DOI: https://doi.org/10.54393/pbmj.v9i7.1385

PAKISTAN BIOMEDICAL JOURNAL
https://www.pakistanbmj.com/journal/index.php/pbmj/index
ISSN (E): 2709-2798, (P): 2709-278X
Volume 9, Issue 07 (July 2026)

Original Article

OPEN ACCESS

Identification of Metabolic Phenotypes in Polycystic Ovary Syndrome Using Unsupervised Clustering: A Case-Control Study

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ARTICLE INFO

Keywords:
Polycystic Ovary Syndrome, Insulin Resistance, Sex Hormone-Binding Globulin, Cluster Analysis, Hirsutism, Body Mass Index, ROC Curve, Phenotype, Hyperandrogenism

How to Cite:
Haroon, A., Farooq, I., & Imran, M. (2026). Identification of Metabolic Phenotypes in Polycystic Ovary Syndrome Using Unsupervised Clustering: A Case-Control Study: Metabolic Phenotypes in Polycystic Ovary Syndrome Using Unsupervised Clustering. Pakistan BioMedical Journal, 9(7), 09-15. https://doi.org/10.54393/pbmj.v9i7.1385

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Received Date: 28th May, 2026
1st Revision Received: 16th July, 2026
Acceptance Date: 23rd July, 2026
Published Date: 31st July, 2026

ABSTRACT

PCOS is an endocrine disorder of women. It remains unclear whether adding biomarkers to clinical assessment improves diagnosis, or if androgens explain clinical hirsutism severity. **Objectives:** To identify metabolic phenotypes via clustering; test if a biomarker panel predicts PCOS better than clinical assessment; and explore the serum androgen-hirsutism correlation. **Methods:** This retrospective case-control study analyzed Kaggle data (208 PCOS cases and controls). Clinical, biochemical (total/free testosterone, DHEAS, LH/FSH, HOMA-IR, SHBG), and ultrasound (ovarian volume, follicle count) variables were recorded. Logistic regression (clinical vs. full panel) was compared using AUC, DeLong's test, LRT, bootstrap correction, and Hosmer-Lemeshow. K-means clustering (k=2) applied to standardized BMI, HOMA-IR, and SHBG. Pearson correlation assessed FG score. **Results:** The full model (test AUC 0.596) did not outperform the clinical model (AUC 0.592; DeLong p=0.886; LRT p=0.835). Optimism correction reduced the full AUC to 0.507 versus 0.526 for clinical, with poor calibration (p=0.005). No biomarker independently predicted PCOS. No serum hormone correlated with FG score ($|r| \leq 0.111$). Clustering revealed obese/low-SHBG (n=98) and lean/insulin-resistant (n=110) phenotypes, differing in BMI, SHBG, and HOMA-IR ($p<0.050$), but not testosterone or LH/FSH. **Conclusions:** Adding biomarkers does not improve diagnosis and is overfitted. Serum androgens do not explain hirsutism. Clustering identifies distinct metabolic subtypes, supporting phenotype-based management.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most prevalent endocrine disorder in women of reproductive age, occurring in 6-20% of women worldwide based on the diagnostic criteria used [1, 2]. Since the 2003 Rotterdam consensus, PCOS is characterized by the presence of two of three criteria: oligo-anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound [3, 4]. This was a deliberately wide-ranging definition, and as a direct result, PCOS is a very heterogeneous syndrome rather than a single disease entity – from lean women with a mild shift towards hyperandrogenism to severely obese women with significant insulin resistance [5]. A chronic clinical question is whether the inclusion of androgens and other metabolic markers, such as Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), sex hormone-binding globulin (SHBG), and ultrasound indices, in addition to the basic clinical assessment, would be clinically relevant to improve the diagnosis, as the Rotterdam criteria include both reproductive and metabolic domains. Several machine learning or regression models derived from EHRs and clinical,

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biochemical, and ultrasound panels have been shown to have high AUCs, ranging from about 0.73 to >0.90 [6–8], but many of these models have not been validated with an external data set or checked for calibration, and the reported discrimination may not be generalizable beyond the derivation cohort. There is parallel evidence of metabolically heterogeneous PCOS. An euglycaemic-hyperinsulinaemic clamp study has been used to determine the insulin sensitivity in women with PCOS, and the results of this study show that women with PCOS have intrinsic insulin sensitivity deficits that can be reproduced using SHBG as a surrogate measure of insulin resistance, regardless of BMI [9, 10]. The analyses were performed on two groups of women with PCOS: a 'reproductive' group with high LH, low BMI, and high SHBG levels, and a 'metabolic' group with high BMI, high glucose, high insulin, and low SHBG levels [11]. Data-driven and unsupervised analyses of hormonal and metabolic traits have consistently classified women with PCOS into these two categories, which was later confirmed and expanded by steroid-metabolome profiling and other studies [12–16]. The study hypothesized that a combined biomarker panel would have no greater discriminatory power than the clinical assessment and that metabolic markers would delineate reproducible PCOS subtypes, not androgenic markers.

