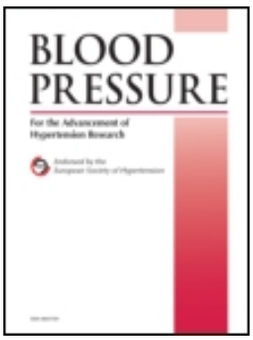

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ORIGINAL ARTICLE

Androgenetic alopecia as an indicator of metabolic syndrome and cardiovascular risk

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ABSTRACT

Numerous studies have investigated a probable association between androgenetic alopecia (AGA) and cardiovascular disease (CVD) by researching limited and dispersed parameters. We aimed to evaluate both traditional and non-traditional cardiovascular risk factors in male patients with early-onset AGA. This case–control study included 68 participants: 51 male patients with early-onset AGA and 17 healthy male controls. Patients with AGA were classified into three groups according to the Hamilton–Norwood scale and the presence of vertex hair loss. Traditional and non-traditional cardiovascular risk factors were examined in all study subjects. Metabolic syndrome was diagnosed in 25 patients with AGA and in two control subjects ($p < 0.05$). The carotid intima–media thickness values were found to be significantly higher in patients with vertex pattern AGA than in patients without vertex baldness and controls ($p < 0.05$). The pulse-wave velocity values were also found to be significantly higher in patients ($p < 0.001$). A limitation of this study was the small study population. In conclusion, vertex pattern AGA appears to be a marker for early atherosclerosis. This finding supports the hypothesis that early-onset AGA alone could be an independent risk factor for CVD and metabolic syndrome.

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Androgenetic alopecia, arterial stiffness, cardiovascular risk factors, carotid intima–media thickness, insulin resistance, metabolic syndrome

Introduction

Androgenetic alopecia (AGA), which is also known as male-pattern baldness, is a common type of hair loss that most frequently occurs in males.[1,2] Androgens and in particular dihydrotestosterone, which is the metabolite of testosterone, have an important role in the development of AGA in males.[3] Numerous diseases including cardiovascular disease (CVD), hypertension, benign prostate hyperplasia and prostate cancer have been reported to have concurrency with AGA. The relation between early-onset AGA and enhanced cardiovascular risk has been reported in many epidemiological studies. These studies demonstrated a relation among obesity, hypercholesterolaemia/dyslipidaemia, impaired insulin sensitivity and vascular pathology-related medical conditions in young adults, such as myocardial infarction (MI), coronary artery disease (CAD) and AGA.[4–11] All these concurrencies appear to be the consequence of vascular/endothelial impairment in patients with AGA. High-sensitivity C-reactive protein (hs-CRP) has been shown to be independently associated with MI, stroke and sudden death.[12–14] Metabolic syndrome (MetS) is a clinical state associated with insulin resistance (IR) and cardiovascular risk factors such as hypertension, obesity and lipid failure.[15] The use of carotid–intima media thickness (CIMT) measurement as a new method to determine atherosclerosis [16] has demonstrated that the annual incidence of stroke and CAD increases as CIMT increases; the risk of MI is enhanced and there is a positive correlation between CIMT and CAD.[17,18] The aortic pulse-wave velocity (PWV) method, which is a non-invasive, simple, cheap and reproducible method used in the measurement of stiffness of the great arteries and considered as the gold standard for the assessment of arterial stiffness, is also an independent determinant for the risk of cardiovascular morbidity and mortality. PWV is also used as a marker for endothelial dysfunction.[19–24]

This prospective case–control study aimed to analyse traditional cardiovascular risk in male patients with AGA by comparing new cardiovascular risk factors including IR, MetS, hormone profile, PWV measurement, CRP and CIMT with the control group.

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Patients and methods

The study was carried out at the Department of Dermatology at Erciyes University. The study protocol was reviewed and approved by the local ethics committee and the study was approved by Erciyes University Research Projects unit (project no. TSI-11-3469).

