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Serum Lipid Profiles in Preeclamptic versus Normotensive Pregnancies: A Case-Control Study

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Abstract

Objectives: To estimate the serum lipid levels in women with preeclampsia and normotensive pregnancy and also determine the correlation between the mean serum lipid levels and the severity of the disease.

Methods: This was a case-control study that involved 116 (58 preeclamptic and 58 normotensive control group) consenting pregnant women at the Federal Medical Centre, Owerri, Nigeria. Five milliliter (5 ml) of their venous blood was collected after an overnight fast of 8–12 hours, and analysed for triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and very low-density lipoprotein cholesterol (VLDL-C) using standard enzymatic methods. The primary outcome measure was the mean serum lipid levels in women with preeclampsia and normotensive pregnancies. Pearson's correlation analysis was employed.

Results: There was a statistically significant difference in the levels of triglyceride (TG) and very low-density lipoprotein cholesterol (VLDL-C) between the preeclamptics and their normotensive control respectively ($p = 0.002$). There was strong positive correlation between the abnormal levels of very low density lipoprotein (LDL) and severe preeclampsia. In addition, there was also a weak positive correlation between the severity of the disease and the levels of triglyceride (TG).

Conclusion: Dyslipidemia, especially elevated LDL and TG levels, is linked to preeclampsia and may influence its progression. Monitoring serum lipid levels in pregnant women may be crucial for managing and detecting preeclampsia early. Further research on lipid-lowering surveillance is recommended.

Keywords: Preeclampsia; Serum lipid level; Low-density lipoprotein; High-density lipoprotein; Hypertension

Introduction

Preeclampsia is a pregnancy-specific disorder characterised by pregnancy-induced hypertension (blood pressure greater than or equal to 140/90 mmHg) on two occasions, at least six hours apart, and proteinuria of greater than or equal to 300 mg/24 hours or 2+ dipstick after 20 weeks of gestation in previous normotensive women. It occurs in about 2–8% of pregnancies [1,2]. Although most affected pregnancies deliver at term or near term with good maternal and foetal outcomes, these pregnancies are at increased risk for maternal and/or foetal mortality or serious morbidity [3,4].

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In Nigeria, about 11% of maternal mortality is caused by preeclampsia/eclampsia [5]. In 2006, a systematic review reported hypertensive disorders to be responsible for 16.1% of all maternal deaths in developed countries, 9.1% in each of Africa and Asia, and 25.7% in Latin America and the Caribbean [6]. Ten million women develop preeclampsia each year around the world. About 76,000 of these women and about 500,000 babies die from this disorder annually [7]. In developing countries, a woman is seven times more likely to develop preeclampsia compared to women in developed countries [8].

Despite extensive research, important pathophysiological aspects of this disease are unknown, hindering the development of preventive and therapeutic strategies. No wonder this disease entity is termed a disease of theories because of the multiple hypotheses involved in its discussion [9], which include immunologic, hematologic, genetic, and environmental factors. Central to the effects of preeclampsia are the resulting presence of uteroplacental hypoxia, an imbalance in angiogenic and anti-angiogenic proteins, oxidative stress, maternal vascular endothelial dysfunction, and elevated systemic inflammation [10,11]. It occurs more often in women with preexisting hypertension, obesity, antiphospholipid syndrome, a prior history of preeclampsia, and low serum calcium, especially in environments where dietary calcium intake is low [10-12]. Preeclampsia is also common in women in their first pregnancy, a change in paternity, and multiple gestation [13-15]. The aforementioned pathophysiologic mechanisms occur in the first half, and the symptoms of hypertension and proteinuria become apparent in the second half of pregnancy.

Physiologically, serum lipid levels in the first trimester are similar to those prior to pregnancy, only for the levels to change significantly in the second and third trimesters, wherein total cholesterol (TC) increases by up to 50%, triglyceride (TG) rises by two to fourfold, high density lipoprotein cholesterol (HDL-C) increases by 30%, and low density lipoprotein cholesterol (LDL-C) increases by 50% [16, 17]. These changes in lipid metabolism lead to the accumulation of fats that serve as calorie reserves for both mother and fetus during this stage of pregnancy and lactation. Additionally, cholesterol and triglycerides are necessary for placental steroid synthesis and placental oxidation/membrane formation, respectively [17-19]. These changes are believed to increase the risk of preeclampsia by inducing endothelial dysfunction secondary to oxidative stress, metabolic syndrome (insulin resistance, hyperglycemia, central obesity, hypertension, and dyslipidemia), and/or dysregulation of lipoprotein lipase that breaks down lipids [20].

