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The role of inflammatory and metabolic biomarkers in polycystic ovary syndrome and their association with cardiovascular risk

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Polycystic ovary syndrome is an endocrine disorder that leads to hormonal and metabolic changes associated with the development of chronic diseases. Our objective was to study the role inflammatory and metabolic markers play in women suffering from polycystic ovary syndrome (PCOS) and the link between these markers and cardiovascular risk. From January to July 2025, cross sectional study was conducted in Al-Habboubi Teaching Hospital to asses inflammatory and metabolic biomarkers in 150 women with PCOS and 50 healthy controls. The diagnosis was made based on the Rotterdam criteria (2003). Exclusion criteria included pregnant women, people with chronic diseases, and people taking medicines that affect the biomarkers. We measured CRP, TNF- α, IL-6, IL-10, lipid profile, insulin, adiponectin, and HOMA-IR in blood samples. Anthropometric measurements included BMI, waist circumference, and blood pressure. Ethical approval was obtained and written informed consents were given. The results of the analysis revealed no age difference between the groups, but the women with PCOS had significantly higher BMI, WC, and BP. High CRP and TNF-α with low IL-10 levels were associated with high inflammatory status. The metabolic abnormalities included elevated levels of fasting glucose, insulin, HOMA-IR, and low level of adiponectin. Cardiovascular risk was independently related to obesity, chronic inflammation, insulin resistance, low adiponectin, and dyslipidaemia in the women with PCOS, which is in line with the results of the correlation and regression analyses. Polycystic ovary syndrome was found to be closely linked to obesity, insulin resistance, dyslipidaemia, and chronic inflammation, all of which contribute to an increased risk of cardiovascular disease. These alterations are due to hormonal and metabolic imbalance leading to increased inflammatory responses, decreased protective adipokines, and endothelial dysfunction.

Keywords: polycystic ovary syndrome; inflammatory biomarkers; insulin resistance; dyslipidemia; cardiovascular risk.

Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrine/metabolic diseases in women of reproductive age, with the prevalence rate worldwide estimated to be 8–13%, depending on diagnostic criteria used. It is characterized by a combination of clinical and biochemical features including hyperandrogenism, oligo or anovulation, and polycystic ovarian morphology (Khan et al., 2019). Besides its reproductive phenotypes, PCOS is becoming more and more a systemic disorder that has massive metabolic and cardiovascular implications. Women having PCOS are likely to develop obesity, insulin resistance, type 2 diabetes mellitus (T2DM), dyslipidemia, and hypertension, all of which predispose them to cardiovascular disease (CVD) during their lifetime (Anagnostis et al., 2018; Joham et al., 2022).

Interest in the role of inflammation as a pathophysiological pathway between PCOS and its cardiometabolic sequelae has been increasing over the past few years. Increasing amounts of evidence indicate that PCOS is a form of low-grade inflammation on a chronic basis, which is manifested through high circulation of inflammatory biomarkers, including C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-alpha), and interleukin-6 (IL-6). These cytokines have been known to disrupt insulin signaling, damage the functionality of endothelium, and mediate atherogenesis (Rostamtabar et al., 2021; Rudnicka et al., 2021). On the other hand, the levels of anti-inflammatory mediators such as interleukin-10 (IL-10) are usually decreased in women with PCOS and contribute to the aggravation of the pro-inflammatory environment. This disproportion between pro-and anti-inflammatory pathways is believed to hasten metabolic failure and vascular injury in PCOS patients (Liu et al., 2021).

Along with the inflammatory factor, metabolic changes are a major focus of the natural history of PCOS. The insulin resistance in PCOS that has up to 70% prevalence has a role in not only the hyperinsulinemia and metabolic derangements but also in ovarian androgen production as a contributor to the vicious cycle of the endocrine dysfunction. The resistance to insulin is often determined by fasting insulin concentrations and the homeostatic model of insulin resistance (HOMA-IR), which are higher in PCOS groups (Ding et al., 2021; Unluhizarci et al., 2021). In addition to that, adipokines such as adiponectin are also dysregulated in PCOS. Adiponectin that is normally insulin-sensitizing, anti-inflammatory, and anti-atherogenic is always reported to be low in PCOS and this further supports the metabolic and vascular risk profile of this disease (Xu & Qiao, 2022). Another manifestation of the metabolic load in PCOS is dyslipidemia. The changes are usually high total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), and reduced high-density lipoprotein cholesterol (HDL-C). Insulin resistance and chronic inflammation mediate these lipid imbalances, and they play a major role in the early development of atherosclerotic changes. Interacting with obesity, especially central adiposity, these abnormalities create a solid mechanistic foundation of the increased cardiovascular risk that PCOS women have compared with their age-matched controls (Liu et al., 2019; Amadi et al., 2023).