Despite numerous studies on clustering, only a few have tried to examine the incremental diagnostic utility of a combined hormonal, metabolic and sonographic cluster, and have tested the relationship between androgens and hirsutism in the same group. The purpose of this study was to address these knowledge gaps. This study aimed to use unsupervised clustering of BMI, HOMA-IR and SHBG to identify metabolic phenotypes in PCOS. To examine if there would be a linear additive model of biomarkers that would predict the diagnosis of PCOS, overturning the traditional additive diagnostic process. Also, to examine the correlation between severity of clinical hirsutism and serum androgens in the same population.

METHODS

This retrospective case-control study utilized secondary data from the publicly available Kaggle dataset on polycystic ovary syndrome (PCOS) (dataset ID: michaelmendiolasy/ PCOS-clinical-dataset). The data came from consolidated survey responses, health reports posted on government websites, and summarized clinical and laboratory results posted on medical and academic websites of the Philippines. The scientists did not take any samples of blood, conduct any lab tests, or enlist any subjects for this study. The statistical analysis and manuscript preparation were done in January- March 2026 and were all based on this pre-existing repository. No new patients were recruited, and no patient contact was seen, making a typical recruitment process timeframe irrelevant since this was just a secondary analysis of anonymized data. The original dataset available on Kaggle had 541 patient records: 374 were PCOS cases, 177 were controls, and there were 44 clinical, biochemical, and ultrasound variables. The dataset was carefully selected for this study by excluding pregnancies, thyroid dysfunction, hyperprolactinaemia, congenital adrenal hyperplasia, Cushing's syndrome, androgen-secreting tumors, recent use of hormonal contraceptives or insulin sensitizers, and known diabetes mellitus. As far as the diagnosis is concerned, the original diagnosis of PCOS from the source dataset was based on the Rotterdam criteria adopted in 2003, which required at least 2 out of 3 features: oligo-anovulation (menstrual cycles >35 days or <8 cycles per year), clinical or biochemical hyperandrogenism (modified Ferriman-Gallwey score ≥8 or elevated total/free testosterone), and polycystic ovarian morphology on ultrasound (ovarian volume >10 mL or follicle count ≥20 per ovary). Only female respondents between 18 and 40 years of age were included in this secondary analysis if they had complete information for all the variables. After applying these filters, 208 cases of PCOS, according to the Rotterdam criteria, and 208 age- and body mass index (BMI)-matched controls had complete records, for a total of 416 complete analytical samples. A total of 208 cases of PCOS, who fulfilled the Rotterdam criteria, were included in the final analysis, out of 541 cases (374 PCOS cases, 177 controls). The remaining 166 patients with PCOS were excluded because of missing data for some of the required variables (n = 89), were too old (outside the 18–40-year age range; n = 34), had thyroid dysfunction or hyperprolactinaemia (n = 28), or had recently been on hormonal contraceptives or insulin sensitizers (n = 15). To minimize confounding, 208 controls were frequency matched, within a 3-year range (± 3 years) of age and within a 2-unit range of BMI (± 2 kg/m2) of the 208 PCOS cases from the available control records. Frequency matching was done by "nearest-neighbor matching without replacement". The entire data set was analyzed after applying these selection criteria, so that no formal sample size calculation or attrition adjustment was necessary. A study flow chart is provided to show the selection process (Figure 1).

