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Evaluation of the levels of the Lipid profile in patients with polycystic ovaries and insulin resistance

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Abstract:

Background: *High levels of male hormone, irregular menstruation, hormone imbalances, and metabolic abnormalities are the diagnostic criteria for polycystic ovarian syndrome, a common endocrine condition that affects 6 to 20 percent of women of reproductive age. An imbalance in the quantity of insulin secreted is known as insulin resistance, and it increases the risk of heart disease and type 2 diabetes.*

Objectives*: Evaluate the Lipid profile (LDL, HDL, triglyceride and cholesterol) in the serum of those with insulin resistance and polycystic ovarian syndrome and its comparison with the control group.*

Materials and Methods*: Eighty women between the ages of 16 and 40 are included in a case and control study. Forty of the first group, who had polycystic ovarian syndrome and insulin resistance, were matched with forty healthy women in the second group. October 2023 and February 2024 were the dates of sample collection for each. The lipid profile was analyzed as follows.*

Results: *The results of the study demonstrated a difference in the biostatistics of the lipid profile (LDL, HDL, triglyceride, and cholesterol) between the PCOS and IR group and the control group. Additionally, when the patient group was compared according to age, the study demonstrated the impact of the patient's woman's body mass index (BMI).*

Conclusions*: It was discovered that patients with PCOS and IR had greater levels of LDL, HDL, triglyceride, and cholesterol than the control group. Additionally, the data indicated that patients older than 30 had significantly higher levels of both LDL and cholesterol. The BMI is highly significant, and among the patients, obesity accounted for 45% of the group's characteristics.*

Key-words*: Polycystic ovarian syndrome, Insulin resistance, Lipid profile.*

Introduction

Since Stein and Leventhal's initial finding in 1935, interest in polycystic ovaries (PCO) and its associated condition (PCOS) has evolved from a "gynecological curiosity to a multisystem endocrinopathy." It is perhaps the most common endocrine disorder in women, as it is responsible for most occurrences of hirsutism,

irregular menstruation, and anovulatory infertility. With a complex pathophysiology that has generated a lot of scholarly discussion, it is also among the least understood endocrinological illnesses[1, 2], Symptoms of polycystic ovaries include irregularity Menstrual cycle, hirsutism, acne, alopecia, dermatitis and its thickness, insomnia or sleep disturbance [3]. Insulin resistance (hyperinsulinemia) is now recognized as a characteristic of polycystic ovarian syndrome (PCOS). Only recently has its importance in the pathophysiology of PCOS been recognized. The effective use of insulin-sensitive drugs in the treatment of PCOS has been made possible by better understanding of the role that insulin resistance plays in the disorder. Insulin resistance seems to be present in PCOS in both fat and lean women [1, 4, 5]. One of the earliest indicators of many diseases that affect people is insulin resistance, which is defined as a failure in insulin-mediated control of glucose metabolism in tissues. It is associated with a number of metabolic abnormalities, which essentially mean that the body's cells do not respond to the insulin hormone, that cells are less sensitive to insulin signaling, and that glucose is not absorbed. Weight gain results from the body retaining glucose since it is not used to produce energy [6].Women with PCOS are more likely to have lipid problems. The risk of cardiovascular disease is increased by a number of lipid abnormalities that are present in PCOS, including lower levels of high density lipoprotein and higher levels of triglycerides, total cholesterol, and low-density lipoprotein cholesterol (LDL-C). A recent study indicated that consuming a high-fat diet stimulated metabolic and ovarian conversion in those with PCOS, which had an impact on the hormonal profile [7]. Infertility in PCOS is associated with dyslipidemia because of ovarian abnormalities, an increase in polycystic follicles, and an increase in follicular layer thickness. Up to 70% of people with PCOS have dyslipidemia. There was a higher percentage of IR diagnoses among obese women than among women with a normal BMI, pointing to a link between IR and dyslipidemia [7, 8]. For overweight/obese women with PCOS, an aerobic exercise training intervention enhanced cardiorespiratory fitness and cardio metabolic health [9].

Aims of the Study

This study addresses the following aims

1-Evaluate of the levels of lipid profile in patients of polycystic ovarian and insulin resistance, and compared with a healthy group.

2-Statement of the effect of age on lipid profile levels.

3- Examine the effect of body mass index (BMI) of patients women.

