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Original Article

Predictors of Adverse Maternal and Perinatal Outcomes in Women with Severe Preeclampsia: A Prospective Cohort Study

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ABSTRACT

Background: Severe preeclampsia is associated with substantial maternal and perinatal morbidity, highlighting the need for early identification of women at increased risk of adverse outcomes. This study aimed to identify predictors of adverse maternal and perinatal outcomes among women with severe preeclampsia. **Methods & Materials:** This prospective cohort study was conducted at the Department of Obstetrics & Gynaecology, Sher-e-Bangla Medical College Hospital, Barishal, Bangladesh, from January 2026 to June 2026, involving 100 women with severe preeclampsia. Participants were prospectively followed for maternal and perinatal outcomes. Data were analyzed using chi-square or Fisher's exact tests and multivariable logistic regression, with $p<0.05$ considered statistically significant. **Results:** Among 100 women, mean age was 27.8 ± 5.1 years and gestational age 34.7 ± 2.8 weeks; 35 (35.0%) presented at <34 weeks. Headache, severe hypertension, and proteinuria ≥3+ occurred in 61 (61.0%), 72 (72.0%), and 54 (54.0%), respectively. Adverse maternal and perinatal outcomes occurred in 34 (34.0%) and 39 (39.0%), respectively. Gestational age <34 weeks independently predicted maternal (aOR 3.82, p=0.003) and perinatal outcomes (aOR 5.24, p<0.001). **Conclusion:** Severe preeclampsia carries substantial maternal and perinatal risk, particularly with early gestation and evidence of hematologic, hepatic, renal, and hypertensive complications.

Keywords: *Severe Preeclampsia, Maternal Outcomes, Perinatal Outcomes*

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INTRODUCTION

Pre-eclampsia (PE) is a pregnancy-specific hypertensive disorder characterized by the development of new-onset hypertension after 20 weeks of gestation, accompanied by maternal organ dysfunction, which may occur with or without proteinuria [1]. As a multisystem disorder unique to pregnancy, PE affects approximately 2%–5% of pregnancies and continues to be a major contributor to maternal morbidity and mortality worldwide despite advances in obstetrical care over recent decades [2]. It is also an important cause of perinatal morbidity and mortality, emphasizing the need for timely diagnosis and appropriate management to minimize serious complications [3]. Hypertensive disorders of pregnancy (HDPs), which encompass gestational hypertension, preeclampsia (PE)/eclampsia, chronic hypertension with superimposed PE, and chronic hypertension, complicate up to 10% of pregnancies globally and remain among the most common causes of adverse maternal and perinatal outcomes.

Severe preeclampsia represents the more serious spectrum of this hypertensive disorder and is characterized by marked elevation of blood pressure and significant proteinuria [3] The clinical manifestations of PE range from mild or asymptomatic hypertension to potentially life-threatening neurological, renal, and cardiopulmonary complications in severe disease. Severe PE may result in major maternal complications, including eclampsia, hemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome, stroke, renal failure, and pulmonary edema [4]. Similarly, PE may have substantial consequences for the fetus, including intrauterine growth restriction, preterm birth, and stillbirth [5]. The clinical course of PE may progress to severe maternal complications such as eclampsia, haemolysis, elevated liver enzymes and low platelet (HELLP) syndrome, stroke, renal failure, and, in severe cases, maternal death [6]. Furthermore, early-onset severe PE has been associated with poorer outcomes than late-onset disease, underscoring the importance of early recognition and intervention [7].

Given the substantial maternal and perinatal consequences of severe PE, early identification of women at increased risk of adverse pregnancy outcomes is clinically important. Identification of women who are likely to develop severe complications at an early stage may facilitate appropriate triage, allocation of healthcare resources, and targeted interventions aimed at preventing adverse outcomes [8]. Evidence-based risk assessment may therefore assist clinicians in recognizing women at high risk of adverse maternal outcomes and determining the appropriate level of care. Accurate risk prediction can further support individualized obstetric surveillance and treatment, as well as decisions regarding the timing and mode of delivery. Early prediction of adverse outcomes in PE is consequently important for improving maternal and neonatal prognosis. Identification of prognostic markers among women with established PE may also facilitate closer monitoring

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and timely clinical intervention, thereby reducing the burden of severe maternal morbidity and mortality [9].

