

Total L-carnitine and insulin resistance in non-obese and obese Iraqi women with polycystic ovary syndrome

DOI: https://doi.org/10.32007/jfacmedbagdad.6512040.

Maad M. Shallal *	FICOG
Najmah M. Meran*	FICOG, CABOG
Zina A. Hussein**	FICOG

\odot \odot

This work is licensed under a Creative Commons Attribution-Noncommercial 4.0 International License

Abstract:

Background: Polycystic ovary syndrome (PCOS) is one of the most frequent endocrine illnesses affecting reproductive - age women. L-carnitine has important roles in oxidative stress, energy production, and glucose metabolism. It affects insulin resistance as decreased plasma carnitine level has been well reported in type II diabetes mellitus. Hence, it means L-carnitine may reduce insulin resistance which is found in PCO disease.

Aim of the study: This study aims to measure the level of L-carnitine and insulin resistance in both obese and non- obese patients with PCOS.

Patients and Methods: Sixty women within the reproductive age with PCOS (30 obese and 30 nonobese) were recruited from the Gynecology and Obstetrics Outpatient Clinic in Baghdad Teaching Hospital from June 2016 to June 2017. The data collected for each case included: Height, weight, waist circumference, blood pressure, obstetrical, medical, and medication history as well as ultrasound results. A physical examination was done to evaluate the clinical signs of hyperandrogenism. Biochemical measurements included fasting blood sugar, leutinizing hormone, follicular stimulating hormone, Testosterone and lipid profile were measured together with total L-carnitine (using L-Carnitine Assay Kit Sigma-Aldrich Co.). Insulin resistance was diagnosed according to National Cholesterol Education Program/Adult Treatment Panel III (NCEP/ATP III). PCOS is diagnosed according to the Rotterdam criteria.

Results: This study revealed that insulin resistance (IR) was present in 51.7% of PCOS patients, which was higher in obese PCOS patients (73.3%) than in the non-obese (30%). Age of patients, serum cholesterol, LH, and FSH were not related to IR. High mean BMI, waist circumference, FBS and triglyceride were significantly associated with IR (p < 0.05), while low serum HDL and L-Carnitine were associated with IR (p < 0.05). The mean serum total L-carnitine in this study was 34.03µmol/L. Obese women had lower carnitine levels than non-obese women and low serum L-Carnitine was associated with IR. Serum triglyceride, FBS and testosterone were correlated negatively with serum L-carnitine (p < 0.05) and serum HDL correlated positively with serum L-carnitine (p value = 0.001). **Conclusions:** The mean value of serum total L-carnitine among the non-obese PCOS women was

Conclusions: The mean value of serum total L-carnitine among the non-obese PCOS women was higher than among the obese ones. Low serum L-carnitine is associated with insulin resistance.

Keywords: L-Carnitine, Insulin resistance, Obesity, Women, PCOS.

Introduction:

PCOS is a complex condition that is most often diagnosed by the following criteria: Hyperandrogenism, ovulatory dysfunction and polycystic ovaries, infertility and obesity. However, obesity is not observed in all women with PCOS. The prevalence of obesity in women with PCOS ranges from 1% to 80% across studies and varies with the definition of obesity as well as with women's race and cultural group. Approximately half the adult women with PCOS are overweight or

*Corresponding Author: Dept. of Obstetrics and Gynecology, college of Medicine, University of Baghdad <u>najmah@comed.uobaghdad.edu.iq</u> <u>maadmahdishalal@comed.uobaghdad.edu.iq</u> ** University of Mustansyria college of medicine Dept. of Obstetrics and Gynecology <u>zinaabdullah@uomustansiriyah.edu.iq</u> Obese (1) The prevalence of type II diabetes and insulin resistance has been reported to be higher among women with PCOS. (2, 3) Women with PCOS have higher levels of total cholesterol, verylow-density lipoprotein, LDL, and triglycerides, and lower levels of high-density lipoprotein (HDL) compared with healthy women. (2) These endocrine and medical disorders associated with PCOS: Dyslipidemia, type II diabetes, obesity, and hypertension which are established risk factors for cardiovascular disease. Chronic anovulation, hyperandrogenemia, and insulin resistance are also associated with increased cardiovascular risk. (4) Several studies have suggested a link between PCOS and different types of malignancies (eg: endometrial, breast, and ovarian cancers) (5), but these studies were generally small, retrospective, and did not employ adequate controls.

