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Pharmacologic Management of Hypertensive Disorders of Pregnancy and Postpartum Hypertension: Current Evidence and Clinical Implications

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ABSTRACT

Hypertensive disorders of pregnancy remain a major cause of preventable maternal morbidity and mortality worldwide, encompassing a spectrum of conditions from chronic and gestational hypertension to severe hypertensive crises associated with preeclampsia and eclampsia. Severe elevations in blood pressure (systolic ≥160 mmHg and/or diastolic ≥110 mmHg) markedly increase the risk of stroke, cardiovascular complications, and adverse perinatal outcomes, underscoring the need for prompt and appropriate pharmacological intervention. This review synthesizes contemporary evidence regarding treatment thresholds, target blood pressure goals, and the comparative efficacy and safety of commonly used antihypertensive agents, including labetalol, nifedipine, hydralazine, and methyldopa, in both antepartum and postpartum settings. Emerging randomized trials and meta-analytic data supporting earlier intervention in non-severe chronic hypertension are discussed, along with clinical considerations unique to the postpartum period, during which blood pressure instability and stroke risk remain elevated. Therapeutic decision-making must balance maternal cardiovascular protection with fetal and neonatal safety, considering placental drug transfer and the relative scarcity of long-term outcome data. An individualized, evidence-based, and multidisciplinary approach throughout the antepartum and postpartum continuum is essential to prevent severe hypertension, minimize end-organ injury, and improve immediate and long-term maternal cardiovascular outcomes.

Keywords

Hypertensive disorders of pregnancy, preeclampsia, postpartum hypertension, antihypertensive therapy, maternal morbidity, and blood pressure control.

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INTRODUCTION

Hypertensive disorders of pregnancy (HDP) affect approximately 5–10% of pregnancies worldwide and remain a major cause of maternal and perinatal morbidity and mortality, particularly in low- and middle-income countries [1,2]. In pregnancy, hypertension is generally defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, confirmed on repeated measurements, whereas severe hypertension (≥160/110 mmHg) represents a medical emergency associated with markedly increased risks of intracerebral hemorrhage, heart failure, placental abruption, and maternal death [3,4]. The global burden of HDP has increased over recent decades, partly due to advancing maternal age, obesity, chronic hypertension, and metabolic diseases [5]. In addition to immediate obstetric risks, women with HDP also face an elevated long-term risk of cardiovascular disease, underscoring the importance of optimal blood pressure control across the peripartum continuum [6].

The management of hypertension in pregnant and postpartum patients presents unique challenges to healthcare professionals. Therapeutic decisions must balance maternal cardiovascular protection against potential fetal and neonatal risks, as most antihypertensive agents cross the placenta, and some are

contraindicated because of their teratogenic or fetotoxic effects [7]. Clinicians must determine appropriate diagnostic criteria, treatment thresholds, target blood pressure goals, and drug selection strategies tailored to the severity and etiology of hypertension, whether chronic, gestational, or related to preeclampsia. Furthermore, the postpartum period warrants particular attention, as blood pressure commonly peaks several days after delivery, and the risk of stroke is highest during this interval [8,9]. Therefore, an evidence-based and individualized pharmacologic approach is essential to reduce severe hypertension, prevent end-organ injury, and optimize maternal and neonatal outcomes [10].

Classification of Hypertensive Disorders in Pregnancy

Hypertensive disorders during pregnancy comprise a spectrum of conditions that vary in terms of the timing of onset, clinical manifestations, and associated maternal-fetal risks. These disorders are commonly categorized as chronic hypertension, gestational hypertension, preeclampsia (with or without severe features), eclampsia, and chronic hypertension with superimposed preeclampsia. Chronic hypertension refers to elevated blood pressure present before conception or diagnosed before 20 weeks of gestation, whereas gestational hypertension describes new-onset hypertension after 20 weeks' gestation without proteinuria or systemic features of end-organ involvement [11-14]. Preeclampsia is defined as hypertension accompanied by proteinuria and/or evidence of maternal organ dysfunction, including renal insufficiency, hepatic abnormalities, thrombocytopenia, neurological symptoms, and uteroplacental dysfunction. Eclampsia is characterized by new-onset generalized seizures in patients with preeclampsia and remains a major cause of severe maternal morbidity worldwide [1,2].

