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Original Research Article

Androgenetic alopecia- Role of androgen levels

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Abstract

Introduction: Androgenetic alopecia is a genetically determined disorder characterized by increased level of local androgen metabolites and increase androgen receptor binding in genetically predisposed men.

AIM is to study the levels of androgens in males between 18 to 35 yrs with progressive male pattern hair loss (Androgenetic alopecia) with respect to certain demographic variables and clinical severity.

Materials and Methods: A cross sectional study on levels of androgens in males between 18 to 35 years was taken up. A total of 27 males clinically diagnosed as androgenetic alopecia were included. Serum dehydro epiandrosterone sulphate and testosterone were estimated. The results were analysed using Statistical Package for Social Sciences (SPSS) version 19.

Results: Mean ages of patients were 25 years. Mean age of onset was 23 years. Family history was positive in 66.70% and significantly related to clinical severity ($p<0.05$). Most common type was Hamilton Type II pattern observed in 49% patients. Clinical severity was moderate (Hamilton Type II & III) in 51.9% cases. DHEA-S levels were elevated in 14.8%cases. Higher mean DHEA-S levels were significantly associated with greater clinical severity ($p<0.05$).Testosterone levels were normal in 92.6%.

Conclusion: In our study there is a significant association of family history to androgenic alopecia. However there is no significant difference in the mean levels of androgens in groups with and without family history. Hamilton – Norwood Type II pattern was the most prevalent clinical pattern noticed in the study. Testosterone levels are normal in 92.6%. Increased mean levels of DHEA-S is significantly ($P<0.05$) associated with increased clinical severity of male pattern androgenic alopecia.

Keywords: Androgenetic alopecia, Serum Testosterone, DHEA-S.

1. Introduction

Androgenetic alopecia (AGA) is the most common form of alopecia in men and women. AGA is a physiologic process induced in genetically predisposed hair follicles under the influence of androgens which leads to progressive miniaturization of hair follicles.[1,2] The risk of developing AGA increases with a positive family history. Genetic factors can modify the response of the hair follicles to the circulating androgens. The enzyme 5α reductase type 1 & 2, responsible for the reduction of testosterone to the potent DHT, is encoded by the SRD5A1 gene on chromosome 5 and SRD5A2on chromosome 2, respectively. Both forms of this enzyme are expressed in hair follicles.[3] The gene encoding androgen receptors, Stu1 restriction fragment length is present on the X chromosome and may play a role in regulating the potency of androgens available to the hair follicle.[2] Enhanced androgen effects at the genetically predisposed hair follicles are mediated by raised androgen receptor density and /or increased activity of 5α reductase type 2.

2. Materials and Methods

A cross sectional study on serum levels of androgens - dehydro epiandrosterone sulphate and serum testosterone. After obtaining consent study was carried out at Gandhi hospital for a period of one year. A total of 27 males between 18 to35 years clinically diagnosed as androgenetic alopecia were included in the study. Patients with history of acute/chronic illness, patients on medication known to cause hair loss, patients with anemia and abnormal thyroid function were not included in the study. Detailed history including age, occupation, duration, evolution, family history, drug history were carefully recorded. The diagnosis and grading were made based on clinical examination using Hamilton-Norwood classification. Routine investigations like complete blood picture, erythrocyte sedimentation rate, complete urine analysis, random blood sugar, liver function test, Blood urea, Serum creatinine, thyriod function tests were done.

3 ml of blood was collected in sterile containers between 10A.M – 11 A.M in each patient. The clotted blood samples were sent to standard laboratory, after meeting the lab requirements for transport wherein the androgens – DHEAS and testosterone were measured by electro-chemiluminiscence assay .The results were analysed using Statistical Package for Social Sciences (SPSS) version 19.

3. Observations and Results

Out of 27 cases studied, most common presenting age group was between 23-27 years (17 cases) followed by 28-32 yrs (6 cases) and between 18-22 yrs (4 cases).

Out of the 27 cases, 12 were students and 15 were in working population. The duration of alopecia is one year in 10 patients, 2 years in12 and ≥3 years in 5patients. Family history of androgenetic alopecia is present in18 (66.70%) and no family history in 9 (33.30%). The average age for onset of androgenetic alopecia, irrespective of family history was 23 yrs. Out of the 27 cases studied 48.15% of the cases have Type II, 44.4% have Type III and 7.41% have Type IV Hamilton-Norwood Pattern of hair loss.(Table:1)

Based on clinical severity, 13 of them were diagnosed as mild (Hamilton Type II) and 14 as moderate (Hamilton type III & IV). (Table: 2) Of the 18 cases with family history of AGA, 6 (33.3%) had mild while 12 (66.75) had moderate severity of AGA. Of the 9 cases without family history of AGA, 7 (77.8%) had mild and 2(22.2%) had moderate severity of AGA. Positive family history was significantly associated with the clinical severity ($P<0.05$).(Table: 3) Out of the 27 cases studied, 23 (85.2%) of them had normal dehydroepiandrosterone sulphate (DHEA-S) levels.(Table: 4) Out of the 27 cases studied, 25 (92.6%) cases had a normal level of testosterone. The mean values of the parameters studied among the 27 subjects did not bear any statistical significance. (Table:5) The duration of andrgenetic alopecia was statistically related to the clinical severity ($P<0.01$). The mean DHEA-S value showed statistically significant relationship with respect to clinical severity ($P<0.05$).(Table:6) The clinical severity co-related with the mean age presentation of ($P<0.01$). The parameters studied did not show any statistical significance with respect to family history.(Table:7)