Although several studies have examined the inflammatory markers, metabolic indices, and the cardiovascular risk factors separately in PCOS, few have examined the relationship of these factors. Majority of the current data show that the interaction among obesity, insulin resistance, and inflammation plays a key role in promoting the cardiovascular morbidity (Abraham Gnanadass et al., 2021). There are however discrepancies on whether or not these biomarkers alone predict cardiovascular outcomes. As an example, although CRP and IL-6 are commonly increased in PCOS, other studies indicate that the differences become insignificant after correction for BMI, suggesting that obesity is possibly a confounding factor. In the same vein, the precise predictive values of adiponectin and dyslipidemia in cardiovascular risk assessment of PCOS are also a subject of debate (Rudnicka et al., 2020). The links between inflammatory and metabolic biomarkers in PCOS is thus vital in improving the characterization of the heterogeneity of the disease, high-risk groups, and prevention of the disease. Clinically, PCOS women carrying a substantial burden of inflammatory and metabolic derangements could be identified early

to allow timely intervention to reduce the cardiovascular risk either by lifestyle changes and pharmacological intervention of insulin resistance or anti-inflammatory interventions (Aboeldalyl et al., 2021; Chen & Pang, 2021).

The current research paper aimed to assess the importance of inflammatory and metabolic biomarkers in PCOS women and investigate their correlations with cardiovascular risk factors. In particular, we aimed to compare inflammatory (CRP, TNF-α, IL-6, IL-10) and metabolic (fasting glucose, insulin, HOMA-IR, adiponectin, lipid profiles) biomarkers of PCOS patients and healthy participants so as to evaluate relationships between these biomarkers and cardiovascular metabolic variables and detect independent predictors of cardiovascular risk. Through a combination of inflammatory and metabolic aspects, this paper offers a more detailed view of the mechanisms through which PCOS is connected to cardiovascular disease.

Materials and methods

The Human Ethics Committee of Al-Habbobi Teaching Hospital approved the research; everyone involved was informed about the research and was requested to sign a consent. An assurance was also given to the patients that their information would remain confidential.

The proposed study was designed as a cross-sectional observational study, carried out at the Al-Habboubi Teaching Hospital / Women Consultation and Gynecology Division, Nasiriyah, Iraq, between January 1, 2025, and July 10, 2025, to explore the role of inflammatory and metabolic biomarkers in patients with polycystic ovary syndrome (PCOS) and their relationship with cardiovascular risk factors. We examined 150 women with PCOS and 50 healthy women as age-matched controls. The assessment of PCOS was made according to the Rotterdam criteria (2003), which included at least two of the following: oligo- or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound. The inclusion criteria were informed consent from women aged 18–35 years with confirmed PCOS, whereas the exclusion criteria included pregnancy or lactation, diabetes mellitus, cardiovascular disease, hepatolithyrenephritis, and current use of drugs that alter hormonal, metabolic, or inflammatory condition. The venous blood samples were taken following an 8–10 hours fasting period, with about 5 mL in plain serum tubes and EDTA plasma tubes. Serum samples were left to clot at room temperature for 30 minutes, then centrifuged at 3,000 rpm for 10 minutes. The plasma samples were centrifuged and stored under –80 °C before analysis. Measurement of inflammatory biomarkers was carried out using enzyme-linked immunosorbent assay (ELISA) kits, including C-reactive protein (CRP), TNF-α, IL-6, and IL-10, whereas metabolic biomarkers were quantified with routine enzyme-linked colorimetric assays (fasting blood glucose and lipid profile [total cholesterol, triglycerides, HDL-C, LDL-C]), and fasting insulin and adiponectin were measured using EL. The formula to calculate HOMA-IR was: fasting insulin (mU/dL) fasting glucose (mg/dL) / 405. Measurements of anthropometric parameters (such as height, weight, waist circumference, and blood pressure) were taken in accordance with standard procedures, and body mass index (BMI) was calculated as weight (kg) / height (m^2). The research was carried out in accordance with the ethical principles and informed written consent was signed by all the participants before joining the research and confidentiality was upheld in the process of conducting the research.

