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Original Article

A Case–Control Study of the Association Between Androgenetic Alopecia and Metabolic Syndrome in Males

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ABSTRACT

Background: Androgenetic alopecia (AGA) is the most common form of hair loss in men, characterized by progressive follicular miniaturization driven by androgen sensitivity and genetic predisposition. Although traditionally regarded as a cosmetic condition, emerging evidence suggests a potential association between AGA and systemic metabolic abnormalities, including metabolic syndrome—a cluster of central obesity, dyslipidemia, hypertension, and impaired glucose metabolism—which significantly elevates cardiovascular risk.

Objectives: To evaluate the association between androgenetic alopecia and metabolic syndrome, and to assess the relationship between AGA severity and individual metabolic risk factors.

Methods: This hospital-based case–control study enrolled 114 male participants aged 18–60 years: 57 clinically diagnosed AGA cases and 57 age-matched controls without alopecia. AGA severity was graded using the Modified Norwood–Hamilton classification. Anthropometric measurements, blood pressure, fasting blood glucose, and fasting lipid profile were recorded. Metabolic syndrome was diagnosed using the revised NCEP ATP III criteria.

Results: Metabolic syndrome was significantly more prevalent in AGA cases (26.3%) than in controls (8.8%; $p < 0.001$). Cases exhibited significantly higher triglyceride levels (139.26 ± 59.24 vs. 116.02 ± 30.77 mg/dL), lower HDL cholesterol (49.26 ± 18.73 vs. 53.00 ± 10.56 mg/dL), elevated fasting blood sugar, and greater central obesity compared to controls. AGA severity correlated significantly with disease duration (Spearman's $\rho = 0.623$, $p = 0.001$) and family history ($p = 0.001$), but not with overall metabolic syndrome prevalence. Reduced HDL showed a significant association with higher AGA grades ($p = 0.04$). Multivariate analysis identified elevated triglycerides (OR = 1.04) and increased waist circumference (OR = 1.18) as independent predictors of metabolic syndrome.

Conclusion: Androgenetic alopecia is significantly associated with metabolic syndrome and adverse metabolic parameters, particularly dyslipidemia and central obesity. AGA, especially in young males, may serve as an early clinical marker of systemic metabolic risk. Routine screening for metabolic syndrome in AGA patients is recommended to facilitate timely intervention and reduce long-term cardiovascular morbidity.

Keywords: *Androgenetic alopecia; Metabolic syndrome; Dyslipidemia; Central obesity; Cardiovascular risk; Norwood–Hamilton classification.*

INTRODUCTION

Androgenetic alopecia (AGA) is the most common form of hair loss in men and is characterized by progressive, patterned, androgen-dependent miniaturization of genetically susceptible hair follicles. The condition predominantly involves the frontotemporal and vertex regions of the scalp, resulting in gradual conversion of terminal hairs into shorter, finer, and less pigmented hairs. The extent and pattern of hair loss in males are commonly assessed using the **Hamilton–Norwood scale**, which provides a standardized clinical grading of AGA severity.[1,2] Although AGA is primarily considered a dermatological and cosmetic condition, increasing attention has been directed toward its possible association with systemic metabolic abnormalities.

Metabolic syndrome (MetS) is a cluster of interrelated cardiometabolic abnormalities comprising central obesity, dyslipidemia, hypertension, and impaired fasting glucose, which collectively increase the risk of cardiovascular disease and type 2 diabetes mellitus. Insulin resistance is considered a central underlying mechanism in the development of these metabolic disturbances.[3,4] Because the individual components of MetS may remain clinically silent for several years, identification of easily recognizable clinical features associated with metabolic risk may provide an opportunity for earlier screening and preventive intervention.

This relationship is particularly relevant in the Indian population because South Asians demonstrate a characteristic tendency toward increased visceral adiposity, insulin resistance, dyslipidemia, and cardiometabolic risk even at comparatively lower body mass index levels. This "South Asian phenotype" contributes to the disproportionately high burden of MetS and its complications in India, often at a relatively younger age.[5] Consequently, identification of potential external clinical markers associated with metabolic abnormalities may be particularly useful in this population.

