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Original research article

Clinical profile of pregnant ladies with preeclampsia

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Abstract
Exact primary cause for the development of preeclampsia is unknown. However abnormal trophoblastic invasion, inappropriate endothelial cell activation, exaggerated inflammatory response all this may play a key role in the development of preeclampsia. All the pregnancies with inclusion criteria were subjected to Doppler examination after recording the clinical history of the patients, clinical examination and ultrasound after taking informed consent gestational age is calculated by CRL measurement at the time of first trimester scan UA doppler recording were obtained prospectively twice, first at 11 to 14 weeks of Gestation and second at 20 to 24 weeks of gestation. In study group H/O FGR was present in 23 patients (46%), H/O HTN in 27 patients (54%) and previous LSCS in 8 patients (16%). In control group H/O previous LSCS was present in 12 cases (24%). In study group totally 13 HTN cases – 10 had severe preeclampsia, 2 had mild preeclampsia and 1 had gestational HTN. In control group totally 3 had HTN cases 1 had severe preeclampsia, 1 had mild preeclampsia and 1 had gestational HTN.
Key words: Preeclampsia, Doppler examination, LSCS

Introduction

Hypertension is the most common problem which occurs during pregnancy and it is a major cause of maternal and perinatal mortality and morbidity. Preeclampsia is a multisystem disorder and represents a major threat to fetus and mother when it emerges [1].

Hypertensive disorders complicate 5 - 10 percent of all pregnancies around the world. In India, incidence is 8-10%, incidence being more in nullipara, around 15% and in multiparas around 10% [2].

Severe growth restriction results in premature delivery, with the related risk of long term respiratory and neuro developmental problems. There is an increased perinatal mortality, particularly in very low birth weight infants. Intrauterine hypoxia which can occur in FGR may contribute to the risk for cerebral palsy. If central redistribution of blood flow in the fetus occurs, there can be ischemia of the gut leading to necrotizing enterocolitis [3].

Exact primary cause for the development of preeclampsia is unknown. However abnormal trophoblastic invasion, inappropriate endothelial cell activation, exaggerated inflammatory response all this may play a key role in the development of preeclampsia.

Primary stage is trophoblastic invasion, where in normal pregnancy the wall of the spiral arteries is invaded by the trophoblasts which converts small Musculoelastic spiral arteries into large tortuous arteries which allows a large blood flow to the intervillous space. These vessels become resistant to the action of vasomotor pressors [4].

These physiological changes fails to get completed in the patient with preeclampsia hence there will be reduced uteroplacental perfusion and vessels will be responsive for vasomotor pressors.

Second stage involves endothelial activation and exaggerated inflammatory response secondary to the release of placental debris into the maternal circulation from intervillous space. Placental debris are formed due repeated episodes of placental hypoxia or hypoperfusion. This will incite a inflammatory response and release of inflammatory cytokines, products which affects angiogenesis and lipid peroxidation. These products will affect the endothelial system of various organs leading to signs and symptoms of multi organ involvement [5, 6].

Methodology

Source of Data: This is a prospective cohort study. All the patients with inclusion criteria attending to department of obstetrics and gynaecology were included.

Inclusion Criteria

- Singleton pregnancy at 11 to 13 weeks of gestation with normal fetus
- Body mass index <30 kg/m2

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- Pregnant women with history and physical findings suggestive of PIH, IUGR, PIH plus IUGR in previous pregnancies

Exclusion Criteria

- Cardiovascular disease
- Multiple gestations
- Fetuses with congenital anomalies
- Renal disease
- Essential hypertension prior to pregnancy and other high risk pregnancies
- Smoking and alcohol and drug addiction chronic diseases and of treatment with aspirin and heparin or antihypertensives before enrollment

Methods of Collection of Data

All the pregnancies with inclusion criteria were subjected to Doppler examination after recording the clinical history of the patients, clinical examination and ultrasound after taking informed consent gestational age is calculated by CRL measurement at the time of first trimester scan UA doppler recording were obtained prospectively twice, first at 11 to 14 weeks of Gestation and second at 20 to 24 weeks of gestation.

All scan performed by experienced radiologist. In first trimester ultrasound will be used to perform UA doppler examination various indices will be calculated like pulsatality index and resistance index of both right and left UA and In second trimester scan will be obtained.

All data thus calculated will be charted in predesigned proforma and tabulated and analyzed with appropriate statistical tests. The different parameters were determined as normal or abnormal for gestational age by using previous studies as reference values. The mode of delivery will be tabulated whether vaginal or caesarean.

Perinatal outcome will also be studied in the form of perinatal death, mean, birth weight and admission to neonatal Intensive Care Unit (NICU).

Results

Table 1: Age distribution between the two groups

Age group (years)	Study group		Control group	
	N	%	N	%
<20	0	0.0	5	10.0
20-25	28	56.0	30	60.0
26-30	17	34.0	9	18.0
31-35	4	8.0	4	8.0
>35	1	2.0	2	4.0
Total	50	100.0	50	100.0
Chi square p value=0.097 (Not significant)				

As seen in above in the table, in the study group main participants belonged between 20 – 25 years (56%) followed by the 26-30 years (34%) and mean age is 25.3.

