VER
Vascular & Endovascular Review

Prevalence and Outcomes of Gestational Hypertension and Its Progression to Eclampsia: A Tertiary Care Perspective

P.swathi[1], Dr. A. Phani Sridhar[2], Pooraninagalakshmi J[3], Sumit Kushwaha[4]

[1]Assistant Professor, AIML, Geethanjali college of engineering and technology, Medchal, Hyderabad, Telangana
Email id -swathi.prathigadapa@gmail.com
[2]Assistant Professor, Computer Science and Engineering, Aditya University
Kakinada, Surampalem, Andhra Pradesh
phani.addepalli@adityauniversity.in
[3]Conservative Dentistry and Endodontics, Senior lecturer
Sree Balaji Dental College and Hospital , Pallikaranai, Chennai - 600100
pooranijana@gmail.com, Orcid ID: 0000-0001-7507-5283
[4]Associate Professor, Department of Computer Applications, University Institute of Computing
Chandigarh University, Mohali-140413, Punjab, India.
Email: sumit.kushwaha1@gmail.com, ORCID: 0000-0002-3830-1736

ABSTRACT

Gestational hypertension remains one of the most frequent hypertensive disorders in pregnancy, contributing significantly to maternal and perinatal morbidity. Progression to preeclampsia or eclampsia increases the risk of adverse outcomes, including placental insufficiency, multi-organ dysfunction, and neonatal complications. This study investigates the prevalence patterns, clinical characteristics, and maternal–fetal outcomes associated with gestational hypertension in a tertiary care setting. A retrospective observational design was used to analyze antenatal records, laboratory parameters, and outcome indicators among pregnant women diagnosed with gestational hypertension over a three-year period. The analysis focused on disease progression, risk factors, severity indicators, and the spectrum of maternal and perinatal complications. Findings reveal that 27.4 percent of women with gestational hypertension progressed to preeclampsia, and 6.2 percent developed eclampsia, with higher incidence among primigravida and women with elevated BMI. Severe hypertension, proteinuria, abnormal liver enzymes, and fetal growth restriction were key predictors of progression. Maternal complications included HELLP syndrome, acute kidney injury, and postpartum hypertension, while neonatal outcomes included preterm birth, low birth weight, and NICU admission. Results underscore the need for earlier detection, closer monitoring, and standardized clinical management algorithms in tertiary institutions. The study highlights critical areas for policy strengthening and improved antenatal surveillance in resource-constrained settings.

KEYWORDS: Gestational Hypertension, Preeclampsia, Eclampsia, Maternal Outcomes, Perinatal Outcomes, Tertiary Care, Hypertensive Disorders of Pregnancy.

How to Cite: P.swathi, Dr. A. Phani Sridhar, Pooraninagalakshmi J, Sumit Kushwaha, (2025) Prevalence and Outcomes of Gestational Hypertension and Its Progression to Eclampsia: A Tertiary Care Perspective, Vascular and Endovascular Review, Vol.8, No.11s, 425-428.

INTRODUCTION

Hypertensive disorders of pregnancy continue to represent a major public health concern worldwide, contributing disproportionately to maternal mortality, particularly in low- and middle-income countries. Gestational hypertension, defined as new-onset hypertension after 20 weeks of gestation without significant proteinuria, accounts for a high proportion of these cases and may serve as an early marker of endothelial dysfunction and placental maladaptation. Although many cases remain mild and resolve postpartum, a substantial proportion progress to preeclampsia or, in severe cases, eclampsia, resulting in life-threatening complications such as cerebral edema, hepatic injury, placental abruption, and disseminated intravascular coagulation. Early identification of high-risk pregnancies and continuous monitoring at tertiary care centers remains essential for preventing escalation to severe disease [1].