A number of clinical and laboratory characteristics have been investigated as potential predictors of adverse outcomes in women with PE. Previous studies have demonstrated associations between risk factors for severe PE and adverse pregnancy outcomes. Li et al.[10] reported that advanced maternal age, multiple pregnancies, use of assisted reproductive technology, rural residence, a history of hypertensive disorders of pregnancy, male fetal sex, multigravida status, polycystic ovary syndrome, HELLP syndrome, intrahepatic cholestasis of pregnancy, cardiovascular disease, gestational diabetes mellitus (GDM), systemic lupus erythematosus, thyroid disease, and liver disease were significantly associated with adverse outcomes among pregnant women with PE. Wu et al.[11], in a study of 136 women with early-onset severe PE, identified elevated red blood cell counts, reduced platelet counts, and earlier gestational age at delivery as important risk factors for poor pregnancy outcomes. Prediction models such as fullPIERS have incorporated readily available clinical and laboratory parameters, including gestational age at delivery, dyspnea or chest pain, oxygen saturation, platelet count, serum creatinine, and serum aspartate transaminase, to estimate the risk of adverse maternal outcomes. While several studies have examined potential predictors of adverse outcomes in PE, their findings have not been consistent, and the relative contribution of individual predictors remains incompletely defined [12].

Although several studies have investigated predictors of adverse outcomes in preeclampsia, findings have been inconsistent, and the relative importance of individual clinical and laboratory predictors remains unclear.15 Moreover, existing prediction models differ in their populations, methodologies, and outcome measures, limiting their generalizability and clinical application. Therefore, further prospective evaluation of readily available clinical and laboratory factors is needed to identify women with severe preeclampsia who are at increased risk of adverse maternal and perinatal outcomes. The present study aimed to identify predictors of adverse maternal and perinatal outcomes among women with severe preeclampsia.

Objective

- To identify predictors of adverse maternal and perinatal outcomes among women with severe preeclampsia.

METHODS & MATERIALS

This prospective cohort study was conducted at the Department of Obstetrics & Gynaecology, Sher-e-Bangla Medical College Hospital, Barishal, Bangladesh, from January 2026 to June 2026. A total of 100 pregnant women diagnosed with severe preeclampsia were included, selected according to predefined inclusion and exclusion criteria. Participants were prospectively followed from admission through delivery and the early postpartum or neonatal period to assess maternal and perinatal outcomes and identify potential predictors of adverse outcomes.

Inclusion Criteria

i. Pregnant women diagnosed with severe preeclampsia according to predefined diagnostic criteria.
ii. Women admitted to the Department of Obstetrics & Gynaecology during the study period.
iii. Women with singleton or multiple pregnancies.
iv. Women who provided written informed consent to participate in the study.
v. Women available for follow-up until delivery and the immediate postpartum/neonatal period.

Exclusion Criteria

i. Women with chronic hypertension predating pregnancy or diagnosed before 20 weeks of gestation.
ii. Women with pre-existing chronic renal or hepatic disease.
iii. Women with pre-existing hematological disorders that could affect platelet count or coagulation parameters.
iv. Women with major medical disorders likely to independently influence maternal or perinatal outcomes.
v. Women who declined participation or had incomplete essential clinical or laboratory data.

Data Collection

After enrollment, relevant sociodemographic, obstetric, clinical, and laboratory data were collected using a structured data collection form. Written informed consent was obtained from each participant before enrollment. Sociodemographic variables included maternal age and residence. Obstetric variables included gravidity, gestational age at admission, previous history of preeclampsia, previous history of hypertension, and pregnancy type. Clinical assessment included headache, visual disturbance, generalized edema, epigastric or right upper abdominal pain, and blood pressure measurement. Laboratory investigations included urinary protein assessment, platelet count, liver enzymes, and serum creatinine. The presence of HELLP syndrome and eclampsia at admission was also recorded.

Operational Definitions

Severe preeclampsia was defined as preeclampsia associated with severe-range blood pressure and/or evidence of maternal organ dysfunction. Severe hypertension was defined as systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥110 mmHg. Significant proteinuria was defined as urinary protein ≥3+ on dipstick assessment. Thrombocytopenia was defined as a platelet count <100,000/mm³, while renal impairment was defined as serum creatinine ≥1.2 mg/dL. Elevated liver enzymes were defined according to the laboratory reference range used by the study hospital. Gestational age <34 weeks was considered early-onset presentation for the purpose of predictor analysis.

