Gazi Med J 2026;37(3):411-418

DOI: http://dx.doi.org/10.12996/gmj.026.4683

The Impact of Obesity on the Metabolic Profile of Patients with Polycystic Ovary Syndrome

Polikistik Over Sendromlu Hastalarda Obezitenin Metabolik Profil Üzerine Etkisi

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ABSTRACT

Objective: The aim of this study was to evaluate the effects of polycystic ovary syndrome (PCOS) and accompanying obesity on various metabolic and biochemical parameters, particularly body composition, insulin resistance (IR), lipid profile, and inflammatory markers. **Methods:** A total of 120 women aged 18–40 years were included in the study: 40 obese patients with PCOS, 40 normal-weight (non-obese) patients with PCOS, and 40 healthy controls. The diagnosis of PCOS was established according to the Rotterdam criteria. Participants were classified based on body mass index (BMI) as obese (BMI ≥30 kg/m²) or non-obese (BMI <25 kg/m²). Body composition was assessed using bioelectrical impedance analysis. Serum fasting glucose, insulin, glycated hemoglobin (HbA1c), lipid profile, C-reactive protein (CRP), alanine aminotransferase, aspartate aminotransferase, anti-Müllerian hormone (AMH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol, and progesterone were analyzed. IR was calculated using the homeostatic model assessment of IR (HOMA-IR) method. Statistical comparisons between groups were performed using the Kruskal–Wallis test followed by Dunn's post-hoc analyses.

Results: Body weight, BMI, total fat mass, and body fat percentage were significantly higher in the obese PCOS group than in the other groups, whereas body water percentage and high-density lipoprotein (HDL) cholesterol levels were significantly lower (p < 0.001). Fasting insulin levels and HOMA-IR were significantly higher in the obese PCOS

ÖZ

Amaç: Bu çalışmanın amacı, polikistik over sendromunun (PCOS) ve eşlik eden obezitenin, özellikle vücut kompozisyonu, insülin direnci, lipid profili ve inflamatuvar belirteçler olmak üzere çeşitli metabolik ve biyokimyasal parametreler üzerindeki etkilerini değerlendirmektir.

Yöntemler: Çalışmaya 18–40 yaşları arasında, PCOS tanılı 40 obez hasta, PCOS tanılı 40 normal kilolu hasta ve 40 sağlıklı kontrol olmak üzere toplam 120 kadın dahil edilmiştir. PCOS tanısı Rotterdam kriterlerine göre konulmuştur. Bireyler vücut kütle indeksine (VKİ) göre obez (VKİ ≥30 kg/m²) ve obez olmayan (VKİ <25 kg/m²) olarak sınıflandırılmıştır. Vücut kompozisyonu Biyoelektrik Empedans Analizi ile değerlendirilmiştir. Serum açlık glukozu, insülin, HbA1c, lipid profili, C-reaktif protein (CRP), alanin aminotransferaz (ALT), aspartat aminotransferaz, anti-Müllerian hormon (AMH), luteinizan hormon (LH), folikül uyarıcı hormon (FSH), östradiol ve progesteron düzeyleri analiz edildi. İnsülin direnci HOMA-IR yöntemi kullanılarak hesaplanmıştır. Gruplar arasındaki istatistiksel karşılaştırmalar Kruskal-Wallis testi ve ardından Dunn post hoc analizleri ile gerçekleştirilmiştir.

Bulgular: Obez PCOS grubunda vücut ağırlığı, vücut kütle indeksi (VKİ), toplam yağ kütlesi ve vücut yağ yüzdesi diğer gruplara göre anlamlı derecede daha yüksek iken, vücut suyu yüzdesi ve HDL kolesterol düzeyleri anlamlı derecede daha düşüktür (p < 0,001). Açlık insülini ve HOMA-IR düzeyleri obez PCOS grubunda hem kontrol grubuna hem de obez olmayan PCOS grubuna göre anlamlı derecede daha yüksek tespit

Cite this article as: Koçoğlu SE, Kavutçu M, Güler İ, Canpunar N, Dashdamirova M, Nehri LN, et al. The impact of obesity on the metabolic profile of patients with polycystic ovary syndrome. Gazi Med J. 2026;37(3):411-418

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Received/Geliş Tarihi: 09.04.2026
Accepted/Kabul Tarihi: 09.05.2026
Publication Date/Yayınlanma Tarihi: 10.07.2026

