

ASSESSMENT OF NEONATAL OUTCOMES IN ECLAMPTIC MOTHERS ADMITTED TO NMCH, PATNA, BIHAR

¹Dr. Samiksha Sharma, ²Dr. Girijanand Jha, ³Dr. Binod Kr Singh, ⁴Dr. Saroj Kumar

¹Senior Resident, Department of Pediatrics, Nalanda Medical College and Hospital Patna, Bihar, India

²Senior Resident, Department of Pediatrics, Nyalanda Medical College and Hospital Patna, Bihar, India

³Professor & HOD, Department of Pediatrics, Nalanda Medical College and Hospital Patna, Bihar, India

⁴Assistant Professor, Department of Pediatrics, Nalanda Medical College and Hospital Patna, Bihar, India

Article Info: Received 15 March 2020; Accepted 28 May 2020

DOI: <https://doi.org/10.32553/ijmbs.v4i8.1374>

Corresponding author: Dr. Samiksha Sharma

Conflict of interest: No conflict of interest.

Abstract

Hypertension during pregnancy is a highly variable disorder unique to pregnancy and a leading cause of maternal and fetal/neonatal morbidity and mortality. Pregnancy-induced hypertension is the general classification for hypertension diseases during pregnancy, which include pregnancy-induced hypertension usually after 20th week of gestation (without proteinuria), pre-eclampsia (with proteinuria), and eclampsia (pre-eclampsia with convulsions). This disease is responsible for high maternal and perinatal morbidity and mortality rates, and is one of the main public health problems. Hence based on above findings the present study was planned for Assessment of Neonatal Outcomes in Eclamptic Mothers Admitted to NMCH, Patna, Bihar.

The present study was planned in Department of Pediatrics, Nalanda Medical College and Hospital, Patna, Bihar, India. The study was planned from March 2019 to October 2019. In the present study 50 females admitted with eclampsia or with pre-eclampsia but subsequently developing eclampsia were enrolled. Also the control females were also evaluated for comparative evaluation.

The data generated from the present study concludes that Prevention of prematurity, treatment of morbidities & prevention of infection among infants should be done to reduce the PMR and improve perinatal outcome. Thus High risk pregnancy should be identified prospectively and then given special care, perhaps a major impact on overall perinatal loss could be reduced.

Keywords: Eclamptic Mothers, Patna, Bihar, etc.

Introduction

Ten percent of all pregnancies are complicated by hypertension. Eclampsia and preeclampsia account for about half of these cases worldwide, and these conditions have been recognized and described for years despite the general lack of understanding of the disease. [1] In the fifth century, Hippocrates noted that headaches, convulsions, and drowsiness were ominous signs associated with pregnancy. In 1619, Varandaeus coined the term eclampsia in a treatise on gynecology. [2, 3]

Eclampsia, which is considered a complication of severe preeclampsia, is commonly defined as new onset of grand mal seizure activity and/or unexplained coma during pregnancy or postpartum in a woman with signs or symptoms of preeclampsia. [4, 5] It typically occurs during or after the 20th week of gestation or in the postpartum period. Nonetheless, eclampsia in the absence of hypertension with proteinuria has been demonstrated to occur in 38% of cases reported in the United Kingdom. [6] Similarly, hypertension was absent in 16% of cases reviewed in the United States. [4]

The clinical manifestations of maternal preeclampsia are hypertension and proteinuria with or without coexisting systemic abnormalities involving the kidneys, liver, or blood. There is also a fetal manifestation of preeclampsia involving fetal growth restriction, reduced amniotic fluid, and abnormal fetal oxygenation. [6] HELLP syndrome is a severe form of preeclampsia and involves hemolytic anemia, elevated liver function tests (LFTs), and low platelet count.

Most cases of eclampsia present in the third trimester of pregnancy, with about 80% of eclamptic seizures occurring intrapartum or within the first 48 hours following delivery. Rare cases have been reported before 20 weeks' gestation or as late as 23 days' postpartum. Although there are numerous studies exploring ultrasound and biomarker prediction of patients at risk of pre-eclampsia, other than early detection of preeclampsia, no reliable test or symptom complex predicts the development of eclampsia. In developed countries, many reported cases have been classified as unpreventable.