Study Variables and Predictors

The potential predictors evaluated in the study included maternal age ≥30 years, primigravida status, gestational age <34 weeks, previous history of preeclampsia, blood pressure ≥160/110 mmHg, proteinuria ≥3+, platelet count <100,000/mm³, elevated liver enzymes, and serum creatinine ≥1.2 mg/dL. These variables were selected based on their clinical relevance and potential association with maternal and perinatal complications in severe preeclampsia.

Outcome Measures

The primary outcomes were adverse maternal and adverse perinatal outcomes. An adverse maternal outcome was defined as the occurrence of one or more of the following: eclampsia, HELLP syndrome, acute kidney injury, pulmonary edema, placental abruption, disseminated intravascular coagulation, intensive care unit admission, or maternal death. An adverse perinatal outcome was defined as the occurrence of one or more of the following: preterm birth <37 weeks, low birth weight <2500 g, 5-minute Apgar score <7, neonatal intensive care unit admission, stillbirth, or early neonatal death. Individual maternal and perinatal complications were also recorded separately.

Follow-up and Outcome Assessment

Participants were followed prospectively from hospital admission through delivery and the immediate postpartum period. Maternal complications, intensive care requirements, and maternal survival were documented. Newborns were assessed at birth for gestational age, birth weight, and 5-minute Apgar score and were followed during the early neonatal period for NICU admission, stillbirth, and early neonatal death.

Statistical Analysis

Data were entered, coded, and analyzed using IBM SPSS Statistics. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies

and percentages. Associations between categorical predictors and adverse maternal or perinatal outcomes were assessed using the chi-square test or Fisher's exact test, as appropriate. Separate multivariable logistic regression models were constructed for adverse maternal and adverse perinatal outcomes. Clinically relevant candidate variables identified a priori and/or showing evidence of association in univariable analysis were considered for inclusion in the multivariable models. Adjusted odds ratios (aORs) with 95% confidence intervals (CIs) were reported. A two-sided *p*-value <0.05 was considered statistically significant.

RESULTS

The majority of women were aged 20–29 years (55 patients, 55.0%), followed by those aged 30–39 years (32 patients, 32.0%), while 8 patients (8.0%) were younger than 20 years and 5 patients (5.0%) were aged ≥40 years, with a mean age of 27.8 ± 5.1 years. Most participants were from urban areas (57 patients, 57.0%), while 43 patients (43.0%) were from rural areas. Multigravida women predominated (58 patients, 58.0%) over primigravida women (42 patients, 42.0%). The mean gestational age at admission was 34.7 ± 2.8 weeks, with 35 patients (35.0%) presenting before 34 weeks of gestation. A previous history of preeclampsia was reported in 18 patients (18.0%), while 12 patients (12.0%) had a previous history of hypertension. Multiple pregnancy was present in 8 patients (8.0%).

Table I
Sociodemographic and Obstetric Characteristics of Women with Severe Preeclampsia (*n* = 100).

Category	Characteristic	No. (%) / Mean ± SD
Age group (years)	<20	8 (8.0)
	20–29	55 (55.0)
	30–39	32 (32.0)
	≥40	5 (5.0)
	Age (years)	27.8 ± 5.1
Residence	Urban	57 (57.0)
	Rural	43 (43.0)
Gravidity	Primigravida	42 (42.0)
	Multigravida	58 (58.0)
Gestational age	Gestational age at admission (weeks)	34.7 ± 2.8
	Gestational age <34 weeks	35 (35.0)
Previous history	Previous history of preeclampsia	18 (18.0)
	Previous history of hypertension	12 (12.0)
Pregnancy type	Multiple pregnancy	8 (8.0)

