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ORIGINAL ARTICLE

Trimester-Specific Serum Lipid Profiles in Pregnant Women in Enugu, Nigeria: A Cross-Sectional Comparative Study

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ABSTRACT

Normal gestation is accompanied by marked maternal metabolic adaptations, including alterations in lipid profiles required to support fetal growth. However, standard laboratory reference intervals derived from non-pregnant populations remain inadequate for evaluating gravid women, particularly in sub-Saharan African settings where localized data are limited. This study evaluated the trimester-specific changes in serum lipid profiles among pregnant women in Enugu metropolis, South-East Nigeria, relative to non-pregnant controls. A hospital-based cross-sectional study was conducted among 200 women categorized into four groups (n = 50 per group): first trimester, second trimester, third trimester, and non-pregnant controls. Venous blood samples were collected after an overnight fast, and serum Total Cholesterol (T-CHL), Triglycerides (TG), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), and Very Low-Density Lipoprotein Cholesterol (VLDL-C) were quantified spectrophotometrically. Data were analyzed using One-Way ANOVA with statistical significance set at $p < 0.05$. Serum T-CHL, TG, HDL-C, and VLDL-C concentrations were significantly higher across all trimesters compared to controls ($p < 0.001$). T-CHL (6.9 ± 0.5 mmol/L), LDL-C (5.1 ± 0.4 mmol/L), and HDL-C (2.8 ± 0.3 mmol/L) peaked significantly during the second trimester. Conversely, TG (5.3 ± 0.4 mmol/L in the second trimester) and VLDL-C (2.4 ± 0.1 mmol/L in the third trimester) demonstrated fluctuating trends as gestation advanced. **Conclusion**: Gestation induces, trimester-specific hyperlipidemia in Nigerian women. Establishing localized trimester-specific reference intervals is critical for distinguishing physiological adaptations from pathological dyslipidemia during routine antenatal care.

Keywords: Antenatal Care; Gestation; Lipid Profile; Maternal dyslipidemia; Pregnancy Trimesters, Nigeria

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1.0 INTRODUCTION

Maternal physiological adaptation during pregnancy involves profound anatomical, biochemical, and metabolic changes designed to support fetal growth and development while simultaneously preparing the maternal organism for parturition and lactation (Herrera, 2002; Kato & Sekiya, 2020). Among these systemic adaptations, alterations in lipid metabolism are particularly striking (Ghio et al., 2016; Sattar et al., 2017). Throughout normal gestation, maternal lipid metabolism undergoes a biphasic transition characterized by an initial anabolic phase in early and mid-pregnancy (marked by enhanced fat storage and increased lipogenesis) followed by a catabolic phase in late pregnancy (characterized by accelerated lipolysis, hyperlipidemia, and enhanced fat mobilization) (Herrera, 2002; Ziegler & Fomon, 2018). These metabolic shifts are tightly regulated by hormonal alterations, specifically marked by progressive increases in circulating concentrations of estrogen, progesterone, human placental lactogen, and cortisol (Ghio et al., 2016; Kato & Sekiya, 2020). Estrogen acts as a primary physiological driver by stimulating hepatic biosynthesis of endogenous triglycerides and upregulating very-low-density lipoprotein (VLDL) production, while concomitantly altering peripheral tissue sensitivity to insulin (Herrera, 2002; Sattar et al., 2017). Consequently,

