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Assessment of adiponectin and lipid profile with polycystic ovarian syndrome

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ABSTRACT

Polycystic ovarian syndrome (PCOS) is an endocrine disorder commonly affecting women of reproductive age. It is characterized by increased androgens, metabolic dysfunction and irregular or absent menstrual periods. The study aimed to estimate the level of adiponectin and lipid profile related with polycystic ovarian syndrome. The study included 90 women between September 2024 to September 2025 with a range of ages (15-45years) (28.666±6.837), divided into two groups: 45 with a mean (31.689±6.842) used as healthy and 45 with mean of age (24.644±5.390) as patients. The serum adiponectin, anti-mullerian hormone (AMH), luteinizing hormone (LH), follicular stimulation hormone (FSH), prolactin, and t.testosterone, were quantified through Enzyme-Linked Immunosorbent Assay (ELISA) techniques. lipid profile (Cholesterol, Triglyceride, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low density lipoprotein (VLDL) measurement by spectrophotometer. The result found a significant decrease ($p \leq 0.01$) in adiponectin, high density lipoprotein (HDL) and follicular stimulation hormone (FSH) in the PCOS patient compared to the healthy group, also in the hormonal level, a significant increase($p \leq 0.01$) in anti-mullerian hormone (AMH), luteinizing hormone(LH), prolactin, testosterone, lipid profile demonstrated a statistically significant increase ($p \leq 0.01$) in the LDL, cholesterol, TG, VLDL, adiponectin AUC:95.704%, with Sensitivity to Specificity 93.333%-97.678%. Low adiponectin and dyslipidemia are connected to the pathophysiology of PCOS and can be uses as clinical marker for PCOS.

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1. INTRODUCTION

Polycystic ovarian syndrome(PCOS) affects women of fertility age. The main symptoms of this disorder include irregular menstrual cycle, hyperandrogenism, Acne, and hirsutism [1]. Diagnosis of PCOS is based on two characteristics, at least this characteristic includes (Hyperandrogenism, anovulation, and PCOS morphology on sonography) according to Rotterdam criteria 2003[2, 3]. the pathophysiology of PCOS is unclear, but most research suggests that hormonal imbalance, environmental factors, genetic factors, inflammation, obesity, and epigenetic factors are implicated in PCOS [4]. Adiponectin, a hormone predominantly released by adipose tissue, has two types of membrane receptors: receptor 1 (Adipo-R1) and receptor 2 (Adipo-R2) is an anti-inflammatory, insulin-sensitizing, and vasodilatory agent essential for healthy metabolic and vascular function [5]. adiponectin has attracted a lot of attention due to its potent anti-inflammatory, anti-atherogenic, and insulin-sensitizing qualities, It is promotes glucose absorption and fatty acid oxidation under physiologically normal circumstances, which aids in preserving metabolic homeostasis, PCOS, where serum adiponectin level are commonly decreased than those in healthy groups, may represent a pathogenic relationship between dysfunctional ovaries, decreased fat cell activity and it was linked to insulin resistance [6,7]. Lipid profiles play a crucial role in evaluating the risk of cardiovascular diseases in women with polycystic ovary syndrome (PCOS). Dyslipidemia is frequently observed in this population, typically characterized by reduced serum of HDL and increased triglycerides. These lipid abnormalities are significant contributors to an increased long-term risk of cardiovascular conditions.

Therefore, regular monitoring of lipid levels is essential for guiding both lifestyle modifications and pharmacological interventions aimed at mitigating cardiovascular risk [8] The objective of this study was to evaluate the role of adiponectin and lipid profile in PCOS.