A total of 60 male patients with early-onset AGA, aged between 18 and 55 years, who agreed to participate in the study and met the eligibility and exclusion criteria, were enrolled in the study. We determined the age limit for early-onset AGA to be 30 years and earlier.[25]

The patients were divided into three groups, which were classified according to the Hamilton–Norwood scale. A control group comprising 20 healthy male subjects was created (Figure 1). In addition, another two groups were created according to hair loss in the vertex in both patients and healthy controls.[5]

We examined the presence of traditional and non-traditional cardiovascular risk factors in male patients with early-onset AGA and healthy controls. According to the primary study objectives, we examined the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), MetS parameters, CIMT and PWV. We accepted HOMA-IR formulation values $\geq$2.7 as IR.[26] Individuals from the control and patient groups were diagnosed with MetS according to the Metabolic Syndrome Guidelines of the Society of Endocrinology and Metabolism of Turkey, which meet the 2005 parameters of the International Diabetes Federation (IDF).[25,26]

All participants underwent arterial blood pressure measurement, baseline transthoracic echocardiography, CIMT and PWV measurements in the Department of Cardiology Hospital. For the measurement of carotid–femoral PWV, sequential pulse-wave recordings were taken first from the carotid area and then from the femoral area using an electrocardiography device.

As secondary study parameters, information on identity, age, smoking status, sedentary lifestyle, alcohol consumption, drug use, diabetes, hypertension, dyslipidaemia, family history of CAD and presence of AGA in first degree relatives was obtained during initial admission using the standard form. The body mass index and the waist/hip ratio were calculated at the first visit.

Laboratory procedures were performed at the Biochemical, Serology, Hormone and Nuclear Medicine Laboratories. Fasting glucose, cholesterol, high-density lipoprotein, low-density lipoprotein, triglyceride, insulin, hs-CRP, dehydroepiandrosterone sulfate (DHEA-S), sex hormone-binding globulin (SHBG) and free testosterone levels were measured. The HOMA-IR formulation = [Fasting plasma glucose (mg/dl) × Fasting plasma insulin (μIU/ml)]/405) was used to determine the IR.

The suitability of data for the normal distribution was assessed by the Shapiro–Wilk test. Between-group