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maternal circulating lipids including Total Cholesterol (T-CHL), Triglycerides (TG), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), and Very Low-Density Lipoprotein Cholesterol (VLDL-C) undergo marked, progressive fluctuations across the first, second, and third trimesters of pregnancy (Okafor & Chime, 2019; Sattar et al., 2017). In clinical practice, understanding the physiological boundaries of gestational hyperlipidemia is critical because extreme deviations from normal trimester-specific lipid profiles are strongly associated with adverse obstetric and neonatal outcomes (Ghio et al., 2016; World Health Organization [WHO], 2021). Exaggerated dyslipidemia during pregnancy has been directly implicated in the pathogenesis of gestational diabetes mellitus (GDM), pregnancy-induced hypertension, preeclampsia, macrosomia, and intrauterine growth restriction (Kato & Sekiya, 2020; Okafor & Chime, 2019). Furthermore, persistent maternal dyslipidemia can induce endothelial dysfunction, oxidative stress, and long-term cardiovascular risks for both the mother and the developing fetus through the mechanism of metabolic programming (Sattar et al., 2017; Ziegler & Fomon, 2018). Despite the acknowledged significance of lipid metabolism in obstetric care, normative reference intervals for gestational lipid parameters remain widely unstandardized across diverse geographic, ethnic, and socio-cultural populations (Kato & Sekiya, 2020; WHO, 2021). Standard laboratory reference ranges derived from non-pregnant populations are entirely inadequate for interpreting lipid panels in gravid women, often leading to misdiagnosis or failure to identify pathological gestational hyperlipidemia (Okafor & Chime, 2019; Sattar et al., 2017). In developing countries, particularly within sub-Saharan Africa, regional data mapping trimester-specific lipid adaptations remain sparse, constraining the capacity of clinicians to establish localized diagnostic thresholds (Okafor & Chime, 2019; WHO, 2021). Geographic and socio-demographic factors—including nutritional status, dietary patterns, urbanization, and lifestyle variations—further modulate maternal lipid profiles during gestation (National Bureau of Statistics [NBS], 2022; WHO, 2021). In urban and semi-urban settings such as the Enugu metropolis in South-East Nigeria, shifting nutritional transitions toward westernized diets rich in refined carbohydrates and saturated fats interact with underlying physiological pregnancy adaptations (NBS, 2022; Okafor & Chime, 2019). This nutritional shift potentially exacerbates gestational lipid burdens among expectant mothers (Ghio et al., 2016; NBS, 2022). Despite these emerging public health concerns, comprehensive empirical data quantifying trimester-specific lipid fluctuations among pregnant populations in South-East Nigeria remain limited (NBS, 2022; Okafor & Chime, 2019). Routine antenatal care protocols in many regional healthcare facilities frequently omit comprehensive lipid profiling, prioritizing basic hematological and infectious screening while overlooking metabolic complications that could herald gestational vascular disorders (Okafor & Chime, 2019; WHO, 2021). Addressing this critical knowledge gap requires rigorous empirical investigation to establish baseline trimester-specific lipid profiles and evaluate their patterns relative to non-pregnant controls (Albers et al., 1978; Fredrickson et al., 1967; Friedewald et al., 1972). Such data are essential for improving maternal-fetal medicine, refining clinical diagnostic guidelines, and designing targeted metabolic monitoring strategies during antenatal care visits (Fossati & Prencipe, 1982; Kato & Sekiya, 2020; WHO, 2021).

This study is underpinned by the pressing clinical need to understand normal versus pathological metabolic adaptations during pregnancy, particularly in resource-constrained sub-Saharan African settings where maternal morbidity and mortality remains elevated (Okafor & Chime, 2019; WHO, 2021). Although pregnancy-induced hyperlipidemia is a recognized physiological phenomenon, the precise temporal kinetics and magnitude of lipid fraction changes across specific trimesters among Nigerian women have not been thoroughly integrated into local obstetric guidelines (NBS, 2022; Okafor & Chime, 2019). Therefore, this study evaluated the trimester-specific changes in serum lipid profiles among pregnant women in Enugu metropolis, South-East Nigeria. By conducting a comprehensive hospital-based cross-sectional study across the first, second, and third trimesters within the Enugu metropolis, this research generates localized, empirical evidence that addresses the over-reliance on generalized or Caucasian-derived reference ranges (NBS, 2022; Okafor & Chime, 2019).

2.0 MATERIALS AND METHODS

2.1 Study area

The study was conducted within the Enugu metropolis, Enugu State, Nigeria. Participants were consecutively recruited from the Antenatal Care (ANC) clinics of major tertiary and secondary healthcare facilities located within the metropolis.

2.2 Study design

A cross-sectional comparative design was employed. Demographic parameters, clinical history, and physiological variables were captured via interviewer-administered questionnaires. The study was conducted between February and July 2023.

2.3 Study population

This hospital-based cross-sectional study recruited a total of 200 female subjects. The cohort was stratified into four groups ($n = 50$ per group): First trimester pregnant women (1-13 weeks), Second trimester pregnant women (14-27 weeks), Third trimester pregnant women (28-40 weeks) and Non-pregnant women of reproductive age (Control group). Both urban and rural dwellers receiving care at the facilities were included. Participation was entirely voluntary, and informed consent was obtained after explaining the research objectives using structured questionnaires and consent forms.

2.4 Inclusion and exclusion criteria

Confirmed singleton pregnancy with defined gestational age (1-40 weeks across trimesters), active and regular attendance at antenatal care follow-up clinics, Provision of signed informed consent and apparently healthy non-pregnant women as controls with regular menstrual cycles. Participants were not included if they had Uncertain gestational age or multiple gestations, Pre-existing metabolic disorders (e.g., diabetes mellitus, pre-existing hypertension, or overt renal/hepatic disease), History of dyslipidemia or use of lipid-lowering medications and Refusal or lack of consent.

