Androgenetic Alopecia: Clinical Grading and Associated Metabolic Risk Factors - A Hospital-Based Case-Control Study

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ABSTRACT

Background
Androgenetic alopecia (AGA) is the commonest form of patterned hair loss and has increasingly been linked to insulin resistance, dyslipidaemia, and the metabolic syndrome. The strength of this association in young Indian patients remains incompletely defined, and clinical severity has not been uniformly correlated with metabolic burden. The present study was undertaken to assess the prevalence and pattern of metabolic abnormalities in patients with AGA and to correlate them with clinical grade.

Methods
A hospital-based case-control study was conducted over eighteen months in the dermatology outpatient department. One hundred patients aged 18 to 50 years with clinically diagnosed AGA were enrolled as cases, and 100 age-matched and sex-matched individuals without AGA served as controls. Severity was graded using the Hamilton-Norwood scale for males and the Ludwig scale for females. Anthropometric measurements, blood pressure, fasting plasma glucose, fasting insulin, lipid profile, and HOMA-IR were recorded. Metabolic syndrome was defined per modified NCEP-ATP III criteria. Data were analysed using SPSS v26.0.

Results
Metabolic syndrome was present in 38% of cases versus 17% of controls ($p = 0.001$). Mean HOMA-IR was significantly higher in cases (2.94 ± 1.18) than controls (1.62 ± 0.71; $p < 0.001$). Cases showed higher waist circumference, triglycerides, and fasting glucose, and lower HDL-C (all $p < 0.05$). Severity of AGA correlated positively with HOMA-IR ($r = 0.512$, $p < 0.001$) and with the number of metabolic syndrome components present.

Conclusion
AGA, particularly of early onset and higher clinical grade, is significantly associated with insulin resistance and the metabolic syndrome. Patients presenting with AGA warrant systematic metabolic screening as part of routine dermatological practice.

Keywords: Androgenetic Alopecia, Hamilton-Norwood, Ludwig Grade, Insulin Resistance, Metabolic Syndrome, HOMA-IR.

INTRODUCTION

Androgenetic alopecia (AGA) is the most prevalent form of progressive, non-scarring hair loss in both sexes and is characterised by miniaturisation of terminal scalp hairs in genetically predisposed androgen-sensitive follicles.[1] In men, the condition typically expresses as bitemporal recession and vertex thinning, while in women it manifests as diffuse central thinning with preservation of the frontal hairline.[2] Epidemiological surveys indicate that nearly 50% of men and 25% of women will exhibit clinically apparent AGA by the fifth decade of life, with prevalence rising steadily with advancing age.[1,3] Although AGA was historically regarded as a cosmetic disorder, mounting evidence over the last two decades has reframed it as a phenotypic marker of systemic metabolic disturbance, drawing parallels with other dermatological signs of insulin resistance.[4]

The pathogenesis of AGA is multifactorial, with androgen sensitivity, polygenic inheritance, and follicular microinflammation contributing to progressive shortening of the anagen phase and reduction in hair shaft diameter.[2] Dihydrotestosterone (DHT), produced locally by the action of 5-alpha reductase on testosterone, binds to androgen receptors in dermal papilla cells and triggers a cascade culminating in follicular miniaturisation.[5] The same androgen axis is implicated in conditions characterised by hyperinsulinaemia, including polycystic ovary syndrome (PCOS) in women and early-onset central obesity in men, suggesting that AGA may share a common endocrine and metabolic background with these disorders.[5]

The first formal description of an association between premature scalp baldness and insulin resistance was provided by Matilainen and colleagues, who demonstrated that men with early AGA exhibited higher fasting insulin levels and a greater prevalence of insulin resistance than age-matched controls without alopecia.[4] This pioneering observation prompted a wave of investigations examining the link between AGA and the constituent elements of the metabolic syndrome, namely central obesity, atherogenic dyslipidaemia, hypertension, and dysglycaemia.[6,7] Su and Chen, in a large community-based survey, reported that men with moderate to severe vertex-pattern AGA had significantly higher odds of meeting the criteria for metabolic syndrome than men without alopecia, even after adjustment for age and family history.[6] Comparable observations have been recorded in cohorts from Spain, Korea, Mexico, Egypt, and India, lending substantial geographical breadth to the association.[7,8]