Presently, no laboratory investigation provides a reliable diagnosis of preeclampsia. This becomes a problem because the definitive treatment is delivery of the fetus and placenta, often times exposing the preterm fetus to risk, not minding the occurrence of atypical preeclampsia, wherein hypertension or proteinuria may be absent [21]. However, the quest for optimal perinatal outcomes and a reduction of maternal risks influences the timing of the delivery [10]. Since endothelial injury plays a major role in the etiopathogenesis of the disease [22], lipid accumulation in macrophages and arterial intimal cells has been shown to cause endothelial injuries [23]. The study is therefore aimed at estimating the mean serum lipid levels in women with preeclampsia and normotensive pregnancies and determining the relationship between the mean serum lipid levels and the severity of the disease.

Methods

1.1 Study design: The study was a case-control study of preeclamptics as cases and normotensive pregnant women as controls.

1.2 Study setting: This study was conducted among pregnant women receiving antenatal care in the Department of Obstetrics and Gynaecology of the Federal Medical Centre (FMC), Owerri, Nigeria.

1.3 Study population: The study population comprised of pregnant women with an established diagnosis of preeclampsia as cases and normotensive pregnant women as controls**.**

1.4 Inclusion criteria: Those included in the study were women with singleton pregnancies with a diagnosis of preeclampsia (cases) and normotensive women with singleton gestation who are more than 20 weeks pregnant.

1.5 Exclusion criteria: The exclusion criteria were pregnant women with molar pregnancy, multiple gestations**,** diabetes mellitus**,** chronic hypertension**,** renal disease**,** obesity, a known history of dyslipidemia, human immunodeficiency virus (HIV) infection, cardiac disease, and liver disease.

Sampling technique

Consecutive, eligible, consenting preeclamptic women were enrolled from among attendees in the antenatal clinic and those who presented to the ward. Each eligible consenting normotensive woman following a preeclamptic was enrolled from the same antenatal population to serve as control. Unbooked preeclamptic presenting for the first time to the labour ward who gave informed consent were also recruited. The cases and the controls were matched for age and parity.

Sample size determination

The sample size calculation was calculated using a 95% confidence interval with 0.05 precision. For the purpose of this study, a prevalence of 1.7% of preeclampsia in Nnewi, Nigeria, as reported by Mbachu et al. [24] was used.

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$n = 2Z^2PQ/d^2$. Where: n = sample size, Z = standard normal deviation at 95% confidence interval, which is 1.96, d = degree of precision (taken as 0.05), P = proportion of the target population (estimated at 1.7%, which is 1.7/100 = 0.017), Q = alternate proportion (1-P). The minimum sample size was estimated to be 52. However, considering possible unforeseen attrition factors, 10% of this value was added to make it up to 58 pregnant women. In this study, 116 patients were recruited (58 each for the study group and the control).

Data collection

A detailed explanation of the study was given to the selected participants. A written informed consent was obtained. The participants were interviewed by the investigators, who were occasionally aided by trained assistants, comprising junior resident doctors and house officers in the department, with the aid of a semi-structured, pre-tested questionnaire.

Study procedure

The participants were allowed to sit and rest for about 5 minutes. The cuff of the Accoson mercury sphygmomanometer was applied around the upper left arm at the level of the heart. The Korotkoff sounds 1 and 5 (disappearance) were used to get the systolic and diastolic blood pressures, respectively [25]. Elevated blood pressure was repeated after at least 4 hours. The subjects were weighed, and urine protein estimation was done using dipstick measurement of a clean midstream urine specimen.

