OPD annual attendance of approximately 1,20,000 and hypothesized frequency of 2% and a confidence interval of 95%. We took 44 cases of AGA and 40 controls in our study. The patients were included in the study as per the following criteria.

Inclusion criteria

Male patients with grade 3 or more of androgenic alopecia and in the age group of 19-35 years were included in the study. This age group was taken because early-onset androgenic alopecia refers to pattern hair loss before 35

years of age. Also, this age group can be influenced by certain primordial prevention and lifestyle modifications.

Exclusion criteria

Patients with established endocrine disorder, diabetes mellitus, cardiovascular disease, who took any steroids or hormonal treatment for hair loss in the past 6 months, chronic telogen effluvium, diffuse alopecia areata, refusing consent, and patients with Norwood Hamilton Grade 1 and 2 androgenic alopecia were excluded from the study. Patients who were on anabolic steroids were also excluded. The exclusion criteria were designed keeping in mind the various factors that can affect the outcome of the study. Patients taking steroids and hormonal treatment for hair loss were excluded because of their effect on the baseline hormonal levels we were going to investigate. Patients suffering from Norwood Hamilton grades 1 and 2 were excluded from the study because these are the early initial stages.

A detailed history of each patient was taken and recorded in the proforma designed for study regarding demographic data of the patients (name, age, sex, occupation, education, marital status, residence, phone number), chief complaint, mode of onset, progression, duration, areas of hair loss, recession of hair line, complain of itching and trichodynia, dietary history, history of drug intake (anabolic steroids), maternal and paternal history and lifestyle habits like smoking.

A general physical and systemic examination was performed on each patient. Cutaneous examination to look for signs of hyperandrogenism like acne and seborrhoea was done. Examination of the scalp was done to look for hair texture, hair thickness (to look for percentage of vellus and terminal hairs), recession of anterior hairline, and presence of seborrheic dermatitis. Clinical tests like hair pull tests and trichoscopy were done to rule out another diagnosis.

From each patient, 5 ml of blood sample was drawn after fasting overnight/ fasting for at least 8 hours. Centrifugation was done in the Remi R-8C centrifugation machine at 2000 rpm for 10 minutes which separated the blood sample into RBC sediment at the bottom and supernatant above which is serum (Figure 1). The serum of patients was then transferred via pipette into screw tight plastic container and kept in a refrigerator at -20° Celsius till estimation was done. Total testosterone, LH, FSH, prolactin, and SHBG were measured from blood samples drawn. Testosterone, LH, FSH, and prolactin were analyzed by Beckman Coulter Access 2 Immunoassay Analyzer and SHBG levels were estimated by ELISA kit (Figure 2). The reference values of LH, FSH, prolactin, total testosterone and SHBG are as follows: LH: 0.5-10 IU/l, FSH: 1.3-11.5 IU/l, PRL: 1.0-18 ng/ml, testosterone: 3.0-12.0 ng/ml, SHBG: 20.0-70.0 nmol/l. Free androgen index (FAI) was calculated by using the formula testosteronex100/SHBG.

All the data was compiled and tabulated in IBM statistical package for the social sciences 25 (SPSS), and the data was analyzed using various tests. Unpaired 't' test was used for the comparison of the parametric variables with continuous data between the two groups. Chi-square test, and Fischer exact test were used for non-parametric variables, and p value <0.05 was considered significant.

RESULTS

In this study 44 cases of androgenetic alopecia and 40 male patients with unrelated disorders were taken. The mean age of cases was 25.25 years and of controls was 25.97 years and the p value was >0.05. Therefore, both the groups were matched for age. Most of the cases of androgenetic alopecia belonged to urban area i.e., 65.90% (n=24). There was no statistical difference between cases and controls (Table 2).

The mean age of onset of androgenetic alopecia was found to be 24.29±3.28 years. Maximum number of patients presenting with androgenetic alopecia were in the age group of 23-26 years (n=18). Fifty-four percent (n=24) patients with androgenetic alopecia showed gradual progression of the disease.