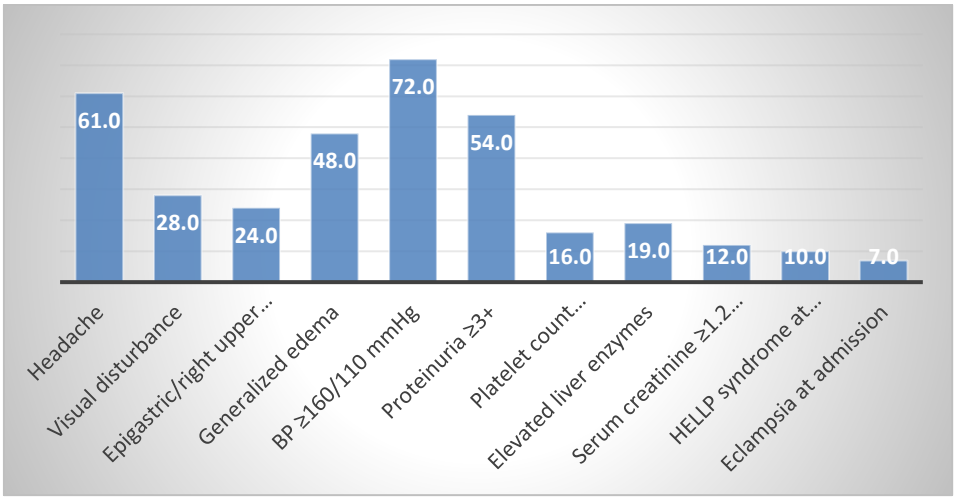


Figure 1: Clinical and Laboratory Characteristics of Women with Severe Preeclampsia at Admission (n = 100).

Figure 1 shows that headache was the most common clinical symptom, reported by 61 patients (61.0%), followed by generalized edema in 48 patients (48.0%), visual disturbance in 28 patients (28.0%), and epigastric/right upper abdominal pain in 24 An adverse maternal outcome occurred in 34 patients (34.0%). Among individual maternal complications, HELLP syndrome was the most frequent (10 patients, 10.0%), followed by ICU admission (9 patients, 9.0%), acute kidney injury (8 patients, 8.0%), eclampsia (7 patients, 7.0%), placental abruption (6 patients, 6.0%), patients (24.0%). Severe hypertension with blood pressure ≥160/110 mmHg was observed in 72 patients (72.0%), while proteinuria ≥3+ was present in 54 patients (54.0%). Thrombocytopenia with platelet count <100,000/mm³ was identified in 16 pulmonary edema (5 patients, 5.0%), disseminated intravascular coagulation (3 patients, 3.0%), and maternal death (2 patients, 2.0%). An adverse perinatal outcome occurred in 39 patients (39.0%). Preterm birth before 37 weeks was the most common perinatal outcome (35 patients, 35.0%), followed by low birth patients (16.0%), elevated liver enzymes in 19 patients (19.0%), and serum creatinine ≥1.2 mg/dL in 12 patients (12.0%). HELLP syndrome and eclampsia at admission were present in 10 patients (10.0%) and 7 patients (7.0%), respectively.
weight <2500 g (38 patients, 38.0%), NICU admission (31 patients, 31.0%), 5-minute Apgar score <7 (18 patients, 18.0%), stillbirth (8 patients, 8.0%), and early neonatal death (4 patients, 4.0%).

Table II
Maternal and Perinatal Outcomes Among Women with Severe Preeclampsia (n = 100).

	Outcome	Frequency (n)	Percentage (%)
Maternal outcomes	Any adverse maternal outcome	34	34.0
	Eclampsia	7	7.0
	HELLP syndrome	10	10.0
	Acute kidney injury	8	8.0
	Pulmonary edema	5	5.0
	Placental abruption	6	6.0
	Disseminated intravascular coagulation	3	3.0
	ICU admission	9	9.0
	Maternal death	2	2.0
Perinatal outcomes	Any adverse perinatal outcome	39	39.0
	Preterm birth <37 weeks	35	35.0
	Low birth weight <2500 g	38	38.0
	5-minute Apgar score <7	18	18.0
	NICU admission	31	31.0
	Stillbirth	8	8.0
	Early neonatal death	4	4.0

Significant predictors of adverse maternal outcomes included primigravida status, gestational age <34 weeks, previous preeclampsia, BP ≥160/110 mmHg, proteinuria ≥3+, thrombocytopenia, elevated liver enzymes, and creatinine ≥1.2 mg/dL (p<0.05). Adverse perinatal outcomes were significantly associated with gestational age <34 weeks, proteinuria ≥3+, thrombocytopenia, elevated liver enzymes, and creatinine ≥1.2 mg/dL (p<0.05). Age ≥30 years was not significantly associated with either outcome.

Table III
Association of Potential Predictors with Adverse Maternal and Perinatal Outcomes Among Women with Severe Preeclampsia.