2.5 Ethical clearance

Ethical clearance and institutional approval were granted by the Department of Medical Laboratory Science and the Institutional Ethics Committee of Enugu State University Teaching Hospital (ESUTH). The study adhered strictly to the ethical principles of the Declaration of Helsinki.

2.6 Sample collection and preparation

Ten milliliters (10 ml) of venous blood was collected aseptically from each participant following an overnight fast (10-12 hours) using sterile disposable syringes. Samples were dispensed into plain vacuum tubes (10 ml) and allowed to clot at room temperature. Serum was harvested by centrifugation at 3000 rpm for 5 minutes. Aliquots of clear, non-hemolyzed serum were stored frozen at $-20°C$ in clean containers until biochemical analysis.

2.7 Assay procedure

Blood sample was collected from each participant in the mornings after an overnight fasting, left to clot for some hours and centrifuged at 3000 rpm, and then serum was obtained for lipid profile determination.

2.8 Lipid Profile Test

Serum lipid parameters including; Total Cholesterol (T-CHL), Triglycerides (TG), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), and Very Low-Density Lipoprotein Cholesterol (VLDL-C), were quantified spectrophotometrically using standard enzymatic assay kits on an SFRI BSA 3000 automated/semi-automated spectrophotometer. Low density Lipoprotein and Very Low Density Lipoprotein Cholesterol was calculated using the Friedewaid's equation (Friedewaid et al, 1972).

$$\text{VLDL-C} = \frac{\text{Triglycerides (TG)}}{2.2} \text{ (mmol/L)}$$

$$\text{LDL-C} = \text{T-CHL} - (\text{HDL-C} + \text{VLDL-C}) \text{ (mmol/L)}$$

2.9 Statistical analysis

Data were analyzed using the Statistical Package for the Social Sciences (SPSS) software for Windows (Version 21.0). Continuous variables are presented as Means ± Standard Deviation (SD), while categorical variables are presented as frequencies and percentages. Group comparisons were evaluated using One-Way Analysis of Variance (ANOVA) followed by Tukey`s HSD post-hoc tests. Statistical significance was established at $p < 0.05$.

3.0 RESULTS

3.1 Demographic Distribution of the sampled population

The socio-demographic distribution reflects typical antenatal clinic attendance demographics in South-East Nigeria. The majority of participants fall within the prime reproductive age brackets (26-41 years, accounting for 74% combined), with married women constituting the overwhelming majority (92.5%). Most participants had attained secondary or primary education, ensuring sufficient baseline understanding during questionnaire administration (Table 1).

3.2 Lipid Profile variations between pregnant and non-pregnant women across different trimesters

Regarding the lipid profile (Table 2), Total Cholesterol (T-CHL) concentrations in first trimester (4.0 ± 0.2 mmol/L), second trimester (6.9 ± 0.5 mmol/L), and third trimester (5.6 ± 0.4 mmol/L) women were all significantly higher ($p < 0.001$) when compared to the non-pregnant control group (3.9 ± 0.3 mmol/L). Similarly, Triglycerides (TG) showed statistically significant elevations ($p < 0.001$) across the first (3.2 ± 0.2 mmol/L), second (5.3 ± 0.4 mmol/L), and third trimesters (4.6 ± 0.4 mmol/L) relative to the control group (2.1 ± 0.3 mmol/L). High-Density Lipoprotein Cholesterol (HDL-C) levels were also significantly elevated ($p < 0.001$) in the first (1.9 ± 0.2 mmol/L), second (2.8 ± 0.3 mmol/L), and third trimesters (2.2 ± 0.3 mmol/L) in comparison with the control group (1.3 ± 0.1 mmol/L). Low-Density Lipoprotein Cholesterol (LDL-C) demonstrated a statistically significant increase ($p < 0.001$) in the second trimester (5.1 ± 0.4 mmol/L) compared to the control (2.5 ± 0.3 mmol/L), whereas first trimester levels (2.3 ± 0.1 mmol/L) showed no significant difference ($p > 0.05$) relative to the control. Lastly, Very Low-Density Lipoprotein Cholesterol (VLDL-C) levels were significantly elevated ($p < 0.001$) across the first (1.3 ± 0.1 mmol/L), second (1.8 ± 0.1 mmol/L), and third trimesters (2.4 ± 0.1 mmol/L) when compared to the control group (0.3 ± 0.1 mmol/L).