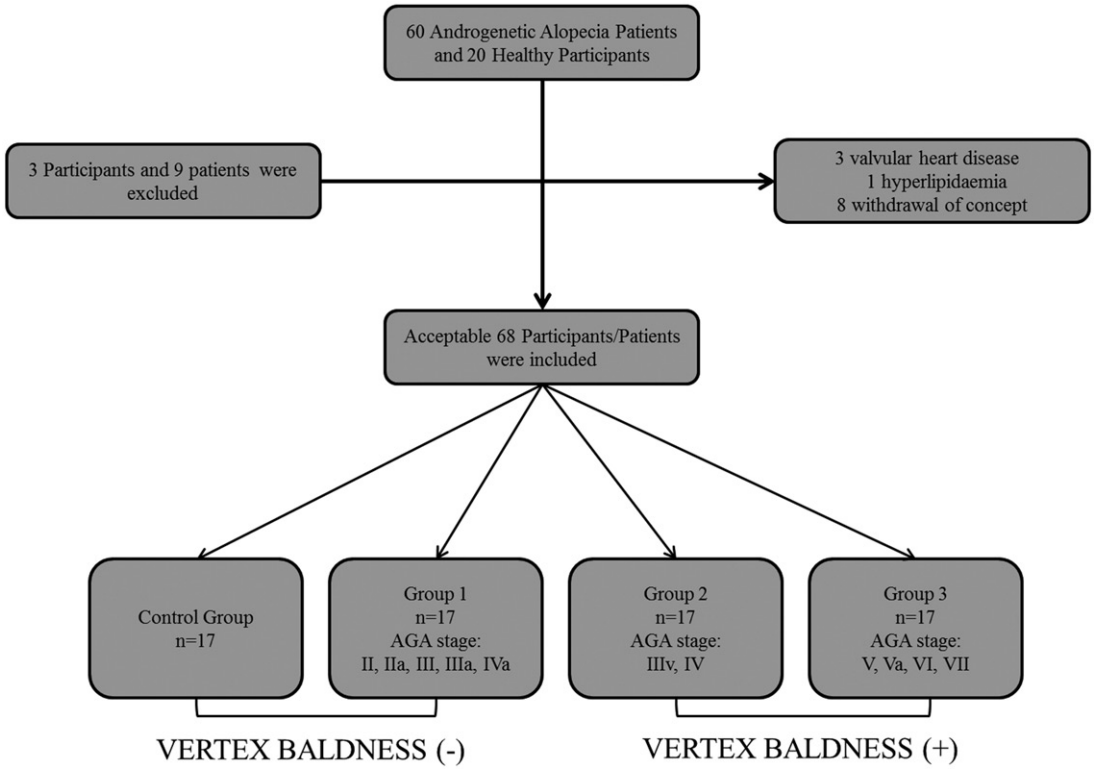

Figure 1. Study sample. AGA, androgenetic alopecia.

comparisons were made using the chi-squared test for categorical variables, and the Kruskal–Wallis *H* test and one-way analysis of variance for quantitative variables; the Tukey test was used as a *post hoc* test. The data are presented as frequency and percentage, mean and standard deviation, or median and 25th and 75th percentiles. Analysis of data was conducted using the SigmaPlot 12.0 program and $p < 0.05$ was considered to be the level of significance.

Results

There were no statistically significant differences between the control group and the patient groups (defined in Figure 1) in terms of age, gender, biochemical measures, hormone profiles and traditional cardiovascular risk factors, as shown in Tables 1–3.

The HOMA-IR value was above 2.7 in one patient in the control group, three patients in Group 1, four patients in Group 2 and five patients in Group 3. No statistically significant difference was determined in the comparison of MetS parameters (Table 3). The number of patients with MetS was lower in the control group than in the patient groups ($p < 0.05$) but there was no difference among the patient groups (Table 4). No significant difference was found among any of the groups in terms of other transthoracic echocardiographic parameters and CRP levels (Tables 5 and 6).

The CIMT in all groups is shown in Figure 2(B), with no significant difference determined between the control

Table 1. Traditional cardiovascular risk factors.

	Control	Group 1	Group 2	Group 3	*p*
Age (years)	29.41 ± 6.82	30.24 ± 9.30	29.35 ± 8.22	34.35 ± 6.78	0.208
Weight (kg)	78.24 ± 11.23	79.06 ± 12.82	76.59 ± 15.03	81.94 ± 11.72	0.669
Height (cm)	176.53 ± 6.74	175.18 ± 7.66	174.29 ± 5.08	174.12 ± 4.78	0.656
BMI (kg/m^2)	25.01 ± 3.28	25.69 ± 3.38	25.10 ± 4.70	27.02 ± 3.55	0.385
Waist circumference (cm)	97 (87.25–103.75)	98 (92.5–105.25)	95 (84.50–102.50)	98 (94.75–106.00)	0.630
Hip circumference (cm)	103 (98.0–110.5)	103 (98.0–110.5)	100 (95.5–108.0)	105 (103.0–112.0)	0.090
Sedentary lifestyle (+/–)	0/17 (0.0%/100%)	5/12 (29.4%/70.6%)	3/14 (17.6%/82.4%)	2/15 (11.8%/88.2%)	0.107
Smoking (+/–)	8/9 (47.1%/52.9%)	8/9 (47.1%/52.9%)	7/10 (41.2%/58.8%)	6/11 (35.3%/64.7%)	0.882
Alcohol consumption (+/–)	1/16 (5.9/94.1)	2/15 (11.8/88.2)	0/17 (0.0/100.0)	2/15 (11.8/88.2)	0.498
Family history of heart disease (+/–)	2/15 (11.8/88.2)	8/9 (47.1/52.9)	8/9 (47.1/52.9)	4/13 (23.5/76.5)	0.064
Family history of AGA (+/–)	11/6 (64.7/35.3)	9/8 (52.9/47.1)	13/4 (76.5/23.5)	15/2 (88.2/11.8)	0.129

