







Original Research

Associations Between Hypertensive Disorders in Pregnancy and Maternal and Fetal Outcomes: A Retrospective Cohort Study on Systolic and Diastolic Blood Pressure Levels at Delivery

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Academic Editors: George Daskalakis and Michael H. Dahan

Submitted: 18 July 2025 Revised: 4 September 2025 Accepted: 25 September 2025 Published: 16 December 2025

Abstract

Background: This study aimed to determine the effect of hypertensive disorders during pregnancy, systolic blood pressure (SBP), and diastolic blood pressure (DBP) on maternal and fetal mortality and morbidity. **Methods:** This retrospective cohort study included 195 women aged ≥ 18 years with hypertensive disorders during pregnancy who underwent caesarean section between 2012 and 2017. Patients were divided into groups based on their hypertensive diagnosis (25 with gestational hypertension (GHT), 164 with preeclampsia, 6 with eclampsia) or the recorded SBP or DBP readings at delivery. Logistic regression and linear regression were used to examine associations between hypertensive diagnosis, DBP at delivery, SBP at delivery, and the development of adverse maternal and fetal outcomes after adjusting for confounders. **Results:** Multivariable regression analysis revealed a trend toward an increased risk of intrauterine growth restriction (IUGR) (adjusted risk ratio (aRR) = 2.92; $p = 0.09$) in patients with eclampsia compared to those with GHT, while patients with preeclampsia had a significantly increased risk of IUGR (aRR = 2.77; $p = 0.003$). Patients with preeclampsia also had a threefold increased risk of premature delivery (aRR = 3.29; $p < 0.004$), while those with eclampsia had a fourfold increased risk (aRR = 4.09; $p = 0.002$) compared to patients with GHT. Both groups also had significantly lower fetal birth weights than the GHT group. A DBP of ≥ 90 mmHg during delivery was associated with significantly reduced fetal birth weight (coefficients (Coef.), -381.5 , 95% confidence interval (CI), -739.6 to -23.4 ; $p = 0.04$). No significant differences were observed in the outcomes between patients with an SBP value < 140 mmHg during delivery and those with a SBP recording ≥ 140 mmHg. **Conclusions:** SBP and DBP are important parameters in the maternal early warning criteria. Strict DBP monitoring may help to increase patient safety, especially in patients with preeclampsia or eclampsia.

Keywords: diastolic blood pressure; systolic blood pressure; hypertensive disorders of pregnancy; gestational hypertension; preeclampsia; eclampsia

1. Introduction

Gestational hypertension (GHT) is defined as systolic blood pressure (SBP) ≥ 140 mmHg, or diastolic blood pressure (DBP) ≥ 90 mmHg, measured after 20 weeks of gestation [1]. The prevalence of GHT in women of reproductive age is 10% [2]. Preeclampsia is the most common cause of hypertensive disorders of pregnancy worldwide (3–5% of pregnancies) and is associated with maternal and fetal mortality, as well as severe morbidity. If proteinuria accompanies SBP > 160 mmHg and/or DBP > 110 mmHg, this is defined as severe pre-eclampsia, or pre-eclampsia with severe features [3].

Hypertensive disorders of pregnancy are associated with higher risks for diabetes mellitus, renal disorders, cardiovascular disorders, and premature birth [4]. It has been

estimated that one in ten maternal deaths in Asia and Africa is due to hypertensive disorders of pregnancy [2], and that GHT is responsible for 16% of stillbirths worldwide. More detailed analyses have shown that high SBP was associated with increased admission to intensive care in the postpartum period, as well as increases in preterm birth, stillbirth, and kidney damage [5]. Moreover, 10% of early neonatal deaths are known to be accompanied by hypertensive disorders of pregnancy [6]. It has been shown that high SBP values increase fetal mortality and morbidity, as well as maternal mortality and morbidity [7]. For this reason, early diagnosis and treatment of hypertensive disorders are important in order to prevent complications [8].

Changes in the cardiovascular system and volume balance between body compartments in patients with GHT



lead to a vicious cycle that involves an uncontrolled increase in blood pressure due to increased cardiac output and intravascular volume, activation of the renin-angiotensin-aldosterone system, sodium retention, and natriuresis [9]. These pathophysiological changes make it difficult to diagnose hypertensive disorders in early pregnancy, resulting in treatment disruption that can lead to serious maternal and fetal complications. One of the most common adverse impacts on the fetus is intrauterine growth restriction (IUGR), resulting from decreased placental blood flow due to increased maternal blood pressure [10].

Changes in SBP and DBP caused by aggressive treatment of hypertension are explained with the concept of the “J curve”, whereby serious reductions and elevations in DBP rather than SBP have negative effects on organ perfusion. Bilo *et al.* [11] reported variations in the “J curve” according to age and concomitant diseases. The Systolic Blood Pressure Intervention Trial (SPRINT) study [12] highlighted that SBP should be kept at <120 mmHg in order to reduce cardiovascular risks that may occur due to hypertension. However, it is known that changes in DBP as well as SBP cause some negative alterations in the overall macrocirculation, starting in microcirculatory compartments [13]. DBP values <70 mmHg have been shown to impair myocardial perfusion by causing ischemia due to reduced coronary blood perfusion [14]. Data from the treatment of preserved cardiac function heart failure with an aldosterone antagonist trial (TOPCAT) study on 3417 patients showed that DBP <70 mmHg was associated with poor outcomes in patients with heart failure [15]. Conversely, some studies have shown that elevated DBP in hypertensive patients was also associated with an increased risk of cardiovascular and neurological complications [16,17]. A study of pregnant women found that, unlike DBP, changes in SBP were not associated with preeclampsia and low birth weight, suggesting that DBP may be more important for microcirculatory haemostasis [18].

To our knowledge, there are still no studies in the literature on the “J curve” in patients with GHT. In pregnant women with uncontrolled hypertension after 37 weeks gestation, emergency delivery is recommended due to the increased risk of maternal and fetal complications. However, there continue to be poor outcomes in this patient group, possibly due to a lack of research with regard to DBP [10]. Therefore, the primary aim of this study was to investigate the effects of hypertensive disorders of pregnancy on maternal and fetal mortality and morbidity. The secondary aim was to examine the effects of elevated DBP and SBP at delivery on maternal and fetal pregnancy outcomes.