Sample collection and biochemical analysis

Following written informed consent and a response to the questionnaire, the participant was registered on the note book and allotted a serial number. Aseptic measures were used to draw 5 ml of venous blood from the antecubital vein of all the participants after an overnight fast of 8–12 hours and deliver it into appropriately labelled plain containers. The sample was collected by the investigator with occasional aid from trained assistants, who comprised junior residents and house officers in the department. The sample was analysed in the chemical pathology laboratory of the institution using a Maxmat auto-analyzer. Prior to the analysis, the samples were centrifuged at 3,000 revolutions per minute for 5 minutes to separate the serum from the red cells, after which the serum was analysed immediately or stored in the refrigerator at 2–8 °C. Analysis was done for serum TG, TC, and HDL-C by using chemical precipitation reagents, viz., the Randox kit for HDL and the Biosystem kits for TG and TC. The chylomicron fraction was separated from the sample with the aid of phototungstate contained in the reagents before the supernatant was used for the final spectrophotometric analysis. VLDL-C was calculated as 1/5 of TG [26]. Serum LDL-C was deduced from Frederickson-Friedwald's formula [27]; LDL-C (mmol/L) = TC-(TG/2.2+HDL). The reference ranges for serum lipid in FMC, Owerri, Nigeria, are: TC ≤ 230 mg/dl; TG 36–165 mg/dl; HDL ≥35 mg/dl; LDL <130 mg/dl; and VLDL 2–30 mg/dl.

Outcome measures

The primary outcome measure was to determine the mean serum lipid levels in women with preeclampsia and in women with normotensive pregnancies. The secondary outcome measures were to establish the relationship between the mean serum lipid levels and fetomaternal parameters (age, parity, and gestational age) in the two groups and then the correlation between the mean serum lipid levels and the severity of the disease.

Statistical analysis

Software Package for Social Science (SPSS) for version 20.0 for Windows® (SPSS Inc., Chicago, IL, USA) was used to analyse the data. The difference between the mean levels of serum lipids in the two groups was determined using a Student's t-test. Categorical variables were analysed using the chi-square, and continuous variables were analysed with ANOVA where applicable. Pearson's correlation was used to find a correlation between lipid fractions and the severity of preeclampsia. The level of statistical significance was set at a 95% confidence interval.

Results

During the study period, 116 participants were recruited for the study (58 preeclamptics and 58 normotensive controls). Table 1 shows the sociodemographic characteristics of the study population. A large proportion of the women, 80 (69.0%), had tertiary education. Most of the patients were either civil servants (31%), traders (27.6%), or housewives (27.6%). Only a small percentage were students (13.8%).

The comparison of clinical characteristics between the cases and the controls is shown in Table 2. There was no statistically significant difference between the mean age (29.83±4.76 years vs. 29.48±4.29 years, p = 0.68), the mean parity (2.62±1.82 vs. 2.43±1.63, p = 0.60), and the mean gestational age (33.81±4.33 weeks vs. 34.97±4.48 weeks, p = 0.26) between the preeclamptic and normotensive control groups, respectively. In addition, there was no difference in the mean body mass index between the preeclamptics and the control groups (24.4±0.44 kg/m^2 vs. 24.13±0.85 kg/m^2, p = 0.18). However, there were statistically significant differences in the mean systolic blood pressure (164.22±12.90 mmHg, vs. 115.17±9.36 mmHg, p < 0.01) and the mean diastolic blood pressure (106.20±12.29 mmHg, vs. 70.00±8.11 mmHg, p < 0.01), respectively, between the preeclamptics and the control normotensive group (Table 2).

The mean levels of triglycerides (TGs) and very low-density lipoprotein (VLDL) were notably higher in the preeclamptic group compared to the control group (p<0.05).

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However, no significant differences were observed in total cholesterol (TC), low-density lipoprotein (LDL), or high-density lipoprotein (HDL) between the groups ($p > 0.05$), as illustrated in Table 3.

Regarding the different age groups between the preeclamptic and normotensive control groups, there was a statistically significant difference in all lipid fractions for ages 26–30 years ($p < 0.05$). In the 36–40 years age group, all lipid fractions except HDL showed significant differences. However, only total cholesterol (TC), HDL, and LDL showed significant differences in the 20–25 years age group between the two groups (Table 4).

Table 5 shows the relationship between mean serum lipid level and gestational age. There was a statistically significant difference in the mean serum levels of TG, HDL, and VLDL between 26 and 30 weeks of gestational age in both groups. Also, there was also a statistically significant difference in the level of TG in the late third trimester (36–40 weeks) between both groups ($p<0.05$).

Table 1: Socio-demographic characteristics of the study population.

