



## Assessment of maternal and fetal outcomes in pre-eclampsia condition in females from Bihar Region

Dr. Madhu Sinha

Senior Consultant, Department of Obstetrics and Gynaecology, Mangalam Nursing Home, Nawada, Bihar, India

### Abstract

Pre-eclampsia refers to recent onset of hypertensive (Systolic blood pressure  $\geq 140$  mm Hg or diastolic blood pressure  $\geq 90$  mm Hg) & proteinuria ( $\geq 0.3$  gm protein in 24 hours urine specimen) after 20 weeks of gestation. [11] It is considered to be severe if SBP  $> 160$  or  $\geq 110$  mm Hg DBP or proteinuria  $\geq 5$  gm in a 24 hours urine specimen or oliguria, cerebral or visual disturbances, pulmonary edema, impaired liver function or thrombocytopenia is present. There is an appreciable variation in the proportion of maternal mortality due to these disorders between the low & high income group, maximally noted to occur in the low-middle income countries. [11] Hence based on above condition the present study was planned for Assessment of Maternal and Fetal Outcomes in Pre-Eclampsia Condition in Females from Bihar Region.

The present study was planned in Department of Obstetrics and Gynaecology, Mangalam Nursing Home, Nawada, Bihar. In the present study 20 females suffered from the Pre-eclampsia condition were enrolled. The comparative study was done by enrolling normal pregnant females without any complication. All information regarding demographic profile, antenatal care, clinical finding, laboratory findings were noted. Seizures were controlled by using Magnesium Sulfate as per the Pritchard regimen. Delivery of the patient was the definitive treatment. The outcome of both, the mother and her newborn was traced till discharge or death.

The data generated from the present study concludes that maternal and newborn deaths due to preeclampsia 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. By detecting and managing pre-eclampsia, judiciously, thus preventing eclampsia, can improve the survival rate of women and babies in developing countries.

**Keywords:** maternal, fetal outcomes, pre-eclampsia condition, Bihar Region, etc

### Introduction

Pre-eclampsia is a multisystem disorder specific to pregnancy and puerperium, it manifests by onset of hypertension and proteinuria after twenty weeks of gestation. It occurs earlier with gestational trophoblastic diseases or multiple pregnancies and resolves by twelve weeks postpartum. Hypertension During pregnancy is diagnosed when the systolic pressure is 140 mmHg or more and Diastolic pressure of 90 mmHg or more measured on two occasions at least 6 hours apart within seven days. A single reading of diastolic above 110 mmHg in a pregnant woman is considered as hypertension.

Preeclampsia is one of the hypertensive (high blood pressure) disorders of pregnancy. It is a major cause of maternal and perinatal mortality (number of stillbirths and deaths of newborn in the first week of life) and morbidity. Hypertensive disorders of pregnancy occur in about 10% of all pregnant women around the world. Preeclampsia affects 3–5% of pregnancies. Along with preeclampsia, other diseases which are included in the group of hypertensive disorders of pregnancy are eclampsia, gestational hypertension and chronic hypertension.

In Asia and Africa, nearly one tenth of all maternal deaths are associated with hypertensive disorders of pregnancy. In India, the incidence of preeclampsia is reported to be 8-10% among the pregnant women. According to a study, the prevalence of hypertensive disorders of pregnancy was 7.8% with preeclampsia in 5.4% of the study population in

India. Preeclampsia is a pregnancy specific hypertensive disease with multisystem involvement. It is a disorder of widespread vascular endothelial malfunction and vasospasm that occurs after 20 weeks of gestation and can present as late as 4-6 weeks postpartum (after child birth).

According to the new guidelines given by American Congress of Obstetricians and Gynaecologists (ACOG) in 2013, the diagnosis of preeclampsia does not require the detection of high levels of protein in the urine (proteinuria) along with hypertension. Evidence shows that changes in kidney and liver can occur without signs of proteinuria, and the amount of protein in the urine does not predict how severely the disease will progress.

