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RESEARCH ARTICLE

Role of chemerin and omentin in predicting ovulatory dysfunction in obese women with polycystic ovary syndrome (PCOS)

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Abstract

Polycystic ovary syndrome (PCOS) is an endocrine condition that is characterized by ovulatory dysfunction, hyperandrogenism, and metabolic imbalances. The aim of the study was to assess the predictive ability of chemerin and omentin-1 in ovulatory dysfunction of obese PCOS women. The study is a case-control study conducted at Al-Nasiriyah General Hospital (Jan 2024 – Jun 2025) with a sample size of 150 obese and 70 normal weight women with PCOS and 50 healthy controls. The diagnosis of PCOS was made according to Rotterdam criteria (2003). They included participants aged 18 years to 35 years old, who were not on hormonal therapy in the recent past; pregnancy, endocrine and chronic diseases were excluded. Blood samples were fasted and serum was separated and stored in the refrigerator at 20°C. Hormonal (Follicle-Stimulating Hormone, Luteinizing Hormone, LH/FSH, testosterone, Sex hormone binding globulin), metabolic (glucose, insulin, HOMA-IR, triglycerides, HDL-C), and adipokine (chemerin, omentin-1) levels were measured using ELISA, spectrophotometry, and Cobas e411 kits (Roche, Germany). There were no major differences in terms of age (p=0.63). BMI, waist circumference, menstrual irregularity and family history of PCOS were more prevalent among the obese PCOS women. There was an increase in the LH, LH/FSH ratio and the total testosterone and a decrease in SHBG. The most pronounced metabolic abnormalities were observed in PCOS obese, with lowest HDL-C, and progressively higher levels of metabolic abnormalities with increasing deficiency of ovulatory dysfunction and insulin resistance. There are significant differences in hormonal, metabolic, and ovarian changes in obese and non-obese PCOS women. High levels of chemerin and low levels of omentin-1 are closely related to insulin resistance and ovulatory dysfunction and suggest their use as predictive biomarkers and mediators of metabolic and reproductive pathophysiology in PCOS.

1. INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common heterogeneous endocrine disorder in women of reproductive age, affecting 6-15 % of them depending on the diagnostic criteria applied. It is defined by hyperandrogenism and ovulatory dysfunction with the presence or absence of polycystic ovarian morphology as determined upon ultrasound (1). In line with the Rotterdam consensus (2003) criteria, PCOS is diagnosed after exclusion of other etiologies like congenital adrenal hyperplasia, androgen-secreting tumors, and adrenocortical dysfunction. Apart from the characteristic

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reproductive aspects, PCOS is being recognized more and more as a multifaceted metabolic disorder that combines insulin resistance, visceral obesity dyslipidemia, and enhanced cardiovascular risk (2,3).

The coexistence of obesity and PCOS aggravates insulin resistance, hyperinsulinemia, and hyperandrogenism, leading to more severe reproductive dysfunction and a poorer response to ovulation induction therapies. Most metabolic and endocrine changes are interdependent, connected via adipokines, a cascade of biologically active proteins synthesized by adipose tissue that influence energy metabolism, inflammation and reproductive physiology (4,5). Among the adipokines associated with PCOS development, chemerin and low Omentin-1 are intriguing candidates, as they exhibit opposite effect on metabolic pathways and inflammation. Chemerin, originally identified as a chemoattractant protein produced by white adipose tissue (6), is a multifunctional adipokine predominantly expressed in AT, liver, and skin with effects that may range from regulating adipocyte differentiation to chemoattracting itself. Increased levels of chemerin are detected in obesity, metabolic syndrome, type 2 diabetes mellitus (T2DM), and polycystic ovary syndrome (PCOS). Mechanistically, chemerin promotes aryl hydrocarbon receptor (AhR)- mediated inflammatory responses in adipose tissue (7) and modulates insulin signaling; in addition, chemerin has been shown to impact ovarian steroidogenesis via its receptor, chemokine-like receptor 1 (CMKLR1), expressed in ovarian granulosa and theca cells (8). These events may lead to anovulation and defective follicular maturation in the ovarian microenvironment (9).