ABSTRACT

group than in both the control group and the non-obese PCOS group ($p < 0.001$). Additionally, HbA1c levels were higher in the obese PCOS group compared with both the control group ($p < 0.001$) and the non-obese PCOS group ($p < 0.01$). Similarly, triglyceride (TG), CRP, and ALT levels were higher in the obese PCOS group than in the other groups. AMH levels were higher in the non-obese PCOS group than in the control group. In contrast, no statistically significant differences were found among the groups in LH levels, FSH levels, the LH/FSH ratio, or progesterone levels. Correlation analyses showed that BMI and fat mass were positively associated with indicators of IR and with levels of TG, CRP, and ALT, and negatively associated with HDL cholesterol and body water percentage.

Conclusion: The severity of metabolic impairment in PCOS is closely associated with obesity and increased fat mass. Obesity is one of the main determinants of IR, dyslipidemia, and inflammation. Therefore, improving body composition is of great importance in the management of PCOS to reduce metabolic risk.

Keywords: Polycystic ovary syndrome, obesity, insulin resistance, body composition, inflammation, lipid profile

ÖZ

edilmiştir ($p < 0,001$). Ayrıca HbA1c düzeyleri obez PCOS grubunda kontrol grubuna ($p < 0,001$) ve obez olmayan PCOS grubuna ($p < 0,01$) göre daha yüksek bulunmuştur. Benzer şekilde trigliserid, CRP ve ALT düzeyleri de obez PCOS grubunda diğer gruplara göre daha yüksekti. AMH düzeyleri obez olmayan PCOS grubunda kontrol grubuna göre daha yüksektir. Buna karşın, LH, FSH, LH/FSH oranı ve progesteron düzeyleri açısından gruplar arasında istatistiksel olarak anlamlı bir farklılık bulunmamıştır. Korelasyon analizleri, VKİ ve yağ kütlesinin insülin direnci göstergeleri, trigliserid, CRP ve ALT düzeyleri ile pozitif; HDL kolesterol ve vücut suyu yüzdesi ile negatif ilişkili olduğunu göstermiştir.

Sonuç: Sonuç olarak, PCOS’ta metabolik bozulmanın şiddeti obezite ve artmış yağ kütlesi ile yakından ilişkilidir. Obezite, insülin direnci, dislipidemi ve inflamasyonun temel belirleyicilerinden biridir. Bu nedenle metabolik riskin azaltılması amacıyla PCOS yönetiminde vücut kompozisyonunun iyileştirilmesi büyük önem taşımaktadır.

Anahtar Sözcükler: Polikistik over sendromu, obezite, insülin direnci, vücut kompozisyonu, inflamasyon, lipid profili

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a heterogeneous disorder characterized by ovulatory dysfunction and hyperandrogenism, with both metabolic and endocrine features. It is one of the most common hormonal disorders, affecting approximately 6%–20% of women worldwide. Most of the clinical manifestations of PCOS emerge during adolescence (1). The Rotterdam Criteria (2003) are currently the most widely accepted international diagnostic criteria for PCOS (2). According to these criteria, PCOS is diagnosed when at least two of the following are present: clinical or biochemical hyperandrogenism (hirsutism, androgenetic alopecia, acne, or elevated serum testosterone levels), oligo-ovulation or anovulation, and polycystic ovarian morphology on ultrasonography (3,4).

PCOS is a multifactorial syndrome that arises from the interaction of metabolic disturbances, genetic predisposition, environmental factors, and intrauterine developmental processes. Genetic and epigenetic mechanisms are considered major determinants of its etiology. Previous studies have shown that PCOS shares a common genetic background with obesity, insulin resistance (IR), type 2 diabetes, and cardiometabolic diseases (5). In addition, environmental factors such as obesity, IR, and inflammation may influence epigenetic regulation and contribute to the transgenerational transmission of the PCOS phenotype (6,7). In this context, obesity and IR are regarded not only as conditions that accompany PCOS but also as key factors that determine the clinical course and severity of PCOS.

The World Health Organization classifies a body mass index (BMI) of 18.5–24.9 kg/m^2 as normal weight, 25.0–29.9 kg/m^2 as overweight, and ≥30.0 kg/m^2 as obesity (8,9). Epidemiological data indicate that the prevalence of obesity among women with PCOS varies across regions. In Australia, the prevalence of obesity in women with PCOS has been reported as 47%, whereas in India, approximately 20.7% of women diagnosed with PCOS were found to be obese and 7.5% to be overweight (10,11). A systematic review including multiple countries showed that the prevalence of obesity in women with PCOS ranges from 38% to 50% (12).