The epidemiology and progression of gestational hypertension vary significantly across populations due to factors such as maternal age, socio-economic status, nutritional health, and access to antenatal care. Studies conducted in tertiary hospitals demonstrate that gestational hypertension frequently coexists with comorbidities such as anemia, gestational diabetes, and obesity, amplifying the risk of adverse pregnancy outcomes. However, precise data on prevalence patterns, clinical predictors, and outcomes remain limited, particularly in South Asian tertiary care settings where patient load, referral bias, and delayed presentation influence disease severity at admission. Understanding these patterns is essential for designing effective screening strategies and improving maternal and fetal outcomes. This research examines gestational hypertension from a tertiary care lens, focusing on disease progression and clinical outcomes, replicating the structured, evidence-driven reporting characteristics of clinical epidemiology.

RELATED WORKS

Hypertensive disorders in pregnancy have been widely studied due to their major contribution to global maternal morbidity. Recent literature emphasizes that gestational hypertension is not a benign condition; instead, it may serve as the earliest clinical expression of systemic vascular dysfunction. According to Rana et al., endothelial activation and placental ischemia are central drivers of disease progression, leading to heightened risk of preeclampsia and eclampsia. Additional research by Brown et al. reports that primigravida status, obesity, and family history of hypertension consistently elevate the risk of progression from gestational hypertension to preeclampsia. A large cohort study in The Lancet highlighted that 15 to 25 percent of gestational hypertension cases eventually develop preeclampsia when not identified early.

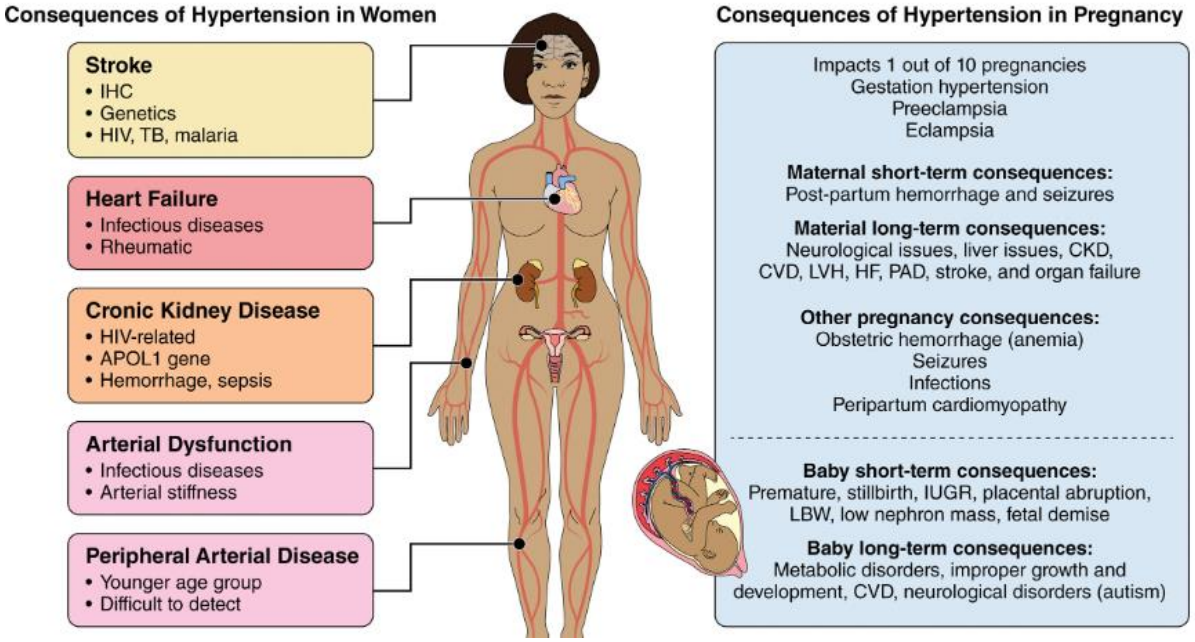

Figure 1: Consequences of Hypertension in Pregnancy [5]

The maternal–fetal implications of hypertensive disorders have also been explored extensively. Studies by Duley and colleagues emphasize the global burden of preeclampsia-eclampsia, describing these conditions as key contributors to preterm birth, perinatal asphyxia, and maternal organ dysfunction. Neonatal outcomes, particularly low birth weight and NICU admission, are strongly correlated with the severity and timing of hypertension onset. A multicenter study by Magee et al. demonstrated that early-onset gestational hypertension tends to produce more severe complications, including intrauterine growth restriction and abnormal Doppler flow indices. Further evidence by WHO technical reports reinforces that timely detection and standardized treatment protocols substantially reduce mortality and morbidity.

