www.iiste.org

Biochemical Changes in Plasma Lipids and Mineral Elements in Preeclampsia Patients in Ekiti State, Nigeria

OYEYEMI A. O* ASAOLU M. F OJO O.C Department of Biochemistry, Ekiti State University, Ekiti State, Nigeria

Abstract

This study has determined the concentrations of Total Cholesterol (TC), High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL), Triacylglyceride (TG), Very Low Density Lipoprotein (VLDL), Mineral Elements and some Electrolytes. Subjects used for this study were made up of 180 women with preeclampsia and 80 normotensive pregnant women, who attended antenatal and postnatal clinics of the Ekiti State University Teaching Hospital, Ado-Ekiti and Federal Medical center, Ido-Ekiti, Ekiti State, Nigeria. In addition, 80 control volunteers were also involved in this study. The control participants comprise of 40 normotensive and 40 hypertensive non-pregnant women randomly selected. They were also non-users of contraceptives therapy. Anthropometric measurements including age, parity, blood pressure, height and weight were recorded for the subjects. Plasma concentrations of each of the biomarkers were determined using test kits, based on established methods, in the subjects at 1st trimester, 2nd trimester and 3-6 days post-partum. The results in the hypertensive pregnant subjects were compared statistically (P<0.05), with the normotensive pregnant subjects. TC, LDLC, VLDLC and TG increase significantly in PIH subjects, but HDL showed significant decrease in the preeclampsia subjects ,as the pregnancy progresses to the 3rd trimester. At post -partum the lipid profile becomes normal again in preeclampsia. Subjects with preeclampsia showed more significant decrease in the levels of Ca, Mg, Na, and P elements. This research work pointed out clearly that, Plasma lipids and some mineral elements are implicated in Pregnancy Induced Hypertension. They may be likely etiological factors in the disease, and may also serve as indicators for early detection.

Keywords: Preeclampsia, Total Cholesterol (TC), High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL), Triacylglyceride (TG), Very Low Density Lipoprotein (VLDL), Mineral elements.

INTRODUCTION

Pregnancy refers to the period from conception to the expulsion of the foetus. This period constitutes in a woman's life a special condition that affects various physiologic and endocrinology systems as a result of adaptation of the mother to accommodate and support the foetus before and after parturition (Davey, 1988). Pregnancy is a natural state that sets up great changes throughout the whole body. Throughout gestation, the physiological changes occur in pregnant women and most of these changes subside quickly after delivery (Guice-Booth, 2005). Pregnancy can be complicated by at least two distinct types of hypertension. The first one is chronic hypertension, which is usually characterized by blood pressure greater than 140/90mmHg. The second type is Pregnancy Induced Hypertension (PIH), and also known as pre-eclampsia. Preeclampsia is a complication that results to the development of hypertension after about twenty (20) weeks of gestation in a woman who had previously been normotensive (with no pre-existing renal disease). A patient with preeclampsia will begin her pregnancy with a normal blood pressure but it will rise sometime in the third trimester in typical cases, but earlier in severe cases. A rise in the blood pressure by 30/20mmHg (e.g. from 90/50mmHg to 120/70rnmHg) over the course of the pregnancy is a diagnostic criterion, even though the final pressure may seem normal (Sibai *et al.*, 2007).

Preeclampsia is one of the major causes of maternal death throughout the world (Chesley, 1987). It has been suggested that one or more of the protective mechanisms are either deficient or fails to functions properly or even gets out of control.

The exact cause of preeclampsia is unknown, and currently there is no sure way to prevent the hypertension (Oyeyemi and Asaolu, 2015).

Lipids are molecular organic compounds composed largely of carbon and hydrogen that are essential for cell growth. Lipids are non soluble in water and combine with carbohydrates and proteins to form the majority of all plants and animal cells. Lipids are more commonly synonymous with the word 'fats' when speaking in terms of personal health and though all fats are not all lipids. Plasma lipids include Cholesterol and Triacylglyceride. Cholesterol is a naturally occurring substance in the body and its composed of lipids. It's a waxy substance produced by the liver and found in certain foods and needed to make vitamin D and some hormone, build cell walls and create bile salts that helps to digest fats. They are found in foods from animal source while vegetables, fruits and grains contain none. The liver produces about 1000mg of cholesterols per day and an individual probably consumes about 150 to 250mg in the foods eaten. Cholesterol is separated into two types, such as high density lipoprotein (HDL) and low density lipoprotein (LDL). In a lipid test, the lipoproteins are separated so that the levels of each can be measured. Lipid tests are part of preventive routines cases, as they

help determine whether there are significant risks of atherosclerosis, a hardening of the arteries that interferes with or interrupts blood flow. The lipoprotein levels are measured and dietary changes are usually in order when the total cholesterol approaches or rise above 200mg per decimeter in the blood (Schaefer *et al.*, 1995)

