

EVALUATION OF SERUM LEVELS OF FASTING LIPID PROFILE IN PRE-ECLAMPTIC WOMEN

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Abstract:

Objectives: This study was designed to evaluate the role of lipid profile alteration in the development of pre-eclampsia.

Methodology: A total of 35 pre-eclamptic subjects (20-40weeks gestation age) aged between 18-40years participated in this study. For comparative assessment 34 aged matched healthy pregnant women with same gestational age was used as control. Serum lipid profile (total cholesterol, triglyceride, HDL-C, LDL-C, and VLDL-C) of the subjects respectively were monitored. **Result:** The serum TG concentration increased significantly (0.95 ± 0.53 to 0.77 ± 0.33 $p < 0.05$). **Conclusion:** Lipid metabolism plays a vital role in the pathophysiology of pre-eclampsia. Increased TG levels and triglyceride clearance and high blood pressure are associated with the development of pre-eclampsia.

Keywords: Pre-eclampsia(PE), High Density Lipoprotein-Cholesterol (HDL-C), Low Density Lipoprotein cholesterol (LDL-C), Very Low Density Lipoprotein-Cholesterol (VLDL-C), Triglycerides (TG), Total Cholesterol (TC).

INTRODUCTION

Complications of pre-eclampsia remain a major cause of maternal morbidity and mortality especially in developing countries (1). Pre-eclampsia is characterized by hypertension, proteinuria, and oedema (2). Pre-eclampsia most commonly occurs during the last trimester of pregnancy. The risk of developing pre-eclampsia appears to be greater in women who have family history of essential hypertension and there may also be a relationship between risk of pre-eclampsia and the metabolic syndrome (2). Pre-eclampsia is a medical condition which affects virtually all maternal organ systems (5). Despite considerable research, the cause or causes of pre-eclampsia remain unclear and there are no clinically useful screening tests to identify women in whom it will develop (4). Early pregnancy dyslipidaemia is associated with an increased risk of PE (5). Women with a history of PE have significant differences in lipid parameters and an increased susceptibility to lipoprotein oxidation when compared with women who had normal pregnancy. Disorders of lipoprotein metabolism are reported to be a major cause of hypertension and proteinuria in PE (6), (2). This present study was designed to ascertain/investigate the alteration in lipid profile in normal and pre-eclamptic women.

MATERIALS AND METHODS

The study was approved by the Nnamdi Azikiwe University Teaching Hospital(NAUTH) ethics committee. A total of 69 participants were selected from the ante-natal clinic and pre-natal wards (obstetrics and gynaecology) of NAUTH Nnewi, Anambra State Nigeria. These subjects were divided into two groups

(a)35 pre-eclamptic subjects (pre-eclampsia was defined as the occurrence after 20 weeks of gestation, a diastolic Bp> 90mmHg systolic Bp> 140mmHg more than two occasions at least 4hours apart, and proteinuria of 0.3g/l or more in a 24hour urine collection period) (mean age 28.29±5.3)

(b) 34 Normotensive pregnant women as controls of matching age (mean age 26.29±4.9). None was known to have chronic hypertension or any renal /other metabolic disease.

Fasting blood specimens were collected from all control and pre-eclamptic subjects. Blood was always collected before onset of labor. Serum was separated for analysis.

Serum total cholesterol (mmol/l): Serum total cholesterol was determined after enzymatic hydrolysis and oxidation, indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine in the presence of phenol and peroxidase. (7)

Triglycerides (mmol/l): Triglycerides were determined after enzymatic hydrolysis with lipases. The indicator was a quinoneimine formed from hydrogen peroxide, 4-amino-phenazone and 4-chlorophenol under the catalytic influence of preoxidase.

HDL-cholesterol (mmol/l): Cholesterol in chylomicron, very low density lipoproteins, and low density lipoproteins are precipitated by adding phosphotungstic acid and magnesium ions to form the sample. Centrifugation leaves only the HDL in the supernatant while the cholesterol content was determined enzymatically. (7) Body Mass Index was calculated by dividing body weight (kg) by square of height (meters).

LDL-cholesterol (mmol/l) and VLDL-cholesterol (mmol/l): were determined using friedewald’s and colleagues formulae (1972).

$LDL-C=TC-(VLDL+HDL)$ and $VLDL-C=TG/2$.

Statistical Analysis: Mean and standard deviation were calculated for both pre-eclaptic and control groups. Level of significance between control and pre-eclamptic were analyzed using student’s t test. Data are presented as mean± standard deviation. Pvalue <0.01 was considered statically significant.

