RESEARCH ARTICLE

Evaluation of Lipid Profile and Sexual Hormones in Women with Polycystic Ovarian Syndrome at Al-Najaf Province

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Received: 16th June, 2023; Revised: 20th July, 2023; Accepted: 12th August, 2023; Available Online: 25th September, 2023

ABSTRACT

Background: A multiple-factorial condition influencing females' reproductive years is polycystic ovarian syndrome (PCOS). This study aims to investigate lipid profiles and sex hormones, and DHEA hormones related to patients suffering from PCOS.

Method: In this investigation, the sex hormones, lipid profile and DHEA hormone of 45 females in their reproductive years who were suffering from PCOS were compared to those of 45 women who were healthy and of a similar age.

Results: Our results showed that the mean ± standard deviation of triglyceride parameter is higher than others parameters in both women patients with polycystic syndrome (73.2 ± 147.3 mg/dl) and control (57.9 ± 123.7 mg/dl) groups with highly significant difference (*p-value* < 0.001). While, mean ± standard deviation of other parameters, including cholesterol, HDL, LDL and VLDL, was higher in women with polycystic syndrome than in the control groupe.

Conclusion: The more active fertile age group, 21 to 30 years old are more susceptible to the incidence of PCOS. Our data shows a significant correlation between DHEA and cholesterol, HDL, VLDL in the patient group. There is a positive statistical correlation between DHEA and LDL and between DHEA and LH, prolactin, and testosterone. The correlation between DHEA and FSH showed a passive association (statistically significant).

Keywords: Polycystic ovary syndrome, Dyslipidemia, Androgen, Triglycerides, DHEA.

International Journal of Drug Delivery Technology (2023); DOI: 10.25258/ijddt.13.3.27

How to cite this article: Hassan AU, Adnan AN. Evaluation of Lipid Profile and Sexual Hormones in Women with Polycystic Ovarian Syndrome at Al-Najaf Province. International Journal of Drug Delivery Technology. 2023;13(3):942-947.

Source of support: Nil.

Conflict of interest: None

INTRODUCTION

One of the women's most prevalent endocrine conditions, polycystic ovarian syndrome (PCOS) is marked by hyperandrogenism, ovulatory failure, and sonographic evidence of polycystic ovaries.[1] affecting (5.6–21.3%) of women of reproductive age worldwide.[2] In order to diagnose this disorder, at least two of the three Rotterdam criteria must be satisfied: Menstrual abnormalities (oligomenorrhea or amenorrhea), clinical or biochemical hyperandrogenism (HA), and ultrasound-verified polycystic ovaries are three examples.[3] It is to blame for 40% of female infertility instances. it is a significant contributor to both reproductive disorders and endometrial cancer. Additionally, various metabolic diseases, including hepatic steatosis, glucose intolerance, dyslipidemia, type 2 diabetes, and hypertension, are linked to PCOS.[4] The main causes of metabolic dysfunction in PCOS women are elevated free fatty acids (FFAs) and rising adiposity. Over 10% of females develop PCOS, which is characterized by polycystic ovaries, high clinical levels of biochemical androgen, and ovulatory dysfunction. Insulin resistance, type 2 diabetes, obesity, and an elevated risk of cardiometabolic disease are metabolic consequences linked with PCOS.[5] There is a complicated relationship between lipid metabolism and PCOS traits, which may help to explain why long-term cardiovascular disease risk is higher in PCOS women with diverse characteristics. These PCOS patients also presented with distinct lipid profiles. Investigating the best patient subgroups with PCOS who require lipid-lowering therapy might be worthwhile.[6]

MATERIAL AND METHODS

Study Design

This study was performed in "The Fertility Center at AL-Sadder Medical City in Najaf Province Iraq, during the period from the 1st October. 2022 to 1st March. 2023. The patients' info included age, address, contraceptive taking, thyroid disease, hypertension, diabetes mellitus. The patient number was 90 women divided into two groups: first group was 45 women who are suffering from (PCOS) according to the Rotterdam Criteria (2003). On ultrasound scan, it necessitates the presence of at least two of the following traits: menstrual irregularities, hyperandrogenism, and polycystic ovaries. The patients' age

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ranged from 17 to 45 years. The second group is the control group, which comprises of 45 women who are eligible if they have a regular menstrual cycle, normal ovulation, and normal ovaries as determined by the gynecologists who do not not contraceptives. The study case-control study. The exclusion criteria consist of a grouping of females having another cause of infertility.

