Gestational hypertension

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INTRODUCTION

Gestational hypertension and preeclampsia/eclampsia are hypertensive disorders induced by pregnancy; both disorders resolve postpartum. Gestational hypertension is the most common cause of hypertension in pregnant women.

This topic will discuss gestational hypertension. Other hypertensive disorders of pregnancy are reviewed separately:

- (See "Preeclampsia: Clinical features and diagnosis".)
- (See "Preeclampsia: Management and prognosis".)
- (See "Eclampsia".)
- (See "HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets)".)

DEFINITION AND PROVISIONAL DIAGNOSIS

Gestational hypertension is a clinical diagnosis defined by the new onset of hypertension (defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg) at ≥20 weeks of gestation in the absence of proteinuria or new signs of end-organ dysfunction [1]. The blood pressure readings should be documented on at least two occasions at least four hours apart; however, if blood pressure is severely elevated (systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥110 mmHg), then it is neither necessary nor desirable to wait hours before confirming and treating severe hypertension. Management of gestational hypertension with severe hypertension is identical to that for preeclampsia with severe features

and underscores the potential for serious adverse events when pregnancy-related blood pressures are severely elevated, even in the absence of proteinuria.

Final diagnosis — Gestational hypertension is a temporary (provisional) diagnosis for hypertensive pregnant women who do not meet criteria for preeclampsia (table 1) or chronic hypertension (hypertension first detected before the 20th week of pregnancy). The diagnosis is changed to:

- Preeclampsia, if proteinuria or new signs of end-organ dysfunction develop (ie, criteria for preeclampsia are met).
- Chronic hypertension, if blood pressure elevation persists ≥12 weeks postpartum. Of note, in 2017, the definition of hypertension in nonpregnant adults was revised as follows (see "Overview of hypertension in adults", section on 'Definitions'):
    - Normal blood pressure – Systolic <120 mmHg and diastolic <80 mmHg
    - Elevated blood pressure – Systolic 120 to 129 mmHg and diastolic <80 mmHg
    - Hypertension:
        - Stage 1 – Systolic 130 to 139 mmHg or diastolic 80 to 89 mmHg
        - Stage 2 – Systolic at least 140 mmHg or diastolic at least 90 mmHg
- Transient hypertension of pregnancy, if blood pressure returns to normal by 12 weeks postpartum.

An important consideration is the availability of prepregnancy blood pressures for comparison with blood pressures during pregnancy. Some women may have had undiagnosed prepregnancy hypertension, which may be in the severe range. When such women present for prenatal care early in gestation, they may have normal or mildly elevated blood pressures because of normal early pregnancy physiology. Development of isolated severe hypertension later in pregnancy may reflect return to their baseline blood pressure level rather than preeclampsia with severe features. Similarly, if they present late in gestation for prenatal care and have isolated severe hypertension, it is difficult to determine whether this reflects chronic hypertension or development of preeclampsia with severe features. Since the management and pregnancy outcome of chronic hypertension is different from the management and pregnancy outcome of preeclampsia with severe features, the distinction is clinically relevant but not always possible during pregnancy.

All patients require postpartum follow-up at 12 weeks to establish whether they had pregnancy-related hypertension or have chronic hypertension, with or without superimposed preeclampsia, as all of these diagnoses have long-term health implications. (See "Preeclampsia: Management and prognosis", section on 'Long-term maternal risks of pregnancy-associated hypertension' and "Overview of hypertension in adults".)

PREVALENCE

Gestational hypertension is the most common cause of hypertension during pregnancy. It occurs in 6 to 17 percent of healthy nulliparous women and 2 to 4 percent of multiparous women [2-4]. The prevalence is highest in women with preeclampsia in a previous pregnancy, women with multifetal gestation, and overweight/obese women [5,6].

RISK FACTORS

Risk factors are similar to those for preeclampsia (see "Preeclampsia: Clinical features and diagnosis", section on 'Risk factors'). However, epidemiologic studies report differences in the magnitude of the associations for the two disorders [7].

DIAGNOSTIC EVALUATION

The main goals in the initial evaluation of pregnant women with newly developed hypertension are to distinguish gestational hypertension from preeclampsia, which has a different course and prognosis, and to determine whether hypertension is severe, which affects management and outcome.

