

Current Understanding on Hypertensive Disorders in Pregnancy

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Abstract: Hypertensive disorders of pregnancy (HDP) are a complex spectrum of conditions that develop during gestation, posing significant risk to maternal and fetal health. This disorder encompasses gestational hypertension, preeclampsia, eclampsia, and chronic hypertension during pregnancy, with notably higher prevalence in low- and middle-income countries. By systematically reviewing latest clinical and experimental evidence concerning about HDP, this review aimed to provide a new understanding on risk assessment and clinical prevention strategies for HDP. In addition, there is still controversy over the antihypertensive treatment for pregnant women with mild to moderate hypertension during pregnancy or hypertension without terminal organ damage. It did not still confirm when to start antihypertensive treatment, the better antihypertensive target and recommended drugs. Therefore, this review also elaborated on used antihypertensive drugs and their effects on mother and fetus in recent years. Summarily, our conclusion suggested preventing HDP still requires a multi-faceted approach including diet, exercise and lifestyle habits. More prospective data were necessary to scientifically clarify preventive and therapeutic measures.

Keywords: hypertensive disorders of pregnancy, risk assessment, prevention strategies, pregnant woman, antihypertensive therapy

Introduction

Pregnancy induced hypertension (HDP) is a global health issue that affects 5% to 10% of pregnancies and leads to 700,000 maternal and 500,000 fetal deaths annually.¹ Although the global HDP mortality rate dropped from 39.8/100,000 in 1990 to 27.8/100,000 in 2019, its incidence rate is still rising, especially in countries with low social population index (SDI).^{1,2} HDP is defined as the onset of sustained hypertension after 20-week gestation, which is characterized by systolic blood pressure (BP) ≥ 140 mmHg and/or diastolic BP ≥ 90 mmHg, with normalization occurring within 12 weeks after postpartum.² This disease spectrum mainly includes gestational hypertension (GH), pre-eclampsia (PE), eclampsia, hypertension in pregnancy and hypertension with superimposed preeclampsia.^{2,3} HDP can be manifested with diverse maternal symptoms including edema, proteinuria, headache, visual disturbances, epigastric discomfort and chest tightness.³ Without timely intervention, it can potentially develop to severe complications such as heart failure, renal impairment and intracranial hemorrhage, posing life-threatening risk to both mother and fetus.⁴ In fact, recent evidence have witnessed growing emphasis on risk prediction for HDP.⁵ Large-scale clinical studies have identified some risk factors, including maternal age, pre-pregnancy body weight, family history and others.⁵⁻⁸ However, considerable controversy persists regarding the management of mild-to-moderate HDP with or without end-organ damage, particularly concerning the optimal timing for initiating antihypertensive therapy, thresholds of target BP and preferred pharmacologic agents.³ For severe hypertension in pregnancy, current guidelines recommend rapid BP reduction to $<160/105$ mmHg within minutes to hours, followed by gradual stabilization at $140-150/90-100$ mmHg.⁹ First-line antihypertensive agents typically include labetalol, methyldopa and nifedipine, though their safety profile during pregnancy remain debated.¹⁰ This review synthesized current evidence on risk factors, pathophysiological mechanisms and

therapeutic approaches for HDP patients, aiming to provide clinical guidance for early prevention and effective BP management among these patients.

Risk Factors for HDP

Common Risk Factors

Age, weight, chronic diseases, pre-pregnancy hypertension, metabolic abnormalities, multiple pregnancies, family history and other factors have been confirmed to be common risk factors for HDP.¹¹ For example, pregnant women over 40 years old or under 20 years old are more likely to develop HDP, and the age of the first pregnancy being under 20 years old or over 35 years old is also one of the high-risk factors.¹² A previous study found that gestational anemia, a history of growth hormone use and spontaneous abortion were the important factors on predicting the progression of GH to PE.¹³ Overweight or obesity with a body mass index (BMI) exceeding the normal range is an independent risk factor for HDP.¹⁴ Chen et al found that patients with chronic diseases such as diabetes, antiphospholipid syndrome and chronic nephritis were more likely to suffer from HDP.¹⁵ As for metabolic factors, fasting blood glucose (FPG), triglycerides (TG), familial hypercholesterolemia (FH), lactate dehydrogenase (LDH), mean platelet volume (MPV)¹⁶ and blood urea nitrogen (BUN) in the circulating blood were all associated with the occurrence of HDP.¹⁷ Therefore, focusing on evaluating the above risk factors might help to predict the risk of developing HDP.