2. Materials and Methods

2.1 Study Design and Participants

This study was conducted at Marmara University Pendik Training and Research Hospital, and ethical ap-

proval was obtained from the Ethics Committee of Marmara University (Approval No: 09.2018.162, dated 06 January 2018). The study was performed in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its subsequent revisions. This was a retrospective cohort study of adult patients with hypertensive disorders of pregnancy. We included pregnant women aged >18 years who underwent caesarean section at our university hospital under regional or general anaesthesia following admission for GHT, preeclampsia, or eclampsia between 2012 and 2017. Patients who underwent surgeries other than caesarean section were excluded, as were those with incomplete data. Also excluded were patients admitted to the intensive care unit (ICU), since these represent a distinct population with a greater severity of illness, as well as different management protocols and outcome trajectories. The inclusion of such patients would likely have introduced confounding factors, thus reducing the internal validity of our study.

2.2 Data Collection and Variables

The requirement for patient consent was waived because of the retrospective nature of this study. Data was entered into a standardised data collection sheet on Microsoft Excel for Mac, Version 16.83 (Microsoft Corporation, Redmond, WA, USA). The following information was collected: (1) baseline demographic characteristics including age, body mass index (BMI), gestational age, concomitant maternal disorders, and laboratory results for creatinine and haemoglobin; (2) SBP and DBP readings at admission and delivery; (3) fetal and neonatal data including fetal birth weight, Appearance, Pulse, Grimace, Activity and Respiration (APGAR) scores at 1 and 5 minutes, prematurity, and IUGR; and (4) postpartum and postoperative outcomes including length of hospital stay, presence of postoperative severe arrhythmia, and pulmonary embolism. The length of hospital stay was defined as the duration between the first day of patient admission to the hospital and discharge. Gestational age was recorded as a continuous variable, but was categorized into the following clinically meaningful groups: preterm (<37 weeks), term (37–41 weeks), and post-term (≥ 42 weeks). This categorization facilitated the interpretation of outcomes and also aligns with standard obstetric practice.

Both SBP and DBP values were recorded to evaluate potential relationships between hypo- or hypertension and various outcomes, including length of hospital stay, fetal birth weight, and risks for pulmonary embolism, IUGR, premature delivery, and arrhythmia. Blood pressure was measured using an automated oscillometric device (Model HKD-02, manufactured by Hunkar Ltd. A.S., Denizli, Turkey). Measurements were obtained in the seated position after at least 5 min rest. For each patient, three consecutive readings were taken at one-minute intervals, with the average of the last two readings used for analysis. All

measurements were performed by trained personnel using appropriately sized cuffs to ensure consistency and accuracy.

Participants were categorized into three different exposure groups to assess outcomes across various hypertensive and blood pressure parameters. The first categorization grouped patients by their specific diagnosis of hypertensive disorder of pregnancy (GHT, preeclampsia, or eclampsia). The second categorization grouped participants based on their DBP at delivery (<90 mmHg or ≥ 90 mmHg), while the third grouped them by their SBP at delivery (<140 mmHg or ≥ 140 mmHg).

Preeclampsia was diagnosed based on the presence of new-onset hypertension after 20 weeks of gestation, along with proteinuria ≥ 300 mg/24 h, or a urine dipstick reading $\geq 2+$. In cases without significant proteinuria, diagnosis was made if there were systemic manifestations, including thrombocytopenia, renal or hepatic dysfunction, pulmonary edema, or neurological symptoms. To prevent the overestimation of cases, a urine dipstick reading of 1+ was not considered diagnostic.

Postoperative severe arrhythmia was defined as any clinically significant disturbance of cardiac rhythm occurring within the first 48 h after surgery and requiring intervention, including pharmacologic therapy or electrical cardioversion, or causing hemodynamic instability. Severe arrhythmias included sustained ventricular tachycardia, ventricular fibrillation, torsades de pointes, new-onset high-grade atrioventricular block, or rapid atrial fibrillation/flutter with hemodynamic compromise. All events were confirmed by continuous electrocardiogram (ECG) monitoring, telemetry, or 12-lead ECG.

2.3 Statistical Analysis

Statistical comparisons were performed to evaluate differences across three groups: GHT, preeclampsia, and eclampsia. Numerical data were summarized as the mean and standard deviation if normally distributed, or as the median and interquartile range if they were not normally distributed. The distribution of continuous variables, such as age, birth weight, and BMI, was assessed using the Shapiro-Wilk test. Groups with normally distributed numerical data were compared with the Student's *t* test and analysis of variance (ANOVA) test, while groups with skewed data were compared with the Kruskal-Wallis test and the Wilcoxon rank-sum test. Categorical data were presented as frequencies and percentages. Pearson's Chi-squared test and Fisher's exact test were used to test hypotheses and compare categorical data.

The relationships between exposures and outcomes of interest were evaluated using three models of multivariable logistic regression and linear regression. The first model examined associations between hypertensive disorders of pregnancy and maternal and fetal outcomes, the second model examined associations between DBP at delivery and

the same outcomes, while the third model examined associations between SBP at delivery and the same outcomes. The logistic regression and linear regression models were adjusted for age and gestational diabetes mellitus (GDM). Logistic regression was utilized for categorical outcomes, which included the development of pulmonary embolism, IUGR, premature delivery, and postoperative arrhythmia. Linear regression was employed for continuous outcomes, including fetal birth weight and the length of hospital stay. For categorical outcomes, modified Poisson regression with robust variance estimation was used to calculate adjusted risk ratios (aRR). We chose this approach because several outcomes were common, and estimating aRRs provides a more interpretable measure of relative risk than odds ratios, which can overestimate the effect size in such scenarios.

The 95% confidence interval (95% CI) for aRRs and regression coefficients (Coef.) was also reported. Exact *p*-values were reported and interpreted as evidence against the null hypothesis. All statistical analyses were carried out using IBM SPSS Statistics 22 (IBM Corp, Armonk, NY, USA) and Stata 18.0 (StataCorp, College Station, TX, USA).

3. Results

Fig. 1 shows the flow chart for this study and the participant selection process. A total of 2246 patient records underwent preliminary screening for eligibility. After exclusion of ineligible cases, 195 participants were available for inclusion, of which 25 had GHT, 164 had preeclampsia, and 6 had eclampsia. Patients with incomplete data were handled using listwise deletion. Four patients with missing data were excluded from the final analysis.

