ORIGINAL RESEARCH PAPER

Volume-8 | Issue-4 | April-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

LIPID PROFILE PREDICTOR OF CARDIOVASCULAR RISK FACTOR AMONG POLYCYSTIC OVARY SYNDROME WOMEN

Biochemistry

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ABSTRACT

Objective : To examine whether Polycystic Ovarian syndrome differ from controls regarding Cardiovascular risk factors like lipid profile Triglyceride, Cholesterol, LDL-C , HDL-C, VLDL-C.

Methods: A cross sectional study and has been carried out in our institute during the period of February 2014 - August 2015. All the study subjects were examined & investigated according to predefined Performa. The study protocol was approved by the Ethical Committee of the Institute. Informed written consent was obtained from all the study subjects enrolled in the study.

Results: PCOS women has mean of serum cholesterol, serum triglycerides, serum HDL, serum LDL, serum VLDL level 188.63 ± 34.33, 109.05 ± 23.94 ,40.66 ±7.34 126.15 ± 35.80 21.8± 4.7 as compared to mean of normal women 136.80 ± 24.44, 93.66 ± 18.12, 39.78 ± 8.14, 78.28 ± 23, 18.7 ± 3.6 respectively.

Conclusion: serum levels of Total Cholesterol, LDL-C, VLDL-C, and triglyceride levels, whereas HDL-C levels were highly significantly decreased in PCOS cases as compared to that of controls.

KEYWORDS

Polycystic Ovarian syndrome, lipid profile, Cardiovascular disease

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the common endocrine disorder that affects 6 -10% of woman in reproductive age.[1,2,3] Polycystic ovaries develop when the ovaries are stimulated to produce excessive amounts of male hormones (androgens), in particular testosterone, by the release of excessive luteinizing hormone (LH) by the anterior pituitary gland. or high levels of insulin in the blood (hyperinsulinaemia) in women whose ovaries are sensitive to this stimulus[5].

Dyslipidemia is likely to be the major risk factors for CVD in women with PCOS. To reduce the incidence of cardiovascular disease in polycystic ovary syndrome women lipid profile use as early predictor.

MATERIAL AND METHOD:

A cross sectional study and has been carried out in our institute during the period of February 2014 - August 2015. All the study subjects were examined & investigated according to predefined Performa. The study protocol was approved by the Ethical Committee of the Institute. Informed written consent was obtained from all the study subjects enrolled in the study.

Group A Consists of 60 clinically diagnosed and biochemically confirmed cases of polycystic ovarian syndrome. These were selected from patients attending the outpatient department of Obstetrics and Gynaecology. Group B Consist of 60 age and sex matched normal individuals were studied as controls were enrolled in the present study All the calculations were done by using Microsoft Office Excel 2007 and statistical analysis was done using the SPSS software, Version 11.5. All statistical data was analysed by, Levene's test for Equality of Variances, t-test for Equality of Means. Mann-Whitney test. P-value less than 0.05 ($P < 0.05$) was considered to be statistically significant (S). P value of less than 0.001 ($P < 0.001$) was considered to be statistically highly significant (HS). P-value more than 0.05 ($P > 0.05$) was considered to be statistically non-significant (NS).

DISCUSSION

Insulin is recognized as one of the primary hormonal regulators of lipid metabolism. It acts on various stages of lipid metabolism. For instance, during the post-prandial state, insulin promotes the uptake of TG into adipose tissue by up regulating the activity of adipose tissue lipoprotein lipase (LPL) [6]

It has been shown that women with central obesity, the type most commonly seen with PCOS, lead to insulin resistance and rise in insulin circulating level defect in insulin action enhance the steroidoeenic effects on theca cell and suppress sex hormone – binding globulin production by hepatocytes, leading to high free androgen levels and exhibit significantly higher levels of insulin insensitivity compared to weight matched controls.[6,7]

In addition to overstimulation of LH by GnRH, the ovarian dysfunction hypothesis proposes that there is a primary defect in the ovarian thecal cell layer that results in exaggerated ovarian androgen secretion), because ovarian theca cells are very sensitive to LH stimulation. In PCOS, the exaggerated response of LH leads to increased levels of 17a-hydroxyprogesterone. Generally, ovarian androgens are converted into estrogens, but in PCOS disproportionate response of 17a-hydroxyprogesterone by gonadotrophins stimulation increases androgen production.[8]

However, it is well known that obesity generates a decrease in the sexual hormone-binding globulin (SHBG), and therefore an increase in the free androgens. Obesity generates an increase of testosterone levels in PCOS patients.[9]

Relatively low FSH and disproportionately high LH concentrations are common in PCOS. Serum a drostenedione and testosterone concentrations (total and free concentrations) are elevated, with mean concentrations 50 to 150% higher than normal. Testosterone is known to facilitate lipolysis, providing increased FFA concentrations.