As in other patients with newly diagnosed hypertension, white coat hypertension (also called isolated clinic or office hypertension) should be excluded by repeating measurement of blood pressure when the patient is relaxed. Results of home blood pressure monitoring can be useful to establish the patient's blood pressure profile. If an automated device is used in the home or office, it should have been validated in a pregnant population [8]. (See "Out-of-office blood pressure measurement: Ambulatory and self-measured blood pressure monitoring".)

In addition, the following evaluation should be performed.

Measure protein excretion — Urinary protein excretion should be determined since the presence or absence of proteinuria is a key clinical criterion that determines whether the

patient will be given a diagnosis of gestational hypertension or preeclampsia. A urine dipstick of negative to trace should not be used to definitively exclude significant proteinuria since false negative results occur with low specific gravity (<1.010), high salt concentration, highly acidic urine, or with nonalbumin proteinuria. A positive urine dipstick value, especially if only +1, also requires confirmation since false positives occur. Urine protein can be quantitated using a urine protein-to-creatinine ratio ≥0.26 mg protein/mg creatinine (30 mg/mmol) on a random urine sample or with a 24-hour urine collection. We prefer the latter, as it conforms to the most widely accepted diagnostic criteria for preeclampsia. However, the urine protein-to-creatinine ratio is convenient for patients who have difficulties collecting and/or transporting a 24-hour specimen due to issues such as childcare, employment, or lack of transportation. (See "Evaluation of proteinuria in pregnancy and management of nephrotic syndrome".)

Even after this evaluation, it can be difficult to exclude preeclampsia conclusively. Studies have shown that 10 percent of women with clinical and/or histologic manifestations of preeclampsia have no proteinuria and 20 percent of women with eclampsia (a form of severe preeclampsia) do not have significant proteinuria prior to their seizure [9]. Therefore, when hypertension is accompanied by any of the signs and symptoms of end-organ dysfunction (table 1), the patient should be managed as a patient with preeclampsia, even if proteinuria is not present [1]. (See "Preeclampsia: Management and prognosis".)

Evaluate for features of severe disease — Patients should be questioned about the presence of severe features of preeclampsia, such as the new onset of cerebral or visual disturbances or epigastric or right upper quadrant pain (table 2). The chest should be auscultated to assess for pulmonary edema. The presence of any of these findings upgrades the diagnosis to preeclampsia with severe features. (See "Preeclampsia: Clinical features and diagnosis".)

Rule out other causes of acute hypertension — Acute hypertension can also be caused by medical disorders, such as pheochromocytoma and use of drugs that can produce a hyperadrenergic state, such as cocaine, amphetamine(s), and phencyclidine. A standardized interview to screen for misuse of substances should be performed; some examples are shown in the table (table 3). The Society for Maternal-Fetal Medicine suggests consideration of drug testing in patients with acute clinical complications such as unexplained severe hypertension [10]. (See "Hypertensive disorders in pregnancy: Approach to differential diagnosis".)

Perform laboratory evaluation — Laboratory evaluation helps to determine whether there is end organ involvement, which occurs with preeclampsia but not gestational hypertension. Changes consistent with preeclampsia with severe features include thrombocytopenia, increase in creatinine concentration to >1.1 mg/dL, and doubling of hepatic transaminases (table 2).

Determine the severity of hypertension — Hypertension in pregnancy is defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg. It is considered severe when systolic blood pressure is ≥160 mmHg and/or diastolic blood pressure is ≥110 mmHg. (See 'Management' below.)

Assess fetal well-being — As with all hypertensive pregnancies, fetal well-being should be assessed with a biophysical profile or nonstress test with amniotic fluid estimation. We also obtain a sonographic estimation of fetal weight; umbilical artery Doppler velocimetry is reserved for fetuses with growth restriction [5]. (See "Overview of antepartum fetal surveillance".)

RISK OF PROGRESSION TO PREECLAMPSIA

Ten to 50 percent of women initially diagnosed with gestational hypertension go on to develop preeclampsia in one to five weeks [11,12]. Progression from preeclampsia to preeclampsia with severe features may occur more rapidly, within days.

