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Polycystic ovary syndrome is associated with a more pronounced atherogenic lipid profile.

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Background:

Polycystic ovary syndrome (PCOS) is associated with a higher frequency of cardiovascular risk factor. Lipid profiles are potent markers for cardiovascular risk.

Aim of the study:

To identify changes in lipid metabolism in women with PCOS and the relative impact of obesity and hyperandrogenism on lipid parameters.

Subject and methods:

The study was conducted in the period from August 2006 till March 2007 on eighty women, who had been diagnosed as PCOS; they were recruited from IVF Institute of Embryo Research &Infertility Treatment in Baghdad. Twenty apparently healthy fertile women were served as control. Endocrine evaluation and Lipid assays were performed in both cases and controls.

Results:

There were significant elevations in LH, LH to FSH ratio, Testosterone and Free testosterone levels in women with PCOS when compared with the normal control group.

For lipid profile there was a significant elevation in levels of TG, Cholesterol, and LDL in combination with low level of HDL and with an increase in atherogenic index in women with PCOS when compared with normal control group.

Conclusion:

Amore atherogenic lipid profile, in particular related to HDL metabolism, was found in women with PCOS, both obesity and hyperandrogenism contribute to these changes, and there was evidence for an additional influence of PCOS on lipid metabolism.

Key words: PCOS, TG, LDL, HDL, Cholesterol, VLDL, Atherogenic index.

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ألخلاصة

مرض تكييس المبايض هو قصور في عمل المبايض من حيث الاباضه ,ويرافق ذلك صعود في تركيز الهرمونات الذكرية في مصل الدم,كما ويرتبط بوجود بعض العوامل التي تمثل خطورة الاصابه بأمراض ألاوعيه ألدموية للقلب .

هدف البحث

يهدف هذا البحث لدراسة التغيرات الايضيه التي تحصل في الدهون في مصل الدم في مريضات تكييس المبايض وتأثير زيادة الهرمونات ألذكريه والسمنة لدى المريضات على هذه التغييرات.

خطة البحث

اجري البحث على ثمانين مريضه من المتزوجات ويعانون من العقم,وقد تم تشخيصهم مسبقا في معهد أبحاث الاجنه وعلاج العقم ,جامعة النهرين ,بغداد.

ولأجل ألمقارنه تم إجراء نفس التحاليل على عشرين امراة لا تعاني من العقم أو المرض .

وقد تم فحص الهرمونات والدهون على المجموعتين.

النتائج

لوحظ في هذه الدراسة زيادة معنوية في تراكيز الهرمونات في المريضات عند مقارنة النتائج مع هرمونات النساء الأصحاء أما بالنسبة للدهون فهناك زيادة في تراكيز الدهون الثلاثية والكلسترول والبروتين ذو الدهون واطئة الكثافة مع نقص معنوي في البروتين ذو الدهون عالية الكثافة عند مقارنتها مع التراكيز في النساء الأصحاء.

الاستنتاج

تم التوصل إلى إن ألسمنه وزيادة الهرمونات لدى مريضات تكيس المبايض يؤثر بشكل معنوي على الايض الحيوي للدهون في مصل الدم ,مما يشكل خطورة الإصابة بأمراض ألاوعيه الدموية للقلب.

Introduction

Polycystic ovary syndrome (PCOS) is a syndrome of ovarian dysfunction that is characterized by anovulation, hyperandrogenism and the presence of polycystic ovary morphology (1).

Obesity and insulin resistance occur frequently in association with this syndrome. Cardiovascular risk factors seem to cluster in women with PCOS compared to the general population(2).The combination of elevated levels of adrenal androgens and obesity leads to increased formation of extra glandular estrogen(3).

The studies that performed both in vivo and vitro (in cultured theca cells) consistently suggested that ovarian theca cells in affected women are more efficient at converting androgenic precursors to testosterone than are normal theca cells (4) .There are several lines of evidence suggesting that women with PCOS are also at increased risk of cardiovascular disease (5). Women with the disorder are at substantial risk for the development of metabolic and cardiovascular abnormalities (6).