Conversely, omentin-1 is an anti-inflammatory adipokine secreted mainly by stromal vascular cells in visceral fat depots with a higher action rate in the omental (visceral) depot (10). Omentin-1 improves insulin-stimulated glucose uptake in adipocytes and is downregulated by obesity, insulin resistance, and inflammation. Decreased plasma omentin-1 levels in obesity, metabolic syndrome, and PCOS have been reported, particularly in women with severe insulin resistance. Experimental data suggest that omentin-1 might regulate the reproductive axis through mechanisms linked to improved insulin sensitivity, decreased androgen production, and attenuation of low-grade chronic inflammatory processes (11).

The relationship between chemerin and omentin-1 in the setting of PCOS is of considerable interest, given that both adipokines have opposing effects on metabolic and inflammatory pathways (12). Whereas, an increase in chemerin can be regarded as part of a broader pro-inflammatory and insulin-resistant milieu deleterious to ovulatory capacity, decreases in omentin-1 indicate that metabolic protection is failing, and contribute to the exacerbation of hormonal perturbations. Therefore, evaluating both adipokines together may offer a better understanding of the intersection between metabolism and reproduction in PCOS (13,14).

Ovulatory dysfunction in PCOS is a continuum from dysfunctional menstrual cycles to absence of ovulation, which may be due to many factors: hyperandrogenism, altered gonadotropin secretion, insulin resistance, and intrinsic ovarian abnormalities (15). The identification of such biomarkers would enable individualization, reducing time to treatment, selecting appropriate empiric treatments, and, as adjuncts, providing efficacy indicators for targeted therapies aimed at correcting ovulatory dysfunction. Although informative, classic predictors, such as hormonal assays and ultrasound data analysis, may not fully capture the metabolic facets of reproductive dysfunction (16).

Several studies have more recently reported that chemerin is positively correlated with insulin resistance indices, androgens, and markers of inflammation, while omentin-1 is negatively associated with these parameters, but this relation remains controversial (17). This was exemplified for chemerin, which showed a strong relationship with elevated LH levels and an increased LH/FSH ratio, both indicative of ovulatory dysfunction in PCOS whereas rank Spearman coefficients could not always be calculated for the other metabolites (18). On the other hand, lower levels of omentin-1 have been linked to better insulin sensitivity and decreased androgen concentrations. Yet, the literature is to some extent inconsistent, as studies report no significant difference between PCOS and control groups or observed in the obese versus lean phenotype of PCOS (19).

The potential diagnostic and prognostic importance of these adipokines in turn suggests the necessity for well-designed studies directly comparing chemerin and omentin-1 profiles in clearly defined PCOS subgroups, especially when stratified by obesity status, alongside measures of ovulatory function. This would provide an answer to whether the observed alterations in adipokines are mainly due to obesity, PCOS per se, or their interaction (20). Therefore, to fill this gap the present study aimed at analyzing serum chemerin and omentin-1 levels in obese and normal-weight women with PCOS compared to healthy controls, and investigating their associations with hormonal, metabolic, as well as clinical markers of anovulation. This work aims to identify anthropometric, endocrine, and metabolic factors associated with these adipokines to identify potential predictive variables for reproductive impairment in PCOS. This awareness could not only potentially enhance the accuracy of diagnosis, but could also give an idea of possible therapeutic interventions that are aimed at the manipulation of adipokines in PCOS in women.

