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Original Research Article

A Study of Lipid Profile in Pregnant and Nonpregnant Women

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Abstract:

Introduction: Maternal metabolism undergoes numerous changes during pregnancy including lipid metabolism to ensure adequate nutrient supply to the foetus. The present study investigates alterations in lipid profile (Total cholesterol, triglycerides, LDL, VLDL and HDL) in normal pregnant women (primigravida) in the three trimesters and non- pregnant women of similar age group. The purpose of the present study is to find out the effects of pregnancy on the lipid profile in order to take steps to minimize cardiovascular complications and promote reproductive health of women.

Aim: To study the changes in lipid profile during normal pregnancy.

Materials and Methods: This was a cross-sectional observational study consisting of 200 subjects that included 100 primigravida pregnant women and 100 non- pregnant women between 20-35 years. Blood samples for the estimation of lipid profile were taken and analysed using clinical chemistry analyser ERBA (XL-300).

Results: Serum Low density lipoprotein (LDL), total cholesterol (TC), triglycerides (TG) and very low density lipoproteins (VLDL) were significantly elevated in the second and third trimesters when compared to first trimester and also in comparison to normal non-pregnant women. Serum High density lipoproteins (HDL) showed a biphasic pattern characterised by significant elevation in first trimester when compared to normal non-pregnant women and then progressively declined in the second and third trimesters when compared to normal non-pregnant women.

Conclusion: An atherogenic lipid profile is observed in most women by the third trimester of normal pregnancy.

Keywords: Pregnancy, Trimester, Lipid Profile.

Introduction

Pregnancy is a physiological variation of the normal female human body and consists of various events after fertilization of ovum [1]. Conception initiates numerous changes in maternal physiology that continue throughout gestation [2].

There is extra demand of energy as pregnancy advances. Hence various maternal metabolic adaptations including lipid metabolism occur during pregnancy that ensures an adequate nutrient supply to the foetus [3].

Early pregnancy is the anabolic phase due to the built up of maternal fat stores. In marked contrast, late pregnancy is the catabolic phase due to net breakdown of adipose tissue depots thereby significantly elevating the lipid profile components by the end of third trimester [4]. Hormonal changes during pregnancy induce the changes in lipid composition. In early pregnancy, there is increase in levels of estrogen and progesterone along with increased secretion of insulin due to hyperplasia of pancreatic beta-cells [5]. Elevated insulin increases peripheral glucose utilization with a corresponding fall in plasma glucose levels. Increased glycogenesis, increased lipogenesis and decreased lipolysis also occur due to the hyperinsulinemia. Late pregnancy is characterised by increased lipolysis thereby causing rise in free fatty acids and glycerol in the maternal circulation. This results from insulin resistance that peaks during third trimester in turn leading to increased activity of the enzyme hormone-sensitive lipase and decrease in lipoprotein lipase activity.

The change in maternal lipid profile depends upon the trimester of pregnancy. A significant increase in plasma total cholesterol, LDL cholesterol and triglycerides is seen during second trimester and maximum in the third trimester. But the change in HDL cholesterol is variable.

The aim of the present study is to investigate the changes in lipid profile during normal pregnancy.

The primary objective is to estimate the lipid profile (serum total cholesterol, triglycerides, HDL, LDL, VLDL) in pregnant women (primigravida) in the three trimesters. The secondary objective is to estimate the lipid profile in non- pregnant women of similar age group and to compare the values of lipid profile of the above two groups.

Materials and Methods

The study was approved by the Institutional Ethics Committee and was conducted in the Department of Obstetrics and Gynaecology in association with the Department of Physiology, Government Medical College Kottayam for a period of one year from April 2011 to April 2012.

Inclusion Criteria

The study group consisted of 200 subjects that included 100 pregnant (primigravida) and 100 non-pregnant women aged between 20-35 years and willing for the study.

