(International Print/Online Journal)

SJIF IMPACT FACTOR: 5.565
PUBMED-National Library of Medicine ID-101739732

ISSN (Print): 2209-2870
ISSN (Online): 2209-2862

International Journal of Medical Science and Current Research (IJMSCR)
Available online at: www.ijmscr.com
Volume 7, Issue 2 , Page No: 157-162
March-April 2024

IJMSCR

Study of Lipid Profile in Women with Polycystic Ovary Syndrome and compare with Healthy Women

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Type of Publication: Original Research Paper
Conflicts of Interest: Nil

Abstract

Background: Polycystic ovary syndrome (PCOS) is a condition in which the ovaries produce an abnormal amount of androgens, male sex hormones that are usually present in women in small amounts. The name polycystic ovary syndrome describes the numerous small cysts (fluid-filled sacs) that form in the ovaries.

Methods: This paper focuses on analyzing the importance of biochemical parameters in Polycystic ovary syndrome including lipid profile and their implications in the evolution of the disease by using standard procedure of selected biochemical parameters.

Results: The present study showed that the value of total cholesterol, Triglyceride, LDL were significantly high and HDL significantly low in Polycystic ovary syndrome patient compare to normal patients.

Conclusions: According to this study, women with PCOS exhibited higher levels of triglycerides, LDL, and cholesterol in their atherogenic lipoprotein profile. particularly in the obese population, which may be a risk factor for subsequently developing cardiovascular complications.

Keywords: TC, TG, HDL, LDL, VLDL, PCOS

Introduction

The most prevalent endocrine condition affecting women who are of reproductive age is called PCOS, or polycystic ovarian syndrome. It has long been known that the presence of persisting ovaries with several [2–8 mm] tiny cysts and hyper vascularized androgen-secreting stroma is associated with menstrual cycle disturbance [oligomenorrhea or amenorrhoea], obesity, and symptoms of excess androgen [hirsutism, alopecia, acne] [1]. The common European perspective is that the illness encompasses any of the endocrine problems [high serum concentrations of luteinizing hormone [LH] and/or androgen] as well as the signs and symptoms listed above [2]. It is generally agreed upon in North America that the illness is identified by the co-occurrence of hyperandrogenism and ovulatory failure in the absence of non-classical adrenal hyperplasia, without the necessity for ultrasonic scanning to determine the presence of polycystic ovaries [3].

Free androgen index [FAI] is frequently elevated due to common observations of higher circulating testosterone concentrations and decreased globulin binding sex hormones [SHBG] concentrations. It is unknown how hormone abnormalities affect lipoprotein deficiencies. On lipoprotein metabolism, however, it is well established that androgen and oestrogens have opposing effects. Hepatic regulation of insulin sensitivity, body fat distribution, and lipoprotein metabolism may mediate these effects [4]..

The lipoprotein profile of obese PCOS individuals is typified by lower HDL-C (high-density lipoprotein cholesterol) concentrations and higher plasma triglycerides, which are similar to those observed in type 2 diabetic participants [5]. In PCOS, low-density lipoprotein cholesterol [LDL-C] is frequently just marginally raised. Nevertheless, a straightforward quantitative estimate of LDL content could be deceptive because LDL does not exist as homogenous particles. On the other hand, there exist many subpopulations of LDL particles that vary in terms of their size, density, atherogenic propensity, and lipid makeup. Even at normal LDL-cholesterol concentrations, small and compact LDL particles (LDL-III) are associated with a higher frequency of coronary heart disease (CHD) and type 2 diabetes in circulation due to their demonstrated greater atherogenic potential compared to bigger LDL species (LDL-1 and LDL-II) [6]. Information about PCOS-related topics is still lacking.

The concentration of low, dense LDL in the normal population is mostly determined by plasma triglyceride levels [7].

Research revealed that when plasma triglyceride concentrations are raised, the activity of the enzyme hepatic lipase [HL] enriches the larger LDL particles, making them amenable for conversion to smaller species [8]. There is a substantial correlation between upregulating androgen activity and downregulating oestrogen activity and increased HL activity. However, it was not documented to evaluate anomalies in the LDL subfraction profiles in women with PCOS based on the link between endogenous hyperandrogenemia and HL activity [9].

A number of factors, including obesity, diabetes mellitus, cigarette smoking, and genetics, might worsen the atherogenic process and coronary heart disease (CHD) in addition to dyslipidemia in PCOS. Delaying the beginning and slowing the progression of atherosclerosis has been the focus of significant public health efforts in recent decades. Managing dyslipidemia, especially in people at high risk, was one of the most effective methods since atherosclerosis is strongly linked to the accumulation of lipoprotein particles. [10].

Materials And Methods

A study was conducted in Departments of Biochemistry and Gynaecology & Obstetrics for a period of three year in Pacific Institute of Medical Sciences, Udaipur.

A total number of 100 patients admitted at Pacific Institute of Medical Sciences Udaipur, was form the subjects of the present study. Out of these 100 patients were suffering from polycystic ovarian syndrome and 50 were normal patients. Efforts will be made to match all anthropometric factors comparable to both the groups of patients.