It is not clear whether gestational hypertension and preeclampsia are independent diseases with a similar phenotype (hypertension) or if gestational hypertension is an early mild stage of preeclampsia. There is consistent evidence that preeclampsia develops in a substantial proportion of women initially diagnosed with gestational hypertension, and that women who progress to preeclampsia have characteristics different from those who continue to have nonproteinuric hypertension. Clinical characteristics at presentation of gestational hypertension that predict an increased risk for progression to preeclampsia include [13]:

- Gestational age less than 34 weeks at diagnosis (sensitivity 85 percent, specificity 60 percent).
- Mean systolic blood pressure >135 mmHg on 24 hour blood pressure monitoring (sensitivity 61 percent, specificity 76 percent).
- Elevated serum uric acid level (>5.2 mg/dL [0.309 mmol/L]) (sensitivity 88 percent, specificity 93 percent) [14,15].

Abnormal uterine artery Doppler velocimetry has also been found to be predictive (sensitivity 86 percent, specificity 90 percent); however, uterine artery Doppler is not routinely performed clinically.

Other characteristics have been reported to be predictive, but are not readily measureable: elevated serum levels of anti-angiogenic markers (eg, sFlt-1), reduced levels of proangiogenic

placental growth factor, and higher total vascular resistance (>1340 dyne seconds/cm^5) [16-19].

Some data suggest preeclampsia and gestational hypertension are independent diseases. Although they share many risk factors, epidemiologic studies report differences in the magnitude of the associations for the two disorders. For example, primiparity is a stronger risk factor for preeclampsia than for gestational hypertension (odds ratio 2.2 versus 1.2) [20] and multiple gestation and diabetes mellitus are stronger risk factors for preeclampsia than gestational hypertension [7,20]. Prognosis is also different: the recurrence rate for gestational hypertension is severalfold higher than that for preeclampsia (>20 percent versus approximately 5 percent for preeclampsia at term) [21,22].

Others have reported physiologic and histologic differences between the two disorders. Total blood and plasma volumes are significantly higher in women with gestational hypertension (3139 mL/m^2 and 2132 mL/m^2, respectively) than in women with preeclampsia (mean 2660 mL/m^2 and 1790 mL/m^2, respectively) [23], Doppler measures of arterial and venous hemodynamics and vascular endothelial function are normal in women with gestational hypertension and abnormal in women with preeclampsia [16,24], and levels of microparticles associated with endothelial cell damage are significantly lower in women with gestational hypertension than in women with preeclampsia [25]. Histologic signs of placental ischemia are less prominent in gestational hypertension than in preeclampsia [26].

MANAGEMENT

The decision to deliver women with preterm gestational hypertension attempts to balance three competing factors: (1) the fetal benefits from expectant management (ie, further growth and maturation), (2) the maternal and fetal benefits from early intervention (ie, avoidance of complications from progression of hypertensive disease over the remainder of pregnancy), and (3) the maternal and fetal risks from expectant management (eg, progression of hypertensive disease and possible sequelae, including stillbirth or asphyxia). We believe close surveillance of pregnancies with nonsevere gestational hypertension managed expectantly can mitigate the risk of development of serious sequelae of gestational hypertension; therefore, we manage these patients expectantly and deliver them when their clinical situation deteriorates or at term. (See 'Timing of delivery' below.)

This strategy is supported by the HYPITAT-II trial, which randomly assigned women with nonsevere hypertensive disorders of pregnancy at 34+0 to 36+6 weeks to immediate delivery or to expectant management with delivery at term or upon development of features of severe preeclampsia [27]. Three percent of women with gestational hypertension (new hypertension

with diastolic blood pressure ≥100 mmHg and no proteinuria) managed expectantly developed one or more adverse maternal outcomes (thromboembolic complications, HELLP syndrome, eclampsia, placental abruption) versus no woman in the immediate delivery group, but immediate delivery also resulted in more cases of neonatal respiratory distress syndrome (4.3 versus 1.1 percent). At age 5, early delivery did not result in poorer neurodevelopmental outcomes compared with expectant management [28].

Blood pressure less than 160/110 mmHg — The management of gestational hypertension is similar to that of preeclampsia without severe features.

