Assessment and management of hypertensive disorders in pregnancy

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Hypertensive disorders in pregnancy are among the most common complications with varied presentations at different stages of pregnancy. Women’s healthcare providers must be vigilant to conduct a thorough assessment and provide evidence-based management to provide exceptional care to women, particularly to prevent potentially life-threatening conditions including severe preeclampsia and HELLP syndrome as well as sequelae. Women’s healthcare providers must understand the diagnostic criteria and treatment options available to appropriately counsel patients through hypertensive disorders in pregnancy and the immediate postpartum period.

Key words: hypertensive disorders in pregnancy, gestational hypertension, preeclampsia, HELLP syndrome

Hypertensive disorders in pregnancy (HDPs) are among the most common pregnancy complications and leading causes of pregnancy-related death in the United States. Among maternal deaths in the US occurring during the same hospitalization as delivery, upward of 32% had a documented HDP. Chronic hypertension was noted in approximately 8% of deaths.1 Although HDPs typically manifest during pregnancy, it should not be overlooked that development may occur during the postpartum period up to 6 weeks following delivery. Women with a history of any HDP continue to have an elevated risk of cardiovascular disease for the rest of their lives.2
This article reviews the definitions and diagnostic criteria for HDPs. Up-to-date recommendations for prevention and management of HDPs are described. Nurse practitioners who provide prepregnancy, interpregnancy, antepartum, and postpartum care can improve maternal health and pregnancy outcomes through preventive measures, early diagnosis, and evidence-based management of HDP.

**Definitions and diagnostic criteria**

An international consensus defining normal blood pressure in pregnancy allows for consistent diagnoses among all clinicians. The definition of a normal blood pressure in pregnancy of less than 140 mm Hg systolic and 90 mm Hg diastolic was originally derived from nonpregnant populations based on normal distribution of blood pressure during pregnancy. More recent prospective cohort data support the longstanding 140/90 mm Hg threshold utilized for defining an elevated blood pressure during pregnancy and the immediate postpartum period. Severe-range blood pressure readings are defined as a systolic blood pressure of at least 160 mm Hg or diastolic blood pressure of at least 110 mm Hg. Although a single blood pressure reading is not diagnostic, sustained elevated or severe-range blood pressure readings require attention and evaluation. Severe hypertension may occur antepartum, intrapartum, or postpartum. Severe hypertension prior to 20 weeks' gestation is typically due to chronic hypertension and requires additional evaluation for end-organ damage and exclusion of secondary causes. Hypertensive disorders in pregnancy include gestational hypertension, preeclampsia, eclampsia, and chronic hypertension with superimposed preeclampsia. Gestational hypertension is defined as hypertension occurring after 20 weeks' gestation in persons with previously normal blood pressure. Preeclampsia is defined as gestational hypertension with new-onset proteinuria. In the absence of proteinuria, preeclampsia is diagnosed in cases of gestational hypertension with new-onset thrombocytopenia, renal insufficiency, impaired liver function, pulmonary edema, visual symptoms, or headache unresponsive to medication and not accounted for by alternative diagnosis.

**Table 1. Diagnostic criteria for preeclampsia**

| Blood pressure       | • SBP ≥ 140 mm Hg or DBP ≥ 90 mm Hg on two occasions at least 4 hours apart after 20 weeks’ gestation in a woman with previously normal blood pressure  
|                      | • SBP ≥ 160 mm Hg or DBP ≥ 110 mm Hg. (Severe preeclampsia may be confirmed with short interval (minutes) to facilitate timely treatment.)  
|                      | and  
| Proteinuria          | • 24-hour urine collection: > 300 mg  
|                      | • Protein/creatinine ratio: > 0.3  
|                      | • Urine dipstick reading: 2+ protein (use only if other quantitative methods unavailable)  
|                      | In the absence of proteinuria, new-onset hypertension with the new onset of any of the following meet diagnostic criteria:  
|                      | • Thrombocytopenia: Platelet count < 100,000  
|                      | • Renal insufficiency: Serum creatinine > 1.1 or doubling of baseline serum creatinine in the absence of another renal disease  
|                      | • Impaired liver function: Elevated LFTs at least twice normal concentration  
|                      | • Pulmonary edema  
|                      | • New-onset headache unresponsive to medication and not accounted for by alternative diagnosis  