2. MATERIALS AND METHODS

This case-control study was conducted at Al Nasiriyah General Hospital in Iraq between January 2024 and June 2025. It involved one hundred and fifty (150) women with polycystic ovary syndrome (PCOS), divided into two categories: obese PCOS (n=80) and normal weight PCOS (n=70), and 50 age-mated healthy controls. The diagnosis of PCOS was made according to the Rotterdam criteria (2003). The eligibility criteria were women between the ages of 18-35 years and with

a confirmed diagnosis of PCOS and no hormonal therapy in the past 3 months. The exclusion criteria were pregnancy, thyroid disorders, diabetes mellitus, Cushing's syndrome, chronic kidney or liver disease, and recent infections. Following the procedure of informed consent (written), sterile vacutainer tubes were used to take the fasting venous blood samples (5 mL) of each participant. Serum was separated by centrifugation at 3,000 rpm for 10 minutes and stored at −20°C until analysis. Hormonal parameters, including LH, FSH, LH/FSH ratio, total testosterone, and SHBG, were measured using commercially available ELISA kits (Biotechne, USA). Metabolic parameters, including fasting glucose, fasting insulin, triglycerides, and HDL-C, were measured using spectrophotometric methods (Biolabo, France) according to manufacturer protocols. HOMA-IR were measured using commercially available Cobas e411 kits (Roche, German). Serum chemerin and omentin-1 concentrations were measured using ELISA kits (Biotechne, USA) following the manufacturer's instructions.

2.1. Statistical Analysis

Quantitative data were analyzed using SPSS version 26. Results are presented as frequencies and percentages. For normally distributed variables, independent and dependent t-tests (two-tailed) were used. For non-normally distributed variables, the Mann-Whitney U test, the Wilcoxon test, and the Chi-square test were applied. A p-value of < 0.05 was considered statistically significant. All continuous variables are expressed as mean ± standard deviation (SD). The standard deviation was used to describe the variability of data distribution within each study group. No standard error of the mean (SEM) values was reported.

3. RESULTS AND DISCUSSION

3.1. Sociodemographic and Clinical Characteristics of The Obese with PCOS, Normal Weight with PCOS, and Control Groups

The results showed that the three groups were homogeneous in terms of age, with no statistically significant differences ($p=0.63$). However, there were significant statistical differences between the groups in all other variables. The group of women with PCOS and obesity had the highest mean BMI (32.6 ± 2.8 kg/m^2) and waist circumference (95.4 ± 6.2 cm), followed by the group of women with normal weight, while the control group recorded the lowest mean for both measurements ($p<0.001$). The prevalence of menstrual irregularity and a family history of PCOS was significantly higher in both PCOS groups (85% and 42%, respectively, for the PCOS + obesity group; 70% and 40%, respectively, for the PCOS group with normal weight), compared to the control group (8% and 12%, respectively), with significant statistical differences ($p<0.001$) as shown in Table 1.

3.2. Comparison of Hormonal Parameters Among the Obese with PCOS, Normal Weight with PCOS, and Control Groups

Hormonal profile analysis in Table 2, showed statistically significant differences between groups in all parameters except FSH. Luteinizing hormone (LH) levels, LH/FSH ratio, and total testosterone were significantly higher in both PCOS groups than in the control group, with the PCOS group with obesity recording the highest values for all parameters. For example, the LH/FSH ratio in the PCOS group with obesity (2.05 ± 0.48) was higher than in the normal-weight PCOS group (1.81 ± 0.42) and the control group (1.05 ± 0.30) ($p < 0.001$). In contrast, sex hormone-binding globulin (SHBG) levels were significantly lower in the PCOS group with obesity (25.8 ± 6.2 nmol/L), gradually increased in the PCOS group with normal weight (32.4 ± 6.8 nmol/L), and reached their highest levels in the control group (42.1 ± 7.4 nmol/L), with statistically significant differences ($p < 0.001$).

