

Hypertensive Emergencies in Pregnancy



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KEYWORDS

- Hypertensive disorders • Pregnancy • Gestational hypertension • Preeclampsia
- Antihypertensive medications

KEY POINTS

- The 4 main categories of hypertensive disorders in pregnancy are chronic hypertension, gestational hypertension, preeclampsia, and chronic hypertension with superimposed preeclampsia.
- Hypertensive disorders contribute to significant maternal, fetal, and neonatal morbidity and mortality.
- The pathophysiology and etiology of hypertensive disorders in pregnancy is not well understood.
- All antihypertensive drugs cross the placenta, and only a few medications have been sufficiently studied in pregnancy.
- Untreated severe maternal hypertension can lead to end-organ injury. First-line medications for severe hypertension include intravenous labetalol, intravenous hydralazine, and oral nifedipine.

INTRODUCTION

Classification of Hypertensive Disorders in Pregnancy

There are 4 categories of hypertensive disorders in pregnancy as outlined by the 2013 American College of Obstetricians and Gynecologists (ACOG) Task Force on Hypertension in Pregnancy.¹ This group of experts was convened to review available data and provide evidence-based guidelines for the diagnosis and management of hypertensive disorders in pregnancy. The 4 categories include chronic hypertension (CHTN), preeclampsia, gestational hypertension (GHTN), and CHTN with superimposed preeclampsia.

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CHTN is an increase in blood pressure that either antedates pregnancy or begins before 20 weeks of gestation. Preeclampsia involves an elevation in blood pressure after 20 weeks of gestation in addition to either proteinuria or the development of signs of end-organ damage known as “severe features” (Box 1). Proteinuria is defined as excretion of 300 mg or greater of protein in a 24-h urine collection or a random protein to creatinine ratio of at least 0.3 mg/dL. Importantly, the ACOG recently emphasized that proteinuria is not requisite for the diagnosis of preeclampsia.¹ Preeclampsia can be subdivided into preeclampsia and preeclampsia with severe features based on the presence of severely elevated blood pressure or the aforementioned severe signs and symptoms.² The amount of protein is no longer used to differentiate preeclampsia and preeclampsia with severe features. GHTN occurs when there is an increase in blood pressure after 20 weeks of gestation in the absence of proteinuria or other severe features of preeclampsia. Finally, preeclampsia can occur in patients with longstanding CHTN. This category is referred to as CHTN with superimposed preeclampsia.¹

A non-pregnancy-related acute increase in blood pressure known as a “hypertensive emergency” may also occur in pregnant patients. This life-threatening presentation necessitates immediate treatment. Examples of this clinical scenario include hypertensive encephalopathy, aortic dissection, left ventricular failure, and increased catecholamines secondary to conditions such as pheochromocytoma or cocaine intoxication.³ Some of these diagnoses, like hypertensive encephalopathy, can be difficult to differentiate from preeclampsia. A retrospective study found that hypertensive encephalopathy is quite rare in comparison with preeclampsia.⁴

Epidemiology of Hypertensive Disorders in Pregnancy

An increasing number of pregnant women in the United States have chronic medical conditions, like CHTN, that increase the risk of adverse outcomes.⁵ The prevalence of hypertensive disorders in pregnancy has been increasing over the last decade with up to 8% of deliveries affected in 2006.⁶ A cross-sectional study of national data found that the increased prevalence of hypertensive disorders was highest for CHTN and GHTN, but rates of preeclampsia are also increasing.⁶ In fact, since 1987, the incidence of preeclampsia has increased by approximately 25% in the United States.⁷ Hypertensive disorders are more common in women with multiple gestations, chronic medical conditions, and gestational diabetes.⁶ The prevalence of hypertension is expected to continue to increase in the future with advancing maternal age and rising rates of obesity.⁸

Hypertensive disorders are a predominant cause of maternal and perinatal morbidity and mortality around the world.² From 2006 to 2010, hypertensive disorders

Box 1

Severe features of preeclampsia

- Severe hypertension (systolic BP \geq 160 mm Hg or diastolic BP \geq 110 mm Hg)
- Thrombocytopenia (platelets $<$ 100,000/ μ L)
- Elevated liver enzymes (twice normal)
- Right upper quadrant or epigastric pain unresponsive to medication
- Unremitting cerebral or visual symptoms
- Renal insufficiency (creatinine $>$ 1.1 mg/dL or twice normal)
- Pulmonary edema

