Original Article

DOI: 10.14235/bas.galenos.2025.38980
Bezmialem Science

Phenotype-specific Variations in Vitamin D, Body Mass Index, Insulin Resistance, Anti-Mullerian Hormone, and Lipid Levels in Women with Polycystic Ovary Syndrome

Polikistik Over Sendromu Fenotipleri Arasında D Vitamini, Vücut Kitle İndeksi, İnsülin Direnci, Anti-Müllerian Hormon ve Lipid Profillerinin Karşılaştırılması

Fatih Şanlıkan[1], İsmail Bağlar[2], Esra Keleş[1], Uğur Kemal Öztürk[1], Emre Mat[2], Metin Ayğar[2]

[1]University of Health Sciences Türkiye, Kartal Dr. Lütfi Kırdar City Hospital, Department of Gynecologic Oncologic, İstanbul, Türkiye
[2]University of Health Sciences Türkiye, Kartal Dr. Lütfi Kırdar City Hospital, Department of Obstetrics and Gynecology, İstanbul, Türkiye

ABSTRACT

Objective: Polycystic ovary syndrome (PCOS) is a heterogeneous endocrine disorder characterized by diverse phenotypes that differ in metabolic and hormonal risk. The integrated profiles of vitamin D, body mass index (BMI), insulin resistance, anti-Mullerian hormone (AMH), and lipid parameters across these phenotypes remain incompletely defined. The aim of the study was to compare serum 25-hydroxyvitamin D [25(OH)D], AMH, insulin resistance [homeostasis model assessment of insulin resistance (HOMA-IR)], BMI, and lipid profiles among the four PCOS phenotypes defined by the Rotterdam criteria.

Methods: This retrospective study included 420 women aged 18-40 years with PCOS classified as phenotype A (hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology), B (hyperandrogenism and ovulatory dysfunction), C (hyperandrogenism and polycystic ovarian morphology), and D (ovulatory dysfunction and polycystic ovarian morphology). Serum 25(OH)D, AMH, fasting glucose, fasting insulin, and lipid parameters were measured; HOMA-IR was calculated. Data were analyzed using one-way ANOVA or Kruskal-Wallis tests ($p<0.05$ was considered significant).

ÖZ

Amaç: Polikistik over sendromu (PKOS), farklı metabolik ve hormonal risk profillerine sahip çeşitli fenotiplerle karakterize heterojen bir endokrin bozukluktur. Serum 25-hidroksivitamin D [25(OH)D], vücut kitle indeksi (VKİ), insülin direnci [homeostatik model değerlendirmesi-insülin direnci (HOMA-IR)], anti-Müllerian hormon (AMH) ve lipid parametrelerinin bu fenotipler arasındaki bütüncül profilleri halen tam olarak ortaya konulamamıştır. Bu çalışmanın amacı, Rotterdam kriterlerine göre tanımlanan dört PKOS fenotipi arasında serum 25(OH)D, AMH, HOMA-IR, VKİ ve lipid profillerini karşılaştırmaktır.

Yöntemler: Bu retrospektif çalışmaya, 2003 Rotterdam kriterlerine göre PKOS tanısı alan 18-40 yaş arası 420 kadın dahil edildi. Katılımcılar A (hiperandrojenizm, ovulatuvar disfonksiyon ve polikistik over morfolojisi), B (hiperandrojenizm ve ovulatuvar disfonksiyon), C (hiperandrojenizm ve polikistik over morfolojisi) ve D (ovulatuvar disfonksiyon ve polikistik over morfolojisi) fenotipleri olarak sınıflandırıldı. Serum 25(OH)D, AMH, açlık glukozu, açlık insülini ve lipid parametreleri ölçüldü; HOMA-IR hesaplandı. Veriler tek yönlü ANOVA veya Kruskal-Wallis testleri kullanılarak analiz edildi ve $p<0,05$ istatistiksel olarak anlamlı kabul edildi.

