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Dyslipidemia In Preeclampsia Risk Factor For Future Maternal Cardiovascular Diseases

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Abstract:
Introduction: *Preeclampsia is a potentially devastating disease of pregnancy that complicates 2%–8% of all pregnancies in the United States and can threaten the life of both the mother and her unborn child (1, 2). Manifesting after 20 weeks of gestation, preeclampsia is a multiorgan disorder defined as de novo hypertension (systolic blood pressure ≥140 mm Hg; diastolic blood pressure ≥90 mm Hg) combined with proteinuria (≥300 mg/24 hours), as defined by the American Congress of Obstetricians and Gynecologists (3).*
Material and Methods: *This cross sectional study was carried out in Department of Obstetrics and Gynaecology, Patliputra Medical College, Dhanbad, Jharkhand. 80 women with preeclampsia and 80 normotensive pregnant women as controls were included in the study after obtaining their informed consent. The diagnosis of Preeclampsia was done as per the norms of American college of Obstetrics and Gynecologists. All the participants were inquired by a questionnaire containing their personal history, family history of PIH, Twins, Hypertension and Diabetes, Drug history and their symptoms.*
Results: *The values of the lipid fractions were tabulated as mean ± SD (Table 1). Total cholesterol, triglycerides, LDL-C, VLDL-C were significantly elevated and HDL-C was significantly decreased in Preeclampsia group compared to control (p< 0.0001). The risk ratios TC/HDL, TGL/HDL, and LDL/HDL were significantly elevated and HDL/VLDL significantly decreased in Preeclampsia group compared to control (p< 0.0001) (Table 2).*
Conclusion: *Pre-eclampsia is an under-recognized risk factor for IHD,chronic hypertension, peripheral vascular disease, and stroke. Potential mechanisms for CVD include endothelial,vascular, and metabolic dysfunction encountered during pre-eclampsia, which does not recover post-partum. Alternatively, pre-eclampsia during pregnancy could be a marker for future CVD, as both conditions share similar genetics,similar pathophysiology, such as hyperlipidemia, and several common risk factors, such as obesity, diabetes mellitus, and renal disease. Given that CVD remains the largest cause of death among women, new studies investigating this high-risk condition should be carried out to understand the disease further, and to develop novel therapeutic strategies to manage this condition in order to reduce the global burden of CVD among women.*

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I. Introduction:

Pre-eclampsia is a multisystem placentally mediated disease, which usually occurs after 32 weeks of gestation, with distinctive features of hypertension and proteinuria. Pre-eclampsia affects 2% to 8% of all pregnancies (1). Therefore, this topic is of significance to the cardiovascular health of over 300 million women worldwide (2). The goal of this review is to determine the association of pre-eclampsia and future cardiovascular risk and to explore the potential management options for these high-risk women.

The blood pressure is normally determined by Cardiac output and Peripheral vascular resistance (1). In normal pregnancy, though cardiac output is increased (7) blood pressure is maintained because of the decrease in peripheral vascular resistance. But in PIH there is increased resistance due to the increased response to vasopressors (6), altered lipid synthesis leading to decrease in the ratio of PGI2/TXA2 and antioxidants/lipid peroxides (6) and changes in the local factors like NO, endothelins.

There is an accumulated evidence stating that abnormal placentation is one of the initial events leading to this disease. Impaired interstitial trophoblasts invasion and failure of vascular invasion leading to inadequate perfusion of feto-placental unit along with Impaired decidual remodeling,(7)Impaired function of uterine natural killer cells and maternal endothelial failure to express adhesion molecules and reduced placental L-arginine concentration due to excessive arginase II expression decrease the production of nitric oxide. This promotes abnormal placental perfusion and microvascular oxidative damage.

