Original article:

Impact of Lipid Profile Parameter as an Indicator of Coronary Artery Disease in Woman with Female Pattern Alopecia

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ABSTRACT

Introduction: Hair loss may be a benign and transient process, or can be a serious and permanent problem. The majority of hair loss complaints seen in both males and females are caused by androgen-dependent or increased telogen hair shedding. Our aim is to investigate the impact of lipid profile parameter as an indicator of coronary artery disease in woman with female pattern alopecia.

Materials and methods: Fifty female patients with female pattern of hair loss and fifty female participants were included in control group. The age range from 20 to 50 years were enrolled as the case and control groups, respectively. The Hamilton-Norwood scale 16 was used forgrading of AGA.

Results: Positive family history of AGA, HT, DM and CAD was significantly higher in cases than the controls. The mean level of Apo A1 was higher in the patients than the controls although they were within the normal limits in both groups. There were no statistically significant differences in the mean level of cholesterol, triglyceride, LDL, HDL– cholesterol, and Apo B between the patients and the controls.

Conclusion: lipid profiles, especially LP (a), should be measured in women with AGA to find out those at risk of CAD.

Keywords: CoronaryArtery Disease, Lipid Profiles, Alopecia, Cholesterol, Triglyceride.

INTRODUCTION

The cosmetic and psychological importance is immense of hairs, although Hair has no vital function in humans. Loss of hair from scalp is even more distressing than growth of the body in woman or facial hair in excess of the culturally acceptable norms.

Hair loss may be a benign and transient process, or can be a serious and permanent problem. The majority of hair loss complaints seen in both males and females are caused by androgen-dependent or increased telogen hair shedding.

The hair follicles in humans show a cyclical activity. The phases of hair cycle include anagen (86%), catagen (1%), telogen (13%).[1] Release of the dead hair from the follicle (exogen) occurs either late in telogen or early in anagen. Besides, these phases another phase of hair growth cycle has been described which is called as the kenogen.[2]

Female pattern hair loss (FPHL) has emerged as the preferred term for androgenetic alopecia (AGA) in females owing to the uncertain relationship between androgens and this entity.[3]It is characterized by a reduction in hair density over the crown and frontal

scalp with retention of the frontal hairline. In 1977, Ludwig clearly described the distinctive features of FPHL and classified it into three grades of severity referred to as Ludwig grades I, II, and III.[4]

Initially, the term "diffuse alopecia in women" was widely used to characterize the disease.[5,6] After 1942, when Hamilton demonstrated of the involvement of male hormones in the development of classic pattern baldness in men, the term androgenetic alopecia was established to emphasize the hormonal and genetic factors associated with the development of the disease.[7,8] Because diffuse pattern alopecia (which often affects women) was thought to be a variant of the same entity, the term female androgenic alopeciaor female pattern baldness, started to be used.[6] FPHL is the condition that most commonly leadsto hair loss in adult females. Its prevalence increases with age and the disease shows an inconsistentresponse to treatment.[9]These elements compromise body image and strongly affect self-esteem, negatively impacting the patients' quality of life.There can be a correlation between androgenic alopecia (AGA) and life-threatening diseases, such as coronary artery disease (CAD). The frequency of CAD has been shown to increase in AGA.[10-12] Furthermore, studies have shown that the pattern of lipid profile of men with AGA predisposes them to CAD.[13,14]

The effect of serum lipid parameters on atherosclerotic heart disease has been well documented. In particular, lipoprotein a (LP a) and apolipoproteins that have been shown as important independent risk factors for CAD.[15]An increased low density lipoproteins to high density lipoproteins (LDL:HDL) ratio has already been considered a sensitive predictor of cardiovascular risk.

Clarifying the potential relationship between androgenetic alopecia and CAD might help to better understand thepathophysiology of coronary heart disease.Our aim is to investigate the impact of lipid profile parameter as an indicator of coronary artery disease in woman with female pattern alopecia.

Materials and Methods

This case control study was undertaken in the department of Dermatology, Era's Lucknow Medical College & Hospital, Lucknow. Data was collected from woman who were attending dermatology department in hospital. 100 females were enrolled in the study, in which fifty female patients with female pattern of hair loss and fifty female participants were included in control group. The age range from 20 to 50 years were enrolled as the case and control groups, respectively.

The study had the approval from the institutional ethical committee and the written consent was taken from all the participants in case and control groups, respectively.

