

INVESTIGATIVE REPORT

A Comparative Study of Dyslipidaemia in Men and Women with Androgenic Alopecia

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Several studies have analyzed the relationship between androgenetic alopecia and cardiovascular disease (mainly heart disease). However few studies have analyzed lipid values in men and women separately. This case-control study included 300 patients consecutively admitted to an outpatient clinic, 150 with early onset androgenetic alopecia (80 males and 70 females) and 150 controls (80 males and 70 females) with other skin diseases. Female patients with androgenic alopecia showed significant higher triglycerides values (123.8 vs 89.43 mg/dl, $p=0.006$), total cholesterol values (196.1 vs 182.3 mg/dl, $p=0.014$), LDL-C values (114.1 vs 98.8 mg/dl, $p=0.0006$) and lower HDL-C values (56.8 vs 67.7 mg/dl, $p<0.0001$) versus controls respectively. Men with androgenic alopecia showed significant higher triglycerides values (159.7 vs 128.7 mg/dl, $p=0.04$) total cholesterol values (198.3 vs 181.4 mg/dl, $p=0.006$) and LDL-C values (124.3 vs 106.2, $p=0.0013$) versus non-alopecic men. A higher prevalence of dyslipidemia in women and men with androgenic alopecia has been found. The elevated lipid values in these patients may contribute, alongside other mechanisms, to the development of cardiovascular disease in patient with androgenic alopecia. **Key words:** male and female androgenic alopecia; cardiovascular risk factors; lipid levels; body mass index; weight.

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Androgenic alopecia (AGA) is the most common type of alopecia in both men and women, but its prevalence and pathogenic mechanisms have been investigated mainly in men. That male AGA could be a risk factor for coronary heart disease (CHD) was first suggested by Cotton et al. (1). Some subsequent studies, almost all conducted in male patients, appear to support this association (2–4). Only a few studies have analysed the relationship between AGA and CHD in women (5–6). Mansouri et al. (5) support the hypothesis that AGA is associated with CHD in women under the age of 55 years, according to angiographic studies.

The mechanism of the association of CHD and AGA has not been elucidated. The relationship between lipid profile and CHD has been analysed in several studies (7); however, few papers have analysed lipid values in men and women with AGA, which was the objective of this study.

SUBJECTS AND METHODS

Subjects

This case-control study included 300 patients, 150 with AGA (80 males and 70 females) and 150 controls (80 males and 70 females) consecutively admitted to the outpatient clinic (Dermatology Department, San Cecilio Hospital, Granada, Spain). The control group presented skin diseases other than alopecia. Diagnosis of AGA was based on family history and clinical findings, including: early age of onset (<35 years), pattern of increased hair thinning on frontal/parietal scalp with greater hair density on occipital scalp for males; retention of frontal hairline (in females); and the presence of miniaturized hairs and diversity of hair diameter (by dermoscopy). Detailed study of the consumption of drugs, iron intake and thyroid metabolism ruled out other causes of alopecia. The degree of AGA was determined by application of Ludwig scales (II–III) and Ebling scales (III–V). Inclusion criteria were: men and women; aged 35–60 years; presence of early-onset AGA (age <35 years) with Ebling degree III or above for males (vertex and frontal alopecia); and Ludwig degree II or above for females; and signing of informed consent to study participation. Exclusion criteria were: receipt of hormone replacement therapy with testosterone or corticoids for >1 month (chronic corticoid therapy). Inclusion criteria for controls were: males or females with other dermatological diseases (mainly naevi, seborrheic keratosis, actinic keratosis and basal cell carcinoma); aged 35–60 years; and signing of informed consent to study participation. Exclusion criteria were as described above and the presence of AGA.