Address for Correspondence: Assoc. Prof., Fatih Şanlıkan, University of Health Sciences Türkiye, Kartal Dr. Lütfi Kırdar City Hospital, Department of Gynecologic Oncologic, İstanbul, Türkiye
E-mail: fatihroland@hotmail.com
ORCID IDs of the authors: F.Ş.: 0000-0002-3166-7129, İ.B.: 0009-0008-0619-7111, E.K.: 0000-0001-8099-8883, U.K.Ö.: 0000-0003-0720-2919, E.M.: 0000-0002-3965-543X, M.A.: 0000-0001-7801-8583

Cite this article as: Şanlıkan F, Bağlar İ, Keleş E, Öztürk UK, Mat E, Ayğar M. Phenotype-specific variations in vitamin D, body mass index, insulin resistance, anti-Mullerian hormone, and lipid levels in women with polycystic ovary syndrome. Bezmialem Science. [Epub Ahead of Print]

Received: 31.07.2025
Accepted: 23.12.2025
Epub: 29.06.2026

ABSTRACT

Results: Phenotype distribution was A: 35.7%, B: 28.6%, C: 21.4%, and D: 14.3%. Significant inter-phenotype differences were found for BMI, HOMA-IR, AMH, high-density lipoprotein (HDL) cholesterol, and triglycerides (all p<0.01). Phenotypes A and B showed higher mean BMI (31.5±6.1, 30.8±5.8 kg/m²) and HOMA-IR [3.9 (2.8-5.5), 3.7 (2.7-5.1)] and lower HDL cholesterol (41.5±8.5, 42.8±9.1 mg/dL) compared with C and D (p<0.001). Triglycerides were higher in A and B, while AMH was elevated in A [8.8 (6.5-11.2) ng/mL] and C [8.5 (6.8-11.0) ng/mL] versus D [7.1 (5.5-9.5) ng/mL] (p=0.008). No significant differences were observed in vitamin D, total cholesterol, or low-density lipoprotein cholesterol (p>0.05); vitamin D deficiency was prevalent in all phenotypes.

Conclusion: Hyperandrogenic and anovulatory PCOS phenotypes (A and B) exhibit more adverse metabolic profiles with higher BMI, insulin resistance, and dyslipidemia. Vitamin D deficiency was universal but phenotype-independent. Phenotype-specific evaluation may improve metabolic risk stratification and individualized management in women with PCOS.

Keywords: Polycystic ovary syndrome (PCOS), phenotypes, metabolic risk factors, vitamin D, anti-Mullerian hormone (AMH), insulin resistance

ÖZ

Bulgular: Fenotip dağılımı A: %35,7, B: %28,6, C: %21,4, D: %14,3 idi. Fenotipler arasında VKİ, HOMA-IR, AMH, yüksek yoğunluklu lipoprotein (HDL) kolesterol ve trigliserid düzeylerinde anlamlı fark bulundu (tümü p<0,01). A ve B fenotiplerinde VKİ (31,5±6,1; 30,8±5,8 kg/m²) ve HOMA-IR [3,9 (2,8-5,5); 3,7 (2,7-5,1)] değerleri daha yüksek, HDL kolesterol (41,5±8,5; 42,8±9,1 mg/dL) düzeyi ise C ve D'ye göre daha düşüktü (p<0,001). Trigliserid düzeyleri A ve B'de daha yüksekti. AMH düzeyleri A [8,8 (6,5-11,2) ng/mL] ve C [8,5 (6,8-11,0) ng/mL] fenotiplerinde D fenotipine [7,1 (5,5-9,5) ng/mL] göre anlamlı derecede yüksekti (p=0,008). Serum 25(OH)D, toplam kolesterol ve LDL düzeyleri arasında fark saptanmadı (p>0,05); D vitamini eksikliği tüm fenotiplerde yaygındı.

Sonuç: Hiperandrojenizm ve anovulasyon ile karakterize PKOS fenotipleri (A ve B), daha yüksek VKİ, insülin direnci ve dislipidemi ile seyreden daha olumsuz metabolik profillere sahiptir. D vitamini eksikliği tüm fenotiplerde yaygın olup fenotipten bağımsızdır. Fenotipe özgü değerlendirme, PKOS'lu kadınlarda metabolik risk sınıflandırmasını ve bireyselleştirilmiş yönetimi iyileştirebilir.

