ORIGINAL ARTICLE

Assessment of Lipid Profile in Pre-Eclampsia

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ABSTRACT

Objective: Assessments of lipid profile in preeclampsia.
Design: A cross-sectional analytical study.
Place and duration of study: Department of Physiology, BMSI, JPMC Karachi in collaboration with Department of Gynecology and obstetrics JPMC Karachi.
Subjects and methods: study included 90 subjects distributed into three groups of 30 subjects each. 30 were apparently healthy subjects. 30 mild preeclamptic and 30 severely preeclamptic. Clinical detailed were collected. Fasting blood samples were drawn by adopting aseptic methods and serum was analyzed for lipid profile.
Results: Among 90 screened subjects Serum triglycerides in 30 normal subjects was (176.76 mg/d1), in 30 mild preeclamptic subjects showing (235.53mg/ldl), was highly significant as compare to normal. However 30 severe preeclamptic subjects show serum triglycerides 242.mg/dl as compare to normal. The difference in both groups were statistically highly significant as compare to normal.
Conclusion: Concluded that lipid profile levels were elevated with preeclampsia which suggests that elevated circulating levels may be involved in pathogenesis of uteroplacental vascular disease associated with preeclampsia, which may be risk marker in pregnant women.
Key words: Lipids, Preeclampsia.

INTRODUCTION

Preeclampsia is defined as development of hypertension and proteinuria (>300 mg urinary protein in 24h), after 20th week gestation. Hypertension is defined as a blood pressure greater than 140/90 mmHg or rise in blood pressure of 30/15 mmHg from the baseline level confirmed by two measurements at 6 hours apart[1]. Preeclampsia is a human pregnancy specific disorder that adversely affects the mother (by vascular dysfunction) and the fetus (by intrauterine growth restriction). Incidence of preeclampsia is between 3% and 10% of pregnancies and there is no evidence that this has changed appreciably. Preeclampsia is characterized by vasospasm, increased peripheral vascular resistance and reduced organ perfusion[2]. It has been proposed that blood pressure decreases from the first trimester to the second trimester and rises again in third trimester for healthy pregnant women. For women who developed gestational hypertension or preeclampsia, blood pressure is stable during first half of pregnancy and then continuously increased until delivery[3].

Risk of preeclampsia is increased in women with a previous history of preeclampsia, in those with

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antiphospholipid antibodies, preexisting diabetes mellitus (DM), twin pregnancy, nulliparity, family history, raised body mass index (BMI) before pregnancy or maternal age below 20 years or above 40 years, risk is also increased with an interval of 10 years or more since a previous pregnancy, autoimmune disease, renal disease and chronic hypertension[4]. Preeclampsia is divided in mild and severe forms, depending upon the severity of hypertension, the amount of proteinuria and the degree to which other organ systems are affected[5]. Despite being one of the leading cause of maternal death, the mechanism responsible for the pathogenesis of preeclampsia is still unclear. Vasospasm is the basic event in PIH. It is possible that vasospasm itself exerts a damaging effect on the blood vessels. Alternating segmental arteriolar spasm and dilatation causes endothelial damage. Angiotensin II causes endothelial contration. These changes lead to interendothelial cell leakage through which platelets, fibrinogen and other blood constituents get deposited sub-endothelially. These vascular changes, along with ischemia of surrounding tissues, lead to hemorrhage, necrosis, and other endorgen changes seen in severe PIH[6]. Lipid peroxidation is a process normally occurring at low levels in all cells and tissues. It involves oxidative conversion of unsaturated fatty acids to primary products known as lipid hydroperoxides and a variety of secondary metabolites. An important mechanism

for lipid peroxidation detectable in all cells is commonly referred to as the free radical process of lipid peroxidation[7]. Any imbalance between prooxidant and antioxidant forces in which former dominates may be broadly defined as oxidative stress of which lipid peroxidation is one important manifestation. Although lipid peroxidation affects many cellular components, the primary reaction sites involve membrane associated polyunsaturated fatty acids and protein thiols[8]. Oxidative stress is a key factor in disease process. In the placenta the synthesis of reactive oxygen species is increased and activities of antioxidant enzymes are reduced. There is also evidence of lipid peroxidation in the maternal blood and placenta[9]. The endothelium seems to be the target organ for preeclampsia process. Altered endothelial cell function in preeclampsia includes activation of coagulation cascade, increased membrane permeability, enhanced response to pressor agents and increased vasoconstriction, which all contribute to reduced perfusion of affected organs[10].

