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The correlation between thyroid hormones, reproductive hormones, body mass index (BMI) and hirsute in Iraqi women with polycystic ovary syndrome (PCOS).

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ABSTRACT

This research was conducted to study the relationship between the polycystic ovary syndrome (PCOS) and thyroid disorders. The study includes 50 infertile Iraqi women with polycystic ovary syndrome (PCOS) and 20 healthy women. Blood samples were collected from the Infertility Center of AL-Yarmok Teaching Hospital in Baghdad, during the period from November, 2010 to May, 2011. The age of infertile and fertile women was ranged from 16 to 45 years. Hormonal study of Estradiol (E2), Luteinizing hormone (LH), Follicle stimulating hormone (FSH), Testosterone (T),Thyroid stimulating hormone (TSH) , Triiodothyronine (T3) and Thyroxin(T4) was done for each patient. The results showed that there is a significant ($P < 0.05$) decrease in E2 and FSH levels in PCOS women comparing with fertile women. Also a significant ($P < 0.05$) increase in LH\FSH was detected in PCOS women and fertile women and non-significant ($P < 0.05$) differences in testosterone, TSH, T3 and T4 levels between infertile and fertile women.The hormonal profile according to Body mass index -BMI was showed to be significantly ($P < 0.05$) decreased in testosterone in obese and overweight PCOS women, significant ($P < 0.05$) decrease in FSH level in obese PCOS women and no significant differences in E2 and LH levels.According to the hirsute in PCOS women the hormonal profile showed a significant ($P < 0.05$) decrease in E2 and FSH levels in hirsute PCOS women, no significant differences in LH levels and elevated in testosterone levels but without significance in hirsute PCOS..

Introduction

Polycystic ovary syndrome (PCOS) was first described in 1935 by Stein and Leventhal **(1)**. PCOS is a common endocrine disorder in women which associated with marked increases in ovarian androgen production (hyperandrogenaemia) and insulin resistance. Although the pathogenesis of PCOS remains uncertain, insulin resistance and hyperinsulinemia are found in approximately 80 % affected women (**2).**

A complete understanding of the underlying pathophysiology of PCOS is still lacking, because of the heterogeneity of this disorder.There are most likely multiple underlying pathophysiologic mechanisms (**3**). Several theories have been proposed to explain the pathogenesis of PCOS such as an increase in (GnRH) and LH secretion (**4**) or alteration in insulin secretion lead to hyperinsulinmia and insulin resistance (**5,6**). A defect in androgen synthesis that results in an increased ovarian androgen production was also proposed (**7**).

The common symptoms associated with POC include elevated serum levels of androgens, specifically testosterone, androstenedione, and

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dehydroepiandrosterone sulfate (DHEAS). These elevated levels lead to hirsutism of facial hair, androgenic alopecia, acne, oily skin and seborrhea (**8**). Occasionally masculinization (**9**) and obesity centered on the lower half of the torso 'apple-shaped" were also noticed *(***10**) in addition to other symptoms.

The current study was conducted to find the correlation between PCOS, hirsutism and obesity.

Materials and Methods

Subjects

Two study groups have been investigated, the first group consist of fifty infertile Iraqi Baghdadian women with PCOS. PCOS patients were selected from the Infertility Center of Al-Yarmouk Teaching Hospital from November, 2010 to May, 2011.The second group is healthy control which consists of twenty healthy fertile women of different ages. All of them are chosen dependent on the following criteria (**11**):

Regular menstrual cycle (26 to 30 days), age 15 to 45 years, normal body mass index (18 to 25 Kg/m²), no history of endocrine disease and no use of medication or oral contraceptives. Ultrasonography and blood sampling were collected from subjects during the follicular phase (3, 4, or 5 day).

The data were collected together with the subject's gynecological history and all their social, medical, and reproductive data according to a questionnaire forma.

Criteria used for the diagnosis PCOS subjects:

To enroll the subjects with PCOS should include at least two of the following three features (**12**):

1. Oligo and /or unovulation.
2. Clinical and /or biochemical features of hyper androgenism.
3. The presence of polycystic ovary morphology.

Blood sampling:

Venous blood sample (5 ml) was collected from each woman of both PCOS and healthy control. The serum obtained by putting the blood samples in a clean dry plain plastic tube and allowed to clot at 37C for 30 minutes before centrifugation. The tubes centrifuged at 5000 rpm for 5 minutes, serum was collected and kept in freezer until used.

Hormonal assay:

Hormonal analysis for E2 , FSH, LH ,T was performed by using Addendum-Mini VIDAS apparatus (VIDAS) 12 mode l0, 1992, BioMerieux Company, France, through an enzyme linked fluorescent assay (ELFA) technique.