Emerging Risk Factors

Recent evidence have shown that lifestyle plays an important role in the development of HDP.^{18–22} Poor lifestyle conditions such as a high-salt diet and physical inactivity were associated with the occurrence of HDP.¹⁸ HDP incidence were significantly higher than that in low altitude, suggesting some environmental factors might have an influence on the HDP risk.¹⁹ In recent years, researchers have also made some new attempts, for example, Li et al performed a prospective evaluation on the association between vaginal microorganisms and HDP by vaginal swab sequencing to identify bacteria.²⁰ They found that *L. crispatus* dominant vaginal community was related to with a reduced HDP risk, supporting that vaginal microbiome characteristics might be helpful to identify individuals at high risk of HDP and might provide a potential target for the development of new prepregnancy intervention methods.²¹ Ohseto et al explored the relationship between dietary electrolyte intake and the prevalence of HDP subtype, by estimating the dietary intake of calcium, potassium, sodium and magnesium with the food frequency questionnaire.²² They came to a conclusion that the intake of calcium and potassium might play an important role in the pathogenesis of PE.²² Briefly, the risk factors of HDP are involved in multiple aspects, and further mechanism exploration for these risk factors can be more accurately and quickly predict the occurrence of HDP.^{1–3}

Risk Models for HDP

Clinical indicators have been found to be one of the commonly used methods to predict HDP.^{1–4} Recently, researchers have explored more valuable clinical indicators, combined with statistical methods to build predictive models for the risk assessment.²³ For instance, Gunderson et al proposed a novel risk assessment model by combining traditional factors with BP patterns within 20 weeks of gestation, to more accurately distinguish the low-risk to intermediate-risk HDP.²⁴ Nurkkala et al also demonstrated the importance of BP for HDP prediction, reporting that the universal BP polygenic risk score (BPPRS) added to the model including clinical risk factors and preeclampsia-specific polygenic risk score (PEPRS) can significantly improve the predictive efficiency of HDP.²⁵ In addition, one study used multi-population meta-analysis to identify 12 independent sites associated with predicted PE and HDP, deriving a genome-wide polygenic risk score.⁵

Biomarker model usually refers to the prediction method by detecting certain biomolecules or metabolites in pregnant women.^{5–8} A previous study by combining the soluble fms-like tyrosine kinase 1/placental growth factor (PIGF) ratio with uterine artery Doppler ultrasound parameters (pulse index and resistivity index) created an effective mid-term pregnancy prediction method for the occurrence of early-onset HDP.^{26,27} Their results showed that this algorithm can improve the performance on predicting severe PE and complications such as prenatal fetal death or acute fetal distress, compared to biochemical markers or uterine Doppler parameters used alone.²⁷ Tejaswi et al used the enzyme colorimetric

method to determine the urinary calcium-creatinine ratio (UCCR) to predict PE, showing that the UCCR indicator was a good predictor of PE in primipara, and can be considered as a routine screening test at 20 to 28 weeks of pregnancy during routine prenatal examination.²⁸

Imaging examination is a non-invasive detection method to predict HDP risk by the observation and analysis of the imaging uterus, fetus and blood flow of pregnant women.²⁹ Common imaging methods include ultrasound examination, magnetic resonance imaging and others.^{30,31} However, these examinations require operation and interpretation by professional technicians, and rely heavily on medical resources. Liu et al extracted radiomics features from diffusion weighted imaging (DWI) and apparent diffusion coefficient (ADC) images, developing a radiomics nomogram by combining it with maternal age and BMI, which showed a good performance in the training and test groups.³² Papastefanou et al showed that placental volume from imaging examination was an independent predictor of pregnancy complications associated with placental insufficiency at 11 to 14 weeks.³³ In addition, it was found that ocular artery Doppler can improve the prediction performance of PE, especially for the PE prediction within 3 weeks after pregnancy.³⁴