It is well recognized that the liver is the primary site of lipogenesis in the body and one of the main sources of lipids for adipocytes alongside dietary lipids. Enhanced hepatic delivery of FFA results in increased generation of VLDL .This situation is further exacerbated by excess synthesis of apolipoprotein B-100 in the liver, which stimulates the transfer of TG in exchange for cholesterol esters resulting in smaller and less dense HDL particles being produced these HDL particles are rapidly metabolized leading to a drop in circulating HDL levels together these disturbances in lipid metabolism lead to the development of dyslipidemia characterized by elevated VLDL, LDL, and reduced HDL.

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.[10]

Testosterone Testosterone Drostenedione, dihydrotestosterone (DHT), dehydroepiandrosterone (DHEA) and DHEA-sulfate (DHEA-S) are the androgens found in males and females. In women, androgens are precursors for estrogen and are produced in the ovaries, adrenal glands, and adipose cells. Once secreted, T is almost entirely bound to transport proteins: a) weakly bound to albumin, b) tightly bound to sex

hormone-binding globulin (SHBG), and c) free or unbound. SHBG is the principal binding protein for T.[11]

Low levels of SHBG are associated with increased concentrations of free testosterone, resulting in a more androgenic profile results in women.[12]

Elevated plasma triglycerides concentration results in increased secretion of VLDL particles by the liver. 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.[10] Cholesterol ester transfer protein may contribute to this. In addition to the insulin resistance, hyperandrogenism also contributes for an altered lipid profile. Hyperandogenism has been associated with increased hepatic lipase activity which has a role in the catabolism of HDL particles.

Thus, in the present study, highly significant alterations in triglyceride, VLDL-cholesterol, LDL-cholesterol and HDL cholesterol, in PCOS patients were observed. Women with PCOS are at higher risk of developing diabetic mellitus and cardiovascular disease.

Comparison of lipid profile parameters in study groups.

Parameter	Group A (n=60) (mean ± SD)	Group B (n=60) (mean ± SD)	P value
Total cholesterol (mg/dl)	188.63 ± 34.33	136.80 ± 24.44	<0.001 (HS)
LDL- cholesterol (mg/dl)	126.15 ± 35.80	78.28 ± 23	<0.001 (HS)
HDL-cholesterol (mg/dl)	40.66 ±7.34	39.78 ± 8.14	<0.05 (S)
Triglycerides (mg/dl)	109.05 ± 23.94	93.66 ± 18.12	<0.001 (HS)
VLDL-cholesterol (mg/dl)	21.8± 4.7	18.7 ± 3.6	<0.001 (HS)

S (P < 0.05) = significant, HS (P < 0.001) = highly significant

In this study, there was highly significant increase in the serum levels of TC, LDL-C, TG, VLDL-C (<0.001) and significant decrease in the serum levels of HDL-C (<0.05) in group A as compared to that of group B.

Group A has mean of serum cholesterol, serum triglycerides, serum HDL, serum LDL, serum VLDL level 188.63 ± 34.33, 109.05 ± 23.94 ,40.66 ±7.34 126.15 ± 35.80 21.8± 4.7 as compared to mean of group B 136.80 ± 24.44, 93.66 ± 18.12, 39.78 ± 8.14, 78.28 ± 23, 18.7 ± 3.6 respectively.

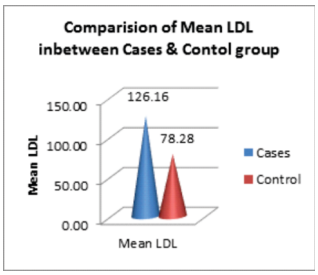
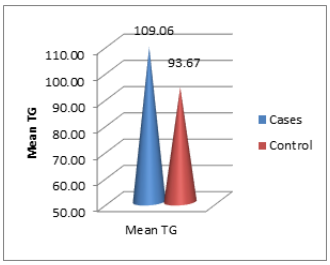
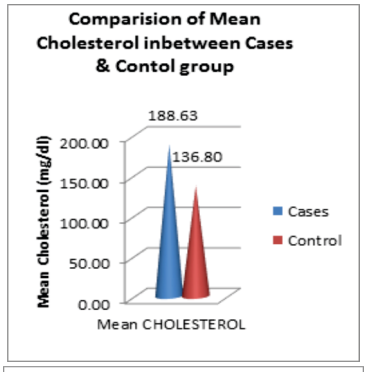
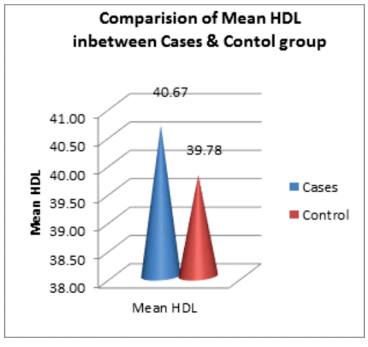


Chart Showing Comparison of Mean LDL & VLDL in between distribution in study groups.



Showing Comparison of Mean Triglycerides in between distribution in study groups.





CONCLUSION

Elevated serum levels of Total-Cholesterol, triglyceride, LDL-C, VLDL-C, as well as depleted levels of protective cholesterol HDL-C indicate increased cardiovascular risk in PCOS patients.

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