Data are presented as mean ± SD, median (25th–75th percentiles) or *n* (%)
Group 1: stage II, IIa, III, IIIa and IVa androgenetic alopecia (AGA); Group 2: stage III, vertex and stage IV AGA; Group 3: stage V, Va, VI and VII AGA.
BMI: body mass index.

Table 2. Hormone, lipid and insulin values.

	Control	Group 1	Group 2	Group 3	*p*
fT (pg/ml)	17.06 ± 4.38	16.50 ± 4.62	18.34 ± 5.42	14.77 ± 483	0.199
DHEA-S (ng/ml)	3117 (2231–4349)	3072 (2499.5–3564)	3154 (2764–4323)	2789 (2057–4528)	0.845
SHBG (nmol/l)	20 (17.0–27.5)	18 (13.0–25.5)	24 (17.0–33.5)	21 (15.0–29.5)	0.311
LDL (mg/dl)	103.92 ± 26.39	95.52 ± 28.47	101.29 ± 31.61	112.89 ± 43.63	0.495
Cholesterol (mg/dl)	167.06 ± 32.87	170.88 ± 28.94	177.94 ± 40.43	173.77 ± 39.78	0.840
Insulin (mU/l)	5.87 (2.97–8.24)	7.65 (4.08–11.67)	6.24 (4.85–10.85)	7.09 (3.95–13.84)	0.608

Data are presented as mean ± SD or median (25th–75th percentiles).
Group 1: stage II, IIa, III, IIIa and IVa androgenetic alopecia (AGA); Group 2: stage III, vertex and stage IV AGA; Group 3: stage V, Va, VI and VII AGA.
fT: free testosterone; DHEA-S: dehydroepiandrosterone sulfate; SHBG: sex hormone-binding globulin; LDL: low-density lipoprotein.

Table 3. Comparison of data according to metabolic syndrome criteria.

	Control	Group 1	Group 2	Group 3	*p*
Glucose (mg/dl)	80.06 ± 9.21	79.06 ± 13.68	79.88 ± 12.91	85.41 ± 9.32	0.359
Triglyceride (mg/dl)	108 (71.5–147.5)	115 (67.0–201.0)	119 (80.0–161.5)	129 (81.0–184.5)	0.713
HDL (mg/dl)	41.18 ± 7.13	43.55 ± 8.83	38.7 ± 12.29	38.88 ± 6.95	0.371
HOMA-IR	1.20 (0.59–1.80)	1.55 (0.82–2.31)	1.19 (0.83–2.20)	1.39 (0.81–3.06)	0.678
Abdominal obesity/waist circumference	97 (87.25–103.75)	98 (92.5–105.25)	9 5 (84.50–102.50)	9 8 (94.75–106.00)	0.630
SBP (mmHg)	123.29 ± 13.82	126.18 ± 13.03	129.0 ± 12.74	128.0 ± 3.90	0.468
DBP (mmHg)	74.41 ± 11.34	74.59 ± 10.58	78.24 ± 11.91	71.65 ± 14.05	0.468

Data are presented as mean ± SD or median (25th–75th percentiles).
Group 1: stage II, IIa, III, IIIa and IVa androgenetic alopecia (AGA); Group 2: stage III, vertex and stage IV AGA; Group 3: stage V, Va, VI and VII AGA.
HDL: high-density lipoprotein; HOMA-IR: Homeostasis Model Assessment of Insulin Resistance; SBP: systolic blood pressure; DBP: diastolic blood pressure.

and patient groups (Table 6). However, with regard to the patients with hair loss in the vertex, values were higher in these patients than in both the other patients and the controls ($p < 0.05$) (Table 7). Statistical analysis of the data revealed that PWV was low in the control group, whereas it was high in the patient groups with AGA ($p < 0.001$) (Table 7) (Figure 2A). When all study participants were separated into those with and without hair loss in the vertex, PWV was found to be significantly higher in the group with hair loss in the vertex ($p < 0.001$) (Table 7).

