



Systematic Review and Meta-analysis of the Association Between Metabolic Syndrome and Androgenetic Alopecia

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The association of androgenetic alopecia with metabolic syndrome has been investigated in several studies, with conflicting results. We conducted a meta-analysis to quantitatively evaluate the risk grade of metabolic syndrome and the metabolic profile in patients with androgenetic alopecia compared with controls. In total, 19 articles (2,531 participants) satisfied the inclusion criteria. The pooled odds ratio for the prevalence rate of metabolic syndrome between the group with androgenetic alopecia and controls was 3.46 (95% CI 2.38–5.05; $p < 0.001$). Female sex, early onset, and African ethnicity were associated with an increased risk of metabolic syndrome. Furthermore, patients with androgenetic alopecia had significantly poorer metabolic profiles, such as body mass index, waist circumference, fasting glucose, blood lipids, and blood pressure. It is important for physicians to screen metabolism-related indicators in patients with androgenetic alopecia. More rigorously designed studies and larger sample sizes are required in future studies.

Key words: alopecia; androgens; metabolic syndrome; systematic review; meta-analysis.

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Androgenetic alopecia (AGA) is the most common pattern of hair loss disorder that occurs around or after puberty. AGA features progressive miniaturization of the hair follicle in predisposed individuals without scarring (1). The incidence of AGA depends on age, sex, and ethnicity. Epidemiological data show that approximately 30% of white men have AGA by age 30 years, increasing to approximately 50% by age 50 years and 80% by age 70 years. In addition, 40–50% of white women may develop AGA during their lifetime. People with Asian and African ethnicity have a lower prevalence of AGA than white people (2). It is generally recognized that the diminishing anagen phase of the hair follicles is triggered by dihydrotestosterone (DHT) binding to androgen receptors, resulting in terminal hair transforming into thinner and shorter vellus hair over time (3).

SIGNIFICANCE

This study showed that androgenetic alopecia was associated with a high risk of metabolic syndrome and patients with androgenetic alopecia had a significantly worse metabolic profile. These findings highlight the need for screening metabolism-related indicators in patients with androgenetic alopecia in order to prevent long-term consequences with respect to cardiovascular abnormalities.

Metabolic syndrome (MetS) is a group of metabolic abnormalities involving central obesity, hypertension, glucose intolerance, insulin resistance (IR) and dyslipidaemia (4). Patients diagnosed with MetS are likely to be more vulnerable to cardiovascular events and diabetes (5). The association of AGA with MetS, as well as with IR, has been investigated in several studies, with contradictory findings (6–8). Few meta-analyses of the relationship between AGA with MetS have been conducted. Although Wu (9) and Caro-Chang (10) showed that patients with AGA had a greater chance of developing MetS, comparison of metabolism-related indicators between patients and healthy individuals were not performed. Trieu et al. (11) reported that alopecia was a potential observable clinical marker for cardiovascular events, because baldness was positively associated with an increased risk of hyperinsulinemia, IR, dyslipidaemia, and hypertension. The aim of the current study was to perform a systematic review and meta-analysis of the literature to clarify and illustrate the prevalence of MetS in patients with AGA compared with control individuals. The study also investigated whether sex, region, or alopecia onset, have an influence on increasing the risk of developing MetS. Finally, the study also evaluated metabolic indicators, such as body mass index (BMI), waist circumference (WC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), fasting blood sugar (FBS), systolic blood pressure (SBP), and diastolic blood pressure (DBP) between patients with AGA and controls.

MATERIALS AND METHODS

This systematic review and meta-analysis was performed following the MOOSE Guidelines of observational studies. The protocol of this meta-analysis was registered at PROSPERO, International Prospective Register of Systematic Reviews (crd.york.ac.uk/prospero/, Identifier: CRD42021269754).

Search strategy

Four main databases, PubMed, EMBASE, Web of Science, and The Cochrane Library, were searched to identify potentially eligible studies from database inception to 20 June 2021. The search terms used were: "metabolic syndrome" OR "syndrome X" AND "alopecia" OR "androgenetic alopecia" OR "pattern hair loss" OR "hair loss" OR "baldness". For example, an advanced search of PubMed comprised: ((("Metabolic Syndrome"[Mesh]) OR ("Metabolic Syndrome"[Title/Abstract]) OR ("Syndrome X"[Title/Abstract])) AND (("Alopecia"[Mesh]) OR ("alopecia"[Title/Abstract]) OR ("androgenetic alopecia"[Title/Abstract]) OR ("pattern hair loss"[Title/Abstract]) OR ("hair loss"[Title/Abstract]) OR ("baldness"[Title/Abstract])). The references of included studies and related reviews were screened to find additional potentially eligible studies.

