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Article

Association of Dyslipidemia With Reproductive Hormone Profiles and Menstrual Regularity in Patients With Polycystic Ovary Syndrome

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Abstract

Aims/Background: Polycystic ovary syndrome (PCOS) is a common endocrine and metabolic disorder among women of reproductive age, often accompanied by dyslipidemia. However, the direct association between dyslipidemia and reproductive hormone profiles and menstrual regularity remains unclear. This study aimed to retrospectively analyze the association of dyslipidemia with reproductive hormone levels and menstrual regularity in patients with PCOS. **Methods:** This was a single-center retrospective cross-sectional study that included 324 patients with PCOS who visited The First Affiliated Hospital of Soochow University between June 2021 and June 2025. Based on the inclusion and exclusion criteria, 236 patients were ultimately included. Patients were divided into a dyslipidemia group (n = 121) and a eulipidemia group (n = 115) based on their lipid profiles. General demographic data, lipid levels, reproductive hormone levels, and menstrual history were collected. Multiple linear regression was used to examine the independent associations between lipid levels and reproductive hormone levels, whereas multivariate logistic regression was used to examine the relationship between lipid levels and menstrual regularity. **Results:** Patients in the dyslipidemia group had significantly higher levels of follicle-stimulating hormone (FSH), luteinizing hormone (LH), and total testosterone (TT) than those in the eulipidemia group (all $p < 0.001$), significantly lower estradiol (E2) levels ($p < 0.001$), and a significantly higher incidence of menstrual irregularities (90.08% vs. 43.48%, $p < 0.001$). Multiple linear regression analysis showed that, after adjusting for age, body mass index (BMI), and homeostasis model assessment of insulin resistance (HOMA-IR), triglycerides (TG) and low-density lipoprotein cholesterol (LDL-C) were independently positively associated with TT, FSH, and LH, whereas high-density lipoprotein cholesterol (HDL-C) was independently and negatively associated with TT and FSH (all $p < 0.001$). HDL-C was positively associated with E2, whereas LDL-C was negatively associated with E2; however, these associations did not remain significant after correction for multiple comparisons. TG and LDL-C were independently and negatively associated with sex hormone-binding globulin (SHBG) (all $p < 0.01$). Multivariate logistic regression analysis showed that TG (OR = 2.126, 95% CI: 1.088–4.156, $p = 0.027$) and HDL-C (OR = 0.153, 95% CI: 0.052–0.448, $p < 0.001$) were independently associated with menstrual irregularity. **Conclusion:** In patients with PCOS, dyslipidemia is closely associated with reproductive hormone imbalances and menstrual irregularity. TG and HDL-C levels were independently associated with menstrual regularity. Dyslipidemia is not only a metabolic abnormality but may also contribute to the development of the endocrine phenotype of PCOS. Clinical management should consider the potential value of lipid-targeted interventions in improving reproductive and menstrual outcomes.

Keywords: polycystic ovary syndrome; dyslipidemia; reproductive hormones; menstrual irregularity; menstruation disturbances

1. Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrine and metabolic disorders affecting women of reproductive age and is a major cause of anovulatory infertility and hyperandrogenism [1]. Epidemiological studies indicate that its prevalence is approximately 5%–15%, depending on the diagnostic criteria applied, and may be even higher in some regions [2]. The diagnosis of PCOS is currently based predominantly on the Rotterdam criteria. Given its considerable phenotypic heterogeneity, PCOS is not considered a single disease entity but rather a heterogeneous syndrome characterized primarily by reproductive dysfunction and frequently accompanied by metabolic disorders [3]. In addition to reproductive manifestations such as menstrual irregularities, acne, hirsutism, and infertility, PCOS patients often exhibit metabolic disturbances, including insulin resistance, obesity, and abnormalities in lipid metabolism, suggesting that its pathophysiology involves both the reproductive and metabolic axes [4].

Among the clinical manifestations of PCOS, menstrual irregularity is one of the most common and often earliest presenting symptoms, which includes oligomenorrhea, prolonged menstrual cycles, and amenorrhea [5]. These disturbances are primarily associated with dysfunction of the hypothalamic-pituitary-ovarian axis and a persistent hyperandrogenic environment [6]. Increased luteinizing hormone (LH) secretion, together with relatively inadequate follicle-stimulating hormone (FSH) activity, may impair dominant follicle development and ovulatory dysfunction. At the same time, excessive androgen production

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by the ovaries and adrenal glands can further interfere with follicular maturation, potentially establishing a vicious cycle [7,8]. Therefore, it is challenging to fully explain the complex clinical manifestations of PCOS from a reproductive perspective alone, underscoring the need to incorporate metabolic factors into a more comprehensive analytical framework.

Lipid metabolism disorders are among the most common metabolic comorbidities of PCOS [9]. It has been reported that approximately 70% of patients with PCOS exhibit dyslipidemia, typically characterized by elevated triglycerides (TG), total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C), as well as decreased high-density lipoprotein cholesterol (HDL-C) [10]. This high-TG, low-HDL lipid profile is closely associated with insulin resistance and other metabolic disturbances. Previous animal studies have suggested that a high-fat diet can induce PCOS-like ovarian morphological changes in prepubertal animal models, including an increased number of follicles and thickened follicular walls, suggesting that disturbances in lipid metabolism may directly contribute to the development of ovarian dysfunction [11]. Clinical studies have also reported that, compared with patients with PCOS and normal blood lipid profiles, those with hypercholesterolemia often have a higher body mass index, more significant fasting hyperinsulinemia and insulin resistance, and elevated oxidative stress indicators [12]. Collectively, this evidence suggests that dyslipidemia may not only represent a metabolic comorbidity of PCOS but may also contribute to the pathophysiological processes underlying its endocrine disorders.