Making a simple summary, the prediction model for HDP is mainly based on the common risk factors such as maternal age, family history, body weight, chronic diseases, prepregnancy hypertension and metabolic abnormalities.¹⁻⁸ These factors are easily accessible and have been widely used in clinical practice, providing doctors with a preliminary basis for risk assessment. However, due to the large individual differences between various pregnant women, the prediction effect of the same risk factor is discrepant in different pregnant women.³⁵ These clinical prediction models usually can only provide a relatively rough risk assessment, and it is difficult to accurately predict the risk of individual pregnant women.⁸ Biomarker prediction models are usually able to provide more objective and accurate predictive information, which might help doctors to more accurately assess the risk of pregnant women, but the detection of some biomarkers is more expensive and may be difficult to popularize in some regions or medical facilities.³⁶ In addition, the predictive effect of some biomarkers needs to be further validated and/or improved to ensure their accuracy and reliability.³⁷ The predictive model of imaging examination is non-invasive, excellent and intuitive, and can provide rich diagnostic information for doctors through ultrasound and magnetic resonance imaging. However, it usually requires professional technical personnel to operate and interpret, which has the disadvantage of great subjectivity.³⁸ More and more new biomarkers or imaging methods are needed to further address the limitations of current risk predictions for HDP in the future.

Pathological Mechanism of HDP

The pathogenesis of HDP is quite complex that can be involved in multiple factors working together, including genetic factors, hormone changes, immune regulation, and others.³⁹

Genetic Factors

Genetics plays a certain role in the onset of pregnancy induced hypertension. Research evidence has already shown that the risk of HDP might be relatively higher if her mother or sister has a history of pregnancy induced hypertension during period of gestation.^{40,41} Polymorphisms in certain genes are associated with susceptibility to gestational hypertension, which might affect vascular function, hormone metabolism and immune system regulation, thereby increasing the HDP risk.⁴²⁻⁴⁴

Hormonal Changes

During pregnancy, the hormone levels in a woman's body tend to undergo significant changes.⁴⁵ The placenta can secrete a large amount of hormones such as placental lactogen, estrogen and progesterone, which not only maintain pregnancy, but also have an impact on the physiological function of the mother body.^{46,47} For example, estrogen and progesterone have a significant relaxing effect on smooth muscle in blood vessels.⁴⁶⁻⁴⁸ In order to ensure normal blood circulation, the mother body can regulate BP through a series of mechanisms about pregnancy-related hormones, potentially leading to a significant increase in BP.⁴⁹ Placental prolactin can also affect insulin sensitivity, leading to abnormal glucose metabolism, indirectly causing abnormal increase in BP.⁵⁰

Immune System

The immune system of women body often undergoes adaptive change to avoid maternal rejection of the fetus during pregnancy.⁵¹ But this adjustment, sometimes, results in excessive activation of the immune system.⁵² The excessive inflammatory factors produced by immune cells can damage endothelial cells and lead to vasoconstriction, thereby causing an abnormal increase in BP.⁵³

Hemodynamic Changes

As the fetus grows, the blood volume of pregnant women gradually increases to meet the demands of the fetus and themselves.⁵⁴ However, some pregnant women's cardiovascular system cannot adapt well to this change. Their heart needs to work harder to pump blood for oxygen demand of whole body tissues, contributing to an increase BP.⁵⁵ At the same time, an enlarged uterus in the abdomen may compress the inferior vena cava during pregnancy, reducing the amount of blood returning to the heart.⁵⁶ In order to maintain sufficient cardiac output, heart might increase pumping pressure, further leading to an increase in BP.⁵⁷

Others

Additionally, the underlying diseases of pregnant women such as chronic hypertension, diabetes, and kidney disease are more likely to increase the risk of HDP.^{1–8} Except that advanced pregnancy (age ≥ 35 years), multiple pregnancies, obesity, and other confirmed factors, there are evidence showing that long-term mental stress and anxiety in pregnant women may also affect the neuroendocrine system, leading to elevated BP.^{58–60} Further understanding these causes can help expectant mothers take preventive measures during pregnancy, ensuring the health of both mother and baby.

