Journal of PharmaSciTech
ISSN: 2231 3788 (Print)
2321 4376 (Online)
Research Article

Lipid Profile Changes During Pregnancy in South Indian Population

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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. Blood lipid concentrations, lipoproteins and apolipoproteins in the Plasma increase significantly during pregnancy. The concentration of serum total cholesterol, serum triglycerides, serum HDL and serum LDL in normal pregnant women increase with increasing gestational age. The increase in maternal lipid profile is in response to the maternal switch from carbohydrate to fat metabolism, which is an alternative pathway for energy generation due to high demand. Pregnancy is a stressful condition in which many physiological and metabolic functions. Lipid metabolism changes during pregnancy. Plasma lipid 30 Pregnant women and 30 Non-pregnant women Blood samples were drawn from all the subjects following a fast of 12 hours and analyzed for Serum Triglycerides (TG), Total cholesterol (TC) and HDL cholesterol, LDL, VLDL, fasting Blood Sugar and Uric acid were analysed. This study results suggest that future lifestyle programs in women of reproductive age with a focus on lowering triglyceride levels (i.e. diet, weight reduction, and physical activity) may help to prevent hypertensive complications during pregnancy and adverse birth outcomes. Additional studies are needed to evaluate whether lowering TG levels by means of lifestyle programs (e.g. diet and physical activity) is beneficial in reducing adverse pregnancy outcome.

Keywords: Pregnancy, Lipid profile, Diet, Pre-eclampsia, Insulin

Introduction

The association of alteration of serum lipid profile in essential hypertension is well documented. An abnormal lipid profile is known to be strongly associated with atherosclerotic cardiovascular diseases and has a direct effect on endothelial dysfunction. Changes in carbohydrate and lipid metabolism occur during pregnancy to ensure a continuous supply of nutrients to the growing fetus despite intermittent maternal food intake. These metabolic changes are progressive and may be accentuated in women who develop gestational diabetes mellitus (GDM). Thus, although both uric acid and changes in lipid profile are associated with metabolic syndrome, these conditions could have opposing or perhaps synergistic effects on maternal and fetal health [1]. Altered lipid synthesis leading to decrease in prostaglandin I2: Thrombaxane I2 (PGI2 : TXA2 ratio) is also supposed to be an important way of pathogenesis in pregnancy induced hypertension [2]. Thus abnormal lipid metabolism seems important in the pathogenesis of pregnancy induced hypertension (PIH) too. The association of alteration in serum lipid profile in essential hypertension is well documented [2]. Hormonal imbalance leading to altered lipid profile in serum is assumed to be the prime factor in etiopathogenesis of pregnancy - induced hypertension (PIH). In this study, we explored uric acid concentrations and lipid profile in pregnant women during the third trimester of gestation.

Material and Methods

30 Pregnant women and 30 Non-pregnant women Blood samples were drawn from all the subjects following a fast of 12 hours and analyzed for Serum Triglycerides (TG), Total cholesterol (TC) and HDL cholesterol (HDL-C) by enzymatic methods with the help of Glaxo kits on ERBA Chem- 5 semi auto analyzer. Serum LDL cholesterol (LDL-C) was calculated by Frederickson-Friedwald's formula according to which LDL cholesterol = Total cholesterol - (HDL cholesterol + VLDL cholesterol). VLDL cholesterol (VLDL-C) was calculated as 1/5 of Triglycerides and Fasting blood sugar and Uric acid were analysed. Data were statistically analyzed by Student's't' test and significance was expressed in term of 'P' value.

Results and Discussion

Some previous studies showed that the most dramatic damage in the lipid profile in normal pregnancy is serum hypertriglyceridemia, which may be as high as two to three folds in the third trimester over the levels in nonpregnant women [3]. In our study also this observation holds true. Here the serum triglyceride concentration showed very significant, increase in the third trimester of normal pregnancy than in the nonpregnant women, the mean value being raised.

