LIPID PROFILE AND OBESITY IN WOMEN WITH PREECLAMPSIA
RUNNING TITLE : HYPERLIPIDEMIA IN PREECLAMPSIA

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ABSTRACT

Background: Maternal predisposition to preeclampsia could be explained by abnormal lipid metabolism which may have a role to play in the promotion of oxidative stress and vascular dysfunction seen in preeclampsia and this is avoidable with timely and effective care. One of such care is close monitoring of the lipid profile which would no doubt improve clinical outcome.

Aim : To estimate lipid profile and assess obesity in women with preeclampsia.

Method: This study had a total of 196 respondents. The subjects were registered pre-eclamptic (PE) ante-natal care (ANC) patients of the Obstetrics and Gynaecology Department of Government Specialist Hospital, Benin City, Edo State, Nigeria. The control subjects were registered normotensive ANC patients (NPW) and analbuminuric hypertensive pregnant (AHPW) patients of the same Department, and within the same age range. Ethical clearance was obtained from the Edo State Health Management Board. Structured questionnaires were administered to the study groups. Body mass index (BMI) was calculated from their weights and heights. Standardized colorimetric assay kit was used for lipid profile. Data were analyzed using SPSS version 16.0 and the level of significance was set at 95% ($p < 0.05$).

RESULTS: The results revealed a significantly ($P<0.05$) higher BMI in PE (33.35±0.75Kg/m^2) when compared with NPW (26.57±0.66Kg/m^2) and AHPW (24.84±1.26Kg/m^2. The mean index systolic and diastolic blood pressures were found to be significantly ($p< 0.05$) higher in PE (160.24±9.34mmHg and 108.56±8.02mmHg) than in NPW (106.00±11.23mmHg and 63.38±4.32mmHg) and AHPW (141.38±14.21mmHg and 98.21±4.94mmHg).The mean total cholesterol (TC), triglyceride (TG) and low density lipoprotein (LDL-C) were significantly ($P<0.05$) increased in PE (228.44±3.39mg/dl, 154.73±3.96mg/dl and 146.17±2.85mg/dl) and AHPW (192.13±21.21mg/dl, 134.51±12.62mg/dl and 141.23±10.25mg/dl) when compared with NPW (179.53±5.97mg/dl, 122.92±5.52mg/dl and 116.83±5.48mg/dl). However mean high density lipoprotein (HDL-C) was significantly ($p < 0.05$) lower in PE (38.54 ± 0.61mg/dl) and AHPW (35.63±8.05mg/dl) than in NPW (45.61±1.32mg/dl). TC and TG were significantly ($P<0.05$) increased in the 2ndtrimester in PE (205.76 ± 5.23 mg/dl, and 176.03 ± 6.20 mg/dl,) and AHPW (190.57 ±11.23mg/dl and 165.65±9.44mg/dl) than in NPW (159.44±9.15mg/dl and 113.63±9.16mg/dl) and the 3rdtrimesters(PE: 211.13±4.63mg/dl and 193.43±4.97mg/dl, AHPW: 202.82±10.13mg/dl and 180.94±11.75mg/dl, NPW: 173.82±8.02mg/dl and 130.35±6.12mg/dl) while HDL-C was significantly ($P<0.05$) lower in PE(38.44±0.91mg/dl) and AHPW(33.98±7.55mg/dl) than NPW(45.67±1.17mg/dl) in the 3rd trimester. Mean serum LDL-C and TG were significantly ($P<0.05$) higher in severe (155.61±5.10mg/dl and 160.72±5.20mg/dl) than in mild (142.79±3.43mg/dl and 147.39±5.60mg/dl) PE.

CONCLUSION: This study showed that abnormalities of lipid metabolism and obesity may contribute to the severity of preeclampsia and thus lipid profile can be used as a biomarker for preeclampsia.