Several shared pathogenic mechanisms provide biological plausibility for an association between AGA and MetS. Hyperinsulinemia, resulting from insulin resistance, may reduce circulating sex hormone-binding globulin (SHBG), thereby increasing the bioavailability of free androgens. Increased androgenic activity in genetically susceptible hair follicles may contribute to progressive follicular miniaturization and worsening of AGA.[6] Insulin resistance and hyperinsulinemia may therefore represent an important metabolic link between the two conditions.

In addition to altered insulin and androgen activity, chronic low-grade inflammation, oxidative stress, and adipokine dysregulation may contribute to both metabolic dysfunction and alterations in the hair follicle microenvironment. Increased levels of pro-inflammatory cytokines, particularly tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6), are associated with metabolic abnormalities and may influence vascular and follicular function.[7,8] The coexistence of hormonal, inflammatory, and metabolic pathways supports the concept of a potential metabolic–dermatological axis linking AGA with systemic metabolic dysfunction.

The clinical importance of this association is greater when AGA develops at a younger age or progresses to more advanced grades. Since scalp hair loss is readily visible and can be recognized during routine clinical examination, AGA has been proposed as a potential external indicator of an underlying adverse metabolic profile. Detection of such an association could help identify apparently healthy men who may otherwise remain unaware of underlying central obesity, hypertension, dyslipidemia, impaired glucose metabolism, or other components of MetS.[9,10]

Furthermore, evaluating the severity of AGA may provide additional clinical information. If the prevalence of MetS increases with advancing Hamilton–Norwood grades, the extent of hair loss could potentially assist in identifying individuals with a greater metabolic risk. However, the relationship between AGA severity and the occurrence of MetS requires further evaluation, particularly in Indian male populations where both androgenetic alopecia and metabolic abnormalities are commonly encountered.[9,10]

Despite the high dual burden of androgenetic alopecia and metabolic syndrome in India, robust case-control data examining their association remain limited. In particular, further evidence is required to establish whether AGA can serve as a useful clinical marker for increased metabolic risk and whether increasing severity of alopecia is associated with a greater prevalence of MetS. Therefore, the present study was undertaken to study the association between metabolic syndrome and androgenetic alopecia in male patients, to determine the utility of AGA as a clinical marker for an increased risk of developing metabolic syndrome, and to correlate the severity of AGA according to Hamilton–Norwood grading with the prevalence of metabolic syndrome.

MATERIAL AND METHODS

Study Design and Setting

This hospital-based case–control study was conducted in the Outpatient Department of Dermatology, Venereology and Leprosy, Prasad Institute of Medical Sciences (PIMS), Lucknow, over a period of 18 months. The study was conducted after obtaining approval from the Institutional Ethics Committee (IEC). Written informed consent was obtained from all participants prior to enrollment in the study.

Sample Size Calculation
The sample size was calculated with reference to previous literature,[1] considering a prevalence of metabolic syndrome of 33.3% among cases and 14% among controls, with a two-sided α of 0.10 and statistical power (1−β) of 80%. The sample size was calculated using the two-proportion formula: $n = (Z\alpha/2 + Z\beta)^2 \times [p_1(1-p_1) + p_2(1-p_2)]/(p_1-p_2)^2$. The calculated minimum sample size was 57 participants per group. Accordingly, a total of 114 participants were enrolled, comprising 57 cases and 57 controls.

Study Population
A total of 114 male patients attending the Dermatology OPD were enrolled and divided into two equal groups. Group A (Cases) consisted of 57 male patients clinically diagnosed with androgenetic alopecia (AGA) based on the Modified Norwood–Hamilton classification. Group B (Controls) consisted of 57 age-matched male patients without any clinical evidence of alopecia who attended the OPD for benign dermatological conditions, including warts, melanocytic nevi, and melasma. Male patients aged 18–60 years who were willing to provide informed consent were eligible for inclusion. For inclusion in the case group, participants were required to have a clinical diagnosis of AGA, whereas controls were required to have no clinical evidence of any form of alopecia. Patients with other types of alopecia, including scarring alopecia, telogen effluvium, and alopecia areata, were excluded. Patients receiving medications known to affect metabolic parameters, including corticosteroids, testosterone therapy, antihypertensive drugs, lipid-lowering agents, or antidiabetic drugs, were also excluded. In addition, patients with a known endocrine disorder independently associated with metabolic derangement were not included in the study.

