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ASSESSMENT OF SERUM IRISIN IN OVERWEIGHT AND OBESE PATIENT WITH POLYCYSTIC OVARY SYNDROME

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ABSTRACT

Background and Objective: Polycystic ovary syndrome (PCOS) is the most common endocrine disorder affecting 10–15 % of women in reproductive age. The objective was to evaluate the effect of irisin in the etiology of obese patient with PCOS.

Study Design and Methods: The study design its a case control study. This study involved fifty obese ladies (50) with confirmed diagnosis of PCOS. The second group served as control group and included forty apparently normal (non-polycystic ovary) obese ladies (40). The following main parameters were measured: Fasting serum levels of total Cholesterol, triglyceride, high density lipoprotein-cholesterol, low density lipoprotein– cholesterol, glycohaemoglobin (HbA1c%), Insulin , Testosterone, FSH, serum LH and Irisin.

Results: The median serum Irisin was significantly higher among PCOS cases (119.1 ng/ml) compared to healthy controls (74.7 ng/ml). The median serum FSH was significantly higher among PCOS cases (6.52 ng/ml) compared to controls (4.72 ng/ml). Serum LH was obviously higher among PCOS cases (6.88 ng/ml) compared to controls (6.08 ng/ml). The mean pulse rate, blood WBC count and serum VLDL were significantly higher (71.6/min, 8.3 mg/dl and 22.5 mg/dl respectively) in cases with PCOS compared to controls (67.5/min, 7.2 mg/dl and 18.5 mg/dl respectively). Conversely, the serum HDL was significantly lower among PCOS cases (43.8 mg/dl) compared to controls (48.4 mg/dl), serum testosterone showed statistically non-significant differences.

Conclusion: Data of the present study showed that serum Irisin was significantly higher among PCOS cases. These findings suggest important role of irisin as a biomarker for PCOS and an important factor in the development of PCOS.

KEYWORD: PCOS, Obese, Irisin.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a prevalent heterogeneous endocrine disorder that affects approximately 10% of women during reproductive age (Bozdag et al., 2016), and is characterized by features of biochemical and/or clinical hyperandrogenism, oligo- or anovulation and polycystic ovaries . Accumulating evidence suggests that PCOS might be the leading cause of anovulatory infertility (Legro et al., 2013), and could predispose affected women to increased risks of cardiometabolic comorbidities including type 2 diabetes, metabolic syndrome and cardiovascular disease(Zhao et al.,2016) , subsequently leading to a negative impact on health-related quality of life for patients with PCOS and a significant burden on the healthcare system (Barry et al., 2011). As a result, specific intervention with a target in the etiology of PCOS is crucial and strongly recommended. However, it's causes remains not fully understood to date.

Insulin resistance, although not necessarily required for the diagnosis of PCOS, occurs in around 50–80% of women with this syndrome (Legro et al., 2004), and is considered to be an important contributor to its aetiology, in addition to hyperandrogenism (Cassar et al, 2016) .Irisin, an exercise-induced hormone that is produced primarily by skeletal muscle and adipose tissue upon proteolytical cleavage of protein fibronectin type-III domain containing protein 5 (FNDC-5) (Boström et al., 2012), is suggested to contribute to the development of PCOS because of its close relationship with insulin resistance (Polak et al., 2017) .

Aim of the study :

1-Evaluating the role of irisin in PCOS patients.
2-Finding out any correlation of irisin with other variants.

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SUBJECTS, MATERIALS AND METHODS

The design of the present study is a case - control study. This study was carried out in the Department of Medical physiology and pharmacology/college of Medicine / University of Duhok , Azadi Teaching Hospital and private gynecology clinic between 1/10/2018 – 1/4/2019.