J Fac Med Baghdad 2023; Vol.65, No. 1 Received: Dec, 2022 Accepted: Mar.., 2023 Published: April 2023 "At a recent joint Rotterdam ESHRE/ASRM (European Society of Human Health Reproduction and Embryology/American Society for Reproduction Medicine) consensus meeting a refined definition of the PCOS was agreed: Namely the presence of two out of the following three criteria: (6) Oligo- and/or anovulation,

hyperandrogenism (clinical and /or biochemical) and polycystic ovaries on ultrasound examination". (7).

Acetyl-L-carnitine is involved in both anabolic and catabolic metabolic pathways in the body. Carnitine is important for energy balance across cell membranes and in the energy metabolism of tissues that rely heavily on fatty acid oxidation for energy, such as skeletal and cardiac muscles. Although carnitine is most known for its involvement in the metabolism of carnitine-free fatty acids, it also improves carbohydrate consumption. (8) D- and Lcarnitine, Acetyl-L-carnitine, and propionyl-Lcarnitine are all molecules that are referred to as carnitine. Only one of the two forms (L-carnitine, not D-carnitine) is physiologically active, meaning it can be used by the body. (9)

Oxidative stress, energy production and glucose metabolism all require L-carnitine. L-carnitine has the ability to maintain mitochondrial membranes, enhance energy supply to the organelle, and protect cells from apoptosis. Since the involvement of Acyl-CoA derivative accumulation in the development of insulin resistance was hypothesized, the use of carnitine in the therapy of IR has gotten a lot of interest. (10)

The beneficial effect of L-carnitine on ovulation quality and rate of pregnancy, as well as on the biochemical profile of PCOS patients, appear to support its usage in clinical practice. The purpose of supplementing with L-carnitine during the follicular phase is to reduce reactive oxygen species (ROS) and function as a scavenger for detrimental oxidative stress compounds collected during earlier cycles of ovulation induction.(11) A better effect on body weight and lipid metabolism improves these patients' quality of life much more. Thus because of its positive effects and triple impacts on lipid metabolism, oxidative stress, and metabolism of glucose, this medication could be used as a first-line therapy for PCOS. (10, 11)

This study aims to measure the level of L-carnitine and insulin resistance in both obese and non- obese patients with PCOS.

Patients and methods

This study was carried out at the Gynecology and Obstetrics Out-Patient Clinic of Baghdad Teaching Hospital. Sixty ladies with PCOS were recruited from June 2016 to June 2017, 30 obese women and 30 non-obese women with PCOS. L-carnitine and IR were measured for all sample members. The data collected from each sample member were: Height, weight, waist circumference, blood pressure, obstetrical history, medical history, medication history and ultrasound results. A physical examination was done to detect the clinical signs of hyperandrogenism. The Rotterdam criteria were used to diagnose PCOS, with at least two of the three criteria: Ovulatory disturbance (oligomenorrhea or amenorrhea); hyperandrogenism, and an ovarian volume larger than 10 ml by ultrasound.

Insulin resistance (IR) was diagnosed according to the National Cholesterol Education Program/Adult Treatment Panel III (NCEP/ATP III) criteria for metabolic syndrome(12) when three or more criteria are present:

Waist circumference > 88 cm.

Fasting triglyceride level of 150 mg/dL or more.

Blood pressure level of 130/85 mm Hg or more.

High-density lipoprotein level of < 50 mg/dL.

Fasting glucose level of 110 mg/dL or more.

Exclusion criteria included:

Age below 16 years and above 45 years. Women taking drugs known to affect weight loss or metabolism of carbohydrate and lipid.

Endocrinopathies including diabetes, androgen secreting tumors, Cushing's syndrome, non-classical 21-hydroxylase deficiency, hyperprolactinemia, thyroid dysfunction.

Alcohol consumption, smoking and use of all drugs which change sex hormones, carnitine metabolism, lipoprotein, or insulin secretion or action.

Consent from all women was obtained.

Except for L-carnitine, all biochemical measures were done on the same day. The samples were kept at 2-20° C until they were tested for total L-carnitine levels. Biochemical tests: Blood samples were taken between the third and seventh days after spontaneous menstrual bleeding, following an overnight fast. Samples were collected by vein puncture. Samples were allowed to clot and kept undisturbed for about thirty minutes and then centrifuged at 400 rpm for 10 minutes at room temperature. The serum was separated, the samples were tested for glucose immediately after separation, and the remainder was stored at $- 20^{\circ}$ C for future analyses.

The following measurements and investigations were done:

Waist circumference and hip circumference was measured with tape measure

BMI was calculated with the following reference values: < 18.5 as under-weight, 18.5 - 24.99 as optimal weight, and ≥ 25 as obesity / overweight.