Cholesterol is transported through the blood stream in the form of large chylomicrons or lipoprotein, LDL transport cholesterol to peripheral tissues and regulates de novo cholesterol synthesis at these sites. HDL picks up cholesterol released in to the plasma from dying cells and from membranes undergoing turnovers. An acyl transferase in HDL esterifies these cholesterol which are then returned by HDL in the liver or to the tissues that uses cholesterol to synthesize steroid hormones (Ostos *et al*; 2002). LDL clogs the blood vessels keeping blood from flowing through the body the way it should. When LDL levels are high, cholesterol is deposited on the walls of the arteries and forms a hard substance called plague. Over time, the plague causes the arteries to become narrower, decreasing blood flow and causing a condition called atherosclerosis or hardening of the arteries. This affects the coronary arteries that supply blood to the muscles of the heart; it is called coronary artery diseases, which puts a person at risk of having heart attack. When atherosclerosis affects the blood levels that supply the brain blood, the condition is called cerebral vascular disease which puts the person at risk of having a stroke. It may also block flow to other vital organs including the kidney and intestines (Ostos *et al*; 2002).

Triglycerides are also a form of fats in the blood stream. People with high triglycerides levels often have high total cholesterol, a high cholesterol LDL, a low cholesterol HDL levels. Many individuals with heart disease also have high triglyceride levels. Several clinical studies have shown that people with above normal triglyceride levels (greater than or equal to 200mg/dl) have an increased risk of heart disease. Also people with diabetes or who are obese are also likely to have high triglycerides (Gotto, 1998).

Mineral elements such as Na, K, P, Mg, Fe and so on, are important part of the body fluids which perform great roles in the body system. Tests that measure the concentrations of mineral elements and electrolytes are needed for both the diagnosis and management of renal endocrine, acid-base, water balance and many other conditions. Their importance lies in part with the serious consequences that follow from the relatively small changes that diseases or abnormal conditions may cause. For example, the reference range of potassium is 3.6–5.0mmol/L. Potassium is first used as STAT (needed immediately) test because values below 3.0mmol/L are associated with arrhythmia (irregular heartbeat), tachycardia (rapid heartbeat) and cardiac arrest and above 6.0mmol/L are associated with bradycardia (slow heart beat) and heart failure. Sodium measurement are also very useful in differentiating the cause of an abnormal potassium results, conditions such as the overuse of diuretics (drugs that promote lower blood pressure) often result in low levels of sodium and potassium. On the other hand, cushing's disease and Addison's disease drive the sodium and potassium in opposing directions (Henry, 2001).

MATERIALS AND METHODS

Subjects

Subjects used for this study were made up of 180 hypertensive pregnant women and 80 normotensive pregnant women, who attended antenatal and postnatal clinics of the Ekiti State University Teaching Hospital, Ado-Ekiti and Federal Medical center, Ido-Ekiti, Ekiti State, Nigeria. In addition, 80 control volunteers were also involved in this study. The control participants comprise of both normotensive and hypertensive non-pregnant women randomly selected. They were also non-users of contraceptives therapy.

Recruitments of Subjects

The subjects used for this work were divided into four (4) groups.

Group 1

In this group, the subjects were 180 hypertensive pregnant women (HPW) with symptoms of Pregnancy Induced hypertension (pre-eclampsia), brought into Ekiti State Teaching Hospital, Ado – Ekiti and Federal Medical Centre, Ido –Ekiti for management and treatment. Eighty (80) of the subjects were in their 2nd trimesters while 40 were in their 3rd trimesters respectively. They were monitored up to three to six days after delivery.

Group 2

Subjects in this group were 80 in number. They were randomly selected and were normotensive pregnant women (NPW) in their 2^{nd} and 3^{rd} trimesters of pregnancy. They were attending the antenatal clinics of Ekiti State Teaching Hospital, Ado – Ekiti and Federal Medical Centre, Ido –Ekiti. They were also mornitored to three to six days after delivery. They serve as controls for group 1.

