Original Article

Androgenetic Alopecia: Traditional Cardiovascular Risk Factors, Metabolic Syndrome, and Component Traits among Nigerian Adults

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Abstract

Background: Androgenetic alopecia (AGA) has been linked to cardiovascular diseases (CVDs) and metabolic syndrome (MetS). Works on AGA, cardiovascular risk factors (CVRFs) and MetS are rare among Nigerians. **Aim:** This study set out to determine the relationship among CVRFs, MetS, and AGA. **Subjects and Methods:** This is a cross-sectional study done among adults who were 18 years and above in selected communities in Ogbomoso on 260 consenting AGA participants as well as 260 age controls without AGA. They were matched for age and sex using a multistage sampling method. Anthropometric measurements, fasting blood glucose, and lipid profile samples were collected. MetS was diagnosed using International Diabetes Federation criteria. Data were analyzed using IBM SPSS version 20. Ethical approval was gotten before commencement of the study (LTH/OGB/EC/2017/162). **Result:** Metabolic syndrome in AGA was higher than in controls (8.08% vs. 7.69%, $p = 0.742$). AGA was significantly associated with elevated mean systolic blood pressure (SBP) ($p = 0.008$), low High Density Lipoprotein (HDL-c) ($p < 0.001$), alcohol intake ($p < 0.001$), dyslipidaemia ($p = 0.002$), and sedentary lifestyle ($p = 0.010$). The correlates of AGA severity in male and female gender are age ($p < 0.001$ and 0.009 respectively), SBP ($p = 0.024$) and abdominal obesity ($p = 0.027$) in male gender. **Conclusion:** AGA in Nigerians is associated with dyslipidaemia, alcohol intake, and sedentary lifestyle. AGA severity is related to age, higher mean SBP, abdominal obesity and low HDL-c in male and age, and Body mass index in females. Nigerians with AGA should be screened for dyslipidaemia and counseled against the use of alcohol and sedentary lifestyle.

Keywords: *Androgenetic alopecia, cardiovascular risk factors, metabolic syndrome, Nigerians*

Introduction

Androgenetic alopecia (AGA) is the progressive decline in structure and function of the hair follicles with eventual loss of the hair. The AGA has been linked to clusters of cardiovascular risk factors (CVRFs) with attendant complications if not well taken care of. Cotton was the first to spotlight the relationship between AGA and the cardiovascular system.[1]

AGA has been suggested as a surrogate marker of atherosclerosis.[2] Ertas *et al*.[3] reported that AGA could be an indicator of metabolic syndrome (MetS) and cardiovascular disease (CVD) as well as a marker for early atherosclerosis. Lin-Hui Su also showed diabetes mellitus to be an independent predictor of mortality in AGA.[4] Premature androgenetic alopecia (especially grades III and IV) has been linked to MetS.[5]

Cardiovascular risk factors, if not prevented or treated on time, could lead to diseases. Cardiovascular diseases,

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accounting for about one third of global death, still tops the death list while coronary heart disease contributes 7.4 million deaths out of the 17.5 million burdens of cardiovascular diseases.[6] They have also been shown to constitute 37% of death under the age of 70, majority occurring in resource poor areas.[6] This study, therefore, aimed at documenting the clusters of traditional CVRFs and component traits of metabolic syndrome associated with AGA and its severity in a resource poor setting.

Methods

It was a cross-sectional study carried out in selected communities on 520 participants consisting of 260 subjects and 260 controls who were matched for age and sex through a multistage sampling technique. The study was approved as part of a larger study on androgenetic alopecia (LTH/OGB/EC/2017/162). Method of participant selection has previously been described.[7]

Relevant history including age, gender, occupation, and ethnicity were documented. Their smoking habit, alcohol intake, and sedentary lifestyle were also documented. Sedentary activity was assessed using the level of physical activities according to participants' occupation—highly skilled academic, skilled workers, manual worker, student, unemployed but can work, unemployed but cannot work.[8,9]

We documented self-reported previous history of cardiovascular events like coronary artery disease and stroke. Participants had all parts of their head examined and diagnosis of AGA was made clinically and graded by the modified Hamilton-Norwood and Ludwig scales, respectively, for male and female.[7] Anthropometric characteristics of participants were assessed using standard methods.[10] The heights of the participants were obtained using stadiometer to the nearest 0.1 cm. Weight was checked using the hospital weighing scale and recorded to nearest 0.1 kg. The waist circumference was taken at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest.[10] All measurements were taken using a stretch-resistant tape measure. Abdominal obesity was diagnosed with the International Diabetes Federation (IDF) criteria.[11] Body mass index (BMI) was calculated as appropriate. The blood pressure (BP) readings were obtained from subjects and control using Accoson mercury sphygmomanometer. All blood pressure measurements were obtained from two recordings in a sitting position at least five minutes apart, and an average was recorded as the study data. Hypertension was diagnosed using the Joint National Committee (JNC-8) guidelines with systolic ≥140 mmHg and diastolic blood pressure ≥90 mmHg.[12]

