Glycemic and Lipedimic Status in Different Trimester of Pregnancy

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Abstract:

The study has been undertaken to assess the lipid profile in different trimester of pregnancy. Under a cross-sectional design, 30 healthy nonpregnant as well as 130 pregnant women, at 3 different trimesters of pregnancy (43 in 1st trimester, 44 in 2nd trimester and 43 in 3rd trimester) were sampled for plasma levels of glucose and lipids profile. All the study groups were age matched [mean ± SD; years: 26.02 ± 2.90 in nonpregnant group; 24.95 ± 4.77 in 1st trimester group; 24.36± 4.95 in 2nd trimester and 25.49± 5.24 in 3rd trimester group respectively]. Plasma total cholesterol, triglyceride, LDL cholesterol and HDL cholesterol level increased throughout the pregnancy. In present study dyslipidemia was found to develop in group with advancing trimester of pregnancy that is the risk of cardio vascular disease.

Introduction:

Pregnancy is associated with an increased in cellular proliferation as a result of uterine enlargement, expansion of blood volume, placental development, and fetal growth [1,2]. Maternal risk factors include disorders associated with endothelial dysfunction such as chronic hypertension, diabetes, kidney diseases and dyslipidemia as well as more specific defects such as Protein C or protein S deficiencies, activated protein C resistance, anticardiolipin antibodies and hyperhomocysteinemia [3,4].

Many pregnant women had elevated serum total cholesterol and triglyceride [5,6,7,8,9,10], compared to non-pregnant women, which would be associated with an increased risk of coronary heart diseases (CHD) and can adversely affect the health of a pregnant woman and her fetus [11]. In numerous studies it has been documented that in normal pregnancy the circulating concentration of triglycerides and cholesterol increases progressively [12,13,14]. Increased in serum lipids are commonly found during the second half of pregnancy. Different studies have repeatedly shown that the concentrations of total and LDL cholesterol and triglyceride were significantly increased in normal pregnancy [5,6,7,8,9,10,15].

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HDL cholesterol was also found significantly increased in normal pregnancy [7,10,15]. However, other workers have reported little change in HDL cholesterol during pregnancy [8,9].

It has been suggested that fasting glucose concentrations are lower in pregnant women than in nonpregnant women, but the postprandial increase in glucose and insulin is substantially higher in the third trimester of pregnancy than in nonpregnant control subjects [16] therefore, pregnancy is a state of increased insulin resistance and insulin secretion and of reduced hepatic insulin extraction [17].

This study was conducted to assess the risk of cardiovascular disease in different trimester of pregnancy.

MATERIAL AND METHODS

Subjects:

Healthy pregnant women (43 in 1st trimester, 44 in 2nd trimester and 43 in 3rd trimester, 18 to 35 years old) and age matched nonpregnant women (n=30, 18 to 35 years old) were sampled. Pregnant women attending antenatal clinics at the BSMMU and Shurwardhy hospital were recruited into the study during antenatal visit at 6-12 week, 13-24 week and 25-36 week of gestation, and the study was conducted as cross sectional design. Trained interviewers collected the information on socioeconomic data, previous obstetric history, and height and weight.

Sample Collection and Analytical Methods:

Venous blood sample was drown from antecubital vein. Fasting blood sample were collected in a haperinized test tube. Blood was allowed to stand for 10 minutes and then centrifuged for 10 minutes at 3000 rpm at 4^0C to separate plasma, which was stored at -70°c until analysis. The plasma samples were analyzed for fasting glucose, TG, Total cholesterol, HDL cholesterol, LDL cholesterol.

Plasma glucose was estimated by enzymatic colorimetric (GOD-PAD) method [18], total cholesterol by enzymatic endpoint method (cholesterol oxidase/peroxidase) in autoanalyzed [19], triglyceride by enzymatic colorimertic method [20], HDL cholesterol by enzymatic colorimetric method [21], LDL cholesterol was calculated by using FriedWald formula [21].

STATISTICAL ANALYSIS

Data are expressed as mean ± SD for parametric values and median (range) for non-parametric values. Comparison between groups were done using Mann-Whitney U test for skewed data and Independent t-test for normally distributed. A p value of

<0.05 was considered as significant. All the statistical analysis were performed with the SPSS data (SPSS Inc, Chicago, IL, USA).

RESULTS

Table-1 depicts the pattern of blood pressure in nonpregnant women and pregnant women in different trimester. All the study groups were age matched [mean± SD ; years: 26.0±2.9 in nonpregnant group; 24.95±4.77 in 1st trimester group, 24.36±4.95 in 2nd trimester group and 25.49±5.24 in 3rd trimester group respectively (p= non-significant)]. Systolic blood pressure in all different trimester of pregnant women were found to be significantly lower compared to nonpregnant women (p<0.005, p<0.001 and p<0.023 in 1st trimester group, 2nd trimester group and 3rd trimester groups respectively).

As depicted in table 2, Family history of hypertension was prominent in nonpregnant group compared to pregnant groups. About 2 (4.7%) pregnant women both in 1st and 3rd trimester group have preeclampsia in their previous pregnancy and 1 (2.3%) of subject in 2nd trimester group have eclampsia of her previous pregnancy.

In clinical examination 25.6% (11), 15.9% (7) and 41.9% (18) of pregnant women were found anemic at 1st, 2nd and 3rd trimester groups respectively. Only 11.6% (5 out of 43) of subjects have visible edema at 3rd trimester group.

