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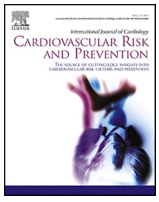

Examining maternal and fetal outcomes across various subtypes of hypertension during pregnancy

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ABSTRACT

Introduction: Hypertensive disorders of pregnancy (HDP) are a leading cause of maternal morbidity and mortality worldwide. It includes chronic hypertension (CH), gestational hypertension (GH), preeclampsia (PRE), and CH with superimposed preeclampsia (SPE).We aim to assess in-hospital maternal and fetal outcomes of women in each of these groups in comparison to normotensive controls.

Methods: Study sample included women in the National Inpatient Sample dataset from 2016 to 2020 who were categorized into the 4 groups of HDP as described above. They were compared to normotensive pregnancies for maternal and fetal outcomes using regression analysis after adjusting for age, race, C-section status, and comorbidities.

Results: The study dataset from October 2015–December 2020 included 19,089,780 delivery admissions with 2,771,809 (14.5 %) of patients affected by HDP. The HDP groups were distributed as follows: GH - 38 %, PRE - 32 %, SPE - 11 %, and CH - 19 %. Women with PRE, SPE, and CH had significantly higher rates of mortality, circulatory shock, peripartum cardiomyopathy, acute kidney injury, preterm labor, stillbirth, and cerebrovascular events as compared to normotensive patients, while GH did not. Specifically, maternal mortality was highest in the SPE group (adjusted odds ratio [aOR] 3.16), followed by PRE (aOR 2.91) and CH (aOR 2.42). Additionally, all HDP groups had higher rates of small for gestational age and significant bleeding as compared to normotensive patients.

Conclusions: Pregnant patients with CH, PRE, and SPE experience higher rates of adverse maternal and fetal outcomes during their delivery admission when compared to normotensive patients. Understanding the graded risk differences across HDP subtypes may enable more tailored interventions, optimizing maternal and fetal outcomes for those at highest risk.

1. Introduction

Hypertensive disorders of pregnancy (HDP) constitute a significant health concern globally, affecting up to 15 % of pregnancies and contributing notably to maternal morbidity and mortality rates [1,2]. In the United States, HDP accounts for 7–12 % of pregnancy-associated maternal deaths [3]. Subcategories of HDP, including chronic hypertension (CH), preeclampsia (PRE), CH with superimposed preeclampsia (SPE), and gestational hypertension (GH), are delineated by the American College of Obstetrics and Gynecology (ACOG) based on the timing of hypertension onset and the presence of end-organ injury [4,5].

HDP are associated with an elevated risk of acute kidney injury (AKI), pulmonary edema, acute respiratory distress syndrome (ARDS), and cardiovascular disease in mothers, along with adverse fetal outcomes such as stillbirth, small for gestational age (SGA), and preterm birth [6–8]. Despite advancements in early diagnosis and management, the various HDP contribute to different extents to maternal mortality

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List of abbreviations:

HDP	Hypertensive Disorders of Pregnancy
CH	Chronic Hypertension
GH	Gestational Hypertension
PRE	Preeclampsia
SPE	Chronic Hypertension with Superimposed Preeclampsia
NIS	National Inpatient Sample
ICD-10-CM	International Classification of Diseases, 10th Revision, Clinical Modification
aOR or OR	Adjusted Odds Ratio and Odds Ratio
CI	Confidence Interval
AKI	Acute Kidney Injury
ARDS	Acute Respiratory Distress Syndrome
SGA	Small for Gestational Age
MI	Myocardial Infarction
PE	Pulmonary Embolism

[3]. Previous studies have noted varying degrees of diastolic changes and left ventricular remodeling among different subtypes of HDP, suggesting potential differences in severity [9]. However, there is limited literature comparing maternal and fetal outcomes across the four subgroups of HDP at a large contemporary scale. This study aims to fill this gap by evaluating in-hospital maternal and fetal outcomes among women in each subgroup of HDP as compared to normotensive controls, utilizing nationally representative data.

2. Methods

2.1. Study design and data source

This retrospective cohort study utilized data from the National Inpatient Sample (NIS) database spanning from October 2015 to December 2020. The NIS is the largest publicly available all-payer inpatient care database in the United States, capturing approximately 20 % of all U.S [10,11]. hospitalizations and providing a representative sample of national hospitalizations. Given that the data is both publicly available and de-identified, Institutional Review Board (IRB) approval was not required.

2.2. Study population

We identified adult women (age ≥18 years) admitted to hospitals for delivery-related hospitalizations (normal delivery, C-section delivery, delivery-related procedures, and labor related complications). HDP subtypes were classified according to the ACOG guidelines, including CH, PRE —which, for this study, encompassed eclampsia and HELLP (Hemolysis, Elevated Liver enzymes, and Low Platelets)— SPE, and GH. The International Classification of Diseases-10 Clinical Modification (ICD-10-CM) diagnostic and procedure codes were used for identification of the sample and various HDP subtypes (Supplemental Table S1).

Cases of HDP were identified based on ICD-10-CM diagnosis codes corresponding to each subtype. CH was defined by ICD-10-CM codes I10.x, PRE by O14.x, CH with SPE by O13.x or O14 with I10.X, and GH by O13.9. Normotensive pregnancies served as the comparison group, identified based on the absence of HDP diagnostic codes. For cases with overlapping codes, those with both GH and PRE codes were categorized as PRE, and those with both CH and PRE or PRE and SPE were labeled as SPE.