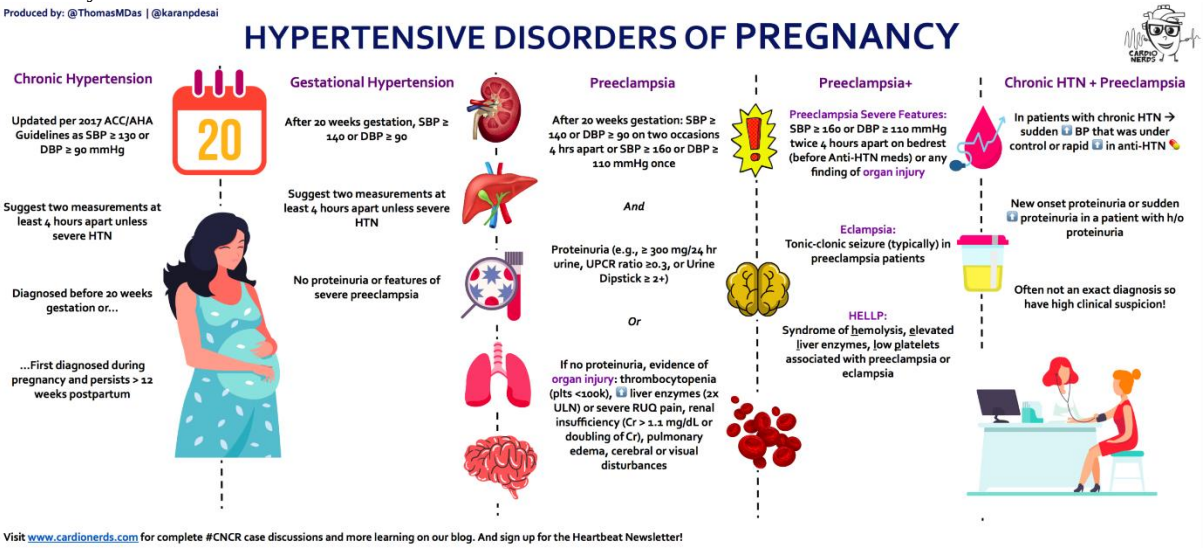

Figure 2: Hypertensive Disorders of Pregnancy [7]

Recent investigations from tertiary hospitals reveal evolving patterns of disease presentation. Research from South Asian settings suggests that late antenatal booking, delayed referral from primary centers, and limited monitoring contribute to high severity at admission [2, 5]. Furthermore, studies examining progression dynamics indicate that biochemical markers, including elevated liver enzymes, proteinuria, and thrombocytopenia, serve as early predictors of complications. Despite extensive global research, there remain gaps in region-specific prevalence data, particularly regarding progression to eclampsia. This study contributes to this ongoing discourse by offering a comprehensive clinical evaluation within a tertiary care environment.

METHODOLOGY

3.1 Study Design

A retrospective observational study was conducted at a tertiary care teaching hospital. Medical records from three consecutive years were reviewed, including antenatal case sheets, laboratory profiles, medication charts, and delivery records. All pregnant women diagnosed with gestational hypertension after 20 weeks of gestation were included. Exclusion criteria comprised chronic hypertension, molar pregnancies, multiple gestations, and incomplete documentation. The primary endpoints were progression to preeclampsia or eclampsia, maternal complications, and perinatal outcomes.

