MERIT RESEARCH JOURNALS
www.meritresearchjournals.org

Merit Research Journal of Medicine and Medical Sciences (ISSN: 2354-323X) Vol. 13(6) pp. 131-138, December, 2025
Available online http://www.meritresearchjournals.org/mms/index.htm

doi: 10.5281/zenodo.18110437

Original Research Article

Preeclampsia Occurrence and its Impact on Maternal and Neonatal Health, Including Contributing Risk Factors

Mais Raad Abass[1], Hind Elia Georges[2*], Edwar Z. Khosho[3]

[1]MBChB. Basrah Maternity and Childhood Hospital, Basrah, Iraq.

[2]MBChB. FICMS. (Family Medicine), Al-Razi Primary Health Centre, Basrah Health Directorate. Basrah, Iraq.

[3]Assistant Professor and consultant of Obstetrics and Gynaecology, College of Medicine, University of Basrah, Basrah, Iraq.

***Corresponding Author's E-mail: Hindelia954@gmail.com**

Abstract

Hypertensive disorders in pregnancy, especially preeclampsia, affect 2–8% of pregnancies globally. Risk factors include prior hypertension, chronic conditions, advanced maternal age, obesity, infections and assisted reproduction. Poor placental development underlies preeclampsia pathogenesis, contributing to significant maternal and foetal risks. This study was conducted to determine the prevalence of preeclampsia and associated risk factors (family history, age, hypertension, diabetes, etc.), and to investigate its foetal and maternal outcomes. A longitudinal study was conducted at Al Basra Hospital (1st of September 2024–1st of Sept 2025). Preeclampsia prevalence, risk factors and outcomes were investigated. Pregnant women diagnosed with preeclampsia, excluding those with chronic conditions or extreme ages were included. Data collected covered socio-demographic, obstetric and medical history, but also family history of hypertension during pregnancy. The study included 75 women with a mean age of 33.2 years; most lived in urban areas and were housewives. Over half had regular antenatal care, and 46.7% had prior hypertension. Non-severe preeclampsia was more common (81.3%). Severe preeclampsia was significantly associated with lower gestational age at delivery, higher rates of eclampsia, emergency Caesarean sections, postpartum haemorrhage, lower birth weights, increased neonatal deaths, as well as more neonatal intensive care unit admissions, indicating worse maternal and foetal outcomes, compared to non-severe preeclampsia cases. Severe preeclampsia increases maternal and neonatal complications, including preterm delivery and neonatal intensive care unit admissions. Major risk factors include obesity, primigravidity, irregular antenatal care and prior hypertension.

Keywords: Fetal outcomes, Hypertensive disorders, Maternal outcomes, Preeclampsia, Pregnancy complications, Risk factors

INTRODUCTION

Hypertensive disorders of pregnancy affect nearly 10% of pregnancies (Ananth et al., 2013) and are defined by the International Society for the Study of Hypertension in Pregnancy (ISSHP) as new-onset hypertension (systolic ≥140 mmHg or diastolic ≥90 mmHg) after 20 weeks of gestation (Brown et al., 2018). They include chronic hypertension, gestational hypertension, and pre-eclampsia, which may also develop as a complication

superimposed on chronic hypertension. These conditions significantly impact maternal and foetal health both during and beyond pregnancy (Fox et al., 2019).

The pathogenesis of preeclampsia involves abnormal placental vasculature development, particularly impaired spiral artery remodelling, leading to placental hypoperfusion, hypoxia and oxidative stress. These changes result in the release of anti-angiogenic factors into maternal circulation, triggering systemic endothelial dysfunction and the clinical features of preeclampsia (Preeclampsia: Clinical features and diagnosis. [cited 2024 Dec 1].

Globally, preeclampsia affects 2–8% of pregnancies, with incidence influenced by healthcare access and maternal risk profiles. In developed countries, it accounts for 2–5% of pregnancies, with better prenatal care aiding early detection (Say et al., 2014; Kovacheva et al., 2024). In developing regions, prevalence is higher (4–8%) and contributes substantially to maternal mortality due to late diagnosis and inadequate treatment. Overall, preeclampsia and related hypertensive disorders are responsible for 10–15% of direct maternal deaths worldwide (Mayrink and Reis, 2024).