Anahtar Kelimeler: Polikistik over sendromu (PKOS), fenotipler, metabolik risk faktörleri, D vitamini, anti-Müllerian hormon (AMH), insülin direnci

Introduction

Polycystic ovary syndrome (PCOS) is a heterogeneous endocrine disorder, and the Rotterdam criteria classify affected women into four phenotypes according to combinations of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology (PCOM) (phenotypes A-D). This classification is not merely descriptive; it reflects meaningful variability in clinical presentation and longer-term metabolic risk and therefore offers a practical framework for risk stratification and individualized care (1-3).

From a pathophysiologic perspective, body mass index (BMI), insulin resistance [commonly estimated by the homeostasis model assessment, homeostasis model assessment of insulin resistance (HOMA-IR)], anti-Mullerian hormone (AMH), vitamin D status, and lipid parameters represent interrelated domains that help explain both the reproductive and cardiometabolic expression of PCOS. Prior studies show that vitamin D deficiency is frequent in PCOS and associates with adverse metabolic features; AMH—although a marker of follicular dynamics—also varies across phenotypes and may align with disease severity; insulin resistance and adiposity are central to metabolic risk; and dyslipidemia [lower high-density lipoprotein (HDL) cholesterol and higher triglycerides] characterizes the atherogenic profile often seen in PCOS. These markers were selected a priori because, together, they capture hormonal milieu (AMH), nutritional/endocrine modulation (vitamin D), core metabolic load (BMI and HOMA-IR), and downstream cardiovascular risk (lipids)—domains that map onto phenotype-level differences and are relevant to routine clinical decision-making (4-7).

Despite this rationale, the combined, phenotype-specific profile of these parameters was incompletely defined. Our study addresses this gap by comparing serum 25-hydroxyvitamin D [25(OH)D], AMH, insulin resistance (HOMA-IR), BMI, and a standard lipid panel across the four Rotterdam phenotypes in a well-characterized cohort, minimizing redundancy by listing the parameters once here and applying them consistently in the analyses that follow (8,9).

This phenotype-anchored approach has direct clinical implications. If hyperandrogenic phenotypes (A and B) demonstrate higher BMI, greater insulin resistance, and a more atherogenic lipid profile than phenotypes C and D—as our data indicate—then clinicians can prioritize early metabolic screening (oral glucose tolerance or HOMA-IR estimation), aggressive weight-centered interventions, lipid management, and cardiometabolic counseling in these groups. Conversely, in phenotypes where AMH is relatively higher without commensurate metabolic burden, emphasis may be placed on reproductive planning and ovulation-focused strategies. Similar vitamin D levels across phenotypes suggest that deficiency screening and replacement should be considered broadly in PCOS rather than restricted to a single phenotype. In sum, classifying patients by Rotterdam phenotype can guide practical, phenotype-sensitive choices in diagnostic work-up (e. g., frequency and intensity of metabolic testing) and in management (e. g., lifestyle targets, pharmacotherapy for

insulin resistance or dyslipidemia), thereby operationalizing precision care in everyday practice (10-12).

Methods

This retrospective cross-sectional study was conducted using data of patients diagnosed with PCOS between January 1, 2020 and January 1, 2025 at University of Health Sciences Türkiye, Kartal Dr. Lütfi Kırdar City Hospital, Department of Gynecology and Obstetrics. Ethical approval was obtained (University of Health Sciences Türkiye, Kartal Dr. Lütfi Kırdar City Hospital, 05 June 2025, decision no: 2025/010.99/17/41), and all data were anonymized. Due to the retrospective design of the study and the use of anonymized data, the requirement for informed consent was waived by the Institutional Ethics Committee.

A total of 420 women aged 18-40 years with a definite diagnosis of PCOS according to the 2003 Rotterdam criteria (at least two of the following: oligo/anovulation, clinical or biochemical hyperandrogenism, PCOM on ultrasound) and complete laboratory data (vitamin D, AMH, insulin resistance parameters, and lipid profile) were included.

