Biotechnology Journal International

26(2): 25-33, 2022; Article no.BJI.87254
ISSN: 2456-7051
(Past name: British Biotechnology Journal, Past ISSN: 2231–2927, NLM ID: 101616695)

Effect of Gravidity on Biochemical Parameters in Normotensive and Hypertensive 3rd Trimester Pregnant Women

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/BJI/2022/v26i230171

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/87254

Original Research Article

Received 20 March 2022
Accepted 22 May 2022
Published 08 June 2022

ABSTRACT

Background: Pregnancy is a period in which a woman carries one or more foetus in her uterus. It is typically divided into three trimesters based on gestational age which is measured in weeks and months. Gravidity is referred to the number of times a woman has been pregnant. Pregnancy comes with several changes in metabolism, resulting to changes biochemical markers in pregnant women, some of which to certain extent may pose health risks in those with existing health conditions such as high blood pressure. The study of these changes becomes necessary to determine and arrest the risks should they exist during pregnancy.
Aim: The study was aimed at evaluating the effects of gravidity on biochemical markers in normotensive and hypertensive 3rd trimester pregnant women.
Materials and Methods: At Rivers State University Teaching Hospital, a cross-sectional study was conducted on 100 women. The consenting patients who met the inclusion criteria were randomly assigned to one of two groups: normotensive (50 normotensive pregnant women at their third trimester) or hypertensive (50 hypertensive pregnant women at their third trimester) (HPW2T). The subjects in each group were subsequently split into three categories depending on gravidity:

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primigravida (one pregnancy), multigravida (two or more), and grand multigravida (five or more).
For the assessment of TC, TG, HDL, and LDL, fasting blood samples were taken using the venepuncture technique.
AIP, CR-I, CR-II, AC, and APoB/APoA1) biochemical indices were computed quantitatively. At a $p<0.05$, the data were examined using ANOVA and the Tukey comparison test.
Result: There was no significant difference in the mean levels of the biochemical parameters among the gravidity groups in the normotensive group except for LDL and APoB levels that was significantly higher, $p<0.05$. The hypertensive group had no significant difference in the mean levels of all studied parameters among the gravidity group, $p>0.05$..
Conclusion: In this study conducted at Rivers State University Teaching Hospital, gravidity had no impact on most biochemical markers in normotensive and hypertensive pregnant women at their third trimester.

Keywords: Gravidity; pregnancy; cardiovascular marker; hypertensive women; diabetes mellitus.

1. INTRODUCTION

Pregnancy is a period in which a woman carries one or more foetus in her uterus (womb) [1]. It is typically divided into three trimesters based on gestational age which is measured in weeks and months. Gravidity is referred to the number of times a woman has been pregnant [1].

Non-complicated (physiological) pregnancy is a dynamic state accompanied by specific metabolic changes. Among these changes, the most interesting for researchers in the last couple of years were specific lipid profile and oxidative stress status, because of their potential influence on women's health later in life and their influence on cardiovascular disease (CVD) development [2]. In addition, these alterations in lipogenesis and oxidative stress status have been linked to perinatal morbidity and mortality, as a popular area for research outcomes [3]. Specific altered lipid profile during non-complicated pregnancy is essential for the normal course of pregnancy and fetal development. Nevertheless, these specific changes in lipid parameters raise the question of their pro-atherogenic potential during pregnancy and its influence on the risk for the development of CVD in women later in life, as well as complications during pregnancy, especially preeclampsia, but also gestational diabetes mellitus and intrauterine growth restriction (IUGR). By the end of the third trimester, most healthy pregnant women develop a lipid profile that could be considered highly atherogenic in healthy nonpregnant women [2]. Non-complicated pregnancy is also associated with alterations in the composition and size of low-density lipoprotein (LDL) and high-density lipoprotein (HDL) particles, which become smaller and denser with higher pro-atherogenic potential and decreased atheroprotective potential [4]. Apolipoprotein A-I (apoAI) and apolipoprotein B (apoB) are considered to be better indicators of pro-atherogenic and atheroprotective lipid components, because of their lower metabolic variations compared to other lipid components [5]. ApoB is an essential structural component of very low-density lipoproteins (VLDLs), intermediate-density lipoproteins (IDLs) and LDL. As each particle in these lipoproteins contains apoB, the total number of atherogenic particles can be estimated by measuring the plasma level of this apolipoprotein. The levels of apoA-I in plasma are strongly correlated with HDL-cholesterol (HDL-C) levels and generally with HDL particles with confirmed antiatherogenic effects [6]. The ratio apoB/apoA-I is considered to be the best indicator of the pro-atherogenic and atheroprotective components of lipoprotein particles [5]. Non-complicated pregnancy is also characterized by increased oxidative stress. Reactive oxygen species (ROS) and their control by antioxidants are involved in the physiology of the female reproductive system [7]. They are important for the normal course of pregnancy and fetal development. When the balance with the antioxidant system is disturbed, oxidative stress in pregnancy may lead to serious complications, such as preeclampsia, gestational diabetes mellitus, IUGR, miscarriage and preterm birth. Increase in oxidative stress is associated with abnormal lipid profile and may cause oxidative modification of lipids, so the studies which were conducted in complicated pregnancies also showed increased concentrations of lipid peroxides [8]. Altered lipid profile, oxidative stress and inflammation are molecular mediators of endothelial dysfunction development, which leads to preeclampsia and other pregnancy complications.

