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Relationship of Serum Lipid Profiles in Preeclampsia and Normal Pregnancy, Kimpese and Lukala Cities, Kongo Central, DR Congo

Malaka AN[1], Tuakashikila YM[1], Kabamba MM[1], Elongi-Moyene JP[2,3] and Tuakuila JK[1,4*]

[1]Faculty of Sciences and Technologies, University of Kinshasa, Kinshasa, DR Congo

[2]Faculty of Medicine, University of Mbandaka, Equateur, DR Congo

[3]General Hospital of Kinshasa, Kinshasa, DR Congo

[4]Faculty of Sciences, University of Sherbrooke, Quebec, Canada

Abstract

Objective: The present study aims to investigate the lipid profile levels and basal body mass index in preeclampsia and normal pregnancy in Kongo Central women, DR Congo.

Material and methods: This hospital-based cross-sectional study was conducted in Kimpese and Lukala cities with 65 participants among 35 preeclampsia women (case) and 30 normal pregnancies (control) were enrolled from January to July 2025. Blood samples were collected for analysis of total cholesterol, triglyceride and high-density lipoprotein by enzymatic assays, while low-density lipoprotein by using Fried Ewald's formula in between 20-40 weeks of gestation.

Results: The mean values of BMI, TC and LDL were significantly higher in pre-eclamptic women as compared with controls, respectively. The serum TG levels was higher, but not statistically significant in pre-eclamptic women as compared with controls. No significant difference was observed between two groups for maternal age, gestational age at enrollment, and HDL.

Conclusion: Abnormal lipid profile and increased BMI are regarded as risk factors of preeclampsia. The lipid profile screening as well as BMI monitoring may help reducing the preeclampsia which enhances maternal and fetal outcomes.

Keywords: Preeclampsia; Normal pregnancy; Lipid profile; Basal body mass index

Introduction

Preeclampsia (PE) is generally defined as Pregnancy-Induced Hypertension (PIH, systolic blood pressure > 140 mm Hg, diastolic blood pressure > 90 mm Hg) that occurs after 20 weeks of gestation, exposing pregnant women to increased risks of disabilities, hospitalization, and death.[1-7] It clinically manifests as a combination of symptoms: proteinuria (>300 mg/24h), seizures, placental abruption, disseminated intravascular coagulation,

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***Corresponding author at:** Tuakuila JK, Analytical Chemistry and Environmental Toxicology Laboratory, Faculty of Sciences, University of Kinshasa, Kinshasa, The DRC. 16 Tel: +243-81-934-7828

cerebral hemorrhage, pulmonary edema, renal failure, hepatic hemorrhage.[8,9] It is the most common cause of maternal and prenatal morbidity and mortality with the overall incidence estimated at 4.6% [1.8 -16.7] with considerable variation between countries.[10-12] Among these risk factors, obesity in women is steadily increasing worldwide and has been regarded as a risk factor for PE since the early last century.[13] Obese pregnant women are known to be at high risk of developing PE.[14] Several studies reported increased in lipid oxidation products and decreased in the levels of antioxidants in the pathogenesis of pre-eclampsia.[15] For example, compared to normal pregnancy, women who develop PE have subsequently elevated levels of Total Cholesterol (TC), oxidized Low-Density Lipoprotein (LDL) and Triglycerides (TG) and lower levels of circulating vitamin C.[16,17] The oxidative stress *via* an oxidative conversion of LDL-cholesterol to oxidized LDL form stress could explain the pathogenesis of this syndrome. The profile of lipids (cholesterols) is therefore essential to understanding the overall effect of this phenomenon. The present study aims to investigate the lipid profile levels and basal Body Mass Index (BMI) in PE and normal pregnancy in Kongo Central women, DR Congo.

Methods

Study site and subjects

This hospital-based cross-sectional study was conducted in Kimpese and Lukala cities from Kongo Central province (Southwest of the DR Congo) which were stratified to have prenatal clinics: Kimpese (Lamba Hospital Center and Christ-vie Health Center) and Lukala Bondeko health and maternity center), Lukula (Lunguana Health Center and Munzinga Health Center). Participants (n = 65) were recruited from pregnant women during the pregnancy hospital visit between January to July 2025. Pregnancy information collected in the questionnaires were clinics, socio-demographics, Anthropometrics, current and previous pregnancies, current and previous PE, diabetes mellitus, smoking during pregnancy, and lifestyle. All pregnant women were divided into two groups such as PE women for case group (n=35) and normal pregnancy women for the control group (n=30). The inclusion criteria of the present study were included that all pregnant women from 17-40 years, and having 20-40 gestational weeks. The research protocol was approved by the Bio-ethics Committee of the School of Public Health at the University of Kinshasa.

