Original Article

Lipid Profile in Polycystic Ovarian Syndrome

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ABSTRACT

Objective: To compare serum lipid profile of the patients with polycystic ovarian syndrome (PCOS) with normal healthy controls.

Methodology: It was a cross-sectional study conducted in the Department of Pathology, Sharif Medical City Hospital, Lahore. The study included 120 subjects between the age of 18- 40 years. They were divided into two groups. Group 1 included 60 patients of PCOS and group II consisted of 60 healthy females of same BMI and age group. Newly diagnosed patients of PCOS based on Rotterdam criteria were included in this study by random sampling technique. Lipid profile was performed on all subjects.

Results: Mean age of the study patients included in group I was 33±7 years and in control group II mean age was 31± 9 years. A statistically significant difference was found when serum lipid profile of both groups was compared. Serum total cholesterol, triglyceride, LDL and VLDL were significantly raised in patients with PCOS while serum HDL was low in the patient group as compared to controls

Conclusion: Total cholesterol, triglycerides, LDL and VLDL levels were high and HDL level was low in patients with PCOS. So, it was concluded that dyslipidemia is an accompanying feature in patients with PCOS.

Keywords: *Polycystic ovarian disease. Dyslipidemia. Insulin resistance. Lipid profile.*

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common causes of endocrine dysfunction in premenopausal women. Its prevalence ranges from 4% to 7% worldwide. Polycystic ovarian syndrome (PCOS) is a reproductive endocrinopathy with multisystem involvement and is characterized by increased adrenal and ovarian androgen secretion and metabolic disturbances. In this disorder patients present with obesity, chronic anovulation, androgen excess and insulin resistance.[1]

It is the most common cause of infertility in females. According to Rotterdam (2003), polycystic ovarian syndrome is defined as having any two of following conditions: 1) Oligo/anovulation, 2) Clinical/ biochemical signs of hyperandrogenism, 3) Polycystic ovaries by scan with the exclusion of other related disorder. The pathophysiology of PCOS involves the hypothalamus-pituitary-ovarian axis, ovarian theca cell hyperplasia, hyperinsulinemia and release of adipocyte-driven factors and other cytokines.[2]

In patients with PCOS binding of insulin to its receptors is inefficient that leads to hyperglycemia and increased insulin secretion from beta cells.[9] Insulin resistance occurs in about 50-80% of patients with PCOS.[4]

Polycystic ovary syndrome has multifactorial etiology with the involvement of genetic, environmental, endocrine and metabolic factors. Patients present with complaints of menstrual irregularity, infertility, hirsutism, acne and various metabolic disorders. Obesity, insulin resistance, dyslipidemia and hypertension are the common metabolic derangements. Women with PCOS can develop cardiometabolic syndrome at an early age.[7] A cardiometabolic syndrome is a group of interlinked risk factors that promote the development of atherosclerosis, coronary artery disease and type 2 diabetes mellitus.[8]

There is a strong influence of obesity on the prevalence of metabolic abnormalities in patients with PCOS. It is associated with a change in lipid and lipoprotein metabolism.[1]

Dyslipidemia is a common metabolic abnormality that occurs in females with polycystic ovary syndrome. Insulin resistance plays an important role in the pathophysiology of dyslipidemia in women with PCOS by stimulation of lipolysis and alteration in expression of hepatic and lipoprotein lipases. Hyperandrogenism in these females leads to increased hepatic lipase. It may also contribute to dyslipidemia and adipocyte dysfunction.[3]

Obesity and insulin resistance can result in increase in long-chain free fatty acids in the portal vein. This causes increased secretion of apolipoprotein B100 by the liver. The long-chain fatty acids affect degradation of apoB in the endoplasmic reticulum. This leads to increased VLDL levels in these patients as compared to the normal and eventually results in hypertriglyceridmia.[4]

In these patients levels of high-density lipoprotein-cholesterol (HDL-C) and apolipoprotein (Apo) A-I are decreased. There is increased secretion of VLDL particle by the liver that results in increased levels of triglycerides (TG), ApoB and very low-density

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lipoprotein.[3-6]
The present study was undertaken to document the lipid abnormalities so that the patients at the risk of cardiometabolic syndrome will be identified.