1. Group 1: 50 Confirm healthy women patients.
2. Group 2: 50 Confirm PCOS women.

Inclusion Criteria

1. Patient who is willing to participate.
2. Radiologically confirmed report that showed multiple small cysts in ovary.
3. Irregular Menses
4. Obese women

Exclusion Criteria

1. Other endocrinal disorders like diabetes mellitus, untreated hypothyroidism 2. Patients those on drug treatment like antihypertensive, antiplatelet, lipid lowering agents, drug affecting glucose.
2. Vitamin D drugs
3. Androgen-secreting Tumor
4. Impaired glucose tolerance
5. Pregnancy
6. Women taking oral contraceptive pills
7. breast feeding
8. Any other chronic disease

The blood samples for analysis were taken at least after minimum of 12 hours of complete fasting. The subject was asked to have a light, fat free diet on the day prior to sampling. The venepuncture was done in the cubital fossa, Tourniquet was used but was released just before sampling to avoid artefactual increase in the concentration of serum lipids. About 5 ml blood was drawn using perfectly dry and sterile disposable syringes. The serum was separated within 2 hours of collection to prevent artefactual changes in concentration of HDL. The sample were analysed the same day or within 48 hours. The lipid and lipoprotein assay was done using the fully automated analyser EM 360.A total number of 100 patients admitted at Pacific Institute of Medical Sciences

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Clinical Methodology

Symptoms (Weakness, loss of appetite, Nausea, Irregular Menses), serum Lipid Profile were recorded by using Autoanalyzer EM-360.

Statistical Analysis

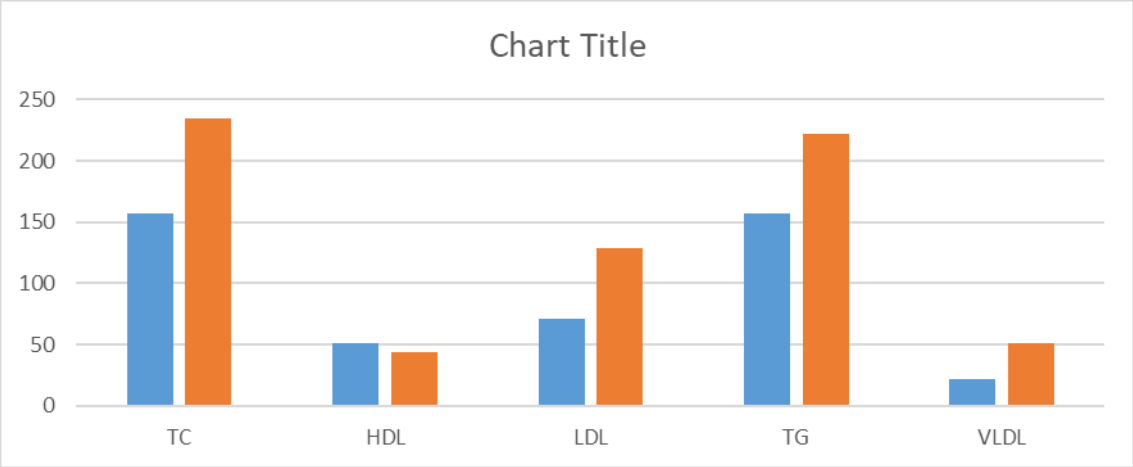
For the quantitative analysis, we used the software SPSS software. In this meta-analysis, all p values reported were two-tailed with the statistical significance set at $\leqslant 0.05$.

Result

Table 1: Comparison of Lipid Profile between Normal Patient and Polycystic ovarian syndrome patients

S. No	Test	Normal Patient		PCOS Cases		P Value
		MEAN	SD	MEAN	SD	
1	TC	157.45	9.5	234.87	24	<0.0001
2	HDL	51.51	5.94	43.65	6.93	<0.005
3	LDL	70.71	15.55	129.09	31.53	<0.005
4	TG	157.06	3.0	221.58	12.5	<0.0001
5	VLDL	21.41	7.15	51.05	18.20	<0.005

Fig 1. Comparison of Lipid Profile between Normal Patient and Polycystic ovarian syndrome patients



The present study showed that the Mean and Standard deviation of Total Cholesterol (TC), Low density Lipoprotein (LDL), Very density Lipoprotein were significantly high in polycystic ovarian syndrome compared to normal patients. And the mean and standard deviation of HDL significantly low in in polycystic ovarian syndrome compared to normal patients (table 1 and fig1).

Discussion

Between PCOS patients and control subjects, we observed non-significant differences in TC, TG, LDL cholesterol, HDL cholesterol, VLDL cholesterol, TC/HDL cholesterol, and LDL cholesterol/HDL cholesterol. However, compared to controls, patients with PCOS had higher mean TC/HDL cholesterol and LDL cholesterol / HDL cholesterol, as well as lower HDL cholesterol and apoA-I. Patients with

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PCOS also had higher TG, TC, and LDL cholesterol levels (Goldstein *et al*) [3].