Exclusion criteria

- History of coronary artery bypass surgery,
- History of acute coronary syndrome
- Women who were on drugs
- Which could affect lipid metabolism, smokers, alcohol consumers, and
- Those with diabetes mellitus (DM), CAD, hypertension (HT), familial hyperlipidemia, thyroid disease, iron deficiency, chronic renal failure, liver disease, polycystic ovary syndrome (PCOS), and cancer were excluded from the study.

The Hamilton-Norwood scale 16 was used for grading of AGA, and the all participants were assessed by the same doctor. The severity of hair loss was assessed according to the Ludwig classification

and for those whose pattern of hair loss was not compatible with the Ludwig pattern. After fasting of 8 hours, 5 ml of venous blood was drawn in a sterile syringe and submitted to the laboratory for lipid profile evaluation including cholesterol, triglyceride, high density lipoprotein (HDL), low density lipoprotein (LDL), lipoprotein (a) [LP(a)], apolipoprotein A1 (Apo A1), and apolipoprotein B (Apo B). The immunoturbidimetric method was used to measure Apo A1 and Apo B levels and LP (a) levels were determined using the nephelometric assay. The levels of thyroid stimulating hormone (TSH), free thyroxine (T4), serum iron, ferritin, TIBC, hemoglobin, and hematocrit were also measured to exclude thyroid disease and anemia in the study group. The analysis of the data was performed by using SPSS computer program software. Data was analyzed with t-test and Chi-square test. A P-value of <0.05 was considered significant.

Results

100 females were enrolled in the study, in which fifty female patients with female pattern of hair loss and fifty female participants were included in control group. The age range from 20 to 50 years were enrolled as the case and control groups, respectively. Mean age was 31.29 ± 11.03 years in the case and 27.3 ± 7.8 years in the control group with no significant difference between them (Table 1, fig.1). Details offamily history regarding AGA, HTN, DM and CADin the case and control groups are shown in table 2,fig.2, in which 88% have positive family history of Androgenic Alopecia, 72% have hypertension, 44% Diabetes Miletus, 60% have Coronary Artery Disease in case group. In control group The positive family history of Androgenic Alopecia found in 2%, hypertension in 6%, Diabetes Miletus in 2% and Coronary Artery Disease found in 0% cases. As shown, a positive family history of AGA, HT, DMand CAD was significantly higher in cases than thecontrols (P<0.05). The levels of total serum cholesterol, triglyceride,HDL–cholesterol, LDL–cholesterol, LP (a), Apo A1,and Apo B are shown in the table 3, fig.3. As it is shownin the table, the level of LP (a) was significantlyhigher in the patients than the controls.The mean level of Apo A1 was higher in thepatients than the controls although they were withinthe normal limits in both groups. Therewere no statistically significant differences in themean level of cholesterol, triglyceride, LDL, HDL–cholesterol, and Apo B between the patients andthe controls.

Table 1:Description of age in case and control group

Variable	Group		
	Case (%) (Mean ± SD)	Control (%) (Mean ± SD)	P value
Age	31.29 ± 11.03	27.3 ± 7.8	0.12

