LIPID PROFILE IN PREMENOPAUSAL WOMEN WITH POLYCYSTIC OVARIAN SYNDROME A CROSS SECTIONAL STUDY

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ABSTRACT

Introduction: The most frequent endocrinopathy affecting women in their reproductive years is PCOS, which affects 5% to 10% of them. PCOS was initially reported by Stein and Leventhal in 1935.

Aims: To assess lipid profile in premenopausal women with polycystic ovarian syndrome.

Materials and method: It's a hospital based, cross-sectional study. The study was conducted in Eden Hospital, Medical College and Hospital, Kolkata over a period from March 2019 to March 2020. A total of 71 samples have been included in this study.

Result: The patients' mean low density lipoprotein (mg/dl) in the control group was 77.0000± 9.0277.The mean low density lipoprotein (mg/dl) of patients in the PCOS group was 141.4080± 36.4961.The difference between the two groups' mean low density lipoprotein (mg/dl) was statistically significant ($p<0.0001$). Additionally, the patients' mean high density lipoprotein (mg/dl) in the control group was 80.3333± 18.7466.Patients in the PCOS group had an average high density lipoprotein of 40.2084± 6.3429 mg/dl.The mean high density lipoprotein (mg/dl) varied significantly between the two groups ($p<0.0001$).

Conclusion: Our study concludes that anthropometric parameters (weight, BMI, waist circumference) and lipid profile have significant correlation with premenopausal patients with PCOS. Dyslipidemia is a significant cardiovascular risk factor that has to be assessed in these people in order to control the condition early and stop more cardiovascular problems.

Keywords: Lipid Profile, PCOS, Hyperandrogenism and Dyslipidemia.

INTRODUCTION

The most frequent endocrinopathy affecting women in their reproductive years is PCOS, which affects 5% to 10% of them. [1] 1935 saw the first description of PCOS by Stein and Leventhal. Since then, there has been a significant shift in our knowledge of the pathophysiology of PCOS, with a focus increasingly on the condition's connection to insulin resistance. PCOS is a chronic hyperandrogenic syndrome that can lead to oligomenorrhea, amenorrhea, infertility, diabetes mellitus, cardiovascular disease, endometrial cancer risk, and hirsutism, or abundant body hair. These are only a few of the major short- and long-term implications that PCOS patients face. PCOS is characterized by the following: (1a menstrual cycle ranging from more than 35 days or fewer than 8 cycles annually to an amenorrheic period, or total lack of menses; (2signs of excess androgen, such as hirsutism, alopecia, acne, acanthosis nigricans, or higher androgen levels in tests; (3) Once these other factors have been ruled out, hyperandrogenism and anovulation might occur. A woman does not always have polycystic ovaries in order to have this condition. Patients with PCOS are 67% to 86% likely to have polycystic ovaries. [2]

For women, the most prevalent cause of infertility is polycystic ovary syndrome. The indications and symptoms of PCOS are diverse and can impact the reproductive, endocrine, and metabolic systems to differing degrees of mildness and severity. [3] The symptoms of ovarian dysfunction associated with polycystic ovary syndrome (PCOS) include hyperanderogenism and anovulation. A masculine pattern of hair distribution may be the outcome of hyperanderogenism in PCOS women. Furthermore, the correlation between obesity and insulin resistance in PCOS raises the likelihood of prediabetes and type II diabetes, both of which exacerbate dyslipidemia. [4] Cardiovascular disorders and atherosclerosis are more likely to affect these people. In 5–10% of women who are of reproductive age, polycystic ovarian syndrome (PCOS) is the most frequent endocrine condition. Hyperandrogenism and persistent anovulation [5] are the hallmarks of this condition, which is linked to a wide variety of clinical and biochemical manifestations such as acne, hirsutism, irregular menstruation, obesity, infertility, hyperinsulinemia, glucose intolerance, and dyslipidemia. It is still debatable if lipid abnormalities are present in PCOS and how they develop. According to some research, PCOS women have greater rates of lipid abnormalities than do healthy women. [6]

MATERIALS AND METHOD

Study Design/ Experimental Design: Hospital based, cross-sectional study
Study Setting-The proposed study will be institute based conducted at OPD and indoor ward of department of obstetrics and Gynecology, MCH, Kolkata
Place of Study: Eden Hospital, Medical College and Hospital, Kolkata
Period of Study: 12 months (March 2019 to March 2020)
Study Population: Based on previous records.
Sample size: 71

Inclusion Criteria

We will include the patients as per the Rotterdam criteria (2003) of polycystic ovarian syndrome.
PCOS Case Definition:
The diagnosis is set if any 2 out of 3 criteria are met, in the absence of other entities that might cause these finding:

1. Oligo ovulation and/or anovulation.
2. Sign of androgen excess (clinical or biochemical)
3. Polycystic ovaries (one or two) on gynecological ultrasound.