Plasma glucose concentrations, with the reference normal values being <110 mg/dl, 111 - 125 mg/dl being impaired, and $\geq 126 \text{ mg}$ / dl considered diabetes as stated by World Health Organization guidelines

Luteinizing Hormone (LH) and Follicle Stimulating Hormone (FSH) levels using commercial enzymelinked immunoassay (EIA) kits. Values of FSH between 3.2-15 ng/ml and of LH between 1.2-12.5 ng/ml were regarded as normal. Testosterone level using electrochemiluminescence's immunoassay value between 0.1-0.9 ng/ml was regarded as normal.

Lipid profile: Triglycerides, cholesterol, and HDL were tested with a fully automated analyzer using established enzymatic procedures

Ultrasound: was done by an expert radiologist in the department of Radiology-Baghdad Teaching Hospital.

Statistical analysis: The two study groups were agematched. The statistical package SPSS-20 (Statistical Packages for Social Sciences- version 20) was used for data analysis. Simple frequency, percentage, mean, and standard deviation measurements were used to present the data. The ttest and chi- square were used, with a P value of <0.05 being considered significant.

Results

The mean \pm SD age of the non-obese group was 26.6 \pm 4.87 and for obese group 28.9 \pm 5.46. The mean \pm SD of BMI of the non-obese group was 22.9 \pm 1.54 Kg/m² (range: 19.5-24.9) and for obese group 28.9 \pm 2.96 Kg/m² (range: 25.20-34.60). The mean \pm SD of waist circumference for the non-obese group was 82.1 \pm 5.11 and for the obese group was 82.6 \pm 7.08. The mean \pm SD of FBS for the non- obese was 97.6 \pm 15.35 mg/dL and for the obese 128.1 \pm 56.70 mg/dL, tables1 and 2.

Table 1: Descriptive statistics of all studied women	with	PCOS	(N=60)
--	------	-------------	--------

Variables	Minimum	Maximum	Mean	SD
Age (Years)	18	40	27.8	5.26
BMI (Kg/m²)	19.50	34.60	25.9	3.81
Waist circumference (cm)	71.0	95.0	82.7	6.13
FBS (mg/dl)	74	325	112.9	43.97
Cholesterol (mg/dl)	159	330	235.7	41.6
Triglyceride (mg/dl)	107	220	148.3	30.83
HDL (mg/dl)	21	68	43.8	12.09
LH (ng/ml)	6.0	17.8	11.5	2.64
FSH ng/ml)	3.1	8.8	5.5	1.36
LH/FSH Ratio	1.60	2.60	2.1	0.18
Testosterone (ng/ml)	0.3	3.3	1.6	0.71
Total L-Carnitine (µmol/L)	8.5	67.3	34.0	17.26

Table 2: Description of the obese and non-obese PCOS study groups

Variable	Study Groups	Min	Max	Mean	SD	P value
Age (Years)	Non-obese	18	39	26.6	4.87	0.095
	Obese	18	40	28.9	5.46	
BMI (Kg/m²)	Non-obese	19.50	24.90	22.9	1.54	0.001
	Obese	25.20	34.60	28.9	2.96	
Waist circumference (cm)	Non-obese	73.0	90.5	82.1	5.11	0.773
	Obese	71.0	95.0	82.6	7.08	
Cholesterol (mg/dl)	Non-obese	159	295	223.8	35.56	0.026
	Obese	174	330	247.6	44.3	
Triglyceride (mg/dl)	Non-obese	107	194	133.7	20.71	0.001
	Obese	112	220	162.9	32.62	
HDL (mg/dl)	Non-obese	33	68	51.8	10.41	0.001
	Obese	21	50	35.9	7.64	
FBS (mg/dl)	Non-obese	74	124	97.6	15.35	0.006
	Obese	77	325	128.1	56.70	
LH (ng/ml)	Non-obese	6.0	16	10.8	2.52	0.058
	Obese	7.4	17.8	12.1	2.64	
FSH (ng/ml)	Non-obese	3.1	7.3	5.2	1.25	0.098
	Obese	3.1	8.8	5.8	1.41	
Testosterone (ng/ml)	Non-obese	0.3	2.1	1.3	0.47	0.001
	Obese	0.9	3.3	1.9	0.75	
Total L-Carnitine (µmol/L)	Non-obese	12.2	67.3	44.5	15.87	0.001
	Obese	8.5	49.3	23.5	11.25	

Of all the women studies 41 (68.3%) had normal FBS (< 110 mg/dl), 15 (25%) had impaired fasting glycaemia (111 - 125 mg/ dL) and 4 (6.7%) had FBS \geq 126 mg/ dL (diabetic). Twenty-six (86.7%) of the non-obese group had normal FBS and 4 (13.3%) had

impaired fasting glycaemia, while 15 (50%) obese women had normal FBS, 11 (36.7%) had impaired fasting glycaemia and 4 (13.3%) were diabetic(table 3).