4- Identify the risk of this syndrome by evaluating some biomarkers(LDL,HDL,Cholesterol, Tirglceraide).

Materials and methods:

The first group in this study consisted of 40 women with PCOS and insulin resistance, whereas the second group consisted of 40 healthy women between the ages of 16 and 40 who were within the reproductive age range. Using a 5 ml medical syringe, 5 ml of blood was drawn into gelatin tubes (also known as gel tubes) on the second and third day of the menstrual cycle (early follicular phase) in order to perform the testing for LDL, HDL, cholesterol, and triglycerides. All samples were collected between October 2023 and February 2024 at the maternity outpatient clinics and the Gynecological and Obstetric Teaching Hospital in Karbala city. The samples were allowed to stand at room temperature for 15 minutes before the serum was separated and maintained at -20° C unless it was immediately needed.

Data Analysis

Statistical analysis was carried out using SPSS version 27. Categorical variables were presented as frequencies and percentages. Continuous variables were presented as (Means ± SD). Pearson Chi-Square test and Fisher's Exact test were used to find the association between categorical variables. A p-value of ≤ 0.05 was considered as significant.

Results

Table1: Given the mean differences of lipid profile including (LDL, HDL,triglyceride andcholesterol) according to the study group including (polycystic ovarian syndrome with insulin resistance, control group).

Table (1) The mean differences of lipid profile according to study group (N=80)

Lipid profile	Study group	N	Mean ± SD	P-value
LDL (mg/dl)	Polycystic ovarian syndrome and Insulin resistance	40	105.20 ± 22.94	**<0.001***
	Control group	40	75.10 ± 21.95	
HDL (mg/dl)	Polycystic ovarian syndrome and Insulin resistance	40	43.11 ± 12.95	**<0.001***
	Control group	40	53.35 ± 10.49	
Triglyceride (mg/dl)	Polycystic ovarian syndrome and insulin resistance	40	144.48 ± 75.61	**<0.001***
	Control group	40	89.80 ± 29.58	
Cholesterol (mg/dl)	Polycystic ovarian syndrome and insulin resistance	40	176.31 ± 29.96	**<0.001***
	Control group	40	146.29 ± 25.04	

*P value ≤ 0.05 was significant.

Table 2: The mean differences of lipid profile including (LDL, HDL, triglyceride and cholesterol) according to age including (< 30 years and ≥ 30 years) among patients polycystic ovarian syndrome with insulin resistance.

Table (2) The mean differences of lipid profile according to age among patients polycystic ovarian syndrome with insulin resistance (N=40)

Lipid profile	Age (years)	N	Mean ± SD	P-value
LDL (mg/dl)	<30	29	99.67 ± 23.85	**0.011***
	≥ 30	11	119.76 ± 11.76	
HDL (mg/dl)	<30	29	43.65 ± 14.90	0.677

	≥ 30	11	41.70 ± 5.39	
Triglyceride(mg/dl)	<30	29	131.95 ± 79.05	0.089
	≥ 30	11	177.52 ± 56.05	
Cholesterol (mg/dl)	<30	29	168.47 ± 28.64	**0.006***
	≥ 30	11	196.97 ± 23.58	

*P value ≤ 0.05 was significant.

Figure (1) shows distribution of patients polycystic ovarian syndrome with insulin resistance according to body mass index (kg/m^2) including (normal (18.5-24.9), overweight (25-29.9) and obese (≥ 30)). Normal body mass index was represented in (N=10, 25.0%) of patients, overweight represent (N=12, 30.0%) of patients and less than half of patients (N=18, 45.0%) presented with obesity. Mean body mass index of patients was (29.93 ± 5.64) kg/m^2.