Low HDL was found to be a dyslipidemia variable in patients in our investigation. This is consistent with data from the South Indian population, where 93.3 percent of PCOS sufferers have low HDL. Hyperinsulinemia and hyperandrogenemia may be the cause of dyslipidemia in PCOS patients. This made it possible for adipocytes to experience an increase in catecholamine-induced lipolysis, which released free fatty acids into the bloodstream. Hypertriglyceridemia is brought on by increased secretion of VLDL due to elevated free liver fatty acids. Reduced HDL cholesterol and increased LDL cholesterol are the results of hypertriglyceridemia via the reverse cholesterol transfer route. The early-life androgenic stimulation of adipocytes PCOS in non-obesity n=20 n=20 t p, non-obese Control [mg/dL] total cholesterol 180.35 ± 7.7 7.73 0.05 209.91 ± 15.28 1.05 minimal density

The clinical features of the control group and the non-obese women with PCOS were similar in this study, and the difference was not statistically significant [P > 0.05]. This suggests that U/S and biochemical markers are necessary for the diagnosis of PCOS, particularly in non-obese women. This outcome concurs with the findings of (Goldstein *et al*) [3].

Total cholesterol and triglycerides were considerably higher in non-obese women in our research of PCOS patients than in patient controls. These findings align with the analysis conducted by Pagotto et al. [14], which found that while plasma, HDL, LDL, and VLDL were not statistically significantly different between the non-obese PCOS group and the control group, serum total cholesterol and serum triglycerides were higher. Furthermore, Cinar *et al* [1] demonstrated that non-obese PCOS patients had statistically significant increases in total cholesterol and LDL when compared to controls, but no statistically significant differences were found in serum HDL or triglycerides.

In our analysis of obese women, we found that the PCOS group had higher total cholesterol, triglycerides, and LDL than the non-PCO group. This difference was statistically significant [P < 0.01] for LDL and extremely statistically significant [P < 0.001] for total cholesterol and triglycerides. The HDL and VLDL groups did not differ statistically significantly from one another. These outcomes concur with those of Khomami *et al* [10].

Besides, Jones et al. [15] revealed that obese PCOS girls had lower HDL, higher triglycerides, and higher cholesterol than the non-obese control group. Additionally, Jayasekara et al. [16] demonstrated that although blood HDL was lower in PCOS than the control group, there were statistically significant increases in serum triglycerides, cholesterol, and LDL compared to the matched control group. According to their research, compared to obese regulation, obese PCOS patients' serum cholesterol and LDL levels increased statistically considerably. According to Goldstein et al.'s investigation [3], the triglyceride rise in obese PCOS patients was not statistically significant. However, the lipid profile of the PCOS group revealed a statistically significant increase in VLDL [P < 0.01] and triglycerides [P < 0.05] when compared to the control group. While HDL and LDL decreased, the rise in cholesterol was statistically nonsignificant [P > 0.05].

An important health concern is that women with PCOS may have an underlying metabolic pathological condition (such as insulin resistance or high insulin levels) that is linked to a long-term risk of coronary heart disease. By using limited classification criteria, many PCOS patients who have a high risk factor for coronary heart disease may be missed [8]. Over a two-year period, Yang *et al* [17] assessed 143 women under 40 who had coronary angiography performed to test for valvular disease or chest discomfort. Forty-two percent of these women had polycystic ovaries when transvaginal ultrasonography was used to assess their ovaries. Compared to women with normal ovaries, those with polycystic ovaries had more severe coronary artery disease. The prevalence of polycystic appearing ovaries in patients with coronary heart disease was reported twice in a general population of women [9].

In our research, we found a statistically significant association between low HDL and high serum total cholesterol in patients with polycystic ovarian syndrome; however, this relationship did not hold true for high LDL and triglycerides. The control groups and the patients did not exhibit a significant difference in mean serum lipid levels. It should be noted that when the results were broken down by

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body mass index, patients with BMI > 30 showed significantly different average blood triglycerides, while patients with BMI < 25 and BMI < 30 showed significantly different total serum cholesterol. The case group outperformed the control group in every one of these subjects. The dyslipidaemia in females with polycystic ovarian syndrome has been examined in several research. Though different studies tend to report conflicting results in their study population due to factors such as race, genetics, diet, lifestyle and differences in economics. Comparing 27 PCOS individuals to 22 weight-stratified (obese and nonobese) eumenorrheic control participants revealed the significance of insulin in lipid abnormalities reported in hyperandrogenic women with PCOS [18]. Significantly reduced HDL concentrations and a weak connection between BMI and blood HDL levels were seen in PCOS-afflicted women.

Conclusion and Summary

According to this study, women with PCOS exhibited higher levels of triglycerides, LDL, and cholesterol in their atherogenic lipoprotein profile. particularly in the obese population, which may be a risk factor for subsequently developing cardiovascular complications.

Ethical Issues

Research project approved by the ethics committee of Pacific Institute of Medical Sciences, Udaipur-313005, Rajasthan, INDIA**.**

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