Ethical Consideration

AL-Najaf Al-Ashraf Health Directorate approved this study according to their instruction. Verbal agreement from the patients and/or their next of kin after a full exploration of the study aims was taken before blood was drawn.

Statistics

Statistical Package for Social Sciences (SPSS) version 25 for iWindows, developed by IBM in the US in 2017, was used to input, organize, and analyze the data of the research participants, polycystic women patients, and controls. Prior to doing the study, all variables were reviewed for mistakes or inconsistencies. Utilizing histogram and normal distribution curves it was determined whether or not the continuous variables age, cholesterol, TG, HDL, LDL, VLDL, FSH, LH, PRL, testosterone, and DHEA, followed the statistical normal distribution. The significance of variations in categorical variable frequencies between polycystic woman patients and controls was evaluated using the chi-square test. *p-values* of 0.05 or less are regarded to be substantial levels of significance. Finally, using the Windows version of Microsoft Word 2013, results and conclusions were presented in tables and/or figures as appropriate.

RESULTS

Lipid Profile among (PCOS) Patients and Control

The results of the current study in Table 1 showed that the mean ± standard deviation of the triglyceride parameter is higher than other parameters in both women patients with polycystic syndrome (73.2 ± 147.3 mg/dl) and control (57.9 ± 123.7 mg/dl) groups with highly significant difference ($p < 0.001$).

While mean ± standard deviation of others parameters, including cholesterol, HDL, LDL and VLDL reaches 50.5 ± 139.2, 28.3 ± 46.7, 27.1 ± 72.8 and 19.5 ± 29.01 mg/dl, respectively, in the patient's group, the in control group reaches 33.5 ± 126.9, 21.7 ± 40.4, 25.7 ± 43.8 and 14.8 ± 25.8 mg/dl, respectively with significant difference (p <0.001 to <0.05) as shown in the Table 2.

Level of Sex Hormones among PCOS Patients and Control

Table 3 shows the results of hormone levels among patients and controls. The results appear that the prolactin hormone level was highest in the PCOS category (7.9 ± 30.7 ng/ml) than the control category (7.1 ± 18.9 ng/mL) and this rise was statistically significant (*p-value*<o.oo1).

From the side, luteinizing hormone level appeared to increase amongst the control group (4.06 ± 12.3 mIU/mL) compared to women patients with polycystic syndrome (3.4 ± 16.6 mIU/mL). Also, this rise was statistically significant ($p < 0.001$).

Also, follicle-stimulating hormone (FSH) level was statistically higher significant in the control group (2.2 ± 6.5 mIU/mL) compared to women patients with polycystic syndrome (1.4 ± 2.7 mIU/mL).

As well as, testosterone hormone levels appear to be statistically significantly raised ($p < 0.001$) amongst women patients with the polycystic syndrome (1.03 ± 1.8 ng/mL) compared to the control group 0.2 ± 0.75 ng/mL).

Level of Dehydroepiandrosterone (DHEA) Hormone among Patients and Control

DHEA hormone amongst studied categories, where the level of DHEA hormone was higher in women patients with polycystic syndrome (40.08 ± 168.9 ng/mL) compared to the control group (26.18 ± 70.4 ng/mL), (*p-value* < 0.001) (Figures 1-8).

DISCUSSION

Hormone imbalance is an important factor contributing to high cholesterol in PCOS patients. Low levels of the female hormone estrogen are linked to an increase in total cholesterol due to greater levels of blood lipid (fat) known as triglyceride and another low-density lipoprotein (LDL), also known as "bad" cholesterol.[7]

High cholesterol in ladies with polycystic ovary syndrome are also overweight or fatty, especially in the belly area, as excess insulin can lead to weight gain. Visceral or belly fat can result in reduced HDL, or "good" cholesterol, and greater triglycerides.[8]

