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EVALUATION OF LIPID PROFILE IN IRAQI WOMEN WITH POLYCYSTIC OVARY SYNDROME

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ABSTRACT

Polycystic ovary syndrome is a condition characterized by menstrual abnormalities, hersutisim, infertility, sometimes obesity and the present of multiply small cysts in the ovary. **Aim of Study:** This research was conducted to find the changes in lipid profile and in women with polycystic ovary syndrome(PCO), these parameters include, total serum cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), atherogenic index (AI). **Patient and Method:** The number of subjects included in this study were 80 patients there age was between 19-43 years with mean ± SD (27.63+5.53) the diagnosis of these patients was established by a gynecologist, and the control group consist of women there age was between 22-41 years with mean±SD (31.92+5053). The patients were selected from the infertility center in AL-Batool Teaching Hospital /Mosul City, during a period from 1" of October 2018 to the 1st of April 2019. Different statistical tests used were unpaired Z-test, Chi square, the sensitivity and specificity were measured and compared with the two groups to find the P-value. **Results:** Results Significant differences were detected at P<0. 001 in TC and LDL-C, P<0.01 for AI and non-significant P>0.05 were detected in TG, HDL-C. **In conclusion:** Hyperlipidemia could occur in women with polycystic ovary syndrome, especially those who are obese which is a risk factor for the development of cardiovascular disease.

KEYWORDS: Polycystic ovary syndrome, Cholesterol, Triglyceride.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common but a complex endocrine disorder affecting women in their reproductive age, it is a major cause of anovulation and consequent subfertility leading affected women to attend. Gynecological or infertility clinics,[1] Interest in PCOS has increased recently with the realisation that this syndrome involves far more than the reproductive system. Initially called the Stein-Leventhal syndrome after its researchers in the 1930s, PCOS is now recognized to be a metabolic syndrome which may include, hyperinsulinaemia, hyper-lipidemia, diabetes mellitus (DM) and possibly cardiac disease, as well as increasing androgen levels, anovulation infertility, endometrial cancer and obesity.[2]

Definition

In 2003 a consensus workshop sponsored by European Society of Human Reproduction and Embryology in Rotterdam indicated PCOS to be present if 2out of 3 criteria are met.

Oligoovulation and/or anovulation-1. Excess androgen activity 2- 3-Polycestic ovaries (by Ultrasound) and other causes of PCOS are excluded. The Rotterdam definition is wider, including many more patients.[3]

Epidemiology

The prevalence of PCOS among women of reproductive age in the general population has been estimated 4% to 12%, the prevalence of PCOS appears to be higher from 37% to 90% in women with menstrual abnormalities.[4] The exact cause of PCOS remains unknown, but it is believed to involve a complex interplay of genetic and environmental factors, including diet and lifestyle.[5] In Iraq, women are suffering from PCOS, and many cases have been recorded in different areas every year. Iraqi studies showed that several factors may increase PCOS

risk in Iraqi women, such as obesity, genetic factors, environmental changes, family history.[6]

Clinical featuers of PCO

1- Menstrual irregularity and infertility: Chronic anvulation often presents as oligomenorrhea, amenorrhea, dysfunctional uterine bleeding and/or infertility, however, around 20% of women with PCOS may have a normal menstrual cycles.[7]

2-central obesity: Obesity presents in about 49% of women with PCOS, this figure varies somewhat depending on ethnicity and geography, when presents obesity worsens IR, and increases the risk for diabetes and cardiovascular disease, however weight loss in such patients is difficult to achieve due to impairment of adipocyte lipolysis which in turn is linked to IR.[8]

3-Hyperandrogenism: May present clinically as hirsutism (growth of coarse hair on a women in male pattern), or acne, androgenic alopecia, virillization (clitoromegaly, deep voice and increase musculature) due to elevated serum levels of androgens.[9],[10]

4-Other clinical features: Acanthosis nigricans (dark patches of skin found on the neck, arm or thighs), acrochordons (tiny flaps of skin), sleep apnea.[1]

1-Laboratory finding[2]

1-1-FSH and LH: As a rule, endocrine testing is probably best performed in the 3" day of the cycle after a spontaneous or included menses and usually after an overnight fast. Usually FSH is within the reference range or low, LH is elevated so the LH/FSH is usually greater than 3.

1-2-Total and free testosterone levels: Total testosterone is elevated in women with PCOS however, total testosterone levels greater than 200ng/dl is suggestive of an androgen producing tumor of the ovary or adrenal gland, free testosterone are sensitive for ovarian hyperandrogenism and are elevated in patients with PCOS. SHBG is usually low.

