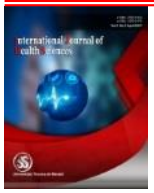

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The Association Between Serum Leptin Levels and Body Mass Index in Polycystic Ovarian Syndrome Patients

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Body Mass Index;
Hyperandrogenism;
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Abstract

Polycystic ovary syndrome (PCOS) is the most common endocrine abnormality in reproductive-age women, which is characterized by hyperandrogenism, anovulation, and polycystic ovaries. Some evidence suggested that leptin also causes PCOS due to its role in the female reproductive system. The physiological function of leptin controls the balance of energy and suppresses the center of appetite. Patients with PCOS may be underweight, normoweight, overweight, and obese, based on their body mass index (BMI), but obesity is a common clinical situation in PCOS. Obesity occurs when the level of leptin increases but cannot decrease appetite, resulting in leptin resistance. This study aimed to discover the connection between body mass index (BMI) and the level of leptin in patients with PCOS. Methods: A case-control sectional study included 100 women, divided between 50 women with polycystic ovary syndrome and 50 healthy women. They're aged from 18 to 40 years old. Samples were collected from Al-Batoul General Hospital and private clinics in Diyala Governorate during the period from December 2024 to end March 2025. Blood samples were collected under fasting conditions from every enrolled participant. Results: The results include several biomarkers; each biomarker (means ± SD) was calculated in two groups. Women with PCOS demonstrated significantly higher levels of BMI, FBG, leptin, LH, FSH, testosterone, TG, TC, and LDL ($p < 0.001$ for all), while their estradiol and HDL levels were significantly lower than those of the control group ($p < 0.001$). No statistically significant difference was observed in age ($p = 0.682$) and height ($p = 0.316$). Leptin exhibited strong positive correlations with BMI ($r = 0.86$) with high statistical significance ($p < 0.001$). Conclusion: Serum leptin levels in PCOS patients were higher than in control individuals. Additionally, observed positive correlation was observed between leptin and BMI. These findings underscore the significance of weight management in decreasing BMI and addressing PCOS. Further research is warranted to explore the role of leptin in the pathogenesis of PCOS.

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Contents

1 Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine system disorder among women of reproductive age, with a prevalence of 6–25% (Rotterdam et al., 2004). It is a heterogeneous disorder characterized by menstrual dysfunction, infertility, increased androgen levels, polycystic ovaries, hirsutism, and/or alopecia (Narayanaswamy et al., 2023). Moreover, PCOS is linked to a higher occurrence of factors contributing to cardiovascular disease (CVD), diabetes mellitus, dyslipidemia, and hypertension (Auda et al., 2023; Veena et al., 2019). Hyperandrogenism is considered a significant factor in the development of PCOS, although insulin resistance and obesity contribute to both direct and indirect mechanisms involved in the excessive production of androgens in PCOS. Furthermore, the present pandemic of obesity predicts a higher occurrence of PCOS in the future (Frederiksen et al., 2013). A recent meta-analysis found that the overall prevalence of hyperhomocysteinemia (HHcy) in Chinese women was 28% and was on an increasing trend. Additionally, there was a correlation between elevated homocysteine (Hcy) levels and obesity, insulin resistance, and elevated androgen levels. Elevated homocysteine levels are associated with insulin resistance and may exacerbate hyperandrogenism, which is a major feature of polycystic ovary syndrome (PCOS) (Abass & Hussein, 2025). Obesity is common in more than half of women with PCO. The risks of cardiovascular disease and diabetes mellitus are increased by central fat distribution, or android obesity (Rotterdam et al., 2004). In addition to the peripheral tissue involvement caused by obesity, women with PCOS also had an increase in intra-abdominal fat, which is unrelated to obesity (Carmina et al., 2007). Adiponectin, leptin, and resistin are among the bioactive cytokines and adipokines secreted by fat tissue (Manneråås-Holm et al., 2011). According to some data, the emergence of obesity-related disorders, such as PCOS, may be linked to dysregulated leptin expression (Lecke SB et al., 2013; Lecke SB et al., 2013). Leptin is a polypeptide hormone, the first fat cell-derived hormone (adipokine) produced and secreted by white adipose tissue, which is crucial for some metabolic functions and transmits metabolic signals to the brain nerve tissue to modulate the hypothalamus-pituitary-ovary. It affects the reproductive systems in a variety of ways and plays a significant role in maintaining energy balance (Murri M et al., 2013). Leptin resistance is associated with obesity and persistent hyperphagia, resulting in hyperinsulinemia that will then lead to insulin resistance. Hyperinsulinemia decreases the level of sex hormone-binding globulin (SHBG) in the liver, leading to an increased androgen level in the ovary. Hyperinsulinemia, together with an increase in luteinizing hormone (LH), can increase the androgen level in the ovary that will trigger hyperandrogenemia as a symptom of PCOS (Chen et al., 2015; Rizk et al., 2015). Hyperleptinemia will affect the receptors in the hypothalamus, causing changes in gonadotropin-releasing hormone (GnRH) secretion. An increase in GnRH level may disrupt LH secretion, which then causes an increase in ovarian androgen level (Yau et al., 2017; Huang-Doran & Franks, 2016). Increased androgens may cause hyperandrogenemia that inhibits follicular development and causes anovulation, infertility, and polycystic ovaries, which comprise the symptoms of PCOS (Rojas et al., 2014) as shown in Figure 1.