Table 1 presents the baseline characteristics of participants. The eclampsia group was the youngest (mean age 27.0 years), while the GHT group was the oldest (mean age 33.8 years). Patients with GHT had a shorter median hospital stay compared to patients with preeclampsia or eclampsia. The mean fetal birth weight was lowest in the eclampsia group, followed by the preeclampsia and then GHT groups. The majority of participants were multigravida, with 52 (26.7%) being primigravida. The majority of patients with GHT delivered between 37 and 41+6 weeks (76%), whereas most deliveries for preeclampsia patients occurred between 31 and 36+6 weeks (43.9%).

3.1 Intergroup Comparisons of Demographic and Clinical Characteristics

A significant difference in patient age was found between groups ($p = 0.04$). Pairwise analysis revealed that patients with preeclampsia were significantly younger compared to those with GHT ($p = 0.015$). Differences in the level of 24-h urine protein between groups were highly significant ($p < 0.001$). Post-hoc analysis revealed that preeclampsia patients had significantly higher protein lev-

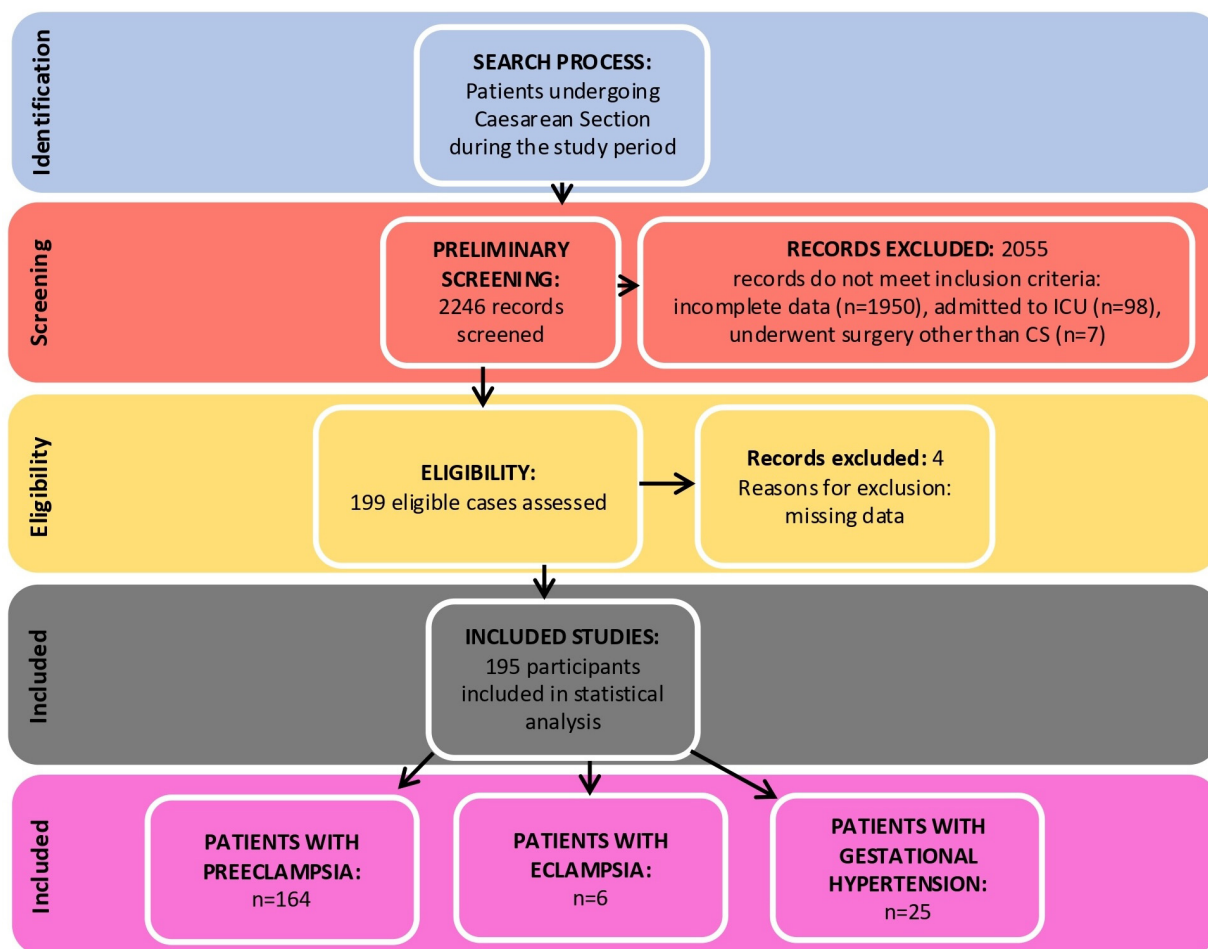


Fig. 1. Flow chart for participant selection. ICU, intensive care unit; CS, cesarean section.

els than GHT patients ($p < 0.001$), while eclampsia patients had significantly higher protein levels than both of the other groups ($p = 0.001$).

3.2 Intergroup Comparisons of Maternal and Fetal Outcomes

A significant difference was found in the gestational age at delivery ($p < 0.001$). Specifically, patients with preeclampsia had significantly earlier deliveries than those with GHT ($p < 0.001$). Birth weight was also significantly different between the groups ($p < 0.001$). Pairwise comparisons showed that preeclampsia patients had infants with a lower birth weight than those of GHT patients ($p < 0.001$). Similarly, eclampsia patients also had infants with a lower birth weight than those of GHT patients ($p = 0.019$).

The duration of hospital stay differed significantly across the groups ($p < 0.001$). Both preeclampsia ($p < 0.001$) and eclampsia ($p = 0.005$) groups had significantly longer hospital stays compared to the GHT group. Primigravid status was significantly associated with the diagnostic group ($p = 0.039$), and was attributable to a significant difference between the preeclampsia and GHT groups ($p = 0.049$).