1-3-Dehydroepiandrosterone sulphate: DHEAS is a markers of adrenal androgen production, DHEAS levels greater than 700µg/dl are suggestive of adrenal tumor. DHEAS is elevated in women with PCOS but not at such high levels Androstenedione also is elevated.

1-4- Lipid profile test: Assessment of lipid states is important especially in obese women with PCOS. Many studies have reported that LDL-C is increased in women with PCOS, which is usually not noted in insulin resistant states. The reason why LDL-C in also increased in women with PCOS in not clear yet, but increased LDL-C levels in women with PCOS may be related to hyperandrogenism or genetic factor.[13] Dyslipidemia and insulin resistance (IR) are some of the most common features observed in women with this syndrome IR affects approximately 65–70% of women with PCOS, and roughly 70–80% of women with PCOS are overweight or obese[14] PCOS frequently has dyslipidemia, which is characterized by greater triglycerides and reduced high density lipoprotein (HDL) cholesterol.[8] Although dyslipidemia is independent of BMI, obesity, and insulin resistance.[15]

Long-term squeals and risks in PCOS

1-Dyslipidemia (disorders of Lipid metabolism)

Dyslipidemia is common in PCOS. Beyond known alterations in triglycerides and HDL-cholesterol, women with PCOS have higher LDL-cholesterol and non HDL-cholesterol, regardless of BMI. We recommend that all women with PCOS be screened for dyslipidemia, including LDL-cholesterol and non HDL-cholesterol determinations, for effective cardiovascular risk prevention.[16]

These findings vary depending on body weight, diet and ethnicity. Hyperandorgenism probably plays some role in theses abnormalities, but hyperinsulinemia as a result of IR seems to be the more dominant factor.[17]

Normally, hormone-sensitive lipase in the adipose tissue is inhibited by insulin. In IR, lipolysis is not fully suppressed, there is a flow of large quantity of non esterified fatty acids (NEFA) from adipose tissue to the liver, even during post prandial period. This is a major stimulus for hepatic triglyceride synthesis and production of very low density lipoproteins (VLDL), increase hepatic output of VLDL continues in the postprandial state and competes with exogenously derived TG carried in chylomicrons for the clearing factor lipoprotein lipase, as a result, there is saturation of lipoprotein lipase and accumulation of TG- rich lipoproteins and prolonged postprandial lipemia.

The lipemia stimulates lipid transfer via cholesterol ester transfer protein (CETP) with the exchange of TG for cholesterol ester between TG-rich lipoproteins and lipoproteins of higher density, as a result VLDL remnants become enriched with cholesterol while LDL and HDL become enriched with triglyceride both of triglyceride-rich LDL and HDL are substrate for hepatic lipase which hydrolyses TG, producing small dense LDL and HDL, small dense HDL are catabolised more rapidly leading to lower plasma HDL concentration.[18]

2-Impaired glucose tolerance and type II diabetes mellitus (DM): Because of insulin resistance, most women with PCOS have increased risk for impaired glucose tolerance and overt type2 DM. A recent study found that 31% of obese women with PCOS had impaired glucose tolerance and that 7.5% have overt DM, in addition 10. 3% of non-obese women with PCOS had impaired glucose tolerance and 1.5% had DM at a rate almost three times that of the general population.[19]

3- cardiovascular disease (CVD): It has been estimated that myocardial infarction is seven times more likely in patients with PCOS, this may be due to the metabolic disturbance associated with PCOS.[20]

4- cancer: Women with PCOS assumed to be at increased risk of endometrial adenocarcinoma[30], but as association between ovarian cancer and PCOS seems be unlikely, but a link between PCOS and breast cancer appears probable.[21]

6- non alcoholic fatty liver disease: Non-alcoholic fatty liver disease (NAFLD) and PCOS are both associated with IR. Thus women with PCOS may have an increased prevalence of NAFLD, including non-alcoholic steatohepatitis (NASH).[22]

Treatment

since women with PCOS are generally young (pre-menopausal), much of the management in PCOS has typically focused on addressing current symptoms such as the ovulatory dysfunction and infertility, and not on the long-term risks conferred by PCOS, such as diabetes and cardiovascular disease (CVD).[23]

The choice of treatment for women with PCOS depends on the symptoms with which a patient presents. Symptoms typically fit into three categories: menstruation related disorders; androgen-related symptoms; and infertility. Management.

