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Efficacy of Prostatic Specific Antigen as a Diagnostic Marker in Polycystic Ovary Syndrome and its Correlation with Lipid Profile

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a complex endocrine disorder affecting a significant proportion of women and can lead to amenorrhea and infertility. High levels of prostatic specific antigen (PSA) were observed in PCOS, however, there is insufficient evidence regarding its efficacy as a diagnostic marker for this condition.
Objectives: To evaluate the efficacy of PSA in the diagnosis of PCOS and to assess its relation with abnormal lipid profile observed in this syndrome.
Materials and methods: A case-control study including 60 patients with PCOS and 60 healthy women as a control group. Demographic data, including age and body mass index (BMI), were collected. Blood samples were obtained from each participant to measure PSA levels and lipid profile parameters. The association of PSA with lipid profile parameters was made.
Results: PCOS patients had a significantly higher BMI compared to normal controls. The mean serum level of PSA in PCOS patients was significantly elevated in comparison to the control group. The cut-off value of PSA at $\geq$ 0.025 ng/ml was observed to be a diagnostic marker for PCOS. The highest percentage of PCOS patients were obese, with a BMI $\geq$ 30 kg/m^2. On the contrary, most women in the control group had a normal weight. Obese women in the PCOS and control groups had significantly higher mean serum PSA levels in comparison to those who were normal or overweight. A non-significant correlation was observed between PSA levels and BMI in patients with PCOS. The mean serum levels of cholesterol (CHO), triglyceride (TG), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) were significantly higher in PCOS patients than in controls. In contrast, the mean high-density lipoprotein (HDL) level was significantly lower in PCOS in comparison to control. A significant positive correlation was observed for PSA with the lipid profile parameters (CHO, TG, LDL, VLDL) in PCOS patients.
Conclusion: Elevated PSA levels in PCOS patients, closely linked with an abnormal lipid profile, indicate its efficacy as a potential marker for the diagnosis and monitoring of the condition.
Keywords: Polycystic ovary syndrome; Prostatic specific antigen; Lipid profile.

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a diverse reproductive endocrine disease affecting more than 10% of females in the childbearing age as well as young adolescents. In addition, a major cause of secondary amenorrhea and infertility due to ovulatory dysfunction [1]. However, its prevalence varies widely depending on the criteria used for diagnosis, ethnicity, genetic and environmental factors [2, 3]. The syndrome is characterized by abnormal ovarian function and morphology accompanied by two cardinal features: Hyperandrogenism and varying degrees of metabolic dysfunction [2]. The associated metabolic abnormalities include insulin resistance, hyperinsulinemia, type II diabetes mellitus, obesity, and dyslipidemia. The typical dyslipidemia pattern in PCOS is characterized by reduced high-density lipoprotein (HDL) and elevated triglyceride (TG), which increases the risk of coronary artery disease in these patient [4]. Androgen excess, on the other hand, is the central pathology in PCOS and is the primary mechanism leading

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to the inhibition of follicular growth. This results in the accumulation of excessively immature follicles and is clinically demonstrated by features of hyperandrogenemia, such as hirsutism, acne, alopecia, and ovulatory dysfunction [5].

The diagnosis of PCOS is still challenging; no single diagnostic measure or laboratory parameter is sufficient. Currently, the "Rotterdam criteria", are based on the presence of at least 2 out of 3 features, 1) Oligo-ovulation and/or anovulation. 2) Biochemical and/or clinical hyperandrogenism. 3) Ultrasound scans showing polycystic ovaries, are the most accepted method for making the diagnosis [6, 7]. Additionally, ultrasound imaging, which is the main component in these criteria, is affected by personal monitoring levels and the technical limitations of the equipment. Consequently, there is a strong emerging need to identify a new marker that is independent of the menstrual cycle and can serve as a reliable diagnostic tool for PCOS [5].