Data collection

The diagnosis of PE was used by the second-time onset of systolic blood pressure ≥ 140 mm Hg and diastolic blood pressure ≥ 90 mm Hg after 20 weeks of gestation and associated with proteinuria, if urinary protein excretion ≥ 300 mg/24 hours.[7] The Blood pressure was measured and repeated after 5 and 10 min in each pregnant woman using a mercury sphygmomanometer.[7,18] The BMI of all pregnant women was recorded such as normal (18.5 kg/m^2 to 24.9 kg/m^2), overweight (25 kg/m^2 - 29.9 kg/m^2), and obesity (≥ 30 kg/m^2) ranges. [7] During their routine hospital checkup, pregnant women provided venous blood samples in 10 mL metal free tubes containing ethylene diamine tetra acetic acid which were immediately centrifuged (10 minutes, 3000 g) and the serum fraction was transferred into 2.5 mL pre-cleaned glass vials (Supelco) and stored at -80°C.

Analytical methods

The analyzed the serum concentration of TC, HDL and TG levels by the method of an automated system (Cobas c 111 Analyzer) with standard enzymatic assays, while LDL was measured by using Fried Ewald's formula.[19]

Statistical analysis

Statistical data analysis was completed using Prism GraphPad 9.41 (GraphPad Soft - ware, San Diego, CA, USA). The normality of residuals (TC, HDL, LDL and TG) was evaluated using Kolmogorov–Smirnov test for continuous variables. For the descriptive statistics, results are presented as mean (± standard deviation), range and p-value from t-test for continuous variables. All results were assessed for data quality based on the coefficient of variation (CV: ± 5%). The Limit of Detection (LOD) was assigned a value of 3 mg/dL, 3 mg/dL, and 4 mg/dL for TC, HDL, and TG respectively. For quality control purposes, the reagent kits and calibrators used in this study were from the Cobas c 111 Analyzer. Each sample was tested according to the manufacturer's instructions with supplied reagents and the results were analyzed statistically performing precision, linearity, accuracy, and reference interval verification of 3 mg/dL - 800 mg/dL for TC, 3 mg/dL - 120 mg/dL for HDL, and 4 mg/dL - 1100 mg/dL for TG.

Results

During the study period from January to July 2025, baseline characteristics of the participants are shown in Table 1. As compared with controls (normotensive), women who subsequently developed PE had significantly higher BMI (25.7 kg/m^2 ± 2.9 kg/m^2 versus 23.1 kg/m^2 ± 2.2 kg/m^2, p < 0.0001), SBP (15.8 ± 1.2 mm Hg versus 9.7 ± 1.0 mm Hg, p = 0.0001), and DBP (10.7 ± 1 mm Hg versus 6.2 ± 0.8 mm Hg, p = 0.0001) with no difference in maternal age (28.9 ± 6.9 years versus 26.8 ± 7.5 years, p = 0.2046) and gestational age at enrollment (29.6 ± 5.5 weeks versus 32.0 ± 5.1 weeks, p = 0.0798).

Table 1: Comparison between preeclamptic cases and normotensive controls regarding maternal age, gestational age, BMI, SBP, and DBP at enrolment

Parameters	Preeclamptic cases (n = 35)	Normotensive controls (n = 30)	p-value
Maternal age (years)			
Mean ± SD	28.9 ± 6.9	26.8 ± 7.5	0.2046
Range	17-40	17-40	NS
GA at enrolment (weeks)			
Mean ± SD	29.6 ± 5.5	32.0 ± 5.1	0.0798
Range	21-40	20-39	NS
BMI (kg/m^2)			
Mean ± SD	25.7 ± 2.9	23.1 ± 2.2	0.0001
Range	20.4-32.0	18.1-27.2	HS
SBP (mm Hg)			
Mean ± SD	15.8 ± 1.2	9.7 ± 1.0	0.0001
Range	14.0-19.3	08-12	HS
DBP (mm Hg)			
Mean ± SD	10.7 ± 1.2	6.2 ± 0.8	0.0001
Range	9.0-13.1	5.1-8.2	HS

BMI-Body mass index, DBP-Diastolic blood pressure, GA-Gestational age, HDL-c- High density lipoprotein cholesterol, LDL-c- Low density lipoprotein-cholesterol, SD-Standard deviation, SBP-Systolic blood pressure, TC-Total cholesterol, TG-Triglyceride, VLDL-c- Very low density lipoprotein-cholesterol, NS: Non-significant, and HS: Highly significant.

