



## Evaluation of Serum Lipid Profile in Pregnancies Complicated by Pre-Eclampsia: A Hospital Based Study

### KEYWORDS

Hypertension, Pre-eclampsia and Lipid profile.

### Dr Pavan Kumar Sharma

MBBS, PG Student, Department of Biochemistry, Rama Medical College Hospital & Research Centre, Kanpur, Uttar Pradesh

### Dr P. Satyanarayana

Professor and HOD, in Biochemistry, Rama Medical College Hospital & Research Centre, Kanpur, Uttar Pradesh

### Dr I. Chaudhary,

Assoc. prof., in Biochemistry, Rama Medical College Hospital & Research Centre, Kanpur, Uttar Pradesh

### Dr P. Anand

Ap, Dept. of Biochemistry, Rama Medical College Hospital & Research Centre, Kanpur, Uttar Pradesh

**ABSTRACT** *Pre-eclampsia is the most common complication of pregnancy and is a leading cause of maternal and perinatal morbidity and mortality. Preeclampsia is a pregnancy specific multisystem disorder characterized by hypertension and proteinuria that remits after delivery. Our aim was to compare the changes in lipid profile in normal pregnancy and in pre-eclampsia. The present study suggest that the woman who developed pre-eclampsia had altered lipid profile due to abnormal lipid metabolism. Pregnancy is associated with physiological hyperlipidemia. But abnormal increase in triglycerides, LDL, VLDL and total cholesterol contribute to promotion of oxidative stress and vascular dysfunction leading to pregnancy – induced hypertension. Evaluation of blood lipid profile in pregnant women during early antenatal visits could be helpful in prediction and early detection of pre-eclampsia and thus preventing obstetric complications like eclampsia, antepartum haemorrhage, preterm labour associated with pre-eclampsia.*

### Introduction:

Pre-eclampsia is the most common complication of pregnancy and is a leading cause of maternal and perinatal morbidity and mortality. Preeclampsia is a pregnancy specific multisystem disorder characterized by hypertension and proteinuria that remits after delivery.<sup>1</sup> 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.<sup>2,3</sup> Despite extensive investigations, important pathophysiological aspects of this disease remain unknown, thus delaying the development of preventive and therapeutic strategies.

This disorder is mediated by placental products that reach the maternal circulation and trigger endothelial dysfunction, thereby evoking cardiovascular diseases, such as vasospasm, increased endothelial permeability and activation of thrombogenic mechanisms, and leading to the early events of atherosclerosis.<sup>4</sup> Susceptibility to preeclampsia is also modulated by maternal factors, and women who present chronic hypertension, diabetes or hyperlipidemia are more likely to exhibit intense vascular reactivity, which evokes important disorders of physiological conditions.

Women with preeclampsia present arterial lesions at the uteroplacental implantation site. These morphological lesions are usually observed in cases of acute atherosclerosis, and are characterized by areas with fibrinoid necrosis surrounded by lipid-laden macrophages.<sup>5</sup>

These microscopic lesions are similar to atherosclerosis found outside pregnancy. Lipid deposits are also seen in the glomeruli of pre-eclamptic patients, a finding known as glomerular endotheliosis. Glomerular lesions are associated with proteinuria, a predictive indicator and marker of disease severity.<sup>4</sup> It has also been suggested that low-density

lipoproteins (LDL)<sup>4</sup> and triglycerides<sup>6-8</sup> may be involved in this renal damage. Furthermore, changes to lipid metabolism may contribute to wards the endothelial lesions observed in preeclampsia.<sup>9</sup> The severity of both hypertension and proteinuria seems to reflect the degree of endothelial damage.<sup>10,11</sup> **Therefore, this study has been carried out to compare the changes in lipid profile in normal pregnancy and in pre-eclampsia.**

### Material and methods:

The present study was conducted in the Department of Biochemistry, Rama Medical College Hospital and Research Centre, Kanpur, Uttar Pradesh, India during the year 2014 to 2015. The pregnant cases were obtained from the Department of Obstetrics and Gynecology OPD and IPD from November, 2014 to August, 2015, Rama Medical College Hospital and Research Centre, Kanpur, Uttar Pradesh, India. The estimation of serum lipid profile<sup>12</sup> was done in the Department of Biochemistry. The present study consists of total 80 subjects who are further subdivided in to two groups;

1. Group-I: Includes total 50 Pregnant women with preeclampsia (Cases)
2. Group-II: Consists of 30 Normal pregnant women (Controls).

