

Lipid Profile Pattern of Pre-Eclamptic and Eclamptic Patients Attending University of Maiduguri Teaching Hospital

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Received Nov 27, 2013; Accepted Apr 24, 2014; Published Apr 30, 2014

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Abstract

Background: Pre-eclampsia and Eclampsia are the most common complications of pregnancy. They are said to be the most common cause of fetal and maternal morbidity and mortality worldwide. These complications are the consequences of the effects of pregnancy on metabolic processes involving lipid and lipoproteins among others

Objective: This study aimed to evaluate the pattern of lipid profile alterations in pregnant women with preeclampsia and eclampsia.

Method: This is a cross-sectional study in which 100 subjects were recruited; 40 pre-eclamptic, 20 eclamptic and 40 normotensive (Control group) pregnant women. Fasting serum lipid profile (Total cholesterol, LDL cholesterol, HDL - cholesterol and triglyceride) were determined.

Result: Mean serum TG concentrations in pre-eclampsia (2.4 ± 0.9 vs 1.9 ± 0.6) and in eclampsia (2.8 ± 1.2 vs 1.9 ± 0.6) were significantly high when compared to normal control ($p < 0.05$). Mean Serum TC concentration (4.9 ± 1.3 vs 6.0 ± 2.1) and HDL-Cholesterol concentration (1.8 ± 0.4 vs 3.0 ± 2.2) were significantly low in women with eclampsia when compared with control group ($p < 0.05$).

Conclusion: The result of the study showed that, although increased plasma lipid (hyperlipidaemia) is associated with normal pregnancy, this is exaggerated in both preeclampsia and eclampsia. The increase is also associated with severity of the disease.

Keywords: Lipid; Pre-Eclampsia; Eclampsia; Hospital

Introduction

Pre-eclampsia is characterized by hypertension, proteinuria and edema. It usually occurs in the later part of the second trimester and in the third trimester, but can occur in the early part of pregnancy [1,2]. Eclampsia, in addition to features of preeclampsia, is characterized by epileptiform convulsions [3]. It is usually a sequel to preeclampsia in untreated cases [4]. Pre-eclampsia and eclampsia are the most common complications of pregnancy [1] and are said to be the most common cause of fetal and maternal morbidity and mortality worldwide [5-7]. A study carried out in 2006 [8], among pregnant women study showed that the direct obstetric causes of maternal death accounted for 79.4% and eclampsia accounted for 31.9%. Development of pre-eclampsia/eclampsia is greater in women with family history of essential hypertension and currently metabolic syndrome has also been implicated [3,7].

Pregnancy is known to affect biochemical metabolic processes involved in lipid and lipoprotein metabolism among others [9]. These metabolic changes are likely to have evolved to meet the metabolic demands of the growing fetus. However, such metabolic changes particularly that of lipid and lipoprotein are found to be exaggerated in pre-eclampsia [9], and this may contribute to some of the

complications of pre-eclampsia/eclampsia, such as intrauterine growth restriction, prematurity and fetal death.

Since alterations in lipid concentrations is one of the characteristic features of pre-eclampsia [10,11] and because preeclampsia and eclampsia are very common and are associated with increased maternal mortality in pregnancy and puerperium [12], it is pertinent to consider the importance of timely laboratory evaluation of the lipid profile pattern among pregnant women complicated pre-eclampsia and eclampsia in University of Maiduguri Teaching Hospital, Maiduguri.

There is scanty data on the subject matter in this environment. The result of this investigation will serve as baseline information for the society and could help in developing strategies for the prevention or early detection of dyslipidemia in preeclampsia and eclampsia. The overall objective of this study therefore is to assess the lipid profile pattern among patients with pre-eclampsia and eclampsia. Specific objectives are:

To estimate serum total cholesterol, triglyceride, high density lipoprotein and low density lipoprotein cholesterol in pre-eclamptic, eclamptic and normal pregnant women.

To compare the results among the three groups.

