



STUDY OF LIPID PROFILE, LIPID PEROXIDATION AND VITAMIN E IN PREGNANCY INDUCED HYPERTENSION

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ABSTRACT:

Pregnancy induced hypertension (PIH) is a common medical complication of pregnancy with a high incidence. The study comprised of 30 normal and 30 PIH cases in their third trimester of pregnancy and the following estimations were done: Serum Malondialdehyde level (MDA), Vitamin E, triglycerides (TG), total cholesterol (TC), HDL-cholesterol (HDL-C) and LDL-C. The PIH cases had significant rise in both systolic and diastolic blood pressure (BP) ($P < 0.0001$). There was a significant rise in the fasting triglycerides, total cholesterol and LDL-C levels in PIH ($P < 0.0001$). MDA was twice in the cases and Vitamin E was half the levels that of controls ($P < 0.0001$). Early detection of these parameters is going to aid in better management of PIH cases.

Key words : lipid profile vitamin E malondialdehyde (MDA) oxidative stress pregnancy-induced hypertension (PIH)

INTRODUCTION

Lipid abnormalities in hypertension are well documented since hypertension is a predisposing risk factor for coronary heart disease. It is important to understand the pattern of lipid profile and associated abnormalities.¹ Studies on lipoprotein abnormalities are now extended to oxidative stress in pregnancy induced hypertension. The role of oxidative stress are important source of vascular injury, in the genesis of this increased risk has also been studied extensively as many antihypertensive drugs have demonstrated antioxidant effect.⁵ Oxidative stress arises due to deficiency in the level of antioxidative enzymes such as superoxide dismutase and glutathione peroxidase which are defensive agents against production of superoxides or reactive oxygen species. These enzymes help to scavenge the oxygen free radical. In the deficiency of these enzymes the reactive oxygen species freely oxidizes the membrane lipids and the lipids of lipoproteins, leading to damage of endothelial cell membrane and production of oxidized LDL which are implicated in atherogenesis.

As a part of lipid peroxidation status the malondialdehyde (MDA) assay is the most generally used to assess the role of oxidative stress. MDA is one of the several products produced during free radical induced oxidation of polyunsaturated fatty acids of membrane.

Hypertensive disorders are common medical complications of pregnancy with a reported incidence of about 10% of first pregnancies and 20-26% of women with chronic hypertension. The association of alteration in serum lipid profile in essential hypertension is well documented. Hormonal imbalance leading to altered lipid profile in serum is attributed to be the prime factor

etiopathogenesis of pregnancy-induced hypertension (PIH). PIH includes a group of hypertensive disorders developed due to the gravid state. Lipid peroxidation occurs at low levels in all cells and tissues. In health, oxidation by free radicals and neutralization by antioxidants remain in balance. When the reactive oxygen species (ROS) are in abundance, oxidative stress occurs which is thought to be the causative factor in PIH.³

For the aforesaid reasons, the present study was conducted to study the lipid profile, lipid peroxidation product, malondialdehyde (MDA) and lipophilic antioxidant, vitamin E in cases of pregnancy and healthy controls.

MATERIALS AND METHODS

The total number of subjects studied were thirty women from normal condition and thirty from pregnant condition, Wardha district in Maharashtra State of India. Blood pressure of each case was measured with a mercury sphygmomanometer and stethoscope. To estimate serum cholesterol and high density lipoprotein cholesterol, 10ml of blood was withdrawn from antecubital vein of each subject under aseptic condition. Serum was separated and used for estimation of serum cholesterol and serum high density lipoprotein cholesterol. Total cholesterol was estimated by CHOD-PAP method, high density lipoprotein was estimated by phosphotungstic acid method. Serum MDA level and vitamin 'E' measured by analyzer. The results were tabulated (Table 1). The mean of the difference of values obtained at these groups were calculated and statistically analyzed by utilizing the student 't' test.