Abbreviation: BP, blood pressure.

accounted for 9.4% of pregnancy-related deaths in the United States.⁵ The majority of maternal deaths related to hypertensive disorders of pregnancy occur within 42 days postpartum. Severe maternal complications occurring significantly more often in women with hypertensive disorders compared with normotensive women include acute renal failure, disseminated intravascular coagulation syndrome, pulmonary edema, pulmonary embolism, cerebrovascular disorders, and respiratory distress. Severe obstetric complications are most likely to occur in preeclampsia with severe features but also occur in GHTN, although the risk is comparatively modest.⁶ Adverse fetal and neonatal outcomes, including iatrogenic preterm birth, low birth weight, and fetal demise, are also more likely in women with hypertensive disorders.^{9,10}

PATHOPHYSIOLOGY

The pathophysiology of hypertensive disorders in pregnancy is not understood fully. In pregnancy, the renin–angiotensin hormone system is upregulated and systemic vascular resistance decreases. As a result, blood pressure initially decreases starting as early as 7 weeks gestation. The decrease in diastolic blood pressure (DBP) tends to be greater than the decrease in systolic blood pressure (SBP). Maternal blood pressure begins to increase again in the third trimester.¹¹ The inciting event in the development of pregnancy-related hypertensive disorders is thought to be abnormal cytotrophoblast invasion of spiral arteries, leading to reduced uteroplacental perfusion. The resultant placental ischemia is assumed to cause an abnormal activation of the maternal vascular endothelium.¹²

The cardiovascular hemodynamics of hypertensive disorders are variable (**Table 1**).¹³ In general, preeclampsia is associated with increased systemic vascular resistance, increased left ventricular afterload, and decreased cardiac output.^{13,14} Conversely, based on a small cohort study using Doppler echocardiography in pregnancy, women with GHTN maintain low systemic vascular resistance and increased cardiac output.¹⁴

Preeclampsia involves a constellation of physiologic changes that include vasoconstriction, hemoconcentration, and possible ischemia in the placenta and other maternal organs. Vascular reactivity is owing to an imbalance and dysfunction in vasodilatory and vasoconstrictive substances. The resultant vasoconstriction decreases placental perfusion and perfusion of maternal organs, which can lead to end-organ damage. As renal blood flow decreases, so does the glomerular filtration rate. In rare cases, profoundly decreased renal perfusion can lead to acute tubular necrosis. Liver hematomas and rupture can occur in cases of preeclampsia, especially in conjunction with severe thrombocytopenia. The exact cause of eclamptic seizures is not well understood, but both hypertensive encephalopathy and ischemia secondary to vasoconstriction have been hypothesized. The visual changes (scotoma) often seen in women with preeclampsia may occur secondary to edema of the posterior cerebral hemispheres.³

Table 1
Hemodynamic physiology of hypertensive disorders in pregnancy

| Disorder | Systemic Vascular Resistance | Cardiac Output |
|--------------------------|------------------------------|----------------|
| Pregnancy | Decreased | Increased |
| Gestational hypertension | Decreased | Increased |
| Preeclampsia | Increased | Decreased |

PATIENT EVALUATION OVERVIEW

Diagnosis of Hypertensive Emergencies

Hypertension is defined as an SBP of greater than or equal to 140 mm Hg or DBP greater than or equal to 90 mm Hg on 2 occasions (≥ 4 hours apart). Hypertension is considered severe with a SBP of 160 mm Hg or greater or a DBP of 110 mm Hg or greater.^{1,11} As mentioned, preeclampsia is diagnosed in the presence of elevated blood pressure and either proteinuria (≥ 300 mg of protein in a 24-h urine collection or protein to creatinine ratio of ≥ 0.3 mg/dL) or severe features as evidence of end-organ damage (**Box 1**). Severe forms of preeclampsia also include eclampsia and HELLP syndrome. HELLP is an acronym for hemolysis (H), elevated liver enzymes (EL) and low platelets (LP; see **Box 2**).³