DBP, diastolic blood pressure; LFTs, liver function tests; SBP, systolic blood pressure.
Zure disorder. Chronic hypertension with superimposed preeclampsia is defined as preeclampsia in women with a history of hypertension before pregnancy or prior to 20 weeks’ gestation.4

Prevalence
The combined prevalence of HDPs is approximately 15%, up from 13% in 2017.4 In a recent maternal mortality review, an HDP diagnosis code was documented in approximately 1 in 3 deaths occurring during hospitalization for delivery.1 Chronic hypertension occurs in approximately 2.2% of all pregnancies in the US and remains a major cause of maternal morbidity and mortality. Risk factors for HDPs such as advanced maternal age, obesity, and diabetes mellitus have increased in the US and might explain the increase in HDP prevalence.

There are substantial disparities in who will experience an HDP. Prevalence of any HDP was higher among delivery hospitalizations to women of advanced maternal age (18% among women ages 35–44 and 31% among women ages 45–55) and Black (20.9%) and American Indian/Alaskan Native (16.4%) women, to those residing in rural counties (15.5%) and in zip codes in the lowest median household-level income quartile (16.4%), or delivering in hospitals in the South (15.9%) or Midwest (15.0%).4 Compared with non-Hispanic White women, non-Hispanic Black women have higher odds of beginning pregnancy with chronic hypertension and developing severe preeclampsia. In a study of racial and ethnic disparities in pregnancy-related deaths, those caused by HDPs among Black and American Indian/Alaskan Native women were found to be substantially higher than those among White women.5 Factors that contribute to racial and ethnic inequities in chronic and pregnancy-induced hypertension include higher prevalence of HDP risk factors as well as differences in access to healthcare and the quality of healthcare delivered.5,7

Racial bias within the US healthcare system can affect HDP care from screening and diagnosis to treatment.8 Furthermore, psychosocial stress from experiencing racism is associated with chronic hypertension.9 Reducing the prevalence of HDPs and improving health outcomes for individuals experiencing an HDP is dependent on systems-level attention to the root causes of existing disparities.

Antepartum clinical management
Clinical guidance for antepartum management of HDPs focuses on prompt identification and preventing progression to severe maternal complications. Increasing evidence suggests that standardized treatment protocols improve patient outcomes, such as the integration of the Alliance for Innovation on Maternal Health (AIM) safety bundles. These multidisciplinary specific safety bundles combine expert opinions to support best practice in communities across the United States to make

Table 2. ACOG/SMFM recommendations for low-dose aspirin prophylaxis for preeclampsia prevention10

Low-dose aspirin (81 mg) prophylaxis initiation recommended for pregnant individuals at high risk for preeclampsia, between 12–28 weeks’ gestation (optimally < 16 weeks) with one or more risk factors:

- History of preeclampsia
- Multifetal gestation
- Chronic hypertension
- Pregestational diabetes
- Renal disease
- Autoimmune disease (ie, systemic lupus erythematosus, antiphospholipid syndrome)
- Combinations of multiple moderate risk factors:
  - Nulliparity
  - Obesity (prepregnancy BMI > 30)
  - First-degree relative with history of preeclampsia
  - Black race
  - Low income/low socioeconomic status
  - Advanced maternal age
  - Personal history factors (low birth weight, SGA, previous adverse pregnancy outcome, > 10-year pregnancy interval)
  - In vitro fertilization

ACOG, American College of Obstetricians and Gynecologists; BMI, body mass index; SGA, small-for-gestational age; SMFM, Society for Maternal-Fetal Medicine.
birth safer.\textsuperscript{10} The two main goals of management of women with pre-eclampsia are prevention of seizures and control of hypertension.

