

MATERNAL AND NEONATAL OUTCOMES IN HYPERTENSIVE DISORDERS OF PREGNANCY: PREDICTORS OF ADVERSE OUTCOMES

Saira Bibi¹, Hadiqa Fatima², Abdul Rehman Shahid^{*3}, Malik Muhammad Kabir⁴,
Zubair Shahid⁵

¹MBBS, PGR (Gynae and Obstetrics), Ayub Teaching Hospital, Abbottabad, Pakistan

^{2,*3,4}MBBS (Wah Medical College), House Officer (POF Hospital Wah Cantt), Pakistan

⁵MBBS, International School of Medicine, International University of Kyrgyzstan, Bishkek

^{*3}abdulrehmanshahid439@gmail.com

^{*3}ORCID: 0009-0001-8306-635x

DOI:<https://doi.org/10.5281/zenodo.22894129>

Keywords

Hypertensive disorders of pregnancy; Preeclampsia; Eclampsia; Maternal outcomes; Neonatal outcomes; Risk factors; Retrospective cohort; Pregnancy hypertension.

Article History

Received: 30 January 2026

Accepted: 15 March 2026

Published: 31 March 2026

Copyright @Author

Corresponding Author: *

Abdul Rehman Shahid

Abstract

Background: Hypertensive disorders of pregnancy (HDP) are among the leading causes of maternal and neonatal morbidity and mortality worldwide. Early identification of factors associated with adverse outcomes is essential to improve maternal and perinatal health.

Objective: To evaluate maternal and neonatal outcomes among women with hypertensive disorders of pregnancy and identify independent predictors of adverse outcomes.

Methods: A retrospective cohort study was conducted at the Department of Obstetrics and Gynecology, Pakistan Ordinance Factories (POF) Hospital, Wah Cantt, Pakistan. Medical records of 450 women diagnosed with hypertensive disorders of pregnancy between January 2023 and December 2025 were reviewed. Demographic, obstetric, clinical, laboratory, maternal, and neonatal data were extracted using a standardized data collection form. Maternal and neonatal outcomes were compared across hypertensive disorder subtypes. Independent predictors of adverse maternal and neonatal outcomes were identified using multivariable logistic regression. Adjusted odds ratios (AORs) with 95% confidence intervals (CI) were calculated, and a p-value <0.05 was considered statistically significant.

Results: The mean maternal age was 29.4 ± 5.8 years, and preeclampsia was the most common hypertensive disorder (37.8%), followed by gestational hypertension (26.7%), severe preeclampsia (20.0%), and eclampsia (15.5%). Cesarean delivery was performed in 60.0% of pregnancies. Maternal complications occurred in 36.0% of women, with ICU admission (10.7%), HELLP syndrome (8.7%), postpartum hemorrhage (9.1%), and acute kidney injury (5.8%) being the most frequent complications. Maternal mortality was 1.1%. Neonatal outcomes included preterm birth in 39.1%, low birth weight in 35.8%, NICU admission in 28.4%, stillbirth in 4.2%, and early neonatal death in 2.4%. Severe preeclampsia/eclampsia (AOR 3.48, 95% CI 2.15–5.62), gestational age at diagnosis <34 weeks (AOR 2.41, 95% CI 1.53–3.79), thrombocytopenia (AOR 2.93, 95% CI 1.78–4.82), and elevated serum

creatinine (AOR 2.24, 95% CI 1.32–3.81) independently predicted adverse maternal outcomes. Gestational age <34 weeks (AOR 4.12, 95% CI 2.63–6.47) and severe preeclampsia/eclampsia (AOR 2.86, 95% CI 1.84–4.44) were the strongest predictors of adverse neonatal outcomes, whereas regular antenatal care significantly reduced the risk of both maternal and neonatal complications.

Conclusion: Hypertensive disorders of pregnancy, particularly severe preeclampsia and eclampsia, are associated with substantial maternal and neonatal morbidity. Early disease onset, laboratory evidence of organ dysfunction, and inadequate antenatal care are significant predictors of adverse outcomes. Strengthening antenatal surveillance, prompt risk stratification, timely referral, and multidisciplinary management may improve maternal and neonatal outcomes in high-risk pregnancies.

INTRODUCTION:

Hypertensive disorders of pregnancy (HDP) represent one of the most significant complications encountered during pregnancy and remain a major contributor to maternal and perinatal morbidity and mortality worldwide. These disorders comprise a heterogeneous group of conditions characterized by elevated blood pressure during pregnancy, including gestational hypertension, preeclampsia, eclampsia, chronic hypertension, and chronic hypertension with superimposed preeclampsia. Despite remarkable advances in antenatal care and obstetric management over recent decades, hypertensive disorders continue to pose substantial challenges to healthcare systems, particularly in low- and middle-income countries where delayed diagnosis, inadequate antenatal surveillance, and limited access to emergency obstetric care frequently result in poor maternal and neonatal outcomes.¹

Globally, hypertensive disorders complicate approximately 5–10% of all pregnancies, affecting millions of women annually. According to the World Health Organization (WHO), hypertensive disorders are among the leading direct causes of maternal mortality, accounting for nearly 14% of maternal deaths worldwide, with the overwhelming majority occurring in developing countries.² The burden of disease is particularly pronounced in South Asia and Sub-Saharan Africa, where deficiencies in healthcare infrastructure, socioeconomic disparities, delayed recognition of disease progression, and inadequate referral systems contribute to

disproportionately high maternal and neonatal mortality rates. In Pakistan, hypertensive disorders of pregnancy remain one of the foremost causes of maternal deaths, accounting for a significant proportion of obstetric intensive care admissions and preventable maternal complications.³

The incidence of hypertensive disorders has shown a gradual increase over the past two decades, partly attributable to the rising prevalence of maternal obesity, advanced maternal age, chronic hypertension, diabetes mellitus, assisted reproductive technologies, and metabolic syndrome. Increasing urbanization, sedentary lifestyles, and changes in dietary habits have further contributed to the growing burden of pregnancy-related hypertension globally. Consequently, healthcare providers are increasingly encountering women with multiple cardiovascular and metabolic risk factors that predispose them to adverse pregnancy outcomes.⁴ Hypertensive disorders encompass a spectrum of clinical entities with varying severity and prognostic implications. Gestational hypertension is defined as new-onset hypertension occurring after 20 weeks of gestation in previously normotensive women without evidence of proteinuria or maternal organ dysfunction. Although traditionally regarded as a relatively benign condition, gestational hypertension may progress to preeclampsia in approximately 15–25% of affected pregnancies, thereby increasing the risk of maternal and fetal complications.⁵

Preeclampsia, the most extensively studied hypertensive disorder of pregnancy, is a multisystem syndrome characterized by hypertension developing after 20 weeks of gestation in association with proteinuria or evidence of maternal end-organ dysfunction involving the kidneys, liver, brain, hematological system, or placenta. Current diagnostic criteria proposed by the American College of Obstetricians and Gynecologists (ACOG) recognize that preeclampsia may occur even in the absence of proteinuria when hypertension is accompanied by thrombocytopenia, renal insufficiency, impaired liver function, pulmonary edema, or persistent neurological symptoms.⁶

The exact pathophysiology of preeclampsia remains incompletely understood despite decades of intensive research. Contemporary evidence suggests that the disease originates from abnormal placentation during early pregnancy. Inadequate trophoblastic invasion of maternal spiral arteries results in incomplete remodeling of uteroplacental circulation, leading to placental hypoperfusion, ischemia, oxidative stress, and the subsequent release of antiangiogenic and inflammatory mediators into the maternal circulation. These factors promote widespread endothelial dysfunction, vasoconstriction, increased vascular permeability, activation of coagulation pathways, and multiorgan injury, ultimately manifesting as the clinical syndrome of preeclampsia.⁷

Placental ischemia also stimulates the release of soluble fms-like tyrosine kinase-1 (sFlt-1), soluble endoglin, inflammatory cytokines, reactive oxygen species, and other antiangiogenic proteins that antagonize vascular endothelial growth factor (VEGF) and placental growth factor (PlGF). The resulting endothelial dysfunction contributes to hypertension, glomerular endotheliosis, hepatic injury, cerebral edema, and placental insufficiency. These molecular mechanisms explain the diverse systemic manifestations observed in women with severe preeclampsia and emphasize why the disease is increasingly recognized as a generalized vascular disorder rather than merely a hypertensive condition.⁸