Treatment for HDP

Antihypertensive drugs for HDP patients should not only consider the treatment effect and cardiovascular prognosis of pregnant woman, but also pay more attention to fetal safety.⁶¹ Some antihypertensive drugs are considered safe and widely used in obstetrics, while others are contraindicated because of teratogenic or toxic effects on the fetus.^{62,63} Therefore, the safety of antihypertensive drugs is the primary issue in the treatment of HDP. We tried to introduce common antihypertensive drugs, precautions for drug treatment and their effects on mother and fetus.

General Principles of Antihypertensive Therapy

The antihypertensive therapy of HDP was based on the BP level, gestational age, and other risk factors related to the safety of maternal and fetal.^{64,65} The main purpose is to reduce the adverse incidence and mortality associated with hypertension in both the mother and fetus and extend gestational duration as much as possible to allow the fetus had more time to develop and mature before delivery.^{64,66,67} However, limited scale of randomized controlled trials, evidence derived from meta-analyses and clinical databases have resulted in controversy regarding the pharmacological treatment of HDP.⁶⁶ Therefore, the safety and efficacy data for evaluating antihypertensive drugs are very limited. Currently, the selection of antihypertensive drugs during pregnancy is mainly based on the patient's race, existing treatments, drug efficacy and safety, clinical experience and familiarity with the drugs.⁶⁸ Moreover, the process of antihypertensive treatment should strive for stability, and maintain a relatively stable value for BP.⁶⁹ When severe hypertension with organ damage such as acute left ventricular failure occurs, it is necessary to urgently lower the BP to the target range, and reach stability within 24–48 hours.⁶⁹

Drug Therapy for Mild to Moderate HDP

At present, the treatment for mild to moderate HDP (140–159 and/or 90–109 mmHg) and hypertension without target-organ damage were still controversial.^{65–67} Existing clinical guidelines have not reached agreement on when to start antihypertensive treatment, optimal antihypertensive target and recommended therapeutic drugs.^{68,69} The 2018 European Society of Cardiology guidelines recommend initiating antihypertensive therapy for all HDP women (with or without proteinuria) with sustained elevation more than BP 140/90 mmHg.⁶⁸ The 2020 American College of Obstetricians and

Gynecologists guidelines recommend that antihypertensive medication should not be required for preeclampsia patients with mild gestational hypertension and/or no end-organ damage.⁷⁰ The 2020 guidelines of the Society of Obstetrics and Gynecology of Chinese Medical Association recommend that antihypertensive treatment should be used for hypertensive pregnant women with systolic BP ≥ 140 mmHg and (or) diastolic BP ≥ 90 mmHg.⁷¹ Methyldopa, labetalol and nifedipine are the preferred drugs recommended.⁷²

The reasons for previous guidelines that antihypertensive treatment was not recommended for mild and moderate hypertension during pregnancy are as follows: (1) Due to the physiological decline in BP in the first trimester, the risk of cardiovascular complications in pregnant women is relatively low, especially in patients without a history of severe hypertension and/or normal renal function;⁶⁸ (2) Fetal perfusion is depends on appropriate maternal BP, and the lowering BP might cause damage to uterine placental perfusion, contributing to an elevated risk of developing preeclampsia and low birth weight;⁷³ (3) Although treatment of mild to moderate HDP can reduce the progression to severe hypertension, it dose not really improve adverse maternal and perinatal outcomes.⁷⁴ Therefore, the current evidence is not clear whether mild or moderate hypertension during pregnancy should be actively intervened by antihypertensive drugs. Closely monitoring the clinical manifestations of pregnant women and fetus and the experience of clinicians might be important to determine whether antihypertensive treatment should be considered individually.