Table 2 reports serum lipid and lipoprotein levels in both groups. The mean values of TC and LDL were significantly higher in pre-eclamptic women as compared with controls (TC=227.1 ± 54.4 mg/dL versus 187.2 ± 27.9 mg/dL, p = 0.0003; LDL=122.5 ± 46.2 mg/dL versus 86.2 ± 23.3 mg/dL, p = 0.0001), respectively. The serum TG levels was higher, but not statistically significant (TG = 175.7 ± 67.7 mg/dL versus 162.3 ± 44.8

mg/dL, p = 0.3385) in pre-eclamptic women as compared with controls. No significant difference was observed between two groups for HDL (69.4 ± 18.4 mg/dL versus 68.4 ± 15.8 mg/dL, p = 0.8226).

Table 2: Second-trimester serum lipid profile in preeclamptic cases and normotensive controls.

Parameters	Preeclamptic cases (n = 35)	Normotensive controls (n = 30)	p-value
TC (mg/dL)			
Mean ± SD	227.1 ± 54.4	187.2 ± 27.9	0.0003
Range	133-348	132-244	HS
TG (mg/dL)			
Mean ± SD	175.7 ± 67.7	162.3 ± 44.8	0.3385
Range	67-314	96-311	NS
HDL (mg/dL)			
Mean ± SD	69.4 ± 18.4	68.4 ± 15.8	0.8226
Range	32-107	37-103	NS
LDL (mg/dL)			
Mean ± SD	122.5 ± 46.2	86.2 ± 23.3	0.0001
Range	55.2-253.2	42.8-133.6	HS

TC=Total cholesterol, TG= Triglyceride, HDL= High density lipoprotein, LDL= Low density lipoprotein, NS: Non-significant, and HS: Highly significant.

In Table 3, TC (234.8 ± 53.8 mg/dL versus 178.6 ± 28.1 mg/dL, p = 0.0055) and LDL (128.4 ± 46.3 mg/dL versus 86.1 ± 25.6 mg/dL, p = 0.01518) levels were significantly higher in severe preeclamptic cases (urinary protein excretion >300 mg/24 hours) compared to mild cases of PE (urinary protein excretion = 300 mg/24 hours); on the other hand, there was no significant difference between both groups for either TG (181.4 ± 69.0 mg/dL versus 140.2 ± 50.7 mg/dL, p = 0.3385) or HDL level (70.1 ± 18.7 mg/dL versus 64.4 ± 17.4 mg/dL, p = 0.5234).

Table 3: Second-trimester serum lipid profile in mild and severe preeclamptic cases.

Parameters	*Mild preeclamptic cases (n = 10)	**Severe preeclamptic cases (n = 25)	p-value
TC (mg/dL)			
Mean ± SD	178.6 ± 28.1	234.8 ± 53.8	0.0055
Range	133-205	146-348	HS
TG (mg/dL)			
Mean ± SD	140.2 ± 50.7	181.4 ± 69.0	0.1569
Range	94-217	67-314	NS
HDL (mg/dL)			
Mean ± SD	64.4 ± 17.4	70.1 ± 18.7	0.5234
Range	42-88	32-107	NS
LDL (mg/dL)			
Mean ± SD	86.1 ± 25.6	128.4 ± 46.3	0.01518
Range	55.2-430.6	67.8-253.2	S

TC=Total cholesterol, TG= Triglyceride, HDL= High density lipoprotein, LDL= Low density lipoprotein, NS: Non-significant, S: significant, HS: Highly significant.

