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METABOLIC AND HORMONAL ALTERATIONS ASSOCIATED WITH EARLY-ONSET ANDROGENETIC ALOPECIA IN YOUNG MEN: CASE–CONTROL STUDY

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Abstract:
Background: There is growing recognition that early-onset androgenetic alopecia (AGA) may be a clinical indicator of systemic metabolic disorders. Its relationship to hormonal and metabolic biomarkers is still unclear, though.
Objective: To compare metabolic and androgenic hormonal profiles between young men with early-onset AGA and healthy controls and to evaluate their relationships with disease severity.
Methods: This case–control study included 160 participants (80 patients with early-onset AGA and 80 age-matched healthy controls) recruited in Kirkuk, Iraq. Body mass index (BMI), fasting glucose, fasting insulin, HOMA-IR, lipid profile, and total testosterone were assessed. Disease severity was graded using the Hamilton–Norwood classification. Correlation analysis, multivariable logistic regression, and receiver operating characteristic (ROC) curve analysis were performed.
Results: Patients with AGA exhibited significantly higher BMI, fasting glucose, fasting insulin, HOMA-IR, total cholesterol, triglycerides, and LDL-C, whereas HDL-C and total testosterone were significantly lower than those of controls (all $P < 0.001$). Disease severity was positively correlated with metabolic abnormalities and inversely correlated with total testosterone. Family history, HOMA-IR, LDL-C, and total testosterone were identified as independent predictors of AGA. HOMA-IR showed the highest diagnostic performance (AUC = 0.871), with 83.8% sensitivity and 80.0% specificity.
Conclusion: Early-onset AGA is associated with significant metabolic and hormonal alterations, particularly insulin resistance and dyslipidemia. HOMA-IR may represent a useful metabolic marker for identifying young men at increased cardiometabolic risk.

Keywords: Androgenetic alopecia; HOMA-IR; Insulin resistance; Testosterone; Dyslipidemia; Biomarkers.

Introduction

Androgenetic alopecia (AGA) is a chronic, non-scarring hair-loss disorder, characterized by progressive miniaturization of susceptible scalp follicles. It is the most frequent form of hair loss in men and may start soon after puberty leading to the characteristic bitemporal recession and vertex thinning of male-pattern hair loss [1]. AGA has a multifactorial biological basis, including a strong genetic predisposition and androgen-dependent follicular activity. Increased local conversion of testosterone to dihydrotestosterone by 5α-reductase and increased androgen-receptor signaling shorten the anagen phase and progressively decrease hair-shaft diameter [2]. Although AGA is medically benign, early onset may be of great concern to young men as hair is closely linked to age, attractiveness and self-image. Prospective evidence indicates that younger age and greater disease severity are associated with greater reduction in hair-related quality of life [3]. Furthermore, a systematic review and meta-analysis have shown that male AGA can have a measurable psychosocial burden, mainly through appearance-related concerns and impaired quality of life, although the extent of psychological effects varies greatly between individuals and study populations [4]. In addition to its effects on appearance and mental health, early-onset AGA has drawn more attention as a possible outward sign of systemic metabolic dysfunction. In early, a case-control study found increased hyperinsulinemia and insulin-resistance-related disorders in men who developed alopecia before 35 years of age [5]. Further studies in young men showed that patients with advanced AGA had significantly higher fasting insulin and homeostatic model assessment of insulin resistance (HOMA-IR) values. The results indicate that metabolic abnormalities can be detected as early as young adulthood [6]. Other case-control studies also noted increased frequencies of insulin resistance and metabolic syndrome in men with early-onset AGA, which further confirms the clinical significance of evaluating glucose metabolism, adiposity, blood pressure and lipid abnormalities in affected patients [7]. But the association is still controversial. Studies in young non-obese subjects indicated that insulin resistance was more correlated with the presence of metabolic syndrome than with AGA itself, stressing the need to control for obesity and other potential confounders [8]. Abnormal lipid metabolism may be another important feature. Men with early-onset AGA have been found to have higher total cholesterol, triglyceride and low-density lipoprotein cholesterol concentrations compared to non-alopecic controls, reflecting a potentially adverse cardiovascular risk profile [9]. Moreover, studies that have evaluated broader cardiovascular risk factors have demonstrated an increased prevalence of metabolic syndrome and alterations in anthropometric, hormonal, inflammatory and vascular parameters in patients with early-onset AGA. However, the clinical importance and causal direction of these associations are still unclear [10]. Furthermore, male AGA hormonal profile is complex. The follicular sensitivity to androgens may be more important than the circulating testosterone concentration and paradoxically some studies have shown lower serum testosterone levels among the affected young men despite the androgen-dependent nature of the disorder [11-12]. Thus, the present study aimed to compare metabolic and androgenic hormonal parameters between young males with early-onset AGA and healthy

