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A Cross Sectional Control Study to Investigate the Potential Association Between Preeclampsia and Adverse Lipid Profile

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ABSTRACT

A multi-system disorder with an unknown aetiology, preeclampsia is distinct in that it is characterized by the development of sustained hypertension during pregnancy, reaching 140/90 mm Hg or higher, along with proteinuria after the 20th week of pregnancy or earlier in the case of twin pregnancy, H. Mole and acute polyhydramnios in a patient who was previously normotensive and nonproteinuric. To determine maternal hyperlipidemia as a risk factor for preeclampsia. The study was performed in the Department of Gynaecology and Obstetrics of Burdwan Medical College and Hospital, Burdwan in collaboration with its Biochemistry Department. Burdwan Medical College and Hospital, Burdwan is a tertiary care center of Gynaecology and Obstetrics. Around 10,000 mothers are delivered in each year. As this is a referral center, booked and unbooked cases are admitted. The majority of patients, or 72% of cases, had slightly increased blood pressure and 28% of cases had high blood pressure in preeclamptic moms. Out of 50 preeclamptic individuals, 56% had proteinuria levels between 300 and 1000 mg within 24 hrs but only 8% had severe proteinuria above 3 g. Since my study is limited to short-term trials, more research is needed to determine whether this elevated lipid profile control could prevent the development of preeclampsia. Long-term, repeated studies are needed to establish this potential association as a risk factor for preeclampsia and adverse lipid profiles.

INTRODUCTION

A multi-system disorder with an unknown aetiology, preeclampsia is distinct in that it is characterized by the development of sustained hypertension during pregnancy, reaching 140/90 mm Hg or higher, along with proteinuria after the 20th week of pregnancy or earlier in the case of twin pregnancy, H. Mole and acute polyhydramnios in a patient who was previously normotensive and nonproteinuric.

Vasospasm, increased peripheral vascular resistance and decreased organ perfusion are the hallmarks of preeclampsia[1]. The condition is polymorphic in that it can impact almost all organ systems. Significant proteinuria, new hypertension development (typically ≥140/90 mm Hg) and sign remission following delivery are indicators of preeclampsia[2].

About 2% of pregnancies result in preeclampsia, which is still largely to blame for perinatal and maternal morbidity and death[3].

Preeclampsia is a leading cause of maternal mortality in the developing countries and is correlated with a five-fold rise in infant death. Reduced uteroplacental perfusion is the main factor contributing to fetal compromise[1]. Pregnancy termination is the only intervention that successfully cures the condition. Iatrogenic prematurity is largely to blame for the rise in perinatal mortality. Preeclampsia is the reason for up to 50% of premature deliveries[4]. The risk of prenatal morbidity and death is much higher when hypertension and proteinuria are present together than when hypertension is present alone[5]. It has long been established that aberrant placentation and reduced placental perfusion are linked to preeclampsia. Preeclampsia is not always the outcome of other disorders that are marked by poor placentation, such as intrauterine growth restriction[6]. This has given rise to the increasingly accepted theory that preeclamptic maternal syndrome requires the combination of placental abnormality and maternal predisposing factors[7]. Duckitt and Harrington[8]. A comprehensive analysis of controlled trials published between 1966 and 2002 was conducted in order to quantify unadjusted relative risks for different preeclampsia risk variables. Preeclampsia is more frequently linked to the following maternal predisposing factors.

MATERIALS and METHODS

Study area: The study was performed in the Department of Gynaecology and Obstetrics of Burdwan Medical College and Hospital, Burdwan in collaboration with its Biochemistry Department. Burdwan Medical College and Hospital, Burdwan is a tertiary care center of Gynaecology and Obstetrics. Around 10,000 mothers are delivered in each year. As this is a referral center, booked and unbooked cases are admitted.

Study population: Pregnant mother [3rd trimester Gestation] attending in the antenatal OPD and Gynaecology and Obstetrics emergency as a booked or unbooked case from different districts of West Bengal and adjacent states mainly Bihar, Jharkhand and Orissa.

Study period: The study was carried out from 1st May 2020 to August 2021 in the Department of Gynaecology and Obstetrics in Burdwan Medical College and Hospital, Burdwan and Kolkata.