Hypercholesterolemia and hyperlipidemia are strongly associated with CVD as they promote

atherosclerosis, a precursor to myocardial infarction, stroke, and peripheral vascular disease [9]. Lipid profile including total cholesterol (TC), high density cholesterol (HDL) and triglycerides (TG) serves as a screening tool for dyslipidemia and the risk of CVD. Using these values low density lipoprotein (LDL) and total cholesterol/ HDL ratio (TC/HDL) are calculated. HDL and its major protein ApolipoproteinA1 (ApoA1) are recognized as independent protective factors against coronary heart disease [10], while elevated Apolipoprotein B (ApoB), LDL and TG are associated with a higher risk of atherosis and cardiovascular disease [11]. Triglycerides are a commonly measured component of lipid profiles for cardiovascular risk assessment [12]. Raised triglycerides are strongly associated with future risk of diabetes as well as cardiovascular disease [13] with elevated TG suggested as an explanation for residual cardiovascular risk even after statin therapy [14]. Limited study on the effect of gravidity on biochemical parameters in hypertensive pregnant women as compared to normotensives in their 3rd trimester necessitates this study.

2. MATERIALS AND METHODS

2.1 Study Design

Women totalling 100 took part in the cross-sectional study, which comprised both pregnant and non-pregnant women. According to the clinical history in their clinical folder, 50 of the participants were normotensive and the other 50 were hypertensive. Both groups (normotensive and hypertensive) had three subgroups depending on gravidity (number of pregnancies): primigravida (number of pregnancies=1), multigravida (number of pregnancies>1), and grand multigravida (number of pregnancies≥5). The primigravida subgroup contained 15 participants, the multigravida group had 27 participants, and the grand multigravida group comprised 8 participants in the normotensive group. The primigravida subgroup comprised 21 participants, the multigravida group had 25 participants, and the grand multigravida group had four participants in the hypertensive group. In Rivers State University Teaching Hospital, their atherogenic characteristics were tested individually to see if gravidity had effect on biochemical markers at 3rd trimester pregnancy.

2.2 Study Area

The research was conducted at the Rivers State University Teaching Hospital (previously known as Braithwaite Memorial Specialist Hospital) in Port Harcourt, which is Rivers state capital.

2.3 Study Population

The population of interest is pregnant women at their third trimester, who are further subdivided into two groups: normotensive third trimester pregnant women and hypertensive third trimester pregnant women.

2.4 Eligibility Criteria

This study included all apparently healthy pregnant women and hypertensive pregnant women, including those on medication, who were for the first time prenatal care attendant during their current pregnancy. Exclusion criteria covered a recent history of blood transfusion, surgery, or inability to offer informed permission.

2.5 Method of Selection

Subjects who qualified the inclusion criteria and presented written consents to participate in the study were chosen using a simple random procedure proposed by some researchers in a study on pregnant mothers [15,16].

2.6 Method of Sample Collection

Total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL), and low density lipoprotein (LDL) were measured in fasting blood samples taken by venepuncture (LDL). Blood was carefully emptied into plain vacutainer tubes, allowed to clot, then centrifuged for 10 minutes at 1500rpm. The serum was separated and kept at -4°C until it was time for the analysis [17, 18].