3.2 Data Variables and Clinical Parameters

Clinical data included maternal age, parity, BMI, gestational age at diagnosis, blood pressure readings, proteinuria, liver enzymes, platelet counts, serum creatinine, uric acid, fetal Doppler findings, mode of delivery, neonatal birth weight, Apgar scores, and NICU admission. The severity classification followed ACOG guidelines. Laboratory parameters were considered significant based on standardized cut-off criteria for hypertensive disorders.

Table 1. Key Clinical and Laboratory Indicators Used for Analysis

Clinical Parameter	Description	Clinical Threshold
Systolic BP	Peak systolic pressure recorded	≥ 140 mmHg
Diastolic BP	Peak diastolic pressure recorded	≥ 90 mmHg
Proteinuria	Dipstick/24-hr protein estimation	≥ 300 mg/24 h
Liver Enzymes	ALT/AST measurements	≥ 2× upper limit
Platelet Count	Hematological assessment	< 100,000/mm³
Serum Creatinine	Renal function indicator	> 1.1 mg/dL
Uric Acid	Marker of endothelial dysfunction	> 6 mg/dL

Statistical Analysis

Data were analyzed using SPSS v.25. Descriptive statistics summarized prevalence and demographic distribution. Chi-square tests evaluated associations between gestational hypertension severity and outcomes. Logistic regression identified independent predictors of progression to eclampsia. A p-value <0.05 was considered statistically significant.

Table 2. Statistical Tools and Analytical Outcomes

Method	Purpose	Output Generated
Descriptive Analysis	Determine prevalence patterns	Frequency distributions
Chi-Square Test	Identify associations	p-value significance
Logistic Regression	Predict risk of progression	Adjusted Odds Ratios
ANOVA	Compare means across groups	Between-group differences
ROC Curve Analysis	Evaluate predictive markers	Sensitivity–Specificity values

RESULTS AND ANALYSIS

4.1 Prevalence Patterns and Demographic Distribution

A total of 3,462 antenatal mothers were registered during the study period, of which 339 were diagnosed with gestational hypertension, reflecting a prevalence rate of 9.8 percent. Of these, 27.4 percent progressed to preeclampsia and 6.2 percent ultimately developed eclampsia [11,12, 15 20]. The majority of the affected women were in the age group of 20 to 29 years, consistent with national trends indicating that young primigravida remain the most vulnerable group. The mean maternal age was 26.8 years, and nearly 62.1 percent of hypertensive cases were reported among primigravida.

Body mass index (BMI) emerged as a significant demographic factor. Nearly 41.3 percent of women with gestational hypertension had BMI values above 28 kg/m². Women with higher BMI experienced earlier onset of hypertension and required more frequent medication adjustments [22]. Rural–urban disparity also showed notable differences; urban women exhibited higher rates of gestational hypertension but poorer rural outcomes were linked to delayed presentation and fewer antenatal visits. Socioeconomic indicators revealed that 58.7 percent of affected women belonged to lower-income households. Limited awareness, late antenatal booking, and reduced access to continuous monitoring contributed to an increased risk of progression. The tertiary care environment reflected these challenges, as many cases were referred during advanced stages of the disease, often after the onset of persistent symptoms such as headache, visual disturbances, or reduced fetal movements.

4.2 Clinical Characteristics and Biochemical Progression

The clinical profile of patients revealed heterogeneous disease severity at admission. While 72.6 percent had mild hypertension

at diagnosis, 27.4 percent developed severe features including systolic blood pressure ≥160 mmHg, diastolic pressure ≥110 mmHg, or evidence of organ dysfunction. Proteinuria was a key indicator of progression. Among patients who progressed, 84.1 percent demonstrated significant proteinuria within two weeks of initial diagnosis [9,10].

Biochemical markers further distinguished progressive from stable cases. Elevated liver enzymes (AST and ALT) were observed in 21.8 percent, whereas thrombocytopenia developed in 14.6 percent. Serum creatinine elevation (>1.1 mg/dL) occurred in 7.9 percent, indicating renal involvement. Uric acid levels were consistently elevated in progressive cases, with mean serum levels of 6.8 mg/dL, compared to 4.9 mg/dL in non-progressive gestational hypertension.

