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Research Article

Investigating the Predictive Role of Leptin–Adiponectin Imbalance in Polycystic Ovary Syndrome Among Overweight Iraqi Females

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Abstract

The polycystic ovary syndrome (PCOS) chain reaction (PCC) is a hormonal enhancement factor closely associated with metabolic disorders. The adenokines leptin and adiponectin play a key role in regulating insulin activity. They also have a reproductive function through fat absorption, and the regulation of their transport may contribute to the development of PCC. This is an additional type of "lipid control," characterised by elevated leptin and adiponectin levels in the blood of 120 Iraqi men aged 18–44 years, including 60 adult men and 60 men over 60. Control groups of men and women were also included. Long menstrual cycles of 2–5 days were analysed, and biochemical markers were measured using spectroscopy. In patients with polycystic ovary syndrome (PCOS), leptin flow is significantly increased, and excess fat is transported via this pathway. A control group was also analysed (P<0.05). Enhanced leptin/adiponectin exhibits high diagnostic power, with an area under the curve (AUC) of 0.80 (80%) and a specificity score of 73% (0.03%). Leptin shows strong positive affinity for carotid intima-media thickness (IMT), triglycerides, and leptin protein (LPD), indicating a positive correlation with metabolic processes associated with diabetes. These results demonstrate that adiponectin's nutritional balance plays a role in metabolic and reproductive processes, as evidenced by its excellent function and superactive protein (SPC) activity. This enhanced leptin/adiponectin activity is an important biomarker for promoting health.

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1. INTRODUCTION

This field uses three criteria to diagnose polycystic ovary syndrome (PCOS), an endocrine disorder affecting women of early reproductive age: the 2006 AE-PCOS criteria, the 2003 Rotterdam (ROT) criteria, and the 1999 criteria established by the National Institutes of Health [1]. These criteria include elevated levels of male hormones such as testosterone, unwanted hair growth in areas like the face and chest, and irregular menstrual cycles (anovulation), leading to enlarged ovaries [2]. PCOS affects between 2% and 26% of the world's population [3, 4], with a significantly higher prevalence among obese women, estimated at 73%. PCOS is closely associated with obesity, characterized by a male body structure, a high waist-to-hip ratio, and fat accumulation in the anterior abdominal region [5]. Polycystic ovary syndrome (PCOS) results from a combination of environmental, genetic, and behavioral factors that interact inefficiently. The most common clinical symptom of PCOS is the secretion of androgens by the ovarian endothelial cells. Increased enzyme production in the steroid synthesis pathway leads to increased androgen secretion [6]. The combination of abdominal obesity and insulin resistance also leads to elevated blood androgen levels. Elevated insulin levels in obese women directly stimulate androgen production in cells, affecting fatty acid metabolism and fat accumulation in the liver, which in turn leads to decreased production of sex hormone-binding globulin (SHBG) [7], as illustrated in Figure 1.