KEY WORDS : Preeclampsia, Hyperlipidemia, Obesity, Overweight,

INTRODUCTION

Nearly one tenth of all maternal deaths in Africa and Asia and one-quarter in Latin America are associated with hypertensive diseases in pregnancy, a category that encompasses pre-eclampsia.[1]Pre-eclampsia(PE) is a disorder of pregnancy characterized by high blood pressure and a large amount of protein in the urineand it is one of the leading causes of maternal and perinatal morbidity and mortality worldwide.[2]

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In pre-eclampsia, the systolic blood pressure is≥140mmHg, diastolic ≥90 mmHg and there is proteinuria of at least 300 mg in 24hour urine sample collection. According to the World Health Organization (WHO), the incidence of preeclampsia ranges from 2-10% of pregnancies worldwide with 1.8 – 16.7% occurring in developing countries while developed countries have a rate of 0.4%.In Nigeria, preeclampsia affects 37,000 women annually with a prevalence rate of 5.6% to 7.6% of pregnancies in Southern Nigeria.[3,4,5] Onset of symptoms occur in the late second or third trimester, most commonly after the 32nd week. Some women will experience pre-eclampsia as early as 20weeks, though this is rare and it may also occur in the immediate postpartum period.[6]Women with mild proteinuria generally have no symptoms. However, women with severe pre-eclampsia (blood pressure ≥160/110 mmHg, proteinuria>2.5g/24h) may have symptoms such as renal insufficiency, liver diseases, haematological and neurological disturbances.[2]

During pregnancy, there is increased lipolysis and mobilization of triglyceride from adipocytes. Increased production of VLDL in combination with impaired lipoprotein lipase activity leads to ineffective clearance of triglyceride-rich lipoproteins such as VLDL and VLDL-remnants which eventually result in increased triglyceride levels.[7] Hepatic lipase is responsible for the increased synthesis of TGs whereas the decreased activity of lipoprotein lipase is responsible for decreased catabolism at the adipose tissue level, the net effect of which is increased circulating TGs.[8] There is also accumulation of LDL due to decreased activity of lipoprotein lipase and LDL, total cholesterol and triglyceride levels increase from the second trimester until the third trimester with increasedrisk of development of atherosclerosis.[9,10]

The prevalence of maternal obesity is rising in some antenatal clinics, in line with the prevalence of obesity in the general population with its attendant complications such as gestational diabetes mellitus (GDM), chronic hypertension and preeclampsia.[11]Obesity is considered a risk factor for preeclampsia and there are many mechanisms that link obesity with a higher risk of developing preeclampsia; such as increased level of proinflammatory cytokines and leptin.[12,13]It has been described that leptin reduces cytotrophoblast proliferation. One of the early alterations observed in preeclampsia is poor cytotrophoblast proliferation.[12,14]The increasing prevalence of maternal obesity worldwide is therefore a major challenge in the care of pregnant women from preconception to postpartum. In this study, we intend to assess the relationship between lipid profile and obesity in women with preeclampsia.

MATERIAL AND METHODS

The subjects were registered pre-eclamptic ante-natal clinic (ANC) patients of the Obstetrics and Gynaecology Department of Government Specialist

Hospital, Benin City, Edo State, Nigeria. The control subjects were non-pre-eclamptic ANC patients and analbuminuric hypertensive pregnant patients of the same Department and within the same age range. Patients were considered hypertensive when blood pressure (BP)>140/90 mmHg.

Sample size was 196 calculated from the Cochran's formula (Cochran, 1977).[15] Before carrying out the study, ethical clearance was obtained from the research and Ethics Committee of the Ministry of Health, Benin City, Edo State, Nigeria. Verbal informed consent was obtained from all participants. Structured questionnaires were administered to the study groups and used to document their personal data, medical history, social, obstetric and family history. A physical examination was carried out to measure their blood pressure. Fresh Urine samples were collected into sterile bottles in the hospital under supervision. The urine was used for urinalysisusing dipstick to determine those with proteinuria. Blood samples were collected from the antecubutal veins following routine aseptic procedure using a 10ml syringe and dispensed into plain specimen bottles for lipid profile. Samples were centrifuged at 3,000 revolutions/min after allowing the sample to stand for 30 minutes to clot. The serum was harvested with clean pasteur pipettes and stored at 2 - 8^{o}C and analyzed within 48hrs.Total cholesterol (TC) and triglyceride (TG) were analyzed using the enzymatic endpoint method described by Roeschlau et. al.[16]high density lipoprotein (HDL-C) was analysed using the precipitation method described by Roeschlau et. al[16] while thelow density lipoprotein (LDL-C) was estimated from the Friedewald's Equation.[17]

Data was collected and entered into a proforma and analyzed using Statistical package for social science (SPSS) version 16.0. The means and standard deviations of the age, BMI, gestational age and lipid profile were calculated. The Pearson correlation was used in calculating the correlations between any two variables. Univariate analysis were presented as frequencies while bivariate or multivariate analysis were presented as means and standard error of means. The level of statistical significance was set at a p-value of < 0.05 for all tests of statistical significance. Data presentation, tables and charts were done using Microsoft Office.