According to NICE guidelines (2019), women with a history of preeclampsia, chronic kidney disease, autoimmune disorders, diabetesand chronic hypertension are at high risk (National Guideline Alliance (UK) Hypertension in Pregnancy: Diagnosis and Management (NG133) [(cited 2024 Dec. 1)]. Moderate risk factors include nulliparity, maternal age ≥40 years, BMI ≥35 kg/m² (2), family history of preeclampsia, multiple pregnancy or inter-pregnancy interval >10 years (National Guideline Alliance (UK) Hypertension in Pregnancy: Diagnosis and Management (NG133) [(cited 2024 Dec. 1)]. Additional risks include elevated mean arterial blood pressure before 15 weeks (North et al., et al., 2011), polycystic ovarian syndrome (Bahri Khomami et al., 2019), sleep-disordered breathing and infections such as periodontal disease, urinary tract infection and *Helicobacter pylori* (Bellos et al., 2018; Nourollahpour et al., 2019). Use of oocyte donation in assisted reproduction also carries higher risk compared with IVF or natural conception (Blazquez et al., 2016).

Preeclampsia is diagnosed after 20 weeks of gestation, when hypertension is accompanied by new proteinuria, maternal organ dysfunction (renal, hepatic, neurological or haematological), or utero-placental dysfunction. It may present intra-partum or post-partum, and can also occur as super-imposed preeclampsia in women with chronic hypertension (Brown et al., 2018). Eclampsia, defined by seizures, is a severe complication (National Guideline Alliance (UK) Hypertension in Pregnancy: Diagnosis and Management (NG133) [(cited 2024 Dec. 1)].

Diagnostic criteria include confirmed hypertension plus at least one complication: proteinuria (spot urine P:Cr ≥30 mg/mmol or A:Cr ≥ 8 mg/mmol), renal impairment (creatinine ≥90 µmol/L), hepatic involvement (raised transaminases ± pain), neurological symptoms (eclampsia, stroke, visual disturbances), haematological abnormalities (thrombocytopenia, DIC, haemolysis) or utero-placental dysfunction such as growth restriction, abnormal Doppler or still-birth (Brown et al., 2018).

Management focuses on maternal stabilization and optimizing delivery timing. As preeclampsia accounts for 20–30% of pre-term births, interventions aim to minimize prematurity-related complications. Antenatal cortico-steroids improve foetal lung maturity, particularly between 26–34 weeks (Roberts et al., 2017; Porto et al., 2011), though some benefit may extend beyond 34 weeks and even at elective caesarean after 37 weeks (Saccone and Berghella, 2016). Risks such as altered off-spring vascular and glucose metabolism must be weighed carefully (Kelly et al., 2012). Magnesium sulphate is used for seizure prophylaxis and foetal neuro-protection, with strong evidence supporting its role in reducing cerebral palsy risk (National Guideline Alliance (UK) Hypertension in Pregnancy: Diagnosis and Management (NG133) [(cited 2024 Dec. 1)]; Doyle et al., 2009). Delivery remains the only definitive cure, and timing must balance maternal condition with foetal maturity (National Guideline Alliance (UK) Hypertension in Pregnancy: Diagnosis and Management (NG133) [(cited 2024 Dec. 1)].

Preeclampsia is a major cause of hypertension in pregnancy and increases the risk of stroke, heart failure and eclampsia (19, 20). Multi-organ dysfunction may develop, including HELLP syndrome (The dangers of preeclampsia: Every mom-to-be should be aware [Internet]. Bangkokhospital.com. [cited 2024 Dec 1]. Placental abruption is a life-threatening complication for both mother and foetus (Mikuscheva et al., 2021). Post-partum, affected women have higher risks of chronic hypertension, kidney disease, cardiovascular disease and haemorrhage, emphasizing the importance of long-term monitoring (Khosla et al., 2021; Madazli et al., 2014).

Preeclampsia is a leading cause of medically indicated pre-term delivery, exposing neonates to respiratory distress, infections and neurodevelopmental disorders (Backes et al., 2011). Intra-uterine growth restriction often results in low birth weight with related complications (Backes et al., 2011). Severe disease may cause still-birth from placental insufficiency (Fox et al., 2019). Furthermore, long-term risks for off-spring include increased susceptibility to hypertension and cardiovascular disease in adulthood (Fox et al., 2019).

Timely recognition, close monitoring and appropriate management significantly improve outcomes. However, preeclampsia still contributes to up to 25% of maternal deaths in some populations, including Caribbean, Latin American, Asian and Black women, highlighting persistent disparities (Gestational Hypertension and Preeclampsia: ACOG Practice Bulletin, 2020).