Sample size: The study included 50 cases of established preeclamptic mother as a case. 50 cases with normal blood pressure and without any medical and obstetrical complications were selected as Control.

Preeclampsia (Case) was diagnosed by:

- Blood pressure more than or equal to 140/90 mm of Hg after 20 weeks of pregnancy with no previous history of hypertension
- Proteinuria more than 300 mg/24 hrs urine specimen with no previous history of proteinuria
- Associated with or without oedema

Sample design: All pregnant mothers admitted in Obstetrics ward under our care fulfilling the inclusion criteria regarded as our case.

Study design: A cross sectional control study.

RESULT

In the preeclampsia group 36 (72%) women were primigravid mothers and 6 (12%) women were multiparous agonist 35 (70%) and 5 (10%) in control group respectively. In preeclamptic mothers the majority of the patients i.e., 72% cases blood pressure was mildly elevated and 28% of cases the blood pressure was severe. Amongst 50 preeclam ptic patients in 56% of cases the level of 24 hrs proteinuria were between 300 mg 1000 mg whereas-severe proteinuria more than 3 g were found only in 8% cases. Relation of lipid profile in severe PE with high blood pressure l >=1EO/110×mm of Hg) with severity of proteinuria. ≥3 g/24 hrs urine. Mean value is higher in high blood pressure in TC, TG: LDL and VLDL but in uric acid and HDL mean value is higher in severe proteinuria. Comparison of the severe PE and PE control group's parameters. The mean values of uric acid, total PE, TC, TG, LDL and VLDL are significantly higher in severe PE. The HDL mean is either almost the same or slightly lower than the control group mean value.

DISCUSSIONS

The study was undertaken to investigate the potential association between preeclampsia and adverse lipid profile, at different gestational period in

patients attending OPD of Gynaecology Obstetrics of Burdwan Medical College and Hospital, Burdwan and Kolkata during the study period of 2007 May to 2008 August. Out of 100 cases investigated equal number of control and preeclampsia mothers were studied. Apart from epidemiological, clinical and sonological assessment, the lipid profile were studied after 12 hrs of fasting of all these subjects.

Preeclampsia accounts for 15% of direct maternal fatalities in the United Kingdom and 24% of all maternal deaths in India. It is a leading cause of maternal and perinatal mortality and morbidity globally[9]. Particularly in developing countries.

Preeclampsia causes a five-fold increase in perinatal death, with iatrogenic preterm being the primary cause.

In non-industrialized nations, eclampsia has a greater burden of prenatal mortality and morbidity (up to 40%).

Understanding the aetiology and pathogenesis of any disease process is essential for prevention, as is having access to techniques for identifying those who are most likely to develop it.

Preeclampsia is most common in nullipara women, whose reported incidence ranges from 6-7%. In our investigation

Preeclampsia is known to occur often in adolescent populations and in older moms, i.e., at extremely high ages, although in our study

According to my research, 8% of instances are in the age category of 30 years and older and 24% of cases are in the adolescent group. The majority, approximately 68% of the population, is in the 20-29 age bracket as a result of the decline in early society. Most likely, this is our marriage.

In this study, middle class families accounted for the majority of patients with preeclampsia (66%) and control (70%) cases.

BMI is related to nutrition and better living condition. Patients with high BMI suffer from various medical disorders and that also aggravates obstetrical complication.

It was found that BMI more than 29 had increased risk of preeclampsia.

Numerous investigations come to the conclusion that individuals with pregestational BMIs greater than 35 were quadruple as likely to develop preeclampsia; nulliparity tripled the risk, while mother age greater than 40 doubled the risk. In our study-most of the patients in control group (68%) and Preeclamptic group (64%) are within 20 to 24.9 of BMI and more than 30 of BMI 6% in control group and 8% in preeclampsia group. And below 19.9 of BMI also reflects 8% in case and 6% in control group. Study. shows BMI >35 kg/m² increase the risk of preeclampsia but in my study most patients are within normal BMI range (20-24.9), most likely it is due to low socioeconomic status and under nutrition in prepregnant state and though it is a predisposing factor but not directly related with preeclampsia.

Despite its limitations, such as measurement errors related to sphygmomanometers and the impact of maternal position on blood pressure in pregnant women, blood pressure monitoring is still the cornerstone of early diagnosis.

