Comparative analysis of hormonal and metabolic indices in phenotypic subgroups of polycystic ovary syndrome

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Abstract

Objective: To compare hormonal and metabolic characteristics across Rotterdam polycystic ovary syndrome (PCOS) phenotypes (A–D) and identify key predictors of hyperandrogenism.

Material and Methods: In this retrospective cohort study, women with PCOS were classified into four Rotterdam phenotypes. Hormonal and metabolic parameters were assessed in the early follicular phase, and composite indices including HOMA-IR, QUICKI, TG/HDL, and free androgen index (FAI) were calculated. Logistic regression and receiver operating characteristic analysis were used to evaluate predictors of hirsutism.

Results: The study included 226 women, with respective phenotype subgroups of: A n=85; B n=29; C n=43; and D n=69. Phenotype A showed the most pronounced hyperandrogenic and metabolic alterations, whereas phenotype D displayed the mildest profile with lower androgen levels and hirsutism scores. Significant differences in insulin resistance and lipid-related indices were observed across phenotypes. FAI was the strongest predictor of hirsutism (area under the curve =0.861), followed by total testosterone and dehydroepiandrosterone sulfate, while sex-hormone binding globulin was inversely associated.

Conclusion: PCOS phenotypes demonstrate distinct hormonal and metabolic patterns. Phenotype A represents the most metabolically and androgenically severe subgroup, whereas phenotype D is comparatively mild. FAI emerges as the most informative marker for hirsutism, supporting a phenotype-oriented approach to clinical assessment and follow-up in PCOS.

Keywords: Polycystic ovary syndrome, Rotterdam phenotypes, insulin resistance, TyG index, free androgen index, hirsutism, LH/FSH, AMH

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Introduction

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder in women of reproductive age, linked to heightened risks of subfertility, metabolic syndrome, type 2 diabetes, and cardiovascular disease (1,2). This condition, characterized by significant heterogeneity in reproductive, metabolic, and dermatological manifestations, is estimated to impact 5% to 15% of women globally (3-5).

The diagnosis of PCOS is based on the 2003 Rotterdam criteria, necessitating the presence of a minimum of two of the following: oligomenorrhea/anovulation; hyperandrogenemia; and polycystic ovarian appearance (6). The phenotypic

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variation arising from these criteria results in substantial disparities in clinical presentation, diagnosis, and treatment approaches (7). The National Institute of Health criteria established in 1990 delineated two principal phenotypes for polycystic ovarian syndrome. phenotype A is characterized by the presence of hyperandrogenism, oligoanovulation, and polycystic ovary morphology, whereas phenotype B is defined by hyperandrogenism and oligoanovulation in the absence of polycystic ovarian morphology. These two phenotypes are frequently designated as the "classic" forms of PCOS. Subsequently, the 2003 Rotterdam criteria and the 2006 Androgen Excess and PCOS Society guidelines refined this classification by introducing phenotype C, characterized by hyperandrogenism and polycystic ovaries without ovulatory dysfunction, and phenotype D, defined by oligo-anovulation and polycystic ovaries in the absence of hyperandrogenism (8,9).

Although the etiology of PCOS is not definitively known, genetic, environmental, and epigenetic factors are thought to be involved. Hormonal imbalances, particularly increased luteinizing hormone (LH), androgen excess, insulin resistance, and decreased sex hormone binding globulin (SHBG), are the frequently observed biochemical changes in PCOS (10). Clinically, it can manifest with symptoms such as oligomenorrhea, anovulation, hyperandrogenism, hirsutism, acne, and hair loss (11).

Recent studies have investigated the relationship between biochemical markers and various symptoms, highlighting their importance for diagnosis and prognosis. Markers such as the LH/follicle stimulating hormone (FSH) ratio, testosterone/SHBG, anti-Müllerian hormone (AMH) levels, and the insulin/glucose ratio have been shown to play different roles in different phenotypes of PCOS. However, combining these parameters to create new indices and investigating their relationship with symptoms remains understudied.