Group 3

This group was made up of 40 hypertensive non pregnant women (HNPW) and non- users of contraceptive

therapy randomly selected.

Group 4

This group also consisted of 40 normotensive non-pregnant women (NNPW) and non- users of contraceptive therapy randomly selected. The subjects here serve as control to group 3.

Selection of subjects

The selection of Hypertensive subjects in this study was made by screening patients admitted from the antenatal and post natal wards of of Ekiti State Teaching Hospital, Ado – Ekiti and Federal Medical Centre, Ido –Ekiti. They were all having a blood pressure greater than 140/90 mmHg at the time of sampling. The blood pressure was measured by means of sphygmomanometer. It was also noted that none of the hypertensive pregnant women had any evidence of chronic renal disease or any other medical Obstetrics and Gyneacology complications apart from hypertension. However some of the subjects were having oedema and proteinuria.

In the case of the normotensive pregnant women, they were all healthy, and none of them gave any history of hypertension or other diseases and they were non- contraceptive users.

Collections of samples

All the subjects in each group while resting and supine had 10ml of their venous non fasting blood samples collected into lithium heparinised anticoagulant bottles. The blood was immediately centrifuged at 4,000 revolutions per minute (rpm) for 15minutes, and separated to obtain the plasma. The samples were then analyzed to obtain the concentrations of all the desired parameters in this study.

Biochemical assay

Plasma Total cholesterol in all the samples collected from all the subjects was analyzed by the cholesterol CHOD-PAP method which is an enzymatic end point method (Trinder, 1988). HDL in the sample was separated by precipitation through the procedure adopted by Lopes- Virella (1977).LDL-Cholesterol concentration in the sample was determined using the relationship described by Frederickson-Frieldwald *et al.*, (1972).The concentration of VLDL-cholesterol in the sample was analysed by a simple relationship described by Frederickson-Frieldwald *et al.*, (1972).GPO-PAP method of Randox diagnostic kit (Trinder, 1988) was used to determine Triacylglerol.

Estimation of sodium and potassium were analyzed using flame emission photometry the method as described by AOAC 1990. Plasma levels of the elements (Zn, Fe, Ca, Mg, Mn, Pb, P, Cd, Cu) were determined using atomic absorption spectophotometry as described by AOAC 1990.

Statistical Analysis

The data collected was analyzed using one –way Analysis of variance (ANOVA) and Duncan multiple range test to compare the data obtained from the experiment to those of the control.

RESULTS AND DISCUSSION

Tables 1a, 1b and 1c represents the lipid profile results.TC, LDLC, VLDLC and TG increase significantly in PIH subjects. HDL on the other hand showed significant decrease in the PIH subjects as the pregnancy progresses to the 3rd trimester. At post-partum, TC, LDLC, VLDLC, and TG significantly decrease, while HDL increases significantly in PIH subjects as compared with the normotensive pregnant group.

TABLE 1a Plasma Lipid Profile, and Total Cholesterol concentrations (mmol/L), in normotensive non
pregnant women [A], hypertensive non pregnant women [B], normotensive pregnant women [C], and
hypertensive pregnant women [D], at 2 nd trimester.

PARAMETERS	Α	В	С	D	Ls
TC	3.71±0.62 ^a	4.32±0.44°	4.00 ± 0.43^{a}	5.99±0.34 ^b	P<0.001
HDLC	2.53±0.42 ^a	1.99±0.30°	2.40±0.31ª	1.08 ± 0.40^{b}	P<0.001
LDLC	$0.84{\pm}0.50^{a}$	$1.60{\pm}0.50^{b}$	$0.85{\pm}0.40^{a}$	1.68 ± 0.42^{b}	P<0.001
VLDLC	0.26±0.18 ^a	$0.42 \pm 0.20^{\circ}$	$0.30{\pm}0.15^{a}$	0.71 ± 0.30^{b}	P<0.001
TG	$0.58{\pm}0.30^{a}$	$0.90{\pm}0.22^{b}$	$0.60{\pm}0.30^{a}$	$1.30{\pm}0.50^{b}$	P<0.001
SBP (mmHg)	98.7±10.42 ^a	161±13.45 ^b	104±4.91ª	151±4.24 ^b	P<0.05
DBP (mmHg)	$67.15{\pm}10.18^{a}$	107±12.45 ^b	66 ± 3.58^{a}	96±7.89 ^b	P<0.05

Results are presented as means \pm standard error of mean. Values with different superscript are significantly different.

