ORIGINAL ARTICLE

STATUS OF LIPID PROFILE IN PREGNANCY

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ABSTRACT

Pregnancy greatly increases demand for metabolic fuels that are needed for growth and development of the fetus and its support structures. The total gestation related energy cost has been estimated at approximately 83000 kcal. The major change in energy expenditure and in the accumulation of fat occurs at different times during pregnancy. In the current study a serial study of serum lipids was performed in a group of women throughout gestation and six weeks post partum while they were having their usual diet and unrestricted daily activity. The control group consisted of non pregnant healthy women of child bearing age. Data was analyzed by SPSS software. All lipid fractions underwent a gradual and persistent rise throughout pregnancy with the exception of HDL-C i.e. a progressive rise was observed in serum total cholesterol, serum triglyceride, serum VLDL and LDL-C as pregnancy advances, while serum HDL-C showed a biphasic pattern, as initial rise and then decline in later third of pregnancy. However, during early pregnancy the values of all lipid fractions do not differ significantly from those for normal non pregnant controls. No significant difference was observed in vegetarian and non-vegetarian group in any of the lipid fractions in different trimester of pregnancy—so diet had no significant influence on lipid synthesis during pregnancy.

KEYWORDS: Lipid profile, pregnancy, fetus, cholesterol

INTRODUCTION

Physiologically, the mother becomes almost a new person during the period of pregnancy. Profound local and systemic changes in maternal physiology are initiated by conception and continued throughout pregnancy.¹Advancement of pregnancy is accompanied by the extra demand of energy. As pregnancy progresses, a well-integrated metabolic shift occurs to ensure an adequate supply of nutrients to a constantly feeding fetus from an intermittently fasting and feeding mother. During early pregnancy, maternal metabolic environment is modified by a rise in serum levels of estrogen and progesterone, pancreatic beta-cell hyperplasia occurs and there is an increase in the secretion of insulin.² Hyperinsulinemia leads to an increase in peripheral glucose utilization, a decline in fasting plasma glucose levels, increased tissue storage of glycogen, increased storage of fats and decreased lipolysis. Freinkel³ was the first to describe the maternal metabolic changes of late pregnancy as "accelerated starvation", when food is unavailable and "facilitated anabolism" when food is ingested. Maternal fuel adjustments during late pregnancy include a sparing of

glucose (for the fetus) and an increased concentration of fatty acids in plasma.

It is known that high concentrations of many of the steroids occur as normal pregnancy advances. Since cholesterol is the source of most of the steroids found in increased amounts in the circulation of normal pregnant patients, the part played by lipid metabolism in pregnancy, becomes all the more intriguing - as cholesterol is a major factor for the development of atherosclerosis.Chauffard⁴ in 1911 undertook the first chemical study of blood lipids during pregnancy and suggested that an increase occurs in the cholesterol level. With the development of more modern techniques various studies observed an increase in various lipid fractions, though the increase was neither consistent in time of appearance nor proportion of changes in various fractions. Most authors believe that the increase in blood lipids is related to the requirements of the fetus and to development of the mammary apparatus. Thus hyperlipidemia is a normal prenatal finding but many questions in this field remained unanswered, such as those pertaining to the exact relationship among the lipid changes during pregnancy. Thus in the present study an attempt has been bade to define more precisely the changes in lipid

fractions during different periods of gestation and at six weeks post partum.

MATERIAL AND METHOD

A total of 325 women were enrolled and the study was carried out from September 2009 to May 2011. They were divided as:

Control group: Comprised 100 non-pregnant, healthy women having normal menstrual function with no evident hormonal deficiency. Among these 59 were vegetarians, while 41 were non-vegetarians.

Study group: Comprised 225 pregnant women, further subdivided into two types according to their dietary habits:

Group I (n=134): Vegetarian women.

Group II (n=91): Non-vegetarian women.

