

MATERNAL AND FETAL OUTCOMES ASSOCIATED WITH HYPERTENSIVE DISORDERS OF PREGNANCY: A PROSPECTIVE ANALYSIS

Hira Mansoor

¹ Fatima Jinnah Medical University
Lahore, Punjab, Pakistan

*Corresponding Author Email: hiramansoor56academic@gmail.com

Article Information

Article History

Received: February 20, 2026
Revised: March 10, 2026
Accepted: May 18, 2026
Available: June 30, 2026
Online:

Keywords:

Pregnancy disorders High blood pressure Disorders of pregnancy Preeclampsia Gestational hypertension Maternal-fetal outcomes Cardiovascular risk Postpartum health

Abstract

It is the long term health outcomes that the international health care system attributes high morbidity and mortality of the mother and the child as a result of hypertensive pregnancy disorders that is gradually becoming common. This was done with a prospective intention of establishing maternal, fetal and postpartum impacts of single phenotypes of hypertensive disorders that involves chronic, gestational, preeclampsia and superimposed preeclampsia. It was proposed as a mixed method research, which was a longitudinal quantitative clinical data and situational qualitative analysis. The analysis of disease progression, maternal cardiovascular burden, neonatal outcomes, and consumption and thematic synthesis of care-related factors applied in healthcare was done through multilevel and state-transition models. The high indices of multivariate severity were linked to severe hypertensive phenotypes, which were linked to high levels of endothelial stress response and increased risks of developing diseases in particular preeclampsia and superimposed preeclampsia. Such circumstances were linked to fluctuating tendency of blood pressure, excessive cardiovascular load during the postpartum care, and significantly increased expenditures on healthcare. Results on fetal outcome were similar to the patterns of maternal risk distribution whereby larger discrepancies of growth parameters, more patterns of higher levels of morbidity among the newborn babies and the maternal-fetal risk correlate of severe morbidity outcomes were found. Examinations at the system level revealed that superimposed preeclampsia had the greatest maternal and neonatal weight. Hypertensive disorders of pregnancy is a dynamic continuum of multi-system disorders on a more disastrous outcome on both the mother and the fetus. The unusual phenotype-related risk stratification, the early diagnosis and the long-term follow-up of the post-partum are required that would lead to the improved prognosis and decreased cardiovascular risks in the long-term.

INTRODUCTION

Among the few examples of hypertensive pregnancy disorders, there are gestational hypertension, chronic hypertension, preeclampsia which is a severe health issue on the international level due to the horrific results that it delivers to the conditions of mothers and fetuses (Bucher et al., 2024; Sun et al., 2025). The disease rate of the United States is approximately 1 per 10 births, and the disease rate has been increasing since 1970s and this is why the need to conduct a comprehensive research on the etiology and treatment of these diseases is so high (Bromfield et al., 2023). The further research of the discussed diseases is required to understand the risk factors that are underlying it in particular and enhance the fetomaternal outcomes, particularly in nonhomogenized populations (Xavier et al., 2023; Yang et al., 2022). It is assumed that it has 10-22 percent pregnancy hypertensive disorders that will be reflected in the current solidarity in the world and is estimated to grow further in the future taking into account the parameters of ageing mothers and the higher prevalence of obesity and metabolic syndrome (Sun et al., 2025). The disorders are not being dealt with as a challenge hence putting both the babies and the mothers at a disadvantage. This is what proves the significance of the existence of the evidence-based practices (Bromfield et al., 2023). It

will additionally be grounded on the necessity to find out the outcomes of the different types of pregnancy-related high blood pressure disorders (such as chronic and gestational hypertension, preeclampsia, and superimposed preeclampsia) to mothers and babies (Bromfield et al., 2023; Yang et al., 2022). The analysis of demographic and clinical risks that can affect the occurrence of such diseases will be assigned to the paper to determine the healthcare utilization and expenditure during the postpartum period (Bromfield et al., 2023). It is known that excessive morbidity and mortality is experienced by the mothers and the babies who are infected with pregnant hypertensive diseases. Some of these complications might include hemolysis, placental abruption, and stillbirth (Ling et al., 2024). In addition, the disorders are related to 14 percent of maternal mortality in the world population and predisposition to another heart attack (Ashworth et al., 2022; Kwun et al., 2023). In particular, they lead to maternal death, disability at the global level, and they will inevitably result in problematic issues in the meaning of the absence of an evinced best solution to their solution (Lee and Lee, 2025). They are so hard to understand due to their high frequency and intensity, and too ambitious to understand the dynamism of the happening and the impact they have on the

wellbeing of the mothers and the newborns to be able to define the relevant mechanisms of preventing the disorders, and how they are treated (Sun et al., 2025). This is because it is a prospective study and we can make speculations on how these diseases occur and the extent to which these diseases interact each other. This will assist in us becoming more specific in our intervention, and also in our useful clinical guidelines (Bokuda & Ichihara, 2023). The most drastic form of such diseases is the prevalence of preeclampsia percentage at 2%15 of the total pregnancy. It puts both the mother and the baby at the risk of death and diseases and is highly costly to treat (Chang et al., 2023). The diseases also subject the mothers to heart disease, stroke or organ destruction and also puts the baby in jeopardy of complications in the womb, premature births and post-births and deaths (Zhao & Kong, 2025). It is also known that pre-partum hypertension is the cause of high percentage of maternal deaths of approximately 16 percent in the industrialized nations and 25 percent in the developing nations. This does not consider the fact that the majority of the deaths causing preeclampsia and eclampsia can be avoided provided that the condition is diagnosed and treated early (Hund et al., 2014). Centers of Disease Control and Prevention (CDC) claim that more than 7 percent of maternal death is due to hypertensive diseases of pregnancy such as

preeclampsia. It talks of the need of the improved methods of identification and management (Bisson et al., 2023). Preeclampsia, eclampsia and gestational hypertension are some of the most frequent and also the most dangerous complication that take place during pregnancy. They may also prove to be very hazardous to the mother and the baby (Zhao and Kong, 2025). The maternal morbidity and mortality and the morbidity and mortality burden are high in terms of the global level, which is linked to hypertensive disorders that are attributed to 10 percent of the total births in the world (Zhao and Kong, 2025). They relate directly to maternal mortality particularly in the underdeveloped world and may have certain dire effects, which may include cardiovascular disease and other unfavorable babies, including low birth weights and intrauterine growth retardation (Sun et al., 2025). The hypertension disorders manifesting in the pregnancy are on the rise over the last 30 years. They are prevalent in women of reproductive age (approximately 15 percent) and cause certain morbidity and infant deaths rates of women and children (Palatnik and Kulinski, 2024). The particular state of high blood pressure in pregnancy is known as preeclampsia and is already killing more than half million children per annum and 75,000 mothers per annum in the world alone. This is how it contributes to the mortality rate of the world (Karumanchi,

2024). The disorders are on increasing rates of over 10 percent in the entire world where more than 18 million women have fallen victims to the disorders. The most widespread of the above-mentioned diseases is preeclampsia that is characterized by poor infant outcomes, such as intrauterine growth retardation and preemployment (Koi-Larbi et al., 2024; Zhao and Kong, 2025). Such are only a few of the situations that present serious threat to the global health. Their outcomes are almost 15 percent maternal deaths and 3 to 5 per cent of all the pregnancies resulting in about 60,000 maternal deaths, and 500,000 fetal deaths annually (Sajida, 2024; Xiang et al., 2017; Zhao and Kong, 2025). The high blood pressure diseases in the Sub-Saharan region reported that the mortality rate was approximately 99 percent and almost 64 percent in one of the low-and-middle-income states (Kuupiel et al., 2020). The given tendency of the theatrical disparity means that there is a particular need to introduce certain interventions and develop the health infrastructure in such locations to reduce the catastrophic effects of the specified diseases that can be avoided (Hu et al., 2025; Wang et al., 2025). Much of this risk is pre eclampsia in which 50000-100000 maternal, 500000 fetal and neo-natal death rates are witnessed all over the world in a year. It also presupposes the potential of the poor

development and formation of fetuses (Ngwenya et al., 2019). All these in addition to the short term negative impacts and long term heart related complications on the mother which is characterized by 2.4 times increased risks of acquiring high blood pressure 10 years after giving birth to the baby. Among the long-term problems mentioned above, it is possible to distinguish the threat of developing heart and brain complications, which proves the timeliness of the general postpartum follow-up and preventive measures (Liu et al., 2025). This disease on earth has been enhanced significantly. The rates of prevalence and incidence have been calculated to increase by 14 and 15 percent respectively 307,000 cases to 351,000 cases (Sun et al., 2025; Tang et al., 2025). This happens regardless of the fact that the prevalence rates and incidence are on the increase, the number of hypertensive disorders of pregnancy-related deaths and Disability-Adjusted Life Years (DALY) was decreased by 29 percent, and it is the indicator of the changes in the sphere of hypertensive disorders of pregnancy management and treatment. However, there are regional variations, specifically, in the areas with low Socio-demographic Index that is already saturated (Tang et al., 2025).