2. METHOD

2.1 The study design

A case-healthy study was conducted to evaluate fertility in PCOS women in Karbala, Iraq. The data were collected from September 2024 to September 2025 at the hospital, Fertility Unit in Karbala. the diagnosis of PCOS was confirmed by a consulting physician and was based on the Rotterdam criteria from 2003, as outlined in the Rotterdam ESHRE/ASRM Consensus of 2004 to be diagnosed through laboratory tests, monitoring of certain with PCOS, patients needed to meet at minimum two out of the following three features:(i) ovulation dysfunction (ii) exhibiting either biochemical or clinical symptoms, and sign of hyperandrogenism, which includes hirsutism, alopecia, or acne, (iii) the presence of polycystic ovaries as observed through transvaginal ultrasound examination by the physician, according to the Rotterdam criteria, indicating morphological changes in the ovaries [9]. and by collecting information for each group using a questionnaire.

2.2 Exclusion ad Inclusion

The exclusion: of this study includes patients with diabetes, smoking, hormonal contraceptives, and ovary surgery.
The inclusion: includes married women diagnosed with PCOS with ages (15-45) years.

2.3 Sample Collection

The study included (90) individuals that was divided into two groups as follows:(45n) of PCOS female patients, (45n) appeared healthy and were used as healthy. After completing the questionnaire on day 2 or 3 of the menstrual cycle A 5 ml blood sample was collected, s was put in gel tubes and allowed to clot at room temperature °C for 10 min before being centrifuged at 5000 rpm, before being utilized to measure research tests, the serum was separated and kept at -18°C in an Eppendorf tube. Serum levels of Adiponectin, anti-mullerian hormone (AMH), luteinizing hormone (LH), follicular stimulation hormone (FSH), prolactin, and testosterone hormone utilized an enzyme-linked immunosorbent test (ELISA) in accordance with the guidelines provided by the manufacturer (Elabscience\USA). ELISA Kit could clone /USA for Lipid profile includes |(TC), (TG), and (HDL) and is determined by a spectrophotometer. Very low-density lipoprotein (VLDL) is calculated by the formula (VLDL= TG/5) [10].

The formula calculated the Low-density lipoprotein (LDL)

$$\text{LDL-c (mg/d L)} = \text{TC (mg/d L)} - \text{HDL-c (mg/dL)} - \text{TG (mg/dl)}$$ [11].

2.4. Estimation of Body mass index (BMI)

Body mass index (BMI) is calculated by.

$$\text{BMI} = \text{Weight (kg)} / \text{Height (m2)}$$ [12]

Serum levels of Adiponectin were determined using a commercially available human ELISA kit, which utilizes the sandwich ELISA technique. The microplate included in the kit was pre-coated with a specific antibody targeting human Adiponectin. A biotin-labeled detection antibody and an avidin–horseradish peroxidase (HRP) conjugate were added sequentially. Upon substrate addition, a blue color developed, which turned yellow after the addition of the stop solution. The intensity of the resulting color was measured spectrophotometrically at a wavelength of 450 ± 2 nm.

2.5. Ethical approval

The study was conducted following ethical standards, and approval was obtained under the reference number: IQ.UOK.CAMS.DCL.REC.2.

2.6. Statistical Analysis

Version 23 of the SPSS statistical programs was used to do the statistical analysis. Descriptive statistics have been used to compress the analysis results. Additionally, the standard deviation and mean have been computed. A probability criterion of $p < 0.01$ was used to evaluate the experimental data's statistical significance. The data's normality has also been evaluated using the Shapiro-Wilk test, and the homogeneity of variance has been examined using the Levene test. Additionally, the Independent T-Test and Mann-Whitney Test were used to determine statistical differences between two distinct groups. Additionally, numerous comparisons between the groups were carried out using variance analysis (ANOVA) [13].

3. RESULTS

3.1. Adiponectin and Lipid Profile Comparison between PCOS female and healthy.

The results in table 1 found a highly significant decrease($p \leq 0.01$) in adiponectin and HDL in the female patients compared to the healthy, also found a significant increase($p \leq 0.01$) in TC, TG, LDL, and VLDL in the PCOS patients compared to the healthy group as in table 1.

