International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	HYPERTENSIVE DISORDERS IN PREGNANCY: FROM PATHOGENESIS TO CLINICAL MANAGEMENT
DOI	https://doi.org/10.31435/ijitss.3(51).2026.6291
RECEIVED	30 April 2026
ACCEPTED	15 July 2026
PUBLISHED	23 July 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

HYPERTENSIVE DISORDERS IN PREGNANCY: FROM PATHOGENESIS TO CLINICAL MANAGEMENT

Maria Sierant *(Corresponding Author, Email: sierant.maria@gmail.com)*
Nicolaus Copernicus Voivodeship Hospital, Koszalin, Poland
ORCID ID: 0009-0002-1012-742X

Kornelia Tyczka
Samodzielny Publiczny Wojewódzki Szpital Zespolony w Szczecinie, Szczecin, Poland
ORCID ID: 0000-0001-8700-2372

Zofia Cepowska
Pomeranian Medical University Hospital No. 1, Szczecin, Poland
ORCID ID: 0009-0005-7910-0833

Maria Nowakowska
Pomeranian Medical University Hospital No. 1, Szczecin, Poland
ORCID ID: 0009-0007-2321-0464

ABSTRACT

Hypertension in pregnancy combines several conditions, such as chronic hypertension, gestational hypertension, unclassified hypertension before delivery, and preeclampsia. According to WHO data, preeclampsia complicates 3-8% of pregnancies and contributes significantly to maternal and neonatal mortality. In recent years, hypertension in pregnancy has been diagnosed more and more frequently, which is associated with the prevalence of conditions such as diabetes, metabolic syndrome, dyslipidemia, and obesity, but also with the increasing average age of women becoming pregnant. The following article discusses the diagnosis, prevention, and non-pharmacological and pharmacological treatment of this condition. The authors also highlight the role of lifestyle modifications during the preconception period in preventing maternal and fetal complications. The safety of pharmacotherapy for both women and newborns is also discussed. In women with preeclampsia, magnesium sulfate should be used to reduce the risk of eclampsia by more than half. The following article is a review of the diagnosis and treatment of hypertensive disorders occurring during pregnancy.

KEYWORDS

Hypertension, Pregnancy, Preeclampsia, Treatment

CITATION

Maria Sierant, Kornelia Tyczka, Zofia Cepowska, Maria Nowakowska. (2026) Hypertensive Disorders in Pregnancy: From Pathogenesis to Clinical Management. *International Journal of Innovative Technologies in Social Science*. 3(51). doi: 10.31435/ijitss.3(51).2026.6291

Introduction

Hypertension in pregnancy is a complex term combining several disease entities. The precise classification of hypertension in pregnancy is presented in Table I. According to data from the World Health Organization (WHO), preeclampsia occurs in 3–8% of pregnant women worldwide, and at the same time, it is one of the pathological conditions occurring in pregnancy associated with the highest risk of complications and mortality for both the mother and the newborn [1]. Hypertension in pregnancy can lead to complications such as preterm labor, fetal growth restriction, HELLP syndrome (hemolysis, elevated liver enzymes, low platelet count), and eclampsia – which involves consciousness disorders with co-occurring tonic-clonic seizures. Therefore, increasing awareness of PE among doctors of all specialties, as well as patients and their families, is crucial for the early identification of risk factors and the prevention of complications [2].

Table 1. Classification of hypertension in pregnant women. Own elaboration based on literature.

Type of Hypertension	Definition	Characteristic Criteria	Postpartum Course
Chronic hypertension	Hypertension present before pregnancy or developing before the 20th week of gestation	May coexist with proteinuria	Persists > 6 weeks postpartum
Gestational hypertension	Hypertension developing after the 20th week of gestation	Absence of proteinuria and signs of organ damage	Usually resolves within 6 weeks postpartum
Unclassified hypertension before delivery	First blood pressure measurement after the 20th week of gestation	Diagnosed hypertension, but impossible to determine whether it is chronic or gestational	Classification is only possible after postpartum observation
Preeeclampsia	Hypertension after the 20th week of gestation	Hypertension + proteinuria (>0.3 g/day or ACR ≥30 mg/mmol) OR maternal organ damage (kidneys, liver, CNS, coagulation system) OR uteroplacental dysfunction	Usually resolves after delivery

Abbreviations: gw (gestational week / weeks of gestation); CNS – Central nervous system; ACR – Albumin/Creatinine Ratio.