Prostatic specific antigen (PSA) is a glycoprotein secreted by the male prostate. It is a protease enzyme splitting the seminal vesicle proteins "seminogelin I and II" leading to liquefaction of the seminal coagulate. It is currently the primary indicator of malignancy, widely utilized in the screening and diagnosis of prostatic carcinoma [8]. PSA secretion in female tissue has been detected in some organs of females, like the breast, ovary, and endometrium, as well as in the milk and amniotic fluid [9]. In females with normal menstrual cycles, PSA is regarded as a mediator of local uterine function; regulating the menstrual blood flow, the cyclical proliferation of the endometrium, the implantation, and the parturition [10]. Steroid hormones such as progestins, androgens, and glucocorticoids regulate PSA production in female tissue, acting through steroid hormone receptors [11, 12].

In recent years, a growing number of studies have demonstrated high levels of PSA in association with hyperandrogenic conditions like PCOS [6]. However, there was no sufficient information regarding the diagnostic value of PSA in this syndrome. In addition, the association of PSA with insulin resistance and metabolic abnormalities in PCOS was analyzed previously [11–13], but there is a paucity in the literature addressing the association of this marker with the lipid profile in these patients. Hence, this study aimed to determine the diagnostic efficacy of PSA in a group of patients with PCOS and evaluate its relationship with the abnormal lipid profile observed in this syndrome.

MATERIALS AND METHODS

A case-control study, including 120 females aged 16-42 years, was conducted at the Department of Gynecology and Obstetrics, "Azadi Teaching Hospital", Kirkuk province, Iraq from the 7th of June 2022 to the 31st of July 2024. The study was conducted according to the principles originating from the Declaration of Helsinki. It was carried out after an informed patients' consent was taken. The study protocol has gained the approval of the Ethical Committee of the University of Kirkuk, College of Medicine according to document number 56 on June 6, 2022.

The sample size was calculated based on a confidence level of 95%, a power of study of 80%, a hypothesized difference of PSA of 0.05 among cases and control, and a population variance of 0.02, which yielded a sample size of 120. This was divided into two groups, the case group included 60 patients with PCOS and the control group comprised of 60 healthy, age-matched women with regular menses and normal-looking ovaries on ultrasound imaging. The Rotterdam criteria was used to define cases with PCOS as having two of the followings: 1) Oligo-menorrhea/amenorrhea. 2) Clinical features of hyperandrogenism. 3) Ultrasound scan showing polycystic ovaries (single or both ovaries with eight or more follicles <10 mm in diameter).

The exclusion criteria included women with the followings: History of previous ovarian surgery, ovulatory dysfunction unrelated to PCOS, idiopathic hirsutism, androgen-secreting or adrenal tumors, receiving hormonal contraception or any drugs known to affect the ovulatory function or lipid metabolism for the previous three months, as well as those with a history of the chronic medical disease (e.g. diabetes mellitus, hypertension, thyroid dysfunction, and coronary vascular disease).

Data were collected, including demographic criteria such as age and body mass index (BMI). Women were considered as normal weight at BMI (18.5–24.9 kg/m^2), overweight at BMI (25–29.9 kg/m^2), and obese at BMI ($\geq$ 30kg/m^2). For all tests, 5 ml of blood was collected after overnight fasting and estimation of the biochemical variables, including PSA level and lipid profile. Total PSA (tPSA) was estimated using an immunoenzymometric assay, committed by the "TOSOH AIA System Analyzer (TOSOH, Japan)". The serum cholesterol (CHO) (reference range <200 mg/dl), triglyceride (TG) (reference range <150 mg/dl), high-density lipoprotein (HDL) (reference range $\geq$ 60 mg/dl), low-density lipoprotein (LDL) (reference range < 100 mg/dl) and very low-density lipoprotein (VLDL) (reference range 2–30 mg/dl) were measured using the "ELITtech" Clinical system enzymatic colorimetric method. The association of PSA level with these variables was made.

Statistical data analysis was performed using the application of the statistical package for the social sciences (SPSS) version 25 software (IBM, Chicago, USA). The mean, standard deviation, and standard error were calculated for continuous data describing the demographic characteristics. To compare between continuous variables the independent t-test was applied. ANOVA test was performed to find the association between different variables in the studied groups. The correlation among the continuous variables was estimated through the application of the Pearson correlation test. For the categorized variables, numbers and percentages were used. To compare between categorized variables the Chi-square test was utilized. To determine the cutoff value of PSA for diagnosing PCOS the receiver operating characteristic (ROC) curve was used. P-value of < 0.05 was regarded to be a statistically significant difference. statistically significant difference.