Study selection

Included original articles met the following inclusion criteria: (i) the study had a case-control design with 2 study arms: 1 group of patients with AGA and a control group, and published data on both patients with AGA and age- and sex-matched controls; (ii) the study population was over age 18 years; (iii) the diagnostic criteria of MetS and severity in patients with AGA were specifically described, and control individuals were also clearly defined; (iv) the study clearly reported the prevalence of each condition associated with MetS in each group (AGA and controls); and (v) the manuscript was in English. Exclusion criteria were: (i) duplicated or overlapping data and (ii) incomplete data or unclear outcome effects. The process of article selection was carried out independently by 2 research fellows (Yueqi Qiu and Xingyu Zhou). Any discrepancies were resolved by mutual discussion.

Data extraction and quality assessment

Two independent reviewers (Yueqi Qiu and Xingyu Zhou) compiled the data from the included studies, including the surname of the first author, publication year, participant ages, sex ratio, study country, study sample size, diagnostic criteria of MetS, severity in patients with AGA, prevalence of MetS in the AGA and control groups, and quality assessment score. The Newcastle–Ottawa Scale (NOS) was used to exactly evaluate the quality of each eligible study (12), which was based on the participant selection process, comparability, outcome assessment, and data processing. This scale varies from 0 to 9 stars. A score greater than 7 represents high quality, 5–7 indicated moderate quality, and a score less than 5 is classified as poor quality.

Statistical synthesis and analysis

Statistical analysis and graphics production were performed using Stata version 16.0 (StataCorp LLC, College Station, TX, USA). The odds ratios (OR) for patients with MetS were obtained for both the group with AGA and the control group, with a 95% confidence interval (95% CI), for each study and values were presented using forest plot graphs. The effect sizes of the mean difference in BMI, WC, TG, HDL-C, FBS, SBP, and DBP between patients with AGA and controls were calculated using the weighted mean difference (WMD) and 95% CI. The I^2 statistic was used to test heterogeneity of the included studies. I^2 identified the extent of true heterogeneity, using the formula $I^2 = [(Q - \text{degrees of freedom}) / Q \times 100\%]$ (13). The pooled effect sizes were obtained with the DerSimonian–Laird random-effects model if heterogeneity was high ($I^2 > 50\%$ or $p < 0.1$). Otherwise, if heterogeneity was low, a fixed-effect model was used (14). To further explore the origin of heterogeneity, we conducted meta-regression as well as subgroup and sensitivity analyses.

Publication bias was detected using Egger's regression asymmetry test and visualized using a funnel plot (15). p -values < 0.05 were considered statistically significant.

RESULTS

Study selection and characteristics

A total of 420 publications were identified in PubMed ($n = 98$), EMBASE ($n = 136$), Web of Science ($n = 120$), and The Cochrane Library ($n = 66$). After screening the title and abstracts, 383 studies were excluded for the following reasons: (a) duplicate article, (b) irrelevance to AGA or MetS, and (c) review, case report, and systematic review. Among the other 37 potentially eligible articles, 19 articles, including 1,342 patients with AGA and 1,189 controls, were included in this meta-analysis. The PRISMA flow diagram of study selection is shown in **Fig. 1**. All 19 articles were in English language and had a case-control design. The included studies were published or posted online between 2009 and 2021 and the studies were conducted in 9 countries on 4 continents. The sample size ranged from 68 to 202 participants. Information on sex was also presented in detail. The mean participant age was between 24.32 and 45.60 years in 17 studies with complete data for age. The scale used to describe the degree of baldness varied. In men, the Hamilton–Norwood scale was used in most studies and the Ebling scale was used in only 1 study. The degree of alopecia in women was classified using the Ludwig scale. In 14 of the 19 studies, MetS was diagnosed using the National Cholesterol Education Programme Adult Treatment Panel III (NCEP ATP III) criteria; 3 studies used criteria of the International Diabetes Foundation (IDF)