Fifty obese Ladies (50) with history of PCOS, were involved in the study. The enrollment of ladies with PCOS chosen according to the Rotterdam Criteria (Rotterdam 2004). All ladies were at child bearing age (14-45 years) and were referred from different areas of Duhok governorates (rural and urban). The second group included forty apparently normal obese ladies (40), who served as control group. Data acquired at study entry included age, address, occupation , habitation , life style exercise, menstrual history (no.of cycle/year , amenorrhea ,oligo menorrhea, short Cycle ,prolonged cycle ,spotting), signs of hyperandrogenism , acne, hirsutism , dark skin, loss of hair ,family history, fertility, pulse rate, blood pressure . Before the blood pressure is recorded the patient should ideally avoid smoking or ingesting caffeine for 30 min, she should be quiet and comfortably warm . The arm selected should be resting & free of clothing . If you have not already felt the brachial pulse , do so to make sure it is intact .Position the arm so that the brachial artery is at heart level (roughly level with the 4^{th} inter costal space). When the patient is seated , resting the arm on a table a little above the patient's waist is suitable. When taking a standing blood pressure , try to support the arm at the midchest level. The inflatable cuff is wrapped around the arm above the elbow & stethoscope is placed over the brachial artery at the elbow .the cuff is inflated until the pressure in it is above the expected systolic pressure in the artery .the cuff occludes the artery , no sound is heard with the stethoscope . The pressure in the cuff is then lowered slowly , at the point at which the systolic pressure in the artery just exceeds the cuff pressure , the blood passes through the artery with each heart beat & with each beat a tapping sound is heard below the cuff . When sounds are heard is the systolic pressure & as the pressure in the cuff is lowered the sounds become louder , then dull & muffled & then disappear . When the sounds begin to muffle is the diastolic pressure in the artery, standard cuff in adults is 12 – 15 cm width , the narrower cuff gives false high results . Weight, height, body mass index, investigations (C.B.C, lipid profile, HbA1c, serum insulin, serum testosterone, FSH, LH, Irisin).

The height and weight for each lady were measured and used for calculation of body mass index (BMI) as follows (Abdullah, 2008): BMI = weight (kg) / height^2 (m^2). Those with BMI $\leq$ 25 were considered "normal", those between 25 and 29.9 were considered "overweight" and those with BMI > 30 were considered "obese" (Sorlie, 1995).

5 ml of venous blood was drawn from each participant, using sterile disposable syringe, 2 ml was placed into EDTA containing tube and sent for assessment of HbA1c% and complete blood count using Coulter Autoanalizer. The obtained serum from remained 3ml was divided into 3 parts and stored in capped tubes and stored frozen at -28° C until the time of analysis for lipid profile , Insulin , Testosterone, FSH, LH and Irisin levels.

By using human irisin ELISA kit for the quantitative determination of human irisin concentrations in serum, irisin measurement is carried out by enzyme-linked immunosorbent assays in plasma or serum (ELISA) or by the expression of Fndc5 mRNA .The validation of both tests has been debated by (Albrecht et al.,2015), after analyzing the different polyclonal antibodies available in the market to measure irisin concentration and the expression of FNDC5 mRNA . In contrast, (Jedrychowski et al.,2015), developed a method for the quantification of irisin using the technique of mass spectrometry in tandem, verifying the existence of irisin, making it possible to quantify it with greater accuracy and, in addition, to demonstrate that irisin is present in similar or even higher concentrations than hormones such as insulin, resistin, and leptin (Munoz .,et al 2018).The sensitivity of the assay was 0.2 ng/ml and the linear range of the standard was 5 to 500 ng/ml. The intra- and inter- assay variations were both less than 10%.(Bing Yan et al.,2014) , the range in our Kit was 3.12 ng/ml-200 ng/ml.

RESULTS

The mean pulse rate, blood WBC count and serum VLDL was significantly higher (71.6/min, 8.3 and 22.5 mg/dl respectively) in cases with PCOS compared to controls (67.5/min, 7.2 and 18.5 mg/dl respectively). Conversely, the mean blood pressure, blood MCV, MCH and serum

HDL was significantly lower among PCOS cases (86.8 mmHg and 43.8 mg/dl) compared to controls (90.5 mmHg, 86.9, 28.3 and 48.4 mg/dl).

The remaining measurements, namely: Body Mass Index, Hb concentration, MCHC, platelets count, RBC count, total serum Cholesterol, TG, LDL and HbA1c% showed no statistical differences between PCOS cases and controls, table 1.

Table (1): The case control difference in mean of selected measurements among overweight / obese study participants (BMI ≥ 25 Kg/m^2).

