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Original Research Article

A Comparative Study of Serum Triglyceride Levels in Normal Pregnancy and Preeclampsia

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ABSTRACT

Background and Objective: Pregnancy is a time of excitement and wonders tempered by natural concerns about the well-being of mother and developing foetus. Hypertensive disorders complicate 5 to10%of all pregnancies along with haemorrhage and infection forms the deadly triad that contributes greatly to maternal mortality and morbidity rates. How pregnancy incites or aggravates hypertension remains unsolved despite decades of intensive research. Indeed hypertensive disorders remain among the most significant and intriguing unsolved problem in obstetrics. Hypertriglyceridemia due to insulin resistance is accentuated in women with preeclampsia and eclampsia. Elevated triglycerides are found to be associated with oxidative stress and vascular dysfunction. The study was done to compare the serum triglyceride levels in normal pregnancy and preeclampsia. The values obtained were compared with normal nonpregnant women.
Methods: The present study was undertaken to find out the alterations in the circulating levels of serum triglyceride in normal pregnancy and preeclampsia when compared to normal nonpregnant women. The study was conducted by taking a statistical sample size of 30 subjects (18- 35 years) in each group. Data were analyzed using ANOVA. Significance level was fixed at $p<0.05$.
Results and Interpretation: The mean serum triglyceride levels of normal pregnant women in the third trimester was (221. 63 ± 39.58 mg /dL), significantly higher than in normal non pregnant women (106.27 ± 13.16 mg /dL). The mean serum triglyceride level of preeclamptic patients in this study (322.17 ± 76.75 mg /dL) was significantly higher than the mean serum triglyceride value (221.63 ± 39.58 mg /dL) in normal pregnant women and normal non pregnant women (106.27 ±13.16 mg /dL).
Conclusion: The high triglyceride levels seen during the last trimester of normal pregnancy is further increased in preeclampsia. The hypertriglyceridemia during preeclampsia is a probable contributor to future cardiovascular disease.

Keywords: Preeclampsia, Triglyceride, Endothelial dysfunction

INTRODUCTION

Pregnancy is a time of excitement and wonders tempered by natural concerns about the well-being of mother and developing foetus. The test of any civilization is a measure of consideration and care which it gives to the weaker sections of the society. Indices that reflect poor obstetrical and perinatal out comes would lead to the assumption that medical care for the entire population is lacking. Hypertensive disorders complicate 5 to 10% of all pregnancies along with haemorrhage and infection forms the deadly triad that contributes greatly to maternal mortality and morbidity rates. [(1)] How pregnancy incites or aggravates hypertension remains unsolved despite decades of intensive research.

Indeed hypertensive disorders remain among the most significant and intriguing unsolved problem in obstetrics.

Preeclampsia is a multifactorial disease. [(2)] However endothelial dysfunction and oxidative stress contributes the pathophysiology of preeclampsia. [(3)] Hypertriglyceridemia due to insulin resistance is accentuated in women with preeclampsia and eclampsia. Elevated triglycerides are found to be associated with oxidative stress and vascular dysfunction. The increase in plasma triglyceride concentrations have been reported in groups of women with preeclamsia before the onset of clinically evident disease. [(4)] Such profile may also be a potential contributor to endothelial cell dysfunction, which is a central feature in the pathophysiology of preeclampsia.

MATERIALS AND METHODS

The case control study was done to find out the alterations in triglyceride levels in preeclampsia and normal pregnant woman when compared with normal nonpregnant woman. The study was done among 90 subjects between 18 – 35 years, divided into 3 groups of 30 each. Preeclampsia patients were diagnosed as blood pressure more than 140 / 90 mm Hg on more than two occasions and urine albuminuria 30 mg / dl (> 1 ± dip stick) after 20 weeks of gestation .

Women with any systemic diseases or bad obstetric history were excluded from the study and all of them abstained from smoking and alcoholism. All antenatal women were in the third trimester, (28- 40 weeks) of gestation. Informed written consent was obtained from all the subjects. Blood pressure recording along with a detailed physical examination was done.

Blood samples were collected after 8-12 hours of fasting. Urine protein was estimated using magistik reagent strips. [(5)] Triglyceride levels in the serum were estimated using the reagent kit; Triglycerides – GPO Trinder method. [(6)]

The results were expressed as mean +/- SD and analyzed using Statistical Package for Social Sciences (SPSS) version 16. Analysis Of Variance (ANOVA) was used to test whether there is significant difference among two or more independent groupsand P value <0.05 was taken as the level of significance.

RESULTS

The mean serum triglyceride levels of normal pregnant women in the third trimester was (221. 63 ± 39.58 mg /dL), significantly higher than in normal non pregnant women (106.27 ± 13.16 mg /dL) (Table 1). The mean serum triglyceride level of preeclamptic patients in this study (322.17 ± 76.75 mg /dL) was significantly higher than the mean serum triglyceride value (221.63 ± 39.58 mg /dL) in normal pregnant women and normal non pregnant women (106.27 ±13.16 mg /dL).

TABLE 1: Comparison of serum Triglyceride levels in three groups

Triglyceride (mg/dL)				
	Normal non- pregnant Control group	Normal pregnant Group I	Preeclampsia Group II	p value
MEAN ±SD	106.27±13.16	221.63±39.58	_	0.001 (significant)
	106.27±13.16	_	322.17±76.75	0.001 (significant)
	_	221.63±39.58	322.17±76.75	0.001 (significant)

In this table the mean values of serum triglyceride in preeclampsia patients (322.17 ± 76.75) is significantly higher than mean triglyceride values in normal nonpregnant (106.27 ± 13.16) and normal pregnant women (221.63 ±39.58). The mean triglyceride value in normal pregnant women (221.63± 39.58) is higher than normal nonpregnant women (106.27± 13.16) and the difference is statistically significant.