Defective placental implantation causes placental ischemia leading to endothelial dysfunction (7). This leads to Reduced perfusion of affected organs(Predominantly kidneys, liver & brain) that leads to the Clinical manifestation of Preeclampsia. Normal pregnancy is hyperlipidemic (i.e.) 3 fold increase in TGL and fatty acids, 50% increase in LDL and HDL. One of the reasons for this increase may be due to the reduction in intestinal motility. Reduction of enterohepatic circulation with increased excretion of cholesterol in the bile also leads to alteration in lipid profile (8). Normal pregnancy is also a state of hyperestrogenemia. Estrogen results in increase in HDL level and TGL level, decrease in LDL level.

II. Materials And Methods:

This cross sectional study was carried out in Department of Obstetrics and Gynaecology, Patliputra Medical College, Dhanbad, Jharkhand. 80 women with preeclampsia and 80 normotensive pregnant women as controls were included in the study after obtaining their informed consent. The diagnosis of Preeclampsia was done as per the norms of American college of Obstetrics and Gynecologists. All the participants were inquired by a questionnaire containing their personal history, family history of PIH, Twins, Hypertension and Diabetes, Drug history and their symptoms.

The study included individuals of any gravida of age between 20 yrs and 45 yrs, after 20 wks of pregnancy with Blood Pressure ≥140/90 and proteinuria (positive dipstick) for preeclampsia and individuals with Blood Pressure ≤120/80 for control. Subjects with history of Diabetes, Hypertension, Hyperlipidemia before 20 wks and Edema, Proteinuria, oliguria, Hepatic disease and Involvement of other Organs were excluded. Clinical examination of participants were carried out to rule out Diabetes, Hypertension, Hyperlipidemia before 20 wks, edema, proteinuria, oliguria, Hepatic disease, involvement of other Organs..

Fasting blood samples were collected; the serum was separated and analyzed for the following parameters. The automated analytical system applying routine methods was used for the following measurements: glucose (GOD-POD method), TC (cholesterol esterase method), and HDL-C after precipitation using phosphotungstate method (cholesterol esterase method), and triglyceride (lipase method). LDL-C was calculated by using the Friedwald equation [LDL-C = TC – (HDL-C + triglyceride/5)], where the triglyceride level was less than 400 mg/dL. The risk ratios were calculated from the estimated values. All the results were tabulated. 24 hrs urine collected and protein estimated by analysis using Roche URS-345 dipstick urinalysis stripe to diagnose preeclampsia.

Statistical Analysis: The various results obtained were statistically analyzed using ANOVA technique. Correlations between the variables were estimated by Pearson's correlation coefficients. Significance was assumed if the *P* value was less than 0.05.

III. Results:

The values of the lipid fractions were tabulated as mean ± SD (Table 1). Total cholesterol, triglycerides, LDL-C, VLDL-C were significantly elevated and HDL-C was significantly decreased in Preeclampsia group compared to control ($p < 0.0001$).

Table 1: lipid fractions

Test	Preeclampsia	Control		p value
TC	203.54 ± 31.50	173.16	± 30.18	<0.0001
TGL	337.24 ± 77.29	196.42	± 36.96	<0.0001
HDL	36.61 ± 7.71	46.50 ± 7.80		<0.0001
LDL	103.46 ± 33.62	88.162	± 30.22	0.0006465
VLDL	66.72 ± 15.42	38.462	± 7.50	<0.0001

(TC-Total cholesterol, TGL-Triglycerides, HDL-High density cholesterol, LDL-Low density cholesterol, VLDL-Very Low density cholesterol)

The risk ratios TC/HDL, TGL/HDL, and LDL/HDL were significantly elevated and HDL/VLDL significantly decreased in Preeclampsia group compared to control ($p < 0.0001$) (Table 2). The results of various lipid sub-fractions and the atherosclerotic risk ratios were compared between the preeclampsia and control groups and shown in FIGURE 1 and FIGURE 2 respectively.