DBP at delivery demonstrated a significant overall difference between groups ($p = 0.036$). This was driven by a difference between the preeclampsia and GHT groups ($p = 0.016$), with no significant difference observed in the other pairwise comparisons. Creatinine levels were significantly different among the groups ($p = 0.005$). Pairwise comparisons confirmed that both preeclampsia ($p = 0.005$) and eclampsia patients ($p = 0.040$) had significantly higher creatinine levels compared to GHT patients.

Table 2 presents the blood pressure measurements, as well as the maternal and fetal outcomes, in each of the three hypertensive groups. No cases of maternal mortality occurred in this cohort. All groups had a similarly low incidence of postoperative arrhythmia and pulmonary embolism. Our analysis also revealed a significant association between prematurity and the diagnostic group ($p < 0.001$), with the difference being primarily between the preeclampsia and GHT groups ($p < 0.001$).

Further, the majority of patients had elevated SBP and DBP readings at delivery. The incidence of seizures was very different across the groups ($p < 0.001$). As expected, a significant difference was observed between the preeclampsia and eclampsia groups ($p < 0.001$), and be-

Table 1. Baseline characteristics of the study participants.

Parameter	Gestational hypertension (n = 25)	Preeclampsia (n = 164)	Eclampsia (n = 6)
Age, years; mean (SD)	33.8 (6.0)	30.5 (6.3)	27.0 (6.0)
BMI, kg/m ² ; mean (SD)	32.7 (2.9)	31.5 (5.0)	30.0 (3.8)
Gestational age, weeks			
24–30+6	0 (0.0%)	28 (17.1%)	3 (50.0%)
31–36+6	6 (24.0%)	72 (43.9%)	3 (50.0%)
37–41+6	19 (76.0%)	64 (39.0%)	0 (0.0%)
Primigravida/Multigravida			
Primigravida	2 (8.0%)	46 (28.0%)	4 (66.7%)
Multigravida	23 (92.0%)	118 (72.0%)	2 (33.3%)
C-section urgency			
Elective	4 (16.0%)	22 (13.4%)	0 (0.0%)
Emergency	21 (84.0%)	142 (86.6%)	6 (100.0%)
Anaesthesia type			
General	14 (56.0%)	89 (54.3%)	6 (100.0%)
Spinal	11 (44.0%)	75 (45.7%)	0 (0.0%)
Birth weight, g; mean (SD)	3279.6 (866.5)	2298.6 (1013.6)	1623.3 (706.9)
APGAR score, 1 min; median (IQR)	9.0 (8.0–9.0)	8.0 (6.0–9.0)	5.0 (5.0–8.0)
APGAR score, 5 min; median (IQR)	10.0 (10.0–10.0)	10.0 (8.0–10.0)	7.5 (7.0–9.0)
Gestational DM	1 (4.0%)	11 (6.7%)	0 (0.0%)
Pre-pregnancy DM	3 (12.0%)	8 (4.9%)	0 (0.0%)
Antihypertensive medication			
None	11 (44.0%)	30 (18.3%)	1 (16.7%)
Methyldopa	1 (4.0%)	23 (14.0%)	0 (0.0%)
Nifedipine	10 (40.0%)	48 (29.3%)	3 (50.0%)
Methyldopa & Nifedipine	3 (12.0%)	61 (37.2%)	2 (33.3%)
Methyldopa & Doxazosin	0 (0.0%)	1 (0.6%)	0 (0.0%)
Nifedipine & Captopril	0 (0.0%)	1 (0.6%)	0 (0.0%)
Urine protein			
Negative	4 (16.0%)	13 (7.9%)	1 (16.7%)
Trace	21 (84.0%)	18 (11.0%)	1 (16.7%)
1+	0 (0.0%)	14 (8.5%)	0 (0.0%)
2+	0 (0.0%)	61 (37.2%)	0 (0.0%)
3+	0 (0.0%)	52 (31.7%)	3 (50.0%)
4+	0 (0.0%)	6 (3.7%)	1 (16.7%)
24-h urine protein, g; median (IQR)	0.0 (0.0–110.0)	1645.5 (597.5–3294.0)	3839 (800.0–8632.0)
Hospital LOS, days; median (IQR)	2.0 (2.0–3.0)	3.5 (3.0–5.0)	4.5 (4.0–5.0)

BMI, body mass index; C-Section, caesarean section; APGAR, Appearance, Pulse, Grimace, Activity and Respiration; DM, diabetes mellitus; SD, standard deviation; IQR, interquartile range; LOS, length of stay.

tween the eclampsia and GHT groups ($p < 0.001$). The rates of seizure were negligible in the preeclampsia and GHT groups (Table 2).

We examined the associations between the type of hypertensive disorder including gestational hypertension, preeclampsia, and eclampsia together with maternal and fetal outcomes. Multivariable logistic regression analysis showed that compared to patients with GHT, patients with preeclampsia and eclampsia had 2.77-fold and 2.92-fold higher risks of developing IUGR, respectively (Table 3). Similarly, patients with preeclampsia and eclampsia had 3-fold and 4-fold higher risks of premature delivery, respectively, compared to those with GHT. The risk for postoperative arrhythmia was lower in the preeclampsia group, but

this difference did not reach statistical significance. There was no difference in the risk of pulmonary embolism between patients with preeclampsia and GHT. Linear regression showed that both preeclampsia and eclampsia were both associated with significantly lower fetal birth weight compared to GHT. Further, they were both associated with a longer hospital stay compared to GHT. The association between eclampsia and the development of pulmonary embolism or postoperative arrhythmia could not be assessed due to an insufficient number of cases with these outcomes.

We evaluated the impact of diastolic blood pressure at delivery, comparing women with DBP <90 mmHg versus ≥ 90 mmHg. Patients with a DBP of <90 mmHg during delivery had consistently lower frequencies of pulmonary em-

Table 2. Blood pressure measurements, and incidence of maternal and fetal complications.