2.7 Laboratory Methods

2.7.1 Serum total cholesterol determination

With an enzymatic technique, total cholesterol was quantified [19].

2.7.2 Procedure

At the end of enzymatic hydrolysis and oxidation, cholesterol is measured. In the presence of phenol and peroxidase, hydrogen peroxide and 4-aminoantipyrine produce the indicator quinoneimine. The amount of colour generated is proportional to the serum cholesterol levels.

2.7.3 Principle

Requirements for the assay were taken into account. Distilled water was used to zero the device.

1ml of cholesterol reagent was pipetted into clean dry test tubes labelled blank, standard, and tests, followed by 10µl of distilled water, standard, and sample. It was thoroughly mixed by tilting the bottoms of the tubes, then incubated at 37°C for 5 minutes on a waterbath. In a spectrophotometer set to 540nm wavelength, the absorption of the standard and test samples was compared to the blank.

2.7.4 Determination of high-density lipoprotein (HDL) cholesterol in serum

HDL-C was quantified using an enzymatic technique [20].

2.7.5 Principle

By adding phosphotungstic acid in the presence of magnesium ions, low density lipoprotein (LDL and VLDL) and chylomicron fractions can be quantitatively precipitated.

Following centrifugation, an enzymatic technique is used to quantify the cholesterol concentration in the HDL fraction that remains in the supernatant.

2.7.6 Procedure

The blood samples were centrifuged for five minutes at 12,000 rpm after being put into tubes. The serum was separated and organized into control, standard, and sample tubes. 200µl of precipitating reagent (R) and 20µl of sample were placed in the test tubes, 20ul of standard in the standard tube, and distilled water in the blank tube. By tilting the bottoms of the tubes, it was adequately mixed and allowed to stand for 10 minutes at room temperature. The tubes were spun at 12,000 rpm for 2 minutes.

The clear supernatant was then removed and the HDL cholesterol level was measured.

2.7.7 Serum triglycerides determination

The enzymatic approach is used to quantify triglycerides [21].

2.7.8 Principle

After hydrolysis by enzyme and oxidation with lipases, measurement for triglycerides is taken. Under the catalytic effect of peroxidase, a quinoneimine is generated by combining hydrogen peroxide, 4-aminophenazone, and 4-chlorophenol. The amount of color generated in the sample is related to the concentration of triglycerides.

2.7.9 Procedure

The experiment circumstances were taken into account. Pure water was used to zero the instrument.
As a blank, standard, and test, 1ml of triglyceride reagent was applied to the tubes. The tubes were filled with 10 l of standard and sample, mixed, and incubated at 370C for 5 minutes. The absorbance of samples was measured against a blank using a 1cm light path (cuvette) at 505 nm wavelength.

2.7.10 Low-Density Cholesterol Measurement (LDL-C)

Friedewald's equation was used in the determination of LDL cholesterol [22]..

LDL – Cholesterol = Total Cholesterol – (TG/2.2) – HDL

The following standard formulas were used to determine the atherogenic index and lipid ratios:

AIP = Log (TG/ HDL-C): Reference Range **=** Low risk (-0.3 – 0.1), Moderate risk (0.1 – 0.24), High risk (>0.24) [23].

CRI-1 = TC/HDL-C: Reference Range **=** Low risk (< 1-3), Moderate risk (3-5), High risk (>5) [23].

CRI-II = LDL-C /HDL-C: Reference Range **=**Low risk (< 1-3), Moderate risk (3- 5), High risk (> 5) [23].

AC = TC – HDL-C/ HDL-C: (Reference >3.0) [23].

Apo B/ Apo A1: Reference range = (low risk 0.30, moderate risk 0.6 and high risk 0.8) [23]

2.8 Statistical Analysis

GraphPad Prism Version 8.0.2.263 was used to analyze the data collected throughout the investigation. The mean and standard deviation were used to represent the data. The one-way analysis of variance (ANOVA) was used to compare the means (ANOVA). At $p<0.05$, the

Tukey comparison test was employed to ensure that there were significant differences between the groups.