3.3. Comparison of Metabolic Parameters Among the Obese with PCOS, Normal Weight with PCOS, and Control Groups

The results in Table 3, revealed statistically significant differences in all metabolic parameters between groups ($p < 0.001$). The group of women with PCOS and obesity had the highest values for fasting glucose (98.6 ± 8.4 mg/dL), fasting insulin (17.8 ± 4.2 mIU/mL), and HOMA-IR (4.31 ± 1.20), indicating more severe insulin resistance in this group. They also had the highest triglycerides levels (160.2 ± 28.5 mg/dL). In contrast, HDL-C cholesterol levels were lowest in the PCOS group with obesity (43.8 ± 6.4 mg/dL), gradually increased in the normal-weight PCOS group, and reached their highest levels in the control group (54.1 ± 7.2 mg/dL). Overall, all metabolic parameters were worse in both PCOS groups compared to the control group, with these values worsening in the obese group.

3.4. Comparison of Serum Chemerin and Omentin-1 Levels Among the Obese + PCOS, Normal Weight + PCOS, and Control Groups

The results showed statistically significant differences in chemerin and omentin-1 levels among all groups ($p < 0.001$). Chemerin levels were significantly highest in the group of women with PCOS and obesity (342.8 ± 40.6 ng/mL),

followed by the normal-weight PCOS group (298.6 ± 35.2 ng/mL), while they were lowest in the control group (245.4 ± 30.8 ng/mL). In contrast, omentin-1 levels were lowest in the PCOS and obesity group (259.2 ± 32.4 ng/mL), then increased in the normal-weight PCOS group (295.8 ± 34.1 ng/mL), and reached their highest levels in the control group (342.6 ± 38.2 ng/ml). These results indicate that chemerin increases with deteriorating metabolic status, while omentin-1 decreases. As shown in Table 4.

3.5. Correlation Between Serum Chemerin and Omentin-1 levels and Markers of Ovulatory Dysfunction and Insulin Resistance

Correlation analysis showed strong statistically significant relationships between chemerin and omentin-1 levels and all parameters of ovulation dysfunction and insulin resistance. Chemerin levels were positively and statistically significantly correlated with the LH/FSH ratio ($r=0.48$, $p<0.001$), total testosterone ($r=0.51$, $p<0.001$), HOMA-IR ($r=0.55$, $p<0.001$), and menstrual irregularity ($r=0.49$, $p<0.001$). In contrast, omentin-1 levels were negatively and statistically significantly associated with the same variables: LH/FSH ratio ($r=-0.42$, $p<0.001$), total testosterone ($r=-0.39$, $p<0.001$), HOMA-IR ($r=-0.46$, $p<0.001$), and menstrual irregularity ($r=-0.44$, $p<0.001$). These results suggest that chemerin may be a strong predictor of increased severity of ovulation disorders and insulin resistance, whereas omentin-1 may be a protective factor or an indicator of better health status as shown in Table 5.

3.6. Multivariate Regression Analysis of Chemerin and Omentin-1 with Ovulatory Dysfunction Markers

After adjusting for confounding factors (age, BMI, and waist circumference), multiple regression analysis results in Table 6, showed that Chemerin levels remained positively and independently associated with LH/FSH ratio, total testosterone, insulin resistance (HOMA-IR), and menstrual irregularities. These values indicated that higher Chemerin levels enhance the severity of hormonal and metabolic imbalances in PCOS patients. In contrast, Omentin-1 showed an independent inverse relationship with all of these variables. Higher levels were associated with lower LH/FSH ratios, total testosterone, HOMA-IR, and lower menstrual irregularities, suggesting a potential protective role against hormonal and metabolic imbalances in this group of women, as shown in Table 6.

Table 1. Sociodemographic characteristics of study participants

Variable	Obese + PCOS (n=80) mean ± SD	Normal weight + PCOS (n=70) mean ± SD	Controls (n=50) mean ± SD	p-value
Age (years, mean ± SD)	27.8 ± 4.6	26.9 ± 4.4	27.1 ± 4.5	0.63
BMI (kg/m^2, mean ± SD)	32.6 ± 2.8	23.1 ± 1.9	22.7 ± 2.0	<0.001
Waist circumference (cm)	95.4 ± 6.2	78.6 ± 5.4	77.9 ± 5.6	<0.001
Menstrual irregularity (%)	85 (106/150)*	70 (49/70)	8 (4/50)	<0.001
Family history of PCOS (%)	42 (34/80)	40 (28/70)	12 (6/50)	<0.001

* Tables may have a footer.