Notably, menstrual irregularity is not only a core clinical manifestation of PCOS but also an important clinical indicator reflecting reproductive dysfunction and, potentially, disease severity. While associations between individual lipid parameters and hyperandrogenism have been reported, existing evidence remains fragmented. Most previous studies have focused on isolated hormone levels rather than on comprehensively evaluating reproductive hormone profiles across multiple lipid fractions. Additionally, findings regarding the independent relationships between dyslipidemia and menstrual cyclicity remain inconsistent. A systematic assessment that simultaneously evaluates the spectrum of reproductive hormone levels and directly examines the association between different lipid fractions and the risk of menstrual irregularity is therefore lacking.

Therefore, exploring the relationships among dyslipidemia, reproductive hormone levels, and menstrual regularity within the framework of PCOS as a multisystem disorder has significant theoretical and clinical implications. On the one hand, such an approach may help elucidate the mechanisms of interaction between the metabolic and reproductive axes; on the other hand, it may provide new insights into risk stratification and individualized management of patients with PCOS. If dyslipidemia is independently associated with hormonal imbalances and menstrual irregularities, early identification and appropriate management may positively affect reproductive function and improve long-term metabolic outcomes. However, given the observational nature of such associations, causal relationships require further validation.

Therefore, the present study aimed to systematically analyze the relationships among blood lipid levels, reproductive hormone profiles, and menstrual regularity in PCOS patients. Specifically, the study assessed whether individual lipid parameters and dyslipidemia were independently associated with reproductive hormone levels and the risk of menstrual irregularity after accounting for metabolic and demographic factors. Unlike previous studies primarily focused on pregnancy outcomes or cardiovascular risk, this study examined the role of dyslipidemia in the endocrine phenotype of PCOS and sought to re-examine the mechanisms underlying menstrual irregularities from a metabolic perspective. By integrating blood lipid indicators, reproductive hormone levels, and clinical menstrual data, this study aimed to provide more targeted evidence to support the comprehensive management of PCOS.

2. Methods

2.1 Participants

This study was a single-center retrospective cross-sectional study. The study subjects were female patients diagnosed with PCOS who visited the outpatient clinic of The First Affiliated Hospital of Soochow University between June 2021 and June 2025. The inclusion criteria were as follows: (1) PCOS patients diagnosed according to the 2003 Rotterdam criteria [13], that is, after excluding other diseases that cause hyperandrogenism or ovulatory disorders, meeting at least two of the following three criteria: oligo- or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology; (2) age 18–40 years; and (3) complete clinical data, including menstrual history, physical measurement indicators, and laboratory test results, including fasting blood lipid profiles, fasting blood glucose, fasting insulin, and reproductive hormone levels. The exclusion criteria were as follows: (1) coexistence of other known endocrine disorders, such as thyroid dysfunction, hyperprolactinemia, congenital adrenal hyperplasia, and Cushing's syndrome; (2) a previous diagnosis of diabetes or other serious metabolic diseases; (3) previous ovarian-related surgery or organic lesions that could significantly affect ovarian function, such as ovarian tumors, stage III or higher endometriosis, or uterine fibroids >4 cm; (4) missing clinical data or incomplete key indicators; and (5) use of oral contraceptives, glucocorticoids, antiandrogens, or other drugs that may affect reproductive hormone levels or glucose and lipid metabolism within 3 months before enrollment. The patient selection flowchart is shown in Fig. 1.

All case data were obtained from the hospital's electronic medical record system and laboratory database. This study categorized patients into groups based on their blood lipid levels. According to the relevant diagnostic criteria for dyslipidemia, patients were defined as having dyslipidemia if they met any of the following criteria: fasting triglycerides (TG) ≥1.7 mmol/L, low-density lipoprotein cholesterol (LDL-C) ≥3.4 mmol/L, or high-density lipoprotein cholesterol (HDL-C) <1.3 mmol/L [14]. Patients who did not meet any of these criteria were assigned to the eulipidemia group. Based on this classification, the general clinical characteristics and metabolic indicators of the two groups were compared, and the relationships between dyslipidemia, lipid parameters, and reproductive hormone levels were further analyzed.

Reproductive hormone indicators included LH, FSH, estradiol (E2), total testosterone (TT), and sex hormone-binding globulin (SHBG). Menstrual status was determined based on medical history. Menstrual irregularity was defined as a menstrual cycle longer than 35 days or fewer than eight menstrual periods per year [15]. Menstrual history was recorded by the attending clinician at the initial visit and reflected the patient's self-reported menstrual cycle pattern during the preceding 12 months, as documented in the electronic medical record.

2.2 Collection of Patient Baseline Information and Clinical Data

All clinical data and laboratory test results of enrolled patients were obtained from the hospital's electronic medical record system and were derived from routine assessments performed during the initial evaluation phase. Laboratory tests were performed during the early follicular phase as specified in the medical records. General demographic data included age, height, and weight. Height and weight were measured by trained medical staff on the morning of the initial visit while patients were in a fasting state. Body mass index (BMI) was automatically calculated by the medical record system using the formula: BMI = weight (kg) / height $(m)^2$. Blood pressure was recorded as the blood pressure of the right upper arm measured in the seated position after resting quietly for at least 5 min at the initial visit. Two consecutive measurements were obtained using an electronic blood pressure monitor (HEM-9700T, Omron Healthcare Co., Ltd., Kyoto, Japan), and the average value was recorded. Systolic and diastolic blood pressure were recorded.