Eclampsia manifests as 1 seizure or more, with each seizure generally lasting 60-75 seconds. The patient's face

initially may become distorted, with protrusion of the eyes, and foaming at the mouth may occur. Respiration ceases for the duration of the seizure. Eclamptic seizures may be divided into 2 phases. Phase 1 lasts 15-20 seconds and begins with facial twitching. The body becomes rigid, leading to generalized muscular contractions. Phase 2 lasts about 60 seconds. It starts in the jaw, moves to the muscles of the face and eyelids, and then spreads throughout the body. The muscles begin alternating between contracting and relaxing in rapid sequence.

A coma or period of unconsciousness, lasting for a variable period, follows phase 2. After the coma phase, the patient may regain some consciousness, and she may become combative and very agitated. However, the patient will have no recollection of the seizure. A period of hyperventilation occurs after the tonic-clonic seizure. This compensates for the respiratory and lactic acidosis that develops during the apneic phase. Seizure-induced complications can include tongue biting, head trauma, broken bones, and aspiration.

The mechanism(s) responsible for the development eclampsia remain(s) unclear. [5] Genetic predisposition, immunology, endocrinology, nutrition, abnormal trophoblastic invasion, coagulation abnormalities, vascular endothelial damage, cardiovascular maladaptation, dietary deficiencies or excess, and infection have been proposed as etiologic factors for preeclampsia/eclampsia. [2] Imbalanced prostanoid production and increased plasma antiphospholipids have also been implicated in eclampsia. [2, 7] In murine models, placental ischemia appears to be associated with an increased susceptibility to seizures and cerebrospinal fluid (CSF) inflammation. [5]

Preeclampsia/eclampsia produces multiple systemic derangements that can involve a diversity of organ systems including hematologic, hepatic, renal, and cardiovascular systems as well as the central nervous system. The severity of these derangements often correlates with maternal medical (eg, preexisting renal or vascular pathology) or obstetric factors (eg, multifetal gestations or molar pregnancy).

Eclampsia is associated with cardiovascular derangements such as generalized vasospasm, increased peripheral vascular resistance, and increased left ventricular stroke work index. Pulmonary capillary wedge pressure (PCWP) may vary from low to elevated. Importantly, central venous pressure (CVP) may not correlate with PCWP in patients with severe preeclampsia or eclampsia. [8]

A prospective observational study by Vaught that included 63 women with pre-eclampsia with severe features reported higher systolic pressure, higher rates of abnormal diastolic function, decreased global right ventricular

longitudinal systolic strain, increased left-sided chamber remodeling, and higher rates of peripartum pulmonary edema in these women when compared with healthy pregnant women.

Many uterovascular changes occur when a woman is pregnant. It is believed that these changes are due to the interaction between fetal and maternal allografts and result in systemic and local vascular changes. It has been shown that in patients with eclampsia, the development of uteroplacental arteries is hindered.

It is believed that in eclampsia there is abnormal cerebral blood flow in the setting of extreme hypertension. The regulation of cerebral perfusion is inhibited, vessels become dilated with increased permeability, and cerebral edema occurs, resulting in ischemia and encephalopathy. With increasing blood pressure, cerebral autoregulation is impaired resulting in cerebral regions of ischemia as well as microhemorrhage, each of which may initiate a seizure focus. In extreme hypertension, normal compensatory vasoconstriction may become defective. Several autopsy findings support this model and consistently reveal swelling and fibrinoid necrosis of vessel walls. [2]

In addition, it is believed that antiangiogenic factors, such as placental protein fms-like tyrosine kinase 1 (sFlt-1) and activin A, antagonize vascular endothelial growth factor (VEGF). [9] Elevated levels of these proteins cause a reduction of VEGF and induce systemic and local endothelial cell dysfunction. [1]

Leakage of proteins from the circulation and generalized edema are sequelae of the endothelial dysfunction and thus a defining factor associated with preeclampsia and eclampsia. Evidence indicates that leptin molecules increase in the circulation of women with eclampsia, inducing oxidative stress, another factor in eclampsia, on cells. (The leptin increase also results in platelet aggregation, most likely contributing to the coagulopathy associated with eclampsia.) [2, 10]

Oxidative stress has been found to stimulate the production and secretion of the antiangiogenic factor activin A from placental and endothelial cells. [9] Studies in pregnant mouse models have proposed that there is a dysregulation in the reactive oxygen species (ROS) signaling pathway. [10, 11]