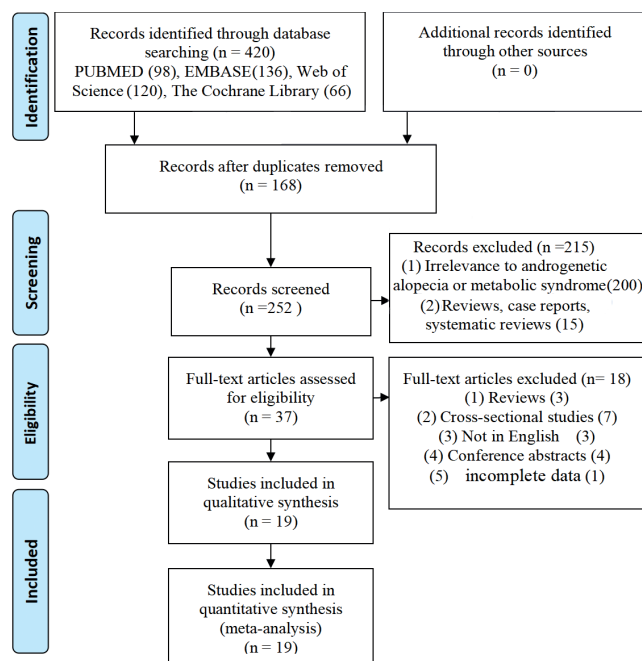


Fig. 1. Identification, inclusion, and exclusion of studies.

and one study applied both of these latter criteria. Quality assessment was determined using the NOS, and the scores of included articles ranged from 5 to 8 stars; thus, the included studies were considered to have relatively high quality. The characteristics of the studies and details regarding NOS quality assessment are shown in **Table I**.

Association of androgenetic alopecia with metabolic syndrome

In this study, 19 studies had adequate data for this analysis (6–8, 16–31). The pooled OR for the prevalence of MetS between the AGA and control group was 3.46 (95% CI: 2.38–5.05; $p < 0.001$) in the DerSimonian–Laird model, owing to significant heterogeneity ($I^2 = 63.7%$, $p < 0.001$; **Fig. 2**). Meta-regression was performed to explore the potential heterogeneity sources. The effects of different covariates on the prevalence of MetS are shown in **Table II**. The publication year, alopecia onset (early-onset or not), region, NOS score, and sample size

(≥ 100 cases or fewer) showed no associations with the observed ORs among study groups. Considering the existence of heterogeneity, further subgroup analysis on the basis of sex, early-onset, and region was performed. In 18 of 19 studies, the prevalence of MetS by sex was clearly reported (in 2,151 men and 220 women). The pooled OR in the 2 subgroups was 3.08 ($z = 3.134$, $p < 0.001$) and 7.34 ($z = 6.010$, $p < 0.001$), respectively. Early-onset AGA is defined as alopecia occurring and developing before age 36 years, which is at least stage III using the Hamilton–Norwood classification (20). Nine of these studies were conducted in early-onset AGA. The pooled ORs in patients without early-onset and with early-onset AGA were 3.13 ($z = 4.093$, $p < 0.001$) and 3.88 ($z = 3.131$, $p < 0.001$), respectively; this result revealed that younger age might have a closer relationship with MetS. In subgroup analysis by region, a significant correlation between AGA and MetS was found in Europe, Asia, Africa, and South America (**Fig. 3**).