Predictor	Adverse Maternal Outcome (n=34)	No Adverse Maternal Outcome (n=66)	p-value	Adverse Perinatal Outcome (n=39)	No Adverse Perinatal Outcome (n=61)	p-value
Age ≥30 years	17 (50.0)	20 (30.3)	0.052	18 (46.2)	19 (31.1)	0.127
Primigravida	19 (55.9)	23 (34.8)	0.045	21 (53.8)	21 (34.4)	0.057
Gestational age <34 weeks	20 (58.8)	15 (22.7)	<0.001	25 (64.1)	10 (16.4)	<0.001
Previous preeclampsia	10 (29.4)	8 (12.1)	0.031	9 (23.1)	9 (14.8)	0.282
BP ≥160/110 mmHg	30 (88.2)	42 (63.6)	0.009	32 (82.1)	40 (65.6)	0.076
Proteinuria ≥3+	24 (70.6)	30 (45.5)	0.019	26 (66.7)	28 (45.9)	0.043
Platelet <100,000/mm³	12 (35.3)	4 (6.1)	<0.001	11 (28.2)	5 (8.2)	0.006
Elevated liver enzymes	14 (41.2)	5 (7.6)	<0.001	12 (30.8)	7 (11.5)	0.017
Creatinine ≥1.2 mg/dL	9 (26.5)	3 (4.5)	0.001	8 (20.5)	4 (6.6)	0.044

Multivariable analysis identified gestational age <34 weeks, BP ≥160/110 mmHg, thrombocytopenia, elevated liver enzymes, and creatinine ≥1.2 mg/dL as independent predictors of adverse maternal outcomes. For adverse perinatal outcomes, independent predictors were gestational age <34 weeks, proteinuria ≥3+, thrombocytopenia, and elevated liver enzymes.

Table IV
Multivariable Logistic Regression Analysis of Predictors of Adverse Maternal and Perinatal Outcomes Among Women with Severe Preeclampsia.

Predictor	Adverse Maternal Outcome			Adverse Perinatal Outcome		
	Adjusted OR	95% CI	p-value	Adjusted OR	95% CI	p-value
Gestational age <34 weeks	3.82	1.55–9.42	0.003	5.24	2.14–12.84	<0.001
BP ≥160/110 mmHg	2.41	1.01–5.77	0.048	1.84	0.78–4.33	0.163
Proteinuria ≥3+	1.72	0.76–3.91	0.193	2.31	1.02–5.25	0.044
Platelet <100,000/mm³	4.76	1.49–15.21	0.009	3.18	1.05–9.62	0.041
Elevated liver enzymes	4.21	1.52–11.65	0.006	2.87	1.04–7.92	0.042
Creatinine ≥1.2 mg/dL	3.67	1.02–13.18	0.046	2.94	0.88–9.82	0.079

DISCUSSION

In this prospective cohort study conducted at Sher-e-Bangla Medical College Hospital, Barishal, a substantial proportion of women with severe preeclampsia experienced adverse maternal and perinatal outcomes. Earlier gestational age, thrombocytopenia, and elevated liver enzymes were independently associated with both maternal and perinatal complications, while severe hypertension and elevated creatinine predicted adverse maternal outcomes and significant proteinuria predicted adverse perinatal outcomes. These findings underscore the importance of early risk assessment and recognition of maternal organ dysfunction to identify women requiring closer monitoring and timely intervention.

In the present study, women with severe preeclampsia had a mean age of 27.8 ± 5.1 years, with the majority belonging to the 20–29-year age group (55.0%). This finding was comparable to that reported by Akhter et al. [13], who observed that the largest proportion of women with severe preeclampsia were aged 21–25 years (41%), with a mean age of 28.32 ± 6.8 years. Similarly, Sultana et al. [14] reported

a mean maternal age of 27.65 ± 5.85 years, which closely corresponded to the present study. Multigravida women predominated in the current study (58.0%), while primigravida women accounted for 42.0%, closely resembling the distribution reported by Sultana et al. [14], where 60% were multiparous and 40% were primiparous. The similar maternal age and parity distributions across these studies suggest a broadly comparable sociodemographic and obstetric profile among women presenting with severe preeclampsia.