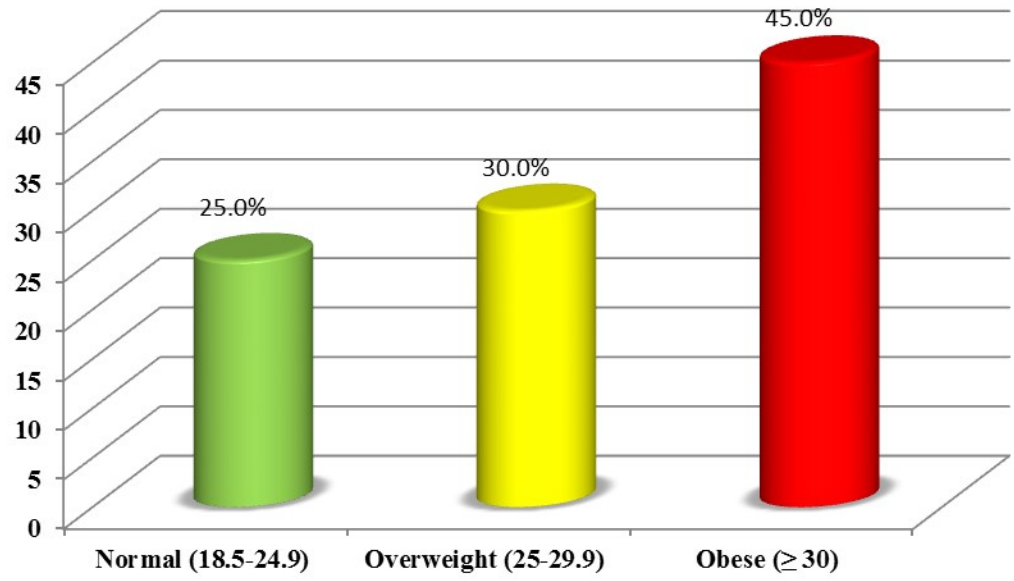

Figure (1) Distribution of patients with polycystic ovarian syndrome with insulin resistance according to body mass index (kg/m^2)

Discussion

The most prevalent endocrine disorder in women of reproductive age is commonly acknowledged to be polycystic ovarian syndrome (PCOS), which can also cause dyslipidemia, hyperandrogenism, hyperinsulinemia, oxidative stress, and infertility. It is now understood that dyslipidemia contributes significantly to the emergence of PCOS[7].The presented study shows an increase in cholesterol, triglycerides, and bad cholesterol (LDL) and a decrease in good cholesterol (HDL) in women patients compared to the control

group, as shown in Table (1). These results are consistent with earlier research that was published and revealed dyslipidemia patterns [10-13]. A recent study showed that mild hypercholesterolemia is typically observed in women with PCOS [14]. A recent animal study indicated that feeding prepubertal rats a high-fat diet caused metabolic and ovarian changes that are typically seen in PCOS, which raises the possibility that hyperlipidemia may have an effect on the hormonal profile [15]. Oocyte quality and early embryo growth are clearly impacted by obesity, which is brought on by apoptosis, mitochondrial malfunction, and endoplasmic reticulum stress brought on by lipotoxicity[16].According to Shaman A. A. et al.'s findings on lipid profiles based on androgen levels[17], androgens may be a key factor in hyperlipidemia. However, recent research has indicated that hypomethylated genes involved in the synthesis of lipids and steroids may encourage the manufacture of androgen and other steroid hormones, which may help to partially explain the causes of hyperandrogenism in PCOS [18]. According to these research, hyperandrogenism is a significant contributor to lipid abnormalities, whereas alterations in lipid-related genes favor the development of hyperandrogenism. In comparison to women with PCOS and normal cholesterol levels, those with mild hypercholesterolemia have higher body mass indexes (BMIs), fasting insulin levels, and IR levels [14]. Dyslipidemia was found to be considerably more common in PCOS and insulin resistance patients in the current investigation. A significant amount of free fatty acids are released from adipose tissues into the systemic circulation as a result of insulin resistance, which inhibits the capacity of insulin to control lipolysis [12].The American College of Obstetricians and Gynecologists and the Androgen Excess and PCOS Society both advise that all PCOS patients undergo a thorough fasting lipid and lipoprotein evaluation as part of their cardiovascular risk assessment [11, 12, 19, 20]. Since LDL-C is the lipoprotein biomolecule that causes atherosclerosis the most, it continues to be the main target [11, 12, 21, 22]. After the examination of PCOS patients for dyslipidemia, Patients with normal fasting lipid profiles will need to be evaluated again in two years, or little period of time if there has been an increase in body weight[11, 12].