RESULTS

A total of 196 respondents consisting of 124 pre-eclamptics (PE), 36 normotensive pregnant women (NPW) and 36 analbuminuric hypertensive pregnant women (AHPW) participated in this study. Most of the PE (44%) and Normotensive pregnant women (41.7%) were in the age bracket of 31-35 years while most of the analbuminuric hypertensive pregnant women (40%) were in the age range of 36-40 years (Fig 1). Amongst the PE 39(31.5%) were mild while 85 (68.5%) had severe PE based on a BP> 160/110mmHg(Fig.2).

The results revealed a significant($P<0.05$) higher BMI in PE(33.35±0.75Kg/m^2) when compared with NPW(26.57±0.66Kg/m^2) and AHPW (24.84±1.26Kg/m^2. Majority 71 (57.30%) of PE were overweight and obese (30{24.2%}) while most of the NPW 18(50%) and AHPW 23 (63.9%) were of normal weight. The mean indexsystolic and diastolic blood pressures were found to be significantly ($p< 0.05$) higher in PE (160.24±9.34mmHg and 108.56±8.02mmHg) than in NPW (106.00±11.23mmHg and 63.38±4.32mmHg) and AHPW (141.38±14.21mmHg and 98.21±4.94mmHg). (Table 1&2)

The mean TC,TG and LDL-C were significantly ($P<0.05$) increased in PE (228.44±3.39mg/dl,154.73±3.96mg/dl and 146.17±2.85mg/dl) and AHPW (192.13±21.21mg/dl, 134.51±12.62mg/dl and 141.23±10.25mg/dl) when compared with NPW(179.53±5.97mg/dl,

122.92±5.52mg/dl and 116.83±5.48mg/dl) However mean HDL-C was significantly ($p < 0.05$) lower in PE (38.54±0.61mg/dl) and AHPW(35.63±8.05mg/dl than in NPW (45.61±1.32mg/dl). (Table 3)

TC and TG were significantly ($P<0.05$) increased inthe 2nd trimester inPE (205.76 ± 5.23 mg/dl, and 176.03 ± 6.20 mg/dl,) and AHPW (190.57 ±11.23mg/dl and 165.65±9.44mg/dl) than in NPW (159.44±9.15mg/dl and 113.63±9.16mg/dl)and also in the 3rd timester(PE: 211.13±4.63mg/dl and 193.43±4.97mg/dl, AHPW: 202.82±10.13mg/dl and 180.94±11.75mg/dl, NPW: 173.82±8.02mg/dl and 130.35±6.12mg/dl). There was also a significant ($p < 0.05$) increase in TG in the 3rd than in the 2nd trimesters within the PE and AHPW. LDL-C was significantly ($P<0.05$) higher in PE(145.86±3.76mg/dl and 157.56±4.29mg/dl) and AHPW(130.38±11.42mg/dl and 141.06±10.93mg/dl) than NPW(105.74±0.81mg/dl and 113.40±7.50mg/dl) in the 2nd and 3rd trimester while HDL-C was significantly ($P<0.05$) lower in PE(38.44±0.91mg/dl) and AHPW(33.98±7.55mg/dl) than NPW(45.67±1.17mg/dl) in the 3rd trimester. (Table 4)

Mean serum LDL-C and TG were significantly ($P<0.05$) higher in severe (155.61±5.10mg/dl and 160.72±5.20mg/dl) than in mild (142.79±3.43mg/dl and 147.39±5.60mg/dl) PE. LDL-C and TC levels were found to be non-significantly ($P<0.05$)higher amongst the Obese (150.51±6.55mg/dl and 214.46±7.25mg/dl) and overweight (145.73±3.29mg/dl and 208.69±4.26mg/dl) than in the normal weight (141.96±7.81mg/dl and 203.26±8.69). In the pre-eclamptics, plasma HDL was found to be non-significantly ($P<0.05$) higher in the normal weight (37.05 1.43mg/dl) than in the overweight (35.46±0.78mg/dl) and Obese (33.85±1.31mg/dl) while mean plasma TG was found to be relatively the same amongst the three groups of pre-eclamptics. (Table 5 & 6)