Variable	N	%
Age (years)		
20-25	28	24.1
26-30	45	38.8
31-35	28	24.1
36-40	15	12.9
Education		
primary	6	5.2
secondary	30	25.9
tertiary	80	69
Occupation		
civil servant	36	31
housewife	32	27.6
student	16	13.8
trader	32	27.6
Husband's education		
primary	6	5.2
secondary	39	33.6
tertiary	71	61.2
Husband's occupation		
civil servant	48	41.4
unemployed	4	3.4
trader	50	43.1
self employed	14	12.1

Table 2: Comparison of clinical characteristics between the cases and the controls.

	Severity	N	Mean	Std. Deviation	T(p-value)
Age	Normotensive	58	29.4828	4.28887	0.41(0.68)
	Pre-eclamptic	58	29.8276	4.75777	
Parity	Normotensive	58	2.431	1.6342	0.59(0.60)
	Pre-eclamptic	58	2.6207	1.82408	
Gestational age	Normotensive	58	34.9655	4.47592	1.12(0.26)
	Pre-eclamptic	58	33.8103	4.33451	
BMI	Normotensive	58	24.1276	0.85481	1.36(0.18)
	Pre-eclamptic	58	24.4397	0.50608	
Systolic blood pressure	Normotensive	58	115.1724	9.36424	23.43(0.00)*
	Pre-eclamptic	58	164.2241	12.90321	
Diastolic blood pressure	Normotensive	58	70	8.11107	18.72(0.00)*
	Pre-eclamptic	58	106.2069	12.29428	

Std = Standard, BMI = Body mass index

Table 3: Comparison between mean serum lipid levels in preeclamptic and normotensive women.

LIPID FRACTIONS	NORMOTENSIVE	MILD PREECLAMPSIA	SEVERE PREECLAMPSIA	F(p value)
	MEAN+SD	MEAN +SD	MEAN+SD	
TG	140.96±49.38	193.51±88.97	234.47±113.04**	6.80(0.002)*
TC	219.71±58.26	211.69±55.46	224.62±81.16	0.25(0.779)
HDL	46.34±15.41	44.33±20.73	45.60±19.03	0.10(0.908)
LDL	130.62±53.79	134.49±64.26	136.89±68.12	0.70(0.932)
VLDL	28.25±9.82	38.69±17.80	46.90±22.62**	6.77(0.002)*

TG = Triglyceride, TC = Total cholesterol, HDL = High density lipoprotein, LDL = Low density lipoprotein,
VLDL = Very low density lipoprotein
Key: **= statistically significant with the post hoc test
*= statistically significant

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Table 4: Relationship between mean serum lipid levels and age.

Lipids	Age Group (years)	Mean Normotensive (mg/dl)	Lipid Mild Preeclampsia (mg/dl)	Level ± SD Severe Preeclampsia (mg/dl)	F	p value
TG						
	20-25	165.05±40.28	189.29±55.58	205.53±40.93	1.24	0.31
	26-30	99.33±11.29	180.61±104.70	259.77±116.13	5.51	0.01*
	31-35	220.77±97.29	181.85±48.09	257.31±143.60	0.72	0.49
	36-40	87.70±10.23	201.30±70.83	159.13±13.58	4.08	0.04*
TC						
	20-25	181.52±27.23	217.64±24.56	184.90±41.07	4.92	0.02*
	26-30	186.87±33.06	199.56±64.34	264.19±82.20	4.63	0.02*
	31-35	244.80±72.49	303.80±29.67	253.03±58.46	1.37	0.27
	36-40	222.60±18.71	252.77±22.61	117.00±25.87	57.42	0.00*
HDL						
	20-25	61.95±17.86	41.61±12.93	40.70±8.21	6.4	0.01*
	26-30	26.73±11.12	50.19±15.65	39.40±18.89	5.75	0.01*
	31-35	48.20±19.87	40.45±15.41	51.97±28.45	0.36	0.69
	36-40	34.40±2.65	38.81±1.63	47.30±29.58	0.48	0.63
LDL						
	20-25	67.75±26.95	148.57±39.92	103.20±40.37	12.68	0.00*
	26-30	140.20±19.87	109.07±48.65	172.83±71.96	5.97	0.01*
	31-35	148.12±70.67	221.95±29.73	158.29±42.75	2.75	0.08
	36-40	170.80±8.88	173.71±9.52	46.37±12.8	69.44	0.00*
VLDL						
	20-25	33.00±8.72	37.81±11.07	41.10±8.18	1.25	0.31
	26-30	20.10±2.32	36.11±20.94	51.97±23.24	5.46	0.01*
	31-35	44.16±19.47	36.35±9.64	51.46±28.73	0.72	0.49
	36-40	17.50±3.67	40.24±14.18	31.83±2.74	4.07	0.04*

TG = Triglyceride, TC = Total cholesterol, HDL = High density lipoprotein, LDL = Low density lipoprotein, VLDL = Very low density lipoprotein
*=statistically significant

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Table 5: Relationship between mean serum lipid level and gestational age.