Clinical parameters and measurements of lipid levels

Serum total cholesterol (C), triglycerides, high-density lipoprotein (HDL)-C and low-density lipoprotein (LDL)-C levels were studied in samples drawn between 08.00 h and 09.00 h after a 12-h fasting period. Also, total-C/HDL-C and LDL-C/HDL-C ratios were calculated. Data were also gathered on age, weight, height and body mass index (BMI = kg/m²) smoking, alcohol consumption, sedentarism, personal or family history of cardiovascular disease. Dyslipidaemia was diagnosed if triglycerides were >150 mg/dl, total C was >200 mg/dl or LDL-C was >130 mg/dl, or if treatment for dyslipidaemia was ongoing.

Statistical analyses

Statistical analyses were performed with the SPSS/PC software (Version 15.0 for windows SPSS Inc, Chicago, IL, USA).

Table I. Mean (SD) values for biometrics and lipid levels age in men and women with androgenic alopecia (AGA) and their respectively controls (no AGA). p-values of the t-test

	Men		p-value	Women		p-value
	AGA (n=80)	No AGA (n=80)		AGA (n=70)	No AGA (n=70)	
Age (years)	47.1 (8.4)	45.3 (9.2)	0.19	48.2 (9.1)	47.9 (8.1)	0.83
Weight (kg)	84.1 (13.4)	82.2 (10.8)	0.32	72.4 (13.7)	68.9 (13.1)	0.12
Height (cm)	173.7 (7.9)	174.8 (8.1)	0.38	162.1 (6.3)	163.7 (7.2)	0.16
BMI (kg/m ²)	27.87 (4.3)	26.95 (3.9)	0.15	27.6 (5.9)	25.9 (5.6)	0.082
Triglycerides (mg/dl)	159.7 (98)	128.7 (97.7)	0.04	123.8 (82.4)	89.43 (63.9)	0.0067
HDL-C (mg/dl)	48.7 (14.2)	48.9 (15.2)	0.93	56.8 (14.7)	67.7(14.1)	<0.0001
LDL-C (mg/dl)	124.3 (34)	106.2 (35.7)	0.0013	114.1 (27.3)	98.8 (24.1)	0.0006
Total cholesterol (mg/dl)	198.3(37.2)	181.4 (39.8)	0.0062	196.1 (33.6)	182.3 (32.1)	0.014
Total cholesterol/HDL	4.07 (1.1)	3.70 (1.2)	0.043	3.45 (1.2)	2.69 (0.64)	<0.0001
LDL-C/HDL-C	2.55(0.95)	2.17(0.97)	0.013	2.1 (1)	1.45 (0.4)	<0.0001

LDL-C: low-density lipoprotein-cholesterol; HDL-C: high-density lipoprotein-cholesterol; BMI: body mass index.

Student's *t*-test was used to compare mean values of quantitative variables, and the Levene test to study the variance. Qualitative variables were analysed with χ^2 test. Correlations among variables were studied using the Pearson's coefficient. $p \leq 0.05$ was considered significant in all analyses.

RESULTS

A total of 150 Caucasian patients with AGA were studied (70 women: 58% II Ludwig degree and 42% III, and 80 men: 29% Ebling degree III, 41% IV and 30% V). Mean age was 47.7 years (SD 9.3) in the AGA patient group and 46.6 years (SD 8.7) in the sex-matched control group ($p=0.31$). Mean time since alopecia onset was 16.7 years (16.2 years for women and 17.1 years for men; $p=0.22$). No significant differences were found between AGA patients and controls with respect to tobacco or alcohol consumption, sedentarism, personal or familiar history of CHD. No AGA patient or control had family hyperlipidaemia or treatment for hyperlipidaemia.

Mean age, weight, height and BMI values are summarized in Table I for men and women with AGA and controls. Men with AGA presented higher significant mean weight (84.1 vs. 72.4 kg; $p < 0.001$) and height (173.7 vs. 162.1 m; $p < 0.0001$) than women with AGA. Men with AGA presented higher significant values in cholesterol total/HDL-C, LDL-C/HDL-C, triglyceridaemia, LDL-C and total cholesterol than non alopecic men (Table I). No significant differences

were observed between Ebling degree (III–V) of AGA and lipid parameters.