Mean values of age, blood pressure, BMI and other anthropometric parameters did not significantly differ between the groups studied. Fasting blood samples were collected from all the subjects. The blood was drawn into plain tubes and subjected to estimation of lipid profile (Serum Cholesterol by modified Roeschlau's method⁵, Serum triglyceride by McGrowan method.⁶, HDL – C by Bursteim et al method⁷, VLDL and LDL – C was calculated by Friedewald's formula⁸). The same lipid profile estimation was also carried out in study group six weeks post partum. Data analysis was performed with the SPSS statistical software. The results for continuous variables are mean \pm S.D. Student t test for independent samples, analysis of variance (ANOVA) were used in the assessment of the significance of difference between group means.

RESULTS

Table 1 and 2 shows the mean values of lipid profile in non-pregnant and pregnant vegetarian and nonvegetarian women respectively, important findings are:

- No significant difference was observed in the values of serum total cholesterol, serum triglycerides, serum HDL-C, serum VLDL and LDL-C during early pregnancy as compared to normal non-pregnant controls.
- A progressive rise was observed in serum total cholesterol, serum triglyceride, serum VLDL and LDL-C as pregnancy advances, while serum HDL-C showed a biphasic pattern, as initial rise and then decline in later third of pregnancy.
- No significant difference was observed in vegetarian and non-vegetarian group in any of the lipid fractions in different trimester of pregnancy.
- Various lipid fractions decreased considerably at sixth weeks post partum, but still remained significantly elevated from control group.

| Table 1. Lipid | Profile values | in non-pregnant ar | nd pregnant vegetariar | 1 subjects |
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|---------------------------|---------------------|---------------------|--|----------------------|------------------------------|
| Parameters | Vegetarian | Group I (n=134) | | | |
| | control subjects | I trimester | II trimester | III trimester | Six weeks post |
| | (n=59) | | | | partum |
| Total cholesterol (mg/dl) | 169.4 <u>+</u> 32.6 | 182.9 <u>+</u> 31.4 | 222.4 <u>+</u> 34.7* | 251.7 <u>+</u> 44.6† | 202.9 <u>+</u> 26.6‡ |
| Triglycerides (mg/dl) | 81.3 <u>+</u> 15.9 | 84.4 <u>+</u> 12.2 | 141.3 <u>+</u> 25.1* | 226.8 <u>+</u> 43.5† | 117.6 <u>+</u> 12.4 <u>+</u> |
| HDL-C (mg/dl) | 46.7 <u>+</u> 5.7 | 47.0 <u>+</u> 6.3 | 55.6 <u>+</u> 5.5* | 51.1 <u>+</u> 6.3 | 47.4 <u>+</u> 6.1 |
| VLDL-C (mg/dl) | 16.2 <u>+</u> 3.1 | 16.8 <u>+</u> 5.8 | 28.2 <u>+</u> 8.4* | 45.3 <u>+</u> 9.7† | 23.8 <u>+</u> 2.4 <u>+</u> |
| LDL-C (mg/dl) | 103.1 <u>+</u> 19.2 | 114.1 <u>+</u> 20.2 | 132.6 <u>+</u> 21.6* | 154.3 <u>+</u> 25.2† | 132.3 <u>+</u> 22.5‡ |
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* p<0.05 (II trimester Vs control group; II trimester Vs I trimester)

⁺ p<0.01 (III trimester Vs control group; III trimester Vs I trimester)

[‡] p<0.05 (Six weeks post partum Vs control group)

DISCUSSION

Present study shows that serum total cholesterol and serum triglyceride levels increased markedly during pregnancy. Although the total cholesterol increased to maximum in the third trimester, maximum rate of increase occurred during second trimester. Serum triglyceride concentration remained constant until the first trimester, gradually increasing thereafter until delivery. At this time the triglyceride level was 2.8 times higher than that of non-pregnant group. Hypertriglycridemia of pregnancy is distinguished from other hypertriglyceridemias by an increase or maintenance of high density lipoprotein cholesterol. Thus fat appears to be the main form of stored energy in pregnancy—As fat deposition, increases early during the first half of pregnancy, reaching a peak before week 30, and finally mobilization and diminish storage in late gestation. The interpretation for this is that the

hypothalamic center somehow becomes "awakened" to the degree of maternal adiposity. Although the mechanism for the more sensitive regulation of appetite in pregnancy is still unknown, a more important is that the increases in the adipose tissue store anticipates the late gestation fetal growth spurt by beginning in early gestation and reaching a maximum in mid gestation.⁹

No significant change in the lipoprotein cholesterol level occurred during the first trimester. During the second trimester HDL-C and VLDL increased progressively. In the third trimester HDL-C showed a slight decline from the second trimester values, whereas the LDL-C rose by about 30% from second trimester. The most striking alternation occurred in the VLDL which increased approximately 180% above the value in non-pregnant women. Herrera¹⁰, has suggested that rise in triglyceride provides maternal fuel, saving the glucose for the fetus. The rise in LDL-C appears to be necessary for placental steroidogenesis. The rise in triglyceride parallels the rise in estrogen levels during pregnancy. The rise in triglyceride rich VLDL particles during pregnancy is dependent more on an increased rate of synthesis caused by estrogens than on a decrease in the rate of removal¹⁰. The increase in estrogen levels during late pregnancy theoretically should decrease the level of LDL-C. This, however, does not occur and instead, elevations in LDL-C and apo-B are observed.

They could be secondary phenomena caused by increased conversion of the abundant VLDL. High progesterone levels during late pregnancy also could be a contributing factor. The initial increase HDL-C is estrogen dependent, while falling HDL-C in the last half of pregnancy, along with LDL-C changes, correlate with rising levels of human placental lactogen, insulin and insulin resistance¹⁰.

| Table 2: Lipid Prof | ile values in non | -pregnant and p | regnant non-vegetar | an subjects |
|---------------------|-------------------|-----------------|---------------------|--------------|
| | ne values in non | -pregnant and p | regnam non-vegetan | an subjects. |

| Parameters | Non-vegetarian | Group II (n=91) | | | |
|---|---------------------|---------------------|----------------------|----------------------|------------------------------|
| | control subjects | I trimester | II trimester | III trimester | Six weeks post |
| | (n=41) | | | | partum |
| Total cholesterol (mg/dl) | 176.9 <u>+</u> 27.1 | 188.3 <u>+</u> 32.6 | 225.4 <u>+</u> 31.6* | 253.8 <u>+</u> 43.9† | 212.2 <u>+</u> 24.1‡ |
| Triglycerides (mg/dl) | 97.7 <u>+</u> 19.4 | 103.5 <u>+</u> 17.7 | 152.1 <u>+</u> 29.5* | 238.2 <u>+</u> 40.8† | 125.9 <u>+</u> 13.4‡ |
| HDL-C (mg/dl) | 45.3 <u>+</u> 6.1 | 45.7 <u>+</u> 5.2 | 54.1 <u>+</u> 5.7* | 50.4 <u>+</u> 6.5 | 46.1 <u>+</u> 5.7 |
| VLDL-C (mg/dl) | 19.2 <u>+</u> 6.6 | 22.2 <u>+</u> 7.0 | 30.4 <u>+</u> 9.9* | 48.1 <u>+</u> 10.3† | 24.8 <u>+</u> 4.4 <u></u> ‡ |
| LDL-C (mg/dl) | 114.1 <u>+</u> 21.1 | 123.3 <u>+</u> 20.4 | 135.2 <u>+</u> 23.1* | 155.8 <u>+</u> 24.9† | 143.3 <u>+</u> 20.2 <u>+</u> |
| * $c < 0.05$ (II trime action $M_{\rm c}$ as a track of II trime action $M_{\rm c}$ I trime action) | | | | | |

* p<0.05 (II trimester Vs control group; II trimester Vs I trimester)

†p<0.01 (III trimester Vs control group; III trimester Vs I trimester)

‡ p<0.05 (Six weeks post partum Vs control group)

Serum total cholesterol and triglyceride levels fell after delivery, but remained elevated in comparison to control group. Further, it was observed that there was no significant difference in lipid profile parameters in vegetarian and non-vegetarian subjects—so diet had no significant influence on lipid synthesis during pregnancy.

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