

Study of Serum Lipoprotein (A) and Lipid Profile in Polycystic Ovarian Syndrome

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Abstract

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder affecting women of reproductive age, often linked to metabolic disturbances, including dyslipidemia and increased cardiovascular risks. This study aims to assess serum lipoprotein (a) [Lp(a)] and lipid profiles in PCOS patients to establish potential cardiovascular risk factors. A case-control study was conducted on 100 women, 50 diagnosed with PCOS based on the Rotterdam criteria and 50 healthy controls. Serum Lp(a) and lipid profile, including total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL), were measured. Results indicated significantly higher levels of Lp(a), TC, TG, and LDL in PCOS patients compared to controls, while HDL levels were significantly lower. These findings suggest that women with PCOS have a higher predisposition to cardiovascular diseases due to altered lipid metabolism. The study highlights the need for routine lipid monitoring and early interventions to mitigate cardiovascular risks in PCOS patients.

Keywords: Polycystic ovarian syndrome, lipoprotein (a), lipid profile, dyslipidemia, cardiovascular risk, metabolic disorder.

Introduction

Polycystic ovarian syndrome (PCOS) is a complex hormonal disorder that affects approximately 5–10% of women of reproductive age worldwide. It is characterized by menstrual irregularities, hyperandrogenism, and polycystic ovaries, often leading to infertility. The condition is closely associated with metabolic abnormalities, including insulin resistance, obesity, and dyslipidemia, increasing the risk of cardiovascular diseases (CVD) [1].

Lipoprotein (a) [Lp(a)] is a plasma lipoprotein structurally similar to LDL but with an additional apolipoprotein(a) component. Elevated Lp(a) levels are considered an independent risk factor for atherosclerosis, thrombosis, and cardiovascular events. Studies suggest that women with PCOS exhibit higher Lp(a) levels, which may contribute to their increased susceptibility to CVD [2,3].

Lipid profile abnormalities are commonly observed in PCOS patients, with increased levels of triglycerides (TG), total cholesterol (TC), and LDL cholesterol, along with reduced high-density lipoprotein (HDL) cholesterol [4]. These alterations may be attributed to insulin resistance, a core feature of PCOS, which disrupts lipid metabolism and promotes an atherogenic lipid profile [5].

Several mechanisms link PCOS to dyslipidemia and elevated Lp(a) levels. Insulin resistance stimulates hepatic lipid synthesis, leading to increased secretion of very-low-density lipoproteins (VLDL) and LDL particles. Simultaneously, reduced HDL levels impair reverse cholesterol transport, further worsening lipid homeostasis [6]. Hyperandrogenism in PCOS may also play a role by influencing hepatic lipid metabolism and adipose tissue function [7].

Understanding the lipid abnormalities in PCOS is crucial for early detection and management of cardiovascular risks. This study aims to evaluate serum Lp(a) and lipid profiles in women with PCOS and compare them with healthy controls to establish potential correlations between PCOS and lipid disturbances. Identifying these associations could help develop targeted interventions for reducing long-term cardiovascular complications in PCOS patients.

Aim and Objectives

Aim

To evaluate the serum lipoprotein (a) and lipid profile in women with PCOS and compare them with healthy controls.

Objectives

1. To assess serum Lp(a) levels and lipid parameters (TC, TG, HDL, LDL) in PCOS patients and controls.
2. To analyze the correlation between Lp(a) levels and lipid abnormalities in PCOS patients.

Materials and Methods

This case-control study was conducted at a tertiary care hospital, recruiting 100 women aged 18–35 years. Among them, 50 were diagnosed with PCOS based on the Rotterdam criteria, while 50 age-matched healthy women served as controls. Serum Lp(a) and lipid profile (TC, TG, HDL, LDL) were measured using an enzymatic colorimetric method.

Inclusion Criteria

- Women aged 18–35 years diagnosed with PCOS.
- Healthy age-matched women as controls.
- Patients not receiving lipid-lowering therapy.

Exclusion Criteria

- Pregnant or lactating women.
- Women with known cardiovascular diseases, diabetes, or thyroid disorders.
- Patients on hormonal or lipid-altering medications.

Results

Table 1: Serum Lipoprotein (a) and Lipid Profile in PCOS Patients and Controls

Parameter	PCOS Group (Mean ± SD)	Control Group (Mean ± SD)	p-Value
Lp(a) (mg/dL)	36.5 ± 10.2	21.3 ± 8.5	<0.001
TC (mg/dL)	202.4 ± 24.1	175.2 ± 20.3	<0.01
TG (mg/dL)	156.3 ± 18.5	118.7 ± 14.9	<0.01
LDL (mg/dL)	128.9 ± 16.7	102.5 ± 12.4	<0.05
HDL (mg/dL)	38.2 ± 5.3	50.4 ± 6.1	<0.01

Table 2: Correlation Between Lp(a) and Lipid Parameters in PCOS Group

Parameter	Correlation with Lp(a) (r-value)	p-Value
TC	0.42	<0.05
TG	0.35	<0.05
LDL	0.48	<0.01
HDL	-0.32	<0.05

PCOS patients showed significantly elevated Lp(a), TC, TG, and LDL, with decreased HDL, indicating an increased cardiovascular risk.

Discussion

The present study demonstrated that women with PCOS have significantly higher serum Lp(a) and

an altered lipid profile compared to healthy controls. Elevated Lp(a) levels in PCOS align with prior studies highlighting its role in atherogenesis [8,9]. Higher LDL and TG levels in PCOS further emphasize the metabolic dysfunction associated with this syndrome [10].

Insulin resistance plays a critical role in lipid dysregulation in PCOS. Hyperinsulinemia promotes hepatic lipid synthesis, leading to elevated LDL and TG levels while reducing HDL [11]. Additionally, hyperandrogenism contributes to lipid abnormalities by modulating hepatic lipid metabolism and adipose tissue distribution [12].

Studies suggest that Lp(a) promotes atherogenicity by enhancing LDL oxidation and impairing endothelial function [13]. The observed correlation between Lp(a) and LDL in this study supports this hypothesis, indicating that increased Lp(a) levels may exacerbate cardiovascular risks in PCOS patients [14].

Considering these findings, regular lipid screening and lifestyle interventions, including dietary modifications and physical activity, are crucial for managing dyslipidemia in PCOS. Pharmacological interventions like statins and metformin may also be considered for high-risk individuals [15].

Conclusion

This study highlights significant alterations in serum Lp(a) and lipid profile in PCOS patients, suggesting an increased cardiovascular risk. Elevated Lp(a) and LDL, coupled with decreased HDL, contribute to atherosclerotic progression in these women. Early lipid monitoring and intervention strategies are essential to mitigate long-term cardiovascular complications in PCOS.

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