2.3. Outcome measures

Maternal outcomes were divided into primary cardiovascular and secondary non-cardiovascular outcomes to provide a more detailed analysis. Primary maternal outcomes included: in-hospital mortality, acute myocardial infarction (MI), cardiac arrest, pulmonary edema or acute decompensated heart failure, cerebrovascular accidents (CVA), pulmonary embolism (PE), circulatory shock, and peripartum cardiomyopathy. Secondary maternal outcomes encompassed: AKI, gestational diabetes mellitus, bleeding requiring transfusion, ARDS, need for mechanical ventilation, and sepsis.

Fetal outcomes included SGA, preterm labor, and stillbirth, as these represent key indicators of adverse fetal health in HDP and may reflect intrauterine growth restriction, early delivery, and perinatal loss.

2.4. Statistical analysis

Descriptive statistics were employed to summarize the demographic and clinical characteristics of the study population across the HDP subtypes. To allow for meaningful comparisons between groups of different sizes, we calculated the incidence of each outcome as the rate per 100,000 for each category. Additionally, regression analyses were then conducted to compare maternal and fetal outcomes between each HDP subtype and a normotensive control group, adjusting for potential confounding variables. The covariates included in the model are age, race, hospital region, income, payer status, Type 1 and Type 2 diabetes, hyperlipidemia, heart failure, chronic kidney disease, coronary artery disease, polycystic ovary syndrome, obesity, smoking status, multiple gestations, and cesarean delivery. Adjusted odds ratios (aORs) with 95 % confidence intervals (CIs) were calculated for each outcome, quantifying the associations of HDP subtypes with maternal and fetal risks.

To further explore the impact of disease severity, a sensitivity analysis was conducted within the PRE and SPE groups, stratifying outcomes by the presence or absence of severe features. All statistical analyses were performed using STATA 17, with adjustments for the complex survey design of the NIS database to ensure accurate estimation of standard errors. Results were considered statistically significant at $p < 0.05$.

3. Results

The study included a total sample size of 19,087,890 women from the NIS dataset spanning from October 2015 to December 2020. Within this cohort, there were 2,771,809 cases of HDP. Amongst those with HDP, 525,645 women (19.0 %) were categorized as having CH, while 1,047,549 women (37.8 %) had GH, 887,495 women (32.0 %) were diagnosed with PRE, and 311,120 women (11.2 %) had SPE.

3.1. Baseline characteristics

Baseline characteristics varied across HDP subtype (Supplemental Table S2). CH and SPE patients were older on average, with CH associated with a higher prevalence of significant comorbidities, including chronic kidney disease, congestive heart failure, and chronic obstructive pulmonary disease. Racial disparities were observed, particularly with a higher proportion of Black individuals among those with CH and SPE. Insurance coverage also varied, with Medicaid more commonly used among patients with PRE and SPE. Cesarean delivery rates were highest in the SPE and PRE groups, and labor induction was more frequent in GH cases.

3.2. Primary maternal cardiovascular outcomes

Maternal and fetal outcomes are reported per 100,000 deliveries in Table 1. The risk of acute MI was highest in SPE (aOR 14.4, 95 % CI: 8.3–24.8, $p < 0.001$), followed by PRE (aOR 9.40, 95 % CI: 4.14–16.6, $p < 0.001$), CH (aOR 3.85, 95 % CI: 1.97–7.52, $p < 0.001$), while GH showed no significant association (Fig. 1).

In terms of cardiac arrest, increased odds were observed in PRE (aOR

Table 1
In-Hospital Maternal and Fetal Outcomes by Hypertensive Disorder Subtype reported per 100,000.

	Normotensive controls (n = 16,316,081)	Chronic Hypertension (n = 525,645)	Gestational HTN (n = 1,047,549)	Pre-eclampsia (n = 887,495)	Superimposed Pre-eclampsia (n = 311,120)	P-Value
Primary Maternal Outcomes						
Composite outcome[a]	15,580 (95.5)	1825 (347.2)	1040 (99.3)	3285 (370.0)	2095 (673.4)	<0.01
Died during hospitalization	800 (4.9)	105 (20.0)	40 (3.8)	170 (19.2)	95 (30.5)	<0.01
Any Shock	9410 (57.7)	950 (180.8)	565 (53.9)	1710 (192.7)	685 (220.2)	<0.01
Cardiogenic Shock	285 (1.7)	45 (8.6)	15 (1.4)	60 (6.8)	65 (20.9)	<0.01
Acute Myocardial infarction	255 (1.6)	85 (16.2)	30 (2.9)	200 (22.5)	235 (75.5)	<0.01
Peripartum Cardiomyopathy	1480 (9.1)	565 (107.5)	180 (17.2)	1035 (116.6)	915 (294.1)	<0.01
Pulmonary Embolism	4255 (26.1)	330 (62.8)	335 (32.0)	635 (71.5)	350 (112.5)	<0.01
Cerebrovascular accident	635 (3.9)	95 (18.1)	65 (6.2)	335 (37.7)	285 (91.6)	<0.01
Pulmonary Edema	3430 (21.0)	1715 (326.3)	515 (49.2)	3745 (421.9)	2940 (945.3)	<0.01
Secondary maternal outcomes						
Sepsis	13,700 (84.0)	1280 (243.4)	1020 (97.4)	2415 (272.1)	975 (313.4)	<0.01
Acute respiratory distress syndrome	8600 (52.7)	1550 (295.0)	705 (67.3)	3820 (430.4)	2545 (818.0)	<0.01
Mechanical ventilation	4870 (29.9)	875 (166.5)	335 (32.0)	2090 (235.5)	1380 (443.6)	<0.01
Gestational Diabetes	1173989 (7187.6)	84,035 (15,985.8)	109680 (10,471.5)	102085 (11,501.4)	47,850 (15,382.6)	<0.01
Disseminated intravascular coagulation	23,810 (146.0)	1285 (244.4)	1950 (186.1)	4430 (499.2)	1230 (395.3)	<0.01
Acute kidney injury	7980 (48.9)	1915 (364.3)	1455 (138.9)	9365 (1055.2)	5105 (1640.1)	<0.01
Blood Transfusion	652,595 (4000.4)	29,420 (5595.8)	32,245 (3076.5)	80,615 (9088.6)	36,000 (11,569.8)	<0.01
Fetal Outcomes						
Small for gestational age	823,420 (5048.6)	36,650 (6972.8)	69,010 (6585.7)	94,605 (10,657.5)	30,050 (9657.8)	<0.01
Fetal distress	515570 (3159.4)	27,700 (5271.1)	40,635 (3879.7)	67,635 (7619.5)	28,540 (9173.1)	<0.01
Preterm labor	424,285 (2.6 %)	18,075 (3438.1)	33,540 (3201.4)	23,395 (2636.8)	8285 (2662.5)	<0.01
Stillbirth	1475 (9.0)	175 (33.3)	120 (11.5)	305 (34.4)	215 (69.1)	<0.01