age-matched controls and to evaluate the relationships of these parameters with disease severity and their capability to discriminate the presence of early-onset AGA independently

2. Materials and Methods

2.1 Study Design and Participants

Case-control study data were collected in Kirkuk Governorate of Iraq from July to November 2025. Methods: One hundred sixty young adult men were included which consists of 80 patients with early-onset androgenetic alopecia (AGA) (case group) and 80 healthy age-matched men without clinical evidence of hair loss. Patients with AGA were obtained from dermatology outpatient clinics and healthy controls were obtained from the local community. A consultant dermatologist diagnosed AGA on the basis of clinical examination and characteristic scalp hair-loss patterns, and severity of disease was graded according to the Hamilton–Norwood classification.

2.2 Inclusion and Exclusion Criteria

Eligible patients were men aged 18-35 years with clinical diagnosis of early-onset androgenetic alopecia confirmed by consultant dermatologist. Controls were healthy age matched men who had no clinical evidence for androgenetic alopecia. Exclusion criteria were: diabetes mellitus, thyroid, hepatic, renal, endocrine, autoimmune, inflammatory or malignant disease, or receipt of hormonal therapy, systemic corticosteroids, finasteride, dutasteride, minoxidil, anabolic steroids or lipid- or glucose-lowering drugs within the previous 3 months.

2.3 Clinical Assessment

Demographic and clinical data including age, smoking status, family history of androgenetic alopecia, duration of hair loss, and medication history were recorded for each participant. Body weight, height and body mass index (BMI) were measured using standardized procedures.

2.4 Blood Sample Collection

After a 10- to 12-hour overnight fasting, venous blood (8–10 mL) was obtained from each subject between 08:00 AM and 10:00 AM. Blood samples were allowed to clot at room temperature for 30 minutes followed by centrifugation (3000 rpm for 10 minutes). The serum that was separated was aliquoted and stored at -20°C for future biochemical analyses.

2.5 Laboratory Investigations

Fasting serum insulin and total testosterone concentrations were measured using the VIDAS® automated immunoanalyzer (bioMérieux, Marcy-l'Étoile, France) based on the enzyme-linked fluorescent assay (ELFA) principle. Fasting blood glucose and serum lipid profile (total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C)) were analysed by using an automated clinical chemistry

analyser FUJI DRI-CHEM (FUJIFILM Corporation, Tokyo, Japan) according to the manufacturer's instructions.

2.6 Calculation of Metabolic Indices

Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), a validated index of insulin sensitivity based on fasting glucose and fasting insulin concentrations, was used to assess IR. For each individual, HOMA-IR was calculated using the formula

HOMA-IR = [Fasting insulin (μIU/mL) × Fasting glucose (mg/dL)] / 405

Body mass index (BMI) as a marker of general adiposity was calculated as body weight (kg) divided by the square of height (m^2). Body weight and height were measured by standardized procedures in light clothing and without shoes. BMI was expressed in kilograms per square meter (kg/m^2).

2.7 Statistical Analysis

IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA) was used for statistical analysis. The Shapiro-Wilk test was used to determine whether the data was normal. Categorical variables were shown as percentages and proportions, while continuous variables were shown as mean ± SD. The Chi-square or Fisher's exact test were used to analyze categorical variables, and the independent-samples Student's t-test or Mann-Whitney U test were used for group comparisons. Pearson or Spearman correlation coefficients were used to compute correlations. To find independent predictors of early onset androgenetic alopecia, binary logistic regression analysis was used. The diagnostic performance of the examined biomarkers was evaluated using receiver operating characteristic (ROC) curve analysis. A two-sided P value of less than.05 was considered statistically significant.

