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Original Research Article

Evaluating Testosterone and Dehydroepiandrosterone Sulfate (DHEA-S) as Biomarkers for Male Androgenetic Alopecia

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Abstract: ***Background*:** Androgenetic Alopecia (AGA) 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. ***Aims and objectives*:** This study evaluated the testosterone and Dehydroepiandrosterone Sulfate (DHEA-S) in males with androgenetic alopecia. ***Methods*:** This case control study was carried out in the department of Dermatology & Venereology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka during the period of January 2024 to December 2024. A total of 70 patients with androgenetic alopecia and without history of androgenetic alopecia were included in the study. The study had 35 male patients presenting with patterned hair loss were recruited as AGA group, whereas 35 had age and sex-matched with no evidence of hair loss were recruited as control group. Serum FSH, LH, prolactin, testosterone, SHBG, DHEA-S and free androgen index were estimated. ***Results*:** In this study, the mean age was 25.7±2.7 years in AGA group and 25.3±2.5 years in control group. The mean BMI was 26.6±2.5 kg/m^2 in AGA group and 26.0±2.7 kg/m^2 in control group. The difference were not statistically significant (p>0.05) between AGA and control groups. Mean serum LH (7.6±1.5 vs. 4.7±1.4 µIU/mL; *P* =0.001), serum prolactin (14.0±3.8 vs. 10.8±4.4 ng/ml; *P* =0.001), serum testosterone (6.5±1.9 vs. 5.2±2.0 ng/ml; *P* =0.005), DHEA-S (297.0±11.7 vs. 260.9±14.7 µg/dl) and free androgen index (72.5±30.8 vs. 49.2±27.1; *P* =0.001) are significantly increased in AGA group than control groups. The mean serum FSH (3.9±1.5 vs. 5.5±2.5 µIU/mL; *P* =0.002) and SHBG (23.4±6.2 vs. 29.2±9.6 nmol/l; *P* =0.004) are significantly decreased in AGA group than control groups. ***Conclusion***: This study was concluded that serum LH, prolactin, serum testosterone, DHEA-S and free androgen index are increased whereas, serum FSH and SHBG are decreased in case of AGA compared to controls.

Keywords: Male Androgenetic Alopecia, Testosterone, Dehydroepiandrosterone Sulfate (DHEA-S).

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 [1].

The term androgenetic alopecia was coined by dermatologist from New York, Norman Orentreich in the year 1960 [2]. 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 [3, 4].

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Androgenetic alopecia (AGA) – also termed male-pattern alopecia, common baldness, and male-pattern hair loss – is the most common type of alopecia occurring after puberty [5]. It is typically manifested as progressive hair thinning and shortening in affected areas. Although regarded as a minor dermatological condition, it affects self-image and is a great cause of anxiety and depression in some patients, particularly younger ones [6, 7].

Male hypogonadism is defined as a gonadal dysfunction resulting in low total testosterone (TT) serum levels [8]. Dehydroepiandrosterone sulfate (DHEA-S) is a molecule with a weak androgenic activity mainly secreted by the adrenal gland and, by a lesser extent, by the gonads, whose role in the pathogenesis of hypogonadism remains debated. Meta-analytic data provide evidence for increased DHEA-S levels and a worse glycolipid profile in male patients with early-onset (<35 years) androgenic alopecia (AGA), defined by a grade of alopecia higher than III according to the Hamilton–Norwood scale, compared to controls [9].

Subnormal levels of testosterone among their men with AGA. Increased levels of testosterone leads to increased dihydrotestosterone levels by 5α reductase and thus increased action of these androgens on the dermal papillae cells of hair follicles in these patients. AGA in patients with normal testosterone levels could be attributed to increased androgen receptors or increased androgen sensitivity [10, 11].

