Original Article

"ASSESSMENT AND EVALUATION OF SERUM LIPID PROFILE IN PREGNANCIES COMPLICATED BY PRE-ECLAMPSIA: A TERTIARY CARE HOSPITAL BASED STUDY"

Pavan Kumar Sharma[1*], Pawan Arun Kulkarni[2], Sushil Kumar[3]

[1]*Assistant Professor, Department of Biochemistry, Major SD Singh Medical College & Hospital, India.

[2]Assistant Professor, Department of Biochemistry, Rama Medical College Hospital and Research Centre, Mandhana, Kanpur, Uttar Pradesh, India

[4 3]Tutor Department of Biochemistry, G.M.C, Banda, India.

***Corresponding author:** Dr. Pavan Kumar Sharma
*Assistant Professor Department of Biochemistry, Major SD Singh Medical College & Hospital, India. .Email ID: doctorpavan1980@gmail.com

ABSTRACT

Pre-eclampsia is the most common complication of pregnancy that leads to considerable morbidity and mortality in both the mother and the foetus. Preeclampsia is a multisystem condition associated with pregnancy that goes away after delivery and is marked by hypertension and proteinuria. Our goal was to examine the changes in lipid profiles between pre-eclampsia and a typical pregnancy. According to the current study, the woman who developed pre-eclampsia may have had abnormal lipid metabolism, which resulted in a distinct lipid profile.

An association exists between pregnancy and physiological hyperlipidemia. Pregnancyinduced hypertension is caused by abnormal increases in triglycerides, LDL, VLDL, and total cholesterol, which in turn stimulate oxidative stress and vascular dysfunction. In order to reduce obstetric complications including eclampsia, antepartum haemorrhage, and preterm labour linked to pre-eclampsia, evaluation of the blood lipid profile in pregnant women during early antenatal visits may help in the prediction and early identification of pre-eclampsia.

Keywords: Serum lipid profile, Pre-eclampsia, Hypertension, Lipid Profile, LDL,VLDL

Introduction:

Pre-eclampsia is a disorder that occurs only during pregnancy and the postpartum period. It affects both the mother and the unborn baby and occurs in approximately 5% of all pregnancies, being an important cause of maternal morbidity and mortality.

The most common pregnancy problem that causes morbidity and mortality in both the mother and the foetus is pre-eclampsia. Preeclampsia is a pregnancy-specific multisystem condition that goes away after delivery and is marked by proteinuria and hypertension [1]. Preeclampsia is an illness

that only appears during pregnancy and the first few weeks after giving birth. It occurs in around 5% of pregnancies and affects both the mother and the foetus, greatly increasing maternal morbidity and mortality [2, 3].Important aspects of this disease's pathophysiology are still unclear despite extensive research, which hinders the development of preventive and therapeutic measures.

In underdeveloped nations, preeclampsia is a frequent medical issue during pregnancy. It is among the most frequent causes of morbidity and mortality in both mothers and foetuses.

Worldwide, preeclampsia occurs in 3-5% of cases.3 [1].

Placental products that enter the mother's circulation and induce endothelial dysfunction are the origin of this problem. This leads to the development of atherosclerosis and other cardiovascular diseases such as vasospasm, increased endothelial permeability, and activation of thrombogenic pathways [4]. Preeclampsia risk is strongly influenced by maternal factors; women who have diabetes, hyperlipidemia, or persistent hypertension are more likely to have severe vascular reactivity, which can result in serious physiological abnormalities.

Women with preeclampsia present arterial lesions at the uteroplacental implantation site. These morphological le-sions are usually observed in cases of acute atherosclero-sis, and are characterized by areas with fibrinoid necrosis surrounded by lipid-laden macrophages [5].

The atherosclerosis observed extrapregnancy is equivalent to these microscopic lesions. Lipid deposits, or glomerular endotheliosis, are another disease that can be seen in the glomeruli of pre-eclamptic patients. Proteinuria, a predictor and marker of disease severity, is associated with glomerular lesions [4]. Triglycerides 6–8 and low-density lipoproteins (LDL) have also been suggested as possible causes of renal damage.Moreover, changes in lipid metabolism might be a factor in the endothelial lesions linked to preeclampsia.

Furthermore, changes to lipid metabolism may contribute towards the endothelial lesions observed in preeclampsia [2]. The severity of both hypertension and proteinuria seems to reflect the degree of endothelial damage.The possible correlation between the altered lipid profile and the severity of renal lesions, as reflected by proteinuria, may contribute towards clarify the complex pathophysiology of preeclampsia [5-9].