RESULTS

The study included 60 patients with PCOS classified into four phenotypes according to their clinical presentation: 9 patients (15%) with oligo/amenorrhea + hyperandrogenism, 10 patients (16.7%) with oligo/amenorrhea + polycystic ovaries, 13 patients (21.7%) with polycystic ovaries + hyperandrogenism and 28 patients (46.6%) with oligo/amenorrhea + polycystic ovaries + hyperandrogenism.

There was no significant difference among the two groups of PCOS and control in term of age (P-value > 0.05); however, PCOS patients had a significantly higher BMI compared to normal controls (P-value < 0.05), additionally, the mean serum level of PSA in patients with PCOS was significantly elevated (P-value < 0.001) in compared to the control group (0.063 *vs.* 0.011 ng/ml), as shown in Table 1.

Table 1. Demographic characteristics and prostatic specific antigen (PSA) level in polycystic ovary syndrome (PCOS) patients and control groups* .

Variables	PCOS Mean ± SD	Control Mean ± SD	P-value
Age (year)	29.99 ± 7.27	28.67 ± 9.24	0.467
BMI (Kg/m^2)	30.68 ± 6.17	26.54 ± 5.43	0.002†
PSA (ng/ml)	0.063 ± 0.023	0.011 ± 0.005	0.001†

* Independent sample *t*-test, BMI: Body mass index, †Significant (P-value < 0.05)

The receiver operating characteristic (ROC) curve analyses revealed that the cut-off value of a PSA at ≥ 0.025 ng/ml was associated with the best combination of sensitivity and specificity (96.7% and 98.9%, respectively) as a diagnostic marker for PCOS, as shown in Figure 1.

The highest percentage of PCOS patients were obese with a BMI ≥ 30 kg/m^2 (50%). in contrast, the majority of women in the control group had a normal weight (56.7%). Obese women in both the PCOS and control groups had significantly higher mean serum PSA levels compared to those who were of normal or overweight (Table 2).

Regarding the correlation between serum PSA level and BMI in PCOS patients, the statistical analysis of the data revealed a non-significant correlation for PSA with this confounder (r = 0.187, p = 0.153) as shown in Figure 2.

The mean serum levels of CHO, TG, LDL, and VLDL were significantly higher in PCOS (239.03, 162.62, 178.85, and 32.92) in comparison to normal controls (142,17, 105.79, 76.22, and 21.16), respectively. In contrast, the mean HDL level was significantly lower in PCOS compared to the control group (35.41 vs. 45.17), as shown in Table 3.

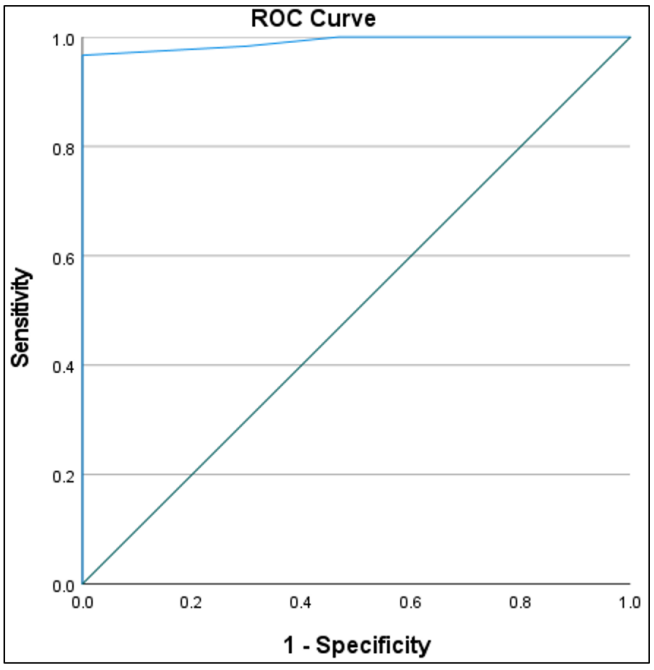

Figure 1. Receiver operating characteristic curve for prostatic specific antigen as a diagnostic marker for polycystic ovary syndrome.