Clinical Examination and Assessment
Each participant underwent a structured clinical evaluation. Sociodemographic characteristics were recorded, followed by a detailed history regarding the duration and progression of hair loss and family history of AGA. General physical examination included measurement of height and weight, from which body mass index (BMI) was calculated. Waist circumference (WC) was measured at the midpoint between the lower costal margin and iliac crest at the end of expiration. Blood pressure (BP) was recorded in the right arm using a calibrated mercury sphygmomanometer. A detailed scalp examination was performed in all participants. Among cases, the severity of androgenetic alopecia was assessed and graded according to the Modified Norwood–Hamilton classification. Clinical photographs were obtained wherever required for documentation.

Laboratory Investigations
Following a minimum 12-hour overnight fast, venous blood samples were collected from all participants. Laboratory investigations included fasting blood glucose (FBG) and fasting lipid profile. The lipid profile comprised serum triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C).

Diagnostic Criteria for Metabolic Syndrome
Metabolic syndrome was diagnosed according to the revised National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III, 2005) criteria.[2] Metabolic syndrome was considered to be present when a participant fulfilled three or more of the following five criteria: waist circumference ≥102 cm; serum triglycerides ≥150 mg/dL; HDL-C <40 mg/dL; blood pressure ≥130/85 mmHg or receiving antihypertensive treatment; and fasting blood glucose ≥100 mg/dL or receiving antidiabetic therapy.

Statistical Analysis
Data were analyzed using EPI INFO software, version 7.0. Continuous variables were expressed as mean ± standard deviation (SD) and compared between the case and control groups using the unpaired Student's *t*-test. Categorical variables were expressed as frequencies and percentages and compared using the Chi-square (χ^2) test. A two-sided *p*-value <0.05 was considered statistically significant.