Lipids	Gestational age (weeks)	Mean Normotensive (mg/dl)	Lipid Mild Preeclampsia (mg/dl)	Level ± SD Severe Preeclampsia (mg/dl)	F	p value
TG						
	20-25	191.90±26.55	0.00±0.00	141.61±0.00	6.37	0.06
	26-30	98.10±13.97	176.90±87.87	184.83±36.03	5.18	0.02*
	31-35	173.37±66.21	150.70±40.93	239.37±158.34	1.78	0.18
	36-40	152.26±55.95	208.20±105.12	276.27±122.41	4.88	0.01*
TC						
	20-25	178.45±22.13	0.00±0.00	150.40±0.00	2.66	0.18
	26-30	164.90±22.74	212.90±60.85	193.18±72.83	0.56	0.58
	31-35	186.94±44.01	217.96±52.80	251.13±101.03	2.8	0.08
	36-40	226.64±59.37	245.99±56.91	244.98±76.60	0.37	0.69
HDL						
	20-25	54.45±28.12	0.00±0.00	85.50±0.00	2.16	0.22
	26-30	23.70±12.93	45.50±10.39	36.88±9.79	4.41	0.03*
	31-35	46.87±14.49	52.20±14.27	58.16±42.86	0.61	0.55
	36-40	47.86±22.22	45.07±14.97	41.85±16.01	0.45	0.64
LDL						
	20-25	90.60±50.57	0.00±0.00	62.10±0.00	0.56	0.49
	26-30	83.85±36.31	132.00±53.25	118.78±63.57	0.76	0.47
	31-35	106.16±43.55	135.60±66.52	165.00±83.20	2.6	0.92
	36-40	146.34±67.50	157.83±49.89	147.91±64.43	0.25	0.78
VLDL						
	20-25	38.35±5.25	0.00±0.00	28.30±0.00	6.51	0.06
	26-30	19.60±2.77	35.40±17.55	36.97±7.21	5.19	0.02*
	31-35	34.66±13.23	30.13±8.21	47.90±31.68	1.78	0.18
	36-40	146.34±67.50	157.83±49.89	147.91±64.42	0.25	0.78

TG = Triglyceride, TC = Total cholesterol, HDL = High density lipoprotein, LDL = Low density lipoprotein, VLDL = Very low density lipoprotein
*=statistically significant

Table 6: Relationship between mean serum lipid levels with parity.

Lipids	Parity	Mean Normotensive (mg/dl)	Lipid Mild Preeclampsia (mg/dl)	Level ± SD Severe Preeclampsia (mg/dl)	F	p value
TG						
	0	213.2±95.89	139.31±43.32	239.30±117.98	3.92	0.03*
	1	143.4±66.27	164.0±44.33	184.58±41.74	0.97	0.39
	≥2	143.45±64.37	184.58±93.49	249.81±126.28	2.73	0.08
TC						
	0	232.04±63.54	238.26±52.31	233.11±77.06	0.04	0.96
	1	152.65±8.60	199.36±43.92	206.58±68.86	1.61	0.23
	≥2	191.0±36.48	215.14±56.73	225.88±90.29	0.42	0.66
HDL						
	0	46.33±19.58	44.01±19.14	46.71±32.15	0.05	0.95
	1	39.90±31.63	48.88±13.74	53.65±15.57	0.71	0.51
	≥2	45.23±10.73	49.70±17.66	41.03±18.59	0.74	0.48
LDL						
	0	140.25±61.40	152.17±64.29	148.50±66.38	0.17	0.84
	1	83.95±36.19	134.24±64.13	116.11±59.22	1.06	0.36
	≥2	112.65±67.03	128.33±40.28	137.22±73.61	0.33	0.72
VLDL						
	0	42.67±19.18	27.97±8.55	47.87±23.60	3.87	0.03*
	1	28.70±13.27	32.80±8.85	36.92±8.36	0.97	0.39
	≥2	28.65±12.87	36.91±18.69	49.96±25.27	2.74	0.08

