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Hypertensive Emergencies in Pregnancy

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Abstract: Hypertensive disorders of pregnancy complicate 7% to 10% of pregnancies and are among the major causes of maternal and perinatal morbidity and mortality. Recently American College of Obstetricians and Gynecologists Taskforce on Hypertension during Pregnancy modified the diagnosis and management of hypertension in pregnancy, recommending prompt diagnosis, admission, close monitoring, and treatment. They strive to decrease maternal mortality and systemic complications. Labetalol, hydralazine, or nifedipine are considered first-line treatment, and either can be used to stabilize the patient with similar outcomes. Definite treatment is delivery of the fetus and should be considered based on the etiology of the hypertensive crisis and gestational age.
Key words: hypertensive emergencies, pregnancy, preeclampsia, hypertension, antihypertensives

Hypertensive Emergencies

Peripartum hypertensive disorders complicate 10% of pregnancies worldwide and are one of the main etiologies of maternal and perinatal morbidity and mortality.[1] They affect not only the parturient but also the fetus primarily because of iatrogenic premature deliveries and by adverse outcomes related directly to preeclampsia and hypertension (HTN) such as oligohydramnios and fetal growth restriction.

The current American College of Obstetricians and Gynecologists (ACOG) taskforce on HTN during pregnancy has modified several components of diagnostics and management of HTN during pregnancy and have simplified classification into only 4 categories: (1) preeclampsia-eclampsia; (2) chronic HTN; (3) chronic HTN with superimposed preeclampsia; and (4) gestational HTN.[2]

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DEFINITIONS

HTN is diagnosed with 2 determinations of either systolic blood pressure (BP) >140 mm Hg or diastolic blood pressure (DBP) >90 mm Hg or both, taken at least 4 hours apart. During pregnancy, it is considered severe if either systolic BP is ≥ 160 mm Hg or DBP is ≥ 110 mm Hg. A hypertensive emergency is considered when severe HTN occurs acutely, persists for >15 minutes, and presents in a

pregnant or postpartum patient with preeclampsia or eclampsia.[3]

Preeclampsia, a disease known to be exclusive to pregnancy and the postpartum period, has multisystem involvement and develops typically after 20 weeks of gestation. The majority of cases occur near term. Preeclampsia is known to be the most common cause of high BP during pregnancy with an incidence that has been increasing during the past 2 decades likely secondary to the obesity epidemic and increased maternal age. Preeclampsia is a dynamic and progressive systemic disease and remains a leading cause of maternal and perinatal morbidity and mortality worldwide.[2,4] It is classified as with or without severe features depending on patient's condition at the time; however, classification evolves as the disease progresses. It usually presents as new-onset HTN with proteinuria. Proteinuria is a late finding and the absence of it should never delay the diagnosis of preeclampsia. Current guidelines do not require the presence of proteinuria to diagnose preeclampsia. Preeclampsia may increase the risk of developing cardiovascular disease later in life.

Gestational HTN is defined as new-onset HTN (>140/90) after 20 weeks' gestation without proteinuria or any other symptoms. Chronic HTN is diagnosed when a patient has HTN before pregnancy or it develops before 20 weeks of gestational age. Chronic HTN with superimposed preeclampsia is diagnosed when either (1) the patient with previously controlled BP presents with either increase in BP or increased requirements of antihypertensive medications; (2) new onset of proteinuria or increase in known proteinuria levels; or (3) other symptoms or signs develop such as decrease in platelets to $<100{,}000/\mu L$; pulmonary congestion or edema; elevation in liver transaminase levels; renal insufficiency of new onset; or visual/central nervous system disturbances.