Table 2. Hormonal profile of study groups

Parameter	Obese + PCOS (n=80) mean ± SD	Normal weight + PCOS (n=70) mean ± SD	Controls (n=50) mean ± SD	p-value
LH (mIU/mL)	11.4 ± 2.8	9.8 ± 2.5	6.3 ± 1.9	<0.001
FSH (mIU/mL)	5.6 ± 1.3	5.4 ± 1.2	6.0 ± 1.1	0.04
LH/FSH ratio	2.05 ± 0.48	1.81 ± 0.42	1.05 ± 0.30	<0.001
Total Testosterone (ng/dL)	78.2 ± 14.6	65.4 ± 13.8	42.6 ± 10.2	<0.001
SHBG (nmol/L)	25.8 ± 6.2	32.4 ± 6.8	42.1 ± 7.4	<0.001

Table 3. Metabolic parameters of study groups

Parameter	Obese + PCOS (n=80) mean ± SD	Normal weight + PCOS (n=70) mean ± SD	Controls (n=50) mean ± SD	p-value
Fasting Glucose (mg/dL)	98.6 ± 8.4	91.4 ± 7.6	89.8 ± 6.9	<0.001
Fasting Insulin (µIU/mL)	17.8 ± 4.2	12.6 ± 3.8	9.4 ± 2.6	<0.001
HOMA-IR	4.31 ± 1.20	2.84 ± 1.05	2.08 ± 0.70	<0.001
Triglycerides (mg/dL)	160.2 ± 28.5	132.4 ± 24.1	110.6 ± 20.8	<0.001
HDL-C (mg/dL)	43.8 ± 6.4	48.2 ± 6.8	54.1 ± 7.2	<0.001

Table 4. Chemerin and omentin levels in study groups

Biomarker	Obese + PCOS (n=80) mean ± SD	Normal weight + PCOS (n=70) mean ± SD	Controls (n=50) mean ± SD	p-value
Chemerin (ng/mL)	342.8 ± 40.6	298.6 ± 35.2	245.4 ± 30.8	<0.001
Omentin-1 (ng/mL)	259.2 ± 32.4	295.8 ± 34.1	342.6 ± 38.2	<0.001

Table 5. Correlation of chemerin and omentin with ovulatory dysfunction markers

Variable	Chemerin (r, p)	Omentin-1 (r, *p*)
LH/FSH ratio	r = 0.48, p <0.001	r = -0.42, p <0.001
Total Testosterone	r = 0.51, p <0.001	r = -0.39, p <0.001
HOMA-IR	r = 0.55, p <0.001	r = -0.46, p <0.001
Menstrual irregularity	r = 0.49, p <0.001	r = -0.44, p <0.001

Table 6. Multivariate regression analysis of chemerin and omentin-1 with ovulatory dysfunction markers (adjusted for age, BMI, and waist circumference)

Dependent Variable	Predictor	β (Standardized)	95% CI	p-value
LH/FSH ratio	Chemerin	0.32	0.18 – 0.45	<0.001
	Omentin-1	−0.29	−0.42 – −0.15	<0.001
Total Testosterone	Chemerin	0.35	0.20 – 0.48	<0.001
	Omentin-1	−0.27	−0.39 – −0.10	0.002
HOMA-IR	Chemerin	0.41	0.28 – 0.54	<0.001
	Omentin-1	−0.34	−0.48 – −0.19	<0.001
Menstrual irregularity	Chemerin	0.30	0.16 – 0.44	<0.001
	Omentin-1	−0.28	−0.41 – −0.12	<0.001