Site of care — Most women with gestational hypertension without severe blood pressure elevation (ie, systolic blood pressure is ≥160 mmHg and/or diastolic blood pressure is ≥110 mmHg) can be managed safely as outpatients with weekly or twice weekly office visits to assess maternal symptoms and fetal well-being, and measure blood pressure, protein excretion, platelet count, serum creatinine, and liver enzymes. Home blood pressure monitoring can be useful to determine the patient's average and peak blood pressure during usual activity.

Patient education and counseling — Patient education and counseling are important components of management since these patients are at increased risk of developing preeclampsia and other pregnancy complications. We instruct patients to promptly report any symptoms suggestive of preeclampsia (headache, visual changes, epigastric or right upper quadrant pain). We also review signs suggestive of possible fetal impairment, such as decreased fetal movement and vaginal bleeding, and signs of preterm labor. Patients should be given appropriate telephone numbers to call care providers. Heavy vaginal bleeding, severe headache ("worse headache of my life"), stroke symptoms, severe breathing problems, or sudden severe pain are considered medical emergencies because they can be associated with a life-threatening condition.

Level of physical activity — Women may maintain most of their normal physical activities. Bedrest at home or in the hospital does not prevent progression to preeclampsia or improve maternal or fetal outcome, but reduces the frequency of worsening hypertension [29]. The decision to place a patient on bedrest should be individualized and should take into consideration the patient's blood pressures, comorbidities, and social factors. Prolonged bedrest should be avoided because it increases the risk of venous thromboembolism [30].

We advise against strength training and pure isometric exercise, such as weight lifting, as these activities can acutely raise blood pressure to severe levels. Aerobic exercise can cause a modest rise in systolic pressure, usually with no change or a slight reduction in diastolic pressure. In the

absence of information about patients' blood pressure responses to their usual aerobic exercise activities, we advise against aerobic exercise.

Whether and how many hours patients continue to work outside the home depend on multiple factors, particularly their blood pressure at work. These decisions should be made on a case-by-case basis. (See "Working during pregnancy", section on 'Work characteristics'.)

Low-dose aspirin — Whether low-dose aspirin prevents progression of gestational hypertension to preeclampsia is unclear. We do not begin aspirin for prevention of preeclampsia after 20 weeks of gestation and therefore do not prescribe it for women with gestational hypertension. Meta-analyses have shown that beginning low-dose aspirin in the second trimester to pregnant women at average or high risk of developing preeclampsia is associated with a modest reduction in preeclampsia and its sequelae (growth restriction, preterm birth) [31]. However, the included trials had a wide variety of inclusion and exclusion criteria, with some including and others excluding women with gestational hypertension. Most women in these trials began low-dose aspirin before 20 weeks of gestation. Practice guidelines consistently recommend initiating low-dose aspirin before 20 weeks for this reason and because preeclampsia is known to affect placental development early in pregnancy [32]. (See "Preeclampsia: Prevention", section on 'Low-dose aspirin'.)

Maternal blood pressure and laboratory monitoring — We agree with the American College of Obstetricians and Gynecologists (ACOG) guidelines that suggest monitoring blood pressure serially with at least one in-office measurement and weekly assessment of proteinuria, platelet count, serum creatinine, and liver enzymes [1]. Proteinuria, thrombocytopenia, renal insufficiency, or elevated liver enzymes change the diagnosis to preeclampsia, and these patients should be managed accordingly. As discussed above, home blood pressure monitoring can be useful to determine the patient's average and peak blood pressure serially during usual activity. (See "Preeclampsia: Management and prognosis".)

Fetal assessment — In our practice, we ask patients with gestational hypertension to monitor fetal movement daily and call if it is decreased or absent. We order either a nonstress test with sonographic estimation of the amniotic fluid index or a biophysical profile weekly. Testing is begun at 32 weeks of gestation, or earlier if an increased risk of fetal demise is identified and delivery for perinatal benefit would be considered if test results are abnormal.

We also perform serial ultrasound examinations to monitor fetal growth every three to four weeks, as hypertension of any etiology may be associated with placental insufficiency [33,34]. (See "Overview of antepartum fetal surveillance".)

The need for, type, and frequency of fetal assessment in women with gestational hypertension that is not severe are controversial [5]. There is no evidence from large randomized trials that any routine surveillance method results in a decreased risk of fetal death or neonatal morbidity in these patients. Nevertheless, antepartum fetal monitoring of pregnancies deemed to be at increased risk of adverse fetal outcome is a routine obstetric practice in the United States.