Table I. Characteristics of included studies

Author, year	Region	Sex	AGA (n)	Control (n)	Age Group (mean \pm SD)	Degree in patients with AGA	Early-onset	Diagnostic criteria for MetS	Outcomes Group (Y/N)	NOS			Total
										Selection	Comparability	Exposure	
González-González, 2009 (26)	Mexico	M	80	80	AGA (25.64 \pm 4.16), Control (26.49 \pm 3.08)	H-N: \geq III	Y	IDF	AGA (16/64), Control (11/69)	4	1	2	7
Acibucu, 2010 (16)	Turkey	M	80	48	AGA (36.28 \pm 7.74), Control (35.14 \pm 6.54)	H-N: \geq III	N	NCEP ATP III	AGA (20/60), Control (5/43)	3	1	3	7
Arias-Santiago, 2010 (18)	Spain	M/F	77	77	Unclear	N: \geq III; Ludwig: \geq II	N	NCEP ATP III	AGA (42/35), Control (8/69)	2	1	2	5
Mumcuoglu, 2011 (6)	Turkey	M	50	40	AGA (24.32 \pm 2.68), Control (23.42 \pm 3.00)	H-N: \geq III	N	NCEP ATP III /IDF	AGA (4/46), Control (2/38)	3	1	3	7
Pengsalae, 2013 (28)	Thailand	M	40	40	AGA (47.38 \pm 6.94), Control (46.20 \pm 6.54)	H-N: \geq II	Y	NCEP ATP III	AGA (17/23), Control (7/33)	3	1	3	7
Bakry, 2014 (19)	Egypt	M	100	100	AGA (40.09 \pm 10.57), Control (37.85 \pm 9.48)	H-N: \geq III	N	NCEP ATP III	AGA (39/61), Control (19/81)	3	1	2	6
Chakrabarty, 2014 (22)	India	M	85	85	AGA (26.44 \pm 2.64), Control (25.65 \pm 3.19)	H-N: \geq I	Y	NCEP ATP III	AGA (37/48), Control (2/83)	3	1	2	6
Banger, 2015 (20)	India	M	100	100	AGA (25 \pm 5.36), Control (26.22 \pm 5.10)	H-N: \geq I	N	NCEP ATP III	AGA (22/68), Control (8/92)	3	1	3	7
Ozbas Gok, 2015 (8)	Turkey	M	74	42	AGA (32.14), Control (34.4) ^a	H-N: \geq II	N	IDF	AGA (11/63), Control (11/31)	3	1	2	6
Vayá, 2015 (31)	Spain	M	50	50	AGA (43 \pm 8), Control (43 \pm 10)	unclear	Y	NCEP ATP III	AGA (5/45), Control (4/46)	3	1	2	6
El Sayed, 2016 (24)	Egypt	F	45	45	AGA (27.8 \pm 7.9), Control (26.8 \pm 7.3)	Ludwig: \geq I	Y	NCEP ATP III	AGA (31/14), Control (9/36)	3	1	3	7
Ertas, 2016 (25)	Turkey	M	51	17	AGA (30.24 \pm 9.30; 29.35 \pm 8.22; 34.35 \pm 6.78) ^b , Control (29.41 \pm 6.82)	H-N: \geq II	Y	IDF	AGA (25/26), Control (2/15)	4	1	2	7
Alam, 2018 (17)	India	M	50	50	Unclear	H-N: \geq II	Y	NCEP ATP III	AGA (15/35), Control (6/44)	2	1	2	5
Behrangi, 2018 (21)	Iran	M/F	57	54	AGA (48 \pm 7), Control (49 \pm 7)	N: \geq II; Ludwig: \geq I	N	unclear	AGA (37/20), Control (29/25)	2	1	3	6
Dharam Kumar, 2018 (23)	India	M/F	100	100	AGA (33.560 \pm 8.533), Control (29.750 \pm 7.736)	N: \geq I; Ludwig: \geq I	N	NCEP ATP III	AGA (53/47), Control (17/83)	3	1	2	6
Swaroop, 2019 (7)	India	M	50	50	AGA (24.18 \pm 2.663), Control (25.12 \pm 2.344)	H-N: \geq III	Y	NCEP ATP III	AGA (15/35), Control (4/46)	3	1	2	6
Taheri, 2019 (30)	Iran	M	52	50	AGA (42.65 \pm 6.07), Control (45.60 \pm 6.33)	Ebling's scale: III–V	Y	NCEP ATP III	AGA (24/28), Control (8/42)	3	1	2	6
Mustafa, 2020 (27)	Egypt	M/F	100	60	AGA (33.82 \pm 6.6), Control (31.1 \pm 7.1)	H-N: \geq II; Ludwig: \geq I	N	NCEP ATP III	AGA (30/70), Control (6/54)	3	1	3	7
Sheikh, 2021 (29)	Pakistan	M	101	101	AGA (27.77 \pm 5.04), Control (27.38 \pm 4.91)	H-N: \geq III	N	NCEP ATP III	AGA (12/89), Control (3/98)	4	1	3	8

^aNo instructions for standard deviation (SD). ^bPatients with AGA were divided into 3 groups: group 1 (H-N: II, IIa, III, IIIa and IVa), group 2 (H-N: III, vertex and stage IV), and group 3 (H-N: V, Va, VI and VII).
N: Hamilton–Norwood; NCEP ATP III: National Cholesterol Education Programme Adult Treatment Panel III; IDF: International Diabetes Foundation; NOS: Newcastle–Ottawa Scale; AGA, androgenetic alopecia; MetS: metabolic syndrome; M: male; F: female; SD: standard deviation; Y: yes; N: no.

