

Manuscript Title - LIPID PROFILE IN POLYCYSTIC OVARIAN DISEASE.

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Title: LIPID PROFILE IN POLYCYSTIC OVARIAN DISEASE.

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ABSTRACT

BACKGROUND: Polycystic ovarian syndrome is the most common cause of infertility in women. Polycystic ovarian syndrome (PCOS) is a syndrome of ovarian dysfunction that is characterized by anovulation, hyperandrogenism. More over the association of PCOS with obesity and insulin resistance increases the risk of developing cardiovascular disease. Hence the study was planned to estimate the levels of lipid profile in PCOS.

OBJECTIVES: Assessment of serum lipid profile in PCOS.

MATERIAL AND METHODS: 30 clinically diagnosed PCOS patients and 30 age and sex matched controls were enrolled in the study.

RESULT AND CONCLUSION: There was a significant rise in serum total cholesterol, LDL-C ($P < 0.001$), triacylglycerol $P < (0.05)$ and a significant fall in HDL-C ($P < 0.05$) levels of PCOS women as compared to controls. The dyslipidaemia associated with women having PCOS may contribute to the risk for developing atherosclerosis.

Hence it can be concluded that women with PCOS should be screened for lipid profile. This can be helpful in assessing the risk of coronary artery disease in these women. The same can be reduced by counseling the PCOS women regarding life style and dietary modifications.

KEY WORDS: Total cholesterol, LDL-C, HDL-C, dyslipidaemia, PCOS.

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INTRODUCTION

Polycystic ovary syndrome is the most common cause of infertility in women. PCOS includes a heterogeneous collection of signs and symptoms with varying degree of mildness and severity in affecting the reproductive, endocrine and metabolic functions.¹ Polycystic ovary syndrome (PCOS) is a syndrome of ovarian dysfunction that is characterized by anovulation and hyperandrogenism.²

Hyperandrogenism in PCOS women may result in a male pattern of lipoprotein distribution. More over the association of PCOS with obesity and insulin resistance

increases the risk of developing prediabetes and diabetes type II, which also contributes to dyslipidaemia as well.³ These patients are more prone to risk for atherosclerosis and cardiovascular diseases. Hence the study was planned to estimate the lipid profile in PCOS.

MATERIAL AND METHODS

The study was carried out on 30 clinically diagnosed PCOS patients, with age group 19 to 25 years, attending the OPD of OB/GYN Department, Bhausahab Sardesai Talegaon Rural Hospital. 30 healthy normal menstruating women were taken as controls.

Patients with history of diabetes mellitus, renal disease, hypertension, liver disease, other endocrine disorders and any disease affecting lipid profile levels were excluded from study.

5ml fasting venous blood was collected under aseptic condition from each subject in a plain bulb. Serum was separated within one hour. Serum Lipid profile was performed in both cases and controls. Total cholesterol levels were estimated by the CHOD-POD method⁴ and serum triacylglycerol by GPO trinder method⁵. HDL cholesterol was analyzed by using direct method⁶, LDL cholesterol was estimated by direct method⁶.

The results were expressed in terms of mean \pm SD. The test of significance used was student “t” test and a p value less than 0.05 was considered statistically significant.

RESULTS

Table 1: Lipid profile of PCOS women and Controls

S.No.	Parameters	Control	PCOS patients
1	Total Cholesterol (mg/dl)	164 \pm 21	230 \pm 31 ^{**}
2	HDL- C (mg/dl)	49.1 \pm 5.8	39.3 \pm 7.7 [*]
3	LDL- C (mg/dl)	98.2 \pm 15	141 \pm 24 ^{**}
4	Triacylglycerol (mg/dl)	116 \pm 22.3	206 \pm 16.8 [*]

*p<0.05 & **P<0.001 indicate significant.

There was a significant rise in serum total cholesterol, LDL-C (P <0.001), triacylglycerol P<(0.05) and a significant fall in HDL-C (P < 0.05) levels of PCOS women as compared to controls.

Discussions

Polycystic ovarian syndrome (PCOS) is a multifactorial and polygenic condition. It is a syndrome of ovarian dysfunction that is characterized by anovulation, hyperandrogenism and /or the presence of polycystic ovary (PCO) morphology.

The present study confirms the presence of a more atherogenic lipid profile in women with PCOS. There was a significant rise in serum total cholesterol, LDL-C, triacylglycerol and a significant fall in HDL-C levels of PCOS women as compared to controls.

The significant increase in the cholesterol indicates the presence of primary alterations in lipid metabolism in patients with PCOS. The significant increase in triacylglycerol may be due to the accumulation of triacylglycerol could as a result of increased lipogenesis, decreased clearance or reduced fatty acid oxidation.⁷

The significant fall of HDL-c may be due to hyperandrogenism.

Hyperandrogenism may affect lipid metabolism by induction of hepatic lipase activity the enzyme which has a role in catabolism of HDL which may lead to decrease in HDL-c.⁸

Another reason for decrease in HDL-C levels may be due to insulin resistance, which is seen in PCOS women. Insulin resistance leads to more catabolism of

HDL particles and formation of LDL particles. Cholesterol ester transfer protein may play role in this.⁹

All the above biochemical alterations along with insulin resistance predispose PCOS subject to metabolic syndrome. This is associated with higher cardiovascular risk at later age.

CONCLUSION

The study concludes that women with PCOS should be screened for lipid profile. This can be helpful in assessing the risk of coronary artery disease in these women. The same can be reduced by counseling the PCOS women regarding life style and dietary modifications.

REFERENCES

1. Berek JS; Novak's Gynaecology. Endocrinedisorders, 14th edition, 2007: 1076-88.
2. Wild RA. Obesity, lipids, cardiovascular risk ,and androgen excess. Am J Med1995;98:(suppl): 27S -32S.
- 3 Yildiz BO, Gedik O. Assessment of glucoseintolerance and insulin sensitivity in polycysticovary syndrome. Reprod Biomed Online2004; 8(6): 649-56.

4. Myers G. L., Kimberly M. et al. A reference method laboratory network for cholesterol : a model for standardisation and improvement of clinical laboratory measurement . *clin chem* 2000; 46 :1762- 72.
- 5 Henry J.B. *Clinical diagnosis and management of laboratory methods*, 18th edition, W. B. Saunders Philadelphia, 204 -11.
6. **W. Greg Miller et al.** Seven Direct Methods for Measuring HDL and LDL Cholesterol Compare with Ultracentrifugation Reference Measurement Procedures. *Clinical Chemistry* 2010, 56: 977-86.
7. Manjunatha S et al. Effect of PCOS on lipid profile .*Sch. J. App.Med.Sci*2014;2(3D):1153-55.
8. Lambrinoudaki I, Christodoulakos G, Rizos D, Economou E, Argeitis J, Vlachou S. Endogenous sex hormones and risk factors for atherosclerosis in health Greek postmenopausal women. *Eur J. Endocrinol*2006; 154(6): 907-16.
9. Barter PJ, Brewer Jr HB, Chapman MJ, Henneckens CH, Rader DJ, Tall AR. Cholesteryl ester transfer protein, a novel target for raising HDL and inhibiting atherosclerosis. *Arterioscler Thromb Vasc Biol.* 2003; 23:160-67.

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