The aim of this study was to compare body composition, IR, lipid profile, and inflammatory markers between obese and non-obese women with PCOS and to elucidate the impact of metabolic impairment on the relationship between PCOS and obesity.

MATERIALS AND METHODS

Study Design

A total of 120 women aged 18–40 years who presented to the Gynecology and Obstetrics Outpatient Clinic of Gazi University Faculty of Medicine were included in the study. The study included three groups: control (n = 40), obese PCOS (n = 40), and non-obese PCOS (n = 40).

PCOS was diagnosed according to the Rotterdam Criteria, and participants were classified based on BMI as obese (BMI ≥30 kg/m^2) or non-obese (BMI <25 kg/m^2).Overweight individuals with a BMI of 25.0–29.9 kg/m^2 were excluded from the study to preserve group homogeneity.

The control group consisted of healthy women who had regular menstrual cycles, normal ovarian morphology on ultrasonographic evaluation, no signs of hirsutism, acne, or alopecia, had not used oral contraceptives within the previous three months, and had no endocrinological disorders. Individuals who were pregnant or suspected to be pregnant; who had a diagnosis of Cushing syndrome, congenital adrenal hyperplasia, adrenal tumor, hyperprolactinemia, thyroid dysfunction, virilizing tumors, diabetes, hepatic or renal dysfunction, any malignancy, central nervous system disease, or hypertension; or were current smokers were excluded from the study across all groups.

Anthropometric Measurements and Biochemical Analyses

Anthropometric measurements, including BMI, height, body weight, body fat percentage, total fat mass, lean body mass, total body water, and body water percentage, were assessed by bioelectrical impedance analysis (BIA).

In blood samples obtained from the patients, levels of anti-Müllerian hormone (AMH), triglycerides (TG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), total cholesterol, fasting glucose, fasting serum insulin, glycated hemoglobin (HbA1c), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and C-reactive protein (CRP) were analyzed in the Medical Biochemistry Laboratory of Gazi University Faculty of Medicine Hospital. IR was calculated using the homeostatic model assessment of insulin resistance (HOMA-IR) formula: fasting glucose (mg/dL) × fasting insulin (µIU/mL)/405.

Serum AMH levels were analyzed using the electrochemiluminescence immunoassay method. TG levels were measured by the enzymatic colorimetric method, while HDL levels were determined by a photometric method. LDL cholesterol levels were calculated using the Friedewald formula when TG levels were below 400 mg/dL; when TG levels were above 400 mg/dL, LDL cholesterol levels were measured using a homogeneous enzymatic colorimetric method. HbA1c analyses were performed using high-performance liquid chromatography. CRP levels were analyzed using a nephelometric assay, whereas ALT and AST levels were measured using the kinetic photometric method.

Ethics Committee Approval

Ethical approval for this study was obtained from the Non-Interventional Clinical Research Ethics Committee of İzmir Bakırçay University (decision no: 1732, dated: 08.08.2024). Written informed consent was obtained from all participants.

Statistical Analysis

Differences among the groups were analyzed using the Kruskal–Wallis test. For multiple comparisons, Dunn's post-hoc pairwise comparisons were performed with Benjamini–Hochberg correction to control the probability of type I error. Associations among clinical, metabolic, and hormonal variables were evaluated using Spearman's rank correlation analysis, both across the entire sample and within each group, to examine group-specific patterns of relationships. All statistical analyses were conducted in the R software environment using RStudio and relevant R packages. A p-value of <0.05 was considered statistically significant.

RESULTS

Baseline and Anthropometric Characteristics

The analyses revealed statistically significant differences among the groups in anthropometric parameters. The obese PCOS group had significantly higher BMI, body weight, total fat mass, and body fat percentage than both the non-obese PCOS group and the control group ($p < 0.001$). Lean body mass and total body water were also higher in the obese PCOS group than in the other groups ($p < 0.001$), whereas the percentage of body water was significantly lower ($p < 0.001$). In contrast, no statistically significant differences were found between the control group and the non-obese PCOS group with respect to BMI, body weight, fat mass, body fat percentage, lean body mass, total body water, or body water percentage ($p > 0.05$) (Table 1).