DISCUSSION

Preeclampsia is one of the most common medical complications during pregnancy with unknown etiology. It is however, characterized by vasoconstriction, metabolic changes, endothelial dysfunction and activation of the coagulation cascade in conjunction with an inflammatory response.[18] In Nigeria, PE has a prevalence rate of 5.6% and it is associated with maternal and fetal morbidity and mortality worldwide.[19,20]

We observed a significantly higher concentration of triglyceride in PE than in the NPW and AHPW which was also reported by Enquobahrie et al[21], Cekmen et al[22] and Phalak and Tilak[23] in their studies of lipid profile in preeclampsia in India. Normal pregnancy results in physiologic hyperlipidemia with an increase in triglyceride and cholesterol due to hyperestrogenemia which induces hepatic biosynthesis of lipids.[7,24]Insulin resistance during pregnancy, leads to increased influx of fatty acids to the liver promoting the synthesis of VLDL with increased TG concentration.[25] There is also an increase in hepatic lipase activity which increases the synthesis of TG in the liver with associated decrease in the activity of lipoprotein lipase leading to decreased catabolism of adipose tissue. The net effect is an increase in serum TG level.[8] In preeclampsia, there is an additional alteration in blood lipids reflecting disordered lipid and lipoprotein metabolism. The release of free fatty acids and apolipoprotein-CIII from triglycerides promote oxidative stress and proatherogenic responses in macrophages and endothelial cells.[26] VLDL remains in the plasma for a longer time due to a decreased activity of lipoprotein lipase

leading to an increase in blood LDL-C and this is associated with development of atherosclerosis. A significant increase in LDL-C was seen in PE and AHPW when compared with NPW in this study. Studies by Gratacos et al and Cassandra et alsupport this finding.[27,28]Also, significantly increased serum total cholesterol in PE than in AHPW and NPW was observed in our study. Abnormal lipid metabolism is not a mere manifestation but is also involved in the pathogenesis of PE and hypercholesterolemia promotes the formation of free radicals which are also implicated in the pathogenesis of preeclampsia.[29,30]

A significantly decreased HDL-C was observed in PE and AHPW than in NPW. HDL-C was lower in the 3rd than in the 2nd trimester in PE and AHPW while the level remained the same in both trimesters in NPW. HDL-C also decreased with severity in PE. This finding is in accordance with studies by Adiga et aland Phalak and Tilak.[29,23] According to Cekmenet al, low HDL-C in PE is due to insulin resistance while Bozkurt et al stated that there is a direct correlation between adipose tissue lipoprotein lipase activity and plasma HDL-C and this may be responsible for the observed low levels of HDL-C.[22,25] Anuradha and Durga also reported significant increases in LDL-C and TC and no significant difference in HDL-C was observed between mild and severe PE.[31]Vidyabati et al observed that the clinical manifestations of PE preceeded dyslipidemia particularly, hypertriglyceridemia and elevated LDL.[32] This indicates that hypertriglyceridemia and high LDL-C may contribute to the aetiologic and pathophysiologic mechanisms responsible for PE.

The association between lipid profile and BMI was not significant, suggesting that maternal lipid levels were independent of overweight or obesity status, similar to findings by Daniel et al.[33]However, we found that more of the preeclamptic women were obesewhen compared to the NPW and the AHPW. Similarly, numerous studies have shown a strong correlation between an increased BMI and the risk of developing preeclampsia.[34,35]

CONCLUSION

Abnormal lipid metabolism particularly high triglyceride and LDL-C and obesity may contribute to oxidative stress and therefore the etiology of PE. Hence, weight control and lipid testing followed by appropriate management pre-conception and during pregnancy would improve the care of women with preeclampsia.

RECOMMENDATIONS

Routine estimation of serum lipids can be useful as a simple screening test to detect dyslipidemia in PE in order to reduce the incidence of complications. Despite the fact that weight loss is not recommended during pregnancy, weight loss should be recommended in women with obesity or overweight that are planning to get pregnant.