According to the medical records, all laboratory tests were performed during the early follicular phase of the patient's natural menstrual cycle (days 3–5). For patients with amenorrhea, blood samples were collected 3–5 days after progesterone-induced withdrawal bleeding to minimize the potential impact of menstrual cycle-related fluctuations on hormone levels. All blood samples were collected the following morning after an overnight fast of at least 10 h, with approximately 5 mL of venous blood obtained. All samples were subsequently sent to the hospital's clinical laboratory for analysis. The relevant testing methods and instrument models are summarized below based on the laboratory records. Fasting plasma glucose (FPG) was measured using the glucose oxidase method with a fully automated biochemical analyzer (BS-240, Shenzhen Mindray Bio-Medical Electronics Co., Ltd., Shenzhen, China). Fasting insulin (FINS) was measured using an electrochemiluminescence immunoassay with an electrochemiluminescence immunoassay analyzer (eCL8600, Lifotronic Technology Co., Ltd., Shenzhen, China). The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated by the researchers based on the FINS and FPG values recorded in the medical records using the following formula:

$$\text{HOMA-IR} = \text{FINS (mIU /L)} \times \text{FPG (mmol/L)} / 22.5.$$

Reproductive hormone levels, including TT, LH, FSH, E2, and SHBG, were measured using chemiluminescent immunoassay (CLIA) with an immunoassay analyzer (UniCel DxI 800 Access Immunoassay System, Beckman Coulter, Inc., Brea, USA). Lipid levels, including TG, HDL-C, and LDL-C, were measured using enzymatic methods with a fully automated biochemical analyzer (LABOSPECT 008 AS, Hitachi High-Tech Corporation, Tokyo, Japan). All assays were performed in the hospital's certified clinical laboratory under its routine internal quality control procedures.

The aforementioned clinical and laboratory data were extracted from the electronic medical record system and entered into the study database independently by two researchers. After cross-checking and verification, the final analysis dataset was compiled to ensure data accuracy and completeness.

2.3 Statistical Analysis Methods

Data analysis was performed using SPSS version 26.0 statistical software (IBM Corp., Armonk, NY, USA). First, the normality of all continuous variables was assessed using the Kolmogorov–Smirnov test to evaluate their distribution characteristics. Normally distributed continuous data were expressed as mean ± standard deviation ($\bar{x} \pm SD$), and independent-samples *t*-tests were used for comparisons between two groups. Homogeneity of variance was assessed using Levene's test; when the assumption of equal variances was violated, Welch's *t*-test was used for correction. For non-normally distributed data, the median and interquartile range were presented, and Mann–Whitney *U* tests were used to compare two groups. Categorical data were expressed as frequencies (n) and percentages (%), and chi-square (χ^2) tests were used to compare groups.

To explore the independent associations between blood lipid indicators and reproductive hormone levels, multiple linear regression analysis was employed. Reproductive hormone indicators (total testosterone, follicle-