Factor	Gestational hypertension (n = 25)	Preeclampsia (n = 164)	Eclampsia (n = 6)
SBP on admission, mmHg; mean (SD)	162.0 (14.8)	163.5 (15.6)	162.7 (15.5)
DBP on admission, mmHg; mean (SD)	96.4 (10.5)	99.4 (12.4)	96.5 (15.3)
HR on admission, bpm; mean (SD)	91.4 (11.5)	94.3 (14.5)	82.0 (12.9)
SBP before induction, mmHg; mean (SD)	151.8 (18.0)	157.6 (23.8)	172.5 (14.1)
DBP before induction, mmHg; mean (SD)	88.6 (13.0)	96.2 (16.3)	102.0 (13.0)
HR before induction, bpm; mean (SD)	103.2 (15.8)	101.4 (16.8)	109.8 (14.0)
SBP at delivery <140 mmHg	2 (8.0%)	23 (14.0%)	0 (0.0%)
SBP at delivery ≥140 mmHg	23 (92.0%)	141 (86.0%)	6 (100.0%)
DBP at delivery <90 mmHg	8 (32.0%)	33 (20.1%)	0 (0.0%)
DBP at delivery ≥90 mmHg	17 (68.0%)	131 (79.9%)	6 (100.0%)
Pulmonary embolism	1 (4.0%)	4 (2.4%)	0 (0.0%)
IUGR	4 (16.0%)	72 (43.9%)	3 (50.0%)
Seizure	0 (0.0%)	0 (0.0%)	6 (100.0%)
Prematurity	5 (20.0%)	108 (65.9%)	5 (83.3%)
Postoperative severe arrhythmia	1 (4.0%)	4 (2.4%)	0 (0.0%)

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; IUGR, intrauterine growth restriction.

Table 3. Multivariable analysis of the associations between hypertensive disorders of pregnancy and maternal and fetal outcomes.

Outcome	Condition	Adjusted effect size*	95% CI	p-value
Birth weight	Preeclampsia	Coef. -992.4	-1411.6 to -573.3	<0.0001
	Eclampsia	Coef. -1599.0	-2485.0 to -713.1	<0.0001
Hospital LOS	Preeclampsia	Coef. 2.06	0.62 to 3.50	0.005
	Eclampsia	Coef. 1.91	-1.13 to 4.95	0.220
Pulmonary embolism	Preeclampsia	aRR 0.90	0.10 to 8.00	0.930
	Eclampsia	-	-	-
IUGR	Preeclampsia	aRR 2.77	1.11 to 6.93	0.003
	Eclampsia	aRR 2.92	0.85 to 10.01	0.090
Prematurity	Preeclampsia	aRR 3.29	1.48 to 7.32	<0.004
	Eclampsia	aRR 4.09	1.69 to 9.90	0.002
Postoperative severe arrhythmia	Preeclampsia	aRR 0.63	0.07 to 5.69	0.680
	Eclampsia	-	-	-

LOS, length of stay; aRR, adjusted risk ratio; CI, confidence interval; Coef., regression coefficients.

* Adjusted for age and gestational diabetes mellitus.

bolism, IUGR, premature delivery, postoperative arrhythmia, and seizures compared to patients with a DBP of ≥90 mmHg during delivery (Table 4). Adjusted linear regression revealed that a DBP of ≥90 mmHg during delivery was associated with a significantly lower fetal birth weight compared to patients with a DBP of <90 mmHg at delivery (Coef. -381.5, 95% CI: -739.6 to -23.4, $p = 0.04$). On the other hand, there was no difference in hospital length of stay or between the high and low DBP groups. Despite weak evidence against the null hypothesis, we observed 20% and 21% increases in the risks of IUGR and premature delivery, respectively, for patients with a DBP of ≥90 mmHg during delivery (Table 4). The risks for pulmonary embolism and seizures could not be analyzed, as none of the patients with a DBP of <90 mmHg at delivery developed these outcomes.

We analyzed the relationship between systolic blood pressure at delivery as <140 mmHg vs. ≥140 mmHg and the same set of outcomes. The effects of SBP during delivery on maternal and fetal outcomes are presented in Table 5. The mean fetal birth weight was slightly higher in patients with SBP ≥140 mmHg during delivery (mean 2423.4 g) compared to patients with SBP <140 mmHg (mean 2269.1 g). Both groups had an equal median hospital length of stay of three days. Almost no differences were observed in the frequencies of maternal and fetal outcomes between the two groups. Multivariable logistic regression showed no differences in the risk of developing IUGR or premature delivery in patients with SBP ≥140 mmHg during delivery compared to those with SBP <140 mmHg at delivery (Table 5).

Table 4. Effects of DBP during delivery on maternal and fetal outcomes.

Factor	DBP <90 mmHg (n = 41)	DBP ≥90 mmHg (n = 154)	Adjusted effect size*	95% CI	p-value
Birth weight, mean (SD)	2658.2 (1037.2)	2335.8 (1042.3)	Coef. -381.5	-739.6 to -23.4	0.04
Hospital LOS, median (IQR)	3.0 (2.0, 5.0)	3.0 (3.0, 5.0)	Coef. 0.38	-0.81 to 1.57	0.53
Pulmonary embolism	0 (0.0%)	5 (3.2%)	-	-	-
IUGR	15 (36.6%)	64 (41.8%)	aRR 1.20	0.77 to 1.89	0.42
Prematurity	22 (53.7%)	96 (62.3%)	aRR 1.21	0.88 to 1.66	0.25
Postoperative severe arrhythmia	1 (2.4%)	4 (2.6%)	aRR 1.06	0.12 to 9.45	0.96
Seizure	0 (0.0%)	6 (3.9%)	-	-	-

* Adjusted for age and gestational diabetes mellitus.

Table 5. Effects of SBP during delivery on maternal and fetal outcomes.

Factor	SBP <140 mmHg (n = 25)	SBP ≥140 mmHg (n = 170)	Adjusted effect size*	95% CI	p-value
Birth weight, mean (SD)	2269.1 (945.1)	2423.4 (1062.2)	Coef. 149.7	-287.3 to 586.8	0.50
Hospital LOS, median (IQR)	3.0 (2.0, 6.0)	3.0 (3.0, 5.0)	Coef. 0.54	-0.89 to 1.98	0.46
Pulmonary embolism	0 (0.0%)	5 (2.9%)	-	-	-
IUGR	11 (44.0%)	68 (40.2%)	aRR 0.91	0.57 to 1.47	0.72
Prematurity	15 (60.0%)	103 (60.6%)	aRR 1.02	0.72 to 1.44	0.91
Postoperative severe arrhythmia	0 (0.0%)	5 (2.9%)	-	-	-
Seizure	0 (0.0%)	6 (3.5%)	-	-	-

* Adjusted for age and gestational diabetes mellitus.

