**Female pattern hair loss: Risk factor for atherosclerosis**

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No Conflict of interest

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Abstract

Introduction & Objectives: Androgenetic alopecia (AGA), is the most common hair loss disorder affecting both men and women, and is characterized by progressive, patterned hair loss from the scalp. Genes and hormones are implicated in the pathophysiology of this hair type disorder. The clinical change in hair density, related to a change in the hair cycle and miniaturization of the hair follicle, is generally considered to be potentially reversible. The aim of this study was to investigate the serum lipids profile in females with female pattern hair loss and their possible effect on the development of atherosclerosis in Egyptian women.

Material & Methods: The current study included twenty seven female patients suffering from AGA and twenty seven ages matched healthy controls. Blood samples were taken from all subjects for CBC and for measurement of lipid levels including serum level of TC, TG, HDL-C a, LDL-C and LP (a). Ratios of TC: HDL-C and LDL-C: HDL-C was also calculated to assess the atheroscelerotic risk in patients compared to healthy controls.

Results: The results revealed that patients have statistically significant higher levels of TC, TG and LDL-C than controls, while we could not find significant changes as regard levels of LP (a) nor HDL-C. In addition, the atheroscelerotic risk was also statistically highly significant in the case group compared to the control group. There was correlation between the various lipid levels and the stage of female AGA. On the other hand, no correlation could be drawn between the various lipid levels and the duration or BMI of female AGA.

Conclusion: Results of this study confirm that there is a true association between the high serum lipid levels and the female AGA. Females with AGA carry risk factor atherosclerosis more than healthy females. Therefore, serum lipid values especially LP(a) should be re-evaluated in women with AGA in order to detect risk of developing cardiovascular disease in patients with early onset AGA and signal a potential opportunity for early preventive treatment.

**Introduction**

Androgenetic alopecia (AGA), is the most common hair loss disorder affecting both men and women, and is characterized by progressive, patterned hair loss from the scalp. Genes and hormones are implicated in the pathophysiology of this hair type disorder. The clinical change in hair density, related to a change in the hair cycle and miniaturization of the hair follicle, is generally considered to be potentially reversible. The aim of this study is to investigate the serum lipids profile in females with female pattern hair loss and their possible effect on the development of atherosclerosis in Egyptian women.

Although hair loss in women increases with advancing age and is usually more prevalent in women following menopause (Vujovic A, Del Marmol V2014) many younger women also experience hair loss. Indeed, there is a substantial need for treatment of hair loss among women aged 25 to 40 years. (Ramos PM, Miot HA 2015). Prevalence of AGA increase with advanced age, 12% of women develop FPHL by age of 29 years, 25% by age of 49 years, 41% by 69 years, and over 50% have some element of FPHL by 79 years (Gan DC Sinclair , 2005)

Loss of hair is a common complaint and may lead to psychosocial problems. 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 (Mark, 1997).

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 (Olsen, 2001). 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 (Ludwig, 1977).

Correlation between AGA and life-threatening diseases, such as coronary artery disease (CAD) has been investigated. The association between male pattern baldness and CAD was first suggested by Cotton et al. 5 but this association in women was not documented. Later on it was found that female AGA, like male pattern baldness, was associated with CAD in women younger than 55 years but the mechanism of this association was not clear (Mansouri et al., 2005).

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. An increased low density lipoproteins to high density lipoproteins (LDL: HDL) ratio has already been considered a sensitive predictor of cardiovascular risk (Ari and Yigitoglu , 1997)

Few studies have focused on the effect of lipid parameters on the relationship between AGA and CAD (Barud et al., 2002) .Level of Lp (a) which is an important and genetically determined risk factor for CAD was found greater in some AGA patients than the critical level for atherosclerosis. So lipid profiles especially LP(a), should be measured in women with AGA to find out those at risk of CAD (Farajzadeh et al., 2010).

**Patients and Methods**

This study was carried out at the outpatient clinics of Dermatology, Venereology and Andrology Department and Internal Medicine department, Faculty of Medicine, Zagazig University Hospitals in the period from December 2013 till October 2014.Fifty-four female participants aged 20 – 50 years were enrolled in the study.

Participants were divided into two groups: the first group included 27 female patients aged 20 – 50 years having female pattern hair loss diagnosed clinically with Ludwig grades. The second group included 27 healthy controls in the same age range.