Insulin resistance is now considered the unifying pathophysiological link between AGA and metabolic syndrome. Hyperinsulinaemia is hypothesised to reduce hepatic sex hormone-binding globulin (SHBG) production, thereby increasing free testosterone and amplifying the conversion to DHT in target follicles.[5] Insulin and insulin-like growth factor-1 (IGF-1) also exert vasoactive and proinflammatory effects on the perifollicular microvasculature, contributing to follicular ischaemia and microinflammation.[5,9] Additionally, chronic low-grade inflammation associated with visceral adiposity may aggravate androgen-mediated follicular regression, providing a mechanistic bridge between metabolic dysfunction and patterned hair loss.[9]

Beyond the metabolic syndrome itself, AGA has been investigated as a surrogate marker of subclinical atherosclerosis and cardiovascular risk. A meta-analysis by Trieu and Eslick concluded that vertex-pattern baldness was associated with a statistically significant increase in the risk of coronary heart disease, with the magnitude of association rising in proportion to severity of hair loss.[10] Yi and colleagues demonstrated a sex-specific gradient, with the risk of metabolic syndrome rising in tandem with Norwood-Hamilton stage in men and Ludwig grade in women.[8] Such findings underscore the potential value of AGA as a readily visible, low-cost clinical sign that might prompt early metabolic screening and lifestyle counselling.

Despite this growing body of evidence, several issues remain unresolved. Studies have reported heterogeneous odds ratios, varying age cut-offs for defining early AGA, and inconsistent use of grading systems. Indian data, while gradually accruing, are largely confined to small single-centre samples, and few studies have systematically correlated severity of AGA, graded with standardised scales, against a comprehensive panel of anthropometric, glycaemic, and lipid parameters in both sexes drawn from the same catchment population. The phenotypic overlap between early AGA in men and PCOS in women, postulated by Sanke and colleagues, also raises the question of whether AGA itself should be regarded as a male equivalent of PCOS, with a shared insulin-resistant substrate.[5]

The present study was therefore designed to characterise the clinical grading of AGA in patients attending a tertiary care dermatology clinic and to evaluate the prevalence and severity of associated metabolic risk factors compared with matched controls. By stratifying outcomes according to standardised severity grades, the study aimed to establish whether the burden of metabolic disturbance scales with the clinical severity of hair loss. The findings are expected to inform routine dermatological practice by highlighting the need for opportunistic metabolic screening in patients presenting with AGA, particularly those of younger age and higher grade.

AIMS AND OBJECTIVES

Aim

The aim of the study was to determine the prevalence and pattern of metabolic risk factors in patients with clinically diagnosed androgenetic alopecia and to correlate the metabolic profile with clinical severity grade.

Primary Objective

The primary objective was to compare the prevalence of the metabolic syndrome, defined by modified NCEP-ATP III criteria, in patients with androgenetic alopecia against age-matched and sex-matched controls without alopecia.

Secondary Objectives

The secondary objectives were to grade androgenetic alopecia in male patients using the Hamilton-Norwood scale and in female patients using the Ludwig scale; to compare anthropometric, glycaemic, and lipid parameters between cases and controls; to assess insulin resistance using the homeostasis model assessment of insulin resistance (HOMA-IR); and to correlate the severity of androgenetic alopecia with individual metabolic parameters and with the number of metabolic syndrome components present.

MATERIALS AND METHODS

Study Design and Setting

A hospital-based, cross-sectional, case-control study was conducted in the Department of Dermatology, Venereology and Leprosy of a tertiary care teaching hospital over a period of eighteen months following institutional ethics committee approval. Written informed consent was obtained from all participants prior to enrolment.

Sample Size

The sample size was calculated assuming a prevalence of metabolic syndrome of approximately 35% in patients with androgenetic alopecia and 15% in the general adult population, with 80% power and a two-sided alpha of 0.05, yielding a minimum of 88 participants in each group. To allow for incomplete data and dropouts, the final sample was rounded to 100 cases and 100 age-matched and sex-matched controls.