Fig 1.: Modified Norwood–Hamilton Grade III

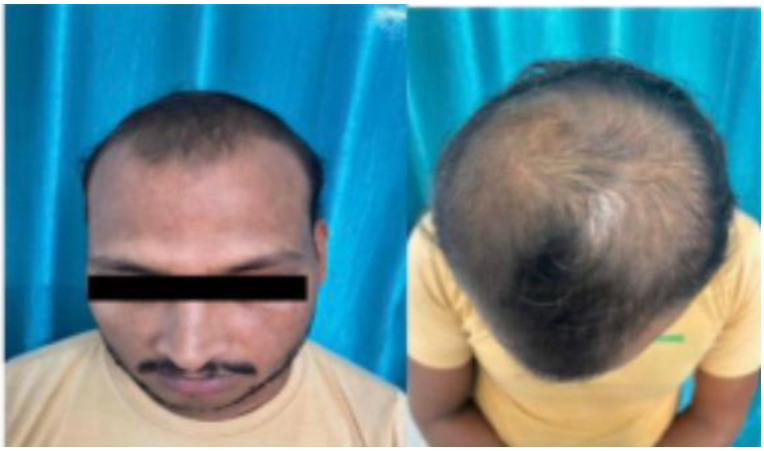

Fig 2.: Modified Norwood–Hamilton Grade IV

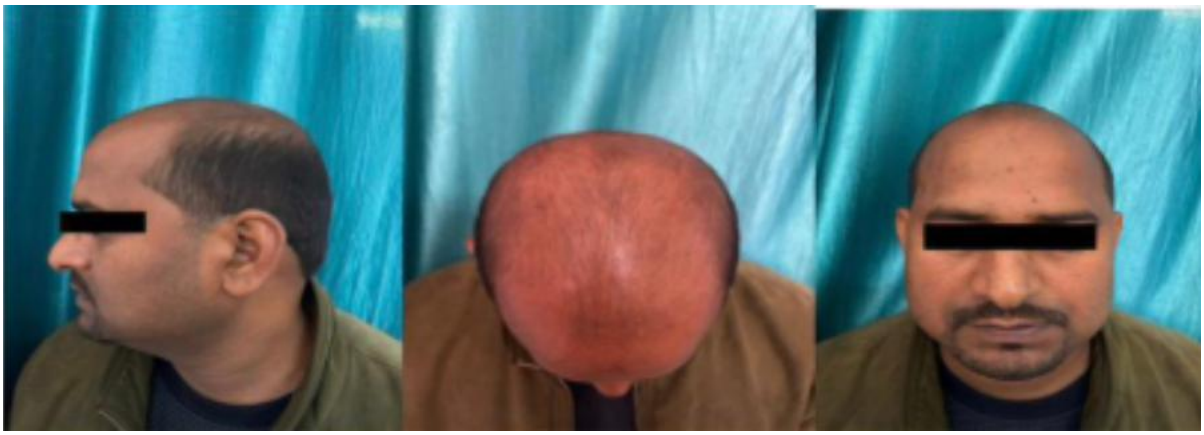

Fig 3.: Modified Norwood–Hamilton Grade VI

RESULTS

Table 1. Demographic Characteristics and AGA Profile of Cases and Controls

Variable	Category	Cases (n=57), n (%)	Controls (n=57), n (%)	p-value
Age Group (years)	18–29	38 (66.67)	36 (63.16)	
	30–39	13 (22.81)	11 (19.30)	
	40–49	5 (8.77)	6 (10.53)	0.55
	50–59	1 (1.75)	4 (7.02)	
Mean Age ± SD (years)	—	28.39 ± 7.76	30.01 ± 9.83	
AGA Grade (Hamilton–Norwood)	Grade 3	12 (21.1)		
	Grade 3A	6 (10.5)		

	Grade 3 Vertex	3 (5.3)		
	Grade 4	10 (17.5)		
	Grade 4A	8 (14.0)		
	Grade 5	9 (15.8)		
	Grade 5A	5 (8.8)		
	Grade 6	4 (7.0)		
Disease Duration (years)	<1 year	3 (5.26)		
	1–2 years	31 (54.39)		
	3–5 years	13 (22.81)		
	>6 years	10 (17.54)		
Family History of AGA	Present	34 (60.0)		
	Absent	23 (40.0)		

Among the 57 cases, 38 (66.67%) were aged 18–29 years, 13 (22.81%) were aged 30–39 years, 5 (8.77%) were aged 40–49 years, and 1 (1.75%) was aged 50–59 years. Among controls, the corresponding values were 36 (63.16%), 11 (19.30%), 6 (10.53%), and 4 (7.02%), respectively. The mean age was 28.39 ± 7.76 years in cases and 30.01 ± 9.83 years in controls, with no statistically significant difference (p=0.55). Among cases, Grade 3 was the most frequent AGA grade, observed in 12 (21.1%) patients, followed by Grade 4 in 10 (17.5%), Grade 5 in 9 (15.8%), Grade 4A in 8 (14.0%), Grade 3A in 6 (10.5%), Grade 5A in 5 (8.8%), Grade 6 in 4 (7.0%), and Grade 3 Vertex in 3 (5.3%). Regarding disease duration, 31 (54.39%) patients had AGA for 1–2 years, 13 (22.81%) for 3–5 years, 10 (17.54%) for >6 years, and 3 (5.26%) for <1 year. A positive family history of AGA was reported by 34 (60.0%) cases, whereas 23 (40.0%) had no family history.

Table 2. Comparison of Biochemical and Anthropometric Parameters Between Cases and Controls