The most common grade of androgenetic alopecia seen in cases was grade 4 accounting for 47.70% (n=21) followed by grade 3 which was 31.80% (n=14) (Figure 3). In our study the duration of the disease ranged from 6 months to >5 years since it is a chronic and gradually progressing disease. In our study, we found that the mean duration of the disease was 30.93 months. Features of hyperandrogenism i.e., acne and seborrhea were seen in 72.70% (n=32) patients of androgenetic alopecia. A positive family history of androgenetic alopecia was seen in 65.90% (n=29) of patients. 41.40% of positive family history patients belonged to the paternal side, 27.50% to the maternal side and 13.90% had both paternal and maternal history. In our study, we found that the initial site of recession most commonly was temporal hairline recession which was seen in 65.9% (n=29) of patients followed by frontal hairline recession which was seen in 20.4% (n=9) patients. Only 13.70% (n=6) of patients presented with the initial site of hair loss from the vertex area. Also, the progression of hair loss in these patients was gradual i.e. occurring over the period of 2-5 years in 54.5% of patients (n=24). Mean testosterone levels among cases (n=44) were 6.44 IU/l and among controls (n=40) were 3.32 IU/l. Similarly, serum LH (8.01 IU/l) and serum FSH (3.82 IU/l) were significantly increased and decreased respectively in comparison to controls and these values were statistically significant with p value<0.05.

Also, we observed that LH/FSH ratio in our patients was >2 i.e. 2.17±0.54 and this was statistically significantly higher with a p value less than 0.05 than levels seen in the control group. We found Prolactin levels to be significantly increased i.e. mean serum prolactin levels were 15.50±5.11 in patients of AGA and when compared

to levels seen in controls it was statistically significant. The mean value of SHBG as found to be 12.72±2.63 and was found to be significantly reduced in comparison to controls with p value being <0.05. Free androgen index (FAI) is calculated by (testosterone/SHBG) multiplied into 100 and the mean value of FAI was found to be significantly raised i.e. 51.03 in patients of AGA and when it was compared with controls, the p value was found to be <0.05. The summary of the above is given in Tables 2 and 3 and the hormonal profile of both groups is given in Table 4.

Table 1: Norwood Hamilton grading.

Grade	Description
Type 1	Absence of bilateral recessions along the anterior border of the hairline in the fronto-parietal region
Type 2	Anterior border of hairline has triangular area of recession in the frontoparietal region
2a	The hairline is anterior to the coronal plane 2 cm anterior to the external auditory meatus
Type 3	Borderline cases which also includes lateral asymmetry in denudation, unusual types of sparseness and thinning of hair or scars
3a	The hairline has receded back to a point between the limit of type IIA and the level of the external auditory meatus
Type 4	Deep frontotemporal recessions, usually symmetrical
4a	Hair is sparse as a broad band along the entire anterior border of hairline
Type 5	Extensive frontoparietal and frontal recessions with a sparseness/absence of hair on the crown
Type 6	The tonsural region of alopecia remains separated from more anteriorly located area of denudation
6a	Peninsula or island of mid frontal hair is sparse or lost
Type 7	The horseshoe-shaped area of sparse hair/denudation is unbroken by any well haired, laterally directed bridge of scalp

Table 1: Variables among cases and controls.

Variables	Cases	Controls	P value
Mean age (years)	25.25±3.49	25.97±4.33	0.73
Residence (%)			
Urban	65.90 (n=29)	60 (n=24)	0.57
Rural	34.10 (n=15)	40 (n=16)	

Table 2: Baseline characteristics of participants with AGA.