ACOG, the Society for Maternal-Fetal Medicine (SMFM), and the US Preventive Services Task Force (USPSTF) recommend low-dose aspirin (81 mg) prophylaxis in pregnant individuals at high risk of preeclampsia and in those with more than one moderate risk factor (Table 2). When indicated, low-dose aspirin should be initiated between 12 and 28 weeks’ gestation (optimally before 16 weeks) and continued daily until delivery.\textsuperscript{11} A dose of ASA 81 mg daily is conventional. However, a 2022 study suggests ASA 162 mg daily has demonstrated the benefit of a 29% reduction in the rate of preeclampsia for high-risk women without an increased risk of bleeding.\textsuperscript{12}

Once an HDP diagnosis is made, management strategies include blood pressure-lowering medication, prevention of eclamptic seizures, and close maternal and fetal monitoring and coordination and continuity of care during the postpartum period. The objectives of treating severe hypertension are to prevent congestive heart failure, myocardial ischemia, renal injury or failure, and ischemic or hemorrhagic stroke.\textsuperscript{13}

**Chronic hypertension during pregnancy**

Historically, treatment for mild chronic hypertension during pregnancy was not recommended based on a lack of data confirming maternal or perinatal benefit with treatment and concerns that antihypertensive therapy may impair fetal growth. As a result of the Chronic Hypertension and Pregnancy (CHAP) trial with results published in 2022, ACOG and SMFM recommend treatment with antihypertensive therapy for mild chronic hypertension in pregnancy. A blood pressure of 140/90 mm Hg was set as the threshold for initiating treatment.\textsuperscript{14,15} Patients with treated chronic hypertension should continue established antihypertensive therapy during pregnancy or change to a regimen compatible with pregnancy. The CHAP trial provided pivotal evidence suggesting that treatment of chronic hypertension in pregnancy reduces both maternal and neonatal morbidity without increasing the risk for SGA (small-for-gestational age) infants as compared to no treatment until hypertension became severe.\textsuperscript{15}

**Gestational hypertension and preeclampsia without severe features**

Outcomes in women with gestational hypertension are generally good, but the notion that gestational hypertension is intrinsically less concerning than preeclampsia is incorrect. Gestational hypertension is associated with adverse pregnancy outcomes and may not represent a separate entity from preeclampsia.\textsuperscript{16} Up to 50% of women with gestational hypertension will eventually develop proteinuria or other end-organ dysfunction consistent with the diagnosis of preeclampsia, and this progression is more likely when the hypertension is diagnosed before 32 weeks’ gestation.\textsuperscript{13}

Among women with gestational hypertension or preeclampsia without severe features, expectant management up to 37 weeks’ gestation is recommended, with frequent fetal and maternal evaluation. Fetal monitoring consists of fetal growth ultrasounds every 3 to 4 weeks, non-stress testing 1 to 2 times weekly, and biophysical profile or modified biophysical profile at least weekly. Delivery may be delayed until 37 weeks in the absence of abnormal antenatal testing.\textsuperscript{16}

**Preeclampsia with severe features**

Expectant management of preeclampsia with severe features before 34 weeks’ gestation is based on strict selection criteria of appropriate candidates and is best accomplished in a setting with resources appropriate for maternal and neonatal care, ideally in a tertiary care facility. Women with gestational hypertension who present with severe-range blood pressures should be managed with the same approach as for women with preeclampsia with severe features.