Hypertension may be absent or mild in up to 50% of patients with HELLP syndrome. The differential diagnosis of HELLP includes acute fatty liver of pregnancy, gallbladder disease, lupus flare, and thrombotic thrombocytopenic purpura/hemolytic uremic syndrome. HELLP syndrome can be differentiated from these other conditions based on normal ammonia levels, mild renal insufficiency and anemia, and the presence of hypertension and proteinuria. A potential complication of HELLP syndrome is the development of a subcapsular liver hematoma. These patients often have phrenic nerve pain, and the diagnosis can be confirmed with imaging studies like computed tomography or abdominal ultrasonography.³

Hypertensive encephalopathy is a type of hypertensive emergency characterized by cerebral edema. This typically occurs in patients with SBP of greater than 220 mm Hg or DBP of greater than 120 mm Hg, although it can occur at lower blood pressures in pregnant women and those with newly increased blood pressures.^{3,4} In hypertensive encephalopathy, there is a failure of the cerebral arteriolar constriction in response to increasing blood pressure. The signs and symptoms of hypertensive encephalopathy, including headache, confusion, and nausea, can develop over several days. Papilledema and retinal hemorrhages are frequently seen with fundoscopic examination in patients with hypertensive encephalopathy.¹⁵ Signs of damage to other organs (eg, cardiac dysfunction or renal failure) may also be present.³ Persistent evidence of neurologic deficits could indicate the presence of a stroke.³

PHARMACOLOGIC TREATMENT OPTIONS

The benefit of antihypertensive medication in mild-to-moderate CHTN remains uncertain and treatment is not recommended for persistent CHTN with a SBP of less than 160 mm Hg and a DBP of less than 105 mm Hg.^{1,16} Likewise, antihypertensive medication is not

Box 2

Diagnosis of HELLP syndrome

- Evidence of hemolysis
 - Schistocytes on peripheral smear
 - Increased lactate dehydrogenase
 - Decreased haptoglobin
 - Increased total bilirubin (≥ 1.2 mg/dL)
 - Decreased hematocrit (hemolysis) or increased hematocrit (hemoconcentration)
- Elevated aspartate and alanine aminotransferase (≥ 70 IU/L)
- Thrombocytopenia (platelets $< 100,000/\mu\text{L}$)

Abbreviation: HELLP, hemolysis, elevated liver enzymes, and low platelets.

recommended for GHTN or preeclampsia with a SBP of less than 160 mm Hg and a DBP of less than 110 mm Hg.¹ Treatment of mild to moderate hypertension results in a 50% decrease in the development of severe hypertension, but no difference in preeclampsia or other outcomes according to a Cochrane systematic review.¹⁷ Conversely, treatment is recommended for severe hypertension (SBP \geq 160 mm Hg and/or DBP $>$ 105–110 mm Hg) persisting for longer than 15 minutes.¹⁸ All antihypertensive drugs cross the placenta and most are Food and Drug Administration category C (risks demonstrated in animal studies but no human studies have been performed; benefits may outweigh the risks). Changes to drug labeling, which include removal of the letter categories took effect June 30, 2015. The new labeling format will be used for all newly submitted drugs while changes to currently approved drugs will be phased in. The problems with medication use during pregnancy can be divided into teratogenicity (first trimester), fetotoxicity (throughout pregnancy), and breast feeding (**Table 2**).¹⁹ Factors that modify the level of risk include agent, dose, and timing.²⁰ As is all too common, unplanned pregnancy often leads to delays in seeking prenatal care and either self-discontinuation or provider advice to stop taking prescription medications in the second trimester, at which point the risk of teratogenesis has already occurred.

Maternal hypertension during pregnancy has been associated with an increased risk of birth defects possibly owing to alterations in uterine blood flow.²¹ These include congenital heart disease, esophageal atresia, and hypospadias.^{21–23} In a systematic review and metaanalysis, Ramakrishnan and colleagues²¹ found that congenital anomalies were more common in both treated and untreated hypertension, although the risk was greater in treated hypertension. Van Zutphen and colleagues²³ demonstrated a higher incidence of hypospadias in untreated hypertension and hypertension treated with nonselective β -blockers, specifically labetalol and propranolol. In this study, labetalol use may have been a surrogate for more severe hypertension. Alternatively, labetalol may affect the urethra during a critical period of development by altering uterine blood flow or may interfere with testosterone production by Leydig cells.

