SIGNIFICANCE OF DYSLIPIDEMIA IN MALES WITH ANDROGENETIC ALOPECIA

D. Asha¹ and Jayakar Thomas²

¹Junior Resident, ²Professor and Head, Department of Dermatology, Sree Balaji Medical College and Bharath University, Chennai, India.

ABSTRACT

Background: Many studies have shown the association between Androgenetic alopecia and cardiovascular disease mainly Coronary artery disease. Aim of the work: This study was designed to evaluate the lipid profile in males with Androgenetic alopecia to find out the incidence of dyslipidemia in these patients. Patients and methods: This case-control study included 30 male patients attending our outpatient department, 15 with Androgenetic Alopecia as case group and 15 with normal hair status as control group. The age group for these patients was between 18-35. Lipid profile including total cholesterol (CHO), triglyceride (TGL), high density lipoprotein (HDL-C), low density lipoprotein (LDL-C), very low density lipoprotein (VLDL), total cholesterol and HDL-C ratio and HDL-C and LDL-C ratio were measured in these patients. Results: Out of the 15 cases with Androgenetic alopecia, 9 cases showed abnormal values in the Lipid profile. There was a significant difference in the serum total Cholesterol/HDL-C ratio (P < 0.05) and LDL-C/HDL-C ratio (P <0.05) between the cases and the controls. Conclusion: We suggest that all males with Androgenetic alopecia must be evaluated for fasting lipid profiles, as abnormal lipid profiles are considered as an early indicator for cardiovascular risk in these patients.

INTRODUCTION

Androgenetic alopecia commonly called as male pattern baldness is the most common cause for hair loss in men which is a progressive, patterned loss of hair when genetically predisposed men are exposed to androgens. High levels of androgen contribute to the development of atherosclerosis and thrombosis leading to the development of hypercholesterolemia. It is considered to be the most common type of baldness in males. It commonly begins in males by the age of 20 years and affects nearly 50% of men by 50 years of age (Hoffmann R and Happle R, 2000; Sinclair RD and Dawber RP, 2001; Al-Sadat Mosbeh et al., 2014). There is androgen-mediated conversion of susceptible terminal hair follicles to vellus hairs. Some twin studies in males indicate that there is a genetic predisposition for Androgenetic alopecia. Androgenetic alopecia can have a negative impact on the quality of life of the affected persons. Several studies have shown the association of Androgenetic alopecia with Diabetes, Hypertension, Coronary artery disease, Cancers and Metabolic syndromes. Coronary artery disease is the major cause for death and disability worldwide (Lopez et al., 2001). Many studies have evaluated Lipids to be the pathogenic factor for Coronary artery disease in Androgenetic alopecia patients. Several studies have shown that baldness is associated with a risk of Coronary artery disease. Obstructive and advanced Coronary artery disease presenting with minimal or no symptoms can progress rapidly and so its detection at an early stage is very important (Lerner and Kannel, 1986).

This study is designed to substantiate the incidence of dyslipidemia in Indian males with androgenetic alopecia, as screening of Androgenetic alopecia patients for dyslipidemia can lead to early intervention and prevention of patients from developing cardiovascular complications like coronary artery disease in the future.
PATIENTS AND METHODS
This cross sectional study included 30 male patients, 15 with Androgenetic alopecia as case group, and 15 with normal hair status as control group attending our Dermatology outpatient department. Androgenetic alopecia was diagnosed based on family history, clinical findings and the pattern of thinning of hair. The pattern and grading of baldness was done using the Basic and Specific (BASP) classification for Androgenetic alopecia.

The age of these patients was between 18 and 35 years. An informed written consent was obtained from each of these patients. These patients were subjected to full history taking, thorough general and dermatological examination. Those patients with systemic disorders, other alopecia’s and on any medication that could affect the lipid metabolism, smoking and alcohol intake, cancer was excluded from the study.

The patients were subjected to a 12 hour fasting period after which a venous blood sample was taken for lipid profile which includes total cholesterol (CHO), high density lipoprotein (HDL-C), low density lipoprotein (LDL-C), triglyceride (TGL), very low density lipoprotein (VLDL), total cholesterol and HDL-C ratio and HDL-C and LDL-C ratio.

Figure 1. Clinical photograph of a male of age 30 years showing Androgenetic alopecia in the vertex region.

Table I. Mean and Standard Deviation for Age and Lipid levels in men with Androgenetic alopecia and their Controls

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Cholesterol</td>
<td>30</td>
<td>169.3667</td>
<td>23.16431</td>
<td>105.00</td>
<td>197.00</td>
</tr>
<tr>
<td>HDL-C</td>
<td>30</td>
<td>45.1260</td>
<td>9.03314</td>
<td>32.00</td>
<td>65.78</td>
</tr>
<tr>
<td>TGL</td>
<td>30</td>
<td>103.4057</td>
<td>25.75392</td>
<td>59.00</td>
<td>160.00</td>
</tr>
<tr>
<td>LDL-C</td>
<td>30</td>
<td>110.3933</td>
<td>19.12561</td>
<td>36.00</td>
<td>146.00</td>
</tr>
<tr>
<td>VLDL</td>
<td>30</td>
<td>21.3867</td>
<td>5.57654</td>
<td>11.80</td>
<td>34.00</td>
</tr>
<tr>
<td>Total Cholesterol/ HDL-C ratio</td>
<td>30</td>
<td>3.8777</td>
<td>.86740</td>
<td>2.20</td>
<td>6.21</td>
</tr>
<tr>
<td>LDL-C/HDL-C ratio</td>
<td>30</td>
<td>2.5563</td>
<td>.73444</td>
<td>.72</td>
<td>4.60</td>
</tr>
<tr>
<td>Age</td>
<td>30</td>
<td>25.4667</td>
<td>4.36074</td>
<td>19.00</td>
<td>35.00</td>
</tr>
</tbody>
</table>

HDL-C: high-density lipoprotein-cholesterol, TGL: triglycerides, LDL-C: low density lipoprotein-cholesterol, VLDL: very low density lipoprotein.