This study will assess the predictive power of biochemical parameters in PCOS patients, including in terms of differences between different PCOS phenotypes, and based on the data obtained, the relationship between indices that can be used in diagnosis and management and PCOS subtypes will be evaluated. This will enable earlier diagnosis of PCOS in clinical practice and the development of personalized treatment plans specific to each symptom.

Material and Methods

This retrospective cohort study was performed at a single center's infertility clinic from April to July 2025. The study protocol was approved by the University of Health Sciences Türkiye, Ankara Etlik City Hospital Ethics Committee (approval number: AEŞH-BADEK2-2025-176, date: 10.06.2024), and all procedures adhered to the Declaration of Helsinki. The study comprised women with PCOS, diagnosed according to the 2018 ESHRE/ASRM criteria, which require the presence of at least two of the following features: ovulatory dysfunction, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasonography (12). Participants were categorized into the four phenotypes of PCOS.

Phenotypes were categorized as follows: phenotype A, Hyperandrogenism + ovulatory dysfunction + polycystic ovarian morphology; phenotype B, Hyperandrogenism + ovulatory dysfunction; phenotype C, Hyperandrogenism + polycystic ovarian morphology; phenotype D, Ovulatory dysfunction + polycystic ovarian morphology (13,14).

Inclusion criteria were women aged 18–35 years who had a pre-existing diagnosis of PCOS and who presented to the outpatient clinic with symptoms related to PCOS, such as menstrual irregularity, hyperandrogenic manifestations, or infertility.

Exclusion criteria included: pregnancy; postpartum or lactation period; the presence of major systemic or psychiatric disorders; non-PCOS endocrine diseases (including thyroid dysfunction, hyperprolactinemia, congenital adrenal hyperplasia, and Cushing syndrome); conditions requiring intensive care monitoring; and incomplete clinical or laboratory data.

Participants' demographic and clinical data were recorded, and body mass index (BMI) was calculated after standardized measurment of height and weight, using the standard formula. Hirsutism was assessed using the modified Ferriman–Gallwey score, with a value of ≥8 being considered to indicate clinical hyperandrogenism. Venous blood samples were collected after an 8–12-hour fast during the early follicular phase of menstruation (days 2–5). Laboratory analyses included fasting glucose, insulin, lipid profile, and hormone levels (total and free testosterone, estradiol, prolactin, dehydroepiandrosterone sulfate (DHEA-S), 17-hydroxyprogesterone, LH, FSH, thyroid stimulating hormone, tri-iodothyronine, thyroxine and SHBG.

The homeostatic model assessment of insulin resistance (HOMA-IR) calculated as [(Fasting insulin × Fasting glucose)/405], quantitative insulin sensitivity check index (QUICKI) calculated using the formula [1/(log insulin + log glucose)] and triglyceride-glucose (TyG) index [ln (triglyceride × glucose/2)] were used to assess insulin resistance and metabolic status. Free androgen index (FAI) was calculated using the formula (total testosterone × 100)/SHBG (9,15-17).

Statistical analysis

Statistical analyses were performed using SPSS, version 29.0 (IBM Corp., Armonk, NY, USA). Data distribution was assessed using visual inspection and the Kolmogorov–Smirnov test. Continuous variables are expressed as median (interquartile range) or mean ± standard deviation, as appropriate, while

categorical variables are presented as counts and percentages. Comparisons between the four PCOS phenotypes (A–D) were conducted using the Kruskal–Wallis test for non-normally distributed continuous variables and one-way ANOVA for normally distributed variables, with post-hoc pairwise comparisons adjusted using Bonferroni correction. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Multinomial logistic regression analysis was used to identify independent hormonal and metabolic predictors of PCOS phenotypes, with phenotype D serving as the reference category. Results were reported as odds ratios (ORs) with 95% confidence intervals (CIs). A two-sided p-value <0.05 was considered statistically significant.