Endothelial cells can be activated in several different ways including free fatty acids, lipoproteins, oxidized lipoproteins or lipid peroxides, fibronectin degradation products and deported syncytotrophoblastic microvillus fragments[11].

PURPOSE OF STUDY

1. To assess the lipid profile in preeclampsia
2. To Explore the possibility of lipid profile being a marker of severity of preeclampsia

MATERIALS AND METHODS

This study was carried out in department of Physiology BMSI, JPMC Karachi with collaboration of department of Gynecology and Obstetrics, JPMC Karachi. This study was performed on 90, pregnant women of age ranging between 16-32 years and having gestational age between 26 to 34 weeks.

Group A = Normal healthy pregnant women as control.

Group B = Mild preeclamptic women
Group C = Severe preeclamptic women

Clinical details were recorded. Blood samples were collected by adopting aseptic methods and serum was analyzed for lipid profile.

RESULTS

Table 1 shows Comparison of Mean (SEM) of age, height and weight between control, mild and sever preeclamptic women show that Mean age of mild preeclamptic group (19.9 ± 0.55) and severe preeclamptic group (21.63 ± 0.75) was significantly low as compared to control (23.96± 0.82). Non significant change was observed in weight and height in control and pre-eclamptic group.

Table 2 shows the Mean value of systolic and diastolic blood pressure were significantly high in mild preeclamptic (149.33 ± 1.43) and (104.16 ± 0.93) in severe preeclamptic women (183.16 ± 0.93) and (121.83 ± 1.40) as compared to control group (112.83 + 2.41) and (76.00 ± 1.48).

Table 3 shows the mean value of serum triglyceride was highly significant in mild preeclamptic (235.53 ± 11.54) and severe preeclamptic group (242.00± 13.56) as compared to control group (176.76 ± 8.05). There was also statistically significant difference in value of serum cholesterol in severe preeclamptic (229.3 ± 8.13) as compared to control (198.53 ± 7.21) non significant difference was found between values of mild preeclamptic as compared to control group.

Table 4 shows Non significant difference between values of HDL cholesterol (28.13 ± 1.37) and LDLC (137.50 ± 6.59) between mild preeclamptic groups when compared to control group. While significant difference was observed in HDL-C (27.33±.97) and LDL-C (153.67 ± 6.10) between severe preeclamptic groups as compared to control. Highly significant difference was found in value of VLDL cholesterol between mild preeclamptic group (47.10 ± 2.3) as well severe preeclamptic group (49.71 ± 3.05) as compared to control (35.28 ± 1.6).

Table 1: comparison of maternal age height, weight in normal pregnant women (control) and preeclamptic groups (All the values are expressed in Mean ± SEM)

Variable	Group A Normal Pregnant Women (Control) n=30	Group B Mild Preeclamptic Women n=30	Group C Severe Preeclamptic Women n=30
Age (years)	23.96±0.82	19.90±0.55	21.63±0.75
Weight (Kg)	58.00±84	60.60±1.20	60.90±1.23
Height (m)	1.54±0.01	1.55±0.01	1.54±0.01

N = Number of subjects
** = <0.05 when compared to control
*** = P<0.001 when compared to control

Table 2: comparison of systolic and diastolic blood pressure in control group and preeclamptic group
(All the values are expressed in Mean ± SEM)