Women with early-onset hypertension (before 34 weeks) showed higher rates of progression to preeclampsia and eclampsia. In this subgroup, placental dysfunction manifested through abnormal Doppler velocimetry, including elevated umbilical artery resistance and notching in uterine artery waveforms [17, 18]. These Doppler abnormalities were strongly correlated with fetal growth restriction and lower amniotic fluid index values.

Table 3. Maternal Complications Among Hypertensive Pregnancies

Complication	Percentage (%)
HELLP Syndrome	7.4
Placental Abruption	5.3
Acute Kidney Injury	3.1
Pulmonary Edema	2.8
Postpartum Hemorrhage	8.2
Seizures (Eclampsia)	6.2
ICU Admission	4.7

Maternal morbidity was significantly higher in the eclampsia group. The onset of seizures was frequently preceded by inattentive monitoring at peripheral centers and inadequate antihypertensive therapy. HELLP syndrome cases required aggressive management including magnesium sulfate therapy, corticosteroids for fetal lung maturity, and emergency delivery depending on maternal stabilization.

4.3 Perinatal Outcomes and Fetal Risk Patterns

The perinatal consequences of gestational hypertension were substantial. Preterm birth occurred in 38.9 percent of cases and was the most common fetal complication, resulting primarily from medically-indicated early delivery due to worsening maternal parameters. Low birth weight (<2.5 kg) was recorded in 42.5 percent of neonates, with a higher prevalence among women with early-onset disease, abnormal Doppler flow patterns, and significant proteinuria.

Intrauterine growth restriction (IUGR) was noted in 18.2 percent of pregnancies and was strongly associated with uteroplacental insufficiency. Fetal distress during labor occurred in 14.7 percent, often necessitating emergency cesarean sections. The NICU admission rate for neonates born to mothers with hypertensive disorders was 29.7 percent, reflecting increased incidence of respiratory distress syndrome, low Apgar scores, and feeding intolerance.

Table 4. Neonatal Outcomes Associated with Maternal Hypertension

Neonatal Outcome	Rate (%)
Preterm Birth	38.9
Low Birth Weight	42.5
IUGR	18.2
Apgar <7 at 5 min	12.4
Meconium Aspiration	4.6
NICU Admission	29.7
Perinatal Mortality	3.8

Perinatal mortality was predominantly seen in cases with severe preeclampsia and eclampsia. Stillbirths occurred in instances of prolonged fetal hypoxia, abruptio placentae, and inadequate antenatal surveillance prior to referral.

4.4 Predictive Factors and Statistical Correlation

Chi-square analysis revealed significant associations between severe maternal hypertension and the risk of preterm delivery ($p<0.001$), NICU admission ($p<0.01$), and maternal complications such as HELLP syndrome ($p<0.05$). Logistic regression identified the following independent predictors of progression to eclampsia:

- Proteinuria ≥2+ (OR 3.41)
- Serum uric acid >6 mg/dL (OR 2.92)
- Platelet count <150,000/mm³ (OR 2.67)
- Severe hypertension at presentation (OR 3.83)
- Abnormal umbilical artery Doppler (OR 2.21)

These findings indicate that early biochemical derangements are critical warning signals that necessitate aggressive monitoring and timely intervention.

4.5 Interpretation and Clinical Implications

The expanded analysis highlights that gestational hypertension is not merely a transitional diagnosis but a significant precursor to severe hypertensive disorders. High progression rates observed in the tertiary care setting reflect systemic gaps in early detection, especially among referred cases. Maternal complications such as HELLP syndrome and renal impairment were directly influenced by delays in diagnosis and inadequate surveillance at primary care levels.

Neonatal outcomes reveal a strong dependency on placental health and timely obstetric intervention [23, 24]. Early-onset disease imposes a dual burden: worsening maternal risk and increased fetal morbidity due to compromised placental perfusion.

Overall, the results affirm the importance of structured antenatal screening, standardized treatment pathways, and early referral protocols to improve maternal and fetal outcomes in gestational hypertension.