The first and second phases of insulin release are 3- to 3.5-fold greater in late pregnancy [4]. Obese pregnant women also develop peripheral and hepatic insulin resistance during the third trimester of pregnancy [5]. The hyperinsulinemic-euglycemic glucose clamp technique indicates that insulin-stimulated glucose disappearance, carbohydrate oxidation, and suppression of endogenous glucose production in obese women are reduced in the third compared with the second trimester. Although the precise mechanism is uncertain, alterations in the hormonal milieu during pregnancy are probably responsible for the reduced insulin sensitivity. Changes in β cell responsiveness occur in parallel with growth of the fetoplacental unit and its elaboration of hormones such as human chorionic somatomammotropin (HCS), progesterone, cortisol, and prolactin. Prevailing insulin resistance produces exaggerated changes in postprandial concentrations of metabolic fuels (eg, glucose, VLDL, and amino acids). Insulin resistance serves to shunt ingested nutrients to the fetus after feeding. n early pregnancy, basal glucose and insulin concentrations do not differ significantly from nongravid values [6]. Basal hepatic glucose production, estimated by using [6,6-2H2]glucose, do not differ at 12–14 wk of gestation. By the third trimester, however, basal glucose concentrations are 10–15 mg/dL (0.56–0.83 mmol/L) lower and insulin is almost twice the concentration of nongravid women. Postprandial glucose concentrations are significantly elevated and the glucose peak is prolonged [7]. Basal endogenous hepatic glucose production (Ra)

increases by 16–30% to meet the increasing needs of the placenta and fetus [3, 8, 9]. Glucose production increases with maternal body weight, such that glucose production per kilogram body weight does not change throughout pregnancy [9]. Endogenous glucose production remains sensitive to increased insulin concentration throughout gestation (90% suppression), in contrast with the progressive decrease in peripheral insulin sensitivity. Increased fasting blood glucose in pregnant women could indicate danger signs which pose a threat to both the woman and the foetus since glucose is an important substrate for metabolism. A high increase in blood glucose during pregnancy could lead to gestational diabetes which is characterized by difficulty during delivery, abnormal foetal weight, adolescent obesity, and neonatal hypoglycaemia. In early pregnancy may be responsible for abnormal foetal development; and neurological defects have been seen in the offspring of diabetic mothers. More specifically, the frequent nocturnal hypoglycaemia some studies observed [9] among insulin-treated diabetic patients may, in severe cases, be a factor responsible for abnormal embryogenesis or perhaps for unexpected death of the foetus during the last trimester of pregnancy.

GDM is accompanied by alterations in fasting, postprandial, and integrated 24-h plasma concentrations of amino acids, glucose, and lipids. These changes include a 3-fold increase in plasma triacylglycerol concentrations during the third trimester of pregnancy, elevation of plasma fatty acids, delayed postprandial clearance of fatty acids, and elevation of the branched-chain amino acids [10]. The principle modulator of this hypertriglyceridemia is oestrogen as pregnancy is associated with hyperoestrogenaemia. Oestrogen induces hepatic biosynthesis of endogenous triglycerides, which is carried by VLDL [11]. This process may be modulated by hyperinsulinism found in pregnancy [12]. Serum triglyceride concentration also rose much more significantly in toxemia of pregnancy in our study which corroborated with the findings of many workers [13, 14]. The above mentioned interactions along with increased endothelial triglyceride accumulation may result in endothelial cell dysfunction in gestosis [15]. Cholesterol is used by the placenta for steroid synthesis and fatty acids are used for placental oxidation and membrane formation. Changes in total cholesterol concentration reflect changes in the various lipoprotein fractions. HDL cholesterol increases by 12 wk of gestation in response to estrogen and remains elevated throughout pregnancy [16]. Total and LDL-cholesterol concentrations decrease initially, but then increase in the second and third trimesters. VLDL and triacylglycerols decrease in the first 8 week of gestation and then continuously increase until term. In the second half of pregnancy, VLDL clearance is altered because of the decreased activity of lipoprotein lipase (LPL) in the adipose and liver and because of the increased activity in the placenta. In the fed state, hepatic LPL is low, but increases with fasting, which increases fatty acid and ketone production for the fetus while the supply of glucose is low.