Analysis of quantitative data was performed using SPSS version 26. The data is expressed in percentage and frequency. In the case of normally distributed variables, t-tests (two tails) were applied for both independent and dependent samples. In the non-normally distributed variables, Chi-square test, Mann-Whitney U-test, and Wilcoxon test were employed. The P-value of less than 0.05 was taken to be statistically significant.

Results

The study results showed no significant difference in the average age between the PCOS patients and the control group (P = 0.64), while highly statistically significant differences were observed in other indicators. The PCOS patients exhibited a higher average body mass index (BMI) (29.6 ± 3.5) compared with the control group (23.4 ± 2.9), with a significant difference (P < 0.001). The average waist circumference was also significantly greater in the PCOS patients (89.5 ± 8.6 cm) compared with the healthy controls (76.8 ± 7.4 cm, P < 0.001). Also, the mean systolic blood pressure of the PCOS patients was higher (126.3 ± 11.2 mmHg) than that of the control group (117.4 ± 9.8 mmHg) with strong statistical significance (P < 0.001), while the diastolic blood pressure showed a similar result, reaching 81.7 ± 7.5 mmHg, compared with 75.9 ± 6.3 mmHg (P < 0.001). These results indicate a close association between PCOS and overweight, central obesity, and hypertension.

Table 1
Sociodemographic and clinical characteristics of the study participants

Variables	PCOS patients (n = 150, mean ± SD)	Controls (n = 50, mean ± SD)	P-value	95% CI
Age, years	26.8 ± 4.9	27.2 ± 5.1	0.64	–1.2–1.9
BMI, kg/m²	29.6 ± 3.5	23.4 ± 2.9	<0.001	5.3–7.2
Waist circumference, cm	89.5 ± 8.6	76.8 ± 7.4	<0.001	10.1–15.2
Systolic BP, mmHg	126.3 ± 11.2	117.4 ± 9.8	<0.001	5.3–12.4
Diastolic BP, mmHg	81.7 ± 7.5	75.9 ± 6.3	<0.001	3.2–8.4

The study results showed clear significant differences in inflammatory markers between the PCOS patients and the control group. C-reactive protein (CRP) levels showed a significant increase in the PCOS patients (5.8 ± 2.1 mg/L) compared with the healthy controls (2.4 ± 1.0 mg/L), with a high statistical significance (P < 0.001). The levels of tumor necrosis factor-alpha (TNF-α) were also significantly elevated in the patients (18.7 ± 4.5 pg/mL) compared with the control group (12.3 ± 3.2 pg/mL, P < 0.001). Similarly, interleukin-6 (IL-6) levels were higher in the PCOS patients (9.4 ± 3.1 pg/mL) than in the controls (4.2 ± 1.9 pg/mL), with a strongly significant difference (P < 0.001). By contrast, interleukin-10 (IL-10) levels were significantly lower in the PCOS patients (3.1 ± 1.2 pg/mL) compared with the healthy controls (5.6 ± 1.5 pg/mL, P < 0.001). These findings suggest that PCOS is associated with an increase in pro-inflammatory mediators and a decrease in anti-inflammatory mediators, which may contribute to an increased risk of cardiovascular events.

Table 2
Inflammatory biomarkers in the study participants

Biomarker	PCOS patients (mean ± SD)	Controls (mean ± SD)	P-value	95% CI
CRP, mg/L	5.8 ± 2.1	2.4 ± 1.0	<0.001	2.8–3.9
TNF-α, pg/mL	18.7 ± 4.5	12.3 ± 3.2	<0.001	5.0–7.8
IL-6, pg/mL	9.4 ± 3.1	4.2 ± 1.9	<0.001	4.2–6.1
IL-10, pg/mL	3.1 ± 1.2	5.6 ± 1.5	<0.001	–3.0––1.8