Obese PCOS: BMI>29.9
Underweight PCOS: BMI<18.5

Exclusion Criteria

1. Other cause of oligo or anovulation
2. Any endocrine dysfunction
3. Other disease impaired lipid profile like thyroid, diabetes mellitus, and pancreatitis
4. Patients under treatment of the following drugs
    - Phenothiazine
    - Metoclopramide
    - Risperidone
    - SSRI
    - Verapamil

RESULT

Table No. 01: Distribution of Mean in all Parameter: Group

		Number	Mean	SD	Minimum	Maximum	Median	P-value
Low density oprotein (mg/dl)	Control	21	77.0000	9.0277	63.0000	92.0000	79.0000	<0.0001
	PCOS	50	141.4080	36.4961	56.0000	204.0000	140.0000	
High density oprotein (mg/dl)	Control	21	80.3333	18.7466	48.0000	114.0000	78.0000	<0.0001
	PCOS	50	40.2084	6.3429	28.0000	56.0000	40.0000	
Total cholesterol (mg/dl)	Control	21	158.7143	20.5600	112.0000	186.0000	163.0000	<0.0001
	PCOS	50	191.8200	26.1360	120.0000	250.0000	193.5000	
Serum triglyceride (mg/dl)	Control	21	133.9524	7.0815	122.0000	145.0000	135.0000	<0.0001
	PCOS	50	163.1600	23.0229	120.0000	200.0000	165.0000	
Fasting blood sugar (mg/dl)	Control	21	93.5714	8.1029	79.0000	108.0000	94.0000	0.0735
	PCOS	50	88.3600	12.0166	70.0000	115.0000	88.0000	
Weight (kg)	Control	21	48.1429	2.6322	44.0000	52.0000	48.0000	<0.0001
	PCOS	50	63.5000	3.1960	56.0000	70.0000	64.0000	
BODY MASS	Control	21	21.0314	1.1159	19.2900	22.5100	21.0500	<0.0001

INDEX (kg/m^2)	PCOS	50	28.1142	1.2800	25.1000	30.7000	27.9800	
Waist circumference (cm)	Control	21	83.4762	2.4211	79.0000	87.0000	84.0000	<0.0001
	PCOS	50	92.7200	2.7408	85.0000	98.0000	92.0000	

The patients' mean low density lipoprotein (mg/dl) in the control group was 77.0000± 9.0277. The mean low density lipoprotein (mg/dl) of patients in the PCOS group was 141.4080± 36.4961. The difference between the two groups' mean low density lipoprotein (mg/dl) was statistically significant ($p<0.0001$). The patients in the Control Group had an average high density lipoprotein of 80.3333± 18.7466 mg/dl. Patients in the PCOS group had an average high density lipoprotein of 40.2084± 6.3429 mg/dl. The mean high density lipoprotein (mg/dl) varied significantly between the two groups ($p<0.0001$). The patients in the Control Group had a mean total cholesterol of 158.7143± 20.5600 mg/dl. The mean total cholesterol (mg/dl) of the PCOS group's patients was 191.8200± 26.1360. The difference between the two groups' mean total cholesterol (mg/dl) was statistically significant ($p<0.0001$). The mean serum triglyceride (mg/dl) of the patients in the control group was 133.9524± 7.0815. The mean serum triglyceride (mg/dl) of patients in the PCOS group was 163.1600± 23.0229. The difference between the two groups' mean serum triglyceride levels (mg/dl) was statistically significant ($p<0.0001$). The patients' mean fasting blood sugar (mg/dl) in the control group was 93.5714± 8.1029. The mean fasting blood sugar (mg/dl) of the PCOS group's patients was 88.3600± 12.0166. The mean fasting blood sugar difference (in milligrams/dl) between the two groups was not statistically significant ($p=0.0735$). In Control Group, the mean weight (kg) of patients was 48.1429± 2.6322. In PCOS Group, the mean weight (kg) of patients was 63.5000± 3.1960. Difference of mean weight (kg) with both Group was statistically significant ($p<0.0001$). In Control Group, the mean body mass index (kg/m2) of patients was 21.0314± 1.1159. In PCOS Group, the mean body mass index (kg/m2) of patients was 28.1142± 1.2800. Difference of mean body mass index (kg/m2) with both Group was statistically significant ($p<0.0001$). In Control Group, the mean Waist circumference (cm) of patients was 83.4762± 2.4211. In PCOS Group, the mean Waist circumference (cm) (mean± s.d.) of patients was 92.7200± 2.7408. Difference of mean Waist circumference (cm) with both Group was statistically significant ($p<0.0001$).