	Study group		
Parameters	**Healthy Controls N=40**	**Cases (PCOS) N=50**	**P value**
Body Mass Index (BMI Kg/m2)			0.09[NS]
Range	(25.2 to 41)	(25.7 to 42.2)	
Mean $_{+ SE}$	30 to0.66	31.5 to0.6	
Pulse rate (/min)			0.007
Range	(60 to 85)	(61 to 89)	
Mean	67.5 to0.85	71.6 to1.14	
Mean blood pressure (mmHg)			0.021
Range	(83.3 to 93.3)	(70 to 130)	
Mean	90.5 to0.52	86.8 to1.32	
Blood Hb concentration (gm/dl)			0.38[NS]
Range	(11.2 to 15.7)	(10.1 to 15.5)	
Mean	13.1to 0.17	12.9 to 0.16	
Blood WBC count mm^3 (µl)			0.013
Range	(4 to 10.7)	(4.6 to 12.7)	
Mean	7.2 to 0.26	8.3 to 0.31	
Blood MCV fL(10^{-15} Liter)			<0.001
Range	(80 to 93.8)	(69.2 to 96.5)	
Mean	86.9 to 0.55	82.6 to 0.89	
Blood MCH **pg(10^{-12} gm)**			<0.001
Range	(26.5 to 31.6)	(20.2 to 31.8)	
Mean	28.3 to 0.19	26.9 to 0.35	
Blood MCHC **g/dL**			0.9[NS]
Range	(30.5 to 35.5)	(29.2 to 35.9)	
Mean	32.5 to 0.14	32.5 to 0.2	
Blood platelets count mm^3 (µl)			0.49[NS]
Range	(144 to 402)	(135 to 414)	
Mean	259.1 to 10.34	269.4 to 10.35	
Blood RBC count			0.08[NS]
Range	(3.8 to 5.8)	(4 to 6.6)	
Mean	4.7 to 0.07	4.8 to 0.06	
Total serum Cholesterol (mg/dl)			0.51[NS]
Range	(124 to 197)	(105 to 227)	

Mean	164.8 to 3.15	161.1 to 4.32	
Serum TG (mg/dl)			0.05[NS]
Range	(60 to 136)	(59 to 357)	
Mean	101.8 to 2.72	121.7 to 8.75	
Serum HDL (mg/dl)			0.001
Range	(40 to 65)	(32 to 67)	
Mean	48.4to 1.11	43.8 to 0.82	
Serum LDL (mg/dl)			0.08[NS]
Range	(74.8 to 95.2)	(36 to 155)	
Mean	85.1 to 0.7	91.6 to 3.28	
Serum VLDL (mg/dl)			0.022
Range	(15.2 to 22)	(12 to 71)	
Mean	18.5 to 0.3	22.5 to 1.49	
HbA1c %			0.84[NS]
Range	(4.5 to 6)	(4.2 to 6.4)	
Mean	5.3 to 0.07	5.3 to 0.07	

As shown in table 2, among the same group of overweight/obese study participants the median serum insulin was significantly lower in cases with PCOS (27.7) compared to controls (67.1). Conversely, the median serum FSH was significantly higher among PCOS cases (6.52) compared to controls (4.72). Serum LH was also obviously higher among PCOS cases (6.88) compared to controls (6.08), but the difference statistically not significant. In addition, the median serum Irisin was significantly higher among PCOS cases (119.1) compared to controls (74.7). The last measurement, which is serum testosterone showed statistically non-significant differences.

The ROC analysis helps to rank the tested measurements in a rank or order from the most affected by PCOS disease process (having the largest ROC area closer to the maximum value of 1) to the least affected one (associated with the smallest ROC area closer to the minimum value of 0.5).

Table (2): The case control difference in median of selected measurements among overweight / obese study participants **.**

	Study group		
	Healthy Controls N=	**PCOS Cases N=**	**P - value**
Serum Irisin			<0.001
Range	(29.7 to 192.5)	(48 to 710)	
Median	74.7	119.1	
Inter-quartile Range	(55.6 to 95.6)	(89.3 to 184.5)	
Serum Testosterone			0.7[NS]
Range	(0.03 to 0.51)	(0.03 to 1.35)	
Median	0.27	0.31	
Inter-quartile Range	(0.18 to 0.38)	(0.18 to 0.45)	
Serum FSH			<0.001
Range	(2.26 to 6.98)	(1.89 to 11.3)	
Median	4.72	6.52	

Inter-quartile Range	(3.84 to 5.79)	(4.98 to 7.22)	
Serum LH			0.06[NS]
Range	(1.24 to 9.69)	(2.01 to 33.79)	
Median	6.08	6.88	
Inter-quartile Range	(3.88 to 7.58)	(4.02 to 10.12)	
Serum Insulin			<0.001
Range	(44.8 to 115)	(11.3 to 258)	
Median	67.1	27.7	
Inter-quartile Range	(55.5 to 85.7)	(18 to 56.4)	

Table (3): Area under ROC curve for selected measurements when used to discriminate between cases with PCO and healthy controls among overweight / obese study participants (BMI ≥ 25 Kg/m^2).