The clinical and laboratory characteristics observed at admission were also broadly consistent with previous reports. Headache was the most common symptom in the present study (61.0%), closely corresponding to the 60.5% reported among women with severe preeclampsia presenting before 34 weeks in the study by Kınay et al. [15]. Visual disturbance and epigastric/right upper abdominal pain were observed in 28.0% and 24.0%, respectively, compared with 15.1% and 5.8% in the study by Kınay et al. [15]. Differences in the frequency of these symptoms may reflect variations in gestational age, disease severity, and the clinical characteristics of the study populations. Severe hypertension with BP ≥160/110 mmHg was present in 72.0% of women, while proteinuria ≥3+ was observed in 54.0%, consistent with the clinical features used to characterize severe preeclampsia in the large cohort reported by Yıldırım et al. [16]. Evidence of maternal organ dysfunction was also observed, including thrombocytopenia (16.0%), elevated liver enzymes (19.0%), and serum creatinine ≥1.2 mg/dL (12.0%). Similarly, Yıldırım et al. [16] reported renal dysfunction, pulmonary edema, and placental abruption among women with severe hypertensive disease, although at different frequencies. The presence of HELLP syndrome (10.0%) and eclampsia (7.0%) in the current study further reflects the severe clinical spectrum of hypertensive disorders in pregnancy.

The maternal and perinatal outcomes in the present study demonstrated the substantial morbidity associated with severe preeclampsia. An adverse maternal outcome occurred in 34.0% of women, with HELLP syndrome (10.0%), ICU admission (9.0%), acute kidney injury (8.0%), eclampsia (7.0%), and placental abruption (6.0%) being the more frequent complications. These findings were broadly comparable with those of Pillai et al. [17], who reported deranged liver function tests in 19%, abnormal renal function in 17%, and DIC-related maternal near-miss events in 3% of women. Notably, the prevalence of elevated liver enzymes (19%) and DIC (3%) in the present study was identical to that reported by Pillai et al. [17]. Patel et al. [18] similarly reported deranged liver function in 26.68%, abnormal renal function in 32.5%, and maternal death in one patient among women with severe preeclampsia/eclampsia. In the present study, maternal death occurred in 2.0%, while acute kidney injury and HELLP syndrome occurred in 8.0% and 10.0%, respectively. Regarding perinatal outcomes, preterm birth (35.0%), low birth weight (38.0%), NICU admission (31.0%), and low 5-minute Apgar score (18.0%) were common, while stillbirth and early neonatal death occurred in 8.0% and 4.0%, respectively. Pillai et al. [17] reported preterm birth in 65%, NICU admission in 39%, and neonatal death in 10%, while Patel et al. [18] reported NICU admission in 70.83% and perinatal death in 4.16%. The higher rates of prematurity and NICU admission in these studies may be related to differences in gestational age at presentation, referral patterns, and severity of disease. Overall, the findings demonstrate the considerable maternal and perinatal morbidity associated with severe preeclampsia and are consistent with the outcomes reported in other clinical cohorts.

Analysis of potential predictors showed that adverse maternal outcomes were significantly associated with primigravida status, gestational age <34 weeks, previous preeclampsia, BP ≥160/110 mmHg, proteinuria ≥3+, thrombocytopenia, elevated liver enzymes, and creatinine ≥1.2 mg/dL. For adverse perinatal outcomes, significant associations were observed with gestational age <34 weeks, proteinuria ≥3+, thrombocytopenia, elevated liver enzymes, and creatinine ≥1.2 mg/dL. These findings were consistent with previous predictive studies. von Dadelszen et al. [19] reported that women with adverse maternal outcomes had lower gestational age and higher creatinine and AST levels, while gestational age and dipstick proteinuria independently predicted adverse maternal outcomes. Similarly, Tan et al. [20] identified gestational age, admission systolic blood pressure, platelet count, AST, creatinine, and qualitative proteinuria as important predictors of severe maternal outcomes. The present findings therefore support the importance of earlier gestational age and evidence of hepatic, hematologic, and renal involvement as markers of increased risk. The significant association between these indicators of maternal organ dysfunction and perinatal outcomes further suggests that greater maternal disease severity may adversely affect fetal and neonatal wellbeing.