Table 1: Lipid profile of blood serum among PCOS pateints and control categories

Parameters	*Study Group*		*Statistical test*	*p-value*
	*Patients Mean ± SD**	*Control Mean ± SD*		
Cholesterol (mg/dl)	50.5 ± 139.2	33.5 ± 126.9	t=1.3	<0.001
Triglyceride (mg/dl)	73.2 ± 147.3	57.9 ± 123.7	t=1.6	<0.001
HDL (mg/dl)	28.3 ± 46.7	21.7 ± 40.4	t=1.1	<0.05
LDL (mg/dl)	27.1 ± 72.8	25.7 ± 43.8	t=5.2	<0.001
VLDL	19.5 ± 29.01	14.8 ± 25.8	t=0.86	<0.05

*: standard deviation; HDL: high-density lipoprotein; LDL: low-density lipoprotein; VLDL: very low-density lipoprotein; t: independent t-test; significance at p<0.05.

Table 2: Hormones level among polycystic women and control

Parameters	*Study group*		*Statistical test*	*p-value*
	*Patients mean ± SD**	*Control mean ± SD*		
FSH (mIU/ml)	1.4 ± 2.7	2.2 ± 6.5	t=9.5	<0.001**
LH (mIU/ml)	3.4 ± 16.6	4.06 ± 12.3	t=5.3	<0.001
Prolactin (ng/ml)	7.9 ± 30.7	7.1 ± 18.9	t=7.3	<0.001
Testosterone (ng/ml)	1.03 ± 1.8	0.2 ± 0.75	t=6.7	<0.001

*: standard deviation; FSH: follicle stimulating hormone; LH: luteinizing hormone; t: independent t-test; **: highly significance.

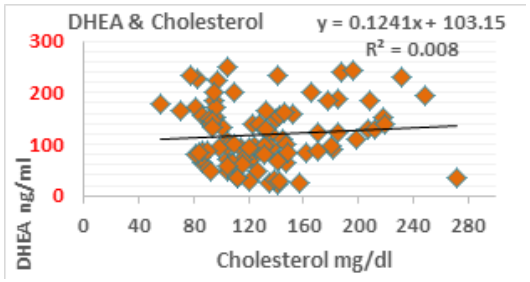

Figure 1: DHEA & Cholesterol

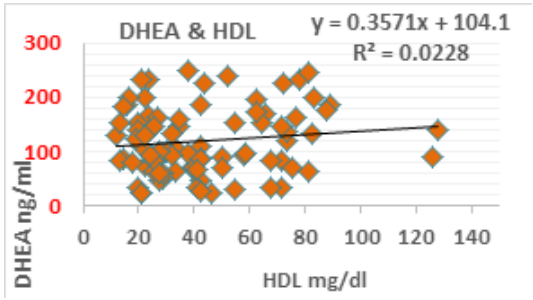

Figure 2: DHEA & HDL

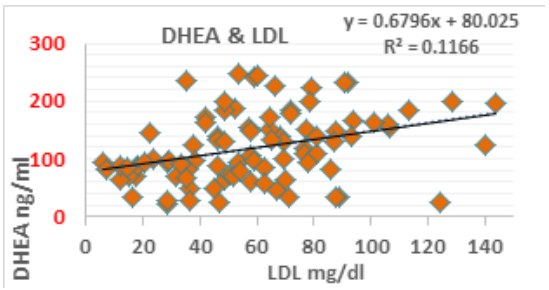

Figure 3: DHEA & LDL

Hyperinsulinemia and hyperandrogenemia may be the causes of dyslipidemia in people with PCOS. As a result, catecholamine-induced lipolysis in adipocytes increased, releasing free fatty acids into the bloodstream. The release of VLDL is triggered by elevated free liver fatty acids, which results in hypertriglyceridemia. decreased high-density lipoprotein cholesterol as well as low-density lipoprotein cholesterol are produced from hypertriglyceridemia via the reverse cholesterol transfer route. Early androgenic stimulation of adipocytes predisposes to dyslipidemia associated with PCOS.[9,10]

Triglyceride (TG) levels may have increased due to body fat buildup, a decrease in the rate at which fat is eliminated, or

Table 3: Level of DHEA hormone among the study groups

Parameter	*Groups*	*Mean ± SD**	*p-value*
DHEA (ng/mL)	Patients No. (45)	40.08 ± 168.9	<0.001 H.S
	Healthy control No. (45)	26.18 ± 70.4	

*:standard deviation; DHEA: dehydroepiandrosterone; H.S: highly significance; two independent t-test.