Metabolic and Biochemical Parameters

When metabolic parameters were compared, fasting insulin and HOMA-IR levels were significantly higher in the obese PCOS group than in the other groups ($p < 0.001$). Additionally, HbA1c levels were found to be higher in the obese PCOS group compared with both the control group ($p < 0.01$) and the non-obese PCOS group ($p < 0.01$). Fasting glucose levels in the obese PCOS group were higher than those in both the control group ($p < 0.001$) and the non-obese PCOS group ($p = 0.02$). In addition, fasting glucose levels in the non-obese PCOS group were significantly higher than those in the control group ($p = 0.02$).

No statistically significant difference was found between the control and non-obese PCOS groups in terms of HDL cholesterol and TG levels. However, HDL cholesterol levels were significantly lower in the obese PCOS group than those in the other groups ($p < 0.001$), whereas TG levels were significantly higher ($p < 0.01$). No statistically significant differences were observed among the groups with respect to LDL cholesterol or total cholesterol levels.

When liver function parameters were examined, ALT levels were found to be significantly higher in the obese PCOS group than in the other groups. This difference was significant compared with those in both the control group ($p < 0.001$) and the non-obese PCOS group ($p = 0.02$). In contrast, no statistically significant differences in AST levels were observed among the groups. CRP levels were

Table 1. Demographic and anthropometric characteristics of the CONT, NON, and OBESE groups.

Variable	CONT (n = 40)	NON (n = 40)	OBESE (n = 40)
Age	23 (22-27)	23 (21–24.5)	27 (23–30)
Height (cm)	164.9 ± 5.0	162.1 ± 4.9	160.9 ± 6.2
Weight (kg)	59.4 ± 4.7	57.1 ± 7.4	84.8 ± 8.2
BMI (kg/m^2)	21.8 ± 1.4	21.7 ± 2.5	32.6 (30.6-34.5)
Body fat percent (%)	28.4 (25.45-30.78)	26.7 ± 5.4	41.4 ± 3.4
Body fat (kg)	16.7 ± 3.5	15.5 ± 4.7	35.2 ± 5.7
Lean mass (kg)	42.7 ± 2.3	41.7 ± 3.7	49.6 ± 3.7
Body water (kg)	31.3 ± 1.7	30.5 ± 2.5	36.5 (33.9–38.9)
Body water percent (%)	52.5 (50.7–54.51)	53.7 ± 3.9	42.8 (40.9–44.2)

Data are presented as mean ± standard deviation or median (minimum–maximum).
CONT: Control group, NON: Non-obese group, OBESE: Obese group, BMI: Body mass index.

significantly higher in the obese PCOS group than in both the control group ($p < 0.001$) and the non-obese PCOS group ($p < 0.001$). No significant difference in CRP levels was found between the control and non-obese PCOS groups. AMH levels were significantly higher in the non-obese PCOS group than in the control group ($p < 0.05$) (Figure 1, Tables 2 and 3).

No statistically significant differences were found among the groups in FSH, LH, LH/FSH ratio, or progesterone levels ($p > 0.05$).

The global significance profile of variables was evaluated across the control, non-obese, and obese groups. The dots represent the −log10-transformed p-values for each variable after Benjamini–Hochberg false discovery rate (FDR) correction. The vertical dashed line indicates the threshold for statistical significance (FDR-adjusted p = 0.05). Variables related to metabolic regulation, such as body weight, body fat parameters, BMI, indicators of IR, and lipid parameters, showed the most pronounced differences among the groups, whereas no statistically significant differences were observed for some hormonal and biochemical markers (Figure 2).

The distribution of anthropometric and metabolic parameters in the control, non-obese, and obese groups is presented using box plots. The graphs illustrate the distributional characteristics of anthropometric, metabolic, and hormonal variables across the study groups. Overall group differences were analyzed using the Kruskal–Wallis test, and Dunn's post hoc pairwise comparisons were performed with Benjamini–Hochberg correction for multiple testing. Compared with the control and non-obese groups, the obese group had lower HDL cholesterol levels and body water percentage, but higher BMI, body fat mass, body fat percentage, insulin, HOMA-IR, TG, CRP, ALT, and body weight. Statistically significant pairwise comparisons are indicated by asterisks (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$) (Figure 3).

The Spearman correlation structure among anthropometric, metabolic, and hormonal parameters was evaluated in the entire sample. In the Spearman correlation heatmap, which visualizes anthropometric, metabolic, biochemical, and hormonal variables using hierarchical clustering, color intensity represents the direction and strength of correlations (red: positive; blue: negative).