Material and Method

Ethical approval was obtained from the ethical committee of the University of Maiduguri Teaching Hospital (UMTH) in accordance with the Helsinki declaration (World Medical Association, 1964).

The study is conducted in UMTH, Maiduguri, Borno State, Nigeria. Hundred (100) subjects were involved in the study: 40 pre-eclamptic and 20 eclamptic patients while 40 normotensive pregnant women were recruited as control. All subjects attended antenatal clinic in UMTH, Maiduguri.

All participant signed informed consent form and answers to the questionnaire were obtained from the selected participants. The purpose of the study was explained to the participants and they were also ensured of the confidentiality of any information obtained from them. They were also told that the participation is voluntary and that they could withdraw at any time.

Inclusion Criteria: Patients with ages between 15 and 45 years, and diagnosed to have pre-eclampsia or eclampsia in the department of Obstetrics and Gynecology of the hospital.

Exclusion Criteria: Patients with history of diabetes mellitus, renal disease, liver disorder, pre-existing hypertension, epilepsy and also those on drugs that can affect lipid metabolism were excluded from the study. Also excluded are women with maternal or fetal abnormal pregnancy (except pre-eclampsia and eclampsia) and subjects who decline to give consent.

Methodology

Five milliliter (5 ml) of fasting blood specimen was collected from each subject for biochemical analysis. The blood was collected aseptically from the antecubital vein using sterile disposable 5 ml syringe and transferred into an appropriately labeled plain container and allowed to clot at room temperature. The sample was centrifuged at 4000 revolution per minute (rpm) for 10 minutes and serum separated into new appropriately labeled sample container and stored frozen until the time for analysis.

Estimation of serum lipid parameters: Total Cholesterol (TC), Triglycerides (TG), Low Density Lipoprotein (LDL), High Density Lipoprotein (HDL)

Serum cholesterol level was determined by enzymatic reactions as described by Mieattini [13]. Serum triglyceride level was determined by enzymatic glycerol phosphate oxidase/peroxidase method as prescribed by Fossati [14]. Serum HDL cholesterol was estimated by enzyme oxidase/peroxidase, after lipid precipitation by phosphotungstate, as described by Grove [15]. Serum LDL cholesterol level was determined by Friederickson-Friedwald's [16] formula below;

$$\text{LDL cholesterol (mmol/L)} = \text{TC} - (\text{TG}/2.2 + \text{HDL})$$

In UMTH, the reference rage of Total Cholesterol (2.5-5.2) mmol/L, Triglycerides (<1.7) mmol/L, HDL-Cholesterol (1.1-1.5) mmol/L, and LDL Cholesterol (<3.9) mmol/L were used in the study Laboratory.

Data obtained was subjected to statistical analysis using statistical software package SPSS version 16.0. The Means, Standard Deviation and their variations were determined using student's t-test. The level of significance was set at a 'p' value of less than 0.05 (<0.05).

Results

Serum lipid profile was compared between cases and controls, and between subgroups of cases. The data are shown as mean ± standard deviation values. Mean TG in pre-eclampsia (2.4 ± 0.9) vs 1.9 ± 0.6) and in eclampsia (2.8 ± 1.2 vs 1.9 ± 0.6) levels were significantly higher when compared to controls (p < 0.05), as shown in Table 1. Mean TC (4.9 ± 1.3 vs 6.0 ± 2.1) and HDL (1.8±0.4 vs 3.0 ± 2.2) levels were significantly lower in women with eclampsia when compared with control groups (p < 0.05) as also shown in Table 1. Mean TC (5.2 ± 1.5 vs 6.0 ± 2.1), HDL (2.5 ± 0.6 vs 3.0 ± 2.2) and LDL (1.7 ± 1.4 vs 2.2 ± 1.3) in preeclampsia and LDL (1.9±1.3 vs 2.2 ± 1.3), in eclampsia were not statistically different when compared to control groups as also shown in Table 1. However, when pre-eclamptic and eclamptic subjects were compared only mean HDL was found to be significantly low (p < 0.05) as shown in Table 2.