Excluded criteria from this study were Participants with symptoms and signs of androgen excess such as menstrual irregularities, history of infertility, hirsutism, severe unresponsive cystic acne, virilization, or galactorrhea; previous diagnosis of polycystic ovary syndrome or other endocrinal disorders; and those receiving hormonal treatment or hormone-releasing intrauterine contraceptive device or drugs that may affect blood glucose levels. None of the patients had other types of alopecia or systemic diseases that may lead to hair fall.

**Patient assessment:** All patients were subjected to the following assessments.

a. Full history-taking.

b. Menstrual history.

c. Full clinical examination.

d. Dermatological examination.

e. Local scalp examination:

Diagnosis of AGA was based on clinical findings (the pattern of hair loss: reduction in hair density over the crown and widening of the central part) and family history of AGA.

Grading of androgenetic alopecia:

Patients were graded as stage I, II or III AGA according to clinical examination of the pattern of hair loss that based on Ludwig’s scale.

f. Investigations:

Patients and controls were subjected to the following:

After fasting for 8 hours, 5 ml of venous blood was drawn into a sterile syringe and submitted to the laboratory for centrifugation and isolation of blood serum and was kept at 20C until the test day.

The blood sample was used for lipid profile evaluation including cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL). They were measured by using **Spin react kit** (**made in Spain**).

Lipoprotein a was also measured by Enzyme Linked immunosorbent Assay (ELISA). Solid phase capture sandwich ELISA assay with a micro well format was used.

**Statistical analysis**

Data were checked, entered and analyzed using SPSS version 16 for data processing and statistics. Data were expressed as number and percentage for qualitative variables and mean ± standard deviation (SD) for quantitative ones. A p value <0.05 indicates significant results. Comparison between the two groups was done using t test and Chi square tests Data were represented as mean ± SD, range, numbers or percentages. P ≤ 0.05 was considered statistically significant, P>0.05 was considered Non-significant, P <0.01 was considered statistically highly significant.

**Ethical considerations**: the study protocol was approved by the Institutional Review Board of the faculty of Medicine, Zagazig University, Egypt. All patients gave written informed consent to participate in the study and for performing all relevant interventions.

**Results**

Fifty-four female were enrolled in the study, twenty-seven female patients with female pattern hair loss by Ludwig grades and twenty-seven normal female participants as a control group. The subjects were divided into two groups (a, b).

Table 1. Characteristics of the two groups.

|  |  |  |  |
| --- | --- | --- | --- |
|  | Cases  (N=27) | Controls  (N=27) | p |
| Age (years) | (37.1 ± 7.9) | (34.7 ± 5.7) | 0.17 |
| AGA | 21(77.8 %) | 2(7.4 %) | 0.00\*\* |
| CVS | 15(55.6 %) | 1(3.7 %) | 0.00\*\* |
| Body mass index (BMI) | 29.3±6.9 | 28 ± 5.9 7.1 | 0.17 |
| TC: HDL-C | (6.7 ± 1.4 ):(4.3 – 10) | (4.5 ± 1.5 ):(1.9–7.6) | 0.00\*\* |
| LDL-C: HDL-C | (4.9±1.2):(2.4 – 8) | (3 ± 1.5):(0.4–5.9) | 0.00\*\* |

P value is significant < 0.05

Group (a) included twenty-seven female patients with AGA; their ages ranged from 23 to 48 years with a mean of (37.1 ±7.9), all patients had normal CBC, liver and renal functions and pelviabdominal ultrasonography.

Group (b) included twenty-seven normal female participants, their ages ranged from 21 to 43 years with a mean of (34.7 ±5.7).There was not a statistically significant difference between the case and the control group regarding age.

The family history of AGA was positive in 21 (77.8%) of case group, versus 2 (7.4%) of the control group, there was statistically significant difference between case and control groups with a highly significant P value (P<0.01).On the other hand, 15 (55.6%) of case group had a family history of cardiovascular disease versus 1 (3.7%) of the control group. Positive family history of cardiovascular disease in the case group was markedly higher compared with that of the control group, with a highly significant P value (P<0.01), table (2).

**Ludwig pattern grades**

According to Ludwig classification 16 patients (59.3%) were Ludwig grade 1, 10 patients (37.0%) were Ludwig grade 2 and one patient (3.7%) was Ludwig grade 3.

Total cholesterol (TC) in the case group ranged from 136 – 354mg/dl, with a mean of 238.7 ± 49.7mg/dl. While TC of the control group ranged from 160 – 225mg/dl with a mean of 192.9 ± 18.1mg/dl. Triglycerides (TGs) level in the case group ranged from 112 – 296mg/dl, with a mean of 200.4 ±65.4mg/dl. Whereas, TG level in the control group ranged from 65 – 160mg/dl, with a mean of 98.7 ±22.3mg/dl.

