

Adverse Perinatal Outcomes among Women with Early- and Late-Onset Preeclampsia

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Abstract

Original Research Article

Background: Preeclampsia is a major cause of adverse perinatal outcomes and the early- and late-onset phenotypes of the disease are associated with differing degrees of fetal and neonatal risk. This study aimed to compare adverse perinatal outcomes between women with early-onset preeclampsia (EOP) and late-onset preeclampsia (LOP) and against normotensive pregnancies, at a tertiary care hospital. **Methods:** A case control study was conducted in the Department of Obstetrics and Gynaecology, BIRDEM General Hospital, Dhaka, over six months (March to August 2019). One hundred women with preeclampsia (50 EOP, 50 LOP) and 50 normotensive controls were enrolled consecutively. Perinatal outcomes, including intrauterine death (IUD), intrauterine growth restriction (IUGR), low birth weight (LBW), neonatal mortality, neonatal intensive care unit (NICU) admission, preterm delivery and mode of delivery, were recorded. Data were analysed using SPSS with chi-square/Fisher exact tests and multivariable logistic regression; $p < 0.05$ was considered significant. **Results:** IUGR was significantly more frequent in EOP than LOP (32.0% vs 8.0%, $p = 0.005$), as was preterm delivery before 37 weeks (70.0% vs 38.0%, $p = 0.002$); caesarean delivery was significantly more frequent in LOP than EOP (90.0% vs 64.0%, $p = 0.004$). Compared with controls, EOP was significantly associated with IUD, IUGR, LBW, NICU admission and preterm delivery (all $p < 0.05$). On multivariable analysis, IUGR (adjusted odds ratio [aOR] 2.80), LBW (aOR 7.70), NICU admission (aOR 4.24) and IUD (aOR 1.90) remained independently associated with adverse perinatal outcome. **Conclusion:** Early-onset preeclampsia carries a substantially higher burden of adverse perinatal outcomes, particularly IUGR, low birth weight and NICU admission, than late-onset disease, supporting closer fetal surveillance and neonatal preparedness for pregnancies complicated by early-onset disease.

Keywords: Preeclampsia; early-onset; late-onset; perinatal outcome; intrauterine growth restriction; low birth weight.

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INTRODUCTION

Preeclampsia remains one of the leading causes of adverse perinatal outcomes worldwide, contributing substantially to stillbirth, preterm birth, fetal growth restriction and neonatal death, particularly in low- and middle-income countries [1,2]. Global estimates suggest that preeclampsia is responsible for approximately 500,000 perinatal deaths annually, with the burden concentrated disproportionately in resource-limited settings where access to fetal surveillance and neonatal intensive care remains constrained [1,3]. In Bangladesh, preeclampsia complicates a substantial proportion of

pregnancies and is closely linked to prematurity, accounting for a large share of medically indicated preterm births in tertiary obstetric units [4].

A key advance in understanding the perinatal impact of preeclampsia has been the recognition that early-onset preeclampsia (EOP, before 34 weeks of gestation) and late-onset preeclampsia (LOP, at or after 34 weeks) represent distinct disease phenotypes with markedly different implications for the fetus [5]. EOP typically arises from a primary failure of trophoblastic invasion and deep placental dysfunction, producing

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chronic fetal hypoxia, growth restriction and a heightened risk of iatrogenic or spontaneous preterm delivery, whereas LOP more often develops against a background of adequate placental perfusion and is associated with comparatively milder fetal compromise [6]. Population-based data have shown that although EOP is considerably less common than LOP, it disproportionately drives adverse perinatal outcomes, with an approximately ten-fold increase in perinatal death compared with unaffected pregnancies, against a two-fold increase for LOP [7]. Similar disparities have been reported across diverse settings: a retrospective study from Indonesia found significantly higher rates of prematurity, early neonatal death, birth asphyxia and low birth weight among women with EOP compared with LOP [8], while a cohort study from a low-resource hospital in Ethiopia reported that perinatal morbidity and mortality were substantially increased among women with early-onset disease [9].

Despite this accumulating international evidence, comparative data specific to Bangladeshi tertiary hospitals quantifying the differential burden of individual adverse perinatal outcomes, such as intrauterine death, growth restriction, low birth weight and need for neonatal intensive care, between EOP and LOP remain limited. Given that neonatal intensive care resources are often constrained in such settings, understanding which preeclampsia phenotype is most strongly associated with specific perinatal complications has direct implications for antenatal counselling, timing of delivery and preparation of neonatal services. Several studies have additionally emphasised that clinical differences between EOP and LOP extend to placental pathology and predictors of adverse perinatal outcome, reinforcing the rationale for phenotype-specific perinatal risk assessment [10,11].