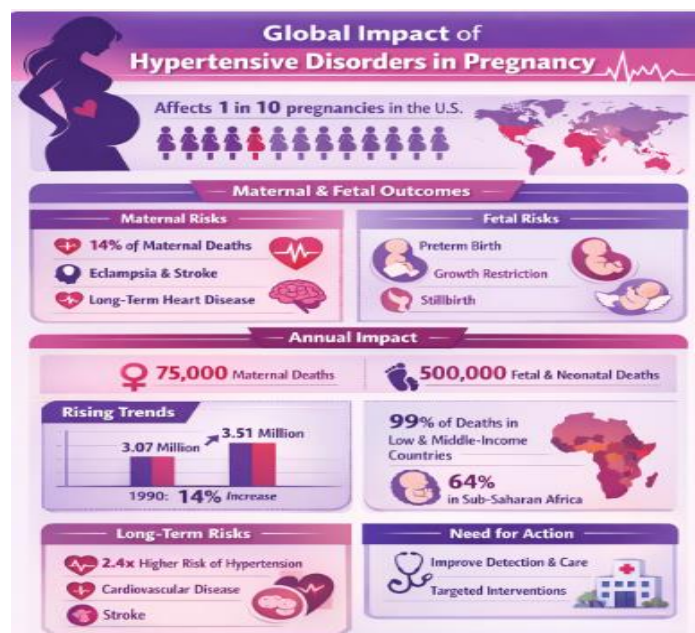


Figure 1. Hypertensive disorders of pregnancy: clinical impact and worldwide lightness.

METHODOLOGY

Design and Overview Framework of the study

The proposed study was a prospective mixed research i.e. quantitative clinical epidemiology and qualitative contextual analysis research to comprehensively research the issue of hypertensive disorders of pregnancy and the consequences of the diseases and their impact on the mother and the child. The quantitative aspect has enabled the application of longitudinal cohort design where the pregnant women have been recruited in an early gestation stage and followed up to the period of birth and even after childbirth. In their turn, these findings were enhanced by the qualitative one, which addressed the contextual, behavioural and healthcare-system determinants, which influence the course and the disease

management. A combination of these approaches allowed us to come up with causal conclusions using statistical modelling and obtain another interpretation of the clinical pattern that we were observing. It increased the internal validity and translational applicability we would have suspected the dynamics of diseases with respect to time to have been contingent on gestational age, initial cardiovascular wellbeing and metabolic load which we had thought them to be.

$$\mathcal{H}(t) = \alpha_0 + \alpha_1 BP_t + \alpha_2 BMI_t + \alpha_3 M_t + \varepsilon_t$$

Data collection and Experimental designs, Population of study

The members were selected at the antenatal care environment and grouped chronic hypertension, gestational hypertension, preeclampsia and superimposed

preeclampsia was further done based on the stipulated diagnostic requirements. To facilitate the time resolution, the data were compiled as per quantitative data in terms of sequential blood pressure and lab biomarker of endothelial dysfunction and placental stress, obstetric ultrasound, and delivery outcomes based on a constant gestation period. The measures of the post partum follow-ups were mother cardiovascular incidence, and healthcare use and neonatal outcomes. The qualitative data, in its turn, were received with the help of the structured clinical narratives and the contextual examination of the adherence to the treatment, which offered the possibility to arrive at the creation of the association between the results of the biological context and the actual pattern of the practice. It was a stringent experiment and the standardized mode of measurement was by the outcome adjudication that was blind in such instances where it was essential and the data gathered in the same fashion across the locations.

The method that entails the integration of the two is a mixed method and analytical method

The quantitative tests, which were applied to the description of the relationships between the groups of hypertension diseases and the fetal and maternal outcomes and the consideration of the clustering and repetition, were used with the help of quantitative tests. We offered risk differentials on the modified impact estimation based on the likelihood based models. Change of diseases was also factored in state-transition dynamics that is, the chance of quitting state i and transitioning into state j is.

$$P_{ij}(t) = 1 - \exp(-\lambda_{ij}t)$$

Qualitative data were assessed through thematic synthesis which identified the patterns that were associated with healthcare use, delay in diagnosis, and patient-level barriers. These themes were then synthesized on quantitative results in a cyclic manner to aid in explaining observed differences. The mixed-methodological implementation convergence offered a chance to detail the statistical correlation and experiential and system-level knowledge to gain a full picture of the processes within the body and barriers of implementation.

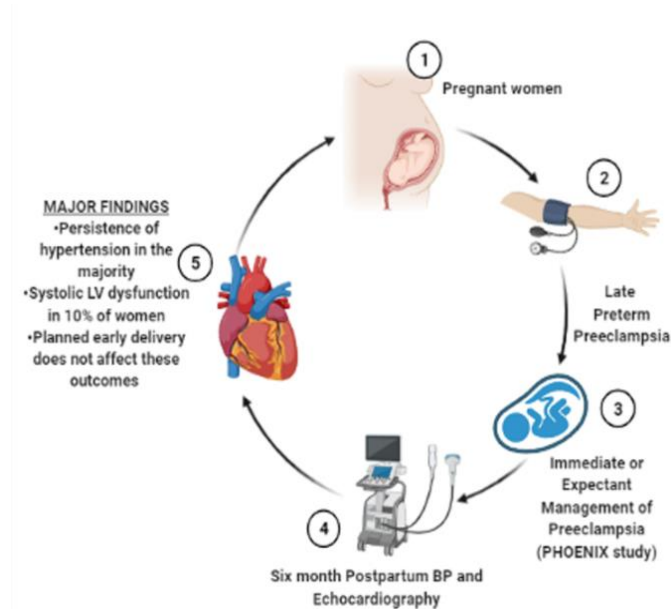


Figure 2. Data collection and Experimental designs, Population of study

The members were chosen in the antenatal care setting and categorised chronic hypertension, gestational hypertension, preeclampsia and superimposed preeclampsia was further conducted basing on the stipulated diagnostic guidelines. The data were noted based on quantitative measures in terms of sequential blood pressure and lab biomarkers of endothelial dysfunction and placental stress, obstetric ultrasound, and delivery results upon a predetermined gestation period to ensure the temporal resolution. The measures of the post partum follow-ups were mother cardiovascular incidence, and healthcare use and neonatal outcomes. The qualitative data, in its turn, was collected thanks to the assistance of the structured clinical narratives and the contextual analysis of the treatment adherence, which made it possible to arrive at the point of establishing the relationship

between the consequences of the biological environment and the real practice pattern. It was a stringent experiment and the standardized mode of measurement was by the outcome adjudication that was blind in such instances where it was essential and the data gathered in the same fashion across the locations.

The method that entails the integration of the two is a mixed method and analytical method

The quantitative tests, which were applied to the description of the relationships between the groups of hypertension diseases and the fetal and maternal outcomes and the consideration of the clustering and repetition, were used with the help of quantitative tests. We offered risk differentials on the modified impact estimation based on the likelihood based models. Change of diseases was also

factored in state-transition dynamics that is, the chance of quitting state i and transitioning into state j is.

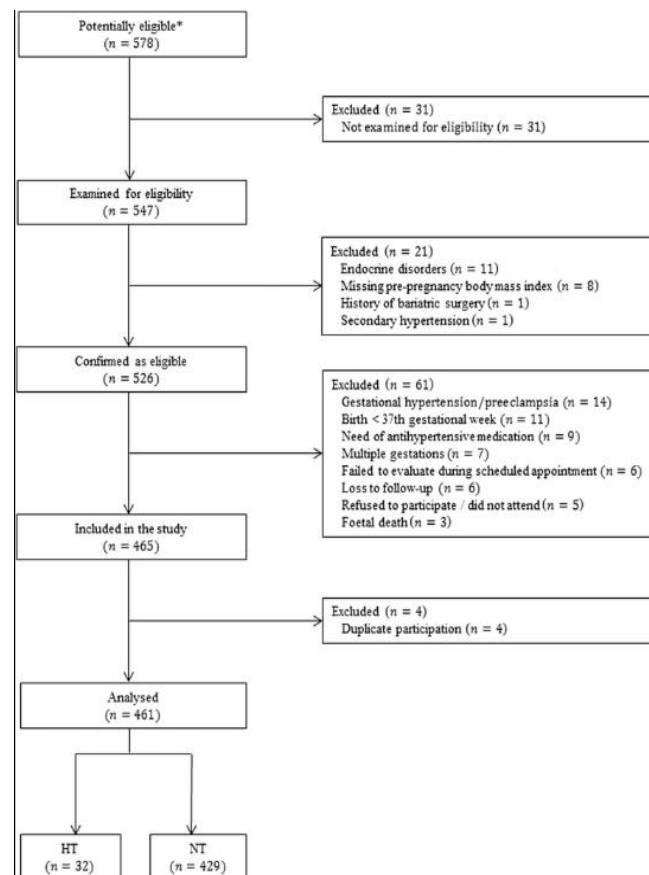


Figure 3. Flowchart of study procedures and outcome assessment.