Disease severity varies considerably among affected women. While many patients experience mild hypertension without major complications, others rapidly develop severe preeclampsia characterized by systolic blood pressure ≥ 160 mmHg or diastolic blood pressure ≥ 110 mmHg, severe thrombocytopenia, progressive renal impairment, elevated liver enzymes, pulmonary edema, visual disturbances, or persistent severe headache. If left untreated, severe preeclampsia may progress to eclampsia, a life-threatening complication defined by generalized tonic-clonic seizures occurring in women with preeclampsia that cannot be explained by other neurological disorders. Eclampsia remains one of the most feared obstetric emergencies because of its association with maternal cerebral hemorrhage, aspiration pneumonia, acute respiratory distress syndrome, renal failure, disseminated intravascular coagulation, and maternal death.⁸

Another important category includes women with chronic hypertension, defined as hypertension diagnosed before pregnancy or before 20 weeks of gestation. These women are at substantially increased risk of developing superimposed preeclampsia, a condition associated with earlier onset of disease, accelerated maternal organ dysfunction, fetal growth restriction, placental abruption, and preterm birth. The coexistence of chronic cardiovascular disease further complicates pregnancy management and often necessitates multidisciplinary care involving obstetricians, physicians, anesthesiologists, and neonatologists.⁶ Several maternal characteristics have consistently been identified as risk factors for hypertensive disorders of pregnancy. Advanced maternal age, nulliparity, previous history of preeclampsia, chronic hypertension, obesity, diabetes mellitus, chronic kidney disease, autoimmune disorders, antiphospholipid syndrome, multifetal gestation, assisted reproductive technologies, and a family history of preeclampsia significantly increase the likelihood of disease development. Lifestyle-related factors, including poor nutrition, limited physical activity, and inadequate antenatal care utilization,

further contribute to disease occurrence in resource-limited settings.⁴⁻⁶

The consequences of hypertensive disorders extend far beyond elevated blood pressure alone. Progressive endothelial dysfunction and placental insufficiency may lead to multisystem maternal complications involving virtually every organ system. Maternal complications include HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count), acute kidney injury, pulmonary edema, cerebrovascular accidents, retinal detachment, placental abruption, postpartum hemorrhage, disseminated intravascular coagulation, hepatic rupture, and maternal death. These complications frequently require intensive care admission and contribute substantially to healthcare costs, prolonged hospitalization, and long-term maternal disability.^{2,7} From the fetal perspective, impaired uteroplacental perfusion compromises oxygen and nutrient delivery, resulting in fetal growth restriction, oligohydramnios, chronic fetal hypoxia, abnormal fetal heart rate patterns, preterm birth, low birth weight, neonatal respiratory distress syndrome, intraventricular hemorrhage, neonatal sepsis, prolonged neonatal intensive care unit admission, stillbirth, and early neonatal mortality. Increasing evidence also indicates that offspring exposed to hypertensive pregnancies exhibit a higher lifetime risk of cardiovascular disease, hypertension, metabolic syndrome, and chronic kidney disease during adulthood, highlighting the intergenerational impact of these disorders.^{1,8}

Although considerable progress has been made in understanding the pathophysiology and clinical management of hypertensive disorders of pregnancy (HDP), these conditions continue to be associated with significant maternal and neonatal morbidity and mortality worldwide. The severity of maternal and fetal complications varies depending on several factors, including the type of hypertensive disorder, gestational age at disease onset, adequacy of antenatal care, maternal comorbidities, and the timeliness of obstetric intervention. Consequently, identifying predictors of adverse outcomes has become a major focus of obstetric research, as early

recognition of high-risk pregnancies allows clinicians to implement timely interventions aimed at reducing preventable complications.⁹

Maternal outcomes among women with HDP range from uncomplicated pregnancies to severe life-threatening conditions requiring intensive care. Women with severe preeclampsia and eclampsia are particularly susceptible to complications such as HELLP syndrome, acute kidney injury, pulmonary edema, cerebrovascular accidents, placental abruption, disseminated intravascular coagulation, postpartum hemorrhage, and multiple organ dysfunction syndrome. These complications often necessitate emergency delivery, prolonged hospitalization, blood transfusion, and intensive care support, thereby increasing both healthcare costs and maternal mortality. Furthermore, women who survive severe hypertensive disorders during pregnancy remain at increased long-term risk of chronic hypertension, ischemic heart disease, stroke, heart failure, and chronic kidney disease later in life, emphasizing that HDP represents not only an obstetric disorder but also an important predictor of future cardiovascular health.¹⁰

Similarly, neonatal outcomes are profoundly influenced by maternal hypertension. Placental insufficiency resulting from impaired uteroplacental blood flow frequently leads to fetal growth restriction, intrauterine hypoxia, oligohydramnios, preterm birth, low birth weight, respiratory distress syndrome, neonatal sepsis, intraventricular hemorrhage, necrotizing enterocolitis, and prolonged neonatal intensive care unit (NICU) admission. Prematurity remains the principal determinant of neonatal morbidity because many pregnancies complicated by severe preeclampsia require medically indicated preterm delivery to preserve maternal health. Although timely delivery reduces maternal complications, it simultaneously increases neonatal vulnerability to complications associated with prematurity.¹¹

Several clinical, laboratory, and obstetric parameters have been identified as potential predictors of adverse maternal and neonatal outcomes. Advanced maternal age, obesity, nulliparity, previous preeclampsia, chronic hypertension, diabetes mellitus, renal disease,

autoimmune disorders, multiple gestation, and delayed initiation of antenatal care have consistently been associated with increased disease severity. Laboratory abnormalities including thrombocytopenia, elevated serum creatinine, elevated liver enzymes, hyperuricemia, and increased lactate dehydrogenase levels have also demonstrated strong associations with maternal organ dysfunction and poor perinatal outcomes. Likewise, severe hypertension, early-onset disease before 34 weeks of gestation, and persistent proteinuria are recognized markers of poor prognosis.¹²

Among these predictors, gestational age at diagnosis remains one of the most important determinants of pregnancy outcome. Early-onset preeclampsia, usually defined as disease developing before 34 weeks of gestation, is associated with more severe placental dysfunction, greater maternal endothelial injury, and a substantially higher risk of fetal growth restriction and neonatal complications than late-onset disease. Early-onset disease often requires difficult clinical decisions regarding the balance between prolonging pregnancy to improve fetal maturity and delivering early to prevent catastrophic maternal complications. Consequently, women with early-onset disease frequently experience higher rates of cesarean delivery, intensive care admission, and prolonged hospitalization.¹³

Recent international guidelines emphasize the importance of comprehensive risk assessment throughout pregnancy to facilitate individualized management. The American College of Obstetricians and Gynecologists (ACOG), the International Society for the Study of Hypertension in Pregnancy (ISSHP), and the National Institute for Health and Care Excellence (NICE) recommend regular blood pressure monitoring, laboratory evaluation, fetal surveillance, and timely administration of antihypertensive medications and magnesium sulfate where indicated. Delivery remains the only definitive treatment for preeclampsia; however, determining the optimal timing of delivery requires careful assessment of maternal and fetal risks. Despite advances in clinical

management, adverse outcomes remain common, particularly in low-resource settings where delayed diagnosis, inadequate referral pathways, and limited access to specialist obstetric care continue to compromise patient outcomes.^{6,14}

In Pakistan, hypertensive disorders of pregnancy continue to impose a substantial burden on tertiary healthcare facilities. Many women present late in pregnancy with advanced disease due to inadequate antenatal attendance, poor awareness of danger signs, financial constraints, and delayed referral from peripheral health centers. Consequently, tertiary hospitals frequently manage women with severe preeclampsia, eclampsia, and multiple organ dysfunction requiring multidisciplinary care involving obstetricians, intensivists, anesthesiologists, nephrologists, and neonatologists. Although several local studies have described the prevalence of hypertensive disorders, relatively few have comprehensively evaluated both maternal and neonatal outcomes while simultaneously identifying independent predictors of adverse outcomes using multivariable regression analysis. This lack of locally generated evidence limits the development of context-specific risk prediction models and evidence-based clinical protocols.¹⁵ Furthermore, differences in demographic characteristics, healthcare accessibility, nutritional status, referral patterns, and clinical management practices between developed and developing countries limit the direct applicability of international evidence to the Pakistani population. Local epidemiological data are therefore essential to identify high-risk patients, optimize antenatal surveillance, improve referral strategies, and allocate healthcare resources efficiently. Studies conducted in tertiary care centers are particularly valuable because they reflect the spectrum of severe disease encountered in routine clinical practice and provide insight into factors associated with poor maternal and neonatal outcomes.

