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Dyslipidemia in Preeclamptic Women From Rural Population

Medical Science

KEYWORDS : Preeclampsia, dyslipidemia, lipid profile

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ABSTRACT

Preeclampsia is a pregnancy specific disease which affects both the mother and the unborn baby. It affects five to eight percent of all pregnancies and is one of the leading causes of maternal mortality and preterm delivery. It is supposed to be associated with high mortality, among the serious complications of pregnancy. This study analyzes the lipid profile among patients with preeclampsia and compares it with healthy pregnant women. Total of 50 samples were collected from the Gynecology and Obstrectics units of NIMS Hospital, 25 rural preeclamptic women were selected as the study group, 25 age-matched healthy pregnant females were selected as controls. The comparison of Fasting sugar, Total cholesterol (mg/dl), Triglyceride (mg/dl), HDL-cholesterol (mg/dl), LDL-cholesterol (mg/dl) and VLDL-cholesterol (mg/dl) between control and study group was analyzed using unpaired "t"-test. Dyslipidemia is evidenced in the form of elevated serum TC, TG, LDL and VLDL with decreased HDL levels in preeclampsia group as compared to healthy pregnant controls

Introduction:

Preeclampsia is a pregnancy specific disease which affects both the mother and the unborn baby. It affects five to eight percent of all pregnancies and is one of the leading causes of maternal mortality and preterm delivery.[1] Preeclampsia most commonly occurs during the last trimester of pregnancy it arises in the early 2nd trimester 14-20 weeks.[2] It is supposed to be associated with high mortality, among the serious complications of pregnancy. Preeclampsia is the most common; it may affect both mother and the fetal survival.[3] Eclampsia defined as tonic colonic seizures in a pregnant or recently delivered woman not attributable to other causes than preeclampsia or gestational hypertension, complicates about 1 to 2% of all cases of severe preeclampsia.[4] Preeclampsia is a complex pregnancy complication associated with increased BP (blood pressure) accompanied by proteinuria, edema, or both.[5] This condition seems to be linked to oxidative stress within the placenta. Increased production of lipid peroxides, thromboxane and cytokine triggered vascular and organic dysfunction have been observed in preeclampsia.[6] Preeclampsia is one of the causes of high morbidity for both mother and fetus, especially in developing countries.[7] It is characterized by hypertension, proteinuria and edema. Several risk factors have been identified in women who develop preeclampsia, they include null parity, history of preeclampsia in previous pregnancy, extremes of maternal age, multi fetal gestation, several preexisting maternal diseases (chronic hypertension, diabetes mellitus, chronic kidney disease, vascular or connective tissue disease, thrombophilia, high body mass index (BMI)), and possibly, long interval between pregnancies. Of these, obesity (where the risk of preeclampsia increases threefold), is the most common. As over 30% of women of reproductive age are obese, increased BMI may be responsible for 30-40% of all cases of preeclampsia. Despite known risk factors, it is not possible to predict which woman will develop preeclampsia during pregnancy. Reproductive implications of pregnancy complicated by hypertensive disorder are well known, and women are advised regarding risk of preeclampsia in subsequent pregnancies.[8]

Early pregnancy dyslipidemia is associated with an increased risk of Preeclampsia.[9] In pregnancy, lipolysis of TG-rich lipoproteins is reduced because of decreased lipolytic activities of the mother[10] whereas placental VLDL receptors are up regulated.[11] This results in are routing of TG-rich lipoproteins to the fetoplacental unit.[12] However, in Preeclampsia, the vascularization of the fetoplacental unit may be impaired, resulting in yet-undefined compensator mechanisms that may further increase synthesis of Maternal Triglyceride (TG) levels. Dyslipidemia is elevation of plasma cholesterol, triglycerides (TGs), or both, or a low high density lipoprotein (HDL) level that contributes to development of atherosclerosis, heightened gestational insulin resistance, abnormally increased concentration of TNF-α, and increased human placental lactogen are thought to contribute to dyslipidemia during pregnancy [13] and early pregnancy dyslipidemia is also said to be associated with and increased risk of preeclampsia therefore, the present study was undertaken to find out the changes in the concentration of blood lipids in preeclamptic women living in the rural areas.