Results

3.1 Demographic and Clinical Characteristics

The study consisted of 160 subjects, 80 patients with early-onset androgenetic alopecia (AGA) and 80 age-matched healthy controls. Table 1 shows the demographic and clinical characteristics of the study population. There was no statistically significant difference in the age between the two groups ($P = 0.324$). In contrast, AGA patients had significantly higher body mass index (BMI) compared to healthy controls ($P < 0.001$). Furthermore, the prevalence of current smoking and a positive family history of AGA was significantly higher in patients than in controls ($P = 0.046$ and $P < 0.001$, respectively). The mean duration of hair loss in AGA patients was 4.2 ± 2.0 years and the median disease severity was Hamilton–Norwood grade III (range II–IV).

Table 1. Demographic and clinical characteristics of the study population

Variable	AGA (n=80)	Controls (n=80)	P value
Age (years)	27.8 ± 4.2	27.1 ± 4.5	0.324
BMI (kg/m²)	27.6 ± 3.8	25.2 ± 3.1	<0.001
Current smokers, n (%)	34 (42.5)	22 (27.5)	0.046
Positive family history, n (%)	56 (70.0)	19 (23.8)	<0.001
Duration of hair loss (years)	4.2 ± 2.0	-	-
Hamilton-Norwood grade	III (II–IV)	-	-

3.2 Comparison of Metabolic and Hormonal Biomarkers

Table 2. Comparison of metabolic and hormonal biomarkers between patients with early onset androgenetic alopecia and healthy controls. The fasting glucose, fasting insulin, HOMA-IR, total cholesterol, triglycerides and LDL-C concentrations were significantly higher in AGA patients than healthy controls (all $P < 0.001$). The AGA group had significantly lower levels of HDL-C and total testosterone than the control group (both $P < 0.001$). Among the assessed biomarkers, the most significant differences between the two groups were HOMA-IR and fasting insulin, suggesting pronounced alterations in insulin sensitivity in patients with early-onset AGA.

Table 2. Comparison of biochemical parameters between patients and controls

Parameter	AGA	Controls	P value
Fasting glucose (mg/dL)	97.8 ± 10.4	91.6 ± 8.2	<0.001
Insulin (μIU/mL)	14.8 ± 4.1	9.6 ± 2.8	<0.001
HOMA-IR	3.61 ± 1.18	2.18 ± 0.74	<0.001
Total cholesterol (mg/dL)	208.3 ± 34.6	181.4 ± 28.2	<0.001
Triglycerides (mg/dL)	168.7 ± 41.5	126.3 ± 30.6	<0.001
HDL-C (mg/dL)	41.8 ± 8.4	49.6 ± 9.1	<0.001
LDL-C (mg/dL)	131.6 ± 29.8	107.5 ± 25.4	<0.001
Total testosterone (ng/mL)	4.38 ± 1.09	5.21 ± 1.16	<0.001

3.3 Correlation Between Disease Severity and Laboratory Parameters

Table 3 summarizes the correlations between Hamilton-Norwood grade and the clinical and biochemical parameters evaluated. Disease severity was significantly and positively correlated with BMI, fasting glucose, fasting insulin, HOMA-IR, total cholesterol, triglycerides, and LDL-C (all $P < 0.01$). Conversely, HDL-C and total testosterone were significantly and inversely correlated with Hamilton-Norwood grade ($P = 0.006$ and $P < 0.001$, respectively). Of the variables assessed, HOMA-IR had the strongest positive correlation with disease severity

(r = 0.54), followed by fasting insulin (r = 0.48) and LDL-C (r = 0.42), whereas total testosterone had the strongest negative correlation (r = −0.45).

Table 3. Correlation between Hamilton–Norwood grade and biochemical parameters

Variable	r	P value
BMI	0.36	<0.001
Fasting glucose	0.29	0.009
Insulin	0.48	<0.001
HOMA-IR	0.54	<0.001
Total cholesterol	0.33	0.003
Triglycerides	0.39	<0.001
HDL-C	−0.31	0.006
LDL-C	0.42	<0.001
Total testosterone	−0.45	<0.001

3.4 Binary Logistic Regression Analysis

Multivariable binary logistic regression analysis results are shown in Table 4. HOMA-IR, LDL-C, positive family history and total testosterone remained significant independent predictors of early onset androgenetic alopecia after adjustment for potential confounding variables. A positive family history exhibited the strongest association with the presence of AGA (adjusted OR = 5.64, 95% CI: 2.58–12.34, P < 0.001), followed by HOMA-IR (adjusted OR = 2.31, 95% CI: 1.52–3.52, P < 0.001). However, BMI and smoking were not independently associated with early-onset AGA after multivariable adjustment (P>0.05).