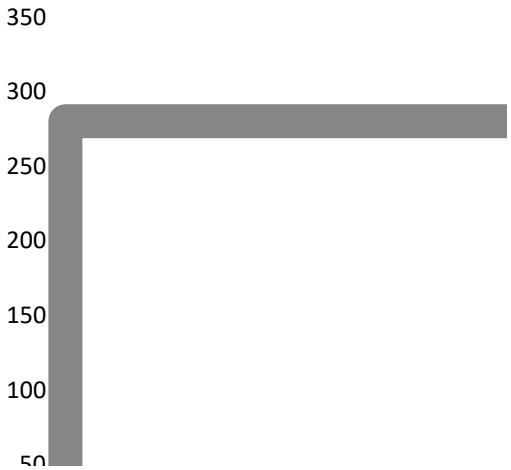

FIGURE 1

DISCUSSION

The significant elevation in fasting triglycerides by late gestation could be attributed mainly to decreased adipose tissue lipoprotein lipase activity due to insulin resistance. Also gestational insulin resistance which is seen to peak during the last trimester causes increased hormonesensitive lipase activity in maternal adipocytes causing adipocyte triglyceride hydrolysis. Thus glycerol and free fatty acid released are available for synthesis of VLDL and thus plasma triglycerides increases towards the last trimester. [(7)] Another principle modulator of this hypertriglyceridemia could be estrogen, as normal pregnancy is associated with elevated estrogen levels. [(8)] Estrogen induces endogenous synthesis of triglycerides in the liver. Some studies have showed that VLDL level is increased in the last trimester of pregnancy. Serum hypertriglyceridemia might be related to enhanced entry of VLDL from liver into the circulation, which carries endogenous triglycerides. [(9)]

The significant hypertriglyceridemia in preeclampsia could be attributed to the markedly high gestational insulin resistance seen during the third trimester. [(10-14)] The insulin resistance causes increased activity of hormone-sensitive lipase in adipocytes and decreased adipose tissue lipoprotein lipase activity than seen during third trimester of normal pregnancy. The expression of LDL receptor and VLDL were also found to be decreased in the placenta of preeclampsia patients. Thus there is decreased clearance of triglycerides from plasma, along with increased production leading to hypertriglyceridemia. The increased lipolytic activity in maternal adipose tissue releases free fatty acids and glycerol. This is taken up by liver and re-esterified for synthesis of VLDL triglycerides. Although the estrogen levels were found to be low in preeclampsia, the exaggerated insulin resistance during the third trimester is mainly responsible for the markedly elevated triglyceride levels. The development of atherosclerosis in the placental spiral arteries of preeclamptic patients indicate that elevated levels of triglycerides are involved in this disorder.

Hypertriglyceridemia may increase the prevalence of ROS by stimulation of leucocyte NADPH oxidase, by lowering the concentration of protective HDL or by increasing the formation of smaller peroxidation susceptible LDL particles. [(11)] ROS, in turn, can decrease the bioavailability of NO, either directly by destruction or indirectly by formation of oxidized lipids that subsequently destroy NO or decrease NO synthase. Also dyslipidemia can adversely affect NO homeostasis.

Studies using gradient gel electrophoresis have reported that the diameter of principal LDL sub class is significantly decreased in preeclampsia relative to normal pregnancy. The smaller variation of LDL has been shown to contribute to endothelial dysfunction in preeclampsia through stimulation of thromboxane synthesis by endothelial cells and increase in intracellular calcium in smooth muscle. [(15)]

Gardner and Stampfer et al have shown that small LDL cholesterol is associated with coronary artery disease. [(16,17)] These small dense particles do not bind readily to LDL receptor and therefore remain in the circulation for longer. [(18)] They also penetrate the arterial intima better than do longer ones and they

are more readily oxidized, possibly because they contain less vitamin E and other antioxidants. Finally, their uptake into macrophages to create foam cells, and thus initiate atherogenesis is facilitated. This may explain their identification as an independent risk factor coronary heart disease.

Thus lipid abnormalities characteristic of insulin resistance are accentuated with established preeclampsia. [19-22] Also the exaggerated hyperinsulinemia relative to normal pregnancy is well described. [23-25] As obesity is a major contributor to insulin resistance and a recognized risk factor for preeclampsia, interventions aimed at weight reduction before pregnancy and/ or avoidance of excessive weight gain during pregnancy may have merit. Also regular exercise, which improves insulin sensitivity, reduces risk. The use of insulin sensitizer drugs like metformin or even hypolipidemic drugs may also warrant study in women at high risk for preeclampsia.

CONCLUSION

The high triglyceride levels seen during the last trimester of normal pregnancy is further increased in preeclampsia. The high triglyceride levels seem to increase the risk of placental vascular disorders, which triggers endothelial dysfunction, atherosclerosis and thrombosis. The findings reported in the present study corroborate the growing number of studies, showing that women with preeclampsia present lipid profile abnormalities and at increased risk factor for future cardiovascular complications. Identification of women who display these markers during or after pregnancy might allow for early interventions like regular exercise, prevention of obesity, antioxidants to diminish or delay future cardiovascular complications.

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