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Original Research Article

Estimation of Lipid Profile and Serum Lipoproteins in Polycystic Ovarian Syndrome in Tertiary Care Teaching Hospital

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Abstract

Background: Dyslipidaemia is the most common metabolic abnormality in Polycystic ovary syndrome (PCOS). Women with PCOS are at an increased risk for cardiovascular diseases. Lipoprotein is an independent risk factor for the development of atherosclerosis. Elevated Lipoprotein (a) along with dyslipidemia and a host of other derangements in PCOS may lead to premature atherosclerosis. We in the current study tried to evaluate the lipid profile and serum lipoprotein (a) in women diagnosed with PCOS in our tertiary care institute.

Methods: This cross-sectional study was conducted in the Department of Obstetrics and gynecology and Biochemistry, Prathima Institute of Medical Sciences, Naganoor, Karimnagar, Telangana State. N=25 cases were studied during the period of the study. N=25 age-matched controls were also included in the study. The serum was isolated from a five mL peripheral venous blood sample taken by venepuncture from both patients and controls following a 12-hour overnight fast. The immunonephelometric technique was used to quantify serum Lipoprotein (a) levels.

Results: The mean levels of total cholesterol in the cases were 208.56 ± 35.67 mg/dl. Taking a cutoff of < 200 mg/dl it was found that 60% of the cases in the study group were below this level and 40% were above 200 mg/dl levels the p-value (<0.001). Serum Triglycerides (TG) among Cases and Controls have shown that 36% of the cases and 80% of the controls are in the normal range (<150 mg/dl). 48% of cases and 20% of controls were in the borderline range (150-199 mg/dl). 16% of cases were found to be having values > 200 mg/dl.

Conclusion: Total cholesterol, triglycerides, low-density lipoproteins, and very-low-density lipoproteins were found to be consistently higher in PCOS patients as compared to the normal controls. Elevated Lipoprotein (a) was also found in most of the PCOS cases in the study. All these abnormalities indicate that polycystic ovarian syndrome may lead to the development of an atherogenic lipid profile and put the patient at risk for future metabolic syndrome.

Keywords: Polycystic ovarian syndrome (PCOS), Lipid profile, Lipoprotein (a), Total cholesterol, Triglycerides

Introduction

PCOS is one of the most prevalent endocrine diseases in women of reproductive age, with a prevalence of 6-10% of women globally based on the National Institute of Health criteria and as high as 15% when the wider Rotterdam

criteria are used. [1] The incidence of PCOS linked endocrinopathy that affects around 7% of women of reproductive age. The triad of hirsutism, amenorrhoea, and obesity is used to constitute the basis for clinical diagnosis. Following that, it was discovered that PCOS has a very diverse clinical picture and that its etiology is multifaceted. [2] This condition is commonly associated with obesity and insulin resistance. When compared to the general population, cardiovascular risk factors appear to cluster in women with PCOS. [3] Obesity, insulin resistance, dyslipidemia, endothelial dysfunction, and the presence of metabolic syndrome are among the risk variables examined in relation to PCOS. [4–7] Given the high prevalence of cardiovascular risk factors in women with PCOS, it's remarkable that a retrospective cohort analysis of women with PCOS revealed no increased coronary heart disease. [8] However, no evidence of long-term follow-up is currently available. It's been suggested that women with PCOS may be exposed to a protective factor, such as estrogens over an extended period. Studies have reported dyslipidemia is associated with PCOS. Women with PCOS have a lipid profile that includes higher TG levels and low levels of high-density lipoprotein-cholesterol (HDL-C). [9] These alterations are in line with the lipid profile that is frequently associated with insulin resistance. Insulin resistance has long been recognized to affect lipid metabolism. Increased plasma TG concentrations are caused by increased secretion of very-low-density lipoprotein (VLDL) particles by the liver. Following that, the activity of CE transfer protein exchanges TGs for cholesteryl ester (CE). As a result of this mechanism, TG-enriched high-density lipoprotein (HDL) particles catabolize more quickly, whereas CE-enriched VLDL particles are transformed into tiny dense low-density lipoprotein (LDL) particles. [10] Insulin resistance leads to lower plasma levels of HDL-C and apolipoprotein (apo) A-I, as well as increased levels of apo B. [11-13] Early detection of modifiable cardiovascular risk factors might aid in the prevention of heart disease. So, the goal of our research is to see if there is a link between an elevated Lipoprotein (a) level and dyslipidemia in patients diagnosed with PCOS in our tertiary care hospital in South India.

Material and Methods

This cross-sectional study was conducted in the Department of Obstetrics and gynecology and Biochemistry, Prathima Institute of Medical Sciences, Naganoor, Karimnagar, Telangana State. Institutional Ethical approval was obtained for the study. Written consent was obtained from all the participants of the study. Voluntarily willing patients were included in the study.