*Mild preeclamptic cases: urinary protein excretion =300 mg/24 hours

**Severe preeclamptic cases: urinary protein excretion >300 mg/24 hours

Discussion

Hypertensive disorders of pregnancy are one of the major causes of severe morbidity, long-term disability, and maternal and neonatal mortality.[14] In Africa and Asia in general, and in the DR Congo in particular, nearly one-tenth of maternal deaths are associated with hypertensive disorders during pregnancy, while in Latin

America, a quarter of these deaths are related to such complications.[9] Among the hypertensive disorders that are complications of pregnancy, pre-eclampsia is one of the major causes of maternal and perinatal morbidity and mortality. The majority of deaths due to these complications are preventable if affected women receive timely and effective care.[9,20]

Although its main risk factors are studied (obesity, multiparity, nulliparity, gestational diabetes, chronic hypertension, family history, smoking, low socioeconomic status, past PE, advanced age (> 35 years) and race, [7] obesity in women is steadily increasing worldwide,[13] and has been regarded as a risk factor for PE since the early last century.[14]

In a systematic review and meta-analysis regarding maternal lipid profile and risk of PE in African pregnant women, Tesfa et al., [21] reported that the maternal serum levels of TG, TC, and LDL were significantly associated with the risk of PE. However, HDL- cholesterol was not significantly associated but it was lower in pre-eclamptic women.

There was a significant rise in TC and LDL levels in PE as compared to normal pregnancy in this study, which was similar to other reports.[21-24] During pregnancy, the plasma cholesterol level increases in response to an increase estrogen induced hepatic synthesis or failure of lipoprotein lipase to clear the plasma lipids.[14,25] High levels of cholesterol may produce free radicals via an oxidative conversion of LDL- cholesterol to oxidized LDL form stress (abnormal lipid profile) could explain the pathogenesis of PE.[9,14] Moreover, LDL-cholesterol transports cholesterol to the peripheral tissue and plays significant role in the development of atherosclerosis and cardiovascular disease.[14,26] Conversely, the mean serum TG level of the current study was higher, but not statistically significant in pre-eclamptic women as compared with controls, probably due to a small number of control (normotensive pregnant women) as reported elsewhere.[21,27,28]

In this study, the mean serum HDL-c level was similar in preeclamptic women as compared to normotensive pregnant women, respectively, similar results were reported in the studies conducted elsewhere, including in Africa.[21,23,24] HDL carries cholesterol from peripheral tissues to the liver, where it is broken down for excretion and used for synthesis of biomolecules. Higher levels of HDL lipoprotein have protective effect against hypertension and cardiovascular diseases.[14,29]

The Present study also observed significant elevated levels of the lipid parameters (TC and LDL), with insignificant altered levels of TG and HDL in the severe preeclamptic participants compared with the mild PE. Several studies observed significant elevated levels of the lipid parameters (TC, TG, LDL), with insignificant altered levels of HDL in the severe preeclamptic participants compared with the mild PE.[23,30,31] On the other hand, women with the most severe form of PE had TG levels similar to normotensive controls.[31]

Obesity and PE are associated with oxidative stress, dyslipidemia, hyperinsulinemia, insulin resistance, and impaired endothelial function.[14,32] Although BMI was significantly higher in preeclamptic cases compared to controls in the present study as also reported elsewhere,[21,23,24] dyslipidemia which is significantly evident among preeclamptic group for the comparable weights may serve as a marker of the pathogenic process of PE.[9,14]

The main limitations of this study are single measurement of lipid profile and the small number of case group (n=35) and normal pregnancy women for the control group (n=30); however, as a pilot study this shows promising results for future larger scale studies to confirm abnormal lipid profile and increased BMI as risk factors of PE.

Conclusion

The findings of this study suggest that the women who develop PE had disturbed lipid profile. The lipid profile screening as well as BMI monitoring may help preventing the PE in normal pregnancy women (control group) or reducing the PE in case group for enhancing maternal and fetal outcomes.

Declarations

Ethical Approval

The research protocol was approved by the Bio-ethics Committee of the School of Public Health at the University of Kinshasa. Kinshasa, DR Congo.

Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Authors' contributions

The first draft of this manuscript has been written by the first author A.N.M. The co-authors Y.M.T. and M.M.K. prepared Tables 1 and 2. And reviewed equally the manuscript. The co-authors JP.E-M, supervised all the manuscript. The J.K.T contributed to supervise all the work and to correspond with the Journal.

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Availability of data and materials

Not applicable. However, the study results will report to individuals sample donors with proper explanations.

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