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A STUDY TO ASSESS THE LEVEL OF LIPID PROFILE AND SERUM LIPOPROTEIN (A) IN POLYCYSTIC OVARIAN SYNDROME WHILE COMPARING CLINICAL OUTCOMES OF CASES AND CONTROLS

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Abstract:

Objective: *The research objective was to assess the level of lipid profile and serum lipoprotein (a) in polycystic ovarian syndrome while dividing the research sample into cases and controls.*

Methodology: *The study started from May 2017 and completed in December 2017 at the Department of Obstetrics and Gynecology of Services Hospital, Lahore. This study included at its best PCOS of 30 cases along with 30 controls. A comparison was made of Lp(a) and Lipid profile among all these cases along with controls and their findings were recorded on an already determined proforma.*

Result: *TG, TC/HDL, LDL, LDL/HDL, VLDL and Serum TC quantities were elevated in all cases. As opposed to the controls, the cases of Lp(a) levels greater than 30 mg/dl were present more in the cases.*

Conclusion: *Elevated Lp(a) and Dyslipidemia, for the patients of PCOS, may become a greater danger of becoming coronary artery disease.*

Keywords: *Lp(a), Lipid Profile and Polycystic Ovary Syndrome.*

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INTRODUCTION:

The polycystic ovarian syndrome is a polygenic and a multifactorial state. This syndrome consists of dysfunction of ovary particularized by hyperandrogenism, anovulation and morphology of PCO i.e., polycystic ovary. In the reproductive age, ranging from six to seven percent women are affected by PCOS i.e., polycystic ovarian syndrome. It is the very common endocrinopathies of the female. Levinthal and Stein in 1935, experimented on the relationship of bilateral polycystic ovaries and amenorrhea, so it was known as Stein-Levinthal syndrome for decades. Earlier, its diagnosis was based on the trio of obesity, hirsutism and amenorrhea. With the passage of time, it was proved that PCOS has multifactorial and etiologically heterogeneous properties clinically [1].

Mostly, PCOS shows the huge neglected population of a female who may become victims of cardiac syndrome. Physical and pathological outcomes of the pituitary of ovarian and hypothalamus axis, hyperplasia of ovarian theca cell, factors driven by adipocyte, many cytokines and hyperinsulinemia [2]. The studies in the previous years have proved that PCOS is responsible for morbidity of the reproductive system and an increase in the risk of diabetic mellitus, endometrial and ovarian cancer, in the near past most of the researches proved that that female having PCOS are prone to cardiac diseases [3 – 5]. The variation of lipoprotein (a) and plasma lipid configuration shows a greater risk for the patient to be a victim of cardiac disease. Metabolic syndrome and female of PCOS have many characteristics in common.

The PCOS causes coronary artery disease type II diabetes mellitus [1, 2]. The CVD in a female having PCOS may be caused by hyperandrogenism, dyslipidemia and insulin resistance [6, 7].

This greater risk of the cardiac diseases is not yet ascertained and even hyperandrogenism is not acknowledged as a risk for cardiac diseases as the research up till now on the pre / post-menopausal female does not show any clear influence of hyperandrogenism on cardiac diseases. dyslipidemia and insulin resistance contribute to the cardiac diseases of a female with PCOS but to which degree that is still unknown [4].

Mostly, Generally, low HDL cholesterol and increased triglycerides are characteristics of the PCOS but some researches prove that dyslipidemia of PCOS is characterized by low HDL-cholesterol but without hypertriglyceridemia to [5]. The value of LDL in patients with PCOS has a direct effect on cardiac diseases [5].