Few drugs have been sufficiently studied for use during pregnancy and there is a lack of randomized, placebo-controlled trials. Commonly recommended medications for the management of severe hypertension include intravenous labetalol and hydralazine, and oral nifedipine. Most guidelines recommend labetalol (66%) or hydralazine (33%) as first-line treatment and oral short-acting nifedipine as second-line treatment, although the ACOG now regards oral nifedipine as a first-line drug in the emergent treatment of severe hypertension.^{18,24,25} Methyldopa, a centrally acting α 2-adrenergic agonist, has also been suggested as a second-line agent for severe hypertensive crisis in some guidelines.²⁴ Methyldopa prevents vasoconstriction by replacing norepinephrine in the synaptic vesicles, thereby reducing catecholamine release and central sympathetic outflow. However, control is gradual over 6 to 8 hours, thus limiting its use in

Table 2
Medication effects based on gestational age

| Gestational Age (From Last Menstrual Period), wk | Effect | Consequence |
|--|------------------|--|
| 1–4 | “All or nothing” | Either miscarriage or no effect. |
| 5–12 | Teratogenesis | Period of organ formation. Birth defects possible during this time. |
| 12–42 | Fetotoxicity | Organs completely formed. Possible damage to the brain and kidneys or impaired growth. |

acute settings. More than 40 years of experience with methyldopa and follow-up in children up to 7.5 years old demonstrates the safety methyldopa during pregnancy, but multiple studies have demonstrated that other agents, namely labetalol, are more effective in controlling blood pressure. There are insufficient data to demonstrate the superiority of 1 drug over another; the choice of antihypertensive in the treatment of very high blood pressure during pregnancy depends on physician experience and knowledge of adverse effects. Drug characteristics and suggested dosing regimens are shown in **Tables 3** and **4** respectively.

In cases of refractory hypertension, labetalol infusion, nicardipine, and sodium nitroprusside are recommended. Refractory hypertension should be treated in conjunction with a specialist in maternal-fetal medicine or critical care. Nicardipine, like nifedipine, is a dihydropyridine calcium channel blocker. Nicardipine is indicated for use in severe hypertension. It has less negative inotropic effect and is less likely to cause reflex tachycardia. Experience with nicardipine in pregnancy shows 91% success in decreasing blood pressure.²⁸ Sodium nitroprusside is a nonselective, direct nitric oxide donor available for use as an intravenous infusion because of its 3-minute duration of action. Sodium nitroprusside is used as a last resort, in part because of the risk of cyanide toxicity, which can occur after 24 to 48 hours. The risk of fetal cyanide poisoning is only theoretic.

Complete treatment of severely increased blood pressure should also include an assessment of fetal wellbeing and close monitoring of blood pressure, symptoms, and laboratory tests. Appropriate laboratory tests are hematocrit, platelet count, serum creatinine and transaminases. In addition, ultrasonography should be performed to assess fetal growth and amniotic fluid volume.³

TREATMENT COMPLICATIONS

Magnesium Toxicity

Magnesium is not an antihypertensive. It should be used for seizure prevention in preeclampsia with severe features and for neuroprotection when delivery is expected before 32 weeks gestation. The mechanism of action for magnesium sulfate in the prevention of seizures is unknown. It can be associated with several adverse effect and potential toxicities (**Table 5**).^{3,29} Magnesium sulfate can cause uterine atony and increase the risk of postpartum hemorrhage.^{3,29} Women receiving magnesium should be monitored closely and magnesium infusion stopped immediately if signs of toxicity develop. Magnesium toxicity can be treated with 10 mL of 10% calcium gluconate solution. Cardiopulmonary arrest, although rare, may require intubation and mechanical ventilation.³

Several case reports have documented adverse effects when combining magnesium and calcium channel blockers. One published case reported neuromuscular blockade,³⁰ and another case involved severe hypotension and subsequent maternal death.¹ However, a retrospective review of patients receiving both magnesium and nifedipine found no increase in neuromuscular weakness, neuromuscular blockade, or hypotension compared with controls.³¹ Regardless, patients receiving both magnesium and nifedipine should be monitored closely given the potential for toxicities with the use of 2 calcium antagonists.¹⁸

