Hypertensive Emergency with pregnancy

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Nothing baffles the researchers more than hypertensive disorders of pregnancy

It is probably the most studied and least understood problem of obstetrics

**OBJECTIVES**

- Defining HTN crisis
- Diagnostic Criteria
- End Organ Consequences
- Medical Management
INTRODUCTION

• Hypertensive disorders of pregnancy are responsible for significant *maternal and perinatal morbidity*

• Complicate *10% to 15%* of all pregnancies

• *17% of maternal deaths* in India

HYPERTENSIVE DISORDERS IN PREGNANCY

• Important cause of "BAD OBSTETRIC OUTCOME"

• *Multisystem* Involvement

• Requiring *close monitoring* of Mother & Fetus

• *Hospital Management* preferred in severe cases

• Prognosis has *improved with advances* in management and team approach
• **Overview**

  - **Hypertension** is the most common medical problem encountered during pregnancy, complicating up to 10% of pregnancies. Hypertensive disorders during pregnancy are classified into 4 categories, as recommended by the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy:

    - **Chronic hypertension**
    - **Preeclampsia-eclampsia**
    - **Preeclampsia superimposed on chronic hypertension**
    - **Gestational hypertension** (transient hypertension of pregnancy or chronic hypertension identified in the latter half of pregnancy).

This terminology is preferred over the older but widely used term "pregnancy-induced hypertension" (PIH) because it is more precise.
When urgent treatment is needed before the establishment of IV access, the oral nifedipine algorithm can be initiated as IV access is being obtained, or a 200-mg dose of labetalol can be administered orally. The latter can be repeated in 30 minutes if appropriate improvement is not observed.

Magnesium sulfate is not recommended as an antihypertensive agent, but magnesium sulfate remains the drug of choice for seizure prophylaxis in severe preeclampsia and for controlling seizures in eclampsia.

Sodium nitroprusside should be reserved for extreme emergencies and used for the shortest amount of time possible because of concerns about cyanide and thiocyanate toxicity in the mother and fetus or newborn, and increased intracranial pressure with potential worsening of cerebral edema in the mother.

There is a need for adoption of standardized, evidence-based clinical guidelines for managing patients with preeclampsia. Individuals and institutions should have mechanisms in place to initiate the prompt administration of medication when a patient presents with a hypertensive emergency.
• 38y pregnant female at 28W of gestation and during routin followup by her obstetrian BLP 170/110 ,she had sever headach ,blurred vision,Epigastric pain , edeoma at face and her hands , with significant proinurea , what's your diagnosis and management???

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**Chronic hypertension**

**Definitions**
- Chronic hypertension is defined as blood pressure exceeding 140/90 mm Hg before pregnancy or before 20 weeks' gestation. When hypertension is first identified during a woman's pregnancy and she is at less than 20 weeks' gestation, blood pressure elevations usually represent chronic hypertension.
- In contrast, new onset of elevated blood pressure readings after 20 weeks' gestation mandates the consideration and exclusion of [preeclampsia](https://www.ncbi.nlm.nih.gov/pubmed/28458284).
  Preeclampsia occurs in 3-6% of all pregnancies and the incidence is 1.5 to 2 times higher in first time pregnancies. Hypertensive disorders in pregnancy may cause maternal and fetal morbidity, and they remain a leading source of maternal mortality.
Chronic Hypertension

Chronic hypertension is a primary disorder in 90-95% of cases and may be either essential (90%) or secondary to some identifiable underlying disorder, such as renal parenchymal disease (eg, polycystic kidneys, glomerular or interstitial disease), renal vascular disease (eg, renal artery stenosis, fibromuscular dysplasia), endocrine disorders (eg, adrenocorticoctosteroid or mineralocorticoid excess, pheochromocytoma, hyperthyroidism or hypothyroidism, growth hormone excess, hyperparathyroidism), coarctation of the aorta, or oral contraceptive use. About 20-25% of women with chronic hypertension develop preeclampsia during pregnancy. [8]

Chronic hypertension occurs in up to 22% of women of childbearing age, with the prevalence varying according to age, race, and body mass index (BMI). Population-based data indicate that approximately 1% of pregnancies are complicated by chronic hypertension, 5-6% by gestational hypertension (without proteinuria), and 3-6% by preeclampsia. [9, 7]