Drug Therapy for Severe HDP

Severe hypertension during pregnancy is defined as systolic BP/diastolic BP $\geq 160/110$ mmHg in previous most guidelines.^{65–69} The goal for lowering BP in severe HDP is to reduce BP under 160/105 mmHg within minutes to hours, and then steadily maintain BP at 140–150/90–100 mmHg to prevent the loss of cerebral vascular autoregulation, thus avoiding stroke and other organ damage.^{75,76} However, there is currently no evidence showing that reducing BP to the “normal” range ($<140/90$ mmHg) can provide additional benefits. Rapidly lowering BP by more than 25% above mean arterial pressure may result in inadequate perfusion, affecting placental perfusion and fetal growth.⁷⁷

Therefore, for the treatment of severe HDP, the principle of lowering BP too quickly should be not followed. For patients with acute severe hypertension during pregnancy, magnesium sulfate should be administered promptly to reduce the risk of eclampsia and maternal morbidity and mortality, without adverse effects on the fetus.⁷⁸ However, magnesium sulfate should not be used as a BP-lowering drug for pregnant woman. Most guidelines still recommend using labetalol, methyldopa or nifedipine for antihypertensive treatment,^{65–69,75,76} because these drugs do not require cardiac monitoring and do not reduce uterine placental blood flow.⁷⁹

Selecting Antihypertensive Drugs for HDP

Calcium Channel Blockers

Existing evidence suggest that calcium channel blockers (CCBs) used during pregnancy are considered safe that their use in early pregnancy is not a major risk for teratogenesis.⁶² For instance, nifedipine has been considered as the most commonly used antihypertensive agent during pregnancy, and most guidelines recommend the short-acting nifedipine as the first-line treatment for hypertension emergency,^{65–69} especially when intravenous access is unavailable or not established. Additionally, sustained-release nifedipine has minimal side effects on the fetus and uteroplacental vessel, which is basically effective in controlling severe hypertensive emergencies when used intravenously with labetalol or hydralazine.⁸⁰ In animal models, diltiazem can cause bone abnormalities, and both verapamil and digoxin can cause fetal growth retardation, hypotension and bradycardia for pregnant woman. Thus, these two drugs are not recommended for the treatment of HDP.⁶⁸

Beta Blockers

Beta (β) receptor blockers have been extensively studied previously,⁸¹ Theoretically, the β blockers might reduce placental perfusion, leading to fetal growth restriction and low birth weight.⁸² However, most studies have ruled out these risks, and there is currently no clear evidence of teratogenicity during pregnancy, with the most extensive research focusing on labetalol. Labetalol is also one of the most widely used drugs in HDP treatment, acting as a non-selective β

receptor blocker.⁸³ Studies have confirmed that labetalol did not alter uteroplacental blood flow with a safe use during pregnancy.⁸⁴ Most guidelines predominantly recommend labetalol as a first-line drug for treating HDP.^{65–70} A previous meta-analysis also showed that β receptor blockers seem to be more effective than methyldopa in reducing the risk of severe hypertension, but there is no significant difference in the PE risk.⁷⁴ However, atenolol can cross the placenta and adversely affect uteroplacental hemodynamics, leading to low birth weight, preterm birth, neonatal hypoglycemia and bradycardia, so that it is not recommended for use during pregnancy.^{68,85}

Angiotensin Converting Enzyme Inhibitor/Angiotensin II Receptor Antagonist

Existing evidence have suggested that angiotensin-converting enzyme inhibitors (ACEIs) and angiotensin II receptor blockers (ARBs) have teratogenic effects.^{86,87} The use of ACEIs or ARBs during pregnancy is associated with fetal death, miscarriage, congenital cardiovascular diseases, central nervous system abnormalities, musculoskeletal malformations, gastrointestinal malformations and renal abnormalities.⁸⁸ Therefore, these drugs should be immediately discontinued after pregnancy, and women planning to conceive should switch to other appropriate antihypertensive medications.