Testosterone decrease lipoprotein lipase activity in abdominal fat cell and insulin resistance impairs the ability of insulin to exert its antilipolytic effects, although these abnormalities would be expected to increase the morbidity and mortality from coronary artery disease and other vascular disorders in women with the PCOS, this has been difficult to establish (8,9,10).

Subjects & Methods

Eighty women in their reproductive age (20-40) years old, who had been diagnosed as PCOS, were recruited from IVF Institute of Embryo Research & Infertility Treatment, Baghdad. They were seen in time period from August 2006 till March 2007.

Twenty apparently healthy fertile women were served as control, which were matched for age and with regular menstrual cycle and normal ultrasound.Twelve hours fasting venues blood sample was taken from each patient, Endocrine evaluation and Lipid assays were performed in both cases and controls. Endocrine evaluation included serum levels of LH, FSH, Testosterone and Free testosterone. Hormone assay were determined by ELFA (Enzyme Linked Fluorescent Assay) technique (11).Serum cholesterol level was measured by enzymatic end point method (12). HDL cholesterol was analyzed by using the direct HDL assay CHOD-PAP method. LDL cholesterol was determined by using formula of Friedwald (13).Triglycerides was determined after enzymatic hydrolysis with lipase, using triglycerides enzymatic PAP 150 BioMerieux.

Results

There were a significant elevation in the mean of Body Mass Index (BMI), LH, and LH to FSH ratio, Serum Testosterone, Free testosterone.(Table1).

TG, VLDL, LDL, Cholesterol and Atherogenic Index with significant decrease in HDL in patients with PCOS when compared with the normal control group.(Table2),(Figure1-6).

Characteristics	PCOS	Control	P value
Number	80	20	
Age (years)	28±6.3	27±3.4	0.3 NS
BMI (kg/m2)	29.3±7.3	22.6±2.7	< 0.05
FSH (mIU/ml)	5.4±1.5	5.8±0.5	>0.05
LH (mIU/ml)	11.9±5.4	7.0±0.6	<0.05
LH/FSH ratio	2.3±1.1	1.2±0.1	<0.05
T-Testo (ng/ml)	50±9.6	24±3.4	<0.05
F-Testo (pg/ml)	8.9±1.0	1.9±0.4	<0.05

Table 1: BMI and Endocrine parameters of PCOS and Healthy control subjects.

Studied parameters	Grouping	N	Mean	Std. Deviation	Std. Error	Sig. (2-tailed)
Triglycerides	Polycystic ovary syndrome	80	115.306	32.368	3.618	0.008**
	Control	20	95.15	12.562	2.809	
Very low density lipoprotein	Polycystic ovary syndrome	80	24.682	16.190	1.810	0.004**
	Control	20	19.03	2.512	0.561	
Low density lipoprotein	Polycystic ovary syndrome	80	146.256	34.526	3.860	0.016*
	Control	20	126.58	18.344	4.101	
High density lipoprotein	Polycystic ovary syndrome	80	36.296	7.728	0.864	0.011*
	Control	20	38.1	3.552	0.794	
Cholesterol	Polycystic ovary syndrome	80	206.8888	35.673	3.988	0.007**
	Control	20	184	18.324	4.097	
Atherogenic index	Polycystic ovary syndrome	80	4.313	1.528	0.170	0.008**
	Control	20	3.37	0.687	0.153	

Table 2 Lipid profiles of PCOS and Healthy control subject.

NS=not significant.
*= the significant level was ≤0.05.
**= the significant level was ≤0.005.