Table 1 shows 72% women in preeclampsia having blood pressure more than 140/90 mm of Hg to <160/110 mm of Hg and in 28% of cases blood pressure remains ≥160/110 mm of Hg.

According to the criteria of the International Society of Hypertension, in pregnancy preeclampsia is defined as presence of hypertension (diastolic BP 90 mm of Hg or more on more than 2 occasions) occurring after 20 weeks of gestation with proteinuria (either more than ≥300 mg protein per day or urinary protein creatinine ratio ≥30) in a previously women. Normotensive and non-proteinuric. While controls do not exhibit any appreciable proteinuria throughout the gestational period, there is a consistent correlation between preeclamptic mothers and urine proteinuria of greater than 300 mg/24 hrs (Table 2).

In our study 56% of preeclampsia patients had proteinuria between 300 mg to 1 g in 24 hrs. 36% had proteinuria between > 1-3 g. Only 8% of them had proteinuria >3 g in 24 hrs urine. Though clinical classification of preeclampsia is arbitrary and is principally dependent on the level of blood pressure for management purpose but proteinuria is more significant than blood pressure to predict fetal outcome. However, proteinuria accompanied by hypertension is the most reliable indicator of fetal morbidity and mortality. Table 3 shows mean values (8.12) of uric acid in proteinuric mother increased compare to mean values (7.2) severe rise in BP of PE mothers. In severe PE with high blood pressure ≥160/110 mm of Hg TC, TG, LDL, VLDL mean are high in comparison to proteinuric group and control group.

Table 1: Parity-wise distribution of control and preeclampsia

	Control		Preeclampsia	
Parity	No.	Percentage	No.	Percentage
P_{0+0}	35	70	36	72
P_{1+0}	10	20	8	16
P_{2+0} and above	5	10	6	12
Total	50	100	50	100

Table 2: Diagnostic criteria for preeclampsia cases according to extent of elevated blood pressure and proteinuria

		No.	Percentage
BP in mm of Hg	?140/90-160/110	36	72
	?160/110	14	28
Total		50	100
Proteinuria (amount in 24 hrs urine)	300 mg-1 gm	28	56
	1 gm - 3 gm	18	36
	>3 gm	4	8
Total		50	100

Table 3: Relation of lipid profile (in mg dL^{-1}) and uric acid in severe preeclampsia with increased blood pressure ≥160/110 mm of Hg and proteinuria >3 g/24 hrs urine

Parameter	Uric acid	TC	TG	LDL	VLDL	HDL
B.P. ≥160/110 mm of Hg	7.2	309.2	356.9	162.5	71.2	54.14
Control	4.32	222.6	230.1	126.6	45.91	54.77
Proteinuria >+++ or ≥3 g/24 hrs. urine	8.12	279.3	313.8	159.8	62.5	56.25

Table 4: Comparison of parameters (mg dL^{-1}) in severe pe and pe and control with statistical significance with case and control

Control	Uric acid	TC	TG	LDL	VLDL	HDL
	4.32	222.55	230.08	126.59	45.91	54.77
PE (case)						
Severe PE	7.2	309.2	356.9	162.5	71.2	54.14
Total PE	6.32	274.62	311	152.07	61.51	53.54
Mild PE	5.44	240.04	265.1	141.64	51.82	52.94
Statistical relation (p-value)	p<0.01	p<0.01	p<0.01	p<0.01	p<0.01	p<0.01

All patients of preeclampsia and control group in this study is between 32 40 weeks of gestation and follow up with bio-chemical analysis of lipid profiles, uric acid, urea and creatinine, 24 hrs urine for total protein, Hb% and platelet count and LFT.

In our study from both group blood samples were collected aseptically after 12 hrs of overnight fasting, 10 samples in postpartum period (with 72 hrs) of both group and 40 samples in group in antepartum period. Samples for blood sugar was also drawn to exclude diabetes which was included in our maternal exclusion criteria. In order to rule out multiple pregnancies and connect the gestational age with L.M.P. for fetal maturity, routine USG for FPP was also performed.

Table 4 shows that serum uric acid level being significantly increased in cases (6.32) compared to control (4.32). In normal pregnant state, most of uric acid filtered is reabsorbed primarily in proximal convoluted tubule. Plasma concentration decreases during pregnancy as glomerular filtration rate increases with an apparent reduction in net tubular re-absorption of uric acid.