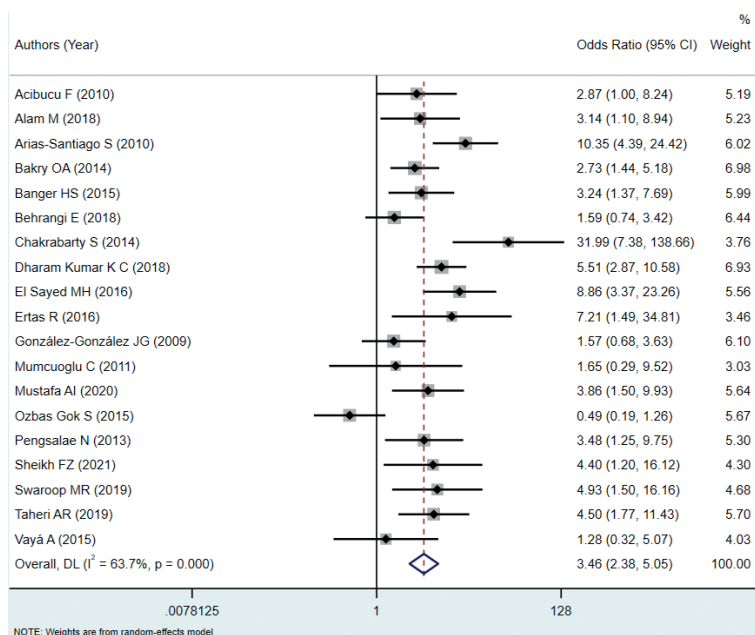


Fig. 2. Forest plot of the association between androgenetic alopecia and metabolic syndrome (6–8, 16–31).

Metabolic profile in patients with androgenetic alopecia

NCEP ATP III is the most commonly-used criteria for classifying MetS among adults (32). The detailed items are as follows: (i) WC ≥ 102 cm in men, ≥ 88 cm in women, (ii) TG value ≥ 150 mg/dl, (iii) HDL-C < 40 mg/dl in men, < 50 mg/dl in women, (iv) FBS ≥ 110 mg/dl, and (v) SBP ≥ 130 mmHg, or DBP ≥ 85 mmHg. Any 3 of the above 5 constitute a diagnosis of MetS (33). To further explore the relationship between AGA and MetS and to assess various established indices of metabolic function, we also conducted meta-analysis of the available data; namely, BMI, WC, TG, HDL-C, FBS, SBP, and DBP. Compared with controls, patients with AGA showed slightly poor metabolic profiles and notably high BMI, WC, TG, FBS, SBP, and DBP values, with low HDL-C levels. Table III presents the detailed data of this meta-analysis.

Sensitivity analyses

Sensitivity analysis was conducted to verify the stability of the above analyses with regard to comorbidity of MetS between patients with AGA and controls. No obvious

Table II. Effects of different covariates on the prevalence of metabolic syndrome

Covariates	Coefficient	Standard error	T-statistic	p-value	95% CI
Years	0.008	0.065	0.12	0.908	(-0.133, 0.148)
Onset	0.376	0.508	0.74	0.472	(-0.721, 1.473)
Region	0.015	0.279	0.05	0.958	(-0.588, 0.618)
NOS	-415	0.298	-1.39	0.187	(-1.060, 0.229)
Sample size	-0.265	0.803	-0.33	0.746	(-2.000, 1.469)

95% CI: 95% confidence interval; NOS: Newcastle-Ottawa Scale.

changes were observed when any 1 study was removed (Fig. 4).

Publication bias

The asymmetry of the funnel plot, which showed an asymmetrical shape, was statistically assessed using Egger’s regression test. The results suggested no significant publication bias overall (t=0.52, p=0.611; Fig. 5).

