

# Study on Serum Lipid Profile in Pregnancies Complicated by Pre-Eclampsia: A Teaching Hospital Based Study.

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## ABSTRACT

**Background:** Preeclampsia is a common medical complication of pregnancy. In India, the incidence of preeclampsia is reported to be 8%–10% of all the pregnancies. It leads significantly to maternal and fetal mortality and morbidity. **Methods:** The present study consists of total 90 subjects who are further subdivided in to two groups; Group-A: 45 pregnant women with preeclampsia as cases and Group-B: 45 Normal pregnant women as controls. **Results:** In present study, we observed an association between maternal dyslipidemia, particularly hypertriglyceridemia and the subsequent risk of preeclampsia. Pregnant women who had preeclampsia had increased total cholesterol, Low density lipoprotein-cholesterol, very Low density lipoprotein-cholesterol, and decreased High density lipoprotein-cholesterol concentrations as compared with pregnant women who were normotensive. **Conclusion:** 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-c, VLDL-c and total cholesterol contribute to promotion of oxidative stress and vascular dysfunction leading to pregnancy – induced hypertension.

**Keywords:** Pre-eclampsia, Dyslipidemia and Hypertension.

## INTRODUCTION

Pre-eclampsia is one of the most common complications of pregnancy. In India, the incidence of preeclampsia is reported to be 8%–10% of all the pregnancies.<sup>[1]</sup> It is a cause of high morbidity for both mother & fetus, especially in developing countries.<sup>[2]</sup> Despite extensive investigations, important pathophysiological aspects of this disease remain unknown, thus delaying the development of preventive and therapeutic strategies. Preeclampsia is a multisystem disorder characterized by hypertension to the extent of 140/90 mmHg or more, proteinuria ( $\geq 300$  mg/day) and edema induced by pregnancy after the 20th week.<sup>[3]</sup> Without any intervention, it progresses to eclampsia, which is characterized by malignant hypertension and epileptiform convulsions requiring emergency cesarean section.<sup>[4]</sup> 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>[5]</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>[6]</sup> These microscopic lesions

are similar to atherosclerosis found out-side 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>[5]</sup> It has also been suggested that low-density lipoproteins (LDL) and triglycerides may be involved in this renal damage.<sup>[7-9]</sup> Furthermore, changes to lipid metabolism may contribute to-wards the endothelial lesions observed in preeclampsia.<sup>[10]</sup> The severity of both hypertension and proteinuria seems to reflect the degree of endothelial damage.<sup>[11,12]</sup> Aim of the present study has to be compare the changes in lipid profile in normal pregnancy and in pre-eclampsia.

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## MATERIALS AND METHODS

This present study was conducted in the Department of Obstetrics and Gynecology, Rama Medical College Hospital and Research Centre, in collaboration with the Department of Biochemistry. The pregnant cases were obtained from the Department of Obstetrics and Gynecology OPD and IPD during the period from September, 2014 to October, 2015, Rama Medical College Hospital and Research Centre, Hapur, Uttar Pradesh, India. The estimation of serum lipid profile was done in the Department of Biochemistry. The present study consists of total 90 subjects who are further subdivided in to two groups; Group-A: 45 Pregnant

women with preeclampsia as cases and Group-B: 45 Normal pregnant women as controls.

**Inclusion criteria for controls and Exclusion criteria:**

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. Triglycerides were measured by GOP-PAP method. Total cholesterol and HDL cholesterol were measured by CHOD-PAP method and low density lipoprotein cholesterol (LDL-cholesterol) were calculated - by Friedwald's formula. Statistical analysis was done using the unpaired T-test and Pearson's correlation.

**RESULTS AND DISCUSSION**

This present study was conducted in the Department of Obstetrics and Gynecology, Rama Medical College Hospital and Research Centre, Hapur, India. Demographic & clinical characteristic of control & study groups are shown in [Table 1]. There was no significant difference of maternal age and gestational age between group A and group B. The mean value of systolic blood pressure in mm of Hg in group A was 146.5 ± 8.62 and in group B 114.2 ± 5.1 there being a significant difference (p < 0.001) between group A and group B. The mean diastolic blood pressure in mm of Hg in Group A and group B were 96.32 ± 6.4 and 78.6 ± 4.7 respectively, there being a significant difference (p < 0.001) between study group and controls group. The mean Body Mass Index in group A was 26.8 ± 4.24 & in group B was 23.2 ± 3.4, 'p' value was (p < 0.001), which was statistically significant.