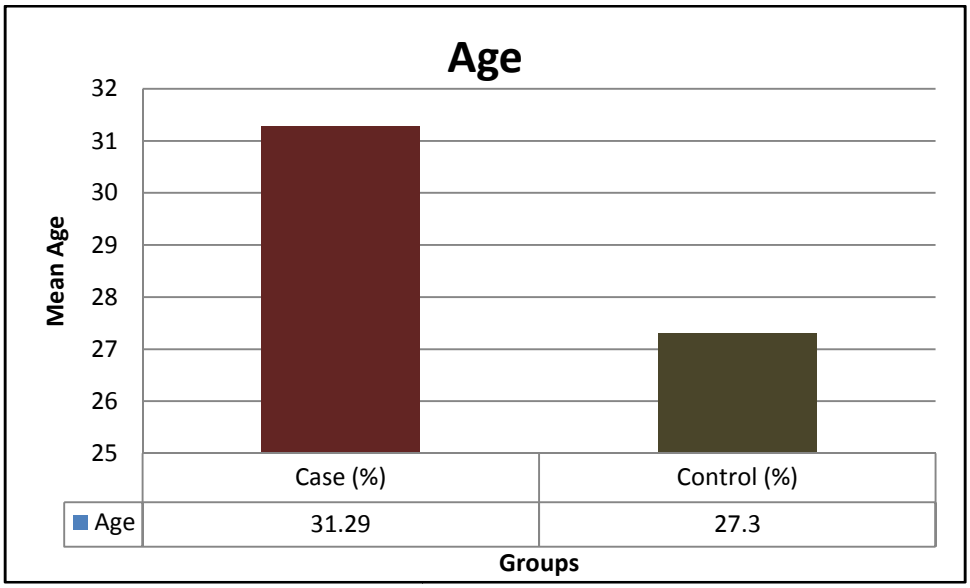

Figure 1: Description of age in case and control group

Table 2: Description of family history of the case and control group

Family history	Group	
	Case (%)	Control (%)
Androgenic Alopecia	44 (88%)	1 (2%)
Hypertension	36 (72%)	3 (6%)
Diabetes Milletus	22 (44%)	1 (2%)
Coronary Artery Disease	30 (60%)	0 (0%)

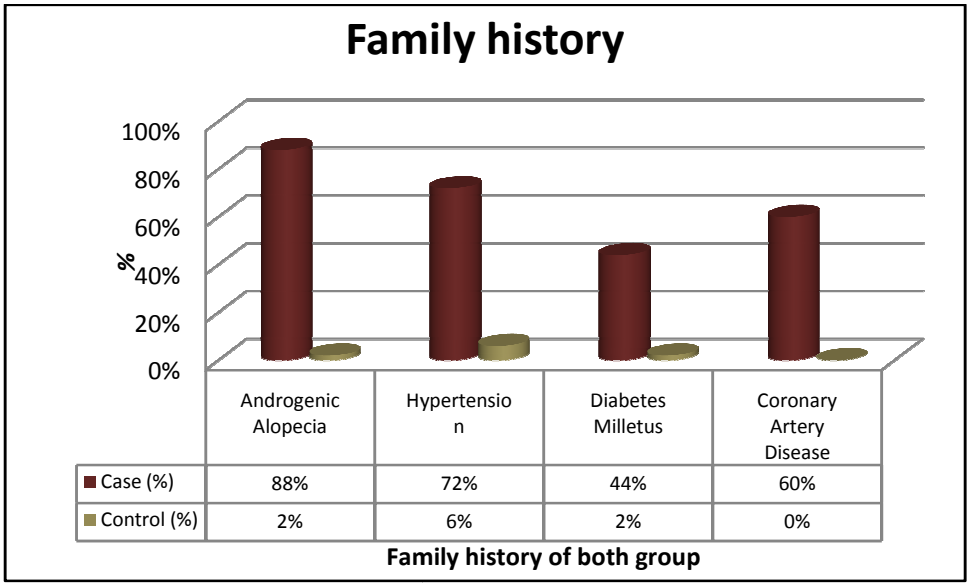

Figure 2: Description of family history of the case and control group

Table 3:The mean values of the lipid profile evaluation between case and control groups

Variable	Case group (Mean ± SD)	Control group (Mean ± SD)	*P*-value
Triglyceride	84.56 ± 32.61	102.91 ± 36.93	0.243
Cholesterol	172.63 ± 87.1	153.68 ± 37.81	0.129
High Density Lipoprotein (HDL	62.73 ± 96.31	51.28 ± 12.39	0.186
Low Density Lipoprotein (LDL),	100.68 ± 43.91	106.11 ± 67.23	0.170
Lipoprotein (A) [LP(A)]	49.53 ± 65.82	22.12 ± 67.73	0.001
ApolipoproteinA1 (Apo A1)	151.71 ± 23.01	107.97 ± 14.53	0.04
ApolipoproteinB (Apo B)	83.78 ± 69.52	84.37 ± 62.51	0.621

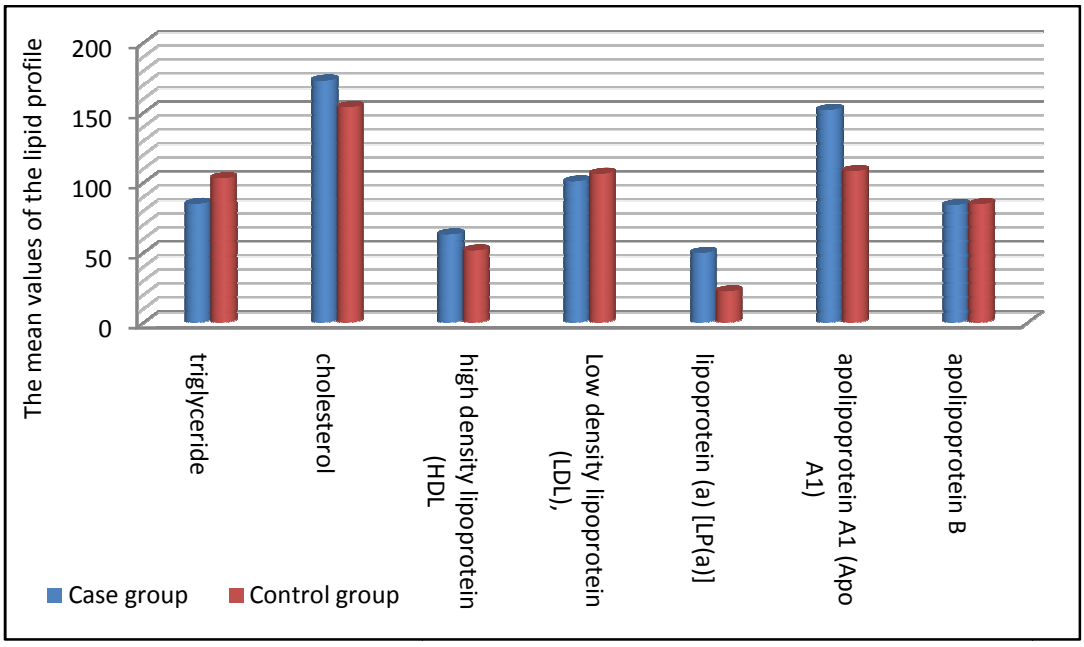

Figure 3: The mean values of the lipid profile evaluation between case and control groups

Discussion

Details of family history regarding AGA, HTN, DM and CAD in the case and control groups are shown in table 2, in which 88% have positive family history of Androgenic Alopecia, 72% have hypertension, 44% Diabetes Miletus, 60% have Coronary Artery Disease in case group. In control group.The positive family history of Androgenic Alopecia found in 2%, hypertension in 6%, Diabetes Miletus in 2% and Coronary Artery Disease found in 0% cases. As shown, a positive family history of AGA, HT, DM and CAD was significantly higher in cases than the controls ($P<0.05$). The level of LP (a) was significantly higher in the patients than the controls. The mean level of Apo A1 was higher in the patients than the controls although they were within the normal limits in both groups. There were no statistically significant differences in the mean level of cholesterol, triglyceride, LDL, HDL– cholesterol, and Apo B between the patients and the controls.

Trevisanet al.[17]showed that patients with fronto-occipital baldness had higher serum cholesterol and blood pressure on the average compared to participants of similar age with no baldness. On the other hand, Matilainenet al.[18]could not report any significant differences in lipid profiles at all between women with AGA and women without AGA.the exact reason for the association betweenbaldness and CAD is unclear, several mechanisms have been proposed. First, elevated androgen levels and increased peripheral sensitivity to androgens have been reported, both in subjects with androgenetic alopecia and CAD.[19-21]

In 1972, Cotton et al.[22] for the first time reported anassociation between CAD and baldness. They found baldnessas a CAD risk factor in 91 patients with overt CAD.However, in 1978, Halimet al.[23] reported that patientswith myocardial infarction had no increase in male patternalopecia. In 1979, Cooke[24] reported that there was noassociation between CAD and either male pattern alopeciaor premature male pattern alopecia in 478 male Caucasianhospital inpatients. In 1993, in a case-control study, Leskoet al.[25]reported that vertex baldness (threefold higher riskof myocardial infarction) but not frontal baldness was associatedwith myocardial infarction among men younger than55 years.Measurement of the LP(a) level hasbeen recommended to determine the risk ofmyocardial infarction.[26,27]LP(a) excess is commonlyseen in patients with premature CHD. In one study,LP (a) excess was present in 18.6% of the patientswith premature CHD, of whom 12.7% had no otherdyslipidemia.[28] In 1999, Sasmaz et al, showed thatmen with AGA had significantly higher levels ofserum LP (a) compared to men with no AGA.[29]Lotufu et al, also showed similar results in their study[30] whileMatilaiuen et al, could not find higher levelsof LP (a) in AGA.[31]

Conclusion

In conclusion as LP (a) is a genetically independent risk factor for CAD the relationship between AGA and CAD can be intervened via this mechanism. we suggest that lipid profiles, especially LP (a), should be measured in women with AGA to find out those at risk of CAD.

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