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Research Article

Effect of PCOS on Lipid Profile

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Abstract: Polycystic ovarian syndrome (PCOS) is one of the most common endocrine problem in women of reproductive age, which is multifactorial in etiology. It is associated with menstrual dysfunction and subfertility. PCOS is common cause for hyperandogenism and hirsutism and associated with obesity and with an increased risk of diabetes mellitus and cardiovascular disease. Dyslipidemia is the most common abnormality in PCOS patients. So our study includes lipid profile variation in women with PCOS in younger age group with normal BMI. A case control study was conducted in 50 PCOS patients (19 to 25 years age) and 50 normal menstruating women matched for age and anthropometric data. Statistical analysis was done by using student "t" test. Serum Triglycerides, Serum Cholesterol, LDL-C, VLDL-C, were significantly raised while HDL-C significantly decreased in PCOS patients compared to normal menstruating women. As PCOS patients are more prone for atherogenic lipid profile and cardiovascular diseases, so they should be screened for altered lipid profile to prevent complications.
Keywords: Polycystic ovarian syndrome, Lipid profile, Hyperandrogenism.

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is one of the most common endocrine disorders in women of reproductive age, which is multifactorial in etiology. 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, 2] . 1% to 5% of female population with 15 to 25 years suffers from pcos [3] and 10% of women in reproductive age [4], because of its association with menstrual dysfunction [5] and subfertility [1, 5].

PCOS is common cause for hyperandogenism and hirsutism and associated with obesity, [6] increased risk of impaired glucose tolerance and type 2 diabetes [7]. 50% of PCOS patients have obesity that to android obesity with an increased risk of diabetes mellitus and cardiovascular disease [6].

Women with PCOS have a greater prevalence of atherosclerosis and cardiovascular diseases [8, 9] and estimated seven fold increased risk for myocardial infarction [10].

Dyslipidemia is the most common abnormality in PCOS [11], with elevated total cholesterol, triglycerides, and low-density lipoprotein (LDL), and low levels of high-density lipoproteins (HDL) [6, 12]. Talbott reported increased level of LDL-Cholestrol in PCOS patients [13] and Conway reported, the most characteristic lipid alteration is decreased levels HDL-C [14]. There are few studies done to know the alteration in serum lipid profile in PCOS patients, thus we have undertaken this study to know the lipid profile variation in women with PCOS in younger age group with normal BMI.

MATERIALS AND METHODS

This study was conducted in the Department of OBG, Hassan Medical College, Hassan, with the assistance of laboratory setup of the Department of Biochemistry, Hassan medical college Hassan. The study and its conduct were cleared by the ethical committee, Hassan Medical College, Hassan.

The study was done by obtaining blood samples from study group and controls, attending OPD, in OB/GYN Department, Hassan medical college Hassan, during study period (May 2013 – April 2014).

The study group consisted of 50 PCOS patients, diagnosed by history and ultrasonographic finding, with age group 19 to 25 years, attending the OPD of OB/GYN Department, Hassan Medical

College, Hassan. 50 normal menstruating women were taken as controls.

Previous history of hypertension, Diabetes Mellitus, renal disease, thyroid disorder, dyslipidemia and any disease affecting lipid profile levels were excluded from study. Subjects with family history of dyslipidemia, were excluded from study. After selecting the subjects and controls, informed consent was taken. Height and weight of the individuals were measured. BMI was calculated as per formula: Weight (Kg)/Height (meter^2) (Quetelet,s Index).

The subjects and controls were examined for vital signs, Pulse, Blood pressure. All vital parameters were within normal physiological limit. After selecting the subjects and controls, appointment was scheduled in prior and they were requested to do an overnight fast prior to the day of the test to get fasting blood sample for lipid profile analysis, in order to avoid changes due to circardian rhythm.

Between 7am to 9am, 2ml of venous blood was collected in a plain bulb by venepuncture under aseptic precaution. Sample was analyzed in Biochemistry Lab, with clinical chemistry Analyzer.

An alysis was done for

- Serum Cholesterol
- Serum Triglygerides
- HDL-C

Very Low Density Lipoprotein (VLDL)

It is obtained by the Friedwald formula [15]

VLDL = TG / 5

Low Density Lipoprotein (LDL)

It is obtained by the formula

LDL Cholesterol = Total Cholesterol – (HDL+VLDL)

Statistical Analysis

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

RESULTS

A case –control study of 50 PCOS patients and regular menstruating women was undertaken. The age, BMI, lipid profile of study and control group is represented in table. There is no statistical difference in age and BMI of study and control group. There is high statistical significant difference in serum Triglycerides, serum total Cholesterol, serum LDL-C levels, serum VLDL-C levels and HDL-C levels. Serum Triglycerides, serum total cholesterol, serum LDL-C levels were raised and serum HDL-C levels were decreased in study group.

Table 1: Anthropometric data of PCOS Patients and Controls

Parameter	Study group (mean ± SD)	Control group (mean ± SD)	't' value	'p' value	Significance
Age(years)	19.04±0.57	18.86±0.57	0.121	>0.05	NS
BMI (kg/m2)	21.46±0.76	21.48±0.81	0.124	>0.05	NS

Table 2: Lipid Profile in PCOS Patients and Controls

Parameters	Study group (mean ± SD)	Controls (mean ± SD)	't' value	'p' value	Significance
Serum Triglycerides (mg/ dl)	120.13±12.88	98.3±18.19	6.93	<0.0001	HS
Serum total Cholesterol (mg/ dl)	202.16 ±16.12	170.8±9.87	11.73	<0.0001	HS
LDL- C (mg/ dl)	121.17±12.13	102.13±7.14	9.56	<0.0001	HS
VLDL-C (mg/ dl)	24.15±2.18	19.2±2.1	11.56	<0.0001	HS
HDL-C (mg/dl)	39.16±6.01	55.45±4.11	15.82	<0.0001	HS

DISCUSSION

The study was conducted to evaluate changes for lipid profile parameters in Polycystic ovarian syndrome (PCOS) patients and normal menstruating women. Polycystic ovarian syndrome is one of the important endocrine disorder causing reproductive abnormalities in women, which has heterogeneous clinical features and multifactorial in etiology [2].

Obesity and insulin resistance occur frequently in association with this syndrome. Cardiovascular risk factors seem to cluster in women with PCOS compared with general population [16]. Dyslipedemia is one of the important risk factor associated with PCOS. In our study we have observed, there is alteration in serum lipid profile. There is significant increase in serum triglycerides, serum cholesterol, serum LDL-C, serum VLDL-C and decrease in the levels of serum HDL-C levels.

The increase in triglycerides may be due the accumulation of triglycerides, which may occur due to the increased lipogenesis, decreased clearance or reduced fatty acid oxidation. Increased secretion of VLDL particles by the liver results in elevated plasma triglycerides concentration. This may occur due to insulin resistance, which is seen in PCOS patients. Insulin resistance also contributes more catabolism of

HDL particles and formation of LDL particles [17]. Cholesterol ester transfer protein may contribute for this [17]. In addition to the insulin resistance, hyperandrogenism also contributes for altered lipid profile. Hyperandogenism has been associated with increased hepatic lipase activity which has role in catabolism of HDL particles. Thus PCOS patients have more atherogenic lipid profile, than controls.

Increased level of triglycerides can be contributory factor adiposity in PCOS women [18]. Altered lipid profile (atherogenic), adiposity, insulin resistance may contribute for cardi-vascular diseases. Thus PCOS patients should be screened and monitored regularly, to prevent complications associated with cardiovascular diseases.

CONCLUSION

As PCOS patients are more prone for atherogenic lipid profile and cardiovascular diseases, so they should be screened for altered lipid profile to prevent complications.

REFERENCES

1. Berek JS; Novak's Gynaecology. Endocrine disorders, 14th edition, 2007: 1076-1088.
2. Zawadic JK, Danif A; Diagnostic criteria for polycystic ovary syndrome towards a rational approch. Polycystic ovary syndrome. Cambridge, England: Blackwell Science, 1992: 377-384.
3. Padubidre VG, Daftary SN editors; Disorders of Ovary. In Shaw's Text book of Gynaecology, 13th edition, 2004: 353-354.
4. Franks S; Polycystic ovary syndrome. New England J Medicine, 1995; 333:853-861.
5. Boomsma CM, Fauser BC, Macklon NS ; Pregnancy complications in women with polycystic ovary syndrome. Semin Reprod Med., 2008: 26(1): 72–84.
6. Wild RA; Obesity, lipids, cardiovascular risk, and androgen excess. Am J Med., 1995:98(suppl): 27S-32S.
7. Yildiz BO, Gedik O; Assessment of glucose intolerance and insulin sensitivity in polycystic ovary syndrome. Reprod Biomed Online, 2004; 8(6): 649-656.
8. Guzick DS1, Talbott EO, Sutton-Tyrrell K, Herzog HC, Kuller LH, Wolfson SK Jr.; Carotid atherosclerosis in women with polycystic ovary syndrome: initial results from a case-control study. Am J Obstet Gynecol., 1996; 174(4):1224-1229.
9. Birdsall MA, Farquhar CM, White HD; Association between polycystic ovaries and extent of coronary artery diseases in women having cardiac catheterization. Ann Intern Med., 1997;126: 32-35
10. Dahlgren E1, Janson PO, Johansson S, Lapidus L, Odén A; Ovary syndrome and risk for myocardial infarction. Evaluated from a risk factor model based on a prospective population study. Acta Obstet Gynecol Scand., 1992; 71(8): 559-604.
11. Lergo RS, Kunselman AR, Dunaif A; Prevelence and predictors of dyslipidaemia in women with polycystic ovary syndrome, Am J Med., 2001; 111: 607-613.
12. Dahlgren E1, Johansson S, Lindstedt G, Knutsson F, Odén A, Janson PO *et al*.; Women with polycystic ovary wedge resected in 1956 to 1965; a long - term follow-up focusing on natural history and circulating hormones. Fertil Steril., 1992; 57: 505-513.
13. Talbott E, Clerici A, Berga SL, Kuller L, Guzick D, Detre K *et al*.; Adverse lipid and coronary heart disease risk profiles in young women with polycystic ovary syndrome, results of a case –control study. J Clin Epidemiol., 1998; 51: 415-422.
14. Conway GS1, Agrawal R, Betteridge DJ, Jacobs HS; Risk factors for coronary artery diseases in lean and obese women with polycystic ovary syndrome. Clin Endrocrinol. (Oxf), 1992; 37(2): 119-125.
15. Friedwald WT, Levy RI, Friedreickson DS; Estimation of concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem., 1972; 18(6):499-502.
16. Wild RA; Long term health consequences of PCOS. Hum Reprod Update, 2002; 8(3): 231-241.
17. 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 atheroscelerosis. Arterioscler Thromb Vasc Biol., 2003; 23:160-167.
18. 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): 907-916.