Table 2: various lipid sub-fractions and the atherosclerotic risk ratios were comparison between the preeclampsia and control groups

Test	Preeclampsia	Control	p value
TC/HDL	5.643 ± 1.72	3.70 ± 0.98	<0.0001
TGL/HDL	9.40 ± 3.25	4.20 ± 1.21	<0.0001
HDL/VLDL	0.56 ± 0.15	1.26 ± 0.37	<0.0001
LDL/HDL	2.94 ± 1.34	1.95 ± 0.85	<0.0001

Figure-1: Comparison Of Lipid Profile In Preeclampsia With That Of Control

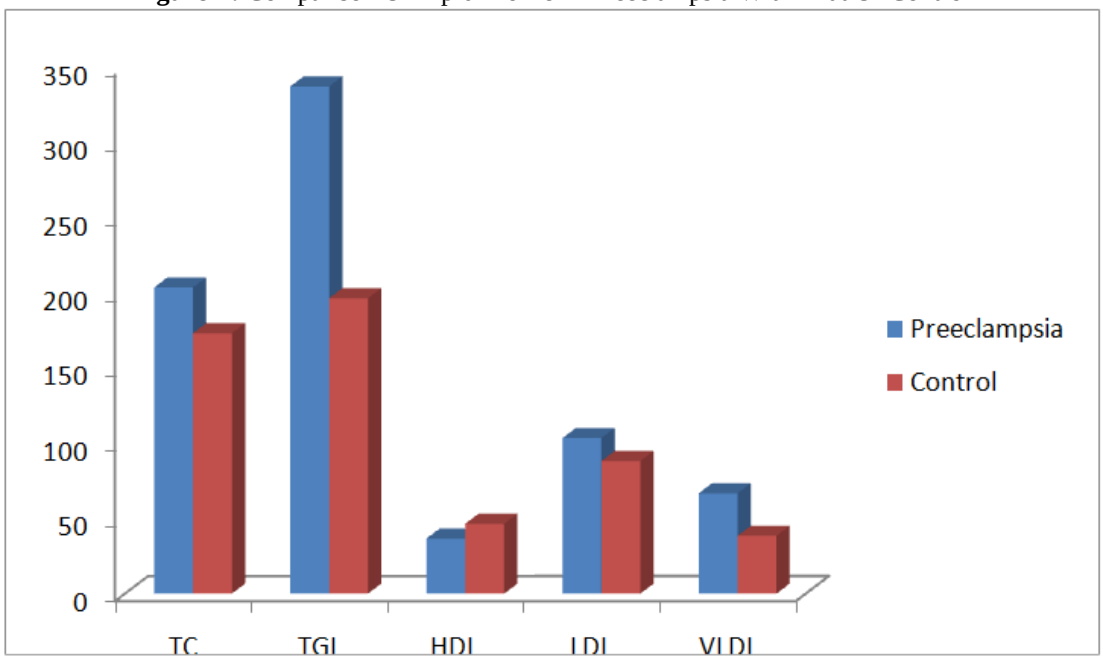
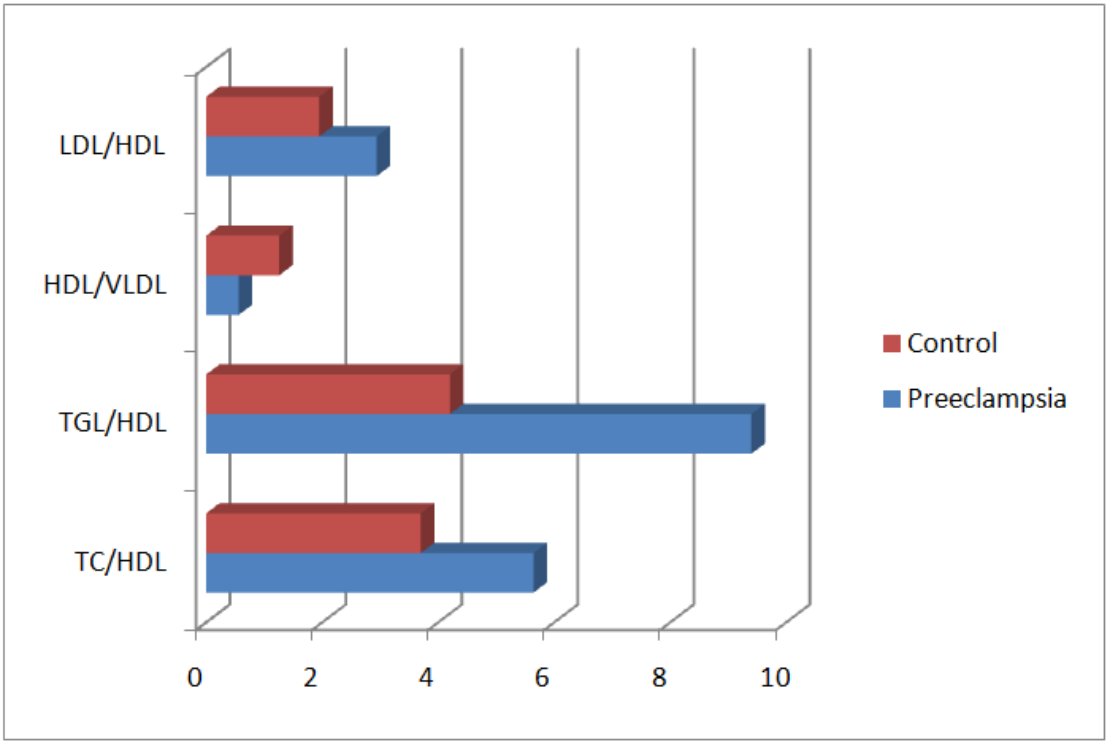