3.2. Comparison of Fertility Hormone between PCOS female and healthy.
Result of Table 2, highly significant elevated (p≤0.01) hormonal parameters that include (LH, prolactin, testosterone, and AMH) were found in female patients respectively compared to the healthy, and significant decrease(p≤0.01) in FSH in the female patient compared to the healthy. Figure 1, 2, 3.

3.3. Adiponectin in the PCOS patient compared to the healthy group based on clinical indicator
Serum adiponectin concentrations were significantly decrease (p≤ 0.01) in PCOS patients regardless of clinical symptoms such as hirsutism and acne, as well as fertility parameters including type of infertility (primary and secondary) and parity status (nulliparousvs. parous), when compared to healthy as in table 3.

3.4. Estimation of adiponectin in PCOS patients according to different demographic criteria.
Significant decreased (p≤ 0.01) of adiponectin in PCOS patients were observed across different demographic criteria, including address (rural vs. urban), age groups (less than 30 and greater than 30 years), type of food (healthy vs. unhealthy), type of delivery (cesarean vs. natural), and BMI categories (normal, overweight, obese), compared to their respective healthy groups as in table 4.

3.5. Receiver Operative Characteristic Curve (ROC) for research parameter
Receiver operating characteristic (ROC) curve analysis demonstrated high diagnostic accuracy of measured biomarkers including adiponectin, AMH, testosterone, prolactin, FSH, and LH, with areas under the curve (AUC) ranging from approximately 95.704% to 98.123%, reflecting excellent sensitivity and specificity for differentiating PCOS patients from healthys, as in table 5.

Table 1. Comparison of lipid profile and adiponectin between the PCOS patients and the healthy group.

Parameters	group	Mean	Std. Deviation	P-value
Cholesterol(mg\dl)	Healthy	175.06	4.23	0.0004**
	Patient	225.32	24.13	
TG (mg\dl)	Healthy	209.12	13.48	0.0006**
	Patient	229.44	20.94	
HDL (mg\dl)	Healthy	63.56	7.02	0.0001**
	Patient	42.15	5.19	
LDL (mg\dl)	Healthy	69.74	8.22	0.0001**
	Patient	135.97	22.41	
VLDL (mg\dl)	Healthy	42.41	3.21	0.0002**
	Patient	45.89	4.19	
Adiponectin (ng\dl)	healthy	2.86	0.49	0.0001**
	Patient	1.26	0.19	

**. The difference is significant in the mean at level (p≤0.0001), HDL: High - density lipoprotein, TG; Triglyceride, LDL: Low density lipoprotein VLDL: Very low-density lipoprotein)

Table 2. The level of different fertility hormones in PCOS patients as compare to healthy.

Parameters	Level	Mean	Std. Deviation	P-value
T.testosterone(ng\ml)	Healthy	1.08	0.05	0.00002**
	Patient	2.73	0.51	
Prolactin (ng\ml)	Healthy	20.74	4.33	0.0001**
	Patient	64.21	11.66	
FSH (mIU/mL)	Healthy	5.72	0.70	0.0004**
	Patient	2.61	0.61	
LH (mIU/mL)	Healthy	0.51	0.16	0.0008**
	Patient	3.33	0.75	
AMH (pg\ml)	Healthy	895.01	198.18	0.0001**
	Patient	2386.14	530.09	

NOT:(FSH- Follicle stimulation hormone, LH- luteinizing hormone, AMH- Anti-mullerian hormone, T. testosterone- Total testosterone, ** (p-vlue≤0.0001)

Table 3. Adiponectin in the PCOS patient compared to the healthy group based on clinical indicator.

Parameters	Classification	group	Mean	Std. Deviation	P-value
Adiponectin (ng\dl)	Type of infertility	Primary	1.301	0.170	0.0001
		Secondary	1.212	0.201	
		Healthy	2.857	0.495	
	Acne	Yes	1.956	0.163	0.0002
		No	1.248	0.213	
		Healthy	1.265	0.495	
	Para\Gravida	Para	1.301	0.170	0.0003
		Gravid	1.212	0.201	
		healthy	2.857	0.495	
	Hirsutism	No	1.215	0.227	0.0002
		Yes	1.268	0.181	
		Healthy	2.857	0.495	

Table 4. Estimation of adiponectin in PCOS patients according to different demographic criteria.