We have also calculated the ratios between different lipids like LDL-C: HDL-C; TC: HDL-C; TG: HDL-C and HDL-C: VLDL-C (Table 1). In present study there was a significant fall in LDLC: HDL-C in normal pregnant women as compared to nonpregnant women. LDL-C: HDL-C however increased significantly in eclamptic women as compared to normal pregnant women [17, 18].

The link between elevated uric acid concentration and metabolic syndrome in the absence of hypertension may be explained in part by elevated insulin levels reducing urinary excretion of uric acid. However, uric acid may also be an independent risk factor for the development of insulin resistance and subsequent diabetes, as elevated uric acid predates the development of type 2 diabetes in nonpregnant adults [19]. The combined effects of second-trimester insulin resistance and hyperuricemia without hypertension on fetal growth are striking [19]. Uric acid is a co-product of an equation that results in production of superoxide and can itself act as a free radical in a setting of low antioxidants [20]. We have also demonstrated that in an *in vitro* system, uric acid reduces the placental uptake of amino acids by the system A amino acid transporter [20].

Uric acid was associated with insulin resistance in mid pregnancy, even among normal-weight women and those who remained normotensive throughout pregnancy [19, 21]. The relationship between uric acid and birth weight was mediated by the presence of insulin resistance. In the absence of insulin resistance, hyperuricemia was associated with an increased risk for reduced fetal growth among women who remained normotensive [19, 22].

Natural rising of plasma lipids is seen in normal pregnancy but this event is not atherogenic and it is believed this process is under hormonal control but in complicated pregnancy, there is a possible defect in the mechanism of adjusting physiologic hyperlipidaemia. There is a need to do routine tests for total cholesterol during pregnancy in order to differentiate between a physiological increase and a pathological one and to establish a national reference range for Indian Population.

The literature is also lacking of Large sample size studies looking at repetitive insults to the cardiovascular system in multiparous women resulting from multiple changes in Lipid profile levels during pregnancy and if this relationship is affected by pregnancy spacing. Additional research is also needed in high risk pregnancies, such as those affected by diabetes and hypertension [23]. And also additional research is needed to analyze the long term effects of fetal plaque build-up and the risk of subsequent cardiovascular health complications in both term and preterm infants. When studying this relationship one must also take into consideration the genetic aspect of hyperlipidemia compared to pregnancy induced hyperlipidemia as well as the environmental effects (i.e. diet and exercise) on cholesterol levels within the newborn and developing child.

Table 1: Serum lipid profile, Concentration of uric acid and fasting blood sugar in control and pregnancy volunteers

S.No.	Parameters (Mean ± S.D)	Non-pregnant women (No.30)	Pregnant women (No.30)	P value
1	Serum Triglyceride (TG)	134.2 ± 8.2	187.3 ± 12.1	<0.001
2	Total cholesterol	185.6 ± 13.2	206.5 ± 8.5	<0.001
3	HDL cholesterol	45.2 ± 7.4	56.8 ± 9.4	<0.001
4	Serum LDL cholesterol (LDL-C)	130.7 ± 12.5	105.1 ± 11.3	<0.001
5	VLDL cholesterol (VLDL-C)	22.1 ± 6.4	41.3 ± 7.2	<0.001
6	Serum Uric acid	3.5 ± 0.6	4.8 ± 0.8	<0.001
7	Fasting blood sugar	95 ± 6	112 ± 12.1	<0.001

Conclusion

Findings reported in this research suggest that the pregnant women studied had elevated TG, TC, and LDL levels. Increased TG levels are usually. High blood pressure, if present at the same time, could lead to the development of pre-eclampsia. This association may be significant in understanding the pathological process of pre-eclampsia and may help in developing strategies for further research is needed to elucidate the mechanisms and consequences of alterations in lipid metabolism during pregnancy. This study results suggest that future lifestyle programs in women of reproductive age with a focus on lowering triglyceride levels (i.e. diet, weight reduction, and physical activity) may help to prevent hypertensive complications during pregnancy and adverse birth outcomes. Additional studies are needed to evaluate whether lowering TG levels by means of lifestyle programs (e.g. diet and physical activity) is beneficial in reducing adverse pregnancy outcome.

Conflicts of interest

The author declares no competing interest.

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