3. RESULT

3.1 Effect of Gravidity on Biochemical Parameters in Normotensives 3rd Trimester

Tables 1.0 (a) and 1.0 (b) show the effect of gravidity on LDL and apo B in Normotensive pregnant women at 3rd trimester. Gravidity showed that LDL was significantly higher in grand multigravida of pregnant women at 3rd trimester compared with primigravida and mutigravida of pregnant women at 3rd trimester (p=0.0174). Apo B was significantly higher in grand multigravida of pregnant women at 3rd trimester compared with primigravida and mutigravida of pregnant women at 3rd trimester (p=0.0473). There was no significance using post Hoc for the two parameters.

3.2 Effect of Gravidity on Biochemical Parameters in Hypertensive 3rd Trimester

Tables 2.0 (a) and 2.0 (b) represents the effect of gravidity on biochemical parameters (TC, TG, HDL, LDL, UA, CRP, VLDL, APO A1, and APO B) in hypertensive pregnant women at 3rd trimester. Gravidity showed no significant effect on all the biochemical parameters in Hypertensive pregnant women at 3rd trimester ($p<0.05$).

4. DISCUSSION

This study analysed Total Cholesterol (TC), triglyceride (TG), High Density Lipoprotein (HDL), Apolipoprotein A1 and B (ApoA1) (ApoB), Low Density Lipoprotein (LDL), Very Low Density Lipoprotein (VLDL) and Uric Acid (UA).

Table 1(a). Effect of Gravidity on Biochemical Parameters in Normotensives 3rd Trimester

Parameters	Normotensive Women			*P*-value	F-value
	Primigravida (1)n = 21	Multigravida(>1) n = 22	Grand Multigravida (≥ 5) n = 7		
TC(mmol/l)	4.53 ± 0.43	4.85 ± 0.53	4.87 ± 0.43	0.0688	2.8340
TG (mmol/l)	1.39 ± 0.34	1.47 ± 0.30	1.43 ± 0.25	0.6602	0.4190
HDL(mmol/l)	0.89 ± 0.26	1.01 ± 0.15	0.87 ± 0.13	0.0954	2.4710
LDL (mmol/l)	3.05 ± 0.15	3.20 ± 0.34	3.37 ± 0.24	0.0174	4.4250
APoA1 (mg/dl)	340.90 ± 34.34	358.90 ± 33.51	370.90 ± 14.86	0.0622	2.9490
APoB (mg/dl)	140.30 ± 30.71	158.20 ± 28.20	172.00 ± 43.19	0.0473	3.2590
CRP(mg/L)	4.67 ± 1.30	5.96 ± 2.32	5.14 ± 1.64	0.0831	2.6240
VLDL (mmol/l)	0.63 ± 0.15	0.67 ± 0.14	0.65 ± 0.11	0.6602	0.4190
UA (mg/dl)	4.88 ± 0.99	4.88 ± 0.71	4.93 ± 0.42	0.9888	0.0113

Table 1(b). The ANOVA Post – Hoc Findings Using Turkey Multiple Comparison Test for Effect of Gravidity on Biochemical parameters (Normotensive 3rd Trimester)

Parameters	Primagravida vs. Multigravida	Primagravida vs Grand multigravida	Multigravida vs Grand Multigravida
TC(mmol/l)	0.0818	0.2394	0.9942
TG (mmol/l)	0.6334	0.9468	0.9430
HDL(mmol/l)	0.1204	0.9855	0.2648
LDL (mmol/l)	0.1640	0.0181	0.2773
APoA1 (mg/dl)	0.1686	0.0927	0.6676
APoB (mg/dl)	0.1617	0.0659	0.5782
CRP(mg/L)	0.0690	0.8311	0.5700
VLDL (mmol/l)	0.6334	0.9468	0.9430
UA (mg/dl)	0.9997	0.9882	0.9905

Table 2(a). Effect of Gravidity on Biochemical Parameters in Hypertensive 3rd Trimester