For males, normal AGA consisted of grade I and II male AGA, mild AGA were IIa-IIIv, moderate AGA as IV-Va while severe AGA was defined by stages VI and VII.[7,13] Similarly in females, mild, moderate and severe FAGA consisted of Ludwig I, II, and III respectively.[7,13]

Five milliliters each of fasting venous blood was taken from participants after 8-12 hour fast into fluoride oxalate and Ethylene Diamine (EDTA) bottles for the analysis of blood glucose and lipids.[7] Samples were centrifuged and serum preserved by refrigeration at -20 degree centigrade until analyzed in accordance with the protocol in the Randox reagent.[7] Samples were analyzed at the Metabolic Research Laboratory of LAUTECH Teaching Hospital Ogbomoso after appropriate collection, transportation, and storage. Glucose oxidase method and reagents of Randox^{R} laboratories Ltd, UK were used to respectively analyze blood glucose and all lipid fractions except LDL-c. The LDL-c was calculated using the Friedwald equation.[7,14] Metabolic syndrome was diagnosed with the International Diabetes Federation (IDF) criteria.[11]

Data was entered using Statistical Package for Social Sciences programmed (SPSS) version 20. Data cleaning was carried out and errors corrected. Quantitative variables were summarized using means and standard deviation, while frequencies and proportions were used for qualitative variables. Associations between AGA and cardiovascular risks were tested using Chi-square test, Students *t* test, and ANOVA (analysis of variance). Level of significance was placed at 5%.

Results

The mean age of subjects with AGA and control population (51.32 ± 16.31 years vs. 51.09 ± 14.09 years) were essentially similar. Majority of subjects were working class individuals (54.5% vs. 45.5%, $p<0.001$). The subjects were taller than the control (1.67 ± 0.08 centimeters vs. 1.64 ± 0.10 centimeters, $p<0.001$), otherwise, other anthropometric characteristics were not markedly different between subjects and control population. Participants with androgenetic alopecia had lower mean HDL-c compared with the control (1.13 ± 0.44 mmol/l vs. 1.29 ± 0.47 mmol/l, $p<0.001$). There existed no marked difference in the mean of total cholesterol, LDL-c, and triglyceride. AGA subjects had higher systolic blood pressure (SBP) compared to the controls (125.96 ± 17.91 mmHg vs. 121.36 ± 21.49 mmHg, $p=0.008$) Table 1.

The prevalence of traditional CVRFs and previously reported CVD in subjects with AGA and controls, Table 2. Dyslipidaemia (61.50% vs. 48.10%, $p=0.002$), sedentary lifestyle (39.60% vs. 28.80%, $p=0.010$), and

Table 1: Socio-demographic and clinical characteristics of study participants

Characteristics	AGA *n* (%)	Control *n* (%)	X^2/t	*p*
Gender				
Mean age	51.32±16.31	51.09±14.09	0.173	0.863
Occupation				
Working	223 (54.5)	186 (45.5)	15.681	<0.001
Not working	37 (33.3)	74 (66.7)		
Anthropometry				
Weight (kilogram)	67.39±12.16	65.62±15.13	1.469	0.143
Height (meter)	1.67±0.08	1.64±0.10	4.478	<0.001
BMI	24.13±4.94	25.18±8.84	-1.669	0.096
Waist circumference (centimeter)	85.43±12.52	84.27±12.86	1.046	0.296
Hip circumference (centimeter)	91.76±11.08	96.20±53.23	-1.314	0.189
Lipid Profile				
Triglyceride (mmol/L)	1.15±0.62	1.06±0.52	1.850	0.065
Total cholesterol (mmol/L)	4.74±1.18	4.85±1.20	-0.992	0.322
HDL-cholesterol (mmol/L)	1.13±0.44	1.29±0.47	-4.225	<0.001
LDL-cholesterol (mmol/L)	3.35±1.18	3.21±1.15	1.407	0.160
Blood Pressure				
SBP (mmHg)	125.96±17.91	121.36±21.49	2.647	0.008
DBP (mmHg)	79.69±10.89	79.07±14.32	0.550	0.583
Fasting Blood Sugar (mmol/L)	5.15±1.83	4.94±1.58	1.388	0.166

