

## Serum Lipid Profile and Uric Acid Concentration during Pregnancy

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### Abstract

**Objectives:** To investigate whether lipid profile and serum uric acid concentration changes during pregnancy or not.

**Method:** 10 non pregnant women and 10 pregnant women were enrolled in the study, have an average age 25-30 years and the same body mass index. Blood samples were drawn from all the subjects following a fasting of 8 hours and analyzed for serum lipid profile and serum uric acid.

**Result:** The results showed that pregnancy period may change lipid profile and serum uric acid, although the volume of sample is small for correlation between the pregnancy period and changes in serum uric acid and lipid profile. This will require large number of volunteers. But, this study give opinion about these changes.

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### Introduction

In pregnancy, elevated serum uric acid and changes in lipid profile is associated with poor perinatal outcomes, including small-for-gestational-age (SGA) infants and preterm birth [1]. This may lead to insulin resistance. Insulin resistance, however, is a component of gestational diabetes and increasing risk for excessive fetal growth, as well as gestational hypertension and pre-eclampsia[2]. 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 [3]. Altered lipid synthesis leading to decrease in prostaglandin I<sub>2</sub>: Thromboxane I<sub>2</sub> (PGI<sub>2</sub> : TXA<sub>2</sub> ratio) is also supposed to be an important way of pathogenesis in pregnancy induced hypertension [4]. 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[4]. Hormonal imbalance leading to altered lipid profile in serum is assumed to be the prime factor in etiopathogenesis of pregnancy - induced hypertension (PIH). PIH includes a group of hypertensive disorders developed due the gravid state [3]. Obviously the association of serum lipid profile with gestational protein uric hypertension is highly suggested to reflect some new diagnostic tools[2]. Moreover, the hormonal imbalance is a prime factor for the etiopathogenesis of PIH and this endocrinal imbalance is well reflected in alteration of serum lipid profile [5]. In this study, we explored uric acid concentrations and lipid profile in pregnant women during the third trimester of gestation.

## Material & Method

The study include the following groups:-

Group A comprises of 10 non pregnant normotensive apparently healthy women.

Group B consist of 10 women having normal uncomplicated pregnancy without hypertension at 18 -20 weeks of gestation. B group has the same range of body mass index (BMI) and in the same range of age 25-30 years . The study was performed in the Department of Biochemistry of pharmacy College in collaboration with Baghdad Teaching Hospital , Obstetrics Department during the period of 1/08/2010 to 1/9/2010.

All the subjects were ranging in age from 25 to 30 years with similar low socio-economic status and dietary habit. They were non smokers and non alcohol drinkers. No subject in the study was suffering from acute or chronic illness during the study period nor they had any past history of cardiac, renal, hepatic dysfunction, gout .This is evaluated by taking medical history, surgical history and drug history from each volunteer. Both group have normal range of renal function test and liver function test.

Blood samples were drawn from all the subjects following a fasting of 8 hours and analyzed for Serum Triglycerides (TG), Total cholesterol (TC) and HDL cholesterol (HDL-C) by enzymatic methods using 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. Fasting blood sugar are measured by using a colorimetric assay from kit G7519 (Pointe Scientific Inc, Canton, MI) using glucose oxidase and a quinone imine dye. Uric acid was measured using a colorimetric assay (Pointe Scientific Inc) kit U7581-120 using uricase. Data were

statistically analyzed by Student's 't' test and significance was expressed in term of 'P' value ( $p < 0.001$ ).

## Results & Discussion

This study show that there was a significant rise in the fasting triglycerides, total cholesterol and VLDL-C levels in pregnancy women ( $P = < 0.001$ ) compared to healthy women . This is also shown by Suchanda Saua *eta* [6].

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 non pregnant women [7] In our study, the serum triglyceride concentration showed highly significant ( $P < 0.001$ ) increase in the third trimester of normal pregnancy than in the non pregnant women. The principle modulator of this hypertriglyceridemia is estrogen, as pregnancy is associated with hyperoestrogenaemia [8,5]. Estrogen induces hepatic biosynthesis of endogenous triglycerides, which is carried by VLDL [9]. This process may be modulated by hyperinsulinism found in pregnancy [10]. Increased TG, found in pregnancy induced hypertension, is likely to be deposited in predisposed vessels, such as the uterine spiral arteries and contributes to the endothelial dysfunction, both directly and indirectly through generation of small, dense LDL [9]. Moreover, this hypertriglyceridemia may be associated with hypercoagulability [9]. Though in our own study, the mean value of HDL-C was higher in the third trimester of normal pregnancy over the non pregnant women. This is due to Oestrogen is responsible for induction of TG and HDL and suppression of serum LDL and oestrogen level falls in pregnant women [Dutta, D.C. (2001) Hypertensive disorders



in pregnancy, In: Textbook of Obstetrics, Ed. Konar, H.L., 5th edition, New Central Book Agency, Kolkata, p 234-55]. The Low level of HDL in pregnant women is however not only because of hypooestrogenaemia but also due to insulin resistance [Kaaja, R., Tirkkanen, M.J., Viinikka, L. and Ylikorkala, O. (1995) Serum lipoproteins, insulin and urinary prostanoid metabolites in normal and hypertensive pregnant women, *Obstet. Gynecol.* 85(3), 353-6.]. In present study, serum VLDL-C level rose significantly ( $P < 0.001$ ) in the third trimester of pregnancy in comparison to non-pregnant women, which is perhaps due to hypertriglyceridemia leading to enhanced entry of VLDL that carries endogenous triglyceride into circulation. The VLDL-C level, as reported by some researchers, might rise upto 2.5 folds at term over the pre-pregnancy level [9]. VLDL level further increase in PIH as evidenced in the present study in corroboration with those of other workers [9] perhaps due to increased VLDL lipoproteins which accumulate over the maternal vascular endothelium, particularly those of uterine and renal vessels [9,10]. Further, VLDL may cause injury to the endothelium, while a specific toxicity-preventing-activity-protein (the pI 5.6 form

of plasma albumin) protects against the VLDL-induced injury in the. A significant fall in LDL-C level in third trimester of normal pregnancy as observed in present study may be attributed to hyperestrogenaemia, while LDL-C level increases significantly in PIH. A significantly higher level of beta-lipoprotein was also reported by many workers in third trimester of gestational proteinuric hypertension (11,12,13). This study showed that serum uric acid increased significantly in pregnant women than non pregnant women. A decreased glomerular filtration rate may contribute to an increased uric acid, but this likely occurs later in pregnancy closer to the time of preeclampsia diagnosis [14]. 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 [14]. The combined effects of second-trimester insulin resistance and hyperuricemia without hypertension on fetal growth are striking [14]. (table 1).

**Table (1):** Serum lipid profile ,concentration of uric acid and fasting blood sugar in control and pregnancy volunteers .

Parameters Mean $\pm$ S.D	Non-pregnant women (No.10)	Pregnant women (No.10)	P value
Serum Triglyceride (TG)	134.2 $\pm$ 8.2	187.3 $\pm$ 12.1	<0.001
Total cholesterol (TC)	185.6 $\pm$ 13.2	206.5 $\pm$ 8.5	<0.001
HDL cholesterol	45.2 $\pm$ 7.4	56.8 $\pm$ 9.4	<0.001
Serum LDL cholesterol (LDL-C)	130.7 $\pm$ 12.5	105.1 $\pm$ 11.3	<0.001
VLDL cholesterol (VLDL-C)	22.1 $\pm$ 6.4	41.3 $\pm$ 7.2	<0.001
Serum uric acid	3.5 $\pm$ 0.6	4.8 $\pm$ 0.8	<0.001
Fasting blood sugar	95 $\pm$ 6	112 $\pm$ 12.1	<0.001

In rats, hyperuricemia caused by blocking uricase leads to systemic and glomerular hypertension, as well as arteriolar damage, and these effects can be reversed by a selective inhibitor of uric acid synthesis.[8] . Uric acid is relevant to oxidative stress and is a prominent antioxidant.[9]

However, 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. [12] 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.[12]

The mechanism by which insulin resistance might attenuate these effects is unclear. Insulin resistance increases substrate availability for the fetus. Perhaps the effects of hyperuricemia are overcome to an extent in the setting of excess glucose [14]. Uric acid was associated with insulin resistance in midpregnancy, even among normal-weight women and those who remained normotensive throughout pregnancy[14]. The relationship between uric acid and birthweight 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 [14].

The study showed that pregnancy period may change lipid profile and serum uric acid ,although the volume of sample is small for correlation between the pregnancy period and changes in serum uric acid and lipid profile .This will require large number of volunteers .But , this study give opinion about these changes.

## References

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