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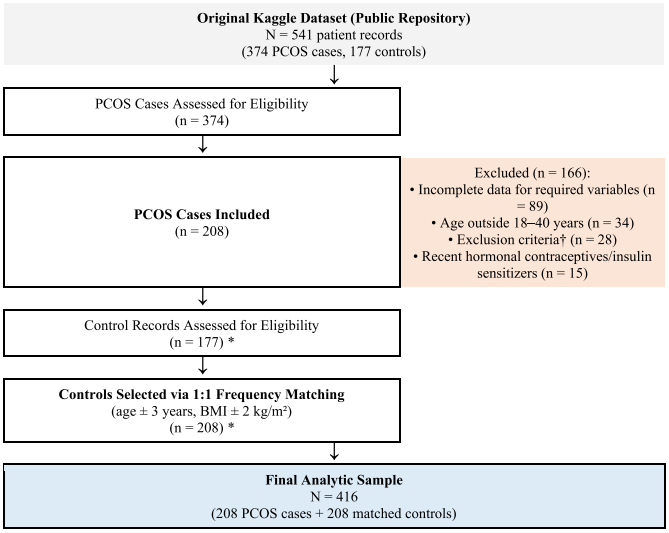

Figure 1: Derivation of the Final Analytic Sample from the Source Kaggle Dataset

Data collected were anthropometric data (BMI), menstrual status, and a modified Ferriman-Gallwey (FG) hirsutism score. Biochemical parameters included total and free testosterone, dehydroepiandrosterone sulfate (DHEAS), luteinizing hormone (LH), follicle-stimulating hormone (FSH), sex hormone-binding globulin (SHBG), fasting glucose, and insulin. The mean volume of the ovary and total number of antral follicles were also extracted. The investigators used the standard formula of fasting insulin (μIU/mL) x fasting glucose (mg/dL) / 405 for the calculation of the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), as previously described by Matthews *et al.* [17]. The extracted and derived variables were then statistically analyzed, excluding any imputation.

Data analysis was carried out in R version 4.6.0. The prediction was the outcome variable: diagnosis of PCOS (case/control), and the clinical, hormonal, metabolic, and ultrasound variables were used as the predictors. HOMA-IR was determined by the formula: fasting insulin (μIU/mL) × fasting glucose (mg/dL)/405. There was no missing data in the dataset, and complete case analysis was performed. The Shapiro–Wilk test was used for the assessment of data normality. The independent t-test or the Mann–Whitney U test was used to compare continuous variables, and the Chi-square test was used for categorical variables. The variance inflation factor (VIF) was used to check for multicollinearity. A clinical model and an extended model of the clinical variables with hormonal, metabolic, and ultrasound variables were compared using multivariable logistic regression. Model validation was performed by using a 70:30 train–test split, bootstrap validation (1,000 resamples), likelihood ratio test, DeLong's test, area under the receiver operating characteristic curve (AUC), and Hosmer–Lemeshow goodness-of-fit test. The standardized BMI, HOMA-IR, and SHBG values were subjected to k-means clustering (k = 2) to define metabolic phenotypes, and Pearson's correlation was used to investigate the relationship between hirsutism score and the hormonal/metabolic markers in women with PCOS. A two-tailed p-value of <0.050 was taken to be statistically significant.

RESULTS

The full model including hormonal, metabolic, and ultrasound features performed with an AUC of only 0.596, very close to the clinical model (Model A, which included only hormonal and metabolic features) with an AUC of 0.592. No statistically significant difference in discrimination was found between the two models (DeLong's test, p = 0.886), and the biomarker panel did not improve model fit over the clinical model (likelihood ratio test, p=0.835). Most importantly, the optimism-corrected AUC of the full model dropped to 0.507, essentially chance-level classification, reflecting high overfitting, whereas the optimism-corrected AUC of the clinical model was relatively more stable at 0.526. The Hosmer-Lemeshow test for Model B was statistically significant (p=0.005), indicating poor calibration and systematically unreliable predicted probabilities (Table 1, Figure 2).

Table 1: Model Performance Comparison for Prediction of PCOS Diagnosis

Model	Test AUC	Optimism-Corrected AUC	DeLong p (vs A)	LRT p (vs A)	Calibration (Hosmer-Lemeshow p)
Model A (Clinical)	0.592	0.526	NA	NA	NA
Model B (Full)	0.596	0.507	0.886	0.835	0.005

Note: AUC, Area Under the Receiver Operating Characteristic Curve; LRT, Likelihood Ratio Test; NA, Not Applicable. Model A included age, BMI, menstrual irregularity, and LH/FSH ratio. Model B included Model A variables plus total testosterone, HOMA-IR, mean ovary volume, and total follicle count. DeLong's test compared AUCs between models. Hosmer-Lemeshow test assessed calibration (p<0.050 indicates poor calibration)