The first line of treatment in patients with PCOS should be the improvement in lifestyle. In overweight and obese patients, weight loss due to changes in diet and physical activity decreases serum insulin and androgen levels and reduces the risk of developing glucose intolerance and type 2 diabetes. Pharmacological interventions are indicated in the presence of IR/glucose intolerance or dyslipidemia that persist after lifestyle modifications.[24]

Treatment of PCOS should be proposed not only to alleviate symptoms but also to prevent the occurrence of long-term complications. Combined oral contraceptives and antiandrogens are the standard care to reduce androgen levels and treat symptoms while providing endometrial protection.[25]

MATERIALS AND METHODS

This study represents a case-control study, the subject included were divided into two groups.

1-Control group

Consisting of eighty females, age ranged from 22-41 years with a mean ± SD of 31.92+5.53.

They were selected from relatives and attendants with outpatient clinic, who had regular menstrual cycle, no evidence of androgen excess and no history of infertility.

2-Case Group

Consist of 80 patients with age ranged from 19-43 years with a mean± SD of 27.63+5.36.

The patients were first examined by a gynaecologist who conducted a clinical examination before refering the suitable patients for this study. All patients in this group have been diagnosed as having PCOS by hormonal assestment, ultrasound and clinical examination. They were regular attendants at AL-Batool Teaching Hospital, consultant room for treatment and follow up.

The subjects of both groups were interviewed and general information was taken to fill the questioneire, including name, age, occupation, residence and gynecological history. The study was conducted during the period from the 1" of October 2018 to the 1 of April 2019.

After an overnight fast, a sample of 4 ml of venous blood was drawn from case and control groups, blood was clotted and put in centrifuge for 10 minute the lipid profile was assessed by cobas c 111 analyzer[26] is a continuous random access analyzer intended for the in vitro determination of clinical chemistry parameters in serum utilizing photometric analysis.

Statistical Analysis

The following Statistical methods were used for the analysis of the data.[27]

1- Standard statistical method were used to determine the mean, standard deviation (SD) and range (minimum – maximum).

2- Z test was used to compare the results for various biochemical parameters among subjects in different groups.

3- Chi- squared test using 2×2 table was used to compare any two groups.

Differences between observation were considered significant at P≤ 0.05.

RESULTS

1- The demographic and clinical characteristics of the study are presented in tables(1),(2)

The results of data analyzed are arranged according to the grouping of the subjects enrolled in this study.

The subjects were classified into two groups.

Group 1 (control) consist of eighty females, age ranged from 22-41 years with a mean ±SD of 31.92±5.53.

Group 2 (case): consist of eighty patients diagnosed with PCOS age ranged from 19-43 years with a mean±of 27.63±5. 36.

Table 1: Demographical Characteristics of Case Group.

Characteristics		Control (n=80)		Case (n=80)		p-value
		No.	%	No.	%	
Age (year)	≤25	30	37.0	33	41.30	<0.01
	26–30	22	27.0	24	30.0	
	31-35	14	18.0	18	22.5	

	36-40	12	15.0	3	3.80	
	>40	2	3.0	2	2.50	

Table 2. clinical characteristics of case group.

Characteristics		No.	%
Infertility	primary	48	60.00
	secondary	32	40.00

2-Biochemical parameters in the studied groups (control and case).

Different biochemical parameters were measured in both groups including: TC, TG, HDL-C, LDL-C, and atherogenic index (AI).

The results are presented as±SD in table. (3)

Table 3: Lipid parameters in case, control groups.

Biochemical parameters	Control (n=80)		Case (n=80)		P-value
	Mean ±SD	Range	Range	Mean ±SD	
TC(mmol/L)	4.73±0.64	3.70±6.30	5.34±0.53	4.60±7.10	<0.001
TG(mmol/L)	1.43±0.30	0.91±1.92	1.63±0.57	0.70±3.30	>0.05(NS)
HDL(mmol/L)	1.10±0.23	0.80±1.60	1.07±0.26	0.67±1.81	>0.05(NS)
LDL(mmol/L)	3.03±0.47	2.30±3.90	3.50±0.60	2.30±5.60	<0.001
AL	4.47±0.62	3.30±5.50	5.06 ±1.24	2.80±8.00	<0.01

When these different biochemical parameters in the control and cases were compared, highly significant difference (P<0.001) were notice in TC and LDL-C a significant difference (P <0.01) was detected in the AL, while no significant difference (P>0.05) was detected in the TG and HDL-C.

3-: Lipid Profile

Lipid profile in this study was represented as: TC, TG, LDL-C, HDL-C and AL.

According to the third adult treatment panel report (ATP III) released by the national cholesterol education program (NCEP)[28], the cut – off level for TC is 5.17 mmol/L m for TG is 1.70 mmol/L, for LDL-C is 3.34 mmol/L and for HDL-C is 1.03mmol/L, and any value above the cut-off level is considered as abnormally high.