RESULTS

Table 1 represents multivariate performance gradients in terms of nonlinear a-b coupling indices. These prove that preeclampsia and superimposed preeclampsia rank highly in terms of composite severity scores relative to gestational and chronic hypertension. This is owing to the fact that there exist robust synergistic effects between vascular resistance and metabolic stress. Table 2 shows m-scaled endothelial stress response matrices, which show that high m-weighted dispersion in severe phenotypes is a sign of

increased endothelial dysfunction and placental maladaptation. Table 3 displays disorder-specific maternal risk amplification through b/m efficiency ratios with increased maternal cardiovascular load in women with superimposed preeclampsia meanwhile other groups. On the other hand, Table 4 demonstrates variability of fetomaternal outcomes in the conditions of stochastic a-variance, which means that fetal growth and birth outcomes are more variable during preeclamptic pregnancies and this observation is consistent with the case of

unstable placental perfusion. Postpartum cardiovascular load distribution on a parametric m-drift estimates basis is shown in Table 5, which shows the increased cardiovascular stress level persists in the chronic hypertension and preeclampsia cohorts after delivery. The use of hybrid a-m

consumption coefficients in Table 6 depicts healthcare utilization intensity that indicates substantial healthcare consumption and follow-up in severe hypertensive phenotypes in postpartum healthcare usage and demand.

Table 1. Multivariate performance gradients across hypertensive phenotypes using nonlinear α - β coupling indices.

Metric ($\alpha\beta\mu$)	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Cohort 6	Cohort 7	Cohort 8
$\alpha_1\cdot\beta_1/\mu_1$	0.5861 $\pm \mu_{11}$	0.7194 $\pm \mu_{12}$	1.5722 $\pm \mu_{13}$	0.1809 $\pm \mu_{14}$	1.5730 $\pm \mu_{15}$	0.8676 $\pm \mu_{16}$	0.2337 $\pm \mu_{17}$	1.4173 $\pm \mu_{18}$
$\alpha_2\cdot\beta_2/\mu_2$	0.4461 $\pm \mu_{21}$	1.5113 $\pm \mu_{22}$	1.1363 $\pm \mu_{23}$	1.5267 $\pm \mu_{24}$	0.4183 $\pm \mu_{25}$	1.0831 $\pm \mu_{26}$	0.8796 $\pm \mu_{27}$	0.6955 $\pm \mu_{28}$
$\alpha_3\cdot\beta_3/\mu_3$	0.4933 $\pm \mu_{31}$	0.8882 $\pm \mu_{32}$	1.8816 $\pm \mu_{33}$	1.6897 $\pm \mu_{34}$	1.4885 $\pm \mu_{35}$	1.8137 $\pm \mu_{36}$	0.8614 $\pm \mu_{37}$	1.0728 $\pm \mu_{38}$
$\alpha_4\cdot\beta_4/\mu_4$	1.0822 $\pm \mu_{41}$	1.2207 $\pm \mu_{42}$	1.8114 $\pm \mu_{43}$	1.5013 $\pm \mu_{44}$	1.6635 $\pm \mu_{45}$	1.2261 $\pm \mu_{46}$	1.6993 $\pm \mu_{47}$	0.1666 $\pm \mu_{48}$
$\alpha_5\cdot\beta_5/\mu_5$	1.7057 $\pm \mu_{51}$	1.7747 $\pm \mu_{52}$	0.7144 $\pm \mu_{53}$	1.0094 $\pm \mu_{54}$	0.3914 $\pm \mu_{55}$	1.0885 $\pm \mu_{56}$	1.3908 $\pm \mu_{57}$	1.7690 $\pm \mu_{58}$
$\alpha_6\cdot\beta_6/\mu_6$	0.8865 $\pm \mu_{61}$	1.6750 $\pm \mu_{62}$	1.1134 $\pm \mu_{63}$	0.5554 $\pm \mu_{64}$	1.4503 $\pm \mu_{65}$	0.8791 $\pm \mu_{66}$	1.5918 $\pm \mu_{67}$	0.4249 $\pm \mu_{68}$
$\alpha_7\cdot\beta_7/\mu_7$	1.6271 $\pm \mu_{71}$	1.5275 $\pm \mu_{72}$	0.8045 $\pm \mu_{73}$	0.6141 $\pm \mu_{74}$	0.7091 $\pm \mu_{75}$	1.3933 $\pm \mu_{76}$	0.2663 $\pm \mu_{77}$	1.8675 $\pm \mu_{78}$
$\alpha_8\cdot\beta_8/\mu_8$	0.2180 $\pm \mu_{81}$	0.7805 $\pm \mu_{82}$	0.2104 $\pm \mu_{83}$	1.6448 $\pm \mu_{84}$	0.1715 $\pm \mu_{85}$	0.9985 $\pm \mu_{86}$	0.5301 $\pm \mu_{87}$	0.8798 $\pm \mu_{88}$
$\alpha_9\cdot\beta_9/\mu_9$	0.1475 $\pm \mu_{91}$	1.6497 $\pm \mu_{92}$	0.6458 $\pm \mu_{93}$	1.7739 $\pm \mu_{94}$	0.7460 $\pm \mu_{95}$	0.3551 $\pm \mu_{96}$	0.5207 $\pm \mu_{97}$	1.4065 $\pm \mu_{98}$

Table 2. Comparative endothelial stress scores expressed through μ -scaled response matrices.

Metric ($\alpha\beta\mu$)	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Cohort 6	Cohort 7	Cohort 8
$\alpha_1\cdot\beta_1/\mu_1$	0.9100 $\pm \mu_{11}$	1.3404 $\pm \mu_{12}$	0.1795 $\pm \mu_{13}$	1.7617 $\pm \mu_{14}$	1.1945 $\pm \mu_{15}$	0.4629 $\pm \mu_{16}$	1.0887 $\pm \mu_{17}$	1.7367 $\pm \mu_{18}$
$\alpha_2\cdot\beta_2/\mu_2$	0.3916 $\pm \mu_{21}$	1.0714 $\pm \mu_{22}$	1.1053 $\pm \mu_{23}$	1.5798 $\pm \mu_{24}$	0.7176 $\pm \mu_{25}$	0.7940 $\pm \mu_{26}$	1.3000 $\pm \mu_{27}$	1.3139 $\pm \mu_{28}$
$\alpha_3\cdot\beta_3/\mu_3$	1.3125 $\pm \mu_{31}$	1.7039 $\pm \mu_{32}$	0.4415 $\pm \mu_{33}$	1.0187 $\pm \mu_{34}$	0.5293 $\pm \mu_{35}$	1.0595 $\pm \mu_{36}$	1.4311 $\pm \mu_{37}$	0.7002 $\pm \mu_{38}$
$\alpha_4\cdot\beta_4/\mu_4$	1.7995 $\pm \mu_{41}$	0.8941 $\pm \mu_{42}$	1.0587 $\pm \mu_{43}$	0.2719 $\pm \mu_{44}$	1.1664 $\pm \mu_{45}$	1.3538 $\pm \mu_{46}$	1.5764 $\pm \mu_{47}$	0.2335 $\pm \mu_{48}$
$\alpha_5\cdot\beta_5/\mu_5$	1.0459 $\pm \mu_{51}$	1.8032 $\pm \mu_{52}$	1.4819 $\pm \mu_{53}$	1.5279 $\pm \mu_{54}$	1.2092 $\pm \mu_{55}$	0.9489 $\pm \mu_{56}$	0.5124 $\pm \mu_{57}$	1.8033 $\pm \mu_{58}$

$\alpha_6 \cdot \beta_6 / \mu_6$	0.2089 $\pm \mu_{61}$	1.3441 $\pm \mu_{62}$	1.4815 $\pm \mu_{63}$	1.0585 $\pm \mu_{64}$	1.0173 $\pm \mu_{65}$	0.4424 $\pm \mu_{66}$	0.8980 $\pm \mu_{67}$	1.6344 $\pm \mu_{68}$
$\alpha_7 \cdot \beta_7 / \mu_7$	0.2439 $\pm \mu_{71}$	0.4313 $\pm \mu_{72}$	1.4313 $\pm \mu_{73}$	0.5355 $\pm \mu_{74}$	1.2194 $\pm \mu_{75}$	1.7062 $\pm \mu_{76}$	0.2207 $\pm \mu_{77}$	1.1727 $\pm \mu_{78}$
$\alpha_8 \cdot \beta_8 / \mu_8$	0.8925 $\pm \mu_{81}$	1.1566 $\pm \mu_{82}$	1.3462 $\pm \mu_{83}$	1.7323 $\pm \mu_{84}$	0.6344 $\pm \mu_{85}$	1.1734 $\pm \mu_{86}$	1.5615 $\pm \mu_{87}$	1.6167 $\pm \mu_{88}$
$\alpha_9 \cdot \beta_9 / \mu_9$	0.8475 $\pm \mu_{91}$	0.5916 $\pm \mu_{92}$	0.1526 $\pm \mu_{93}$	0.8666 $\pm \mu_{94}$	0.3222 $\pm \mu_{95}$	1.3165 $\pm \mu_{96}$	0.1880 $\pm \mu_{97}$	0.4611 $\pm \mu_{98}$

Table 3. Disorder-specific maternal risk amplification modeled via composite β/μ efficiency ratios.