Parameters		Hypertensive Women		*P*-value	F-value
	Primigravida (1) n = 20	Multigravida(>1) n=16	Grand Multigravida (≥ 5) n = 14		
TC(mmol/l)	4.98 ± 0.46	4.81 ± 0.44	4.89 ± 0.45	0.5438	0.6171
TG (mmol/l)	1.53 ± 0.37	1.43 ± 0.27	1.65 ± 0.38	0.2101	1.6130
HDL(mmol/l)	0.94 ± 0.27	0.93 ± 0.11	0.99 ± 0.17	0.6342	0.4599
LDL (mmol/l)	3.37 ± 0.28	3.23 ± 0.33	3.17 ± 0.25	0.1200	2.2180
APoA1 (mg/dl)	382.00 ± 21.10	380.30 ± 17.60	375.70 ± 21.58	0.6670	0.4085
APoB (mg/dl)	140.50 ± 17.21	136.40 ± 10.01	135.90 ± 17.04	0.6152	0.4908
CRP(mg/L)	7.90 ± 2.32	7.62 ± 2.18	8.06 ± 2.39	0.8639	0.1468
VLDL (mmol/l)	0.70 ± 0.17	0.65 ± 0.12	0.75 ± 0.17	0.2101	0.6130
UA (mg/dl)	4.53 ± 0.38	4.83 ± 0.40	4.72 ± 0.39	0.0815	2.6460

Table 2(b). The ANOVA Post – Hoc Findings Using Turkey Multiple Comparison Test for Effect of Gravidity on Biochemical parameters (Hypertensive 3rd Trimester)

Parameters	Primagravida vs. Multigravida	Primagravida vs Grand multigravida	Multigravida vs Grand Multigravida
TC(mmol/l)	0.5144	0.8447	0.8779
TG (mmol/l)	0.6339	0.5768	0.1819
HDL(mmol/l)	0.9735	0.7357	0.6332
LDL (mmol/l)	0.2999	0.1289	0.8678
APoA1 (mg/dl)	0.9639	0.6471	0.8133
APoB (mg/dl)	0.7079	0.6588	0.9940
CRP(mg/L)	0.9292	0.9770	0.8568
VLDL (mmol/l)	0.6339	0.5768	0.1819
UA (mg/dl)	0.0736	0.3471	0.7510

All lipids and apolipoproteins have been shown to be significantly elevated in pregnancy, the most prominent change being a 2.7-fold increase in triglycerides in the third trimester [24]. As pregnancy progresses, lipids levels steadily increase during the pregnancy with a noticeable increase in the third trimester [25]. This lipid metabolism throughout pregnancy allows for proper nutrients for the fetus.

This study showed that LDL was significantly higher in grand multigravida of pregnant women at 3rd trimester compared to the levels in primigravida and multigravida groups (p=0.0174). This suggests that the more the number of pregnancy, the higher the LDL concentration in Normotensive pregnant women in their 3rd trimester. This is capable of predisposing these pregnant women to CVD. The result also showed that Apo B which is an essential structural component of very low-density lipoproteins (VLDLs), intermediate-density lipoproteins (IDLs) and LDL was significantly higher in grand multigravida of normotensive pregnant women at 3rd trimester compared with primigravida and multigravida of normotensive pregnant women at 3rd trimester (p=0.0473). This also indicates that as the number of pregnancy increases, the ApoB concentration also increases significantly from primigravida to multigravida and Grand multigravida in normotensive pregnant women in their 3rd trimester. This may predispose these pregnant women to CVD. The result also showed that there was no significance difference when comparing between the groups using post Hoc. There was also no significant difference in other biochemical parameters such as TC, TG, HDL, ApoA1, CRP, VLDL and UA in normotensive pregnant women at 3rd trimester.

In the hypertensive group, there was no significant effect of gravidity on all the biochemical parameters among the groups ($p<0.05$). This suggests that gravidity has no effect on the biochemical parameters of hypertensive pregnant women at their 3rd trimester.

This work disagrees with Enquobahrie *et al*. [26] that there was a significant rise in LDL

concentration in the hypertensive women than in normal pregnant women. But in this study the rise in LDL was seen in normotensive rather than hypertensive women. This work agrees with Clausen et al. [8] that there was no significant difference in mean total cholesterol concentration in the hypertensive group when compared with that in normal pregnant group. The findings of Tam *et al*. [27] recorded that maternal serum uric acid concentration was a good prognostic factor for monitoring, and prognosis of fetal/neonatal outcomes in women with preeclampsia/ eclampsia. They also observed a relationship between high uric acid level and the risk of preterm birth, low Apgar index, and neonatal death, but not fetal death, but there was no effect of gravidity on uric acid concentration recorded in both the normotensive and hypertensive group in this study, therefore this study is not in consonance with their work. The difference observed in this study from other works is probably the age gestation. Most study focused on first and second trimester while this present study focused on third trimester pregnancy.