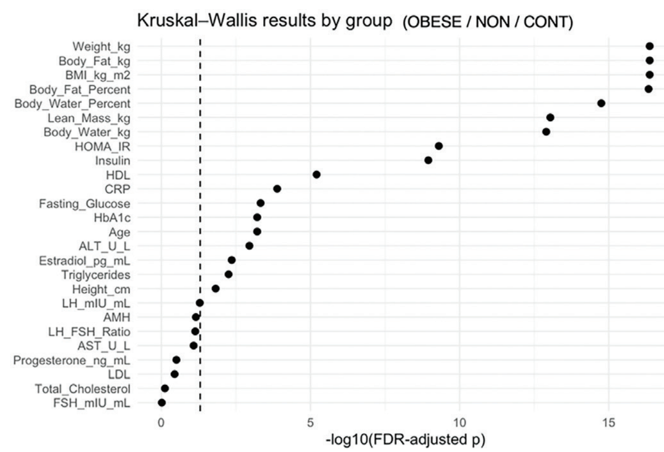

Figure 1. Global significance profile of variables across the CONT, NON, and OBESE groups.

CONT: Control group, NON: Non-obese group, OBESE: Obese group.

Table 2. Comparison of metabolic, biochemical, and hormonal parameters among the CONT, NON, and OBESE groups.

Variable	CONT (n = 40)	NON (n = 40)	OBESE (n = 40)
Fasting glucose (mg/dL)	78.5 ± 7.6	82.3 ± 6.3	86.3 ± 8.4
Insulin (µU/mL)	8.1 ± 3.6	8.6 (5.6–11.1)	15.4 (10.7–22)
HOMA-IR	1.6 ± 0.7	1.8 (1.1–2.3)	3.1 (2.3–4.9)
HbA1c (%)	5.1 ± 0.3	5.2 ± 0.3	5.4 (5.2–5.6)
Triglycerides (mg/dL)	68 (56.1–87.7)	64.5 (58.1–92.2)	87.1 (76.2–104)
Total cholesterol (mg/dL)	167.2 ± 24.2	171.9 ± 20.8	172.1 ± 33.9
LDL (mg/dL)	93.2 ± 18.9	92.7 ± 17.3	99.2 ± 24.6
HDL (mg/dL)	60.1 ± 9.0	63.9 ± 13.4	49.7 ± 11.8
AMH (ng/mL)	2.5 (1.8–4.5)	4.0 (2.9–6.3)	3.9 (1.7–5.4)
CRP (mg/dL)	2.5 (2.5–2.5)	2.5 (2.5–2.5)	2.5 (2.5–5.2)
ALT (U/L)	14 (11.8–20)	16.1 (13–22.7)	21.2 (14.7–29.2)
AST (U/L)	16.5 (15.1–20.6)	18.4 (17–22.2)	19.1 (16.8–21.1)
LH (mIU/mL)	9.1 (5.4–11.6)	14.4 (7.8–20.3)	8.7 (4.8–16.6)
FSH (mIU/mL)	4.8 (4.0–6.7)	5.4 ± 2.1	5.3 ± 2.0
LH/FSH ratio	1.8 (1.2–2.2)	2.7 (1.7–3.6)	2.1 ± 1.1
Progesterone (ng/mL)	0.6 (0.2–3.8)	0.3 (0.1–1.5)	0.3 (0.1–1.7)
Estradiol (pg/mL)	101.9 (59.2–181.5)	73.4 (49.5–123)	51.1 (32.3–79.8)

Data are presented as mean ± standard deviation or median (minimum–maximum).

CONT: Control group, NON: Non-obese group, OBESE: Obese group, BMI: Body mass index, HOMA-IR: Homeostatic model assessment of insulin resistance, HbA1c: Glycated hemoglobin, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, AMH: Anti-Müllerian hormone, CRP: C-reactive protein, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, LH: Luteinizing hormone, FSH: Follicle-stimulating hormone.

Hierarchical clustering analysis revealed that BMI, body fat mass, insulin, HOMA-IR, TG, and CRP levels were strongly positively correlated with one another, whereas these variables were negatively correlated with HDL cholesterol and body water percentage. In contrast, hormonal parameters exhibited weaker interrelationships and formed a relatively independent cluster separate from the metabolic indicators.