TABLE 1b: Plasma Lipid Profile, and Total Cholesterol concentrations (mmol/L), in normotensive non
pregnant women [A], hypertensive non pregnant women [B], normotensive pregnant women [C], and
hypertensive pregnant women [D], at 3 rd trimester.

PARAMETERS	Α	В	С	D	Ls
ТС	3.71±0.62ª	4.32±0.44°	4.00±0.50 ^a	6.12±0.30 ^b	P<0.001
HDLC	2.53±0.42ª	1.99±0.30°	2.52±0.30 ^a	0.88 ± 0.40^{b}	P<0.001
LDLC	$0.84{\pm}0.50^{a}$	1.60±0.50°	1.35±0.45ª	2.61±0.30b	P<0.001
VLDLC	0.26±0.18ª	$0.52 \pm 0.20^{\circ}$	$0.30{\pm}0.04^{a}$	0.82 ± 0.32^{b}	P<0.001
TG	$0.58{\pm}0.30^{a}$	0.90±0.22°	0.63±0.10 ^a	1.51±0.45 ^b	P<0.001
SBP (mmHg)	98.7±10.42ª	161±13.45 ^b	104±4.91ª	151±4.24 ^b	P<0.05
DBP (mmHg)	67.15 ± 10.18^{a}	107±12.45 ^b	66±3.58ª	96±7.89 ^b	P<0.05

Results are presented as means \pm standard error of mean. Values with different superscript are significantly different.

TABLE 1c: Plasma Lipid Profile, and Total Cholesterol concentrations (mmol/L), in normotensive non pregnant women [A], hypertensive non pregnant women [B], normotensive pregnant women [C], and hypertensive pregnant women [D], at 3-6 days post-partum.

PARAMETERS	Α	B	С	D	Ls
ТС	3.71±0.62 ^a	4.32±0.44°	4.03±0.51ª	5.71±0.30 ^b	P<0.001
HDLC	2.53±0.42 ^a	1.99±0.30°	2.51±0.30 ^a	1.72 ± 0.40^{b}	P<0.001
LDLC	$0.84{\pm}0.50^{a}$	1.60 ± 0.50^{b}	1.23±0.40 ^a	1.61 ± 0.40^{b}	P<0.001
VLDLC	0.26±0.18 ^a	$0.42 \pm 0.20^{\circ}$	$0.27{\pm}0.20^{a}$	0.72±0.33 ^b	P<0.001
TG	$0.58{\pm}0.30^{a}$	0.90±0.22°	0.65±0.31ª	1.29 ± 0.32^{b}	P<0.001
SBP (mmHg)	98.7±10.42 ^a	161±13.45 ^b	104±4.91ª	151±4.24 ^b	P<0.05
DBP (mmHg)	67.15±10.18 ^a	107±12.45 ^b	66±3.58ª	96±7.89 ^b	P<0.05

Results are presented as means \pm standard error of mean. Values with different superscript are significantly different.

Tables 2a, 2b and 2c reveal the results or the elements and electrolytes determined in this study. Subjects with PIH showed more significant decrease in the levels of Ca, Mg, Na, and P. However these elements showed significant increase at post–partum in PIH group. Other elements and electrolytes; K, Cl, HCO3, Fe and Zn, did not show any significant change in their levels in both normotensive and PIH patients as compared with the control subjects.

TABLE 2a: Plasma concentrations of elements, electrolytes, urea and creatinine in normotensive non
pregnant women [A], hypertensive non pregnant women [B], normotensive pregnant women [C] and
hypertensive pregnant women [D] at 2 nd trimester.