Table 3: Fasting blood sugars in obese and non-obese women with PCOS

			0111011 111				
FBS	Non-ob	ese	Obese		Total		P Value
	Ν	%	Ν	%	Ν	%	_
Normal	26	86.7	15	50.0	41	68.3	
Impaired fasting glycaemia	4	13.3	11	36.7	15	25.0	0.006
Diabetic	0	0	4	13.3	4	6.7	

Insulin resistance was found in 31 (51.7%) women with PCOS. Nine (30%) non-obese women had insulin resistance and 22 (73.3%) obese women had insulin resistance, (p value= 0.001), table 4.

Table 4: Insulin Resistance in obese and non-obese women with PCOS

Insulin Resistance	Non-ob	ese	Obese		Total		P Value
	Ν	%	Ν	%	Ν	%	_
Present	9	30.0	22	73.3	31	51.7	
Not present	21	70.0	8	26.7	29	48.3	0.001
Total (100%)	30		30		60		

Among all 60 PCOS women serum triglyceride, FBS and testosterone were correlated negatively with serum L-carnitine (p value= 0.001, 0.001 and 0.027 respectively) while serum HDL correlated positively with serum L-carnitine (p value= 0.001). Among obese women, serum HDL correlated positively and serum FBS correlated negatively with serum L-carnitine (p value= 0.008 and 0.002 respectively). table 5.

Table 5: Correlation of clinical and biochemical features with total L-Carnitine in obese and non-obese women with PCOS

Variables	All patients	All patients		ien	Non-obese v	Non-obese women	
	r	P value	r	P value	r	P value	
Age	0.217	0.095	0.010	0.956	0.292	0.117	
Waist circumference	-0.070	0.596	0.005	0.978	0.107	0.574	
Cholesterol	-0.203	0.120	-0.052	0.787	-0.023	0.905	
Triglyceride	-0.401	0.001	-0.320	0.085	0.001	0.999	
HDL	0.498	0.001	0.476	0.008	-0.013	0.946	
FBS	-0.481	0.001	-0.540	0.002	-0.271	0.147	
LH	-0.124	0.345	-0.143	0.450	0.168	0.373	
FSH	-0.132	0.315	-0.171	0.365	0.138	0.468	
Testosterone	-0.285	0.027	0.021	0.912	-0.0001	0.999	

r= (Pearson Correlation Coefficient)

The mean BMI, waist circumference, FBS and triglyceride were significantly higher in women with insulin resistance (p value < 0.05) than those

without. On the other hand, mean serum HDL and L-Carnitine were lower in women with insulin resistance (p value < 0.5) than those without, table 6.

Variables	Insulin Res	Insulin Resistance					
	Present (N=	=31)	Not Presen	Not Present (N=29)			
	Mean	SD	Mean	SD			
Age	27.3	5.45	28.3	5.09	0.443		
BMI	28.2	3.71	23.4	1.94	0.001		
Waist circumference	84.3	6.86	80.3	4.51	0.012		
FBS	128.3	55.37	96.3	13.6	0.004		
Cholesterol	240.2	43.11	230.9	40.11	0.393		
Triglyceride	155.9	32.92	140.1	26.62	0.047		
HDL	40.3	11.83	47.6	11.39	0.018		
LH	11.7	2.67	11.2	2.61	0.459		
FSH	5.7	1.45	5.4	1.25	0.388		
Testosterone	1.7	0.8	1.5	1.59	0.245		
Total L-Carnitine	29.1	16.17	39.3	17.1	0.021		

Discussion

Polycystic Ovary Syndrome, is a prevalent endocrine disorder that affects females of reproductive age. The incidence of PCOS differs depending on the diagnostic criteria employed, with estimates ranging from 9% to 18% in females of reproductive age according to Rotterdam criteria.(7) Just over one half of the women in the current study (PCOS patients) had IR, which is higher than the findings of Vrbikova et al (13) (40.2%) of PCOS women and those of Li et al (43.23%) (14). However, another study revealed that IR did not differ significantly between PCOS and their control group.(15) IR was found to be higher in obese women with PCOS (73.3%) compared to (30%) in the non-obese. Popovska et al (16) found that 58.1% of obese women with PCOS had IR while Li et al (17) reported that 28% of obese patients with PCOS had IR.