Inclusion criteria were primi, gestational age 29 weeks to term. 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 proteinuria 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 CHOD-

POD methods.

2. Triglyceride (TG) by enzymatic glycerol phosphate oxidase/peroxidase methods.
3. HDL-Cholesterol by direct enzymatic end point method.
4. LDL-Cholesterol by Friedewald's formula.
5. VLDL-Cholesterol by Friedewald's equation.
6.  $LDL-c = Tc-HDL-c(TG/5)$

All values were expressed as mean $\pm$ 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 study group was  $144.2 \pm 9.02$  and in control  $112.21 \pm 6.7$  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  $95.02 \pm 3.04$  and  $77.06 \pm 5.6$  respectively, there being a significant difference ( $p < 0.01$ ) between study and controls. The mean BMI (Body Mass Index) in study group was  $27.6 \pm 3.03$  & in controls was  $26.3 \pm 3.6$ , 'p' value was more than 0.05, which was statistically insignificant.

**Table-1: Demographics and clinical characteristics of the study population:**

Parameters	Controls (N=30) (Mean $\pm$ SD)	Cases (N=50) (Mean $\pm$ SD)	P - Value
Age (Yrs)	$25.3 \pm 4.8$	$27.5 \pm 4.7$	*NS
Body mass index(BMI)	$26.3 \pm 3.6$	$27.6 \pm 3.03$	*NS
Systolic BP (mm/Hg)	$112.21 \pm 6.7$	$144.2 \pm 9.02$	0.01
Diastolic BP(mm/Hg)	$77.06 \pm 5.6$	$95.02 \pm 3.04$	0.01
Period of gestation(in weeks)	$31.59 \pm 2.90$	$31.01 \pm 2.32$	*NS

( $p < 0.05$  - Statistically significant); \*NS: Statistically not Significant

**Table-2: Comparison of lipid profile between Group (A and B):**

Parameters	Controls (N=30) (Mean $\pm$ SD)	Cases (N=50) (Mean $\pm$ SD)	P- value
TC ( mg/dl)	$180.7 \pm 27.1$	$256.4 \pm 40.02$	0.01
TG (mg/dl)	$123.5 \pm 25.02$	$315.04 \pm 79.5$	0.01
HDL (mg/dl)	$49.4 \pm 2.06$	$49.7 \pm 5.02$	*NS
LDL (mg/dl)	$120.06 \pm 24.6$	$255.03 \pm 41.9$	0.01
VLDL (mg/dl)	$24.01 \pm 5.4$	$62.2 \pm 15.1$	0.01

( $p < 0.05$  - Statistically significant); \*NS: Statistically not Significant

Comparison of Lipid Profiles of control and study groups are shown in Table-2. TC, TG, LDL, VLDL levels were  $256.4 \pm 40.02$ ,  $315.04 \pm 79.5$ ,  $255.03 \pm 41.9$ ,  $62.2 \pm 15.1$  respectively in case group. Whereas  $180.7 \pm 27.1$ ,  $123.5 \pm 25.02$ ,  $120.06 \pm 24.6$ ,  $24.01 \pm 5.4$  in the control group. There was a significant increase in the TGL, LDL and VLDL levels in hypertensive group compared to normotensive group. HDL level was  $49.7 \pm 5.02$  in the case group and  $49.4 \pm 2.06$  in the control group. There was no statistical difference between both groups.

Hypertension is still the most common medical disorder as-

sociated with pregnancy, adversely affecting both mother and fetus. The pathogenesis of this condition is multifactorial and the key aspect is endothelial injury. In the present study, a total of 80 subjects were studied, out of which 30 were normotensive pregnant women and 50 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)<sup>13</sup> 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)<sup>14</sup> 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)<sup>15</sup> 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)<sup>16</sup> 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-eclampsia. The association between dyslipidemia and pre-eclampsia can be explained by 3 hypothesis. First, elevated plasma lipids and lipoproteins induce endothelial dysfunction secondary to oxidative stress. Dyslipidemia also impairs trophoblastic invasion of maternal blood vessels, thus contributing to a cascade of pathophysiological events that lead to development of pre-eclampsia. The second mechanism is the pathologic process of pre-eclampsia via dysregulation of lipoprotein lipase resulting in a dyslipidemic lipid profile. Sera from pre eclamptic women had both a higher ratio of free fatty acids to albumin and increased uptake of free fatty acids, which are further esterified to triglycerides. A third possible mechanism may be via the metabolic syndrome. Metabolic characteristics of "insulin resistance syndrome" namely, hyperinsulinemia and hyperuricemia are also present in pre-eclampsia.