Recent recommendations by the ACOG Taskforce on Hypertension in Pregnancy are to not administer antihypertensive treatment to patients with mild gestational HTN or preeclampsia if BP remains < 160 mm Hg systolic or 110 mm Hg diastolic due to risks for the fetus and lack of significant benefit to the mother.[5,6] Treatment of chronic HTN outside of pregnancy is aimed at reduction of long-term complications such as stroke, coronary heart disease, congestive heart failure (CHF), and kidney disease (Table 1). During pregnancy these long-term goals collide with short-term maternal and fetal safety. Treatment of severe gestational HTN and preeclampsia with severe features are indicated to reduce the

TABLE 1. Classification of Hypertension

Classification	Systolic Blood Pressure (mm Hg)	Diastolic Blood Pressure (mm Hg)
Normal	< 120	< 80
Prehypertension	120-139	80-89
Stage 1 hypertension	140-159	90-99
Stage 2 hypertension	> 160	> 100
Hypertensive crisis (nonpregnant)	> 179	> 120
Hypertensive crisis in pregnancy	> 160	> 110
Chronic hypertension	Hypertension presenting before 20 wk gestation	
Gestational hypertension	Hypertension presenting after 20 wk gestation without proteinuria or other manifestations	
Preeclampsia	Hypertension presenting after 20 wk gestation associated with proteinuria, thrombocytopenia, renal insufficiency, impaired liver function, pulmonary edema, or cerebral or visual symptoms	

Data derived from: Chobanian et al[7] and American College of Obstetricians and Gynecologists Taskforce on Hypertension in Pregnancy.[8]

risk of CHF, pulmonary edema, cerebrovascular accidents, and death.

CAUSES OF COMPLICATIONS

Severe elevations of BP may result in stroke (hemorrhagic or ischemic), pulmonary edema, and myocardial ischemia. It is unknown at which exact BP levels the latter complications may occur.

Hypertensive crisis during pregnancy differs in both diagnosis and treatment from nonpregnant individuals and so do the rate of complications. In a nonpregnant individual, a hypertensive crisis is diagnosed when the BP is >179 systolic or >109 mm Hg diastolic and is further classified as either an urgency in the presence of symptoms or an emergency in the presence of end-organ damage (Table 2).[11] Hypertensive urgencies among nonpregnant individuals require urgent treatment and the goal of this treatment is to reduce BP within 24 to 48 hours. During hypertensive emergencies, the treatment should start immediately and is not intended to decrease BP to normal levels but to achieve a more appropriate DBP range, usually by decreasing it 10% to 20% or to a DBP of 100 to 110 mm Hg within 30 to 60 minutes.[11] During pregnancy, severe HTN (BP > 160/110) warrants close observation and treatment as well as appropriate management of pregnancy depending on gestational age. Maternal mortality rate due to hypertensive disorders despite treatment remains high, and accounts to over 10% of all causes of maternal mortality.[1]

Two recent COCHRANE reviews[12,13] analyzed the effect of treatment of severe

TABLE 2. Different Definitions of Hypertensive Crisis and Management Criteria[8–10]

	AHA Urgent	AHA Emergent	ACOG Emergent
BP range	SBP > 180 DBP > 120	SBP > 180 DBP > 120	SBP > 160 DBP > 110
Associated symptoms	Headache Shortness of breath Nose bleeds Severe anxiety No end-organ involvement	Neurological changes Hypertensive encephalopathy Cerebral infarction Intracranial hemorrhage Myocardial ischemia Acute pulmonary edema Aortic dissection Renal insufficiency Left ventricular dysfunction Eclampsia	IUGR < fifth percentile Severe oligohydramnios Pulmonary edema Disseminated intravascular coagulation Placental abruption HELLP syndrome Reversed end-diastolic flow Progressive renal insufficiency Severe thrombocytopenia Neurological symptoms Eclampsia
End-organ involvement/ damage	No	Yes	Yes
Target BP and time for stabilization (h)	Within 24-48 h—follow-up fist day after event as outpatient	Reduce 10% first hour, additional 15% next 2-3 h	Decrease within first hours 10%-20% Monitor fetus Decision on delivery according to gestational age, fetal assessment, and maternal condition

Management and treatment goals may differ in patients with specific pathology like intracranial hemorrhage and aortic dissection.