[a] Composite: stroke, Acute coronary syndrome, peripartum cardiomyopathy, stroke, and death.

3.21, 95 % CI: 2.42–4.25, p < 0.001), SPE (aOR 2.76, 95 % CI: 1.82–4.20, p < 0.001), and CH (aOR 1.90, 95 % CI: 1.26–2.87, p = 0.002), while GH showed no significant association. For pulmonary edema or acute decompensated heart failure (ADHF), the highest odds were noted in SPE (aOR 20.8, 95 % CI: 16.3–25.6, p < 0.001) and PRE (aOR 21.7, 95 % CI: 18.8–25.0, p < 0.001), followed by CH (aOR 1.80, 95 % CI: 1.33–2.42, p < 0.001) and GH (aOR 1.88, 95 % CI: 1.14–2.52, p = 0.001). CVA risk was highest in SPE (aOR 10.9, 95 % CI: 7.8–15.3, p < 0.001) and PRE (aOR 6.67, 95 % CI: 5.0–8.8, p < 0.001), followed by CH (aOR 2.62, 95 % CI: 1.64–4.18, p < 0.001), while GH was not significantly associated.

PE showed significant risk with SPE (aOR 4.13, 95 % CI: 2.65–6.42, p < 0.001) and PRE (aOR 2.83, 95 % CI: 1.96–4.09, p < 0.001), whereas CH (aOR 1.39, 95 % CI: 0.78–2.45, p = 0.261) and GH (aOR 1.20, 95 % CI: 0.71–2.02, p = 0.491) were not significantly associated. For circulatory shock, both PRE (aOR 2.45, 95 % CI: 2.16–2.78, p < 0.001), CH (aOR 1.74, 95 % CI: 1.45–2.08, p < 0.001), and SPE (aOR 1.66, 95 % CI: 1.34–2.06, p < 0.001) showed significant associations, while GH did not. Peripartum cardiomyopathy risk was highest in SPE (aOR 17.5, 95 % CI: 14.1–21.8, p < 0.001), followed by PRE (aOR 9.96, 95 % CI: 8.2–12.1, p < 0.001), GH (aOR 1.72, 95 % CI: 1.13–2.47, p = 0.003), and CH (aOR 6.74, 95 % CI: 5.25–8.64, p < 0.001). Finally, inpatient mortality was significantly associated with CH (aOR 2.43, 95 % CI: 1.49–3.97, p < 0.001), SPE (aOR 3.17, 95 % CI: 1.87–5.37, p < 0.001), and PRE (aOR 2.92, 95 % CI: 1.95–4.43, p < 0.001), while GH showed no significant association.

For the composite outcome, CH (aOR 2.44, 95 % CI: 2.13–2.80, p < 0.001), PRE (aOR 3.22, 95 % CI: 2.82–3.67, p < 0.001), and SPE (aOR 4.36, 95 % CI: 3.75–5.63, p < 0.001) were significantly associated with increased odds, while GH showed no significant association. The central figure illustrates the trend of composite outcomes per 1000 deliveries across five groups over the study period. Normotensive and GH women consistently showed the lowest rates (below 2 per 1000), while those with SPE exhibit the highest rates (6–10 per 1000). CH and PRE showed intermediate rates (2–4 per 1000). None of the groups demonstrated statistically significant trends over time.

3.3. Secondary maternal non-cardiovascular outcomes

Fig. 2 shows secondary non-cardiovascular maternal outcomes comparing HDP subtypes to normotensive patients. AKI was significantly associated with all hypertensive disorders, with the highest odds in SPE (aOR 17.7, 95 % CI: 9.36–19.5, p < 0.001) and PRE (aOR 15.7, 95 % CI: 9.52–17.0, p < 0.001), followed by CH (aOR 4.54, 95 % CI: 4.01–5.12, p < 0.001) and GH (aOR 2.64, 95 % CI: 2.31–3.01, p < 0.001). Regarding GDM, elevated risks were observed for all HDP categories: CH (aOR 1.84, 95 % CI: 1.81–1.88, p < 0.001), GH (aOR 1.41, 95 % CI: 1.38–1.43, p < 0.001), PRE (aOR 1.52, 95 % CI: 1.50–1.55, p < 0.001), and SPE (aOR 1.77, 95 % CI: 1.73–1.81, p < 0.001).