4. Discussion

This study of patients with hypertensive disorders of pregnancy found that preeclampsia and eclampsia increased the risk of developing IUGR by 2.77-fold and 2.92-fold, respectively, compared to GHT. Further, elevated DBP during delivery (≥ 90 mmHg) was associated with significantly reduced fetal birth weight, as well as increased risks of developing IUGR (20%) and premature delivery (21%), although these did not reach statistical significance. In contrast, patients with SBP ≥ 140 mmHg during delivery showed no differences in the risk of developing these complications compared to those with SBP < 140 mmHg. In this context, it has been observed that elevated DBP values at the time of delivery may be superior to SBP in terms of predicting maternal and fetal outcomes in patients with hypertensive disorders of pregnancy. DBP ≥ 90 mmHg was associated with a significantly lower birth weight, but the wide confidence interval (-739.6 to -23.4 g) indicates considerable uncertainty in the estimate. Although this warrants clinical attention, the magnitude of the effect should be interpreted cautiously, as the true impact may vary widely. Therefore, both statistical and clinical significance must be considered when evaluating the implications of elevated maternal DBP on fetal growth.

Prolonged high blood pressure leads to cardiac chamber remodeling, concentric hypertrophy, and diastolic dysfunction, thereby creating conditions that can facilitate arrhythmias, including atrial fibrillation [19]. Various studies have shown that elevated SBP and DBP readings are associated with atrial and ventricular arrhythmia in both ob-

stetric and non-obstetric hypertensive individuals [20,21]. A meta-analysis in a non-obstetric hypertensive population found that individuals with SBP of 120–130 mmHg and DBP of 60–69 mmHg had a lower incidence of arrhythmia, highlighting the importance of optimal management of hypertension [22].

The tendency toward arrhythmia increases in pregnancy due to hormonal shifts, reduced autonomic tone, and hemodynamic changes. It often arises in conjunction with a sudden rise in cardiac output and heart rate triggered by pain during delivery [23]. Leon *et al.* [24] identified an association between elevated blood pressure and postpartum arrhythmia in obstetric patients. However, to our knowledge, no studies have yet compared DBP and SBP in this context. In the present study, although we observed that patients with SBP ≥ 140 mmHg or DBP ≥ 90 mmHg showed a higher incidence of postoperative arrhythmia, the difference in this outcome compared to patients with lower blood pressure readings was not statistically significant. Further studies with a large sample size are required to address this subject.

During early pregnancy, uterine circulation has high-capacity and low resistance due to trophoblastic invasion and the remodeling of spiral arteries. Severe preeclampsia is defined as SBP > 160 mmHg or DBP > 110 mmHg, and leads to maternal and fetal complications such as renal dysfunction, pulmonary oedema, and IUGR [25]. Elevated maternal pulse pressure causes a high uterine artery pulsatility index and notching, both of which are associated with preterm birth [26]. Nathan *et al.* [5] reported that increased

SBP was associated with maternal kidney damage, admission to ICU, preterm birth, and stillbirth, but these authors did not detect correlations between elevated DBP and maternal and fetal outcomes. Furthermore, Magee *et al.* [27] reported that maternal DBP ≥ 90 mmHg in patients with pregnancy-related hypertension was associated with low birth weight, preterm delivery, prolonged hospitalization, and worsening of the course of maternal disease. In our study, elevated DBP at the time of delivery was also associated with significantly lower fetal birth weight. Furthermore, we detected associations between DBP ≥ 90 mmHg during birth and worse maternal and fetal outcomes, including premature delivery and IUGR, although only weak evidence was obtained against the null hypothesis. In contrast, however, we found that patients with elevated SBP at delivery showed no differences in maternal and fetal outcomes compared to those with SBP < 140 mmHg.

Studies in the literature have emphasized the importance of managing blood pressure in severe preeclampsia, with uncontrolled decreases in blood pressure having negative effects on patient outcomes. Improvements in maternal and fetal outcomes have been found in patients with SBP < 140 mmHg [2,5]. Additionally, it has been demonstrated that fetal outcomes are adversely affected not only in the case of low SBP, but also in pregnancies where DBP is < 60 mmHg [28]. The current study found that elevated DBP at the time of delivery (≥ 90 mmHg) was associated with a significantly lower fetal birth weight. While we did not find any significant associations between DBP < 90 mmHg and adverse fetal outcomes or maternal morbidity, we acknowledge that our study was not designed to assess the effects of DBP < 60 mmHg. This issue warrants further investigation in future research.

Our findings are consistent with recent evidence highlighting the prognostic importance of maternal DBP in hypertensive disorders of pregnancy. Emerging literature increasingly underscores the nuanced predictive value of both SBP and DBP trajectories in hypertensive disorders of pregnancy. A multicenter retrospective study in women with chronic hypertension found that maintaining SBP below 130 mmHg before 15 weeks reduced the risk of early-onset superimposed preeclampsia and small-for-gestational-age neonates compared with SBP between 140–159 mmHg [29]. Similarly, a Mendelian randomization analysis has provided causal evidence that genetically higher SBP and DBP increase the risk of preeclampsia, preterm birth, and placental abruption [30]. In addition, a large Chinese cohort that examined combined SBP and DBP trajectories showed that higher maternal DBP during both early and late pregnancy was linked to reduced left and right ventricular end-diastolic volumes in offspring, suggesting long-term cardiovascular effects [31]. Taken together, these studies support our observation that DBP may be a more sensitive indicator than SBP for predicting adverse maternal and fetal outcomes in hypertensive pregnancies.

Limitations

The present study has several strengths. It included all eligible adult patients diagnosed with hypertensive disorders of pregnancy and who underwent caesarean section at our university hospital. Additionally, it investigated the impact of high and low SBP and DBP readings on pregnancy outcomes in a high-risk population. This provides valuable insights into the complications associated with uncontrolled blood pressure, highlighting the importance of proper blood pressure management. Our findings contribute to the development of management algorithms and guidelines for patients with hypertensive disorders of pregnancy.