In control group main participants belonged between 20-25 years (60%) followed by 26-30 years (9%) and mean age is 24.1

Table 2: Parity distribution between the study and control group

Parity	Study group		Control group	
	N	%	N	%
Primi	0	0.0	28	56.0
Multi	50	100.0	22	44.0
Total	50	100.0	50	100.0
Chi square p value=<0.001 (Significant)				

As seen in the above table, study group comprised only of multiparas (100%) While control group had 56% primigravida and 44% multigravida

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Table 3: Comparison of previous obstetric history (POH) between the two groups

POH	Study group		Control group	
	N	%	N	%
FGR	23	46.0	0	0
HTN	27	54.0	0	0
Previous LSCS	8	16.0	12	24.0
Nil	2	4.0	38	76.0
Total	50	-	50	-

*Multiple responses possible

In study group H/O FGR was present in 23 patients (46%), H/O HTN in 27 patients (54%) and previous LSCS in 8 patients (16%).
In control group H/O previous LSCS was present in 12 cases (24%)

Table 4: Family history of HTN

Family History	Study group		Control group	
	N	%	n	%
FGR	1	2.0	0	0.0
HTN	3	6.0	1	2.0
Nil	46	92.0	49	98.0
Total	50	100.0	50	100.0
Chi square p value=0.424 (Not significant)				

In study group 1 had the family history of FGR and 3 had family history of HTN while
In control group only one had family history of HTN.

Table 5: GHTN, Mild and Severe PE in study and control groups

PE	Study group (n=48)		Control group (n=49)	
	n	%	n	%
GHTN	1	2.1	1	2.1
Mild	2	4.2	1	2.1
Severe	10	20.8	1	2.1
Total	13	100.0	3	100.0

In study group totally 13 HTN cases – 10 had severe preeclampsia, 2 had mild preeclampsia and 1 had gestational HTN.
In control group totally 3 had HTN cases 1 had severe preeclampsia, 1 had mild preeclampsia and 1 had gestational HTN.

Discussion

Preeclampsia is a pregnancy specific multisystem multifactorial disorder accounting for 14% of maternal deaths worldwide. Incidence of this disorder is around 8-10%. Uterine artery Doppler screening meets all the requirements of a worthwhile screening program in prediction of preeclampsia. Uterine artery screening at 22 to 24 weeks gestation is superior to first trimester screening in prediction of preeclampsia and other adverse pregnancy outcomes. Despite these impressive results, few hospitals have established uterine artery screening programs in the second trimester as there is no effective preventive therapy when treatment is commenced after 24 weeks and also patients may develop adverse pregnancy outcome before 24 weeks gestation [7].

A study was conducted in our hospital to know the predictive value of uterine artery Doppler at 11- 14 weeks and 21 to 24 weeks gestation using pulsatility index as the abnormal test results in both the high risk and low risk groups.

The results showed that abnormal uterine artery Doppler had a good predictive value in predicting women who developed preeclampsia, more so in the high risk group and that pulsatility index is a better Doppler index in the prediction of preeclampsia. This was in accordance to various other studies [8].

Doppler ultrasound is a non-invasive and reliable method for prediction of preeclampsia and adverse pregnancy outcome, but currently there are no effective interventions to prevent adverse outcomes based on an abnormal result. Studies are needed to find out such an intervention. Until such time, routine uterine artery Doppler screening of women is not required. Only screening in high risk women will suffice as to be more cautious during the pregnancy [9].

In case of mild preeclampsia there will be increase in maternal cardiac output but as the severity of disease increases there will be decrease in cardiac output and peripheral vascular resistance which increases both systolic and diastolic BP. When there is increase in both cardiac output and peripheral vascular resistance it may result in intravascular hemolysis.

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Increased sympathetic activity will lead increase in the maternal cardiac output, if not well tolerated it may precipitate left ventricular failure.
Decrease in oncotic pressure due to capillary leak, decreased prefusion to renals and increased hydrostatic pressure may precipitate pulmonary oedema if it's accompanied by the fluid overload.
Oedema in preeclampsia is due to decreased oncotic pressure, which is because of increased capillary permeability to plasma protein while oedema in normal pregnancy is due to increase in hydrostatic pressure.
Women with preeclampsia has hemoconcentration due to lack of hypervolemia which is usually seen in normal pregnancy. Hypovolemia leads to FGR, oligohydramnios and preterm labour.
After delivery, plasma volume increases and hematocrit decreases due to blood loss during delivery and also due to shift in the extracellular fluid in to intravascular compartment.
Women who are destined to develop preeclampsia will loss the resistance to the pressor activity.
Most common haematological abnormality in preeclampsia is mild thrombocytopenia where as overt thrombocytopenia indicates severe disease, Termination of pregnancy is indicated in severe cases which usually improves following delivery [10].
Thrombocytopenia occurs due to increased platelet activation, aggregation and consumption.
Haematocrit interpretation should be done considering haemoconcentration and hemolysis.
Even in the presence of hemolysis there may not be decrease in hematocrit due to hemoconcentration.
High level of lactate dehydrogenase may indicate hemolysis. Hematological abnormalities ranges from thrombocytopenia, DIC and HELLP which are serious complication of preeclampsia.
HELLP syndrome is the triad of hemolysis, elevated liver enzyme and low platelet count which cause serious maternal and fetal morbidity and mortality. It is characterized by microangiopathic haemolytic anemia which cause multiorgan damage especially renals and liver and ultimately result in DIC.

Conclusion

Preeclampsia is a major cause of maternal and perinatal mortality and morbidity accounts for 10% of perinatal mortality and 14% of maternal mortality and morbidity. Early recognition of women of preeclampsia will help in identifying high risk women who may benefit from early prophylaxis & enhanced surveillance and better outcome.

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