Pathogenesis

Pregnancy involves profound hemodynamic and hormonal changes that under normal conditions enable fetal development. However, in some women, deviations in the physiological adaptive mechanisms of the maternal body can lead to the development of hypertension (HA). PE represents a specific form of hypertension in pregnancy, where placental pathology and a secondary, generalized vascular-inflammatory response of the maternal organism play a key role. In a normal pregnancy, there is a significant increase in plasma volume (by approximately 40–50%) and cardiac output, accompanied by a simultaneous reduction in systemic vascular resistance. These changes are modulated by pregnancy hormones, including progesterone, estrogens, and relaxin, which promote vasodilation and cardiovascular adaptation. The renin–angiotensin–aldosterone system is also activated, leading to sodium and water retention, which physiologically expands the circulating blood volume. In some cases, compensatory mechanisms prove insufficient or become deregulated, resulting in an excessive increase in blood pressure (BP), particularly in the second half of pregnancy. Endothelial dysfunction, increased vascular reactivity to vasoconstrictors, and relative resistance to the action of vasodilators can contribute to the development of pathological BP values.

Despite intensive research, the pathogenic mechanisms of preeclampsia remain not fully understood, and its development involves alterations at the cellular, vascular, immunological, and metabolic levels. The most widely accepted model explaining the pathogenesis of PE is the two-stage hypothesis [3]. The first stage of the disease occurs in early pregnancy and is associated with abnormal trophoblast implantation and impaired remodeling of the uterine spiral arteries. Trophoblast cells do not penetrate deeply enough into the decidua and myometrium, and the spiral arteries retain their muscular layer, failing to transform into high-caliber, low-resistance vessels. This results in restricted blood flow through the placenta, its chronic hypoxia, and perfusion instability [2]. In response to hypoxia and environmental stress, the placenta releases numerous bioactive factors into the maternal circulation, initiating the second stage of the disease – a systemic vascular-inflammatory response. Antianigogenic factors play a crucial role here, particularly sFlt-1 (Soluble Fms-like

Tyrosine Kinase-1) and sEng (Soluble Endoglin). sFlt-1, which is a soluble form of the VEGFR-1 (Vascular Endothelial Growth Factor Receptor 1) receptor, binds and neutralizes proangiogenic factors such as VEGF (Vascular Endothelial Growth Factor) and PlGF (Placental Growth Factor), leading to their functional deficiency. Meanwhile, sEng disrupts TGF-β (Tumor Growth Factor-β)-dependent signaling, which is essential for maintaining endothelial integrity [4].

An essential component of the pathogenesis of PE and hypertension in pregnancy is oxidative stress arising from placental hypoxia. Excessive production of reactive oxygen species leads to damage to the trophoblast, endothelial cells, and maternal internal organs. Disruption of mitochondrial antioxidant mechanisms further intensifies free radical production, promoting the progression of vascular dysfunction [5,6].

Immunological mechanisms also play a significant role in the pathogenesis of PE. Improper adaptation of the maternal immune system to the presence of the semi-allogeneic fetus leads to an imbalance between NK (Natural Killer) cells and regulatory T lymphocytes. The result is an intensified inflammatory reaction and abnormal remodeling of the spiral vessels, which secondarily promotes both the development of hypertension and placental damage [7-9].

Predisposition to the development of hypertension in pregnancy and PE can also be genetically and epigenetically determined. Polymorphisms in genes related to angiogenesis, including Flt-1 (Fms-like tyrosine kinase-1), as well as epigenetic modifications such as DNA methylation or altered microRNA expression, influence the regulation of pathways responsible for angiogenesis, immune response, and oxidative stress [10-13].

Preeeclampsia

This is the most severe form of hypertension in pregnancy, occurring in approximately 2% of pregnant women [14]. There are many recognized risk factors described by various scientific societies. They have been divided into moderate-risk and high-risk factors, and their compilation is presented in Table II.

Table 2. Risk factors for developing PE. Own elaboration based on literature.