Inclusion Criteria

Patients aged between 18 and 50 years of either sex, with a clinical diagnosis of androgenetic alopecia of any grade, who were willing to provide written informed consent, were included as cases. Age-matched and sex-matched individuals attending the outpatient department for non-dermatological complaints, with no clinical evidence of alopecia and willing to provide consent, were enrolled as controls.

Exclusion Criteria

Patients with other forms of hair loss including alopecia areata, telogen effluvium, cicatricial alopecia, and trichotillomania were excluded. Individuals with known endocrine disorders, including thyroid dysfunction, Cushing syndrome, and clinically diagnosed polycystic ovary syndrome on therapy, were also excluded. Participants receiving systemic medications known to affect lipid or glucose metabolism, such as corticosteroids, antipsychotics, oral contraceptives, antiretrovirals, statins, and oral hypoglycaemic agents, were excluded. Pregnant or lactating women, patients with a history of malignancy, chronic liver or renal disease, and those who had received topical or systemic therapy for alopecia within the preceding six months were also excluded.

Clinical Assessment and Grading

A detailed history was elicited covering age of onset of hair loss, family history of AGA in first-degree relatives, duration of symptoms, dietary habits, physical activity, smoking, alcohol intake, and concomitant medications. General physical examination included measurement of height, weight, waist circumference, hip circumference, and blood pressure. Body mass index was calculated as weight in kilograms divided by height in metres squared. Waist circumference was measured midway between the lower costal margin and the iliac crest in the standing position at the end of normal expiration. Blood pressure was recorded after at least five minutes of rest, in the sitting position, on two separate occasions, using a calibrated mercury sphygmomanometer.

Severity of androgenetic alopecia in male patients was graded using the modified Hamilton-Norwood scale (Grades I to VII), and in female patients using the Ludwig scale (Grades I to III). Photographic documentation was obtained for record purposes after due consent. Early-onset AGA was defined as clinical onset before the age of 30 years.

Laboratory Investigations

After an overnight fast of at least eight hours, venous blood samples were collected for fasting plasma glucose, fasting serum insulin, total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Plasma glucose was estimated by the hexokinase method, lipid parameters by enzymatic methods, and serum insulin by chemiluminescent immunoassay. Insulin resistance was estimated using the homeostasis model assessment (HOMA-IR), calculated as the product of fasting insulin (μIU/mL) and fasting glucose (mmol/L) divided by 22.5. A HOMA-IR value of 2.5 or above was taken to indicate insulin resistance.

Definition of Metabolic Syndrome

Metabolic syndrome was diagnosed when three or more of the following five criteria, based on the modified NCEP-ATP III definition for the South Asian population, were fulfilled: (a) waist circumference greater than 90 cm in men or greater than 80 cm in women; (b) triglycerides at or above 150 mg/dL or specific treatment; (c) HDL-C less than 40 mg/dL in men or less than 50 mg/dL in women or specific treatment; (d) systolic blood pressure at or above 130 mmHg or diastolic blood pressure at or above 85 mmHg or antihypertensive therapy; and (e) fasting plasma glucose at or above 100 mg/dL or established diagnosis of type 2 diabetes mellitus.

Statistical Analysis

Data were entered into a Microsoft Excel master chart and analysed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean and standard deviation, and categorical variables as frequencies and percentages. The Kolmogorov-Smirnov test was used to assess the normality of distribution. Between-group comparisons for continuous variables were performed using the unpaired Student t-test for normally distributed data and the Mann-Whitney U test for non-normal data. Categorical variables were compared using the Pearson chi-square test or Fisher exact test, as appropriate. Spearman rank correlation was used to assess the relationship between severity of AGA and metabolic parameters. Multivariate logistic regression was performed to identify independent predictors of AGA, with adjustment for age, sex, family history, smoking, and body mass index. A two-sided p-value less than 0.05 was considered statistically significant.

