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Original Research Article

PREECLAMPSIA: EFFECT ON THE FETUS AND NEWBORN

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Abstract

Background & Methods: The aim of the study is to study Preeclampsia Effect on the Fetus and Newborn. The management of pregnancies that involve early severe PE is an issue of concern and debate between obstetricians and neonatologists because expectant management can positively affect perinatal outcome but may not be safe for the mother.

Results: In our study we found maximum cases in Gestational Hypertension (46%). The chi-square statistic is 0.8524. The *p*-value is .035888. The result is significant at $p < .05$. We found maximum cases, Respiratory Distress Syndrome (28%) followed by Thrombocytopenia (25%) followed by Sepsis (23%). The chi-square statistic is 1.9377. The *p*-value is .049617. The result is significant at $p < .05$.

Conclusion: PE remains a major obstetric problem because it is typically an unpredictable maternal disease with variable degrees of fetal involvement. Progress has been made in understanding the complex immunologic, vascular, and genetic factors involved in the pathophysiology of the disease, but to date, such progress has not been translated into clinical practice. No significant improvement has been observed in pregnancy and perinatal outcomes. PE remains an important cause of maternal and fetal morbidity and mortality. Early severe PE and IUGR deserve special attention from obstetricians and neonatologists.

Keywords: preeclampsia, fetus and newborn.
Study Design: Observational Study.

1. Introduction

Eclampsia (convulsions during pregnancy) has been a feared complication since Hippocrates' time[1]. It was discovered in the 1840s that eclampsia was accomplished by proteinuria and in the late nineteenth century the technique of indirect measurement of arterial blood pressure was developed. Hypertension and proteinuria became accepted as warning signs of eclampsia (pre-eclampsia), and screening of pregnant women was introduced[2]. Toxicosis is another term for preeclampsia, commonly used in the past, which expresses a view that toxins were part of the etiology.

Hypertension occurring during pregnancy is a major pregnancy complication, with an incidence of 2-10% of all pregnancies, and is associated with preterm delivery, fetal growth

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retardation, abruptio placentae, maternal morbidity and mortality[3]. The term pregnancy-induced hypertension is quite commonly used and encompasses hypertension, either with proteinuria (preeclampsia) or without proteinuria (gestational hypertension). Unfortunately, different definitions are used, which makes it difficult to evaluate and draw conclusions from the results of different studies[4]. The differences in the definitions and classifications of preeclampsia and gestational hypertension probably originate in how the view and knowledge have changed over time.

Preeclampsia is described as disease with immune maladaptation with lower proportion of T-helper cells, and deposition of immunoglobulins, complement, and fibrin has been noted in the walls of preeclamptic spiral arteries (the terminal branches of the uterine artery) [5]. There is a systemic activation of maternal inflammatory cell responses in preeclampsia, with activation of both granulocytes and monocytes, and increased release of proinflammatory cytokines.

High blood pressure in preeclampsia is mainly due to a reversal of the vasodilation characteristic of normal pregnancy, replaced by marked increases in peripheral vascular resistance[6]. It has been shown that the maternal pressor response to angiotensin-II is reduced in normal pregnancy, but increased in women who develop preeclampsia. Recently, agonistic autoantibodies to the angiotensin-I receptor were described, but the relevance of this finding remains to be elucidated.

2. Material and Methods

Institutional based study was conducted among 100 severe preeclamptic and eclamptic mothers for 01 Year. The study was conducted at Atal Bihari Vajpayee Government Medical College, Vidisha. The sample size was determined by counting severe preeclampsia and eclampsia cases. The management of pregnancies that involve early severe PE is an issue of concern and debate between obstetricians and neonatologists because expectant management can positively affect perinatal outcome but may not be safe for the mother.

3. Result

Table No. 1: Incidence and classification of hypertensive disorders in pregnancy.

Parameter	No.	Percentage	P Value
Preeclampsia	45	45	.035888
PE Chronic Hypertension	03	03	
Gestational Hypertension	46	46	
Chronic Hypertension	06	06	

In our study we found maximum cases in Gestational Hypertension (46%). The chi-square statistic is 0.8524. The *p*-value is .035888. The result is significant at $p < .05$.

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Table No. 2: Management of Preeclampsia

Severity	Gestational Age	No.	Percentage	P Value
Mild	Any	41	41	.049324
Severe	<24 weeks	39	39	
	24 to 34 weeks	13	13	
	>34 weeks	07	07	

In our study we found maximum cases in mild (41%) & severe (39%). The chi-square statistic is 1.6927. The *p*-value is .049324. The result is significant at $p < .05$.

Table No. 3: Perinatal mortality in preeclampsia according to gestational age.

Parameter	No.	Percentage	P Value
≤24 weeks	87	87	.014591
24 to 34 weeks	09	09	
>34 weeks	04	04	

In our study we found maximum cases in ≤24 weeks (87%). The chi-square statistic is 5.9651. The *p*-value is .014591. The result is significant at $p < .05$.