In general, overweight or obese women with PCOS had increased fasting glucose levels and insulin resistance.(13-18) Studies reported that women with both PCOS and abnormal glucose tolerance had significantly higher IR (18) and that the prevalence of IR was significantly higher in the PCOS group compared to the controls.(15) The metabolic syndrome is prevalent in about 43-47 % of PCOS patients, which is two times greater than the proportion in the general population. (19) These results augment our results that showed a higher incidence of IR in PCOS women especially obese ones.

The current study found that age of patients, serum cholesterol, LH and FSH were not associated with insulin resistance while high BMI, waist circumference, FBS triglyceride and low serum HDL and L-Carnitine were associated with insulin resistance. Celik et al found in a follow-up study (of 2-4.17 years) for women with PCOS that 78% of them had normal FBS at baseline and 11.5% converted to impaired glucose tolerance. Among those women with impaired glucose tolerance at baseline, 33.3% converted to type II diabetes mellitus. (18) The present study showed that 13.3% of non-obese and 36.7% of obese patients had impaired fasting glycaemia which agreed with the findings of other studies. (13, 18, 19)

Mahnaz et al showed no significant differences in fasting glucose levels between the control group and PCOS patients, as well as no significant differences in the prevalence of impaired fasting glucose (IFG) between the control group and PCOS, in a casecontrol study. These findings could be due to the small number of cases and controls in that study. (15)

In the current study, women with PCOS had significantly higher testosterone levels which agrees with the findings of previous studies. (16, 19) The multiple functions of insulin may have a role in hyperandrogenism. Although multiple studies have found a link between fasting insulin levels and androgen levels, it is still unclear if hyperandrogenism is caused by hyperinsulinemia or vice versa. Insulin and insulin-like growth factor-1 (IGF-1) are both effective stimulators of ovarian androgen synthesis, with the insulin receptor likely playing a role. (20) The mean LH/FSH ratio in PCOS women was 2.09. However obese women had slightly higher LH and FSH levels than non-obese, indicating the low sensitivity of this test as a diagnostic tool in Iraqi patients with PCOS. Obese PCOS women have significantly greater LH levels than their normal-weight counterparts, according to clinical research (21,22). This was also found in our patients. with LH and FSH was not affected by the status of IR while other studies showed lower LH concentrations in PCOS women. (23)

In our series, obese women had higher serum triglyceride and cholesterol levels and lower HDL

levels than non-obese which agrees with published literature. (17, 20, 24) Obesity is a common symptom of PCOS, with incidence rates ranging from 12.5 % (25) to 100 % (26) according to a recent meta-analysis (24), with an estimated incidence of 49% and 58.1 % of obese females with PCOS being IR.(27) Obesity also worsens IR and exacerbates most of the reproductive and metabolic symptoms of PCOS.(28) Females with PCOS have more triglycerides, LDL-cholesterol, and total cholesterol, and lower HDL-cholesterol levels, according to a meta-analysis (29) compared to control females, regardless of BMI. Women with PCOS are also more liable to develop type 2 diabetes. High waist circumference is associated with IR, in agreement with our study. (24)

The mean level of serum total L-carnitine in the current study of 34.03μ mol/L is close to that reported by Fenkci et al in which the mean level was $40.5 \pm 5.7 \mu$ mol/L . (21) We also found that the obese had carnitine levels lower than the non-obese and that low serum L-Carnitine was associated with IR, both in agreement with the studies of Essah et al (20) and Fenkci et al. (21)

"Researchers from Pamukkale University's School of Medicine in Turkey found that women with PCOS had 50% less L-carnitine in their blood serum than healthy women who are not diagnosed with PCOS (21). They also found a link between low serum L-carnitine levels and a high free androgen index (FAI), which greatly contributes to the growth of excess facial and body hair, as well as the loss of scalp hair in women with PCOS.

Pharmacological treatment, like metformin, target symptoms and are usually helpful, although they have unpleasant gastrointestinal side effects. Most females with PCOS need long-term treatment, thus it is critical to consider additional nonpharmacological treatment options. (30)

Conclusions

The mean value of serum total L-carnitine among the non-obese PCOS women was higher than among the obese ones. Low serum L-carnitine is associated with insulin resistance.