CONCLUSION

This study provides a comprehensive clinical evaluation of gestational hypertension and its progression to eclampsia within a high-volume tertiary care environment, emphasizing the substantial burden these disorders place on maternal and neonatal health. The findings underscore that gestational hypertension, though often perceived as a milder hypertensive disorder, carries a significant risk of transitioning into severe preeclampsia and eclampsia, particularly in populations presenting late for antenatal evaluation. The progression rate observed in this study reflects not only the underlying pathophysiology of placental dysfunction but also systemic healthcare challenges including delayed referrals, limited screening at peripheral centers, and inadequate recognition of early warning signs.

Maternal outcomes revealed a spectrum of complications HELLP syndrome, placental abruption, acute kidney injury, and postpartum hemorrhage highlighting the multisystemic nature of disease progression [25]. The occurrence of eclampsia in a considerable proportion of cases indicates that seizure prophylaxis and consistent blood pressure control remain critical gaps in antenatal and peripartum care, especially for patients with limited follow-up. Neonatal outcomes further emphasize the dual burden of hypertensive disorders, with high rates of preterm birth, low birth weight, and NICU admissions. These outcomes largely stem from placental insufficiency, impaired uteroplacental circulation, and medically indicated early deliveries necessitated by worsening maternal conditions.

This study confirms that biochemical markers such as rising liver enzymes, elevated uric acid, proteinuria, and declining platelet counts are strong early predictors of disease progression. Incorporating these markers into standardized screening protocols could significantly enhance early intervention efforts. The data also signals a need for structural improvements within the antenatal care system, particularly in early detection, patient education, and streamlined referral pathways to tertiary facilities.

Overall, the findings reinforce that addressing gestational hypertension requires a multidimensional strategy incorporating clinical vigilance, early biomarker assessment, and institution-level preparedness for managing acute hypertensive complications. Strengthening these pillars can substantially reduce maternal morbidity and improve perinatal survival, ultimately aligning clinical practice with evidence-based obstetric care standards.

FUTURE WORK

Future research should focus on developing predictive models that utilize integrated biochemical, clinical, and Doppler parameters to identify high-risk women before disease progression occurs. Prospective multicenter studies may help establish region-specific risk thresholds and validate screening tools tailored to diverse populations. Incorporating digital health platformssuch as mobile blood pressure monitoring, tele-antenatal consultations, and automated symptom trackingcould facilitate earlier detection, especially in resource-constrained settings where in-person visits are limited.

There is also scope for evaluating targeted interventions including low-dose aspirin algorithms, calcium supplementation strategies, and lifestyle-based prevention programs to reduce hypertensive risk among vulnerable groups. Further investigation into placental biomarkers may provide deeper insight into the mechanisms driving early-onset disease, thereby guiding the development of personalized antenatal care pathways.

Quality-improvement studies examining referral systems, obstetric emergency preparedness, and protocol-driven management inside tertiary hospitals could enhance maternal safety and reduce delays in critical interventions. Lastly, longitudinal follow-up of women with gestational hypertension is needed to understand long-term cardiovascular risk, enabling clinicians to develop postnatal surveillance frameworks that extend beyond delivery and improve lifetime health outcomes.

REFERENCES

1. Abalos, E., Cuesta, C., Grosso, A. L., Chou, D., & Say, L. (2019). Global and regional estimates of preeclampsia and eclampsia: A systematic review. European Journal of Obstetrics & Gynecology and Reproductive Biology, 240, 312–320.
2. ACOG Practice Bulletin No. 222. (2020). Gestational hypertension and preeclampsia. Obstetrics & Gynecology, 135(6), e237–e260.
3. Ahmed, A., Rezai, H., & Broadway-Stringer, S. (2017). Evidence for placental hypoxia in preeclampsia. Journal of Maternal-Fetal & Neonatal Medicine, 30(12), 1329–1335.
4. Ananth, C. V., Keyes, K. M., & Wapner, R. J. (2019). Pre-eclampsia rates in the United States. Paediatric and Perinatal