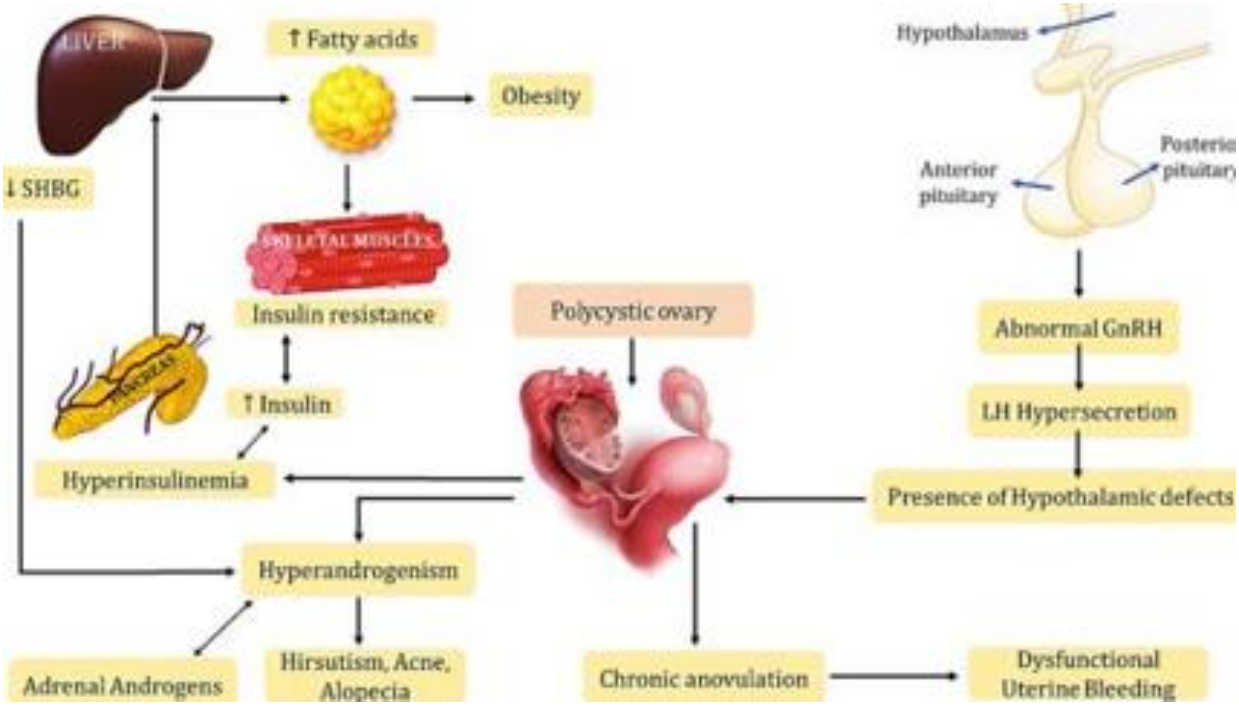

Figure 1: polycystic ovarian syndrome and obesity [8] Symbols: Luteinizing hormone (LH), gonadotropin-releasing hormone (GnRH), polycystic ovarian syndrome (PCOS), and sex hormone binding globulin (SHBG).

Adipokines, a type of adipokine, are chemoprecipitates that secrete a secretory lipid substance. Obesity is linked to leptin and adiponectin [9]. [10] Leptin, a hormone that regenerates body cells, controls neuronal function and maintains homeostasis. Its receptor is involved in music [11]. Leptin is one of the most potent metabolites of fat, secreting it into the body and transporting it to the body's aerobic system. [12] For vitality, it is a very easy-to-produce energy product, enhancing energy and physical activity. It is also involved in reproduction, as in the production of folic acid [13]. Leptin digestion has been more successful than other substances, predating Ogerini and SPC [14-16]. As described in the second section, insulin controls leptin concentration in all directions [17], allowing it to bind to its specific receptors and flow through the bloodstream. This ratio exists in living organisms [11].

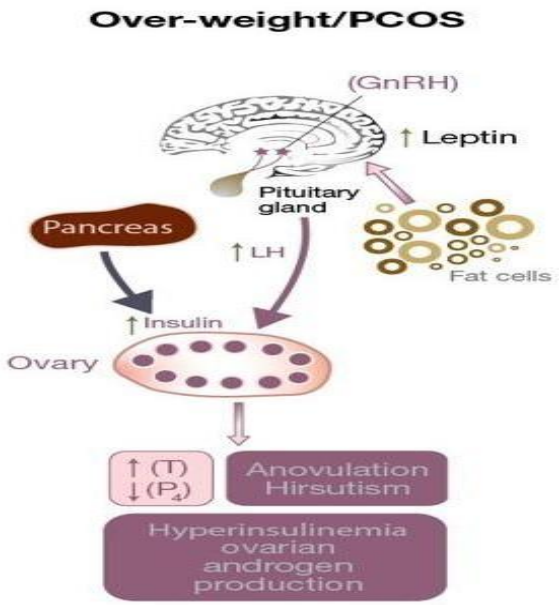

Figure 2: Effect of leptin on females with polycystic ovarian syndrome [11]

This study looked at blood leptin and adiponectin levels in females with PCOS and how they related to lipid profiles and anthropometric obesity indicators.