This study agree in part with Timur *et al.* [28]. In their work on the Apolipoprotein levels in women with preeclampsia, they found out that Apo B and Apo B / Apo A1 were significantly increased in normotensives, but Apo A1 was significantly decreased and advocated that Apo A1 and Apo B/Apo A1 ratio be useful markers in patients with preeclampsia. However, more research works are encouraged to confirm these findings [29-30].

5. CONCLUSION

At the end of this study conducted at Rivers State University Teaching Hospital, a discovery showing that gravidity had no influence on biochemical markers in normotensive and hypertensive pregnant women in the third trimester was made.

ETHICAL CLEARANCE AND CONSENT

The Ethics Committee of the Rivers State Ministry of Health provided ethical clearance.

Before being authorized to participate in the study, all eligible subjects signed an informed consent form.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Huda MM, Leung TM, Zhou L, Abu- Merhi S. Regulating intestinal function to reduce Atherogenic Lipoproteins. Clinical Lipidology. 2009;8(4):481-490.
2. Garduno-Alanís A, Vázquez-de Anda G, Valdés-Ramos R, Talavera JO, Herrera-Villalobos JE, Huitrón-Bravo GG. Predictors of hyperlipidemia during the first half of pregnancy in Mexican women. Nutrition and Hospitality. 2015;31:508–13.
3. Wild R, Weedin EA, Wilson D. Dyslipidemia in pregnancy. Cardiology and Clinicals. 2015; 33:209–15.
4. Ogura K, Miyatake T, Fukui O, Nakamura T, Kameda T, Yoshino G. Low-density lipoprotein particle diameter in normal pregnancy and preeclampsia. Journal of Atherosclererosis and Thrombosis. 2002; 9:42–7.
5. Kaneva AM, Potolitsyna NN, Bojko ER, Odland J. The Apolipoprotein B/Apolipoprotein A-I ratio as a potential marker of plasma atherogenicity. Disease Markers. 2015;591454.
6. Kim Yj, Park H, Lee HY, Ahn Y, Ha EH, Suh SH. Paraoxonase gene polymorphism, serum lipid, and oxidized low-density lipoprotein in preeclampsia. European Journal of Obstetrics and Gynecology. 2007;133;47–52.
7. Al-Gubory K, Fowler PA, Garrel C. The roles of cellular reactive oxygen species, oxidative stress and antioxidants in pregnancy outcomes. Cell Biology. 2010;42:1634–50.
8. Clausen T, Đurovic S, Henriksen T. Dyslipidemia in early second trimester is mainly a feature of women with early onset preeclampsia. British Journal of Obstetrics and Gyneacology. 2001; 108:1081–7.
9. Boullart ACI, de-Graaf J, Stalenhoef AF. Serum triglycerides and risk of cardiovascular disease. Biochemistry and Biophysics Acta. 2012;1821(5):867–75.
10. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease. The Framingham Study. American Journal of Medicine. 1977;62(5):707–14.11.
11. Murphy AJ, Woollard KJ. High-density lipoprotein: A potent inhibitor of inflammation. Clinical Experiment in

Pharmacology and Physiology. 2010; 37(7):710–8.

