Original

Cardiometabolic comorbidities associated with androgenetic alopecia: Findings from a sex-stratified, propensity-matched national cohort

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Abstract

Background: To examine associations between androgenetic alopecia (AGA) and cardiometabolic comorbidities in a large, diverse United States cohort with adjustment for socioeconomic and behavioral factors. **Methods:** We conducted a cross-sectional, 1:2 propensity score–matched analysis using data from the All of Us Research Program. Participants with AGA (n = 1015) were matched to controls on age, sex, race, and ethnicity. Thirteen cardiovascular and metabolic conditions were assessed using electronic health records (EHRs). Conditional logistic regression, adjusted for smoking, insurance, income, healthcare access, and marital status, was performed overall and by sex. **Results:** AGA was significantly associated with hyperlipidemia in males (adjusted odds ratio [OR], 2.32; 95% confidence interval [CI], 1.56–3.46) and females (adjusted OR, 2.55; 95% CI, 1.98–3.28). In females, AGA was also independently associated with obesity (adjusted OR, 1.48; 95% CI, 1.17–1.86), but this association was not observed in males. Associations with hypertension and valvular heart disease did not persist after adjustment. The cross-sectional design and reliance on structured EHR data limited causal inference. **Conclusion:** AGA is independently associated with hyperlipidemia in both sexes and with obesity in females, suggesting its potential as a clinical marker of cardiometabolic risk.

Introduction

Androgenetic alopecia (AGA) is the most prevalent form of hair loss worldwide, affecting approximately 50% of both males and females over their lifetime.[1] Although historically regarded as a cosmetic concern, emerging evidence suggests that AGA may be an important clinical marker of systemic disease, particularly disorders involving cardiovascular and metabolic health. The potential link between AGA and cardiometabolic risk is biologically plausible, as both conditions share pathophysiologic mechanisms such as androgen signaling, chronic inflammation, and endothelial dysfunction.[2] Importantly, AGA often develops early in adulthood, creating a potential opportunity for early identification of individuals at increased cardiometabolic risk before the onset of overt clinical disease.[1]

Existing literature examining the association between AGA and metabolic or cardiovascular outcomes has been constrained by small sample sizes, recruitment from limited or homogeneous populations, and insufficient adjustment for socioeconomic and behavioral factors, often with contradictory findings.[3] As a result, the clinical relevance of AGA as a marker for systemic disease remains uncertain. Given the global burden of cardiometabolic disease, large-scale studies are needed to determine whether AGA may serve as an early, accessible signal for cardiovascular and metabolic risk.

In the present study, we leveraged data from the All of Us Research Program, a nationally representative United States cohort, to examine associations between AGA and a range of cardiovascular and metabolic comorbidities. By applying robust propensity score matching and adjusting for key behavioral and socioeconomic variables, we aimed to clarify whether AGA is independently associated with adverse cardiometabolic outcomes.

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Methods

We conducted a cross-sectional, 1:2 propensity score–matched analysis using the All of Us Research Program participants with at least 1 year of electronic health record (EHR) data. AGA cases (n = 1015), identified using concept identifier (ID) 432867, were matched to 2030 controls on age, race, ethnicity, and sex using nearest-neighbor matching. Demographic and socioeconomic variables (smoking, insurance, income, healthcare access, marital status) were derived from survey responses.

We evaluated 13 cardiovascular and metabolic comorbidities based on clinical relevance, inclusion in major guidelines (American Heart Association/American College of Cardiology, American Diabetes Association, Kidney Disease: Improving Global Outcomes), and prior use in cardiovascular disease risk modeling frameworks using the All of Us Research Program database.[4-7] These included atherosclerotic endpoints (coronary artery disease, myocardial infarction, and peripheral artery disease) as well as cerebrovascular outcomes such as ischemic stroke and transient ischemic attack. Additional cardiovascular endpoints included heart failure, valvular heart disease, atrial fibrillation, and venous thromboembolism (deep vein thrombosis and pulmonary embolism). We also assessed key modifiable cardiometabolic risk factors, including hypertension, type 2 diabetes mellitus, hyperlipidemia, and obesity (body mass index, ≥ 30 kg/m^2). Chronic kidney disease was included as a vascular disease equivalent and cardiovascular risk enhancer. Each comorbidity was identified using curated condition and source concept IDs from the All of Us Researcher Workbench.

Baseline characteristics were compared using χ^2 tests for categorical variables and *t* tests for continuous variables. Comorbidities with statistically significant bivariate associations ($P < .05$) were advanced to conditional logistic regression models, stratified by matched subclass and adjusted for smoking status, insurance coverage, household income, marital status, and healthcare utilization. Sex-stratified regression analyses were performed to evaluate potential effect modification. Adjusted odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. Analyses were conducted using R in the All of Us Researcher Workbench.