Expectant management may include attempts to delay delivery for antenatal corticosteroid benefit or longer if safe for both mother and fetus.\textsuperscript{16} Delivery is recommended when preeclampsia with severe features is diagnosed at or beyond 34 weeks’ gestation after maternal stabilization. Delivery is recommended at any time in the case of deterioration of maternal or fetal condition and should not be delayed for the administration of steroids in the late preterm period.\textsuperscript{15} Because expectant management is intended to provide neonatal benefit at the expense of maternal risk, it is not advised when neonatal survival is not expected. Mode of delivery in women with gestational hypertension or preeclampsia (with or without severe features) should be determined by routine obstetric considerations.

Antihypertensive treatment should be initiated expeditiously for acute-onset severe hypertension that is confirmed as persistent (two readings at least 15 minutes apart). Intravenous hydralazine, intravenous labetalol, and oral nifedipine are the three most common agents used for this purpose. Although parenteral antihypertensive therapy may be needed initially for acute control of blood pressure, oral med-
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Pharmacologic regimens, typically oral labetalol and extended-release nifedipine, should be used during periods of expectant management. One approach is to begin an initial regimen of labetalol 200 mg orally every 12 hours and increase the dose up to 800 mg orally every 8 to 12 hours as needed (up to maximum 2,400 mg/day). If the maximum dose is inadequate to achieve the desired blood pressure goal, or the dosage is limited by adverse effect, then short-acting or extended-release nifedipine can be added gradually.16

The prevention of eclampsia is empirically based on the concept of timely delivery once preeclampsia has been diagnosed. Magnesium sulfate is the most effective available agent in reducing eclampsia and should be considered the drug of choice in the intrapartum and postpartum periods. Benzodiazepines and phenytoin are justified only in the context of antiepileptic treatment or when magnesium sulfate is contraindicated (myasthenia gravis, hypocalcemia, moderate-to-severe renal failure, cardiac ischemia, heart block, or myocarditis) or unavailable.17

A significant body of evidence attests to the efficacy of magnesium sulfate to prevent seizures in women with preeclampsia with severe features.17 The evidence regarding the benefit-to-risk ratio of magnesium sulfate prophylaxis is less supportive of routine use in pre-eclampsia without severe features.5

Optimal magnesium sulfate dosing remains a contested point among professionals as inadequate data exist supporting one dosage over another. Seizures may occur even with magnesium at a therapeutic level. Further complicating aspects are that steady magnesium levels are reached more slowly during the antepartum period than postpartum period related to the larger volume of distribution and higher body mass index during the antepartum period.18 Magnesium should continue for 24 hours following delivery.

Given magnesium sulfate is excreted almost exclusively in the urine, measuring urine output should be part of the clinical monitoring, in addition to monitoring of respiration status and tendon reflexes. If renal function is impaired, serum magnesium levels will increase quickly, which places the patient at risk of significant adverse effects. In patients with mild renal failure or oliguria, the loading dose of 4 to 6 g should be followed by a maintenance dose of only 1 g/hr rather than 2 g/hr.

HELLP syndrome
In the setting of HELLP syndrome between viability and 34 weeks’ gestation, delivery would ideally be delayed 24 to 48 hours for fetal corticosteroid benefit assuming both maternal and fetal conditions remain stable. Contraindications to delayed delivery for fetal corticosteroid benefit include uncontrolled hypertension, eclampsia, pulmonary edema, suspected placental abruption, disseminated intravascular coagulation, nonassuring fetal status, or intrauterine fetal demise.2

The clinical course of HELLP syndrome often is characterized by progressive and sometimes sudden deterioration in maternal and fetal condition. Because the management of patients with HELLP syndrome requires the availability of neonatal and obstetric intensive care units and personnel with special expertise, patients with HELLP syndrome who are remote from term should receive care at a tertiary care center. Very close monitoring including oxygen saturation. Only subsequently is attention directed to the administration of magnesium sulfate as most eclamptic seizures are self-limiting. Magnesium sulfate is not administered to stop a seizure but to prevent recurrent seizures. During eclamptic seizures, there are usually prolonged fetal heart rate decelerations, even fetal bradycardia, and sometimes an increase in uterine contractility and baseline tone. After a seizure, because of maternal hypoxia and hypercarbia, the fetal heart rate tracing may show recurrent decelerations, tachycardia, and reduced variability. Delivery should not proceed until maternal hemodynamic stabilization is achieved. Furthermore, maternal resuscitation is typically followed by the return of a normal fetal tracing.