Discussion

Numerous previous studies in patients with AGA have evaluated the risk of mortality due to MI or for cardiac reasons, and the cardiovascular risk factors.[4–11] It remains unclear why the prevalence of CAD is high in males with AGA. In the present study, these parameters showed a homogeneous distribution among control and all patient groups. One of the important differences between the studies is the selection of age at the onset. Mumcuoğlu et al. [25] also took the age limit for early onset as 30 years in their study carried out in 2011, in which they compared male patients aged between 18 and 30 years with early-onset AGA and an age-matched control group in terms of the presence of IR.

Androgen metabolism is the most frequently suspected mechanism for the association between CVD and AGA in males. Dihydrotestosterone is transformed from testosterone, of which only a small proportion circulates freely in the plasma, by the 5α-reductase enzyme; it is five times more potent than testosterone, and is the main androgen responsible for the miniaturization of hair follicles encountered in AGA.[27,28] The 5α-reductase enzyme, which is found in the muscular layer of the heart and cardiac vessels, transforms testosterone into dihydrotestosterone and leads to proliferation and thickening in the vascular smooth muscle layer.[29] Within this context, it is thought that androgens cause AGA in hairy skin and may lead to cardiovascular events by the same mechanism in the heart as the result of increased activity both in hairy skin and in the cardiac vessels.

It has been demonstrated that there is no relation between the severity of alopecia and serum androgen concentrations.[30] In support of these findings, hair loss, which is generally encountered in both males and females, is considered to result from the abnormal sensitivity of hair follicles to the circulating androgens in genetically susceptible individuals.[27] In line with the literature, the present study found no statistically significant difference between the control and patient groups in terms of free testosterone, DHEA-S and SHBG hormone concentrations.

A 2004 study showed an association between AGA and polycystic ovary syndrome (PCOS).[31]

Table 4. Patients with metabolic syndrome and insulin resistance.

		Control	Group 1	Group 2	Group 3	*p*
Metabolic syndrome	+	2 (11.8)	8 (47.1)	6 (35.3)	11 (64.7)	0.015
	–	15 (88.2)	9 (52.9)	11 (64.7)	6 (35.3)	
Insulin resistance	+	1 (5.9)	3 (17.6)	4 (23.5)	5 (29.4)	0.344
	–	16 (94.1)	14 (82.4)	13 (76.5)	12 (70.6)	

Data are presented as *n* (%).
Group 1: stage II, IIa, III, IIIa and IVa androgenetic alopecia (AGA); Group 2: stage III, vertex and stage IV AGA; Group 3: stage V, Va, VI and VII AGA.

Table 5. Transthoracic echocardiography parameters.

	Control	Group 1	Group 2	Group 3	*p*
LVSD (mm)	30.53 ± 2.70	31.65 ± 2.32	29.24 ± 2.49	30.94 ± 3.63	0.099
LVDD (mm)	48 (46.0–52.0)	48 (47.0–51.5)	49 (47.0–50.5)	49 (47.0–52.0)	0.968
IVS (mm)	9.7 ± 1.2	9.4 ± 1.3	9.4 ± 1.1	9.9 ± 1.4	0.502
PD (mm)	10 (9.0–10.5)	9.0 (9.0–10.5)	10 (10–10)	10 (10–10)	0.368
EF (%)	65.18 ± 3.25	64.65 ± 4.15	68.24 ± 3.85	65.06 ± 3.86	0.028

Data are presented as mean ± SD or median (25th–75th percentiles).
Group 1: stage II, IIa, III, IIIa and IVa androgenetic alopecia (AGA); Group 2: stage III, vertex and stage IV AGA; Group 3: stage V, Va, VI and VII AGA.
LVSD: left ventricle systolic diameter; LVDD: left ventricle diastolic diameter; IVS: interventricular septum thickness; PW: posterior wall thickness; EF: ejection fraction.