Nonetheless, certain limitations should be acknowledged. First, this was a single-center study, which may limit the generalizability of our findings to other settings or populations. In particular, the sample size for the eclampsia group was small, and hence the findings for these patients should be interpreted with caution. Second, the retrospective cohort study design is subject to incomplete data and does not permit the establishment of causality. Future multicenter studies with larger sample sizes and longitudinal designs are recommended to further explore this topic, establish causality, and examine long-term effects. Although our regression analyses were adjusted for age and GDM, other important potential confounders such as BMI, parity, use of antihypertensive medication, and severity of proteinuria were not included. These unmeasured factors may have influenced outcomes, particularly since the eclampsia patients in our cohort were younger and more likely to be primigravida, both of which may independently affect maternal and fetal outcomes. The study draws exclusively on data collected 7–9 years ago (2012–2017). Over this extended interval, diagnostic algorithms, therapeutic guidelines, and demographic characteristics have evolved substantially. Contemporary clinical practice now incorporates newer pharmacologic agents, refined monitoring modalities, and earlier intervention strategies that may materially alter patient outcomes.

5. Conclusions

In conclusion, hypertensive disorders of pregnancy can significantly affect fetal outcomes. Preeclampsia and eclampsia were associated with markedly increased risks of IUGR compared to GHT. Elevated DBP (≥ 90 mmHg) during delivery was associated with lower fetal birth weight, as well as trends for higher risks of IUGR and premature delivery. In contrast, no such associations were observed for SBP ≥ 140 mmHg during delivery. These findings suggest that DBP at delivery may be a more important predictor of fetal outcomes than SBP. Because we did not assess maternal outcomes in this study, no conclusions can be drawn regarding maternal complications.

Availability of Data and Materials

The data used in this work are available upon reasonable request from the corresponding author.

Author Contributions

AS and KCY designed the research study. GC and UZ performed the research. SS and AS contributed to data collection. KCY and BMA analyzed the data. AS and SS drafted the manuscript. All authors contributed to critical revision of the manuscript for important intellectual content. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was performed in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its subsequent revisions. This study received ethics committee approval with protocol number 09.2018.162 from University of Marmara Pendik Training and Research Hospital. Because of the retrospective design, the requirement for informed consent was waived by the Ethics Committee.

Acknowledgment

Not applicable.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at <https://doi.org/10.31083/CEOG45125>.

References

- [1] Jenner B. Hypertension in pregnancy and pre-eclampsia. *Medicine*. 2024; 52: 620–626. <https://doi.org/10.1016/j.mpmed.2024.07.011>.
- [2] Braunthal S, Brateanu A. Hypertension in pregnancy: Pathophysiology and treatment. *SAGE Open Medicine*. 2019; 7: 2050312119843700. <https://doi.org/10.1177/2050312119843700>.
- [3] Dimitriadis E, Rolnik DL, Zhou W, Estrada-Gutierrez G, Koga K, Francisco RPV, *et al.* Pre-eclampsia [published erratum in *Nature Reviews. Disease Primers*. 2023; 9: 35]. *Nature Reviews. Disease Primers*. 2023; 9: 8. <https://doi.org/10.1038/s41572-023-00417-6>.
- [4] Brown MA, Roberts L, Hoffman A, Henry A, Mangos G, O'Sullivan A, *et al.* Recognizing Cardiovascular Risk After Preeclampsia: The P4 Study. *Journal of the American Heart Association*. 2020; 9: e018604. <https://doi.org/10.1161/JAHA.120.018604>.
- [5] Nathan HL, Seed PT, Hezelgrave NL, De Greeff A, Lawley E, Anthony J, *et al.* Early warning system hypertension thresholds to predict adverse outcomes in pre-eclampsia: A prospective cohort study. *Pregnancy Hypertension*. 2018; 12: 183–188. <https://doi.org/10.1016/j.pregphy.2017.11.003>.
- [6] Tlaye KG, Endalfer ML, Kassaw MW, Gebremedhin MM, Ayalem YA. Preeclampsia management modalities and perinatal death: a retrospective study in Woldia general hospital. *BMC Pregnancy and Childbirth*. 2020; 20: 205. <https://doi.org/10.1186/s12884-020-02909-9>.
- [7] Panda S, Das R, Sharma N, Das A, Deb P, Singh K. Maternal and Perinatal Outcomes in Hypertensive Disorders of Pregnancy and Factors Influencing It: A Prospective Hospital-Based Study in Northeast India. *Cureus*. 2021; 13: e13982. <https://doi.org/10.7759/cureus.13982>.
- [8] WHO Recommendations for Prevention and Treatment of Pre-Eclampsia and Eclampsia. World Health Organization: Geneva. 2011.
- [9] Kepley JM, Bates K, Mohiuddin SS. *Physiology, Maternal Changes*. StatPearls Publishing: Treasure Island (FL). 2025.
- [10] Zia A, Sahebdel F, Er-Reguyeg Y, Desjarlais M, Mars JC, Lodygensky GA, *et al.* Role of Cellular Senescence in IUGR: Impact on Fetal Morbidity and Development. *Cells*. 2025; 14: 1097. <https://doi.org/10.3390/cells14141097>.
- [11] Bilo G, Dolan E, O'Brien E, Facchetti R, Soranna D, Zambon A, *et al.* The impact of systolic and diastolic blood pressure variability on mortality is age dependent: Data from the Dublin Outcome Study. *European Journal of Preventive Cardiology*. 2020; 27: 355–364. <https://doi.org/10.1177/2047487319872572>.
- [12] SPRINT Research Group, Wright JT, Jr, Williamson JD, Whelton PK, Snyder JK, Sink KM, *et al.* A Randomized Trial of Intensive versus Standard Blood-Pressure Control. *The New England Journal of Medicine*. 2015; 373: 2103–2116. <https://doi.org/10.1056/NEJMoa1511939>.
- [13] Jälmy M, Edvinsson C, Lykou D, Karampas G, Erlandsson L, Hansson SR, *et al.* Long-term microvascular and blood pressure dysregulation after Preeclampsia. *Hypertension Research*. 2025; 48: 1972–1982. <https://doi.org/10.1038/s41440-025-02176-1>.
- [14] Niu X, Li Z, Kang Y, Li M, Zhong R, Tian J. Effect of different blood pressure levels on short-term outcomes in hospitalized heart failure patients. *International Journal of Cardiology*. Cardiovascular Risk and Prevention. 2023; 16: 200169. <https://doi.org/10.1016/j.ijcrp.2023.200169>.
- [15] Tsujimoto T, Kajio H. Low diastolic blood pressure and adverse outcomes in heart failure with preserved ejection fraction. *International Journal of Cardiology*. 2018; 263: 69–74. <https://doi.org/10.1016/j.ijcard.2018.04.031>.
- [16] Sobieraj P, Lewandowski J, Siński M, Gaciong Z. Low on-treatment diastolic blood pressure and cardiovascular outcome: A post-hoc analysis using NHLBI SPRINT Research Materials. *Scientific Reports*. 2019; 9: 13070. <https://doi.org/10.1038/s41598-019-49557-4>.
- [17] Sahutoglu T, Sakaci T. Diastolic blood pressure variability in 24 hour-ABPM and outcomes of chronic kidney disease. *Clinical Nephrology*. 2018; 90: 46–52. <https://doi.org/10.5414/CN109311>.
- [18] Gunnarsdottir J, Akhter T, Högberg U, Cnattingius S, Wikström AK. Elevated diastolic blood pressure until mid-gestation is associated with preeclampsia and small-for-gestational-age birth: a population-based register study. *BMC Pregnancy and Childbirth*. 2019; 19: 186. <https://doi.org/10.1186/s12884-019-2319-2>.
- [19] Scantlebury DC, Kattah AG, Weissgerber TL, Agarwal S, Mielke MM, Weaver AL, *et al.* Impact of a History of Hypertension in Pregnancy on Later Diagnosis of Atrial Fibrillation. *Journal of the American Heart Association*. 2018; 7: e007584. <https://doi.org/10.1161/JAHA.117.007584>.