Phenotype Assignment and Diagnostic Definitions

Patients were categorized into four phenotypes based on the combination of diagnostic features: A=hyperandrogenism + ovulatory dysfunction + polycystic ovaries; B=hyperandrogenism + ovulatory dysfunction; C=hyperandrogenism + polycystic ovaries; D=ovulatory dysfunction + polycystic ovaries.

Clinical hyperandrogenism was defined using the modified Ferriman-Gallwey (mFG) score, which assesses terminal hair growth in nine body areas (upper lip, chin, chest, upper and lower back, upper and lower abdomen, upper arm, thigh). Each site was scored from 0 (no terminal hair) to 4 (extensive growth), and scores were summed to yield a total between 0 and 36. An mFG score ≥8 was accepted as indicative of clinical hyperandrogenism. Biochemical hyperandrogenism was defined when at least one androgen parameter (total testosterone, free androgen index, or dehydroepiandrosterone sulfate) exceeded laboratory reference ranges.

Waist circumference was measured in centimeters with a flexible non-elastic tape at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest, while the participant was standing and at the end of gentle expiration. BMI was calculated as weight (kg) divided by height squared (m^2). Ovulatory dysfunction was defined as oligomenorrhea (cycle length >35 days or <8 cycles per year) or amenorrhea (≥90 days without menses). Menstrual cycle length was recorded as the average number of days between spontaneous bleeding episodes in the preceding 12 months. PCOM was defined as ≥12 follicles measuring 2-9 mm in diameter in at least one ovary and/or an ovarian volume >10 cm^3 on transvaginal ultrasound.

Exclusion Criteria

Women with endocrinopathies mimicking PCOS (non-classical congenital adrenal hyperplasia, Cushing syndrome, hyperprolactinemia, thyroid dysfunction, androgen-secreting tumors), serious systemic diseases (chronic hepatic/renal failure, cardiovascular disease, uncontrolled diabetes, active malignancy, autoimmune disorders), pregnancy, use of relevant medications (hormones, steroids, high-dose vitamin D in the previous 3 months), or missing data were excluded.

Laboratory Assessments

Morning fasting venous blood samples were analyzed in the central laboratory using standardized and quality-controlled methods.

- 25(OH)D: Automated chemiluminescent immunoassay (ng/mL).
- AMH: Automated enzyme immunoassay (ng/mL).
- Fasting glucose: Hexokinase method; fasting insulin: automated immunoassay; HOMA-IR=[fasting insulin (μIU/mL)×fasting glucose (mg/dL)]/405.
- Lipid profile: Total cholesterol, HDL, low-density lipoprotein (LDL), and triglycerides via enzymatic colorimetric assays (mg/dL).

Statistical Analysis

Analyses were performed using SPSS. Normality was assessed with the Shapiro-Wilk test and histograms. Continuous variables were compared among phenotypes using one-way analysis of variance (post-hoc Tukey/Bonferroni) or Kruskal-Wallis (post-hoc Dunn's) tests as appropriate. A p-value <0.05 was considered statistically significant. Post-hoc pairwise comparisons were explicitly performed to determine which phenotypes differed from each other. In all tables, statistically significant between-group differences are indicated by superscript letters or footnotes (e. g., A>C&D, $p<0.05$).

Rationale for the Absence of a Control Group

Because the study aimed to examine intra-PCOS phenotype differences, not PCOS versus healthy controls, no control group was included. Healthy population metabolic data are well-established in the literature, and our focus was to delineate phenotype-specific risk variation among PCOS patients.

Results

The basic demographic and clinical characteristics of the 420 PCOS patients included in the study, according to phenotypes, are presented comparatively in Table 1. Phenotype A constituted 35.7% of the study population, phenotype B 28.6%, phenotype C 21.4%, and phenotype D 14.3%. While no statistically significant difference was