Scientific Society	Moderate Risk Factors	High Risk Factors
ACOG	First pregnancy Age >35 years Interpregnancy interval >10 years BMI >30 kg/m² Family history of PE History of SGA Low socioeconomic status	PE in a previous pregnancy Chronic hypertension Systemic lupus erythematosus Type I or II diabetes Chronic kidney disease Multiple pregnancy Antiphospholipid syndrome
NICE	First pregnancy Age >40 years Interpregnancy interval >10 years BMI >30 kg/m² Family history of PE Multiple pregnancy	PE in a previous pregnancy Chronic hypertension Autoimmune diseases Type I or II diabetes Chronic kidney disease Antiphospholipid syndrome

Abbreviations: ACOG – American College of Obstetricians and Gynecologists; NICE – National Institute for Health and Clinical Excellence; BMI – Body Mass Index; SGA – Small for Gestational Age.

It is worth emphasizing that obesity is one of the modifiable risk factors for PE. It has been shown how critical patient education in the periconceptional period regarding weight control and reduction can be. A study of over 28,000 nulliparous women with singleton pregnancies shows that pre-pregnancy BMI is one of the key modifiable risk factors for the development of preeclampsia, especially its form occurring before the 37th week of gestation, which is associated with a higher risk of complications for both the mother and the fetus. Additionally, women who developed PE before the 37th week of gestation were significantly more likely to be obese before becoming pregnant (33.1% vs 25.3%). The highest risk was found in women with a BMI ≥ 40 kg/m² – for preterm preeclampsia, the relative risk was RR 5.23 (95% CI: 3.86–7.09), which means a more than fivefold increase in the likelihood of this complication compared to women with a normal body weight [15].

The diagnostic criteria for preeclampsia proposed by various organizations differ slightly. The main difference is that proteinuria is no longer a required parameter for diagnosing preeclampsia according to the ISSHP guidelines. The criteria proposed by the ISSHP (The International Society for the Study of Hypertension in Pregnancy) are presented below in Table III [16].

Table 3. Criteria for diagnosing preeclampsia according to the ISSHP

Preeeclampsia is pregnancy-induced hypertension accompanied by ≥1 of the following clinical situations, the development of which occurred ≥20th week of gestation:
• Proteinuria
• Acute kidney injury (serum creatinine concentration ≥1 mg/dl; 90 μmol/l)
• Hepatic complications (elevated transaminase activity, e.g., AST or ALT >40 IU/l) with or without pain in the right upper quadrant or epigastrium
• Hematological complications (platelet count <150 000/μl, disseminated intravascular coagulation syndrome, hemolysis)
• Neurological complications (e.g., eclampsia, altered mental status, blindness, stroke, clonus, severe headaches, and persistent scotomas)
• Signs of fetal distress (IUGR, abnormal umbilical artery flow in Doppler ultrasound, or intrauterine fetal demise)

Abbreviations: ALT – alanine aminotransferase, AST – aspartate aminotransferase.

Due to the multifactorial background of this disorder, the diagnostic process must take into account both the risk factors for preeclampsia and appropriate biophysical and biochemical markers. Biophysical markers include mean arterial pressure (MAP) and the uterine artery pulsatility index (UtA PI). On the other hand, biochemical markers include pregnancy-associated plasma protein A (PAPP-A) and placental growth factor (PlGF) [17]. Based on these parameters and data obtained from the patient's medical history, an individual risk assessment for preeclampsia in the first trimester of pregnancy is possible using the FMF (Fetal Medicine Foundation) calculator. This tool also allows for the estimation of the risk of IUGR.

This method is recommended as the standard for assessing individual preeclampsia risk. If a risk value higher than 1:150 is obtained, it is recommended to implement prophylaxis in the form of acetylsalicylic acid (ASA) at a dose of 100–150 mg, taken daily in the evening [13]. The FMF calculator algorithm allows for the identification of over 90% of early preeclampsia cases, with a false-positive rate of approximately 10% [18].

An increasing number of reports indicate the possibility of predicting preeclampsia in the second trimester of pregnancy as well, i.e., between the 19th and 24th week of gestation. The risk assessment takes into account parameters such as the number of previous pregnancies, the uterine artery PI index, MAP, plasma PlGF concentration, and sFlt-1 levels [19]. It has also been shown that in the second trimester, the sFlt-1 concentration begins to rise gradually, while the PlGF level decreases as early as the end of the first trimester [20].