Preeclampsia is now to be diagnosed by persistent high blood pressure that develops during pregnancy or during the postpartum period and is associated with a lot of protein in the urine or the new development of decreased blood platelets, changes in the kidney or liver function, fluid in the lungs, or signs of brain disorder such as seizures and/or visual disturbances. HELLP (haemolysis elevated liver enzyme low platelet count) syndrome and eclampsia are the serious complications of the preeclampsia. The majority of deaths related to preeclampsia can be prevented by providing timely and effective care to pregnant women presenting with such complications<sup>[1]</sup>.

Pre-eclampsia (PE) is a disorder of pregnancy characterized by the onset of high blood pressure and often a significant amount of protein in the urine. When it arises, the condition

begins after 20 weeks of pregnancy. In severe cases of the disease there may be red blood cell breakdown, a low blood platelet count, impaired liver function, kidney dysfunction, swelling, shortness of breath due to fluid in the lungs, or visual disturbances. Pre-eclampsia increases the risk of poor outcomes for both the mother and the baby. If left untreated, it may result in seizures at which point it is known as eclampsia [2].

Risk factors for pre-eclampsia include obesity, prior hypertension, older age, and diabetes mellitus. It is also more frequent in a woman's first pregnancy and if she is carrying twins. The underlying mechanism involves abnormal formation of blood vessels in the placenta amongst other factors. Most cases are diagnosed before delivery. Rarely, pre-eclampsia may begin in the period after delivery. While historically both high blood pressure and protein in the urine were required to make the diagnosis, some definitions also include those with hypertension and any associated organ dysfunction. Blood pressure is defined as high when it is greater than 140 mmHg systolic or 90 mmHg diastolic at two separate times, more than four hours apart in a woman after twenty weeks of pregnancy. Pre-eclampsia is routinely screened for during prenatal care [3].

Recommendations for prevention include: aspirin in those at high risk, calcium supplementation in areas with low intake, and treatment of prior hypertension with medications. In those with pre-eclampsia delivery of the baby and placenta is an effective treatment. When delivery becomes recommended depends on how severe the pre-eclampsia and how far along in pregnancy a woman is. Blood pressure medication, such as labetalol and methyldopa, may be used to improve the mother's condition before delivery. Magnesium sulfate may be used to prevent eclampsia in those with severe disease. Bedrest and salt intake have not been found to be useful for either treatment or prevention [4]. Pre-eclampsia affects 2–8% of pregnancies worldwide. Hypertensive disorders of pregnancy (which include pre-eclampsia) are one of the most common causes of death due to pregnancy. They resulted in 46,900 deaths in 2015. Pre-eclampsia usually occurs after 32 weeks; however, if it occurs earlier it is associated with worse outcomes. Women who have had pre-eclampsia are at increased risk of heart disease and stroke later in life. The word "eclampsia" is from the Greek term for lightning. The first known description of the condition was by Hippocrates in the 5th century BC [5].

Swelling (especially in the hands and face) was originally considered an important sign for a diagnosis of pre-eclampsia. However, because swelling is a common occurrence in pregnancy, its utility as a distinguishing factor in pre-eclampsia is not high. Pitting edema (unusual swelling, particularly of the hands, feet, or face, notable by leaving an indentation when pressed on) can be significant, and should be reported to a health care provider.

In general, none of the signs of pre-eclampsia are specific, and even convulsions in pregnancy are more likely to have causes other than eclampsia in modern practice. Further, a symptom such as epigastric pain may be misinterpreted as heartburn. Diagnosis, therefore, depends on finding a coincidence of several pre-eclamptic features, the final proof being their regression after delivery.

Complications of pre-eclampsia can affect both the mother and the fetus. Acutely, pre-eclampsia can be complicated by eclampsia, the development of HELLP (haemolysis elevated

liver enzyme low platelet count) syndrome, hemorrhagic or ischemic stroke, liver damage and dysfunction, acute kidney injury, and acute respiratory distress syndrome (ARDS) [6].