Preeclampsia

Preeclampsia

Although the exact pathophysiologic mechanism is not clearly understood, preeclampsia is primarily a disorder of placental dysfunction leading to a syndrome of endothelial dysfunction with associated vasospasm. In most cases, pathology evaluation demonstrates evidence of placental insufficiency with associated abnormalities such as diffuse placental thrombosis, an inflammatory placental decidual vasculopathy, and/or abnormal trophoblastic invasion of the endometrium. These findings support abnormal placental development or placental damage from diffuse microthrombosis as being central to the development of this disorder. There is also evidence to indicate an altered maternal immune response to fetal/placental tissue may contribute to the development of preeclampsia.
The widespread endothelial dysfunction may manifest as a **maternal syndrome**, **fetal syndrome**, or both. The pregnant woman may manifest dysfunction of multiple organ systems, including the central nervous, hepatic, pulmonary, renal, and hematologic systems. Endothelial damage leads to **pathologic capillary leak** that can present in the mother as **rapid weight gain**, **nondependent edema** (face or hands), **pulmonary edema**, **hemoconcentration**, or a combination thereof. The diseased placenta can also affect the fetus via **decreased uteroplacental blood flow**. This decrease in perfusion can manifest clinically as **nonreassuring fetal heart rate testing**, **low scores on a biophysical profile**, **oligohydramnios**, or as **fetal growth restriction**.
• in contrast, women who develop preeclampsia typically show a hyperresponsiveness to these hormones, an alteration that may be seen even before the hypertension and other manifestations of preeclampsia become apparent. In addition, blood pressures in preeclampsia are labile, and the normal circadian blood pressure rhythms may be blunted or reversed. One study found increased arterial stiffness in women with preeclampsia, as well as in those with gestational hypertension, compared with normotensive controls; treatment with alpha methyldopa significantly improved the vascular stiffness in preeclampsia but did not normalize it. [10]

• Risk factors
  • Preeclampsia is more common at the extremes of maternal age (< 18 y or >35 y). The increased prevalence of chronic hypertension and other comorbid medical illnesses in women older than 35 years may explain the increased frequency of preeclampsia among older gravidas. In addition, black women have higher rates of preeclampsia complicating their pregnancies compared with other racial groups, mainly because they have a greater prevalence of underlying chronic hypertension. Among women aged 30-39 years, chronic hypertension is present in 22.3% of black persons, 4.6% of non-Hispanic white persons, and 6.2% of Mexican Americans. Hispanic women generally have blood pressure levels that are the same as or lower than those of non-Hispanic white women.
  • Women who develop preeclampsia during pregnancy have an increased risk of recurrent preeclampsia during subsequent pregnancies. The overall risk is about 18%. The risk is higher (50%) in women who develop severe early preeclampsia (ie, before 27 weeks' gestation). These women are also at increased risk for cardiovascular disease later in life. Whether the preeclampsia increases cardiovascular risk or the 2 conditions share a common underlying cause remains unclear. [11]
• **Maternal personal risk factors for preeclampsia**
  • The following are maternal personal risk factors for preeclampsia:
    • First pregnancy
    • New partner/paternity
    • Age younger than 18 years or older than 35 years
    • History of preeclampsia
    • Family history of preeclampsia in a first-degree relative
    • Black race
    • Obesity (BMI ≥30)
    • Interpregnancy interval less than 2 years or longer than 10 years

• **Maternal medical risk factors for preeclampsia**
  • The following are maternal medical risk factors for preeclampsia:
    • Chronic hypertension, especially when secondary to such disorders as hypercortisolism, hyperaldosteronism, pheochromocytoma, or renal artery stenosis
    • Preexisting diabetes (type 1 or type 2), especially with microvascular disease
    • Renal disease
    • Systemic lupus erythematosus
    • Obesity
    • Thrombophilia
    • History of migraine\[^{12}\]
    • Use of selective serotonin uptake inhibitor antidepressants (SSRIs) beyond the first trimester\[^{13}\]
• Placental/fetal risk factors for preeclampsia
• The following are placental/fetal risk factors for preeclampsia:
  • Multiple gestations
  • **Hydrops fetalis**
  • Gestational trophoblastic disease
  • Triploidy