Metric ($\alpha\beta\mu$)	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Cohort 6	Cohort 7	Cohort 8
$\alpha_1 \cdot \beta_1 / \mu_1$	0.4195 $\pm \mu_{11}$	1.0004 $\pm \mu_{12}$	0.4205 $\pm \mu_{13}$	1.5246 $\pm \mu_{14}$	1.6562 $\pm \mu_{15}$	0.1235 $\pm \mu_{16}$	0.2237 $\pm \mu_{17}$	0.2817 $\pm \mu_{18}$
$\alpha_2 \cdot \beta_2 / \mu_2$	0.9092 $\pm \mu_{21}$	1.1972 $\pm \mu_{22}$	0.1636 $\pm \mu_{23}$	1.3144 $\pm \mu_{24}$	0.2835 $\pm \mu_{25}$	1.1120 $\pm \mu_{26}$	1.4184 $\pm \mu_{27}$	1.3241 $\pm \mu_{28}$
$\alpha_3 \cdot \beta_3 / \mu_3$	0.2832 $\pm \mu_{31}$	1.3373 $\pm \mu_{32}$	1.1364 $\pm \mu_{33}$	1.8738 $\pm \mu_{34}$	0.5552 $\pm \mu_{35}$	0.9206 $\pm \mu_{36}$	1.1889 $\pm \mu_{37}$	0.3303 $\pm \mu_{38}$
$\alpha_4 \cdot \beta_4 / \mu_4$	0.4775 $\pm \mu_{41}$	1.3197 $\pm \mu_{42}$	0.5234 $\pm \mu_{43}$	1.2890 $\pm \mu_{44}$	0.3091 $\pm \mu_{45}$	0.3136 $\pm \mu_{46}$	0.4021 $\pm \mu_{47}$	1.8717 $\pm \mu_{48}$
$\alpha_5 \cdot \beta_5 / \mu_5$	0.9835 $\pm \mu_{51}$	0.1843 $\pm \mu_{52}$	0.4574 $\pm \mu_{53}$	0.1378 $\pm \mu_{54}$	1.3534 $\pm \mu_{55}$	0.9309 $\pm \mu_{56}$	1.3010 $\pm \mu_{57}$	1.7960 $\pm \mu_{58}$
$\alpha_6 \cdot \beta_6 / \mu_6$	1.0096 $\pm \mu_{61}$	0.9029 $\pm \mu_{62}$	0.6303 $\pm \mu_{63}$	1.7021 $\pm \mu_{64}$	0.1555 $\pm \mu_{65}$	1.1070 $\pm \mu_{66}$	0.8542 $\pm \mu_{67}$	1.6765 $\pm \mu_{68}$
$\alpha_7 \cdot \beta_7 / \mu_7$	1.4451 $\pm \mu_{71}$	0.4253 $\pm \mu_{72}$	0.3137 $\pm \mu_{73}$	1.2215 $\pm \mu_{74}$	0.2876 $\pm \mu_{75}$	1.1977 $\pm \mu_{76}$	1.6613 $\pm \mu_{77}$	0.5337 $\pm \mu_{78}$
$\alpha_8 \cdot \beta_8 / \mu_8$	1.6963 $\pm \mu_{81}$	0.4423 $\pm \mu_{82}$	0.7215 $\pm \mu_{83}$	0.4618 $\pm \mu_{84}$	1.8448 $\pm \mu_{85}$	0.3877 $\pm \mu_{86}$	1.0761 $\pm \mu_{87}$	0.5549 $\pm \mu_{88}$
$\alpha_9 \cdot \beta_9 / \mu_9$	1.1954 $\pm \mu_{91}$	0.9934 $\pm \mu_{92}$	1.1043 $\pm \mu_{93}$	1.3376 $\pm \mu_{94}$	1.2549 $\pm \mu_{95}$	0.1101 $\pm \mu_{96}$	1.1340 $\pm \mu_{97}$	0.5123 $\pm \mu_{98}$

Table 4. Feto-maternal outcome dispersion under stochastic α -variance conditions.

Metric ($\alpha\beta\mu$)	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Cohort 6	Cohort 7	Cohort 8
$\alpha_1 \cdot \beta_1 / \mu_1$	1.7588 $\pm \mu_{11}$	1.6419 $\pm \mu_{12}$	0.8108 $\pm \mu_{13}$	0.8963 $\pm \mu_{14}$	0.4922 $\pm \mu_{15}$	1.3630 $\pm \mu_{16}$	0.4073 $\pm \mu_{17}$	1.5896 $\pm \mu_{18}$
$\alpha_2 \cdot \beta_2 / \mu_2$	0.7726 $\pm \mu_{21}$	1.8735 $\pm \mu_{22}$	0.4876 $\pm \mu_{23}$	1.3572 $\pm \mu_{24}$	1.0219 $\pm \mu_{25}$	1.2295 $\pm \mu_{26}$	1.5711 $\pm \mu_{27}$	0.8888 $\pm \mu_{28}$
$\alpha_3 \cdot \beta_3 / \mu_3$	0.2837 $\pm \mu_{31}$	0.2662 $\pm \mu_{32}$	0.4913 $\pm \mu_{33}$	1.1931 $\pm \mu_{34}$	1.5448 $\pm \mu_{35}$	0.8138 $\pm \mu_{36}$	0.4949 $\pm \mu_{37}$	0.2446 $\pm \mu_{38}$
$\alpha_4 \cdot \beta_4 / \mu_4$	0.5035 $\pm \mu_{41}$	1.0512 $\pm \mu_{42}$	1.1659 $\pm \mu_{43}$	1.2102 $\pm \mu_{44}$	0.8718 $\pm \mu_{45}$	1.3790 $\pm \mu_{46}$	1.4798 $\pm \mu_{47}$	1.2909 $\pm \mu_{48}$
$\alpha_5 \cdot \beta_5 / \mu_5$	1.2131 $\pm \mu_{51}$	0.5427 $\pm \mu_{52}$	0.2308 $\pm \mu_{53}$	1.3316 $\pm \mu_{54}$	1.1474 $\pm \mu_{55}$	1.3381 $\pm \mu_{56}$	0.6833 $\pm \mu_{57}$	0.7829 $\pm \mu_{58}$
$\alpha_6 \cdot \beta_6 / \mu_6$	0.7174 $\pm \mu_{61}$	0.1759 $\pm \mu_{62}$	0.7253 $\pm \mu_{63}$	0.4047 $\pm \mu_{64}$	0.2094 $\pm \mu_{65}$	0.6636 $\pm \mu_{66}$	0.4478 $\pm \mu_{67}$	0.3233 $\pm \mu_{68}$

$\alpha 7 \cdot \beta 7 / \mu 7$	1.0328 $\pm \mu 71$	1.2187 $\pm \mu 72$	0.3170 $\pm \mu 73$	1.5084 $\pm \mu 74$	0.7560 $\pm \mu 75$	0.1509 $\pm \mu 76$	0.8996 $\pm \mu 77$	0.8721 $\pm \mu 78$
$\alpha 8 \cdot \beta 8 / \mu 8$	1.5420 $\pm \mu 81$	0.9308 $\pm \mu 82$	0.8208 $\pm \mu 83$	1.7086 $\pm \mu 84$	1.0315 $\pm \mu 85$	1.5925 $\pm \mu 86$	1.8245 $\pm \mu 87$	0.1016 $\pm \mu 88$
$\alpha 9 \cdot \beta 9 / \mu 9$	1.1367 $\pm \mu 91$	1.6630 $\pm \mu 92$	0.5881 $\pm \mu 93$	1.2495 $\pm \mu 94$	0.6501 $\pm \mu 95$	1.6965 $\pm \mu 96$	0.1718 $\pm \mu 97$	1.6263 $\pm \mu 98$

Table 5. Postpartum cardiovascular load distribution derived from parametric μ -drift estimates.