ACOG indicates The American College of Obstetricians and Gynecologists; AHA, American Heart Association; BP, blood pressure; DBP, diastolic blood pressure; HELLP syndrome, hemolysis, abnormal liver function tests and thrombocytopenia syndrome; SBP, systolic blood pressure.

BP during pregnancy. These included 35 trials and over 3500 patients. These studies suggest that drug therapy should be started when with DBPs are >100 to 110 mm Hg; overly aggressive treatment is discouraged due to concerns of affecting utero-placental blood flow.

MATERNAL ASSESSMENT

Pregnant women presenting with a hypertensive crisis should be triaged promptly on arrival and a full review of systems should be undertaken to evaluate the patient for chest pain or cardiovascular symptoms, neurological symptoms, such as headaches or visual abnormalities, consciousness, and difficulty in breathing. A thorough history of recent drug abuse, current medications, and past medical history is necessary. A physical examination focusing on end-organ damage should be undertaken. Blood work should be ordered to evaluate patient status and diagnose preeclampsia when necessary and should include a complete blood count with differential and platelet count, a comprehensive metabolic panel, and urinalysis. In a patient complaining of chest pain or shortness of breath and depending on the physical examination, additional tests as a chest x-ray or ECG may be required to rule out myocardial injury or pulmonary edema. The use of order sets and protocols to aid the physicians and all the staff managing patients with a hypertensive crisis has been recommended since the guidelines in 2000 and a recent report by Clark et al[14] showed a reduction in morbidity and complications when these order sets were instituted as protocols. Prompt involvement of maternal-fetal medicine specialists, anesthesiologists, neonatologists, and additional specialists involved in the multidisciplinary management of the maternal-fetal unit is recommended.

When patients present with any end-organ symptoms, involvement, or damage, they warrant pharmacologic treatment and in-hospital monitoring. Depending on the gestational age and presentation of the hypertensive crisis, the mother may need to be transferred to a facility with intensive care units for both mother and neonate. Continuous fetal heart rate monitoring during stabilization of the mother is required if the fetus is viable at the time of the hypertensive crisis. Frequent maternal pulse and BP monitoring is necessary and depending on the severity of the symptoms and the medications required, invasive maternal BP monitoring may be warranted with an arterial line (commonly in the radial artery).

The initial goal is to optimize and stabilize the mother to avoid end-organ damage and depending on preexisting conditions and gestational age, assess for delivery of the fetus. Avoidance of relative or actual hypotension is mandatory in these women as this may affect the uterine blood flow and the fetus and complicate management in a fetus with nonreassuring heart rate tracing.

Nonpregnant patients have higher BP tolerance and patients presenting with hypertensive emergencies with BP > 200/120 may be at increased risk of myocardial infarction, stroke, hypertensive encephalopathy, development of dissecting aneurisms, acute renal failure, and pulmonary edema.[11,15] During pregnancy, patients may develop hypertensive emergencies at lower levels of HTN when compared with their nonpregnant counterparts and have increased risk of developing the aforementioned complications. Parturient have an increased risk of presenting with pulmonary edema during a hypertensive emergency due to an increase in hydrostatic pressure from increased effective blood volume and decreased colloid osmotic pressure.[16,17] During pregnancy, severe systolic HTN (≥160 mm Hg) has been linked to a higher risk of stroke and death.[3]

During hypertensive emergencies acute kidney damage may occur. Most hypertensive emergencies are associated with an acute elevation of the systemic vascular resistances resulting in endothelial damage. Such injury activates platelets and

coagulation leading to small vessel occlusion and end-organ hypoperfusion injury (including the kidney). In patients with preeclampsia initial glomerular damage usually manifests as proteinuria, which can worsen with progression of the disease. These patients may experience dysfunction of the glomerular apparatus in the kidney with decreased filtration rate and renal insufficiency increasing the risks of pulmonary edema.[18] In addition, patients may develop severe renal artery vasospasm that may contribute to the development of oliguria.