Bleeding requiring transfusion was significantly associated with CH (aOR 1.49, 95 % CI: 1.41–1.57, p < 0.001), GH (aOR 1.37, 95 % CI: 1.31–1.44, p < 0.001), PRE (aOR 2.92, 95 % CI: 2.82–3.02, p < 0.001), and SPE (aOR 2.39, 95 % CI: 2.26–2.51, p < 0.001). ARDS risk was highest in SPE (aOR 6.30, 95 % CI: 5.56–7.14, p < 0.001) and PRE (aOR 5.47, 95 % CI: 4.98–5.99, p < 0.001), with CH (aOR 2.76, 95 % CI: 2.38–3.19, p < 0.001) also showing significant risk; however, GH was not associated with ARDS. Mechanical ventilation was more likely in SPE (aOR 5.83, 95 % CI: 4.92–6.91, p < 0.001), PRE (aOR 5.32, 95 % CI: 4.70–6.02, p < 0.001), and CH (aOR 2.74, 95 % CI: 2.26–3.33, p < 0.001), while GH was not significantly associated. Lastly, sepsis risk was increased in CH (aOR 2.11, 95 % CI: 1.82–2.46, p < 0.001), PRE (aOR 2.63, 95 % CI: 2.27–3.04, p < 0.001), and SPE (aOR 1.98, 95 % CI: 1.59–2.47, p < 0.001), with no association in GH.

3.4. Fetal outcomes

Fetal outcomes in HDP subtypes, particularly SPE and PRE, were associated with an increased risk of adverse fetal outcomes (Fig. 3). The odds of preterm birth were highest in SPE (aOR 2.7; 95 % CI: 2.6–2.8, p < 0.01), followed by PRE (aOR 2.2; 95 % CI: 2.1–2.3, p < 0.01). Additionally, SGA infants were more common among SPE (aOR 2.9; 95 % CI: 2.8–3.0, p < 0.01) and PRE (aOR 2.4; 95 % CI: 2.3–2.4, p < 0.01) groups. Stillbirth risk was also elevated for CH (aOR 2.0; 95 % CI: 1.9–2.1, p <

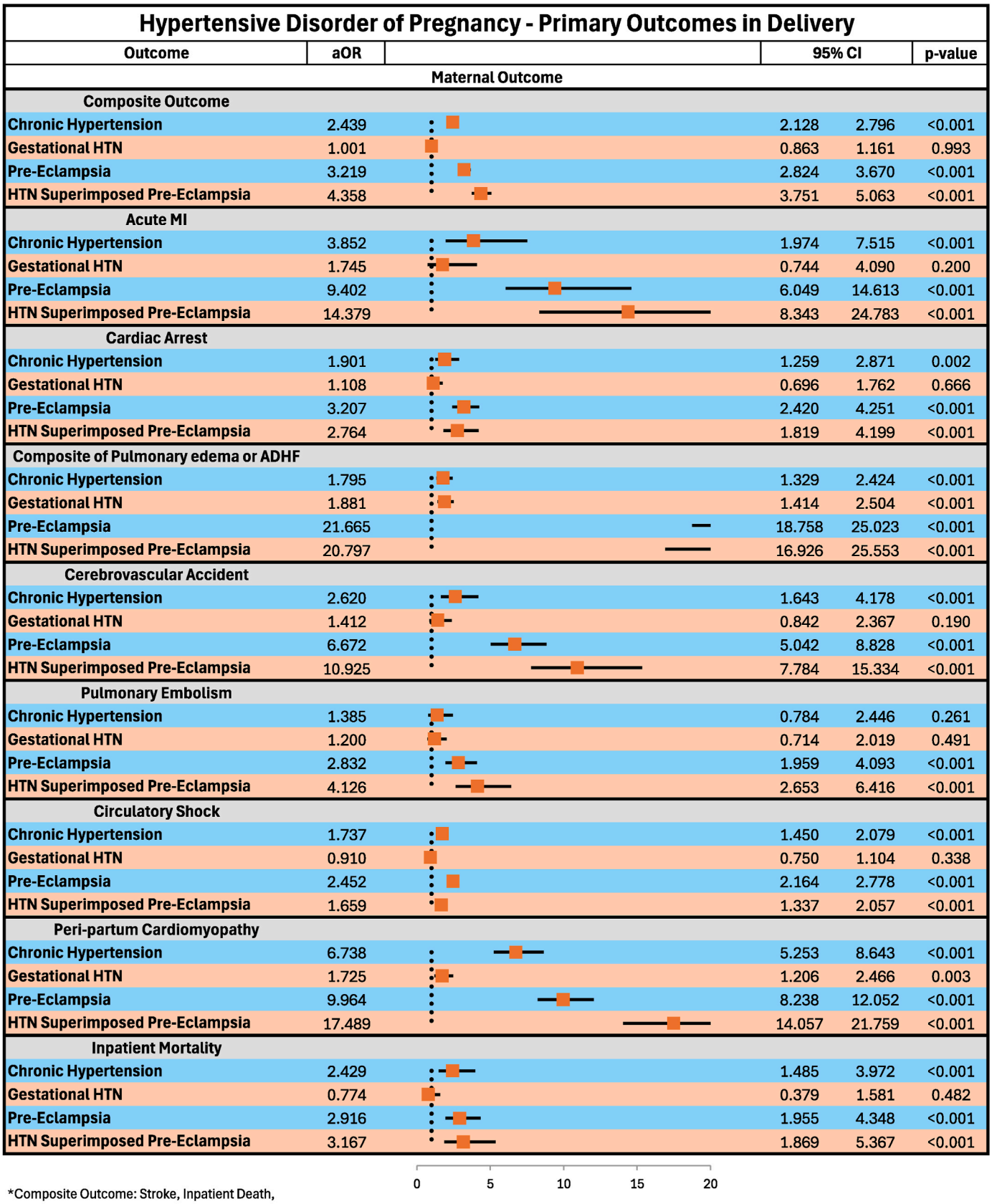

Fig. 1. Regression analysis of Primary Maternal Outcomes by Hypertensive Disorder Subtype. aOR: Adjusted Odds Ratio, CI: Confidence Interval, HTN: Hypertension, ACS: Acute Coronary Syndrome, ADHF: Acute Decompensated Heart Failure, MI: Myocardial Infarction, HELLP: Hemolysis, Elevated Liver enzymes, and Low Platelets.