Table 1. Socio-demographic characteristics of the study participants

Demography	Frequency	Percentage (%)
Age groups		
18−25	33	16.5
26− 33	58	29.0
34−41	90	45.0
42−49	19	9.5
Total	200	100
Religion		
Christianity	150	75
Muslim	35	17.5
Traditional	15	7.5
Total	200	100
Education Background		
Primary School	50	25.0
Secondary School	95	47.5
Tertiary Institution	25	12.5
None	30	15.0
Total	200	100
Marital Status		
Single	10	5
Married	185	92.5
Divorced	5	2.5
Total	200	100

Values expressed as frequency and percentages (%)

Table 2. Comparative Lipid Profile of Pregnant Women Across Gestational Trimesters Versus Non-Pregnant Controls

Parameters (mmol/l)	Control (Non−Pregnant)	1st trimester	2nd trimester	3rd trimester	p−value
T-CHL	3.9 ± 0.3^{a}	4.0 ± 0.2^{b}	6.9 ± 0.5^{d}	5.6 ± 0.4^{c}	0.001
TG	2.1 ± 0.3^{a}	3.2 ± 0.2^{b}	5.3 ± 0.4^{d}	4.6 ± 0.4^{c}	0.001
HDL-C	1.3 ± 0.1^{a}	1.9 ± 0.2^{b}	2.8 ± 0.3^{d}	2.2 ± 0.3^{c}	0.001
LDL-C	2.5 ± 0.3^{a}	2.3 ± 0.01^{a}	5.1 ± 0.4^{b}	3.2 ± 0.4^{ab}	0.001
VLDL-C	0.3 ± 0.1^{a}	1.3 ± 0.01^{b}	1.8 ± 0.1^{c}	2.4 ± 0.1^{d}	0.001

*T-CHL=Total Cholesterol, TG= Triglycerides, HDL-C==High density lipoprotein cholesterol, LDL-C= Low density lipoprotein cholesterol, VLDL-LDL-C= Very Low density lipoprotein cholesterol, SD = Standard deviation, n= Number of subjects, S=Significant.*P<0.05 is considered statistically significant. NB: TG, HDL, and VLDL increased significantly (P=0.00) in the third trimester. While T-CHL and the LDL were significantly (p=0.00) elevated in the second trimester compared to the control*

4.0 DISCUSSION

Pregnancy represents a unique physiological state characterized by metabolic adaptations designed to meet the growing energy demands of the fetus while ensuring maternal metabolic stability and preparing for lactation (Gluckman et al., 2019; Murphy et al., 2020). The present study demonstrated significant, trimester-specific perturbations in maternal serum lipid profiles, marked by progressive increases in Total Cholesterol (T-CHL), Triglycerides (TG), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), and Very Low-Density Lipoprotein Cholesterol (VLDL-C) relative to non-pregnant controls ($p < 0.001$). The profound rise in Total Cholesterol and Low-Density Lipoprotein Cholesterol observed during gestation peaking during the second trimester (6.9 ± 0.5 mmol/L and 5.1 ± 0.4 mmol/L, respectively) is primarily driven by gestational endocrinological shifts. Rising circulating concentrations of placental estrogen and progesterone upregulate hepatic cholesterol synthesis and suppress hepatic lipase activity (Brizzi et al., 2018; Lain & Catalano, 2017). Estrogen stimulates the nuclear receptor sterol regulatory