Studies also suggest that increased systemic leukocyte activity plays a role in the mediation of oxidative stress, inflammation, and endothelial cell dysfunction. Histochemistry studies indicate that there is predominantly an increase in neutrophil infiltration of vasculature in patients with eclampsia. [11]

Eclampsia always should be considered in a pregnant patient with a seizure episode. Although trauma-associated cerebral injury must be ruled-out, a pregnant patient who has been involved in an unexplained trauma (such as a single-vehicle auto accident) and has exhibited seizure activity should be evaluated for eclampsia. Eclampsia can occur during the antepartum, intrapartum, and postpartum periods. Ninety percent of eclampsia cases occur after 28 weeks' gestation. [2]

Preeclampsia can quickly develop into eclampsia. The natural progression of the disease is from symptomatic severe preeclampsia (differentiated from preeclampsia by specific vital signs, symptoms, and laboratory abnormalities) to seizures.

As many as 56% of patients with eclampsia may have transient deficits, including cortical blindness. However, studies have failed to demonstrate evidence of persisting neurologic deficits after uncomplicated eclamptic seizures during the follow-up period. Studies suggest that there is an increased risk for cerebrovascular accidents (CVAs) and coronary artery disease (CAD) in eclamptic mothers later in life.

Although some women who have had eclampsia or preeclampsia have reported subsequent cognitive difficulties even years later, a long-term follow-up study by Postma et al utilizing standardized testing was unable to find objective evidence of such problems. The reported neurocognitive difficulties have seemingly been associated with concentration and memory, as well as with vision-related tasks of daily living. In the study, 46 women who had been eclamptic and 51 who had been preeclamptic were given neurocognitive tests an average of about seven years following the index pregnancy; 48 controls, who had normotensive pregnancies, were also involved. [12-13]

The eclamptic and preeclamptic women in the study did not perform as well as the controls on motor-function tests. (They also performed more poorly on the Hospital Anxiety and Depression Scale.) However, they scored similarly to the control subjects with regard to attention, executive functioning, visual perception, and working and long-term memory. The investigators suggested that the reported cognitive difficulties in previously eclamptic or preeclamptic women occur during complex, stressful situations of daily life and may be exacerbated by anxiety and depression. [12-13]

Hypertension during pregnancy is a highly variable disorder unique to pregnancy and a leading cause of maternal and fetal/neonatal morbidity and mortality. Pregnancy-induced hypertension is the general classification for hypertension diseases during pregnancy, which include pregnancy-induced hypertension usually

after 20th week of gestation (without proteinuria), pre-eclampsia (with proteinuria), and eclampsia (pre-eclampsia with convulsions). This disease is responsible for high maternal and perinatal morbidity and mortality rates, and is one of the main public health problems. Hence based on above findings the present study was planned for Assessment of Neonatal Outcomes in Eclamptic Mothers Admitted to NMCH, Patna, Bihar.

Methodology:

The present study was planned in Department of Pediatrics, Nalanda Medical College and Hospital, Patna, Bihar, India. The study was planned from March 2019 to October 2019. In the present study 50 females admitted with eclampsia or with pre-eclampsia but subsequently developing eclampsia were enrolled. Also the control females were also evaluated for comparative evaluation.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Following was the inclusion and exclusion criteria for the present study.

Inclusion Criteria: All pregnant women are at or beyond 28 weeks of gestation, with singleton pregnancy and in the age group between 20-40 years are included.

Exclusion Criteria: Women with chronic hypertension, renal disease, cardio vascular disease, thyroid disease, liver disease, diabetes mellitus, twin pregnancy and molar pregnancy are excluded. Blood samples were collected with the consent of the patient and centrifuged and analysed immediately for serum calcium and magnesium levels.

Results & Discussion:

Hypertension is one of the most common medical complication of pregnancy. It contributes significantly to the cause of maternal and perinatal morbidity and mortality. Hypertensive disorders of pregnancy predispose women to acute or chronic uteroplacental insufficiency, resulting in ante or intrapartum asphyxia that may lead to fetal death, intrauterine growth retardation and/or preterm delivery. [14]

Essential hypertension cases were less in this study, probably because majority of the mothers did not receive antenatal care and were admitted as emergency cases, hence no blood pressure record during the antenatal period was available. Seventy three per cent of the study cases were emergency admissions, the figures being similar to other studies. [15] The perinatal mortality was also higher in them as compared to the booked cases.