The current case-control study gives valuable perspectives on the relationship between chemerin and omentin-1 and the prediction of ovulatory dysfunction in women with PCOS. Our results reveal that obese and non-obese PCOS women show significant structural, hormonal, and metabolic changes in ovarian tissue, with obesity having an added burden to the alteration. It is worth noting that the high serum chemerin levels and low omentin-1 were closely associated with glucose metabolism and ovulatory function disorders. These findings indicate that chemerin and omentin-1 can be reliable biomarkers to diagnose and predict ovulatory dysfunction in PCOS, especially when it concerns obesity.

As it was reported earlier on obesity in PCOS, this is demonstrated by greater BMI and waist circumference in the obese group compared with normal-weight and control PCOS patients. The rate of menstrual irregularity was also indicated more in PCOS group, particularly, in obese women; which was also similar to Shi et al. (21) reported, a relationship between increased adiposity and reproductive dysfunction.

It was found that there were no significant differences in age before and after stratification, which ensures that the differences observed are not age dependent but rather depend on the metabolic status (22). These findings paralleled increased LH, higher LH/FSH ratios, and total testosterone levels in the PCOS cohort compared to lean controls, particularly among the obese subgroup. This finding is consistent with Pratama et al, (23). This has been referred to as core PCOS features by Yang et al. (24), who described hyperandrogenemia and altered gonadotropin secretion. Thus, lower SHBG in obese PCOS patients has been proposed to be part of the insulin resistance-SHBG axis, as hyperinsulinemia suppresses hepatic synthesis of SHBG Xu et al, (25). By contrast, another study Kotlyar et al, (26) demonstrated less marked differences in LH-to-FSH ratios that may relate to diagnostic criteria used, ethnic differences, or the ability of assay.

The features characterizing obese PCOS were increased fasting glucose, fasting insulin, and HOMA-IR, and higher triglycerides, and lower HDL-C, compared with both normal-weight PCOS subjects and controls. These findings are consistent with the established association of obesity, insulin resistance, and dyslipidemia in PCOS (27). The normal-weight PCOS group exhibited milder endocrine–metabolic disturbances; however, these findings still support the concept of a metabolically obese normal-weight (MONW) phenotype among lean women with PCOS. In contrast, Nowak-Ciołek et al. (28) reported an adjusted hazard ratio (aHR) for glioma risk, demonstrating that approximately 16% of the Methyl Easy-analyzed samples displayed either predominantly methylated or predominantly unmethylated epigenetic profiles. Lean PCOS and controls of this study had similar HOMA-IR levels possibly due to variability in dietary intake, genetic constitution, or environmental factors that modulate insulin sensitivity (29).

The most striking changes in the current study were increased chemerin and decreased omentin-1 levels in our PCOS patients, especially those who were obese. Chemerin is a potent pro-inflammatory adipokine, which may link the inflammation process in the adipose tissue to both insulin resistance and reproductive dysfunction (30). Our findings that overweight PCOS subjects had higher chemerin levels than lean PCOS and normal weight controls support earlier work by Mehrabani et al., (31) who showed that chemerin was overexpressed in adipose tissue and in metabolic syndrome and PCOS. Only small elevations in chemerin were reported by Niepsuj et al. (32) in lean PCOS (using a slightly adapted diagnosis), likely reflecting the smaller sample size difference and differences among diagnostic thresholds, despite the large extent to which adiposity is accounted for through matching on absolute BF mass.

Lin et al. (33) reported significantly lower omentin-1, an anti-inflammatory adipokine, in PCOS groups, especially in obese women, identified as inversely associated with BMI, insulin resistance and inflammatory. Omentin-1 may also decrease as a a result of reduced insulin sensitivity and the aggravating of hyperandrogenism. However, some contrasting

works also existed Biegański et al, found no difference in omentin-1 between PCOS and controls, which may be due to differences in assay methodology or ethnic variation of the populations (34).

Further analysis demonstrated a strong positive correlation of chemerin with the LH/FSH ratio, total testosterone, HOMA-IR, and menstrual irregularity, while omentin-1 showed significant inverse correlations. This is consistent with the hypothesis that adipokines, in addition to regulating metabolism, may participate in the pathophysiology of ovulatory dysfunction in PCOS through both metabolic and reproductive pathways (35). Increased chemerin could alter ovarian steroidogenesis via pro-inflammatory stimulation, whereas decreased omentin-1 may cause insulin resistance and hyperandrogenemia, leading to impaired follicular growth (36).

We suggest that PCOS pregnancies may relate to chronic low-grade inflammation and dysregulation of adipose tissue. During obesity, the increased numbers of hypertrophic adipocytes and macrophage infiltration in white adipose tissue promote elevated production of pro-inflammatory cytokines, including TNF-α and IL-6, which in turn induce chemerin expression (37). Thereby activating CMKLR1 receptors in ovarian theca cells, leading to increased androgen synthesis. In contrast, omentin-1 is primarily produced by visceral fat and increases insulin-mediated glucose uptake; its inhibition in this PCOM picture might be due to the inflammatory state and hyperinsulinemia that are part of PCOS pathogenesis, underlying endocrine abnormalities (38).

This is in agreement with the findings of Guvenc et al, (39) and Johnson et al. (40). The trends in deregulation of chemerin and omentin-1 are seen among PCOS patients, especially the obese ones. The stronger associations in our study are likely due to the strict exclusion of confounding comorbidities and the use of standardized ELISA kits with high sensitivity (39). On the other hand, unsuccessful findings are possible because the studies were too small, involved mixed PCOS phenotypes, and used non-fasting blood samples (which may confound adipokine measurements) (40). This research is new in categorizing women with PCOS as obese and normal-weight to determine the exact effects of obesity on metabolic and reproductive dysfunction. We offer a dual adipokine approach to the relationship between adipose tissue dysfunction, insulin resistance, and ovulatory disturbances by measuring chemerin and omentin-1. The correlation of these adipokines with the LH/FSH ratio, total testosterone, HOMA-IR, and menstrual irregularity is strong, providing information lacking in the literature on ovulatory dysfunction.

Given the incredible findings, this study should mention several limitations. To begin with, the case-control design limits the ability to establish causality between changes in adipokines and ovulatory dysfunction; therefore, longitudinal studies are essential to determine cause-and-effect relationships. Second, the size of the sample could be too small and limited to generalize the results because ethnic, genetic, and environmental factors might cause changes in both adipokine levels and PCOS phenotypes. Third, multivariate regression has been conducted to account for significant confounders (age, BMI, and waist circumference), but it is impossible to rule out residual confounding due to unmeasured confounders (e.g., dietary intake, physical activity, or genetic predisposition). Fourth, the biochemical tests were conducted at a single time point, which does not account for intra-individual variance or cyclic hormonal changes. Lastly, even with standardized ELISA kits, differences in study methods can still lead to variability in reported chemerin and omentin-1 concentrations. Overcoming these limitations in further research with larger, multicenter, prospective studies involving longer metabolic and genetic profiling will strengthen the case for adipokines serving as biomarkers of ovulatory dysfunction in PCOS.

4. CONCLUSIONS

In conclusion, our analysis shows that obese women with PCOS have elevated levels of chemerin and reduced levels of omentin-1 relative to normal-weight PCOS patients and controls, and are strongly correlated with indicators of ovulatory dysfunction and metabolic dysfunction. These findings provide insight into the complexity of metabolic and reproductive disorders in PCOS and the importance of adipokines as biomarkers and potential therapeutic agents.

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Conflict of interest: The authors state that they do not have any conflict of interest connected with the publication of the manuscript.

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