Table 1. Classification of Hypertensive Disorders in Pregnancy and Postpartum

Disorder	Timing	Diagnostic Criteria	Clinical Implications
Chronic hypertension	Before pregnancy or <20 weeks	BP ≥140/90 mmHg	Increased risk of superimposed preeclampsia and preterm birth
Gestational hypertension	≥20 weeks	New-onset BP ≥140/90 mmHg without proteinuria or organ dysfunction	May progress to preeclampsia
Preeclampsia	≥20 weeks	Hypertension with proteinuria and/or maternal organ dysfunction	Multisystem disease; risk of severe maternal and perinatal complications
Severe preeclampsia	≥20 weeks	BP ≥160/110 mmHg and/or severe organ involvement	High risk of stroke, HELLP syndrome, and adverse perinatal outcomes
Eclampsia	Pregnancy or postpartum	Seizures in a patient with preeclampsia	Life-threatening obstetric emergency
Chronic hypertension with superimposed preeclampsia	After 20 weeks	Chronic hypertension with new proteinuria or organ dysfunction	Increased maternal–fetal morbidity

Evolving diagnostic frameworks reflect an improved understanding of placental and endothelial pathophysiology. While earlier definitions required proteinuria for diagnosis, contemporary guidelines recognize that significant maternal organ dysfunction may occur in the absence of proteinuria [1-3]. Accurate classification is clinically essential because it determines surveillance strategies, pharmacological treatment thresholds, delivery timing, and postpartum follow-up [15]. Moreover, women with a history of HDP are at an increased long-term cardiovascular risk, emphasizing the importance of precise phenotyping and risk stratification beyond the peripartum period [16].

Technique for Accurate Blood Pressure Measurement

An accurate blood pressure measurement is critical for the diagnosis and management of hypertensive disorders during pregnancy. Even small technical errors may result in misclassification of disease severity and inappropriate pharmacological interventions [3]. Patients should be advised to avoid caffeine and nicotine

intake for at least 30–60 min before measurement. Measurements should be obtained after at least 5 min of quiet rest in a stable and supported position. When seated, the patient's back should be supported, feet placed flat on the floor without leg crossing, and arms supported at heart level to minimize hydrostatic variation.

Appropriate cuff selection is essential, particularly in obese patients. An undersized cuff may substantially overestimate blood pressure, whereas an oversized cuff may underestimate it. The inflatable bladder should ideally encircle approximately 80% of the upper arm circumference and cover approximately 40% of the width. Devices should be calibrated; when using the auscultatory method, the first Korotkoff sound corresponds to the systolic pressure, and the disappearance of the sound corresponds to the diastolic pressure. Validated automated devices may also be used, provided they have been evaluated in pregnant women [4]. Elevated readings should be confirmed by repeated measurements or serial assessments. Home blood pressure monitoring is useful for detecting white coat or masked hypertension and for the early recognition of worsening blood pressure between visits, particularly postpartum [9].

Preconception Management of Chronic Hypertension

Optimal control of chronic hypertension before conception is a critical component of risk reduction in women planning to conceive. Patients with established hypertension should ideally achieve blood pressure targets that are consistent with contemporary guidelines for non-pregnant adults prior to conception, as elevated preconception blood pressure is associated with adverse pregnancy outcomes [11]. Observational data have shown that pre-pregnancy blood pressure levels are associated with pregnancy outcomes, supporting preconception optimization and risk stratification. Women with chronic hypertension also face an increased risk of developing hypertensive complications during pregnancy and postpartum hypertension, reinforcing the importance of preconception counseling and follow-up planning [14].

Findings from the Chronic Hypertension and Pregnancy (CHAP) trial demonstrated that active treatment of mild chronic hypertension during pregnancy, targeting blood pressure <140/90 mmHg, reduced the incidence of severe hypertension and adverse obstetric outcomes without increasing the risk of small-for-gestational-age infants [10]. These results underscore the importance of blood pressure optimization across the reproductive continuum and support early and sustained antihypertensive management in women with chronic hypertension. When pregnancy is anticipated, clinicians must carefully evaluate the safety of current antihypertensive medications. Some agents commonly used in non-pregnant adults are contraindicated during pregnancy because of established fetotoxic effects; therefore, transitioning to medications with reassuring pregnancy safety data (e.g., labetalol, extended-release nifedipine, or methyldopa) may be appropriate prior to conception [7].

Selection of Antihypertensive Therapy in Women Planning Pregnancy

In women with chronic hypertension who are planning to become pregnant, antihypertensive therapy should prioritize agents with established safety data to minimize teratogenic or fetotoxic risks. Commonly used options include labetalol, extended-release nifedipine, hydralazine, and methyldopa. In practice, extended-release nifedipine or labetalol is often selected as first-line therapy, with the choice individualized according to patient characteristics and comorbidities. When monotherapy is insufficient, combination therapy (commonly labetalol plus extended-release nifedipine) may be required, with close monitoring and shared decision-making.

Antihypertensive Agents to Avoid in Women Planning Pregnancy

Agents that interfere with the renin–angiotensin–aldosterone system (RAAS), including ACE inhibitors and ARBs, should be avoided in women planning pregnancy or at risk of unplanned pregnancy because of fetotoxicity, particularly later in gestation. Early recognition of pregnancy is essential in women receiving these medications so that therapy can be discontinued promptly to minimize embryonic exposure [7]. Mineralocorticoid receptor antagonists are generally avoided owing to limited safety data.