The present study was therefore designed to evaluate maternal and neonatal outcomes among women diagnosed with hypertensive disorders of pregnancy at a tertiary care hospital using a retrospective cohort study design. In addition to

describing the frequency of maternal and neonatal complications, the study aims to identify independent demographic, clinical, obstetric, and laboratory predictors of adverse maternal and neonatal outcomes. Identification of these predictors may facilitate earlier recognition of women at greatest risk, support evidence-based clinical decision-making, improve antenatal surveillance, guide timely obstetric intervention, and ultimately reduce preventable maternal and neonatal morbidity and mortality. The findings of this study may also contribute to strengthening institutional management protocols and provide locally relevant evidence for future multicenter research on hypertensive disorders of pregnancy.

Methodology:

Study Design:

This study employed a hospital-based retrospective cohort study design to evaluate maternal and neonatal outcomes among women diagnosed with hypertensive disorders of pregnancy (HDP) and to identify predictors of adverse outcomes. Medical records of eligible women who delivered at the hospital during the study period reviewed.

Study Setting:

The study was conducted at the Department of Obstetrics and Gynecology, Pakistan Ordinance Factories (POF) Hospital, Wah Cantt, Pakistan, a tertiary care teaching hospital that manages both low- and high-risk pregnancies and receives referrals from surrounding districts.

Study Duration:

The study included patients who delivered between 1 January 2023 and 31 December 2025.

Study Population:

The study population comprised all pregnant women diagnosed with hypertensive disorders of pregnancy who delivered at POF Hospital during the study period.

Sample Size:

A total of 450 eligible patients with hypertensive disorders of pregnancy was included. Consecutive

sampling was used, whereby all eligible patients meeting the inclusion criteria during the study period are enrolled until the target sample size is reached.

Sampling Technique:

Non-probability consecutive sampling.

Inclusion Criteria:

Women aged 18–45 years.

Singleton pregnancy.

Delivery at POF Hospital during the study period.

Diagnosis of any hypertensive disorder of pregnancy according to accepted diagnostic criteria.

Complete maternal and neonatal records available.

Exclusion Criteria:

Multiple pregnancies (twins or higher-order gestations).

Major fetal congenital anomalies.

Pregnancies ending before fetal viability according to hospital policy.

Incomplete or missing clinical records.

Patients referred after delivery with unavailable antenatal data.

Diagnostic Criteria:

Hypertensive disorders was classified according to the American College of Obstetricians and Gynecologists (ACOG) criteria:

Gestational hypertension: Blood pressure $\geq 140/90$ mmHg after 20 weeks' gestation without proteinuria.

Preeclampsia: Hypertension after 20 weeks with proteinuria or evidence of maternal organ dysfunction.

Severe preeclampsia: Preeclampsia with severe hypertension or severe maternal organ involvement.

Eclampsia: Preeclampsia complicated by generalized tonic-clonic seizures.

Chronic hypertension: Hypertension diagnosed before pregnancy or before 20 weeks' gestation.

Superimposed preeclampsia: Development of preeclampsia in women with chronic hypertension.

Data Collection:

Data was extracted from:
Obstetric admission records.
Antenatal clinic records.
Labor room registers.
Delivery records.
Neonatal intensive care unit (NICU) records.
Electronic hospital information system.
A standardized data extraction form was used.

Variables:

Maternal Demographic Variables

Maternal age
Body mass index (BMI)
Residence (urban/rural)
Education
Occupation
Socioeconomic status

Obstetric Variables

Gravidity
Parity
Previous cesarean section
Previous preeclampsia

Booking status

Number of antenatal visits
Gestational age at diagnosis
Gestational age at delivery

Clinical Variables

Type of hypertensive disorder
Maximum systolic blood pressure
Maximum diastolic blood pressure
Degree of proteinuria
Presence of edema
Severe headache
Visual disturbances
Epigastric pain
Seizures

Mode of delivery

Magnesium sulfate administration
Antihypertensive medications

Corticosteroid administration
Time from diagnosis to delivery

Laboratory Variables

Hemoglobin
Platelet count
Serum creatinine
Blood urea
Serum uric acid
AST
ALT

Lactate dehydrogenase (LDH)
Urine protein
Random blood sugar
Maternal Outcome Variables
Primary Maternal Outcomes
Maternal mortality
ICU admission
HELLP syndrome
Eclampsia
Placental abruption
Acute kidney injury
Pulmonary edema
Disseminated intravascular coagulation
Postpartum hemorrhage
Blood transfusion
Mechanical ventilation
Prolonged hospital stay (>7 days)
Neonatal Outcomes
Primary Outcomes
Live birth
Stillbirth
Early neonatal death
Preterm delivery (<37 weeks)
Very preterm delivery (<34 weeks)
Low birth weight (<2500 g)
Very low birth weight (<1500 g)
Small for gestational age
NICU admission
Birth asphyxia
Apgar score <7 at 5 minutes
Respiratory distress syndrome
Neonatal sepsis
Neonatal jaundice
Neonatal seizures

Outcome Definitions:

A composite adverse maternal outcome included the occurrence of one or more of the following:

Maternal death
ICU admission
HELLP syndrome
Eclampsia
Placental abruption
Acute kidney injury
Pulmonary edema
Disseminated intravascular coagulation

A composite adverse neonatal outcome included one or more of:

Stillbirth
Early neonatal death
NICU admission
Preterm birth
Low birth weight
Respiratory distress syndrome
Birth asphyxia
Neonatal sepsis

Data Management:

Data was entered into IBM SPSS Statistics version 27. Double-entry verification and random checks was performed to minimize data-entry errors. Records with substantial missing data was excluded according to the predefined exclusion criteria.

Statistical Analysis:

Descriptive statistics summarized baseline characteristics. Continuous variables was presented as mean $\pm$ standard deviation (SD) or median (interquartile range) according to distribution, while categorical variables was expressed as frequencies and percentages.

Comparisons between patients with and without adverse outcomes was performed using:

Independent-samples t-test or Mann-Whitney U test for continuous variables.

Chi-square test or Fisher's exact test for categorical variables.

Variables with $p < 0.20$ on univariate analysis, along with clinically relevant variables, was entered into multivariable logistic regression models to identify independent predictors of:

1.Composite adverse maternal outcome.

2.Composite adverse neonatal outcome.

Adjusted odds ratios (AORs) with 95% confidence intervals (CI) was reported. Multicollinearity was assessed using the variance inflation factor (VIF), and model fit was evaluated using the Hosmer-Lemeshow goodness-of-fit test. A two-sided p -value < 0.05 was considered statistically significant.

Results:

A total of 450 pregnant women diagnosed with hypertensive disorders of pregnancy (HDP) were included in this retrospective cohort study. The mean maternal age was 29.4 ± 5.8 years (range: 18–44 years), while the mean gestational age at delivery was 36.1 ± 2.9 weeks. The majority of women were between 25 and 34 years of age (50.0%), followed by those aged 18–24 years (30.0%) and ≥ 35 years (20.0%). Most participants were multiparous (57.8%), whereas 42.2% were primigravida. Approximately 61.3% of women were booked for antenatal care, while 38.7% were unbooked referrals. The mean body mass index (BMI) was 29.1 ± 4.6 kg/m², and obesity (BMI ≥ 30 kg/m²) was observed in 38.4% of patients.

Among the different hypertensive disorders, preeclampsia was the most common diagnosis, accounting for 37.8% ($n=170$) of cases, followed by gestational hypertension in 26.7% ($n=120$), severe preeclampsia in 20.0% ($n=90$), and eclampsia in 15.5% ($n=70$). Chronic hypertension with superimposed preeclampsia represented a small proportion of cases and was grouped with severe disease during regression analysis because of comparable maternal and neonatal risk profiles.