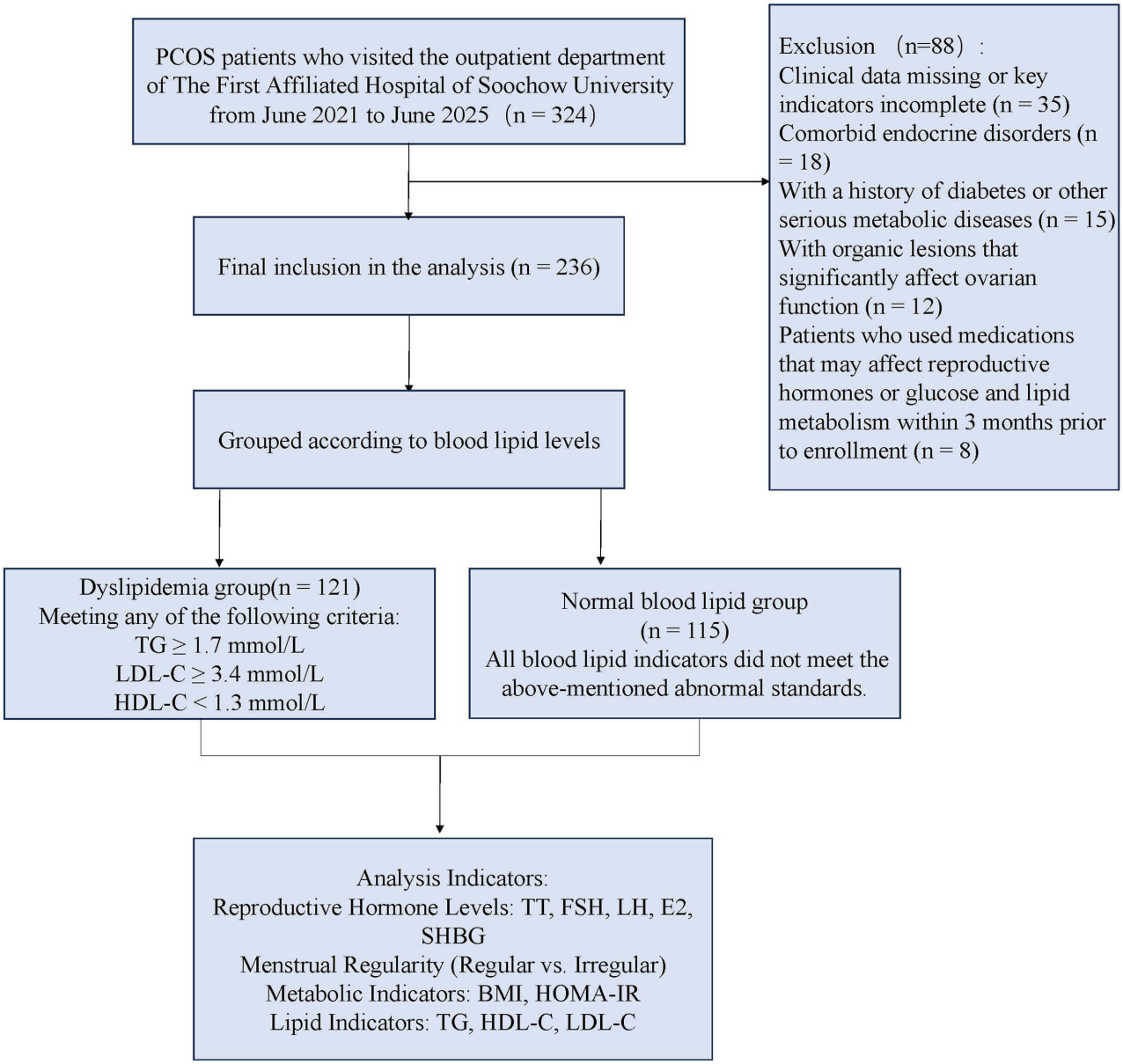


Fig. 1. Flowchart of patient selection. PCOS, polycystic ovary syndrome; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; TT, total testosterone; FSH, follicle-stimulating hormone; LH, luteinizing hormone; E2, estradiol; SHBG, sex hormone-binding globulin; BMI, body mass index; HOMA-IR, homeostasis model assessment of insulin resistance.

stimulating hormone, luteinizing hormone, estradiol, and sex hormone-binding globulin) were used as dependent variables, and blood lipid indicators (triglycerides, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol) were used as the principal independent variables. All principal independent variables were forcibly retained in the final model. A forward stepwise regression method was used, incorporating age, body mass index, and the homeostatic model assessment of insulin resistance as confounding factors, with pre-screening performed based on the results of univariable analyses. Variables with $p < 0.05$ were retained in the final model. The regression coefficients (β) and their 95% confidence intervals (CIs) were calculated. The normality of residuals was visually assessed using Q-Q plots and was confirmed for all linear regression models. Additionally, to assess the robustness of the results against potential violations of distributional assumptions, all models were re-estimated using heteroscedasticity-consistent standard errors based on the Huber-White sandwich estimator, implemented using the heteroscedasticity-consistent covariance matrix option in SPSS version 26.0. This sensitivity analysis did not alter the statistical significance of any reported associations.

To assess the association between blood lipid levels and menstrual regularity, multivariate logistic regression analysis was employed. Menstrual regularity (regular = 0, irregular = 1) was used as the dependent variable. A backward stepwise regression approach was used, with TG, HDL-C, LDL-C, SBP, DBP, age, BMI, and HOMA-IR entered as candidate variables. The variable with the highest *p*-value was then iteratively removed one at a time until all remaining variables in the model had $p < 0.05$. The odds ratios (ORs) and their corresponding 95% CIs were calculated.

All regression models underwent multicollinearity diagnostics. The variance inflation factors (VIFs) for all variables included in the regression models were below 5, indicating no substantial multicollinearity in this study. All statistical tests were two-tailed, with the significance level set at $p < 0.05$. To further assess the robustness of the key findings and account for multiple comparisons, a post-hoc sensitivity analysis using a conservative Bonferroni correction was also performed. The adjusted significance threshold for the five hormone-specific regression analyses was set at $p < 0.0033$ (0.05/15), accounting for three lipid predictors across five hormone outcomes.

3. Results

3.1 Comparison of Patient Baseline Characteristics

This study included 236 patients with PCOS, with 121 in the dyslipidemia group and 115 in the normal blood lipid group. There were no statistically significant differences between the two groups in age, BMI, SHBG, HOMA-IR, or blood pressure (all $p > 0.05$). Regarding reproductive hormones, the dyslipidemia group exhibited significantly higher levels of FSH, LH, and TT than the normal blood lipid group (all $p < 0.001$), while the dyslipidemia group had significantly lower E2 levels ($p < 0.001$). In terms of clinical characteristics, the proportion of patients with menstrual irregularity was significantly higher in the dyslipidemia group than in the normal blood lipid group (90.08% vs. 43.48%, $p < 0.001$) (Table 1).

3.2 Linear Regression Analysis of Blood Lipid Indicators and Reproductive Hormone Levels

Using each reproductive hormone indicator as the dependent variable, univariate and multivariate linear regression models were constructed, respectively. The results are shown in Tables 2,3,4,5,6.

In the analysis of TT, univariate results showed that TG and LDL-C were positively associated with TT, while HDL-C was negatively associated with TT (all $p < 0.001$). In the forward stepwise multivariate models, these relationships remained statistically significant. The independent positive associations of TG ($B = 0.443$, $p < 0.001$) and LDL-C ($B = 0.335$, $p < 0.001$) with TT, as well as the independent negative association of HDL-C ($B = –0.414$, $p < 0.001$) with TT, were all statistically significant (Table 2).

The results of the FSH analysis were largely similar to those of the TT analysis. In the univariate analysis, TG, LDL-C, and BMI were positively associated with FSH, while HDL-C was negatively associated with FSH (all $p < 0.05$). After multivariate adjustment, TG ($B = 0.599$, $p < 0.001$), LDL-C ($B = 0.760$, $p < 0.001$), and BMI ($B = 0.057$, $p = 0.008$) remained independently and positively associated with FSH, while HDL-C ($B = –0.757$, $p < 0.001$) remained independently and negatively associated with FSH (Table 3).

The results of the LH analysis showed that TG, LDL-C, and BMI were positively associated with LH, while HDL-C was negatively associated with LH (all $p < 0.05$). In the multivariate model, TG ($B = 0.769$, $p < 0.001$), LDL-C ($B = 0.523$, $p < 0.001$), and BMI ($B = 0.063$, $p = 0.017$) remained independently and positively associated with LH. HDL-C was independently and negatively associated with LH ($B = –0.393$, $p = 0.021$). However, the negative association between HDL-C and LH did not remain statistically significant after Bonferroni correction and should therefore be interpreted with caution (Table 4).