RESULTS

Demographic and Baseline Characteristics

A total of 200 participants were enrolled, comprising 100 cases of androgenetic alopecia and 100 age-matched and sex-matched controls. Cases and controls were comparable for age and sex distribution (Table 1). The mean age of cases was 32.4 ± 7.8 years compared with 31.9 ± 8.1 years among controls ($p = 0.654$). Males constituted 78% and females 22% of each group. Early-onset AGA (clinical onset before 30 years of age) was observed in 52 (52.0%) cases. The mean body mass index was significantly higher in cases (27.8 ± 3.6 kg/m²) than controls (24.9 ± 3.1 kg/m²; $p < 0.001$). A family history of AGA was reported by 64 (64.0%) cases compared with 12 (12.0%) controls ($p < 0.001$). Current smoking was more frequent among cases (36.0%) than controls (22.0%; $p = 0.029$), as was sedentary lifestyle (62.0% versus 44.0%; $p = 0.011$). Alcohol consumption did not differ significantly between groups ($p = 0.131$).

Parameter	Cases (n=100)	Controls (n=100)	p-value
Mean age (years)	32.4 ± 7.8	31.9 ± 8.1	0.654
Age range (years)	18–49	18–50	—
Male, n (%)	78 (78.0)	78 (78.0)	1.000
Female, n (%)	22 (22.0)	22 (22.0)	1.000
Early onset (<30 years), n (%)	52 (52.0)	—	—
BMI (kg/m²)	27.8 ± 3.6	24.9 ± 3.1	<0.001
Family history of AGA, n (%)	64 (64.0)	12 (12.0)	<0.001
Current smoker, n (%)	36 (36.0)	22 (22.0)	0.029
Alcohol use, n (%)	28 (28.0)	19 (19.0)	0.131
Sedentary lifestyle, n (%)	62 (62.0)	44 (44.0)	0.011

Table 1. Demographic and baseline characteristics of cases and controls

Values are mean ± SD unless otherwise indicated. p-values from Student t-test (continuous) or chi-square test (categorical). AGA, androgenetic alopecia; BMI, body mass index.

Clinical Grading of Androgenetic Alopecia

Among the 78 male cases, the modal Hamilton-Norwood grade was IV, observed in 22 (28.2%) patients, followed by Grade III in 18 (23.1%), Grade V in 14 (17.9%), Grade II in 14 (17.9%), Grade VI in 8 (10.3%), and Grade VII in 2 (2.6%). Among the 22 female cases, Ludwig Grade I was the commonest, present in 12 (54.5%), followed by Grade II in 8 (36.4%) and Grade III in 2 (9.1%). For the purpose of stratified analysis in males, Grades II–III were combined as mild, Grades IV–V as moderate, and Grades VI–VII as severe (Table 2).

Grading scale and grade		Number	Percentage	Cumulative %
Hamilton-Norwood (males, n=78)	Grade II	14	17.9	17.9
	Grade III	18	23.1	41.0
	Grade IV	22	28.2	69.2
	Grade V	14	17.9	87.1
	Grade VI	8	10.3	97.4
	Grade VII	2	2.6	100.0
Ludwig (females, n=22)	Grade I	12	54.5	54.5
	Grade II	8	36.4	90.9
	Grade III	2	9.1	100.0