Non-pharmacological Treatment and Prevention

Non-pharmacological interventions, such as smoking cessation and low- to moderate-intensity physical activity, serve as predictive factors, especially if introduced in the first trimester of pregnancy. In women with low daily dietary calcium intake (<600 mg per day), oral supplementation at a dose of 0.5–2 g daily is recommended [12]. An individual PE risk assessment should be performed for every patient at the beginning of pregnancy. If high or moderate risk is identified, acetylsalicylic acid at a dose of 100–150 mg/day should be initiated before the 16th week of gestation, with the recommendation to take it in the evening [2].

Chronic Hypertension – Treatment

Pregnancy in a woman suffering from chronic hypertension should be planned in consultation with a hypertensiologist or a cardiologist. Before pregnancy, laboratory and electrocardiographic tests should be performed to assess, among other things, kidney, liver, and heart function. In the preconception period, treatment modification should be considered to discontinue drugs potentially teratogenic to the fetus and introduce medications deemed safe. The use of drugs from the groups of mineralocorticoid receptor

antagonists, angiotensin II receptor antagonists, angiotensin II converting enzyme inhibitors, and renin inhibitors is not recommended in women of childbearing potential, except for patients with specific indications. In women planning a pregnancy, changing the current antihypertensive treatment to medications typically used in the treatment of hypertension in pregnancy should be considered. Pregnancy in women with chronic hypertension is a high-risk pregnancy and can be burdened with numerous complications. Daily monitoring of blood pressure and performing a urinalysis to evaluate for proteinuria are very important. The division of drugs used in chronic therapy is shown in Table IV.

Table 4. Medications in chronic antihypertensive therapy in pregnant women. Own elaboration based on literature.

Recommendation	Drug
First-choice drugs	Methyldopa Labetalol Nifedipine Metoprolol
Relatively safe drugs	Dihydralazine Prazosin
Contraindicated drugs	Diuretics Angiotensin-converting enzyme inhibitors Angiotensin receptor antagonists

Pharmacological Treatment of Mild Hypertension

In cases where hypertension is diagnosed in a pregnant woman, the European Society of Cardiology (ESC) recommends initiating antihypertensive treatment to reduce the risk of developing severe hypertension, and consequently, complications for both the pregnant woman and the fetus. Among the drugs used in the therapy of hypertension in pregnant women, we distinguish β-adrenergic receptor antagonists, dihydropyridine calcium channel antagonists, and centrally acting drugs.

From the β-blocker group, labetalol (a non-selective β-receptor antagonist and a postsynaptic α1-receptor antagonist), metoprolol, and bisoprolol (selective β-adrenergic receptor antagonists) can be used in pregnant women [2,12]. They belong to the most frequently used cardiac medications in patients with heart disease – according to ROPAC (Registry of Pregnancy and Cardiac disease), they are taken by as many as 15% of pregnant women with cardiac burdens. Ramlakhan et al. conducted a multivariate regression analysis based on data collected by ROPAC, which is part of the EURObservational Research Programme observational study registry conducted by the ESC and constitutes a prospective, worldwide registry of pregnancies in women with structural heart diseases. The researchers analyzed 5,739 pregnancies across 138 centers in 53 countries between January 2008 and January 2018, adjusting for maternal factors such as BMI, heart disease, cardiomyopathies, primiparity, smoking, diabetes, maternal age, and a propensity for risky behaviors. Data analysis revealed that the use of atenolol was more frequently associated with the occurrence of SGA in newborns (OR 2.3, 1.1–4.9), whereas it occurred less frequently with the use of propranolol (OR 0.2, 95% CI 0.1–0.9) and labetalol (OR 0.2, 0.1–0.4). It was found that the use of atenolol compared to bisoprolol was more often linked to the occurrence of SGA (OR 2.7, 1.2–5.6), while labetalol was less likely to cause fetal growth restriction (OR 0.2, 0.1–0.8).

Furthermore, in newborns whose mothers took β-blockers during pregnancy, a difference in mean birth weight of –177g or –5.8% was noted [21].