Table 1: Basic Details

Group	Group A	Group B
Group of	Eclampsia Females	Control Females
No. of Cases	50	50
Age:		
20 – 30 years	41	36
31 – 40 years	9	14
Parity:		
0	43	40
1	7	10
Antenatal Care:		
Less than 2 visits	6	41
More than 3 visits	44	9
Socio Economic		
Status:		
Lower	8	10
Middle	36	40
Upper	8	0
Weight kg	41 – 56	43 – 53

Table 2: Neonatal Outcomes

Group	Group A	Group B
Group of	Eclampsia Females	Control Females
No. of Cases	50	50
Preterm	20	11
Low Birth Weight	31	15
Intrauterine Growth Retardation	6	1
Birth Asphyxia	17	6
Hypoxic Ischemic encephalopathy	5	0
Early Onset Sepsis	5	2
Early Neonatal Death	2	0
Still Birth	4	2

Maternal morbidity includes severe bleeding from abruption placentae with its resulting coagulopathy, pulmonary edema, aspiration pneumonia, acute renal failure, cerebrovascular haemorrhage, retinal detachment and PRES. Perinatal mortality and morbidity is another impact factor in eclampsia patients, as the definitive treatment is the only termination of pregnancy irrespective of gestational age. The primary target in eclampsia is achieving control of convulsions, control of blood pressure and terminating pregnancy within optimal time frame. At all health providing levels appropriate use of anticonvulsants, anti-hypertensives along with safe culmination of pregnancy should be encouraged for these patients. If need is felt referral to a well-equipped higher center should be done promptly without wasting time along with by appropriate emergency obstetric care.

The major cause of intra uterine death was placental insufficiency producing severe intra uterine growth retardation. The major cause of still birth also is placental insufficiency. From our study it is seen that women with severe IUGR had poor neonatal out come. Respiratory distress syndrome was the major neonatal complication

followed by sepsis and convulsions. All these complications were seen to decrease with increasing gestational age rather than the birth weight. Steroids when given were definitely seen to help reduce the neonatal respiratory distress syndrome. Expectant management can be undertaken by experienced team offering continuous monitoring and care. It is best that such patients be moved to a tertiary care centre with advanced neonatal care facility before the management is offered.

The frequency of hypertensive disorders of pregnancy continues to remain high and majority are due to toxemia of pregnancy. Perinatal mortality is significantly high in mothers with hypertensive disorders. The frequency of both preterm and intrauterine growth retarded babies is higher in these mothers and birth asphyxia is the commonest neonatal complication.

Severe hypertension, a feature of severe pre-eclampsia and preexisting hypertension and advanced maternal age (Barton et al., 2011) [16] are associated with adverse neonatal outcomes because these mothers are prone to abruptio placenta, cerebro-vascular and cardiovascular complications, disseminated intravascular coagulation, acute renal failure and maternal death, IUGR, preterm delivery and neonatal deaths (Haddad & Sibai, 2009). [17]

The nurse midwife plays a significant role in providing care for high risk pregnant women. She should recognize that the mainstay of treatment for pre-eclampsia remains ending the pregnancy by delivering the fetus (and the placenta). This can be a significant problem for the baby if pre-eclampsia occurs at 24-28 week of gestation. Thus, many strategies have been proposed to delay the need for delivery. The nurse midwife could assist with early recognition of the symptomless syndrome. She should also be aware of the serious nature of the condition in its severest form, adhere to agreed guidelines for admission to hospital, and have a great knowledge of investigations and the use of antihypertensive and anticonvulsant therapy. In addition, she can provide postnatal follow up and counselling for future pregnancies (Yoder et al., 2009). [18]

Eclampsia is associated with significant maternal and perinatal morbidity and mortality. The higher death is due to high percentage of the patient being unbooked; majority receive no therapeutic intervention until admission. The delay in the diagnosis, and early detection of warning symptoms is preceding eclampsia, like, edema, headache, nausea, vomiting, epigastric pain, blurring of vision and thereby delay in management, leading to various complications and resulting high mortality and morbidity. Maternal and newborn deaths due to preeclampsia/ eclampsia are preventable: by increasing

community awareness about the condition, improving antenatal care quality, and scaling up proven best practices to prevent mild preeclampsia's escalation to severe pre-eclampsia and eclampsia. By detecting and managing pre-eclampsia, judiciously, thus preventing eclampsia, can improve the survival rate of women and babies in developing countries.

