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Original Research Article

Comparative Assessment of Lipid Profile in Pre-Eclampsia.

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Abstract:
Pre-eclampsia is common medical complication of pregnancy. The association of altered lipid profile in essential hypertension is well documented. The present study was planned to study the role of altered lipid profile in the development of pre- eclampsia.
Methodology: The study comprised of 30 normal pregnant women and 30 pre-eclamptic women in their third trimester of pregnancy. Serum Total cholesterol, Triglycerides, LDL Cholesterol and HDL- Cholesterol by enzymatic colorimetric method were done.
Results: There was significant rise in Serum Total cholesterol, Triglycerides, LDL Cholesterol and a significant decrease in HDL Cholesterol in pre-eclamptic group as compared to normal healthy pregnant women.
Conclusion: Altered lipid profile also has a potential role in the genesis of endothelial dysfunction and expression of pre-eclampsia. Early detection of these parameters may help patient by preventing complications in pre-eclampsia and is going to aid in better management of Pre-eclampsia.
Keywords: Lipid Profile, Dyslipidemia, Pre-eclampsia.

Introduction

Pre-eclampsia is common medical complication of pregnancy. In India, the incidence of pre-eclampsia is reported to be 8-10% of the pregnancies [1]. It contributes significantly to maternal and fetal mortality and morbidity. Pre-eclampsia is a multisystem disorder characterized by hypertension to the extent of 140/90 mm Hg or more, proteinuria (≥300mg/day) and edema induced by pregnancy after 20th week [2]. Without intervention, pre-eclampsia progresses to eclampsia which is characterized by malignant hypertension and epileptiform convulsions requiring emergency caesarian section [3]. The association of altered lipid profile in essential hypertension is well documented. In early pregnancy, there is increased body fat accumulation associated with increased lipogenesis, while in late pregnancy, there is accelerated breakdown of fat depots which play an important role in fetal development [4]. Early pregnancy dyslipidemia is associated with an increased risk of pre- eclampsia [5].

Women with a history of pre-eclampsia have significant differences in lipid parameters and an increased susceptibility to lipoprotein oxidation when compared with women who had normal pregnancy [6]. Therefore, simple measurement of serum lipid profile may be of good predictive value in pre- eclampsia. With this background in mind the present study was conducted to assess lipid profile in cases of pre- eclampsia and compare it with normal healthy pregnant women.

Materials and Methods

The present study was carried out in the Department of Obstetrics and Gynecology, at National Institute of Medical Sciences and Research, Jaipur. Informed consent was taken from all individual subjects inducted into the study. The study comprised of 30 normal healthy pregnant women and 30 pre-eclampsia cases attending antenatal OPD in their third trimester of pregnancy. The diagnosis of pre-eclampsia was based on the definition of American College of Obstetrics and Gynecologists [7].

1) Systolic blood pressure greater than140 mm Hg or a rise of at least 30 mmHg or

2) Diastolic blood pressure greater than 90 mm Hg or a rise of at least 15 mmHg (manifested on two occasions at least 6 hours apart) and

3) Proteinuria of 300 mg or greater in 24 hours urine collection or protein concentration of 1 gm/L (on two occasions of at least 6 hours apart).

Inclusion Criteria for Controls: Primigravida with normal BP, no proteinuria and without any other systemic or endocrine disorder. They were age matched with the cases. All subjects included were in their third trimester (gestational age of ≥28 weeks).

Exclusion Criteria: included diabetes mellitus with or without treatment, obesity, severe anemia (Hb <

6 gm%) or subjects suffering from any other systemic or endocrine disorder. Fasting blood samples (8ml) were collected by venipuncture and analyzed for Serum Triglycerides (TG), Total cholesterol (TC), HDL cholesterol (HDL-C), LDL cholesterol (LDL-C) by the Central Laboratory. Data were statistically analyzed by unpaired T test and expressed in terms of 'P' value.