The 100 pregnant women (primigravida) were divided into three groups as:

First trimester	0-12 weeks	30 subjects
Second trimester	13-24 weeks	30 subjects
Third trimester	25-40 weeks	40 subjects

Exclusion Criteria

Pregnant women with pregnancy induced hypertension, pre- eclampsia, diabetes mellitus, cardiac, renal and hepatic dysfunction were excluded from the study.

Non-pregnant women with known history of diabetes, hypertension, renal and hepatic disorders, polycystic ovarian disease, obesity and women on oral contraceptive pills were excluded from the study. Subjects with family history of hypercholesterolemia were also excluded from the study. Informed consent and a detailed history were taken from all the subjects using a proforma.

Collection of Blood Samples: Five ml of blood sample was taken from all the subjects after 8 hours of fasting under aseptic precautions and transferred into clean dry bottle for estimation of lipid profile.

Biochemical Analysis: All readings were taken using clinical chemistry analyser ERBA (XL-300). Total cholesterol was estimated by enzymatic method using cholesterol reagent set, HDL by enzymatic assay, triglycerides by the enzymatic glycerol phosphate oxidase/ peroxidase method. LDL and VLDL were estimated indirectly using Friedewald formula.

$$\text{LDL} = \text{Total Cholesterol} - [\text{HDL} + \frac{\text{Triglyceride}}{5}]$$

$$\text{VLDL} = \frac{\text{Triglycerides}}{5}$$

Statistical Analysis and Results

The statistical data was entered in Microsoft Excel. The statistical analysis was done using software Epi info version 3.4.3. Mean, standard deviation, standard error and 95% confidence interval were calculated for each parameter.

Significant differences in the means of different parameters of lipid profile in primigravid pregnant women in the three trimesters were done by using the student 't' test. Also, significant differences in the means of different parameters of lipid profile between the pregnant women (primigravida) in the three trimesters and non- pregnant women were done by using the student 't' test.

Table 1: Comparison of various parameters of lipid profile (mg/dl) in the three trimesters of normal pregnant primigravida women

Lipid Profile Parameter (mg/dl)	I Trimester	II Trimester	III Trimester
HDL	75.6	70.3*	65.05 ┼
LDL	86.5	128.26**	161.4 ┼┼
TC	178.6	234.7**	264.3 ┼┼
TG	84.00	180.43**	187.20
VLDL	16.56	36.1**	37.42

* p value < 0.05 (II Trimester Vs I Trimester), ** p value ≤ 0.001 (II Trimester Vs I Trimester), ┼ p value < 0.05 (III Trimester Vs II Trimester), ┼┼ p value ≤ 0.001 (III Trimester Vs II Trimester)

Table 2: Comparison of various parameters of lipid profile ((mg/dl) between non-pregnant women and each trimester of normal pregnancy

Lipid Profile Parameter (mg/dl)	I Trimester	II Trimester	III Trimester	Non- Pregnant
HDL	75.6*	70.3	65.05 ╪	71.83
LDL	86.5	128.26 ┼┼	161.4 ╪	89.93
TC	178.6	234.7 ┼┼	264.3 ╪	178.06
TG	84.00	180.43 ┼┼	187.20 ╪	79.97
VLDL	16.56	36.1 ┼┼	37.42 ╪	15.98

* p value < 0.05 (I Trimester Vs Non- Pregnant), ┼┼ p value ≤ 0.00001 (II Trimester Vs Non-Pregnant), ╪ p value ≤ 0.001 (III Trimester Vs Non- Pregnant)

In the present study, a statistically significant decrease in mean HDL was observed as pregnancy progressed [Table 1]. When compared to normal non pregnant women, pregnant women showed a statistically significant increase in mean HDL value in the first trimester but a statistically significant decrease in mean HDL value in the third trimester [Table 2].

An increase in mean LDL, TC, TG and VLDL values were observed as pregnancy progressed [Table 1]. The LDL, TC, TG and VLDL values showed a statistically significant increase in the second and third trimesters as compared to normal non pregnant women [Table 2]. However, the increase in mean LDL, TC, TG and VLDL values in the first trimester as compared to normal non pregnant women was not statistically significant.