- [20] Uzakova M, Babajanova GS, Uzokov JK. Prevalence and characteristics of arrhythmias during the pregnancy. *Europace*. 2023; 25: euad122.767. <https://doi.org/10.1093/europace/eua122.767>.
- [21] Harding BN, Norby FL, Heckbert SR, McKnight B, Psaty BM, Soliman EZ, *et al.* Longitudinal Measures of Blood Pressure and Subclinical Atrial Arrhythmias: The MESA and the ARIC Study. *Journal of the American Heart Association*. 2021; 10: e020260. <https://doi.org/10.1161/JAHA.120.020260>.
- [22] Lip GYH, Coca A, Kahan T, Boriani G, Manolis AS, Olsen MH, *et al.* Hypertension and cardiac arrhythmias: a consensus document from the European Heart Rhythm Association (EHRA) and ESC Council on Hypertension, endorsed by the Heart Rhythm Society (HRS), Asia-Pacific Heart Rhythm Society (APHRs) and Sociedad Latinoamericana de Estimulación Cardíaca y Electrofisiología (SOLEACE). *Europace*. 2017; 19: 891–911. <https://doi.org/10.1093/europace/eux091>.
- [23] Conti E, Cascio ND, Paluan P, Racca G, Longhitano Y, Savio G, *et al.* Pregnancy Arrhythmias: Management in the Emergency Department and Critical Care. *Journal of Clinical Medicine*. 2024; 13: 1095. <https://doi.org/10.3390/jcm13041095>.
- [24] Leon LJ, McCarthy FP, Direk K, Gonzalez-Izquierdo A, Prieto-Merino D, Casas JP, *et al.* Preeclampsia and Cardiovascular Disease in a Large UK Pregnancy Cohort of Linked Electronic Health Records: A CALIBER Study. *Circulation*. 2019; 140: 1050–1060. <https://doi.org/10.1161/CIRCULATIONAHA.118.038080>.
- [25] Chen Y, Huang L, Zhang J, Guo N. Serum sFlt-1/PlGF ratio combined with ANXA2 correlates with adverse pregnancy outcomes in women with suspected preeclampsia. *The Journal of Maternal-Fetal & Neonatal Medicine*. 2025; 38: 2506008. <https://doi.org/10.1080/14767058.2025.2506008>.
- [26] Chatzakis C, Papavasiliou D, Mansukhani T, Nicolaides KH, Charakida M. Maternal vascular-placental axis in the third trimester in women with gestational diabetes mellitus, hypertensive disorders, and unaffected pregnancies. *American Journal of Obstetrics and Gynecology*. 2025; 232: 489.e1–489.e11. <https://doi.org/10.1016/j.ajog.2024.08.045>.
- [27] Magee LA, von Dadelszen P, Singer J, Lee T, Rey E, Ross S, *et al.* The CHIPS Randomized Controlled Trial (Control of Hypertension in Pregnancy Study): Is Severe Hypertension Just an Elevated Blood Pressure? *Hypertension*. 2016; 68: 1153–1159. <https://doi.org/10.1161/HYPERTENSIONAHA.116.07862>.
- [28] Eraslan Sahin M, Sahin E, Kirlangic MM. Third-trimester persistent maternal hypotension effects on late-onset small for gestational age and adverse perinatal outcomes. *PLoS ONE*. 2023; 18: e0286900. <https://doi.org/10.1371/journal.pone.0286900>.
- [29] Ueda A, Hasegawa M, Matsumura N, Sato H, Kosaka K, Abiko K, *et al.* Lower systolic blood pressure levels in early pregnancy are associated with a decreased risk of early-onset superimposed preeclampsia in women with chronic hypertension: a multicenter retrospective study. *Hypertension Research*. 2022; 45: 135–145. <https://doi.org/10.1038/s41440-021-00763-6>.
- [30] Ardissino M, Reddy RK, Slob EAW, Griffiths J, Girling J, Ng FS. Maternal hypertensive traits and adverse outcome in pregnancy: a Mendelian randomization study. *Journal of Hypertension*. 2023; 41: 1438–1445. <https://doi.org/10.1097/HJH.0000000000003486>.
- [31] Bongers-Karmaoui MN, Wiertsema CJ, Mulders AGMGJ, Helbing WA, Hirsch A, Roest AAW, *et al.* Gestational hypertensive disorders and blood pressure and childhood cardiac outcomes: A prospective cohort study. *BJOG: an International Journal of Obstetrics and Gynaecology*. 2023; 130: 1226–1237. <https://doi.org/10.1111/1471-0528.17468>.