S. no.	Characteristics of patients	Values
1	Mean age of onset of androgenetic alopecia	24.29±3.28 years
2	Duration of disease	30.93±14.62 months
3	Age of onset (%)	
	19-22	31.80 (n=14)
	23-26	40.90 (n=18)
	27-30	22.70 (n=10)
	>30	4.60 (n=2)
4	Progression of androgenetic alopecia	
	Gradual (2-5 years)	54.50 (n=24)
	Moderate (1-2 years)	31.80 (n=14)
	Rapid (<1 year)	13.70 (n=6)
5	Signs of hyperandrogenemia (%)	
	Present	72.70 (n=32)
	Absent	27.30 (n=12)
6	Family history (%)	
	Present	65.90 (n=29)
	Absent	34.10 (n=15)

Table 3: Comparisons of various hormones, ratio and index between case and control group.

Hormones	Mean value in cases	Mean value in controls	P value
Testosterone (ng/ml)	6.44±2.58	3.32±1.53	<0.05

Continued.

Hormones	Mean value in cases	Mean value in controls	P value
LH (IU/l)	8.01±2.64	3.01±1.16	<0.05
FSH (IU/l)	3.82±1.33	5.07±1.27	<0.05
Prolactin (ng/ml)	15.50±5.11	9.84±3.91	<0.05
SHBG (nmol/l)	12.72±2.63	29.18±4.90	<0.0001
FAI	51.03±21.78	11.40±4.66	<0.05

Figure 1: REMI centrifuge.

Figure 2: Beckman Coulter access 2 immunoassay analyser.

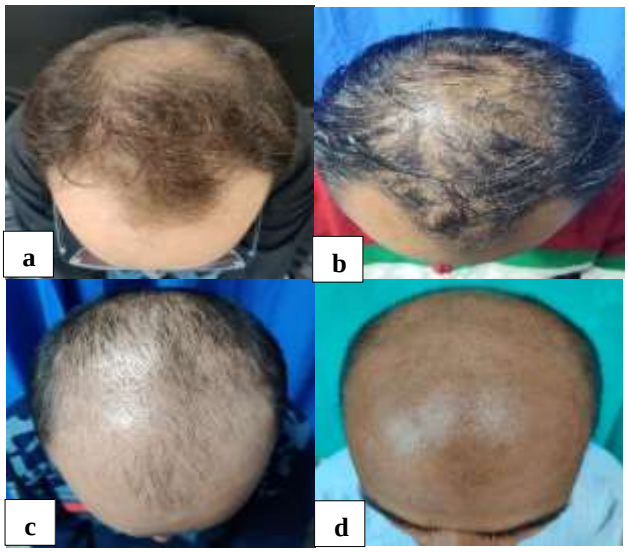

Figure 3: (a) Grade 3 AGA, (b) grade 4 AGA, (c) grade 5 AGA, and (d) grade 6 AGA.

DISCUSSION

Hairs by virtue of their natural qualities and their capacity to be modelled artistically contribute in a significant manner in the perception of self-image. Androgenetic alopecia is the most common type of baldness which results in progressive hair loss and is one of the most common reasons for dermatological consultation. The pathogenesis of androgenetic alopecia is multifactorial and is an interplay of genes, hormones, and environmental factors. Among the 84 patients included in our study, 44 patients were of androgenetic alopecia. The age of patients ranged from 19-35 years with a mean age of 25.25±3.49 years. Maximum proportions of patients were in the age group 25-30 years (47.70%, n=21) followed by the 19-24 years age group (38.60%, n=17). It was observed that most of the patients were of mid 20's age group and our findings were consistent with findings in other studies. The mean age of AGA was 24.7 years in study by Sanke et al.[19] Like the Sanke study, Schimdt et al reported the mean age as 24.31 years.[20] However, this differs from the mean age of AGA reported by Tahir et al who found it to be 29.01±8.62 years.[21] This may be because the subjects ranged from age of 18 years to 55 years whereas we took the patients in the age group of 19-35 years i.e., early AGA. These findings can also be explained by the psychological and social impact of hair loss in the younger population and their immediate consultation for their problem.