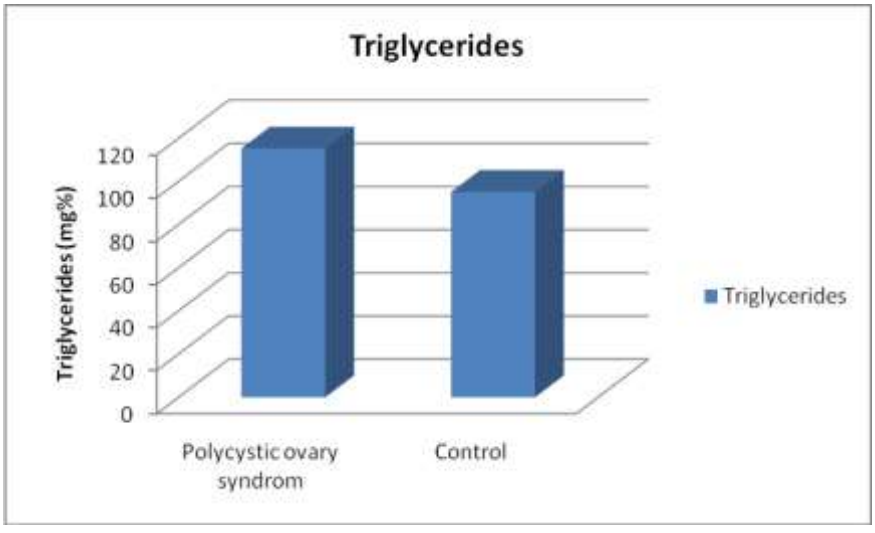

Figure 1: A diagram showing the distribution of mean of Triglyceride in PCOS and control.

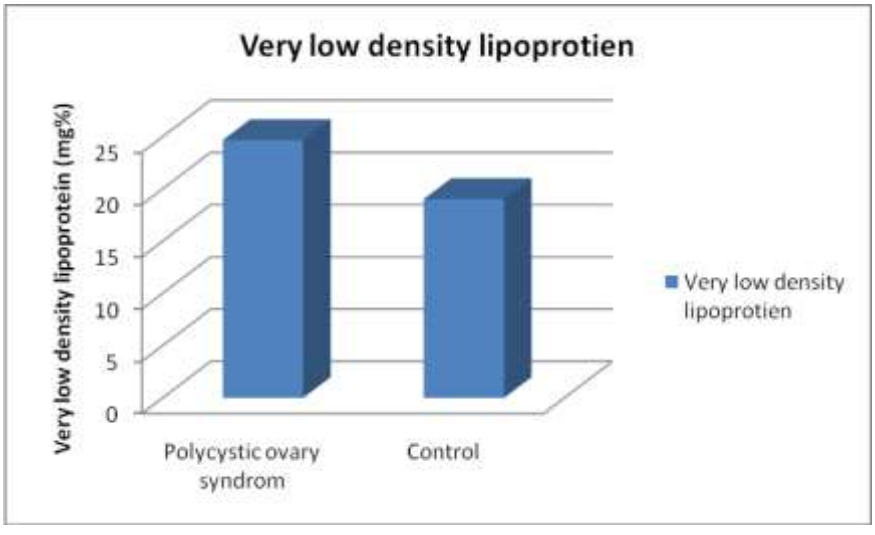

Figure 2: A diagram showing the distribution of mean of VLDL in PCOS and control.