observed between the groups in mean age (p=0.152), statistically significant differences were observed between the phenotypes in terms of BMI, waist circumference, mFG score, and mean menstrual cycle length (p<0.001). Specifically, BMI and waist circumference were statistically significantly higher in hyperandrogenic and anovulatory phenotypes A and B compared to the other phenotypes. Similarly, the mFG score was statistically significantly higher in phenotypes A, B, and C, which included hyperandrogenism, as expected, compared to phenotype D. Median menstrual cycle length values were statistically significantly longer in phenotypes A, B, and D, which included anovulation, than in phenotype C, which had regular menstruation. Comparison of baseline hormonal and metabolic parameters examined in our study among PCOS phenotypes revealed statistically significant differences (Table 2). Median values of BMI, which is critical for metabolic risk, and HOMA-IR, which reflects insulin resistance, were observed to be statistically significantly higher in the hyperandrogenic phenotypes phenotype A and phenotype B compared to the normoandrogenic phenotype D and the ovulatory phenotype C (p<0.001). A similar trend was observed in the lipid profile; median triglyceride values were statistically significantly higher in phenotypes A and B (p<0.001), and mean HDL cholesterol, known to be protective, was statistically significantly lower in these two groups compared to phenotypes C and D (p<0.001).

AMH levels, which are associated with ovarian reserve, also demonstrated statistically significant differences between phenotypes (p=0.008); specifically, phenotypes A and C had higher median values than phenotype D. However, serum 25(OH)D levels, which were generally low across the entire patient group, were not statistically significantly different among the four phenotype groups (p=0.315). Similarly, mean total cholesterol and LDL cholesterol levels did not differ statistically significantly between phenotypes (p=0.188 and p=0.240, respectively).

Discussion

The present study provides a detailed phenotype-based assessment of metabolic and hormonal characteristics among women with PCOS. Our findings confirm that the hyperandrogenic phenotypes (A and B) exhibit a more adverse metabolic profile, with statistically significantly higher BMI, waist circumference, insulin resistance (HOMA-IR), and triglyceride levels, as well as lower HDL cholesterol, compared to the ovulatory or normoandrogenic phenotypes (C and D). These results are consistent with several

Table 1. Baseline demographic and clinical characteristics of patients according to phenotypes

Feature	Phenotype A (n=150)	Phenotype B (n=120)	Phenotype C (n=90)	Phenotype D (n=60)	p-value
Age (years, mean ± SD)	28.1±4.9	28.8±5.3	28.5±5.0	28.0±5.5	0.152
BMI (kg/m², mean ± SD)	31.5±6.1^{ab}	30.8±5.8^{ab}	28.1±5.2^{c}	27.5±4.9^{c}	<0.001
Waist circumference (cm, mean ± SD)	96.5±11.2^{ab}	94.8±10.5^{ab}	89.1±9.8^{c}	87.5±9.1^{c}	<0.001
mFG score [median (IQR)]	11 (8-15)^{a}	10 (7-14)^{a}	9 (6-13)^{a}	4 (2-6)^{b}	<0.001
Cycle length [days, med (IQR)]	55 (40-90)^{a}	60 (45-95)^{a}	29 (27-31)^{b}	50 (38-85)^{a}	<0.001

P-value calculated by ANOVA, Kruskal-Wallis or chi-square test. There is a statistically significant difference in post-hoc tests between groups with different superscript letters (^{a,b,c}) in the same row (p<0.05)
ANOVA: Analysis of variance, BMI: Body mass index, mFG: Modified Ferriman-Gallwey, SD: Standard deviation, IQR: Interquartile range

Table 2. Comparison of clinical and laboratory parameters according to PCOS phenotypes

Parameter	Phenotype A (n=150)	Phenotype B (n=120)	Phenotype C (n=90)	Phenotype D (n=60)	p-value
BMI (kg/m², mean ± SD)	31.5±6.1^{ab}	30.8±5.8^{ab}	28.1±5.2^{c}	27.5±4.9^{c}	<0.001
25(OH)D (ng/mL, mean ± SD)	16.2±7.5	17.1±8.0	17.5±8.8	18.0±9.1	0.315
AMH [ng/mL, median (IQR)]	8.8 (6.5-11.2)^{a}	8.2 (6.1-10.8)^{ab}	8.5 (6.8-11.0)^{a}	7.1 (5.5-9.5)^{b}	0.008
HOMA-IR [median (IQR)]	3.9 (2.8-5.5)^{ab}	3.7 (2.7-5.1)^{ab}	2.9 (2.0-4.0)^{c}	2.6 (1.9-3.8)^{c}	<0.001
Total cholesterol (mg/dL, mean ± SD)	195±35	192±38	188±33	185±30	0.188
HDL cholesterol (mg/dL, mean ± SD)	41.5±8.5^{ab}	42.8±9.1^{ab}	46.5±9.8^{c}	47.8±10.2^{c}	<0.001
LDL cholesterol (mg/dL, mean ± SD)	115±30	112±32	110±28	108±25	0.240
Triglycerides [mg/dL, median (IQR)]	155 (110-210)^{ab}	148 (105-195)^{ab}	125 (90-160)^{c}	118 (85-155)^{c}	<0.001