TG = Triglyceride, TC = Total cholesterol, HDL = High density lipoprotein, LDL = Low density lipoprotein, VLDL = Very low density lipoprotein
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The relationship between mean serum lipid levels and parity is shown in Table 6. There was a statistically significant difference in the mean serum lipid levels of TG and VLDL in primigravidae (p = 0.03) between the preeclamptic and the control normotensive participants. Though there was no difference in mean serum TG and VLDL between primiparous and multiparous participants in both groups (p > 0.05).

Table 7 shows the correlation between mean serum lipid levels and the severity of preeclampsia. Using Pearson's correlation coefficient, considering only people who had preeclampsia, it was found out that there was a strong positive correlation between the abnormal levels of very low density lipoprotein (LDL) and severe preeclampsia. In addition, there was also a weak positive correlation between the severity of the disease and the levels of triglyceride (TG) (Table 7).

The distribution of parity among normotensive, mild, and severe preeclampsia is shown in Figure 1. Figure 2 shows the distribution of mean serum lipid levels among the normotensive, mild, and severe preeclamptic groups.

Table 7: The correlation between mean serum lipid levels and the severity of preeclampsia.

Lipid fraction	R	P value
TG	0.29	0.03*
TC	0.22	0.1
HDL	-0.075	0.57
LDL	0.127	0.34
VLDL	0.51*	0.001*

TG = Triglyceride, TC = Total cholesterol, HDL = High density lipoprotein, LDL = Low density lipoprotein, VLDL = Very low density lipoprotein
*= statistically significant.

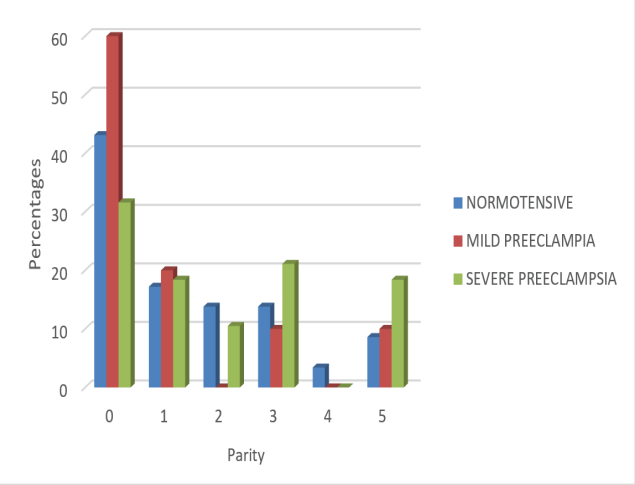

Figure 1: Distribution of parity among normotensive, mild and severe preeclampsia.

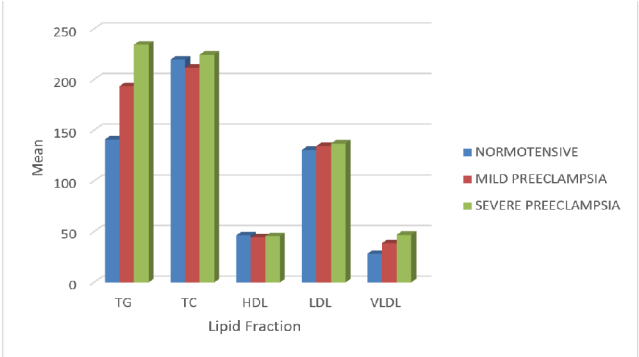

Figure 2: Distribution of mean serum lipid levels among the normotensive, mild and severe preeclamptic groups.

Discussion

The principal findings from the study showed that there was a statistically significant difference in the levels of triglyceride (TG) and very low-density lipoprotein (VLDL-C) between the preeclamptics and their normotensive control. In addition, there was a positive correlation between these lipids and the severity of the disease.

In this study, the cases and the controls were matched for age and parity. It was found that the participants were ages between 20 and 40, which is the reproductive age group. The mean age for the cases and controls was similar to the findings in some other studies [28-30]. Pre-eclampsia was found most in primigravidae, representing 60% of those with a mild form of the disease and 31.6% of those who had a severe disease. This is in keeping with the available body of knowledge [31].