Parameter	Cases Mean ± SD / n (%)	Controls Mean ± SD / n (%)	p-value
Systolic BP (mmHg)	122.23 ± 14.93	121.16 ± 11.06	0.665
Diastolic BP (mmHg)	78.47 ± 9.61	77.86 ± 6.92	0.696
Triglycerides (mg/dL)	139.26 ± 59.24	116.02 ± 30.77	**<0.01**
HDL Cholesterol (mg/dL)	49.26 ± 18.73	53.00 ± 10.56	**0.019**
Fasting Blood Sugar (mg/dL)	96.09 ± 19.30	90.91 ± 11.00	**0.048**
Waist Circumference Distribution			
<80 cm	9 (15.8%)	15 (26.3%)	
80–89 cm	18 (31.6%)	22 (38.6%)	
90–101 cm	17 (29.8%)	13 (22.8%)	
≥102 cm	13 (22.8%)	7 (12.3%)	
Fasting Blood Sugar Distribution			
Normal (<100 mg/dL)	43 (75.4%)	48 (84.2%)	
Prediabetes (100–125 mg/dL)	8 (14.0%)	6 (10.5%)	
Diabetes (≥126 mg/dL)	6 (10.5%)	3 (5.3%)	
Triglyceride Distribution			
Normal (<150 mg/dL)	40 (70.2%)	47 (82.5%)	
Borderline (150–199 mg/dL)	7 (12.3%)	5 (8.8%)	
High (200–499 mg/dL)	10 (17.5%)	5 (8.8%)	
HDL Cholesterol Distribution			
Low (<40 mg/dL)	19 (33.3%)	11 (19.3%)	
Normal (40–59 mg/dL)	18 (31.6%)	27 (47.4%)	
Protective (≥60 mg/dL)	20 (35.1%)	19 (33.3%)	

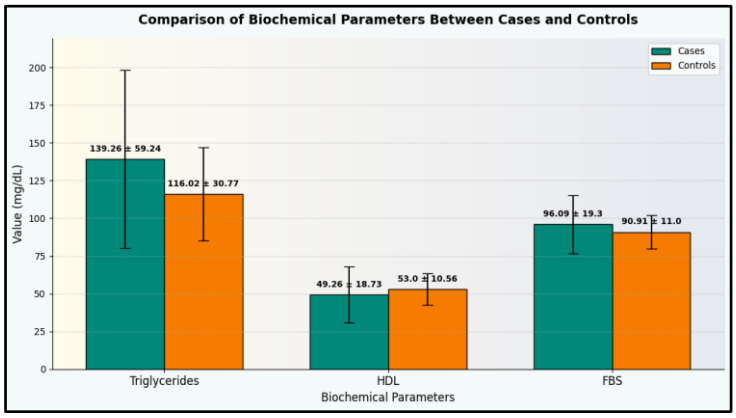

Fig 4 Comparison of Biochemical Parameters Between Cases and Controls

Mean systolic blood pressure was 122.23 ± 14.93 mmHg among cases and 121.16 ± 11.06 mmHg among controls (p=0.665), while mean diastolic blood pressure was 78.47 ± 9.61 mmHg and 77.86 ± 6.92 mmHg, respectively (p=0.696), with neither difference reaching statistical significance. In contrast, mean triglyceride levels were significantly higher among cases (139.26 ± 59.24 mg/dL) than controls (116.02 ± 30.77 mg/dL; p<0.01). Mean HDL cholesterol was significantly lower in cases (49.26 ± 18.73 mg/dL) than controls (53.00 ± 10.56 mg/dL; p=0.019), while fasting blood sugar was significantly higher in cases (96.09 ± 19.30 mg/dL) compared with controls (90.91 ± 11.00 mg/dL; p=0.048).

For waist circumference, 9 (15.8%), 18 (31.6%), 17 (29.8%), and 13 (22.8%) cases had measurements of <80 cm, 80–89 cm, 90–101 cm, and ≥102 cm, respectively, compared with 15 (26.3%), 22 (38.6%), 13 (22.8%), and 7 (12.3%) controls. Normal fasting blood glucose was observed in 43 (75.4%) cases and 48 (84.2%) controls, whereas prediabetes was present in 8 (14.0%) cases and 6 (10.5%) controls, and diabetic-range values were observed in 6 (10.5%) and 3 (5.3%), respectively. Normal triglyceride levels were found in 40 (70.2%) cases and 47 (82.5%) controls; borderline levels in 7 (12.3%) and 5 (8.8%); and high levels in 10 (17.5%) and 5 (8.8%), respectively. Low HDL cholesterol was more frequent among cases (19; 33.3%) than controls (11; 19.3%), while normal HDL was present in 18 (31.6%) cases and 27 (47.4%) controls and protective HDL levels in 20 (35.1%) and 19 (33.3%), respectively.