Preferred Calcium Channel Blockers in Women Planning Pregnancy

Among calcium channel blockers, nifedipine (extended-release or intermediate-acting formulations) has robust pregnancy safety data and is generally preferred for use during pregnancy. Comparative analyses, including network meta-analysis evidence, support nifedipine as an effective option for reducing the progression to severe hypertension, and it is commonly used in the management of pregnancy-related hypertension [15]. Decisions should remain individualized, balancing the evidence base with patient tolerance.

Preferred Beta-Blockers in Women Planning Pregnancy

Labetalol is generally favored because of extensive pregnancy safety data and broad guideline support [14]. Atenolol is typically avoided because of its association with fetal growth restriction in observational studies, whereas metoprolol may be considered when a selective beta-blocker is clinically indicated.

Use of Thiazide and Thiazide-Like Diuretics

The roles of thiazide and thiazide-like diuretics should be individualized. Concerns regarding interference with physiological plasma volume expansion are largely theoretical, because chronic users often reach a steady state. In selected patients requiring multidrug regimens, continuation of low-dose thiazides may be considered with close monitoring [16,17]. If withdrawal leads to inadequate control, a pregnancy-compatible agent can be added or substituted [14].

When to Initiate Antihypertensive Therapy in Pregnancy

Severe hypertension is an obstetric emergency. Pregnant or postpartum patients with confirmed systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥110 mmHg require urgent pharmacological intervention. The American College of Obstetricians and Gynecologists recommends treatment as soon as feasible, ideally within 30–60 min after confirmation [17]. Evidence underscores the critical role of systolic blood pressure in the prediction of cerebrovascular complications. In a case series of maternal stroke associated with preeclampsia/eclampsia, severe systolic hypertension was common before the event. These findings reinforce the fact that rapid treatment of severe hypertension is essential to prevent avoidable maternal morbidity and mortality [18].

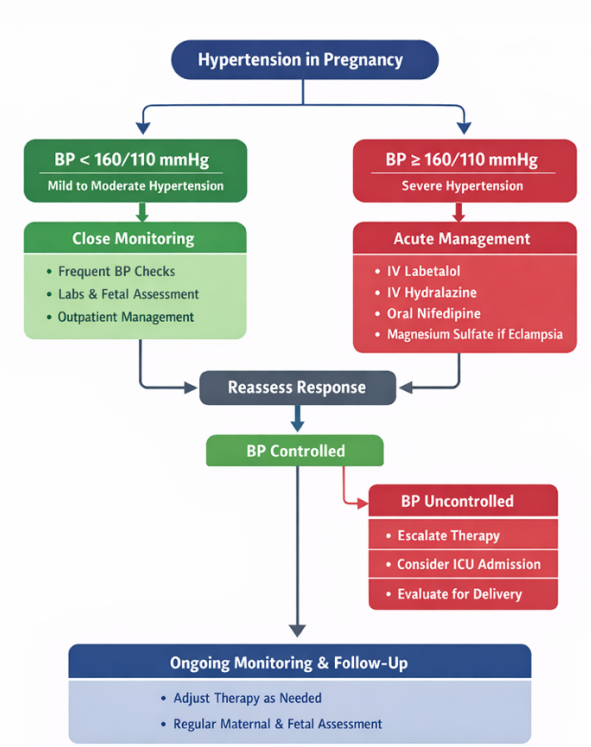

Figure 1. Clinical Algorithm for Management of Hypertension in Pregnancy

Management of Non-severe Hypertension During Pregnancy

Non-severe hypertension (SBP, 140–159 mmHg and/or DBP, 90–109 mmHg) requires individualized risk-based management. Women with chronic hypertension, a prior history of preeclampsia, renal disease, diabetes, obesity, or advanced maternal age may have a higher likelihood of disease progression and may benefit from earlier intervention [14].

Pharmacologic Treatment of Non-severe Hypertension in Pregnancy

Chronic non-severe Hypertension

In pregnant patients with chronic hypertension, current evidence supports active treatment, even in the absence of severe blood pressure elevation. The Clinical Trials of Hypertension Pregnancy (CHAP) trial demonstrated that targeting <140/90 mmHg reduced severe hypertension and adverse outcomes without increasing the risk of small-for-gestational-age [10].

Pregnancy-related non-severe Hypertension

For gestational hypertension and preeclampsia without severe features, escalation of therapy may be reasonable when SBP persistently approaches 150–159 mmHg and/or DBP 100–109 mmHg and delivery is not imminent. Observational studies have linked higher blood pressure to an increased risk of adverse maternal and perinatal outcomes [19,20]. Neurological complications may resemble posterior reversible encephalopathy syndrome (PRES), which can occur at variable blood pressure thresholds in susceptible individuals [21,22]. Guideline-based approaches increasingly emphasize the proactive prevention of progression to severe hypertension through timely therapy and monitoring of appropriate patients [3].