Pre-eclampsia is also associated with increased frequency of Caesarean section, preterm delivery, and placental abruption. Furthermore, an elevation in blood pressure can occur in some individuals in the first week postpartum attributable to volume expansion and fluid mobilization. Fetal complications include fetal growth restriction and potential fetal or perinatal death. Long-term, an individual with pre-eclampsia is at increased risk for recurrence of pre-eclampsia in subsequent pregnancies.

Eclampsia is the development of new convulsions in a pre-eclamptic patient that may not be attributed to other cause. It is a sign that the underlying pre-eclamptic condition is severe and is associated with high rates of perinatal and maternal morbidity and mortality [4]. Warning symptoms for eclampsia in an individual with current pre-eclampsia may include headaches, visual disturbances, and right upper quadrant or epigastric abdominal pain, with a headache being the most consistent symptom [7]. Magnesium sulfate is used to prevent convulsions in cases of severe pre-eclampsia.

HELLP syndrome is defined as hemolysis (microangiopathic), elevated liver enzymes (liver dysfunction), and low platelets (thrombocytopenia). This condition may occur in 10–20% of patients with severe pre-eclampsia and eclampsia [7] and is associated with increased maternal and fetal morbidity and mortality. In 50% of instances, HELLP syndrome develops preterm, while 20% of cases develop in late gestation and 30% during the postpartum period.

There is also an increased risk for cardiovascular complications, including hypertension and ischemic heart disease, and kidney disease. Other risks include stroke and venous thromboembolism. It seems pre-eclampsia does not increase the risk of cancer. Lowered blood supply to the fetus in pre-eclampsia causes lowered nutrient supply, which could result in intrauterine growth restriction (IUGR) and low birth weight. The fetal origins hypothesis states that fetal undernutrition is linked with coronary heart disease later in adult life due to disproportionate growth [8].

Because pre-eclampsia leads to a mismatch between the maternal energy supply and fetal energy demands, pre-eclampsia can lead to IUGR in the developing fetus. Infants suffering from IUGR are prone to suffer from poor neuronal development and in increased risk for adult disease according to the Barker hypothesis. Associated adult diseases of the fetus due to IUGR include, but are not limited to, coronary artery disease (CAD), type 2 diabetes mellitus (T2DM), cancer, osteoporosis, and various psychiatric illnesses [9].

The risk of pre-eclampsia and development of placental dysfunction has also been shown to be recurrent cross-generationally on the maternal side and most likely on the paternal side. Fetuses born to mothers that were born small for gestational age (SGA) were 50% more likely to develop pre-eclampsia while fetuses born to both SGA parents were three-fold more likely to develop pre-eclampsia in future pregnancies [10].

Pre-eclampsia refers to recent onset of hypertensive (Systolic blood pressure  $\geq$  140 mm Hg or diastolic blood pressure  $\geq$  90mm Hg) & proteinuria ( $\geq$  0.3 gm protein in 24 hours urine specimen) after 20 weeks of gestation [11]. It is

considered to be severe if SBP > 160 or ≥ 110mm Hg DBP or proteinuria ≥ 5gm in a 24 hours urine specimen or oliguria, cerebral or visual disturbances, pulmonary edema, impaired liver function or thrombocytopenia is present. There is an appreciable variation in the proportion of maternal mortality due to these disorders between the low & high income group, maximally noted to occur in the low-middle income countries [11]. Hence based on above condition the present study was planned for Assessment of Maternal and Fetal Outcomes in Pre-Eclampsia Condition in Females from Bihar Region.

**Methodology**

The present study was planned in Department of Obstetrics and Gynaecology, Mangalam Nursing Home, Nawada, Bihar. In the present study 20 females suffered from the Pre-eclampsia condition were enrolled. The comparative study was done by enrolling normal pregnant females without any complication. All information regarding demographic profile, antenatal care, clinical finding, laboratory findings were noted. Seizures were controlled by using Magnesium Sulfate as per the Pritchard regimen. Delivery of the patient was the definitive treatment. The outcome of both, the mother and her newborn was traced till discharge or death. 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: Patient with BP ≥ 140/90 mmHg after 20 weeks of gestation admitted in our tertiary care centre.