Antihypertensive therapy — We do not prescribe antihypertensive drugs for antepartum treatment of gestational hypertension, unless hypertension is severe or approaching the severe range or the patient has preexisting end organ dysfunction (eg, renal, cardiac) that may be worsened by hypertension. Data from randomized trials show that drug therapy of mild hypertension does not improve maternal or neonatal outcome. These data are reviewed separately. (See "Treatment of hypertension in pregnant and postpartum women".)

For most women undergoing treatment of severe hypertension, the blood pressure goal is 130 to 150 mmHg systolic and 80 to 100 mmHg diastolic, similar to that in preeclampsia. (See "Treatment of hypertension in pregnant and postpartum women".)

Antenatal corticosteroids — If the clinician believes that an individual patient is at increased risk for delivery within seven days and before 34 weeks of gestation (eg, coexistent pregnancy complications, development of preeclampsia), then corticosteroids should be administered. However, a course of antenatal corticosteroids is not routinely administered to women with nonsevere gestational hypertension because preterm birth <34 weeks is uncommon. A review of pregnancy outcomes in women with nonsevere gestational hypertension found that delivery before 34 weeks occurred in only 1 to 5 percent of cases [5].

Use of steroids at late preterm gestational ages is more controversial. We believe a course of betamethasone is reasonable for patients suspected to be at risk for rapid progression to preeclampsia. (See "Antenatal corticosteroid therapy for reduction of neonatal respiratory morbidity and mortality from preterm delivery".)

Timing of delivery — We deliver patients with gestational hypertension at term rather than preterm, in general agreement with guidelines from multiple major societies [32]. We individualize timing of delivery in these cases based on the degree of hypertension, presence of comorbidities, and the presence of risk factors for adverse pregnancy outcome.

- For pregnancies with gestational hypertension and frequent blood pressures ≥140/90 mmHg and <160/110 mmHg, comorbidities, or other risk factors for adverse outcome, we deliver at 37+0 weeks.

- For uncomplicated pregnancies (no comorbidities or other risk factors for adverse outcome) with gestational hypertension in which only a few blood pressures are ≥140/90 mmHg and <160/110 mmHg, we deliver at 38+0 to 39+0 weeks since neonatal morbidity decreases with advancing gestational age and maternal morbidity is unlikely to be increased with only occasional episodes of nonsevere blood pressure elevation.

Our approach is supported by findings of a retrospective cohort study from the Consortium on Safe Labor that found that induction of labor between 38 and 39 weeks of gestation achieved the optimal balance of low maternal and low neonatal morbidity/mortality [35].

Our approach is similar to that of a consensus opinion of a workshop held by the Eunice Kennedy Shriver National Institute of Child Health and Human Development and the Society for Maternal-Fetal Medicine that suggested delivery at 37+0 to 38+6 weeks for all women with any degree of gestational hypertension because of the risk of progression to preeclampsia [36]. It differs from an ACOG practice bulletin, based largely on expert opinion, that suggests delivery rather than expectant management at ≥37+0 weeks for all women with uncomplicated gestational hypertension [1].

By contrast, for pregnancies with gestational hypertension and blood pressures ≥160/110 mmHg at ≥34 weeks, we and ACOG recommend delivery after maternal stabilization [1]. Expectant management until ≥37+0 weeks is not advised. (See 'Blood pressure greater than 160/110 mmHg' below.)

Intrapartum management — During labor, we monitor women with gestational hypertension for development of proteinuria, worsening hypertension, and symptoms of severe disease since preeclampsia can manifest intrapartum. We do not administer magnesium sulfate seizure prophylaxis unless the patient develops severe hypertension or symptoms or laboratory abnormalities associated with severe preeclampsia. ACOG suggests use of magnesium sulfate seizure prophylaxis for women with gestational hypertension with severe features (table 2), preeclampsia with severe features, or eclampsia [1].

Blood pressure greater than 160/110 mmHg — Women who develop severe gestational hypertension have rates of pregnancy complications comparable to those with preeclampsia with severe features, and thus, ACOG recommends managing these patients similarly. Delivery after maternal stabilization is advised for pregnancies ≥34+0 weeks. (See "Preeclampsia: Management and prognosis", section on 'Preeclampsia with features of severe disease'.)