PARAMETERS	Α	В	С	D	Ls
Na (mmol/L)	141±2.0ª	140±2.0ª	136±2.3 ^b	130±2.4°	P<0.005
K (mmol/L)	4.2±1.0 ^a	4.3±1.1ª	4.2±1.2 ^a	3.9±1.2 ^a	P<0.005
Ca (mmol/L)	4.8±0.2 ^a	4.6±0.2ª	4.2±0.3 ^b	3.4±0.2°	P<0.005
Mg (mmol/L)	0.74±0.1ª	0.71±0.1ª	0.62±0.1 ^b	0.50±0.2°	P<0.001
Fe (mmol/L)	0.031±0.1ª	0.029 ± 0.10^{a}	0.028 ± 0.12^{a}	$0.028{\pm}0.13^{a}$	P<0.001
Cl (mmol/L)	102±2.3ª	103±2.5 ^a	101±2.4ª	100±2.3ª	P<0.005
HCO ₃ (mmol/L)	24.1±2.2 ^a	24.4±2.1ª	24.1±2.0 ^a	23.3±2.2ª	P<0.005
P (mmol/L)	0.9±0.13ª	$0.87{\pm}0.12^{a}$	0.72 ± 0.12^{b}	0.51±0.14°	P<0.005
Zn (mmol/L)	0.011 ± 0.004^{a}	0.010±0.003 ^a	$0.010{\pm}0.003^{a}$	$0.009{\pm}0.002^{a}$	P<0.001
SBP(mmHg)	98.7±10.42ª	161±13.45 ^b	104±4.91ª	151±4.24 ^b	P<0.05
DBP(mmHg)	67.15±10.18 ^a	107±12.45 ^b	66±3.58 ^a	96±7.89 ^b	P<0.05

Results are presented as means \pm standard error of mean. Values with different superscript are significantly different.

TABLE 2b: Plasma concentrations of elements, electrolytes, urea and creatinine in normotensive non
pregnant women [A], hypertensive non pregnant women [B], normotensive pregnant women [C] and
hypertensive pregnant women [D] at 3 rd trimester.

PARAMETERS	Α	В	С	D	Ls
Na(mmol/L)	141±2.0 ^a	140±2.0ª	125.1±2.3 ^b	110.5±2.5°	P<0.005
K(mmol/L)	4.2±1.0 ^a	4.3±1.1 ^a	3.74±1.2 ^a	3.77±1.2 ^a	P<0.005
Ca(mmol/L)	4.8±0.2 ^a	4.6±0.2ª	4.29±0.3 ^b	3.15±0.3°	P<0.005
Mg(mmol/L)	0.74±0.1ª	0.71±0.1ª	0.61±0.1 ^b	0.41±0.2°	P<0.001
Fe(mmol/L)	0.031±0.01 ^a	0.029 ± 0.010^{a}	0.025 ± 0.012^{a}	0.23±0.013 ^a	P<0.001
Cl(mmol/L)	102±2.3ª	103±2.5ª	97.9±2.4ª	96.5±2.3ª	P<0.005
HCO ₃ (mmol/L)	24.1±2.2 ^a	24.4±2.1ª	23.0±2.0 ^a	22.5±2.2ª	P<0.005
P(mmol/L)	0.9±0.013ª	$0.87{\pm}0.012^{a}$	0.69 ± 0.012^{b}	$0.45 \pm 0.014^{\circ}$	P<0.005
Zn(mmol/L)	0.011 ± 0.004^{a}	0.010±0.003ª	0.09 ± 0.003^{a}	$0.76{\pm}0.002^{a}$	P<0.001
SBP mmHg	98.7±10.42ª	161±13.45 ^b	104±4.91ª	151±4.24 ^b	P<0.05
DBP mmHg	67.15±10.18 ^a	107±12.45 ^b	66 ± 3.58^{a}	96±7.89 ^b	P<0.05
Zn(mmol/L) SBP mmHg DBP mmHg	$\begin{array}{c} 0.011{\pm}0.004^{a} \\ 98.7{\pm}10.42^{a} \end{array}$	$\begin{array}{c} 0.010{\pm}0.003^{a} \\ 161{\pm}13.45^{b} \\ 107{\pm}12.45^{b} \end{array}$	0.09±0.003 ^a 104±4.91 ^a 66±3.58 ^a	0.76 ± 0.002^{a} 151±4.24 ^b 96±7.89 ^b	P<0.001 P<0.05 P<0.05

Results are presented as means \pm standard error of mean. Values with different superscript are significantly different.

TABLE 4.4c: Plasma concentrations of elements, electrolytes, urea and creatinine in normotensive non pregnant women [A], hypertensive non pregnant women [B], normotensive pregnant women [C] and hypertensive pregnant women [D] at 3-6 days post-partum.