Body Mass Index:

The female body mass index (BMI) was measured according to the following equation: dividing the weight in kilograms by the height in squared meters (kg/m2) (**13**)**.**The parameters of Body mass index which was used was according to the European Society of Human Reproduction and Embryology**,** 2009 and as follow:

Underweight ≤ 18.5 , Normal 18.5-24.9, Overweight 25-29.9, Obesity ≥ 30.

Hirsutism:

Modified Ferryman-Galway with single observer was used to assess the degree of hirsutism .Score of at least 8 of 36 was taken as significant (upper

lip ,face ,jaw and neck, upper back, lower back, arm ,thigh ,chest, upper abdomen , lower abdomen and perineum (**14**).

Statistical analysis:

The statistical analysis system –SAS (2004) program was used to the find the effect of different factors in traits in this study. Least significant difference (LSD) test was also used for the significant comparision between means.

Results and Discussion

Age

The age of all PCOS women was ranged < 20 to > 40 years. Table (1) revealed that 44% of the study PCOS women were between(21-30) years , followed by 32% of patients whose age ranged between 31-40 years and 14% of patients whose age (<20 years) , and older age group (> 40years) were only 10 % of total PCOS patients. This may lead to conclude that group 2 and group 3 constituted the greatest number groups in the present study. The results disagreed with **Bronstien *et al*. (15)** who reported that 74% of PCOS were adolescences. In Iraq girls might not be diagnosed in the early pubertal

years therefore, the results revealed the high percentage in age group (21-30 years).

Table (1): Distribution of PCOS women and control group mean according to their ages.

Age group	PCOS		Control group	
	No.	%	No.	%
<20	7	14	1	5
21-30	22	44	16	80
31-40	16	32	3	15
> 40	5	10	0	0
Total	50	100%	20	100%

Hormonal profile:

1. Reproductive Hormones

The results obtained from hormonal analysis revealed that the E2 and FSH have a significant lower levels in PCOS women (34.89±2.39pg/ml ; 6.99 ± 0.41 µIU/ml respectively) than healthy control group (54.07 ± 7.02 pg/ml; 13.56 ±3.79 µIU/ml respectively). Other parameters such as LH and LH/FSH ratio showed no significant levels. On the other hand, testosterone levels showed elevated level (1.43±0.29) ng /ml than control group (0.60 ±0.13) ng/ml in PCOS women with no significant (P>0.05) different (Table -2).

Table (2): Mean endocrine –metabolic values (± SE) of polycystic ovary syndrome and healthy women.

Hormones	Mean ± SE		LSD value
	PCOS	Healthy control	
E2 (pg/ml)	34.89 ± 2.39	54.07 ± 7.02	17.464 *
T (ng/ml)	1.43 ± 0.29	0.60 ± 0.13	2.102 ns
LH (µIU/ml)	10.04 ± 0.98	13.51 ± 3.88	7.247 ns
FSH (µIU/ml)	6.99 ± 0.41	13.56 ± 3.79	3.517 *
LH/FSH	1.49 ± 0.168	0.99 ± 0.008	1.203 ns

P<0.05) , ns: non-significant.
T:Testosterone, E2: 17 β-Estradiol, LH: Luteinizing Hormone, FSH: Follicle Stimulating Hormone, SE: Standard error, PCOS: Polycystic ovary syndrome, LSD: Least Significant Differences .

The current results agreed with **Chang and Katiz (16)** who's showed the E2 hormone level in PCOS women may be low to normal. The increase in serum AMH level in PCOS women resulted from an increased production o this hormone per follicle (**17**); this amount led to an inhibits of aromatase activity therefore the follicle did not produce a sufficient amount of E2 hormone (**18**). The elevated of testosterone was in agreement with the study of **Carmina *et al*.(19)** who explained the LH hyper secretion which was in positive correlation with the elevated serum of 17-hydroxyprogestrone , androstenedione and testosterone There were additional causes of hyper androgenism (**20**) as : An increased synthesis of testosterone precursors due to a dysregulation of theca cell androgen production. An inhibin augmentation of LH-mediated androstenedione production. Hyper insulinemia, which has been proposed as the primary event leading to hyper androgenism. An increased serine phosphorylation of the insulin receptor, resulting in an activation of both ovarian and adrenal P450c17α enzymes and promoting androgen synthesis. Genomic variants in genes related to the regulation of androgen biosynthesis, function, and the availability of androgens to target tissues, insulin resistance and the metabolic syndrome. The high level of LH which was noticed in this study was explained by **MecCartney *et al*. (21**) who found that the PCOS women as exhibiting an accelerated frequency and / or higher abundance of LH pulses ,augmentation of LH secretory burst mass, a more disorder in LH secretion. One study reported that 75% of PCOS women have an elevated LH level, because of the elevated insulin levels that cause the abnormalities in hypo thalamicpituitary-ovarian axis that lead to PCOS (**20,23).**