DISCUSSION

PCOS is one of the most common endocrine disorders in women of reproductive age and a complex metabolic and endocrine condition characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. However, PCOS is not limited to the reproductive system; it is also closely associated with various metabolic disturbances, including IR, obesity, dyslipidemia, and chronic low-grade inflammation. The findings of the present study indicate that the metabolic impairments observed in women with PCOS are largely associated with increased fat mass and obesity. The current literature also reports that the prevalence of obesity, particularly visceral/abdominal adiposity, is higher in women with PCOS than in healthy individuals, and that this higher prevalence markedly worsens the cardiometabolic risk profile. Obesity is considered a major determinant in the pathophysiology of PCOS, and increased visceral adiposity has been suggested to impair insulin signaling, thereby accelerating the development of metabolic complications such as IR and dyslipidemia (13). In addition, adipokines and proinflammatory cytokines secreted by increased adipose tissue are reported to play an important role in the disruption of metabolic and hormonal balance, promoting chronic low-grade inflammation through elevations in inflammatory markers such as CRP (14). These inflammatory processes are thought to adversely affect insulin signaling pathways and to play a critical role in the development of IR and the progression of metabolic dysfunction. Therefore, the combined evaluation of body composition and hormonal and metabolic parameters in individuals with PCOS is important to better understand the metabolic consequences of the disorder and the underlying pathophysiological mechanisms.

Table 3. Significant pairwise comparisons of biochemical and hormonal parameters between groups.

Variables	Groups	p-value
Fasting glucose (mg/dL)	CONT-NON	p < 0.05
Fasting glucose (mg/dL)	CONT-OBESE	p < 0.001
Fasting glucose (mg/dL)	NON-OBESE	p < 0.05
Insulin (µU/mL)	CONT-OBESE	p < 0.001
Insulin (µU/mL)	NON-OBESE	p < 0.001
HOMA-IR	CONT-OBESE	p < 0.001
HOMA-IR	NON-OBESE	p < 0.001
HbA1c (%)	CONT-OBESE	p < 0.001
HbA1c (%)	NON-OBESE	p < 0.01
Triglycerides (mg/dL)	CONT-OBESE	p < 0.01
Triglycerides (mg/dL)	NON-OBESE	p < 0.01
HDL (mg/dL)	CONT-OBESE	p < 0.001
HDL (mg/dL)	NON-OBESE	p < 0.001
AMH (ng/mL)	CONT-NON	p < 0.05
CRP (mg/dL)	CONT-OBESE	p < 0.001
CRP (mg/dL)	NON-OBESE	p < 0.001
ALT (U/L)	CONT-OBESE	p < 0.001
ALT (U/L)	NON-OBESE	p < 0.05
Estradiol (pg/mL)	CONT-OBESE	p < 0.01
Estradiol (pg/mL)	NON-OBESE	p < 0.05

HOMA-IR: Homeostatic model assessment of insulin resistance, HbA1c: Glycated hemoglobin, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, AMH: Anti-Müllerian hormone, CRP: C-reactive protein, ALT: Alanine aminotransferase, CONT: Control group, NON: Non-obese group, OBESE: Obese group.

In our study, which used BIA, body weight, BMI, total fat mass, and body fat percentage were significantly higher in the obese PCOS group than in the other groups. In addition, lean body mass and total body water were higher in the obese PCOS group, whereas body water percentage was lower in that group. The markedly elevated fat mass and body fat percentage observed in the obese PCOS group are consistent with previous studies in the literature (15-17). In line with our classification criteria, and given that the non-obese PCOS and control groups had similar BMI and body weight, no significant differences were found between the groups in fat mass or body fat percentage. Similarly, other studies comparing healthy women and non-obese women with PCOS have reported no significant differences in fat mass or body fat percentage (18). These findings suggest that obesity, independent of PCOS, has a substantial effect on body composition. The results presented in Figure 1 further demonstrate that the differences among the groups were largely driven by variables related to body composition, IR, and lipid metabolism.

IR is one of the key pathophysiological mechanisms associated with both obesity and PCOS. In our study, levels of fasting insulin, HOMA-IR, fasting glucose, and HbA1c were significantly higher in the obese PCOS group than in both the non-obese PCOS group and the control group. In addition, fasting glucose levels were significantly higher in all PCOS groups than in healthy controls. The literature indicates that, in the presence of PCOS, obesity contributes to increases in fasting insulin, HOMA-IR, fasting glucose, and HbA1c levels (15-21). Consistent with previous reports, fasting insulin and HOMA-IR values were higher in the non-obese PCOS group than in the control group, although this difference did not reach statistical significance ($p > 0.05$) (15,17,18). These findings suggest that, in addition to PCOS itself, obesity plays a determining role in the severity of IR.