The arterial tissues conveniently take little-condensed LDL due to less antioxidant concentrations and more oxidative susceptibility [3]. The increased little-condensed LDL may increase the risk of cardiac disease up to 3 times and the National Cholesterol Education Programme Adult Treatment Panel III has declared it as an evolving cardiac diseases factor. ALP, atherogenic lipid profile, less HDL-cholesterol and a greater quantity of little LDL having hypertriglyceridemia lead to the danger more cardiac diseases [5]. Fatty acids are freely circulated due to hyperandrogenemia and hyperinsulinemia. The increase of these acids stimulate the liver to secrete very low-density lipoprotein (VLDL) and it causes hypertriglyceridemia. Insulin resistance surrounds PCOS it overproduces VLDL and apoB which leads to hypertriglyceridemia. The near past researches show that variations of Lp, apoB and plasma lipids contribute to greater risk of cardiac diseases [3].

A mixed group of lipoproteins, a molecule of apo(a) attached to apoB-100 along with a lipid, is called lipoprotein(a). The concentration of Lp(a) of the person remains constant during his life. The levels of Lp(a) are genetically specified and it has a different metabolism as compared to LDL. Higher levels of Lp(a) lead to cardiac diseases, myocardial infarction and stroke [6]. The recent researches show that high Lp(a) concentrations predict cardiovascular diseases. Some mechanisms have also been suggested for the part played by Lp(a) in IHD. Its incorporation into atherosclerotic plaque and high-affinity binding to glycosaminoglycan and fibronectin suggest a direct atherogenic action in combination with elevated cholesterol. LDL elements are more prone to oxidation than Lp(a), that is why Lp(a) particles are engaged by scavenger receptors, the formation of plaque, foam cells and proliferation of smooth muscle cell. Moreover, Lp(a), by contending plasminogen, plasminogen (tissue- type) activator or to have direct fibrin binding may damage fibrinolytic action. This linkage of cardiac diseases with Lp(a) is free of the factor of insulin resistance [4]. In addition, the independent causes leading to apoA-I in PCOS affected women are estradiol (E2), body mass index, sex hormone binding globulin and free androgen index, determined through multivariate and univariate analysis. It proves that ovarian sex steroids are responsible for the pathogenesis of dyslipidemia of female having PCOS even though it is 17% of the total variance [3]. The growth of cardiac diseases may be prevented due to in time analysis of the risk causes. Now we have completed our analysis of ascertaining the presence of dyslipidemia and an elevated Lp(a) level in PCOS.

MATERIAL AND METHODS:

The study started from May 2017 and completed in December 2017 at the Department of Obstetrics and Gynecology of Services Hospital, Lahore. The research protocol was sanctioned by the ethical committee of the institution. A learned consensus of the patients was also acquired. They all were from 18-35 years of age range and were without any drug history. As controls, of the same age group, 30 healthy women were chosen.

All those patients who were taking regular medications, drugs or having any renal infection, a disorder of endocrine or liver failure etc., were not included in the research. Hormonal or non-hormonal patients were also not included in the study. A learned consensus was also obtained from the controls.

A sample of blood of 5 ml blood after 12 hours fasting of all night was obtained from the cases and the controls. Then centrifugation and processing of the blood started. Assessments of blood urea, fasting blood glucose, serum creatinine, serum triglycerides and serum total cholesterol, serum Lipoprotein (a) and HDL cholesterol were done of the serum. was counted the following formula was used for counting the VLDL Cholesterol of the serum: VLDL=S.TG/5. From total cholesterol, the value of LDL cholesterol was obtained and from Fried Ewald's equation, the value of HDL cholesterol and triglycerides were obtained. For TG values exceeding 400mg/dl, LDL-C was assessed directly. The ratios of LDL/HDL and TC/HDL were calculated. Fasting urine was assessed for albumin and sugar. SPSS was used to analyze the data. SD and Mean were determined through numerical data while through categorical data the frequencies were determined.