Results

Table 1 shows that age was similar across PCOS phenotypes, while BMI was significantly higher in phenotype A compared with phenotype D (p<0.001). Ovulatory dysfunction was present in phenotypes A, B, and D but absent in phenotype C. Hyperandrogenism was observed in phenotype A–C and not in phenotype D, and polycystic ovarian morphology differed significantly among phenotypes (all p<0.001).

Table 2 demonstrates significant hormonal and metabolic differences. Total and free testosterone were highest in phenotype A and lowest in phenotype D. Phenotype D had lower DHEA-S and higher SHBG levels (p<0.001), consistent with this being the mildest phenotype. Fasting insulin, triglycerides, and very low density lipoprotein levels were significantly elevated in phenotype A compared with other phenotypes.

Table 3 indicates that androgenic and metabolic indices differed across phenotypes. Phenotype A showed higher FAI, insulin resistance indices (HOMA-IR, TyG), and lower insulin sensitivity (QUICKI), whereas phenotype D displayed the most favorable metabolic profile (all p<0.05).

Table 4 shows that, using phenotype D as the reference, higher FAI was independently associated with phenotype (p<0.001). HOMA-IR was positively associated with phenotype A and B, QUICKI was inversely associated with phenotype A, and AMH was independently associated only with phenotype B.

As presented in Table 5, the FAI showed the strongest association with hyperandrogenism and the highest discriminative performance [OR =1.83, p<0.001; area under the curve (AUC) =0.861]. Total testosterone, SHBG, and DHEA-S also demonstrated significant predictive value, whereas metabolic indices showed limited or no discriminatory ability.

Discussion

This study compared the hormonal and metabolic profiles of PCOS subgroups in women of reproductive age, differentiated according to Rotterdam phenotypes (A–D). It was demonstrated that phenotype A was characterized by a more unfavorable metabolic profile (higher BMI, HOMA-IR, TyG, Tg/HDL; lower QUICKI) and significant hyperandrogenemia, while phenotype D exhibited the mildest phenotype, as expected, in terms of hyperandrogenemia markers including total testosterone, FAI and hirsutism. The study highlights the clinical implications of phenotypic heterogeneity and supports the individualization of screening and monitoring strategies based on Rotterdam phenotype.

Significant differences in reproductive and metabolic markers are reported between phenotypes in PCOS. In brief, it has been demonstrated in many studies that phenotype A has the

Table 1. Comparison of demographic, clinical, ultrasonographic, and laboratory characteristics among the four Rotterdam phenotypes of polycystic ovary syndrome

Variables	Phenotype A n=85	Phenotype B n=29	Phenotype C n=43	Phenotype D n=69	p-value
Demographics, median (IQR)					
Age (years)	25.0 (22.0, 27.0)	23.0 (21.0, 27.0)	26.0 (22.0, 29.0)	25.0 (23.0, 28.0)	0.298
BMI (kg/m²)	29.0 (25.0, 33.0)	25.0 (21.0, 34.0)	25.0 (23.0, 31.5)	24.0 (21.0, 27.0)	**<0.001**[a]
Clinical, ultrasonographic and laboratory findings, n (%), median (IQR)					
Ferriman-Gallwey score	12.0 (9.7, 15.0)	12.0 (11.0, 14.0)	12.0 (10.0, 13.0)	6.0 (5.0, 6.0)	**<0.001**[b]
Ovulatory dysfunction	85 (100.0)	29 (100.0)	0 (0.0)	69 (100.0)	**<0.001**
Hyperandrogenism	85 (100.0)	29 (100.0)	43 (100.0)	0 (0.0)	**<0.001**
PCOM	85 (100.0)	0 (0.0)	43 (100.0)	69 (100.0)	**<0.001**