DISCUSSION

Although AGA itself is associated with a lower risk of systemic disorders, growing evidence has revealed the relationship between hair loss and other diseases. In the 1970s, AGA was first confirmed to be related to cardiovascular disease (34). In subsequent years, a great many other relevant diseases have been reported, including metabolic-related illness, such as MetS, hypertension, dyslipidaemia, and obesity (35). However, the pathological

mechanisms underlying the relationships between AGA and these metabolic disorders have not yet been clearly established. The current findings support our hypothesis that there are several factors related to this relationship. Androgens, particularly DHT, play a major role in the pathogenesis of AG by, binding to androgen receptors on androgen-sensitive follicles, which leads to follicular miniaturization (36). High levels of serum androgen also have a potential role in the development of hypertension by binding to receptors in the arterial wall, thereby promoting endothelial proliferation of smooth muscle cells (37). In addition, androgens interfere with lipid metabolism. Decreased HDL-C concentrations and increased concentrations of total cholesterol are observed with high levels of testosterone, which contribute to the development of dyslipidaemia (6). Elevated aldosterone in women with early-onset AGA has been reported. Hypertension, and the enhancement of peripheral sensitivity to androgens, may result from increased mineralocorticoid production (18). Moreover, IR and hyperinsulinemia participate in the development of AGA by producing local androgens from cholesterol and enhancing the local conversion of testosterone to DHT. Furthermore, IR underpins a number of metabolic diseases, especially type 2 diabetes and MetS (38).

The pooled results of the current study indicated that patients with AGA have 3.46 times the risk of MetS as in controls, which is similar to the outcome (OR 2.70, 95% CI: 1.67–4.37) reported by Wu et al. (9) in 2014. However, the significantly higher pooled OR for MetS in women was quite different from the lower OR in the above study. The reason could be owing to the smaller number of female participants and the fact that only

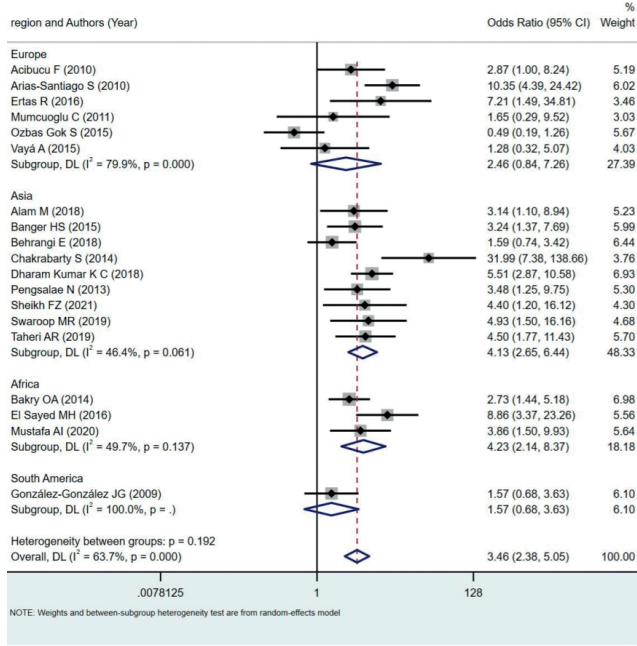
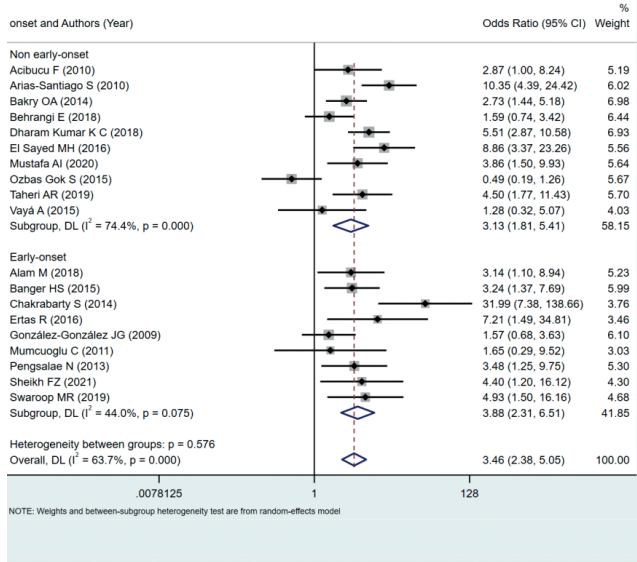
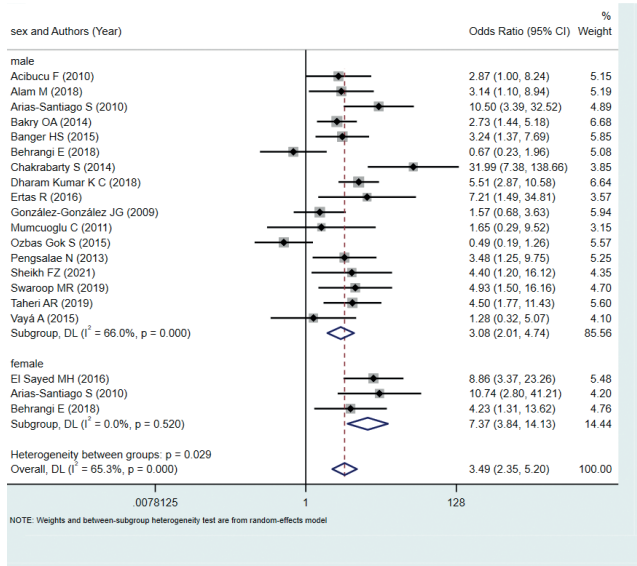