The higher levels of DHEA-S found in men with early-onset AGA compared to controls suggests that this hormone might play a role in the development of AGA. In fact, in the hair follicle, DHEA-S is converted into molecules having higher androgen activity (testosterone, dihydrotestosterone) [12]. Moreover, these higher DHEA-S levels may be involved in the pathogenesis of the observed slightly worse metabolic profile and might represent a biochemical feature of the male PCOS-equivalent. This study will ultimately help to solve this physical and social problem more meticulously. The purpose of this study was to evaluate the testosterone and Dehydroepiandrosterone Sulfate (DHEA-S) in males with androgenetic alopecia.

MATERIALS AND METHODS

This case control study was carried out in the department of Dermatology & Venereology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka during the period of January 2024 to December 2024. A total of 70 patients with androgenetic alopecia and without history of androgenetic alopecia were included in the study. The study had 35 male patients presenting with patterned hair loss were recruited as AGA group, whereas 35 had age and sex-matched with no evidence of hair loss were recruited as control group. Men who had any established endocrine disorder, diabetes mellitus, or cardiovascular disease and those who took any oral medication or hormonal treatment for hair loss were excluded from the study.

A detailed history of each patient was taken and recorded in the proforma designed for study regarding demographic data of the patients (name, age, occupation, residence, phone number). The BMI was calculated applying the formula: weight (kg)/[height (m) × height (m)].

Detailed anamneses were recorded for each individual. All participants were assessed by the same physician. Serum FSH, LH, prolactin, Total testosterone, SHBG and DHEA-S were measured from blood samples drawn. Free androgen index (FAI) was calculated by using the formula testosterone x 100/SHBG.

All the relevant collected data were compiled on a master chart first and then statistical analysis of the results were obtained by using window based computer software devised with Statistical Packages for Social Sciences (SPSS-23) (SPSS Inc, Chicago, IL, USA). Unpaired t-test was used for quantitative *variables and Chi square test used for qualitative variables.* Significant value of 'p' was decided to be at a level of 0.05 in two tailed tests.

RESULTS

In this study, mean age was found 25.7±2.7 years in AGA group and 25.3±2.5 years in control group. The difference regarding age, occupation, residence and BMI were not statistically significant (p>0.05) between AGA and control groups (Table-1). In AGA patients, mean age at onset of androgenetic alopecia was found 23.4±2.5 years and duration of disease was 27.1±8.3 months. The most common androgenetic alopecia showed gradual progression of the disease in 23(65.7%) patients, 26(74.3%) was found signs of hyperandrogenemia, 25(41.4%) was temporal hairline recession, 15(42.9%) was positive family history of androgenetic alopecia and 14(40.0%) was grade 4 of androgenetic alopecia (Table-2). Mean serum LH (7.6±1.5 vs. 4.7±1.4 µIU/mL; *P* =0.001), serum prolactin (14.0±3.8 vs. 10.8±4.4 ng/ml; *P* =0.001), serum testosterone (6.5±1.9 vs. 5.2±2.0 ng/ml; *P* =0.005), DHEA-S (297.0±11.7 vs. 260.9±14.7 µg/dl) and free androgen index (72.5±30.8 vs. 49.2±27.1; *P* =0.001) are significantly increased in AGA group than control groups. The mean serum FSH (3.9±1.5 vs. 5.5±2.5 µIU/mL; *P* =0.002) and SHBG (23.4±6.2 vs. 29.2±9.6 nmol/l; *P* =0.004) are significantly decreased in AGA group than control groups (Table-3).

Table 1: Demographic characteristics of the study participants

	AGA (n=35)		Control (n=35)		P value
	n	%	n	%	
Age (years)					
21-25	18	51.4	17	48.6	0.616
26-30	17	48.6	18	51.4	
Mean±SD	25.7	±2.7	25.3	±2.5	
Occupation					
Businessman	8	22.9	10	28.6	0.584
Service	27	77.1	25	71.4	
Residence					
Rural	7	20.0	10	28.6	0.403
Urban	28	80.0	25	71.4	
BMI (kg/m^2)					
18.5-24.9	9	25.7	11	31.4	0.350
25.0-29.9	26	74.3	24	68.6	
Mean±SD	26.6	±2.5	26.0	±2.7	