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>OR or RR (95% CI)</th>
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<tr>
<td>Antiphospholipid antibody syndrome</td>
<td>9.7 (4.3–21.7)</td>
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<tr>
<td>Renal disease</td>
<td>7.8 (2.2–28.2)</td>
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<tr>
<td>Prior pre-eclampsia</td>
<td>7.2 (5.8–8.8)</td>
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<tr>
<td>Systemic lupus erythematosus</td>
<td>5.7 (2.0–16.2)</td>
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<tr>
<td>Nulliparity</td>
<td>5.4 (2.8–10.3)</td>
</tr>
<tr>
<td>Chronic hypertension</td>
<td>3.8 (3.4–4.3)</td>
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<tr>
<td>Diabetes mellitus</td>
<td>3.6 (2.5–5.0)</td>
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<tr>
<td>High altitude</td>
<td>3.6 (1.1–11.9)</td>
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<tr>
<td>Multiple gestations</td>
<td>3.5 (3.0–4.2)</td>
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<tr>
<td>Strong family history of CV disease (heart disease or stroke in ≥2 first-degree relatives)</td>
<td>3.2 (1.4–7.7)</td>
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<tr>
<td>Obesity</td>
<td>2.5 (1.7–3.7)</td>
</tr>
<tr>
<td>Family history of pre-eclampsia in first-degree relative</td>
<td>2.3–2.6 (1.8–3.6)</td>
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<tr>
<td>Advanced maternal age (&gt;40 years)</td>
<td>1.68 (1.23–2.29) for nulliparas</td>
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<td></td>
<td>1.96 (1.34–2.87) for multiparas</td>
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Abbreviations: CI, confidence interval; OR, odds ratio; RR, relative risk; CV, cardiovascular.
Danger of hypertension with pregnancy

• **Hypertensive disorders in pregnancy are among the leading causes of maternal mortality**, along with thromboembolism, hemorrhage and nonobstetric injuries. Between 2011 and 2013, pregnancy-induced **hypertension caused 7.4% of maternal deaths in the United States**. Furthermore, hypertension before pregnancy or during early pregnancy is associated with a twofold increased risk of **gestational diabetes mellitus**. Transient hypertension of pregnancy (ie, the development of isolated hypertension in a woman in late pregnancy without other manifestations of preeclampsia) is associated strongly with later development of chronic hypertension.

• Although maternal diastolic blood pressure (DBP) greater than 110 mm Hg is associated with an increased risk for **placental abruption** and fetal growth restriction, superimposed preeclamptic disorders cause most of the morbidity due to chronic hypertension during pregnancy (see Complications).

• **Next: Evaluation**

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**Evaluation**

• Determining whether elevated blood pressure identified during pregnancy is due to chronic hypertension or to preeclampsia is sometimes a challenge, especially if no recorded blood pressures from the first half of the gestation are available. Clinical characteristics obtained via history, physical examination, and certain laboratory investigations may be used to help clarify the diagnosis. See Introduction for important maternal and fetal risk factors for preeclampsia that are essential to the history.
• **Symptoms of preeclampsia**

Symptoms of preeclampsia may include visual disturbances, typically scintillations and scotomata, presumed to be due to cerebral vasospasm. The woman may describe new-onset headache that is frontal, throbbing, or similar to a migraine headache, and gastrointestinal complaints of sudden, new-onset, constant epigastric pain that may be moderate to severe in intensity and due to hepatic swelling and inflammation, with stretch of the liver capsule.

Although mild lower extremity edema is common in normal pregnancy, descriptions of rapidly increasing or nondependent edema may be a signal of developing preeclampsia; note, however, that edema is no longer included among the criteria for the diagnosis of preeclampsia. In addition, rapid weight gain is a result of edema due to capillary leak as well as renal sodium and fluid retention.

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**Danger Signs Of Severe Preeclampsia.**

- Headache
- Upper quadrant abdominal pain
- Visual disturbance
- Decreased urine output
- History of convulsion(s)
- Respiratory symptoms that might indicate pulmonary edema (dyspnea, chest pain, cough)
- Nausea/vomiting
- Blindness
• **Cardiovascular findings in preeclampsia**
  • Home and ambulatory blood pressure measurements are increasingly being used in the pregnant population. Assuming the blood pressure device is accurate (validated relative to an office measurement), the measurements may provide valuable additional data regarding hypertension severity and control during pregnancy.
  • In most normal pregnancies, the woman has some lower extremity edema by the third trimester. In contrast, a sudden worsening in dependent edema, edema in nondependent areas (such as the face and hands), or rapid weight gain suggest a pathologic process and warrant further evaluation for preeclampsia. Preeclampsia is a multisystem disease with various physical signs.