Ethical Clearance for the study: Ethical clearance was obtained from Baghdad Teaching Hospital, Medical city, Ministry of Health,

Authors' declaration:

Conflicts of Interest: None

We hereby confirm that all the Figures and Tables in the manuscript are ours. Besides, the Figures and images, which are not ours, have been given permission for re-publication attached with the manuscript

Ethical Clearance: The project was approved by the local ethical committee in Department of Gynecology, College of Medicine, University of Baghdad, according to the code number (08. 20.4.2016).

Authors' contributions:

Dr. Maad Mehdi Shallal: Concept of the study, reviewing manuscript.

Dr. Najmah Mahmood Meran: Writing the project, collecting data, writing draft.

Dr. Zina Abdullah Hussein: Collecting data, writing the manuscript.

References

1. Baer TE, Milliren CE, Walls C, DiVasta AD. Clinical Variability in Cardiovascular Disease Risk Factor Screening and Management in Adolescent and Young Adult Women with Polycystic Ovary Syndrome. J Pediatr Adolesc Gynecol. October 2015, Volume 28, Issue 5, Pages 317-323.

2. Lebbe M, Woodruff TK. Involvement of androgens in ovarian health and disease. Mol Hum Reprod. 2013 Dec; 19(12):828-37.

3. Gottschau M, Kjaer SK, Jensen A, Munk C, Mellemkjaer L. Risk of cancer among women with polycystic ovary syndrome: A Danish cohort study. Gynecol Oncol. 2014 Nov 20; 14: 1475-9.

4. Li L, Baek KH. Molecular genetics of polycystic ovary syndrome: an update. Curr Mol Med. 2015; 15(4):331-42.

5. Ha L, Shi Y, Zhao J, Li T, Chen ZJ. Association Study between Polycystic Ovarian Syndrome and the Susceptibility Genes Polymorphisms in Hui Chinese Women. PLoS One. 2015; 10(5):e0126505.

6. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group: Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome Society. Fertil Steril 2004, 81:19-25.

7. March WA, Moore VM, Willson KJ, Phillips DI, Norman RJ, Davies M J. The prevalence of polycystic ovary syndrome in a community sample assessed under contrasting diagnostic criteria. Hum Reprod. 2010 Feb; 25(2):544-51.

8. Moledina DG, Wilson FP. Pharmacologic Treatment of Common Symptoms in Dialysis Patients: A Narrative Review. Semin Dial. 2015 Apr 25:212-216.

9. Rebouche CJ: Kinetics, pharmacokinetics, and regulation of L-carnitine and acetyl-L-carnitine metabolism. Ann NY Acad Sci 2004, Vol 1033, Issue 1, page 30-41.

10. Mescka CP, Guerreiro G, Hammerschmidt T, Faverzani J, de Moura Coelho D, Mandredini V, et al. L-Carnitine supplementation decreases DNA damage in treated MSUD patients. Mutat Res. May 2015, Volume 775, Pages 43-47

11. Judith L Flanagan, Peter A Simmons, Joseph Vehige, Mark DP Willcox and Qian Garrett. Role of carnitine in disease. Nutrition and Metabolism 2010, 7: 30- 54.

12. Tong PC, Kong AP, So WY, Yang X, Ho CS, MA RC, et al. The Usefulness of the International Diabetes Federation and the National Cholesterol Education Program's Adult Treatment Panel III Definitions of the Metabolic Syndrome in Predicting Coronary Heart Disease in Subjects with Type 2 Diabetes, Diabetes Care, 2007;30(5):1206–1211

13. Li R, Zhang Q, Yang D, Li S, Lu S, Wu X, et al. Prevalence of polycystic ovary syndrome in women in China: a large community-based study. Hum Reprod. 2013 Sep; 28(9):2562-9.

14. Li X, Lin JF. Clinical features, hormonal profile, and metabolic abnormalities of obese women with obese polycystic ovary syndrome. Zhonghua Yi Xue Za Zhi. 2005 Dec 7; 85 (46):3266-71.

15. Lankarani M, Valizadeh N, Heshmat R, Peimani M, and Sohrabvand F. Evaluation of insulin resistance and metabolic syndrome in patients with polycystic ovary syndrome. Gyne cological Endocr inology, August 2009; 25(8): 504–507

16. Popovska-Dimova Z, Krstevska B. The frequency of insulin resistance calculated upon the basis of a fasting glucose to insulin ratio and characteristics of insulin resistant women with polycystic ovary syndrome. Prilozi. 2006 Jul;27(1):87-95.

17. Vrbikova J, Dvorakova K, Grimmichova T, Hill M, Stanicka S, Cibula D, et al. Prevalence of insulin resistance and prediction of glucose intolerance and type 2 diabetes mellitus in women with polycystic ovary syndrome. Clin Chem Lab Med. 2007; 45(5):639-44.