Observations:

The study was conducted over a total sample size of 60 patients, comprising of 30 patient each in case and control group. The observations showed a significant difference in the lipid profile of cases when comparted to controls. (Table 1)

Table I: Comparison of serum lipid profile in the study groups:

Parameter	Study Group	Sample Size (N)	Mean±SD (mg/dl)
Total Cholesterol	Patients	30	195.40±15.11**
	Controls	30	176.34±12.10
LDL	Patients	30	134.5±11.65**
	Controls	30	114.2±11.32
HDL	Patients	30	42.26±12.03**
	Controls	30	55.20±10.02
TG	Patients	30	149.75±20.42**
	Controls	30	125.20±18.30

***p<0.001-highly significant*

Discussion:

Pre-eclampsia has long known to be associated with abnormal placentation and impaired placental perfusion. However, other conditions characterized by poor placentation, such as intrauterine growth retardation, do not necessarily result in pre-eclampsia [8]. This has led to the growing concept that maternal predisposing factors must combine with the placental disorder to result in preeclamptic maternal syndrome [9]. In the recent last years, several lines of evidence have suggested that part of this maternal predisposition could be explained by abnormal lipid metabolism. In this study, we observed significantly increased serum TC, TG and LDL-C and a decreased HDL-C in cases as compared to controls. Normal human pregnancy results in physiologic hyperlipidemia involving a gestational rise in blood triglyceride (TG) and cholesterol. There is a marked rise in serum TG in normal pregnancy as compared to nonpregnant women, which may be as high as two to three folds in the third trimester [10].

The principle modulator of this hypertriglyceridemia is hyperoestrogenemia in pregnancy that induces hepatic biosynthesis of TGs [11]. The anabolic phase of early pregnancy encourages lipogenesis and fat storage in preparation for rapid fetal growth in late pregnancy [12]. Lipolysis is increased as a result of insulin resistance, leading to increased flux of fatty acids to the liver promoting the synthesis of very low- density lipoproteins and increased triglyceride (TG) concentrations [13].

During pregnancy, there is an increase in the hepatic lipase activity and decrease in lipoprotein lipase activity[14]. Hepatic lipase is responsible for the increased synthesis of the TGs at the hepatic level, whereas the decreased activity of lipoprotein lipase is responsible for the decreased catabolism at the adipose tissue level, the net effect of which will be an increase in circulating TGs [15].

Because of a decrease in the activity of lipoprotein lipase, VLDL remains in the plasma for longer and leads to the accumulation of LDL. An increase in LDL is associated with the development of atherosclerosis16. Women with preeclampsia display additional alterations in blood lipids reflecting disordered lipid and lipoprotein metabolism. Serum TG levels rose much more in pre- eclampsia as reported by other studies [5, 17] and as seen in our study also. Increased TG, found in pre-eclampsia is likely to be deposited in predisposed vessels, such as the uterine spiral arteries and contributes to the endothelial dysfunction [14].

There was a significant rise in TC levels in pre-eclampsia as compared to normal pregnancy in our study, which was similar to other reports [15, 16] however other studies reported no alteration in TC levels [8, 13]. A significant rise in the LDL-C levels was seen in pre-eclampsia as compared to controls in our study and also by other workers [6,19]. In our study there was significant fall in HDL-C in pre-eclampsia cases as compared to controls. Low HDL-C in pre- eclampsia is due to insulin resistance [17]. According to Pirzado et al, there is a direct correlation between adipose tissue lipoprotein lipase activity and plasma HDL cholesterol [18]. This direct correlation may be responsible for low levels of HDLcholesterol. Thus, dyslipidemia mediated activation of the endothelial cells and the placentally derived endothelial disturbing factors like lipid peroxides could be a possible cause in the pathogenesis of PIH [20]. This association may be

significant in understanding the pathological process of pre- eclampsia and may help in developing strategies for prevention and early diagnosis of pre-eclampsia. Thus, estimation of lipid profile may have a predictive role in the assessment of the extent of endothelial damage in pre- eclampsia and may help patient by preventing or foreseeing the effects of complications in pre-eclampsia.

Conclusion:

Hence early detection of these parameters is going to aid in better management of pre- eclampsia cases which is important to improve the maternal and fetal outcome in pre- eclampsia.

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