Exclusion Criteria: 1. Multifetal gestation 2. Hypertension due to renal disorders 3. Hypertension with other medical complications like heart disease, epilepsy etc.

**Results & Discussion**

Hypertension is one of the common medical complications of pregnancy known to increase the risk of maternal and perinatal morbidity and mortality [12]. Hypertensive disorders of pregnancy include chronic hypertension, gestational hypertension, pre-eclampsia, eclampsia and chronic hypertension with superimposed pre-eclampsia. Pre-eclampsia is a multisystem disorder of unknown etiology characterized by development of hypertension to the extent of 140/90mmHg or more with significant proteinuria after the 20th week of gestation in a previously normo tensile and non proteinuric women [13]. Gestational hypertension is characterized by BP140/90mmHg or more diagnosed for the first time in pregnancy without proteinuria [13, 14].

Pre-eclampsia (PE) is one of the leading causes of maternal and fetal morbidity and mortality world-wide. Preeclampsia is a unique pregnancy-specific condition that is characterized by hypertension and proteinuria with onset after 20 weeks of gestation. The exact true etiology of preeclampsia is not clear and it is still regarded as disease of theories. There is significant evidence that abnormal trophoblastic invasion of placental blood vessels, altered endothelial cell function, oxidative stress with production of reactive oxygen species play an important role in the pathogenesis of preeclampsia. 3-5 Preeclampsia is still considered to be a major obstetric problem in pregnant women despite advancements in the field of medical sciences.

Pre-eclampsia/ eclampsia have remained a leading cause of

maternal mortality throughout the world, incidence being higher in developing countries due to illiteracy, poor antenatal care and poverty. Incidence of eclampsia and associated maternal mortality and morbidity remain high in the developing countries [15].

According to National eclampsia registry (NER) incidence of hypertensive disorders in India is observed to be 10.08% (11,266 out of 1, 11,725 deliveries) out of which 2.5% presented with eclampsia [16].

**Table 1:** Comparison of Clinical Findings

Cases of	Pre-eclampsia Females	Normal Females
No. of Cases	20	20
Age	22 – 30	21 – 29
Systolic Blood Pressure		
(mmHg)	124 – 148	112 – 130
Diastolic Blood Pressure		
(mmHg)	85 – 99	74 – 89
Haemoglobin (gm %)	11.1 – 13.9	11.7 – 14.2
Socio-economic status		
Lower class	6	5
Middle class	14	15
Maternal education		
Illiterate	6	4
primary	14	16
Pregnancy Induced		
Hypertension	3	4
Anaemia	10	8
Caesarean section		
delivery	11	9

**Table 2:** Maternal and Fetal Outcome

Cases of	Pre-eclampsia Females	Normal Females
Maternal Outcome		
Maternal deaths	2	0
Maternal near miss	3	1
Maternal severe outcomes	10	2
Fetal Outcome		
Fetal death	2	1
Early neonatal death	2	0
Perinatal death	3	2
Preterm birth	4	2
NICU admission	3	2

In a study by Tuffnell *et al* where 88.9% patients were unbooked suggested that adequate antenatal care facilitates early identification of risk factors and appropriate intervention to prevent progression to eclampsia similar to our study where 86.4% patients were not booked in antenatal period [17].

Complications of PE and eclampsia affect both mother and fetus. According to Qublan *et al.* [18] and Sarkar *et al.* [19] the multi organ dysfunction in preeclampsia which is caused by vascular endothelial damage, including maternal liver, lungs, kidney, neuronal system, coagulation pathway system will leads to excessive LDH leakage due to cellular dysfunction, which may lead to the occurrence of preeclampsia and present with elevated serum LDH.

There have been limited studies examining the role of maternal symptoms in predicting outcomes. Menzies *et al.* [20] have stated that the preeclampsia severity criteria identified by both the Canadian Hypertension Society and the National High Blood Pressure Education Program were

not predictive of maternal or perinatal morbidity. Current guidelines that make use of these severity criteria, such as those written by the Society of Obstetricians and Gynecologists of Canada [21] and the American College of Obstetricians and Gynecologists [22], for evaluating the severity of preeclampsia are not uniform and have not been proven effective.