Study Design

This study was designed as a case-comparative study of overweight Iraqi women with polycystic ovary syndrome (PCOS) to examine leptin and adiponectin hormone levels and investigate the relationship between these hormones and blood lipid levels as variables contributing to the development of PCOS**.**

2. MATERIALS AND METHODS

A sample of 120 independent women, aged 18–44 years, with a body mass index (BMI) greater than 25, was recruited from private clinics in the Babil region of Iraq. The participants were divided into two groups:

1. Control group: 60 healthy women with a normal weight (BMI 20–25).
2. Patient group: 60 overweight women (BMI >25) with PCOS.

Blood samples were collected from the women between the second and fifth day of their menstrual cycle. Three millilitres of blood were placed in a normal test tube to prepare the serum, which was used in the tests after being separated and centrifuged at a speed of 3000 rpm for five to ten minutes.

Biochemical Analysis

Lipolipid levels (triglycerides, cholesterol, high-density lipoprotein, low-density lipoprotein, and very-low-density lipoprotein) were assessed using a 520 nm optical spectrometer, while adenostin and leptin levels were assessed using an ELISA (enzyme-linked immunosorbent assay) assay kit.

Calculation Results

Instead of focusing on the x-axis and plotting a best-fit curve passing through a point on the graph, as shown in Figures 3 and 4, the calibration curve was plotted by determining the optical intensity of each parameter on the y-axis.

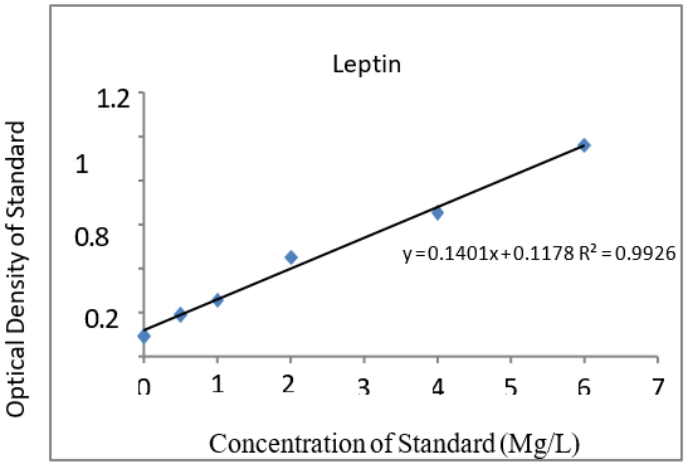

Figure 3: The standard curve for leptin concentration

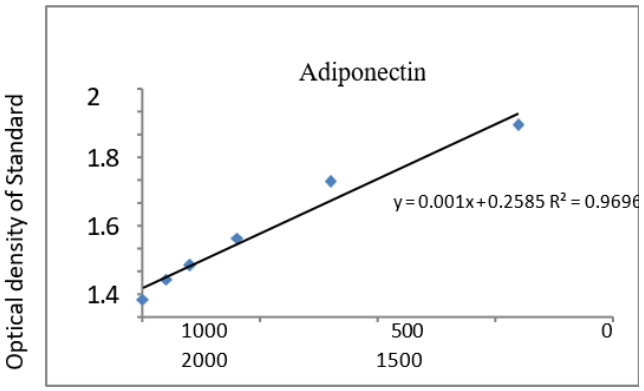

Figure 4: The standard curve for adiponectin concentration

Statistical Analysis

This study used the SPSS statistical software, version 23. All descriptive data were quantitative (measurement) variables, presented as mean ± standard error. Quantitative factors, such as body mass index and age, followed a statistically normal distribution. The studied parameters were compared between the study groups using Student's t-test for two groups. The correlation coefficient (r) was used to compare two quantitative variables, and a p-value of 0.05 or less was considered statistically significant.

3. RESULTS

Table 1 summarises the demographic information of the groups under study. The BMI was significant; the P-value for age between the patient and control groups was not.

Table 1: Demographic data of patient and control groups

Variable	Study Groups	No.	Mean ± SD	P-value
Age (years)	PCOS-Patient	60	25.13 ± 2.2	0.23
	Control	60	25.58 ± 1.8	
BMI (Kg/m²)	PCOS-Patient	60	27.59 ± 0.69	<0.00
	Control	60	25.68 ± 1.28	

The following criteria were used to assess polycystic ovary syndrome (PCOS): body mass index (BMI) and standard deviation. P-values greater than 0.05 were not considered statistically significant, while P-values less than 0.05 were. Table 2 shows the hormonal variables. When comparing patients and control groups, the P-values for leptin, adiponectin, and the leptin/adiponectin ratio were statistically significantly different.

Table 2: Hormonal Variables Between Patients and Control Groups

Variable	Study Groups	No.	Mean ± SD	P-value
Leptin (µg/L)	Patient	60	2.80 ± 0.5	0.025
	Control	60	2.53 ± 0.7	
Adiponectin (µg/L)	Patient	60	74.3 ± 14.5	<0.00
	Control	60	103.9 ± 15.0	
Leptin/Adiponectin Ratio	Patient	60	0.04 ± 0.01	<0.00
	Control	60	0.029 ± 0.01	

Standard deviation (SD), polycystic ovary syndrome (PCOS), and a p-value less than 0.05 were considered statistically significant, while a p-value greater than 0.05 was not. When comparing the patient groups to the control group, lipid levels were significantly different, as shown in Table 3.

Table 3: Lipid Level Data between Patient and Control Groups

Variable	Study Groups	No.	Mean ± SD	P-value
TG (mg/dl)	Patient	60	294.1 ± 20.3	<0.00
	Control	60	148.03 ± 14.8	
CHO (mg/dl)	Patient	60	224.8 ± 16.6	<0.00
	Control	60	166.8 ± 14.4	
HDL (mg/dl)	Patient	60	16.7 ± 2.0	<0.00
	Control	60	29.0 ± 3.5	
LDL (mg/dl)	Patient	60	149.2 ± 17.8	<0.00
	Control	60	108.2 ± 7.7	
VLDL (mg/dl)	Patient	60	58.8 ± 10.8	<0.00
	Control	60	29.6 ± 2.05	

Standard deviations (SD), triglycerides (TG), cholesterol (CHO), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL) were considered statistically significant when the p-value was less than 0.05. Very-low-density lipoprotein (VLDL) was also considered statistically significant. Furthermore, as shown in Table 4, the current study found positive correlations between body mass index (BMI) and leptin levels in overweight individuals ($p < 0.001$, r = 0.83). In contrast, Table 4 shows a positive correlation between leptin and the leptin/adiponectin ratio in patients ($P < 0.001$, r = 0.051), and a positive correlation between leptin and triglycerides and very low-density lipoprotein (VLDL) cholesterol (P < 0.001, r = 0.60 and 0.631, respectively). Although the distribution of leptin and adiponectin levels directly influences reproductive hormone regulation and contributes to the symptoms of polycystic ovary syndrome (PCOS), leptin levels increase with increasing body mass index (BMI), leading to a higher leptin-to-adiponectin ratio in women with PCOS.

Table 4: Relationship between variables in patients

Variable		Leptin	Lep/ADP Ratio	TG	VLDL
BMI	r	.843**	.213	.613**	.603**
	Sig.	.000	.103	.000	.000
Leptin	r	1	.511**	.601**	.631**
	Sig.		.000	.000	.000
Lep/ADP Ratio	r	.511**	1	.370**	.370**
	Sig.	.000		.004	.004
TG	r	.601**	.370**	1	1.000**
	Sig.	.000	.004		.000
VLDL	r	.631**	.370**	1.000**	1
	Sig.	.000	.004	.000	

Indicators of correlation at the 0.01 level include triglycerides, very low-density lipoprotein (VLDL) cholesterol, leptin-to-adenonectin ratio, and Pearson correlation coefficient. At a cutoff value of 0.03, the leptin-to-adenonectin ratio showed good predictive power for polycystic ovary syndrome (PCOS) in overweight women compared to women of normal weight.

This suggests that it may be a better tool for confirming a PCOS diagnosis, as illustrated in Figure 5.

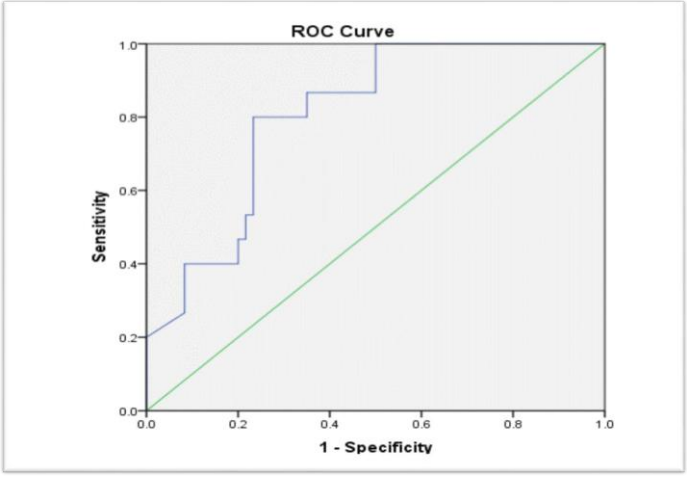

Figure 5: Leptin/adiponectin ratio receiver operating characteristic (ROC) curve for PCOS patients under management; AUC stands for area under the curve.

4. DISCUSSION

To avoid discrepancies in the results of parameters that might arise from a large age difference, age-matched testing was used in this study between the patient and control groups. Body mass index (BMI) is considered an anthropometric indicator in the assessment of obesity [18]. Polycystic ovary syndrome (PCOS) is closely associated with obesity, increasing leptin levels and decreasing adiponectin levels compared to the control group. Therefore, obese women have higher levels of leptin synthase in their adipose tissue compared to women of normal weight. As in some studies [19], [20-22], our study shows a positive correlation between leptin and BMI in women with PCOS. The results of this study show that all plasma lipid levels were significantly higher in the PCOS group compared to the age-matched control group. There is a strong correlation between leptin and triglycerides and very low-density lipoprotein (VLDL), according to several studies [23]. Similar studies have shown that dyslipidemia, common in women with polycystic ovary syndrome (PCOS), is characterized by elevated levels of very-low-density lipoprotein (VLDL-C), low levels of high-density lipoprotein (HDL-C), and elevated triglyceride levels [24, 25]. Abdominal adipose tissue increases due to elevated androgen levels produced by the ovarian endothelial cells; subcutaneous tissues become insulin resistant because insulin inhibits glucose uptake by protein kinase C [26]. Effects of leptin in women with PCOS: Elevated leptin levels lead to:

1. Leptin is produced and stored by granulosa cells. High leptin levels inhibit aromatase expression, affecting the dominant follicle's ability to produce sufficient estrogen, resulting in androgen accumulation and conversion to estrogen [27, 28].
2. Absence of follicular development is associated with elevated leptin levels [29].
3. High levels of adenoctin lead to inadequate gluconeogenesis and free fatty acid uptake [30]. In addition, they affect ovulation, progesterone and estrogen production, and decrease pituitary secretion of GnRH and LH [31].

5. CONCLUSION

This study demonstrates that obese Iraqi women with polycystic ovary syndrome (PCOS) exhibit marked changes in adenokinase levels, characterised by elevated leptin and significantly reduced adenoctin concentrations. The strong correlation between leptin and body mass index (BMI), triglycerides, and very low-density lipoprotein cholesterol (VLDL-C) underscores its pivotal role in the metabolic dysfunction associated with PCOS. Furthermore, the leptin/adenonectin ratio demonstrated high diagnostic value, suggesting its potential as an additional biomarker for detecting PCOS in obese women. Given the marked lipid disturbances in patients with this condition, periodic assessment of lipid levels, particularly triglycerides and VLDL-C, is recommended to mitigate the long-term risks of cardiovascular and metabolic diseases. In general, the results obtained confirm the role of adenokinase imbalance in the development and progression of polycystic ovary syndrome, and emphasise the importance of monitoring metabolic indicators alongside hormonal indicators in clinical evaluation.

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