X^2=Chi-square, *n*=number, % = percentage, t=Student's t-value, LDL=Low Density Lipoprotein, HDL=High-Density Lipoprotein, SBP=Systolic Blood Pressure, DBP=Diastolic Blood Pressure, t=student's *t* value

Table 2: Prevalence of traditional cardiovascular risk factors and cardiovascular diseases in subjects with androgenetic alopecia and controls

Variables	AGA *n* (%)	Control *n* (%)	Total *n* (%)	X^2/t	*p*
Traditional Cardiovascular Risk factors					
Hypertension	50 (19.20)	42 (16.20)	92 (17.70)	0.845	0.358
Diabetes	14 (5.40)	13 (5.00)	27 (5.20)	0.715	0.699
Dyslipidaemia	160 (61.50)	125 (48.10)	285 (54.80)	9.511	0.002
Sedentary	103 (39.60)	75 (28.80)	178 (34.20)	6.697	0.010
Alcohol Intake	73 (28.10)	37 (14.20)	110 (21.20)	15.933	<0.001
Smoking	7 (2.70)	2 (0.8)	9 (1.70)	2.830	0.278
Obesity	21 (8.10)	41 (15.80)	62 (11.90)	7.325	0.007
Past cardiovascular diseases					
Coronary artery diseases	1 (0.4)	1 (0.4)	2 0.4	0.000	1.000
Stroke	5 (1.9)	1 (0.4)	6 (1.2)	2.698	0.216

X^2=Chi-square, AGA=Androgenetic alopecia; Hypertension diagnosed using JNC-8 criteria of systolic blood pressure and diastolic blood pressure greater than 140 mmHg and 90 mmHg, respectively. Diabetes mellitus diagnosed using American Diabetes Association criteria (2018) as fasting plasma glucose (at least 8 hours) ≥ 7.0 mmol/l. Dyslipidemia diagnosed as: Low HDL (male) <1.03 mmol/l, (female) <1.29 mmol/l; High LDL more than 4.1 mmol/l; High triglyceride > 1.7 mmol/l; High total cholestero l > 6.2 mmol/l. Stroke and coronary artery diseases were reported by patients. Sedentary: level of physical activities according to participants' occupation grouping- highly skilled academic, skilled workers, manual worker, student, unemployed but can work, unemployed but cannot work

alcohol intake (28.10% vs. 14.20%, p<0.001) were more common in AGA participants. However, obesity (15.80% vs. 8.10%, p = 0.007) was higher in controls. There prevalence of past cardiovascular diseases between the two groups did not show any remarkable difference.

The prevalence of metabolic syndrome and component traits was examined among subjects with AGA and controls in Table 3 using the International Diabetes Federation (IDF) criteria. The prevalence of MetS (8.08%) was higher compared to the control (7.30%). Similarly, men and women with AGA had significantly lower HDL-c than control (44.23% vs. 24.23%, p < 0.002 and 5.77% vs 5.38%, p < 0.002). There were no remarkable differences in the other metabolic syndrome traits between the two groups.

In Table 4, the metabolic risk factors in relation to the severity of male androgenetic alopecia (MAGA) and female androgenetic alopecia (FAGA) were compared.

Table 3: The prevalence of metabolic syndrome and its component traits in androgenetic alopecia and control using the international diabetes federation (IDF) criteria

Variables	AGA *n* (%)	Control *n* (%)	Total *n* (%)	X^2	*p*
Metabolic Syndrome	21 (8.08)	19 (7.30)	40 (7.69)	0.108	0.742
Elevated FBS	33 (12.69)	30 (11.54)	63 (12.12)	0.163	0.687
Low HDL (male)	115 (44.23)	63 (24.23)	178 (34.23)	32.260	<0.001
Low HDL (female)	15 (5.77)	14 (5.38)	29 (5.57)	14.588	<0.002
High TG	33 (12.69)	36 (13.85)	69 (13.27)	0.150	0.698
Hypertension	48 (18.46)	45 (17.31)	93 (17.88)	0.118	0.731
High AC (male)	41 (15.77)	38 (14.62)	79 (15.19)	0.194	0.979
High AC (female)	25 (9.62)	27 (10.38)	52 (10.0)	0.194	0.979

X^2=Chi-square, AGA=Androgenetic alopecia, IDF=International Diabetes Federation, FBS=Fasting blood sugar, HDL=High-Density Lipoprotein, LDL=Low-Density Lipoprotein, TG=Triglyceride, BP=Blood pressure, AC=Abdominal circumference, LDL=Low Density Lipoprotein, HDL=High-Density Lipoprotein