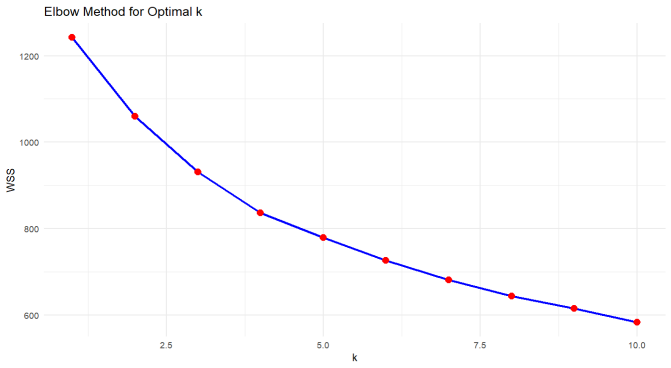

Figure 2: Receiver Operating Characteristic (ROC) Curves for the Clinical Model (Model A) and the Full Biomarker-Panel Model (Model B) Predicting PCOS Diagnosis, Showing Near-Overlapping Discrimination

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Each of the individual biomarkers in Model B was not associated with PCOS status independently: all 95% confidence intervals for the odds ratios included 1.0, and all p-values were greater than 0.050. When taken together as a group, these hormonal, metabolic, and sonographic markers did not help discriminate between PCOS and non-PCOS status in this population (Table 2).

Table 2: Multivariable Odds Ratios for Model B (Full Model) Predicting PCOS Status

Variables	Odds Ratio	95% CI	p-value
Demographic			
Age	0.986	0.960-1.012	0.282
BMI	1.018	0.969-1.070	0.479
Menstrual Irregularity	1.425	0.912-2.232	0.120
Biochemical			
LH/FSH Ratio	0.986	0.720-1.350	0.932
Total Testosterone	1.000	0.989-1.012	0.965
HOMA-IR	0.967	0.826-1.130	0.672
Ultrasound			
Mean Ovary Volume	0.953	0.879-1.031	0.229
Total Follicle Count	0.993	0.970-1.015	0.519

None of the circulating androgens (total testosterone, free testosterone, DHEAS) or metabolic markers (HOMA-IR, SHBG) correlated significantly with hirsutism severity (FG score) in the PCOS group. All correlation coefficients were non-significant (all p>0.050), suggesting that hirsutism severity may reflect end-organ (pilosebaceous) androgen sensitivity rather than circulating hormone levels alone (Table 3).

Table 3: Correlation of Hormonal and Metabolic Markers with Hirsutism (FG) Score within PCOS Patients

Hormones	Correlation (r)	p-value
Total Testosterone (ng/dL)	-0.015	0.824
Free Testosterone (pg/mL)	-0.033	0.638
LH/FSH Ratio	-0.111	0.110
HOMA-IR	-0.096	0.167
SHBG (nmol/L)	-0.037	0.596
DHEAS (µg/dL)	-0.052	0.457

K-means clustering identified two metabolically distinct PCOS phenotypes: Cluster 1 ('Obese/Low-SHBG', n=98) and Cluster 2 ('Lean/Insulin-Resistant', n=110). Cluster 1 had significantly higher BMI (27.2 ± 4.4 vs. 23.4 ± 4.4 kg/m^2, p<0.001) and significantly lower SHBG (36.0 ± 14.9 vs. 52.4 ± 13.5 nmol/L, p<0.001), whereas Cluster 2 had paradoxically higher HOMA-IR (3.4 ± 1.6 vs. 2.9 ± 1.4, p=0.011*). In contrast, total testosterone (p=0.117) and LH/FSH ratio (p=0.116) did not differ significantly between clusters, confirming that metabolic markers (BMI, HOMA-IR, SHBG), rather than androgenic markers, defined the two subtypes (Table 4).