Table 2. Distribution of cases according to Hamilton-Norwood and Ludwig grading

Anthropometric and Metabolic Parameters

Cases demonstrated a significantly more adverse anthropometric and metabolic profile than controls across nearly every variable measured (Table 3). Mean waist circumference was 94.6 ± 9.8 cm in cases versus 86.2 ± 8.4 cm in controls ($p < 0.001$), and the mean waist-to-hip ratio was 0.94 ± 0.06 versus 0.88 ± 0.07 ($p < 0.001$). Systolic and diastolic blood pressures were both significantly elevated in cases (128.4 ± 13.6 mmHg and 82.6 ± 8.8 mmHg) compared with controls (121.8 ± 11.4 mmHg and 78.4 ± 8.2 mmHg, with $p < 0.001$ and $p = 0.001$ respectively). Fasting plasma glucose was higher in cases (102.4 ± 18.6 mg/dL) than controls (91.2 ± 12.4 mg/dL; $p < 0.001$), as was fasting serum insulin (13.6 ± 5.2 versus 8.4 ± 3.4 µIU/mL; $p < 0.001$). HOMA-IR was significantly elevated in cases (2.94 ± 1.18) compared with controls (1.62 ± 0.71; $p < 0.001$). The lipid profile showed higher total cholesterol (198.4 ± 32.6 versus 184.2 ± 28.8 mg/dL; $p = 0.001$), higher triglycerides (168.4 ± 56.8 versus 124.6 ± 42.4 mg/dL; $p < 0.001$), higher LDL-C (126.4 ± 28.6 versus 114.8 ± 24.6 mg/dL; $p = 0.003$), and lower HDL-C (38.6 ± 8.2 versus 46.4 ± 9.4 mg/dL; $p < 0.001$) in cases.

Parameter	Cases (n=100)	Controls (n=100)	p-value
Waist circumference (cm)	94.6 ± 9.8	86.2 ± 8.4	<0.001
Waist-to-hip ratio	0.94 ± 0.06	0.88 ± 0.07	<0.001
Systolic BP (mmHg)	128.4 ± 13.6	121.8 ± 11.4	<0.001
Diastolic BP (mmHg)	82.6 ± 8.8	78.4 ± 8.2	0.001
Fasting plasma glucose (mg/dL)	102.4 ± 18.6	91.2 ± 12.4	<0.001
Fasting insulin (µIU/mL)	13.6 ± 5.2	8.4 ± 3.4	<0.001
HOMA-IR	2.94 ± 1.18	1.62 ± 0.71	<0.001
Total cholesterol (mg/dL)	198.4 ± 32.6	184.2 ± 28.8	0.001
Triglycerides (mg/dL)	168.4 ± 56.8	124.6 ± 42.4	<0.001

HDL-C (mg/dL)	38.6 ± 8.2	46.4 ± 9.4	<0.001
LDL-C (mg/dL)	126.4 ± 28.6	114.8 ± 24.6	0.003

Table 3. Comparison of anthropometric and metabolic parameters between cases and controls

Values are mean ± SD. BP, blood pressure; HOMA-IR, homeostasis model assessment of insulin resistance; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

Prevalence of metabolic syndrome and its components

The prevalence of metabolic syndrome, as defined by the modified NCEP-ATP III criteria, was 38.0% in cases compared with 17.0% in controls, with the difference being highly significant ($\chi^2 = 11.18$, $p = 0.001$; odds ratio 3.00, 95% confidence interval 1.55 to 5.81). Every individual component of the metabolic syndrome was more frequent among cases than controls. Central obesity was present in 56 (56.0%) cases versus 32 (32.0%) controls ($p = 0.001$). Hypertriglyceridaemia was documented in 44 (44.0%) cases compared with 24 (24.0%) controls ($p = 0.003$). Low HDL-C was observed in 48 (48.0%) cases versus 26 (26.0%) controls ($p = 0.001$). Elevated blood pressure was present in 32 (32.0%) cases compared with 18 (18.0%) controls ($p = 0.022$), and impaired fasting glucose was noted in 28 (28.0%) cases versus 12 (12.0%) controls ($p = 0.005$). Insulin resistance, defined as HOMA-IR of 2.5 or above, was present in 58 (58.0%) cases compared with 22 (22.0%) controls ($p < 0.001$) (Table 4).