Also the hormonal assay showed that the FSH was significantly lower in PCOS compared with healthy group. These result was in agreement with the finding by **Begawy *et al* .(24)** .The reduction levels of FSH could be due to high levels of inhibin that have been reported by others in the PCOS women which lead to FSH reduction (**25**) or to over expression of Follistatin leading to the increase of ovarian androgen production (**26**). The increased level of androstenedione from adipose tissue of the PCOS additionally stimulates LH and inhibits FSH (**1**). The results obtained revealed that there was LH/FSH >1.5 in PCOS women (1.493±0.168) compared with healthy group (0.991 ± 0.008),these results were in agreement with **Arroyo *et al* .(27**) who demonstrated that >75% of PCOS women with dyesregulation in gonadotropic function and explained that the normal pulsatile secretion of LH was increased by an increased frequency and amplitude of pulses, while that of FSH is unchanged or muted ,therefore the LH values may be elevated, and the LH:FSH ratio can

be increased to more than 2.5 . On the other hand, these values may be normal in as many as 10% to 20% of women with PCOS.

2. Thyroid Hormones:

Data listed in table (3) showed that there were no significant differences in TSH , T4 and T3 levels in 82% of PCOS women, but all data showed there were elevated levels than normal ranges.

Table (3): The levels of TSH, T4 and T3 among polycystic ovary syndrome infertile and fertile healthy women.

Hormones	Mean ± SE		LSD value
	PCOS	Healthy control	
TSH µIU/ml	9.22 ± 3.51	2.15±0.50	25.127 ns
T3 n. mol/L	4.15 ± 1.58	1.97±0.16	11.317 ns
T4 n. mol/L	101.54 ± 4.87	96.25±5.13	34.954 ns

*** (P<0.05) , ns: non-significant.**
TSH : Thyroid-Stimulating Hormone, T3: Triiodothyronine T4: Thyroxin ,SE: Standarderror, PCOS: Polycystic Ovary Syndrome, LSD: Least Significant Differences.

Both the thyroid and ovaries are part of the endocrine system and belong to a common hormonal axis consisting of hypothalamus - pituitary – thyroid – ovaries (**23**) and according to recent studies, many evidences showed that women who suffered from PCOS present, in most cases, thyroid disorders which is often associated with hypothyroidism or at risk of future hypothyroidism (**20**). The hypothyroidism may lead to lower levels of sex hormone binding globulin (SHBG), which in turn leads to high concentrations of testosterone, one of the factors that contribute to the onset of some symptoms of PCOS such as infertility, polycystic ovaries, hirsutism and acne(**28**). In the current study we did not detect any abnormal levels of thyroid hormones among PCOS patients which may link the PCOS with another factor such as insulin resistance(**29).**

Body mass index and hormonal changes:

The results of this study shown in table(4) indicate that there has been significantly lower ($p<0.05$) level in T hormone levels in obese PCOS women (1.09±0.15ng/ml) and overweight PCOS women (1.26±0.54 ng/ml) when compared with normal weight PCOS women (2.78±1.19 ng/ml). The current results also showed the FSH levels which were significantly lower ($p<0.05$) in obese PCOS women (5.96 ± 0.43µIU/ml) when compared with normal weight PCOS women (7.94± 1.33µIU/ml) . There was no significant difference in serum levels of E2 and LH.

Table (4): Body mass index of polycystic ovary syndrome women and hormonal profile (Mean ± SE).

Hormones	BMI			LSD
	Normal weight	Obesity	Overweight	
E2 pg/ml	35.11 ± 6.30	39.02 ± 3.93	29.79 ± 2.99	9.237 ns
T ng/ml	2.78 ± 1.19	1.09 ± 0.15	1.26 ± 0.54	1.231 *
LH µIU/ml	11.63 ± 3.04	8.28 ± 1.27	11.49 ± 1.61	5.924 ns
FSH µIU/ml	7.94 ± 1.33	5.96 ± 0.43	7.85 ± 0.72	1.663 *

*** (P<0.05) , ns: non-significant.**
E2: 17 β-Estradiol, T: Testosterone, LH: Luteinizing Hormone, FSH: Follicle Stimulating Hormone, SE: Standard error, LSD: Least Significant Differences, BMI: Body Mass Index weight/heigth^2)

The decrease levels of FSH in this study is in agreement with the findings by **Beydoan *et al*. (30),** and **Liou *et al* .(31).** Yet the present findings were in disagreement with **Benson *et al*.(32)** who reported the obese and overweight PCOS women who had increase levels of T hormone and LH. **Wang *et al*.(33)** reported that there was high prevalence of overweight/obesity in PCOS women ,the obesity is associated with insulin resistance and correlated with decreased SHBG which caused an increase in circulating testosterone(**34**). Obesity represents a probable pathogenetic factor since the FSH secretion of the pituitary is inhibited by the increased synthesis of E2 in the adipose tissues, the additional cyclic E2 lead to an increase secretion of LH of the pituitary **(35)**. **Freytag (36)** proved that hyperandrogenaemia possibly causes android fat after hirsutism in PCOS women and confirmed the suggestion that obesity caused by hyperandrogenaemia is mostly adrenocortical obesity. She also reported that both hyperandrogenaemia and obesity were related to the insulin metabolism.