18. Celik C, Tasdemir N, Abali R, Bastu E, Yilmaz M. Progression to impaired glucose tolerance or type 2 diabetes mellitus in polycystic ovary syndrome: a controlled follow-up study. Fertil Steril. 2014 Apr; 101(4):1123-8.e1

19. Suresh S, Vijayakumar T. Correlations of Insulin Resistance and Serum Testosterone Levels with LH:FSH Ratio and Oxidative Stress in Women with Functional Ovarian Hyperandrogenism. Indian J Clin Biochem 2015 Jul; 30(3): 345-50.

20. Essah PA, Nestler JE. The metabolic syndrome in polycystic ovary syndrome. J Endocrinol Invest. 2006 Mar; 29(3):270-80.

21. Fenkci SM, Fenkci V, Oztekin O, Rota S and Karagenc N. Serum total Lcarnitine levels in nonobese women with polycystic ovary syndrome. Human Reproduction 2008; 23(7): 1602 – 1606.

22. Vigerust NF, Bohov P, Bjørndal B, Seifert R, Nygård O, Svardal A, et al. Free carnitine and acylcarnitines in obese patients with polycystic ovary syndrome and effects of pioglitazone treatment. Fertil Steril. 2012 Dec; 98(6):1620-6.

23. Gambineri A, Pelusi C, Vicennati V, Pagotto U, Pasquali R. Obesity and the polycystic ovary syndrome. Int J Obes. 2002, 26(7): 883–896

24. Barrios MR, Arata-Bellabarba G, Valeri l, Velázquez-Maldonado E. Relationship between the triglyceride/high-density lipoprotein-cholesterol ratio, insulin resistance index and cardiometabolic risk factors in women with polycystic ovary syndrome. Endocrinol Nutr. 2009;56(2):59-65

25. Lee H, Oh JY, Sung YA, Chung H, Cho WY. The prevalence and risk factors for glucose intolerance in young Korean women with polycystic ovary syndrome. Endocrine 2009; 36:326–32

26. Peppard HR, Marfori J, Iuorno MJ, Nestler JE. Prevalence of polycystic ovary syndrome among premenopausal women with type 2 diabetes. Diabetes Care. 2001; 24:1050-2.

27. Graff SK, Mário FM, Alves BC, Spritzer PM. Dietary glycemic index is associated with less favorable anthropometric and metabolic profile in PCOS women with different phenotypes. Fertil Steril. 2013; 100(4):1081-8.

28. Flores-Martínez SE, Castro-Martínez AG, López-Quintero A, García-Zapién AG, Torres-Rodríguez RN, Sánchez-Corona J. [Association analysis of SNP-63 and indel-19 variant in the calpain-10 gene with polycystic ovary syndrome in women of reproductive age]. Cir Cir. 2015 Jan-Feb;83(1):35-42.

29. Wild RA, Rizzo M, Clifton S, Carmina E. Lipid levels in polycystic ovary syndrome: systematic review and meta-analysis. Fertil Steril. 2011; 95:1073-9.

30. Zheng Y, Stener-Victorin E, Ng EHY, Li J, Wu X, and Ma H. How does acupuncture affect insulin sensitivity in women with polycystic ovary syndrome and insulin resistance? Study protocol of a prospective pilot study. BMJ Open. 2015; 5(4): e007757.

How to Cite this Article

M. Shallal M, Mahmood N, A. Hussein Z. Total L-carnitine and insulin resistance in non-obese and obese Iraqi women with polycystic ovary syndrome. JFacMedBagdad [Internet]. 2023 Apr. 27 [cited 2023 May 11];65(1):20-6. Available from: https://iqjmc.uobaghdad.edu.iq/index.php/19JF acMedBaghdad36/article/view/2040

الليفوكارنتين الكلي ومقاومة الانسولين لدى النساء العراقيات غير البدينات والبدينات المصابات بمتلازمة تكيس المبايض

/كلية الطب جامعة بغداد / قسم النسائية	الاستاذ الدكتور معد مهدي شلال
/ كلية الطب جامعة بغداد / قسم النسانية	الدكتورة نجمة محمود ميران
كلية الطب جامعة المستنصرية / قسم النسانية	الدكتورة زينه عبدالله حسين /

الخلاصة

الخلفية: تعد متلازمة المبيض المتعدد الكيسات من أكثر أمراض الغدد الصماء شيوعًا التي تصيب النساء في سن الإنجاب، حيث يلعب الليفوكارنتين دورًا مهمًا في الإجهاد التأكسدي وإنتاج الطاقة واستقلاب الجلوكوز . يؤثر على مقاومة الأنسولين حيث تم الإبلاغ عن انخفاض مستوى الكارنيتين في البلازما بشكل جيد في النوع الثاني من داء السكري، مما يعني أن الليفوكارنتين قد تقلل من مقاومة الأنسولين الموجودة في مرض المبيض المتعدد الكيسات.