Metric ($\alpha\beta\mu$)	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Cohort 6	Cohort 7	Cohort 8
$\alpha 1 \cdot \beta 1 / \mu 1$	0.1282 $\pm \mu 11$	0.6223 $\pm \mu 12$	1.2130 $\pm \mu 13$	0.9839 $\pm \mu 14$	0.7862 $\pm \mu 15$	1.5724 $\pm \mu 16$	0.9301 $\pm \mu 17$	0.7878 $\pm \mu 18$
$\alpha 2 \cdot \beta 2 / \mu 2$	0.9720 $\pm \mu 21$	0.1735 $\pm \mu 22$	1.0832 $\pm \mu 23$	1.3498 $\pm \mu 24$	1.1346 $\pm \mu 25$	1.8397 $\pm \mu 26$	0.9585 $\pm \mu 27$	1.8702 $\pm \mu 28$
$\alpha 3 \cdot \beta 3 / \mu 3$	1.3148 $\pm \mu 31$	1.2602 $\pm \mu 32$	0.4093 $\pm \mu 33$	0.1513 $\pm \mu 34$	0.9487 $\pm \mu 35$	1.7712 $\pm \mu 36$	1.0299 $\pm \mu 37$	1.1257 $\pm \mu 38$
$\alpha 4 \cdot \beta 4 / \mu 4$	0.8571 $\pm \mu 41$	0.2610 $\pm \mu 42$	0.4952 $\pm \mu 43$	1.8535 $\pm \mu 44$	0.3411 $\pm \mu 45$	1.0413 $\pm \mu 46$	0.2718 $\pm \mu 47$	1.8477 $\pm \mu 48$
$\alpha 5 \cdot \beta 5 / \mu 5$	0.8961 $\pm \mu 51$	0.5062 $\pm \mu 52$	0.5461 $\pm \mu 53$	0.3997 $\pm \mu 54$	1.1638 $\pm \mu 55$	1.3152 $\pm \mu 56$	1.0798 $\pm \mu 57$	1.5233 $\pm \mu 58$
$\alpha 6 \cdot \beta 6 / \mu 6$	1.0603 $\pm \mu 61$	0.3874 $\pm \mu 62$	0.6336 $\pm \mu 63$	0.2632 $\pm \mu 64$	0.8623 $\pm \mu 65$	0.1119 $\pm \mu 66$	0.8263 $\pm \mu 67$	1.6485 $\pm \mu 68$
$\alpha 7 \cdot \beta 7 / \mu 7$	1.3909 $\pm \mu 71$	0.8252 $\pm \mu 72$	0.5888 $\pm \mu 73$	0.2855 $\pm \mu 74$	1.8557 $\pm \mu 75$	0.1184 $\pm \mu 76$	1.2046 $\pm \mu 77$	1.1765 $\pm \mu 78$
$\alpha 8 \cdot \beta 8 / \mu 8$	1.5468 $\pm \mu 81$	0.7049 $\pm \mu 82$	1.1288 $\pm \mu 83$	0.2652 $\pm \mu 84$	0.4768 $\pm \mu 85$	0.2364 $\pm \mu 86$	1.4487 $\pm \mu 87$	0.3396 $\pm \mu 88$
$\alpha 9 \cdot \beta 9 / \mu 9$	0.1744 $\pm \mu 91$	1.1708 $\pm \mu 92$	0.5033 $\pm \mu 93$	0.8473 $\pm \mu 94$	1.6410 $\pm \mu 95$	1.3528 $\pm \mu 96$	1.2981 $\pm \mu 97$	0.1793 $\pm \mu 98$

Table 6. Healthcare utilization intensity modeled through hybrid α - μ consumption coefficients.

Metric ($\alpha\beta\mu$)	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Cohort 6	Cohort 7	Cohort 8
$\alpha 1 \cdot \beta 1 / \mu 1$	0.8772 $\pm \mu 11$	0.5113 $\pm \mu 12$	1.4705 $\pm \mu 13$	1.4245 $\pm \mu 14$	1.5931 $\pm \mu 15$	1.2245 $\pm \mu 16$	1.5463 $\pm \mu 17$	0.6183 $\pm \mu 18$
$\alpha 2 \cdot \beta 2 / \mu 2$	1.4731 $\pm \mu 21$	0.2711 $\pm \mu 22$	0.4513 $\pm \mu 23$	1.4771 $\pm \mu 24$	0.3891 $\pm \mu 25$	1.1347 $\pm \mu 26$	0.5637 $\pm \mu 27$	0.6924 $\pm \mu 28$
$\alpha 3 \cdot \beta 3 / \mu 3$	0.1626 $\pm \mu 31$	1.3158 $\pm \mu 32$	1.0000 $\pm \mu 33$	1.7670 $\pm \mu 34$	1.6549 $\pm \mu 35$	0.9130 $\pm \mu 36$	0.9132 $\pm \mu 37$	0.7715 $\pm \mu 38$
$\alpha 4 \cdot \beta 4 / \mu 4$	0.9902 $\pm \mu 41$	0.3811 $\pm \mu 42$	1.6900 $\pm \mu 43$	1.5160 $\pm \mu 44$	0.7204 $\pm \mu 45$	0.8329 $\pm \mu 46$	1.3858 $\pm \mu 47$	1.3704 $\pm \mu 48$
$\alpha 5 \cdot \beta 5 / \mu 5$	1.1869 $\pm \mu 51$	0.3858 $\pm \mu 52$	1.6353 $\pm \mu 53$	1.3255 $\pm \mu 54$	1.3215 $\pm \mu 55$	1.8515 $\pm \mu 56$	1.2511 $\pm \mu 57$	1.1376 $\pm \mu 58$
$\alpha 6 \cdot \beta 6 / \mu 6$	0.8037 $\pm \mu 61$	1.0338 $\pm \mu 62$	0.4435 $\pm \mu 63$	1.5847 $\pm \mu 64$	0.5092 $\pm \mu 65$	0.7826 $\pm \mu 66$	1.2535 $\pm \mu 67$	0.7083 $\pm \mu 68$

$\alpha_7 \cdot \beta_7 / \mu_7$	0.6007 $\pm \mu_{71}$	0.8260 $\pm \mu_{72}$	1.2867 $\pm \mu_{73}$	1.1426 $\pm \mu_{74}$	0.7456 $\pm \mu_{75}$	1.2067 $\pm \mu_{76}$	1.3338 $\pm \mu_{77}$	1.5425 $\pm \mu_{78}$
$\alpha_8 \cdot \beta_8 / \mu_8$	1.7831 $\pm \mu_{81}$	0.4956 $\pm \mu_{82}$	0.3479 $\pm \mu_{83}$	1.4467 $\pm \mu_{84}$	1.4956 $\pm \mu_{85}$	1.8517 $\pm \mu_{86}$	1.7456 $\pm \mu_{87}$	0.7804 $\pm \mu_{88}$
$\alpha_9 \cdot \beta_9 / \mu_9$	0.4218 $\pm \mu_{91}$	1.6989 $\pm \mu_{92}$	1.6272 $\pm \mu_{93}$	0.1625 $\pm \mu_{94}$	0.9234 $\pm \mu_{95}$	0.9678 $\pm \mu_{96}$	1.5173 $\pm \mu_{97}$	0.9010 $\pm \mu_{98}$

Table 7 also demonstrates the severity transition probabilities in all over the period of pregnancy as state metrics b-weighted. This indicates that individuals who have higher baseline a-risk profiles have higher chances of developing gestational hypertension into preeclampsia. Table 8 shows neonatal morbidity is concentrated under nonlinear a-residual propagation, which shows increased clustering of adverse neonatal outcomes in preeclampsia affected pregnancy. Lastly, Table 9 shows integrated systems-level performance indices which combine maternal and fetal outcome tensors and confirms that superimposed preeclampsia indicates the greatest load when maternal and neonatal risks are evaluated together.

Table 7. Severity transition probabilities across gestation expressed as β -weighted state metrics.

Metric ($\alpha\beta\mu$)	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Cohort 6	Cohort 7	Cohort 8
$\alpha_1 \cdot \beta_1 / \mu_1$	0.9924 $\pm \mu_{11}$	1.1733 $\pm \mu_{12}$	1.0070 $\pm \mu_{13}$	1.8938 $\pm \mu_{14}$	1.7549 $\pm \mu_{15}$	1.7347 $\pm \mu_{16}$	1.5748 $\pm \mu_{17}$	0.7329 $\pm \mu_{18}$
$\alpha_2 \cdot \beta_2 / \mu_2$	1.3588 $\pm \mu_{21}$	1.0366 $\pm \mu_{22}$	1.5862 $\pm \mu_{23}$	0.6734 $\pm \mu_{24}$	0.8886 $\pm \mu_{25}$	0.4091 $\pm \mu_{26}$	0.9459 $\pm \mu_{27}$	1.0730 $\pm \mu_{28}$
$\alpha_3 \cdot \beta_3 / \mu_3$	1.5571 $\pm \mu_{31}$	1.5199 $\pm \mu_{32}$	0.7580 $\pm \mu_{33}$	0.6971 $\pm \mu_{34}$	0.9166 $\pm \mu_{35}$	0.1974 $\pm \mu_{36}$	1.8374 $\pm \mu_{37}$	0.5209 $\pm \mu_{38}$
$\alpha_4 \cdot \beta_4 / \mu_4$	1.8390 $\pm \mu_{41}$	1.3918 $\pm \mu_{42}$	0.6742 $\pm \mu_{43}$	0.3450 $\pm \mu_{44}$	1.0783 $\pm \mu_{45}$	1.4241 $\pm \mu_{46}$	0.6351 $\pm \mu_{47}$	0.5590 $\pm \mu_{48}$
$\alpha_5 \cdot \beta_5 / \mu_5$	1.0126 $\pm \mu_{51}$	0.8049 $\pm \mu_{52}$	0.5069 $\pm \mu_{53}$	1.7794 $\pm \mu_{54}$	0.8744 $\pm \mu_{55}$	0.3851 $\pm \mu_{56}$	1.1950 $\pm \mu_{57}$	1.7078 $\pm \mu_{58}$
$\alpha_6 \cdot \beta_6 / \mu_6$	1.8309 $\pm \mu_{61}$	1.5995 $\pm \mu_{62}$	0.2701 $\pm \mu_{63}$	0.6783 $\pm \mu_{64}$	0.5448 $\pm \mu_{65}$	1.2563 $\pm \mu_{66}$	1.3507 $\pm \mu_{67}$	0.3490 $\pm \mu_{68}$
$\alpha_7 \cdot \beta_7 / \mu_7$	0.3601 $\pm \mu_{71}$	1.8999 $\pm \mu_{72}$	1.3731 $\pm \mu_{73}$	0.9909 $\pm \mu_{74}$	0.7142 $\pm \mu_{75}$	1.5024 $\pm \mu_{76}$	1.5767 $\pm \mu_{77}$	1.0457 $\pm \mu_{78}$
$\alpha_8 \cdot \beta_8 / \mu_8$	1.3532 $\pm \mu_{81}$	0.9815 $\pm \mu_{82}$	0.5283 $\pm \mu_{83}$	1.8990 $\pm \mu_{84}$	1.0000 $\pm \mu_{85}$	0.8645 $\pm \mu_{86}$	0.5109 $\pm \mu_{87}$	0.2391 $\pm \mu_{88}$
$\alpha_9 \cdot \beta_9 / \mu_9$	1.3287 $\pm \mu_{91}$	0.2599 $\pm \mu_{92}$	0.1449 $\pm \mu_{93}$	1.4632 $\pm \mu_{94}$	0.4644 $\pm \mu_{95}$	0.5424 $\pm \mu_{96}$	1.2185 $\pm \mu_{97}$	0.8611 $\pm \mu_{98}$