Women with AGA showed higher significant mean values than non-alopecic women for all the parameters (triglyceridaemia, LDL-C, total cholesterol, total cholesterol/HDL-C and LDL-C/HDL-C; Table II) and lower significant HDL-C than controls. No significant differences were observed between Ludwig degree (II–III) of AGA in lipid parameters. Men and women with AGA presented significantly higher prevalence of dyslipidaemia than controls (Table II).

Comparing lipid profile in men and women with AGA, we found significant differences in HDL-C, higher for alopecic women (48.7 vs. 56.8 mg/dl; $p=0.0009$). Men with AGA presented significantly higher triglyceridemia mean values (159.7 vs. 123.8 mg/dl; $p=0.01$), LDL-C values (124.3 vs. 114.1 mg/dl; $p=0.04$), total cholesterol/HDL-C (4.07 vs. 3.45; $p=0.0012$) and LDL-C/HDL-C (2.55 vs. 2.1; $p=0.005$) than women with AGA. There was no significant correlation between all these lipids parameters and weight between men and women with AGA except for HDL-C in alopecic men ($r=-0.48$, $p < 0.001$).

DISCUSSION

This study found significantly higher lipid levels in men and women with AGA than healthy controls.

Sadighha et al. (8) studied the lipid profile of men with AGA, and found significantly higher levels of triglyceride and total cholesterol/HDL-cholesterol ratio in men with AGA and significantly lower HDL-C values. However, in our study we did not find significant differences in HDL-C values between men with AGA and the control group. Guzzo et al. (9) evaluated lipid levels in men with Ebling degree III compared with randomly obtained serum lipid profiles of age-matched men referring to the reference laboratory. Although no statistically significant difference was measured in lipid indices between AGA patients and controls, their finding

Table II. Prevalence of dyslipidaemia in men and women with androgenic alopecia (AGA) and their respectively controls. p-values of the χ^2 test and odds ratio with 95% confidence interval

Gender		Dyslipidaemia	χ^2 test	OR (95% CI)
		(%)	(p-value)	
Men (n=80)	AGA	66.2	<0.0001	4.58 (2.35–8.91)
	No AGA	30		
Women (n=70)	AGA	55.7	0.0006	3.63 (1.77–7.42)
	No AGA	25.7		

OR: odds ratio; CI: confidence interval.

is questioned because of confounding factors affecting serum lipid profiles (secondary hyperlipidaemia and familial hyperlipidaemia) were ignored.

Few studies have analysed the relationship between AGA in women and cardiovascular disease. Matilainen et al. (10) support the hypothesis that women with some markers of insulin resistance have significantly increased risk for female AGA, but they did not find significant differences in lipid profiles. Giltay et al. (11) analysed 81 female-to-male transsexual subjects treated with testosterone esters and 38.3% developed AGA; however, no differences in lipid values or weight were found.

The pathogenetic mechanisms of atherosclerosis are quite well known, but the pathogenetic link between alopecia and atherosclerosis is not clear (12, 13). Higher cholesterol- and triglyceride-levels participate along with other mechanisms initiating atheromatous plaque. HDL-C on the other hand protects the vascular wall from aggressive factors (endothelial adhesion, migration of monocytes, etc.) and facilitates the reverse transport of cholesterol. The unfavourable lipid profile in men and women with AGA could explain its association with CHD. In addition, Matilainen et al. (13) found higher LDL-C and triglycerides in men with AGA in patients who had undergone coronary artery bypass graft or percutaneous transluminal coronary angioplasty.

Increased LDL-cholesterol/HDL-cholesterol ratio has already been considered a sensitive predictor for CHD in men, and total cholesterol/HDL-C ratio has been found to be an even better predictive metabolic index for CHD risk in a large study (14). Sharrett et al. (15) stated that high levels of triglycerides and low levels of HDL-C are associated with the transition from atheroma to atherothrombosis. Therefore, control of these two cardiovascular risk factors is essential in patients with subclinical disease.

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