Conclusion:

The data generated from the present study concludes that Prevention of prematurity, treatment of morbidities & prevention of infection among infants should be done to reduce the PMR and improve perinatal outcome. Thus High risk pregnancy should be identified prospectively and then given special care, perhaps a major impact on overall perinatal loss could be reduced.

References:

1. Craici I, Wagner S, Garovic VD. Preeclampsia and future cardiovascular risk: formal risk factor or failed stress test?. *Ther Adv Cardiovasc Dis*. 2008 Aug. 2(4):249-59.
2. Gabbe. *Obstetrics: Normal and Problem Pregnancies. Hypertension*. 5th ed. Churchill Livingstone, An Imprint of Elsevier; 2007.
3. ACOG. ACOG Practice Bulletin: Diagnosis and Management of Preeclampsia and Eclampsia: The American College of Obstetricians and Gynecologists Number 33. Jan 2002.
4. Mattar F, Sibai BM. Eclampsia. VIII. Risk factors for maternal morbidity. *Am J Obstet Gynecol*. 2000 Feb. 182(2):307-12.
5. Warrington JP. Placental ischemia increases seizure susceptibility and cerebrospinal fluid cytokines. *Physiol Rep*. 2015 Nov. 3 (11).
6. Douglas KA, Redman CW. Eclampsia in the United Kingdom. *BMJ*. 1994 Nov 26. 309(6966):1395-400.
7. Nodler J, Moolamalla SR, Ledger EM, Nuwayhid BS, Mulla ZD. Elevated antiphospholipid antibody titers and adverse pregnancy outcomes: analysis of a population-based hospital dataset. *BMC Pregnancy Childbirth*. 2009 Mar 16. 9:11.
8. Bolte AC, Dekker GA, van Eyck J, van Schijndel RS, van Geijn HP. Lack of agreement between central venous pressure and pulmonary capillary wedge pressure in preeclampsia. *Hypertens Pregnancy*. 2000. 19 (3):261-71.
9. Reddy A, Suri S, Sargent IL, Redman CW, Muttukrishna S. Maternal circulating levels of activin A, inhibin A, sFlt-1 and endoglin at parturition in normal pregnancy and pre-eclampsia. *PLoS One*. 2009. 4(2):e4453.
10. Banerjee S, Randeva H, Chambers AE. Mouse models for preeclampsia: disruption of redox-regulated signaling. *Reprod Biol Endocrinol*. 2009 Jan 15. 7:4.
11. Cadden KA, Walsh SW. Neutrophils, but not lymphocytes or monocytes, infiltrate maternal systemic vasculature in women with preeclampsia. *Hypertens Pregnancy*. 2008. 27(4):396-405.
12. Janeczko LL. Formal Testing Fails to Confirm Cognitive Problems Years After Eclampsia or Preeclampsia. *Medscape*. Feb 24 2014.
13. Postma IR, Bouma A, Ankersmit IF, et al. Neurocognitive functioning following preeclampsia and eclampsia: a long-term follow-up study. *Am J Obstet Gynecol*. 2014 Jul. 211(1):37.e1-9.
14. Naeye RL, Friedman EA. Causes of perinatal death associated with gestational hypertension and proteinuria. *Am J Obstet Gynecol* 1979; 133: 8-10.
15. Deorari AK, Arora NK, Paul VK, Singh M. Perinatal outcome in hypertensive disease of pregnancy. *Indian Pediatr* 1985; 22: 877-881.
16. Barton JR. and Sibai BM. (2008): Prediction and prevention of preeclampsia. *ObstetGynecol*; 112(2Pt1): 359-372.
17. Haddad B. and. Sibai BM(2009): Expectant Management in Pregnancies with Severe Pre-eclampsia. *Semin Perintol* :33:143-151.
18. Yoder SR., Thornburg LL., Bisognano JD. (2009) : Hypertension in pregnancy and women of childbearing age.