P value reached from unpaired t-test and chi square test

Table 2: AGA parameter of the study participants

Variables	Mean	±SD
Age at onset of androgenetic alopecia (years)	23.4	±2.5
Duration of disease (months)	27.1	±8.3
Progression of androgenetic alopecia		
Gradual	23	65.7
Moderate	8	22.9
Rapid	4	11.4
Signs of hyperandrogenemia		
Present	26	74.3
Absent	9	25.7
Initial site of hair loss		
Frontal	6	17.1
Temporal	25	41.4
Vertex	4	11.4
Family history of AGA		
Present	15	42.9
Absent	20	57.1
Grade of AGA		
3	6	17.1
4	14	40.0
5	12	34.3
6	3	8.6

Table 3: Hormonal parameters of the study participants

Variables	AGA (n=35)		Control (n=35)		P value
	Mean	±SD	Mean	±SD	
Serum FSH (µIU/mL)	3.9	±1.5	5.5	±2.5	0.002
Serum LH (µIU/mL)	7.6	±1.5	4.7	±1.4	0.001
Serum prolactin (ng/ml)	14.0	±3.8	10.8	±4.4	0.001
Serum testosterone (ng/ml)	6.5	±1.9	5.2	±2.0	0.005
SHBG (nmol/l)	23.4	±6.2	29.2	±9.6	0.004
DHEA-S (µg/dl)	297.0	±11.7	260.9	±14.7	0.001
Free androgen index	72.5	±30.8	49.2	±27.1	0.001

P value reached from unpaired t-test

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 [1]. Therefore, the purpose of the study was to evaluate the testosterone and Dehydroepiandrosterone Sulfate (DHEA-S) in males with androgenetic alopecia.

In this study observed that mean age was found 25.7±2.7 years in AGA group and 25.3±2.5 years in control group. The difference was not statistically significant (p>0.05) between AGA and control groups. In a study done by Pathak *et al*., [1] reported that the mean age was 25.55±3.49 years in AGA group and 25.97±4.33 years in controls group. The difference was not statistically significant between two groups. Cannarella *et al*., [13] obtained that the mean age was 24.33±3.4 years in AGA group and 23.3±2.7 years in controls group. The difference was not statistically significant between two groups. Sanke *et al*., [14] also showed that the mean (SD) age was 24.7 (2.8) years in AGA group and 24.2 (2.6) years in control group. The difference was not statistically significant between two groups.

In the present study the mean BMI was found 26.6±2.5 kg/m^2 in AGA group and 26.0±2.7 kg/m^2 in control group. The difference was not statistically significant (p>0.05) between AGA and control groups. Cannarella *et al*., [13] revealed that patients with early-onset AGA had higher mean (±SD) BMI (25.5±3.8 vs. 23.7±3.0 kg/m^2; $P < 0.05$) compared to controls. Sanke *et al*., [14] also observed that the mean (±SD) BMI was high in cases compared with controls (22.9±3.6 vs 21.1±2.4; $P = 0.01$).

This study observed that in AGA patients mean age at onset of androgenetic alopecia was a found 23.4±2.5 year and duration of disease was 27.1±8.3 months. The most common androgenetic alopecia showed gradual progression of the disease in 23(65.7%) patients, 26(74.3%) was found signs of hyperandrogenemia, 25(41.4%) was temporal hairline recession, 15(42.9%) was positive family history of androgenetic alopecia and 14(40.0%) was grade 4 of androgenetic alopecia. In a study conducted by Pathak *et al*., [1] demonstrated that the mean age of onset was found to be 24.29±3.28 years. Mean duration of the disease was 30.93 months. Fifty-four percent (n=24) patients with androgenetic alopecia showed gradual progression of the disease. Positive family history was seen in 65.90% of patients. 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%. Another study also done by Sanke *et al*., [14] described the mean age of onset was 22.17±3.66 years. Mean duration of the disease was 24.75±20.19 months. Fifty-four percent (n=31) patients with androgenetic alopecia showed rapid progression of the disease. Positive family history was seen in 33% of patients. The most common grade of androgenetic alopecia seen in cases was grade 5 accounting for 35% (n=20) followed by grade 4 which was 33% (n=19).