RESULTS:

This research is conducted to judge the importance of lipoprotein(a)levels and serum lipid for polycystic ovary syndrome. For the purpose of this research cases of 30 PCOS and 30 healthy individuals of the same age were taken as controls. Statistically, the mean age amid the cases and controls is not important ($P>0.05$). Statistically, mean glucose amid the cases and the controls are not important ($P>0.05$). Statistically, mean urea amid the cases and the controls are not important ($P>0.05$). Statistically, mean creatinine amid the cases and the controls are not important ($P>0.05$). The mean of total cholesterol is high in the cases and low in controls, but statistically the difference of mean is not important ($P>0.05$). The mean of triglyceride is high in the cases as compared to controls but statistically, the difference of mean is not important ($P>0.05$). The mean of LDL is high in the cases as compared to the controls but statistically, the difference of mean is not important ($P>0.05$). The mean of HDL is low in the cases as compared to controls but statistically, the difference of mean is not important ($P>0.05$). The mean of Lp(a) is high in the cases as compared to the controls but statistically, the difference of mean is not important ($P>0.05$). Lp(a) levels > 30mg/dl is was present more in the cases as compared to controls. Statistically, this is important by $P < 0.05$.

Table – I: Demographic and Clinical Features

Group		Mean	±SD	P-Value
Age	Controls	26.43	4.57	0.92
	Cases	24.53	4.12	
Average Glucose	Controls	91.33	11.22	0.559
	Cases	92.5	11.66	
Mean Urea	Controls	18.23	2.6	0.000
	Cases	18.77	1.74	
Mean Creatinine	Controls	0.91	0.14	0.964
	Cases	0.88	0.16	
Mean TG	Controls	120.2	36.49	0.336
	Cases	146.03	75.07	
Mean LDL	Controls	91.13	26.5	0.218