Table 3. Prevalence of Metabolic Syndrome and its Association with AGA Grade

Metabolic Syndrome Prevalence

Category	Cases (n=57), n	Cases %	Controls (n=57), n	Controls %	p-value
MetS Present	15	26.3	5	8.8	**<0.001**
MetS Absent	42	73.7	52	91.2	

MetS by AGA Grade (Cases Only)

AGA Grade	MS Present	%	MS Absent	%	p-value
Grade 3	5	33.3	16	38.1	0.99
Grade 4	5	33.3	13	31.0	—
Grade 5	4	26.7	10	23.8	—
Grade 6	1	6.7	3	7.1	—
Total (MS+)	**15**	**100**	**42**	**100**	**$\chi^2 \approx 0.13$**

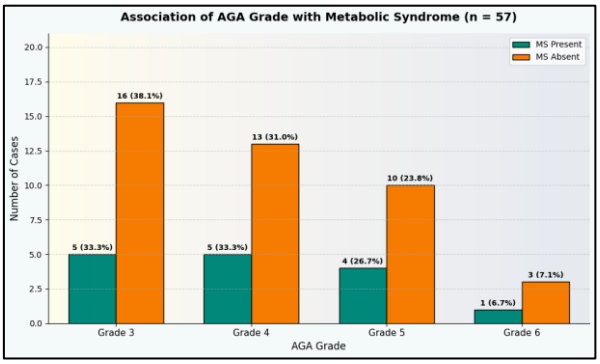

Fig 5 Association of AGA Grade with Metabolic Syndrome (n = 57)

Metabolic syndrome was present in 15 (26.3%) of the 57 AGA cases compared with 5 (8.8%) of the 57 controls, whereas MetS was absent in 42 (73.7%) cases and 52 (91.2%) controls. The prevalence of metabolic syndrome was therefore significantly higher among cases than controls (p<0.001). Among AGA cases with MetS, 5 (33.3%) had Grade 3, 5 (33.3%) had Grade 4, 4 (26.7%) had Grade 5, and 1 (6.7%) had Grade 6 AGA. Among cases without MetS, 16 (38.1%) had Grade 3, 13 (31.0%) had Grade 4, 10 (23.8%) had Grade 5, and 3 (7.1%) had Grade 6. There was no statistically significant association between AGA grade and metabolic syndrome status ($\chi^2 \approx 0.13$, p=0.99).

Table 4. Association of AGA Severity with Family History, Disease Duration, and MetS Components

Variable	Grade 3/3A/3V	Grade 4/4A	Grade 5+/6	p-value
Family History vs AGA Grade				
Family History Present	6	13	15	**0.001**
Family History Absent	15	5	3	—
Total	**21**	**18**	**18**	—
MetS Components vs AGA Grade				
Central Obesity (↑WC)	4	5	8	0.11

Hypertriglyceridemia	5	4	8	0.21
Reduced HDL Cholesterol	3	6	10	**0.04***
Raised Blood Pressure	3	4	6	0.56
Elevated Fasting Blood Glucose	4	3	7	0.19
Disease Duration vs AGA Grade (Spearman ρ = 0.623, p = 0.001)				
<1 year	3	0	0	—
1–2 years	16	11	4	—
3–5 years	2	5	5 (5 grade) +1 (6 grade)	—
>6 years	0	2	5 (5 grade) +3 (6 grade)	—

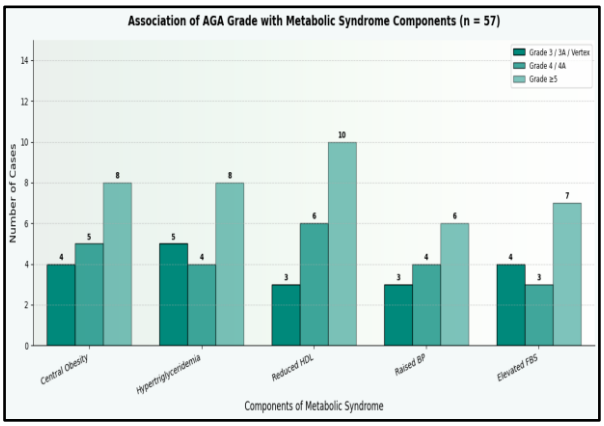

Fig 6 Association of AGA Grade with Metabolic Syndrome Components (n = 57)

A positive family history of AGA was observed in 6 patients with Grade 3/3A/3V, 13 with Grade 4/4A, and 15 with Grade 5+/6, whereas an absent family history was recorded in 15, 5, and 3 patients, respectively. The association between family history and increasing AGA severity was statistically significant (p=0.001).