Nifedipine, belonging to the dihydropyridine calcium channel blockers, is the most frequently mentioned drug in the antihypertensive therapy of pregnancy among medications in this class [2]. Leonard et al. analyzed data concerning 6,724 pregnant women with chronic hypertension who took either nifedipine or labetalol. Target blood pressure values were achieved in 44% of patients taking nifedipine and 42% of patients in the group treated with labetalol. The incidence of SGA in newborns exposed to both of these drugs prenatally was also compared. It was found that the percentage of newborns with SGA was 13% in the labetalol group and 12% in the nifedipine group. The researchers concluded that the use of labetalol and nifedipine is associated with a comparable risk of delivering an SGA newborn. Neither drug significantly increases or decreases the risk of SGA compared to the other, suggesting a similar safety profile in this regard [22].

Among centrally acting drugs, methyldopa is the most frequently used in the therapy of hypertension in pregnancy. It is a decarboxylase inhibitor, an enzyme involved in catecholamine metabolism. Furthermore, it also reduces renin secretion. At the same time, it does not affect placental circulation or fetal hemodynamics [2]. A study conducted in the Psychiatry Department of a tertiary referral hospital in Mumbai included 100 women presenting to a vaccination clinic at the 6th week postpartum. Patients taking psychotropic medications, those with a positive psychiatric history, or those suffering from somatic and psychiatric conditions were

excluded from the study. Based on the Edinburgh Postnatal Depression Scale (EPDS), the risk of postpartum depression in the study group was assessed. A maximum score of 19 points and a minimum score of 0 points were obtained, with a mean score of 6.43 ± 5.97. Thirty-nine percent of the women had an elevated score on the EPDS scale, and 28% were burdened with pregnancy-induced hypertension. The researchers found that 77.78% of patients treated with methyldopa developed postpartum depression. However, it should be kept in mind that the study described above has its limitations, such as a small sample size [23].

Management of Severe Hypertension in Pregnancy

According to ESC guidelines, if severe hypertension, symptoms of preeclampsia, or eclampsia are detected (even at lower BP values), the patient should be referred to a hospital to extend diagnostics and lower blood pressure under conditions that allow monitoring of the patient's and fetus's status, as this is a life-threatening emergency. Antihypertensive therapy should be conducted in such a way as to initially aim to lower MAP by less than 25%, and subsequently lower blood pressure values to <160/110 mmHg by a maximum of 10% per hour. After stabilizing BP values, maintenance treatment should be initiated to keep SBP within 110–140 mmHg and DBP within 80–85 mmHg over the next few days. To lower BP in a pregnant patient with severe hypertension, ESC experts recommend using intravenous dihydralazine, nitroglycerin, urapidil, and labetalol, or oral nifedipine. Particular caution when conducting antihypertensive therapy should be exercised in the presence of coexisting fetal growth restriction. It must be remembered that a sudden drop in blood pressure can cause a reduction in blood flow through maternal organs, as well as the placenta and uterus, which can result in intrauterine fetal demise [2,12]. Initiating an intravenous infusion of magnesium sulfate for eclampsia prophylaxis is indicated. An analysis of randomized trials involving pregnant women with severe preeclampsia showed that seizures occurred in 2.8% of women treated with antihypertensive drugs alone, and much less frequently, in 0.9% of patients treated with magnesium sulfate [24].

Summary

Hypertension in pregnant women is a common problem, estimated to affect approximately 7–10% of pregnancies. Notably, over the last two decades, the scale of this problem has increased by 25% [25]. Despite this, at the initial stage, an increase in BP may remain asymptomatic, which is why measuring blood pressure in pregnant women at every medical visit is so important. Ineffectively treated hypertension in pregnancy can lead to many maternal complications such as eclampsia, HELLP syndrome, disseminated intravascular coagulation syndrome, and kidney failure, as well as fetal complications, which include IUGR, placental abruption, intrauterine hypoxia, and prematurity. Therefore, awareness and education of the pregnant woman on this subject, alongside close cooperation between the patient and the doctor, are of paramount importance [2].