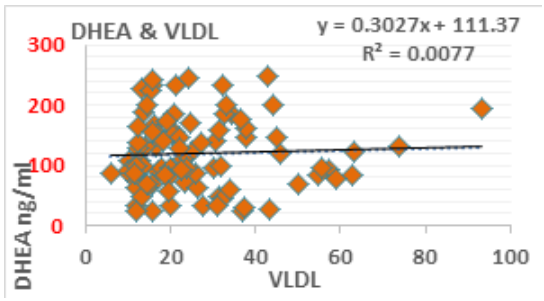

Figure 4: DHEA & VLDL

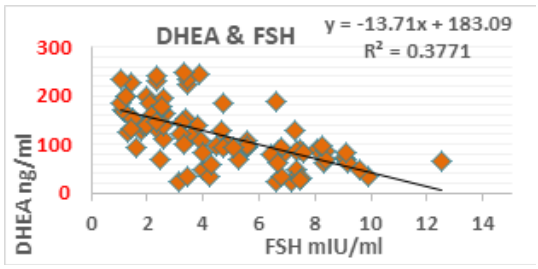

Figure 5: DHEA & FSH

a decrease in the oxidation of fatty acids. High concentrations of plasma TGs were caused by the liver secreting more very-low-density lipoprotein (VLDL) than usual. Insulin resistance, which is seen in PCOS patients, could be to blame for this. Additionally, insulin resistance accelerates the breakdown of HDL-C molecules and LDL-C production. In addition to changing the structure of lipids, hyperandrogenism has been linked to an increase in hepatic lipase activity, which is important for the metabolism of HLD-C molecules. Increased blood triglyceride levels are thought to be a risk factor for cardiovascular disease.[12]

The current study showed a high mean concentration level of HDL cholesterol in PCOS group (28.3 ± 46.7 mg/dl) other than control group (21.7 ± 40.4 mg/dl). The findings of the current investigation, which demonstrated that ladies suffered PCOS had a significant rise in the lipid concentration (total

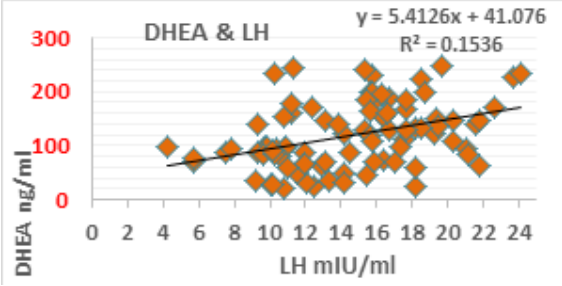

Figure 6: DHEA & LH

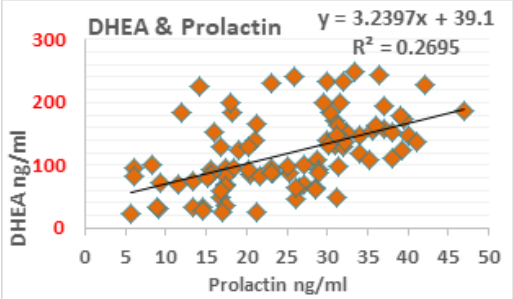

Figure 7: DHEA & prolactin

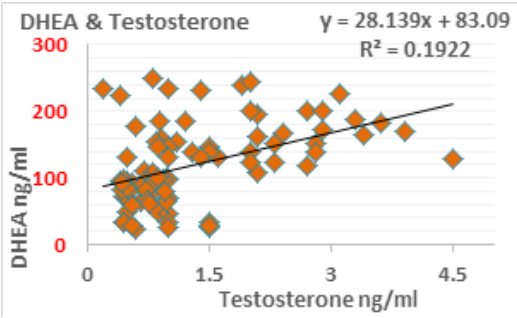

Figure 8: DHEA & Testosterone

cholesterol, triglyceride, high-density lipoprotein (HDL), and LDL, were consistent with the findings of earlier studies.[11-13]

The findings of the current study, however, were at odds with,[14-16] which founds no significant difference in the levels of total cholesterol, triglycerides, HDL, and LDL lipids between women with PCOS and the control group.