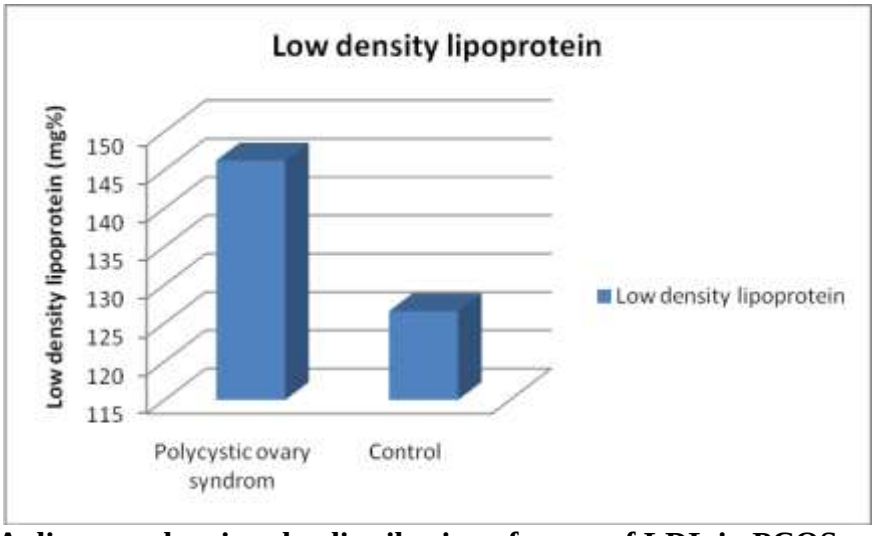

Figure 3: A diagram showing the distribution of mean of LDL in PCOS and control.

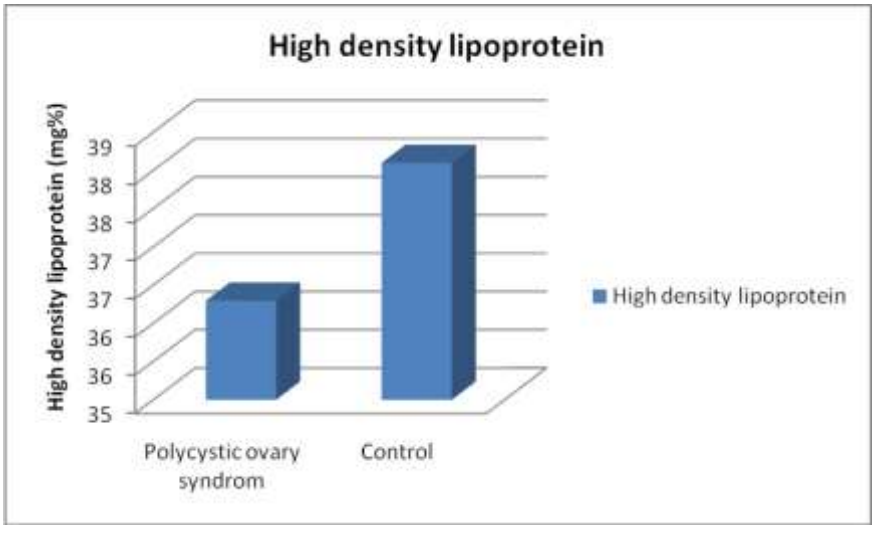

Figure 4: A diagram showing the distribution of mean of HDL in PCOS and control.

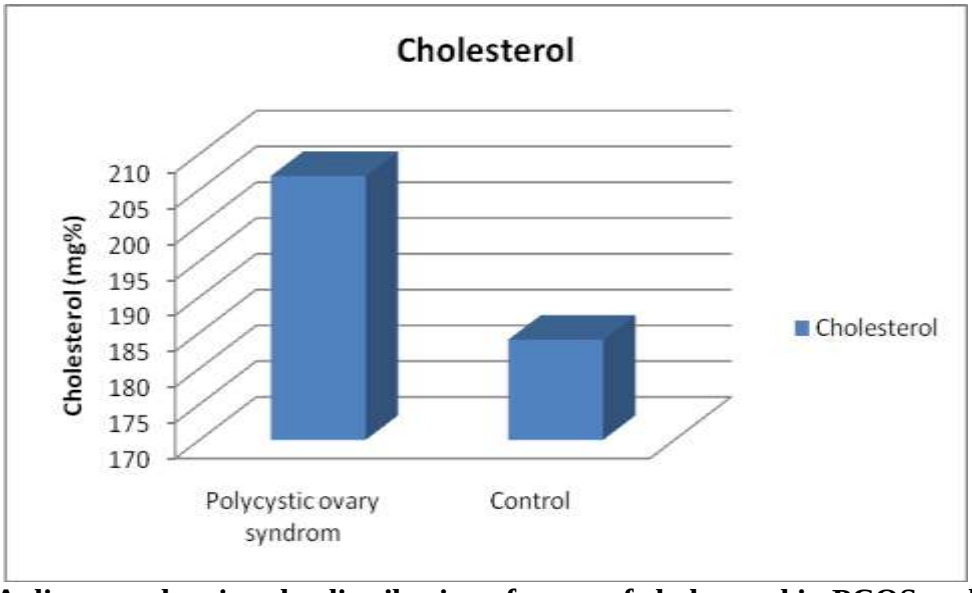

Figure 5: A diagram showing the distribution of mean of cholesterol in PCOS and control.