Hyperandrogenism refers to the presence of clinical and/or biochemical hyperandrogenism as defined by the Rotterdam criteria
Variables with normal distribution were compared using one-way analysis of variance (ANOVA), while non-normally distributed variables were analyzed using the Kruskal–Wallis test. Categorical variables were compared using the chi-square test. Data are presented as median (interquartile range) or n (%), as appropriate. A p-value <0.05 was considered statistically significant
[a]Statistically significant differences were observed between phenotypes A vs. D
[b]Statistically significant differences were observed between phenotypes, D vs. A-C
BMI: Body mass index, PCOM: Polycystic ovary morphology, IQR: Interquartile range

most intense hyperandrogenemia and metabolic risks, while phenotype D has a profile characterized by predominant ovulatory dysfunction and weak hyperandrogenemia (18-22). Our findings are consistent with this general framework.

Previous studies have suggested that alterations in gonadotropin dynamics, particularly higher LH levels and an increased LH/FSH ratio, are more prominent in the classic PCOS phenotypes, especially phenotype A, and may help distinguish between phenotypic subgroups (18,20,23,24). In the present study, however, this pattern was not consistently replicated. In the multinomial logistic regression analysis, the LH/FSH ratio was not significantly associated with phenotype A or B when compared with the reference phenotype D, and a significant difference was observed only between phenotype C and D. Notably, this association was modest in magnitude, limiting its clinical interpretability. Overall, these findings suggest that the LH/FSH ratio may have a limited role in discriminating between PCOS phenotypes. Its clinical usefulness appears to be influenced by population characteristics and methodological variability, indicating that the LH/FSH ratio alone is unlikely to represent a reliable marker for phenotypic classification in PCOS in women of reproductive age.

Furthermore, we found AMH to be higher in phenotype A than in B and C, and lower in B than in D. Our findings support a recent meta analysis which found that AMH levels are associated with phenotypes in the sequence A > D > C > B (25), implying that the follicle pool and antral follicle number may differ according to phenotypes.

The high HOMA-IR and TyG and low QUICKI in phenotype A suggests that this phenotype is more prone to insulin resistance. Numerous studies have supported the association of the TyG index with IR and metabolic syndrome in PCOS, and its good diagnostic performance in distinguishing PCOS (26-28). In the present study, TyG values were significantly higher in phenotype

Table 2. Comparison of hormonal and biochemical parameters among the four Rotterdam phenotypes of polycystic ovary syndrome