In the analysis of E2, univariate results showed that TG and LDL-C were negatively associated with E2, while HDL-C was positively associated with E2 (all $p < 0.05$). After multivariate adjustment, the positive correlation between HDL-C ($B = 4.428$, $p = 0.043$) and E2 and the negative association between LDL-C ($B = –2.315$, $p = 0.040$) and E2 remained statistically significant. In contrast, the association between TG and E2 was no longer statistically significant after adjustment ($p = 0.151$). However, these associations between lipid parameters and E2 were no longer statistically significant after applying a conservative Bonferroni correction for multiple comparisons (adjusted significance threshold: $p < 0.0033$) and should therefore be interpreted with caution (Table 5).

The results of the SHBG analysis differed from those observed for the other reproductive hormones. Univariate analysis showed that TG, LDL-C, age, BMI, and HOMA-IR were all negatively associated with SHBG (all $p < 0.05$). In the multivariate model, the independent negative associations between TG ($B = –0.920$, $p = 0.001$), LDL-C ($B = –0.760$, $p < 0.001$), and BMI ($B = –0.473$, $p < 0.001$) with SHBG remained statistically significant. In contrast, the associations of age and HOMA-IR with SHBG were no longer statistically significant after adjustment (all $p > 0.05$) (Table 6).

3.3 Univariate Logistic Regression Analysis of Blood Lipid Levels and Menstrual Regularity

With menstrual regularity (regular = 0, irregular = 1) as the dependent variable, univariate logistic regression was performed to assess associations between blood lipid indicators, potential confounding factors, and menstrual irregularity. The results are shown in Table 7.

Table 1. Baseline characteristics of patients with PCOS.

Variables	Dyslipidemia group (n = 121)	Normal blood lipid group (n = 115)	Test statistic	*p*-value
Age (years)	33.00 (26.00–38.00)	32.00 (26.00–39.00)	$Z = -0.292$	0.770
BMI (kg/ m^2)	22.20 ± 1.77	22.21 ± 2.09	$t = -0.045$	0.964
FSH (IU/L)	7.45 ± 0.85	6.64 ± 0.76	$t = 7.646$	<0.001
LH (IU/L)	10.92 ± 0.84	10.18 ± 0.90	$t = 6.476$	<0.001
E2 (pmol/L)	127.66 ± 10.14	132.38 ± 9.72	$t = -3.642$	<0.001
SHBG (nmol/L)	29.88 ± 2.06	30.21 ± 2.28	$t = -1.154$	0.250
TT (μg/L)	1.06 (0.86–1.30)	0.51 (0.35–0.74)	$Z = -9.977$	<0.001
TG (mmol/L)	1.39 ± 0.41	0.92 ± 0.38	$t = 9.011$	<0.001
HDL-C (mmol/L)	1.15 (0.92–1.27)	1.52 (1.42–1.67)	$Z = -11.384$	<0.001
LDL-C (mmol/L)	2.61 (2.12–2.97)	2.40 (2.03–2.75)	$Z = -3.054$	0.002
HOMA-IR	3.17 (2.86–3.60)	3.17 (2.71–3.54)	$Z = -0.876$	0.381
SBP (mmHg)	121.00 (112.00–130.00)	121.00 (113.00–129.00)	$Z = -0.153$	0.879
DBP (mmHg)	82.00 (71.00–91.00)	82.00 (71.50–91.00)	$Z = -0.352$	0.725
Menstrual status, n (%)			$\chi^2 = 58.259$	<0.001
Irregular	109 (90.08)	50 (43.48)		
Regular	12 (9.92)	65 (56.52)		

Abbreviations: BMI, body mass index; FSH, follicle-stimulating hormone; LH, luteinizing hormone; E2, estradiol; SHBG, sex hormone-binding globulin; TT, total testosterone; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table 2. Univariable and multivariable linear regression analyses of the association between blood lipid indicators and TT levels.

Variables	Univariable analysis				Multivariable analysis			
	SE	*t*-value	*p*-value	B (95% CI)	SE	*t*-value	*p*-value	B (95% CI)
TG	0.037	15.692	<0.001	0.581 (0.508–0.653)	0.017	25.916	<0.001*	0.443 (0.410–0.477)
HDL-C	0.065	–10.255	<0.001	–0.663 (–0.789– –0.536)	0.025	–16.575	<0.001*	–0.414 (–0.463– –0.365)
LDL-C	0.033	11.908	<0.001	0.393 (0.328–0.457)	0.013	26.063	<0.001*	0.335 (0.310–0.360)
Age	0.003	0.082	0.935	0.000 (–0.006–0.007)				
BMI	0.013	–0.127	0.899	–0.002 (–0.026–0.023)				
HOMA-IR	0.044	–0.596	0.552	–0.026 (–0.113–0.060)				

Note: * indicates statistical significance after Bonferroni correction (corrected significance threshold: $p < 0.0033$, based on three lipid predictors across five hormone outcomes). SE, Standard Error; B, Unstandardized Regression Coefficient.

Table 3. Univariable and multivariable linear regression analyses of the associations between blood lipid indicators and FSH levels.