Metabolic indices showed significant differences between PCOS patients and the control group. The mean fasting blood glucose level was significantly higher in the PCOS patients (101.5 ± 12.8 mg/dL) compared with the healthy controls (89.6 ± 10.5 mg/dL), with strong statistical significance (P < 0.001). Fasting insulin levels were also significantly higher in the PCOS patients (17.3 ± 5.9 μIU/mL) than in the controls (9.6 ± 3.7 μIU/mL), with a highly significant difference (P < 0.001). Similarly, the insulin resistance index (HOMA-IR) was higher in the PCOS patients (4.3 ± 1.5) compared with the controls (2.1 ± 0.9, P < 0.001). By contrast, there was a significant decrease in the mean adiponectin levels (6.8 ± 2.2 μg/mL) in the PCOS patients compared with the healthy controls (11.4 ± 3.1 μg/mL), and the difference was statistically highly significant (P < 0.001). The findings indicate that there was metabolic imbalance in these patients, reflected by high glucose and insulin levels and insulin resistance accompanied by low level of adiponectin, which may be an important risk factor for cardiovascular and metabolic diseases in PCOS patients.

Statistically significant difference between the lipid analysis results of PCOS patients and control group were found. The mean total cholesterol level of the PCOS patients (205.6 ± 32.4 mg/dL) was significantly higher than that of the healthy controls (178.9 ± 28.7 mg/dL, P < 0.001). The average measured level of triglycerides

weas also significantly higher in the PCOS patients (165.3 ± 40.1 mg/dL) than in the control group (121.7 ± 31.5 mg/dL, P < 0.001). The PCOS patients, however, had significantly lower levels of HDL-C (42.6 ± 8.9 mg/dL) than their healthy counterparts (53.1 ± 9.2 mg/dL, P < 0.001). Also, mean low-density lipoprotein cholesterol (LDL-C) was higher in the PCOS patients (124.8 ± 29.7 mg/dL) than in the controls (104.6 ± 25.3 mg/dL, P < 0.001). The findings indicate that PCOS is linked to the lipid parameter disturbances with increased total cholesterol, triglycerides, and LDL-C and reduced HDL-C, contributing to the risk of metabolic syndrome and cardiovascular diseases.

Table 3
Metabolic biomarkers in the study participants

Biomarker	PCOS patients (mean ± SD)	Controls (mean ± SD)	P-value	95% CI
Fasting glucose, mg/dL	101.5 ± 12.8	89.6 ± 10.5	<0.001	8.5–15.2
Fasting insulin, μIU/mL	17.3 ± 5.9	9.6 ± 3.7	<0.001	6.1–9.3
HOMA-IR	4.3 ± 1.5	2.1 ± 0.9	<0.001	1.8–2.7
Adiponectin, μg/mL	6.8 ± 2.2	11.4 ± 3.1	<0.001	–5.7– –3.3

Table 4
Lipid profile of the study participants

Lipid parameter	PCOS patients (mean ± SD)	Controls (mean ± SD)	P-value	95% CI
Total cholesterol, mg/dL	205.6 ± 32.4	178.9 ± 28.7	<0.001	17.3–35.6
Triglycerides, mg/dL	165.3 ± 40.1	121.7 ± 31.5	<0.001	30.7–56.4
HDL-C, mg/dL	42.6 ± 8.9	53.1 ± 9.2	<0.001	–12.7– –8.2
LDL-C, mg/dL	124.8 ± 29.7	104.6 ± 25.3	<0.001	12.5–28.9

The correlation analysis revealed statistically significant positive correlations between most inflammatory markers and the cardiometabolic factors of the PCOS patients. C-reactive protein (CRP) was positively correlated with the body mass index (r = 0.46, P < 0.001), waist circumference (r = 0.42, P < 0.001), systolic blood pressure (r = 0.39, P < 0.001), and HOMA-IR (r = 0.51, P < 0.001). Tumor necrosis factor-alpha (TNF-α) also had a statistically significant positive correlation with the same factors, ranging from 0.36 to 0.44, all with high statistical significance (P < 0.001). Likewise, interleukin-6 (IL-6) levels showed positive correlations with BMI, WC, SBP, and HOMA-IR (r = 0.35–0.47, P < 0.001). Interleukin-10 (IL-10), however, had significant negative associations with these variables, ranging from –0.27 to –0.34 (P = 0.006). These results indicate that an increase in the inflammatory mediators is linked to an increase in central obesity, blood pressure, and insulin resistance, whereas a decrease in the anti-inflammatory mediator IL-10 leads to these metabolic abnormalities.