Also our results are conflicted with[9] in addition,[17] found that obese PCOS females had lower HDL and higher cholesterol compared to control group. Furthermore, according to,[18] while blood HDL was lower in the PCOS group than in the matched control group, there appeared to be a statistically significant rise in blood seum low density lipoprotein, triglyceride, and cholesterol. In contrast with obesity regulation, this investigation discovered excess in these patients' blood seum low density lipoprotein and cholesterol.

PCOS, the most prevalent illness of endocrine among females in age of reproduction, is linked with several metabolic syndrome symptoms, including obesity, insulin resistance, hyperlipidemia, hyperpiesia, sleep apnea, and irregular menstruation. These days, the prevalence of PCOS is rising quickly, which could be attributed to dietary and lifestyle changes as well as hormonal imbalances.[19]

The aberrant ovarian hormonal dynamics associated with PCOS are caused by fast gonadotropin-releasing hormone, including recurrence with enhanced pituitary gland sense to gonadotropin-releasing hormone.[20] As a result, there is an increase in the excretion of luteinizing hormone associated with follicle stimulation hormone.

Along with chronic decreasing, continuous estrogen levels more strongly limit follicle stimulation hormone than luteinizing hormone. The sustained rise among gonadotropin-releasing hormones and recurrence is thought as a primary factor among higher production of leutinizing hormone compared to FSH from the pituitary. Obesity reduces PCOS-related women's aberrant gonadotropin dynamics. Higher levels of leptin and/or insulin are thought to have a direct inhibitory effect on the release of gonadotropins, mediating this action only at the pituitary.[20]

According to[21] PCOS affects women with hyperandrogenism, which causes insulin resistance (IR) among skeletal muscles and adipose tissue.

According to some researches[22] serum PRL and glucose metabolism interact, with different levels of serum prolactin having varied effects on how glucose is metabolized. As a result, hyperprolactinemia patients are more prone to experience impaired glucose tolerance and insulin resistance.

The adrenal cortex's zona reticularis produces DHEA, the precursor to androgen primarily made from cholesterol. More than 95% of the DHEA produced by PCOS patients comes from their adrenal glands, and the remaining 5% comes from their ovaries.[23]

Patients with PCOS have higher levels of DHEA, which can be attributed to the increased androgen production in PCOS, which comes from the ovaries and adrenal glands. According to some researches[24] between 40 and 60% of PCOS patients have excessive androgen production from the adrenal, and this is indicated via elevated DHEA level in blood serum or plasma.

Two of the key characteristics of women with PCOS include hyperandrogenicity and ovulatory failure. Given that the zona reticularis of the adrenal cortex is responsible for more than 95% of its production, DHEAS is a helpful measure for adrenal androgen production in clinical practice.[25]

Breast cancer and other hormone-sensitive cancers are more likely to strike women with polycystic ovaries and high levels of the hormone DHEA.[26]

The correlation between DHEA and cholesterol our data shows a significant correlation between DHEA and cholesterol, HDL, VLDL in the patient's group (R= 0.089, R=0.151, R=0.088, respectively, the *p-value* was > 0.05). And this may due to DHEA might decrease HDL or good cholesterol levels. While there is a positive statistical correlation between DHEA and LDL (R=0.341, *p-value* was < 0.001). And this positive linking due to the fact that DHEA did improve total body fat and LDL (bad cholesterol) (Figures 1-4).

As for the correlation between DHEA and hormones, the results showed a passive association (statistically significant) between [DHEA and FSH] (R=-0.614, *p-value* was < 0.001).

DHEA affects early follicle maturation by regulating androgen receptor (AR) transcription, increasing FSH receptor (FSHR) expression, modulatingi FSH activity ini granulosa cells (GCs), and increasing the number of growing preantral and small antral follicles (Figure 5).[27]

Also, we observed that there is a significant positive correlation between DHEA and LH, prolactin, and testosterone (R=0.392, R= 0.519, R=0.438 respectively, *p-value* was < 0.001) (Figures 6-8). DHEA is a steroid hormone capable of modifying responses of hormones from the pituitary gland into hypothalamic organizers. Its effect on the pituitary gland LH in addition to prolactin releasing by a limited reflection of dopamine inactivation, as well as via a simplification of type two ang excretory work.[28] DHEA also converted by the body into testosterone and other sex hormones. Therefore this is commonly seen in polycystic ovarian syndrome.[29]

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