In the present study, The group of polycystic ovaries and insulin resistance patients was divided into two groups according to age (<30years and ≥ 30 years), showed that the results of patients ≥ 30 years old a significant increased the levels of both LDL, Cholesterol,No significant value was found in Triglycerides,HDL, as shown in Tables (2), The condition of women with PCOS might change with age [23].The prevalence of PCOS appears to decline with aging as the diagnostic criteria for PCOS may diminish or even normalize during the reproductive lifetime [24]. The most significant characteristics of PCOS are irregular menstrual cycles, polycystic ovary morphology, and hyperandrogenism, which may alter by age, It is unknown how long has passed since the onset of a specific symptom before the disease has appeared [25]. In other several studies, it has been shown Triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and total cholesterol (TC) were found to rise with aging in PCOS-affected women[26-28].

The prevalence of obesity among women worldwide has increased 2.5-fold from 6% to 15% over the past 40 years. Over a same period, there has been a corresponding rise in the prevalence of co-morbidities associated with obesity, of which there are more than 50 and which together represent a significant burden on both global health and socioeconomic development. Insulin resistance, which results from weight increase, compensatory hyperinsulinaemia, and the associated metabolic dysfunction are major mediators in the development of many obesity-related diseases. The metabolic syndrome's symptoms, such as type 2 diabetes (T2D), dyslipidaemia, hypertension, and obesity-related cancers such endometrial carcinoma, as well polycystic ovarian syndrome. Usually throughout adolescence, weight increase causes PCOS to become clinically evident [29]. And in the patients' group, the dominant characteristic was obesity by 45%, as shown in Figure(3.3), This is a common finding among women with the syndrome, according to many studies that confirmed the current study findings [30-32].

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References

1. Guzar SH, Jawad ES, Altahan MA, Hameed NN. The Effects of Trace Element levels on Poly cystic Ovary Syndrome in human female Sat Thi-Qar governorate Iraq. Research Journal of Pharmacy and Technology. 2019;12(9):4447-53.
2. Bostanci MS, Sagsoz N, Noyan V, Yucel A, Goren K. Comprasion of ovarian stromal and uterin artery blood flow measured by color doppler ultrasonography in polycystic ovary syndrome patients and patients with ultrasonographic evidence of polycystic. Journal of Clinical Gynecology and Obstetrics. 2013;2(1):20-6.
3. Joham AE, Norman RJ, Stener-Victorin E, Legro RS, Franks S, Moran LJ, et al. Polycystic ovary syndrome. The Lancet Diabetes & Endocrinology. 2022.
4. Kovacs GT, Norman R. Polycystic Ovary Syndrome: Cambridge University Press; 2007.
5. Zhu T, Goodarzi MO. Causes and consequences of polycystic ovary syndrome: insights from Mendelian randomization. The Journal of Clinical Endocrinology & Metabolism. 2022;107(3):e899-e911.
6. Crook M. Lipoprotein X: clinical implications. SAGE Publications Sage UK: London, England; 2013. p. 93-4.
7. Liu Q, Xie Y-j, Qu L-h, Zhang M-x, Mo Z-c. Dyslipidemia involvement in the development of polycystic ovary syndrome. Taiwanese Journal of Obstetrics and Gynecology. 2019;58(4):447-53.
8. Radons J. The human HSP70 family of chaperones: where do we stand? Cell Stress and Chaperones. 2016;21(3):379-404.
9. Costa EC, Sá JCFd, Stepto NK, Costa IBB, Farias Junior LF, Moreira SdNT, et al. Aerobic training improves quality of life in women with polycystic ovary syndrome. 2018.
10. Manikkumar R, Roy DD, Krishnan V, Vijayakumar T. Association of DNA damage and dyslipidemia with polycystic ovarian syndrome. Journal of Medical & Allied Sciences. 2013;3(1).
11. Wild RA, Rizzo M, Clifton S, Carmina E. Lipid levels in polycystic ovary syndrome: systematic review and meta-analysis. Fertility and sterility. 2011;95(3):1073-9. e11.
12. Wild RA, Carmina E, Diamanti-Kandarakis E, Dokras A, Escobar-Morreale HF, Futterweit W, et al. Assessment of Cardiovascular Risk and Prevention of Cardiovascular Disease in Women with the Polycystic Ovary Syndrome: A Consensus Statement by the Androgen Excess and Polycystic Ovary Syndrome (AE-PCOS) Society. The Journal of Clinical Endocrinology & Metabolism. 2010;95(5):2038-49.
13. Enechukwu CI, Onuegbu AJ, Olisekodiaka MJ, Eleje GU, Ikechebelu JI, Ugboaja JO, et al. Oxidative stress markers and lipid profiles of patients with polycystic ovary syndrome in a Nigerian tertiary hospital. Obstetrics & Gynecology Science. 2019;62(5):335-43.
14. Pergialiotis V, Trakakis E, Chrelias C, Papantoniou N, Hatziagelaki E. The impact of mild hypercholesterolemia on glycemic and hormonal profiles, menstrual characteristics and the ovarian morphology of women with polycystic ovarian syndrome. Hormone Molecular Biology And Clinical Investigation. 2018;34(3).
15. Patel R, Shah G. High-fat diet exposure from pre-pubertal age induces polycystic ovary syndrome (PCOS) in rats. Reproduction. 2018;155(2):139-49.
16. Broughton DE, Moley KH. Obesity and female infertility: potential mediators of obesity's impact. Fertility and sterility. 2017;107(4):840-7.
17. Spałkowska M, Mrozińska S, Gałuszka-Bednarczyk A, Gosztyła K, Przywara A, Guzik J, et al. The PCOS patients differ in lipid profile according to their phenotypes. Experimental and Clinical Endocrinology & Diabetes. 2018;126(07):437-44.
18. Pan J-X, Tan Y-J, Wang F-F, Hou N-N, Xiang Y-Q, Zhang J-Y, et al. Aberrant expression and DNA methylation of lipid metabolism genes in PCOS: a new insight into its pathogenesis. Clinical Epigenetics. 2018;10:1-12.