This study was conducted to determine the prevalence of preeclampsia and associated risk factors as family history, age, hypertension and diabetes, and to investigate its maternal and foetal outcomes.

METHODS

A longitudinal study was conducted at Al Basra Hospital for Maternity and Children from the 1st of September 2024 to the 1st of September 2025. The aim was to determine the prevalence of preeclampsia, explore-associated risk factors such as family history, age, hypertension and diabetes, but also to investigate maternal and foetal outcomes.

The study included all pregnant women diagnosed with preeclampsia during the study period, based on the clinical criteria of blood pressure ≥140/90 mmHg after 20 weeks of gestation, accompanied by proteinuria or other systemic involvement. Exclusion criteria were multiple gestations; women with pre-existing hypertension, chronic kidney disease, *Diabetes mellitus*, hemoglobino-pathy, primary aldosteronism, Cushing syndrome or pheochromocytoma/paraganglioma; other secondary causes of hypertension, as well as women younger than 15 years or older than 45 years.

Data were collected in the outpatient clinic using a structured questionnaire. Socio-demographic variability included age, educational level, occupation and residency. Obstetric history covered gravidity, parity, abortion, gestational age and history of hypertension in previous pregnancies. Past medical history was reviewed for autoimmune diseases, chronic kidney disease, hypertension, *Diabetes mellitus* or endocrine causes of hypertension. Surgical and drug histories were also documented, along with family history of hypertension during pregnancy.

All women underwent clinical examination, including assessment of vital signs (blood pressure, heart rate, respiratory rate and temperature). Blood pressure was measured using a calibrated sphygmomanometer, with hypertension defined as ≥140/90 mmHg. Anthropometric measurements of height, weight and body mass index (BMI) were recorded. Proteinuria was screened by urine dipstick: (+) for 30 mg/dL, (++) for 100 mg/dL and (+++) for 300 mg/dL. Laboratory tests included complete blood count, liver and renal function tests, coagulation profile, and serum uric acid levels. Doppler ultrasound was performed to evaluate placental and foetal circulation.

Women diagnosed with preeclampsia were categorized into severe and non-severe groups. Severe preeclampsia was defined by blood pressure ≥160/110 mmHg, proteinuria ≥++ or ≥3 g on semi-quantitative assessment, serum creatinine >100 mmol/L, thrombo-cytopenia, markedly elevated liver transaminases (>50 IU/L), foetal growth restriction, clonus, papilledema, liver tenderness, headache, visual disturbance, epigastric pain, oliguria and convulsions. Non-severe preeclampsia included blood pressure <160/110 mmHg, proteinuria + or ~1 g, normal creatinine, minimal liver enzyme elevation and absence of severe features.

Each woman was followed-up until delivery, and the mode of delivery (elective or emergency caesarean section or vaginal delivery) was recorded. Neonatal outcomes were also documented, including birth weight, sex, placental weight, NICU admission and overall survival.

Approval for the study was obtained from the Ethical Committee of the College of Medicine, Basrah University and the Basrah Health Directorate. Informed verbal consent was obtained from all participants. Confidentiality and privacy of data were maintained throughout the study.

Data were analysed using SPSS version 26. Categorical variables were expressed as frequencies and percentages,and analysed with the Chi-square test; Fisher's exact test was applied when expected counts were fewer than five. Continuous variables were presented as mean ± standard deviation and compared using independent-samples *t*-tests. A *p*-value ≤ 0.05 was considered statistically significant.

RESULTS

The study included 75 women. Their mean age was 33.2 ± 2.4 years, and 52% of them were between 20-29 years old. Regarding their residency, 69.3% lived in an urban area, 38.7% had a secondary education, and 85.3% were housewives. These data are shown in Table 1.

Table 2 shows the pregnancy-related characteristics of the participants. 57.3% of them had regular ANC visits. The mean gestational age at presentation was 34.2 weeks. Concerning gravidity, 42.7% of the tested women had 1-4 previous pregnancies, 46.7% had a history of HTN in previous pregnancy, and 36.0% had a family history of gestational HTN. Regarding gestational DM, 33.3% of women had positive history.

The clinical characteristics of the patients were presented in Table 3. 50.7% of women were overweight, and the mean BMI of participants was 29.91. The mean systolic blood pressure was 160.8 mmHg, and the mean diastolic blood pressure was 108.9 mmHg. According to the diastolic and systolic BP, the women were divided into women with severe and non-severe PE; 81.3% of patients had a non-severe PE.