2. Material and methods:

2.1 Ethical clearance: This study was carried out in the Department of Biochemistry in collaboration with the Department of Gynecology and Obstrectics NIMS Medical College and Hospital, Shobha Nagar, Jaipur, Rajasthan. The institu-

tional ethical clearance was obtained from Ethical Committee of the college.

2.2 Study population: The total number of subjects in the study were 50, 25 Preeclamptic women and 25 healthy pregnant women. The subjects were selected from the Department of Obstetrics and Gynecology, NIMS University Hospital, Shobha Nagar, Jaipur.

The selected subjects were divided into two groups

GROUP 1: Preeclamptic women as control. n = 25

GROUP 2: Healthy pregnant women as study group. n=25

Personal and clinical history of patients was recorded on a well-designed questionnaire which included information regarding socio demographic characters, maternal age, age at marriage, Paragravida, Weight, Height, BMI and Blood Pressure.

2.3 Specimen Collection and Processing
Following an overnight fast, 5ml of blood sample was collected from both control and study groups through routine method by applying aseptic technique and tourniquet as short a time as needed in a vacutainer tube. After coagulation of blood the sample was centrifuged for 10 minutes at 3000rpm to get a clear and cell free serum. The samples were isolated, properly labeled and stored.

2.4 Biochemical analysis
Biochemical analysis of Fasting blood sugar and Lipid Profile parameters viz. Triglycerides (TG), Total Cholesterol (TC), High Density Lipoprotein- Cholesterol (HDL-C), Low density Lipoprotein Cholesterol (LDL-C) and Very Low Density Lipoprotein Cholesterol (VLDL-C) was done using kit methods on Auto analyzer (NANOLAB 150) in Biochemistry Laboratory of NIMS Hospital and Medical College.

Plasma fasting blood glucose level estimated by glucose Oxidase/peroxidase method (Tinder, 1969).

Serum Triglycerides and total cholesterol were measured by using enzymatic method cholesterol oxidase phenolaminoantipyrine (CHOD/PAP) and Glycerol peroxidase, Adenosine Diphosphate (GPO/ADPS) (Herbert 1984)

Serum HDL-C was measured by using kit of polyethylene glycol cholesterol oxidase, phenolaminoantipyrine (PEG/CHOD-PAP) method (Herbert 1984).

Serum LDL –C was calculated by Frederickson – Friedwald's Formula according to which LDL-C =TC – (HDL Cholesterol – VLDL Cholesterol). VLDL cholesterol was calculated as 1/5 of triglycerides.

2.5 Measurements
Height
Height of Subjects was determined by the use of a vertical calibrated scale in the standing position. Subjects removed their shoes prior to height measurement. Heel to head, Crown length was measured in centimeters.

Weight
Weight of subjects was measured by means of an accurate physical balance in the standing position and with minimum clothing. The balance was pre- tested with known standard weights.

Body Mass Index (BMI)

This was calculated from the measurements of weights and heights by using the following formula.

BMI = Weight (Kg)/Height (meters2)

Normal range = 18 – 24kg/m2

Blood Pressure Recording
Both systolic and diastolic blood pressure was measured with a standard mercury sphygmomanometer and standard arm cuff at the right arm. The recordings were taken at interval of 5minutes each and their mean values taken. The degrees of hypertensive were screened for presence of protein in urine by using dipstick method.

Normal blood pressure (BP): Systolic -120mmHg

Diastolic- 80mmHg

3. Observations and results: Table 1 shows that control and study groups were age- matched and there were significant differences in weight and BMI in the study and control ($p< 0.001$).

Table 1: - Demographic and clinical parameters of control and patient groups.

PARAMETERS	CONTROL	PATIENT
Age (Yr.)	26.0 ± 1.24	25.12 ± 1.36
Weight (Kg)	48.12 ± 2.20	52.8 ± 5.66*
BMI (Kg/m^2)	21.36 ± 1.014	23.58 ± 2.31*

Result in Mean ± S * = $p< 0.01$ Statistically significant change

Tabel 2: Comparison of Fasting blood sugar & Lipid Profile of Control & Study group.