Variables	Phenotype A n=85	Phenotype B n=29	Phenotype C n=43	Phenotype D n=69	p-value
Estradiol	38.9 (31.4, 50.0)	42.0 (35.0, 54.0)	36.0 (25.0, 48.4)	41.0 (33.6, 53.0)	0.147
FSH (mIU/mL)	5.4 (4.5, 6.3)	5.9 (5.1, 6.5)	5.6 (5.0, 6.1)	5.5 (4.7, 6.5)	0.460
LH (mIU/mL)	9.2 (7.0, 13.0)	8.4 (5.7, 12.0)	9.0 (6.2, 11.9)	11.0 (8.2, 16.0)	0.064
Prolactin (ng/mL)	15.0 (10.0, 21.0)	19.0 (14.0, 25.0)	19.0 (13.0, 23.0)	16.0 (12.0, 21.0)	0.096
TSH (μIU/mL)	1.9 (1.4, 2.8)	2.2 (1.4, 3.2)	2.4 (1.5, 3.0)	2.1 (1.5, 2.7)	0.500
T3 (ng/mL)	3.2 (2.9, 3.5)	3.2 (2.9, 3.5)	3.2 (2.9, 3.4)	3.2 (3.0, 3.4)	0.778
T4 (μg/dL)	1.1 (1.1, 1.2)	1.1 (1.0, 1.3)	1.2 (1.0, 1.3)	1.2 (1.1, 1.3)	0.675
Total testosterone (ng/dL)	60.9±21.1	48.7±11.8	51.4±16.2	40.6±13.3	**<0.001**[a]
Free testosterone (pg/mL)	2.5 (1.8, 3.3)	2.4 (1.5, 3.2)	2.6 (1.4, 3.4)	2.0 (1.4, 2.6)	0.022[b]
17-hydroxyprogesterone (ng/mL)	1.1 (0.9, 1.5)	1.0 (0.7, 1.3)	1.1 (0.8, 1.9)	1.0 (0.8, 1.3)	0.232
DHEA-S (μg/dL)	325±119.0	317±159.0	318±115.0	232±80.2	**<0.001**[c]
SHBG (nmol/L)	22.0 (17.3, 32.0)	33.0 (22.0, 45.0)	25.0 (19.5, 36.0)	42.0 (35.0, 49.0)	**<0.001**[c]
Fasting glucose (mg/dL)	91.0 (83.0, 96.0)	88.0 (81.0, 95.0)	86.0 (82.5, 94.0)	85.0 (81.0, 92.0)	0.119
Fasting insulin (μIU/mL)	18.0 (10.0, 28.8)	12.0 (9.0, 19.0)	11.3 (6.7, 19.6)	11.0 (7.6, 16.0)	**0.001**[d]
Total cholesterol (mg/dL)	177 (151, 199)	166 (147, 190)	175 (158, 188)	172 (148, 198)	0.757
LDL (mg/dL)	116±33.3	108±28.7	115±21.3	113±28.4	0.711
Triglycerides (mg/dL)	132 (102, 181)	100 (60, 156)	112 (71, 141)	98 (69, 156)	**0.019**[b]
HDL (mg/dL)	47.2±10.6	48.6±12.0	49.1±11.6	51.6±11.9	0.138
VLDL (mg/dL)	23.0 (18.0, 36.0)	18.0 (12.0, 24.0)	16.5 (13.3, 25.5)	18.0 (14.5, 25.5)	0.007[d]

Variables with normal distribution were compared using one-way analysis of variance (ANOVA), while non-normally distributed variables were analyzed using the Kruskal–Wallis test. Categorical variables were compared using the chi-square test. Data are presented as median (interquartile range) or n (%), as appropriate. A p-value <0.05 was considered statistically significant

[a]Statistically significant differences were observed between phenotypes, A vs. B-D, D vs. B, C

[b]Statistically significant differences were observed between phenotypes A vs. D

[c]Statistically significant differences were observed between phenotypes, D vs. A-C

[d]Statistically significant differences were observed between phenotypes, A vs. C, D

FSH: Follicle-stimulating hormone, LH: Luteinizing hormone, TSH: Thyroid-stimulating hormone, T3: Total triiodothyronine, T4: Total thyroxine, DHEA-S: Dehydroepiandrosterone sulfate, AMH: Anti-Müllerian hormone, SHBG: Sex hormone-binding globulin, HOMA-IR: Homeostatic model assessment of insulin resistance, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, VLDL: Very low-density lipoprotein

A, supporting the concept that metabolic dysregulation is most pronounced in the classic PCOS phenotype. Given the strongest association with hirsutism was found for the FAI. Moreover, the high AUC value reaffirmed the usefulness of FAI in detecting biochemical hyperandrogenemia in the clinic. It has been reported that FAI offers significant accuracy in the diagnosis and phenotyping of PCOS (29). The inverse association of SHBG with hirsutism was also an expected finding (30) as a low SHBG increases the free androgen fraction, increasing the risk of clinical hyperandrogenemia.

Study limitations

Our study has several limitations. The relatively small overall sample size may have reduced the statistical power of some analyses, particularly for subgroup comparisons. In addition, the unequal distribution of participants across PCOS phenotypes (with smaller sample sizes in certain groups) may have further limited the power to detect modest differences between phenotypes. The retrospective design also limited the ability to establish causal relationships. Furthermore, unmeasured confounders such as lifestyle and genetic factors could not be completely excluded. Larger, prospective, multicenter studies with more balanced phenotype group sizes are needed to confirm and extend our findings.