	Area Under ROC curve	P
Serum Insulin	0.806	<0.001
Serum Irisin	0.803	<0.001
Serum FSH	0.763	<0.001
Blood MCV	0.742	<0.001
Blood MCH	0.719	<0.001
Serum HDL (mg/dl)	0.687	0.002
Mean blood pressure (mmHg)	0.681	0.003
Pulse rate (/min)	0.641	0.022
Blood WBC count	0.638	0.026
Serum LH	0.616	0.06*
Blood RBC count	0.606	0.08 *
Serum VLDL (mg/dl)	0.595	0.12 *
Serum LDL (mg/dl)	0.583	0.18 *
Total serum Cholesterol (mg/dl)	0.552	0.4 *
Serum TG (mg/dl)	0.538	0.54 *
Blood platelets count	0.535	0.57 *
Blood Hb concentration (gm/dl)	0.530	0.63*
Blood HbA1c %	0.525	0.69 *
Serum Testosterone	0.524	0.7 *
Blood MCHC	0.523	0.71 *

*= nonsignificant

Table 4:

Positive if ≥ cut-off value	Sensitivity	Specificity	Accuracy
Pulse rate (/min)			
≥ 65.500	48.0	60.0	54.0
Blood WBC count			
≥ 7.400	48.0	52.5	50.3
Blood MCHC			
≥ 32.350	54.0	57.5	55.8
Blood platelets count			
≥ 261.000	56.0	62.5	59.3
Serum TG (mg/dl)			
≥ 90.000	52.0	57.5	54.8
Serum Testosterone			

0.225	58.0	52.5	55.3
Serum FSH			
≥ 5.595	68.0	75.0	71.5
Serum LH			
≥ 7.325	58.0	85.0	71.5

Table 5:

Positive if < cut-off value	Sensitivity	Specificity	Accuracy
Mean blood pressure (mmHg)			
<84.150	34.0	90.0	62.0
Blood Hb concentration (gm/dl)			
<13.450	66.0	45.0	55.5
Blood RBC count			
<4.815	64.0	52.5	58.3
Blood MCV			
<86.250	62.0	55.0	58.5
Blood MCH			
<27.350	42.0	85.0	63.5
Total serum Cholesterol (mg/dl)			
<142.500	46.0	67.5	56.8
Serum HDL (mg/dl)			
<44.500	90.0	55.0	72.5
Serum LDL (mg/dl)			
<84.100	50.0	62.5	56.3
Serum VLDL (mg/dl)			
<17.050	44.0	70.0	57.0
Blood HbA1c %			
<5.350	54.0	60.0	57.0
Serum Insulin			
<50.450	56.0	80.0	68.0
Serum Irisin			
<68.900	58.0	57.5	57.8

DISCUSSION

Polycystic ovary syndrome (PCOS) is complex heterogeneous disorder which has several aspects in terms of pathology such as metabolic, endocrine, reproductive and psychological. However, the etiology of PCOS remains poorly understood.(Günalan et al.,2018) .

Several studies suggest that insulin resistance and hyperandrogenism play a central role in progression of PCOS pathophysiology. Obesity is present in 30-75% of women with PCOS. Adipose dysfunction contributes to the

development of glucose intolerance and hyperinsulinemia, which in turn can exaggerate the manifestations of hyperandrogenism. Obese women with PCOS are at increased risk of anovulation and consequent sub-fertility (Diamanti-Kandarakis et al.,2012) . New insights into the regulation of hormones and cytokines in muscle and fat tissue support the concept that PCOS is a systemic syndrome (Ana et al.,2019), skeletal muscle has also been identified as a secretory organ that releases cytokines and other peptides, called myokines. Irisin has been identified as an exercise-induced myokine and has been proposed to mediate the beneficial effects of exercise on metabolism (Ana et al.,2019) and thus far, no report has demonstrated the relationship of circulating irisin and insulin resistance in PCOS women (Ana et al.,2019) . As far we are aware, this is the first report, in our locality, describing the plasma level of irisin in ladies with PCOS.