In obese individuals, prolonged sedentary behavior and low levels of physical activity adversely affect lipid metabolism. Increased sedentary time has been reported to be particularly associated with decreased HDL cholesterol levels and increased TG levels (22,23). In the systematic review and meta-analysis conducted by Isayeva et al. (22), sedentary lifestyle duration was reported to be associated with abdominal obesity, dyslipidemia, and low HDL cholesterol levels. The authors emphasized that low physical activity negatively affects lipid metabolism and increases metabolic risk (22). Similarly, in the review by Silveira et al. (23), a sedentary lifestyle was reported to be associated with obesity and IR, as well as decreased HDL levels and increased TG levels.

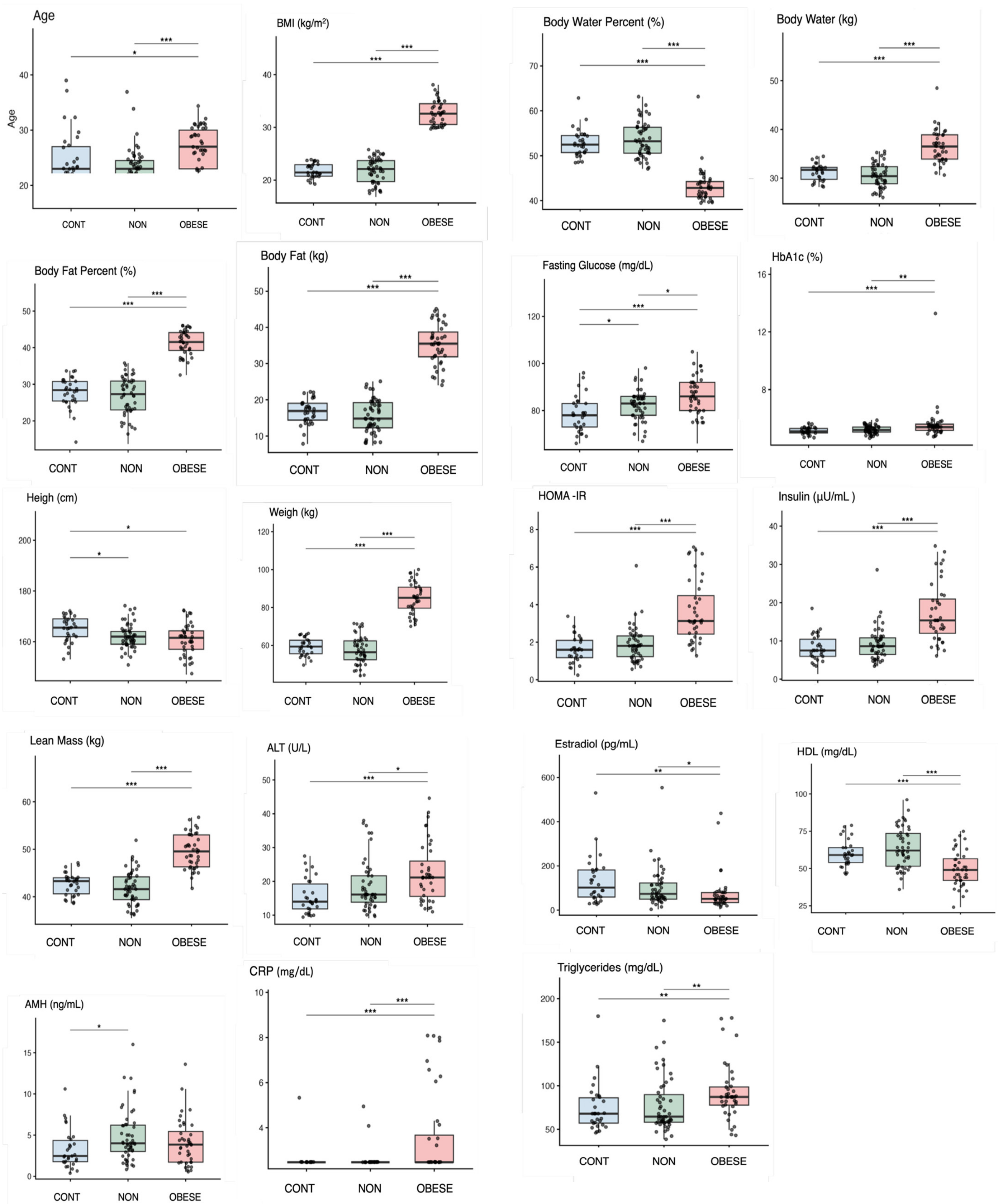

Figure 2. Distribution of anthropometric and metabolic parameters in the CONT, NON, and OBESE groups.