In our study, the mean age of onset of androgenetic alopecia was found to be 24.29±3.28 years. This is in contrast to the findings of Salman et al who reported the age of onset in the 3rd-4th decade of life.[22] This difference in mean age of onset could be possibly explained because in this study age group taken was till >70 years of age. Also, in our study, the maximum proportion of patients were in the age group 23-26 years i.e., 40.9% (n=18). The findings further correlate with the earlier onset of androgenetic alopecia and its chronicity gradually progressively increasing over the span of life. In our study, most of the patients who visited the dermatology OPD were from urban backgrounds accounting for 65.90% (n=29) of the cases. These findings were consistent with 61% of urban patients observed by Gupta et al.[23] This can be possibly explained by the easy availability of health facilities in urban areas and also the greater social and psychological impact of hair loss in urban males.

Out of 44 patients of androgenetic alopecia grade 3 of the disease was seen in 31.80% (n=14), grade 4 in 47.70% (n=21), and grade 5 in 13.70% (n=6) of patients. We excluded grade 1 and 2 androgenetic alopecia because of

exclusion criteria as the hormonal profile is usually normal in the initial stages of androgenetic alopecia. Therefore, commenting on the commonest type of AGA based on Norwood Hamilton classification is difficult. Features of hyperandrogenism such as acne and seborrhoea were seen in 72.70% (n=32) of patients with androgenetic alopecia. Although, there is no data on the prevalence of acne in patients with androgenetic alopecia. Hamilton, et al. showed prepubertal castration prevents seborrhoea and androgenetic alopecia.[24] Also, these findings can be correlated by the supporting evidence of shorter poly Q polymorphism in AR gene which causes increased prevalence of acne and androgenetic alopecia in the same patient. Family history was found to be positive in 65.90% (n=29) of patients with androgenetic alopecia. Similar results were seen by Salman et al who observed that 78.28% of patients showed a positive family history.[22]

Mean testosterone levels among cases (n=44) were 6.44 IU/l and among controls (n=40) was 3.32 IU/l and this difference was statistically significant with p value <0.05. Similarly, Sanke et al reported higher levels of mean testosterone.[19] Their mean testosterone level was 24.61. The test used by them had different normal value ranges which is why commenting of similar values is difficult, although they also reported statistically significant differences in the mean of testosterone among the case and control groups. Also, Schimdt et al, Narad et al and Yildiz et al findings are consistent with our findings.[25-27] Like the Duskova study, Starka et al showed subnormal levels of testosterone in their studies.[17,28] AGA in patients with normal testosterone can be attributed to increased androgen sensitivity. Increased levels of testosterone lead to increased dihydrotestosterone levels by 5α-reductase and then finally acting on the dermal papillae and causing hair loss.

Similarly, serum LH (8.01 IU/l) and serum FSH (3.82 IU/l) were significantly increased and decreased respectively in comparison to controls and these values were statistically significant with p value <0.05. Raised LH levels were also seen by Cohen et al similar to our study.[29] However, Narad et al did not show a significant difference in the levels of LH and FSH in patients with androgenetic alopecia.[26] LH influences testosterone and other metabolites thereby contributing to the pathogenesis of androgenetic alopecia. Increased LH leads to increased testosterone levels and thus contributes to increased male pattern baldness. In addition, LH also stimulates the adrenal gland to produce androstenedione, a weaker androgen. Increased levels of LH thus suggest that the hypophyseal-adrenal axis contributes to the pathogenesis of premature AGA. FSH-reduced levels were also seen by Starka et al in men with AGA.[28] The role of FSH in androgenetic alopecia is explained by the induction of Sertoli cells to produce testosterone.

Also, we observed that the LH/FSH ratio in our patients was >2 i.e., 2.17±0.54 (p<0.05). This finding is supported by a similar finding reported by Duskova et al, Sanke, et al, Narad et al.[17,19,26] They too saw a similar profile of LH/FSH ratio i.e., >2. The significance of this LH/FSH ratio is that a similar raised level of this ratio is seen in patients of polycystic ovarian syndrome (PCOS) and this finding thus supports one of the parameters in our study and helps to support the proposition of AGA being phenotypic equivalent to PCOS in females.

We found Prolactin levels to be significantly increased i.e., mean serum prolactin levels were 15.50±5.11 in patients of AGA and when compared to levels seen in controls had p value <0.05. Prolactin plays its role in androgenetic alopecia by stimulating suprarenal androgen release and it also modulates 5α-reductase activity. The mean value of SHBG was found to be 12.72±2.63 and was found to be significantly reduced in comparison to controls, p value <0.05. Narad et al observed similar findings of decreased SHBG levels in patients with androgenetic alopecia.[26] Similar findings were observed in study by Sanke et al, Duskova et al also reported relatively decreased SHBG levels in AGA.[17,19] SHBG is the protein that binds testosterone and when its levels are reduced there is a relatively increased concentration of free testosterone in the blood thereby leading to a state of hyperandrogenaemia which in turn acts as a stimulator in the process of androgenetic alopecia. Also, lower levels of SHBG are also associated with impaired glucose tolerance.[30]

FAI is calculated by (testosterone/SHBG)×100 and the mean value of FAI was found to be significantly raised i.e., 51.03±21.78 in patients of AGA and when it was compared with controls, and the p-value was found to be <0.05. Similar to our study Sanke et al also observed raised FAI in patients of AGA and thus supports our findings.[19] FAI is homologous to free testosterone. Free testosterone causes the gradual transformation of miniaturized follicles in a predisposed individual. It is a form that binds to tissue receptors.

The hormonal parameters seen in PCOS are increased testosterone levels, raised LH levels, increased prolactin levels, raised LH/FSH ratio >2, and normal to low FSH values. In the males with androgenetic alopecia, it was seen that in comparison with controls, the mean testosterone levels, mean LH levels, mean prolactin levels were significantly raised. The FSH was significantly reduced on comparison and the LH/FSH ratio was >2. Similar types of findings were seen by Sanke et al and Narad et al.[19,26] They too observed that the alteration in hormonal parameters of patients with AGA was quite similar to that seen in PCOS females. This finding thus helps in the proposition of AGA being phenotypically equivalent to PCOS in females.

CONCLUSION

Androgenetic alopecia in males is one of the commonest hair loss dermatological conditions. Androgen hormones i.e., testosterone and other hormones like FSH, LH, and prolactin, and factors affecting the concentration of free

testosterone in the body like SHBG were found to be significantly altered in our patients of AGA. Our study concluded that serum testosterone, serum prolactin, LH, LH/FSH ratio, and FAI are increased whereas serum FSH, and SHBG are decreased in cases of androgenetic alopecia compared to controls. This presentation of hormonal parameters in the patients of AGA has not only a local effect on the scalp but can also have wider implications for the homeostasis of the body. Researchers have proposed the male phenotypic equivalent of PCOS as AGA. These findings were based on the similarities of mode of inheritance, genetic involvement, and almost similar hormonal parameters. PCOS is one of the most common causes of infertility in females and it not only interferes with reproductive function but also is notorious for causing alteration in insulin and lipid homeostasis leading to complications like type 2 diabetes mellitus, hypertension, obesity, atherosclerosis, and non-classic cardiovascular risk factors, including mood disorders, such as depression and anxiety. It can be proposed that males with androgenetic alopecia are also at risk of various metabolic abnormalities and insulin resistance. The patients of AGA in the future may suffer complications like diabetes mellitus, atherosclerosis, coronary artery disease, hypertension, obesity, and mood disorders due to psycho-social impact.

Therefore, we would like to conclude that AGA should not be considered a meagre scalp disorder of hair loss that worries the patient. But it is imperative to consider it as a disorder with systemic implications and so these patients should be addressed and counseled to adopt various lifestyle modifications so as to act at a primordial level of prevention, and to prevent these above modifiable dreadful complications in the future.

More studies in the future are required to interlink the genetic and hormonal similarities between androgenetic alopecia in males and PCOS in females and to ascertain this phenotypic equivalence between the two disorders for their integrated and comprehensive management.

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