Table 6. Comparison of new cardiovascular risk factors.

	Control	Group 1	Group 2	Group 3	*p*
hs-CRP (mg/l)	3.34 (3.34–3.34)	3.34 (3.08–3.41)	3.08 (3.08–3.34)	3.34 (3.08–3.41)	0.161
CIMT (mm)	0.52 (0.46–0.58)	0.52 (0.48–0.63)	0.59 (0.47–0.69)	0.62 (0.56–0.72)	0.111
PWV (m/s)	5.90 (5.35–6.85)	7.60 (6.65–8.60)	7.60 (6.60–8.95)	8.20 (7.40–10.50)	< 0.001

Data are presented as median (25th–75th percentiles).
Group 1: stage II, IIa, III, IIIa and IVa androgenetic alopecia (AGA); Group 2: stage III, vertex and stage IV; Group 3: stage V, Va, VI and VII AGA.
hs-CRP: high-sensitivity C-reactive protein; CIMT: carotid intima–media thickness; PWV: pulse-wave velocity.

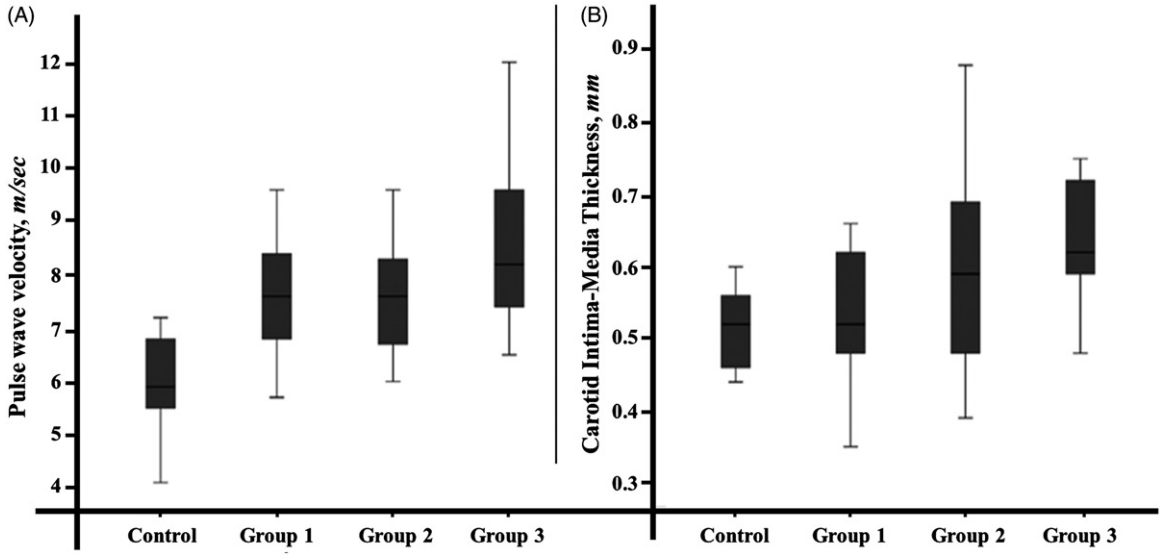

Figure 2. (A) Distribution of pulse-wave velocity values; (B) distribution of carotid intima–media thickness values.

Table 7. Evaluation of some parameters according to vertex baldness.