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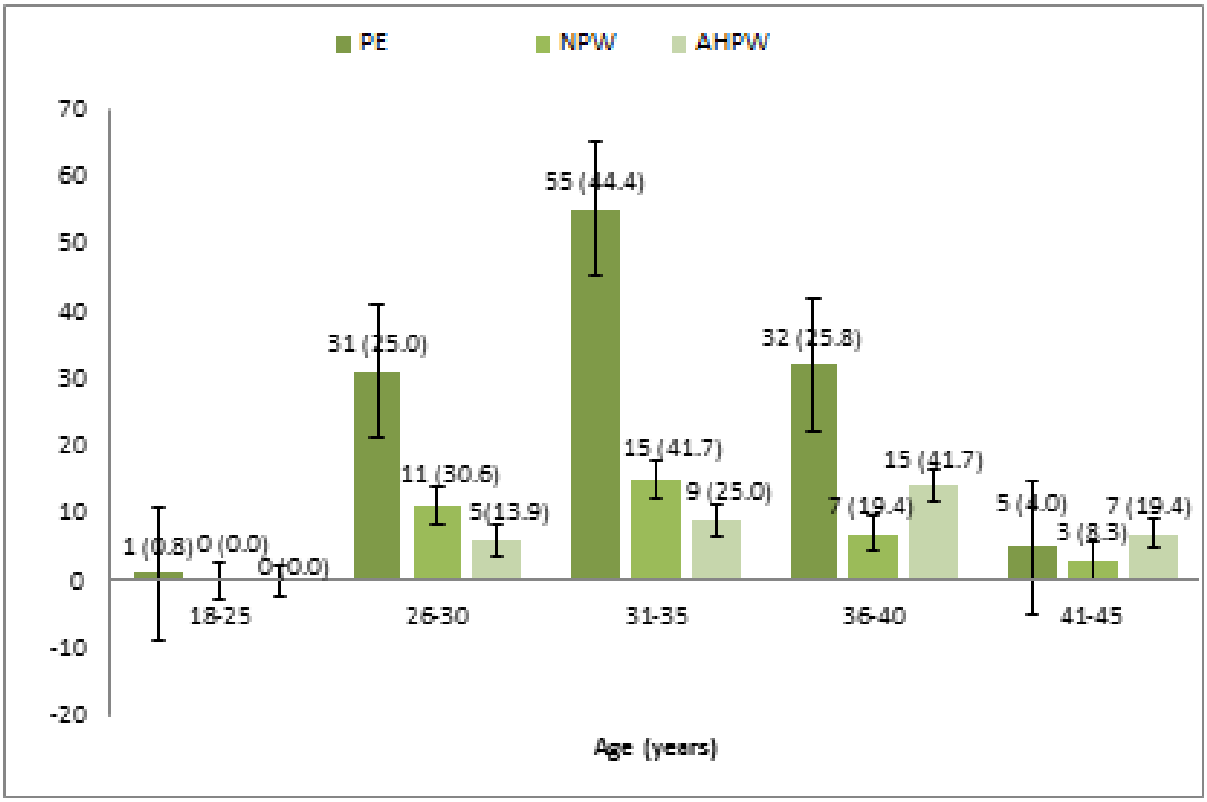

Fig 1:AGE GROUP DISTRIBUTION OF SUBJECTS

Values in parenthesis represent percentage distribution, while the bars on top of the histogram represent standard error.

KEY:

PE Preeclampsia

NPW Normotensive pregnant women

AHPW Analbuminuric hypertensive pregnant women

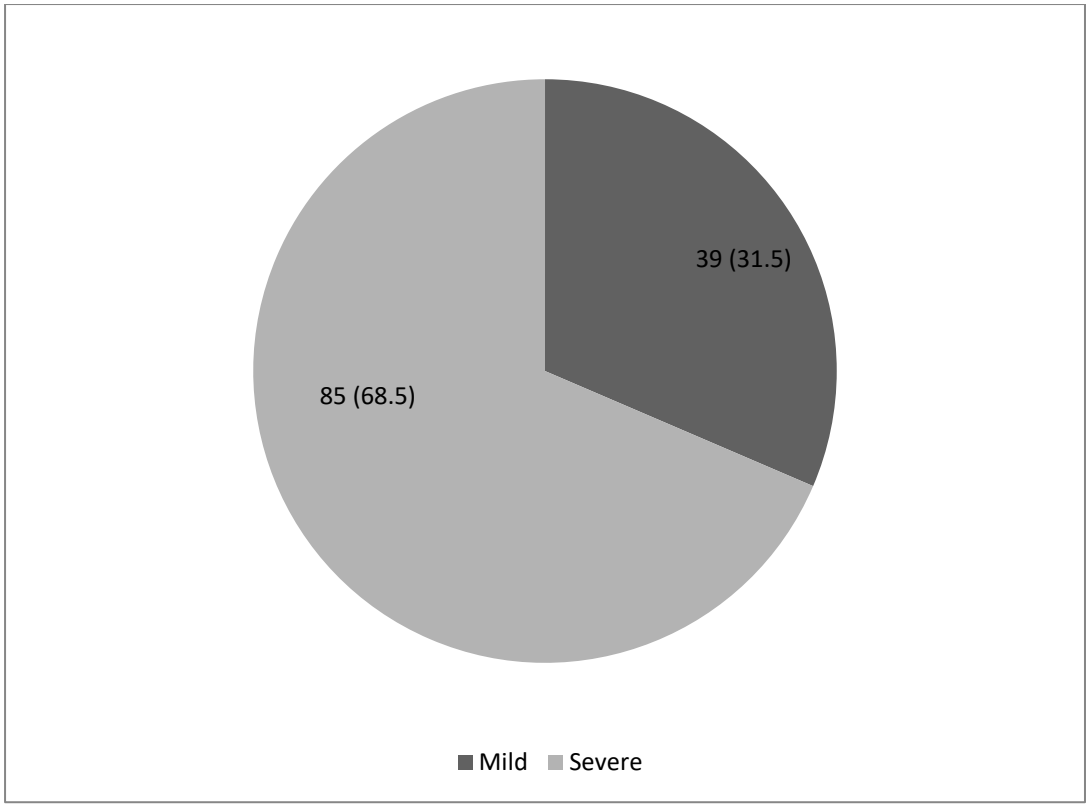

Fig2: SEVERITY OF PRE-ECLAMPSIA

TABLE 1. CLINICAL AND ANTHROPOMETRIC CHARACTERISTICS OF PE, NPW AND AHPW

Clinical/Anthropometric parameter	PE	NPW	AHPW
Booking SBP (mmHg)	136.03 ±23.01[a]	103.05 ± 12.60[b]	140.00 ± 25.52[ac]
Booking DBP (mmHg)	85.08 ± 1.46[a]	62.77 ± 7.01[b]	80.00 ± 5.16[ac]
Index SBP (mmHg)	160.24 ± 9.34[a]	106.00 ± 11.23[b]	141.38 ± 14.21[cd]
Index DBP (mmHg)	108.56 ± 8.02[a]	63.38 ± 4.32[b]	98.21 ± 4.94[cd]
BMI (kg/m^2)	33.35 ± 0.75[a]	26.57 ± 0.66[b]	24.84 ± 1.26[cb]

Values are represented as Mean ± SEM
Values in the same row with different alphabets differ significantly ($p<0.05$)
Key:
SBP Systolic blood pressure

DBP Diastolic blood pressure
BMI Body mass index

PE Pre-eclampsia
NPW Normotensive pregnant women
AHPW Analbuminuric hypertensive pregnant women

TABLE 2. DISTRIBUTION OF PE, NPW AND AHPW SUBJECTS BASED ON BMI

BMI(kg/m^2)	PE (n = 124)	NPW (n = 36)	AHPW (n = 36)
Normal weight (18-24.9)	23 (18.50)	18 (50.00)	23 (63.90)
Overweight(25-29.9)	71 (57.30)	9 (25.00)	6 (16.70)
Obese (>30)	30 (24.20)	9 (25.00)	7 (19.40)
Total	**124**	**36**	**36**

Values in parenthesis represent percentages

Key:

BMI Body mass index PE Pre-eclampsia

NPW Normotensive pregnant women AHPW Analbuminuric hypertensive pregnant women

TABLE 3. LIPID PROFILE STATUS IN PE, NPW AND AHPW, SUBJECTS.