Table 4. Binary logistic regression analysis

Predictor	Adjusted OR	95% CI	P value
HOMA-IR	2.31	1.52-3.52	<0.001
LDL-C	1.03	1.01-1.05	0.002
BMI	1.08	0.99-1.18	0.081
Family history	5.64	2.58-12.34	<0.001
Smoking	1.69	0.89-3.18	0.109
Testosterone	0.58	0.41-0.82	0.002

3.5 Diagnostic Performance of Metabolic and Hormonal Biomarkers

The diagnostic performance of the investigated biomarkers for discrimination of patients with early-onset androgenetic alopecia from healthy controls is presented in Figure 1and Table 5.

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HOMA-IR showed the highest diagnostic accuracy (AUC = 0.871), followed by fasting insulin (AUC = 0.846), LDL-C (AUC = 0.791), total testosterone (AUC = 0.781) and BMI (AUC = 0.742). Among the studied biomarkers, HOMA-IR had the highest sensitivity (83.8%) and specificity (80.0%), indicating a greater discriminatory power than the other parameters studied.

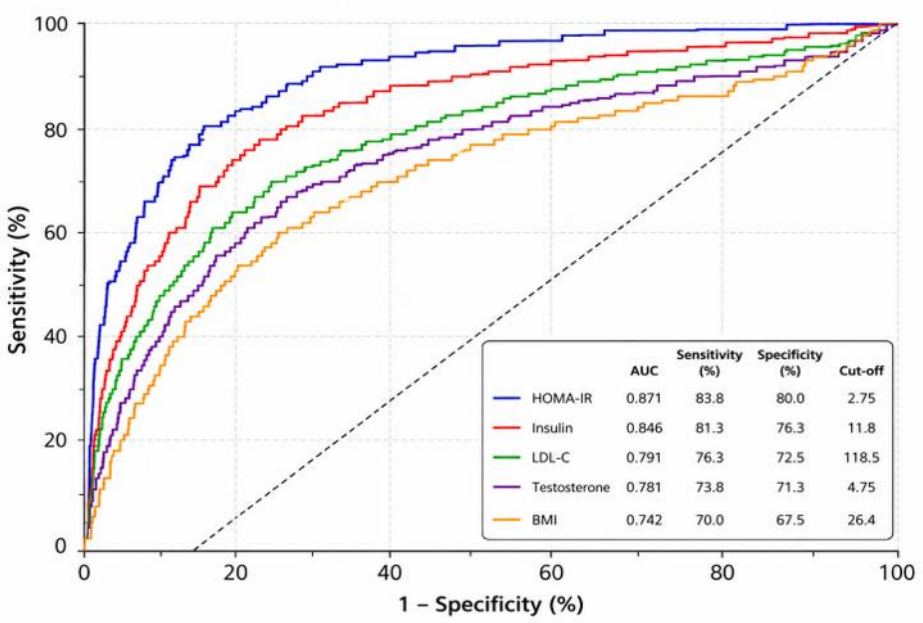

Table 5. ROC curve analysis of selected biomarkers

Biomarker	AUC	Sensitivity (%)	Specificity (%)	Cut-off
HOMA-IR	0.871	83.8	80.0	2.75
Insulin	0.846	81.3	76.3	11.8
LDL-C	0.791	76.3	72.5	118.5
BMI	0.742	70.0	67.5	26.4
Testosterone	0.781	73.8	71.3	4.75

3.6 Distribution of HOMA-IR, Fasting Insulin, and Total Testosterone

Distribution of HOMA-IR, fasting insulin and total testosterone levels in patients with early-onset androgenetic alopecia (AGA) and in healthy controls is shown in Figure 2. The patients with AGA had significantly higher HOMA-IR ($P < 0.001$) and fasting insulin levels ($P < 0.001$) than healthy controls. Conversely, the AGA group had significantly lower levels of total testosterone compared to the control group ($P < 0.001$). The box-and-whisker plots also demonstrate a clear separation between the two groups across all three biomarkers with little overlap in their distributions.