METHODOLOGY

It was a cross-sectional study conducted in the Department of Pathology, Sharif Medical City Hospital, Lahore. The study included 120 subjects which were divided into two groups. Group 1 included 60 patients of PCOS and group II consisted of 60 healthy females of same BMI and age group. Newly diagnosed patients of PCOS based on Rotterdam criteria were included in this study by random sampling technique. The institutional ethical committee approved the study and informed consent was obtained from all the study subjects.
A proforma which contained details of age, menstrual history, medical history, family history of type 2 diabetes mellitus and polycystic ovarian syndrome was filled by all subjects. Using aseptic techniques, 5 ml of venous blood sample was collected from all study subjects after 12 hours overnight fast. Lipid profile was performed on all samples and results of both groups were compared. Total cholesterol, triglycerides and high density lipoprotein cholesterol were performed on selectra. Total cholesterol was performed by cholesterol oxidase method, triglycerides by glycerol phosphate oxidase and peroxidase method and HDL-C was done by phosphotungstic acid method. LDL-C and VLDL-C were calculated using the Friedewald's formula.

STATISTICAL ANALYSIS

The data was analyzed by using SPSS version 21. Mean & standard deviation was calculated for quantitative variables. Two groups were compared by using t-test.

RESULTS

Mean age of the study patients included in group I was 33±7 years and in control group II mean age was 31±9 years. Mean levels of triglycerides, total cholesterol, HDL and LDL in group I and II were tabulated in table 1. A statistically significant difference was found when serum lipid profile of both groups was compared. Serum total cholesterol, triglyceride, LDL, VLDL were significantly raised in patients with PCOS while serum HDL was low in the patient group as compared to controls (Figure 1).

Table 1: Comparison of serum lipid profile of study subjects

	Cholesterol (mg/dL)	Triglyceride (mg/dL)	HDL (mg/dL)	LDL (mg/dL)	VLDL (mg/dL)
Group I	225.3±15.1	219.6±18.9	28.7±3.08	139.3±4.4	30.5±5.1
Group II	144.4±12.5	126.5±17.2	57.7±72.0	81.9±9.7	22.3±2.1

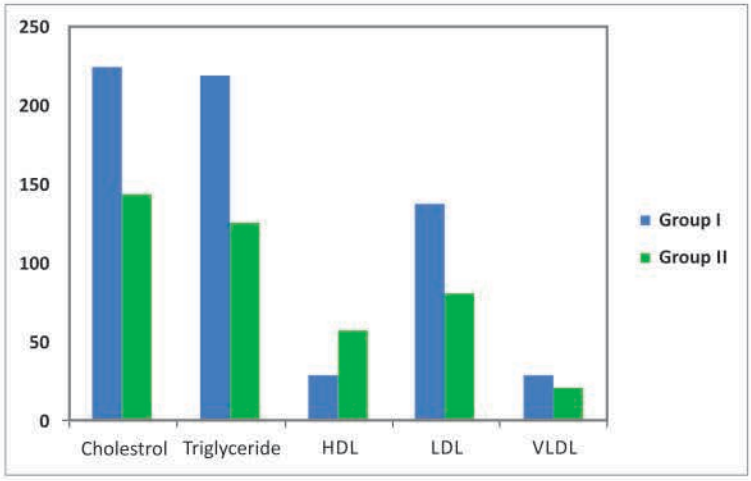

Figure 1: Bar chart showing lipid profile of study subjects

DISCUSSION

Polycystic ovary syndrome is the most frequently occurring endocrine disorder seen in women of reproductive age. It is characterized by menstrual irregularities, insulin resistance and hyper-androgenism.[6]

Dyslipidemia is one of the most perplexing metabolic disturbances that occur in patients with PCOS. Insulin resistance, obesity and hyperandrogenism play a significant pathophysiological role in the development of dyslipidemia in this syndrome.[4]