The decision for preterm delivery of the fetus in the setting of preterm preeclampsia (at 34 weeks' gestation) is based on the estimated risk of an adverse outcome balanced with the considerable benefit to the fetus if pregnancy is prolonged. Expectant management is usually attempted in women thought to be at high risk for complications until 34 weeks' gestation, after which the neonatal outcomes are excellent and the benefit for the fetus is usually outweighed by the estimated risk to the mother [22].

Management with immediate delivery leads to high neonatal mortality and morbidity rates and prolonged hospitalization in the neonatal intensive care unit because of prematurity. Conversely, attempts to prolong pregnancy with expectant management may result in fetal death or asphyxial damage in utero and increased maternal morbidity. The risk of prolonging pregnancy is associated with worsening maternal endothelial dysfunction and continued poor perfusion of major maternal organs with the potential for severe end organ damage to the brain, liver, kidneys, placenta, fetus, and hematologic and vascular systems. Fetal risks include progressive intrauterine growth restriction and fetal demise associated with abruption or uteroplacental insufficiency [23]. The most common causes of perinatal death are fetal asphyxia, prematurity, fetal growth restriction and acidosis. Perfect quality antenatal services can diagnose preeclampsia, and its appropriate management can reduce the incidence of eclampsia and subsequent morbidity and mortality. Vigorous and prompt management of eclampsia will reduce the maternal and perinatal morbidity and mortality.

Maternal morbidity includes severe bleeding from abruption placenta 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 preeclampsia patients, as the definitive treatment is the only termination of pregnancy irrespective of gestational age. The primary target in preeclampsia 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 centre should be done promptly without wasting time along with by appropriate emergency obstetric care.

Many referring doctors have little or no experience regarding the management of preeclampsia. It is recommended that physicians and nurses referring such cases should consult physicians at the perinatal centre before transport. The women should be stabilized regarding blood pressure and control of convulsions before transport and they should be sent in an ambulance with medical personnel in attendance. Tertiary care centre should have a backup with facilities to manage critical maternal complications and provide intensive care for the immature infant.

## Conclusion

The data generated from the present study concludes that maternal and newborn deaths due to preeclampsia 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. By detecting and managing pre-eclampsia, judiciously, thus preventing eclampsia, can improve the survival rate of women and babies in developing countries.

## References

1. [https://www.nhp.gov.in/disease/gynaecology-and-obstetrics/preeclampsia#:~:text=Preeclampsia%20is%20a%20pregnancy%20specific,postpartum%20\(after%20child%20birth\).](https://www.nhp.gov.in/disease/gynaecology-and-obstetrics/preeclampsia#:~:text=Preeclampsia%20is%20a%20pregnancy%20specific,postpartum%20(after%20child%20birth).)
2. Al-Jameil N, Aziz Khan F, Fareed Khan M, Tabassum H. "A brief overview of preeclampsia". *Journal of Clinical Medicine Research*. 2014; 6(1):1-7. doi:10.4021/jocmr1682w. PMC 3881982. PMID 24400024.
3. Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R (August 2010). "Pre-eclampsia". *Lancet*. 2014; 376 (9741):631-44. doi:10.1016/S0140-6736(10)60279-6. PMID 20598363.
4. WHO recommendations for prevention and treatment of pre-eclampsia and eclampsia (PDF), 2011. ISBN 978-92-4-154833-5. Archived (PDF) from the original on 2015-05-13.
5. Mohler ER. *Advanced Therapy in Hypertension and Vascular Disease*. PMPH-USA, 2006 pp.407-408. ISBN 9781550093186. Archived from the original on 2015-10-05.
6. Arulkumaran N, Lightstone L. "Severe pre-eclampsia and hypertensive crises". *Best Practice & Research. Clinical Obstetrics & Gynaecology*. 2013; 27(6):877-84. doi:10.1016/j.bpobgyn.2013.07.003. PMID 23962474.
7. Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R. "Pre-eclampsia". *Lancet*. 2010; 376(9741):631-44. doi:10.1016/S0140-6736(10)60279-6. PMID 20598363.
8. Barker DJ. "Fetal origins of coronary heart disease". *BMJ*. 1995; 311(6998):171-4. doi:10.1136/bmj.311.6998.171. PMC 2550226. PMID 7613432.
9. Calkins K, Devaskar SU. "Fetal origins of adult disease". *Current Problems in Pediatric and Adolescent Health Care*. 2011; 41(6):158-76. doi:10.1016/j.cppeds.2011.01.001. PMC 4608552. PMID 21684471.
10. Wikström AK, Svensson T, Kieler H, Cnattingius S. "Recurrence of placental dysfunction disorders across generations". *American Journal of Obstetrics and Gynecology*. 2011; 205(5):454.e1-8. doi:10.1016/j.ajog.2011.05.009. PMID 21722870.
11. Ducarme G, Herrnberger S, Pharisien I, Carbillon L, Uzan M. [Eclampsia: retrospective study about 16 cases]. *Gynecol Obstet Fertil*. 2009; 37(1):11-7. doi:10.1016/j.gyobfe.2008.11.011. Epub 2008 Dec 31.
12. Pairu J, Bharati KN, George K. Maternal and perinatal outcome in pregnancy induced hypertension and preeclampsia. *Int J Reprod Contracept Obstet Gynecol*. 2016; 5:2166-70.