Parameters	Classification	Groups	Healthy		Patient		P-value
			Mean	Std. Deviation	Mean	Std. Deviation	
Adiponectin(ng\dl)	Last of the delivery	None	2.477	0.282	1.301	0.170	0.00001
		One Year	3.324	0.560	1.387	0.058	0.0397
		Two Year	2.925	0.568	1.230	0.278	0.0009
		More than two years	2.847	0.479	1.188	0.197	0.00002
	Type of delivery	Cesarean	2.705	0.398	1.233	0.212	0.0001
		Natural	2.901	0.516	1.192	0.198	0.0001
	Address	Rural	2.889	0.553	1.274	0.178	0.00002
		Urban	2.810	0.401	1.250	0.196	0.00004
	Age	Less than 30 years	2.864	0.507	1.258	0.199	0.00002
		More than 30 years	2.852	0.495	1.256	0.166	0.0001
	Nature of food	Healthy	2.010	0.456	1.202	0.207	0.00006
		Unhealthy	2.815	0.542	1.280	0.180	0.00003
	BMI (km/m2)	Normal	2.708	0.422	1.288	0.220	0.00008
		overweight	2.907	0.523	1.225	0.200	0.00001
		Obese	2.952	0.523	1.278	0.168	0.00003

Table 5. Receiver operation characteristic (ROC)analysis for research parameters.

Metrics		T.testosterone	Prolactin	FSH	LH	Adiponectin	AMH
Std. Error		0.018	0.013	0.018	0.021	0.026	0.015
Asymptotic Sig.		0.001	0.003	0.009	0.002	0.008	0.008
Asymptotic 95% Confidence Interval	Lower Bound	0.938	0.955	0.939	0.925	0.906	0.946
	Upper Bound	1.000	1.000	1.000	1.000	1.000	1.000
Cutoff Point		1.198	35.143	4.329	2.455	1.845	1465.212
Area Under Curve (AUC)		97.333%	98.123%	97.432%	96.642%	95.704%	97.531%
Sensitivity		95.556%	97.778%	95.452%	91.111%	93.333%	93.432%
Specificity		95.355%	95.456%	93.333%	95.535%	97.678%	97.780%
Accuracy		95.556%	96.667%	94.444%	93.333%	95.556%	95.556%
Positive Predictive Value		95.455%	95.652%	93.478%	95.349%	97.674%	97.674%
Negative Predictive Value		93.478%	97.727%	95.455%	91.489%	93.617%	93.617%