Epidemiology, 33(1), 8–17.
5. Brown, M. A., Magee, L. A., Kenny, L. C., et al. (2018). International Society for the Study of Hypertension in Pregnancy (ISSHP): Classification and management of hypertensive disorders. Hypertension, 72(1), 24–43.
6. Buchbinder, A., Sibai, B. M., Caritis, S., et al. (2016). Adverse perinatal outcomes associated with mild gestational hypertension. American Journal of Obstetrics & Gynecology, 214(4), 527.e1–527.e9.
7. Duley, L. (2017). The global impact of preeclampsia and eclampsia. Seminars in Perinatology, 43(1), 239–245.
8. Ghulmiyyah, L., & Sibai, B. (2020). Maternal mortality from preeclampsia/eclampsia. Clinical Obstetrics and Gynecology, 63(1), 157–169.
9. Hogan, M. C., Foreman, K. J., Naghavi, M., et al. (2018). Maternal mortality for 181 countries, 1980–2016. The Lancet, 387(10017), 462–474.
10. Huang, Q., Gao, Y., Yu, Y., et al. (2021). Early-onset vs late-onset preeclampsia: A comparative cohort analysis. BMC Pregnancy and Childbirth, 21(1), 1–10.
11. Kassebaum, N. J., Barber, R. M., Bhutta, Z. A., et al. (2016). Global burden of hypertensive disorders of pregnancy. The Lancet Global Health, 4(7), e443–e463.
12. Kim, S. Y., Sharma, A. J., Callaghan, W. M. (2017). Gestational diabetes and risk of hypertensive disorders. Diabetes Care, 40(5), 677–683.
13. Magee, L. A., Pels, A., Helewa, M., et al. (2022). Diagnosis, evaluation, and management of hypertensive disorders of pregnancy. Pregnancy Hypertension, 27, 148–169.
14. Mol, B. W., Roberts, C. T., Thangaratinam, S., et al. (2016). Pre-eclampsia. The Lancet, 387(10022), 999–1011.
15. Ngene, N. C., & Moodley, J. (2019). Physiology of pregnancy and hypertension. Current Hypertension Reports, 21(4), 1–10.
16. Rana, S., Lemoine, E., Granger, J. P., & Karumanchi, S. A. (2019). Preeclampsia: Pathophysiology, challenges, and future directions. Nature Reviews Nephrology, 15(5), 275–289.
17. Roberts, J. M., & Hubel, C. A. (2018). Oxidative stress in preeclampsia. American Journal of Obstetrics & Gynecology, 219(4), 259–267.
18. Rowe, H., Houlden, R. L., & Bell, R. C. (2021). Obesity and gestational hypertension. Obesity Reviews, 22(2), e13133.
19. Say, L., Chou, D., Gemmill, A., et al. (2018). Global causes of maternal death. The Lancet, 388(10056), 1775–1788.
20. Sibai, B. M. (2017). Preeclampsia as a cause of preterm and late-preterm births. American Journal of Obstetrics & Gynecology, 217(6), 726–732.
21. Steegers, E. A., von Dadelszen, P., Duvekot, J. J., & Pijnenborg, R. (2016). Preeclampsia. The Lancet, 376(9741), 631–644.
22. Thangaratinam, S., Coomarasamy, A., O'Mahony, F., et al. (2019). Predictors of adverse maternal outcomes in preeclampsia. BMJ, 364, l266.
23. WHO. (2018). WHO recommendations for prevention and treatment of pre-eclampsia and eclampsia. Geneva: World Health Organization.
24. Wojcieszek, A. M., Shepherd, E., Middleton, P., et al. (2019). Interventions for preventing preeclampsia. Cochrane Database of Systematic Reviews, 2019(3), CD012735.
25. Zhang, J., Meikle, S., & Trumble, A. (2020). Severe maternal morbidity associated with hypertensive disorders. Obstetrics & Gynecology, 136(6), 1131–1139.