The maternal outcomes concerning PE severity were presented in Table 4. The mean gestational age at delivery was lower among women with severe PE in comparison to those with non-severe PE (35.7 vs 37.5 weeks for both groups, respectively). The difference was statistically significant in p = 0.037. Regarding the development of eclampsia, 28.6% of women with severe PE compared to 1.6% of women in the non-severe PE

Table 1. The socio-demographic distribution of the participants

Variables		No.	%
Age	Mean ±SD	33.2 ± 2.4	
	>20	6	8.0
	20-29	39	52.0
	30-39	25	33.3
	≥40	5	6.7
Residency	Rural	23	30.7
	Urban	52	69.3
Educational level	Illiterate	11	14.7
	primary	25	33.3
	Secondary	29	38.7
	Higher education	10	13.3
Occupation	Housewives	64	85.3
	Employed	11	14.7

Table 2. The pregnancy-related characteristics of the participants

Variables		No.	%
ANC visits	Regular	43	57.3
	Irregular	32	42.7
Gestational age	Mean ± SD	34.2 ± 2.9	
Gravida	Primigravida	31	41.3
	1-4	32	42.7
	>5	16	16.0
History of HTN in previous pregnancy		35	46.7
Family history of HTN during pregnancy		27	36.0
Gestational DM		25	33.3

Table 3. The clinical characteristics of the patients

Variables		No.	%
BMI	Mean± SD	29.91 ± 1.9	
	Normal	11	14.7
	Overweight	38	50.7
	Obese	26	34.6
Blood pressure	Systolic	160.8 ± 10.32	
	Diastolic	108.9 ± 8.7	
PE severity	Severe PE	14	18.7
	Non severe PE	61	81.3

Table 4. The association between PE severity and maternal outcome

Maternal outcome	Severe PE N=14	Non-severe PE N=61	*p*-value
Gestational age at delivery (mean)	35.7 ±1.3	37.5 ±1.9	
Term	5 (35.7)	50 (81.9)	0.037
Preterm	9 (64.3)	11 (18.1)	
Eclampsia	4 (28.6)	1 (1.6)	0.001
Mode of delivery			
Emergency CS	11 (78.6)	18 (29.5)	
Elective CS	3 (21.4)	21 (34.4)	0.006
Normal vaginal delivery	0 (0.0)	22 (36.1)	
Postpartum hemorrhage	4 (28.6)	2 (3.3)	0.001

Table 5. The association between PE Severity and fetal outcome

Fetal outcome	Severe PE	Non-severe PE	*p*-value
Baby fate (alive/died)	12 (85.7)	60 (98.4)	0.027
	2 (14.3)	1 (1.6)	
Weight	2.1 ± 0.8	3.4 ±1.3	0.001
Sex	8 (57.1)	29 (47.6)	0.516
	6 (42.9)	32 (52.5)	
NICU admission	7 (50.0)	14 (22.9)	0.016

group, and this difference is statistically significant in p=0.001. The mode of delivery also shows a statistically significant difference between the two groups, as 78.6% of women in the first group underwent emergency CS in comparison to 29.5% of women in the second group, p =0.006. Regarding PPH, 28.6% of the women with severe PE and 3.3% of these in the second group had post-partum hemorrhage, p =0.001.

The proportion of alive babies was significantly lower in the severe preeclampsia (PE) group (85.7%) compared to the non-severe PE group (98.4%). The p-value (0.027) indicates a statistically significant association between PE severity and baby survival. The mean birth weight was significantly lower in the severe PE group (2.1 ± 0.8 kg) compared to the non-severe PE group (3.4 ± 1.3 kg). The p-value (0.001) confirms that PE severity is significantly associated with lower birth weight. The sex distribution (male/female) did not significantly differ between the severe PE and non-severe PE groups (p = 0.516). The percentage of NICU admissions was significantly higher in the severe PE group (50.0%) compared to the non-severe PE group (22.9%). The p-value (0.016) indicates a significant association, highlighting that severe PE is a risk factor for adverse neonatal conditions, requiring intensive care.

