ORIGINAL ARTICLE

A LONGITUDINAL COMPARATIVE STUDY OF LIPID PROFILE IN THIRD TRIMESTER OF PREGNANCY AND END OF PUERPERIUM

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Background: The postpartum period is associated with maternal metabolic changes geared towards returning the maternal homeostasis to the pre-pregnancy state. This study aimed at investigating the lipid profile in the postpartum period in comparison to the third trimester and control values. **Methods:** Blood Pressure (BP), Body Mass Index (BMI) and lipid profile were assessed using standard procedures. Results were presented in tables and graphs and $p<0.05$ was considered significant. **Results:** The systolic and diastolic blood pressures were 120.1±11.3 and 77.2±10.1 mmHg, 119.4±10.3 and 78.3±11.1 mmHg, 121.5±9.2 and 79.2±9.0 mmHg for the control, third trimester and 6 weeks postpartum respectively. There were no significant difference between the systolic and diastolic blood pressures of the test subjects and control. The BMI were 22.5±3.1 Kg/m^2, 28.4±2.1 Kg/m^2 and 29.4±2.4 Kg/m^2 for control, third trimester and 6 weeks postpartum respectively. There was a statistical difference ($p<0.05$) between the BMI of control and third trimester. The lipid profile in the postpartum period was higher than the control values and this was statistically significant ($p<0.05$) for all lipids studied except the high-density lipoprotein (HDL). **Conclusion:** The clinically non-significant elevated lipid profile in the pregnancy period and decline in the postpartum period are physiological.

Keywords: Postpartum, Lipids, BMI, Trimesters, Pregnancy

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INTRODUCTION

Lipid is a generic term used to describe any fatty or fat-like substance that occurs naturally and capable of being utilized by plants and animals; for energy storage, as structural components of cell membranes, and as important signalling molecules.[1,2] Lipids are bound to specific proteins to form lipoproteins which provide solubility in the aqueous environment and can be metabolized.[2] The importance attached to the need for routine examination of the serum lipid and lipoprotein profile in human subjects, especially during pregnancy is well-established.[3] During pregnancy, maternal metabolism must satisfy the demands of the developing foetus in addition to the energy requirements of the mother. The growth of the foetus is a complex process guided by an interplay among the mother, the placenta, the foetus, the environment *viz a viz* the availability of nutrients such as glucose, lipids, and amino acids. The anabolic phase of early pregnancy results in an increased deposition of fat in maternal adipose tissue. In contrast, late pregnancy is referred to as the catabolic phase involving the release of free fatty acids from the adipocytes. This catabolic phase is enhanced by relative insulin resistance and stimulation of hormone-sensitive lipase by placental hormones. As a consequence, the maternal lipid metabolism is specifically altered during pregnancy.[4] In addition to this hormonal anabolic/ catabolic interplay, other maternal factors such as Body Mass Index (BMI), maternal weight gain, maternal nutrition, pre-pregnancy lipid levels and various medical complications of pregnancy have been documented to have significant effects on lipid metabolism and plasma levels.[5] The catabolic phase of late pregnancy is an essential requirement for the recovery from the stress of parturition, the peripartum and postpartum blood loss and the energy requirement for the breast feeding of the baby during puerperium and beyond. The puerperium is commonly defined as a period of 6 weeks after childbirth. It is the period during which the woman's physiologic, anatomic and biochemical adaptations of pregnancy returns to the pre-pregnancy state. Though lipids and lipoproteins are central to the metabolism of the body, a postpartum reversal of the high lipid profile is paramount in the prevention of atherosclerosis which is associated with coronary heart disease.[6] This study is, therefore, aimed at studying the lipid profile in third trimester of pregnancy and at the end of puerperium.

MATERIAL AND METHODS

This study was a longitudinal study in St. Philomena Catholic Hospital Benin City, Edo State. A total of 60 women between the ages of 20 and 30 years volunteered for the study. The controls were 30 non-pregnant staff of St. Philomena Catholic Hospital. Another 30 were the subjects. They were studied at the 37th week of pregnancy and at the end of puerperium, i.e., end of 6 weeks postpartum. Ethical clearance was obtained from the Ethics and Collaboration Committee of the St. Philomena Catholic Hospital. Informed consent was obtained from the subjects.