There is an increase in plasma lipids even during a normal pregnancy, but this rise is not atherogenic and may instead be physiological as a result of hormonal regulation.Pregnancy difficulties arise whenever this system of regulating physiologic hyperlipidemia is changed [10–12]. Therefore, estimating serum lipid profiles during pregnancy aids in identifying highrisk individuals who may develop preeclampsia.1. A higher risk of unfavourable foetal, neonatal, and maternal outcomes—such as preterm birth, intrauterine growth restriction (IUGR), perinatal death, acute renal and hepatic failure, antepartum haemorrhage, postpartum haemorrhage, and maternal death—is linked to pregnancies complicated by hypertension [13– 15]. Consequently, the current study was conducted at a hospital to assess the serum lipid profile in pregnancies complicated by pre-eclampsia.

Material and methods:

The present study was conducted in the Department of Biochemistry, at Major SD Singh Medical College & Hospital, India for a duration of 6 months 1.e, January 2019 to June 2019. The pregnant cases were obtained from the Department of Obstetrics and Gynecology OPD and IPD from. The estimation of serum lipid profile was done in the Department of Biochemistry. The present study consists of total 150 subjects who are further subdivided in to two groups;

1. Group-I: Includes total 100 Pregnant women with preeclampsia (Cases) 2. Group-II: Consists of 50 Normal pregnant women (Controls).

Inclusion criteria: Patients with gestational age 29 weeks to term.

Exclusion criteria: The cases and controls having past history of diabetes mellitus, hypertension, renal disease, liver disorders, multiple pregnancies and history of treatment with drug influencing lipid profile were excluded.

The pre-eclamptic patients were diagnosed by the presence of persistent hypertension (140/90 mm of Hg or more) gross proteinurea with or without oedema. Blood samples were drawn from all the subjects following a fast of 12-14 hours and were studied for following parameters.

1. Total Cholesterol (TC) by enzymatic end point CHODPOD methods.
2. Triglyceride (TG) by enzymatic glycerol phosphate oxi-dase/peroxidase methods.
3. HDL-Cholesterol by direct enzymatic end point meth-od.
4. LDL-Cholesterol by Friedewald's formula.
5. VLDL-Cholesterol by Friedewald's eqution.
6. LDL-c = Tc-HDL-c(TG/5)

All values were expressed as mean±SD. We used student t-test and pearson's correlation coefficient to find the statistical significance. A P-value <0.05 was to be considered statistically significant.

Results and Discussion:

Table-1 shows the Demographic and clinical characteristics of control and study groups. There was no significant difference of maternal age and gestational age between control and study groups. The mean value of systolic blood pressure in mm of Hg (SBP) in control was 134.20 ± 2.6 and in study group 160.2 ± 6.8 there being a significant difference ($p < 0.01$) between study and control groups. The mean diastolic blood pressure in mm of Hg (DBP) in study and control group were 98.8 ± 9.1 and 83.23 ± 4.6 respectively, there being a significant difference ($p < 0.01$) between study and controls. The mean BMI (Body Mass Index) in study group was 20.9 ± 7.3 & in controls was 23.1±4.3

'p' value was more than 0.05, which was statistically insignificant.

Parameters	**Controls (N=50) (Mean±SD)**	**Cases (N=100) (Mean±SD)**	**P – Value**
Age (Yrs)	26.7 ± 2.3	24.4 ± 4.2	0.08*
Body mass index(BMI)	23.1 ± 4.3	20.9 ± 7.3	0.065*
Systolic BP (mm/Hg)	134.20 ± 2.6	160.2 ± 6.8	0.03
Diastolic BP(mm/Hg)	83.23 ± 4.6	98.8 ± 9.1	0.01
Period of gestation(in weeks)	34.43 ± 3.1	34.01 ± 2.8	0.062*

Parameters	Controls (N=50) (Mean±SD)	Cases (N=100) (Mean±SD)	P- value
TC (mg/dl)	192.2 ± 18.6	252.4 ± 25.7	0.01
TG (mg/dl)	135.6 ± 49.3	299.3 ± 40.8.5	0.03
HDL(mg/dl)	46.2 ± 3.06	43.6. ± 6.32	0.07*
LDL(mg/dl)	128.36 ± 19.3	243.2 ± 17.9	0.01
VLDL(mg/dl)	27.01 ± 3.9	53.07 ± 9.5	0.1

Comparison of Lipid Profiles of control and study groups are shown in Table-2. TC, TG, LDL, VLDL, HDL levels were 192.2 ± 18.6, 135.6 ± 49.3, 128.36 ± 19.3, 27.01 ± 3.9, 46.2 ± 3.06 respectively in controls. Whereas 252.4 ± 25.7, 299.3 ± 40.8.5, 243.2 ± 17.9, 53.07 ± 9.5, 43.6. ± 6.32 in the study group. There was a significant increase in the TGL, LDL and VLDL levels in hypertensive group compared to normotensive group.. There was no statistical difference between both groups in case of HDL(mg/dl).