Low-density lipoprotein cholesterol (LDL-C) in the case group ranged from 69 – 255.8mg/dl, with a mean of 155.2 ± 42.1mg/dl. On the other hand, LDL-C in the control group ranged from75.2 –154.5mg/dl, with a mean of 125.1 ± 20.8mg/dl.

High-density lipoprotein cholesterol (HDL-C) in the case group ranged from 23.5 – 69.8mg/dl, with a mean of 43.1 ±10.5mg/dl. While HDL-C in the control group ranged from 33.1 – 52.7mg/dl, with a mean of 44.1 ± 5.8mg/dl.Lipoprotein an (LP (a)) in the case group ranged from 10 – 81mg/dl, with a mean of 28.1 ± 16.5 mg/dl. While LP (a) in the control group ranged from 10 – 30 mg/dl, with a mean of 9.6 ± 6.8 mg/dl. Patients had significantly high mean values of total cholesterol (TC), triglyceride (TG) level, and low-density lipoprotein (LDL-C). HDL-C was found to be lower than in healthy controls despite that the mean value of HDL-C in both groups was within the normal range. LP (a) is higher in case group but statistically insignificant, table 4.

Table (2): Shows the mean values of the lipid parameters in cases and control groups

|  |  |  |  |
| --- | --- | --- | --- |
| Lipid level (mg/dl) |  |  | P |
| TC | (238.7 ± 49.7) | (192.9 ± 18.1) | 0.00\*\* |
| TGs | (200.4 ± 65.4) | (98.7 ± 22.3) | 0.00\*\* |
| LDL-C | (155.2 ± 42.1) | (125.1 ± 20.8) | 0.00\*\* |
| HDL-C | (43.1 ± 10.5) | (44.1 ± 5.8) | 0.65 |
| LP(a) | (28.1 ± 16.5) | (19.6 ± 68) | 0.07 |

It is worth mentioning that there was a statistically significant correlation between cholesterol level, LDL and lipoprotein-a in patients of AGA and the severity of AGA according to Ludwig criteria as (P <0.05) while the levels of HDL showed no significant correlation as (P > 0.05), table 5.

There was no significant difference between Body mass index (BMI) of cases and controls (P >0.05).

The ratio of TC: HDL-C in the case group ranged from 4.3 to 10, with a mean of 6.7 ± 1.4 SD. On the other hand, the ratio of TC: HDL-C in the control group ranged from 1.9 to 7.6, with a mean of 4.5 ± 1.5 SD.

Furthermore, the ratio of LDL-C: HDL-C in the case group ranged from 2.4 to 8, with a mean of 4.9 ± 1.2 SD. While the ratio of LDL-C: HDL-C in the control group ranged from 0.4 to 5.9, with a mean of 3 ± 1.5 SD. TC: HDL-C, and LDL-C: HDL-C in the case group were markedly higher compared with that of the control group, with a highly significant P value (P<0.01.

Table (3): Shows correlation between AGA severity (Ludwig criteria) and lipid profile in patients:

|  |  |  |
| --- | --- | --- |
| Variable | Ludwig criteria | P |
| Cholesterol | 0.33 | 0.03\* |
| HDL | - 0.23 | 0.06 |
| LDL | 0.52 | 0.02\* |
| Lipoprotein (a) | 0.28 | 0.04\* |

There was no significant correlation detected between mean values of TC, TGs, HDL-C, LDL-C, LP(a) and different variables like the age, BMI and duration of female AGA (P>0.05).

Ratios of TC: HDL-C and LDL-C: HDL-C had no significant correlation with different variables including the age, BMI, duration and stage of female AGA (P>0.05).

Table (4): shows Correlation between lipid parameters and different variables in AGA patients:

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  |  | TC | TG | HDL-C | LDL-C | LP(a) |
|  |  |  |  |  |  |  |
| Age (years) | r | -0.163 | -0.093 | 0.161 | -0.023 | 0.211 |
| P | 0.258 | 0.523 | 0.264 | 0.877 | 0.260 |
| BMI (kg/m2) | r | -0.091 | 0.032 | 0.091 | 0.105 | 0.091 |
| P | 0.530 | 0.826 | 0.529 | 0.467 | 0.520 |
| Duration of AGA (years) | r | 0.059 | 0.046 | 0.243 | 0.154 | 0.032 |
| P | 0.685 | 0.750 | 0.089 | 0.285 | 0.810 |