Parameters (mg/dL)	Control group@	Study group
Fasting Sugar (FBS)	88.6 ± 7.9	89.9 ±9.64^{NS}
Triglyceride (TG)	135.4 ± 50.24	206.24 ± 39.66@
Total Cholesterol (CHO)	170.6±26.2	203.18 ± 27.6@
HDL Cholesterol	50.1 ± 9.57	41.13 ± 6.26@
LDL Cholesterol	93.1 ± 25.81	123.94 ± 25.2@
VLDL Cholesterol	27.2 ± 9.28	38.48 ± 10.2@

Result in Mean ± SD * $p< 0.001$ NS = Not Significant

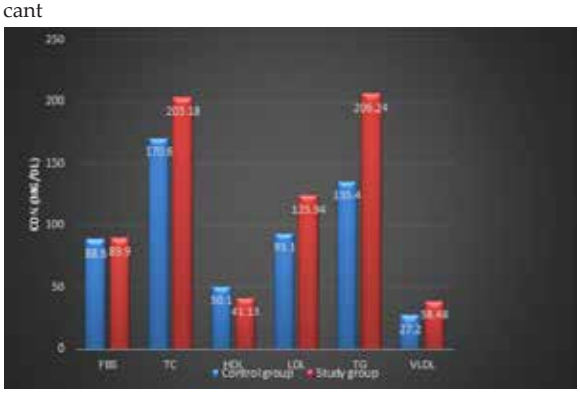

Fig.1: Comparison of blood sugar and lipid profile in control and study group.

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Table No. 2 & Figure 1 show that the level of fasting blood sugar did not show any significant change. The level of triglyceride, total cholesterol, LDL and VLDL was found to be significantly increased in study group as compared to control ($p < 0.001$). The value of HDL in study group was significantly decreased ($p<0.001$) as compared to control group.

4.Discussion:

In present study, the level of triglyceride, total cholesterol, HDL cholesterol, VDL cholesterol, VLDL cholesterol shows significance difference as compared with control group. The mean value of fasting blood sugar (FBS) in study group was 89.9 ± 9.64 mg/dl and triglyceride was 206.14± 39.66 mg/dl. In control group the mean value of FBS was 88.8 ± 7.9mg/dl and triglyceride was 135.4 ± 50.24 mg/dl. It was found that the level of TG was significantly increased in study group as compared to control group ($P<0.001$). The mean value of Total cholesterol in study group was 203.18 ±27.6, HDL cholesterol was 41.13 ± 6.26, LDL cholesterol was 123.94 ± 25.2, VLDL cholesterol was 38.48 ± 10.28. In control group the mean value of Total cholesterol was 170.6 ± 26.2, HDL cholesterol was 50.1 ± 9.57, LDL cholesterol was 93.1 ± 25.81, VLDL cholesterol was 27.2 ± 9.28, It was found that the level of Total cholesterol, HDL cholesterol, LDL cholesterol, VLDL cholesterol shows significance difference as compared with control group. A similar study by Enquobahrie et al., (2004) also shows similar results and explains that hypertriglyceridemia is probably a consequence of competition between chylomicrons and very low-density lipoprotein cholesterol for the lipoprotein lipase. Akhavan et al, 2009 reported that the association between hyperlipidemia and severity of preeclampsia was evaluated and patients with severe preeclampsia had significant increase in plasma triglyceride, cholesterol, and LDL-C levels compared with control. Gohil et al (2011) also measured lipid profile in subjects of preeclampsia and found that dyslipidemia is significantly evident in preeclampsia and plays an important pathological role.

5.Conclusion:

Dyslipidemia is evidenced in the form of elevated serum TC, TG, LDL and VLDL with decreased HDL levels in preeclampsia group as compared to healthy pregnant controls. Whereas, there was no significant difference in FBS level between preeclampsia and control groups.

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