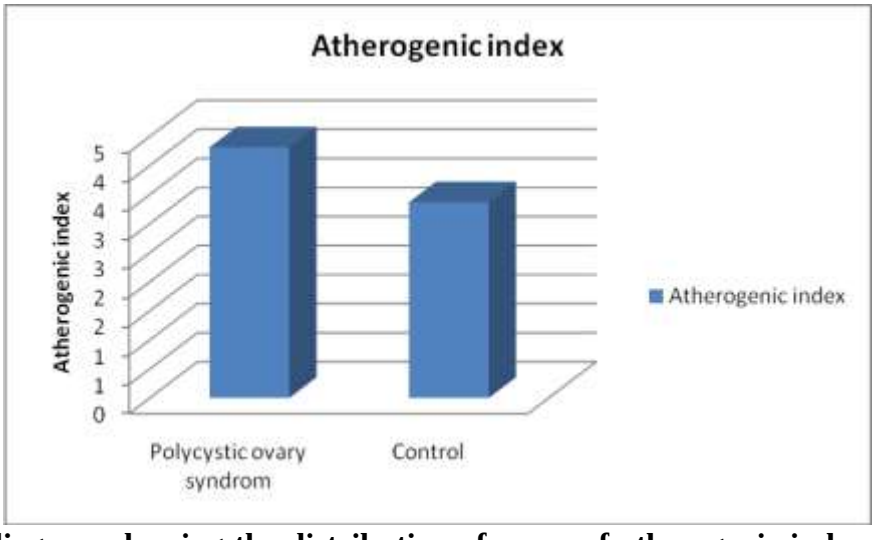

Figure 6: A diagram showing the distribution of mean of atherogenic index in PCOS and control.

DISCUSSION

The present study confirms the presence of amore atherogenic lipid profile in women with PCOS. There were high levels of TG, Cholesterol, and LDL in combination with low level of HDL and with an increase in atherogenic index in women with PCOS .These data are consistent with prior studies of dyslipidemia in women with PCOS (10, 14).

Most studies of dyslipidemia and PCOS have reported on cholesterol levels and TG. The lipid profile that is found in women with PCOS consists of elevated TG levels together with low levels of HDL (15).Increased secretion of VLDL particles by the liver results in elevated plasma TG concentrations. Subsequently, TG is exchanged for cholesteryl ester by the activity of cholesteryl ester transfer protein.

This process results in TG-enriched HDL particles that are catabolised more rapidly, and

cholesteryl ester-enriched VLDL particles that are converted into small dense LDL particles (10, 16).

Lipid metabolism in women with PCOS may also be affected by ovarian and-or adrenal secretion of sex-steroid. The effects of sex-steroids on lipid metabolism are complex and involve the actions of both androgens and estrogens. Hyperandrogenism has been associated with increased hepatic lipase (HL) activity.

This enzyme, that has a role in the catabolism of HDL particles (partially responsible for the hepatic removal of the HDL particles) exhibits strong dimorphism with exogenous androgens up regulating, and estrogens down-regulating its activity (17, 18).

This lead to: increased conversion of intermediate-density to LDL and of HDL to the smaller fractions (which was catabolized faster), decrease elimination of circulating LDL and reduced formation of HDL (19).

However some groups found a marked increase in the prevalence of cardiovascular risk factors in the PCO group, including hypertension, diabetes, hypercholesterolemia, hypertriglyceridemia, and elevated waist-to-hip ratio (20).

In summary, the high atherogenic lipid profile, in particular related to HDL metabolism, was found in women with PCOS, both obesity and hyperandrogenism contribute to these changes, and there was evidence for an additional influence of PCOS on lipid metabolism.

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