	Cases	98.63	19.7	
Mean HDL	Controls	39.92	10.7	0.383
	Cases	37.62	9.58	

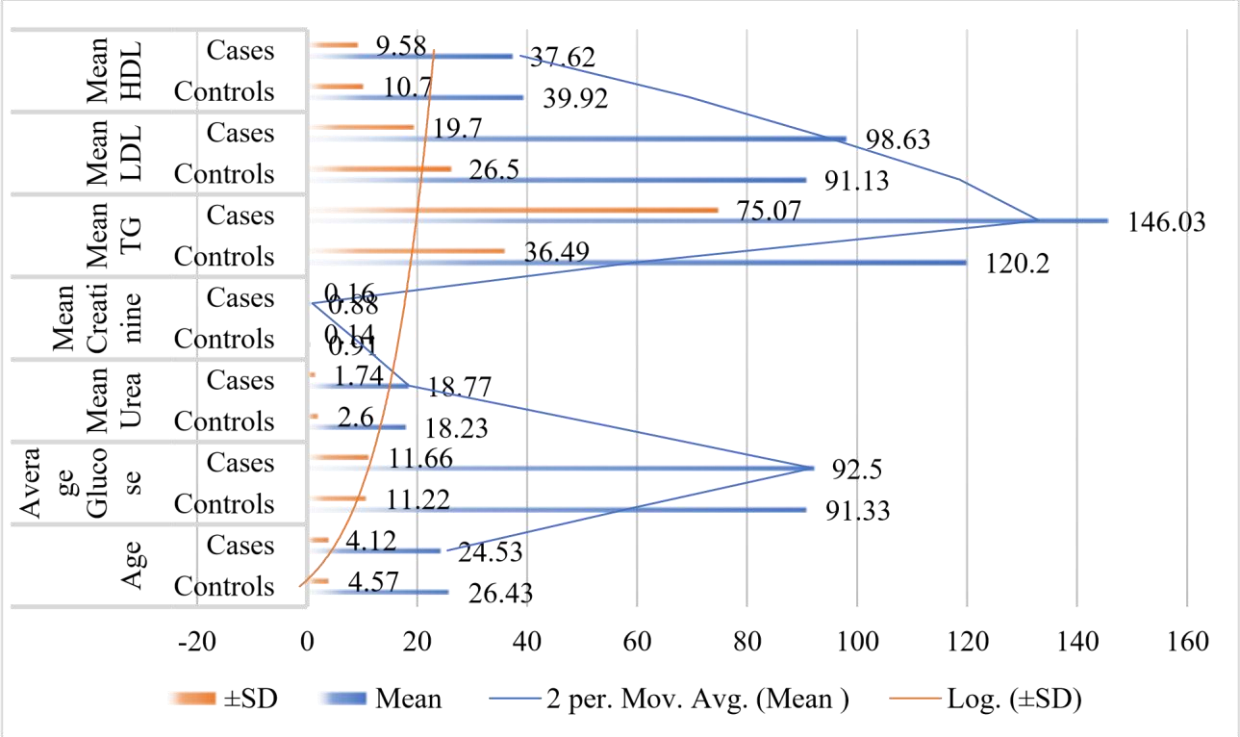
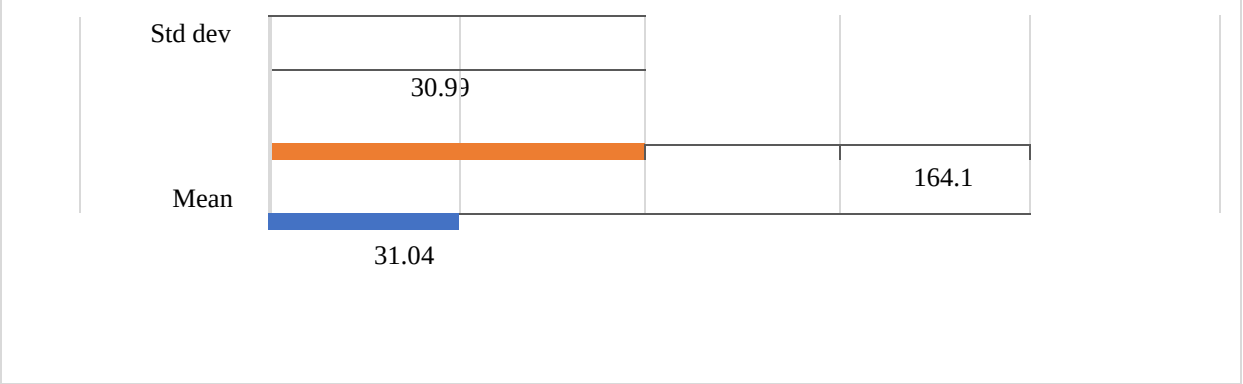


Table – II: Comparison of Controls and Cases

Group	Mean	Std dev	SE of Mean	Mean difference	t	P-Value
Controls	155.2	30.99	5.66	-8.897	-1.11	0.271
Cases	164.1	31.04	5.67			