**Table 1:** Demographics and clinical characteristics of the study groups:

Variabless	Group A (N=45) Mean ± SD	Group B (N=45) Mean ± SD
Age in year**	26.7 ± 6.2	26.4 ± 4.3
Body mass index*	26.8 ± 4.24	23.2 ± 3.4
Systolic BP (mm/Hg)*	146.5 ± 8.62	114.2 ± 5.1
Diastolic BP(mm/Hg)*	96.32 ± 6.4	78.6 ± 4.7
Period of gestation (in weeks)**	30.21 ± 4.23	31.26 ± 2.01

(\*Statistically significant; p<0.05; \*\*statistically not significant)

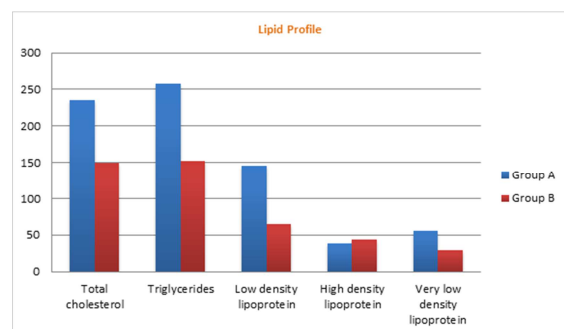
[Table 2] shows the lipid profiles of group A and group B. The mean triglycerides was found to be 257.24 ± 52.61 mg/dl in group A and 152.5 ± 25.2

mg/dl in group B. The increase level of triglycerides in preeclamptic group was found to be highly significant (P < 0.001) as compared to normotensive group [Table 2]. Total cholesterol level in both group as follows: Group a 235.4 ± 42.5 mg/dl and in the group B 150.07 ± 26.1 mg/dl. The group a (preeclamptic group) showed significant rise with P < 0.001. [Table 2] also shows that the mean Low density lipoprotein-cholesterol was found to be 145.32 ± 31.5 mg/dl in group A and 65.42 ± 20.3 mg/dl in the group B. The LDL showed a significant rise with P < 0.001. From [Table 2]: High density lipoprotein-cholesterol level was found to be 47.2 ± 5.21 mg/dk in the group A (preeclamptic) and in the group B (normotensive) 38.4 ± 2.4 mg/dl. The High density lipoprotein-cholesterol was found to be marginally lowered (P < 0.01) in the group a (preeclamptic). [Table 2] reveals Very low density lipoprotein-cholesterol level as 56.2 ± 12.4 mg/dl in group A and 29.04 ± 5.02 mg/dl in group B. The increase in Very low density lipoprotein-cholesterol in preeclamptic group A was found to be highly significant with P < 0.0001.

**Table 2:** Mean & standard deviation of lipid profile between Group (A and B):

Parameters (mg/dl)	Group A (N=45) Mean ± SD	Group B (N=45) Mean ± SD
Triglycerides*	257.24 ± 52.61	152.5 ± 25.2
Total cholesterol*	235.4 ± 42.5	150.07 ± 26.1
Low density lipoprotein-cholesterol*	145.32 ± 31.5	65.42 ± 20.3
High density lipoprotein-cholesterol*	38.4 ± 2.4	44.2 ± 5.21
Very low density lipoprotein-cholesterol*	56.2 ± 12.4	29.04 ± 5.02

(\*Statistically significant; p<0.05; \*\*Statistically not significant)



**Figure 1:** Shows the mean of lipid profile between Group (A and B)

In this study we investigated the role of lipid profile in the occurrence of pre-eclampsia. Preeclampsia still remains as one of the most serious complications of pregnancy and the pathophysiology of the disease is not clearly understood. The possible involvement of genetic and immune mechanism in the etiology of preeclampsia has been increasingly attracting

attention. Damage from free radicals has been implicated in many pathological conditions, and the activity of free radicals may increase during pregnancy and preeclampsia. In this study, we observed an association between maternal dyslipidemia, particularly hypertriglyceridemia and the subsequent risk of preeclampsia. Pregnant women who had preeclampsia had increased total cholesterol, Low density lipoprotein-cholesterol, very Low density lipoprotein-cholesterol, and decreased High density lipoprotein-cholesterol concentrations as compared with pregnant women who were normotensive. Hypertension is still the most common medical disorder associated 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 90 subjects were studied, out of which 45 were normotensive pregnant women and 45 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 ( $p < 0.001$ ) in pregnancies complicated by hypertension. This finding is consistent with findings of Aziz R et al (2007),<sup>[13]</sup> and other workers. A significant rise in the level of LDL-c ( $p < 0.001$ ) and VLDL ( $p < 0.0001$ ) 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 ( $p < 0.001$ ), which is similar to the observation noted by Cekman B et al (2003),<sup>[16]</sup> and others. The level of high density lipoprotein cholesterol showed that the group A had lower value of HDL-C over patients of group B (normotensive), pregnancy which is consistent with study done by Mishra et al.<sup>[14]</sup> Statistically, the difference was highly significant ( $P < 0.001$ ). Estrogen is responsible for induction of triglycerides and high density lipoproteins and inhibition of serum low density lipoproteins and estrogen level falls in PIH.<sup>[15]</sup> Low levels of HDL in PIH are not only because of hypoestrogenemia but also are due to insulin resistance.

## CONCLUSION

In conclusion, the woman who developed preeclampsia had altered lipid profile due to abnormal lipid metabolism. Pregnancy is associated with physiological hyperlipidemia. But abnormal increase in triglycerides, LDL-c, VLDL-c and total cholesterol contribute to promotion of oxidative stress and vascular dysfunction leading to pregnancy induced hypertension. Hence, early detection of these parameters is going to aid in

better management of preeclampsia cases which is important to improve the maternal and fetal outcome.

## REFERENCES

1. Kamath SA. Hypertension in pregnancy. *J Assoc Physicians India* 2006;54:269-70.
2. Vanderjagt DJ, Patel RJ, El-Nafaty AU, Melah GS, Crossey MJ, Glew RH. High density lipoprotein and homocysteine levels correlate inversely in pre-eclamptic women in northern Nigeria. *Acta Obstet Gynaecol Scand.* 2004;83(6):536-42.
3. Dutta DC. Hypertensive disorders in pregnancy. In: Konar H.L., editor. *Textbook of Obstetrics*. 5th ed. Kolkata: New Central Book Agency; 2001. p. 234-55.
4. Packer CS. Biochemical markers and physiological parameters as indices for identifying patients at risk of developing preeclampsia. *J Hypertens* 2005;23:45-6.
5. Airolidi J, Weinstein L. Clinical significance of proteinuria in pregnancy. *ObstetGynecolSurv.* 2007;62(2):117-24.
6. Ross R. Atherosclerosis--an inflammatory disease. *N Engl J Med.* 1999;340(2):115-26.
7. Sattar N, Bedomir A, Berry C, et al. Lipoprotein subfraction concentrations in preeclampsia: pathogenic parallels to atherosclerosis. *Obstet Gynecol.* 1997;89(3):403-8.
8. 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.
9. Winkler K, Wetzka B, Hoffmann MM, et al. Triglyceride-rich lipoproteins are associated with hypertension in preeclampsia. *J ClinEndocrinolMetab.* 2003;88(3):1162-6.
10. 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.
11. 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.
12. Roberts JM, Hubel CA. Is oxidative stress the link in the two-stage model of pre-eclampsia? *Lancet.* 1999;354(9181):788-9.
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. Mishra PK, Yadav MK, Yadav KP, Simlai S. Evaluation of Serum Uric Acid and Lipid Profile in Pre-eclamptic Women: A Hospital Based Study. *J Med Sci and Clin Res* 2016;4:11314-20.
15. Dutta DC. Hypertensive disorders in pregnancy. In: Konar H.L., editor. *Textbook of Obstetrics*. 5th ed. Kolkata: New Central Book Agency; 2001. p. 234-55.
16. 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.
17. 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.

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