This study aimed to discover the connection between body mass index (BMI) and the level of leptin in patients with PCOS.

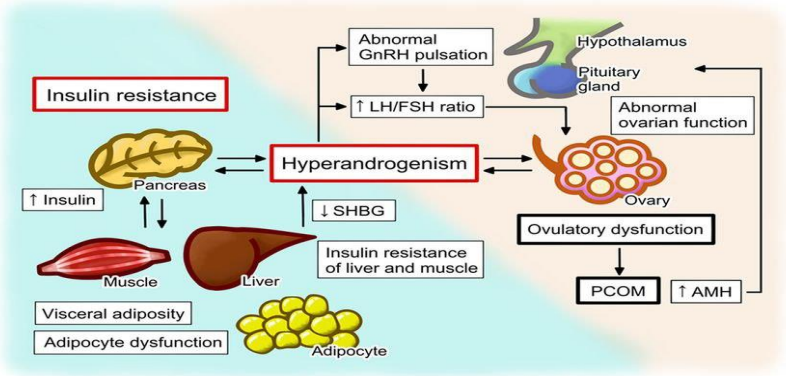

Figure 1: Pathophysiology of Polycystic Ovary Syndrome and Obesity

Symbol: LH stands for luteinizing hormone, FSH for follicle–stimulating hormone, GnRH for gonadotropin-releasing hormone, PCOS for polycystic ovarian syndrome, and SHBG for sex hormone binding globulin, AMH for anti-Mullerian hormone.

2 Materials and Methods

Sample Collection

A case-control sectional study included 100 women, distributed between 50 women with polycystic ovary syndrome and 50 healthy women. They are aged from 18 to 40 years old. Samples were collected from Al-Batoul General Hospital and private clinics in Diyala Governorate during the period from December 2024 to the end of March 2025. Patients with PCOS are diagnosed based on Rotterdam consensus criteria, the presence of at least two of the following features: (1) irregular menstrual periods with more than a 35-day delay between cycles; (2) biochemical and/or clinical evidence of hyperandrogenemia; and (3) polycystic ovaries in USG. While healthy women had regular menstrual cycles, normally-sized ovaries, and no sign of hyperandrogenism.

Exclusion criteria

Exclusion criteria from this study comprised individuals with hyperprolactinemia, thyroid disorders, adrenal disorders, hypothalamic disorders, and Participants who were currently using hormone supplements such as estrogen, progesterone.

Blood Sampling

Blood samples were collected under fasting conditions from every enrolled participant. Using a medical syringe, ten milliliters of blood were withdrawn from the cubital vein. The samples were retained in a special Gel tube and left for ten to fifteen minutes to extract the serum. The samples were centrifuged for 15 minutes at 4000 rpm for one minute. The serum was then divided between small Eppendorf tubes, and in order to avoid repeated thawing, duplicates of each sample were kept. Until the analysis was carried out, the samples were stored in a storage room at-20 degrees Celsius. Total cholesterol, TG, LDL, HDL, and FBG were determined via spectrophotometric analysis and shown in mg/dL. The concentrations of many hormones, including total testosterone, follicle-stimulating hormone (FSH), luteinizing hormone (LH), Estradiol (E2), and Leptin, were measured using the Cobas E-411 chemical analysis device, which works according to the principle of photoelectrochemical immunoassay.

Statistical analysis

All statistical evaluations were carried out using **IBM SPSS Statistics (version 27.0)**, with a threshold for statistical significance set at $p < 0.05$. A combination of descriptive, comparative, correlational, and effect size analyses was used to explore the relationships between metabolic and hormonal variables in individuals with and without Polycystic Ovary Syndrome (PCOS).

3 Results and Discussions

3.1 Results

Descriptive statistics were employed to summarize the dataset. Continuous variables such as age, body mass index (BMI), fasting blood glucose (FBG), lipid profile (TC, TG, HDL, and LDL), hormone levels (LH, FSH, testosterone, and estradiol), and leptin were expressed as **means ± standard deviation (SD)**. In contrast, categorical variables—such as BMI classification (normal weight vs. overweight)—were presented using **frequencies and percentages** for a clearer understanding. **Group comparisons** were conducted to assess significant differences between the PCOS and control groups. **Independent samples *t*-tests** revealed notable disparities across several continuous variables. Women with PCOS demonstrated significantly higher levels of BMI, FBG, leptin, LH, FSH, testosterone, TG, TC, and LDL ($p < 0.001$ for all), while their estradiol and HDL levels were significantly lower than those of the control group ($p < 0.001$). No statistically significant difference was observed in age ($p = 0.682$) and height ($p = 0.316$), suggesting age and height homogeneity between the two cohorts.

Table 1 displays characteristics (age, height, weight, BMI, fasting blood glucose, leptin, LH, FSH, testosterone, estradiol, TC, TG, HDL, LDL) for both the control and PCOS groups, including p-values indicating statistical differences.

Table 1
Participant characteristics for all parameters in two groups

Variable	Control Group (n=50)	PCOS Group (n=50)	p-value
Age (years)	28.4 ± 6.2	27.9 ± 6	0.682
Height (cm)	166.1 ± 6.8	167.5 ± 7.1	0.316
Weight (kg)	61.2 ± 6.3	72.8 ± 10.5	<0.000
BMI (kg/m^2)	22.1 ± 0.9	26.4 ± 1.3	<0.001
FBG (mg/dl)	86.3 ± 5.4	99.1 ± 9.0	<0.001
Leptin (ng/ml)	13.1 ± 4.6	24.1 ± 6.2	<0.001
LH (IU/L)	5.2 ± 0.5	10.2 ± 1.2	<0.000
FSH (IU/L)	6.2 ± 0.5	4.2 ± 0.8	<0.000
Testosterone (ng/dl)	31.5 ± 3.7	60.2 ± 14.8	<0.001
Estradiol (pg/ml)	103.2 ± 8.6	63.8 ± 10.1	<0.001
TG (mg/dl)	81.5 ± 8.2	185.3 ± 15.7	<0.000
TC (mg/dl)	178.5 ± 10.2	222.6 ± 18.3	<0.000
HDL (mg/dl)	65.4 ± 5.5	39.2 ± 5.8	<0.001
LDL (mg/dl)	100.8 ± 8.1	138.6 ± 14.5	<0.001

Table 2 presents the distribution of participants in the control and PCOS groups based on their BMI classification (normal weight or overweight). All control participants were of normal weight, while all PCOS participants were overweight. To evaluate the distribution of BMI classifications across the groups, a Chi-square test was applied. The results indicated a complete divergence in BMI categories ($p < 0.001$): 100% of the control group fell within the normal BMI range (18.5–24.9 kg/m^2), while 100% of the PCOS group were classified as overweight (25–29.9 kg/m^2). This categorical distinction underscores the direct link between PCOS and altered body weight profiles.

Table 2
Distribution of individuals for the study depending on BMI

Group	Normal (18.5-24.9)	Overweight (25-29.9)	χ^2 p-value
Control	50 (100%)	0 (0%)	<0.001
PCOS	0 (0%)	50 (100%)	

For assessing linear associations between serum leptin levels and various metabolic and hormonal parameters, Pearson's correlation coefficients were calculated. Leptin exhibited strong positive correlations with BMI (r = 0.86), body weight (r = 0.84), and LDL (r = 0.73) and strong negative correlations with HDL (r = −0.75) with high statistical significance (p < 0.001). Additionally, leptin demonstrated moderate correlations with both FBG (r = 0.63), triglycerides (r = 0.71), LH (r = 0.53), TC (r = 0.69), and testosterone (r = 0.64), again statistically significant (p < 0.001). Conversely, no meaningful correlations were observed with age (r = −0.10, p = 0.32) or height (r = 0.07, p = 0.48), estradiol (r = 0.02, p = 0.89), or FSH (r =- 0.24, p = 0.09), suggesting leptin's influence is more closely tied to metabolic rather than demographic factors. Table 3 presents the Pearson correlation coefficients (r-values) and corresponding p-values between leptin and various variables in the study population.

Table 3
Correlation of serum leptin levels with all parameters

Variable	Leptin (ng/ml)	
	r-value	p-value
Age (years)	-0.10	0.32
Height (cm)	0.07	0.48
Weight (kg)	0.84	<0.001
BMI (kg/m^2)	0.86	<0.001
FBG (mg/dl)	0.63	<0.001
LH (IU/L)	0.53	<0.001
FSH (IU/L)	-0.24	0.09
Testosterone (ng/dl)	0.64	<0.001
Estradiol (pg/ml)	0.02	0.89
TG (mg/dl)	0.71	<0.001
TC (mg/dl)	0.69	<0.001
HDL (mg/dl)	-0.75	<0.001
LDL (mg/dl)	0.73	<0.001

Effect size analysis was conducted using fold change calculations to quantify the magnitude of hormonal differences between the PCOS and control groups. The results indicated that testosterone levels were 2.3 times higher in the PCOS group (55–90 ng/dl) compared to controls (25–39 ng/dl). Similarly, luteinizing hormone (LH) levels were 2.1 times higher in PCOS subjects (9.0–11.9 IU/L versus 4.5–5.9 IU/L in controls). In contrast, estradiol levels were 1.7 times lower in women with PCOS (50–70 pg/ml) than in the control group (90–120 pg/ml), emphasizing the hormonal dysregulation characteristic of PCOS.

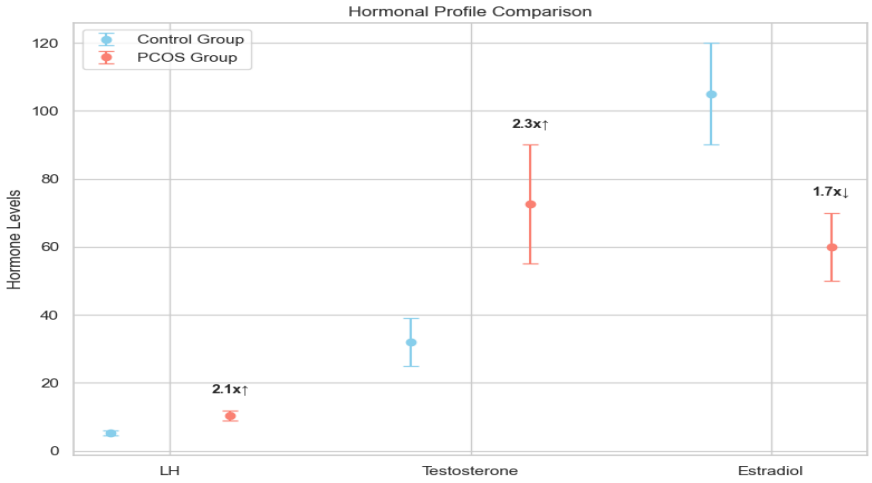

Figure 2. Hormonal Profile Comparison

3.2 Discussions

Polycystic Ovarian Syndrome (PCOS), the common dysovulatory infertility, is characterized by chronic anovulation and hyperandrogenemia (Azziz et al., 2009). Obesity has a strong association with PCOS, which leads to increased leptin levels compared to controls. Leptin synthases in adipose tissue increase in obese females more than in normal-weight individuals (Jahromi et al., 2017). In the present study, the possibility of a relationship between leptin and BMI in women with PCOS was investigated. Our results indicate that serum leptin is significantly higher in PCOS women (24.1 ± 6.2) compared with controls (13.1 ± 4.6), with *a p*-value (<0.001). This finding is comparable with the study of Mohiti-Ardekani and Taarof, which showed elevated levels of serum leptin in 27 Iranian women with PCOS (Mohiti-Ardekani & Taarof, 2010), and agrees with a meta-analysis reported in 2016, which included a total of 19 studies, observed that leptin levels are higher (statistically significant) for women with PCOS as compared to controls (Seth et al., 2021). A high level of leptin plays a role in the pathogenesis of hyperandrogenism in PCOS. Jalilian et al. revealed that there is an association between elevated serum leptin levels and BMI in PCOS (Jalilian et al., 2016). Leptin resistance and lifestyle (uncontrolled diet and exercise) can cause Leptin levels to increase in PCOS patients who have excessive BMI (overweight and obese). Leptin resistance is caused by impaired leptin signal transduction on neurons in the hypothalamus (Allahbadia & Merchant, 2011). These leptin signals are proteins of suppressors of cytokine signaling 3 (SOCS3), protein tyrosine phosphatase 1B (PTP1B), and leptin receptor of signal transducers and activator of transcription 3 (STAT3). They are molecules known to weaken leptin signaling. The high leptin level due to leptin resistance interacts with the hypothalamic axis, hence causing reproductive dysfunction (Amitani et al., 2013). Leptin resistance plays a role in steroidogenesis in ovarian granulosa cells, which contributes to increased androgens in ovarian theca cells (Chakrabarti et al., 2013; Zieba et al., 2005). Leptin levels are high but ineffective in leptin resistance. The BMI stays higher than average. Excessive BMI can be influenced by uncontrolled lifestyle factors, such as irregular exercise and eating habits. Our findings in the present study strongly support these observations, indicating that serum leptin levels exhibited a strong positive correlation with BMI (r =0.86) with high statistical significance ($p < 0.001$) in PCOS patients, as well as agreeing with other studies (El-Gharib et al., 2014; Jalilian et al., 2016) it was This finding is obviously anticipated. Since adipocytes are the primary source of leptin synthesis, a higher BMI causes more fatty tissue and an increase in the production of leptin. The findings of this present research demonstrate that serum lipid (TG, TC, LDL) levels were significantly higher in the PCOS patients (185.3 ± 15.7, 222.6 ± 18.3, 138.6 ± 14.5) than in control (81.5 ± 8.2, 178.5 ± 10.2, 100.8 ± 8.1) with ($p < 0.001$), while the levels of serum HDL is low in PCOS (39.2 ± 5.8) comparison with control (65.4 ± 5.5) this agree with other studies (Wild et al., 2011; Abdulzahra et al., 2023) shows that dyslipidemia, which is common in patients with PCOS, it is characterized by elevated triglycerides and LDL and low in the levels of HDL. In the present study, show that LH, testosterone level were higher in PCOS patients (10.2 ± 1.2), (71.2 ± 14.8) than in controls (5.2 ± 0.5),(31.5 ± 3.7), while the levels of Estradiol and FSH were low in PCOS patients (63.8 ± 10.1), (4.2 ± 0.8) comparison

with control (103.2 ± 8.6), (6.2 ± 0.5), this can also be seen in the fig 2 where testosterone and LH levels are elevated and FSH levels are decreased in PCOS patients compared to the normal levels in healthy individuals. High levels of LH support increasing levels of androgens along with low levels of FSH secretion, compared to the control group (Al-Musawi et al., 2022).

4 Conclusion

In conclusion, Serum leptin levels in PCOS patients were higher than in control individuals. Additionally, a positive correlation was observed between leptin and BMI. These findings underscore the significance of weight management in decreasing BMI and addressing PCOS. Further research is warranted to explore the role of leptin in the pathogenesis of PCOS.

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