Table 4: Analysis of variance (ANOVA) of metabolic risk factors in relation to the severity of male and female androgenetic alopecia

	Male Androgenetic Alopecia					Female Androgenetic Alopecia				
	Total *n*=212	Disease Severity Group 1 *n*=103	Disease Severity Group 2 *n*=60	Disease Severity Group 3 *n*=49	*p*	Total *n*=44	Disease Severity Group 1 *n*=20	Disease Severity Group 2 *n*=18	Disease Severity Group 3 *n*=6	*p*
Age	45.87±12.37	42.49±12.41	46.65±11.38	52.02±11.07	<0.001	29.84±6.19	28.60±2.23	28.89±2.30	36.83±15.00	0.009
FBS	5.22±1.92	5.30±2.17	5.08±1.38	5.22±1.92	0.775	5.01±1.03	5.23±1.15	4.82±0.72	4.87±1.37	0.450
TC	4.76±1.21	4.81±1.23	4.82±1.02	4.59±1.36	0.536	4.83±1.18	4.60±0.94	5.08±1.32	4.81±1.50	0.476
TG	1.15±0.64	1.15±0.627	1.05±0.43	1.30±0.84	0.134	1.11±0.42	1.12±0.42	1.21±0.45	1.00±0.36	0.556
HDL-c	1.14±0.43	1.10±0.39	1.23±0.47	1.13±0.47	0.204	1.11±0.39	1.19±0.36	1.01±0.40	1.14±0.43	0.374
LDL-c	3.34±1.17	3.42±1.19	3.30±1.01	3.23±1.29	0.589	3.37±1.22	3.22±1.03	3.54±1.20	3.32±1.91	0.718
SBP	123.78±17.04	121.14±16.22	125.02±17.02	127.82±18.13	0.024	114.66±11.06	111.75±11.79	116.22±10.14	119.67±10.09	0.230
DBP	79.65±10.57	79.04±10.41	79.13±10.63	81.55±10.82	0.356	76.41±10.26	74.95±9.73	76.00±10.86	82.50±9.52	0.286
WC	85.19±12.75	83.61±11.76	85.21±15.36	88.49±10.66	0.027	77.78±8.44	79.82±8.82	74.61±6.73	80.50±10.03	0.113
BMI	24.20±5.09	23.65±6.17	24.44±3.67	25.06±3.88	0.257	22.73±3.17	23.91±3.48	21.27±2.22	23.21±3.21	0.031

FBS=Fasting blood sugar, HDL=High-Density Lipoprotein, LDL=Low-Density Lipoprotein, TG=Triglyceride, BMI=Body mass index, WC=waist circumference, kg-kilogramme, DSG-1: Modified Hamilton Norwood scale I-III, DSG-2- Modified Hamilton Norwood scale IV-V, DSG-3: Modified Hamilton Norwood scale VI-VII

The severity of the AGA increased with mean age for the MAGA (p<0.001) and FAGA (p=0.009). Also, mean SBP (121.14 ± 16.22 vs. 125.02 ± 17.02 vs. 127.82 ± 18.13 mmHg, p=0.024) and waist circumference (83.61 ± 11.76 vs. 85.21 ± 15.36 vs. 88.49 ± 10.66 centimeters, p=0.027) increased across MAGA disease severity group. Although there was a decrease in trend of BMI between DSG I and II, BMI increased between DSG II and III of FAGA (23.91 ± 3.48 vs 21.27 ± 2.22 vs. 23.21 ± 3.21, p=0.031), Table 4.

Discussion

The study shows that AGA was significantly associated with traditional CVRFs such as dyslipidaemia, sedentary lifestyle, and alcohol intake. Although the prevalence of MetS was insignificantly higher in AGA, HDL-c was significantly lower in AGA compared to control. Although there was no difference in the prevalence of hypertension, diabetes, smoking, and existence of previous CVD between the two groups, but AGA was associated with a significant higher mean SBP. Meanwhile, obesity was significantly found among the control than the AGA population. The older the more severe MAGA and FAGA while the higher the SBP and waist circumference the more severe MAGA. Increase in BMI was significantly associated with increase in AGA severity between DSG 2 and 3 only in FAGA.