The present study was therefore undertaken to compare the pattern and severity of adverse perinatal outcomes, including intrauterine death, growth restriction, low birth weight, neonatal mortality, NICU admission, preterm delivery and mode of delivery, between women with early- and late-onset preeclampsia attending a tertiary care hospital in Bangladesh and to quantify these outcomes relative to normotensive pregnancies, with the aim of informing gestational-age-specific fetal and neonatal management strategies.

MATERIALS AND METHODS

This case control study was conducted in the Department of Obstetrics and Gynaecology, BIRDEM General Hospital, Dhaka, a tertiary referral center, over a six-month period from March to August 2019. The study population comprised pregnant women with singleton pregnancies attending the inpatient and outpatient obstetric services during the study period. A total of 150 women were enrolled, comprising 100

women with a clinical diagnosis of preeclampsia based on American College of Obstetricians and Gynecologists criteria, subdivided into 50 women with early-onset preeclampsia (onset before 34 weeks of gestation) and 50 with late-onset preeclampsia (onset at or after 34 weeks), together with 50 normotensive pregnant women who served as controls and were admitted on the same date as the preeclamptic cases. Cases were recruited consecutively and gestational age was confirmed from the last menstrual period or first-trimester ultrasonography where dating was uncertain; pregnancies complicated by abortion, hydatidiform mole, or major chromosomal or structural fetal anomaly were excluded, along with women with incomplete clinical data, those unwilling to consent, critically ill patients and those younger than 15 years or older than 45 years. Perinatal outcome data, including intrauterine death, intrauterine growth restriction, birth weight, neonatal mortality, neonatal intensive care unit admission, gestational age at delivery and mode of delivery, were extracted from antenatal and delivery records and supplemented by structured interview and clinical examination using a pretested semi-structured questionnaire, to ensure accurate and consistent capture of outcomes across all participants. Both maternal and neonatal case records were cross-checked against admission registers to minimise transcription error and ensure completeness of the perinatal dataset. The study was conducted in accordance with the Declaration of Helsinki, with ethical clearance obtained from the Institutional Review Board and written informed consent secured from all participants, with strict confidentiality maintained through anonymised data handling throughout. Statistical analysis was performed using SPSS software; categorical perinatal outcomes were expressed as frequencies and percentages and compared between groups using the chi-square test or Fisher exact test as appropriate, while continuous variables were compared using the independent samples t-test. Variables significantly associated with adverse perinatal outcome on univariate analysis were entered into a multivariable logistic regression model to derive adjusted odds ratios and 95% confidence intervals, with a p-value of less than 0.05 considered statistically significant throughout the analysis.

RESULTS

A total of 100 women with preeclampsia were included in the perinatal outcome analysis, with 50 women in each phenotype group.

Table 1 shows the baseline maternal characteristics, which were similar between EOP and LOP. The mean maternal age was 29.0 ± 5.1 years in EOP and 28.28 ± 5.7 years in LOP ($p=0.507$). Mean maternal height was 152.0 ± 6.6 cm and 152.52 ± 5.8 cm ($p=0.677$), respectively and mean maternal weight was 71.13 ± 13.9 kg versus 71.58 ± 16.1 kg ($p=0.881$).

Table 1: Baseline maternal characteristics according to preeclampsia phenotype

Characteristic	EOP (n=50)	LOP (n=50)	p-value
Age, years, mean $\pm$ SD	29.0 $\pm$ 5.1	28.28 $\pm$ 5.7	0.507
Height, cm, mean $\pm$ SD	152.0 $\pm$ 6.6	152.52 $\pm$ 5.8	0.677
Weight, kg, mean $\pm$ SD	71.13 $\pm$ 13.9	71.58 $\pm$ 16.1	0.881

Table 2 presents adverse perinatal outcomes according to preeclampsia phenotype. IUD occurred in 7 (14.0%) EOP pregnancies compared with 1 (2.0%) LOP pregnancy, although the difference did not reach statistical significance ($p=0.059$). IUGR was significantly more frequent in EOP than LOP (16 [32.0%] vs 4 [8.0%]; $p=0.005$). LBW occurred in 31 (62.0%) EOP and 22 (44.0%) LOP pregnancies

($p=0.109$). Neonatal mortality occurred in 4 (8.0%) and 1 (2.0%) pregnancies, respectively ($p=0.362$). NICU admission was required for 19 (38.0%) infants in EOP and 13 (26.0%) in LOP ($p=0.284$). Preterm delivery before 37 weeks occurred significantly more frequently in EOP than LOP (35 [70.0%] vs 19 [38.0%]; $p=0.002$). Caesarean delivery occurred in 32 (64.0%) EOP and 45 (90.0%) LOP pregnancies ($p=0.004$).