Discussion

In a previous study by Deepak Parchwani [3], serum HDL showed an initial rise in first trimester and then progressive decline in the second and third trimesters of normal pregnant women. The findings of the present study are consistent with the previous reference.

A statistically significant increase in HDL was observed in first trimester when compared to normal non- pregnant women. But there was a significant decrease in HDL in the third trimester in comparison to normal non- pregnant women. The rise in HDL initially could be attributed to estrogen, while decrease in HDL in the last two - third of pregnancy could be due to insulin resistance [6]. The insulin resistance in late gestation reduces lipoprotein lipase activity leading to accumulation of triglycerides in VLDL. This in turn may enhance Cholestryl Ester Transfer Protein (CETP) mediated lipid exchange between VLDL and HDL thereby enriching HDL particles with triglycerides and depleting them of cholesterol thus lowering HDL levels.

In the present study, serum LDL was significantly elevated in the second and third trimesters when compared to the first trimester and also the normal non-pregnant women. However, there was no significant difference in serum LDL between normal pregnant women in the first trimester and normal non- pregnant women. It is consistent with the findings of Mankuta et al [7] and Fahraeus et al[8]. The rise in LDL could be due to raised levels of progesterone during late pregnancy. It could also be a secondary phenomenon caused by the increased conversion of the abundant VLDL in late gestation.

A study by Munoz et al [9] showed a significant elevation in serum TC in the second and third trimesters in comparison to first trimester and also the normal non-pregnant women. However, there was no significant difference in serum TC between normal pregnant women in the first trimester and normal non- pregnant women. The findings of the current study are consistent with the above reference.

In the current study, the triglycerides were elevated in the second and third trimesters when compared to the first trimester and also the normal non-pregnant women. However, there was no significant difference in serum TG between normal pregnant women in the first trimester and normal non- pregnant women. It correlates with the findings of Belo et al [10], Patrizia et al [11] and Lippi G et al[12]. The elevated estrogen levels during pregnancy leads to the rise in TG [13]. The synthesis of endogenous triglycerides is induced by estrogen in the liver. Another reason could be due to decrease in activity of adipose tissue lipoprotein lipase and increase in activity of hormone – sensitive lipase enzymes induced by insulin resistance in late gestation [14]. Increase in activity of hormone sensitive lipase in late gestation results in increased adipose tissue lipolysis. It in turn leads to more hepatic delivery of free fatty acids and glycerol and they are re- esterified to form triglycerides. Maternal triglyceride catabolism is also reduced due to the decreased activity of lipoprotein lipase activity.

In our study, serum VLDL was significantly elevated in the third trimester when compared to pregnant women in the first trimester and also the normal non-pregnant women. The findings correlate with the study of Jayanta De et al in which VLDL was significantly elevated in third trimester of normal pregnant women [15]. Elevation in serum VLDL in late pregnancy could be due to raised maternal estriol and insulin concentrations.

Conclusion

From the present study, it is concluded that serum HDL showed a biphasic pattern. HDL showed significant elevation in the first trimester and then progressively declined in the second and third trimesters in comparison to normal non-pregnant women. Serum LDL, TC, TG and VLDL were elevated in the second and third trimesters when compared to first trimester in normal pregnant women (primigravida) and also when compared to normal non-pregnant women. However, there was no significant difference in serum LDL, TC, TG and VLDL in normal pregnant women in the first trimester (primigravida) when compared to normal non- pregnant women.

Most women have an atherogenic lipid profile by the third trimester of normal pregnancy. The rise in lipid profile in normal pregnancy may be important for the long-term health profile of women because hyperlipidemia is a recognised risk factor for

coronary heart diseases. Further studies need to be done to determine whether hyperlipidemia during pregnancy is a risk factor for cardiovascular diseases in later life.

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