Results

A total of 1015 individuals with AGA were matched 1:2 to 2030 controls on age, sex at birth, race, and ethnicity, with identical distributions across these variables ($P > .99$ for all). The mean age of both cohorts was 60.62 ± 16.76 years, and the sex distribution was 68.4% female and 29.2% male (Table 1).

Compared with controls, individuals with AGA were more likely to be nonsmokers (67.3% versus 57.8%; $P < .001$), have health insurance (96.2% versus 90.0%; $P < .001$), and fall into higher healthcare utilization strata ($P < .001$). They were also more likely to report annual household income greater than $50,000 (22.4% versus 18.7%; $P < .001$) and to be married (48.7% versus 42.1%; $P < .001$) (Table 1)

In bivariate analyses, AGA was associated with hyperlipidemia (60.6% versus 42.1%; $P < .001$), obesity (38.1% versus 28.5%; $P < .001$), hypertension (50.9% versus 44.2%; $P = .001$), and valvular heart disease (17.0% versus 12.3%; $P < .001$). No significant differences were observed for coronary artery disease, myocardial infarction, stroke/transient ischemic attack, heart failure, peripheral artery disease, atrial fibrillation, venous thromboembolism, type 2 diabetes, or chronic kidney disease (all $P > .05$) (Table 1)

In multivariable models, hyperlipidemia remained associated with AGA in both males (OR, 2.32; 95% CI, 1.56–3.46; $P < .001$) and females (OR, 2.55; 95% CI, 1.98–3.28; $P < .001$). Obesity was associated with AGA in females (OR, 1.48; 95% CI, 1.17–1.86; $P < .001$) but not in males (OR, 0.83; 95% CI, 0.55–1.25; $P = .36$). Hypertension and valvular heart disease were not significant after adjustment (Table 2).

Discussion

In this large, propensity score–matched analysis of the All of Us Research Program cohort, we found that AGA was significantly associated with hyperlipidemia in both sexes and with obesity in females, independent of key socioeconomic and behavioral confounders. These associations persisted after multivariable adjustment for smoking status, insurance coverage, household income, healthcare access, and marital status, suggesting that AGA may reflect underlying cardiometabolic risk beyond shared behavioral and socioeconomic factors.

Although unadjusted analyses indicated higher rates of hypertension and valvular heart disease in individuals with AGA, these associations lost significance after adjustment for healthcare access, socioeconomic status, and utilization. This attenuation suggests that the observed cardiovascular differences may be attributable to structural or behavioral factors rather than direct pathophysiology. This finding is consistent with the mixed evidence linking AGA to specific cardiovascular endpoints in prior literature.

For example, Dharam Kumar et al[8] examined 100 males and 100 controls and reported a higher prevalence of metabolic syndrome in individuals with AGA, marked by abnormalities in triglycerides, HDL cholesterol, and blood pressure. However, their study was limited by small sample size, single-center recruitment, and lack of socioeconomic adjustment, which restricts generalizability. Behrangi et al[9] observed increased metabolic risk in patients with AGA but did not stratify results by sex or control for healthcare access, which may have obscured important patterns. The clinic-based case-control study by Arias-Santiago et al[10] showed that females with AGA had higher triglycerides, total cholesterol, and LDL-C and lower HDL-C compared with controls; males with AGA also had higher triglycerides and LDL-C. A Taiwanese

Table 1. *Baseline Characteristics Stratified by Presence of AGA.*

Characteristic	No AGA (n = 2030)	AGA (n = 1015)	*P* Value[a]
Age, mean (SD)	60.62 (16.76)	60.62 (16.76)	1.000
Female, n (%)	1388 (68.4)	694 (68.4)	
Male, n (%)	592 (29.2)	296 (29.2)	
Race, n (%)			1.000
Asian	128 (6.3)	64 (6.3)	
Black	250 (12.3)	125 (12.3)	
White	1210 (59.6)	605 (59.6)	
Other	442 (21.8)	221 (21.8)	
Ethnicity, n (%)			1.000
Hispanic	318 (15.7)	159 (15.7)	
Non-Hispanic	1630 (80.3)	815 (80.3)	
Ever smoker, n (%)	853 (42.0)	332 (32.7)	< .001
Insurance, n (%)			< .001
No	192 (9.5)	32 (3.2)	
Yes	1826 (90.0)	976 (96.2)	
Income, n (%)			< .001
≤ $50,000	914 (45.0)	276 (27.2)	
> $50,000	380 (18.7)	227 (22.4)	
Missing	736 (36.3)	512 (50.4)	
Healthcare utilization, n (%)			< .001
Low	729 (35.9)	456 (45.9)	
High	183 (9.0)	110 (10.8)	
Missing	1118 (55.1)	449 (44.2)	
Marital status, n (%)			< .001
Married	854 (42.1)	494 (48.7)	
Divorced/separated/widowed	568 (28.0)	219 (21.6)	
Never married	498 (24.5)	266 (26.2)	
Hypertension, n (%)	898 (44.2)	517 (50.9)	.001
Hyperlipidemia, n (%)	854 (42.1)	615 (60.6)	< .001
Coronary artery disease, n (%)	251 (12.4)	144 (14.2)	.176
Myocardial infarction, n (%)	107 (5.3)	54 (5.3)	1.000
Stroke/ transient ischemic attack, n (%)	67 (3.3)	46 (4.5)	.111
Congestive heart failure, n (%)	129 (6.4)	70 (6.9)	.622
Peripheral artery disease, n (%)	< 20 (0.6)	< 20 (0.6)	1.000
Atrial fibrillation, n (%)	143 (7.0)	68 (6.7)	.781
Venous thromboembolism, n (%)	163 (8.0)	94 (9.3)	.279
Type 2 diabetes, n (%)	431 (21.2)	236 (23.3)	.221
Chronic kidney disease, n (%)	210 (10.3)	112 (11.0)	.602
Obesity, n (%)	578 (28.5)	387 (38.1)	< .001
Valvular heart disease, n (%)	250 (12.3)	173 (17.0)	< .001