After adjustment for potential confounders, gestational age <34 weeks emerged as the strongest and most consistent independent predictor, increasing the odds of adverse maternal outcome by nearly four-fold (aOR 3.82) and adverse perinatal outcome by more than five-fold (aOR 5.24). Thrombocytopenia and elevated liver enzymes were also independently associated with both adverse maternal and perinatal outcomes, while creatinine ≥1.2 mg/dL independently predicted adverse maternal outcome. These findings closely correspond with the prediction model developed by Rana et al. [21], which incorporated gestational age, admission systolic blood pressure, platelet count, AST, creatinine, and qualitative proteinuria as key predictors of severe maternal outcomes. In the present study, severe hypertension remained an independent predictor of adverse maternal outcome (aOR 2.41), whereas proteinuria ≥3+ was independently associated with adverse perinatal outcome (aOR 2.31). This latter finding is also consistent with Chan et al. [22], who identified greater proteinuria as an independent predictor of adverse outcomes in a cohort of 353 women with preeclampsia. The lack of statistical significance of proteinuria for maternal outcome in the present multivariable model, despite its significant unadjusted association, may reflect the influence of other correlated markers of disease severity. Overall, the multivariable findings emphasize that earlier gestational age, severe hypertension, proteinuria, thrombocytopenia, hepatic dysfunction, and impaired renal function are clinically important indicators of increased maternal and perinatal risk in severe preeclampsia.

LIMITATIONS

The study had a few limitations:

- The study was conducted at a single tertiary-care hospital with a relatively small sample size of 100 participants, which may limit the generalizability of the findings.
- The relatively short study period and follow-up limited assessment of long-term maternal and neonatal outcomes.

CONCLUSION

Severe preeclampsia is a major hypertensive complication of pregnancy associated with considerable maternal and perinatal morbidity. Gestational age <34 weeks was the strongest independent predictor of both adverse maternal and perinatal outcomes, while thrombocytopenia and elevated liver enzymes independently predicted both outcomes. Severe hypertension and elevated creatinine further predicted adverse maternal outcomes, whereas significant proteinuria predicted adverse perinatal outcomes. Early recognition of these risk factors may facilitate timely intervention and improve maternal and perinatal outcomes.