Parameters (mmol/L)	Pre-Eclamptic	Eclamptic	Control
TC	5.2 ± 1.5	4.9 ± 1.3*	6.0 ± 2.1
TG	2.4 ± 0.9*	2.8 ± 1.2*	1.9 ± 0.6
HDL	2.5 ± 0.6	1.8 ± 0.4*	3.0 ± 2.2
LDL	1.7 ± 1.4	1.9 ± 1.3	2.2 ± 1.3

Values are presented as (mean + S.D in mmol/L). *Difference in mean is significant (p<0.05), TC: Total Cholesterol; TG: Triglyceride; HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein

Table 1: Lipid Profile Pattern of Pre-eclamptic, Eclamptic and control groups

Parameters (mmol/L)	Pre-Eclamptic	Eclamptic	P Value
TC	5.2 ± 1.5	4.9 ± 1.3	0.45
TG	2.4 ± 0.9	2.8 ± 1.2	0.184
HDL	2.5 ± 0.6	1.8 ± 0.4	0.000*
LDL	1.7 ± 1.4	1.9 ± 1.3	0.594

Values are presented as (mean ± S.D in mmol/L)
*Significantly different (p < 0.05), TC: Total Cholesterol; TG: Triglyceride; HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein

Table 2: Lipid Profile Pattern of Pre-eclamptic and Eclamptic groups

Discussion

Hyperlipidaemia develops with normal pregnancy [4]. Plasma TG and Total cholesterol concentrations rise particularly during the third trimester and as pregnancy progresses both become normal [5].

During pregnancy hormonal dynamism occur which contributes to maternal hypertriglyceridemia [17]. There is consistent increase in estrogen concentration throughout the gestation period. This hyperestrogenaemia causes increase endogenous production of VLDL-TGs [17] (estrogen decreases hepatic lipase activity, reduces adipose tissue LPL activity and inhibits hepatic lipase activity) [17,18]. These combined effects of enhanced liver production of VLDL, decrease removal of these particles from the circulation due to low LPL activity,

high Cholesterol Ester Transfer Protein (CETP) activity, and low hepatic lipase activity could be responsible for the accumulation of TGs-rich particles VLDL and LDL [17]. These particles contain sub-fractions of various sizes, densities and compositions which differ in their ability to initiate atherogenesis [17,18]. These proatherogenic lipid profiles characterized by increase TG levels and reduce HDL concentration depicted by this study. In this study, the lipid profile pattern of pre-eclamptic and eclamptic patients revealed significant changes in the lipid parameters as shown in table 1 and 2 and are similarly reported by another study [7].

There was a significant increase in triglycerides during pre-eclampsia and eclampsia and a significant decrease in HDL cholesterol during eclampsia. The result demonstrated an abnormal lipid metabolism in the patients and as reported by others on pre-eclampsia [6]. The study similarly showed a significant decrease in HDL - cholesterol as pre-eclampsia progresses to eclampsia, indicating worsening of lipid derangement with progression of disease (from pre-eclampsia to eclampsia).

The hypertriglyceridemia and decrease in HDL cholesterol observed in these patients might be a result of the peculiarity of the disease process in pre-eclampsia/eclampsia and not only due to the hyperestrogenaemia which is associated with pregnancy in general. This requires further investigation.

Conclusion

The study suggests that there is disturbed lipid profile as a result of abnormal lipid metabolism in preeclamptic and eclamptic women. This association may be worthy of note in the understanding of the pathological process of preeclampsia and eclampsia and may help in developing strategies for prevention and diagnosis of maternal and fetal complications.

Acknowledgement

The authors are grateful for the management of University of Maiduguri Teaching Hospital for providing the facilities to work in the department of Chemical Pathology and also permission to collect samples.

We are equally grateful to Dr. Jibrin Musa Dibal of the department of Agric Engineering and Dr. DS Mshelia of the Department of Chemical Pathology, University of Maiduguri for sparing their time in scrutinizing this manuscript.

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