Fig. 3. Forest plot of the association between androgenetic alopecia and metabolic syndrome based on the subgroup analysis by the sex of patients, onset of alopecia and region of participants (6–8, 16–31). 95% CI: 95% confidence interval.

case-control studies (and no cross-sectional studies) were included in our analysis. Also, female patients with polycystic ovary syndrome (PCOS) have a higher risk of AGA and MetS (39), which might be another explanation for this result. It is possible that, in some studies, women were not screened for PCOS or these cases were not reported. Another meta-analysis of 7,394 participants in studies before 2017 showed that patients with AGA had a greater chance of developing MetS (OR 2.59, 95% CI 1.51–4.44; $p < 0.0005$; $I^2 = 86\%$). The conclusions of that meta-analysis showed that female patients and patients with early-onset AGA had significant associations with MetS (OR 5.59 and OR 3.69, respectively), which was consistent with our research (10). In Trieu's research (11), alopecia was positively associated with an increased risk of hyperinsulinemia (OR 1.97, 95% CI: 1.20–3.21), IR (OR 4.88, 95% CI: 2.05–11.64), and MetS (OR 4.49, 95% CI: 2.36–8.53). Moreover, individuals with alopecia had higher serum cholesterol levels, higher TG levels, and higher SBP and DBP values. However, in this study, they did not conduct a specific subgroup analysis of AGA. In terms of the onset of alopecia, heterogeneity was reduced in the early-onset group compared with the aggregate data. In a recent study, Cannarella et al. (40) proved the presence of slightly worse glycaemic and lipid profiles in early-onset AGA, which was in line with the strong association between early-onset AGA and MetS. However, they did not find any evident differences in HDL-C between patients with AGA and controls and also did not quantify the strength of the association between MetS and patients with early-onset AGA diagnosed with MetS, who would be likely to experience a cardiovascular event within the subsequent 5–10 years (5). It is necessary to examine metabolism-related indicators in patients with AGA to prevent long-term consequences with respect to cardiovascular abnormalities.

There are several special aspects in our review. The included studies were all case-control studies; similar past meta-analyses usually include cross-sectional studies (9, 11). Cross-sectional studies were excluded because patients and controls were not matched for age and sex, which could be a source of heterogeneity. To obtain more credible results, we eliminated cross-sectional studies with a lower level of evidence and used the NOS score. The NOS is a combination of case-control studies and cohort studies, derived from the randomized controlled trial evaluation method, and can be applied to the systematic evaluation of non-randomized controlled trials. The current study also included meta-regression and subgroup and sensitivity analysis for further heterogeneity screening. In addition to the pooled OR, WMDs of metabolic indicators (BMI, WC, TG, HDL-C, FBS, SBP,

Table III. Summary of the meta-analyses for each indicator of the National Cholesterol Education Programme Adult Treatment Panel III (NCEP ATP III)

Analysis item	Studies <i>n</i>	Heterogeneity		Analysis model	Statistical method	WMD (95% CI)	<i>p</i> -value
		<i>I</i> ²	<i>p</i> -value				
Body mass index (kg/cm ²)	12	73.0%	< 0.001	Random effect	DerSimonian-Laird method	1.22 (0.62, 1.81)	0.002
Waist circumference (cm)	13	82.6%	< 0.001	Random effect	DerSimonian-Laird method	4.53 (2.53, 6.53)	0.001
Triglyceride (mg/dl)	15	92.2%	< 0.001	Random effect	DerSimonian-Laird method	19.33 (7.16, 31.49)	0.025
High-density lipoprotein cholesterol (mg/dl)	13	84.0%	< 0.001	Random effect	DerSimonian-Laird method	-2.57 (-4.29, -0.86)	0.025
Fasting blood sugar (mg/dl)	13	84.1%	< 0.001	Random effect	DerSimonian-Laird method	6.51 (2.85, 10.17)	0.011
Systolic blood pressure (mmHg)	13	94.1%	< 0.001	Random effect	DerSimonian-Laird method	7.94 (4.90, 10.98)	0.003
Diastolic blood pressure (mmHg)	13	79.0%	< 0.001	Random effect	DerSimonian-Laird method	4.66 (3.41, 5.91)	< 0.001

WMD: weighted mean difference; 95% CI: 95% confidence interval.

and DBP) were calculated between patients and controls. The results of our meta-analysis showed statistical significance, which confirmed a strong correlation between AGA and MetS.