الهدف من الدراسة: خططت هذه الدراسة لقياس مستوى الليفوكارنتين ومقاومة الأنسولين في كل من مرضى السمنة وغير البدناء المصابين بمتلاز مة تكيس المبايض.

المرضَى والطرق: النساء في سن الإنجاب المصابات بمتلازمة تكيس المبايض 60 سيدة (30 بدينات و 30 غير بدينات) تم جمعهن من العيادة الخارجية لأمراض النساء والتوليد في مستشفى بغداد التعليمي في الفترة من يونيو 2016 إلى يونيو 2017. المعلومات التي تم الحصول عليها من كل عضوة كانت: الطول، الوزن ومحيط الخصر وضغط الدم وتاريخ الولادة والتاريخ الطبي ونتائج الموجات فوق الصوتية وتاريخ الدواء كلها عوامل يجب مراعاتها. تم إجراء فحص جسدي لتقييم العلامات السريرية لفرط الأندروجين. تم قياس القياسات البيوكيميائية لسكر الدم الصائم وهرمون اللوتين وهرمون تحفيز الجريب والتستوستيرون وملف الدهون مع قياس إجمالي الليفوكارنتين باستخدام (NCEP / ATP III (NCEP / ATP). تم تشخيص مقاومة الأنسولين وفقًا للبرنامج الوطني لتعليم الكوليسترول / لوحة علاج البالغين (NCEP / ATP). (III يتم تشخيص متلازمة نكيس المبايض وفقًا للمعايير روتردام.

النتائج: كشفت هذه الدراسة أن مقاومة الأنسولين (IR) كانت موجودة في 51.7% من مرضى متلازمة تكيس المبايض ، والتي كانت أعلى لدى مرضى متلازمة تكيس المبايض ، والتي كانت أعلى لدى مرضى متلازمة تكيس المبايض ، والتي كانت أعلى لدى مرضى متلازمة تكيس المبايض ، والتي كانت أعلى لدى مرضى متلازمة تكيس المبايض ، والتي كانت أعلى لدى مرضى متلازمة تكيس المبايض ، والتي كانت أعلى لدى مرضى متلازمة تكيس المبايض ، والتي كانت أعلى لدى مرضى متلازمة تكيس المبايض (73.3%) البدناء (73.3%) عنها في غير البدينين (30%). لم يكن عمر المرضى والكوليسترول في الدم و LH و مرضى متلازمة تكيس المبايض ، والتي من رضى متلازمة تكيس المبايض (73.3%) البدناء (73.3%) عنها في غير البدينين (30%). لم يكن عمر المرضى والكوليسترول في الدم و FSH مرضى متلازمة المطي FSH مرتبطين بمقاومة الانسولين. ارتبط مؤشر كتلة الجسم المرتفع ومحيط الخصر و FSS والدهون الثلاثية بشكل كبير مع مقاومة الانسولين (p <0.05%) ، بينما ارتبط انخفاض HDL و L-Carnitine في المصل بمقاومة الانسولين (p<0.05%).

كان متوسط إجمالي مصل L-carnitine في هذه الدراسة ق34.03 ميكرولتر / لتر. كان لدى النساء البدينات مستويات كارنيتين أقل من النساء غير البدينات وكان انخفاض مصل L-Carnitine مرتبطًا بمقاومة الانسولين. ارتبطت الدهون الثلاثية في الدم و FBS و هرمون التستوستيرون سلبًا مع مصل FB (0.05> L-carnitine (PL المصل مرتبط بشكل إيجابي مع مصل) Ersدهمة المحمة المحمة م

(p = 0.001).

الاستنتاجات: كمان متوسط قيمة الليفوكارنتين الكلي في المصل بين النساء غير البدينات المصابات بمتلازمة تكيس المبايض أعلى منه بين النساء البدينات. يرتبط انخفاض الليفوكارنتين في المصل بمقاومة الأنسولين.

ا**لكلمات المفتاحية:** الليفوكارنتين، مقاومةَ الأنسولين، السمنة، النساء، متلازمة تكيس المبايض.