PARAMETERS	Α	B	С	D	Ls
Na(mmol/L)	141±2.0 ^a	140±2.0ª	130.3±2.2 ^b	115.9±2.4°	P<0.005
K(mmol/L)	4.2 ± 1.0^{a}	4.3±1.1 ^a	3.75±1.2 ^a	3.89±1.2ª	P<0.005
Ca(mmol/L)	4.8 ± 0.2^{a}	4.6±0.2 ^a	4.40±0.3 ^b	3.45±0.3°	P<0.005
Mg(mmol/L)	0.74±0.1ª	0.71 ± 0.1^{a}	0.71 ± 0.1^{b}	0.45±0.1°	P<0.001
Fe(mmol/L)	$0.031{\pm}0.011^{a}$	0.029±0.010 ^a	0.027 ± 0.001^{a}	0.23±0.001ª	P<0.001
Cl(mmol/L)	102±2.3ª	103±2.5ª	97.9±1.3ª	96.4±2.4ª	P<0.005
HCO ₃ (mmol/L)	24.1±2.2ª	24.4±2.1ª	23.1±2.4 ^a	22.4±2.3ª	P<0.005
P(mmol/L)	0.9±0.13 ^a	0.87 ± 0.12^{a}	0.74±0.21 ^b	0.69±0.14°	P<0.005
Zn(mmol/L)	0.011 ± 0.004^{a}	0.010±0.03ª	0.09±0.013ª	0.89±0.3ª	P<0.001
SBP mmHg	98.7±10.42 ^a	161±13.45 ^b	104±4.91ª	151±4.24 ^b	P<0.05
DBP mmHg	67.15±10.18 ^a	107±12.45 ^b	66±3.58ª	96±7.89 ^b	P<0.05

Results are presented as means \pm standard error of mean. Values with different superscript are significantly different.

The present study showed clearly plasma lipid elevations in PIH group and the results obtained from the present study showed significantly higher levels (p< 0.001) of plasma TC, TG, LDLC, and VLDLC and significantly lower levels of HDLC levels in hypertensive pregnant women compared to the normotensive pregnant women (Tables 1a, and 1b) This agrees with the report of Franz and Wendler, (1992). The TC level in PIH cases was found to be higher than the normotensive pregnant women. These findings are in agreement with the studies reported by Sattar *et al.* (1997). Also the result obtained in the present study, which shows the plasma TG levels of hypertensive cases to be higher than the controls, tallies with what has been reported in previous studies by Das and Ischei (1996). Also the mean VLDLC in PIH cases rose significantly compared to the controls, which may be due to hyper triglyceridemia leading to enhanced entry of VLDL that carries endogenous triglyceride into circulation. The increased VLDLC levels in the hypertensive pregnant women in this present study correlates with the findings of other researchers (Sattar *et al.* 1997).

Plasma LDLC levels in this study which are significantly higher in PIH corroborated with the findings of other studies (Hubel *et al.*, 1989 and Sattar *et al.*, 1997). In addition, the HDLC levels in PIH cases are low as compared with the controls. These findings are also similar to the studies done by Kaaja *et al.* (1995). It is suggested that the low levels of HDLC in hypertensive pregnancy may not only be because of hypo-oestrogenaemia but, may also be due to insulin resistance which is enhanced by low level of Ostrogen (Kaaja *et al.*, 1995). Therefore it is evident that dyslipidemia was found in PIH cases. The elevated TC, TGL, VLDLC, and LDLC levels and reduced HDLC levels may be due to exaggerated insulin resistance and low estrogen levels which may contribute to the pathogenesis.

Table 1c reveals the result for all the plasma lipids at 3-6 days post partum. It was found that TC, TG, VLDLC, and LDLC levels significantly decreases from the values obtained in the 3^{rd} trimester in PIH subjects (P<0.001). In normotensive pregnancy, the values also reduce insignificantly from what was observed in the 3^{rd} trimester. This result correlates with the work of Das and Isichei (1996). The result of this study on the plasma lipids suggest that PIH subjects may have as much or even more of cardiovascular risk index as a result of the

increase in lipids.