DISCUSSION

Preeclampsia (PE) remains a leading cause of maternal and perinatal morbidity and mortality worldwide, particularly in low- and middle-income countries (Karrar et al., 2025). This study aimed to investigate the clinical severity of preeclampsia and its impact on maternal and neonatal outcomes in a cohort of women admitted to Al-Basrah Hospital for Maternity and Children. The rationale behind this research is the high burden of preeclampsia in Iraq, where regional epidemiological data are limited, and early identification of high-risk cases can significantly improve outcomes through timely intervention and resource allocation. The majority of participants were aged between 20–29 years (52%) and 30–39 years (33.3%), with a mean age of 33.2 years. This distribution aligns with the typical reproductive age range and is consistent with previous studies reporting the highest incidence of preeclampsia among women in their 20s and 30s as reported by Sheen et al. (2020). Most participants resided in urban areas (69.3%) and were housewives (85.3%), reflecting the local population socio-demographic profile (Sheen et al., 2020). Interestingly, 48% of participants had only primary or no formal education. This finding supports earlier literature, linking lower educational attainment with increased preeclampsia risk due to poor health literacy, reduced antenatal care uptake, as well as delayed complication recognition as mentioned by (Chang et al. (2023) and Atulri et al. (2023).

Only 57.3% of participants had regular antenatal care (ANC), indicating a substantial proportion (42.7%) with inadequate follow-up. This is concerning given that early and consistent ANC is crucial for identifying preeclampsia and managing complications before they escalate as reported by Dasgupta et al. (2025). The mean gestational age at presentation was 34.2 weeks, with a large number being primigravida (41.3%). Both primigravidity and irregular ANC are well-documented risk factors for preeclampsia (Dasgupta et al., 2025; English et al., 2015). Additionally, 46.7% had a history of hypertension in a previous pregnancy, and 36% reported a family history of hypertensive disorders during pregnancy, further supporting the genetic and recurrent risk patterns of preeclampsia (Kivioja et al., 2022). A third of participants had gestational diabetes, a known comorbidity that exacerbates PE risk and adverse outcomes, which is in agreement with Yang et al. (2022). A striking finding was the elevated average BMI of 29.91, with more than 85% of women being overweight or obese. This reinforces the established association between obesity and preeclampsia through mechanisms involving chronic inflammation, endothelial dysfunction and insulin resistance (Lopez-Jaramillo et al., 2018).

The average systolic and diastolic pressure in this cohort (160.8/108.9 mmHg) indicate the severity of hypertensive burden. Among the participants, 18.7% were diagnosed with severe PE. Though not the majority, this subset had substantially worse clinical outcomes, which is in line with Turbeville et al. (2020). Severe PE was significantly associated with earlier delivery, with a mean gestational age of 35.7 weeks compared to 37.5 weeks in non-severe cases (p = 0.037). Pre-term delivery occurred in 64.3% of severe PE cases, which aligns with prior studies that link severe preeclampsia with indicated

pre-term births due to fetal or maternal compromise as reported by van Esch et al. (2017) and Guida et al. (2017). Furthermore, severe PE was strongly associated with eclampsia (28.6% vs. 1.6%, $p = 0.001$), emergency Cesarean section (78.6% vs. 29.5%, $p = 0.006$), and post-partum hemorrhage (28.6% vs. 3.3%, $p = 0.001$). These findings are consistent with the literature describing increased obstetric interventions and complications in severe PE cases (38, 39). The higher emergency CS rate among severe PE patients reflects the urgent need for delivery due to deteriorating maternal or fetal conditions as explained by Chang et al. (2023), while the increased PPH incidence may relate to uterine atony from prolonged labor, coagulation defects or *abruptio placentae* (Wormer et al., 2025). Fetal outcomes were notably worse in the severe PE group. Neonatal mortality was significantly higher (14.3% vs. 1.6%, $p = 0.027$), and mean birth weight was significantly lower (2.1 kg vs. 3.4 kg, $p = 0.001$). This supports findings by Almuhaytib et al. (2023) and Srinivas et al. (2019), who reported that intrauterine growth restriction (IUGR) and low birth weight are common in severe PE due to utero-placental insufficiency (Almuhaytib et al., 2023; Srinivas et al., 2019). Furthermore, NICU admission was significantly more common among mother infants with severe PE (50% vs. 22.9%, $p = 0.016$), indicating higher neonatal morbidity, which is in agreement with Mendola et al. (2015). The lack of significant association between fetal sex and PE severity ($p = 0.516$) suggests that sex-linked genetic factors did not influence PE severity in this cohort, consistent with previous conflicting evidence on this topic (Campbell et al., 2023).