Medications to Avoid in Pregnancy

There are several antihypertensive medications that are not recommended for use in pregnancy. Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, and mineralocorticoid receptor antagonists should be avoided without a specific

Table 3**Characteristics of antihypertensive drugs commonly used for severe hypertension during pregnancy**

| Drug (FDA Risk Category) | Mechanism | Perinatal Concerns | Side Effects |
|---------------------------------|--|--|---|
| Labetalol (C) | Nonselective β -blocker and vascular α_1 -receptor blocker | β -Blockers associated with congenital heart disease, cleft lip/palate, and open neural tube defects but causality not yet proven ²⁶ ; generally regarded as safe Concern for growth restriction with atenolol but recent study suggests similar risk with labetalol ²⁷ Fetal distress secondary to abrupt maternal hypotension Neonatal bradycardia and hypoglycemia | Caution in women with asthma (bronchoconstriction) Fatigue, lethargy, exercise intolerance, peripheral vasoconstriction, sleep disturbance |
| Hydralazine (C) | Direct vasodilator Relaxes arteriolar smooth muscle, exact mechanism unknown | Fetal distress secondary to abrupt maternal hypotension Cesarean section, abruption, APGAR <7 more common compared with other agents Rarely neonatal thrombocytopenia and neonatal lupus | Palpitations, tachycardia, headache, nausea/vomiting, flushing Side effects may mimic worsening preeclampsia Rarely polyneuropathy or drug-induced lupus |
| Nifedipine (C) | Dihydropyridine calcium channel blocker acting predominantly on the arterial smooth muscle | Increased liver clearance may require higher doses Fetal distress secondary to abrupt maternal hypotension | Tachycardia, palpitations, peripheral edema, headaches, flushing Risk of neuromuscular blockade, myocardial depression, and hypotension when combined with magnesium unsubstantiated Sublingual preparations associated with MI and death |

Abbreviations: FDA, US Food and Drug Administration; MI, myocardial infarction.

| Table 4 Initial approach to the management of severe hypertension | | |
|---|--|--|
| Drug | | |
| Labetalol | Hydralazine | Nifedipine |
| If BP remains ≥ 160 mm Hg systolic or ≥ 110 mm Hg diastolic for more than 15 min: | | |
| Administer 20 mg IV over 2 min | Administer 5–10 mg IV over 2 min | Administer 10 mg orally |
| Repeat BP in: | | |
| 10 min | 20 min | 20 min |
| If BP remains ≥ 160 mm Hg systolic or ≥ 110 mm Hg diastolic: | | |
| Administer 40 mg IV over 2 min | Administer 10 mg IV over 2 min | Administer 20 mg orally |
| Repeat BP in: | | |
| 10 min | 20 min | 20 min |
| If BP remains ≥ 160 mm Hg systolic or ≥ 110 mm Hg diastolic: | | |
| Administer 80 mg IV over 2 min | Administer labetalol 20 mg IV over 2 min | Administer 20 mg orally |
| Repeat BP in: | | |
| 10 min | 10 min | 20 min |
| If BP remains ≥ 160 mm Hg systolic or ≥ 110 mm Hg diastolic: | | |
| Administer hydralazine 10 mg IV over 2 min | Administer labetalol 40 mg IV over 2 min | Administer labetalol 40 mg IV over 2 min |
| Repeat BP in: | | |
| 20 min | 10 min | 10 min |
| If BP remains ≥ 160 mm Hg systolic or ≥ 110 mm Hg diastolic: | | |
| Obtain emergency consultation and treat as recommended | | |

These algorithms are appropriate for antepartum, intrapartum, and postpartum severe hypertension.

Choice of agents should be guided by clinician experience and knowledge of adverse effect.

Management should also include physician notification, documentation, and fetal surveillance.

Once target BP is achieved, check BP every 10 minutes \times 1 hour, then every 15 minutes \times 1 hour, then every 30 minutes \times 1 hour, then hourly for 4 hours.

Abbreviations: BP, blood pressure; IV, intravenous.