The current meta-analysis also had several limitations. Based on observational analytic studies, our review was more subject to bias than systemic reviews with other designs, especially randomized controlled trials,

although the age-matched controls used in case-control studies could mitigate potential bias in the results owing to age. Therefore, the ideal study design would be a cohort study, which prospectively follows both AGA and healthy individuals for the development of MetS or its relevant risk factors. We did not include other risk factors that may contribute to the diagnosis of AGA and MetS, especially family history and smoking status. Moreover, different diagnostic criteria for MetS may also be sources of heterogeneity. The severity of AGA was not included in subgroup analysis owing to the absence of some raw data. Further studies with larger sample sizes are still needed.

In conclusion, in our meta-analysis, AGA was remarkably associated with an increased OR of MetS, which was greater in women and in patients with early-onset AGA and African ethnicity, based on subgroup analysis. We also demonstrated that patients with AGA had significantly poor metabolic profiles, such as BMI, waist circumference, triglyceride, HDL-C, FBS, SBP, and DBP values, compared with controls. However, more rigorously designed studies with larger sample sizes are necessary in future studies. The results of this study highlight the need to screen metabolism-related indicators in patients with AGA to prevent long-term consequences with respect to cardiovascular abnormalities.

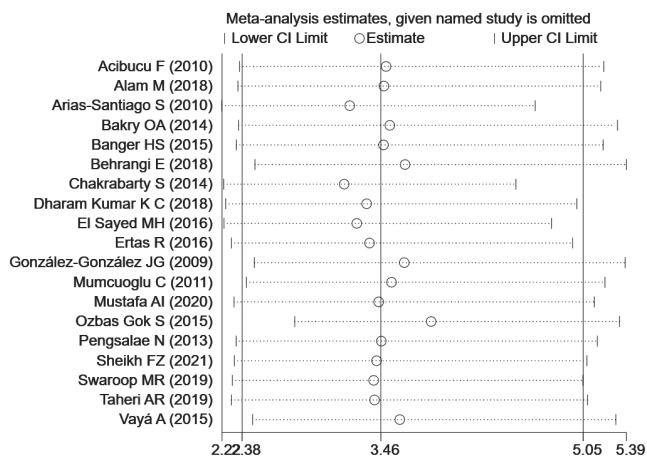


Fig. 4. Sensitivity analysis for the strength of the association between androgenetic alopecia and metabolic syndrome (6–8, 16–31). Plot of sensitivity analysis, excluding 1 study at a time, and the pooled estimate for the remaining studies. 95% CI: 95% confidence interval.

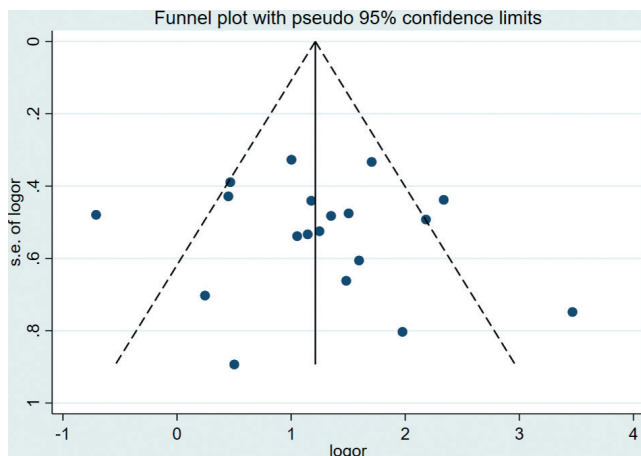


Fig. 5. Begg's funnel plot. 95% CI: 95% confidence interval.

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The data supporting this meta-analysis were extracted from previously published studies and reported datasets, which were also cited. The processed data are available from the corresponding author upon request.

The authors have no conflicts of interest to declare.

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