### SEVERITY OF PREECLAMPSIA

<table>
<thead>
<tr>
<th>Mild</th>
<th>Severe</th>
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<tr>
<td>• Systolic blood pressure between 140 and 160 mm Hg</td>
<td>• Systolic blood pressure &gt;160 mm Hg</td>
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<tr>
<td>• Diastolic blood pressure between 90 and 110 mm Hg</td>
<td>• Diastolic blood pressure &gt; 110 mm Hg</td>
</tr>
<tr>
<td>• Proteinuria between 3 and 5 gm on a 24-hour sample</td>
<td>• Proteinuria &gt; 5 gm on a 24-hour sample</td>
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</table>
• Women should be allowed to sit *quietly for 5-10 minutes before each blood pressure measurement*. Blood pressure should be measured in the sitting position, with the cuff at the level of the heart. Inferior vena caval compression by the gravid uterus while the patient is supine can alter readings substantially, leading to an underestimation of the blood pressure. Similarly, blood pressures measured in the left lateral position may yield falsely low values if the blood pressure is measured in the higher arm, unless the cuff is carefully maintained at the level of the heart.

• Korotkoff sounds I (the first sound) and V (the disappearance of sound) should be used to denote the systolic blood pressure (SBP) and DBP, respectively. In about 5% of women, an exaggerated gap exists between the fourth (muffling) and fifth (disappearance) Korotkoff sounds, with the fifth sound approaching zero. In this setting, both the fourth and fifth sounds should be recorded (e.g., 120/80/40, with sound I = 120, sound IV = 80, and sound V = 40), because the fourth sound will more closely approximate the true DBP.

• Any automated blood pressure cuffs provide reasonable estimates of true blood pressure during normal pregnancy (especially those validated for pregnancy) but tend to underestimate blood pressure in preeclamptic women. Only a few automated blood pressure cuffs have been validated in preeclampsia. Manual blood pressure measurement with a mercury sphygmomanometer remains the criterion standard in this setting.

• Maternal SBP greater than 160 mm Hg or DBP greater than 110 mm Hg denotes severe disease; depending on the gestational age and maternal status, delivery should be considered for sustained pressures in this range.
• **Diagnostic Overview**
  • Determining whether elevated blood pressure identified during pregnancy is due to chronic hypertension or to preeclampsia is sometimes a challenge, especially if no recorded blood pressures from the first half of the gestation are available. Clinical characteristics obtained via history, physical examination, and certain laboratory investigations may be used to help clarify the diagnosis. Fetal well-being must also be considered with the workup of the mother.
  • Preeclampsia is rare before the third trimester, and the diagnosis of severe hypertension or preeclampsia in the first or early second trimester necessitates exclusion of *gestational trophoblastic disease* and/or molar pregnancy. Mild lower extremity edema is common in normal pregnancy, but rapidly increasing or nondependent edema may be a signal of developing preeclampsia. However, edema is no longer included among the criteria for the diagnosis of preeclampsia.
  • New seizures in pregnancy suggest preeclampsia-eclampsia, but primary neurologic disorders must be excluded.

• **Routine Tests**
  • Laboratory testing to evaluate chronic hypertension (if not done previously or recently) includes testing for target organ damage, potential secondary causes of hypertension, and other risk factors.
  • Blood tests to order when evaluating eclampsia include those suggested to evaluate for preeclampsia. Such studies include: urinalysis; complete blood cell (CBC) count; and serum sodium, potassium, creatinine, and glucose levels (the presence of high levels of progesterone, an aldosterone antagonist, during a normal pregnancy may mask the hypokalemia from hyperaldosteronism).
  • Other suggested tests include measurements of creatinine clearance, blood urea nitrogen (BUN), albumin, 24-hour urinary protein, serum calcium, uric acid, glycosylated hemoglobin, thyroid-stimulating hormone (TSH), liver enzymes and bilirubin, and a urine dip for protein.
• Renal and Hepatic Evaluation