Analyte	PE (n = 124)	NPW (n = 36)	AHPW (n = 36)
Serum TC (mg/dl)	228.44 ± 3.39^a	179.53 ± 5.97^b	192.13 ± 21.21^{ac}
Serum TG (mg/dl)	154.73 ± 3.96^a	122.92 ± 5.52^b	134.51 ± 12.62^{cb}
Serum LDL-C (mg/dl)	146.17 ± 2.85^a	116.83 ± 5.48^b	141.23 ± 10.25^{ac}
Serum HDL-C(mg/dl)	38.54 ±0.61^a	45.61±1.32^b	35.63 ±8.05^{ac}

Values are represented as mean ± SEM

Values in the same row with different alphabets differ significantly ($p<0.05$).

keys: TC Total cholesterol, TG Triglyceride, LDL-C Low density lipoprotein cholesterol HDL-C High density lipoprotein cholesterol, PE Pre-eclampsia,

NPW Normotensive pregnant Women

AHPW Analbuminuric hypertensive pregnant women

TABLE 4. LIPID PROFILEIN VARIOUS TRIMESTERS IN PE, NPW AND AHPW.

Analytes	Trimester	PE n = 124	NPW n = 36	AHPW n = 36
Serum TC (mg/dl)	2nd	205.76±5.23[a]	159.44±9.15[b]	190.57±11.23 [cd]
	3rd	211.13±4.36[a]	173.82±8.025[b]	202.82±10.13[ac]
	p-value	0.19	0.03	0.07
Serum TG (mg/ml)	2nd	176.03±6.20[a]	113.63±9.16[b]	165.65±9.44[ac]
	3rd	193.43±4.97[a]	130.35±6.12[b]	180.94±11.75[cd]
	p-value	0.04	0.14	0.04
Serum LDL-C (mg/dl)	2nd	145.86±3.76[a]	105.74±0.81[a]	130.38±11.42[ac]
	3rd	157.56±4.29[a]	113.40±7.50[b]	141.06±10.93[ac]
	p-value	0.52	0.77	0.53
Serum HDL (mg/dl)	2nd	42.65±2.79[a]	45.80±2.15[b]	43.07±8.33[ac]
	3rd	38.44±0.91[a]	45.67±1.17[b]	33.98±7.55[cd]
	p-value	0.62	0.64	0.02

Values are represented as mean ± SEM

Values in the same row with different alphabets differ significantly ($p < 0.05$)

TC Total cholesterol TG Triglyceride

LDL-C Low density lipoprotein cholesterol HDL –CHigh density cholesterol

NPW Normotensive pregnant women

AHPWAnalbuminuric hypertensive pregnant women PE Pre-eclampsia

TABLE 5. LIPID PROFILE IN PE SUBJECTS AT DIFFERENT PHASES OF PE

	Pre-eclamptics	
Analyte	**Mild (n=39)**	**Severe (n=85)**
Serum TC (mg/dl)	**209.94 ±5.02[a]**	**207.27 ± 4.42[a]**
Serum LDL-C (mg/dl)	**142.79± 3.43[a]**	**155.61 ± 5.10[b]**
Serum HDL-C (mg/dl)	**38.28 ± 1.06[a]**	**35.450 ± 0.73[a]**
Serum TG (mg/dl)	**147.39 ±5.60[a]**	**160.72±5.20[b]**

Values are represented as mean ± SEM

Values in the same row with different alphabets differ significantly ($p < 0.05$)

TC Total cholesterol

TG Triglyceride

LDL-C Low density lipoprotein cholesterol

HDL-C High density cholesterol

PE Pre-eclampsia
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TABLE 6. RELATIONSHIP BETWEEN BMI AND LIPID PROFILE IN PE

Analyte	BMI Normal weight N=23	 Overweight N=71	 Obese N=30
Serum TC (mg/dl)	**203.26 ± 8.69[a]**	**208.69±4.26[a]**	**214 ± 7.25[a]**
Serum TG (mg/dl)	**157.08 ±9.41[a]**	**157.00±4.99[a]**	**157.08 ± 9.4 [a]**
Serum LDL-C(mg/dl)	**141.96 7.81[a]**	**145.73±3.29[a]**	**150.51± 6.55[a]**
Serum HDL-C (mg/dl)	**37.05 ±1.43[a]**	**35.46±0.78[a]**	**33.85±1.31[a]**

Values are represented as mean ± SEM
Values in the same row with different alphabets differ significantly ($p < 0.05$)
TC Total cholesterol
TG Triglyceride
LDL-C Low density lipoprotein cholesterol
HDL-C High density cholesterol
PE Pre-eclampsia
NPW Normotensive pregnant women
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