Component	Cases, n (%)	Controls, n (%)	p-value
Central obesity	56 (56.0)	32 (32.0)	0.001
Hypertriglyceridaemia	44 (44.0)	24 (24.0)	0.003
Low HDL-C	48 (48.0)	26 (26.0)	0.001
Elevated blood pressure	32 (32.0)	18 (18.0)	0.022
Impaired fasting glucose	28 (28.0)	12 (12.0)	0.005
Metabolic syndrome (≥3 components)	38 (38.0)	17 (17.0)	0.001
Insulin resistance (HOMA-IR ≥2.5)	58 (58.0)	22 (22.0)	<0.001

Table 4. Prevalence of the metabolic syndrome and its components in cases and controls

Severity of AGA and Metabolic Profile

Among male cases, the metabolic profile worsened progressively with rising Hamilton-Norwood grade (Table 5). Mean BMI rose from 26.2 ± 3.1 kg/m² in mild AGA (Grades II–III) to 28.4 ± 3.4 kg/m² in moderate AGA (Grades IV–V) and 30.1 ± 3.8 kg/m² in severe AGA (Grades VI–VII), with a significant linear trend ($p = 0.002$). Mean HOMA-IR similarly increased from 2.34 ± 0.94 in the mild group to 3.06 ± 1.12 in the moderate group and 3.84 ± 1.28 in the severe group ($p < 0.001$). The prevalence of metabolic syndrome among male cases rose from 18.8% in the mild group to 44.4% in the moderate group and 70.0% in the severe group ($p < 0.001$ for trend). The mean number of metabolic syndrome components was 1.6 ± 0.9 in the mild group, 2.4 ± 1.1 in the moderate group, and 3.2 ± 1.2 in the severe group ($p < 0.001$). Mean fasting glucose, triglycerides, and waist circumference all increased significantly with grade, while HDL-C declined significantly (all $p < 0.01$).

Variable	Mild (II–III) n=32	Moderate (IV–V) n=36	Severe (VI–VII) n=10	p-value (trend)
BMI (kg/m²)	26.2 ± 3.1	28.4 ± 3.4	30.1 ± 3.8	0.002

Waist circumference (cm)	90.4 ± 8.6	95.8 ± 9.2	101.6 ± 9.8	<0.001
Fasting glucose (mg/dL)	96.2 ± 14.2	104.4 ± 18.6	112.6 ± 22.4	0.004
Triglycerides (mg/dL)	146.8 ± 48.2	172.4 ± 56.4	204.6 ± 64.8	<0.001
HDL-C (mg/dL)	41.6 ± 7.8	37.8 ± 7.6	34.2 ± 8.4	0.008
HOMA-IR	2.34 ± 0.94	3.06 ± 1.12	3.84 ± 1.28	<0.001
MetS, n (%)	6 (18.8)	16 (44.4)	7 (70.0)	<0.001
Mean MetS components	1.6 ± 0.9	2.4 ± 1.1	3.2 ± 1.2	<0.001

Table 5. Stratification of metabolic parameters by Hamilton-Norwood grade among male cases (n=78)

p-values for linear trend across severity strata (one-way ANOVA for continuous; chi-square for trend for categorical)

Correlation and Multivariate Analysis

Spearman rank correlation analysis demonstrated a significant positive correlation between the severity grade of androgenetic alopecia and HOMA-IR ($r = 0.512$, $p < 0.001$), waist circumference ($r = 0.418$, $p < 0.001$), fasting plasma glucose ($r = 0.394$, $p < 0.001$), triglycerides ($r = 0.382$, $p < 0.001$), and the total number of metabolic syndrome components present ($r = 0.486$, $p < 0.001$). A significant inverse correlation was observed between severity grade and HDL-C ($r = -0.346$, $p = 0.002$). On multivariate logistic regression with AGA as the dependent variable and adjustment for age, sex, family history, smoking, and BMI, insulin resistance (HOMA-IR ≥2.5) emerged as the strongest independent metabolic predictor of AGA, with an adjusted odds ratio of 3.42 (95% confidence interval 1.74 to 6.73; $p < 0.001$). Family history of AGA carried the largest overall effect size (adjusted odds ratio 8.96, 95% confidence interval 3.92 to 20.48; $p < 0.001$). BMI of 25 kg/m² or above (adjusted odds ratio 2.18; $p = 0.034$), hypertriglyceridaemia (adjusted odds ratio 2.06; $p = 0.045$), and low HDL-C (adjusted odds ratio inverse 0.46; $p = 0.038$) also retained statistical significance, whereas smoking did not ($p = 0.115$) (Table 6).