The median of serum Irsin was significantly higher among obese PCOS cases (119.1ng/ml) compared to control(74.7 ng/ml).These results are consistent with ((Minyan et al., 2015) showed that irisin levels was significantly elevated in PCOS patients even in the absence of risk factors for metabolic syndrome. These findings suggest important role of irisin as a biomarker for PCOS and an important factor in the development of PCOS (Gutch et al.,2016).

In adults, irisin levels are affected by exercise, age, sex, muscle mass obesity, and cold exposure but not by meals and diurnal rhythms (Löffler et al .,2015) . Besides these factors, irisin levels are affected in metabolic diseases such as type 2 diabetes mellitus, gestational diabetes mellitus , polycystic ovary syndrome, nonalcoholic fatty liver disease and chronickidneydisease (Chen et al.,2015).It was suggested that normal ovarian physiology is maintained by intreractions between endocrine factors from muscle,and ovarian tissues. The abnormal regulation of irisn may represent early events in PCOS (Chang et al.,2014) . It is considered that the rising circulating irisin level in obesity is an accommodative compensatory response to obesity-induced metabolic dysfunction, such as a decline of insulin levels or irisin resistance (Qiu et al., 2016).

The high levels of irisin in PCOS patients can be explained as an Irisin resistance state (Sesti et al.,2014) similar to insulin resistance (Chang et al., 2014) in which high circulating hormone levels fail to induce the desired physiologic effect, or by the suggestion that it acts as a protective mechanism to counteract excess energy inflow, because irisin normally increases energy expenditure in brown and adipose tissues (Bostrom et al.,2012).

The elevated serum irisin level was suggested to be a protection in the pre-diabetic state of PCOS before diabetes mellitus develops. The abnormal changes of irisin may contribute to the development of risk factors resulting in increased morbidities such as insulin resistance, dyslipidemia, and obesity in PCOS patients (Chang et al., 2014).

In the present paper there was also lower level of serum HDL, higher VLDL in obese PCOS ladies than control .These findings were agree with (Bing Yan et al.,2014, Munoz .,et al 2018). It was found that irisin levels increased in patients with metabolic syndrome compared to those of patients without the syndrome(Salazar et al. ,2011). The most accepted theory to explain the pathophysiology of the metabolic syndrome is insulin resistance (Foda et al.,2018). There was lower level of insulin in obese PCOS ladies compared with corresponding control, in contrary to the most other researches (Foda et al ,2018) **(**Ibrahim et al ,2018).

The only explanation in our thought could be that the PCOS ladies were selected from private clinic and they were already on metformin (anti diabetic drug) treatment. According to the ASRM/ESHRE criteria definition, PCOS is frequently associated with insulin resistance and metabolic syndrome (Rotterdam Criteria 2003) (Bostanci et al ,2015) . Insulin has a key role in carbohydrate metabolism and insulin resistance is associated with PCOS at a reported prevalence of 50%-70% occurring independently of obesity (Kurdiova et al.,2014).

In this study, we found that women with PCOS had higher circulating WBC compared with BMI matched female controls which is consistent with (Orio et al.,2005) .Chronic, low-grade inflammation is a characteristic of many metabolic diseases, including obesity, metabolic syndrome and type 2 diabetes (Hotamisligil,2006).Given the metabolic derangement in women with PCOS, this syndrome has also been hypothesized to be associated with increased levels of low grade inflammation (Escobar-Morreale.,2011) .

The level of LH, FSH were higher in obese PCOS than control .this result is contrary to (Abdulrazak et al.,2007) whom concluded that

no significant statistical correlation was found between LH/FSH ratio, BMI, menstrual pattern and hirsutism. This disproves the traditional concept about PCOS. In women with PCOS, the frequency of LH secretion is increase. This change happens in response to receiving stimulation by GnRH and increase bioavailability of LH. The high level of LH, lead to ovarian hyperplasia and production of androgen from ovarian stromal and thecal cells. This condition fixes the chronic anovulation. It is not clear that the impairment in hypothalamic-pituitary-ovarian axis leads to PCOS or this disturbance happen as an outcome of PCOS (Bostanci et al.,2015).Although serum testosterone level was of no any statistic difference in between two groups (obese PCOS and obese control), but the interquartile range and even mean rank was different serum testosterone were more in PCOS than control and this result is agree with almost all other researches (Chang et al.,2019). Hyperandrogenism is an important clinical characteristic of the syndrome since it is associated with worse prognosis and higher risk of metabolic and cardiovascular disease (Daan et al.,2015) . However, recent genetic findings suggested that it may not be the only driver of PCOS manifestations (Day et al.,2018).As diagnosed by the Rotterdam criteria, hyperandrogenism is present in about 60 to 80% of cases.