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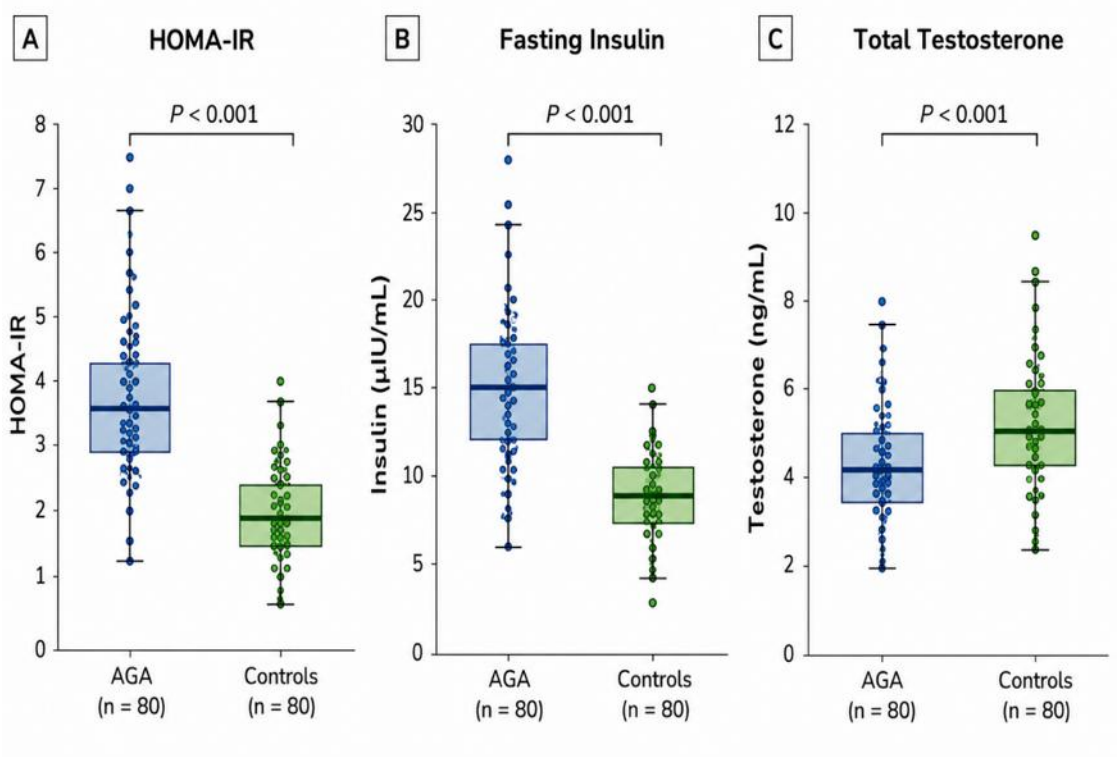

Figure 2. Box-and-whisker plots of HOMA-IR, fasting insulin and total testosterone levels in patients with early-onset androgenetic alopecia (AGA) and healthy controls. P values indicate between-group differences Individual data points are shown, with.

4. Discussion

The present study demonstrated that young men with early-onset androgenetic alopecia had a less favorable metabolic and hormonal profile compared to controls of similar age. The main findings were significantly higher BMI, fasting glucose, fasting insulin, HOMA-IR, total cholesterol, triglycerides and LDL-C and lower HDL-C and total testosterone concentrations. Furthermore, higher Hamilton-Norwood grade was linked to worse metabolic indices and family history, and HOMA-IR, LDL-C and testosterone were identified as independent factors associated with AGA by multivariable analysis. These findings support the hypothesis that early-onset AGA may be more than a localized cosmetic disorder and may be an externally visible phenotype associated with underlying metabolic changes.

The significantly higher levels of fasting insulin and HOMA-IR in the patients are consistent with the early report of Matilainen et al. who suggested that premature AGA may be a marker of hyperinsulinemia and disorders related to insulin resistance [5]. González-González et al. reported similar results, with significantly higher HOMA-IR in young men with AGA of Hamilton–Norwood grade III or more [6]. Moreover, Acibucu et al. [7] observed the increased prevalence of insulin resistance and metabolic syndrome in patients with early AGA. More recent data support these observations: Erden et al. reported significantly higher frequencies of

insulin resistance and metabolic syndrome in men aged between 18 and 35 years with early-onset AGA, with insulin resistance progressively more frequent with increasing Hamilton–Norwood stages [16].