MATERNAL PROGNOSIS

Postpartum course — Most women with gestational hypertension become normotensive within the first postpartum week [37]. If blood pressure returns to normal by 12 weeks postpartum, their diagnosis is changed to transient hypertension of pregnancy. If they remain hypertensive at the 12th postpartum week, they are given the diagnosis of chronic hypertension, which happens in approximately 15 percent of cases [38].

The mean time to normalization of blood pressure postpartum after preeclamptic pregnancies is approximately two weeks. The slower rate of recovery in preeclampsia may reflect the time required for resolution of the endothelial injury, which may not be present in gestational hypertension.

The decision to use nonsteroidal anti-inflammatory agents for postpartum analgesia should be individualized, as these drugs are known to cause elevations in blood pressure in nonpregnant individuals with hypertension. If blood pressure is elevated in the postpartum period, we recommend avoiding these drugs. (See "Treatment of hypertension in pregnant and postpartum women", section on 'Analgesia'.)

Recurrence risk — A 2015 meta-analysis of individual patient data from almost 24,000 women with gestational hypertension who became pregnant again reported that 22 percent developed hypertension in a subsequent pregnancy (gestational hypertension: 15 percent, preeclampsia: 7 percent) [39]. Given these data and other data that women with a high risk factor or several moderate risk factors for preeclampsia may benefit from low-dose aspirin therapy in pregnancy, we offer women with a history of gestational hypertension and blood pressures ≥160/110 mmHg low-dose aspirin in future pregnancies to reduce their risk of developing preeclampsia. (See "Preeclampsia: Prevention", section on 'Candidates'.)

Long-term prognosis — Gestational hypertension is associated with development of hypertension later in life, and also with development of diseases related to hypertension (cardiovascular disease, hyperlipidemia, chronic kidney disease, diabetes mellitus) [38,40-47]. A prospective study of over 15,000 women with a first singleton birth observed that women with gestational hypertension in three consecutive pregnancies had significantly higher blood pressure later in life than women who remained normotensive (systolic pressure 27 mmHg higher, diastolic pressure 12 mmHg higher) [41]. They also had more unfavorable lipid and glycemic profiles, but these differences appeared to be due to higher body mass index at follow-up in women with a history of hypertension in pregnancy. In another study, women with both gestational hypertension and gestational diabetes were at particularly high risk for future diabetes (hazard ratio [HR] 36.9, 95% CI 26.0-52.3), hypertension (HR 5.7, 95% CI 4.9-6.7), and cardiovascular disease/mortality (HR 2.4, 95% CI 1.6-3.5) compared with women who had neither disorder, but no information on maternal weight was available [48]. In a third study,

gestational hypertension was associated with a twofold increased risk of cardiovascular disease during 14 years of postpartum follow-up, and the risk increased in those with small-for-gestational-age infants and/or preterm delivery [46].

Gestational hypertension and preeclampsia appear to have similar long-term cardiovascular risks, including chronic hypertension. Many long-term outcome studies evaluate outcomes among women with a history of "pregnancy-associated hypertension," given some uncertainty in the distinction between gestational hypertension and preeclampsia. The long-term risks of preeclampsia are reviewed in detail separately. (See "Preeclampsia: Management and prognosis", section on 'Long-term maternal risks of pregnancy-associated hypertension'.)

Clinical monitoring, risk factor evaluation, and early intervention might benefit women with a history of hypertension of any etiology in pregnancy. (See "Overview of primary prevention of cardiovascular disease".)

PERINATAL OUTCOME

- **Nonsevere hypertension** – Pregnancy outcomes of patients with nonsevere gestational hypertension are generally favorable [3-5,12,49-51]. Most studies report that the mean birth weight and rates of fetal growth restriction, preterm birth, abruption, and perinatal death are similar to those in the general obstetric population. However, one population-based cohort study reported that the risk of delivering a small for gestational age newborn at term increased by 2 percent for each mmHg rise in diastolic blood pressure from early to late pregnancy, even in the absence of overt hypertension [52].

- **Severe hypertension** – