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Monitoring of leptin hormone levels in child bearing – age women in Baghdad / Iraq – with polycystic ovary syndrome, and the impact of BMI on leptin levels

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Abstract

At Poly-cystic ovarian syndrome (PCOS) pathway and duration, there are Hormonic elevations in Androgen and testosterone. Since PCOS is a metabolic disorder, the present study is designed to monitor Leptin levels at PCOS, being there is a common cause between metabolic disorder and PCOS.

All potential biological factors that affect Leptin levels were isolated, in order to monitor exclusively BMI effect on serum Leptin levels at PCOS. The result illustrated that serum leptin level is not associated with the duration of PCOS in proportion to non-significant statistics of the present results which showed between leptin and PCOS duration, hence leptin hormone couldn't be a biomarker for PCOS investigation or prediction. MI are associated statistically with PCOS duration according to A two-tailed t-test, whereas BMI doesn't influence serum blood Leptin levels.

Keywords: Leptin, polycystic ovary syndrome, PCOS, BMI, metabolic disorder.

مراقبة مستويات هرمون اللبتين لدى النساء في سن الانجاب المصابات بمتلازمة تكيس المبايض في بغداد – العراق , وتأثير مؤشر كتلة الجسم على مستويات اللبتين

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الخلاصة

في مسار و فترة متلازمة تكيس المبايض (PCOS) ، هناك ارتفاعات هرمونية في الأندروجين والتستوستيرون. و نظرًا لأن متلازمة تكيس المبايض لها علاقة بالاضطراب الاستقلابي ، فقد تم تصميم الدراسة الحالية لمراقبة مستويات اللبتين في متلازمة تكيس المبايض ، نظرًا لوجود سبب مشترك بين اضطراب التمثيل الغذائي ومتلازمة تكيس المبايض.

تم عزل جميع العوامل البيولوجية المحتملة التي تؤثر على مستويات اللبتين ، من أجل مراقبة تأثير مؤشر كتلة الجسم بصورة حصرية على مستويات مصل اللبتين في متلازمة تكيس المبايض.

أوضحت النتائج الحالية أن مستوى اللبتين في الدم لا يرتبط بمتلازمة تكيس المبايض حسب الحسابات الإحصائية والتي اظهرت نتيجة غير مجدية سريريا ما بين مستويات مصل اللبتين ومتلازمة تكيس المبايض ، وبالتالي لا يمكن أن يكون قياس هرمون اللبتين مؤشراً بيولوجياً للتنبؤ او تشخيص متلازمة تكيس المبايض.كما اظهرت النتائج ارتباط مؤشر كتلة الجسم (BMI) إحصائيًا مع متلازمة تكيس المبايض وفقًا لاختبار تي ثنائي الذيل ، بينما لا يؤثر هذا مؤشر كتلة الجسم على مستويات اللبتين في الدم. الكلمات المفتاحية: اللبتين ، متلازمة تكيس المبايض ، متلازمة تكيس المبايض ، مؤشر كتلة الجسم ، اضطراب التمثيل الغذائي.

Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder with a high prevalence of 6–20% of reproductive-aged women. Typically, PCOS is first identified during the early reproductive years. The clinical expression varies but commonly includes oligo-ovulation or anovulation, hyperandrogenism, and the presence of polycystic ovaries. The aetiology of the syndrome is obscure yet, and the variability in phenotype expression continues to render clinical care and research concerning this heterogeneous condition challenging. [1, 2].

Leptin, a peptide hormone secreted by adipose tissue, is essential for controlling hunger and energy use. This hormone, which has a considerable effect on ovarian function, is also involved in the regulation of reproduction, insulin action, and fat metabolism. Leptin levels are higher in PCOS patients, according to recent studies. It might be associated with both obesity and glucose tolerance decrease. [3,4] Multiple human studies have examined the hypothesis that leptin contributes to the emergence of PCOS. Mixed findings have emerged from PCOS leptin research, with some studies reporting elevated leptin levels, (10) while others found no difference in leptin levels between PCOS patients and healthy women [5,6]. Hence, we design the present study performed on individuals with no diabetes or cardiovascular disorders to prevent multi- effect on leptin levels.

Material and Method

50 serum blood specimens of Iraqi bearing-age women with PCOS have been collected from outpatients in Baghdad-Iraq, alongside 25 healthy individuals of beg-age women involved as control. All the individuals of the two groups had no diabetes or cardiovascular disorders to prevent the evident effect of these diseases on Leptin levels as documented in previous studies that have the same object of the present study. Also, BMI of the individuals of the two studied groups is obtained by direct questioning during sample collecting. Leptin levels have been measured by the ELISA technique. Blood samples were collected at random times. The data of the obtained results are analyzed statistically by SPSS 25 for windows.

Results and Discussion

From obtained results, the number of women with polycystic ovary syndrome (PCOS) represented as a disease group of 50 patients as compared to a group of 25 women with no PCOS as a healthy control group, as shown in Table 1:

-		0 1
	Groups	Frequency
	Women with PCOS	50
	Women without PCOS	25
	Total	75

Table (1): frequency of women as disease and control groups.

The results of the descriptive statistics show that women with PCOS have higher values for the dependent variable BMI (Kg/m2) (M = 27.16, SD = 4.68) than the women with no PCOS (M = 23.51, SD = 2.4). A two-tailed t-test for independent samples showed that the difference between the two groups with respect to a dependent variable BMI (Kg/m2) was statistically significant, t (40) = 2.56, p = .014 as shown in table 2 and figure 1. This agrees with many previous studies that showed the same result [9].

Table (2): The mean BMI values of the two studied groups.

		Ν	Mean	SD	t (df)	P – Value
BMI (kg/m2)	Women w PCOS	5 0	2 7.16	2 56	4 0	014
	Women W no PCOS	2 5	2 3.51	2 4		

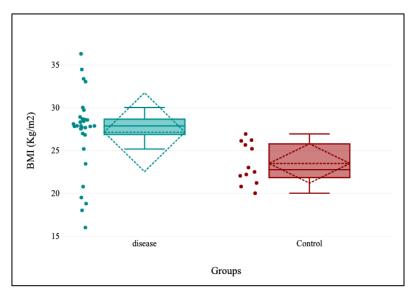


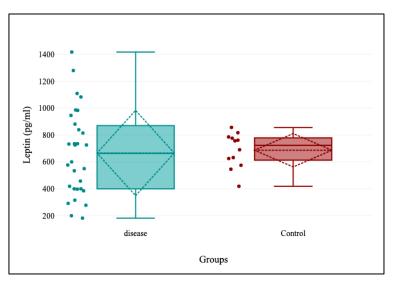
Fig. (1): SD \pm mean of BMI for the two studied groups.

Women with PCOS has slightly lower values for the dependent variable Leptin (pg/ml) (M = 666.14, SD = 22.59) than the Control group (M = 686.87, SD = 12.75). A two-tailed t-test for independent samples showed that the difference between disease and Control with respect to the dependent variable Leptin (pg/ml) was not statistically significant, t (39.97) = -0.3, p = .768, hence this result illustrated that Leptin is n not related to the pathology of PCOS.

Insulin resistance affects Leptin levels [10], the present study disagrees with the previous studies that didn't consider the Insulin resistance influence and cardiovascular disorders [11] on Leptin levels.

Table (3): The mean of leptin levels in each of the two studied groups. The two studied groups.

		Ν	Mean	SD	t(df)	P- value
Leptin (pg/ml)	Women w PCOS	50	666.14	22.59	-0.3(39.9)	0.768
	Women w no PCOS	25	686.87	12.75		



have no Insulin resistance and no cardiovascular disorder.

Fig. (2): SD means of Leptin concentration in the two studied groups.

 Table (4): Obesity levels in the individuals of the two groups.

		Gro		
		Women w PCOS	Women w no PCOS	Total
	Moderate overweight	22	2	24
	normal weight	9	9	18
ity	underweight	7	2	9
obesity	Obese	11	3	14
0	slight overweight	7	7	14
	sever obese	6	2	8
	Total	50	25	75

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A Chi2 test was performed between Groups and obesity. There was a statistically significant relationship between Groups and obesity, $\chi^2(5) = 22.53$, p = <.001

Chi ²	22.53
Df	5
Р	<.001

A Pearson correlation was performed to test whether there was an association between BMI (Kg/m2) and Leptin (pg/ml). The result of the Pearson correlation showed that there was no significant association between BMI (Kg/m2) and Leptin (pg/ml), r (40) = 0.05, p = .774. This result unlike previous studies [12] and this suggest that Leptin secretion didn't affect by adipose tissue mass or BMI values, it may be controlled exclusively by the hypothalamus.

 Table (5): Correlation between BMI and Leptin.

	r	P-value
BMI (Kg/m2) and Leptin	0.05	.774
(pg/ml)		

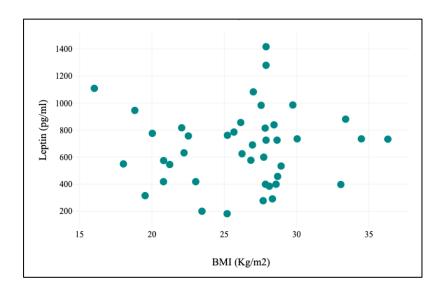


Fig. (3): scatter diagram between Leptin and BMI.

Reference

- 1. Meier, R.K. (2018) Polycystic ovary syndrome. Nursing Clin. N. Am. 53, 407–420.
- **2.** Teede, H.J. et al. (2018) Recommendations from the international evidence-based guideline for assessing and managing polycystic ovary syndrome. Hum. Reprod. (Oxford, England) 33, 1602–1618.
- **3.** Pehlivanov B, Mitkov M. Serum leptin levels correlate with clinical and biochemical indices of insulin resistance in women with polycystic ovary syndrome. Eur J Contracept Reprod Health Care. 2009;14(2):153-159.
- **4.** Chakrabarti J. Serum leptin level in women with polycystic ovary syndrome: correlation with adiposity, insulin, and circulating testosterone. Ann Med Health Sci Res. 2013;3(2):191-196.
- **5.** Calvar CE, Intebi AD, Bengolea SV, Hermes R, Spinedi E. Leptin in patients with polycystic ovary syndrome. Direct correlation with insulin resistance [In Spanish]. Medicine (B Aires). 2003; 63(6):704-710.
- **6.** Bideci A, Camurdan MO, Yesilkaya E, Demirel F, Cinaz P. Serum ghrelin, leptin and resistin levels in adolescent girls with polycystic ovary syndrome. J Obstet Gynaecol Res. 2008;34(4): 578-584.
- 7. Bell BB, Rahmouni K. Leptin as a Mediator of Obesity-Induced Hypertension. *Curr Obes Rep* 2016; 5: 397–404.
- **8.** Pérez-Pérez A, Vilariño-García T, Fernández-Riejos P, Martín-González J, Segura-Egea JJ, Sánchez-Margalet V. Role of leptin as a link between metabolism and the immune system. Cytokine Growth Factor Rev 2017; 35: 71–84.
- **9.** Polycystic ovary syndrome patients with high BMI tend to have functional disorders of androgen excess: a prospective study.
- **10.** (Relationship between serum leptin and insulin resistance among obese Nigerian women, 2016).
- **11.** Kim SH, Despres JP, Koh KK. Obesity and cardiovascular disease: friend or foe? *Eur Heart J.* (2016) 37:3560–8. 10.1093/eurheartj/ehv509.
- Considine RV, Sinha MK, Heiman ML, Kriauciunas A, Stephens TW, Nyce MR, et al. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. *N Engl J Med.* (1996) 334:292–5. 10.1056/NEJM199602013340503.