Table (5): Shows Correlation between ratios of TC: HDL-C and LDL-C: HDL-C and different variables in patients:

|  |  |  |  |
| --- | --- | --- | --- |
|  | | | |
|  |  | TC:HDL-C | LDL-C:HDL-C |
| Age (years) | r | -0.163 | -0.070 |
| P | 0.258 | 0.628 |
| BMI (kg/m2) | r | -0.099 | 0.013 |
| P | 0.492 | 0.928 |
| Duration of AGA (years) | r | -0.091 | -0.016 |
| P | 0.530 | 0.910 |
| Stage of AGA | r | 0.034 | 0.090 |
| P | 0.813 | 0.532 |

**DISCUSSION**

Androgenetic alopecia is the most common hair loss disorder, affecting both men and women. It leads to progressive miniaturization of the hair follicle with a usually characteristic pattern distribution in genetically predisposed men and women (Blume-Peytavi et al., 2011).

Females have been found to have higher levels of 5-α reductase, more androgen receptors, and lower levels of cytochrome P450 (which converts testosterone to estrogen)(Drake et al.,1996).Although androgen excess in females leads to the growth of unwanted body hair or hirsutism, it is generally not a prerequisite for FPHL (Olsen et al., 2005). Nearly all men affected with AGA have normal circulating androgen levels. The predisposition to AGA is predominately due to genetic factors. The role of androgens in female AGA is less certain than in men and other factors may be involved (Chumlea et al., 2004). The diagnosis of AGA is normally made clinically, however aggravating associated factors and other diseases affecting scalp and hair growth need to be excluded (Blume-Peytavi et al., 2011).

Cardiovascular disease is the leading cause of death worldwide. Prevention and early detection of it are the major goal of health care (Günes et al., 2008). The main cause of heart disease and stroke is atherosclerosis which is a progressive disease characterized by the accumulation of lipids and ﬁbrous elements in the large arteries. Furthermore, it is considered to be the underlying cause of approximately half of all deaths in westernized countries (Lusis, 2000). Sharrett et al. (1999) stated that high levels of (TGs) and low levels of (HDL-C) are associated with the transition from atheroma to atherothrombosis.

Dyslipidemia has been studied as a valuable predictor of cardiovascular disease. For instance, increased LDL-c/HDL-c ratio has already been considered a sensitive predictor for CHD in men, and TC/HDL-C ratio has been found to be an even better predictive metabolic index for CHD risk in a large study (Lemieux et al., 200).

Furthermore, LP (a) has been shown as an important independent risk factor for CAD in many studies. Measurement of the LP (a) level has been recommended to determine the risk of myocardial infarction (Sathanur and Berenson, 1995 & Ari and Yigitoglu, 1997).

Increased level of serum LP(a) is said to be associated with endothelial dysfunction and CAD as it has a structure similar to LDL but is attached to a glycoprotein called apolipoprotein- a. It has sticky adhesive nature and can easily attach to LDL, calcium, and other components in an atherosclerotic plaque on the blood vessel wall. Moreover, due to its similarity with plasminogen, it competes with plasminogen for the binding with fibrin inhibiting its breakdown thus promotes blood clot formation. It also activates immune cells including monocytes and macrophages which help in inducing inflammation. These effects altogether help in inducing plaque formation and promote clot formation after the plaque is ruptured (Kamstrup et al., 2009).

The pathogenetic mechanisms of atherosclerosis are quite well known, however, the pathogenetic link between alopecia and atherosclerosis is not clear yet (Arias-Santiago., 2009). The pathogenic mechanism explaining the increase in cardiovascular risk in AGA patients may be attributed to the greater peripheral sensitivity of the receptors to androgens produced in AGA patients. The free testosterone is transformed by the action of 5αreductase into DHT. As the 5α-reductase and DHT receptors are present in blood vessels and heart as well as the hair follicles, this increased sensitivity leads eventually to follicular miniaturization at the follicular level and proliferation of vessel smooth muscle cells, a key phenomenon in atherosclerosis alongside lipid deposits, at the vascular level (AriasSantiago et al., 2010d).Cotton et al. (1972) was the first one to show an association between the occurrence of coronary artery disease and baldness, therefore he suggested that male androgenetic alopecia (MAGA) could be a risk factor for cardiovascular disease. However, few later studies have analyzed the relationship between AGA in women and cardiovascular disease and supported the hypothesis that AGA mainly in women < 55 years is associated with coronary artery disease (Matilainen et al., 2003 and Mansouri et al., 2005).