A hypertensive crisis during pregnancy can cause acute disruption of uterine artery blood flow to the placenta and jeopardize the fetus. These fetuses may already have a limited reserve due to preexisting chronic HTN or preeclampsia that have affected normal placental development and perfusion. These changes may lead to restricted fetal growth and oligohydramnios and may ultimately cause alterations in umbilical artery perfusion in the fetus.

Patients are also at risk of developing eclampsia. Furthermore, they have a higher incidence of intracranial hemorrhage, stroke, and subcortical edema. Preeclamptic women with severe features and hypertensive crisis can develop cortical and subcortical hemorrhages in the occipital lobe, small cortical infarctions, and subcortical edema that can subsequently be associated to posterior reversible encephalopathy syndrome (PRES). PRES develops because of failure of cerebral autoregulation (mainly in the posterior vertebra basilar circulation) and disruption of the blood brain barrier leading to vasogenic edema primarily in the parietal and occipital lobes. These changes may persist for several weeks after delivery and resolution of the hypertensive emergency.[19–21]

Patients with a hypertensive crisis require an immediate complete neurological examination. When lateralization is found on the clinical examination, immediate neuroimaging is required with computed tomography being more readily available and better for visualizing intracranial hemorrhages and magnetic resonance imaging been a better choice if PRES is suspected. Magnetic resonance imaging is more sensitive to detect early ischemic changes allowing a differential diagnosis between vasogenic (hydrostatic) and cytotoxic (ischemic) edema. Blurred vision, amaurosis, and scotoma may be present with hypertensive emergencies and may be due to either occipital involvement or due to retinal detachment. Fundoscopic visualization may be necessary to make the differential diagnosis.

Patients with hypertensive emergencies require prompt maternal stabilization. If it is diagnosed in an outpatient setting, patients need to be transferred to a hospital as soon as possible for treatment. If transfer to a tertiary center is required (in case of prematurity and severe HTN), initial stabilization is prudent before transfer.[3] Monitoring is required for a viable fetus. Once treatment has been started to stabilize the patient and lower the BP, the clinician needs to make decisions about the management of pregnancy depending in part on the gestational age and the cause of the hypertensive emergency. After 34 weeks delivery of the fetus after stabilizing the mother may be indicated. Before 34 weeks, maternal stabilization should be attempted at institutions where maternal and neonatal intensive care resources are available.

TREATMENT ALTERNATIVES

A recent Chochrane review published in 2013 by Duley et al[12] compared the use of hydralazine, labetalol, or calcium channel blockers in preeclampsia. They found no major difference between the 3 medications and recommended each one be started based on patient individual characteristics and clinical scenario, adverse effects, and clinicians own experience.

Once the hypertensive emergency is diagnosed, treatment with intravenous (IV) labetalol, IV hydralazine, or oral (PO) nifedipine should be started in the hospital setting (Table 1). Fetal monitoring should be started when the patient is admitted if the fetus is viable. Noninvasive BP with an adequately fitting cuff may be used but should be cycled frequently (every 5 min or more frequently) until the patient is stabilized. Invasive BP monitoring (arterial line) is indicated for unstable patients requiring continuous infusions of these medications. Invasive BP monitoring is also indicated in conditions in which noninvasive monitoring is unreliable such as morbid obesity.

Despite theoretical concerns, there is no clinical evidence that combining magnesium sulfate with calcium channel blockers, such as nifedipine or nicardipine increases the risk of respiratory arrest.

PHARMACOLOGIC MANAGEMENT OF HYPERTENSIVE CRISIS

When patients present with a hypertensive crisis the goal of treatment is to rapidly lower the BP to a level that decreases the risk of CHF and myocardial ischemia, renal injury, and ischemic or hemorrhagic stroke. There are no data as to the exact safe range of BP that needs to be achieved, but clinicians should target a BP < 160/110 in most women during pregnancy. First-line medications have classically been hydralazine, labetalol, and oral nifedipine (Table 3).