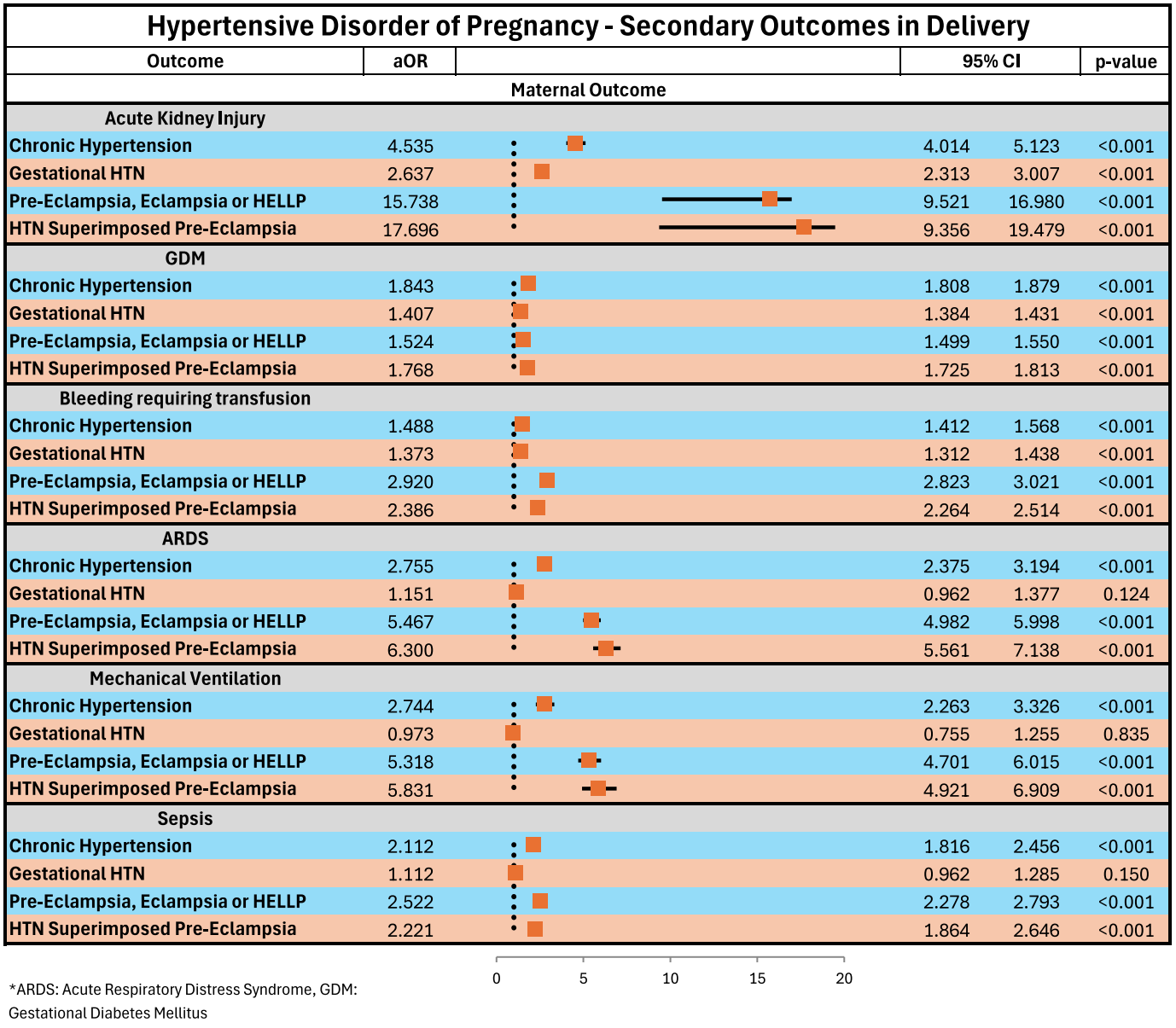

Fig. 2. Trends in secondary Maternal Outcomes Over Time by Hypertensive Disorder Subtype aOR: Adjusted Odds Ratio, CI: Confidence Interval, HTN: Hypertension; HELLP: Hemolysis, Elevated Liver enzymes, and Low Platelets.

0.01), SPE (aOR 1.8; 95 % CI: 1.6–1.9, $p < 0.01$), and PRE (aOR 1.5; 95 % CI: 1.5–1.6, $p < 0.01$). In contrast, GH generally presented a reduced risk for preterm birth (aOR 0.76; 95 % CI: 0.74–0.78, $p < 0.01$) and stillbirth (aOR 0.52; 95 % CI: 0.48–0.56, $p < 0.01$) relative to normotensive controls.

3.5. Resource utilization

Both length of stay and hospital charges were significantly higher for patients with HDP compared to normotensive controls ($p < 0.01$). Normotensive patients had an average length of stay of 2.5 ± 2.0 days and charges of $19,855 ± 17,522.1. In comparison, the length of stay was 3.2 ± 3.3 days for CH, 3.0 ± 2.3 days for GH, 4.0 ± 3.5 days for PRE, and 4.9 ± 5.0 days for SPE. Corresponding hospital charges were $25,754.3 ± 32,365.2 for CH, $23,800.8 ± 19,498.9 for GH, $32,856.2 ± 30,968.8 for PRE, and $39,186.2 ± 38,728.1 for SPE.