Thus the objective of this study was to evaluate lipid levels in women with AGA and in healthy controls to evaluate the possibility of cardiovascular risk factors in females with AGA.

There was a high statistically significant positive family history of cardiovascular disease in patients of the current study compared to healthy controls. On the contrary, Arias-Santiago et al. (2010d) found no significant differences between women with AGA and women without AGA as regard family history of cardiovascular disease.

Regarding lipid parameters, Female patients with AGA showed marked dyslipidemia when compared to healthy controls as they had significantly higher mean values of (TC) , (TG) , (LDL-C) ,TC:HDL-Cand LDL-C:HDL-C.

This was in agreement with the study done by (Arias-Santiago et al., 2010 d and a)who reported that women with AGA have higher significant mean values than non-alopecic women for TC (196.1 ± 33.6) P value(0,01) ,TG (123.8± 82.4), P value (0.00) LDL-C (114.1± 27.3) P value (0.00) , and LDL-C:HDL-C (2.1± 1)P value (0.00) and lower significant HDL-C (56.8 ± 14.7) P value (0.00) than healthy controls. However, as regard HDL-C, the present study showed a low but insignificant decrease of the mean value of HDL-C (43.1 ± 10.5) P value (0.65) in patients versus controls. Trevisan et al. (1993) showed that patients with frontal-occipital baldness had higher serum cholesterol and blood pressure on the average compared to participants of similar age with no baldness. (Sharma et al., 2013) showed that the TG (166, 79± 57.94) P value (<0.0001) and LDL (70.38± 16.34) P value (< 0.0001) levels in patients with AGA were significantly higher than controls as in our study.

On the other hand, Matilainen et al. (2003) could not report any significant differences in lipid profiles at all between women with AGA and women without AGA. This may be due to the fact that they didn’t exclude patients receiving hypocholesterolemic drugs as we did.

As regard to the LP (a) level, our patients showed high level of LP (a) but insignificant in comparison to the controls (28.1 ± 16.5) P value (0.07), although previous studies showed that LP (a) levels in patients with AGA were significantly higher than in controls (Farajzadeh et al., 2010 & Farajzadeh et al., 2011 & Sharma et al., 2013 and Mosbeh et al., 2014). The values were (26.01 ± 29.50) P value(0,002) , (47.10 ± 52.54) P value (0,001) , (27.37±6.117) P value(<0.00001) ( 48.10 ± 52.53) P value (0,001)respectively .That may be due to larger sample size in their studies, so further studies should be done on a larger number of GA patients to confirm the correlation.

In this study, there was a significant correlation between lipid parameters and Ludwig stage of AGA, and this is in contrary to the study done by Arias-Santiago et al., (2010 d and a) who reported that no significant differences were observed between Ludwig degree of AGA and lipid parameters. However, other studies done on MAGA showed that there was a correlation between the pattern of AGA and lipid parameters (Lesko et al 1993 and Rebora, 2001). Regarding BMI, the present study showed no statistically significant differences between cases and healthy controls. In addition, there was no statistically significant correlation between BMI and lipid parameters, and this was consistent with the study done by (Arias-Santiago et al., 2010 d). On the other hand, Matilainen et al. (2003) found higher mean BMI values in women with AGA in relation to controls.

Finally, lipid values may be regarded as a risk factor for cardiovascular disease and AGA could be considered an alarming sign for cardiovascular diseases. The unfavorable lipid profile in men and women with AGA could explain its association with CHD. Conclusion

Results of this study confirm that there is a true association between the high serum lipid levels and the female AGA but the pathogenetic link is still not clarified. This true association based on the presence of significant high values of TC, TG and LDL-C in females with AGA compared to age-matched healthy females.

Females with AGA could carry risk factor for cardiovascular disease due to their high risk of atherosclerosis more than healthy females as appeared from the measurement of the ratio of TC:HDL-C and LDL-C:HDL-C.

Therefore, serum lipid values especially LP(a) should be evaluated in women with AGA in order to detect risk of developing cardiovascular disease in patients with early-onset AGA and signal a potential opportunity for early preventive treatment

In conclusion, the results suggest that female AGA associated with dyslipidemia but the pathogenetic link could be still not clarified.

Further studies are, however, still recommended on a large scale of patients in a trial for finding the pathogenetic link between female AGA and dyslipidemia. Lipid values may be regarded as a risk factor for cardiovascular disease, and AGA could be considered an alarming sign for cardiovascular diseases.

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