19. Mosca L. Guidelines for prevention of cardiovascular disease in women: a summary of recommendations. Preventive Cardiology. 2007;10:19-25.
20. Bulletins-Gynecology ACoP. ACOG Practice Bulletin no. 108: Polycystic ovary syndrome. Obstet Gynecol. 2009;114(4):936-49.
21. Ahmed Alobaidi AH AM, Ahmad SS, Alsamarai AM. Dyslipidemia and oxidative stress in Iraqi women with poly cystic ovary syndroms. World J Pharm Pharm Sci. 2015;4(09, 39-48).
22. Kim JJ, Choi YM. Dyslipidemia in women with polycystic ovary syndrome. Obstetrics & gynecology science. 2013;56(3):137-42.
23. Pavlik E, DePriest P, Gallion H, Ueland F, Reedy M, Kryscio R, et al. Ovarian volume related to age. Gynecologic oncology. 2000;77(3):410-2.
24. Lauritsen MP, Bentzen J, Pinborg A, Loft A, Forman J, Thuesen L, et al. The prevalence of polycystic ovary syndrome in a normal population according to the Rotterdam criteria versus revised criteria including anti-Müllerian hormone. Human reproduction. 2014;29(4):791-801.
25. Kushnir VA, Halevy N, Barad DH, Albertini DF, Gleicher N. Relative importance of AMH and androgens changes with aging among non-obese women with polycystic ovary syndrome. Journal of ovarian research. 2015;8(1):1-7.
26. Carmina E, Campagna AM, Lobo RA. A 20-year follow-up of young women with polycystic ovary syndrome. Obstetrics & Gynecology. 2012;119(2 Part 1):263-9.
27. Liang S-J, Hsu C-S, Tzeng C-R, Chen C-H, Hsu M-I. Clinical and biochemical presentation of polycystic ovary syndrome in women between the ages of 20 and 40. Human Reproduction. 2011;26(12):3443-9.
28. Teede HJ, Misso ML, Costello MF, Dokras A, Laven J, Moran L, et al. Recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Human reproduction. 2018;33(9):1602-18.
29. Barber TM, Franks S. Obesity and polycystic ovary syndrome. Clinical endocrinology. 2021;95(4):531-41.
30. Barber TM, Hanson P, Weickert MO, Franks S. Obesity and polycystic ovary syndrome: implications for pathogenesis and novel management strategies. Clinical Medicine Insights: Reproductive Health. 2019;13:1179558119874042.
31. Glueck CJ, Goldenberg N. Characteristics of obesity in polycystic ovary syndrome: Etiology, treatment, and genetics. Metabolism. 2019;92:108-20.
32. Zhou L, Ni Z, Yu J, Cheng W, Cai Z, Yu C. Correlation between fecal metabolomics and gut microbiota in obesity and polycystic ovary syndrome. Frontiers in Endocrinology. 2020;11:628.