Labetalol is a competitive antagonist with selective α-1 sympathetic receptor blockade and nonselective β-receptor blockade. Labetalol inhibits neuronal uptake of norepinephrine and has some direct vasodilating ability. It is metabolized in the liver and its clearance is affected by hepatic perfusion. Its onset of action is around 5 to 10 minutes with a peak effect at around 10 to 20 minutes. Serum half-life is 6 hours. Dosing during pregnancy, is usually an initial 10 to 20 mg IV bolus over 2 minutes; the bolus can be repeated every 20 minutes, increasing the bolus dose depending upon the response to the prior dose to up to 80 mg per bolus with a maximum total IV dose of 300 mg.[8] Maximum BP response occurs within 5 minutes of each bolus. Labetalol can also be given as a constant infusion of 1 to 2 mg/min when control of the BP is not achieved by bolus treatment, but consideration of invasive monitoring before the starting of the infusion is recommended. A major advantage of labetalol is that it decreases BP through vasodilation (from the α-blockade) without accompanying tachycardia thanks to the β-blockade effect. This agent is contraindicated in asthma, bradycardia, heart blocks, and in acute CHF. It can be given in patients with aortic dissection as monotherapy and is the only β-blocker not contraindicated in patients with hypertensive crisis and recent cocaine use.

Hydralazine is a direct arteriolar vasodilator that directly affects the smooth muscle in precapillary resistance vessels with minimal effect on postcapillary venous capacitance vessels resulting in decreased systemic vascular resistances. It has a slower onset of action (as compared with labetalol) and is metabolized by hydroxylation and glucuronidation in the liver. Approximately 15% of the drug is excreted unchanged by the kidneys. Hydralazine has been associated with increased risk of maternal hypotension and oliguria.[22] The latter side effects are mainly seen when administered to patients that are hypovolemic (eg, preeclampsia). Other side effects include nausea, headache, dizziness, sweating and tremors, and reflex tachycardia. Onset of action occurs between 5 and 30 minutes after administration and may last for up to 2 to 6 hours when given IV. Commonly the drug is administered as boluses of 2.5 to 5 mg IV every 20 minutes. No maximum dose has been clearly described. Hydralazine may cause cerebral vasodilation worsening cerebral edema

TABLE 3. Drugs Used to Treat Hypertensive Emergencies During Pregnancy

	Labetalol IV	**Hydralazine IV/IM**	**Nifedipine PO**
Mechanism of action	Sympathetic α-1 and β-blocker	Direct vasodilator	Calcium channel blocker
Onset	Quick (within 5 min)	Gradual (around 20 min)	Gradual
Dose	10-20 mg IV over 2 min. May repeat bolus in escalating doses up to 80 mg per bolus every 20 min (maximum 300 mg) OR 1-2 mg/min infusion—titrate by increasing 1 mg/min every 10 min	2.5-5 mg IV slowly Repeat 5-10 mg IV every 20-40 min.	10 mg PO every 20 min. May increase to 20 mg per dose Maximum 50 mg
Effect on heart rate	Less tachycardia	Compensatory tachycardia is frequent	Reflex tachycardia is frequent
Side effects/ considerations	May be first-line treatment	Higher doses associated with maternal hypotension, headache and decreased placental perfusion with fetal heart rate abnormalities. May produce Lupus like syndrome. Adjust dosing in renal impairment.	Maternal headache, dizziness, flushing, lightheadedness, and nausea/heartburn
Contraindication	Asthma- may block effects of bronchodilators during asthma attack Heart disease and bradycardia or advanced AV block CHF, cardiogenic shock or decompensated systolic CHF Care if given in pheochromocytoma	Mitral valve rheumatic heart disease due to increase in pulmonary artery pressure Coronary artery disease—tachycardia may cause MI Caution in patients with stroke	Aortic stenosis, coronary artery disease; severe hypotension, acute MI; cardiogenic shock
Pregnancy category	C	C	C

AV indicates atrioventricular block; CHF, congestive heart failure; IM, intramuscular; IV, intravenous; MI, myocardial infarction; PO, by mouth. Data derived from: American College of Obstetricians and Gynecologists Taskforce on Hypertension in Pregnancy. Hypertension in pregnancy.[8,11]

in patients with elevated intracranial pressure.