In this study observed that mean serum LH (7.6±1.5 vs. 4.7±1.4 µIU/mL; P =0.001), serum prolactin (14.0±3.8 vs. 10.8±4.4 ng/ml; P =0.001), serum testosterone (6.5±1.9 vs. 5.2±2.0 ng/ml; P =0.005), DHEA-S (297.0±11.7 vs. 260.9±14.7 µg/dl) and free androgen index (72.5±30.8 vs. 49.2±27.1; P =0.001) are significantly increased in AGA group than control groups. The mean serum FSH (3.9±1.5 vs. 5.5±2.5 µIU/mL; P =0.002) and SHBG (23.4±6.2 vs. 29.2±9.6 nmol/l; P =0.004) are significantly decreased in AGA group than control groups. In a study done by Pathak *et al*., [1] reported that 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. 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. Zhang *et al*., [5] revealed that there were significant differences in the serum androgen levels between the alopecia group and the normal controls for all measured levels except for serum testosterone. SHBG was similar between the AGA group and normal controls. This indicated that the severity of AGA was independent of sexual hormones levels and was affected by other factors, such as the duration of alopecia or the sensitivity of hair follicle cells to androgens. This result should be elucidated by further investigation. Furthermore, they assessed the serum levels of FSH and LH in these patients, and did not find any significant differences between the patients and healthy controls. Similarly, Sanke *et al*., [14] obtained the mean levels of total testosterone (P = .04), DHEA-S (P = 0.02), LH ($P < 0.001$), and prolactin (P = 0.01) were significantly higher in the AGA group, while mean values of FSH ($P < 0.001$) and SHBG ($P < 0.001$) were significantly decreased in the AGA group. The mean FAI was significantly higher in those with AGA than in controls ($P < 0.001$). Like the Starka *et al*., [11] and Duskova *et al*., [15] study showed subnormal levels of testosterone in their studies. 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 causinghair loss [11, 15].

Narad *et al*., [16] found normal values of DHEA-S among men with AGA in their studies. Increased levels of DHEA-S indirectly point toward increased androgen levels, which result in premature balding by acting on the dermal papillae cells. Interestingly, substantially elevated levels of DHEA-S were reported by Legro *et al*., [17] in their study of acne in men, supporting it as a marker of male hyperandrogenism. Furthermore, DHEA-S has special properties such as long half-life and lack of pulsatility, which make it useful as a marker of hyperandrogenism in men. Similar findings were noted by Duskova *et al*., [15] and Narad *et al*., [16] the lower the levels of SHBG, the higher the free testosterone levels, and thus hyperandrogenism aggravates AGA. Narad *et al*., [16] did not show significant difference in LH levels between cases and controls. Increased LH leads to increased testosterone levels and thus contributes to increased male patternbaldness. 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. Significantly low FSH levels were also demonstrated by Starka *et al*., [11] in men with AGA. In men, FSH stimulates the Sertoli cells in the testes to produce testosterone and inhibin. Inhibin decreases the output of FSH bya negative feedback loop. Increased testosterone level also createsa negative feedback loop that decreases FSH.

There were few limitations in our study, including small sample size. In addition, the parameters of assessing alopeciagrade are subjective, and the clinical data were based on patients' memory. The men in the AGA group in our study were not related to women with PCOS.

We suggest that all patients with premature AGA should be assessed for baseline risk factors such as positive family history of AGA, obesity, and abnormal blood glucose levels. Large, multicenter case-control studies should be undertaken to study the endocrinological profile in men with early AGA and these risk factors. We also suggest the same for cases of adolescent alopecia (patterned hair loss occurring before age 18 years), which are increasing in number.

CONCLUSION

This study was concluded that 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, DHEA-S and factors affecting the concentration of free testosterone in the body like SHBG were found to be significantly altered in our patients of AGA. In this study serum LH, prolactin, serum testosterone, DHEA-S and free androgen index are increased whereas, serum FSH and SHBG are decreased in case of AGA 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.

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