Hirsutism and hormones changes:

Data listed in table (5) showed the E2 was significantly lower ($p<0.05$) in PCOS women with

hirsutism (33.17±2.37pg/ml) compared with PCOS women (42.75 ±7.50pg/ml). The results obtained showed that there were no ($p>0.05$) significant differences in serum levels of LH in PCOS hirsute and PCOS women. In spite of the T hormone levels that was with no significant

Difference between PCOS hirsute women (1.50±0.35)ng/ml and PCOS women (1.07±0.31ng/ml). While these results were significantly ($p<0.05$) higher than the control subjects (0.6±0.13) ng/ml.

Table (5): Effect of hirsutism in level of hormones (Mean ± SE) in polycystic ovary syndrome women.

Hormones	Hirsutism Mean ± SE		LSD value
	PCOS without hirsute	PCOS with hirsute	
E2 pg/ml	42.75 ± 7.50	33.17 ± 2.37	8.856 *
T ng/ml	1.07 ± 0.31	1.50 ± 0.35	1.180 ns
LH µIU/ml	13.87 ± 2.19	9.20 ± 1.06	5.680 ns
FSH µIU/ml	8.05 ± 0.54	6.76 ± 0.48	1.594 ns*

*** (P<0.05) , ns: non-significant.**
E2: 17 β-Estradiol, T: Testosterone, LH: Luteinizing Hormone, FSH: Follicle Stimulating Hormone, PCOS: Polycystic Ovary Syndrome SE: Standard error, LSD: Least Significant Differences,

The results noticed by this study were in agreement with **Deugarte *et al*. (37)** and **Azziz *et al*. (38). Ovalle and Azziz, (39)** concluded the source of hyperandrogenism due to the genetic abnormalities in insulin receptor resulting in the thickening of the ovarian theca that increased the androgen production and inhibition of SHBG synthesis. The degree of hirsutism might be influenced by the relative activity of the 5α reductase that convert testosterone to the more active metabolic dihydrotestosterone (4,**20**).

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العلاقة بين هرمونات الدرقية والتكاثر وكتلة الجسم و المشعرانية عند النساء العراقيات المصابات بمتلازمة تعدد الاكياس المبيضية

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الخلاصة

هدفت الدراسة الى أيجاد العلاقة بين متلازمة تعدد الاكياس المبيضية وأختلال الهرمونات الدرقية. شملت الدراسة ٥٠ من النساء غير الخصيبات مصابات بمتلازمة تعدد الاكياس المبيضية و ٢٠ من النساء الخصيبات الطبيعيات. جمعت عينات الدم من المشمولات بالدراسة من مركز العقم في مستشفى اليرموك التعليمي في بغداد بين نوفمبر ٢٠١٠ الى مي ٢٠١١. تراوح عمر المشمولات بالدراسة بين ١٦ و٤٥ سنة. حسب مستوى هرمونات الاسترادول وهرمونات التكاثر LH, FSH والتستوستيرون وهرمونات الدرقية T3,T4 والهرمون المحفز TSH لهما ولجميع المشمولات بالدراسة. بينت النتائج وجود أنخفاض معنوي بأحتمالية ٠.٠٥ في مستوى هرمونات الاسترادول و FSH وزيادة معنوية في مستوى LH\FSH عند النساء المريضات مقارنة مع الطبيعيات. كما بينت النتائج عدم وجود فروق معنوية في مستويات هرمونات التستوستيرون والهرمونات الدرقية T3, T4 و TSH بين النساء المريضات والطبيعيات. قياس مقدار كتلة الجسم BMI والمشعرانية تم أيضا في هذه الدراسة. بينت النتائج وجود انخفاض معنوي في مستوى التستوستيرون في النساء المريضات بمتلازمة تعدد الاكياس المبيضية والسمينات وزائدات الوزن وانخفاض معنوي في مستوى هرمون FSH وعدم وجود فروق معنوية في مستوى هرمونات الاسترادول و LH في المريضات السمينات. وبينت نتائج المشعرانية ان هناك انخفاض معنوي في مستوى هرمونات الاسترادول و FSH وعدم وجود فروق معنوية في مستوى هرمون LH وأرتفاع غير معنوي في مستوى التستوستيرون عند النساء المشعرات المريضات بالمتلازمة.