P-value was calculated by ANOVA or Kruskal-Wallis tests. There is a statistically significant difference in post-hoc tests between groups with different superscript letters (^{a,b,c}) in the same row (p<0.05)
ANOVA: Analysis of variance, SD: Standard deviation, IQR: Interquartile range, PCOS: Polycystic ovary syndrome, BMI: Body mass index, 25(OH)D: Serum 25-hydroxyvitamin D, AMH: Anti-Mullerian hormone, HOMA-IR: Homeostasis model assessment of insulin resistance, HDL: High-density lipoprotein, LDL: Low-density lipoprotein

previous reports emphasizing that hyperandrogenism and anovulation jointly contribute to worsened cardiometabolic risk in PCOS (3,8,9).

When analyzed individually, phenotype A (hyperandrogenic + anovulatory + PCOM) demonstrated the most pronounced metabolic deterioration, aligning with Yilmaz et al. (8) and Panidis et al. (9), who reported that phenotype A carried the highest insulin resistance and dyslipidemia burden. Phenotype B, which also includes hyperandrogenism and anovulation but lacks PCOM, demonstrated a similar but slightly less severe metabolic profile, suggesting that ovulatory dysfunction and androgen excess rather than ovarian morphology primarily drive metabolic abnormalities. Phenotype C (hyperandrogenic + PCOM with regular cycles) maintained higher AMH levels and mFG scores than phenotype D but exhibited more favorable metabolic indices, reflecting the potential mitigating effect of preserved ovulatory function. Finally, phenotype D, characterized by ovulatory dysfunction and PCOM but without hyperandrogenism, represented the mildest metabolic profile, corroborating prior evidence that normoandrogenic phenotypes have lower cardiovascular and metabolic risk (9,13,14).

Waist circumference and BMI, both markers of central and total adiposity, were statistically significantly elevated in phenotypes A and B. Central adiposity plays a crucial role in insulin resistance and androgen excess, and our data support the strong correlation between abdominal fat accumulation and metabolic risk in PCOS (13,15).

Similarly, the mFG score, which quantifies the degree of hirsutism, was statistically significantly higher in phenotypes A, B, and C—all of which include hyperandrogenism—consistent with the pathophysiologic link between androgen excess and clinical hair growth (3,9). Menstrual cycle length was also markedly prolonged in anovulatory phenotypes A, B, and D, aligning with the expectation that ovulatory dysfunction manifests clinically as oligomenorrhea or amenorrhea. These findings collectively reinforce that both reproductive and metabolic features vary systematically across Rotterdam phenotypes.

AMH levels differed significantly among PCOS phenotypes in the present study, with higher concentrations observed in phenotypes A and C compared with the normoandrogenic phenotype D. This finding is consistent with previous reports demonstrating that AMH is closely related to follicle number, granulosa cell activity, and androgen excess rather than directly reflecting metabolic severity (10,11). Importantly, although phenotypes A and C exhibited higher AMH levels, our study design does not allow conclusions regarding a causal relationship between AMH and metabolic disturbances. The observed differences in AMH should therefore be interpreted as phenotype-associated hormonal variations rather than indicators of metabolic burden.

In this context, AMH may be considered a complementary biomarker reflecting ovarian and endocrine characteristics across PCOS phenotypes, rather than a direct marker of insulin resistance or cardiometabolic risk. The coexistence of elevated AMH and adverse metabolic features in phenotype A likely reflects the clustering of hyperandrogenism, anovulation, and ovarian dysfunction within this phenotype, rather than a direct pathophysiological role of AMH in metabolic dysregulation. Future prospective studies incorporating longitudinal follow-up and multivariable analyses are needed to clarify whether AMH has any independent predictive value for metabolic outcomes in women with PCOS.