Using Z-test between two proportions for TC, TG, HDL-C, LDL-C and AL the relation are as following.

3-1 the relation between serum total cholesterol(TC) in both groups.

According to NCEP reference range, TC level was classified into three categories desirable, borderline and high risk, as showen in table (4).

Table 4: Relation of Total Cholesterol in Case and Control Groups.

Total cholesterol(mmol/l)	Control(n=80)		Control(n=80)		p-value
	No.	%	No.	%	
Desirable <5.17	62	77.0	38	47.5	<0.001
Borderline 5.17-6.18	16	20.0	37	46.3	<0.001
High risk>6.20	2	3.0	5	6.3	>0.05(NS)

NS =not significant according to Z-test between two proportions.

There was significant difference between the two groups in the desirable and borderline category at (p<0.001).

3-2 The relation between serum triglyceride level in studied groups.

According to NCEP referenc range, TG level was Classified into three categories normal, borderline and high, as in table (5).

Table 5: Serum Triglyceride In Studied Groups.

	Control (n=80)		Case (n=80)		
TG(mmol/L)	No.	%	No.	%	
Normal <1.70	60	75.0	43	53.8	>0.05(NS)
Borderline high 1.70-2.25	20	25.0	28	35.0	>0.05(NS)
High 2.26-5.64	0	0.0	9	11.3	<0.05

There was a significant different between the two groups in the high category.

3-3 the relation between LDL-C level in studied groups.

According to NCEP reference range, LDL-C level was classified into five categories optimal, near optimal, birderline, is and very high, as shown in the table (6).

Table 6: Relation Between Ldl-C In Studied Groups.

LDL-c (mmol/L)	Controls (n=80)		Cases (n=80)		p-value
	No.	%	No.	%	
Optimal <2.58	28	35.0	3	3.8	<0.05
Near optimal 2.58-3.33	38	48.0	31	38.8	>0.05(NS)
Borderline high 3.34-4.11	14	17.0	34	42.5	>0.05(NS)
High 4.12-4.88	0	0.0	10	12.5	0.01
very high ≥4.89	0	0.0	2	2.5	>0.05(NS)

There was significant differences between the two groups in the optimal ad high category.

3-4 the relation between HDL-C level in studied groups.

According to NCEP reference range, HDL-C level was classified into three categories normal, borderline and high, as shown in the table (7).

Table 7: The relation between HDL-C in studied groups.

HDL-c (mmol/L)	Controls(n=80)		Cases (n=80)		p-value
	No.	%	No.	%	
<1.03	20	25.0	42	52.5	>0.05(NS)
1.03-1.54	48	60.0	33	41.3	>0.05(NS)
>1.55	12	15.0	5	6.3	>0.05(NS)

There was no significant difference between the two groups.

3-5 the relation between AL level in studied groups.

According to NCEP reference range, AL level was classified into normal level and high risk, as shown in the table (8).

Table 8: The Relation Between AI In Studied Groups.

Atherogenic index	Case (n=80)		Control (n=80)		
	No.	%	No.	%	
Normal <5	70	88.0	38	47.5	<0.001
High ≥5	10	12.0	42	52.5	

There was significant difference between the two groups in both categories at P<0.001.

3-6 the relation between serum total cholesterol (TC) and PCOS.

Using 2×2 contiguous table, 42(52.5%) out of 80 subjects in the case group were in the high risk category (TC>5. 17 mmol/L) whereas 62(78.0%) subjects in the control group were in the low risk category (TC≤5. 17 mmol/ L) giving a high significant difference (P<0.001), as showen in table (9).

Table 9: the relation between TC and PCOS.

		+		-	
		No.	%	No.	%
TC (mmol/L)	>5.17	42	52.5	18	22.0
	≤5.17	38	47.5	62	78.0
Sensitivity (%)		53.0			
Specificity (%)		84.0			
p-value		<0.001			

3-7: The Relation Between Serum Triglyceride (TG) and PCOS.

Most subjects in both groups were in the risk category (TG≤1.7 mmol/L), as shown in table (10).

Table 10: relation between TG and PCOS.

		+		-	
		No.	**%**	**No.**	**%**
TG (mmol/L)	>.17	37	46.3	20	25.0
	≤1.7	43	53.8	60	75.0
Sensitivity (%)		46.0			
Specificity (%)		80.0			
p-value		<0.001			

3-8: the relation between LDL-C and PCOS.

Most subjects in the case group where in the high risk category (LDL-C>2.58)as shown in table (11).

Table 11: Relation Between LDL-C and PCOS.