Figure 2: Comparing Calculated Risk R atios Between Preeclamptic and Control Groups



The risk ratio TGL/HDL had a significant positive correlation with triglycerides (r = 0.762) and a negative correlation with HDL-C (r = -0.674 in patients). The correlati on was highly significant (P<0.001). From fac tor analysis by rotated component matrix, the 3 factors TGL/HDL, TGL or VLDL & TC acco unted for 89.644 percent of the variance and he nce contribute more to the preeclampsia. The significant elevation of Triglycerides and the rati o TGL/HDL in preeclamptic women compared to the normotensive pregnant women can b e seen in the following charts.

IV. Discussion:

There was dramatic alteration in lipid profile which has been elevated since 20 weeks of gestation. In our study all the lipid sub fractions were significantly elevated, more in the case of triglycerides (figure 1) and

HDL was significantly decreased. The significant rise in serum triglyceride concentration in pre-eclampsia in our study was established in the studies of many workers (12, 13). The exaggerated TGL rise leads to elevated atherogenic small dense low density lipoprotein concentrations (7, 14-16). Hepatic lipase activity has been shown to be elevated in PE and could contribute to increased small dense low density lipoprotein concentration (7), via increased TGL exchange into LDL, followed by hepatic lipase induced lipolysis of the particle (17). HDL cholesterol levels are reduced, probably as a consequence of the increased TGL levels and elevated hepatic lipase activity (18).

The recently published results of the ongoing Copenhagen Male Study, which studied the effect, the TGL/HDL ratio has on the long-term development of heart disease showed that TGL/HDL ratio > 6 had a much higher risk of developing heart disease(29)(30) . This was observed in our study also, (the TGL/HDL ratio in preeclamptic population was 9.41 (high risk) against 4.30 in normal pregnancy). The significant increase of TGL/HDL ratio in preeclamptic population is shown in figure 4. The dyslipidemia of elevated triglycerides and lowered HDL in our study was similar to that of previous studies (31- 33).

V. Conclusion:

Pre-eclampsia is an under-recognized risk factor for IHD,chronic hypertension, peripheral vascular disease, and stroke. Potential mechanisms for CVD include endothelial,vascular, and metabolic dysfunction encountered during pre-eclampsia, which does not recover post-partum. Alternatively, pre-eclampsia during pregnancy could be a marker for future CVD, as both conditions share similar genetics,similar pathophysiology, such as hyperlipidemia, and several common risk factors, such as obesity, diabetes mellitus, and renal disease. Given that CVD remains the largest cause of death among women, new studies investigating this high-risk condition should be carried out to understand the disease further, and to develop novel therapeutic strategies to manage this condition in order to reduce the global burden of CVD among women.

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