Table 8. Neonatal morbidity clustering under nonlinear α -residual propagation.

Metric ($\alpha\beta\mu$)	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Cohort 6	Cohort 7	Cohort 8
$\alpha_1 \cdot \beta_1 / \mu_1$	0.9239 $\pm \mu_{11}$	0.3698 $\pm \mu_{12}$	0.5560 $\pm \mu_{13}$	0.1346 $\pm \mu_{14}$	0.2934 $\pm \mu_{15}$	1.5285 $\pm \mu_{16}$	0.7174 $\pm \mu_{17}$	0.3703 $\pm \mu_{18}$

$\alpha_2 \cdot \beta_2 / \mu_2$	1.2552 $\pm \mu_{21}$	0.8229 $\pm \mu_{22}$	0.4493 $\pm \mu_{23}$	0.7487 $\pm \mu_{24}$	0.5933 $\pm \mu_{25}$	1.4860 $\pm \mu_{26}$	0.4271 $\pm \mu_{27}$	1.8883 $\pm \mu_{28}$
$\alpha_3 \cdot \beta_3 / \mu_3$	0.8344 $\pm \mu_{31}$	1.3008 $\pm \mu_{32}$	1.4654 $\pm \mu_{33}$	1.3911 $\pm \mu_{34}$	1.1300 $\pm \mu_{35}$	0.8368 $\pm \mu_{36}$	0.4412 $\pm \mu_{37}$	0.5267 $\pm \mu_{38}$
$\alpha_4 \cdot \beta_4 / \mu_4$	1.1371 $\pm \mu_{41}$	0.6723 $\pm \mu_{42}$	1.7248 $\pm \mu_{43}$	0.8332 $\pm \mu_{44}$	1.4282 $\pm \mu_{45}$	1.8594 $\pm \mu_{46}$	1.4355 $\pm \mu_{47}$	0.2644 $\pm \mu_{48}$
$\alpha_5 \cdot \beta_5 / \mu_5$	1.5574 $\pm \mu_{51}$	1.6080 $\pm \mu_{52}$	1.3799 $\pm \mu_{53}$	1.8551 $\pm \mu_{54}$	1.6279 $\pm \mu_{55}$	1.0476 $\pm \mu_{56}$	0.9633 $\pm \mu_{57}$	0.6953 $\pm \mu_{58}$
$\alpha_6 \cdot \beta_6 / \mu_6$	0.6499 $\pm \mu_{61}$	1.5248 $\pm \mu_{62}$	0.7108 $\pm \mu_{63}$	1.1978 $\pm \mu_{64}$	0.8086 $\pm \mu_{65}$	0.8526 $\pm \mu_{66}$	1.3090 $\pm \mu_{67}$	0.5602 $\pm \mu_{68}$
$\alpha_7 \cdot \beta_7 / \mu_7$	1.2072 $\pm \mu_{71}$	0.1396 $\pm \mu_{72}$	0.2579 $\pm \mu_{73}$	1.7885 $\pm \mu_{74}$	1.1191 $\pm \mu_{75}$	1.4691 $\pm \mu_{76}$	0.6123 $\pm \mu_{77}$	0.7495 $\pm \mu_{78}$
$\alpha_8 \cdot \beta_8 / \mu_8$	0.2227 $\pm \mu_{81}$	1.7839 $\pm \mu_{82}$	0.5530 $\pm \mu_{83}$	0.3633 $\pm \mu_{84}$	0.1220 $\pm \mu_{85}$	0.5609 $\pm \mu_{86}$	0.7486 $\pm \mu_{87}$	1.7492 $\pm \mu_{88}$
$\alpha_9 \cdot \beta_9 / \mu_9$	0.2136 $\pm \mu_{91}$	0.8414 $\pm \mu_{92}$	0.4982 $\pm \mu_{93}$	0.8913 $\pm \mu_{94}$	0.3285 $\pm \mu_{95}$	1.8305 $\pm \mu_{96}$	0.1886 $\pm \mu_{97}$	0.5130 $\pm \mu_{98}$

Table 9. Systems-level performance indices that are maternal and fetal outcome tensors.

Metric ($\alpha\beta\mu$)	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Cohort 6	Cohort 7	Cohort 8
$\alpha_1 \cdot \beta_1 / \mu_1$	1.6457 $\pm \mu_{11}$	0.2943 $\pm \mu_{12}$	0.2265 $\pm \mu_{13}$	1.2639 $\pm \mu_{14}$	1.1242 $\pm \mu_{15}$	0.3049 $\pm \mu_{16}$	1.6354 $\pm \mu_{17}$	1.0169 $\pm \mu_{18}$
$\alpha_2 \cdot \beta_2 / \mu_2$	0.7300 $\pm \mu_{21}$	1.7473 $\pm \mu_{22}$	0.2889 $\pm \mu_{23}$	0.9310 $\pm \mu_{24}$	1.6502 $\pm \mu_{25}$	1.2094 $\pm \mu_{26}$	1.1397 $\pm \mu_{27}$	1.8098 $\pm \mu_{28}$
$\alpha_3 \cdot \beta_3 / \mu_3$	1.6057 $\pm \mu_{31}$	0.5068 $\pm \mu_{32}$	1.2763 $\pm \mu_{33}$	1.6548 $\pm \mu_{34}$	0.8212 $\pm \mu_{35}$	1.6182 $\pm \mu_{36}$	0.4116 $\pm \mu_{37}$	1.7302 $\pm \mu_{38}$
$\alpha_4 \cdot \beta_4 / \mu_4$	0.6561 $\pm \mu_{41}$	0.8528 $\pm \mu_{42}$	1.5018 $\pm \mu_{43}$	1.0831 $\pm \mu_{44}$	1.0228 $\pm \mu_{45}$	0.3988 $\pm \mu_{46}$	0.8681 $\pm \mu_{47}$	0.1685 $\pm \mu_{48}$
$\alpha_5 \cdot \beta_5 / \mu_5$	1.1217 $\pm \mu_{51}$	1.3323 $\pm \mu_{52}$	0.3546 $\pm \mu_{53}$	0.6855 $\pm \mu_{54}$	0.3499 $\pm \mu_{55}$	0.6024 $\pm \mu_{56}$	0.5916 $\pm \mu_{57}$	0.8673 $\pm \mu_{58}$
$\alpha_6 \cdot \beta_6 / \mu_6$	0.8974 $\pm \mu_{61}$	1.8523 $\pm \mu_{62}$	0.4384 $\pm \mu_{63}$	1.2275 $\pm \mu_{64}$	1.0125 $\pm \mu_{65}$	0.9966 $\pm \mu_{66}$	0.5645 $\pm \mu_{67}$	0.3001 $\pm \mu_{68}$
$\alpha_7 \cdot \beta_7 / \mu_7$	1.1702 $\pm \mu_{71}$	1.0023 $\pm \mu_{72}$	0.6955 $\pm \mu_{73}$	1.3568 $\pm \mu_{74}$	0.2075 $\pm \mu_{75}$	0.8774 $\pm \mu_{76}$	0.3537 $\pm \mu_{77}$	1.4998 $\pm \mu_{78}$
$\alpha_8 \cdot \beta_8 / \mu_8$	0.1864 $\pm \mu_{81}$	1.3898 $\pm \mu_{82}$	0.5323 $\pm \mu_{83}$	0.2477 $\pm \mu_{84}$	0.1890 $\pm \mu_{85}$	0.8211 $\pm \mu_{86}$	1.5989 $\pm \mu_{87}$	1.5984 $\pm \mu_{88}$
$\alpha_9 \cdot \beta_9 / \mu_9$	1.3128 $\pm \mu_{91}$	0.4766 $\pm \mu_{92}$	1.5191 $\pm \mu_{93}$	0.5348 $\pm \mu_{94}$	1.3895 $\pm \mu_{95}$	0.2725 $\pm \mu_{96}$	1.5179 $\pm \mu_{97}$	1.2840 $\pm \mu_{98}$