3.6. Sensitivity analysis

In a sensitivity analysis examining outcomes in HDP with and without severe features, significant findings emerged for both maternal and fetal outcomes (Supplemental Fig. S1). Severe features in PRE and SPE significantly increased maternal risks, including AKI (PRE: aOR 1.838; SPE: aOR 1.327), heart failure (PRE: aOR 2.785; SPE: aOR 2.118), and peripartum cardiomyopathy (PRE: aOR 1.942; SPE: aOR 3.194).

In fetal outcomes, severe features in PRE were associated with significantly higher odds of *preterm labor* (aOR 2.223, 95 % CI 2.142–2.308, $p < 0.001$) and *small-for-gestational-age* fetuses (aOR 1.732, 95 % CI 1.667–1.799, $p < 0.001$). The risk of *stillbirth* was also elevated in PRE with severe features, with an aOR of 1.513 (95 % CI 1.361–1.683, $p < 0.001$), though this association was not seen with SPE.

4. Discussion

In this comprehensive study, we undertook a large-scale analysis of

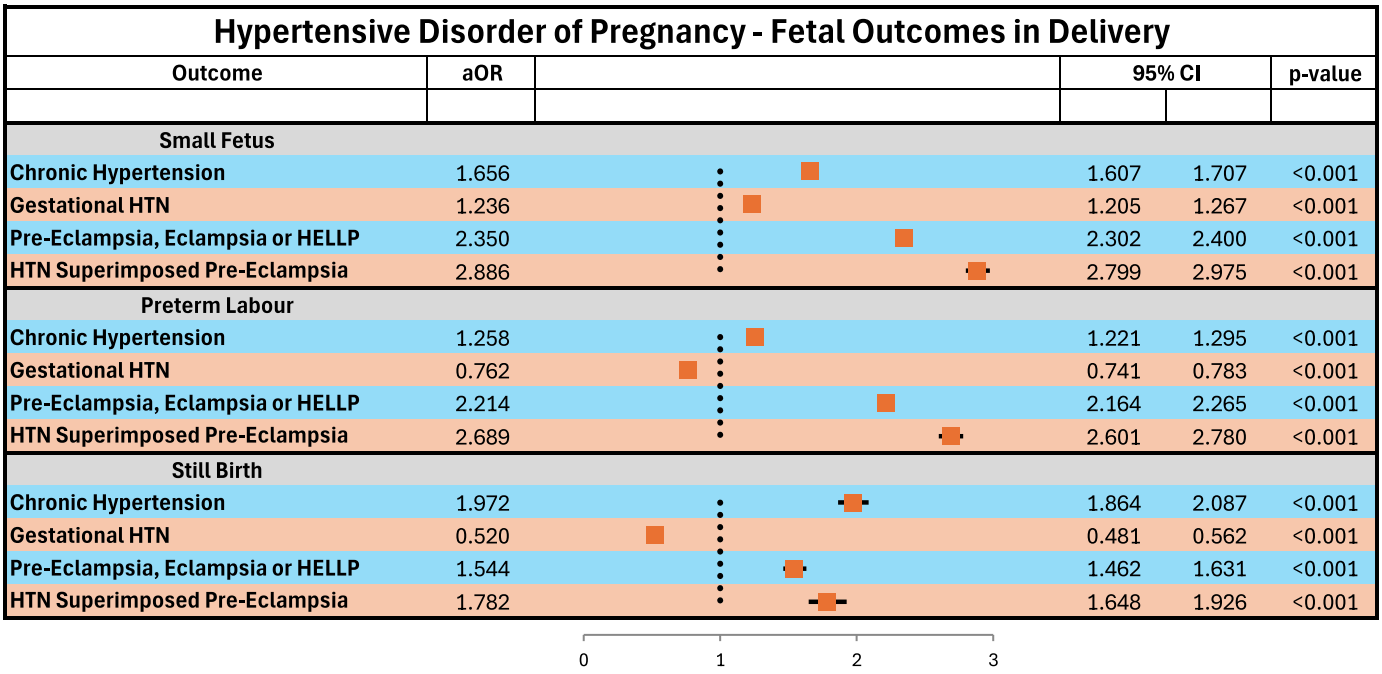

Fig. 3. Fetal Outcomes by Hypertensive Disorder Subtype. aOR: Adjusted Odds Ratio, CI: Confidence Interval, HTN: Hypertension; HELLP: Hemolysis, Elevated Liver enzymes, and Low Platelets.

delivery hospitalizations in the United States spanning from October 2015 to December 2020. Our primary aim was to investigate the differences in maternal and fetal outcomes among different HDP subtypes and how they compare to normotensive controls. Our analysis identified significant disparities in outcomes among the different subtypes of HDP. Specifically, individuals with PRE, SPE, and CH exhibited higher rates of adverse outcomes severe maternal complications when compared to normotensive controls, including in-hospital mortality, acute MI, CVA, circulatory shock, and peripartum cardiomyopathy. These groups also experienced heightened risks of AKI, PE, and need for mechanical ventilation. Additionally, adverse fetal outcomes such as SGA, preterm labor, and stillbirth were more prevalent among these subtypes compared to normotensive patients. Notably, women with GH did not demonstrate significantly higher rates of adverse outcomes as compared to normotensive controls.

The classification of HDP into distinct subtypes, including CH, GH, PRE, and SPE, acknowledges the heterogeneous nature of these disorders [12]. This classification system has now evolved, with various societies acknowledging that each subtype may display distinct pathophysiological mechanisms and clinical presentations [13,14]. Moreover, the classification of HDP subtypes allows for a more refined understanding of their clinical implications [13,14]. For instance, PRE and SPE are characterized by endothelial dysfunction, which contributes to the development of multisystem organ involvement and adverse outcomes [15]. On the other hand, CH represents a chronic condition with persistent hypertension, posing long-term risks to maternal and fetal health [13]. Our study's approach of analyzing outcomes for each subtype offers a nuanced understanding of the impact of HDP on maternal and fetal health.