Inclusion criteria

1. Patients diagnosed with PCOS
2. No history of Diabetes Mellitus, Hypertension, Renal Disease, and Liver disorders
3. No history of other endocrine abnormalities
4. Females aged between 18 to 35 years
5. No clinical evidence of hyperandrogenemia

Exclusion criteria

1. History of menstrual disorders
2. Abnormal menstrual cycle
3. Do not want to participate in the study voluntarily

Based on the inclusion and exclusion criteria n=25 cases were studied during the period of the study. N=25 age-matched controls were also included in the study. Detailed history including menstrual history was obtained. Baseline data including age, body-mass index (BMI; kg/m2), detailed medical history, clinical examination, and relevant investigations were recorded. The serum was isolated from a five mL peripheral venous blood sample taken by venipuncture from both patients and controls following a 12-hour overnight fast. The immunonephelometric

technique was used to quantify serum Lipoprotein (a) levels. On the Erba Chem 7 Semi-Automatic Biochemistry Analyzer, total cholesterol (TC), serum triglycerides (TG), HDL cholesterol, fasting blood glucose, blood urea, and serum creatinine were measured using commercial kits. Friedewald's method was used to determine serum very-low-density lipoprotein (VLDL) cholesterol and LDL cholesterol from the values of TC, TG, and HDL cholesterol. The ratios of TC/HDL cholesterol and LDL cholesterol/HDL cholesterol were calculated in this study. [9]

Statistical analysis: The data was collected and uploaded on an MS Excel spreadsheet and analyzed by SPSS version 19 (Chicago, IL, USA). Quantitative variables were expressed on mean and standard deviations and qualitative variables were expressed in proportions and percentages. Fisher's exact test has been used to find the difference between two proportions.

Results

The distribution of the study samples according to the age is given in table 1. The cases and controls are divided into 4 groups <20 years, 21-25 yrs, 26-30 yrs, >31 – 35 yrs. Maximum numbers of cases are in the age group of 26 - 30 yrs (44%). The mean age of the cases in the study was 27.5 ± 8.5 years and the mean age of the controls group in the study was 24.5 ± 9.5 years. The mean age between the cases and controls was not statistically significant (P>0.05)

Table 1: Distribution of cases and control included in the study

Age group in years	*PCOS* Cases		Controls		P-value
	Frequency	*percentage*	*Frequency*	*percentage*	
18 - 20	3	12.0	3	12.0	0.99
21 – 25	8	32.0	10	40.0	0.25
26 – 30	11	44.0	8	32.0	0.33
31 – 35	3	12.0	4	12.0	0.87
Total	25	100	25	100	-

A comparison of BMI has been done between the two groups and has been shown in table 2. A slightly higher mean BMI of 25.82 ± 2.65 kg/m^2 was recorded in cases than in controls with a BMI of 24.59 ± 1.95 Kg/m^2 but the difference in mean BMI between the two groups was not statistically significant (P>0.05).

Table 2: BMI distribution of cases and control of the study

BMI scores Kg/m^2	*PCOS Causes*		*Controls*	
	Frequency	*percentage*	*Frequency*	*percentage*
< 18	4	16.0	3	12.0
18.1 – 25.0	8	32.0	12	48.0
25.1 – 30.0	10	40.0	7	28.0
> 30.0	3	12.0	3	12.0
Mean BMI	25.82 ± 2.65		24.59 ± 1.95	

Estimation of lipid profile between cases and controls has been shown in table 3. The mean levels of total cholesterol in the cases were 208.56 ± 35.67 mg/dl. Taking a cutoff of < 200 mg/dl it was found that 60% of the cases in the study group were below this level and 40% were above 200 mg/dl levels. The mean cholesterol levels were significantly higher in cases as compared to

the controls p-value (<0.001). Serum Triglycerides (TG) among Cases and Controls have shown that 36% of the cases and 80% of the controls are in the normal range (<150 mg/dl). 48% of cases and 20% of controls were in the borderline range (150-199 mg/dl). 16% of cases were found to be having values > 200 mg/dl. Distribution of Serum Low-Density Lipoproteins (LDL) among Cases and Controls have been given in table 14. 92% of the cases and 98% of the controls were in the normal range (≤129 mg/dl). Distribution of Serum Very Low-Density Lipoprotein (VLDL) among Cases and Controls shows 88% of the cases and 96% of the controls are in the normal range (≤40 mg/dl). Distribution of Serum High-density Lipoprotein (HDL) among Cases and Controls has been given in table 3. 96% of cases and 92% of controls were having values of HDL below 40 mg/dl. Higher mean Lp(a) is recorded in cases compared to controls but the difference in mean Lp(a) between the two groups was not statistically significant (P>0.05) However, when the distribution of Lipoprotein(a) is divided into those > 30mg/dl, samples with Lp (a) ≥ 30mg/dl are found 20% of cases were above this value (Table 3)

Table 3: Comparison of lipid profile and Lp(a) between controls and cases

Lipid parameters	*PCOS Cases*		*Controls*		*P-value*
	Mean	*± SD*	*Mean*	*± SD*	
Cholesterol (mg/dl)	208.56	35.67	175.94	18.87	0.001*
Triglycerides (mg/dl)	165.33	50.32	135.43	22.34	0.013*
LDL (mg/dl)	125.21	25.64	120.22	15.61	0.251
HDL (mg/dl)	38.99	8.52	43.21	7.23	0.324
VLDL (mg/dl)	25.74	6.63	23.17	6.50	0.661
Lp(a) (mg/dl)	17.55	6.8	22.03	12.15	0.312

* Significant

In Table 4 we have tried to compare total cholesterol levels and triglyceride levels among obese and non-obese PCOS patients. Interestingly we found that the difference is statistically not significant (p-value 0.034) was obtained between obese and non-obese cases for Total cholesterol. However, the differences between the serum triglycerides in both groups were not found to be significant.

Table 4: Comparison of lipid parameters among obese and non-obese PCOS patients

Body Mass Index (BMI)	Cases (n)	Total Cholesterol (mg/dl)	Triglycerides (mg/dl)
		Mean ± SD	Mean ± SD
Obese PCOS (BMI > 25 kg/m^2)	17	208.41 ± 35.12	171.24 ± 50.24
Non-Obese PCOS (BMI < 25 kg/m^2)	8	194.72 ± 40.31	148.60 ± 40.80
P value	-	0.034*	0.236

* Significant

Discussion

The most prevalent endocrine condition affecting women is polycystic ovary syndrome. It is characterized by hyperandrogenism, amenorrhoea, oligomenorrhoea, and infertility in women of reproductive age. Chronic anovulation, clinical or biochemical hyperandrogenism, obesity, and polycystic ovaries are all hallmarks of PCOS. Hyperandrogenism relates to oligomenorrhoea or amenorrhoea, and clinical signs of hirsutism or acne may

be present. [2] The majority of dyslipidemia and PCOS investigations have focused on cholesterol and triglyceride levels (TGs). Women with PCOS have a lipid profile that includes higher TG levels and low levels of high-density lipoprotein-cholesterol (HDL-C). [14] These alterations are in line with the lipid profile that is frequently associated with insulin resistance. Insulin resistance has long been recognized to affect lipid metabolism. Increased plasma TG concentrations are caused by increased secretion of very-low-density lipoprotein (VLDL) particles by the liver. Following that, CE transfer protein activity trades TGs for cholesteryl esters (CE). TG-enriched high-density lipoprotein (HDL) particles catabolize more quickly because of this process, whereas CE-enriched VLDL particles are converted into microscopic dense low-density lipoprotein (LDL) particles. [15] Insulin resistance causes plasma levels of HDL-C and apoprotein (apo) A-I to drop, while apo B levels rise. [16-18] In women with PCOS, ovarian and/or adrenal steroid production may alter lipid metabolism in addition to insulin resistance. Sex steroids have a complex effect on lipid metabolism, including both androgens and estrogens. Hyperandrogenism has been associated with increased hepatic lipase (HL) activity. This enzyme, which is involved in the breakdown of HDL particles, is sexually dimorphic, with exogenous androgens increasing activity and estrogens decreasing it. [19, 20] In our study, PCOS subjects had higher mean values of TC, TG, and LDL than controls. This is in line with similar research by Olivier et al., [21] who found an increase in triglycerides, cholesterol, and LDL-C, as well as a reduction in HDL-C and apo A-I. According to Berneis et al., [22] low HDL-C is prevalent in PCOS patients, although hypertriglyceridemia is unusual. On the contrary, they discovered that the most prevalent lipid change determining cardiovascular risk, an increase in LDL-C, is not present in all PCOS groups. In addition to overall LDL-C levels, the quality of LDL may have a direct impact on CV risk. The Adult Treatment Panel III of the National Cholesterol Education Program recognizes that tiny, dense LDL has a 3-fold higher risk of coronary artery disease and is classified as an emerging CV risk factor. In this study, we discovered that PCOS patients had lower HDL-C than controls, whereas PCOS patients have greater mean VLDL. This is in line with the findings of Wild et al., who found that women with PCOS had greater triglycerides and VLDL-C, as well as lower HDL2-C and A1:A2 ratios. In PCOS, researchers discovered decreased levels of HDL2 cholesterol and greater levels of apolipoprotein B. Obese women showed lower HDL-C and apo A-I levels, as well as greater triglycerides and VLDL-C levels. [23] In comparison to the controls, we found a substantially larger number of patients with Lp(a) >30 mg/dl (P 0.05). Even though the mean Lp(a) in cases is greater than in controls, the difference is not statistically significant. Our findings are consistent with those of Ahmed M Mohamadin et al., [24] who found that PCOS patients had greater levels of circulating ADMA, total homocysteine, hsCRP, Lp(a), and fibrinogen than healthy controls. [25]

Conclusion

Within the limitations of the current study, it can be concluded that total cholesterol, triglycerides, low-density lipoproteins, and very-low-density lipoproteins were found to be consistently higher in PCOS patients as compared to the normal controls. Elevated Lipoprotein (a) was also found in most of the PCOS cases in the study. All these abnormalities indicate that polycystic ovarian syndrome may lead to the development of an atherogenic lipid profile and put the patient at risk for future metabolic syndrome.

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