The results revealed a gradual increase in mean serum levels of CHO, TG, LDL, and VLDL and a reduction in HDL level with increasing BMI in both PCOS and control groups, furthermore, NW, OW, and OB PCOS patients had significantly higher levels of atherogenic lipids and lower HDL compared to controls with the same BMI (Table 4).

Further analysis of the data in the PCOS group demonstrated a significant positive (P-value < 0.05) correlation for PSA with lipid profile parameters (CHO, TG, LDL, and VLDL) (r = 0.638, r = 0.368, r = 0.644, and r = 0.414, respectively), a negative but non-significant (P-value > 0.05) correlation was observed for PSA concerning HDL (r = -0.197) as shown in Figure 3.

DISCUSSION

PCOS remains a syndrome with complex pathogenesis influenced by several environmental and genetic factors and regulated by multiple hormones [14]. Several hormonal markers, such as follicle-stimulating hormone (FSH), luteinizing hormone (LH), anti-mullerian hormone (AMH), and testosterone, have been proposed as diagnostic tools for PCOS. However, it appears that these hormones have variable diagnostic performance and are subjected to variations throughout the menstrual cycle [6]. The main outcomes of the current research were, firstly, the level of tPSA was found to be substantially elevated in patients with PCOS. Furthermore, the serum level of this marker was found to be significantly linked with the abnormal lipid profile, the potentially life-threatening metabolic abnormality, observed in this syndrome.

The source, physiological role, and enzymatic activity of PSA in PCOS are not yet fully understood. However, PSA might reflect androgenic activity in the androgen-sensitive tissue. Recent advancements in highly sensitive methods for measuring PSA have enabled its evaluation as a potential biomarker for PCOS [5, 11]. In the current study, patients with PCOS exhibited significantly elevated tPSA levels compared to controls. A cutoff level of PSA ≥ 0.025 ng/ml demonstrated excellent sensitivity and specificity for detecting the condition. To the best of our knowledge, only two studies have established PSA cutoff points for PCOS diagnosis. In the study by Mardanian et al. [15], a cutoff value of > 0.07 ng/ml was identified, yielding a sensitivity of 91% and specificity of 81.2%. Similarly, Wang et al. [16] compared PSA, nesfatin-1, and AMH in 200 PCOS cases and reported a PSA cutoff value of 16.56 pg/ml, with a sensitivity and specificity (95.3% and 83.5%, respectively) greater than the other two markers. The differences in the cut-off values between the studies may stem from variations in the methodologies and units used for PSA measurement.

Several studies have explored PSA in PCOS to uncover potential associations. Some researchers suggest that, due to the low PSA levels in females, it could serve as a monitoring index for the management of the disease [6, 16]. Notably, Nagar et al. [10] reported non-significant variation in PSA levels across different menstrual phases or between pre- and post-menopausal women, indicating that PSA is unaffected by hormonal fluctuations and can be tested at any point in the menstrual cycle. However, a single study on adolescent girls with PCOS found no diagnostic benefit for serum tPSA [17].

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Table 2. Prostatic specific antigen level in polycystic ovary syndrome and control groups in relation to body mass index (BMI)*.

BMI(Kg/m^2)	PCOS		Control		P-value
	No (%)	PSA (ng/ml)ϒ Mean ± SE	No (%)	PSA (ng/ml)ϒ Mean ± SE	
NW	8 (13.3)	0.051 ± 0.007	34 (56.7)	0.009 ± 0.001	0.001†
OW	22 (36.7)	0.065 ± 0.005	12 (20)	0.015 ± 0.002	0.001†
OB	30 (50)	0.067 ± 0.004	14 (23.3)	0.017 ± 0.001	0.001†

* Chi-square test, ϒ Independent sample *t*-test, NW: Normal weight, OW: Over-weight, OB: Obese, †Significant (P-value < 0.05).