Tables 2a and 2b show the results of the elements and electrolytes determined in both normotensive pregnancy and PIH patients during 2nd and 3rd trimesters respectively. The levels of these elements; Ca, Mg, Na, and P were seen to decrease insignificantly (P<0.05) in normotensive, but significantly in hypertensive pregnancy as compared with the non-pregnant subjects. The result of the hypertensive non pregnant group has no significant change from the normotensive non pregnant group. However a significant decrease was seen in the levels of the elements during the 3rd trimester. Patients with PIH showed more significant decrease compared with those with normotensive pregnancy (P < 0.05). Renal excretion of calcium and phosphate increases during pregnancy (Mozdzien et al, 1995). Excretion usually increases during each trimester, with maximum levels reached during the third trimester. It is reported that a daily supplement of 2000 mg calcium had significant results in lowering the incidence of toxemia (Cong et al, 1995). There was a significant decrease in the plasma magnesium levels in the normal pregnancy cases as compared to those in the non- pregnant controls, and Mg significantly decreased more in PIH. Our findings are consistent with the reports of other researchers, (Seydoux et al, 1992). Magnesium affects the cardiac and smooth muscle cells by altering the transport of calcium and its binding to the membrane and organ cells. Magnesium acts peripherally to produce peripheral vasodilatation and a fall in blood pressure. Thus, low levels of magnesium predispose to an increase in the arterial pressure(Standley et al, 1997). Magnesium is known to increase the prostacycline release from the endothelial cells of the blood vessels, which acts as a potent vasodilator. In addition, magnesium depletion increases the vasoconstrictor effect of angiotensin II and nor-adrenaline.

Plasma sodium levels was also found to be significantly decreased in the PIH cases as compared to that in the non-pregnant controls and in the normotensive pregnant women. The findings are in accordance with those reported by other authors, Clarke (1997). In PIH, sodium transport is altered across the cell membrane and this leads to the accumulation of sodium in the extravascular spaces and a decrease in the plasma sodium levels.

Other element and electrolytes (K, Cl, HCO3, Fe and Zn) as seen from both Tables 2a and 2b did not show any significant in their levels in both normotensive and PIH patients as compared with the control subjects. This result in agreement with the work of Indumati *et al* 2011, suggests that these electrolytes may not really be implicated in the etiology of PIH.

The plasma levels of Ca, Mg, Na and P that significantly decreased in 2nd and 3rd trimesters began to show significant increase (P<0.05) at post-partum in PIH patients. For normotensive pregnant patients, an insignificant increase was seen (P>0.05). The significant increase at post-partum in these electrolytes further suggests their implication in PIH. This result agrees with the work of Hurd *et al.*,(2002).

CONCLUSION

In conclusion, the result obtained in this study on the Plasma lipids suggests dyslipidemia in preeclampsia cases. The abnormal lipid metabolism particularly high triglycerides, high TC, high LDLC, and low HDLC levels may favor formation of atheromatous plaques that lead to cardiovascular disease. Therefore, it is recommended that blood lipid concentrations should be evaluated in pregnant women and women with PIH during antenatal care which may provide information to clinician about high risk pregnancies and helpful in prevention of complications. Further diets which can raise HDLC levels are recommended to preeclampsia patients. Furthermore, elements such as Ca, Na, Mg, and P have been implicated in PIH as seen in this study. They all significantly decreased in preeclampsia. Moreover, from the results in this study, though calcium and magnesium deficiencies cannot be pin pointed as the sole factors for the etiology of preeclampsia, but their relationship with preeclampsia cannot be denied. Both magnesium and sodium are known to decrease the intracellular calcium by different mechanisms, thus leading to smooth muscle contraction and an elevation in blood pressure. The recommended daily allowance of calcium for a pregnant woman is 1200 mg. Thus, along with a dietary restriction of sodium, a dietary supplementation of calcium and magnesium in the form of milk, cheese, soybean products, leafy vegetables, etc. during pregnancy, could result in a reduction in the incidence of preeclampsia.

ACKNOWLEDGEMENT

All the Professors in the department of Biochemistry, Ekiti state university, Nigeria and the chief technologists of the university are highly appreciated for their contributions that made this research work a success.

REFERENCES

AOAC. (1990): Association of official, chemists, official methods of analysis. 15thEdition, Washington DC, U.S.A.