In this study, we observed a statistically significant difference in the mean levels of total cholesterol, triglyceride, LDL-C and VLDL-C between group I and group II. The mean values of total cholesterol, triglyceride, LDL-C and VLDL-C were higher in patients than controls whereas mean HDL-C values were lower in group I patients as compared to controls. Similar results were found in another study which indicated that levels of triglycerides, cholesterol and LDL-C were increased and HDL-C and apoA-I levels were decreased.[11] A study conducted in Iran showed no significant difference in lipid profile of women with PCOS and healthy controls.[20] This is contrary to our results.

Insulin resistance impairs the ability of insulin to suppress lipolysis. It increases the mobilization of free fatty acids from adipose stores resulting in increased hepatic delivery of free fatty acids. Inhibition of hepatic very low-density lipoprotein synthesis by the insulin is impaired in these patients. All these factors lead to altered catabolism of very low-density lipoprotein.[4]

A study conducted by Cheung et al. compared serum lipid profile in patients with PCOS and controls. They observed that high levels of triglyceride and low HDL were the most common characteristic findings seen in patients with PCOS. These findings were present even in patients with low BMI.[17,18]

Our results are in accordance with the above observations. Elevated triglycerides levels may occur due to resistance to the action of insulin on lipoprotein lipase present in peripheral tissues. The subsequent exchange of triglycerides for cholesteryl ester (CE) by the activity of CE transfer protein (CETP) results in triglycerides enriched high-density lipoprotein (HDL) particles that are catabolized more rapidly and CE-enriched VLDL particles that are converted into small dense low-density lipoprotein (LDL) particles. Insulin resistance may also be responsible for the decreased levels of HDL cholesterol. In spite of increased synthesis of HDL cholesterol, the plasma level of HDL is low due to an increased rate of degradation of apolipoprotein A1/HDL cholesterol. These metabolic derangements might predispose women with PCOS to earlier onset of cardiovascular disease.[4]

Hyperinsulinemia can result from a decrease in insulin clearance as well as from increased insulin secretion. Therefore, in PCOS hyperinsulinemia is probably the result of increased basal insulin secretion and decreased hepatic insulin clearance.[5]

A number of studies showed that the women with PCOS had dyslipidemia in the form of low HDL and high triglycerides.[18,19]

In a study conducted by Sadananjali et al. obese and lean patients with PCOS were compared with control subjects. They also observed decreased levels of HDL cholesterol and high levels of apolipoprotein B, triglycerides and VLDL-C in patients with PCOS. They found no association between lipid profile and BMI.[1]

A study conducted in India showed that in women with PCOS the lipid profile was significantly different as compared to control group. The difference between the mean values of triglycerides ($P<0.001$), total cholesterol ($P = 0.002$) and HDL ($P < 0.001$) was statistically significant two groups but the level of low-density lipoprotein (LDL) ($P <or=0.07$) was not statistically significant.[12]

A study conducted on Korean women with PCOS observed an increased prevalence of dyslipidemia in these patients. Dyslipidemia in PCOS is consistent with the findings that occur in the insulin resistant state i.e decreased levels of ApoA-I and HDL-C and increased levels of triglycerides. Women with PCOS have both qualitative and quantitative changes in LDL-C. Not only LDL-C level is increased in these women but the proportion of atherogenic small dense LDL is also raised.[2]

The present study showed significant atherogenic lipid profile in women with PCOS. This leads to increased risk of cardiovascular disease in these patients. Therefore, increasing awareness of the risk factors, screening of high risk population and early interventions in the form of lifestyle modification and medication will help in prevention of complications of this disease.

CONCLUSION

Total cholesterol, triglycerides, LDL and VLDL levels were high and HDL level was low in patients with PCOS. So, it was concluded that dyslipidemia occurs in patients with PCOS.

It is recommended that lipid profile should be performed regularly in women with PCOS. There is a limitation to this study that in this study effect of BMI on the lipid profile of the patients was not noted.

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