DISCUSSION

Our study population comprises of 71 subjects of which Cases and Control were 50 (70.4%) and 21 (29.6%) respectively.

We compared the mean values of serum cholesterol, LDL, HDL, triglycerides, TSH, weight, BMI, waist circumference, and fasting blood sugar between the cases and controls in our research. In order to determine whether there was a significant association, we also compared the mean values of several lipids and anthropometric factors between cases and controls.

Saghafi-Asl M *et al* [7] (2013) study was aimed to investigate fat composition in connection to insulin resistance and anthropometric measures in overweight or obese women with polycystic ovarian syndrome (PCOS). In this cross-sectional study, 63 overweight or obese PCOS patients were divided into insulin-resistant (IR) and non-insulin-resistant (NIR) groups. The anthropometric

indices, such as body mass index (BMI), waist and hip circumference, waist to hip ratio (WHR), and waist to height ratio (WHtR), were also evaluated along with the lipid profile.

Our study demonstrated strong correlation between lipid profile and premenopausal PCOS patients. Triglycerides, cholesterol, and low density lipoprotein mean values were higher in case studies than in control, showing a positive association; in contrast, low density lipoprotein mean values were lower in case studies than in control, indicating a negative correlation. According to the study, the mean total cholesterol (mg/dl) of the patients in the Control Group was 158.7143± 20.5600. The mean total cholesterol (mg/dl) of the PCOS group's patients was 191.8200± 26.1360. The difference between the two groups' mean total cholesterol (mg/dl) was statistically significant ($p<0.0001$). The mean serum triglyceride (mg/dl) of the patients in the control group was 133.9524± 7.0815. The mean serum triglyceride (mg/dl) of patients in the PCOS group was 163.1600± 23.0229. The difference between the two groups' mean serum triglyceride levels (mg/dl) was statistically significant ($p<0.0001$). The patients' mean low density lipoprotein (mg/dl) in the control group was 77.0000± 9.0277. The mean low density lipoprotein of the PCOS group's patients was 141.4080± 36.491. The difference between the two groups' mean low density lipoprotein (mg/dl) was statistically significant ($p<0.0001$). The patients in the Control Group had an average high density lipoprotein of 80.3333± 18.7466 mg/dl. Patients in the PCOS group had an average high density lipoprotein of 40.2084± 6.3429 mg/dl. The mean high density lipoprotein (mg/dl) varied significantly between the two groups ($p<0.0001$).Thus, the current study validates that premenopausal PCOS patients have a higher atherogenic lipid profile. When comparing the PCOS group to the control, there was a substantial decrease in HDL levels and a significant increase in blood total cholesterol, LDL, and triglycerides.

Wild RA *et al* [8](2011) found that In PCOS, dyslipidemia is prevalent. Regardless of BMI, women with PCOS have increased LDL and nonHDL cholesterol in addition to documented changes in triglycerides and HDL cholesterol. It is recommended that all women diagnosed with PCOS undergo screening for dyslipidemia, which includes measuring LDL and non-HDL cholesterol levels, in order to effectively reduce cardiovascular risks.

Our study also included In premenopausal women with PCOS, the thyroid hormone profile, fasting blood sugar, and LFT are measured to look for any correlations.The mean values of these parameters did not significantly differ between the two groups. The investigation found no evidence of a significant relationship between these characteristics and our patients.

Valkenburg O *et al* [9](2008) found that ApoA-I, apoB, cholesterol, high-density lipoprotein (HDL)-cholesterol, triglycerides (TGs), and fasting insulin were measured. Using the Friedewald formula, low-density lipoprotein (LDL)-cholesterol was computed. There were 295 controls and 557 PCOS-afflicted women in total. When age and body mass index were taken into account, PCOS women showed lower levels of apoA-I (118 vs. 146 mg/dl) and HDL-cholesterol (46 vs. 55 mg/dl), along with higher median levels of insulin (10.1 vs. 6.9 mU/liter), TGs (95 vs. 81 mg/dl), cholesterol (196 vs. 178 mg/dl), and LDL-cholesterol (125 vs. 106 mg/dl) when compared to controls (a l P values ≤ 0.01). The apoB levels in the patients and controls were comparable. In women with PCOS,

the free androgen index, body mass index, SHBG, and estradiol were all found to be independent predictors of apoA-I levels.