Chesley L.C, (1987); Hypertensive disorders in pregnancy, New York Applaton, Centry-Crofts, pp.57 Clarke S. L (1997): Ed. Critical care obstetrics, 3rd edition, USA, Black well science Ltd. 251-256pp. Cong K, Chi S, Liu G (1995) Calcium supplementation during pregnancy for reducing pregnancy induced hypertension. *Chinese Medical Journal Engl* 108:57-9,

- Das S.C and Isichei, U.P., (1996): Serum total cholesterol, triglycerides, HDL Cholesterol and LDL Cholesterol in pregnant women. *Tropical Journal Obstetrician Gynaecology*. 13:159-163
- Davey DA, MacGillivray I. (1998) The classification and definition of the hypertensive disorders of pregnancy. American Journal of Obstetrics and Gynecology 158(4):892–8
- Franz, H and Wendler, D. (1992): A contolled study of marternal serum concentration of lipoprotein in pregnancy induced hypertension. *Archives of Gynaecology and Obstetrician*. 252:81-86
- Fredrerickson- Friedewald, W.T (1972): Method for the determination of LDL- Cholesterol. *Clinical Chemistry*. 18:499-502
- Gotto AM (1998) Triglyceride: The Forgotten Risk Factor. Circulation 97: 1027-1028
- Guice-Booth E (2005); Pregnancy changes and healthy living in pregnancy: Q and A, 1sted, Mreadobrook Press, 214-247
- Henry, J.B (2001): Clinical diagnosis and management of laboratory methods 20th edition, philadelphia: W.B., Saunders Company.
- Hubel, C.A, Roberts, J.M., Taylor, R. N, Masc, T.S, Roaens, G.M, and Mclangtilin, M.K, (1989): Lipid peroxidation in pregnancy. New perspective on preeclampsia. *American Journal of Obstetrician and Gynecology* 161:1025-1034
- Hurd W.W, Fommin V.P, Natarajan V. (2002): Magnesium sulphate inhibits the oxytocin-induced production of inositol 1,4,5- tris phosphate in cultured human myometrial cells. *American Journal of Obstetrician* and Gynecology 187:419-24
- Indumati V, Kodliwadmath M, Sheela M (2011): The role of serum electrolytes in pregnancy induced hypertension. *Journal of Clinical Diagnostic Research* 2011, 5:66–69.
- Kaaja, R., Tikkanen, M., Viinikka, L. and Ylikorkala, O. (1995): Serum lipoproteins, insulin, and urinary prostanoid metabolites in normal and hypertensive pregnant women. *Obstetrics and Gynecology*. 85: 353–356
- Lopes-Virella, (1977): HDL- Cholesterol Precipitant. Chemistry 23: 882-885
- Mozdzien G, Schinninger M, Zazgornik (1995): Kidney function and electrolyte metabolism in healthy pregnant women. *Wien. Med. Wochenschr* 145:12-7,
- Ostos M.A, Conconi M, Vergnes L, Baroukh N, Ribalta J, Girona J, Caillaud J.M, Ochoa A, Pearson T.A, Mensah G.A, and Alexander R.W (2003): Markers of inflammation and cardiovascular disease: application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation*, 107:499-511.
- Oyeyemi A.O and Asaolu M.F (2015): Evaluation of some Marker Enzymes, Prostaglandins, C-Reactive Protein and Plasma Total Protein, in Pregnancy Induced Hypertension among women in Ekiti State, Nigeria. *Journal of Biology, Agriculture and Healthcare* 5 (15):170-175
- Sattar, N., Bendomin, A., Berry, C., Shepherd, J, and Greer, I.A and Packard, C.T. (1997): Lipoprotein Subtraction concentration in preeclampsia. *Obstetrics and Gynaecology*. 89(3): 403-408
- Schaefer E.J, Lichtenstein A.H, Lamon-Fava S. (1995): Lipoproteins, nutrition, aging and atherosclerosis. *American Journal of Clinical Nutrition* 61:726S-740S
- Seydoux, J., Girardin, E., Paunier, L. and Beguin, F. (1992) :Serum and intracellular magnesium during normal pregnancy and in patients with pre-eclampsia. *British. Journal of Obstetric Gynaecology*. 99: 207–211.
- Sibai B.M, Gabbe S.G, Niebyl J.R and Simpson J.L (2007); Hypertension. Obstetrics- Normal and problem pregnancies 5th ed. Philadelphia, Pa; Elsevier Churchill Livingstone, chapter 33
- Standley, C. A., Whitty, J. E., Mason, B. A. & Cotton, D. B.(1997):Serum ionized magnesium levels in normal and preeclamptic gestation. *Obstetrics. Gynaecology* 89: 24–27.
- Trinder, P., (1988): Report of the national Cholesterol Educational Program. Expert panel on detection, evaluation and treatment of high blood cholesterol in adults. Arch. Intern. Med. 148:36-39