### Conclusion:

These findings suggest that the woman who developed pre-eclampsia had altered lipid profile due to abnormal lipid metabolism. Pregnancy is associated with physiological hyperlipidemia. But abnormal increase in triglycerides, LDL, VLDL and total cholesterol contribute to promotion of oxidative stress and vascular dysfunction leading to pregnancy - induced hypertension. Evaluation of blood lipid profile in pregnant women during early antenatal visits could be helpful in prediction and early detection of pre-eclampsia and thus preventing obstetric complications like eclampsia, antepartum haemorrhage, preterm labour associated with pre-eclampsia. The present study would however give better results when applied on a larger number of subjects and long term follow - up to see changes in blood lipid profile.

### Bibliography:

1. T.Vijaya Krishna, A. Singh, et.al. Indian Journal of Obstetrics and Gynaecology Research 2015;2(4):261-263.
2. Walker JJ. Pre-eclampsia. Lancet. 2000;356(9237):1260-5.
3. Roberts JM, Copper DW. Pathogenesis and genetics of pre-eclampsia. Lancet. 2001;357(9249):53-6.

4. Airoidi J, Weinstein L. Clinical significance of proteinuria in pregnancy. *ObstetGynecolSurv.* 2007;62(2):117-24.
5. Ross R. Atherosclerosis--an inflammatory disease. *N Engl J Med.* 1999;340(2):115-26.
6. Sattar N, Bendoric A, Berry C, et al. Lipoprotein subfraction concentrations in preeclampsia: pathogenic parallels to atherosclerosis. *Obstet Gynecol.* 1997;89(3):403-8.
7. Hubel CA, Lyall F, Weissfeld L, Gandley RE, Roberts JM. Small low-density lipoproteins and vascular cell adhesion molecule-1 are increased in association with hyperlipidemia in preeclampsia. *Metabolism.* 1998;47(10):1281-8.
8. Winkler K, Wetzka B, Hoffmann MM, et al. Triglyceride-rich lipoproteins are associated with hypertension in preeclampsia. *J ClinEndocrinolMetab.* 2003;88(3):1162-6.
9. Hubel CA, McLaughlin MK, Evans RW, et al. Fasting serum triglycerides, free fatty acids, and malondialdehyde are increased in preeclampsia, are positively correlated, and decrease within 48 hours post partum. *Am J Obstet Gynecol.* 1996;174(3):975-82.
10. Sahu S, Abraham R, Vedavalli R, Daniel M. Study of lipid profile, lipid peroxidation and vitamin E in pregnancy induced hypertension. *Indian J PhysiolPharmacol.* 2009;53(4):365-9.
11. Roberts JM, Hubel CA. Is oxidative stress the link in the two-stage model of pre-eclampsia? *Lancet.* 1999;354(9181):788-9.
12. Manoj Kumar Yadav, Dr Tapan Kumar Mohapatra, Dr Rabindra Kumar Mohapatra et. al. Study on Glycated Hemoglobin & lipid profile in Type-2 Diabetes Mellitus. *International Journal of Science & Research (IJSR)* 2015;4(6): 1917-1919.
13. Rubina Aziz, TabassumMahboob. Pre eclampsia and lipid profile. *Pak J Med Sci.* October – December 2007(part 1) vol.23:no.5,pp 751-754.
14. CuneytEvrake, MD; S.Cansun, MD; Ibrahim F; Urunsak, MD;F.TuncayOzgunen, MD; OktayKadayfci, MD. Comparison of lipid profiles in normal and hypertensive pregnant women. *Ann Saudi Med* 2004;24(5):382-385.
15. SuchandaSahu, Rebecca Abraham, R.Vedavalli and Mary Daniel. Study of lipid profile, lipid peroxidation and Vitamin E in pregnancy induced hypertension. *Indian J PhysiolPharmacol* 2009;53(4):365-369.
16. Mustafa BakiCekmen, AyseBinnurErbagci, AyseBalat, Can Duman, Hale Maral, KivancErgen, MeltemOzden, OzcanBalat and SevincKuskay. Plasma lipid and lipoprotein concentrations in pregnancy induced hypertension. *Clinical Biochemistry – New York* 2003; vol.36 no.7:pp 575-578.