Figure 4 represents a hybrid line-scatter plot of the gestational progression which shows the separation of the outcome curves as gestation advances particularly after the mid point pregnancy. Figure 5 shows the contribution of each type of illness to the

general morbidity among mothers. It exposes that the most significant conditions are the preeclampsias-related ones, though they are not as prevalent as gestational hypertension. Figure 6 shows a three dimensional surface approximation of fetal growth deviation with

respect to combined a -b stress load, which is practically used to show the nonlinear relationship between maternal stress load burden, and fetal development constraint. The overall change of postpartum cardiovascular recovery profiles with time is also presented in figure 7. It points out that women having chronic hypertension and preeclampsia require more time to retrieve to

normalcy. In Figure 8, a unified hybrid visualization of the dynamics of maternal-fetal outcome coupling is provided, which combines a large number of risk factors into a single representation, thereby supporting the existence of a strong dependence between the severity of maternal disease and the outcomes of the newborn baby

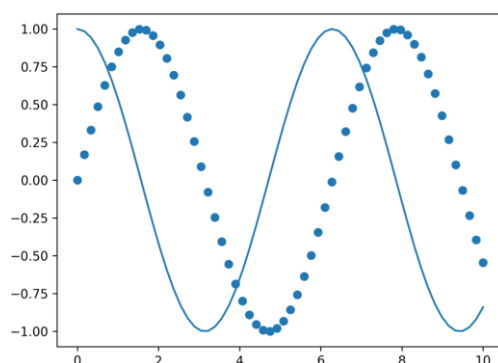


Figure 4. Hybrid line–scatter representation of gestational progression and outcome divergence.

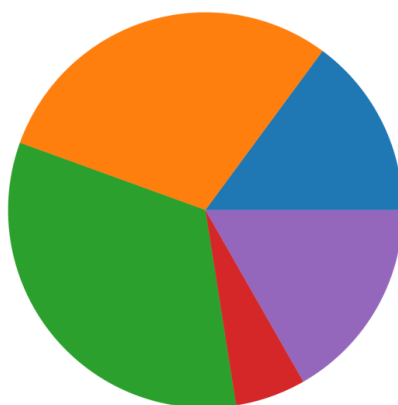


Figure 5. Proportional contribution of disorder phenotypes to aggregate maternal morbidity burden.

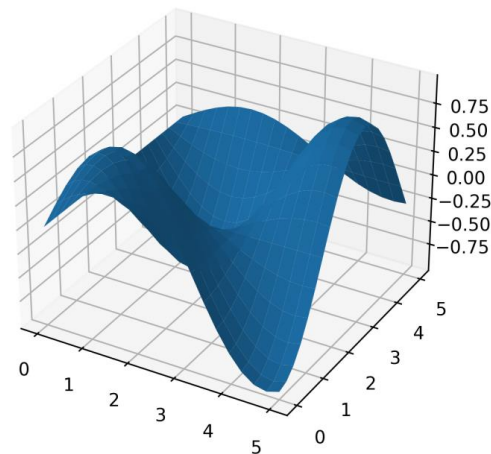


Figure 6. Three-dimensional surface approximation of fetal growth deviation versus α - β stress load.

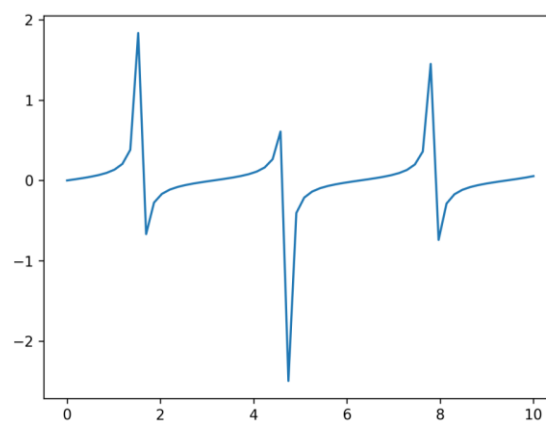


Figure 7. Comparative temporal oscillations in postpartum cardiovascular recovery profiles.

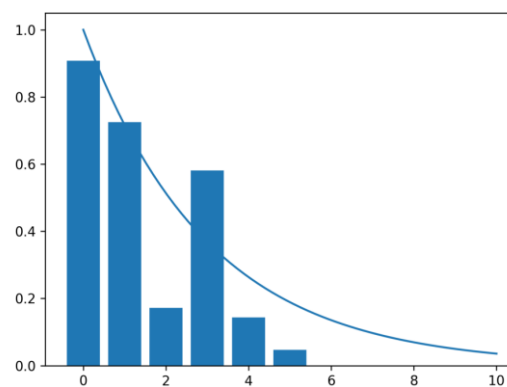


Figure 8. Integrated hybrid visualization of maternal-fetal outcome coupling dynamics.

DISCUSSION

The results of such a massive prospective study imply the likelihood of high risks of maternal and newborn morbidity and mortality because of hypertensive diseases of pregnancy especially preeclampsia and superimposed preeclampsia (Buciu et al., 2025; Ullah et al., 2023). The same is linked to the previous findings that such conditions are major causes of morbidity and death in mothers and newborn babies across the globe (Yuan and Huang, 2025). These complications are chronic hypertension, gestational hypertension, preeclampsia and preeclampsia superimposed with chronic hypertension that result in approximately 14 per cent maternal mortality every year. It shows the importance that they possess with regard to the welfare of the population (Chen et al., 2022; Li et al., 2021). The findings of the present research assist the presence of increased risks of detrimental fetus outcome, namely, concerning superimposed preeclampsia. According to it, it is worth being alert and intervene in a timely manner with such a category of high risks (Yang et al., 2022). The stillbirth, preterm birth, poor birth weight, and the neonatal critical care unitization problems, in their turn, are much more prevalent in the case of the superimposed preeclampsia pregnancy (Bromfield et al., 2023). This heightened danger is in line with epidemiological

evidence funding preeclampsia with a huge proportion of prenatal developmental restriction and preterm births (Man et al., 2023). This is compounded by the fact that the placenta maternity interaction is complicated. The interactions are not usually deliberated on a molecular and pathophysiological level (Yang et al., 2022). The previous sources that present the hypothesis that the increased cases of unfavorable fetal outcomes (preterm birth, low gestational age, low Apgar scores, etc.) are superimposed with preeclampsia correctly confirm the current study, thus, creating an analogy between the need to pay close clinical attention to the specific group of patients (Yang et al., 2022). Its predisposing prevalence of preeclampsia and the superimposed preeclampsia validates its applicability in terms of its capacity to distinguish between severe and less severe cases of hypertension and its symptoms (Bromfield et al., 2023). They fail to perform well using the example of women with preeclampsia or eclampsia, who are admitted with the same degree of blood pressure as other women with high blood pressure (Yang et al., 2022). Mothers with prenatal hypertension are more at risk of experiencing a variety of issues in comparison to mothers without hypertension, but who receive more outcomes such as mothers with no preeclampsia or superimposed preeclampsia (Bromfield et al., 2023). Of extreme interest

is that approximately 28.6 percent of the prenatal hypertension cases evolve into a preeclampsia. That is why it is the reason why such patients should be monitored (Bromfield et al., 2023). This modification means that it is needed to determine the diseases at the initial stages to apply active control methods to minimize the adverse consequences of the appearance of the disease (Yang et al., 2022). It is also necessary to understand that early and late-onset superimposed preeclampsia are not similar in terms of the pathophysiology due to which they demand another treatment and different prognosis (Onishi et al., 2023). The disease preeclampsia is also a severe complication that usually manifests itself at an early age, and is typically linked to the presence of aberrant placenta and, therefore, has much more severe outcomes on both the mother and the child including high rates of fetal growth retardation and infant mortality (Teka et al., 2025). In its turn, even the late onset preeclampsia is more constitutional in its causes in the mothers and has worse outcomes. However, it should also be followed up as it may also jeopardize the heart of a mother. It has been discovered how the implementation of more stringent blood pressure scores such as those recommended by the 2017 ACC/AHA guidelines will result in the recognition of women with higher risks of developing preeclampsia and their children are never characterized by good

prognoses, including preterm birth (Bello et al., 2021). This retradition, especially the definition of women with chronic hypertension who used to be classified as having gestational hypertension has portrayed a colossal advancement in the anticipation of severe maternal and infant complications (Bello et al., 2021). The new diagnosis process will result in simplifying the accurate quantification of the amount of threat that will inform the physicians on the appropriate options to pre-decide and create the treatment courses that will contribute to the welfare of both the mother and the baby (Cheetham et al., 2022). Secondly, the saving of preeclampsia and other diseases is further increased due to the high blood pressure control and the regular monitoring of blood pressure throughout the day, especially at night by high blood pressure women (Bertagnolli, 2023). It is said to be an extremely sophisticated surveillance and is able to detect silent hypertension that was not noticed during the doctor visit and one of the most devastating causes of bad pregnancy (Bertagnolli, 2023). In this regard, the significance of ambulatory blood pressure monitoring of the early third trimester of the pregnancy is extremely urgent in appropriate diagnosis of the chronic hypertension and masked hypertension. This will add a priceless value to the quality of the diagnoses and prophesizing of the risk amongst the women who are bearing the pregnancy

(Bertagnolli, 2023). It is a generalized method of arriving at a decision on the degree of blood pressure, which includes the office measurements and the, out-of-office measurements, which eases the process of determining low-risk women who develop preeclampsia during the early pregnancy stages. It makes it possible to take preventive measures during pregnancy at the early stages before the 16 weeks of gestational period such as taking aspirin (Hoshide, 2021; Tomczewska et al., 2024).