Regarding metabolic syndrome components, central obesity was present in 4, 5, and 8 patients across Grade 3/3A/3V, Grade 4/4A, and Grade 5+/6 groups, respectively (p=0.11). Hypertriglyceridemia was observed in 5, 4, and 8 patients (p=0.21), reduced HDL cholesterol in 3, 6, and 10 patients (p=0.04), raised blood pressure in 3, 4, and 6 patients (p=0.56), and elevated fasting blood glucose in 4, 3, and 7 patients (p=0.19), respectively. Among these components, only reduced HDL cholesterol showed a statistically significant association with increasing AGA severity (p=0.04).

Disease duration showed a significant positive correlation with AGA severity (Spearman ρ=0.623, p=0.001). For disease duration <1 year, the values across the three severity groups were 3, 0, and 0/0; for 1–2 years, 16, 11, and Apr-00; for 3–5 years, 2, 5, and 1-May; and for >6 years, 0, 2, and 3-May, respectively.

Table 5. Multivariate Logistic Regression: Independent Predictors of Metabolic Syndrome

Risk Factor	Odds Ratio (OR)	95% Confidence Interval	p-value
Waist Circumference (cm)	1.18	1.06–1.32	**0.003**
Triglycerides (mg/dL)	1.04	1.01–1.07	**0.009**
HDL Cholesterol (mg/dL)	0.97	0.94–1.01	0.18
Systolic BP (mmHg)	1.01	0.98–1.05	0.42
Diastolic BP (mmHg)	1.02	0.97–1.06	0.36
Fasting Blood Sugar (mg/dL)	1.01	0.99–1.03	0.21

Multivariate logistic regression identified waist circumference and triglyceride levels as statistically significant independent predictors of metabolic syndrome. Each one-unit increase in waist circumference was associated with an 18% increase in the odds of MetS (OR=1.18, 95% CI: 1.06–1.32; p=0.003). Similarly, increasing triglyceride levels were independently associated with MetS (OR=1.04, 95% CI: 1.01–1.07; p=0.009). HDL cholesterol showed an inverse association with MetS (OR=0.97, 95% CI: 0.94–1.01), but this was not statistically significant (p=0.18). Systolic blood pressure (OR=1.01, 95% CI: 0.98–1.05; p=0.42), diastolic blood pressure (OR=1.02, 95% CI: 0.97–1.06; p=0.36), and fasting blood sugar (OR=1.01, 95% CI: 0.99–1.03; p=0.21) were also not independently associated with MetS in the multivariate model.

DISCUSSION

The present case and control study evaluated the prevalence of metabolic syndrome and its association with androgenetic alopecia (AGA) in males. AGA, traditionally considered a cosmetic concern, has increasingly been recognized as a potential clinical marker of underlying metabolic abnormalities. A significantly higher prevalence of metabolic syndrome was observed among cases compared to controls, suggesting a strong association between AGA and metabolic dysregulation.

The majority of participants belonged to the 18–29 years age group (66.67% of cases; 63.16% of controls), with no significant intergroup difference ($p = 0.55$). Mean age was 28.39 ± 7.76 years in cases and 30.01 ± 9.83 years in controls. This age-matched distribution minimizes confounding and aligns with prior reports by Sheikh F et al.[20] (mean age 27.77 ± 5.04 years) and Kumar et al.[18] The younger age profile is clinically significant, as early-onset AGA carries higher cardiovascular and metabolic risk.[7]