This study showed significantly higher values of tryglyceride and very low-density lipoprotein in the preeclamptic groups when compared with the normotensive group. This finding has been commonly demonstrated in other studies [30, 32-34]. There was no significant difference in the values of total cholesterol and low-density lipoprotein. This is comparable with other studies [30,33]. Singh et al. [35], apart from finding a significant difference in very low-density lipoprotein, also found a significant difference in the values of total cholesterol and low density lipoprotein [35,36]. This is in contradistinction to the findings of this study. The difference might be due to variation in the study population. While in this study, women were selected from 20 weeks gestational age and above, in the above study by Singh et al. [35], the study population was chosen from pregnant women prior to 20 weeks gestational age.

This study also found a significant difference in all lipid fractions in the age group of 26 to 30 years. A similar finding was seen in the age group of 36 to 40 years, where all lipid fractions but the high-density lipoprotein were found to be significantly different across the groups. This may indicate an increase in the chances of dyslipidemia with increasing age, as is known [37,38].

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The period between the late second trimester and early third trimester (26–30 weeks) and the period around term (36–40 weeks) showed significantly higher values of triglyceride and very low-density lipoprotein in the preeclamptic groups, albeit abnormal. There was also a significant difference in the values of HDL in the gestational age group of 26–30 weeks. This is similar to the findings of Irinyenikan et al. [28], where triglyceride, high-density lipoprotein, and very low-density lipoprotein were significantly higher in the third trimester in preeclamptics when compared to normotensive women [28]. Other studies have made a similar suggestion [37,39].

Primigravidity is a common risk factor for preclampsia, and studies have often depicted the disease condition that commonly occurs in primigravidae [14]. In this study, it was equally found that primigravidity was not just a risk factor for the disease, but the findings of statistically significant differences in the triglyceride and very low density lipoprotein were reflective of what was found in the general population studied.

There was a positive correlation between serum levels of triglyceride and very low-density lipoprotein and the severity of the disease. In this study, even though it is only the VLDL that shows a strong positive correlation, this is similar to what was found in some other studies [28,32]. The study by El Khouly et al. [36] did not find a positive correlation between VLDL and the severity of the disease, probably due to the recruitment of women of gestational age less than 20 weeks into the study population. It is believed that the elevated level of very low density lipoprotein is due to hypertriglyceridemia, leading to enhanced entry of very low density lipoprotein that carries endogenous triglyceride into circulation and, by doing so, gets deposited on the maternal uterine and renal vascular endothelium, causing injury to it [40].

The strength of this study lies in the selection of controls from eligible, consenting normotensive pregnant women immediately following each preeclamptic participant, ensuring that cases and controls were matched for age and parity, thus minimizing bias. Additionally, the large sample size of 116 enhances the study's power compared to previous studies. However, a limitation is the lack of consideration for dietary differences among participants. Furthermore, the results may not be applicable to participants who developed preeclampsia early, around 20 weeks of gestation, due to the inclusion criteria of women with ≥ 20 weeks of gestation.

Future Direction

In the face of disappointing biochemical and clinical tests done in predicting the women at risk of developing the disease, testing the triglyceride levels and very low density lipoprotein in high-risk women may be useful in the early detection of parturients who will develop the disease.

Conclusion

Preeclamptic women have higher lipid levels compared to their normotensive counterparts. Although lipid fractions increase during pregnancy, the rise is more pronounced in preeclamptic women, particularly in triglycerides and very low-density lipoprotein (VLDL). The increase in VLDL levels is directly proportional to the severity of preeclampsia. Given the limitations of current biochemical and clinical tests in predicting those at risk, measuring triglyceride and VLDL levels in high-risk women could be valuable for early detection of preeclampsia. Further research on lipid lowering surveillance is recommended.

Declaration of Competing Interest

The authors declare no conflict of interest.

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Data and materials availability

The data and materials for this study are available upon formal request from the authors.

Ethical approval

The ethical approval for the study was obtained from the Federal Medical Center, Owerri, Nigeria, Ethics Review Committee with reference number; 03294. The study was conducted according to ethical principles for human subjects according to Helsinki declaration.

Consent for the study

A written informed consent was obtained from all the participants prior to recruitment into the study.

Consent for publications

This was obtained from the study participants.

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