Eclampsia
The initial steps in the management of a woman with eclampsia include basic supportive measures to prevent maternal injury and aspiration. The patient is placed in lateral position, oxygen is administered, and vital signs are monitored.
is required until delivery and in the postpartum period, with laboratory testing (LFTs, platelet count, and H/H) occurring no less than every 12 hours. With supportive care, 90% of patients with HELLP syndrome will have platelet count more than 100,000 and down trending liver enzymes within 7 days’ postpartum.19

**Postpartum clinical management**

Hypertensive disorders of pregnancy may occur during the postpartum period up to 6 weeks following delivery. Postpartum hypertension and preeclampsia may present as persistent or exacerbated hypertension in women with previous hypertensive disorders of pregnancy or as a new-onset condition. Most women who experience resultant eclampsia, stroke, pulmonary edema, or hypertensive encephalopathy in the postpartum period have symptoms that may include increasing peripheral edema, headache, visual disturbances, altered mental status, and/or decreased level of consciousness for hours to days before presentation. Risk factors for postpartum stroke include massive transfusion for postpartum hemorrhage, hypertensive disorders, advanced maternal age, connective tissue disorders, HIV, sepsis, thrombophilia, and congenital heart disease.20

All patients should receive information on preeclampsia during postpartum discharge teaching including signs/symptoms, importance of reporting signs/symptoms to a healthcare provider as soon as possible, and severe features warranting return to hospital emergency department/obstetric triage after discharge. In patients discharged with a preeclampsia diagnosis, arranging appropriate outpatient surveillance is vital including routine home and clinical blood pressure monitoring within 3 to 5 days and again in 7 to 10 days.21

Several systematic reviews and meta-analyses have linked preeclampsia with an increased risk of cardiovascular disease including hypertension, myocardial infarction, congestive heart failure, cerebrovascular events, peripheral arterial disease, and cardiovascular mortality later in life, with an estimated doubling of odds compared with women unaffected by preeclampsia.22 Lifelong attention should be paid to these women related to their elevated cardiovascular risks, particularly related to ongoing health and preventive measures.17,23

**Prepregnancy and interpregnancy care**

Research has shown a positive correlation between prepregnancy blood pressure control and improved maternal and neonatal outcomes.24 Blood pressure optimization prior to pregnancy plays a crucial component to decreased maternal morbidity and mortality. Consideration should be given to adequate antihypertensive therapy appropriate to women of childbearing age. Ideally blood pressure would be well-controlled prior to pregnancy on either labetalol or nifedipine. For those with difficult-to-control hypertension, other agents may be appropriate following consultation with maternal-fetal medicine and risk-benefit analysis. Currently, the prescribing rates of angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARB) to women of childbearing age remain high despite known teratogenic effects. Among those evaluated, less than half have documented contraception. If a woman presents for a routine gynecologic or prepregnancy visit on an ACE inhibitor or ARB, patient education related to the importance of effective contraception or a change to her antihypertensive regimen is crucial to potentially prevent an adverse neonatal outcome related to the teratogenic effects of these medications.25

**Conclusion**

The prevalence of HDPs has continued to increase over time with notable racial and ethnic, sociodemographic, and geographic disparities. Severe HDP-associated maternal complications and mortality are preventable with equitable implementation of public health education, particularly during the perinatal period, and clinical strategies. These include efforts across the life course for preventing HDPs; identifying, monitoring, and appropriately treating those with HDPs with continuous and coordinated care; increasing awareness of urgent maternal warning signs; and implementing quality improvement initiatives to address severe hypertension.

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