Adapted from American College of Obstetricians and Gynecologists. Committee opinion no. 623: emergent therapy for acute-onset, severe hypertension during pregnancy and the postpartum period. *Obstet Gynecol* 2015;125:521–5.

indication like proteinuric renal disease.¹ Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers interfere with fetal renal hemodynamics leading to congenital anomalies in the first trimester and oligohydramnios, kidney damage, and death in the second and third trimesters. Mineralocorticoid receptor antagonists

| Table 5 Magnesium toxicity associated with serum levels | |
|--|------------------------------|
| Serum Magnesium Level (mg/dL) | Toxicity |
| 8–12 | Loss of deep tendon reflexes |
| 10–12 | Somnolence, Slurred speech |
| 12–16 | Respiratory paralysis |
| 20–35 | Cardiac arrest |

like spironolactone can have anti-androgenic effects in pregnancy with feminization of the male fetus found in animal studies.³² β -Blockers are commonly used antihypertensives in pregnancy, but there is ongoing debate about the risk of congenital anomalies and growth restriction (see [Table 3](#)).

EVALUATION OF OUTCOME

Goals of Treatment

The purpose of treatment of severe hypertension in pregnancy is to decrease maternal and fetal complications. Women with severely increased blood pressure are at risk of stroke, myocardial infarction, renal failure, uteroplacental insufficiency, placental abruption, and death.^{11,33,34} Severe hypertension is the only modifiable end-organ complication of preeclampsia.³⁴ The blood pressure target for women with CHTN requiring antihypertensive therapy is to maintain a SBP of 160 mm Hg or less and a DBP of 105 mm Hg or less, or 160/110 mm Hg or less in GHTN and preeclampsia.¹ It is important to try to avoid an abrupt decrease in pressure which can lead to potential harmful fetal effects.¹¹ Therapy should aim to decrease mean arterial pressure by approximately 20% to 25% over minutes to hours then further decrease blood pressure to less than 160/110 mm Hg over the subsequent hours. The signs and symptoms of hypertensive encephalopathy (headache and confusion) often improve rapidly with treatment.

Fetal Evaluation

Monthly growth ultrasounds in the third trimester are recommended for women with CHTN. Umbilical artery Doppler velocimetry testing should be performed weekly if there is any evidence of fetal growth restriction or superimposed preeclampsia. In addition, antenatal fetal testing (e.g. nonstress testing) is suggested in women with CHTN and either coexisting need for medication, signs of fetal growth restriction, or superimposed preeclampsia.¹ Women with GHTN or preeclampsia in pregnancy should have ultrasounds to measure fetal growth, antenatal testing, twice weekly blood pressure checks, and weekly laboratory evaluations. Candidates for expectant management of preeclampsia with severe features require closer surveillance, including daily nonstress testing, weekly ultrasound for Doppler of the umbilical artery and amniotic fluid volume measurement, ultrasound every 2 to 3 weeks for growth, daily fetal movement counting, and periodic laboratory evaluation (frequency depends on patient stability).³ Administration of corticosteroids for fetal lung maturity is recommended in women with preeclampsia at less than 34 weeks of gestation.¹

Delivery Planning

Induction of labor is recommended for hypertensive disorders in pregnancy given the associated maternal and fetal morbidities in addition to the high risk of progression of disease. However, the optimal timing of delivery remains controversial. There have been no randomized, controlled trials to guide timing of delivery in women with CHTN. A cohort study of women with CHTN found that delivery at 38 to 39 weeks gestation was optimal for balancing fetal and neonatal risks.³⁵ The ACOG endorses delivery at 38 to 39 weeks for women with CHTN not requiring medication, 37 to 39 weeks for women controlled with medication, and 36 to 37 weeks for women with severe uncontrolled hypertension.³⁶

A randomized, controlled trial in women with either GHTN or preeclampsia without severe features found that induction of labor at 37 weeks gestation was associated with a significant decrease in composite maternal morbidity as compared with

expectant management.³⁷ The ACOG suggests delivery at 37 to 38 weeks for women with GHTN and induction of labor for preeclampsia without severe features at 37 weeks gestation.³⁶