Table No. 4: Newborn Effects

Newborn Effects		No.	Percentage	P Value
IUFD		46	46	.160589
Still Birth		22	22	
Still Birth	Preterm Births	09	09	
	Low Birth Weight	05	05	
	Meconium Aspiration Syndrome	08	08	
Hypotonic		17	17	
HIE		15	15	

In our study we found maximum cases in IUFD 46%, followed by still birth 22%. The chi-square statistic is 1.9687. The *p*-value is .160589. The result is *not* significant at $p < .05$.

Table No. 5: Neonatal Complications in preterm newborn

Neonatal Complications	No.	Percentage	P Value
Respiratory Distress Syndrome	28	28	
Thrombocytopenia	25	25	.049617
Sepsis	23	23	
Necrotizing Enterocolitis	15	15	
Intraventricular Haemorrhage	09	09	

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In our study we found maximum cases in preterm, Respiratory Distress Syndrome (28%) followed by Thrombocytopenia (25%) followed by Sepsis (23%). The chi-square statistic is 1.9377. The *p*-value is .049617. The result is significant at $p < .05$.

Table No. 6: Neonatal Complications in term newborn

Neonatal Complications	No. (100)	Percentage	P Value
Respiratory Distress Syndrome	13	13	.802839
Thrombocytopenia	01	01	
Sepsis	09	09	
Necrotizing Enterocolitis	01	01	
Intraventricular Haemorrhage	00	00	

In our study we found maximum cases in term, Respiratory Distress Syndrome (13%) followed by Sepsis (09%) by Thrombocytopenia (01%). The chi-square statistic is 0.0623. The *p*-value is .802839. The result is *not* significant at $p < .05$.

4. Discussion

PE is considered a two-stage disease that begins with poor placentation and reduced uteroplacental blood supply, resulting in placental hypoxia[7]. This first stage, with silent placental events, is followed by the release of several mediators: growth factors and their soluble receptors, inflammatory cytokines, placental debris, and products of placental oxidative stress. Such mediators cause endothelial cell dysfunction and the systemic inflammatory syndrome, leading to the clinical manifestation of PE[8]. The existence of two PE subsets of early and late disease that have different biochemical and clinical features was supported in a mouse model. The model showed marked pathologic changes in placentas, lower fetal survival, and more severe intrauterine growth restriction (IUGR) in early PE but no significant changes in placental and fetal growth in late PE, thus suggesting that early PE is a placental disease and late PE is a maternal systemic disease. This finding could explain the poor outcome of fetuses and infants whose mothers develop early PE[9].

Oxidative stress results from an imbalance between the increased generation of reactive oxygen species, including free radicals and the intermediates derived from the mitochondrial metabolism, and deficiency in antioxidant defense mechanisms[10].

Placental oxidative stress is regarded as an intermediate event in the pathogenesis of PE, and although its cause remains unclear, there is strong evidence that the triggering event is abnormal placentation with underperfusion, suggesting that increased oxidative stress could be generated in the placenta through the hypoxia-reperfusion mechanism[11]. Oxidative stress promotes lipid peroxidation in placental cell membranes and can lead to endothelial cell dysfunction through increased production of thromboxane A2, release of toxic products or activation of several intracellular signaling cascades, and secretion of soluble factors, which can subsequently activate the maternal inflammatory response. Evidence that oxidative stress contributes to endothelia dysfunction, leading to PE, is supported by a number of reports showing increased concentrations of oxidative stress markers (usually

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malondialdehyde, a major breakdown product split off from lipid peroxides) in contrast with decreased antioxidant concentrations in maternal circulation and placentas of women who have PE.

This hospital-based study showed that the magnitude of maternal adverse outcome was intrapartum onset of HDP was found to be a predictor of both maternal and perinatal adverse outcomes. The magnitude of maternal complications in the study was higher than that in a study conducted in India [12]. This may be due to difference in health seeking behavior, access to roads, or transport as well as getting optimum management, early identification of high risk women, and postpartum follow-up. This study was the first study conducted in an institution, and it is considered as the strength of the study specific to maternal outcome. In our study we found complications maximum cases in preterm, Respiratory Distress Syndrome (28%) followed by Thrombocytopenia (25%) followed by Sepsis (23%). In our study we found complication with maximum cases in term, Respiratory Distress Syndrome (13%) followed by Sepsis (09%) by Thrombocytopenia (01%).

5. Conclusion

PE remains a major obstetric problem because it is typically an unpredictable maternal disease with variable degrees of fetal involvement. Progress has been made in understanding the complex immunologic, vascular, and genetic factors involved in the pathophysiology of the disease, but to date, such progress has not been translated into clinical practice. No significant improvement has been observed in pregnancy and perinatal outcomes. PE remains an important cause of maternal and fetal morbidity and mortality. Early severe PE and IUGR deserve special attention from obstetricians and neonatologists.

6. References

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