Table 5
Correlation of inflammatory biomarkers with cardiometabolic risk factors in the PCOS patients

Biomarker	BMI (r, P)	Waist circumference (r, P)	Systolic BP (r, P)	HOMA-IR (r, P)
CRP	0.46, <0.001	0.42, <0.001	0.39, <0.001	0.51, <0.001
TNF-α	0.41, <0.001	0.38, <0.001	0.36, <0.001	0.44, <0.001
IL-6	0.43, <0.001	0.40, <0.001	0.35, <0.001	0.47, <0.001
IL-10	–0.31, 0.002	–0.29, 0.004	–0.27, 0.006	–0.34, <0.001

The results of the logistic regression analysis indicated that several metabolic and inflammatory factors were independent predictors of an increased risk of cardiovascular complications in the PCOS patients. Obesity (BMI ≥ 30 kg/m²) was found to increase the risk by 2.8-fold (OR = 2.8, 95% CI: 1.6–4.9, P < 0.001). Elevated levels of C-reactive protein (CRP ≥ 5 mg/L) were associated with a 3.4-fold increased risk (OR = 3.4, 95% CI: 1.9–6.0, P < 0.001). Furthermore, insulin resistance (HOMA-IR ≥ 3) showed a significant effect, increasing the risk by 2.6-fold (OR = 2.6, 95% CI: 1.5–4.3, P < 0.001). While low adiponectin levels were associated with a 2.1-fold increased risk (OR = 2.1, 95% CI: 1.2–3.8, P = 0.006), dyslipidemia was also shown to increase the risk of cardiovascular events by 1.9-fold (OR = 1.9, 95% CI: 1.1–3.3, P = 0.021). These findings confirm that obesity, chronic inflammation, insulin resistance, low adiponectin, and dyslipidemia are key factors contributing to the increased risk of cardiovascular disease in PCOS patients.

Table 6
Logistic regression analysis for predictors of cardiovascular risk in the PCOS patients

Variable	OR (Odds ratio)	95% CI	P-value
BMI ≥ 30 kg/m²	2.8	1.6–4.9	<0.001
CRP ≥ 5 mg/L	3.4	1.9–6.0	<0.001
HOMA-IR ≥ 3	2.6	1.5–4.3	<0.001
Low adiponectin	2.1	1.2–3.8	0.006
Dyslipidemia	1.9	1.1–3.3	0.021

Discussion

Table 1 results show that the PCOS women had significantly greater values of body mass index (BMI), waist circumference, systolic and diastolic blood pressure than the healthy controls, with no difference in age. It is in line with a number of studies that have shown that obesity and central adiposity are more common in PCOS women, which are both significant predisposing factors to metabolic and cardiovascular dysfunction (Zhuang et al., 2022). The high blood pressure level of the PCOS group is also consistent with the results of Mellembakken et al. (2021), who indicated that PCOS women develop premature endothelial dysfunction and hypertension at a rather early age. However, some of the contradicting studies propose that blood pressure differences may decrease when PCOS patients and controls are matched, and the BMI factors are held constant, therefore suggesting that obesity is a mediating factor in its own right (Gunning et al., 2019). However, our results also highlight central obesity and hypertension as early risk factors of the pathogenesis of PCOS.

Table 2 results indicate that C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6) were significantly higher, while interleukin 10 (IL-10) was lower in the PCOS women than in the controls. This type of biomarker supports the original idea of PCOS being a chronic low-grade inflammation. The results of our study are consistent with the systematic review and meta-analysis of Vasyukova et al. (2023), establishing the existence of significantly higher CRP and IL-6 in PCOS regardless of BMI. Likewise, Azarbayjani et al. (2024) had also found pro-inflammatory dominance and low anti-inflammatory cytokines in PCOS. Conversely, other studies have failed to reveal substantial differences in TNF-α, especially among the lean PCOS women (Abraham Gnanadass et al., 2021). Such discrepancy could be attributed to variation in the phenotype of PCOS, the extent of insulin resistance, and physiological features. The IL-10 decrease in our cohort also shows an imbalance in pro- and anti-inflammatory signal transmission, which may worsen metabolic imbalance (Hafizi Moori et al., 2023; Mustafa et al., 2024).