Our findings highlight the importance of a phenotype-sensitive clinical screening and management approach: (i) early glycemic and lipid monitoring (TyG, HOMA-IR, Tg/HDL) and weight management for phenotype A; (ii) less focus on

Table 3. Comparison of hormonal and metabolic ındices among the four rotterdam phenotypes of polycystic ovary syndrome

Variables	Phenotype A n=85	Phenotype B n=29	Phenotype C n=43	Phenotype D n=69	p-value
FAI	8.80 (5.70, 13.00)	5.80 (2.80, 8.30)	6.50 (4.25, 10.50)	3.40 (2.10, 4.00)	**<0.001**[a]
LH/FSH ratio	1.75 (1.38, 2.22)	1.58 (1.02, 2.25)	1.57 (1.11, 2.20)	2.22 (1.36, 2.81)	**0.021**[b]
AMH	7.20 (5.60, 9.10)	4.20 (3.20, 6.50)	5.60 (4.45, 7.75)	6.85 (5.27, 8.77)	**<0.001**[c]
HOMA-IR	4.00 (2.20, 6.80)	2.70 (1.90, 4.40)	2.60 (1.40, 4.20)	2.30 (1.60, 3.30)	**<0.001**[d]
TyG index	8.74 (8.39, 9.07)	8.44 (8.01, 8.81)	8.51 (8.02, 8.71)	8.43 (7.88, 8.77)	**0.006**[e]
QUICKI	0.312 (0.290, 0.338)	0.329 (0.314, 0.345)	0.335 (0.309, 0.362)	0.335 (0.319, 0.354)	**<0.001**[e]

Variables with normal distribution were compared using one-way analysis of variance (ANOVA), while non-normally distributed variables were analyzed using the Kruskal–Wallis test. Categorical variables were compared using the chi-square test. Data are presented as median (interquartile range) or n (%), as appropriate. A p-value <0.05 was considered statistically significant
[a]Significant differences between A vs. B,D, and D vs. B, C
[b]Significant difference between C vs. D
[c]Significant differences between A vs. B, C and B vs. D
[d]Significant difference between A vs. D
[e]Significant differences between A vs. C, D
FAI: Free androgen index, LH: Luteinizing hormone, FSH: Follicle-stimulating hormone, AMH: Anti-Müllerian hormone, Tg: Triglyceride, HDL: High-density lipoprotein cholesterol, HOMA-IR: Homeostatic model assessment of ınsulin resistance, TyG index: Triglyceride-glucose index, QUICKI: Quantitative insulin sensitivity check index

Table 4. Multinomial logistic regression analysis of hormonal and metabolic predictors across polycystic ovary syndrome phenotypes, using phenotype D as the reference category

Reference category Phenotype D	Phenotype A		Phenotype B		Phenotype C	
Variables	**OR (95% CI)**	**p-value**	**OR (95% CI)**	**p-value**	**OR (95% CI)**	**p-value**
FAI	1.90 (1.56-2.31)	**<0.001**	1.64 (1.33-2.04)	**<0.001**	1.90 (1.56-2.31)	**<0.001**
LH/FSH ratio	0.82 (0.61-1.08)	0.170	0.67 (0.42-1.06)	0.092	0.62 (0.41-0.94)	**0.025**
AMH	1.00 (0.99-1.00)	0.979	0.66 (0.53-0.82)	**<0.001**	1.00 (0.99-1.00)	0.801
HOMA-IR	1.28 (1.12-1.48)	**<0.001**	1.23 (1.05-1.44)	**0.009**	1.15 (0.98-1.35)	0.078
TyG ratio	1.01 (0.76-1.36)	0.898	0.79 (0.47-1.31)	0.361	0.72 (0.45-1.15)	0.171
QUICKI	0.98 (0.97-1.00)	**<0.001**	0.98 (0.97-1.00)	0.097	0.99 (0.98-1.00)	0.483