RESULTS
30 patients attending our outpatient department, 15 with Androgenetic alopecia as cases and 15 with normal hair status as controls were studied based on the BASP classification. The mean and standard deviation for cases and controls are shown in Table I. Significant differences were found between Androgenetic alopecia patients and the controls with respect to the Total Cholesterol and HDL-C ratio (p = .006). Significant difference was observed also with respect to the LDL-C and HDL-C ratio between the cases and controls (p = .006). No significant differences were found between the cases and controls with respect to the Total Cholesterol, Triglycerides, HDL-C, LDL-C, VLDL. The p-value for each variable is shown in Table II.
**REFERENCES**


**DISCUSSION:**

Androgenetic alopecia or male pattern hair loss is the most common cause for baldness in males which is characterized by progressive loss of hair. It is a common psychosocially and cosmetically distressing condition. Genetic studies have shown the association of Androgenetic alopecia with polymorphism in the androgen receptor gene on X chromosome. There is androgen-mediated conversion of terminal hairs into vellus hairs. The thick, pigmented terminal hairs gradually transform to thin, short, indeterminate hairs and finally to non-pigmented vellus hairs (Lata Sharma et al., 2013).

The dihydrotestosterone is responsible for the shrinkage of the hair follicle that is genetically sensitive to it. 5 α-reductase is responsible for the conversion of free testosterone into dihydrotestosterone (Ellis et al., 1988; Nyholt et al., 2003). Many studies have shown that the 5 α-reductase are also present in the heart and blood vessels. The dihydrotestosterone receptors are involved in the proliferation of smooth muscles which is a main feature that may lead to atherosclerosis along with deposition of lipids (Fujimoto et al., 1994).

This study was based on the Basic and Specific (BASP) classification. The BASP classification is for patterned hair loss that is universal for both women and men. It is based on the pattern of hair loss, including the shape of the anterior hair line and the density of hair on the frontal and vertex areas. The basic type represent the shape of the anterior hair line, the specific type represent the density of hair on specific areas like the vertex and frontal areas and the final type is based on the combination of the two types.

Early onset of androgenetic alopecia leads to psychological stress, and has been regarded as an important risk factor for cardiovascular diseases (Lotufo et al., 2000), especially Coronary artery disease, metabolic syndromes (Denmark Wahnefried et al., 2000) and prostate cancer (Su LH, Chen TH, 2010). Dyslipidemia is a major risk factor for Coronary artery disease. Cotton et al in 1972 was the first person to suggest the association between Androgenetic alopecia and Coronary artery disease (Cotton et al., 1972; Lemieux et al., 2000; Won-Soo Lee et al., 2007). In this study, there is significant difference in the Total Cholesterol and HDL-C ratio and LDL-C and HDL-C ratio between the cases and controls.

These two ratios have been found to be a sensitive predictor for coronary heart disease in men. Hence control of these two ratios is very important to prevent the development of complications of the cardiovascular system in these patients.

**CONCLUSION**

This study shows the association of Androgenetic alopecia with dyslipidemia, indirectly with Coronary artery disease. So every patients diagnosed with Androgenetic alopecia must be evaluated for fasting lipid profiles, as abnormal lipid profiles are considered as an indicator for Cardiovascular disease, and an early intervention can be taken in preventing these patients from developing Coronary artery disease in the future.

**ACKNOWLEDGEMENT:** NONE

**CONFLICT OF INTEREST:**

The authors declare that they have no conflict of interest.

**STATEMENT OF HUMAN AND ANIMAL RIGHTS**

All procedures performed in human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

**Table II. The p-value of Lipid Profiles in cases and controls**

<table>
<thead>
<tr>
<th>Lipid Profile</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Cholesterol</td>
<td>.759</td>
</tr>
<tr>
<td>TGL</td>
<td>-</td>
</tr>
<tr>
<td>HDL-C</td>
<td>.500</td>
</tr>
<tr>
<td>LDL-C</td>
<td>.500</td>
</tr>
<tr>
<td>VLDL</td>
<td>.264</td>
</tr>
<tr>
<td>Total Cholesterol/HDL-C ratio</td>
<td>.006 (S)</td>
</tr>
<tr>
<td>LDL-C/HDL-C ratio</td>
<td>.006 (S)</td>
</tr>
</tbody>
</table>

TGL: triglycerides, HDL-C: high density lipoprotein-cholesterol, LDL-C: low density lipoprotein-cholesterol, VLDL: very low density lipoprotein. (S): significant difference: p-value < 0.05