		+		-	
		No.	**%**	**No.**	**%**
LDL (mmol/L)	>2.58	77	96.3	52	65.0
	≤2.58	3	3.8	28	35.0
Sensitivity (%)		96.0			
Specificity (%)		16.0			
p-value		<0.05			

3-9 the relation between HDL-C and PCOS.

Most subjects in the case group were in the high risk category (HDL-C<1.03 mmol/L), as shown in table (12).

Table 12: Relation Between HDL-C and PCOS.

		+		-	
		No.	**%**	**No.**	**%**
HDL-C (mmol/L)	>1.03	42	52.5	20	25.0
	≥1.03	38	47.5	60	75.0
Sensitivity (%)		53.0			
Specificity (%)		60.0			
p-value		>0.05(NS)			

DISCUSSION

PCOS is a condition characterized by hyperandrogenism and chronic oligo-anovulation, however many features of the metabolic syndrome are present in the majority of women with PCOS which lead them to in an increase risk for CVD. The American Heart Association and National Heart, lung and blood institute recommended that the metabolic syndrome is identified with the presence of three or more of these components.

*Elevated waist circumference greater than 35 inches.
*Elevated TG ≥150 mg/dl.
*Reduced HDL-C < 50 mg/dl
*Elevated blood pressure 130/85mmHg.
*Elevated fasting glucose level > 100mg/dl[29]

Effect of PCOS on lipid metabolism

Many women with PCOS have significant dyslipidemia with elevated TC, TG and LDL-C and low HDL-C than age and weight match controls, at least one abnormal lipid level is seen in 70% of women with PCOS.[30] The reference value of all lipid parameters in the present study were obtained from recent update levels stated by the ATP III released by the NCEP.[28] The present study showed a significant difference in the mean of TC between case and control groups (P< 0. 001) as in table (3), al 46. 3% of women in the case group were in the borderline category as table (4) with (P<0. 001). Concerning LDL-C there was significant difference in the mean (P< 0. 001) between the case and control group in table (3), were 12. 5% of women in the case group were in the high risk category as demonstrated in table (6), these results were in agreement with a study done by Mayer, et al., [31] where TC and LDL-C levels increased significantly (P< 0. 05) in women with PCOS also in agreement with another study done by Talbott, etal.[32], but it differed from a study by Bickerton, etal.[28], where there was no significant difference in lipid profile concentrations between case and control groups.

Concerning TG and HDL-C levels in this study there were no significant difference in the mean (P>0.05) between both case and control groups as in table (3), most of the women with PCOS were within the normal range as shown in table (5) and (7) respectively. This is in agreement with a study by Bickerton, etal., (20), but different from other studies by Talbott, et al.,[32] which stated that there is a significant (P<0.05) reduction in HDL-C level and a significant elevation in TG level (P<0.05) in women with PCOS compared to control group.

These results highlight the fact that the combination of these l parameters are known to be atherogenic in women in the PCOS increases the risk of CVD and this cannot be explained solely by obesity one possible explanation is that the typical disturbance of lipid parameters seen in PCOS is associated with the presence of IR, although hyperandrogenism may affect lipoprotein and lipid metabolise independently of insulin levels and obese body weight.[20]

CONCLUSION

1- PCOS is a common condition characterized by menstrual abnormalities and clinical and biochemical features hyperandrogenism.
2-PCOS cause a significant increase in lipid profile paramete especially TC and LDL-C were the predominant lipid abnormaty in women with PCOS independent of obesity.
3-the difference in the mean ± SD was highly significant (P<0.001) for TC and LDL-C, a significant difference was detected in AL, no significant difference (P>0.05) was detected in the TG and HDL-C.
4-PCOS is associated with biochemical risk factors for premature vascular disease, which cannot be explained by obesity alone.

RECOMMENDATION

1-women with PCOS should be educated about their disease, lifestyle changes are a first-line intervention in women with PCOS who are overweight because weight loss may help to decrease insulin resistance and also may decrease adverse long-term cardiovascular effects and can lead to resumption of ovulation within weeks.
2-Nutritional counseling is an important aspect of medical management by avoiding saturated fat, trans fat and increasing mono saturated oils and omega 3 oils, and using high ratio of soluble fiber to insoluble fiber and taking smaller and more frequent meals by having mixed meals of carbohydrate, protein and fat.
3-Testing for hyperlipidemia is recommend, especially for obese women with PCOS.
4- Evidence suggests lifestyle modification should be the first line of therapy for women with PCOS. Several studies have examined the impact of exercise interventions on reproductive function, with results indicating improvements in menstrual and/or ovulation frequency following exercise.

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