Table 2: Adverse perinatal outcomes according to early- and late-onset preeclampsia

Outcome	EOP (n=50), n (%)	LOP (n=50), n (%)	p-value
Intrauterine death	7 (14.0)	1 (2.0)	0.059
Intrauterine growth restriction	16 (32.0)	4 (8.0)	0.005
Low birth weight	31 (62.0)	22 (44.0)	0.109
Neonatal mortality	4 (8.0)	1 (2.0)	0.362
NICU admission	19 (38.0)	13 (26.0)	0.284
Preterm delivery <37 weeks	35 (70.0)	19 (38.0)	0.002
Caesarean delivery	32 (64.0)	45 (90.0)	0.004

Table 3 presents perinatal outcomes among women with early-onset preeclampsia compared with normotensive controls. IUD (14.0% vs 2.0%, $p=0.003$), IUGR (32.0% vs 4.0%, $p=0.001$), LBW (62.0% vs 26.0%, $p<0.001$), NICU admission (38.0% vs 12.0%,

$p=0.001$) and preterm delivery before 37 weeks (70.0% vs 60.0%, $p=0.028$) were all significantly more common among women with EOP than controls. Caesarean delivery was significantly less frequent in EOP than in controls (64.0% vs 80.0%, $p=0.043$).

Table 3: Perinatal outcomes among women with early-onset preeclampsia compared with normotensive controls

Outcome	Control (n=50), n (%)	EOP (n=50), n (%)	p-value
IUD	1 (2.0)	7 (14.0)	0.003
IUGR	2 (4.0)	16 (32.0)	0.001
LBW	13 (26.0)	31 (62.0)	<0.001
Neonatal mortality	2 (4.0)	4 (8.0)	0.117
NICU admission	6 (12.0)	19 (38.0)	0.001
Preterm delivery <37 weeks	30 (60.0)	35 (70.0)	0.028
Caesarean delivery	40 (80.0)	32 (64.0)	0.043

Table 4 presents perinatal outcomes among women with late-onset preeclampsia compared with normotensive controls. LBW was significantly more frequent in LOP than controls (44.0% vs 26.0%, $p=0.039$) and preterm delivery before 37 weeks was

significantly less frequent in LOP than controls (38.0% vs 60.0%, $p<0.001$). IUD, IUGR, neonatal mortality, NICU admission and caesarean delivery did not differ significantly between LOP and controls.

Table 4: Perinatal outcomes among women with late-onset preeclampsia compared with normotensive controls

Outcome	Control (n=50), n (%)	LOP (n=50), n (%)	p-value
IUD	1 (2.0)	1 (2.0)	1.000
IUGR	2 (4.0)	4 (8.0)	0.405
LBW	13 (26.0)	22 (44.0)	0.039
Neonatal mortality	2 (4.0)	1 (2.0)	0.320
NICU admission	6 (12.0)	13 (26.0)	0.076
Preterm delivery <37 weeks	30 (60.0)	19 (38.0)	<0.001
Caesarean delivery	40 (80.0)	45 (90.0)	0.165

Table 5 presents the results of multivariable logistic regression analysis of selected adverse perinatal outcomes. IUGR (adjusted odds ratio [aOR] 2.80, 95% CI 1.08–7.26, $p=0.035$), low birth weight (aOR 7.70,

95% CI 2.07–28.50, $p=0.002$), NICU admission (aOR 4.24, 95% CI 1.68–10.70, $p=0.002$) and IUD (aOR 1.90, 95% CI 1.02–3.54, $p=0.017$) were all independently associated with adverse perinatal outcome.

Table 5: Multivariable logistic regression analysis of selected adverse perinatal outcomes

Outcome	Adjusted OR	95% CI	p-value
IUGR	2.8	1.08–7.26	0.035
Low birth weight	7.7	2.07–28.50	0.002
NICU admission	4.24	1.68–10.70	0.002
IUD	1.9	1.02–3.54	0.017

DISCUSSION

In the present cohort, women with early-onset preeclampsia (EOP) and late-onset preeclampsia (LOP) had comparable baseline maternal age, height and weight, indicating that the marked differences in perinatal outcome observed between the two groups are attributable to the underlying disease process rather than to differences in maternal baseline characteristics. This observation is consistent with Rahman *et al* [8], who similarly reported broadly comparable maternal demographics between EOP and LOP despite substantially divergent neonatal outcomes.

