A TERTIARY CARE CENTRE BASED STUDY ON SERUM LIPID LEVELS IN HEALTHY NON-PREGNANT WOMEN, NORMAL PREGNANCY AND IN PREECLAMPSIA

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ABSTRACT

BACKGROUND
Preeclampsia is more prevalent in the first pregnancy and is associated with very high maternal and foetal morbidity and mortality. Although, the exact cause of preeclampsia is still unknown, the basic pathology lies in the endothelial cell dysfunction and intense vasospasm. Dyslipidaemia is believed to play a key role in altering the microenvironment around endothelium.

So, the objective is to study the serum lipid levels in healthy non-pregnant women, normal pregnancy and in preeclampsia.

MATERIALS AND METHODS
Fasting serum lipid levels were estimated in 40 healthy non-pregnant women (Group-1), 40 normotensive primigravida (Group-2) and in 40 preeclamptic primigravida in their third trimester (Group-3). Serum lipid levels were estimated using fully automated analyser. Data was analysed using SPSS Version 18 software. P value < 0.05 was considered as statistically significant.

RESULTS
Compared with normal pregnancy, in preeclampsia, the level of serum total cholesterol, triglycerides, VLDL and LDL were significantly increased and HDL was decreased significantly. In normal pregnant women, serum total cholesterol, triglycerides, HDL, VLDL and LDL levels were significantly high compared to healthy non-pregnant women.

CONCLUSION
Dyslipidaemia in pregnancy may be associated with increased risk of developing preeclampsia. Screening of the above parameters may help in developing strategies for prevention and better management of preeclampsia.

KEY WORDS
Preeclampsia; Dyslipidaemia; Endothelial Cell.

Study Group 2
Normotensive age matched primigravida with gestational age between 34 - 40 weeks.

Study Group 3
Preeclamptic age matched primigravida with gestational age between 34 - 40 weeks with blood pressure ≥ 140/90 mmHg in more than two occasions and proteinuria of at least 1+ in dipstick testing.

Exclusion Criteria
Any history of chronic hypertension, diabetes, kidney disease, liver disease, coagulation disorders or multiple pregnancies. Prior informed consent was taken from all. A detailed history was taken from all subjects that include age, gravida, parity and history of hypertension, dyslipidaemia, cardiac illness, renal disease, hepatic dysfunction or any other acute or chronic illness. Details of drug intake was also noted. Blood pressure recording along with a detailed physical examination was done. Urine protein was detected using dipstick method.

Statistical Analysis
The present study was designed as a comparative study and statistical analysis has been done to determine the differences between 3 groups. Data was analysed using Statistical Package for Social Sciences [SPSS] version 18. Results were expressed as Mean + SD. Mean differences between the groups were analysed using ANOVA. The p-value of < 0.05 was taken as the level of significance.

From the above three groups of subjects under aseptic precautions, blood was collected by venous puncture using disposable syringes and needles. Samples for lipid profile were obtained after 8 to 12 hours fasting. 2 mL of blood was drawn. Serum lipid profile was estimated using fully automated analyser- Clinical chemistry analyser- XL-300 (ERBA-TRANSASIA).

RESULTS

Figure 1. Comparison of Serum Total Cholesterol Levels (mg/dL), p= 0.000 (Highly Significant)

Figure 2. Comparison of Serum Triglyceride Levels (mg/dL), p= 0.000 (Highly Significant)

Figure 3. Comparison of Serum HDL-Cholesterol Levels (mg/dL), p= 0.000 (Highly Significant)

Figure 4. Comparison of Serum VLDL-Cholesterol Levels (mg/dL), p= 0.000 (Highly Significant)
The mean level of HDL in normal pregnant women was significantly higher than in healthy young women. The mean value was significantly decreased in preeclampsia. Oestrogen may be responsible for the induction of HDL. The increase in HDL in normotensive pregnant women can be explained by hyperoestrogenaemia. In preeclampsia, the oestrogen level decreases, so reduced HDL may be due to hypoestrogenaemia. The decrease in HDL may also be due to exaggerated insulin resistance seen in preeclampsia. This reduced HDL level may also contribute to the reduced prostacyclin level seen in preeclampsia. The effects of HDL on prostacyclin synthesis are mediated via up-regulation of cyclooxygenase-2 expression. Prostacyclin is an important regulator of vascular homeostasis. Lower serum HDL level may also reduce antioxidative protection due to paraoxonase-1 on other lipoproteins. Paraoxonase-1 is the enzyme responsible for HDLs antioxidant function. It is closely bound to the HDL particle.

The rise in VLDL in normal pregnancy may be due to hypertriglyceridaemia. Increased triglycerides leads to enhanced entry of VLDL, as endogenous triglycerides are carried into circulation by VLDL from the liver. The rise in triglyceride-rich VLDL particles during pregnancy is dependent more on an increased rate of synthesis caused by oestrogen. Increased VLDL in preeclampsia may be explained by heightened insulin resistance, which leads to increased inhibition of lipolytic activity. It will result in overproduction of VLDL in liver. Increased VLDL lipoproteins accumulate in the maternal vascular endothelium of uterine and renal vessels.

The mean serum LDL is increased in normal pregnancy compared to healthy young women. But the increase is not statistically significant in our study. In preeclamptic patients, the value obtained is significantly higher than other two groups. The increase of LDL in normal pregnant women could be secondary to increased conversion of abundant VLDL. In preeclampsia, increase in LDL level may be due to decreased level of oestrogen. The greatest effect of oestrogen on LDL metabolism was to increase the fractional catabolic rate of LDL particles. It was indicated that small dense LDL had a greater capacity to stimulate thrombocytopenia synthesis and release by endothelial cells, thereby causing vasocostriction.
Small dense LDL are also more susceptible to oxidative modifications. It leads to formation of peroxides that inhibit endothelium-derived relaxation factor and also leads to foam cell formation in decidua. By Uphol et al supports this hypothesis, as they observed increased levels of free radical products of lipid peroxidation (Malondialdehyde) in preeclamptic condition compared to normotensive pregnant women and healthy non-pregnant women. Oxidised LDL impairs endothelial function by expression of adhesion molecules, inhibition of endothelial prostacyclin synthesis, increased endothelin production and release and increased platelet aggregability.

CONCLUSION
In our study, preeclampsia is associated with decreased HDL and increased serum total cholesterol, triglycerides, VLDL and LDL levels in their third trimester. These factors are found to cause endothelial dysfunction in several ways. Endothelial cell dysfunction is said to be an important factor in the aetiopathogenesis of preeclampsia. Women with a history of preeclampsia are reported to be associated with increased risk of cardiovascular disease, hypertension, ischaemic heart attack, venous thromboembolism and death. These findings confirm the possible association between hypertension during pregnancy and future cardiovascular disease. In order to implement preventive healthcare protocols, it is important to identify risk factors for preeclampsia. So, changes in the serum levels of above parameters can be used as an effective marker in the early diagnosis of preeclampsia. Also, it may prevent the possible risk of developing cardiovascular disease in future.

REFERENCES