12. Goldberg IJ, Eckel RH, McPherson R. Triglycerides and heart disease: still a hypothesis? Arteriosclerosis, Thrombosis and Vasculature. 2011;31(8):1716–25.
13. Kannel WB, Gordon T, Castelli WP. Role of lipids and lipoprotein fractions in atherogenesis: the Framingham study. Progcress in Lipid Research. 1981; 20:339–48.
14. Wierzbicki AS, Clarke RE, Viljoen A, Mikhailidis DP. Triglycerides: A case for treatment? Current Opinion on Cardiology. 2012; 27(4):398–404.
15. Catherine I, Biambo KG, Nyebuchi J, Fyneface AC, Goodnews N. Evaluation of Nutrient Composition in Breast Milk of Breast Feeding Mothers in Urban and Sub-urban Subjects in Rivers State. Asian Journal of Pediatric Research. 2021;7(2):39-44.
16. Faith D, Kalaotaji GB, Nyebuchi J, Chikadibia FA, Felix EK. Comparative study of heavy metals in breast milk of breast feeding mothers in urban and sub-urban subjects in Rivers state. Journal of Applied Life Sciences International. 2021;28(8):31-36.
17. Oladapo- Akinfolarin TT, Bartimeaus ES, Nwachukwu EO, Nduka N. Assessment of C-reactive protein levels in normotensive and hypertensive pregnant subjects in Port Harcourt, Nigeria. World Journal of Pharmaceutical and Medical Research. 2017;3(10):292-296.
18. Oladapo- Akinfolarin TT, Bartimeaus ES, Nwachukwu EO, Nduka N. Assessment lipoprotein levels in normotensive and hypertensive pregnant women in Port Harcourt, Nigeria. American Journal of Biomedical Sciences. 2018; 10(1):18-27.
19. Allain CC, Poon LS, Chan CSG, Richmond W, Fu PC. Enzymatic Determination of Total Serum Cholesterol. Clinical Chemistry. 1974;20 (4):470-475.
20. Tietz NW. Laboratory Methods. In: Fundamentals of Clinical Chemistry, 3rd Edition, W.B. Saunders Company, Philadelphia. 1987;927-962
21. Fraser CG, Hearne CR. Assessment of colorimetric enzymatic determination of triglyceride, by manual and centrifugal analyzer techniques, and comparison with a CDC standardized method. Clinical Biochemistry. 1981;14 (1):28-31.
22. Friedwald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma, without use of preparative ultracentrifuge. Clinical Chemistry, 1972;18:499-502.
23. World Health Organization. Non-communicable diseases. Cardiovascular Diseases (CVDs). Last accessed on 2014 may 10, at 9:40 pm;2014.
24. Rymer J, Constable S, Lumb P, Crook M. Serum lipoprotein (a) and apolipoproteins during pregnancy and postpartum in normal women. Journal of Obstetrics and Gynaecology. 2002; 22(3):256–9.
25. Wiznitzer A, Mayer A, Novack V, Sheiner E, Gilutz H, Malhotra A. Association of lipid levels during gestation with preeclampsia and gestational diabetes mellitus: A population-based study. American Journal of Obstetrics and Gynecology. 2009; 201(5):482 1–4828.
26. Enquobahrie DA, Williamss MA, Butter CL, Federick IO, Miller RS, Luthy DA. Maternal plasma lipid concentrations in early pregnancy and risk of pre-eclampsia. America Journal of Hypertension. 2004; 17(7):578-581.
27. Tam M, Lelong H, Nguyennam L, Phan DD, Lehuy VQ, Nguyen A. Maternal Serum Uric Acid Concentration and Pregnancy Outcomes in Women with Preeclampsia/ Eclampsia. International Journal of Gynecology and Obstetrics. 2018;144: 1.
28. Timur H, Daglar HK, Kara O, Kirbas A, Inal HA, Turkmen GG, Yilmaz Z, Elmas B, Uygur D. A Study of Serum Apo A1 and Apo B -100 Levels in Women with Preeclampsia. Pregnancy Hypertension. 2016;6(2):121–125.
29. Dashty M, Motazacker MM, Levels J, de-Vries M, Mahmoudi M, Peppelenbosch MP. Proteome of human plasma very low-density lipoprotein and low-density lipoprotein exhibits a link with coagulation and lipid metabolism. Thrombosis and Haemostasis. 2014;23(111):518–30.
30. Enquobaline DA, Williams MA, Butler CL, Frederick JD, Muller RS, Luthy D. Maternal Plasma Lipid Concentrations in Early Pregnancy and Risk of Preeclampsia. American Journal Hypertension. 2004;17: 574 – 581.

31. Macekova D, Kovac G, Hinst J, Illek B, Pereckova J, Baraskova Z. Lipid peroxidation and biochemical parameters in maternal pre-delivery and post-delivery plasma. Biologia. 2010; 65:170–4.

Peer-review history:
The peer review history for this paper can be accessed here:
https://www.sdiarticle5.com/review-history/87254