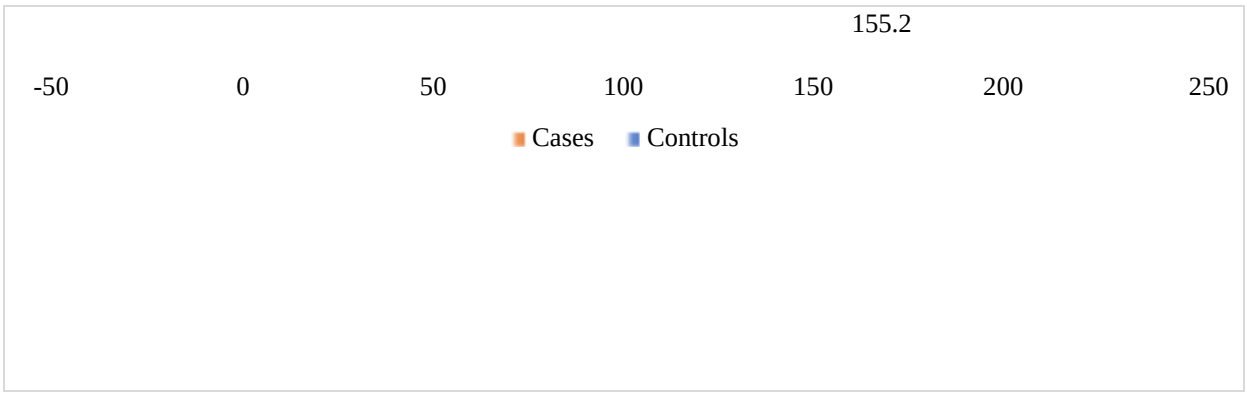
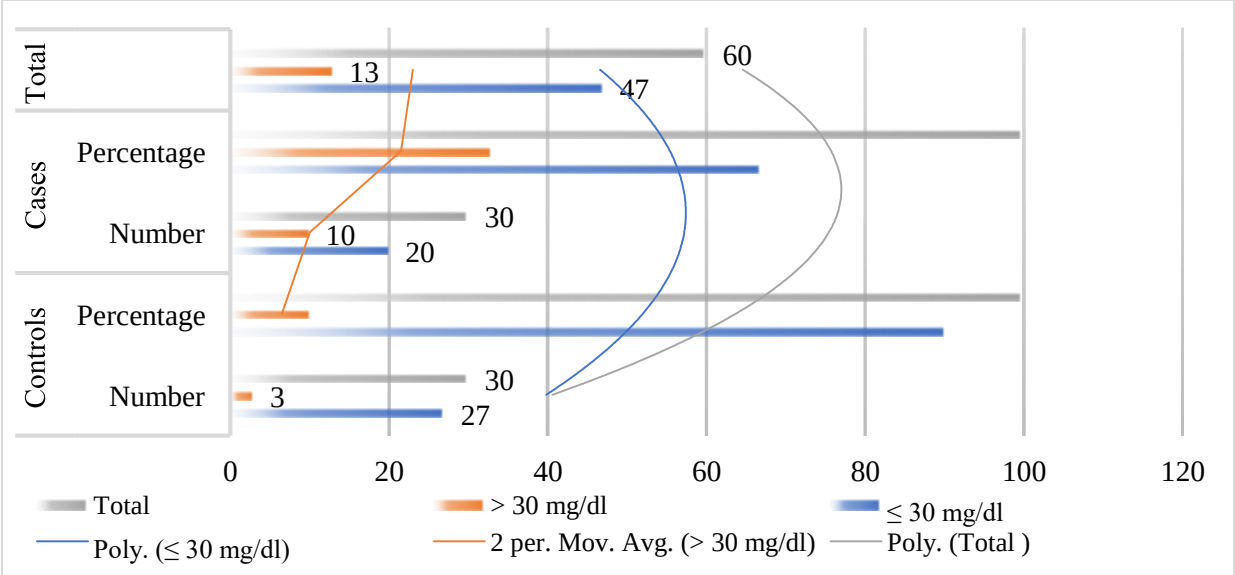


Table – III: Serum Lipoprotein (a) comparison among cases and controls

Lp(a)	Controls		Cases		Total	P-Value
	Number	Percentage	Number	Percentage		
≤ 30 mg/dl	27	90.0	20	67.0	47	0.028
> 30 mg/dl	3	10.0	10	33.0	13	
Total	30	100.0	30	100.0	60	



DISCUSSION:

We did this research on 30 PCOS cases and on 30 controls which were healthy to ascertain the disorders of serum lipid and levels of lipoprotein(a) in polycystic ovarian syndrome. The changes of lipid profile are related to polycystic ovary syndrome.

This research was carried out at Kempegowda Institute of Medical Sciences & Hospital. All the patients ranged from 18 – 35 years of age. Statistically mean age amid the cases and the controls were not important (P>0.05). It reinforces the researches by Ahmed M Muhammad and Shou-Kui Xiang, who concluded that there is no variance in age of PCOS and controls [6, 8]. LDL, TG, and TC of high levels means were in cases of PCOS as compared to controls. Research of Olivier also proves that LDL-C, cholesterol and triglycerides are elevated when combined with reduced apoA-I and HDL-C [3]. Research by Berneis

showed that low HDL-C is frequently observed as opposed to hypertriglyceridemia in the cases of PCOS. They also concluded that lipid variations and high LDL-C are not found in every patient of PCOS but the value of LDL has an undeviating effect on cardiac diseases. The presence of small LDL in dense form is also considered as an increased factor of risk for the cardiac diseases by The National Cholesterol Education Program Adult Treatment Panel III [5].