Metabolic parameter	Spearman r	p-value	Adjusted OR (95% CI), p
Waist circumference	0.418	<0.001	2.18 (1.06–4.48), 0.034
Fasting plasma glucose	0.394	<0.001	1.94 (0.96–3.92), 0.064
Triglycerides	0.382	<0.001	2.06 (1.02–4.18), 0.045
HDL-C	−0.346	0.002	0.46 (0.22–0.96), 0.038
HOMA-IR	0.512	<0.001	3.42 (1.74–6.73), <0.001
Number of MetS components	0.486	<0.001	—
Family history of AGA	—	—	8.96 (3.92–20.48), <0.001
Current smoking	—	—	1.84 (0.86–3.92), 0.115

Table 6. Correlation and multivariate logistic regression of metabolic parameters with AGA

Adjusted odds ratios from multivariate logistic regression, adjusted for age, sex, family history, smoking, and BMI. Dependent variable: presence of androgenetic alopecia

DISCUSSION

The present case-control study provides further evidence that androgenetic alopecia is associated with a measurable and clinically meaningful burden of metabolic disturbance. Cases exhibited significantly higher waist circumference, blood pressure, fasting glucose, fasting insulin, HOMA-IR, total cholesterol, triglycerides, and LDL-C, and significantly lower HDL-

C than age-matched and sex-matched controls. The prevalence of the metabolic syndrome was more than double in cases compared with controls (38.0% versus 17.0%), and the severity of AGA correlated positively and significantly with HOMA-IR and the number of metabolic syndrome components.

These findings are consistent with the foundational observations of Matilainen and colleagues, who first demonstrated higher fasting insulin and a greater prevalence of insulin resistance in men with early AGA.[4] Bakry and colleagues, in an Egyptian case-control study, reported a metabolic syndrome prevalence of 38.5% in patients with AGA compared with 14.6% in controls, with insulin resistance independently associated with the presence of AGA, figures that align closely with the present results.[11] Acibucu and colleagues, in a Turkish cohort of young men with early-onset AGA, similarly reported significantly higher HOMA-IR and a greater prevalence of metabolic syndrome than in controls, with insulin resistance emerging as the most consistently associated parameter.[12]

Among Indian studies, Banger and colleagues reported a metabolic syndrome prevalence of approximately 35% in young men with early-onset AGA, along with higher carotid intima-media thickness, suggesting that AGA may also serve as a surrogate marker of subclinical atherosclerosis.[13] Chakrabarty and colleagues found that 28% of young Indian men with AGA met the criteria for metabolic syndrome, a prevalence intermediate between the present cohort and earlier reports, and noted that the strength of association rose with the severity of alopecia.[14] Sharma and colleagues, in a north Indian population, observed significantly higher waist circumference, triglycerides, and fasting glucose, along with lower HDL-C, in patients with AGA than in matched controls, mirroring the lipid and anthropometric pattern documented in the present study.[15]

Su and Chen, in a community-based survey of over 700 Taiwanese men, reported that moderate to severe vertex-pattern baldness was associated with significantly higher odds of metabolic syndrome, particularly central obesity and low HDL-C, even after adjustment for age and family history.[6] Yi and colleagues, in a middle-aged Korean population, demonstrated a graded increase in the prevalence of metabolic syndrome with rising Norwood-Hamilton stage in men and Ludwig grade in women,[8] closely paralleling the trend observed in the present cohort where metabolic syndrome rose from 18.8% in mild AGA to 70.0% in severe AGA. Mumcuoglu and colleagues also confirmed a significant association between insulin resistance and early-onset AGA in young Turkish men, with HOMA-IR rising in parallel with severity.[16]

Ertas and colleagues, evaluating cardiovascular risk in AGA, demonstrated higher blood pressure, atherogenic index of plasma, and arterial stiffness in patients with vertex-pattern baldness,[17] supporting the view that the metabolic disturbances documented here are likely to translate into elevated long-term cardiovascular risk. Arias-Santiago and colleagues extended this observation to both sexes, reporting that women with female-pattern hair loss also exhibited higher prevalence of hypertension, insulin resistance, and dyslipidaemia than controls.[7] Pengsalae and colleagues, in a Thai case-control study, similarly demonstrated a significant association between AGA and metabolic syndrome, particularly in patients with onset before 35 years of age.[18]