	Vertex baldness		
	–	+	*p*
Age	29.82 ± 8.04	31.85 ± 7.84	0.296
Waist circumference (cm)	97.00 (88.00–105.25)	97.00 (91.00–104.25)	0.792
BMI (kg/m^2)	25.35 ± 3.30	26.06 ± 4.22	0.440
Triglyceride (mg/dl)	108.5 (70.0–161.0)	120.0 (85.0–163.0)	0.443
HDL (mg/dl)	42.38 ± 7.99	38.82 ± 9.83	0.108
HOMA-IR	1.31 (0.71–2.00)	1.28 (0.82–2.71)	0.465
fT (pg/ml)	16.78 ± 4.44	16.55 ± 5.37	0.851
DHEA-S (ng/ml)	3116 (2485.75–3728.25)	3074 (2201.75–4281.00)	0.980
SHBG (nmol/l)	19.50 (15.75–26.00)	23.00 (15.00–32.50)	0.220
SBP (mmHg)	124.94 ± 13.27	124.91 ± 13.57	0.993
DBP (mmHg)	74.50 ± 10.80	74.94 ± 13.26	0.881
hs-CRP (mg/l)	3.34 (3.08–3.41)	3.09 (3.08–3.36)	0.104
CIMT (mm)	0.52 (0.47–0.61)	0.60 (0.48–0.71)	0.043
PWV (m/s)	6.80 (5.7–7.7)	7.90 (7.05–9.60)	< 0.001

Data are presented as mean ± SD or median (25th–75th percentiles).
Vertex baldness (–): Control group and Group 1 [stage II, IIa, III, IIIa and IVa androgenetic alopecia (AGA)]; Vertex baldness (+): Group 2 (stage III, vertex and stage IV AGA) and Group 3 (stage V, Va, VI and VII AGA).
BMI: body mass index; HDL: high-density lipoprotein; HOMA-IR: Homeostasis Model Assessment of Insulin Resistance; fT: free testosterone; DHEA-S: dehydroepiandrosterone sulfate; SHBG: sex hormone-binding globulin; SBP: systolic blood pressure; DBP: diastolic blood pressure; hs-CRP: high-sensitivity C-reactive protein; CIMT: carotid intima–media thickness; PWV: pulse-wave velocity.

Starka et al. revealed that men with early-onset alopecia have a hormone profile similar to women with PCOS.[32] Therefore, the idea that "early-onset AGA in men is the male phenotype of PCOS" is becoming important. This idea is supported by a study performed by Matilainen et al., which showed that when the male group with early-onset AGA between the ages of 19 and 50 years was compared to the control group, dyslipidaemia, hypertension, obesity, hyperinsulinaemia, IR and disorders associated with MetS were higher in the patient group.[6]

In a 2011 study, it was shown that significantly higher HOMA-IR and fasting IR index were present in patients with early-onset AGA.[33] It was suggested that the reason for increased cardiovascular risk in AGA patients is IR and endothelial dysfunction caused by defective MetS parameters. In our study, no statistically significant difference was found in the patient and control groups for IR, which is shown as one of the parameters of MetS, or for the HOMA-IR value, which is accepted as the indicator of IR.[34] Thus, the number of patients with IR is increased in direct proportion to the grade of alopecia. The small study population is a limitation of this study.

In previous studies performed in the general population, the prevalence of MetS was found to be between 7.5% and 20%.[35,36] A strong relation between MetS

and CVD was shown by Lehto et al.[37] Trevisan et al. noted a relation between AGA in young patients and high cholesterol and blood pressure levels, which are the diagnostic criteria of MetS, and they stated that this relation declined with increasing age.[38] In 2010, Arias-Santiago et al. showed that lipid levels were higher in the patient group, and the number of patients diagnosed with MetS according to Adult Treatment Panel-III MetS criteria was higher in the AGA group than in the control group.[39] In our study, there was no statistically significant difference for MetS parameters. Recruitment of young patients and the similarity of mean age between the patient groups and control group may be suggested as the cause of this difference. We used the last issue of IDF MetS criteria (2005), which classified abdominal obesity according to ethnic groups, for all of the recruited patients including the control group.[25,26] The number of patients diagnosed with MetS was significantly higher in the patient groups with AGA in our study. In line with the literature, in the control group, the proportion of patients with MetS was 11.8%.