The relationship between AGA and CVRFs is replete in the literature but with inconsistent findings globally and even within regions. We found there exists relationship among dyslipidaemia, alcohol intake, sedentary lifestyle, and AGA. Park *et al*.[15] similarly found low HDLc as a correlate of AGA and in addition, clusters of CVD such as stroke, higher prevalence of hypertension, smoking, fasting glucose, and triglyceride were associated with AGA in difference to our study. Unlike in the present

study, Arias-Santiago *et al*.[16] found abdominal obesity, SBP, triglycerides, and blood glucose levels associated with AGA. In similarity to our study, Yeo *et al*. and other workers[17,18] reported association between AGA and alcohol consumption.[19]

AGA was significantly associated with dyslipidaemia in this study. This is similar to the reports by Bakry *et al*. and Arias-Santiago *et al*. who had documented that AGA was significantly associated with high triglyceride and LDLc.[16,20] Subjects in this study were found to have a significantly lower HDLc than controls, a finding similar to some reports.[21-23] Higher prevalence of hypercholesterolemia in AGA has also been documented.[3,5]

Vora *et al*. also reported lower HDLc in premature AGA while Iram *et al*. reported deranged lipid profile in severe AGA.[24,25] Otsuka *et al*.[26] found dyslipidaemia related to more tendencies with hypertension. Kim *et al*.[27] was of the opinion that dyslipidaemia may be due to the relationship between AGA and CVD. However, in the present study, we found no association between the CVD examined and AGA. This may be due to the relatively low prevalence of MetS and CVRFs in the index population. Contrary to the findings of this study, Park *et al*.[15] found significant association between male-type AGA, and hypertension, stroke, and smoking, but AGA was only associated with hypertension and diabetes mellitus in Kim's *et al*. study.[27] The finding of increased alcohol intake among AGA (28.1% vs. 14.2%) compared to the controls was comparable to the work by Nargis *et al*. and Salman *et al*.[18,19] Alcohol consumption has been linked to dyslipidemia and hypertension As Moreira *et al*.[28] related quantity of alcohol consumed with blood pressures irrespective of age, smoking, education, and blood pressure lowering drugs.

Concerning association and severity of CVRFs and AGA, the severity of AGA increased with age for males and females. The severity of the MAGA increases, the more severe the mean SBP and waist circumference in males. Park *et al*.,[15] in addition to higher BMI, documented wider waist circumference, higher diastolic blood pressure (DBP), and higher prevalence rate of hypertension with more severe FAGA. Agamia *et al*.[29] in Egypt found a higher waist circumference and BMI in AGA compared to non-AGA. Arias-Santiago also documented abdominal obesity, SBP, triglyceride, and blood glucose levels were significantly greater in AGA.[16] Danesh-Shakiba *et al*.[30] documented a significantly higher SBP and DBP with Norwood-Hamilton IV-VII compared to type II and III.[30]

Smoking was not found related to AGA in this study, as reported by Nargis *et al*., while Su *et al*. reported an association.[18,31] The smoking status, the quantity as well as intensity if smoking of smoking were related to AGA in their study[31] as also found in a previous Nigerian study that reported the relationship between smoking and early-onset AGA.[32] We reported no important link between AGA, diabetes mellitus, and abdominal obesity as found by other studies.[21-23]

Metabolic syndrome has been associated with AGA in several studies.[5,29,33-35] AGA was reported as a risk factor for MetS that increased the possibility of atherosclerotic CVD.[3-5,36] The prevalence of metabolic syndrome in AGA (8.08%) in this study is lower than some previous reports (14.1%-60%) but close to 8.0% reported by Batra *et al*.[5,29,33-35] These previously reported works vary in age, race, and settings as compared to this study. Early onset AGA has been found to be related to MetS in several studies.[22,23,33] Gopinath *et al*. and others[1,23] suggested early screening for MetS, while association signaled an indication for coronary artery disease in the study by Acibucu *et al*.[37] Other studies, mostly in the Asian nations (Wu and Su), found late onset AGA was associated with Metabolic syndrome.[31,38] While Wu *et al*., identified AGA as risk factor for MetS, Su suggested early intervention in people with AGA could reduce risk of complication of CVD and type 2 diabetes mellitus.[31,38]

The study being cross-sectional lacks the strength to establish causality. The recall of the questions on past CVD is also fraught with possibilities of negative or positive recall errors. These limitations notwithstanding, the strength of the study lies in being a community-based study, robust sample size and carefully designed probabilistic sampling that give credence to the statistics.

Conclusion

The AGA was significantly associated with dyslipidaemia, sedentary lifestyle, and alcohol intake. Metabolic syndrome is insignificantly prevalent in AGA (8.08% vs 7.30%) compared to control. The severity of AGA was associated with increasing age in male and female. Increasing severity of MAGA was associated with increase in SBP and waist circumference. Increase in BMI was significantly associated with increase in severity between DSG 2 and 3 only in FAGA. Subjects with AGA should be screened for dyslipidemia and counseled against the use of alcohol and sedentary lifestyle.

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Conflicts of interest

There are no conflicts of interest.

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