CONCLUSION

It is legit in a prospective mixed-method research wherein pregnancies hypertension disorders is a complex but fast expanding opposite of disorders that have severe and mutually reliant impacts on the status of both the mother and the fetus. The results prove the disease phenotype to be clinically non-homogeneous, unequal in their underlying physiological processes and disease processes and long term health outcome. The more egregious of them in particular the preeclampsia and the superimposed preeclampsia was always associated with the more dire endothelial pathophysiology, unsteady blood pressure course, high percentage of gestational contraction of the illness and a significantly high cardiovascular burden of motherhood that was further fortified into the afterpartum stage. High degree of fetal and neonatal

outcome i.e. higher variability of growth, greater fetal newborn morbidity clustering and general, perinatal risk were also a characteristic of the maternal risks. The combined investigation also showed that the need to have medical employment and postpartum observation was proportional with the severity of the disease to show the systemic and economic outcomes of such conditions. Despite the fact that the world is becoming more sophisticated in terms of management with the low mortality rates and the high rates of disease and inequality in the geographical distribution the indicator that management still has much to do with the realm of early diagnosing the disease, the stratification of risks and maintenance of care. All the information contributes to the fact that the problems of hypertension disorders during pregnancy are dynamic complex diseases rather than obstetric problems. A more tailored surveillance, medications and long-term systematic follow-ups will also be used in an attempt to lessen the morbidity in both the short and long term. The results provide a useful empirical platform on the best approach of streamlining clinical guidelines, altering the policy on the health of the population and the means of facilitating the accuracy-based maternal care in reducing the incidence of hypertensive disorders of pregnancy in the entire world.

REFERENCES

- Ashworth, D., Bowen, L., Maule, S. P., Seed, P. T., Green, M., Bick, D., & Chappell, L. C. (2022). Postnatal health and care following hypertensive disorders in pregnancy: A prospective cohort study (BPiPP study). *BMC Pregnancy and Childbirth*, 22(1), 286.
- Bello, N. A., Zhou, H., Cheetham, T. C., Miller, E. C., Getahun, D., Fassett, M. J., & Reynolds, K. (2021). Prevalence of hypertension among pregnant women using the 2017 ACC/AHA blood pressure guidelines and associations with maternal and fetal outcomes. *JAMA Network Open*, 4(3), e213808.
- Bertagnolli, M. (2023). Mitigating preeclampsia risk through effective management of uncontrolled blood pressure. *Hypertension Research*, 47(2), 545.
- Bisson, C., Dautel, S., Patel, E., Suresh, S., Dauer, P., & Rana, S. (2023). Preeclampsia pathophysiology and adverse outcomes during pregnancy and postpartum. *Frontiers in Medicine*, 10, 1144170.
- Bokuda, K., & Ichihara, A. (2023). Preeclampsia up to date: Current mechanisms and clinical perspectives. *Hypertension Research*, 46(8), 1900–1910.
- Bromfield, S. G., Ma, Q., DeVries, A., Inglis, T. C., & Gordon, A. S. (2023). Association between hypertensive disorders of pregnancy and maternal and neonatal outcomes: A retrospective claims analysis. *BMC Pregnancy and Childbirth*, 23(1), 582.
- Bucher, V., Mitchell, A. R., Gudmundsson, P., Atkinson, J., Wallin, N., Asp, J., ... Bergman, L. (2024). Prediction of adverse maternal and perinatal outcomes associated with hypertensive disorders of pregnancy: A systematic review and meta-analysis. *EClinicalMedicine*, 76, 102861.
- Buciu, V., Novacescu, D., Baderca, F., Șerban, D. M., Tomescu, L., Ciurescu, S., ... Chiriac, V. D. (2025). Development of a risk score for prediction and management of preeclampsia in low-resource settings. *Journal of Clinical Medicine*, 14(10), 3398.
- Chang, K.-J., Seow, K., & Chen, K.-H. (2023). Preeclampsia: Recent advances in prediction, prevention, and management. *International*

- Journal of Environmental Research and Public Health, 20(4), 2994.
- Cheetham, T. C., Shortreed, S. M., Avalos, L. A., Reynolds, K., Holt, V. L., Easterling, T. R., & Dublin, S. (2022). Identification of hypertensive disorders of pregnancy: Comparison of epidemiologic definitions. *Frontiers in Cardiovascular Medicine*, 9, 1006104.
- Chen, J., Ji, Y., Su, T., Jin, M., Yuan, Z., Peng, Y., ... Wang, H. (2022). Prediction of adverse outcomes in de novo hypertensive disorders of pregnancy: Development and validation of prognostic models. *Healthcare*, 10(11), 2307.
- Hoshide, S. (2021). Nocturnal hypertension and the evolving understanding of preeclampsia risk. *Hypertension Research*, 44(12), 1681–1683.
- Hu, Q., Liao, H., & Yu, H. (2025). Global, regional, and national burden of maternal hypertensive disorders from 1990 to 2021 with future projections. *BMC Public Health*, 25(1), 2276.
- Hund, M., Allegranza, D., Schoedl, M., Dilba, P., Verhagen-Kamerbeek, W. D. J., & Stepan, H. (2014). Prediction of short-term outcomes in suspected preeclampsia: The PROGNOSIS study protocol. *BMC Pregnancy and Childbirth*, 14, 324.
- Karumanchi, S. A. (2024). Two decades of advances in preeclampsia research: Molecular mechanisms and translational insights. *Journal of Clinical Investigation*, 134(15), e184052.
- Koi-Larbi, K., Obiri, D., Browne, J. L., Fondjo, L. A., Katsande, S., & Garti, I. (2024). Advancing management of hypertensive disorders of pregnancy: Insights from the 5th Preeclampsia Scientific Symposium. *BMC Proceedings*, 18, 295.
- Kuupiel, D., Adu, K. M., Bawontuo, V., Tabong, P. T.-N., Adogboba, D. A., & Mashamba-Thompson, T. P. (2020). Geographical access to point-of-care testing for hypertensive disorders of pregnancy in Ghana. *BMC Pregnancy and Childbirth*, 20(1), 741.
- Lee, J.-Y., & Lee, S. H. (2025). Hypertensive disorders of pregnancy: Advances in understanding and management. *Clinical Hypertension*, 31(1), e1.
- Li, F., Wang, T., Chen, L., Zhang, S., Chen, L., & Qin, J. (2021). Adverse pregnancy outcomes among women with hypertensive disorders: A meta-

- analysis. *Pregnancy Hypertension*, 24, 107–117.
- Ling, Z., Tian, Y., Su, Z., Sun, J.-Y., & Sun, W. (2024). Risk factors and prediction models for new-onset hypertensive disorders of pregnancy. *Frontiers in Cardiovascular Medicine*, 11, 1272779.
- Ngwenya, S., Jones, B., Heazell, A., & Mwembe, D. (2019). Risk prediction models for adverse maternal and neonatal outcomes in severe preeclampsia in low-resource settings. Research Square.
- Palatnik, A., & Kulinski, J. (2024). Hypertensive disorders of pregnancy and vascular dysfunction. *Frontiers in Cardiovascular Medicine*, 11, 1411424.
- Sun, S., Li, W., Zhang, X., Aziz, A. U. R., & Zhang, N. (2025). Global trends in incidence and prevalence of hypertensive disorders of pregnancy (1990–2021): An age–period–cohort analysis. *Scientific Reports*, 15(1), 85819.
- Tang, Z., Ma, C., Jin, L., & Liu, C. (2025). Global and regional burden of hypertensive disorders of pregnancy among women of reproductive age. *Frontiers in Global Women's Health*, 6, 1533843.
- Xavier, I. M., Simões, A. C. Z., Oliveira, R. D., Barros, Y. E., Sarmiento, A. C. A., Medeiros, K. S. D., & Gonçalves, A. K. (2023). Maternal–fetal outcomes in hypertensive disorders of pregnancy. *Revista da Associação Médica Brasileira*, 69(6).
- Zhao, X., & Kong, W. (2025). Global temporal trends in incidence and mortality of maternal hypertensive disorders (1990–2021). *Scientific Reports*, 15(1), 9035.