Review of Evidence: Treatment of Non-severe Chronic Hypertension

Treatment thresholds for mild chronic hypertension have historically varied; however, the Change in A-haemoglobin level (CHAP) study shifted practice by showing improved maternal and obstetric outcomes when treatment was initiated and titrated to <140/90 mmHg rather than withheld until severe hypertension developed [10]. In the CHAP study, the primary composite outcome occurred in 30.2% of treated patients versus 37.0% in controls (RR 0.82; 95% CI 0.73–0.92) [10]. Secondary analyses further suggested that participants achieving a mean clinic blood pressure <130/80 mmHg experienced lower composite adverse outcomes than those maintaining 130–139/80–89 mmHg (adjusted RR 0.45; 95% CI 0.38–0.54) [21].

Evidence in Mixed Populations with Chronic and Pregnancy-Related Hypertension

Evidence syntheses and contemporary reviews consistently show that pharmacologic therapy reduces the progression to severe hypertension, an outcome recognized as clinically meaningful by major professional societies [23]. The CHIPS trial demonstrated that tighter control reduced severe hypertension compared with less-tight control [24-30]. and post-hoc analyses supported an association between severe hypertension and adverse maternal outcomes [31]. Although reductions in broader obstetric outcomes have been less consistent across heterogeneous studies, available evidence supports the prevention of severe hypertension as a key strategy for reducing maternal complications while maintaining fetal safety.

Acute Therapy of Severe Hypertension

Choice of First-Line Agents

Acute severe hypertension during pregnancy or the postpartum period requires immediate pharmacological intervention. Intravenous labetalol and hydralazine are accepted first-line agents, and immediate-release oral nifedipine is an effective alternative when intravenous access is delayed or unavailable [25]. Selection should consider the clinician’s experience, side effects, comorbidities, and drug availability. Continuous fetal monitoring is recommended when the fetus is viable [17].

Table 2. Acute Pharmacologic Management of Severe Hypertension in Pregnancy and Postpartum

Drug	Initial Dose	Repeat / Titration	Key Clinical Notes
Labetalol (IV)	20 mg IV	20–80 mg every 10 min (max 300 mg) or infusion 1–2 mg/min	First-line agent; avoid in asthma or significant bradycardia
Hydralazine (IV)	5 mg IV	5–10 mg every 20 min (max 20–30 mg)	Alternative first-line; may cause reflex tachycardia
Nifedipine (Immediate-release oral)	10 mg	10–20 mg every 20 min if needed	Useful when IV access unavailable
Nicardipine (IV infusion)	5 mg/h	Increase to max 15 mg/h	Preferred in refractory hypertension; requires infusion pump
Nitroglycerin (IV infusion)	5 mcg/min	Titrate up to 100 mcg/min	Preferred in severe hypertension with pulmonary edema
Nitroprusside (IV infusion)	0.25 mcg/kg/min	Max 2 mcg/kg/min (short duration only)	Last-resort therapy; risk of fetal cyanide/thiocyanate toxicity

Postpartum Considerations

Blood pressure often peaks between the postpartum days 3 and 6 due to the mobilization of extracellular fluid and hemodynamic shifts. This rise may occur even in women who were initially normotensive after delivery and is more pronounced in those with HDP [8]. Given that many events occur after discharge, structured postpartum monitoring is essential. The American College of Obstetricians and Gynecologists recommends blood pressure assessment within 7–10 days postpartum (earlier for severe disease) and emphasizes patient education regarding warning symptoms [9]. Long-term cardiovascular risk reduction requires coordinated follow-up and transition to primary care/cardiovascular prevention pathways when indicated [32,33].

CONCLUSION

Hypertensive disorders of pregnancy remain a major cause of preventable maternal and perinatal morbidity and mortality. Early treatment and prompt control of chronic non-severe hypertension are essential to reduce the risk of stroke and other life-threatening complications. Antihypertensive therapy should balance maternal cardiovascular protection with fetal and neonatal safety through individualized treatment strategies and close monitoring. A multidisciplinary approach across the antepartum and postpartum periods is crucial for optimizing maternal outcomes and mitigating long-term cardiovascular risks.

DECLARATIONS

The authors declare no conflicts of interest related to this review.

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AUTHORS' CONTRIBUTIONS

AIT conceptualized the review, conducted the literature search, synthesized the evidence, and drafted the manuscript. The author critically revised the manuscript, approved the final version for publication, and accepts full responsibility for the integrity and accuracy of the content.

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