In the research of Burghen, 1980, the relation of hyperinsulinemia and hyperandrogenism of PCOS female while in Chang, 1983, explained the resistance of insulin in non-obese of PCOS female. The study of Legro showed that female having PCOS are seven times more prone to have type II diabetes mellitus. The study of Ehrmann explained that six percent PCOS female of impaired glucose tolerance is converted to type II diabetes annually. The dysfunction of pancreatic β-cell and profound resistance of peripheral insulin are the causes of intolerance of glucose in PCOS female [9]. Cardiac diseases are predicted by hyperinsulinemia too.

In this research, we observed that mean of LDL was high and HDL-C was low in PCOS cases as opposed to controls. It reinforces the research by Wild, which show that PCOS female have less Apolipoprotein A1: A2 ratios and HDL2-C while they have high VLDL-C and triglycerides.

The research by Koval shows that overweight PCOS female of African-American nature, compared with Caucasians, have a level of high HDL-C by 5.1 mg/dl. It proposes that the difference of HDL-C in races can be a cause in the classification of risk in cardiac diseases. We observed during our research that PCOS female, opposed to controls, are prone to have a mean of high LDL-C/HDL-C and TC/HDL. It conforms to the findings of Shou-Kui Xiang [8]. The irregularities lipid, excluding being overweight, are linked with insulin resistance. They explained that with PCOS female insulin resistance is directly influenced by the ratios of serum lipoprotein and this can be utilized as an economic, simple and reliable gauge for assessing the resistance of insulin. So clinically it can be useful for diagnosing and treating patients of PCOS. Comparing controls of ($P<0.05$) with cases of Lp(a) >30mg/dl, our research indicate that the number of cases is greater than controls. Statistically, the difference of the higher mean in Lp(a) cases as compared to controls is not important ($P>0.05$). The conclusions of our research conform to those of Ahmed M Mohamad, as he explains there are high total homocysteine, fibrinogen, hs-CRP, Lp(a) and circulating ADMA in PCOS patients as compared to controls [6].

Most of the researches show that PCOS female is more prone to cardiac diseases, high carotid intima-media thickness, more carotid plaques, the disapproving pattern of lipoprotein. But a study of longer duration in the UK indicated that there has not been any increase in CVD. It leads to the question there are other factors of protection in the patient of PCOS. The study conducted in the UK consisted of an age having mean 58 years while CVD in a female does not increase up to 7^{th} and 8^{th} decades [8 – 10].

Now a day's nonproductive feature of PCOS is getting more attention. In recent years, increasing attention has been paid to the non-reproductive aspects of PCOS. The effects, in future, of the disturbances of metabolism on the health of women has got attention to intervention and follow-up studies bases.

CONCLUSION:

This study intended to show the impact on the profile of lipid by polycystic ovary syndrome where the strongest parameter of atherogenic lipid is Lipoprotein(a). In the research, the following parameters were elevated such as TG, TC, VLDL, LDL, LDL/HDL ratio, TG/HDL ratio and TC/HDL and the levels of HDL were reduced. In cases, the levels of Lp(a) were observed high like > 30mg/dl as compared to controls. The research concludes that the patient is at greater risk to have metabolic syndrome as polycystic ovarian syndrome causes the profile of atherogenic lipid. It is imperative to have more studies in this context with a greater number of participants to verify the changes caused in lipoprotein(a) by polycystic ovarian syndrome and to consider the importance of family history, physical activity, social parity and education.

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