Pregnant patients with PRE, SPE, and CH exhibit higher rates of inpatient mortality as compared to normotensive cohorts. HDP represents a significant contributor to maternal mortality, exacerbating the already concerning trend of increasing maternal mortality rates in the US [16]. In fact, according to the Centers for Disease Control and Prevention (CDC), maternal mortality rates in the United States have been steadily increasing, with cardiovascular-related conditions, including HDP, identified as leading causes of maternal death [17]. Over a 40-year period, Anant et al. reported an excess of 2.1 hypertension-related maternal deaths per 100,000 live births [18], emphasizing the gravity of the issue. Furthermore, CH in pregnancy triples the risk of maternal mortality, as shown in a study by Bateman et al. [19] The elevated maternal mortality rates observed in our PRE, SPE, and CH groups as compared to normotensive controls aligns with findings from existing literature [20,21].

Broadly, women with PRE, SPE, and CH had higher rates of adverse events such as circulatory shock, PE, peripartum cardiomyopathy, kidney failure, and CVA as compared to the normotensive cohort. In a study based on the National Readmissions Database (NRD) evaluating risk factors for peripartum cardiomyopathy, pre-eclampsia (with and without severe features) conferred a 7-to-19-fold higher risk of peripartum cardiomyopathy as did CH demonstrating a 10-fold increased risk of peripartum cardiomyopathy [22]. We similarly demonstrate SPE increased the risk 17-fold, PRE 9-fold, and CH 6.7-fold. A Danish study found that PRE was associated with a 1.5-fold higher risk of deep vein thrombosis as well as PE both during pregnancy and post-partum, we observed that the prevalence of PE was similar across PRE, SPE and CH groups [23]. PRE also confers a 4-fold increased risk of stroke [24]. In our cohort, the incidence of CVA was highest among those with SPE. Existing literature reflects that AKI as a complication of pre-eclampsia affects about 1 % of patients, which is very similar to the incidence noted in our PRE and SPE cohorts [25].

A study by Wu et al. examining deliveries from 2004 to 2014 found that women with SPE had the highest odds for stroke (OR 7.83, 95 % CI 6.25 to 9.80), MI (OR 5.20, 95 % CI 3.11 to 8.69), and peripartum cardiomyopathy (OR 4.37, 95 % CI 3.64 to 5.26) as compared to women without HDP [26]. We assessed the trend of our composite outcome (stroke, acute coronary syndrome, peripartum cardiomyopathy, and death) in a more contemporary period. Over our study period, women with SPE had the worst outcomes, followed by those with PRE and CH, as compared to normotensive patients. Circulatory shock has not been well described as an outcome before in women with HDP. Our study showed that patients with PRE, CH and SPE all had significantly higher odds of developing shock as compared to normotensive patients, while GH did not confer a higher risk of shock.

Our study findings suggest that GH does not confer significantly higher rates of acute adverse in-hospital outcomes during delivery

admission compared to normotensive controls. This distinction may stem from differences in pathophysiology and clinical severity. GH is characterized by new-onset hypertension after 20 weeks of gestation without proteinuria or end-organ dysfunction, distinguishing it from PRE/SPE [14].

Physiologically, GH and PE appear to diverge. PE is characterized by placental ischemia, angiogenic imbalance (elevated sFlt-1, reduced PlGF), and systemic endothelial dysfunction, which contribute to multi-organ injury [27]. In contrast, GH may represent a transient maternal hemodynamic response without significant placental pathology or angiogenic disruption [27]. Supporting this distinction, echocardiographic studies have shown lower rates of left ventricular remodeling and diastolic dysfunction in GH compared to PRE and SPE [9]. Similarly, Hauspurg et al. found that individuals with PRE had higher odds of developing postpartum hypertension than those with GH, with aORs of 2.35 (95 % CI, 1.63–3.41) and 1.61 (95 % CI, 1.09–2.39) at one year, respectivel [28].

A study from Canada compared risk factors and outcomes between GH and PRE and found that PRE is associated with higher rates of severe maternal complications like eclampsia and HELLP syndrome. It was also associated with earlier gestational age at delivery, and poorer perinatal outcomes, such as increased preterm births and small-for-gestational-age infants, compared to GH. PRE patients also had a higher rate of cesarean deliveries due to the severity of the condition [29]. However, it is important to note that GH can progress to more severe disease. PRE develops in up to 35 % of women initially diagnosed with GH and in 25 % of those with CH [30]. The HYPITAT trial showed that delivering GH patients at 37 weeks can reduce the risk of disease progression and adverse maternal outcomes compared to expectant management, supporting current practice guidelines [31]. Identifying biomarkers or predictive factors for the progression of GH to more severe forms of HDP could aid in risk stratification and inform clinical management strategies. Not only does it emphasize more research into it but raises questions about the necessity and intensity of intervention in GH cases and emphasizes the importance of accurate diagnosis and risk stratification.

Our study demonstrated multiple poor fetal outcomes associated with PRE, SPE, and CH, including preterm labor, SGA, and still birth. Endothelial dysfunction, characteristic of HDP, leads to increased cytokine release, triggering inflammation and elevated blood pressure [30,32]. This process, along with impaired spiral artery remodeling, reduces fetal perfusion, thus impeding fetal growth. Similar to our results The Hokkaido study showed that women with HDP had 2.1-, 3.5-, and 3.6-fold higher risks of having SGA infants, preterm birth, and infants with low birth weight than those with normotensive pregnancies [33]. Prior studies including BOSHI study and others demonstrated that the trajectory of maternal blood pressure during pregnancy is also an indicator of infant birth weight [34,35]. In an extensive meta-analysis of 55 studies, women with CH had high pooled incidences of preterm delivery <37 weeks' gestation, birth weight <2500 g (16.9 %, 13.1 %–21.5 %), neonatal unit admission and perinatal death [36].