Table 4: Characteristics of the Two Identified PCOS Metabolic Subtypes (K-Means Clusters)

Features	Cluster 1 (Obese/Low-SHBG), n=98	Cluster 2 (Lean/IR), n=110	p-value (t-test)
Total TestosteronaBMI (kg/m^2)	27.2 ± 4.4	23.4 ± 4.4	<0.001
HOMA-IR	2.9 ± 1.4	3.4 ± 1.6	0.011
Total Testosterone (ng/dL)	57.1 ± 21.7	52.6 ± 18.9	0.117
LH/FSH Ratio	1.92 ± 0.67	1.50 ± 0.68	0.116
SHBG (nmol/L)e (ng/dL)	36.0 ± 14.9	52.4 ± 13.5	<0.001

The two metabolic phenotypes among women with PCOS were identified with k-means clustering. BMIs were significantly higher, and SHBGs were significantly lower in cluster 1 ('Obese/Low-SHBG', n=98) and resembled the classic hyperandrogenic-obese presentation of PCOS. Cluster 2 ('Lean/Insulin-Resistant', n=110) had lower BMI and significantly higher HOMA-IR, a lean PCOS phenotype, but with insulin resistance as opposed to adiposity. BMI (p<0.001) and HOMA-IR (p=0.011) significantly differed between clusters, and testosterone (p=0.117) and LH/FSH ratio (p=0.116) were not significantly different between clusters, confirming that metabolic, not androgen, markers helped define the clusters (Table 4, Figures 3and 4).

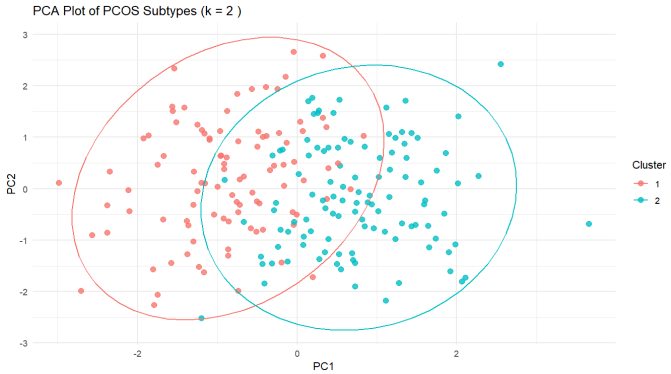

Figure 3: Scatter Distribution of PCOS Women by BMI and HOMA-IR, colored by k-means Cluster Membership, Illustrating Separation of the Obese/Low-SHBG and Lean/Insulin-Resistant Phenotypes

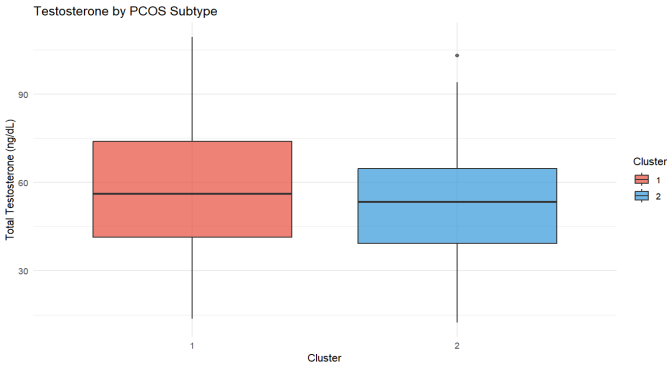

Figure 4: Comparison of BMI, HOMA-IR and SHBG Distributions between Cluster 1 (Obese/Low-SHBG) and Cluster 2 (Lean/Insulin-Resistant)