Grade 3 AGA was the most common severity (21.1%), followed by Grades 4 (17.5%), 5 (15.8%), 4A (14%), and 3A (10.5%), consistent with Tilwani M et al.[14] and Sheikh F et al.[20] A significant positive correlation was observed between disease duration and AGA severity (Spearman's $\rho = 0.623$, $p = 0.001$), reflecting androgen-mediated follicular miniaturization.[11] Family history was present in 60% of cases and was significantly associated with higher grades ($p = 0.001$), consistent with a 2–5-fold increased genetic risk for AGA.[11]

Blood pressure did not differ significantly between groups (SBP: 122.23 ± 14.93 vs. 121.16 ± 11.06 mmHg, $p = 0.665$; DBP: 78.47 ± 9.61 vs. 77.86 ± 6.92 mmHg, $p = 0.696$), consistent with Alam M et al.[16] but contrasting with Kumar et al.[18] and Taheri A et al.[19] Biochemically, cases showed significantly higher triglycerides (139.26 ± 59.24 vs. 116.02 ± 30.77 mg/dL; $p < 0.01$), lower HDL (49.26 ± 18.73 vs. 53.00 ± 10.56 mg/dL; $p = 0.0192$), and higher fasting blood sugar (96.09 ± 19.30 vs. 90.91 ± 11.00 mg/dL; $p = 0.0481$). High triglycerides (200–499 mg/dL) were more frequent in cases (17.5% vs. 8.8%), as were low HDL levels (33.3% vs. 19.3%), confirming an atherogenic lipid profile consistent with Chakrabarty S et al.[9] and Mahitha Devi M et al.[17]

Prediabetes and diabetes were more prevalent among cases (24.5% vs. 15.8%), indicating impaired glucose metabolism possibly mediated by insulin resistance—a central mechanism linking AGA and metabolic syndrome through hyperinsulinemia, reduced SHBG, and elevated free androgens promoting follicular miniaturization.[7] Waist circumference ≥102 cm was also more frequent among cases, consistent with Bakry O et al.[10] who identified central obesity as the strongest predictor of metabolic syndrome.

The prevalence of metabolic syndrome was significantly higher in cases (26.3%) versus controls (8.8%; $p < 0.001$), consistent with Gopinath H et al.[13] (22.4% vs. 9.4%), Swaroop M et al.[15] (30% vs. 8%), Chaudhari ND et al.[22] (48% vs. 18%), and the meta-analysis by Qi Y et al.[7] which reported a 3.46-fold increased risk of metabolic syndrome in AGA patients.

No significant association was found between AGA grade and metabolic syndrome ($p = 0.99$), echoing Chakrabarty S et al.[9] and Almudimeegh A et al.[21] though Tilwani M et al.[14] and Majella S et al.[24] reported otherwise. Reduced HDL alone showed a significant association with AGA severity ($p = 0.04$), suggesting lipid abnormalities are most closely tied to disease progression.

Multivariate logistic regression identified waist circumference (OR = 1.18, $p = 0.003$) and triglycerides (OR = 1.04, $p = 0.009$) as independent predictors of metabolic syndrome, underscoring the primacy of visceral adiposity and dyslipidemia. Shared pathophysiological pathways—insulin resistance, chronic inflammation, oxidative stress, endothelial dysfunction, and adipokine imbalance—form an integrated metabolic–alopecia axis.[7,12] Contrasting findings from Gok SO et al.[12] and Zhu H et al.[23] may reflect differences in study design, ethnicity, and sample size.

CONCLUSION

The present study establishes a significant association between androgenetic alopecia and metabolic syndrome in males, with a notably higher prevalence of metabolic syndrome among cases (26.3%) compared to controls (8.8%). Individuals with AGA demonstrated an adverse metabolic profile, including elevated triglycerides, reduced HDL cholesterol, higher fasting blood glucose, and increased central obesity. Waist circumference and triglycerides were identified as independent predictors of metabolic syndrome on multivariate analysis.

Although AGA severity correlated with disease duration and family history, it did not consistently correlate with metabolic syndrome, suggesting that AGA—irrespective of its grade—may serve as an early clinical marker of systemic metabolic risk. These findings highlight the importance of routine metabolic screening in AGA patients, particularly young males, to enable timely intervention and reduce long-term cardiovascular morbidity.

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