Our subgroup analysis also demonstrated that severe features in HDP, particularly in PRE, are associated with significantly higher risks of AKI, heart failure, and peripartum cardiomyopathy. For example, severe-feature PRE was linked to nearly a three-fold increase in heart failure risk (aOR 2.785, 95 % CI 2.079–3.730). Fetal outcomes were also affected, with severe-feature PRE increasing the odds of preterm labor (aOR 2.223, 95 % CI 2.142–2.308) and small-for-gestational-age births. This highlights another important element to take into consideration when looking at subtypes of HDP.

Our supplemental data (Table S2) highlight demographic and clinical characteristics associated with HDP that are consistent with established risk factors reported in prior studies. Women with CH and SPE were significantly older than normotensive controls in our cohort, consistent with prior literature indicating increased risk of HDP with advancing maternal age [37,38]. Furthermore, significant racial disparities were observed, with Black women having disproportionately higher rates of HDP, a finding supported in prior population-based analyses highlighting higher susceptibility of HDP among Black patients [39]. Moreover, a significantly higher prevalence of chronic metabolic and endocrine comorbidities—including diabetes mellitus, polycystic ovarian syndrome, chronic kidney disease, and obesity—in the HDP subgroups. This aligns with well-established associations between pre-pregnancy metabolic dysfunction and increased risk for pre-eclampsia [38,40]. Similarly, multiple gestation was more prevalent in the HDP cohort than normotensive controls, consistent with its established association with HDP in prior literature [37,38].

HDP represents a significant public health concern, and our study demonstrates that different HDP subtypes have varying impact on maternal morbidity and mortality. The graded severity observed across these subtypes demonstrates the need for individualized clinical approaches to effectively reduce morbidity and mortality. Our data supports a stratified risk management strategy, particularly for patients with PRE and SPE. Strategies as has previously been suggested should continue to include early identification through systematic screening and risk stratification, supported by biomarkers predictive of severe disease [38,41]. Enhanced surveillance, timely initiation of antihypertensive therapy, and carefully timed delivery planning are crucial, especially for patients with PRE and SPE [41,42]. Additionally, innovative strategies such as blood pressure self-monitoring, telemedicine, and involvement of community health workers can improve patient monitoring, enhance adherence to management protocols, and address disparities in healthcare access and quality [41]. The variation in severity across HDP types may reflect underlying differences in pathophysiology, offering potential avenues for targeted research and treatments.

4.1. Limitations

Our study offers valuable insights into maternal and fetal outcomes in HDP, but has several limitations. First, the retrospective nature and reliance on inpatient data from the NIS database restricts our ability to capture long-term outcomes beyond hospitalization. Additionally, the NIS database relies on diagnostic codes for case identification and classification, which may introduce inherent limitations related to coding accuracy and completeness. Misclassification or underreporting of HDP cases, as well as other comorbidities and complications, could potentially affect the validity and generalizability of our findings. Furthermore, our study design precludes the establishment of causal relationships between hypertensive disorders and adverse outcomes. While we adjusted for various confounding factors in our regression analysis, the possibility of residual confounding remains, and causality cannot be inferred from our observational data alone. Moreover, the NIS database lacks detailed clinical information such as medication use, disease severity, obstetric history, and socioeconomic factors, which may influence outcomes but were not accounted for in our analysis. Medication use can influence outcomes, making it crucial to understand antihypertensive adherence in pregnant individuals, particularly those with CH or GHTN, who often exhibit low adherence [43–45]. Future studies incorporating more comprehensive clinical data are warranted to provide a deeper understanding of the factors contributing to adverse outcomes in HDP.

4.2. Conclusion

Pregnant patients with CH, PRE, and SPE experience higher rates of adverse maternal and fetal outcomes when compared to normotensive patients. Understanding the graded risk differences across HDP subtypes may enable more tailored interventions, optimizing maternal and fetal outcomes for those at highest risk.

CRediT authorship contribution statement

Laith Alhuneafat: Writing – review & editing, Writing – original draft, Visualization, Supervision, Project administration, Methodology, Investigation, Formal analysis, Conceptualization. **Fares Ghanem:** Writing – review & editing, Methodology, Investigation, Formal analysis. **Sneha Nandy:** Writing – review & editing, Writing – original draft. **Sana Khan:** Writing – review & editing, Writing – original draft. **Anushree Puttur:** Writing – original draft. **Ahmad Jabri:** Writing – review & editing. **Alaq Haddad:** Writing – review & editing, Writing – original draft. **Bhavadharini Ramu:** Writing – review & editing, Supervision. **Bethany Sabol:** Writing – review & editing, Validation. **Jessica Schultz:** Writing – review & editing, Validation. **Selma Carlson:** Writing – review & editing, Validation, Project administration.

Disclosure

BR: Speaker's honoraria: Abbott Inc.

Data availability

The National Inpatient Sample (NIS) is a large publicly available all-payer database in the United States. Healthcare Cost and Utilization Project Agency for Healthcare Research and Quality (HCUP AHRQ) has restrictions on data sharing based on a Data Use Agreement any questions should be directed to them.

Ethical compliance

The research qualifies as no risk or minimal risk to subjects and our institution does not require ethical approval for NIS database studies.

Study funding

No funding applied.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Bhavadharini Ramu reports a relationship with Abbott that includes: speaking and lecture fees. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Biorender.com was used to create the central illustration.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcrp.2025.200413.

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