It is recommended that whether treatment started with labetalol or hydralazine, the clinician should consider switching medications if HTN is uncontrolled after administering 3 doses of the initial medication provided sufficient time has passed since last dose.

Nifedipine is a calcium channel blocker (dihydropyridine) and can only be administered orally. In obstetrics, it has a major role as a tocolytic agent but has also been used during hypertensive emergencies. In nonobstetric patients, its use in hypertensive emergencies has been discouraged due to tachycardia that accompanies the drop in BP, but this has not been considered a contraindication in pregnant women without heart disease. The dose for hypertensive emergencies is usually 10 mg PO and can be repeated

every 30 minutes for up to 3 doses. Once BP is controlled it may be scheduled every 2 to 6 hours. It is a renal artery vasodilator and also acts as a natriuretic. Most frequent side effects include flushing, peripheral edema, dizziness, lightheadedness, headache, and nausea.

Second-line agents include the use of nicardipine,[12,23] nitroglycerine, and sodium nitroprusside. *Nicardipine* is a parenterally administered second-generation dihydropyridine derivative calcium channel blocker with an onset of action within 5 to 15 minutes and a duration of action between 4 and 6 hours. Its main site of action is the smooth muscle on blood vessels decreasing the peripheral vascular resistance with minimal negative inotropic effect. The latter makes it a good option in patients with systolic dysfunction. Nicardipine also decreases cardiac ischemia by dilating coronary arteries and increases stroke volume.[24] It is contraindicated in severe aortic stenosis. The initial infusion rate is 2.5 to 5 mg/h with slow titration of 2.5 mg/h every 5 minutes titrated to desired effect up to a maximum dose of 15 mg/h. Side effects are flushing, headache, and reflex tachycardia. Nicardipine could theoretically decrease uterine tone and predispose to postpartum hemorrhage secondary to uterine atony.

FETAL ASSESSMENT

When patients present with a hypertensive crisis during pregnancy, gestational age is an important factor in determining treatment and monitoring strategies. After fetal viability is reached, continuous monitoring during treatment of maternal hypertensive crisis is mandatory.

HTN Crisis With Preeclampsia

When the hypertensive crisis is accompanied with preeclampsia and the patient presents with end-organ damage such as pulmonary edema, renal failure, placental abruption, severe thrombocytopenia, disseminated intravascular coagulation, neurological symptoms, or nonreassuring fetal tracings, irrespective of gestational age, prompt delivery of the fetus should be considered. Maternal stabilization should be attempted before delivery of fetus.

If the hypertensive crisis does not present with end-organ damage and BP improves with treatment, delivery of the fetus is determined by gestational age. If the fetus is at or beyond 34 weeks' gestation, delivery may be recommended to avoid continuous and progressive deterioration of both mother and fetus. If the gestational age is < 34 weeks, expectant management may be warranted until 34 weeks' gestational age or fetal maturation benefit with corticosteroids unless the condition worsens.[8]

HTN Crisis With Chronic HTN

Patients with chronic HTN presenting with a hypertensive crisis require prompt treatment to avoid end-organ damage. Treatment should be started in the hospital setting, and both fetus and mother require close monitoring for deteriorating signs. Surveillance of fetal growth and Doppler velocimetry is fundamental to evaluate fetal status during pregnancy.[25] In patients with hypertensive crisis and a gestational age < 34 weeks, the use of corticosteroids for fetal lung maturity should be considered. If patients with chronic HTN present with hypertensive crisis and additionally have superimposed preeclampsia with severe features, it is recommended to use intrapartum and postpartum parenteral magnesium sulfate to prevent the development of eclamptic seizures. Delivery of the fetus in these patients should be considered depending on gestational age and managed in the same algorithm as patients with preeclampsia with severe features.

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