RESULTS.

TABLE 1: ANTHROPOMETRIC AND BLOOD PRESSURE DATA (MEAN±SD) OF PRE-ECLAMPTIC AND NORMOTENSIVE SUBJECTS.

PARAMETERS	PRE-ECLAMPSIA n=35	CONTROL n=34	t-TEST	P-Value
AGE(Yr)	28.29±5.8	26.29±4.9	1.54	0.127
SBP(mmHg)	158.3±3.8	117.94±6.4	31.86	<0.001*
DBP(mmHg)	106.0±8.1	80.29±7.4	13.75	<0.001*
BMI(Kgm ²)	27.23±3.9	26.62±3.8	0.66	0.514
PULSE PRESSURE(mmHg)	52.29±8.77	37.65±9.39	6.69	<0.001*
MEAN ARTERIAL PRESSURE(mmHg)	123.43±5.63	92.84±5.51	22.79	<0.001*

*P < 0.01 significant as compared to normal and control.

TABLE 2: LIPID PROFILE DATA(MEAN±SD) OF PRE-ECLAMPTIC AND NORMOTENSIVE SUBJECTS.

PARAMETERS	PREECLAMPSIA n=35	CONTROL n=34	t-Test	P-Value
TG (mmol/l)	0.95±0.53	0.77±0.33	1.64	0.03*
TC (mmol/l)	4.81±1.44	5.04±1.35	-0.71	0.48
HDL-C (mmol/l)	1.48 ±0.42	1.56± 0.33	-0.80	0.26
LDL-C (mmol/l)	2.42± 1.27	2.68± 1.20	-0.86	0.64
VLDL-C (mmol/l)	0.40± 0.21	0.35± 0.15	1.25	0.11

* P< 0.05 significant as compared to normal and control.

Mean Triglyceride (0.95±0.53 vs 0.77±0.33) levels are significantly higher in the pre-eclamptic group compare to the control group (P< 0.05) as shown in table 2 while mean HDL-C (1.48 ±0.42 vs 1.56± 0.33), Total cholesterol (4.81±1.44 vs 5.04±1.35), LDL-C (2.42± 1.27 vs 2.68± 1.20) and VLDL-C (0.40± 0.21 vs 0.35± 0.15) levels were not statistically different between pre-eclamptic and normal subjects as shown in table 2.

DISCUSSION.

In this study we investigated the role and relationship between lipid profile and the incidence of pre-eclampsia. There was a positive correlation between pre-eclampsia and lipid parameters as shown in table 2.

We observed higher levels of triglycerides during pre-eclampsia which provide evidence of abnormal lipid metabolism. Pre-eclampsia is characteristically associated with hypertriglyceridemia. Higher levels of triglycerides with high blood pressure have been observed in our study as shown in table 1 and 2. These types of higher results also have been reported in other studies in pre-eclamptic women (5, 6, 8-11).

During the course of normal pregnancy, plasma triglyceride and cholesterol concentrations rise and as pregnancy progresses both become normal. Hormonal variations during pregnancy affect lipid metabolism. The endogenous female sex hormones have significant effect on serum lipids (11). During pregnancy there is an increase in the hepatic lipase activity and decrease in lipoprotein lipase activity. Hepatic lipase is responsible for the increased synthesis of the triglycerides 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 triglycerides and the second step uptake of the remnant chylomicrons by the liver is delayed so it leads to accumulation of triglycerides in serum as observed during the present study shown in table 2.

Another hypothesis is that hypertriglyceridemia is probably as a result of the competition between chylomicrons and VLDL-C for lipoprotein lipase. Classically chylomicron clearance occurs in two sequential steps (1) triglycerides hydrolysis by lipoprotein lipase (2) uptake of the remnant by the liver. Delay in the second step leads to accumulation of remnants in plasma and is generally thought to represent the atherogenic risk of hypertriglyceridemia. The conclusion of another study also showed that there exists a consistent positive relationship between elevated maternal TG and the risk of pre-eclampsia. (12)

Summarizingly, the findings reported in this article implies that women who developed pre-eclampsia had altered lipid profile due to abnormal lipid metabolism. Elevated triglyceride levels, delayed triglycerides clearance and high blood pressure are the reasons for the development of pre-eclampsia. This association maybe significant in understanding the pathophysiology process of pre-eclampsia and may help in developing strategies for early detection, better prevention, treatment and management.

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