• Kidneys
  • Urinalysis may be used as a screen for proteinuria. Trace levels to +1 proteinuria are acceptable, but levels of +2 or greater are abnormal and should be quantified with a 24-hour urine collection or spot urine protein-creatinine ratio.
  • In a 24-hour urine collection, the reference range for protein excretion in pregnancy is up to 300 mg/d. Higher levels are abnormal and may reflect renal involvement in preeclampsia. Creatinine clearance increases approximately 50% during pregnancy, and levels less than 100 mL/min suggest renal dysfunction that is either chronic or due to preeclampsia.
  • Serum creatinine is usually less than 0.8 mg/dL during pregnancy; higher levels suggest intravascular volume contraction or renal involvement in preeclampsia. A serum uric acid level greater than 5 mg/dL is abnormal; this is a sensitive but nonspecific marker of tubular dysfunction in preeclampsia.

• Liver
  • Elevated levels of hepatic transaminases may reflect hepatic involvement in preeclampsia; these increased values may occur in the absence of epigastric/RUQ pain.
• **Magnetic Resonance Studies of the Brain**
  • **MRI**
  • An MRI may be performed to evaluate for abnormalities of the cerebral cortex (ie, edema, infarction, hemorrhage) in preeclamptic women with severe visual disturbance, seizures, or altered mental status, as seen in the image below. An MRI is more sensitive than a CT scan for detecting cerebral cortical abnormalities but less useful in detecting cerebral hemorrhage.

• **Ultrasonography**
  • Ultrasonography or CT scanning of the liver may be used to evaluate for subcapsular hemorrhage or infarction in the setting of persistent severe RUQ pain or markedly elevated hepatic transaminases.
• **Echocardiography and Electrocardiography**

  Limited echocardiography may be performed to evaluate for LVH in chronic hypertension and to exclude cardiomyopathy or occult valvular disease in pregnant women with pulmonary edema.

  Perform a 12-lead electrocardiogram (ECG) to evaluate for LVH in women with chronic hypertension.

• **Electroencephalography**

  An electroencephalogram (EEG) may be indicated to evaluate recurrent seizure activity, persistent altered level of consciousness, or altered mental status. Following eclampsia, the EEG may reveal epileptiform activity. More commonly, the test shows nonspecific diffuse slowing that may persist for several weeks after delivery.
• **Ophthalmologic findings in preeclampsia**
  • Retinal vasospasm is a severe manifestation of maternal disease; consider delivery. In addition, retinal edema is known as serous retinal detachment. This can manifest as severely impaired vision if the macula is involved. It generally reflects severe preeclampsia and should also lead to prompt consideration of delivery. The condition typically resolves upon completion of pregnancy and resolution of the hypertension and fluid retention.

• **Fetal Monitoring**
  • Close fetal monitoring under the direction of an obstetrician is essential in pregnant women with preeclampsia. Preeclampsia is a disease of the placenta. When the placenta is severely affected, subtle hypoperfusion of the fetus can occur, which may initially manifest as a decrease in the amniotic fluid level (oligohydramnios), fetal growth restriction, and intrauterine fetal death as a consequence of placental insufficiency. If these problems with the fetus occur, they may be an indication for delivery. Monitoring usually consists of monthly ultrasounds to assess fetal growth after viability and fetal surveillance by biophysical profile weekly or non-stress test twice weekly.
Gestational hypertension

- **Gestational Hypertension**
  - Gestational hypertension refers to hypertension with onset in the latter part of pregnancy (>20 weeks' gestation) without any other features of preeclampsia, and followed by normalization of the blood pressure postpartum. Of women who initially present with apparent gestational hypertension, about one third develops the syndrome of preeclampsia. As such, these patients should be observed carefully for this progression. The pathophysiology of gestational hypertension is unknown, but in the absence of features of preeclampsia, the maternal and fetal outcomes are usually normal. Gestational hypertension may, however, be a harbinger of chronic hypertension later in life.