It is thought that preeclampsia's increased uric acid is a reflection of compromised renal function. Hypovolemia is linked to preeclampsia. Reduced excretion and higher absorption of uric acid are linked to hypovolemia. Additionally, in preeclampsia, insulin promotes urate absorption in the proximal tubules. A little increase in insulin resistance occurs throughout a typical pregnancy.

Uric acid is an index of purine catabolism in normal metabolism. phages, In steps of hypercatabolic often associated with faulty renal clearance, the level goes up. In our study we also found uric acid level is higher in severe PE (7.2) compare to mild PE (5.44) and control group (4.32).

The table's uric acid value shows a statistically significant difference between preeclampsia instances and greater ranges in those situations.

Recent research has concentrated on the connection between blood rheology during preeclampsia and the development of the fetus[10]. It is now well established that the rise in lipid and lipoprotein levels is substantially higher during preeclampsia leading to an assumption that these changes may have a role in producing endothelial damage characteristic of preeclampsia. About normal non-pregnant lipid profiles as follows:

In total cholesterol: <200 mg dL^{-1} desirable, - 200-239 mg dL^{-1}-borderline high, ≥240 mg dL^{-1}-high; in TG <160 mg dL^{-1}, LDL- <100 mg dL^{-1} optimal, 100- 129-near or above normal, 160-189 mg dL^{-1} high-≥190 mg dL^{-1}-very high, HDL Cholesterol <40 low-≥60 mg dL^{-1}-high.

The average TG concentration in preeclamptic patients was shown to be considerably greater in 14 investigations compared to unaffected controls; in seven more research, there was a nonsignificant trend in the same direction. Usually, the risk of preeclampsia quadrupled for every increase in TG Category. There was roughly a four-fold higher risk of preeclampsia in the highest TG category compared to the lowest in four trials that controlled for potential confounders like maternal age, parity and body mass index. The study concludes that there is a consistent positive association between elevated TG and the risk of preeclampsia[11]. During pregnancy, total cholesterol increased on average 58%, being more marked in the second trimester. Triglycerides increased on average (45% and this increase was constantly evident throughout the second and third trimesters. No single woman had triglycerides >4 mmol L^{-1} at any time during pregnancy.

While there was a modest decline in the other three trimesters at the end of the third trimester, HDL cholesterol climbed significantly between the first and second trimesters and plateaued at the end of the third. Although not statistically significant, the shift in LDL density during pregnancy was more pronounced in multiparous women than in primiparous ones.

Women with a history of preeclampsia have been found to have aberrant lipid metabolism in earlier research. Two investigations on preeclamptic women a few months after birth have found decreased high density lipoprotein (HDL) cholesterol and increased plasma levels of triglycerides and LDL cholesterol.

Munoz *et al*.[12] In their investigation, they found that during the 25th week of gestation, there is a considerable increase in the levels of plasma TG, LDL cholesterol and TC. These increases occur gradually throughout pregnancy.

Serum hypertriglyceridemia, which may increase by two to three times in the third trimester compared to levels in non-pregnant women, appears to provide the most significant harm to the lipid profile throughout a typical pregnancy, according to some earlier research. This observation is also valid in our investigation. Here the serum triglyceride concentration should very significant ($p<0.01$) increase in the third trimester of normal pregnancy (mean 230.082±24.59) compare to PE patients [311±96.0049 (mean±SD)]. The principal modulator of this hypertriglyceridemia is oestrogen as pregnancy is associated with hyperoestrogenemias. Endogenous triglycerides are synthesized in the liver and transported by VLDL as estrogen. Pregnancy-related hyperinsulinism may influence this process. In our investigation, serum triglyceride concentration also increased much greater in pregnancy-related toxaemia, which was consistent with the findings of several researchers.

Endothelial cell dysfunction during gestosis may be caused by the aforementioned interactions in addition to increased endothelial triglyceride buildup. Increased TG, which is present in pregnancy-induced hypertension, is probably deposited in vessels that are susceptible to it, such as the uterine spiral arteries and it both directly and indirectly causes endothelial dysfunction by producing tiny, dense LDL[13].

In our study, in contrast to normal pregnant women, the rise in serum TG was statistically significant $p<0.01$ in preeclamptic patients.