Among acute-phase reactants, the coexistence of hs-CRP, which is considered as a marker of microinflammation, and increased cardiovascular risk has been demonstrated.[40] Hirsso et al. reported that hs-CRP values increased in line with the waist/hip ratio in patients with moderate and severe AGA.[10] Arias-Santiago et al. compared AGA patients with the control group and did not find a significant difference in hs-CRP values.[39] In our study, no significant difference was found between the control and patient groups in terms of hs-CRP, waist, hip and weight measurements.

The relation between CIMT and AGA has been demonstrated in only a few studies. Among these studies, Shahar et al. evaluated a total of 5056 patients aged between 52 and 75 years, of whom 767 had a history of past MI, in terms of the presence and degree of baldness, presence of vertex baldness, having had MI and CIMT, but found no relation between them.[41] They compared the patients with and without a history of MI; however, those who died of MI or other CVD or those with asymptomatic coronary disease in the control group were not taken into consideration.

It has been demonstrated that vertex baldness is a risk factor for increased CVD, particularly in the presence of hypertension and high serum cholesterol.[5] In a recent study, with regard to the CIMT values of the four groups, a significant difference was found only between the control group and the patient group with serious vertex baldness. The CIMT values were found to be significantly higher in patients with vertex pattern AGA than in patients without vertex baldness and controls. No difference was found when the control group was compared with the other two AGA groups.[42]

In our study, likewise, no difference was determined among the control group and three patient groups in terms of CIMT values. However, consistent with the literature, the difference between CIMT values was significant when all participants were divided into two groups, comprising those with and without vertex hair loss. The basic reason for this difference was thought to be the difference between the methodologies used in grouping the patients. Vertex baldness could be suggested as an indicator of a risk factor for atherosclerosis, as an increase in CIMT values indicates premature atherosclerosis.

PWV measurement, which is the gold standard method in assessing arterial stiffness, is also used as a marker for endothelial dysfunction. The present study is unique in the literature in terms of the method used. In addition, various cardiovascular risk factors such as hypertension, diabetes, hyperlipidaemia, smoking, sedentary lifestyle, obesity and MetS, CAD and heart failure, which cause endothelial dysfunction, lead to arterial stiffness. Measurement of carotid–femoral PWV, which has been demonstrated in many studies as an independent risk factor for CVD, is a direct measurement and increased values in young patients may predict CAD. In addition, it was determined that increased arterial stiffness is associated with extensiveness of atherosclerotic disease and the risk of CVD in patients with essential hypertension.[43,44] In the present study, we also demonstrated the difference between the PWV values of the healthy control group and the patient group, which had no difference in terms of traditional CVD risk factors. PWV values were significantly lower in the control group. Moreover, PWV values were significantly higher in the patients with vertex hair loss when all study patients were separated into those with and without hair loss in the vertex. The very high PWV values found in the present study may explain the increased risk for CVD, which has not been clearly defined before, in these patients.

The present study found MetS and CIMT values to be significantly risky in cardiovascular terms. Nevertheless, as the arterial stiffness value was found to be more correlated in young patient groups and in patients with early-onset AGA, it is extremely difficult to explain this independently from MetS alone or other pathophysiological conditions that have been mentioned before.

In conclusion, the present study demonstrates that being an AGA patient alone and independently is a risk factor for CVD and MetS. The risk is particularly higher in those with hair loss in the vertex. The risk is not directly correlated with any laboratory or parametric

measures. Therefore, more comprehensive studies with a longer follow-up period are needed.

Declaration of interest

No potential conflict of interest was reported by the authors.

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