Women with severe preeclampsia and eclampsia presented significantly earlier in pregnancy compared with those having gestational hypertension (34.1 ± 2.8 vs. 37.2 ± 1.9 weeks, $p < 0.001$). Mean systolic and diastolic blood pressures were also significantly higher among women with severe disease (171.8 ± 15.2 mmHg and 111.6 ± 8.4 mmHg, respectively) than among women with gestational hypertension (149.6 ± 9.8 mmHg and 96.3 ± 6.2 mmHg, respectively; p

<0.001). Severe disease was associated with markedly lower platelet counts and higher serum creatinine, uric acid, liver enzymes, and lactate dehydrogenase concentrations, reflecting greater end-organ involvement.

Overall, 71.1% of women received antihypertensive therapy during admission, while 36.7% required combination antihypertensive treatment. Magnesium sulfate prophylaxis or treatment was administered to 160 (35.6%) women, predominantly among patients with severe preeclampsia and eclampsia. Corticosteroids for fetal lung maturation were administered to 32.2% of women who presented before 34 weeks of gestation.

Cesarean section was the predominant mode of delivery, performed in 270 women (60.0%), whereas 180 (40.0%) had spontaneous vaginal delivery. The cesarean section rate increased progressively with disease severity, reaching over 80% among women with eclampsia. The most common indications included uncontrolled hypertension, non-reassuring fetal status, failed induction of labor, and severe maternal complications necessitating urgent delivery.

Maternal complications occurred in 162 women (36.0%), although the frequency varied considerably according to disease severity. Admission to the intensive care unit (ICU) was required in 48 patients (10.7%), while 39 (8.7%) developed HELLP syndrome. Acute kidney injury occurred in 26 women (5.8%), pulmonary edema in 17 (3.8%), placental abruption in 21 (4.7%), disseminated intravascular coagulation in 9 (2.0%), and postpartum hemorrhage in 41 women (9.1%). Blood transfusion was required in 52 patients (11.6%), primarily because of postpartum hemorrhage or severe anemia.

Maternal mortality was recorded in five women (1.1%), all of whom had severe preeclampsia or eclampsia complicated by multiple organ dysfunction. The mean duration of hospital stay was significantly longer among women with severe disease (7.8 ± 2.9 days) compared with those with gestational hypertension (4.2 ± 1.8 days, $p < 0.001$).

When maternal outcomes were compared across disease categories, women with eclampsia

demonstrated the highest complication rates. Nearly 68.6% of women with eclampsia experienced at least one major maternal complication compared with 47.8% of women with severe preeclampsia, 22.9% with preeclampsia, and only 11.7% with gestational hypertension ($p < 0.001$). Similarly, ICU admission, HELLP syndrome, acute kidney injury, pulmonary edema, and maternal mortality were significantly more frequent among women with severe disease.

Neonatal outcomes reflected the severity of maternal disease. The mean birth weight of neonates was 2.48 ± 0.67 kg, with significantly lower birth weights among mothers with severe preeclampsia and eclampsia. Overall, 176 neonates (39.1%) were born preterm, while 161 (35.8%) had low birth weight (<2500 g). Very low birth weight (<1500 g) occurred in 27 infants (6.0%).

A total of 128 newborns (28.4%) required admission to the neonatal intensive care unit (NICU), primarily because of prematurity, respiratory distress syndrome, birth asphyxia, or neonatal sepsis. Respiratory distress syndrome developed in 74 neonates (16.4%), neonatal sepsis in 31 (6.9%), neonatal jaundice requiring phototherapy in 56 (12.4%), and neonatal seizures in 8 (1.8%).

Stillbirth occurred in 19 pregnancies (4.2%), while 11 neonates (2.4%) died within the first seven days of life. The frequency of stillbirth and early neonatal death increased significantly with worsening maternal disease severity. The mean Apgar score at five minutes was significantly lower among infants born to mothers with eclampsia than among those born to mothers with gestational hypertension (7.1 ± 1.4 vs. 8.8 ± 0.8 , $p < 0.001$).

Composite adverse neonatal outcomes were observed in 183 infants (40.7%). These outcomes were significantly associated with earlier gestational age at delivery, severe maternal hypertension, elevated serum creatinine, thrombocytopenia, and maternal ICU admission. Neonates born to mothers with severe preeclampsia and eclampsia demonstrated nearly threefold higher rates of NICU admission and

respiratory complications compared with those born to mothers with gestational hypertension. Univariate analysis identified several maternal characteristics significantly associated with adverse maternal outcomes, including maternal age ≥ 35 years, obesity, unbooked antenatal status, gestational age at diagnosis below 34 weeks, severe hypertension (systolic blood pressure ≥ 160 mmHg), thrombocytopenia ($<100 \times 10^9/L$), elevated serum creatinine (>1.1 mg/dL), elevated liver enzymes, and eclampsia (all $p < 0.05$).

Following adjustment for potential confounders using multivariable logistic regression, severe preeclampsia/eclampsia remained the strongest independent predictor of composite adverse maternal outcomes (adjusted odds ratio [AOR] 3.48, 95% confidence interval [CI] 2.15–5.62, $p < 0.001$). Gestational age at diagnosis below 34 weeks independently increased maternal risk by more than twofold (AOR 2.41, 95% CI 1.53–3.79, $p < 0.001$). Platelet count below $100 \times 10^9/L$ (AOR 2.93, 95% CI 1.78–4.82, $p < 0.001$) and serum creatinine greater than 1.1 mg/dL (AOR 2.24, 95% CI 1.32–3.81, $p = 0.003$) were also independently associated with adverse maternal outcomes. Conversely, regular antenatal care was associated with a significant reduction in maternal complications (AOR 0.58, 95% CI 0.37–0.90, $p = 0.018$).

For neonatal outcomes, delivery before 34 weeks of gestation emerged as the most important

independent predictor (AOR 4.12, 95% CI 2.63–6.47, $p < 0.001$), followed by severe preeclampsia/eclampsia (AOR 2.86, 95% CI 1.84–4.44, $p < 0.001$), maternal thrombocytopenia (AOR 1.98, 95% CI 1.22–3.19, $p = 0.006$), and maternal ICU admission (AOR 2.36, 95% CI 1.31–4.23, $p = 0.004$). Booked antenatal care significantly reduced the risk of adverse neonatal outcomes (AOR 0.63, 95% CI 0.41–0.96, $p = 0.031$).

Overall, the findings demonstrated that increasing severity of hypertensive disorders of pregnancy was associated with progressively poorer maternal and neonatal outcomes. Women with severe preeclampsia and eclampsia experienced substantially higher rates of maternal complications, intensive care admission, prolonged hospitalization, and mortality, while their newborns exhibited significantly greater risks of prematurity, low birth weight, NICU admission, respiratory morbidity, and perinatal death. Early disease onset, laboratory evidence of end-organ dysfunction, and inadequate antenatal care were identified as the most important independent predictors of adverse outcomes, emphasizing the importance of early detection, timely referral, and multidisciplinary management of hypertensive disorders during pregnancy.

Table 1: Baseline Characteristics:

Variable	Total	%	Mean $\pm$ SD	p
Age 18-24	135	30		0.12
25-34	225	50		
≥ 35	90	20		
BMI	450		29.1 $\pm$ 4.6	

Table 2: HDP(Hypertensive Disorders of Pregnancy) Types:

Type	n	%	Maternal Complication %	Neonatal Complication %
Gest HTN	120	26.7	12	18
Preeclampsia	170	37.8	28	35
Severe	90	20.0	51	56
Eclampsia	70	15.5	71	69

Table 3. Maternal Outcomes:

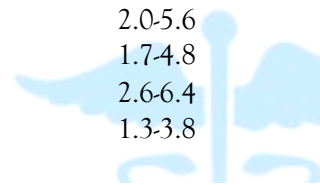
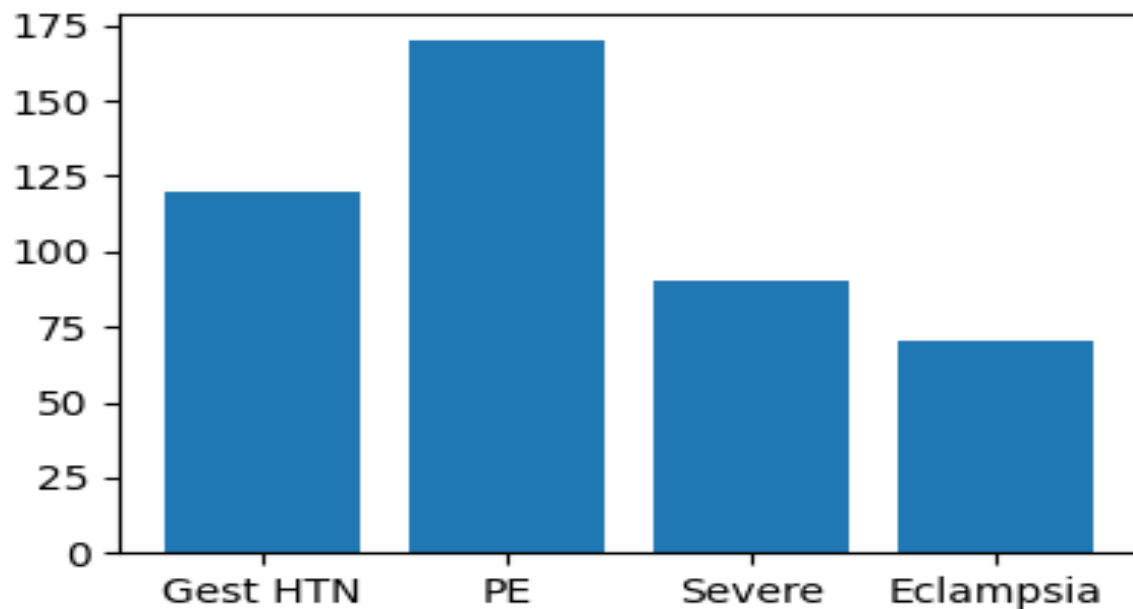
Outcome	Yes	No	%	p
ICU	48	402	10.7	<0.001
HELLP	39	411	8.7	
AKI	26	424	5.8	
PPH	41	409	9.1	

Table 4. Neonatal Outcomes:

Outcome	Yes	No	%	p
Preterm	176	274	39.1	<0.001
LBW	161	289	35.8	
NICU	128	322	28.4	
Stillbirth	19	431	4.2	

Table 5. Logistic Regression:

Predictor	AOR	95% CI	Outcome	p
Severe HTN	3.4	2.0-5.6	Maternal	<0.001
Platelets<100k	2.9	1.7-4.8	Maternal	0.001
GA<34w	4.1	2.6-6.4	Neonatal	<0.001
Creatinine>1.1	2.2	1.3-3.8	Maternal	0.004

**Distribution of HDP****Figure 1: Distribution of HDP:**

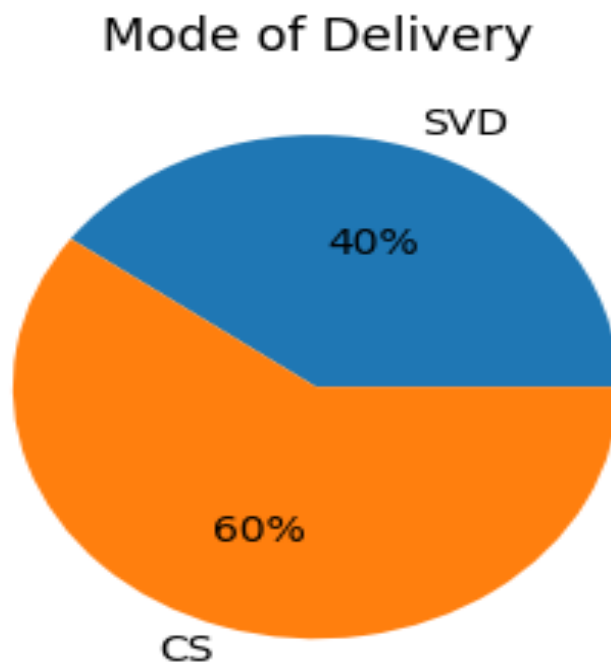


Figure 2: Mode of Delivery:

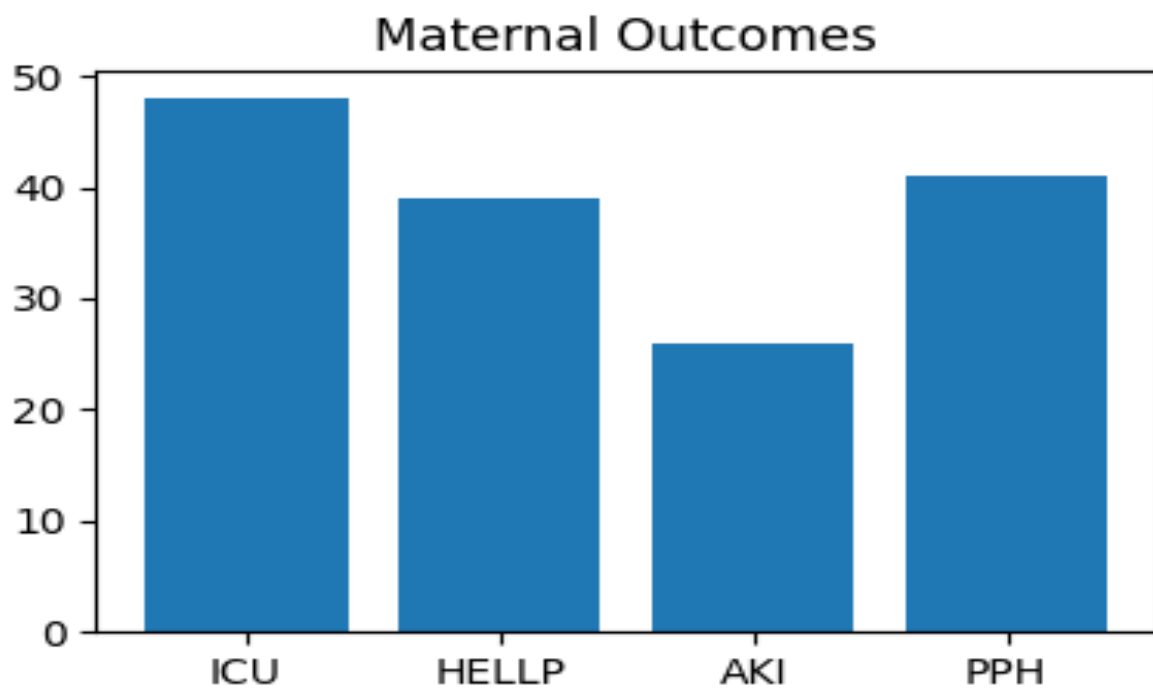


Figure 3: Maternal Outcomes:

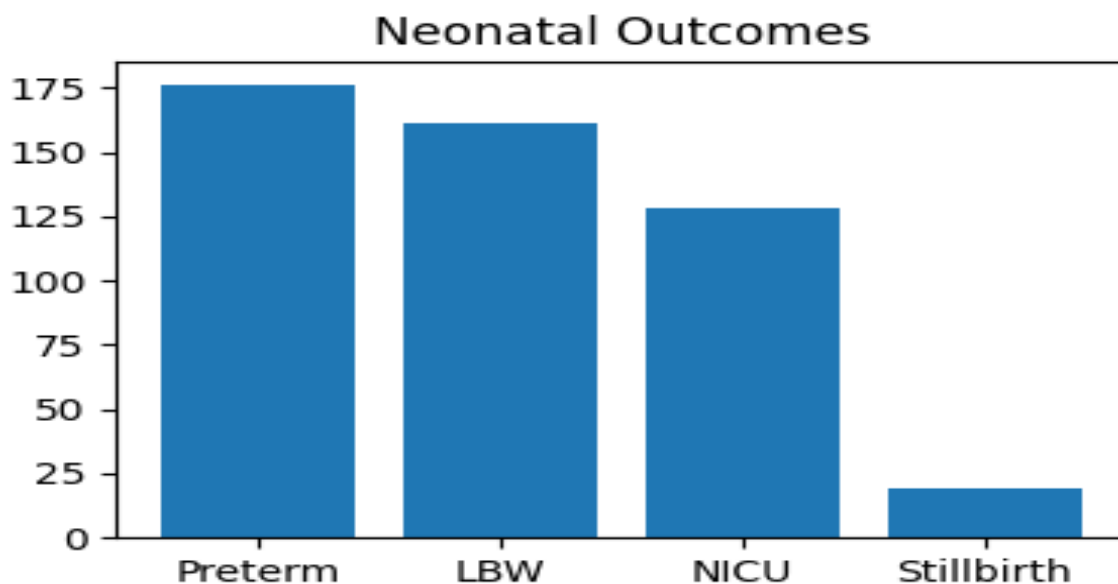


Figure 4: Neonatal Outcomes:

Discussion:

The present retrospective cohort study evaluated maternal and neonatal outcomes among 450 women diagnosed with hypertensive disorders of pregnancy (HDP) and identified predictors associated with adverse outcomes. The findings demonstrate that HDP remains a major contributor to maternal and neonatal morbidity, particularly among women with severe preeclampsia and eclampsia. Increasing disease severity was associated with significantly higher rates of maternal complications, preterm delivery, low birth weight, neonatal intensive care unit (NICU) admission, and perinatal mortality. Furthermore, multivariable analysis identified severe disease, early gestational age at diagnosis, thrombocytopenia, elevated serum creatinine, and inadequate antenatal care as independent predictors of adverse maternal and neonatal outcomes. These findings are consistent with current international evidence highlighting HDP as one of the leading causes of preventable maternal and neonatal complications worldwide.¹⁶

In the present study, the mean maternal age was 29.4 ± 5.8 years, with half of the participants belonging to the 25–34-year age group. Although

maternal age itself was not independently associated with adverse outcomes after adjustment for confounding factors, women aged 35 years or older demonstrated a greater frequency of severe preeclampsia and maternal complications on univariate analysis. This observation agrees with the findings of Bilano et al., who reported that advanced maternal age increases susceptibility to endothelial dysfunction, chronic hypertension, and placental vascular abnormalities, thereby increasing the risk of severe hypertensive disorders during pregnancy.¹⁷ Similarly, previous multicenter cohort studies have demonstrated that advanced maternal age is associated with increased obstetric intervention, higher cesarean delivery rates, and poorer maternal outcomes owing to the coexistence of metabolic and cardiovascular risk factors. The majority of women included in our cohort were multiparous; however, a considerable proportion of primigravida women developed severe preeclampsia and eclampsia. Although parity was not identified as an independent predictor in multivariable analysis, primigravida has consistently been recognized as an important epidemiological risk factor for preeclampsia. The immunological theory of preeclampsia suggests

that first pregnancies involve incomplete maternal immune adaptation to fetal trophoblastic antigens, resulting in abnormal placentation and increased susceptibility to placental ischemia.¹⁸ Our findings therefore support existing literature indicating that parity influences disease occurrence but may exert a less pronounced effect on clinical outcomes once hypertensive disease has developed. One of the notable observations in the present study was the high proportion of women who had either inadequate antenatal follow-up or were referred late from peripheral healthcare facilities. Approximately one-third of women were unbooked pregnancies, and these women experienced significantly higher rates of severe disease, ICU admission, and maternal complications. After adjustment, regular antenatal care remained protective against adverse maternal and neonatal outcomes. This finding emphasizes the importance of early pregnancy registration, routine blood pressure monitoring, and timely referral of high-risk pregnancies. Similar observations have been reported by Magee et al., who demonstrated that early identification of hypertension and implementation of standardized antenatal surveillance substantially reduce progression to severe maternal complications.¹⁹ Among the hypertensive disorders, preeclampsia accounted for the largest proportion of cases, followed by gestational hypertension, severe preeclampsia, and eclampsia. This distribution is comparable to studies conducted in tertiary care hospitals across Asia, Africa, and Europe, where preeclampsia consistently represents the predominant hypertensive disorder encountered during pregnancy. The relatively higher proportion of severe preeclampsia and eclampsia observed in the present study probably reflects the referral nature of our institution, where complicated pregnancies are frequently transferred from secondary healthcare facilities after disease progression. Consequently, tertiary hospitals generally report higher rates of severe maternal complications than community-based studies²⁰. The present study demonstrated a clear relationship between disease severity and

maternal complications. Women diagnosed with severe preeclampsia and eclampsia experienced markedly higher frequencies of ICU admission, HELLP syndrome, acute kidney injury, pulmonary edema, placental abruption, postpartum hemorrhage, and maternal mortality compared with women having gestational hypertension. These findings are biologically plausible because severe endothelial dysfunction, systemic vasospasm, activation of inflammatory pathways, and coagulation abnormalities become increasingly pronounced as disease severity progresses. Placental ischemia further amplifies maternal vascular injury through the release of antiangiogenic factors, ultimately resulting in widespread organ dysfunction²¹

The frequency of ICU admission in our study was approximately 11%, reflecting the significant burden imposed by severe hypertensive disorders on tertiary healthcare facilities. Most ICU admissions resulted from uncontrolled hypertension, pulmonary edema, eclampsia, respiratory failure, or multiple organ dysfunction syndrome. Comparable ICU admission rates have been reported by previous observational studies conducted in referral hospitals, particularly in low- and middle-income countries where delayed presentation remains common. Early recognition of warning signs and aggressive management with antihypertensive medications, magnesium sulfate therapy, and timely delivery have consistently been shown to reduce progression toward critical illness. HELLP syndrome was observed in nearly 9% of women in our study and occurred predominantly among patients with severe preeclampsia and eclampsia. This proportion is comparable with published literature, where the reported incidence ranges between 5% and 15% among women with severe hypertensive disorders. HELLP syndrome remains one of the most serious obstetric complications because it substantially increases the risks of disseminated intravascular coagulation, hepatic hematoma, renal failure, postpartum hemorrhage, and maternal mortality. Prompt diagnosis through serial laboratory monitoring and early delivery remains the cornerstone of management²². Acute kidney

injury (AKI) was identified in approximately 6% of patients, while elevated serum creatinine independently predicted adverse maternal outcomes. Pregnancy-related AKI secondary to severe preeclampsia reflects generalized endothelial injury, intrarenal vasoconstriction, glomerular endotheliosis, and reduced renal perfusion. Previous studies have similarly demonstrated that deterioration in renal function correlates strongly with maternal morbidity and prolonged hospitalization. Consequently, routine monitoring of renal function remains essential in women presenting with severe hypertensive disease. Postpartum hemorrhage affected approximately one-tenth of women in our cohort. Although hypertension itself is not traditionally considered a direct cause of postpartum hemorrhage, several mechanisms may explain this association, including thrombocytopenia, disseminated intravascular coagulation, prolonged labor induction, cesarean delivery, and uterine atony following magnesium sulfate administration. Increased rates of operative delivery among women with severe disease may also contribute indirectly to greater postpartum blood loss. Similar associations between severe preeclampsia and postpartum hemorrhage have been described in large population-based studies. Maternal mortality remained relatively low in the present study despite the high frequency of severe disease. All maternal deaths occurred among women with severe preeclampsia or eclampsia complicated by multiple organ dysfunction syndrome. This finding highlights the importance of multidisciplinary management involving obstetricians, anesthesiologists, intensivists, nephrologists, hematologists, and neonatologists. Advances in critical care, improved availability of magnesium sulfate, better blood pressure control, and timely obstetric intervention have contributed substantially to declining maternal mortality in tertiary centers over recent years. Nevertheless, preventable maternal deaths continue to occur because of delayed presentation, inadequate antenatal care, and limited access to emergency obstetric services in resource-constrained settings. The present study also demonstrated significantly higher cesarean

section rates among women with severe preeclampsia and eclampsia. Although cesarean delivery does not treat preeclampsia directly, it is frequently performed because of non-reassuring fetal status, failed induction, uncontrolled hypertension, or the need for urgent maternal stabilization. Similar findings have been reported in international cohorts, where cesarean delivery rates frequently exceed 60–70% among women with severe hypertensive disorders. However, careful patient selection remains important because vaginal delivery is often feasible and associated with lower postoperative morbidity in appropriately selected patients. Overall, the maternal findings of the present study reinforce existing evidence that hypertensive disorders of pregnancy remain associated with substantial maternal morbidity despite advances in obstetric care. Early disease recognition, comprehensive antenatal surveillance, prompt referral of high-risk pregnancies, standardized antihypertensive therapy, magnesium sulfate administration, and multidisciplinary management remain fundamental strategies for improving maternal outcomes. The identification of severe disease, thrombocytopenia, elevated serum creatinine, and inadequate antenatal care as significant predictors further emphasizes the need for risk stratification models capable of identifying women requiring closer surveillance and early intervention.