Both the ACOG and the Society for Maternal-Fetal Medicine recommend induction of labor at 34 weeks of gestation or greater in patients with preeclampsia with severe features.³⁶ A systematic review of induction of labor for preeclampsia with severe features before 34 weeks gestation found an association with decreased birth weight, greater rates of admission to the neonatal intensive care unit, longer neonatal hospitalization, and increased neonatal complications including intraventricular hemorrhage and respiratory distress syndrome.³⁸ However, induction of labor for preeclampsia with severe features is recommended before 34 weeks in women who are not candidates for expectant management (**Box 3**). In these women, delivery can be delayed for 48 hours for administration of corticosteroids for fetal lung maturity in the absence of uncontrollable severe hypertension, eclampsia, pulmonary edema, placental abruption, disseminated intravascular coagulation, or nonreassuring fetal status. The management of preeclampsia with severe features before viability (23–24 weeks in most centers) should involve consultation with a specialist in maternal-fetal medicine.

The mode of delivery should be decided based on the usual obstetric indications. Blood pressure should be controlled before delivery, especially in women undergoing cesarean section with general anesthesia because blood pressure can increase during endotracheal intubation. The use of neuraxial anesthesia is advised unless contraindicated by maternal thrombocytopenia.¹ The exact platelet level below which neuraxial anesthesia is contraindicated is controversial and varies from institution to institution based on platelet count (usually 50,000–100,000/ μ L), trend, and evidence of platelet dysfunction. Patients with severe thrombocytopenia (platelets <50,000) may require a platelet transfusion en route to the operating room if a cesarean delivery is indicated.³

Postpartum Management

Women with GHTN, preeclampsia, or CHTN with superimposed preeclampsia should have their blood pressure monitored for at least 72 hours postpartum and again 7 to

Box 3

Contraindications to expectant management of preeclampsia with severe features

- Severe hypertension refractory to medication
- Eclampsia
- Pulmonary edema
- Placental abruption
- Disseminated intravascular coagulation
- Thrombocytopenia (platelets <100,000/ μ L)
- Abnormal hepatic enzyme concentrations (twice normal)
- Renal dysfunction (creatinine >1.1 mg/dL or twice normal)
- Right upper quadrant or epigastric pain unresponsive to medication
- Unremitting cerebral or visual symptoms
- Nonreassuring fetal status
- Fetal demise

10 days after delivery.¹ Normalization of blood pressure typically occurs within 10 days of delivery in patients with pregnancy-related hypertension.³ The ACOG recommends antihypertensive treatment in women with persistently increased blood pressures postpartum defined as SBP of 150 mm Hg or higher or DBP of 100 mm Hg or higher. Nonsteroidal antiinflammatory agents used to treat postpartum pain may exacerbate blood pressure owing to sodium and water retention.³⁹ An alternate analgesic can be considered in women with increased blood pressure that persists for more than 1 day postpartum.¹

Women should be counseled and receive discharge instructions regarding the need to contact their health care provider if they develop warning signs or symptoms, including an unremitting headache, visual disturbances, or right upper quadrant pain. New-onset hypertensive disorders of pregnancy can also occur in the postpartum period. Postpartum women with newly elevated blood pressure should be evaluated and treated similarly to antenatal patients.¹

Seizure prophylaxis is continued in patients with preeclampsia with severe features for 24 hours postpartum. Approximately 25% of cases of eclampsia occur in postpartum patients, and seizures can develop up to 4 weeks after delivery. Patients with preeclampsia with severe features are also at risk of developing pulmonary edema in the postpartum period. Furosemide can be used as deemed necessary in these patients.³

Women with pregnancies complicated by hypertensive disorders, especially preeclampsia requiring delivery at less than 34 weeks gestation, are at risk for the development of cardiovascular disease later in life. The ACOG suggests that women with a history of preeclampsia requiring a preterm delivery undergo yearly monitoring of blood pressure, lipids, fasting glucose, and body mass index.¹

SUMMARY

Hypertensive disorders of pregnancy are associated with an increased risk of maternal and perinatal morbidity and mortality. Appropriate management of hypertension and its associated complications can optimize outcomes. Several antihypertensive agents can be used safely in pregnancy, although evidence from randomized controlled trials is lacking. A greater understanding of the pathophysiology, etiology, and natural history of hypertensive disorders in pregnancy would allow for improved prevention strategies and ultimate elimination of associated morbidity and mortality.

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