Table 3 shows mean values of serum uric acid (7.2), TC (309.2), TG (356.9), LDL (162.5), VLDL (71.2) are higher level in severe PE patients in comparison to total PE patients and control group which are statistically significant whereas HDL (z value 0.74) in all the group are not statistically significant ($p>0.05$).

According to my research, third trimester normal pregnancy and any of the groups PE show a substantial change in total cholesterol, which is corroborated by a small number of other studies.

A low values of LDL cholesterol level in 3rd trimester of normal pregnancy mean 126.59±18.51 in comparison to non-pregnant women mentioned in 140.6 15.24 previous studies as observed in present study may be due to hyperestrogenaemia, while LDL cholesterol level increases 152.76±47.56 significantly ($p<0.01$) in preeclampsia. A significantly higher level of beta lipoprotein was also reported by many workers in third trimester of gestational proteinuric hypertension, Endothecal dysfunction is thought to be mostly caused by hypoestrogenaemia, a preponderance of smaller and denser serum LDL particles and a substantial amount of appropriate vascular cell adhesion molecule-I (VCAM-I)[14].

In our present study, serum VLDL cholesterol level raised (45.91±4.49) significantly ($p<0.01$) in the third trimester of pregnancy in comparison to non-pregnant women study 34,. which (24.1 is+2.37) in recent perhaps due to hypertriglyceridemia leading to enhanced entry of VLDL that carries endogenous triglyceride into circulation. According to some researches, the VLDL cholesterol may increase up to 2.5 times at term compared to the pre-pregnant level. The current study and those of other researchers have demonstrated that there is an additional rise in VLDL levels in preeclampsia. This may be because of an accumulation of lipoproteins on the vascular endothelium, notably in the renal and uterine veins. While a particular toxicity-preventing activity protein guards against the VLDL-induced damage in the pathogenic process of toxaemia, more VLDL may still harm the endothelium.

Though in our study, the mean value of HDL cholesterol did not change significantly ($p>0.05$), the mean value is slightly lower in preeclamptic group l i.e., 53.54±8.9196 in comparison to 54.77±7.89). The low level of HDL in preeclampsia is however, hypooestrogenaemia resistance[15].

In Table 4 all statistically significant lipid parameters show strength of positive association as all parameters shows odds ratio >2 and as follows. In-TC OR 7.33, in TG OR 6 in LDL OR - - - 5.06; in VLDL OR - 4.69 [considering upper limit of TC $\geq$250 mg dL^{-1}; TG >210 mg dL^{-1}, LDL$\geq$160: mg dL^{-1}, VLDL >50 as suspected risk factor].

In our study it is also observed that the mean value of serum uric acid (6.32), platelet count (1.60/lacs/cnm^3), serum urea (34.62) and serum creatinine (0.89) was higher in comparison to control group as shown in Table 4.

In my study Table 2 shows 14 cases (28%) out of 50 PE cases were severe preeclampsia (BP$\geq$160/110 mm of Hg). Table 4 shows out of 14 severe PE cases 9 cases (64.28%) were symptoms of severe occipital headache and 3 cases (21.42%) shows severe epigastric pain and 3 cases (21.42%) shows platelet count <100,000/mm^3, one patient features of HELLP syndrome and 4 cases (28.57%) developed eclampsia and shows pulmonary edema subsequently in 2 eclamptic mothers (14.28%). Only 2 severe PE mothers shows ophthalmological changes due to severe preeclampsia in the form of papilledema and retinal exudes. No cases of retinal detachment or blindness found in my study. No cases of oliguria is found in my study though proteinuria shows +++>3 g- $\leq$5 g in 4 cases are found but no cases of >5 g proteinuria/24 hrs urine was found.

With prompt delivery via vaginal route or by LSCS, as well as preventative and therapeutic magnesium sulphate medication as per instance, all moms were effectively treated. The patient with HELLP syndrome did not require a platelet transfusion and was managed conservatively. Both the control group and the case group in my study did not smoke. Among the patients in our research, there was no maternal death.

CONCLUSION

Since my study is limited to short-term trials, more research is needed to determine whether this elevated lipid profile control could prevent the development of preeclampsia. Long-term, repeated studies are needed to establish this potential association as a risk factor for preeclampsia and adverse lipid profiles.

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