The findings in the presented study strongly support the understanding that severe preeclampsia significantly increases the risks of adverse maternal and neonatal outcomes. These outcomes are largely driven by the diseases patho-physiological mechanisms — endothelial dysfunction, systemic inflammation, placental hypo-perfusion and multi-organ involvement (Phipps et al., 2019). The high rates of pre-term birth, eclampsia, NICU admission and emergency Cesarean section in severe PE cases emphasize the importance of early identification and multidisciplinary management. The considerable burden among women with low education and irregular ANC underscores the need for public health interventions focusing on maternal education, accessible antenatal services and community health awareness. This study adds valuable Iraqi-specific data to the global understanding of PE and supports the call for targeted antenatal strategies in developing regions.

The presented study was conducted at a single tertiary hospital in Basrah, which may limit the gene-ralizability of the findings to other regions in Iraq or to different healthcare settings. The sample size, though adequate for detecting significant associations, may not capture the full spectrum of disease severity or long-term outcomes. Self-reported data on antenatal care visits and obstetric history may be subject to recall bias.

CONCLUSIONS AND RECOMMENDATIONS

The current study demonstrates that severe preeclampsia is strongly associated with increased maternal and neonatal morbidity, including higher risks of pre-term delivery, eclampsia, post-partum haemorrhage, low birth weight, neonatal death and NICU admission. The main risk factors identified were obesity, primi-gravidity, irregular antenatal care and prior hypertension. Strengthening antenatal care services with routine screening is essential for early detection and prevention of complications. Health education programs should target women of reproductive age to improve awareness of nutrition, weight management and warning signs of preeclampsia. Risk-based surveillance should be implemented for women with a family or personal history of hypertension and related disorders. In addition, emergency obstetric care must be adequately equipped, ensuring access to NICU support and blood transfusion services. Finally, multi-centre studies are recommended to validate these findings and to investigate the long-term outcomes of preeclampsia in Iraq.

Conflicts of Interests: None

Funding: No funding body was involved in this study.

Ethical Approvals: Ethical approval for the study was obtained from the relevant institutional review board, and informed consent was acquired from all participants prior to their inclusion in the study.

REFERENCES

Almuhaytib FA, AlKishi NA, Alyousif ZM (2023). Early Onset Preeclampsia and Intrauterine Growth Restriction: A Case Report. Cureus. Jan 18;15(1):e33919.

Ananth CV, Keyes KM, Wapner RJ (2013). Pre-Eclampsia Rates in the United States, 1980–2010: Age-Period-Cohort Analysis. BMJ. 347:f6564.

Atluri N, Beyuo TK, Oppong SA, Moyer CA, Lawrence ER (2023). Challenges to diagnosing and managing preeclam-psia in a low-resource setting: A qualitative study of obstetric provider perspectives from Ghana. PLOS Glob Public Health.;3(5):e0001790.

Backes CH, Markham K, Moorehead P, Cordero L, Nankervis CA, Giannone PJ (2011). Maternal preeclampsia and neonatal outcomes. J Pregnancy. 2011:214365.

Bahri Khomami M, Joham AE, Boyle JA, Piltonen T, Silagy M, Arora C, Misso ML, Teede HJ, Moran LJ (2019). Increased Maternal Pregnancy Complications in Polycystic Ovary Syndrome Appear to Be Independent of Obesity-A Systematic Review, Meta-Analysis, and Meta-Regression.