Variables	Univariable analysis				Multivariable analysis			
	SE	*t*-value	*p*-value	B (95% CI)	SE	*t*-value	*p*-value	B (95% CI)
TG	0.115	7.530	<0.001	0.863 (0.638–1.087)	0.093	6.443	<0.001*	0.599 (0.417–0.782)
HDL-C	0.173	–6.397	<0.001	–1.106 (–1.445– –0.767)	0.136	–5.551	<0.001*	–0.757 (–1.024– –0.490)
LDL-C	0.083	10.389	<0.001	0.866 (0.702–1.029)	0.070	10.787	<0.001*	0.760 (0.622–0.898)
Age	0.008	0.711	0.478	0.006 (–0.010–0.021)				
BMI	0.030	2.192	0.029	0.066 (0.007–0.125)	0.021	2.669	0.008	0.057 (0.015–0.098)
HOMA-IR	0.106	1.618	0.107	0.172 (–0.036–0.380)				

Note: * indicates statistical significance after Bonferroni correction (corrected significance threshold: $p < 0.0033$, based on three lipid predictors across five hormone outcomes).

Univariate analysis showed that TG was significantly positively associated with the risk of menstrual irregularity (OR = 3.115, 95% CI: 1.658–5.854, $p < 0.001$), while HDL-C was significantly negatively associated with the risk of menstrual irregularity (OR = 0.107, 95% CI: 0.038–0.301, $p < 0.001$). Thus, higher HDL-C levels were associated with a lower risk of menstrual irregularity. LDL-C was significantly positively associated with the risk of menstrual ir-

Table 4. Univariable and multivariable linear regression analyses of the associations between blood lipid indicators and LH levels.

Variables	Univariable analysis				Multivariable analysis			
	SE	*t*-value	*p*-value	B (95% CI)	SE	*t*-value	*p*-value	B (95% CI)
TG	0.120	7.690	<0.001	0.920 (0.685–1.154)	0.115	6.686	<0.001*	0.769 (0.544–0.995)
HDL-C	0.190	–4.128	<0.001	–0.783 (–1.155– –0.411)	0.169	–2.327	0.021	–0.393 (–0.723– –0.062)
LDL-C	0.097	6.512	<0.001	0.633 (0.442–0.823)	0.087	6.006	<0.001*	0.523 (0.352–0.694)
Age	0.008	–0.472	0.637	–0.004 (–0.021–0.013)				
BMI	0.032	2.155	0.032	0.068 (0.006–0.130)	0.026	2.404	0.017	0.063 (0.012–0.115)
HOMA-IR	0.111	1.725	0.086	0.192 (–0.026–0.410)				

Note: * indicates statistical significance after Bonferroni correction (corrected significance threshold: $p < 0.0033$, based on three lipid predictors across five hormone outcomes).

Table 5. Univariable and multivariable linear regression analyses of the associations between blood lipid indicators and E2 levels.

Variables	Univariable analysis				Multivariable analysis			
	SE	*t*-value	*p*-value	B (95% CI)	SE	*t*-value	*p*-value	B (95% CI)
TG	1.431	–2.395	0.017	–3.427 (–6.232– –0.623)	1.491	–1.441	0.151	–2.149 (–5.072–0.774)
HDL-C	2.093	2.737	0.007	5.727 (1.626–9.828)	2.180	2.032	0.043	4.428 (0.156–8.700)
LDL-C	1.129	–2.384	0.018	–2.691 (–4.903– –0.478)	1.121	–2.064	0.040	–2.315 (–4.513– –0.117)
Age	0.091	–1.669	0.096	–0.152 (–0.329–0.026)				
BMI	0.345	0.640	0.523	0.221 (–0.456–0.898)				
HOMA-IR	1.211	–0.246	0.806	–0.297 (–2.671–2.077)				

Table 6. Univariable and multivariable linear regression analyses of the associations between blood lipid indicators and SHBG levels.

Variables	Univariable analysis				Multivariable analysis			
	SE	*t*-value	*p*-value	B (95% CI)	SE	*t*-value	*p*-value	B (95% CI)
TG	0.304	–2.684	0.008	–0.816 (–1.412– –0.220)	0.281	–3.279	0.001*	–0.920 (–1.471– –0.370)
HDL-C	0.453	–0.698	0.486	–0.316 (–1.203–0.571)	0.406	–1.520	0.130	–0.617 (–1.413–0.178)
LDL-C	0.235	–4.249	<0.001	–0.997 (–1.457– –0.537)	0.212	–3.580	<0.001*	–0.760 (–1.175– –0.344)
Age	0.019	–2.477	0.014	–0.048 (–0.085– –0.010)	0.017	–1.743	0.083	–0.029 (–0.062–0.004)
BMI	0.066	–7.811	<0.001	–0.513 (–0.641– –0.384)	0.069	–6.890	<0.001	–0.473 (–0.608– –0.339)
HOMA-IR	0.256	–2.249	0.025	–0.575 (–1.075– –0.074)	0.239	0.125	0.901	0.030 (–0.439–0.499)

Note: * indicates statistical significance after Bonferroni correction (corrected significance threshold: $p < 0.0033$, based on three lipid predictors across five hormone outcomes).

Table 7. Univariable logistic regression analysis of the associations between blood lipid levels and menstrual irregularity in patients with PCOS.

Variables	β	SE	*Z*-value	*p*-value	OR (95% CI)
TG	1.136	0.322	3.530	<0.001	3.115 (1.658–5.854)
HDL-C	–2.238	0.529	–4.230	<0.001	0.107 (0.038–0.301)
LDL-C	0.585	0.247	2.363	0.018	1.794 (1.105–2.914)
Age	0.003	0.019	0.138	0.890	1.003 (0.966–1.041)
BMI	–0.037	0.072	–0.511	0.609	0.964 (0.837–1.110)
HOMA-IR	–0.017	0.253	–0.067	0.947	0.983 (0.599–1.614)
SBP	–0.012	0.013	–0.940	0.347	0.988 (0.963–1.013)
DBP	–0.012	0.012	–1.043	0.297	0.988 (0.965–1.011)

regularity (OR = 1.794, 95% CI: 1.105–2.914, $p = 0.018$), indicating that higher LDL-C levels were associated with a higher risk of menstrual irregularity. Age, BMI, HOMA-IR, systolic blood pressure, and diastolic blood pressure demonstrated no statistically significant associations with menstrual regularity in the univariate analysis (all $p > 0.05$).