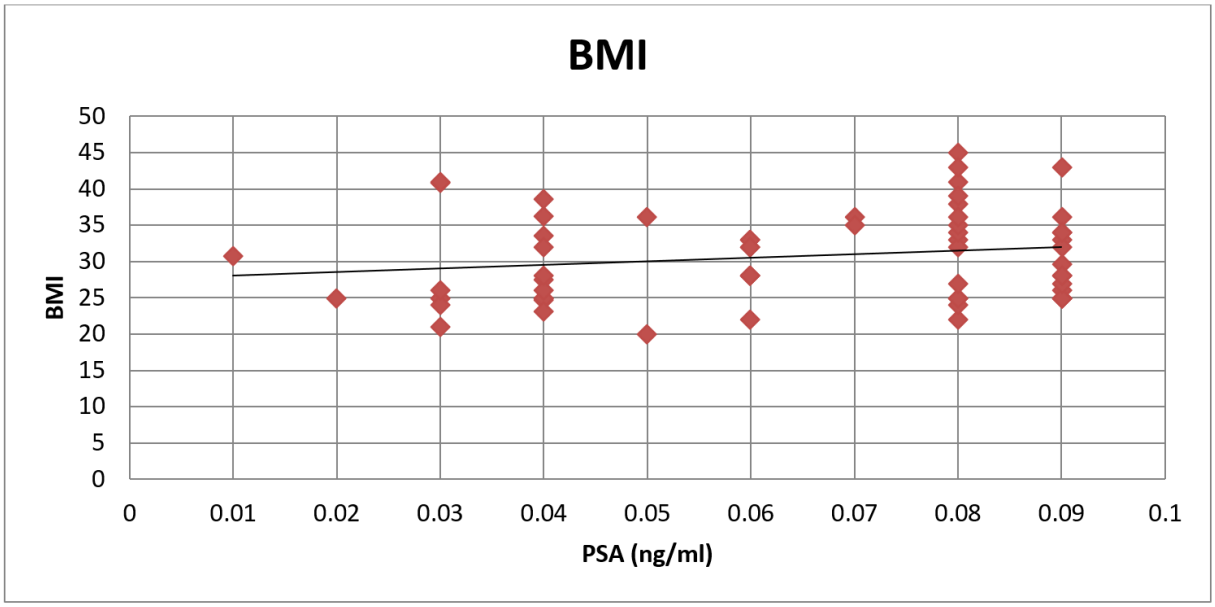

Figure 2. Correlation of prostatic specific antigen (PSA) with body mass index (BMI) in polycystic ovary syndrome*.

* Pearson correlation test (r = 0.187, P-value = 0.153).

Table 3. Lipid profile in polycystic ovary syndrome and control groups*.

Lipid profile	PCOS		Control		P-value
	Mean	SD	Mean	SD	
CHO (mg/dl)	239.03	40.55	142.50	29.38	0.001†
TG (mg/dl)	162.62	32.68	105.97	13.96	0.001†
HDL (mg/dl)	35.41	8.23	45.17	5.99	0.001†
VLDL (mg/dl)	32.92	6.70	21.16	2.76	0.001†
LDL (mg/dl)	178.85	33.80	76.22	31.48	0.001†

* Independent sample *t*-test, CHO: Cholesterol, TG: Triglyceride, HDL: High-density lipoprotein, VLDL: Very low-density lipoprotein, LDL: Low-density lipoprotein, †Significant (P-value < 0.05).

Our study also revealed that PCOS patients had significantly higher BMI, with nearly half suffering from obesity. While higher PSA levels were observed in women with elevated BMI, no significant correlation was detected between PSA and BMI in the PCOS group. Obesity remains a whole mark of PCOS, contributing to hyperandrogenism, ovulatory dysfunction, and metabolic disturbance [4]. The elevated PSA levels in overweight and obese PCOS patients may be attributed to lower sex hormone binding globulin levels, leading to an elevation in the free androgen levels and promoting PSA expression.

Additionally, obese and overweight women in both groups displayed more pronounced lipid profile abnormalities, including a significantly higher total CHO, TG, LDL, and VLDL, along with lower HDL levels. PCOS patients exhibited a greater degree of dyslipidemia compared to controls, even after adjusting for BMI. The marked increase in atherogenic lipids among overweight and obese women may result from

Table 4. Lipid profile in relation to body mass index (BMI) in polycystic ovary syndrome (PCOS) patients and control*.