Obes. Rev. 20:659–674.
Bellos I, Daskalakis G, Pergialiotis V (2018). Helicobacter Pylori Infection Increases the Risk of Developing Preeclampsia: A Meta-Analysis of Observational Studies. Int. J. Clin. Pract. 72:349–353.
Blazquez A., Garcia D., Rodriguez A., Vassena R., Figueras F., Vernaeve V (2016). Is Oocyte Donation a Risk Factor for Preeclampsia? A Systematic Review and Meta-Analysis. J. Assist. Reprod. Genet.;33:855–863.
Brown MA, Magee LA, Kenny LC, Karumanchi SA, McCarthy FP, Saito S, Hall DR, Warren CE, Adoyi G, Ishaku S (2018). Hypertensive Disorders of Pregnancy: ISSHP Classification, Diagnosis, and Management Recommendations for International Practice. Hypertension.;72:24–43.
Bushnell C, Chireau M (2011). Preeclampsia and Stroke: Risks during and after Pregnancy. Stroke Res Treat. 2011: 858134.
Campbell N, Solise D, Deer E, LaMarca B (2023). Sex differences in offspring of preeclamptic pregnancies. Curr Opin Physiol.;34(100688):100688.
Chang KJ, Seow KM, Chen KH (2023). Preeclampsia: Recent Advances in Predicting, Preventing, and Managing the Maternal and Fetal Life-Threatening Condition. Int J Environ Res Public Health. 20(4):2994.
Dasgupta DP, Suman SM, Sultana N, Sujon MSH, Fatema K, Toma MJ, Hossain MM (2025). Comparative study on the factors influencing pre-eclampsia symptoms at different pregnancy stages in Bangladeshi women: urban vs. rural perspectives. BMC Pregnancy Childbirth. 25(1):450.
Doyle LW, Crowther CA, Middleton P, Marret S, Rouse D (2009). Magnesium sulphate for women at risk of preterm birth for neuroprotection of the fetus. Cochrane Database Syst Rev. (1):CD004661.
English FA, Kenny LC, McCarthy FP (2015). Risk factors and effective management of preeclampsia. Integr Blood Press Control.;8:7-12.
Fox R, Kitt J, Leeson P, Aye CYL, Lewandowski AJ (2019). Preeclampsia: Risk Factors, Diagnosis, Management, and the Cardiovascular Impact on the Offspring. J Clin Med.;8(10):1625.
Gestational Hypertension and Preeclampsia: ACOG Practice Bulletin, Number 222. Obstet Gynecol. 2020; 135(6):e237-e260.
Guida JPS, Surita FG, Parpinelli MA, Costa ML (2017). Preterm Preeclampsia and Timing of Delivery: A Systematic Literature Review. Rev Bras Ginecol Obstet. 39(11):622-631.
Karrar SA, Martingano DJ, Hong PL (2025). Preeclampsia. [Updated 2024 Feb 25]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK570611/
Kelly BA, Lewandowski AJ, Worton SA, Davis EF, Lazdam M, Francis J, Neubauer S, Lucas A, Singhal A, Leeson P (2012). Antenatal Glucocorticoid Exposure and Long-Term Alterations in Aortic Function and Glucose Metabolism. Pediatrics.;129:e1282–e1290.
Khosla K, Heimberger S, Nieman KM, Tung A, Shahul S, Staff AC, Rana S (2021). Long-Term Cardiovascular Disease Risk in Women After Hypertensive Disorders of Pregnancy: Recent Advances in Hypertension. Hypertension. 78(4):927-935.
Kivioja A, Toivonen E, Tyrmi J, Ruotsalainen S, Ripatti S, Huhtala H, Jääskeläinen T, Heinonen S, Kajantie E, Kere J, Kivinen K, Pouta A, Saarela T, Laivuori H (2022). Increased Risk of Preeclampsia in Women With a Genetic Predisposition to Elevated Blood Pressure. Hypertension. 79(9):2008-2015.
Kovacheva VP, Venkatachalam S, Pfister C, Anwer T (2024). Preeclampsia and eclampsia: Enhanced detection and treatment for morbidity reduction. Best Pract Res Clin Anaesthesiol. 38(3):246–56.
Lopez-Jaramillo P, Barajas J, Rueda-Quijano SM, Lopez-Lopez C, Felix C (2018). Obesity and Preeclampsia: Common Pathophysiological Mechanisms. Front Physiol. 9:1838.
Madazli R., Yuksel M.A., Imamoglu M., Tuten A., Oncul M., Aydin B., Demirayak G (2014). Comparison of Clinical and Perinatal Outcomes in Early- and Late-Onset Preeclampsia. Arch. Gynecol. Obstet. 290:53–57.
Mayrink J, Reis ZSN (2024). Pre-eclampsia in low and middle-income settings: What are the barriers to improving perinatal outcomes and evidence-based recommendations? Int J Gynaecol Obstet. 164(1):33-39.
Mendola P, Mumford SL, Männistö TI, Holston A, Reddy UM, Laughon SK (2015). Controlled direct effects of preeclampsia on neonatal health after accounting for mediation by preterm birth. Epidemiology.;26(1):17-26.
Mikuscheva A, Strassding F, MacKenzie E (2021). Three cases of severe placental abruption as a first symptom of preeclampsia. Case Rep Obstet Gynecol [Internet]. 2021:3863607.
National Guideline Alliance (UK) Hypertension in Pregnancy: Diagnosis and Management (NG133) [(cited 2024 Dec. 1)];2019 Available online: https://www.nice.org.uk/guidance/ng133
North RA, McCowan LME, Dekker GA, Poston L, Chan EHY, Stewart AW, Black MA, Taylor RS, Walker JJ, Baker PN, et al. (2011). Clinical Risk Prediction for Pre-Eclampsia in Nulliparous Women: Development of Model in International Prospective Cohort. BMJ. 342:d1875.
Nourollahpour Shiadeh M., Riahi S.M., Adam I., Saber V., Behboodi Moghadam Z., Armon B., Spotin A., Nazari Kangavari H., Rostami A (2019). Helicobacter Pylori Infection and Risk of Preeclampsia: A Systematic Review and Meta-Analysis. J. Matern. Fetal. Neonatal Med.;32:324–331.
Pan Y, Wang Y, Miao J, Ji X, Wu C, Wang Y, Ding H (2024). Risk Factors for Postpartum Hemorrhage in Severe Pre-Eclampsia: A Retrospective Single-Centre Study of 1953 Cases. Med Sci Monit. 30:e943772.
Pasokpuckdee K, Boriboonhirunsarn D (2023). Incidence of Preeclampsia and Cesarean Section Rate According to the Robson Classification. Cureus. 15(12):e49845.
Phipps EA, Thadhani R, Benzing T, Karumanchi SA (2019). Pre-eclampsia: pathogenesis, novel diagnostics and therapies. Nat Rev Nephrol. May;15(5):275-289. doi: 10.1038/s41581-019-0119-6. Erratum in: Nat Rev Nephrol. 2019;15(6):386.
Porto AMF, Coutinho IC, Correia JB, Amorim MMR (2011). Effectiveness of Antenatal Corticosteroids in Reducing Respiratory Disorders in Late Preterm Infants: Randomised Clinical Trial. BMJ.;342:d1696.
Preeclampsia: Clinical features and diagnosis. [cited 2024 Dec 1]. 2024. [Internet]. Available from: https://www.uptodate.com/contents/preeclampsia-clinical-features-and-diagnosis
Roberts D, Brown J, Medley N, Dalziel SR (2017). Antenatal Corticosteroids for Accelerating Fetal Lung Maturation for