REFERENCES

1. World Health Organization. (2025, December 10). *Pre-eclampsia*. https://www.who.int/news-room/fact-sheets/detail/pre-eclampsia
2. Bręborowicz, G. (2021). *Położnictwo i ginekologia* (T. 1–2, wyd. 3). PZWL.
3. Dimitriadis, E., Rolnik, D. L., Zhou, W., Estrada-Gutierrez, G., Koga, K., Francisco, R. P. V., Whitehead, C., Hyett, J., da Silva Costa, F., Nicolaides, K., & Menkhorst, E. (2023). Pre-eclampsia. *Nature Reviews Disease Primers, 9*(1), Article 8. https://doi.org/10.1038/s41572-023-00417-6
4. Levine, R. J., Maynard, S. E., Qian, C., Lim, K., England, L. J., Yu, K. F., Schisterman, E. F., Thadhani, R., Sachs, B. P., Epstein, F. H., Sibai, B. M., Sukhatme, V. P., & Karumanchi, S. A. (2004). Circulating angiogenic factors and the risk of preeclampsia. *New England Journal of Medicine, 350*(7), 672–683. https://doi.org/10.1056/NEJMoa031884
5. Feng, H., Wang, L., Zhang, G., Zhang, Z., & Guo, W. (2020). Oxidative stress activated by Keap-1/Nrf2 signaling pathway in pathogenesis of preeclampsia. *International Journal of Clinical and Experimental Pathology, 13*(3), 382–392.
6. Tossetta, G., Fantone, S., Piani, F., Crescimanno, C., Ciavattini, A., Giannubilo, S. R., & Marzioni, D. (2023). Modulation of NRF2/KEAP1 signaling in preeclampsia. *Cells, 12*(11), Article 1545. https://doi.org/10.3390/cells12111545
7. Raguema, N., Moustadraf, S., & Bertagnolli, M. (2020). Immune and apoptosis mechanisms regulating placental development and vascularization in preeclampsia. *Frontiers in Physiology, 11*, Article 98. https://doi.org/10.3389/fphys.2020.00098