- **Preeclampsia/Gestational Hypertension Screening**
  - Researchers in the United Kingdom have developed a method for first-trimester screening to identify women at risk for the development of preeclampsia or gestational hypertension. The screening algorithm uses a combination of maternal variables, including mean arterial pressure, uterine artery pulsatility index, pregnancy-associated plasma protein-A, and placental growth factor. The algorithm proved especially effective for predicting early preeclampsia (ie, requiring delivery before 34 weeks). Screening strategies for preeclampsia other than evaluating for risk factors through a detailed medical history are not currently recommended by ACOG or SMFM.
  - A cohort study that covered over 13,000 pregnancies by Egeland et al found that gestational hypertension and preeclampsia share several preconception risk factors, some of which could be modified to reduce the risk of adverse pregnancy outcomes. The baseline risk factors shared among gestational hypertension and preeclampsia were: family history of diabetes mellitus, a women's own diabetes status prior to conception, a high total cholesterol/high-density lipoprotein cholesterol ratio (>5), overweight and obesity, and elevated blood pressure status. For preeclampsia but not gestational hypertension, a family history of myocardial infarction before 60 years of age and elevated triglyceride levels also predicted risk.
  - Next: Hematologic Evaluation
BASIC MANAGEMENT OBJECTIVES

- Termination of pregnancy with least possible trauma to the mother and the fetus
- Birth of an infant who subsequently thrives
- Complete restoration of health to the mother

GOALS OF TREATMENT

Within 1-2 hours
Reduce MAP 20-25%
Controlled environment
Use IV medications

- This is a special situation when we need to decrease B. P. quickly and effectively

- A single drug is better in small intermittent doses, combinations should be avoided as they may have compound side effects
• Medical Therapy
• Treatment of Acute Hypertension in Pregnancy
• Acute severe hypertension in pregnancy is a medical emergency requiring treatment to lower blood pressures within 30 minutes of confirmation to reduce risk of maternal stroke. According to the February 2015 ACOG Committee Opinion #623 “Emergent Therapy for Acute-Onset, Severe Hypertension During Pregnancy and the Post-Partum Period,” first line options for treatment include oral immediate-release nifedipine, IV labetalol, and IV hydralazine. [4]

• In 2015 and 2017, the American College of Obstetricians and Gynecologists Committee on Obstetric Practice issued updated guidelines regarding the emergency treatment of acute onset severe hypertension during pregnancy, including the following [4, 5, 6]:
  • Acute-onset, severe hypertension that is accurately measured using standard techniques and is persistent for 15 minutes or longer is considered a hypertensive emergency.
  • Intravenous (IV) labetalol and hydralazine have long been considered first-line medications for the management of acute-onset, severe hypertension in pregnant women and women in the postpartum period. Available evidence suggests that oral nifedipine also may be considered as a first-line therapy.
  • Parenteral labetalol should be avoided in women with asthma, heart disease, or congestive heart failure.
• When urgent treatment is needed before the establishment of IV access, the oral nifedipine algorithm can be initiated as IV access is being obtained, or a 200-mg dose of labetalol can be administered orally. The latter can be repeated in 30 minutes if appropriate improvement is not observed.

• **Magnesium sulfate is not recommended as an antihypertensive agent**, but magnesium sulfate remains the drug of choice for seizure prophylaxis in severe preeclampsia and for controlling seizures in eclampsia.

• **Sodium nitroprusside** should be reserved for extreme emergencies and used for the shortest amount of time possible because of concerns about cyanide and thiocyanate toxicity in the mother and fetus or newborn, and increased intracranial pressure with potential worsening of cerebral edema in the mother.

• There is a need for adoption of standardized, evidence-based clinical guidelines for managing patients with preeclampsia. Individuals and institutions should have mechanisms in place to initiate the prompt administration of medication when a patient presents with a hypertensive emergency.

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**TREATMENT OF ACUTE SEVERE HYPERTENSION DURING PREGNANCY**

• Parenteral hydralazine is most commonly used

• Parenteral labetalol is second-line drug (avoid in women with asthma and CHF)

• Oral nifedipine used with caution. (Short-acting nifedipine is not approved by FDA for managing hypertension)
• **Bedrest and hospitalization**
  • Women with worsening hypertension during pregnancy often are placed on bed rest or restricted activity, although no scientific evidence demonstrates that this is beneficial in prolonging gestation or reducing maternal or fetal morbidity/mortality.
  • Women with hypertension and suspected preeclampsia are typically admitted to a hospital for close observation and investigation. Those with established preeclampsia must be observed very closely, either in hospital or in a comprehensive home monitoring program under the care of an obstetrician.
• Women with preeclampsia remote from term (ie, < 34-36 weeks’ gestation) should be promptly transferred to a facility with adequate resources to care for premature newborn infants. This is essential because worsening preeclampsia disease activity may require urgent delivery at any time.

• When preeclampsia develops remote from term (ie, < 34-36 weeks’ gestation), attempts are often made to prolong the pregnancy to allow for further fetal growth and maturation. In this setting, both maternal and fetal status must be very closely monitored in a high-risk obstetric center. Fetal testing should be performed at least twice weekly, using a combination of biophysical profiles and nonstress testing supervised by an obstetrician (see Fetal Monitoring). Facilitated delivery should occur if either maternal or fetal deterioration is noted, with the mode of delivery decided by obstetric indications.

• **Chronic hypertension**

• Women with mild chronic hypertension often do not require antihypertensive therapy during most of pregnancy. Pharmacologic treatment of mild hypertension does not reduce the likelihood of developing preeclampsia later in gestation and increases the likelihood of intrauterine growth restriction. If maternal blood pressure exceeds 160/100 mm Hg, however, drug treatment is recommended.

• Three treatment options are available in cases of mild chronic hypertension in pregnancy. Antihypertensive medication may be withheld or discontinued, with subsequent close observation of blood pressure. Because blood pressure drops during normal pregnancy and no data support the use of medication in patients with blood pressures less than 160/100 mm Hg, the authors recommend this option most often.
Pharmacologic considerations

Although the primary risk of chronic hypertension in pregnancy is development of superimposed preeclampsia, no evidence suggests that pharmacologic treatment of mild hypertension reduces the incidence of preeclampsia in this population.[20] A study by Magee et al that analyzed 987 women at 14 to 34 weeks gestation to test the outcomes of less-tight vs. tight control of hypertension found that although primary-outcome rates were similar, severe hypertension (≥160/110 mm Hg) developed at a higher frequency in the less-tight control group (40.6% vs. 27.5%).[21]
• In normal pregnancy, women's mean arterial pressure drops 10-15 mm Hg over the first half of pregnancy. Most women with mild chronic hypertension (ie, SBP 140-160 mm Hg, DBP 90-100 mm Hg) have a similar decrease in blood pressures and may not require any medication during this period. Conversely, DBP greater than 110 mm Hg has been associated with an increased risk of placental abruption and intrauterine growth restriction, and SBP greater than 160 mm Hg increases the risk of maternal intracerebral hemorrhage. Therefore, pregnant patients should be started on antihypertensive therapy if the SBP is greater than 160 mm Hg or the DBP is greater than 100-105 mmHg.

• The goal of pharmacologic treatment should be a DBP of less than 100-105 mm Hg and an SBP less than 160 mm Hg. Women with preexisting end-organ damage from chronic hypertension should have a lower threshold for starting antihypertensive medication (ie, >139/89) and a lower target blood pressure (< 140/90).\(^3\)

• If a pregnant woman’s blood pressure is sustained greater than 160 mm Hg systolic and/or 110 mm Hg diastolic at any time, lowering the blood pressure quickly with rapid-acting agents is indicated for maternal safety.\(^4\) Anticonvulsant therapy may be undertaken in the setting of severe preeclampsia (primary prophylaxis) or in the setting of eclamptic seizures (secondary prophylaxis). The most effective agent is IV magnesium sulfate; phenytoin is an alternative, although less effective, therapy.
Take home massage

- Hypertension represent the commonest CV disorder with pregnancy represent 10-15% of all cases and represent 7% of total maternal mortality
- 4 common types of hypertension with pregnancy: Chronic hypertension, preeclampsia, chronic hypertension superimposed by preeclampsia and gestational hypertension
- Hypertensive Emergancy with pregnancy when BLP more than 160/110 more than 30 min and require emergant ttt esp in situation of preeclampsia due to high risk of maternal and fetal mortality
- IV Hydralizin, IV labetalol and oral Nifidiben are the mistro of management in such situation, mg sulfate and Na nitroprussid in special cases
- Management of chronic hypertension in pregnancy include alpha methyle dopa and CCB and avoid ACEs and ARBs