Hypertensive disorders during pregnancies, named preeclampsia, are a pregnancy-specific disorder that affects 3–5% [12,13] of pregnant women worldwide.

The pathogenesis of this condition is multifactorial and the key aspect is endothelial injury. In the present study, a total of 150 subjects were studied, out of which 50 were normotensive pregnant women and 100 were hypertensive pregnant women. The difference in blood pressure is statistically significant ($p < 0.01$). Comparing the lipid profiles between cases and controls (Table 2), it is observed that the level of triglyceride is significantly high ($p < 0.01$) in pregnancies complicated by hypertension. This finding is consistent with findings of Aziz R et al (2007) [13] and other workers. The level of HDL showed statistically not significant difference between the two groups, which is similar to the observations of Cuneyt Evruke et al (2004) [14] and others, while few workers have shown decrease in the level of HDL.

A significant rise in the level of LDL ($p < 0.01$) and VLDL ($p < 0.01$) was seen in the present study, which is similar to the findings of Sahu S.et al(2009) [15] and other workers ($p < 0.01$). In the present study, the pregnant women who subsequently developed hypertensive disorder in pregnancy showed high level of total cholesterol ($p < 0.01$), which is similar to the observation noted by Cekman B et al (2003) [16] and others.

The association between dyslipidemia and risk of pre-eclampsia is biologically possible and is compatible with what is known about the pathophysiology of pre-eclamp-sia. The association between dyslipidemia and pre-ec-lampsia can be explained by 3 hypothesis.

The fact that the patients with preeclampsia presented dyslipidemia, characterized by high levels of triglycerides and VLDL, indicates that there are common interfaces between preeclampsia and the endothelial lesions that occur in atherosclerosis. Our results allow us to hypothesize that these lesions may evoke adverse cardiovascular events later on during the adulthood of these women. In a systematic review of the literature, Bellamy et al.reported that women with a history of preeclampsia presented increased risk of cardiovascular disease (risk relative, RR = 3.7), hypertension (RR = 2.16), ischemic heart attack (RR = 1.81), venous thromboembolism (RR = 1.79) and death (RR = 1.49). These findings confirm the possible association between hypertension during pregnancy and future cardiovascular disease [17].

First, oxidative stress causes endothelial dysfunction due to increased plasma lipids and lipoproteins. Additionally, dyslipidemia hinders the trophoblasts' ability to invade the mother's blood vessels, which sets off a series of pathophysiological events that culminate in the development of pre-eclampsia. The second mechanism involves the dysregulation of lipoprotein lipase, which leads to a dyslipidemic lipid profile and the pathologic process of pre-eclampsia. Sera from pre-eclamptic women exhibited enhanced uptake of free fatty acids, which are subsequently esterified to triglycerides, as well as a greater ratio of free fatty acids to albumin. Metabolic syndrome could be a third potential route. Pre-eclampsia also exhibits the metabolic features of "insulin resistance syndrome," including hyperinsulinemia and hyperuricemia [18].

Disorders of lipoprotein metabolism are reported to be a major cause of hypertension and proteinuria in Pre-eclampsia . In view of the above findings it is postulated that alteration of lipid metabolism may play a key role in the development of symptoms of Pre-eclampsia and Eclampsia.

Prenatal blood lipid profile evaluation may be useful in predicting and identifying preeclampsia early on, hence averting obstetric problems such as eclampsia, antepartum haemorrhage, and preterm labour linked to pre-eclampsia. However, when applied to a larger number of individuals and long-term follow-up to observe changes in blood lipid profile, the current study would yield superior results.

Conclusion:

These results point to aberrant lipid metabolism as the cause of the woman's altered lipid profile and pre-eclampsia. The condition of physiological hyperlipidemia is linked to pregnancy. However, an aberrant rise in total cholesterol, LDL, VLDL, and triglycerides promotes oxidative stress and vascular dysfunction, which in turn causes pregnancy-induced hypertension. Therefore, these women should receive adequate counseling to urge them to adopt healthier habits and lifestyles and to seek periodic checkups, in order to detect cardiovascular disease in its early stages, before irreparable damage or even death ensues.

Declarations:

Conflicts of interest: There is no any conflict of interest associated with this study Consent to participate: We have consent to participate.

Consent for publication: We have consent for the publication of this paper.

Authors' contributions: All the authors equally contributed the work.

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