Despite the widespread vitamin D deficiency observed across all groups, we observed no statistically significant inter-phenotype difference. This aligns with the findings of Eftekhar et al. (7) and He et al. (16), though some studies suggested lower vitamin D levels in hyperandrogenic phenotypes (7,12). Given that vitamin D deficiency is nearly universal in PCOS, factors such as sun exposure, dietary intake, and genetic variation likely override phenotype-based differences (16,17). Nevertheless, consistent deficiency underscores the need for population-wide vitamin D screening in women with PCOS, regardless of phenotype.

The novelty of this study lies in its comprehensive evaluation of both hormonal and metabolic parameters across all four Rotterdam phenotypes within a large, single-center cohort, integrating anthropometric, biochemical, and endocrine indices into a unified comparison. By highlighting that each phenotype exhibits a distinct combination of metabolic, reproductive, and biochemical characteristics, this work strengthens the rationale for phenotype-specific risk stratification in clinical practice. From a practical standpoint, phenotypes A and B should be monitored more closely for metabolic syndrome, dyslipidemia, and insulin resistance, whereas phenotype C may primarily benefit from reproductive-focused management. Phenotype D patients, though metabolically milder, may still require long-term follow-up for reproductive dysfunction. These phenotype-based distinctions can guide individualized counseling, diagnostic testing, and early intervention strategies, aligning with precision medicine principles in PCOS management (13,14,18).

Study Limitations

This study has several limitations that should be acknowledged. First, its retrospective and single-center design precludes causal inferences between PCOS phenotypes and metabolic or hormonal parameters; therefore, the observed associations should be interpreted descriptively rather than as cause-effect relationships. Second, because data were collected from medical records, variability in clinical and laboratory measurements could not be entirely excluded. In particular, serum 25(OH)D

levels were measured at different times of the year, and information on seasonal variation, sun exposure, dietary intake, and supplementation was not available, which may have attenuated potential phenotype-related differences. Third, insulin resistance was assessed using HOMA-IR, a widely accepted but indirect surrogate marker; more advanced methods such as euglycemic-hyperinsulinemic clamp studies were not feasible in this retrospective setting. Additionally, lifestyle-related factors including physical activity, dietary habits, smoking, and alcohol consumption were not systematically recorded and therefore could not be included in the analyses. The absence of a healthy control group limits comparisons with the general population; however, the primary aim of this study was to evaluate intra-PCOS phenotype differences rather than PCOS versus non-PCOS comparisons. Despite these limitations, the major strength of the study lies in its relatively large sample size and comprehensive, phenotype-based evaluation of anthropometric, metabolic, and hormonal parameters within a well-defined PCOS cohort.

Conclusion

By expanding the comparative analysis beyond metabolic indices to include anthropometric and clinical features such as waist circumference, mFG score, and menstrual cycle length, our study provides a nuanced understanding of how distinct PCOS phenotypes manifest across multiple biological dimensions. The differential pattern of AMH further emphasizes the endocrine diversity of the syndrome. Collectively, these findings add novel evidence supporting integrated, phenotype-based evaluation as a cornerstone for improving diagnostic accuracy and optimizing personalized management in PCOS, particularly in guiding phenotype-oriented metabolic screening strategies in routine clinical practice.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Ethics Committee of University of Health Sciences Türkiye, Kartal Dr. Lütfi Kırdar City Hospital, on 05 June 2025 (decision no: 2025/010.99/17/41).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: F.Ş., İ.B., E.M., M.A., Concept: F.Ş., E.K., U.K.Ö., M.A., Design: F.Ş., İ.B., E.K., E.M., Data Collection or Processing: F.Ş., İ.B., M.A., Analysis or Interpretation: F.Ş., İ.B., E.K., U.K.Ö., E.M., Literature Search: F.Ş., İ.B., U.K.Ö., E.M., M.A., Writing: F.Ş., İ.B., E.K., M.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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