Abbreviations: AGA, androgenetic alopecia; TIA, transient ischemic attack.
[a] *P* values were calculated using *t* test for continuous variables and the $\chi 2$ test for categorical variables.

Table 2. *Multivariable Conditional Logistic Regression of AGA-Associated Comorbidities, Stratified by Sex.*

Condition[a]		OR (95% CI)	*P* Value
Hyperlipidemia			
	Male	2.32 (1.56–3.46)	< .001
	Female	2.55 (1.98–3.28)	< .001
Hypertension			
	Male	1.02 (0.67–1.55)	.935
	Female	1.12 (0.88–1.44)	.350
Obesity			
	Male	0.83 (0.55–1.25)	.361
	Female	1.48 (1.17–1.86)	< .001
Valvular heart disease			
	Male	1.46 (0.81–2.61)	.206
	Female	1.11 (0.85–1.45)	.435

Abbreviations: AGA, androgenetic alopecia; CI, confidence interval; OR, odds ratio.
[a] Models stratified by matched subclass (age, race, ethnicity) and adjusted for smoking status, insurance coverage, household income, healthcare access, and marital status.

study[11] of 740 males found that moderate-to-severe AGA was associated with metabolic syndrome (adjusted OR, 1.67) and low HDL-C (adjusted OR, 2.36) after controlling for age, family history, and smoking, although the findings were limited by population homogeneity. Our findings are consistent with these reports of dyslipidemia, demonstrating that hyperlipidemia remains associated with AGA after adjustment for socioeconomic and behavioral variables.

One of our most notable findings is the sex-specific association between AGA and obesity, which was significant in females but not in males. This supports previous evidence that female-pattern AGA may indicate more substantial underlying metabolic dysfunction. For example, meta-analysis by Qiu et al[3] found that the odds of metabolic syndrome were more than twice as high in females with AGA (OR, 7.34) compared with males (OR, 3.08). Our results reflect this disparity and suggest that female AGA may serve as a clinical signal of cardiometabolic risk.

Several biological pathways may explain these associations. Elevated dihydrotestosterone activity, a key factor in AGA, increases expression of proinflammatory cytokines such as TGF-β1, IL-1α, and TNF-α in dermal papilla cells, disrupting the hair cycle and promoting follicular miniaturization. Chronic low-grade inflammation, oxidative stress, and insulin resistance (hallmarks of metabolic syndrome) may further exacerbate perifollicular microinflammation and impair nutrient delivery to hair follicles. Environmental exposures such as smoking and ultraviolet radiation, along with microbiota-related inflammation, may also contribute to this shared pathogenic process.[12,13]

This study has limitations. Its cross-sectional design precludes assessment of causality or temporal directionality. Reliance on structured EHR data may lead to underreporting or misclassification of AGA and metabolic conditions, particularly among individuals with limited healthcare engagement. We lacked detailed information on physical activity, dietary habits, and use of lipid-lowering or antiandrogenic medications, which could confound the observed associations. In addition, the absence of standardized clinical grading for AGA severity prevented analysis of dose–response relationships. However, notable strengths include the large, nationally representative cohort and rigorous propensity score matching with adjustment for behavioral and socioeconomic confounders often unmeasured in prior studies.

Despite these limitations, our findings have important clinical implications. The association between AGA and hyperlipidemia, particularly in females, persisted after accounting for relevant confounders, suggesting a possible independent biological relationship. Clinicians should consider AGA, especially in younger females, as a potential indicator of underlying metabolic risk. Routine screening for dyslipidemia and obesity in this population may allow for earlier identification and management of cardiometabolic disease.

Conclusion

Our analysis of the All of Us Research Program cohort confirms and extends prior evidence that AGA is significantly associated with hyperlipidemia in both sexes and with obesity in females. These findings support the growing recognition that AGA may serve as a dermatologic window into cardiovascular risk. Early identification and preventive screening in this population may be an important step in mitigating long-term health consequences.

Potential conflicts of interest

The authors declare no conflicts of interest.

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