All subjects fasted overnight for a minimum of 8 hours. Five millilitre of fasting venous blood was collected from the antecubital vein from each subject

and placed into plain bottles. The blood was then centrifuged at 4000 rpm for 5 minutes after clotting and serum removed and stored at 4 °C pending assay for lipid profile. Serum Triglycerides (TG), Total Cholesterol (TC) and High Density Lipoprotein (HDL) Cholesterol were analysed by enzymatic methods with Glaxo kits on ERBA Chem-5® semi-auto analyser. Plasma total cholesterol was estimated by cholesterol oxidase-pheny antipyrine method (CHOD-PAP).[7] Plasma triglycerides (TG) were estimated using glycerol phosphate oxidase method (GPD method).[8] High density lipoprotein-cholesterol was estimated using phosphotungstic acid and magnesium chloride method.[9] LDL-cholesterol was estimated using the Friedewald formula.[10]

LDL-cholesterol=Total cholesterol-(TG/2.2-HDL-cholesterol)

Dyslipidemia was defined according to the Evaluation and Treatment of High Blood Cholesterol in Adults executive summary of the third report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III): LDL cholesterol 100 mg/dl=optimal, 100–129 mg/dl=near optimal/above optimal, 130–159=borderline high, 160–189=high, ≥190=very high. Total cholesterol <200 mg/dl=desirable, 200–239 mg/dl=borderline high, ≥240 mg/dl=very high. HDL cholesterol 40 mg/dl=low, ≥60=high. Triglyceride <150 mg/dl=normal, 150–199=borderline high, 240–499=high, ≥500=very high.[11]

Body weight was measured in Kg using bathroom scale. Subjects put off their shoes and any extra dress items before they mounted the scale. Height was measured without footwear in meters using a standard stadiometer. Body Mass Index (BMI) was calculated using the formula:

Weight in Kg/height in meter squared

Blood Pressure was measured following a rest period of about 30 minutes in the hospital; the systolic and diastolic pressures were measured in each subject on the brachial artery using auscultator method. The Helsinki Declaration was strictly followed for data collection.[12]

Data were presented as Mean±SD using the Microsoft Excel 2010. Student's *t*-test was used for analyses of data and $p<0.05$ were considered statistically significant.

RESULTS

The mean ages were 24.5±3.2 and 25.5±3.5 year for control and test subjects respectively. The systolic and diastolic blood pressures were 120.1±11.3 and 77.2±10.1 mmHg, 119.4±10.3 and 78.3±11.1 mmHg, 121.5±9.2 and 79.2±9.0 mmHg for the control, third trimester, and end of puerperium respectively. There were no significant difference between the systolic and diastolic blood pressures of the test subjects and controls.

The BMI were 22.5±3.1 Kg/m^2, 28.4±2.1 Kg/m^2 and 29.4±2.4 Kg/m^2 for control, third trimester and end of puerperium respectively. There were statistical differences ($p<0.05$) between the BMI for control and third trimester, and between the BMI of controls and subjects at the end of puerperium. The mean plasma lipid profile values for control and test subjects are shown in Table-1.

Table-1: Lipid profile of subjects

Parameters	Control	3rd trimester	6 Weeks Postpartum
TC	141.4±10.4	199.3±12.2	165.3±13.4
HDL-C	43.1±3.3	50.9±3.8	45.0±2.1
LDL-C	86.8±9.5	161.5±12.6	116.7±9.9
TG	45.7±5.4	171.7±10.8	148.5±10.4

DISCUSSION

The importance attached to need for routine examination of serum lipid and lipoprotein profile in human subjects especially during pregnancy is well established.[3] The maternal lipid metabolism is specifically altered during pregnancy[4], such that all lipids and lipoprotein cholesterol fractions increase with increasing age of pregnancy[13]. Normal pregnancy is associated with high concentrations of oestrogens which may contribute to the rise in plasma lipids especially in latter half of pregnancy.[14] Total cholesterol, LDL-C, HDL-C, and triglyceride levels rise throughout the second and third trimester, peaking just before term at approximately 36 weeks of gestational age.[14]

The increase in lipid profile in third trimester of pregnancy was also observed in this study. The pattern of increase of total cholesterol from the control subjects to the third trimester was the same as the pattern of decrease from the third trimester to the end of puerperium. This same mirror image pattern was observed for the high density lipoprotein and low density lipoprotein. However, for the triglyceride there was a steep rise from the control to the third trimester value with a non-mirror image decrease at the post-partum period. Overall, the physiological mechanism in the postpartum period for return of the elevated lipid profile to pre-pregnant value is optimum. Studies have revealed that the mean values of serum TC, TG, and LDL are significantly higher among the hypertensive patients compared to normotensives.[15] However, in this study the elevated lipid profile levels in the third trimester of pregnancy and the postpartum period were not associated with elevated blood pressures. This suggests that the dyslipidemia in pregnancy and postpartum is physiological.