Women at Risk of Preterm Birth. Cochrane Database Syst. Rev.;3:CD004454.
Saccone G., Berghella V (2016). Antenatal Corticosteroids for Maturity of Term or near Term Fetuses: Systematic Review and Meta-Analysis of Randomized Controlled Trials. BMJ. 355:i5044.
Say L, Chou D, Gemmill A, Tunçalp Ö, Moller AB, Daniels J, et al. (2014). Global causes of maternal death: a WHO systematic analysis. The Lancet Global Health. 2(6):e323–33.
Sheen JJ, Huang Y, Andrikopoulou M, Wright JD, Goffman D, D'Alton ME, Friedman AM (2020). Maternal Age and Preeclampsia Outcomes during Delivery Hospitalizations. Am J Perinatol. 37(1):44-52.
Srinivas SK, Edlow AG, Neff PM, Sammel MD, Andrela CM, Elovitz MA (2019). Rethinking IUGR in preeclampsia: dependent or independent of maternal hypertension? J Perinatol. t;29(10):680-4.
The dangers of preeclampsia: Every mom-to-be should be aware [Internet]. Bangkokhospital.com. [cited 2024 Dec 1]. Available from: https://www.bangkokhospital.com/en/ content/preeclampsia-pregnant-woman-must-be-careful
Turbeville HR, Sasser JM (2020). Preeclampsia beyond pregnancy: long-term consequences for mother and child. Am J Physiol Renal Physiol. 318(6):F1315-F1326.
van Esch JJA, van Heijst AF, de Haan AFJ, van der Heijden OWH (2017). Early-onset preeclampsia is associated with perinatal mortality and severe neonatal morbidity. J Matern Fetal Neonatal Med. 30(23):2789-2794.
Wormer KC, Jamil RT, Bryant SB (2025). Postpartum Hemorrhage. [Updated 2024 Jul 19]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; Available from: https://www.ncbi.nlm.nih.gov/ books/NBK499988/
Yang Y, Wu N (2022). Gestational Diabetes Mellitus and Preeclampsia: Correlation and Influencing Factors. Front Cardiovasc Med. 9:831297.