REFERENCES

1. Magee LA, Brown MA, Hall DR, Gupte S, Hennessy A, Karumanchi SA, Kenny LC, McCarthy F, Myers J, Poon LC, Rana S. The 2021 International Society for the Study of Hypertension in Pregnancy classification, diagnosis & management recommendations for international practice. Pregnancy hypertension. 2022 Mar 1;27:148-69.
2. Facca TA, Famá EA, Mastroianni-Kirsztajn G, Sass N. Why is preeclampsia still an important cause of maternal mortality worldwide?. Revista Brasileira de Ginecologia e Obstetrícia/RBGO Gynecology and Obstetrics. 2020 Sep;42(09):586-7.
3. Lisonkova S, Bone JN, Muraca GM, Razaz N, Wang LQ, Sabr Y, Boutin A, Mayer C, Joseph KS. Incidence and risk factors for severe preeclampsia, hemolysis, elevated liver enzymes, and low platelet count syndrome, and eclampsia at preterm and term gestation: a population-based study. American Journal of Obstetrics & Gynecology. 2021 Nov 1;225(5):538-e1.
4. Hansson SR, Nääv Å, Erlandsson L. Oxidative stress in preeclampsia and the role of free fetal hemoglobin. Frontiers in physiology. 2015 Jan 13;5:516.
5. Backes CH, Markham K, Moorehead P, Cordero L, Nankervis CA, Giannone PJ. Maternal preeclampsia and neonatal outcomes. Journal of pregnancy. 2011;2011(1):214365.
6. Chang KJ, Seow KM, Chen KH. Preeclampsia: recent advances in predicting, preventing, and managing the maternal and fetal life-threatening condition. International journal of environmental research and public health. 2023 Feb 8;20(4):2994.
7. Teka H, Yemane A, Abraha HE, Berhe E, Tadesse H, Gebru F, Yahya M, Tadesse Y, Gebre D, Abrha M, Tesfay B. Clinical presentation, maternal-fetal, and neonatal outcomes of early-onset versus late onset preeclampsia-eclampsia syndrome in a teaching hospital in a low-resource setting: A retrospective cohort study. PloS one. 2023 Feb 27;18(2):e0281952.
8. MacDonald TM, Walker SP, Hannan NJ, Tong S, Kaitu'u-Lino TU. Clinical tools and biomarkers to predict preeclampsia. EBioMedicine. 2022 Jan 1;75.
9. von Dadelszen P, Payne B, Li J, Ansermino JM, Pipkin FB, Côté AM, Douglas MJ, Gruslin A, Hutcheon JA, Joseph KS, Kyle PM. Prediction of adverse maternal outcomes in pre-eclampsia: development and validation of the fullPIERS model. The Lancet. 2011 Jan 15;377(9761):219-27.
10. Li X, Zhang W, Lin J, Liu H, Yang Z, Teng Y, Duan S, Lin X, Xie Y, Li Y, Xie L. Risk factors for adverse maternal and perinatal outcomes in women with preeclampsia: analysis of 1396 cases. The Journal of Clinical Hypertension. 2018 Jun;20(6):1049-57.
11. Wu SW, Wu LF, Wang Q, Zhang WY. Risk factors of adverse pregnancy outcomes during expectant management of early onset severe pre-eclampsia. Zhonghua fu chan ke za zhi. 2010 Mar 1;45(3):165-9.
12. Townsend R, Khalil A, Premakumar Y, Allotey J, Snell KI, Chan C, Chappell LC, Hooper R, Green M, Mol BW, Thilaganathan B. Prediction of pre-eclampsia: review of reviews. Ultrasound in Obstetrics & Gynecology. 2019 Jul;54(1):16-27.
13. AKHTER N, BEGUM A, MONI M, DISHA M, RUMMAN R, HOLY M, SULTANA S. THE OUTCOME OF PREECLAMPSIA WITH SEVERE FEATURES. M ABDUR RAHIM MEDICAL COLLEGE JOURNAL Учредители: M Abdur Rahim Medical College. 2025;18(1):54-66.
14. Sultana S, Siddique M, Hoque S, Shahriar SM, Karim K. Evaluation of Pregnancy Outcome in Severe Preeclampsia. Obs Gyne Review J Obstet Gynecol. 2024;10(1):9–15.
15. Kınay T, Küçük C, Kayıkçıoğlu F, Karakaya J. Severe Preeclampsia versus HELLP Syndrome: Maternal and Perinatal Outcomes at <34 and ≥34 Weeks' Gestation. Balkan Med J. 2015 Oct;32(4):359-63.
16. Yıldırım G, Güngördük K, Aslan H, Gül A, Bayraktar M, Ceylan Y. Comparison of perinatal and maternal outcomes of severe preeclampsia, eclampsia, and HELLP syndrome. J Turk Ger Gynecol Assoc. 2011 Jun 1;12(2):90-6.
17. Pillai SS. Fetomaternal outcome in severe preeclampsia and eclampsia: a retrospective study in a tertiary care centre. Int J Reprod Contracept Obstet Gynecol. 2017 Sep 1;6(9):3937.
18. Patel AJ, Patel BS, Shah AC, Jani SK. Maternal and perinatal outcome in severe pre-eclampsia and eclampsia: a study of 120 cases at a tertiary health care center in Western India. International Journal of Reproduction, Contraception, Obstetrics and Gynecology. 2021 Mar 1;10(3):1011-7.
19. von Dadelszen P, Magee LA, Devarakonda RM, Hamilton T, Ainsworth LM, Yin R, Norena M, Walley KR, Gruslin A, Moutquin JM, Lee SK, Russell JA. The prediction of adverse maternal outcomes in preeclampsia. J Obstet Gynaecol Can. 2004 Oct;26(10):871-9.
20. Tan J, Yang M, Liao Y, Qi Y, Ren Y, Liu C, Huang S, Thabane L, Liu X, Sun X. Development and validation of a prediction model on severe maternal outcomes among pregnant women with pre-eclampsia: a 10-year cohort study. Sci Rep. 2020 Sep 24;10(1):15590.
21. Rana S, Powe CE, Salahuddin S, Verlohren S, Perschel FH, Levine RJ, Lim KH, Wenger JB, Thadhani R, Karumanchi SA. Angiogenic factors and the risk of adverse outcomes in women with suspected preeclampsia. Circulation. 2012 Feb 21;125(7):911-9.
22. Chan P, Brown M, Simpson JM, Davis G. Proteinuria in pre-eclampsia: how much matters? BJOG. 2005 Mar;112(3):280-5.