However, the association of AGA with insulin resistance has not been established universally. Nabaie et al. reported that insulin resistance was associated with coexisting metabolic syndrome rather than AGA itself in young non-obese individuals [8]. Likewise, Swaroop et al. found a significant association between early-onset AGA and metabolic syndrome but not with insulin resistance considered independently [15]. Such discrepancies might be due to differences in sample size, adiposity of participants, severity of AGA, ethnic background, diagnostic cut-offs for HOMA-IR and selection of controls . But in the current analysis, HOMA-IR remained significant after adjustment for BMI and smoking, suggesting that the relationship was not explained only by general adiposity or lifestyle exposure.

The increased BMI and unfavorable lipid profile in the AGA group are in line with previous evidence linking early hair loss to cardiometabolic derangements. Arias-Santiago et al. reported significantly higher levels of total cholesterol, triglycerides, and LDL-C in patients with androgenetic alopecia [9], and in a larger cardiovascular evaluation, adverse anthropometric, metabolic, and vascular characteristics were found in patients with androgenetic alopecia [10]. Similarly, Bakry et al. found increased BMI, fasting glucose, triglycerides, insulin and HOMA-IR and decreased HDL-C in patients with moderate-to-severe AGA [13]. Su and Chen in community-based survey showed independent association between AGA and metabolic syndrome after adjustment for age, smoking and family history, with reduced HDL-C as an important metabolic component [14].

The positive associations of HOMA-IR, insulin, LDL-C, triglycerides, and BMI with Hamilton-Norwood grade also suggest that metabolic disturbances could be more severe with increasing disease severity. HOMA-IR had the strongest positive correlation and highest ROC performance with AUC of 0.871, sensitivity of 83.8% and specificity of 80.0%. ROC analysis in a cross-sectional study does not prove causation or support the use of HOMA-IR as a stand-alone diagnostic test for AGA. However, it suggests significant discrimination between patients and controls. Recent systematic review and meta-analysis of over 11000 AGA cases confirmed the association of insulin resistance, elevated fasting insulin, overweight/obesity, smoking and family history with the presence or progression of AGA [18].

It may seem paradoxical that the total testosterone concentration is lower in patients because AGA is androgen dependent. However, the disorder is a function of follicular androgen sensitivity, androgen-receptor activity, and local conversion of testosterone to dihydrotestosterone rather than elevated circulating testosterone alone. Similarly, González-González et al. reported elevated free testosterone in controls compared with young men with AGA [6]. Recent hormonal research has also suggested that systemic measurements of androgens may not be a direct reflection of local androgen signaling on the scalp [17]. Therefore, the inverse association between testosterone and AGA in this study should not be

taken to imply that lower testosterone causes hair loss; residual confounding and the limitations of a single morning measurement should also be considered.

A positive family history was the strongest independent association, while smoking was no longer significant after adjustment for multivariable factors. This pattern is biologically plausible given the large inherited component of AGA and suggests that the crude association with smoking may partly reflect covariance with metabolic or familial characteristics. In conclusion, the results support metabolic risk assessment in young men with early or advanced AGA particularly in those with increased BMI, dyslipidemia or strong family history [12,19].

Conclusion

Early-onset androgenetic alopecia was associated with significant metabolic and hormonal changes including increased BMI, insulin resistance and adverse lipid profile along with lower total testosterone levels. Metabolic abnormalities correlated positively with disease severity. AGA was independently predicted by HOMA-IR, LDL-C, family history and total testosterone. HOMA-IR had the best diagnostic performance among all the investigated biomarkers, implying that insulin resistance could be an important metabolic feature of early-onset AGA. These findings are consistent with the idea that AGA may be an easily recognizable clinical sign of underlying metabolic dysfunction in young men. These findings need to be confirmed in further large scale prospective studies and to determine whether early metabolic assessment and intervention can improve long term clinical outcomes.

Conflict of Interest

The authors declare that they have no conflict of interest.

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