The patients' mean low density lipoprotein (mg/dl) in the control group was 77.0000± 9.0277. The mean low density lipoprotein (mg/dl) of patients in the PCOS group was 141.4080± 36.4961. The difference between the two groups' mean low density lipoprotein (mg/dl) was statistically significant ($p<0.0001$). The patients in the Control Group had an average high density lipoprotein of 80.3333± 18.7466 mg/dl. Patients in the PCOS group had an average high density lipoprotein of 40.2084± 6.3429 mg/dl. The mean high density lipoprotein (mg/dl) varied significantly between the two groups ($p<0.0001$).

Nascimento JX *et al* [10] (2015) found that waist circumference, systolic and diastolic blood pressure, total cholesterol, triglycerides, low density lipoprotein cholesterol, high density lipoprotein cholesterol, fasting glucose, and body mass index. With the exception of HDL, all cardiovascular risk indicators had a greater probability of changing when the lipid accumulation product was over the 37.9 cm.mmol/L cut off value, according to logistic regression analysis. In women with polycystic ovary syndrome, the lipid buildup result appears to be significant to suggest a risk of cardiovascular illnesses.

Jahan S *et al* [11] (2018) found that Triglycerides, LDL, and mean total cholesterol were all substantially (p0.05) linked to PCOS. The probability of developing dyslipidemia in patients with elevated total cholesterol HDL ratio is projected to be 11.16 (95% CI = 3.9-33.1) times greater in PCOS patients than in group II. The lipid profile was analyzed using multivariate logistic regression, and the results showed that PCOS was substantially linked with only elevated LDL-C (>130 mg/dl).

Saha S *et al* [12](2008) found that There was a substantial increase in the levels of blood total cholesterol, triglycerides, LDL-C, and carotid intima-media thickness in young women with polycystic ovarian syndrome. There was no discernible difference in the HDL-C levels of the two groups of women. Women with Polycystic Ovary Syndrome showed a significant correlation between carotid intima-media thickness and serum LDL-C, triglycerides, and total cholesterol, but a negative correlation with HDL-C. According to their research, women with Polycystic Ovary Syndrome are predisposed to atherosclerosis from a young age.

Paneri S *et al* [13](2018) found that In comparison to control patients, PCOS cases showed significantly lower levels of HDL, triglycerides, LDL, and VLDL as well as cholesterol. The study found that high AMH levels raise the risk of hyperlipidemia linked to PCOS, polycystic ovarian syndrome, and other problems.

According to our research, the patients' mean total cholesterol (mg/dl) in the Control Group was 158.7143± 20.5600. The mean total cholesterol (mg/dl) of the PCOS group's patients was 191.8200± 26.1360. The difference between the two groups' mean total cholesterol (mg/dl) was statistically significant ($p<0.0001$). The mean serum triglyceride (mg/dl) of the patients in the control group was 133.9524± 7.0815. The mean serum triglyceride (mg/dl) of patients in the PCOS group was

163.1600± 23.0229. The difference between the two groups' mean serum triglyceride levels (mg/dl) was statistically significant (p<0.0001). The patients' mean waist circumference (in centimeters) in the control group was 83.4762± 2.4211. The mean patient waist circumference (in centimeters) in the PCOS group was 92.7200± 2.7408. The mean waist circumference (in centimeters) differed between the two groups in a statistically significant way (p<0.0001). The patients' mean fasting blood sugar (mg/dl) in the control group was 93.5714± 8.1029. The mean fasting blood sugar (mg/dl) of the PCOS group's patients was 88.3600± 12.0166. The mean fasting blood sugar difference (in milligrams/dl) between the two groups was not statistically significant (p=0.0735). In Control Group, the mean TSH (mIU/L) of patients was 3.7000± .2966. In PCOS Group, the mean TSH (mIU/L) of patients was 3.3260± 1.1032. Difference of mean TSH (mIU/L) with both Group was not statistically significant (p=0.1319).

Macut D *et al* [14](2008) found that dyslipidemia characterized by substantial LDL oxidation ($p < 0.001$), increased triglycerides ($p = 0.010$), and reduced HDL cholesterol ($p < 0.01$). In conclusion, only women with PCOS had the distinctive dyslipidemia of insulin resistance and unfavorable proatherogenic lipoprotein ratios; controls did not have these conditions. Women with PCOS may be more susceptible to developing atherosclerosis too soon, according to evidence from elevated OxLDL, the relationship between apoE and nonesterified fatty acids, and insulin resistance.

CONCLUSION

Our study concludes that anthropometric parameters (weight, BMI, waist circumference) and lipid profile have significant correlation with premenopausal patients with PCOS. Dyslipidemia is a significant CVD risk factor that has to be assessed in these people in order to control the condition early and stop more cardiovascular problems.

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