Changes in the plasma lipids during pregnancy have been recognised and thought to be mostly due to alterations in the hormonal milieu in the form of rise in insulin, progesterone, 17-B estradiol and human placental lactogen.[9,16] Lipids and lipoprotein cholesterol fractions have been reported to reduce to their normal

reference value at the end of puerperium.[17] This study revealed a reduction in the triglyceride, LDL-C and total cholesterol at the end of the puerperium. But these lipids at the end of the puerperium were still significantly higher than the control values. The reduction observed in triglyceride levels is consistent with the study done by Ola and Adedeji[18], although their observation was at the 4-week postpartum. However, the high-density lipoprotein cholesterol reduced to the pre-pregnancy value at the end of puerperium, as there was no statistical difference between the control and the 6th week postpartum value.

National Cholesterol Education Program describes serum concentration of lipids as; desirable, within normal range, optimal, high, near optimal, normal and borderline.[11] Thus, the total cholesterol level for control and test subjects was desirable. The HDL-C was within normal range for control and test subjects. However, for the LDL-C the control was optimal while the LDL-C for the third trimester was high and the 6 weeks postpartum value was near optimal. The triglyceride value for the control subjects was normal while the third trimester value was borderline high, the 6-weeks postpartum value was normal.

CONCLUSION

The elevated lipid profile in pregnancy is physiological and declines to the pre-pregnant values to restore the non-gravid maternal homeostasis.

LIMITATIONS

The control subjects were different from test subjects.

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REFERENCES

1. Frier KN. Lipid metabolism. Clinical Biochemistry. (3rd ed). McGraw Hill Companies. Philadelphia, London. 2002; pp. 532–41.
2. Ojule AC, Akani CI, Opurum HC. Plasma lipids during pregnancy in Women Port Harcourt, Nigeria. Nig J Med 2005;14(4):155–60.
3. Seymour LH. Prevention and treatment of hyperlipidemia in quick reference to clinical nutrition, a guide to clinicians. JB Lippincott Company, Philadelphia Toronto. 1979:137–51.
4. Guan MX, Wenk MR. Biochemistry of Inositol lipids. Frontiers in Bioscience 2008;13:3239–51.
5. Brasaemle DL. Thematic review series: Adipocyte Biology. The perilipin family of structural lipid droplet proteins: stabilization of lipid droplets and control of lipolysis. J Lipid Res 2007;48(12):2547–59.
6. Hevonoja KL, Chait MH, Howard NT. Metabolism of low density lipoprotein from patients with diabetic hyperglyceridemia by cultured human skin fibroblasts. Diabetes 2000;34:8–14.
7. Wakatsuki A, Ikenoue N, Izumiya C, Okatani Y. Effects of estrogen and simvastatin on low-density lipoprotein subclass in hyper-cholesterolemic postmenopausal women. Obstet Gynecol 1998;33(7):367–71.
8. Tietz NW. (Ed). Clinical Guide to Laboratory Tests, 3rd ed. Philadelphia; WB Saunders: 1995.
9. Alvarez J, Montelongo A, Iglesias A, Lasuncion M. Longitudinal study on lipoprotein profile, high density lipoprotein subclass, and postheparin lipases during gestation in women. J Lipid Res 1996;37:299–308.
10. Friedwald WT, Levy RJ, Fredrickson DS. Estimation of the concentration of Low Density Lipoprotein Cholesterol in plasma without use of preparative ultracentrifuge. Clin Chem 1972;18(1):499–502.
11. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third report of the National Cholesterol Education Program Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. Circulation 2002;106(25):3143–421.
12. World Medical Association Declaration of Helsinki: Ethical principles for medical research involving human subjects. JAMA 2000;284(23):3043–5.
13. Di Cianni G, Miccoli R, Volpe L, Lencioni C, Del Prato S. Intermediate metabolism in normal pregnancy and in gestational diabetes. Diab Metab Res 2003;19(9):259–70.
14. Emeka E, Neboh JK, Emeh UU, Aniebue EJ, Ikekpeazu IC, Maduka OE. Relationship between lipid and lipoprotein metabolism in trimesters of pregnancy in Nigerian women: Is pregnancy a risk factor? J Nat Sci Biol Med 2012;3(1):32–7.
15. Choudhury KM, Mainuddin AKM, Wahiduzzaman M, Mohammed S, Islam S. Serum lipid profile and its association with hypertension in Bangladesh. Vasc Health Risk Manag 2014;10:327–32.
16. Coleman RA, Lee DP. Enzymes of triacylglycerol synthesis and their regulation. Prog Lipid Res 2004;43(2):134–76.
17. Salawu AA, Ajose OA, Adegoke OA, Adedosu AN, Adeyemi AB Aboderin AO. Pattern of changes in plasma lipids and lipoprotein cholesterol levels through normal pregnancy and puerperium in Ile-Ife, Nigeria. J Dent Med Sci 2014;13(7):77–9.
18. Ola R Adedeji OO. Plasma lipids during first trimester in pregnant Nigeria women. Nig Qt J Hosp Med 1997;7(1):111–3.

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