Values are presented as odds ratios (OR) with 95% confidence intervals (CI). Phenotype D (non-hyperandrogenic PCOS) was used as the reference category in the multinomial logistic regression model. A p-value <0.05 was considered statistically significant.
FAI: Free androgen index, LH: Luteinizing hormone, FSH: Follicle-stimulating hormone, AMH: Anti-Müllerian hormone, HOMA-IR: Homeostatic model assessment of insulin resistance, TyG index: Triglyceride-glucose index, QUICKI: Quantitative insulin sensitivity check index, PCOS: Polycystic ovary syndrome

Table 5. Predictive performance of hormonal and metabolic markers for hyperandrogenism

	OR	95% CI	p-value	AUC
FAI	1.83	1.51-2.22	**<0.001**	0.861
LH/FSH ratio	0.74	0.57-0.96	**0.026**	0.617
AMH (ng/mL)	1.00	0.99-1.00	0.818	0.435
HOMA-IR	1.24	1.08-1.42	**0.001**	0.624
TyG ratio	0.91	0.69-1.20	0.509	0.475
QUICKI	0.98	0.98-0.99	0.004	0.624
T. Testosterone/DHEA-S ratio	0.76	0.03-17.14	0.866	0.490
Total testosterone	1.06	1.03-1.08	**<0.001**	0.755
Free testosterone	1.47	1.10-1.97	**0.008**	0.625
17-hydroxyprogesterone	1.10	0.75-1.59	0.616	0.545
DHEA-S	1.00	1.00-1.01	**<0.001**	0.723
SHBG	0.98	0.97-0.99	**0.008**	0.796

FAI: Free androgen index, LH: Luteinizing hormone, FSH: Follicle-stimulating hormone, AMH: Anti-Müllerian hormone, HOMA-IR: Homeostatic model assessment of insulin resistance, TyG index: Triglyceride-glucose index, QUICKI: Quantitative insulin sensitivity check index, OR: Odds ratio, CI: Confidence interval, AUC: Area under the curve, SHBG: Sex hormone-binding globulin, DHEA-S: Dehydroepiandrosterone sulfate
Significant results are shown in bold ($p<0.05$ was considered statistically significant)

markers of hyperandrogenemia and more on management of ovulatory dysfunction for phenotype D; and (iii) strengthening biochemical confirmation with FAI and SHBG in cases of suspected hirsutism. These recommendations will reduce the impact of phenotypic heterogeneity on clinical workload and patient education.

Conclusion

PCOS phenotypes have distinct hormonal–metabolic profiles. Phenotype A was characterized by insulin resistance and hyperandrogenemia (higher HOMA-IR, TyG, TG/HDL, lower QUICKI), whereas phenotype D was the mildest with the lowest hirsutism burden. FAI was the most informative marker for hirsutism, and TyG remained associated with phenotype A after BMI adjustment, supporting simple, low-cost metabolic risk stratification. LH/FSH is population- and method-sensitive and should be interpreted contextually. Care should be phenotype-guided, and findings warrant prospective multicenter validation and threshold refinement.

Ethics

Ethics Committee Approval: *The study protocol was approved by the University of Health Sciences Türkiye, Ankara Etlik City Hospital Ethics Committee (approval number: AEŞH-BADEK2-2025-176, date: 10.06.2024), and all procedures adhered to the Declaration of Helsinki.*

Informed Consent: *Waived due to the retrospective study design.*

Footnotes

Author Contributions: *Surgical and Medical Practices: F.B.F., Concept: A.K., Design: B.S.Ü., Data Collection or Processing: E.Ö., T.D.A., Analysis or Interpretation: C.O.U., Literature Search: Ö.V.A., Writing: B.S.Ü.*

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