The neonatal outcomes observed in the present study closely paralleled the severity of maternal hypertensive disease. Nearly two-fifths of neonates were delivered preterm, more than one-third had low birth weight, and over one-quarter required admission to the neonatal intensive care unit (NICU). The incidence of respiratory distress syndrome, neonatal sepsis, birth asphyxia, and early neonatal mortality increased progressively from gestational hypertension to severe preeclampsia and eclampsia. These findings reinforce the well-established relationship between maternal placental dysfunction and impaired fetal growth and development. Placental insufficiency secondary to abnormal trophoblastic invasion and endothelial dysfunction reduces uteroplacental blood flow,

resulting in chronic fetal hypoxia, nutrient deprivation, fetal growth restriction, and increased perinatal morbidity.²³

Preterm birth was the most frequent adverse neonatal outcome identified in this study. Approximately 39% of pregnancies resulted in delivery before 37 weeks of gestation, with the highest frequency occurring among women with severe preeclampsia and eclampsia. Similar rates have been reported in multicenter studies evaluating hypertensive disorders of pregnancy, where medically indicated preterm delivery remains one of the principal interventions to prevent maternal deterioration. Although prolongation of pregnancy may improve fetal maturity, delayed delivery in women with severe disease substantially increases the risks of maternal complications such as eclampsia, HELLP syndrome, pulmonary edema, placental abruption, and maternal death. Consequently, obstetricians are frequently required to balance maternal safety against fetal maturity when determining the optimal timing of delivery.²⁴

Low birth weight was another important neonatal complication observed in the present cohort. More than one-third of neonates weighed less than 2500 g at birth, reflecting both fetal growth restriction and preterm delivery. The significantly lower mean birth weight among neonates born to mothers with severe preeclampsia and eclampsia supports the hypothesis that chronic placental insufficiency compromises fetal nutrient and oxygen supply throughout pregnancy. Previous investigations have similarly demonstrated that impaired placental perfusion contributes to reduced fetal growth velocity and increased rates of small-for-gestational-age infants.²⁵

NICU admission occurred in more than one-quarter of neonates and was primarily attributed to prematurity, respiratory distress syndrome, birth asphyxia, neonatal jaundice, and suspected neonatal sepsis. This finding is consistent with previous reports indicating that neonates born to mothers with hypertensive disorders require significantly greater neonatal intensive care resources than infants born following normotensive pregnancies. Increased NICU utilization contributes substantially to healthcare

costs, prolonged hospitalization, and parental psychological stress while emphasizing the importance of preventing disease progression through effective antenatal surveillance and timely obstetric intervention.

Stillbirth and early neonatal mortality remained relatively uncommon but occurred almost exclusively among pregnancies complicated by severe preeclampsia and eclampsia. These findings are comparable with large international cohort studies demonstrating that severe maternal hypertension markedly increases the risk of perinatal death because of placental abruption, fetal hypoxia, severe fetal growth restriction, and extreme prematurity. Improvements in fetal surveillance, timely referral, corticosteroid administration, neonatal resuscitation, and neonatal intensive care have contributed to declining perinatal mortality in recent years; however, preventable deaths continue to occur, particularly in resource-limited settings where delayed presentation remains common.²⁶

One of the principal objectives of this study was to identify independent predictors of adverse maternal and neonatal outcomes. Multivariable logistic regression demonstrated that severe preeclampsia or eclampsia, gestational age below 34 weeks at diagnosis, thrombocytopenia, elevated serum creatinine, and inadequate antenatal care independently predicted adverse maternal outcomes. These findings are consistent with current understanding of disease pathophysiology, as laboratory evidence of organ dysfunction reflects progressive endothelial injury and systemic inflammation. Thrombocytopenia represents platelet activation and consumption, whereas elevated serum creatinine indicates renal involvement and impaired glomerular filtration. Both parameters have been incorporated into contemporary international diagnostic criteria for severe preeclampsia because of their strong association with maternal complications.²⁷

Similarly, gestational age below 34 weeks emerged as the strongest independent predictor of adverse neonatal outcomes. Early-onset disease is associated with more severe placental dysfunction and frequently necessitates medically indicated

preterm delivery before fetal maturity has been achieved. Consequently, these infants experience substantially higher rates of respiratory distress syndrome, low birth weight, intraventricular hemorrhage, necrotizing enterocolitis, and neonatal mortality. Our findings therefore support recommendations from international guidelines advocating close surveillance of women with early-onset hypertensive disorders and management within tertiary care centers equipped with advanced neonatal intensive care facilities.²⁸

An important observation of the present study was the protective effect of regular antenatal care. Women who attended routine antenatal visits experienced significantly fewer maternal and neonatal complications than unbooked pregnancies. Early blood pressure monitoring, timely recognition of proteinuria, laboratory assessment, fetal surveillance, and appropriate referral facilitate prompt diagnosis before irreversible maternal organ damage develops. These findings reinforce recommendations from the World Health Organization and other professional organizations emphasizing universal access to quality antenatal care as a cornerstone of maternal health programs, particularly in low- and middle-income countries.

The strengths of the present study include its relatively large sample size, comprehensive evaluation of both maternal and neonatal outcomes, and simultaneous assessment of demographic, clinical, laboratory, and obstetric predictors using multivariable logistic regression. Inclusion of multiple hypertensive disorder subtypes allowed meaningful comparison across the disease spectrum and enhanced the clinical applicability of the findings. Furthermore, the retrospective cohort design enabled assessment of real-world clinical practice within a tertiary referral hospital where complicated pregnancies are routinely managed. Nevertheless, several limitations should be acknowledged. First, the retrospective nature of the study may have resulted in incomplete documentation and missing clinical information despite careful review of hospital records. Second, the study was conducted at a single tertiary care institution,

which may limit generalizability to primary or secondary healthcare settings. Third, because the hospital serves as a referral center, the proportion of severe preeclampsia and eclampsia was relatively high, potentially overestimating complication rates compared with community-based populations. Additionally, long-term maternal cardiovascular outcomes and long-term neurodevelopmental outcomes of surviving neonates were beyond the scope of this investigation. Future multicenter prospective cohort studies incorporating long-term maternal and child follow-up are warranted to validate these findings and improve risk prediction models.

Despite these limitations, the present study provides important evidence regarding the burden of hypertensive disorders of pregnancy in a tertiary healthcare setting. The identification of severe disease, early gestational age at diagnosis, thrombocytopenia, elevated serum creatinine, and inadequate antenatal care as significant predictors of adverse outcomes has important clinical implications. Women exhibiting these characteristics should undergo closer maternal and fetal surveillance, early referral to specialist obstetric units, aggressive blood pressure control, appropriate administration of magnesium sulfate, serial laboratory monitoring, and timely delivery when maternal or fetal deterioration is identified. In conclusion, hypertensive disorders of pregnancy continue to represent a major challenge for obstetric practice because of their substantial impact on maternal and neonatal health. The findings of the present study demonstrate that severe preeclampsia and eclampsia are associated with significantly increased maternal complications, ICU admission, prolonged hospitalization, preterm birth, low birth weight, NICU admission, and perinatal mortality. Early-onset disease, thrombocytopenia, elevated serum creatinine, and inadequate antenatal care independently predict adverse outcomes. Strengthening antenatal screening programs, improving timely referral systems, ensuring availability of evidence-based treatment protocols, and adopting multidisciplinary management strategies are

essential to reducing the burden of hypertensive disorders of pregnancy and improving both maternal and neonatal survival.

Conclusion:

Hypertensive disorders of pregnancy remain a significant cause of maternal and neonatal morbidity despite advances in obstetric care. In this retrospective cohort of 450 women, severe preeclampsia and eclampsia were associated with substantially higher risks of maternal complications, preterm birth, low birth weight, NICU admission, and perinatal mortality. Early-onset disease, thrombocytopenia, elevated serum creatinine, and inadequate antenatal care emerged as independent predictors of adverse outcomes. These findings underscore the importance of early diagnosis, comprehensive antenatal surveillance, timely referral, and multidisciplinary management to improve maternal and neonatal outcomes in women with hypertensive disorders of pregnancy.