As shown in Table 3, fasting blood glucose, fasting insulin, and HOMA-IR of the PCOS women were significantly higher, and adiponectin was significantly lower. These findings support the existing role of insulin resistance in the pathophysiology of PCOS. The results are in agreement with Shirazi et al. (2021), who demonstrated consistent increases in insulin resistance markers in case-control studies of PCOS (Al-Hussaniy et al., 2025). Lowered adiponectin levels are well-established in women with PCOS (Mishra et al., 2022), which is a manifestation of insensitivity to insulin and anti-inflammatory control. Some of the studies within lean PCOS subgroups do not indicate such notable glucose or insulin abnormalities but our results indicate that the existence of obesity in this group exacerbated the metabolic malfunction. When combined, these changes support the idea that insulin resistance and low adiponectin are at the core in associating PCOS with diabetes and cardiovascular disease risky outcomes in the future (Kumawat et al., 2021; Mahameed et al., 2026).

The PCOS women had a significantly higher levels of total cholesterol, triglycerides, and LDL-cholesterol, but lower levels of HDL-cholesterol than the controls (Table 4). This lipid profile shows the obvious dyslipidemia, which is a common symptom of PCOS. We found agreement with large-cohort studies indicating that insulin resistance and androgen excess are the causal factors behind hepatic

overproduction of VLDL, loss of HDL production, and lipid deposition (Ali et al., 2020). It was also reported in the study by Jabczyk et al. (2023) that there were similar lipid abnormalities, which they associated with chronic inflammation and endothelial dysfunction (Al Alouci et al., 2026). Some of the studies conflicting with this have reported milder or no significant differences in lipid levels in lean or young PCOS groups (Mellembakken et al., 2021), indicating that the extent of dyslipidemia varies depending on obesity, insulin resistance, and PCOS phenotype.

Table 5 demonstrates that CRP, IL-6, and TNF-a had positive correlations with BMI, waist circumference, systolic blood pressure, and HOMA-IR, whereas IL-10 had positive correlations with the mentioned variables. These correlations are good indications of a direct relationship between obesity and central adiposity and systemic inflammation and insulin resistance in PCOS. Such associations are consistent with the mechanistic research that indicates that inflammatory cytokines are released by enlarged adipocytes and invading macrophages, and hence, disrupt insulin signaling and vascular activity (Battineni et al., 2021). The anti-inflammatory protective effect of IL-10 on cardiometabolic variables indicates the loss of anti-inflammatory protection of PCOS women. Although other past research documented less robust correlations between cytokines and metabolic indices in lean PCOS patients (Ullah et al., 2024), we support the notion that obesity is a major enhancer of the inflammatory and metabolic interaction.

Table 6 indicates that cardiovascular risk in PCOS women was independent of BMI 30 kg/m^2, CRP 5mg/L, HOMA-IR 3, reduced adiponectin, and dyslipidemia. These results are consistent with previous evidence that obesity, insulin resistance, and inflammation are significant causative factors of cardiovascular complications in PCOS (Mu et al., 2019). Predictive value of CRP is especially significant because meta-analyses have proved that it is closely related to CVD in women (Kałużna et al., 2022). Low adiponectin also became an effective independent predictor, which showed involvement in the endothelial functionality and glucose-lipid metabolism (Sohaei et al., 2019). Even though there are studies that indicate that hyperandrogenism alone causes cardiovascular risk, our findings indicate that the synergistic role of metabolic and inflammatory factors play a pivotal role in determining the level of risk. This implies that prevention of obesity and inflammation may become a major aspect of prevention of PCOS at the early stages (Jena et al., 2018).

Conclusion

Obesity, insulin resistance, dyslipidemia, and a persistent state of pro-inflammatory activity are all closely associated with polycystic ovary syndrome (PCOS), which leads to an increased risk of cardiovascular diseases. These metabolic and hormonal disequilibriums exacerbate the inflammatory condition of the system, lower defensive adipokines such as adiponectin and omentin, and interfere with endothelial action. Hyperandrogenism, insulin resistance, and changes in adipokine secretion are combined factors that lead to oxidative stress, vascular dysfunction, and increased risk of metabolic and cardiovascular complications in PCOS women, which is the result of the interplay between endocrine and inflammatory pathways.

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