The diagnosis of pregnancy was based on the definition of American College of Obstetrics and Gynecologists.⁴

Inclusion criteria for cases: Primigravida with diagnosed pregnancy according to the definition of American College of Obstetrics and Gynecologists with an age ranging from 18-35 years. Inclusion criteria for controls: Primigravida with normal BP, 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 >24 weeks).

RESULTS

, Vitamin E was half the levels of that of healthy controls ($P < 0.0001$)

(Table I) : General characters of Control & Cases

Parameters	Control (n = 30)	Cases (n = 30)	P
Systolic BP (mm Hg)	109.3 \pm 9.3	142 \pm 4.2	<0.001
Diastolic BP (mm Hg)	80.2 \pm 4.0	94.8 \pm 12.37	< 0.001
Total Cholesterol (mg/dl)	182.1 \pm 9.2	297.3 \pm 17.2	< 0.001
Triglycerides (mg/dl)	87.7 \pm 12.3	255.00 \pm 31.6	< 0.001
LDLC (mg/dl)	91.2 \pm 11.6	200.7 \pm 15.2	< 0.001
HDLC (mg/dl)	65.2 \pm 4.7	42 \pm 2.7	< 0.001
VLDL (mg/dl)	15.2 \pm 2.1	45.7 \pm 5.2	< 0.001
Malondialdehyde (nmol/ml)	3.12 \pm 0.4	7.04 \pm 0.3	< 0.001
Vitamin 'E'	6.32 \pm 0.2	4.26 \pm 0.1	<0.001

In our study the maternal age was significantly high ($P = 0.005$) in the cases as compared to controls so we tried to correlate the maternal age with systolic and diastolic BP. There was no consistent significant correlation. The level of rises of serum lipids did not significantly correlate with the rise or fall in MDA in both the cases and controls. There was a rise in MDA levels with the rise in systolic BP (P value not significant). In the pregnancy cases with the rise in diastolic BP there was a negative correlation with MDA ($P < 0.05$).

DISCUSSION

There is a marked rise in serum TG in normal pregnancy as compared to non-pregnant women, which may be as high as two to three folds in the third trimester⁶. The principle modulator of this hypertriglyceridemia is hyperoestrogenemia in pregnancy that induces hepatic biosynthesis of TGs¹⁰. Serum TG levels much more in pregnancy as reported by other studies^{11, 12} and as seen in our study also. Increased TG levels results in endothelial cell dysfunction and in pregnancy gets deposited in predisposed vessels¹³, causes generation of small dense LDL¹⁴.

There was a significant rise in TC levels in pregnancy as compared to normal condition in our study. In our study there was

The pregnancy cases had significant rise in both systolic and diastolic blood pressure (BP) as compared to healthy pregnant subjects ($P < 0.0001$). There was a % significant rise in the fasting triglycerides, total cholesterol and LDL-C levels in PIH ($P < 0.0001$). The lipid peroxidation product, MDA was almost twice in the cases as compared to the controls ($P = < 0.0001$). The antioxidant

significant fall in HDL-C in pregnancy condition. Estrogen is responsible for induction of TG and HDL-C but in PIH there is a fall in estrogen levels as compared to normal condition. Hence the low HDL-C in pregnancy is due to hypoestrogenemia. A significant rise in the LDL-C levels was seen in pregnancy as compared to controls in our study and also by other workers^{15, 16}. It is observed that MDA-LDL and oxidized LDL increase in pregnancy. This enhanced lipid peroxidation is involved in the foam cell formation of decidua in pathogenesis of pregnancy.

We observed that MDA levels increased in PIH cases as compared to normal condition. There was a decrease in antioxidant levels that is Vitamin E as a response to oppose the oxidative stress.

The present study clearly indicates that significant rise in MDA with decreased vitamin E levels and altered serum lipid levels are possible causative factors for the pathogenesis of PIH. Hence early detection of these parameters is going to aid in better management of pregnancy cases which is important to improve the maternal fetal status in pregnancy.

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