<https://doi.org/10.18203/2320-1770.ijrcog20162085>

13. Hiralal DCK. Dutta's Textbook of Obstetrics. 9th ed, 2018, 207-8.
14. Cunningham FG, Williams JW. Williams Obstetrics. 24th ed. New York: McGraw-Hill Medical, 2010, p. 728-30.
15. Ghulmiyyah L, Sibai B. Maternal mortality from preeclampsia/eclampsia. *Semin Perinatol.* 2012; 36(1):56-9. doi: 10.1053/j.semperi.2011.09.011. [PubMed]
16. Gupte S, Wagh G. Preeclampsia-eclampsia. *J Obstet Gynaecol India.* 2014; 64(1):4-13. doi: 10.1007/s13224-014-0502-y. Epub 2014 Jan 31.
17. Tuffnell DJ, Jankowicz D, Lindow SW, Lyons G, Mason GC, Russell IF, *et al.* Yorkshire Obstetric Critical Care Group. Outcomes of severe pre-eclampsia/eclampsia in Yorkshire 1999/2003. *BJOG.* 2005; 112(7):875-80.
18. Qublan HS, Ammarin V, Bataineh O, Al-Shraideh Z, Tahat Y, *et al.* Lactic dehydrogenase as a biochemical marker of adverse pregnancy outcome in severe pre-eclampsia. *Med Sci Monit.* 2005; 11(8):393-397.
19. Sarkar PD, Sogani S. Evaluation of serum lactate dehydrogenase and gamma glutamyl transferase in preeclamptic pregnancy and its comparison with normal pregnancy in third trimester. *Int J Res Med Sci.* 2013; 1(4):365-368.
20. Menzies J, Magee LA, Macnab YC, *et al.* Current CHS and NHBPEP criteria for severe preeclampsia do not uniformly predict adverse maternal or perinatal outcomes. *Hypertens Pregnancy.* 2007; 26:447-62.
21. Magee LA, Helewa ME, Moutquin JM, *et al.* Diagnosis, evaluation, and management of the hypertensive disorders of pregnancy. SOGC Clinical Practice Guidelines, No. 206, March 2008. *J Obstet Gynaecol Can.* 2008; 30(Suppl 3):S1-48.
22. American College of Obstetricians and Gynecologists Committee on Practice Bulletins—Obstetrics. Diagnosis and management of preeclampsia and eclampsia. ACOG Practice Bulletins, No. 33, January 2002. *Obstet Gynecol.* 2002; 99:159-67.
23. Swamy MK, *et al.* Maternal and perinatal outcome during expectant management of severe preeclampsia between 24 and 34 weeks of gestation. *The journal of obstetrics and gynecology of India,* July-August. 2012; 62(4):413-418.