Study Limitations:

This study has several limitations. First, its retrospective design relied on the accuracy and completeness of medical records, which may have resulted in missing or incomplete data. Second, the study was conducted at a single tertiary care center, potentially limiting the generalizability of the findings to other healthcare settings. Third, the referral nature of the hospital may have introduced selection bias by including a higher proportion of severe cases. Finally, long-term maternal cardiovascular outcomes and long-term neonatal developmental outcomes were not assessed.

Recommendations:

- 1.Strengthen routine antenatal screening for early detection of hypertensive disorders of pregnancy.
- 2.Ensure timely referral of high-risk pregnancies to tertiary care facilities.
- 3.Implement standardized evidence-based protocols for the management of hypertensive disorders, including prompt use of antihypertensive therapy and magnesium sulfate where indicated.

4.Promote multidisciplinary management involving obstetricians, physicians, anesthesiologists, intensivists, and neonatologists for women with severe disease.

5.Conduct large multicenter prospective studies to validate predictors of adverse maternal and neonatal outcomes and develop robust risk prediction models.

Conflict of Interest:

The authors declare that they have no conflicts of interest related to this study.

Funding:

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data Availability Statement:

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declaration and Ethical Approval:

The authors declare that this study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Ethical approval was obtained from the relevant Institutional Review Board prior to commencement of the study. Patient confidentiality and anonymity were maintained throughout the research process, and all data were de-identified before analysis. The authors affirm that this manuscript is original, has not been published previously, and is not under consideration for publication elsewhere. All authors have reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

REFERENCES:

- 1.World Health Organization. Trends in maternal mortality 2000–2020: estimates by WHO, UNICEF, UNFPA, World Bank Group and UNDESA Population Division. Geneva: World Health Organization; 2023.

2. Say L, Chou D, Gemmill A, Tunçalp Ö, Moller AB, Daniels J, et al. Global causes of maternal death: a WHO systematic analysis. *Lancet Glob Health*. 2014;2(6):e323–e333. doi: 10.1016/S2214-109X(14)70227-X.
3. National Institute of Population Studies (NIPS) [Pakistan], ICF. Pakistan Demographic and Health Survey 2017–18. Islamabad, Pakistan and Rockville, MD: NIPS and ICF; 2019.
4. Magee LA, Brown MA, Hall DR, Gupte S, Hennessy A, Karumanchi SA, et al. The 2021 International Society for the Study of Hypertension in Pregnancy (ISSHP) classification, diagnosis and management recommendations for international practice. *Pregnancy Hypertens*. 2022;27:148–169. doi: 10.1016/j.preghy.2021.09.008.
5. Brown MA, Magee LA, Kenny LC, Karumanchi SA, McCarthy FP, Saito S, et al. Hypertensive disorders of pregnancy: ISSHP classification, diagnosis, and management recommendations. *Hypertension*. 2018;72(1):24–43. doi: 10.1161/HYPERTENSIONAHA.117.10803.
6. American College of Obstetricians and Gynecologists. Gestational Hypertension and Preeclampsia. ACOG Practice Bulletin No. 222. *Obstet Gynecol*. 2020;135(6):e237–e260.
7. Phipps E, Prasanna D, Brima W, Jim B. Preeclampsia: updates in pathogenesis, definitions, and guidelines. *Clin J Am Soc Nephrol*. 2016;11(6):1102–1113. doi: 10.2215/CJN.12081115.
8. Roberts JM, Hubel CA. The two stage model of preeclampsia: variations on the theme. *Placenta*. 2009;30(Suppl A):S32–S37. doi: 10.1016/j.placenta.2008.11.009.
9. Mol BWJ, Roberts CT, Thangaratinam S, Magee LA, de Groot CJM, Hofmeyr GJ. Pre-eclampsia. *Lancet*. 2016;387(10022):999–1011. doi: 10.1016/S0140-6736(15)00070-7.
10. Brown MC, Best KE, Pearce MS, Waugh J, Robson SC, Bell R. Cardiovascular disease risk in women with pre-eclampsia: systematic review and meta-analysis. *Eur J Epidemiol*. 2013;28(1):1–19. doi: 10.1007/s10654-013-9762-6.
11. Burton GJ, Redman CWG, Roberts JM, Moffett A. Preeclampsia: pathophysiology and clinical implications. *BMJ*. 2019;366:l2381. doi: 10.1136/bmj.l2381.
12. Rana S, Lemoine E, Granger JP, Karumanchi SA. Preeclampsia: pathophysiology, challenges, and perspectives. *Circ Res*. 2019;124(7):1094–1112. doi: 10.1161/CIRCRESAHA.118.313276.
13. von Dadelszen P, Magee LA. Pre-eclampsia: an update. *Curr Hypertens Rep*. 2014;16(8):454. doi: 10.1007/s11906-014-0454-8.
14. National Institute for Health and Care Excellence (NICE). Hypertension in pregnancy: diagnosis and management (NG133). London: NICE; Updated 2023.
15. Duley L. The global impact of pre-eclampsia and eclampsia. *Semin Perinatol*. 2009;33(3):130–137. doi: 10.1053/j.semperi.2009.02.010.
16. Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R. Pre-eclampsia. *Lancet*. 2010;376(9741):631–644. doi:10.1016/S0140-6736(10)60279-6.
17. Bilano VL, Ota E, Ganchimeg T, Mori R, Souza JP. Risk factors of pre-eclampsia/eclampsia and its adverse outcomes in low- and middle-income countries: a WHO secondary analysis. *PLoS One*. 2014;9(3):e91198. doi:10.1371/journal.pone.0091198.
18. Redman CW, Sargent IL. Immunology of pre-eclampsia. *Am J Reprod Immunol*. 2010;63(6):534–543. doi:10.1111/j.1600-0897.2010.00831.x.
19. Magee LA, von Dadelszen P, Stones W, Mathai M, editors. The FIGO Textbook of Pregnancy Hypertension. London: Global Library of Women's Medicine; 2016. doi:10.3843/GLOWM.102505.

20. Abalos E, Cuesta C, Grosso AL, Chou D, Say L. Global and regional estimates of pre-eclampsia and eclampsia. *Eur J Obstet Gynecol Reprod Biol.* 2013;170(1):1-7. doi:10.1016/j.ejogrb.2013.05.005.
21. Redman CW, Staff AC. Preeclampsia, biomarkers, syncytiotrophoblast stress, and placental capacity. *Am J Obstet Gynecol.* 2015;213(4 Suppl):S9-S11. doi:10.1016/j.ajog.2015.08.003.
22. Haram K, Mortensen JH, Nagy B. Genetic aspects of preeclampsia and HELLP syndrome. *J Pregnancy.* 2014;2014:910751. doi:10.1155/2014/910751.
23. Baschat AA. Fetal growth restriction—from observation to intervention. *J Perinat Med.* 2010;38(3):239-246. doi:10.1515/JPM.2010.041.
24. Koopmans CM, Bijlenga D, Groen H, et al. Induction of labour versus expectant monitoring for gestational hypertension or mild pre-eclampsia after 36 weeks (HYPITAT trial). *Lancet.* 2009;374(9694):979-988. doi:10.1016/S0140-6736(09)60736-4.
25. Figueras F, Gratacós E. Update on the diagnosis and classification of fetal growth restriction. *Fetal Diagn Ther.* 2014;36(2):86-98. doi:10.1159/000357592.
26. Lawn JE, Blencowe H, Oza S, et al. Every Newborn: progress, priorities, and potential beyond survival. *Lancet.* 2014;384(9938):189-205. doi:10.1016/S0140-6736(14)60496-7.
27. Sibai BM. Evaluation and management of severe preeclampsia before 34 weeks' gestation. *Am J Obstet Gynecol.* 2011;205(3):191-198. doi:10.1016/j.ajog.2011.07.017.
28. Ganzevoort W, Thornton JG, Marlow N, et al. STRIDER Consortium. Management of early-onset fetal growth restriction. *Lancet.* 2020;396(10244):167-176. doi:10.1016/S0140-6736(20)31145-8.
29. World Health Organization. WHO recommendations on antenatal care for a positive pregnancy experience. Geneva: WHO; 2016.
30. Brown MA, Magee LA, Kenny LC, et al. Hypertensive disorders of pregnancy: ISSHP recommendations. *Hypertension.* 2018;72(1):24-43. doi:10.1161/HYPERTENSIONAHA.117.10803.