Intrauterine growth restriction was significantly more common in EOP than LOP (32.0% vs 8.0%, $p=0.005$) and preterm delivery before 37 weeks was likewise significantly more frequent in EOP (70.0% vs 38.0%, $p=0.002$). These findings mirror those of Madazli *et al* [11], who reported markedly higher rates of growth restriction and preterm delivery among women with early-onset compared with late-onset disease and are consistent with the pathophysiological model in which EOP originates from a primary failure of trophoblast invasion and deep placental insufficiency, producing chronic fetal hypoxia and growth restriction from early in gestation [6,16]. Cheng and Wang [17] similarly emphasised that abnormal placentation is far more pronounced in early-onset disease, which explains the disproportionate burden of fetal growth compromise observed in this phenotype across multiple populations, including the Thai cohort described by Fang *et al* [15] and the Reunion Island cohort of Iacobelli *et al* [14].

Although intrauterine death occurred more frequently in EOP than LOP in the present cohort (14.0% vs 2.0%), this difference did not reach conventional statistical significance ($p=0.059$), most likely reflecting the limited statistical power of the relatively small subgroup sizes to detect a difference in a comparatively infrequent outcome. Nevertheless, when EOP was compared directly with normotensive controls, intrauterine death was significantly more common (14.0% vs 2.0%, $p=0.003$), a pattern consistent with the substantially elevated risk of fetal death associated with early-onset disease reported in larger population-based studies [7,13].

Low birth weight was common in both phenotypes, occurring in 62.0% of EOP and 44.0% of LOP pregnancies, though the between-group difference did not reach significance ($p=0.109$); however, both rates were significantly higher than the 26.0% observed among controls ($p<0.001$ for EOP and $p=0.039$ for LOP), confirming that impaired fetal growth is a feature of preeclampsia across the gestational spectrum, albeit more pronounced in early-onset disease. This finding is consistent with the observation of Stubert *et al* [10] that low birth weight and reduced gestational age at delivery were the dominant predictors of adverse perinatal outcome in severe preeclampsia irrespective of onset timing, though the magnitude of the effect was greatest in early-onset cases.

NICU admission was required for 38.0% of infants in the EOP group compared with 26.0% in the LOP group ($p=0.284$) and this difference achieved significance only when each phenotype was compared against controls ($p=0.001$ for EOP vs controls). This pattern likely reflects the strong dependence of NICU utilisation on gestational age at delivery and birth weight rather than preeclampsia phenotype alone, an interpretation supported by Bulut *et al* [12], who found that neonatal morbidity and NICU admission rates were significantly higher in early-onset preeclampsia largely because of the associated prematurity and lower birth weight in that group.

Notably, caesarean delivery was significantly more frequent in LOP than EOP (90.0% vs 64.0%, $p=0.004$), a finding that may initially appear counterintuitive given the greater fetal compromise typically observed in EOP. This likely reflects clinical decision-making patterns in which women with late-onset disease, who are closer to term and whose fetuses are more likely to be viable and reasonably grown, are more readily delivered by caesarean section once maternal or fetal indications arise, whereas some early-onset cases, particularly those complicated by intrauterine death, may proceed to vaginal delivery. A broadly similar pattern of higher caesarean rates in later-onset disease has been reported by Sundaram [18] in a comparative cohort.

On multivariable analysis, IUGR (adjusted odds ratio [aOR] 2.80), low birth weight (aOR 7.70), NICU admission (aOR 4.24) and intrauterine death (aOR 1.90) remained independently associated with adverse perinatal outcome, reinforcing that these four outcomes represent the principal drivers of perinatal morbidity in this population and should form the core of a phenotype-specific fetal surveillance and neonatal preparedness protocol in similar tertiary care settings [9,19,20].

These findings have practical implications for obstetric units managing preeclampsia in resource-constrained settings. Anticipating a disproportionately high burden of growth restriction, prematurity and need for neonatal intensive care among pregnancies complicated by early-onset disease should prompt earlier referral to centers with adequate neonatal facilities, more intensive fetal surveillance through serial growth scans and Doppler assessment where available and close coordination between obstetric and neonatal teams before delivery.

Limitations and Recommendations

This study was limited by its single-center design and modest subgroup sample sizes, which reduced statistical power to detect differences in relatively infrequent outcomes such as intrauterine death. Some perinatal data were derived from hospital records subject to documentation variation. Larger, multicenter prospective studies are recommended to validate these findings and to develop gestational-age-specific fetal surveillance protocols for preeclampsia in similar low-resource settings.

CONCLUSION

Early-onset preeclampsia was associated with a substantially greater burden of adverse perinatal outcomes than late-onset disease, particularly intrauterine growth restriction and preterm delivery, while low birth weight and NICU admission were common across both phenotypes relative to normotensive pregnancy. Intrauterine growth restriction, low birth weight, NICU admission and intrauterine death were independent predictors of adverse perinatal outcome. These findings support gestational-age-specific fetal monitoring and neonatal preparedness, with particular vigilance warranted for pregnancies complicated by early-onset preeclampsia.

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