CONT: Control group, NON: Non-obese group, OBESE: Obese group.

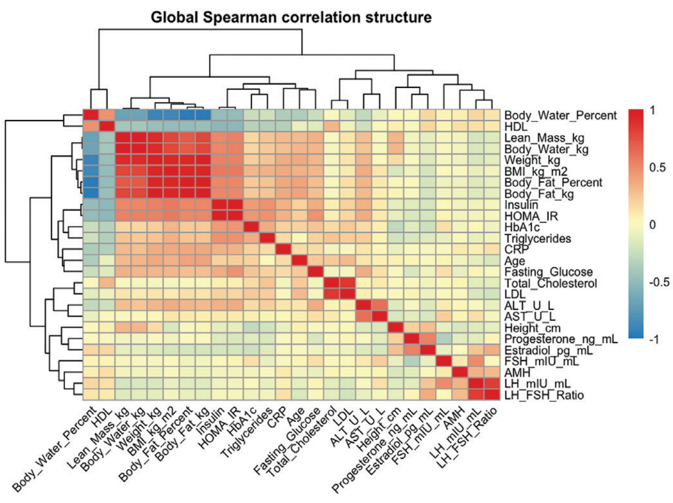

Figure 3. Spearman correlation structure among anthropometric, metabolic, and hormonal parameters in the entire sample.

In our study, HDL cholesterol levels were significantly lower and TG levels were significantly higher in the obese PCOS group than in the other groups. No significant differences were observed among the groups with respect to LDL or total cholesterol levels. Our findings are supported by previous studies reporting lower HDL levels and higher TG levels in individuals with obese PCOS (19,24).

The markedly elevated CRP levels observed in the obese PCOS group may reflect obesity-associated chronic inflammation, given that no significant difference in CRP was found between healthy women and the non-obese PCOS group. Previous studies have likewise reported elevated CRP levels in patients with obese PCOS (21,25). When ALT and AST levels were examined, both parameters were higher in the obese PCOS group; however, the increase was statistically significant only for ALT. The absence of a significant difference in AST levels does not suggest marked hepatocellular injury. No statistically significant differences were found among the groups in terms of FSH, LH, LH/FSH ratio, or progesterone levels.

Correlation analyses showed that BMI and body weight were positively and significantly associated with fat mass, body fat percentage, fasting insulin, HOMA-IR, HbA1c, TG levels, ALT, and CRP levels, and negatively associated with HDL cholesterol and body water percentage.

CONCLUSION

This study demonstrates that metabolic disturbances in PCOS are strongly associated with obesity and increased fat mass. The obese PCOS group exhibited a clearly more unfavorable profile in terms of body composition parameters, markers of IR, and lipid profile, and elevated inflammatory markers. In contrast, no significant differences were observed between the non-obese PCOS group and healthy controls in many metabolic parameters.

The findings of this study indicate that obesity is not merely a condition accompanying PCOS, but also one of the key determinants of the metabolic severity of the disorder. Therefore, we believe that strategies aimed at improving body composition should play a critical role in the management of PCOS, particularly in preventing metabolic complications such as IR, dyslipidemia, and inflammation.

Ethics

Ethics Committee Approval: Ethical approval for this study was obtained from the Non-Interventional Clinical Research Ethics Committee of İzmir Bakırçay University (decision no: 1732, dated: 08.08.2024).

Informed Consent: Written informed consent was obtained from all participants.

Footnotes

Authorship Contributions

Concept: S.E.K., M.K., C.K., Design: S.E.K., C.K., Data Collection or Processing: S.E.K., C.K., N.C., M.D., L.N.N., Analysis or Interpretation: S.E.K., L.N.N., C.K., Literature Search: S.E.K., C.K., Writing: S.E.K., L.N.N., C.K.

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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