Table-3 depicts the glycemic and lipidemic status of study subjects. Fasting blood glucose level were mmol/l median (rang);: 4.53(3.64–5.49) in nonpregnant women, 4.65 (2.78–5.72) in 1st trimester, 4.70(3.4–5.68) in 2nd trimester and 4.0(3.0–5.8) in 3rd trimester groups respectively. Plasma total cholesterol was significantly lower in early pregnancy compared to nonpregnant group mg/dl median (rang);:165 (130-233) in nonpregnant group vs 150 (80-219) in 1st trimester group (p<0.012), but significantly higher in 3rd trimester group 209 (31-308), p<0.001. Compared to nonpregnant women concentration of plasma triglyceride mg/dl median (rang);: 86 (32-350)] was found to increase throughout pregnancy [vs 95 (39-242) in 1st trimester group, (p<0.442); 121 (62-194) in 2nd trimester group (p<0.001); 220 (96-384) in 3rd trimester group (p<0.008)]. Both HDL and LDL concentration were found to be lower in 1st and 2nd trimester groups but higher in 3rd trimester group compared to nonpregnant group.

Table 1: **Pattern of blood pressure in nonpregnant women and pregnant women in different trimester.**

Groups	Age (Years)	SBP (mm Hg)	DBP (mm Hg)
Nonpregnant (n = 30)	26.0±2.90	111±8.90	71±13.88
1st Trimester (n=43)	24.95±4.77	104±12.60	67±9.30
2nd Trimester (n=44)	24.36±4.95	101±11.61	66±7.85
3rd Trimester (n=43)	25.49±5.24	106±10.03	68±8.40

Table -2: Distribution of the study subjects by clinical history, previous obstetric history, and family history of hypertension and Diabetes

	Non Pregnant (n = 30)	1st Trimester (n = 43)	*2nd Trimester (n = 44)*	3rd Trimester (n = 43)
Previous Obstetric History				
PE	-	2 (4.7%)	-	2 (4.7%)
GDM	-	-	-	-
Eclampsia	-	-	1 (2.3%)	-
Anemia	-	11 (25.6%)	7 (15.9%)	18 (41.9%)
Edema	-	-	1(2.3%)	5 (11.6%)
Family history of Hypertension	18 (60.0%)	12 (27.9%)	15 (34.1%)	16 (37.2%)
Family history of DM	8 (26.7%)	7 (16.3%)	12 (27.3%)	7 (16.3%)

Data are presented as number (percentage); n= Number of subject; PE=Preeclampsia; GDM= Gestational Diabetes Mellitus.

Table -3: Glycemic and lipidemic status of the study subjects

Parameter	Group			
	Nonpregnant (n=30)	1st trimester (n=43)	2nd trimester (n=44)	3rd trimester (n=43)
Fasting Glucose (mmol/l)	4.53[a] (3.64 –5.49)	4.65[ab] (2.78-5.72)	4.70[bc] (3.4-5.68	4.0[d] (3.0-5.8)
Cholesterol (mg/dl)	165[a] (130-233)	150[b] (80-219)	165[ac] (99-236)	209[d] (131-308
Triglycerides (mg/dl)	86[a] (32-350)	95[ab] (39-242)	121[c] (62-194)	220[d] (96-384)
HDL (mg/dl)	43[a] (25-68)	34[b] (16-63)	41[ac] (22-77)	50[d] (12-72)
LDL (md/dl)	104[a] (59-175)	96[ab] (17-169)	99[abc] (42-178)	111[acd] (49-208)

Data are presented as median (range) for nonparametric value. n= Number of subject; HDL= High Density lipoprotein; LDL=Low Density Lipoprotein; Values in the same lining not sharing common superscript letter are significantly different $p<0.05$.

DISCUSSION:

Dyslipidemia is a well-established risk factor for coronary heart disease (CHD) in both men and women [22,23,24,25]. Pregnancy is also well documented to produce dyslipidemia, which would be associated with an increased risk of coronary heart diseases (CHD) and can adversely affect the health of a pregnant woman and her fetus (11). Pregnancy has been shown to increase serum total cholesterol and triglyceride [5,6,7,8,9,10]. In the present study plasma cholesterol, triglyceride and LDL cholesterol are found to be higher in 3rd trimester group. These results are similar to previous reports [5,6,7,8,9,10,15]. In the present study HDL cholesterols is also higher in 3rd trimester group than in nonpregnant group .Other workers have reported little change in HDL cholesterol during pregnancy (8,9). Elevated concentration of total cholesterol, triglyceride and LDL cholesterol have proatherogenic effect to develop CVD. Although HDL cholesterol also increases in pregnancy but its antiatherogenic effect is nullified because of higher level of others proatherogenic lipids like cholesterol and triglyceride.

The mechanism whereby pregnancy induces dyslipidemia has not been fully elucidated. The complementary and opposing actions of the individual pregnancy hormones and their changing concentrations during pregnancy would be expected to lead to pronounced alterations in lipoprotein metabolism as gestation progresses.

Desoye *et al.* [7] found a positive correlation between changes in the lipid and lipoprotein concentrations and the changes in the concentrations of the pregnancy hormones estradiol, progesterone, and human placental lactogen (HPL) during gestation [7]. Triglyceride was also positively correlated with increasing concentration of insulin in the second half of gestation. Estrogens can increase the concentration of plasma triglyceride by stimulating hepatic production of the triglyceride rich in very low-density lipoproteins (VLDL) [26] and by inhibition of hepatic and adipose tissue lipoprotein lipases [27]. Estrogens increase the concentration of HDL cholesterol by directly stimulating the production of apolipoproteins AI and AII [27] and indirectly by reducing the catabolism of HDL2 to HDL3 by hepatic lipase. Some authors have suggested that the estrogen-progesterone ratio, which is low in early and in very late pregnancy, is important in the balance of alterations in lipoprotein metabolism throughout pregnancy [7,28]. In conclusion present study indicate , the higher level of TG, total cholesterol and LDL cholesterol in advancing stages of pregnancy.

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