element-binding protein 2 (SREBP-2) in hepatocytes, which enhances the expression of 3-hydroxy-3-methylglutaryl-CoA (HMG-CoA) reductase, the rate-limiting enzyme in cholesterol biosynthesis (Lain & Catalano, 2017; Woollett, 2011). Cholesterol serves as an indispensable substrate for fetal cellular membrane formation and is the primary precursor for placental steroidogenesis, particularly the synthesis of progesterone and estrogens required to maintain pregnancy (Sattar & Greer, 2019; Woollett, 2011). The subsequent physiological decline in T-CHL and LDL-C toward term in the third trimester (5.6 ± 0.4 mmol/L and 3.2 ± 0.4 mmol/L) likely reflects accelerated placental cholesterol uptake mediated by enhanced LDL receptor-related protein (LRP) expression in syncytiotrophoblasts (Gluckman et al., 2019; Woollett, 2011). The progressive elevation in serum Triglycerides (2.1 ± 0.3 mmol/L in controls to 5.3 ± 0.4 mmol/L in the second trimester) and VLDL-C (0.3 ± 0.1 mmol/L in controls to 2.4 ± 0.1 mmol/L in the third trimester) illustrates the biphasic switch from early anabolic fat storage to late catabolic lipid mobilization (Brizzi et al., 2018; Herrera & Ortega-Senovilla, 2014). Mechanistically, hypertriglyceridemia during gestation is driven by two main pathways: augmented hepatic VLDL secretion and delayed peripheral VLDL clearance (Herrera & Ortega-Senovilla, 2014; Sivan et al., 2021). Elevated hyperinsulinemia and placental growth hormone promote physiological maternal insulin resistance in adipose tissue (Lain & Catalano, 2017; Murphy et al., 2020). This insulin resistance unleashes hormone-sensitive lipase (HSL) activity in maternal adipocytes, accelerating intracellular lipolysis and flooding the portal circulation with free fatty acids (FFAs) (Herrera & Ortega-Senovilla, 2014; Wild, 2022). The liver utilizes these FFAs to re-esterify glycerol and assembly-pack VLDL particles via microsomal triglyceride transfer protein (MTP) (Brizzi et al., 2018; Wild, 2022). Simultaneously, high estrogen levels inhibit capillary lipoprotein lipase (LPL) activity in adipose tissue, suppressing peripheral VLDL and triglyceride clearance (Herrera & Ortega-Senovilla, 2014; Sattar & Greer, 2019). This mechanism conserves glucose and amino acids for fetal transport while utilizing maternal lipids as the primary maternal energy substrate (Gluckman et al., 2019; Murphy et al., 2020). High-Density Lipoprotein Cholesterol (HDL-C) concentrations were also significantly elevated across all trimesters, peaking in the second trimester (2.8 ± 0.3 mmol/L) compared to non-pregnant baselines (1.3 ± 0.1 mmol/L). Estrogen directly induces the hepatic transcription of apolipoprotein A-I (ApoA-I), the chief protein constituent of HDL particles (Wild, 2022; Woollett, 2011). Furthermore, estrogen reduces hepatic lipase activity, slowing the catabolism of HDL subfractions (Brizzi et al., 2018; Sattar & Greer, 2019). The physiological increase in HDL-C provides crucial anti-atherogenic, anti-inflammatory, and antioxidant protection, buffering the maternal vascular endothelium against potential injury from the concomitant surge in atherogenic ApoB-containing particles (LDL and VLDL) (Sattar & Greer, 2019; Sivan et al., 2021).

Limitations

The cross-sectional nature of the study design precluded long-term longitudinal tracking of individual participants across all three trimesters. Serum samples were collected from distinct cohorts of pregnant women in each trimester rather than following a single cohort prospectively from early gestation through delivery. Follow-up assessments were not conducted post-parturition to establish the precise timeframe required for maternal serum lipid parameters to resolve back to non-pregnant pre-gestational baselines. While basic socio-demographic parameters (age, marital status, education, and religion) were documented, granular dietary intake profiles, micro-nutritional variations, physical activity levels, and body mass index (BMI) were not controlled for, which may exert subtle confounding influences on maternal lipid kinetics. Participant recruitment was restricted to major antenatal clinics within the Enugu metropolis. Although the cohort included both urban and rural dwellers receiving care in the metropolis, the findings may not fully represent rural populations with distinctly different dietary and lifestyle patterns across broader geographical regions.

Conclusion

This study demonstrates that physiological pregnancy in Nigerian women is accompanied by significant, trimester-specific alterations in maternal serum lipid profiles compared to non-pregnant controls. The progression of gestation is characterized by marked hypercholesterolemic and hypertriglyceridemic shifts, with Total Cholesterol (T-CHL), Low-Density Lipoprotein Cholesterol (LDL-C), and High-Density Lipoprotein Cholesterol (HDL-C) reaching peak concentrations in the second trimester, whereas Triglycerides (TG) and Very Low-Density Lipoprotein Cholesterol (VLDL-C) peak during the third trimester. These observed variations reflect essential endocrinological and metabolic adaptations designed to fulfill fetal developmental requirements and prepare the maternal organism for lactation. Because standard non-pregnant laboratory reference ranges are inadequate for evaluating gravid women, establishing localized, trimester-specific reference intervals is essential to prevent the misdiagnosis of normal physiological gestational hyperlipidemia as pathological dyslipidemia. Incorporating routine lipid panel screening into standard antenatal care protocols will enable early identification of exaggerated lipid excursions, allowing clinicians to mitigate associated vascular and metabolic risks such as preeclampsia and gestational diabetes in South-East Nigeria.

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Conflict of interest

The authors declare no conflict of interest

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