α_2 Receptor Agonists

Phentolamine is a non-selective α_1 and α_2 adrenergic receptor blocker. Relevant guidelines from the Chinese Medical Association's Obstetrics and Gynecology Society mentioned that phentolamine can be administered intravenously for the treatment of HDP.⁷¹ Urapidil has a dual mechanism of action at both central and peripheral levels, and it can block post-synaptic α_1 receptors in the periphery and modulates the activity of the center in the brain by stimulating 5-HT1A receptors, preventing BP elevation.⁷² The ESC guidelines recommend its use for the treatment of severe hypertension and hypertensive crises during pregnancy.⁶⁸ Prazosin is a selective post-synaptic α_1 receptor blocker and is not recommended for the treatment of HDP, although some guidelines suggested it as a second-line treatment for mild to moderate hypertension during pregnancy.⁸⁹

α Receptor Agonists

Methyldopa and clonidine can stimulate the brainstem α_2 receptors to reduce central sympathetic nerve output.^{83,90} Methyldopa was first used in obstetrics as early as 1963, which is still widely used because of its safety and effectiveness now.⁸³ It can gradually control BP over 6 to 8 hours without affecting the hemodynamics of the placenta and fetus or causing teratogenic effect, making it widely used in the treating HDP.⁹⁰ However, compared to β receptor antagonists, methyldopa might significantly increase the risk of preeclampsia and preterm birth.^{90,91} Exposure to methyldopa or clonidine in mid-to-late pregnancy may increase the risk of small for gestational age but does not increase perinatal mortality.⁸³ In addition, methyldopa and clonidine can cause neonatal hypotension and maternal depression,⁶⁸ which should be avoided after postpartum.

Other Antihypertensive Drugs

Sodium nitroprusside is a potent vasodilator with very rapid and strong arterial and venous vasodilation.⁹² But it can produce cyanide and thiocyanate through biotransformation that might have toxic effect to the fetus, making it unsuitable for use during pregnancy.⁹² It is only considered in cases of severe preeclampsia or hypertensive emergencies with left ventricular failure and refractory pulmonary edema that cannot be controlled by other medications.^{69–74,93,94} Nitroglycerin acts on nitrous oxide synthase and can dilate both arteries and veins, alleviating the anterior and posterior load.⁹⁵ It is mainly used in the treatment of preeclampsia combined with pulmonary edema and/or coronary artery disease.^{65–69} Intravenous nitroglycerin is equally effective as oral nifedipine.^{95,96} Thiazides and loop diuretics can cross the placenta but do not cause fetal malformations.⁹⁷ However, these drugs can lead to adverse reactions in the pregnant woman, such as reduced blood volume, decreased amniotic fluid, reduced platelets, hypoglycemia and electrolyte disturbances.⁶⁸ In women with preeclampsia or during lactation, who already have a reduced blood volume, the use of diuretics can further lead to reduced amniotic fluid and decreased milk production, or even inhibit lactation.^{68,83,90–92} Therefore, the use of these drugs is not recommended during pregnancy.

Summary

Poor control of blood pressure during pregnancy can affect maternal and fetal health as well as pregnancy outcomes. Although multiple countries and academic guidelines have provided specific recommendations for antihypertensive treatment during pregnancy, there is currently a lack of high-quality data on the long-term prognosis of mother and fetus. Large randomized trial data are also limited and pharmacokinetic studies of antihypertensive drugs during pregnancy are scarce, and there is heterogeneity in pharmacokinetic descriptions between different studies. In order to achieve evidence-based and personalized use of antihypertensive drugs, further studies are needed on the relationship between pregnancy pathology, pregnancy pharmacokinetics and pharmacodynamics in the future, in order to prevent under-treatment and overtreatment and minimize adverse reaction. In clinical practice, higher quality evidence is necessary to provide a basis for refined management of gestational hypertension treatment and to safeguard maternal and infant health.

Disclosure

Jiahua Liao is the first author. There is no conflict of interest to be clarified for our work.

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