Occupation in the group studied is not found to be significantly related to duration of presentation and age at onset.(Table:8)

Table 1: Clinical Classification as per Hamilton-Norwood Pattern

S. No.	Hamilton-Norwood Pattern	No. of Cases	Percent %
1	Type II	13	48.15%
2	Type III	12	44.40%
3	Type IV	2	7.41%

Table 2: Clinical Severity

S. No.	Clinical Severity	No. of Cases	Percent %
1	Mild	13	48.10%
2	Moderate	14	51.90%
	Total	27	100%

Table 3: Association of Family history with Clinical Severity

Association of Family history with clinical severity				P* Value
No. of Cases / Clinical Severity	Mild	Moderate	Total	
Cases without family history	7	2	9	<0.05
Cases with family history	6	12	18	

*Probable value

Table 4: DHEA-S Levels

S. No.	DHEA-S Levels	No. of Cases	Percent %
1	Normal	23	85.20%
2	Abnormal	4	14.80%
	Total	27	100%

Table 5: Mean values of the parameters studied

Variable parameters in the study	No. of Cases	Minimum	Maximum	Mean	Standard deviation
Duration	27	1	3	1.81	0.736
Testosterone Levels	27	1.98	8.45	4.467	1.63353
DHEA – S Levels	27	82	700	292.98	171.502
T3	27	0.73	1.50	1.615	0.1912
T4	27	3.20	12.30	8.17	1.9518
TSH	27	1.17	7.24	2.4944	1.41194

Table 6: Comparison of variables with respect to clinical severity

Variable Parameters in the study	Clinical Severity#	No. of cases	Mean	Standard Deviation	P* Value
Duration	Mild	13	1.38	0.506	
	Moderate	14	2.21	0.699	< 0.01
Testosterone Value	Mild	13	4.6115	2.03186	NS**
	Moderate	14	4.3329	1.21888	
DHEA – S Value	Mild	13	221.15	106.24	0.033
	Moderate	14	359.69	196.101	<0.05
T3	Mild	13	1.1454	0.15629	NS
	Moderate	14	1.1764	0.22377	
T4	Mild	13	7.905	2.266	NS
	Moderate	14	8.416	1.6564	
TSH	Mild	13	2.5754	1.41308	NS
	Moderate	14	2.4193	1.4599	
Age	Mild	13	23.62	2.181	<0.01
	Working	15	26.21	2.517	

*Probability **Not Significant

Table 7: Comparison of parameters with respect to family history

Variable Parameters in the study	Family History	No. of cases	Mean	Standard Deviation	P* Value
Duration	Absent	9	1.56	0.726	NS**
	Present	18	1.94	0.725	
Testosterone Value	Absent	9	4.3322	1.90541	NS
	Present	18	4.5344	1.53563	
DHEA – S Value	Absent	9	267.52	159.234	NS
	Present	18	305.71	180.383	
T3	Absent	9	1.1378	0.18774	NS
	Present	18	1.1733	0.19719	
T4	Absent	9	8.613	1.8794	NS
	Present	18	7.948	2.0019	
TSH	Absent	9	2.5367	1.46074	NS
	Present	18	2.4733	1.42949	
Age	Absent	9	24.67	2.872	NS
	Present	18	25.11	2.632	

*Probability **Not Significant

Table 8: Comparison of parameters with respect to Occupation

Variable Parameters in the study	Occupation	No. #	Mean	Standard Deviation	P* Value
Duration	Student	12	1.50	0.674	P<0.05
	Working	15	2.07	0.704	
Testosterone Value	Student	12	4.3608	1.18306	NS**
	Working	15	4.552	1.95921	
DHEA – S Value	Student	12	252.87	152.574	NS
	Working	15	325.07	183.987	
T3	Student	12	1.1683	0.10241	NS
	Working	15	1.156	0.2441	
T4	Student	12	7.73	2.0261	NS
	Working	15	8.521	1.8844	
TSH	Student	12	2.3467	1.11135	NS
	Working	15	2.6127	1.64263	
Age	Student	12	22.67	0.985	P<0.01
	Working	15	26.80	2.077	

*Probability **Not Significant # Number of cases

4. Discussion

Androgenetic alopecia is an important cause of concern, especially in the young individuals. This study hence focuses on the age group 18 to 35 years which includes both students and working population. Mean age of participants is 25 years, which is comparable to the group studied by Dirk Kranz *et al* (24years).[4] This age group is epidemiologically significant because of various social factors effecting the individual at that point of life including marriage, image building and important career options amongst many others.