Variable	Group A Normal Pregnant Women (Control) n=30	Group B Mild Preeclamptic Women n=30	Group C Severe Preeclamptic Women n=30
Systolic blood pressure (mmHg)	112.83±2.41	149.33±1.43	183.16±2.49
Diatolic blood pressure (mmHg)	76.00±48	104.16±0.93	121.83±1.40

N = Number of subjects
*** = P<0.001 when compared to control

Table 3: comparison of fasting triglycerides and total cholesterol in control group and preeclamptic group
(All the values are expressed in Mean ± SEM)

Variable	Group A Normal Pregnant Women (Control) n=30	Group B Mild Preeclamptic Women n=30	Group C Severe Preeclamptic Women n=30
Triglycerides (md/dl)	176.76±8.05	235.53±11.54	242.00±13.56
Total Cholesterol (mg/dl)	198.53±7.21	212.73±7.12	229.30±8.13

N = Number of subjects
** = P<0.01 when compared to control
*** = P<0.001 when compared to control

Table 4: comparison of fasting high density lipoprotein cholesterol, low density lipoprotein cholesterol level in control group and preeclamptic group (All the values are expressed in Mean ± SEM)

Variable	Group A Normal Pregnant Women (Control) n=30	Group B Mild Preeclamptic Women n=30	Group C Severe Preeclamptic Women n=30
High density lipoprotein cholesterol (mg/dl)	31.30±1.50	28.13±1.37	27.33±0.97
Low density lipoprotein cholesterol (mg/dl)	131.86±7.18	137.50±6.59	163.07±6.10
Very low density lipoprotein cholesterol (mg/dl)	35.28±1.61	47.10±2.30	49.71±3.05

N = Number of subjects
** = P<0.01 when compared to control
*** = P<0.001 when compared to control

DISCUSSION

Preeclampsia is still one of the leading cause of maternal and fetal morbidity and mortality. Despite active research for many decades, the etiology of this disorders is Unknown. Recent evidence suggests that there may be several underlying causes or predisposing factors leading to endothelial dysfunction and causing signs of hypertension, proteinuria and oedema. Many hypotheses have been offered for the pathogenesis of disease and include prostacyclin – thromboxane imbalance, endothelial dysfunction, immunogenetic and absolute or relative placental ischemia[12]. The current study was undertaken to assess the lipid profile in preeclampsia and explore possibility of lipids being as a marker of severity of preeclampsia. There was significant difference in maternal age between normal pregnant and mild and severe preeclamptic women, though no difference was found in weight and height in normal pregnant and preeclamptic groups. Similar

findings regarding weight and height were observed by hannele etal[13]. Normal human pregnancy results in pronounced physiological hyperlipidemia involving rise in blood triglycerides and cholesterol. Women with preeclampsia display additional alteration in blood lipids, reflecting a disordered lipid and lipoprotein metabolism[14]. In present study, the patients of mild preeclamptic group has nosignificant difference in total Cholesterol as compare to control subject. Although mean level was slightly higher in mild preeclamptic, while significant difference was observed in Cholesterol level in severe preeclamptic women as compare to normal pregnant women. This is in agreement with study conducted by Hubel etal[15]. In this study significant difference in triglyceride levels in preeclamptic groups as compare to control group were found. These findings are in agreement with work of power etal[16]. In this study HCL- Cholesterol was lower in mean values in mild and severe

preeclamptic as compare to normal pregnant women. This is partly in agreement with study conducted by kaaja[17] non-significant difference in low density lipoprotein Cholesterol (LDL-C) was found in mild preeclamptic group as compared to control group, while severe preeclamptic patients has significant high levels of LDL- Cholesterol as compare to control group. These results are in agreement with study of sattar[18]. Hubel and his colleagues reported that very low density lipoprotein Cholesterol is increased in preeclamptic patients. Significantly higher values of VLDL Cholesterol were found in preeclamptic groups as compare to normal pregnant women.

CONCLUSION

We have observed that lipid profile levels are strongly associated with preeclampsia. This suggests that elevated lipids may be involved in pathogenesis with preeclampsia and risk marker of this in women.

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