Lipid profile		BMI (Kg/m^2)						P-value
		NW		OW		OB		
		Mean	SD	Mean	SD	Mean	SD	
CHO (mg/dl)	PCOS	172.38	35.83	249.91	34.52	248.83	27.53	0.001†
	Control	118.59	10.85	169.00	11.22	177.86	4.66	
TG (mg/dl)	PCOS	149.13	35.66	162.27	32.30	166.47	32.32	0.001†
	Control	95.71	6.48	117.33	8.67	121.14	8.44	
HDL (mg/dl)	PCOS	50.75	4.95	36.28	5.24	30.68	4.83	0.001†
	Control	48.76	5.29	39.67	2.61	41.14	2.68	
VLDL (mg/dl)	PCOS	29.83	7.13	32.45	6.46	34.09	6.67	0.001†
	Control	19.14	1.30	23.47	1.73	24.09	1.69	
LDL (mg/dl)	PCOS	123.75	36.97	184.59	24.21	189.33	24.37	0.001†
	Control	50.80	12.72	105.83	12.23	112.57	6.05	

* ANOVA test, CHO: Cholesterol, TG: Triglyceride, HDL: High-density lipoprotein, VLDL: Very low-density lipoprotein, LDL: Low-density lipoprotein, †Significant (P-value < 0.05).

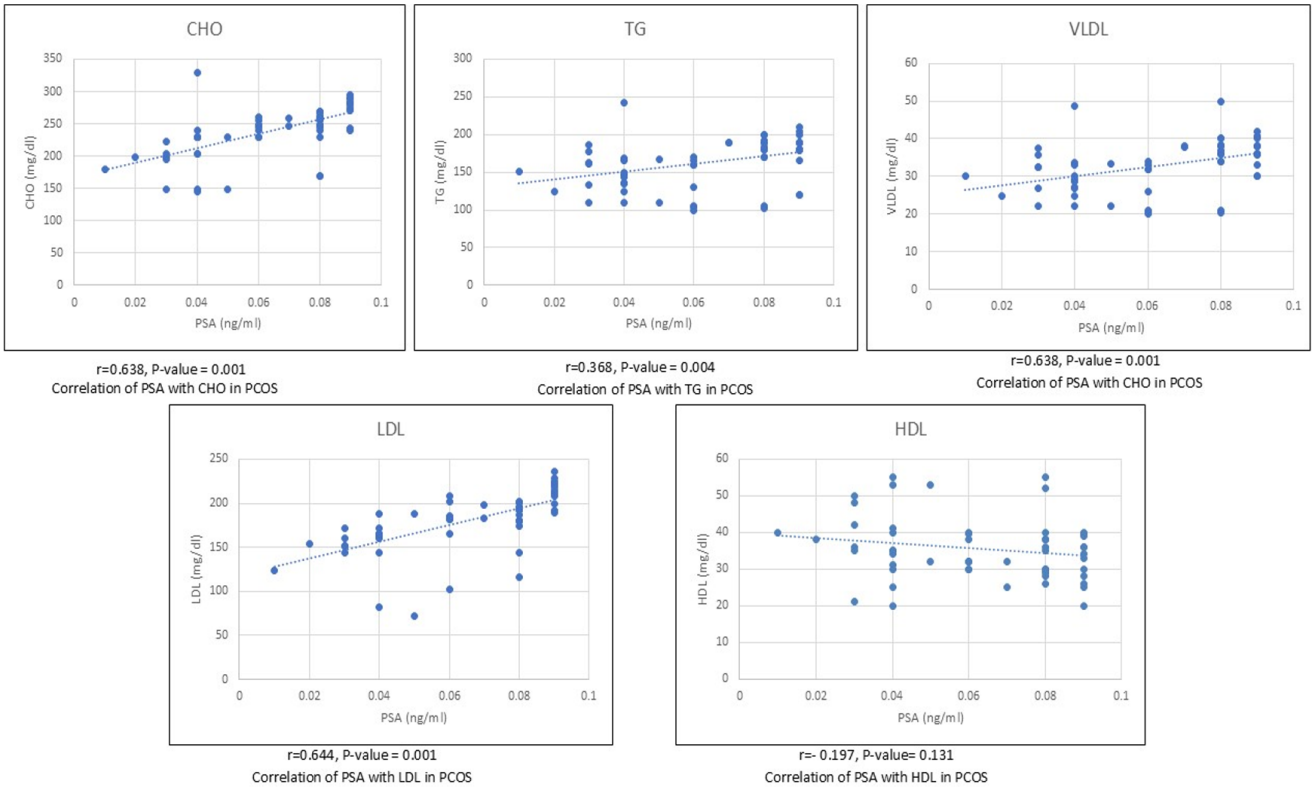

Figure 3. Correlation of prostatic specific antigen (PSA) with lipid profile in polycystic ovary syndrome (PCOS). CHO: Cholesterol, TG: Triglyceride, HDL: High-density lipoprotein, VLDL: Very low-density lipoprotein, LDL: Low-density lipoprotein.

enhanced lipogenesis and impaired clearance or oxidation of fatty acids. Pronounced dyslipidemia in PCOS patients, independent of BMI, may be linked to central obesity, which profoundly impacts the lipid profile [18]. Previous studies on lipid profile and dyslipidemia in PCOS have reported conflicting results. Findings from Korea [19] and Turkey [20] align with our observations of elevated CHO, TG, LDL, VLDL, and reduced HDL. Conversely, in a study from Iran [4] noted higher CHO in controls and an increased TG and decreased HDL in PCOS cases, with BMI adjustment eliminating the significant differences. Rocha et al. [21] reported double the incidence of dyslipidemia in PCOS patients compared to controls, with decreased HDL and increased TG but no differences in CHO or LDL. These discrepancies between studies may reflect regional, ethnic, genetic, dietary, lifestyle, and economic variations among the studied populations.

The current study reported a significant positive correlation between PSA and the lipid profile variables (CHO, TG, LDL,

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and VLDL) in the PCOS group. Both androgens and estrogens exert opposing effects on hepatic lipoprotein metabolism, though the precise impact of hormonal disturbance on dyslipidemia remains unresolved. Androgen excess promotes lipolysis, releasing free fatty acids into the circulation and increasing VLDL production, which raises TG, reduces HDL, and elevates LDL levels [22]. A study by Rajbanshi et al. [23] corroborated the relationship between hyperandrogenemia, hepatic fat accumulation, and adverse lipid profile. While no prior study directly correlates PSA and dyslipidemia, our findings align with previous findings suggesting the role of androgens in the development of dyslipidemia, given PSA's association with hyperandrogenism [4, 22].

However, it is worthy to mention that our study has certain limitations as all the cases were recruited from a single center in Kirkuk province with a relatively small number which might act as a selection bias and affect certain demographic and clinical characteristics of the studied groups. In addition, the tPSA level was evaluated rather than the free component which might affect the accuracy of the results.

CONCLUSION

Our research showed a significantly higher PSA levels in PCOS and was closely linked to the abnormal lipid profile seen in these patients. This suggests that PSA could be used as a single marker for diagnosing and monitoring the disease, avoiding the problems associated with combined diagnosis using two separate markers. Further studies from multiple centers with larger sample sizes, exploring the association of PSA with other hormonal and metabolic parameters of PCOS, are needed before its wide application in clinical practice.

ETHICAL DECLARATIONS

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Ethics Approval and Consent to Participate

The study protocol has gained the approval of the Ethical Committee of the University of Kirkuk, College of Medicine, according to document number 56 on June 6, 2022. Informed consent was obtained from every participant.

Consent for Publication

Not applicable (no individual personal data included).

Availability of Data and Material

Data generated during this study are available from the corresponding author upon reasonable request.

Competing Interests

The author declares that there is no conflict of interest.

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Authors' Contributions

Rifat AG was responsible for the design and implementation of the study, patients' selection, data collection and analysis, and writing of the manuscript. Rifat AG read and approved the final version of the manuscript.

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