Not all reports are unanimously concordant. Some studies have failed to find a significant association between AGA and individual lipid parameters once adjustment for BMI and central obesity is applied, suggesting that the lipid and glucose abnormalities observed in AGA may be largely mediated by adiposity rather than representing an independent dermatometabolic axis.[12] González-González and colleagues, while confirming the association of AGA with insulin resistance in young men, found no significant difference in serum total testosterone or DHT between cases and controls, indicating that the link between

AGA and metabolic syndrome may be driven primarily by insulin resistance rather than by circulating androgen excess.[9] The relatively modest effect size for smoking observed in the present cohort, with an adjusted odds ratio that failed to reach statistical significance, is also at variance with some reports that have implicated smoking as an independent risk factor for AGA, possibly reflecting differences in sample size and smoking intensity across populations.

The mechanistic basis for these consistent observations is now reasonably well delineated. Hyperinsulinaemia suppresses hepatic SHBG synthesis, elevating the free androgen fraction available for 5-alpha reductase-mediated conversion to DHT at the follicular level.[5] Insulin and IGF-1 also act directly on dermal papilla cells, promoting follicular ischaemia through peripheral vasoconstriction and reduced microvascular perfusion, and contributing to perifollicular microinflammation.[5,9] The substantial overlap in clinical, endocrine, and metabolic phenotype between early AGA in men and PCOS in women, highlighted by Sanke and colleagues,[5] supports the conceptualisation of AGA as a cutaneous marker of underlying insulin resistance, and reinforces the rationale for opportunistic metabolic screening in dermatology practice.[19]

From a public health perspective, the strong dose-response relationship between AGA severity and metabolic burden observed in the present study has practical implications. AGA is readily visible at presentation, requires no laboratory test for clinical recognition, and is frequently the only medical complaint that brings young men and women into contact with a healthcare facility. A dermatology visit for AGA therefore represents an opportunity to identify individuals at elevated cardiometabolic risk well before symptomatic disease develops, and to initiate lifestyle counselling and targeted screening for diabetes, dyslipidaemia, and hypertension. The harmonised definition of the metabolic syndrome provides a framework that is straightforward to apply in routine clinical practice.[20]

The present study has certain limitations. Its cross-sectional design precludes causal inference between AGA and metabolic disturbance; only a temporal association can be established. Recruitment from a single tertiary care centre may limit external generalisability, and the relatively small number of female cases restricts subgroup analysis stratified by Ludwig grade. Serum androgen profiling, including total testosterone, free testosterone, DHT, and SHBG, was not performed and would have strengthened the mechanistic interpretation. Longitudinal studies with serial assessment of both AGA severity and metabolic indices are warranted to clarify the directionality of the association and to determine whether early intervention on metabolic risk modifies the natural history of AGA.

CONCLUSION

Androgenetic alopecia in this hospital-based cohort was significantly associated with insulin resistance, central obesity, atherogenic dyslipidaemia, and the metabolic syndrome, and the burden of metabolic disturbance scaled with clinical severity. The prevalence of metabolic syndrome rose from 18.8% in mild AGA to 70.0% in severe AGA among male cases, and HOMA-IR emerged as the strongest independent metabolic predictor of AGA on multivariate analysis. These findings support the view that androgenetic alopecia, particularly when of early onset and higher grade, should not be regarded merely as a cosmetic concern but as a clinically useful, readily visible marker of underlying insulin resistance and cardiometabolic risk. Routine clinical evaluation of patients presenting with AGA should therefore incorporate measurement of waist circumference, blood pressure, fasting plasma glucose, and lipid profile, with opportunistic estimation of HOMA-IR in selected cases. Early identification of metabolic abnormalities in this group offers a window for lifestyle intervention and risk-factor modification long before overt cardiovascular disease develops.

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