8. Raguema, N., Moustadraf, S., & Bertagnolli, M. (2020). Immune and apoptosis mechanisms regulating placental development and vascularization in preeclampsia. *Frontiers in Physiology, 11*, Article 98. https://doi.org/10.3389/fphys.2020.00098
9. Figueiredo, A. S., & Schumacher, A. (2016). The T helper type 17/regulatory T cell paradigm in pregnancy. *Immunology, 148*(1), 13–21. https://doi.org/10.1111/imm.12595
10. Kamrani, A., Alipourfard, I., Ahmadi-Khiavi, H., Yousefi, M., Rostamzadeh, D., Izadi, M., & Ahmadi, M. (2019). The role of epigenetic changes in preeclampsia. *BioFactors, 45*(5), 712–724. https://doi.org/10.1002/biof.1542
11. Hansen, A. T., Jensen, J. M. B., Hvas, A., & Christiansen, M. (2018). The genetic component of preeclampsia: A whole-exome sequencing study. *PLOS ONE, 13*(5), Article e0197217. https://doi.org/10.1371/journal.pone.0197217
12. casusBTL, G. (n.d.). *Wytyczne ESC 2024 dotyczące postępowania w podwyższonym ciśnieniu tętniczym i nadciśnieniu tętniczym*. Polskie Towarzystwo Kardiologiczne. https://ptkardio.pl/wytyczne/1-wytyczne_esc_2024_dotyczace_postepowania_w_podwyzszonym_cisnieniu_tetniczym_i_nadcisnieniu_tetniczym
13. Prejbisz, A., Dobrowolski, P., Kosiński, P., Bomba-Opoń, D., Adamczak, M., Bekiesińska-Figatowska, M., Kądziela, J., Konopka, A., Kostka-Jeziorny, K., Kurnatowska, I., Leszczyńska-Gorzelak, B., Litwin, M., Olszanecka, A., Orczykowski, M., Poniedziałek-Czajkowska, E., Sobieszczańska-Małek, M., Stolarz-Skrzypek, K., Szczepaniak-Chicheł, L., Szyndler, A., . . . Januszewicz, A. (2019). Management of hypertension in pregnancy—Prevention, diagnosis, treatment and long-term prognosis: A position statement of the Polish Society of Hypertension, Polish Cardiac Society and Polish Society of Gynaecologists and Obstetricians. *Arterial Hypertension, 23*(3), 117–182. https://doi.org/10.5603/AH.a2019.0011
14. Steegers, E. A., von Dadelszen, P., Duvekot, J. J., & Pijnenborg, R. (2010). Pre-eclampsia. *The Lancet, 376*(9741), 631–644. https://doi.org/10.1016/S0140-6736(10)60279-6
15. Young, O. M., Twedt, R., & Catov, J. M. (2016). Pre-pregnancy maternal obesity and the risk of preterm preeclampsia in the American primigravida. *Obesity, 24*(6), 1226–1229. https://doi.org/10.1002/oby.21412
16. Brown, M. A., Magee, L. A., Kenny, L. C., Karumanchi, S. A., McCarthy, F. P., Saito, S., Hall, D. R., Warren, C. E., Adoyi, G., & Ishaku, S. (2018). The hypertensive disorders of pregnancy: ISSHP classification, diagnosis, and management recommendations for international practice. *Pregnancy Hypertension, 13*, 291–310. https://doi.org/10.1016/j.preghy.2018.05.004
17. Tan, M. Y., Syngelaki, A., Poon, L. C., Rolnik, D. L., O'Gorman, N., Delgado, J. L., Akolekar, R., Konstantinidou, L., Tsavdaridou, M., Galeva, S., Ajdacka, U., Molina, F. S., Persico, N., Jani, J. C., Plasencia, W., Greco, E., Papaioannou, G., Wright, A., Wright, D., & Nicolaides, K. H. (2018). Screening for pre-eclampsia by maternal factors and biomarkers at 11–13 weeks' gestation. *Ultrasound in Obstetrics & Gynecology, 52*(2), 186–195. https://doi.org/10.1002/uog.19112
18. Poon, L. C., & Nicolaides, K. H. (2014). Early prediction of preeclampsia. *Obstetrics and Gynecology International, 2014*, Article 297397. https://doi.org/10.1155/2014/297397
19. Gallo, D. M., Wright, D., Casanova, C., Campanero, M., & Nicolaides, K. H. (2016). Competing risks model in screening for preeclampsia by maternal factors and biomarkers at 19–24 weeks' gestation. *American Journal of Obstetrics and Gynecology, 214*(5), 619.e1–619.e17. https://doi.org/10.1016/j.ajog.2015.11.016
20. Agrawal, S., Cerdeira, A. S., Redman, C., & Vatish, M. (2018). Meta-analysis and systematic review to assess the role of soluble FMS-like tyrosine kinase-1 and placenta growth factor ratio in prediction of preeclampsia. *Hypertension, 71*(2), 306–316. https://doi.org/10.1161/HYPERTENSIONAHA.117.10182
21. Ramlakhan, K. P., Roos-Hesselink, J. W., Basso, T., Greenslade, J., Flint, R. B., Krieger, E. V., Shotan, A., Budts, W., de Backer, J., Hall, R., Johnson, M. R., & Parsonage, W. A. (2024). Perinatal outcomes after in-utero exposure to beta-blockers in women with heart disease: Data from the ESC EORP Registry of Pregnancy and Cardiac Disease (ROPAC). *International Journal of Cardiology, 410*, Article 132234. https://doi.org/10.1016/j.ijcard.2024.132234
22. Leonard, S. A., Siadat, S., Huybrechts, K. F., Main, E. K., Hlatky, M. A., Hernández-Díaz, S., & Bateman, B. T. (2025). Comparative effectiveness and safety of labetalol versus nifedipine for treatment of chronic hypertension during pregnancy. *JACC: Advances, 4*(9), Article 102054. https://doi.org/10.1016/j.jacadv.2025.102054
23. Nayak, A. S., & Nachane, H. B. (2018). Risk analysis of suicidal ideations and postpartum depression with antenatal alpha methyldopa use. *Asian Journal of Psychiatry, 38*, 42–44. https://doi.org/10.1016/j.ajp.2018.10.024
24. Witlin, A. (1998). Magnesium sulfate therapy in preeclampsia and eclampsia. *Obstetrics & Gynecology, 92*(5), 883–889. https://doi.org/10.1016/S0029-7844(98)00277-4
25. Leszczyńska-Gorzelak, B., & Mierzyński, R. (2026, May 25). *Przewlekłe nadciśnienie tętnicze a ciąża*. Podyplomie. https://podyplomie.pl/ginekologia/31231,przewlekle-nadcisnienie-tetnicze-a-ciaza