The mean age at onset of andregenetic alopecia is 23 years in our study and it was 28 years in the study by Won-soo Lee *et al*.[5] The age of onset and the duration of clinical presentation appeared to be influenced by strong genetic factors. In our study the age at onset was statistically related to positive family history ($P<0.05$).

The clinical severity is dependent upon shortening of anagen phase and hair miniaturization as observed by Therese Leroy *et al*.[6] In our study 44.40% of cases had duration for 1 year , 37% for 2 years and 18.50% of the cases had duration of more than or equal to 3 years. The clinical severity was moderate (Hamilton Type III & IV) in 51.90% and mild (Hamilton Type II) in 48.10% cases. In the 27 cases studied, it is noticed that the moderately severe variety Type III vertex accounted for 44.4% of cases which is comparable to the study by Oslen *et al* (40%).[7] Hamilton Type II was the most common type in the study comparable to study done by V. N. Sehgal.[8]

In our study, 66.7% of cases have a positive family history of androgenetic alopecia. Of these positive paternal history is noted in majority of the cases. This is in accordance with study done by Won-Soo Lee *et al*.[5]

Ina M. Hadshiew *et al* in an article, "Burden of Hair Loss" has mentioned various psychosocial factors and stress with relation to androgenic alopecia. The result of greater number of cases in the working population in the study might be related to increased stress levels compared to students and also the impact of longer duration of the effect of DHT in the working population due to their increased age.[9]

In our study, DHEA-S levels were raised in 14.80% (4 cases). This is contrast to the results where elevation of DHEA-S levels were observed in 100% of cases by pitts RL *et al*.[10]

In our study, 92.60% of the cases have normal testosterone levels. This is in contrast to studies by Starka *et al*,[11] where subnormal levels of testosterone were reported. In individuals with androgenic alopecia, over all levels of testosterone may be normal, however the activity of 5α reductase would be greater than normal so that the amount of DHT in the hair follicle is increased there by leading to androgenetic alopecia.

5. Summary

Our study showed that the mean age group of patients affected was 25 years. Mean age of onset was 23 years. Family history was positive in 66.70% and significantly related to clinical severity ($p<0.05$). Most common type of presentation was Hamilton Type II pattern 49%.Clinical severity was moderate (Hamilton Type II & III) in 51.9%. DHEA-S levels were elevated in 14.8%. Higher mean DHEA-S levels were significantly associated with greater clinical severity ($p<0.05$).Testosterone levels were normal in 92.6% patients.

6. Conclusions

Androgenic alopecia is found to be more common in the younger age group. There is a significant association of family history to the androgenic alopecia in relation to time of onset and severity. However there is no significant difference in the mean levels of androgens in groups with and without family history. Screening of cases with strong family history and counselling them early will help individuals to cope up better and avoid landing in unscientific treatments.

References

[1] Hamilton JB. Disorders of Hair. Rook's textbook of Dermatology (Tony Burns, Stephen Breathnach, Neil Cox *et al*) 8th edition. Willey – Blackwell, 2010:66.13.

[2] Whiting DA. Possible Mechanisms of miniaturization during androgenetic alopecia or pattern hair loss. *J Am Acad Dermatol* 2001; 45:81-6.

[3] Justine A. Ellis, Rodney D. Sinclair. Male Pattern baldness: current treatments, future prospects. *Drug Discovery Today*. 2008; 13(17/18): 791-796.

[4] Dirk Kranz *et al*. Young men's coping with androgenic alopecia: Acceptance accounts when hair gets thinner, Body Image; 8(4):343-348.

[5] Won-Soo Lee *et al*. Analysis of familial factors using the basic and specific (BASP) classification in Korean patients with androgenic alopecia. *Journal o the American Academy of Dermatology* 2011; 65:40-47.

[6] Therese Leroy and Dominique Van Neste. Contrast enhanced phototrichogram pinpoints scalp hair changes in androgen sensitive areas of male androgenetic alopecia. *Skin Research and Technology* 2002:106-111.

[7] E. A Oslen *et al*. Natural history o Androgenetic alopecia. *Clinical and Experimental Dermatology* 1990; 15:34-36.

[8] Ronald L. Pitts. Serum elevation of Dehydropiandrosterone sulphate associated with male pattern baldness in young men. *Journal of American Academy of Dermotology* 1987; 16:71-573.

[9] Ina M Hadshiew, Kertin Foitzik , Petra C Arck and Ral Paus. Burden o Hair loss: Stress and the Underestimated psychosocial Impact of Telogen Efluvium and Androgenetic Alopecia. *Journal o Investigative Dermatology* 2004; 123: 455-457.

[10] Nicole E Rogers and Marc R. Avram. Medical treatment for male and female pattern hair loss. *Journal of American Academy o Dermatology* 2008; 59:547-566.

[11] Starka *et al*. Hormonal Profile of men with premature balding. *Exp. Clin Endocrinol Diabetes* 2004; 112: (1): 24-8.