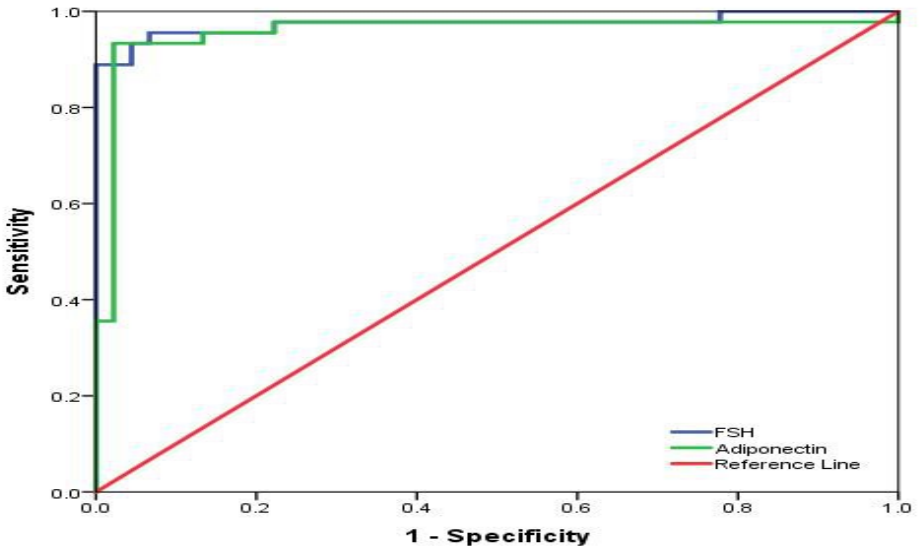

Figure 1. Receiver operation characteristic curve for FSH and adiponectin parameter.

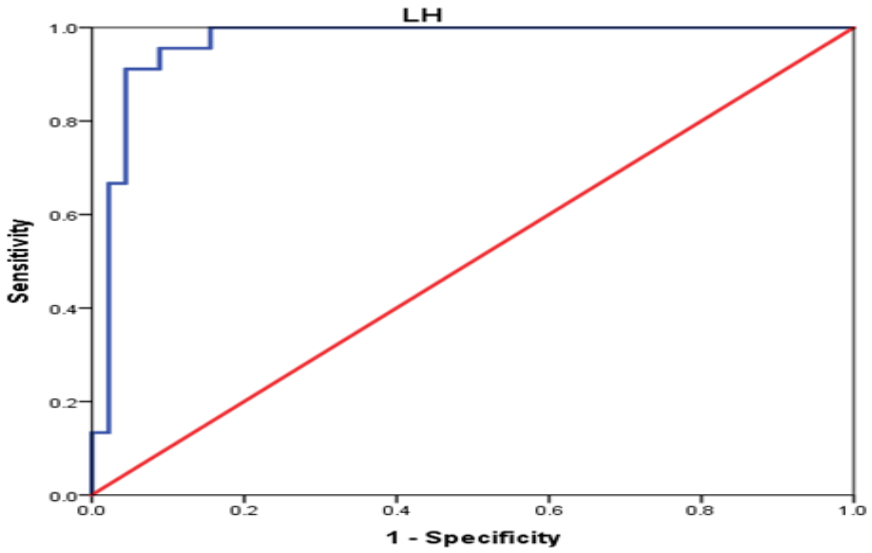

Figure 2. Receiver operation characteristic curve for LH parameter.

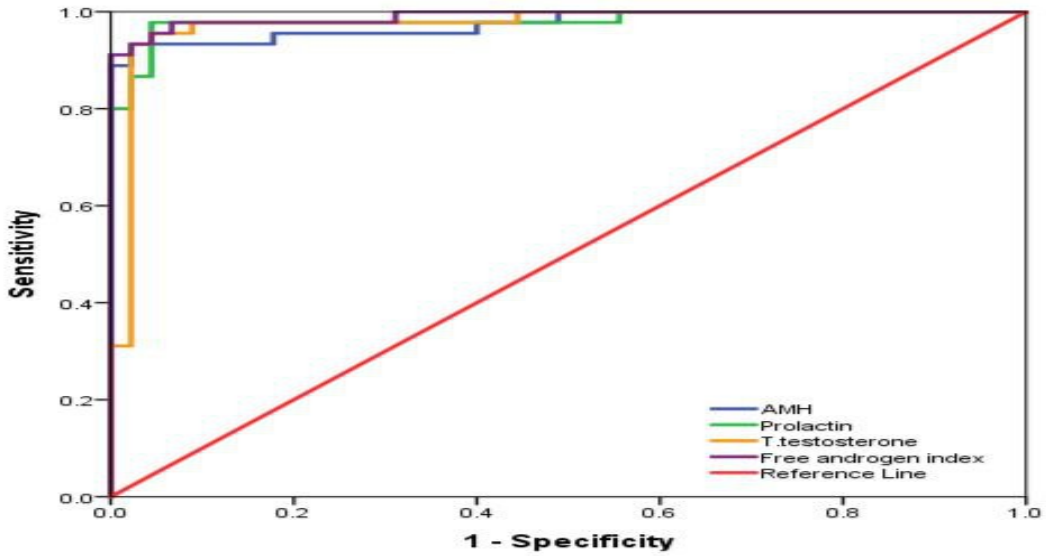

Figure 3. Receiver operation characteristic curve for the testosterone, prolactin, AMH parameter.

4. DISCUSSION

Polycystic ovarian syndrome endocrine disorder characterized by oligoanovulation, irregular menstrual cycle and high androgen levels, in our study found that dyslipidemia is one of the most common complications in PCOS patients that occurs due to decreased adiponectin, glucose intolerance, the nature of food and physical activities and Insulin resistance promotes lipolysis and alters lipase activity, resulting in increased triglyceride and decreased high-density lipoprotein (HDL). Chronic hyperinsulinemia may further contribute to increased hepatic production of very-low-density lipoprotein (VLDL), thereby raising circulating levels of triglycerides and total cholesterol. Moreover, androgen excess in polycystic ovary syndrome (PCOS) can worsen lipid abnormalities by disrupting hepatic lipid metabolism play an essential part in lipid profile in PCOS patient [14]. This agreement with a study [15].

In our study found that women with polycystic ovarian syndrome have a significant decrease in adiponectin levels that were negatively related to insulin resistance and obesity, compared to people without PCOS, those with PCOS exhibit reduced GLUT-4 and elevated levels of lipolysis, which limit insulin-mediated glucose absorption. IR is linked to decreased GLUT-4 expression in adipose tissue in people with or without PCOS [16] reduced expression of adiponectin may contribute to disruptions in energy homeostasis, impaired insulin action, and dysregulation of fertility hormones all of which are key mechanisms underlying the pathogenesis of PCOS, this underscores the critical role of adiponectin in the development of PCOS [17,18]. this agreement with the study [19] but in the study of Iranian women found no association between insulin and adiponectin with PCOS risk [20]. We found that a primary indicator of gonadotropin metabolism disorders in women have PCOS is an elevated level of luteinizing hormone, additionally, women with PCOS exhibit significantly higher total testosterone levels compared to healthy individuals, This finding is likely due to the abnormal frequency and amplitude of gonadotropin-releasing hormone (GnRH) secretion from the hypothalamus in PCOS, which stimulates the pituitary gland to secrete more LH, thereby enhancing androgen production. Furthermore, body mass index (BMI) and hormonal markers including prolactin, LH, and testosterone were found increased in women diagnosed with PCOS [21]. That agreement with [22]. We found elevated level of AMH due to the preantral and tiny antral follicles' enhanced production and release of AMH, AMH levels rise proportionately to the antral follicle count (AFC) at a steady 0.2 ng/ml for each follicle, Furthermore, granulosa cells in PCOS patients' follicles have been demonstrated to generate 75 times as much AMH as normal cells [23]. which agreement with the study [24].

Previous studies documented comparable results showing that Prolactin's sensitivity and specificity were (83.2%,88%) [25]. And Testosterone (92.9%, 95.6%) [26]. LH (93.2%; 86.3%: 95[27]. FSH sensitivity of 65.00% and specificity of 87.80% [28]. AMH had AUC (0.999) with (sensitivity and specificity 99%,100%) [25]. Adiponectin had specificity and sensitivity of 46.07%, 82.33% [29]. Another study had the specificity and sensitivity of adiponectin of 63.6%, 72.7% [30].

5. CONCLUSION

The importance of adiponectin levels and their correlation with hormonal and metabolic abnormalities in women with PCOS are highlighted by this study. In PCOS patients, decreased adiponectin levels were closely associated with abnormalities in reproductive hormones. These results open the door for further investigation into focused treatment approaches by highlighting the possible function of adiponectin as a biomarker for metabolic dysfunction in PCOS.

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