Biochemical hyperandrogenism remains a diagnostic challenge because the assay methods are poorly standardized, there are no universal cutoffs for diagnosis, and some assays for free testosterone quantification are unreliable (Wierman et al.,2014).

About cut off value for serum irisin, serum insulin, Blood HbA1c % , serumVLDL, serum LDL, serum HDL, total serum cholesterol, Blood MCH, Blood MCV, Blood RBC count, Blood Hb concentration, Mean blood pressure, serum LH, serum FSH, serum testosterone, serum TG, Blood platelets count, Blood MCHC, Blood WBC count and pulse rate are collectively positive.

The capacity of serum irisin to differentiate between cases of PCOS and healthy control was assessed with a receiver – operating characteristic (ROC) curve analysis. The Roc area for serum irisn was (0.803,p< 0.001).Serum irisn was of high validity in differentiating PCOS cases from healthy control. This result is in agreement with (Foda et al.,2018).When he suggested that the analysis of ROC curves may suggest that serum irisin may be a valuable biomarker for diagnosis and for monitoring cases with PCOS during treatment.

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الخلاصة

تعتبر متلازمة المبيض المتعدد الكيسات التي هي اضطرابات الغدد الصماء الأكثر شيوعًا والتي تؤثر على 10-15٪ من النساء في سن الإنجاب. كان الهدف هو تقييم تاتير هرمون الايريسين في مسببات المصابات بمتلازمة المبيض المتعدد التكييس

تصميم وطرق الدراسة:

مع تشخيص موكد لمتلازمة المبيض المتعدد التكييس .

تضمنت هذه الدراسة خمسين سيدة بدينة (50) وكذلك مجموعة تانية كمجموعة ضابطة وتضمنت اربعين سيدة سمينات عاديات

تم قياس المتغيرات الرئيسية التالية:

مؤشر كتلة الجسم في المجموعتين و مؤشر كتلة الجسم و تقدير مستوى الدهون في الدم: مجموع الكوليسترول في الدم و الدهون الثلاثية في الدم والبروتين الدهني عالي الكثافة في الدم - الكولسترول ، LDL - الكوليسترول و تقدير الجلايكوهيموغلوبين (HbA1c ٪) ، قياس مستويات الأنسولين وهرمون التستوستيرون و FSH و لوتين (LH) و هرمون الايريسين في الدم.

النتائج:

كان متوسط هرمون إيريسين المصل أعلى بشكل ملحوظ بين حالات متلازمة تكيس المبايض (119.1 نانوغرام / مل) مقارنة بالمجموعة الضابطة (74.7 نانوغرام / مل) و كان متوسط FSH المصل أعلى بشكل ملحوظ بين حالات متلازمة تكيس المبايض (6.52 نانوغرام / مل) مقارنة بالمجموعة الضابطة (4.72 نانوغرام / مل). اما LH المصل فكان أعلى بشكل واضح بين حالات متلازمة تكيس المبايض (6.88 نانوغرام / مل) مقارنة بالمجموعة الضابطة (6.08 نانوغرام / مل).

اما متوسط معدل النبض و عدد خلايا الدم البيضاء في الدم VLDLفي المصل كان أعلى بشكل ملحوظ (71.6 / دقيقة ، 8.3 مجم / ديسيلتر و 22.5 مجم / ديسيلتر على التوالي) في الحالات مع متلازمة تكيس المبايض مقارنة بالمجموعة الضابطة (67.5 / دقيقة ، 7.2 مجم / ديسيلتر و 18.5 مجم. / دل على التوالي). في المقابل ، كان HDL المصل أقل بشكل ملحوظ بين حالات متلازمة تكيس المبايض (43.8 مجم / ديسيلتر) مقارنة بالمجموعة الضابطة (48.4 مجم / ديسيلتر).

الاستنتاجات:

أظهرت بيانات الدراسة الحالية أن إيريسين العضلات المصلي كان أعلى بشكل ملحوظ في حالات متلازمة تكيس المبايض مما قد يعزز دوره في حدوثها.