Table 8. Multivariable logistic regression analysis of the associations between blood lipid levels and menstrual irregularity in patients with PCOS.

Variables	β	SE	*Z*	*p*-value	OR (95% CI)
TG	0.754	0.342	2.206	0.027	2.126 (1.088–4.156)
HDL-C	–1.877	0.548	–3.428	<0.001	0.153 (0.052–0.448)
LDL-C	0.495	0.261	1.893	0.058	1.640 (0.983–2.739)

3.4 Multivariate Logistic Regression Analysis of Blood Lipid Levels and Menstrual Regularity

Backward stepwise logistic regression was performed with TG, HDL-C, LDL-C, age, BMI, and HOMA-IR as candidate variables. The results are shown in Table 8. In the final model, TG remained independently and positively associated with menstrual irregularity (OR = 2.126, 95% CI: 1.088–4.156, $p = 0.027$), while HDL-C remained independently and negatively associated with menstrual irregularity (OR = 0.153, 95% CI: 0.052–0.448, $p < 0.001$). LDL-C did not reach statistical significance in the final model (OR = 1.640, 95% CI: 0.983–2.739, $p = 0.058$). These findings suggest that elevated TG and reduced HDL-C levels are independently associated with menstrual irregularity, with HDL serving as a protective factor and TG as a risk factor.

4. Discussion

This single-center retrospective study systematically examined the potential associations of dyslipidemia with reproductive hormone profiles and menstrual regularity in patients with PCOS. The results showed that patients in the dyslipidemia group had significantly elevated LH, FSH, and TT levels, lower E2 levels, and a significantly higher prevalence of menstrual irregularity compared with those in the normal lipidemia group. Further linear regression analyses demonstrated significant associations between individual lipid indicators and reproductive hormone levels. Multivariate logistic regression analysis further showed that HDL-C was independently associated with a lower risk of menstrual irregularity. In contrast, TG was independently associated with an increased risk, although LDL-C did not remain statistically significant after accounting for other lipid fractions. These results suggest that, in PCOS, a disorder involving both metabolic and reproductive dysfunction, dyslipidemia is not only a metabolic comorbidity but may also be associated with endocrine disorders and menstrual irregularities. Notably, HOMA-IR did not differ significantly between the dyslipidemia and eulipidemia groups in our cohort, and HOMA-IR was included as a candidate variable in all stepwise regression models. In the final models in which HOMA-IR was retained, the lipid–hormone associations remained significant, whereas in several models HOMA-IR was not significant. This pattern suggests that the observed associations between lipid fractions and reproductive outcomes were not fully explained by insulin resistance and may involve pathways independent of insulin resistance.

Dyslipidemia is one of the most common metabolic abnormalities in PCOS, typically characterized by elevated TG and LDL-C levels and reduced HDL-C levels. The metabolic abnormalities of PCOS are closely intertwined with reproductive endocrine disorders [16,17]. In this study, TG and LDL-C showed significant positive associations with TT, while HDL-C showed a negative association; these associations remained statistically significant after adjustment for relevant confounding factors. Our findings both replicate and extend previous work in this area. Early landmark studies by Legro et al. [18] and Apridonidze et al. [19] first reported that dyslipidemia, particularly elevated TG and reduced HDL-C, clustered with hyperandrogenism in PCOS [20]. However, these earlier analyses were largely univariate or did not comprehensively adjust for key confounding factors, such as BMI and insulin resistance. In our stepwise regression models, age, BMI, and HOMA-IR were entered as candidate variables alongside lipid fractions, and only those that significantly contributed to each model were retained. The associations between lipid fractions and androgen levels persisted after multivariable modeling, thereby strengthening the evidence for a metabolic–endocrine interplay.

More recently, Mao et al. [21] reported that LDL-C and TG were positively associated with free androgen index after adjustment, consistent with our observations. Importantly, our study extends beyond hormone levels to a clinically meaningful endpoint, menstrual regularity. Our logistic regression analysis revealed that, among the lipid fractions examined, HDL-C and TG showed the most robust independent associations with menstrual irregularity. To our knowledge, the independent associations between individual lipid fractions and menstrual cyclicity, evaluated with adjustment for relevant demographic and metabolic factors, have been insufficiently characterized in previous PCOS studies. This finding helps bridge the gap between the well-documented metabolic–hormonal associations and their potential clinical manifestation in menstrual and ovulatory dysfunction.

From a hormonal perspective, the results of this study can be summarized into two main characteristics. First, patients with dyslipidemia exhibited a more pronounced hyperandrogenic profile. Cholesterol, a precursor of steroid hormone synthesis, may increase substrate availability for ovarian steroidogenesis when LDL-C levels are elevated. Simultaneously, lipid abnormalities are often accompanied by oxidative stress and a chronic low-grade inflammatory

environment. This metabolic background may enhance the responsiveness of theca cells to gonadotropin stimulation, thereby potentially exacerbating the hyperandrogenic phenotype [22,23,24]. Second, dyslipidemia was associated with altered gonadotropin secretion patterns. This study also found that TG and LDL-C were independently positively associated with FSH and LH, while HDL-C was negatively associated with these gonadotropins. These findings suggest that dyslipidemia is not only associated with peripheral hormone levels but may also be related to dysregulation of the hypothalamic-pituitary-ovarian axis. PCOS patients commonly exhibit relatively increased LH secretion, and accompanying metabolic disturbances may further amplify this neuroendocrine abnormality. Hyperinsulinemia, for instance, is recognized as a co-gonadotropin that can amplify LH-driven ovarian androgen production and has been shown to impair hypothalamic sensitivity to progesterone-mediated negative feedback on LH pulse frequency [25]. In parallel, elevated free fatty acids and inflammatory cytokines may directly modulate gonadotroph function, while hyperleptinemia and leptin resistance may alter kisspeptin-mediated GnRH secretion [26]. These converging signals may contribute to further disruption of hypothalamic-pituitary axis activity and promote a pattern of relatively increased LH secretion. Estrogen levels were relatively lower in the dyslipidemia group, suggesting that follicular development and granulosa cell function may be affected by the altered metabolic microenvironment.

The changes in SHBG provide an important complement to the above hormonal pattern. Dyslipidemia was associated with lower SHBG levels, suggesting that abnormalities in lipid metabolism may influence hepatic SHBG synthesis through metabolic or inflammatory pathways. At the clinical level, the most practically significant finding of this study is the independent association between dyslipidemia and the risk of menstrual irregularity. Menstrual disorders in PCOS are commonly associated with ovulatory dysfunction, which represents one of the core pathological features of the syndrome. This study showed that elevated TG was independently associated with an increased risk of menstrual irregularity, while elevated HDL-C was independently associated with a lower risk. LDL-C was associated with menstrual irregularity in univariate analysis but was not statistically significant in the multivariate model, suggesting that its association may be partly explained by other lipid fractions. These findings suggest that alterations in the lipid profile may interfere with the ovulatory rhythm by affecting androgen levels, SHBG concentrations, and follicular development. A previous study has also indicated a positive association between the severity of metabolic abnormalities and the degree of ovulatory dysfunction, which, to some extent, supports the observations of this study [27].

The findings of this study offer several insights into PCOS management strategies. In clinical practice, interventions for PCOS primarily focus on weight control and the improvement of insulin resistance, while dyslipidemia is often considered primarily as a long-term cardiovascular risk factor. If lipid metabolic disorders are also associated with reproductive axis dysfunction, early identification and management of lipid abnormalities, particularly elevated TG and reduced HDL-C levels, may be valuable in optimizing the endocrine environment. However, whether improving lipid profiles can directly improve reproductive outcomes remains uncertain, and this possibility requires further verification through prospective studies.

5. Limitation

This study also has several limitations. First, the retrospective cross-sectional design limits the potential to infer causality. Second, the study sample was derived from a single center, which may limit the generalizability of the findings. The mean BMI of our cohort was approximately 22.2 kg/m^2, suggesting that our findings may be more representative of relatively lean PCOS phenotypes and should be extrapolated to populations with higher adiposity with caution. Third, total cholesterol was not consistently available for all patients and was therefore not included in the dyslipidemia classification. Although the combination of TG, HDL-C, and LDL-C captures important features of the atherogenic lipid profile observed in PCOS, the omission of total cholesterol may have resulted in slight misclassification of a small subset of patients with isolated hypercholesterolemia. Fourth, the absence of inflammatory markers, adipokines, and oxidative stress markers precluded further investigation into the specific molecular mechanisms linking lipid abnormalities to reproductive axis regulation. Furthermore, the separate assessment of multiple hormone-specific models may have increased the risk of a Type I error. Although the core findings remained robust after a post-hoc Bonferroni correction, these findings should be interpreted as exploratory and require prospective verification. Future studies could incorporate longitudinal follow-up and investigate lipid-targeted interventions to determine whether improvements in blood lipid profiles are accompanied by synchronous improvements in reproductive hormone profiles and menstrual regularity, thereby clarifying the potential causal pathways.

6. Conclusion

In summary, this study demonstrates that dyslipidemia is closely associated with reproductive hormone imbalances and menstrual irregularity in patients with PCOS. Abnormal lipid metabolism may contribute to the development of the PCOS phenotype through multiple endocrine regulatory pathways, and its clinical significance should not be limited to cardiovascular risk assessment; it may also warrant consideration within the framework of reproductive function management. These findings provide further clinical evidence of the interaction between metabolic abnormalities and reproductive dysfunction in PCOS.

Key Points

• This single-center retrospective study of 236 PCOS patients found that dyslipidemia was associated with a significantly higher prevalence of menstrual irregularity and altered reproductive hormone profiles, including elevated total testosterone, LH, and FSH levels.

• Multivariable regression analyses demonstrated that TG and LDL-C were independently and positively associated with androgen and gonadotropin levels, whereas HDL-C was independently and negatively associated with these hormones; similar inverse associations were observed between TG and LDL-C and SHBG.

• In logistic regression analysis, higher TG levels were independently associated with a greater risk of menstrual irregularity, while higher HDL-C levels were independently associated with lower risk, suggesting that lipid abnormalities may be associated with ovulatory dysfunction beyond their established cardiovascular implications.

• These findings are limited by the cross-sectional single-center design, and the loss of some lipid–estradiol associations after multiple-comparison correction underscores the need for prospective validation.

Availability of Data and Materials

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions

XYS, YYZ and YPP designed the research study. XYS and XPL performed the research. YYZ and QZ analyzed the data. YPP drafted the article. All authors contributed to important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study protocol was approved by the Ethics Committee of The First Affiliated Hospital of Soochow University (approval number: 2026311). Since this study involved analysis of previously collected data and no additional interventions were performed on patients, the requirement for informed consent was waived by the Ethics Committee. The study was conducted following the Declaration of Helsinki.

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Conflicts of Interest

The authors declare no conflicts of interest.

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