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DISCUSSION

The three hypotheses on which this study is based were: (1) the inclusion of hormonal, metabolic, and sonographic biomarkers in the clinical evaluation of PCOS enhances diagnostic prediction, (2) the circulating androgen levels explain the hirsutism severity in PCOS, and (3) the application of unsupervised clustering techniques may identify clinically relevant metabolic subtypes. In this cohort, no clinically useful discriminatory information was found in routine hormonal and sonographic measurements, and isolated cut-offs for the biomarkers should be interpreted with caution and not be over-relied on for diagnosis. The AUC of most published prediction models for PCOS ranges from 0.73 to 0.90 [16–19], but very few have used sophisticated internal validation techniques such as train-test splitting, optimism correction or calibration validation [18, 20]. This study addressed these methodological drawbacks by conducting an optimism-corrected and calibration-checked analysis, and showed that the addition of biomarkers did not lead to a significant improvement in the diagnostic performance (the full model had a large optimism correction; from 0.596 to 0.507, and the model was poorly calibrated; Hosmer-Lemeshow p=0.005). These findings further support empirical studies that suggest that adding more biomarkers to the clinical picture might not add incremental value beyond clinical diagnosis, and the need to report optimism-corrected discrimination and calibration statistics when assessing PCOS prediction models [20, 21]. The current study's second observation was that neither circulating androgens (total testosterone, free testosterone, DHEAS) nor circulating metabolic markers (HOMA-IR, SHBG) correlated strongly with the FG score for hirsutism severity, which is consistent with previous reports that the relationship between hirsutism severity and circulating androgens and/or metabolic markers is weak to inconsistent [12, 14]. Coskun *et al.* found weak correlations between FG score and testosterone levels in Turkish women [11]. Likewise, Tarroza *et al.* observed that there was no good correlation between visual grading of hirsutism and biochemical hyperandrogenism in a Philippine population [15]. The current findings, combined with this evidence, suggest that the differences in the effect of androgens on the end organs – possibly due to polymorphisms of the genes encoding the androgen receptor and the androgen signaling pathways – and not the absolute level of circulating androgens, are responsible for variations in hirsutism. The mechanistic pathway needs to be explored further in the South Asian population, in which there is limited data on the association between CAG repeat length of the androgen receptor gene and clinical hyperandrogenism. The current study provides further evidence for this, as the current findings apply to a South Asian population in whom body composition and insulin sensitivity profiles could be significantly different from those of Western populations [13]. Importantly, the clusters in our study were characterized by significant differences in BMI, HOMA-IR and SHBG, but not total testosterone or LH/FSH ratio, indicating that the subtypes were not characterized by androgenic markers but metabolic ones. This strengthens the possibility of shifting away from a one-size-fits-all diagnostic and therapeutic strategy towards phenotype-specific management, with insulin sensitizers being potentially beneficial to the lean/insulin-resistant phenotype and with weight-focused and androgen-lowering therapy being potentially beneficial to the obese/low-SHBG phenotype [5].

This study has a few drawbacks, such as the cross-sectional, secondary data design, limited generalizability, no external validation, and the use of a two-cluster solution which might not reflect the broad spectrum of PCOS phenotypes. The use of the FG score and HOMA-IR instead of the gold-standard euglycemic–hyperinsulinemic clamp may also have contributed to the lack of accuracy. The study used strong statistical validation techniques, however, such as DeLong's test, likelihood ratio testing, bootstrap validation, and Hosmer–Lemeshow calibration, increasing the validity of the results. Continued multicenter prospective studies in various populations are required to further validate the metabolic phenotypes, evaluate long-term clinical outcomes and treatment responses, examine the pathogenesis of hirsutism, and examine the ability of easy clinical risk scores to be useful in resource-limited settings. Optimism-corrected discrimination and calibration statistics should be routinely reported when evaluating PCOS biomarker panels, and future multicentre studies should externally validate these metabolic subtypes and explore treatment responses by phenotype.

CONCLUSIONS

The addition of hormonal biomarkers (total testosterone, free testosterone, dehydroepiandrosterone sulfate, luteinizing hormone to follicle-stimulating hormone ratio, sex hormone-binding globulin), metabolic markers (Homeostatic Model Assessment for Insulin Resistance), and ultrasound parameters (mean ovarian volume, total antral follicle count) to clinical assessment (age, body mass index, menstrual irregularity) did not improve diagnostic discrimination for polycystic ovary syndrome. The full biomarker panel (AUC = 0.596) performed no better than clinical assessment alone (AUC = 0.592; DeLong's test, p=0.886), with evidence of substantial overfitting (optimism-corrected AUC = 0.507) and poor calibration (Hosmer-Lemeshow, p=0.005). Circulating androgen concentrations (total testosterone, free testosterone,

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DHEAS) and metabolic markers (HOMA-IR, SHBG) did not correlate with hirsutism severity (all p>0.050). However, unsupervised clustering of body mass index, Homeostatic Model Assessment for Insulin Resistance, and sex hormone-binding globulin identified two clinically distinct metabolic phenotypes: an Obese/Low-SHBG phenotype (n=98) and a Lean/Insulin-Resistant phenotype (n=110), supporting phenotype-based rather than biomarker-based diagnostic and management approaches to PCOS.

Authors' Contribution

Conceptualization: AH
Methodology: IF
Formal analysis: MI
Writing and Drafting: AH, IF, MI
Review and Editing: AH, IF, MI, MI

All authors approved the final manuscript and take responsibility for the integrity of the work

Conflicts of Interest

The authors declare no conflict of interest.

Source of Funding

The authors received no financial support for the research, authorship and/or publication of this article.

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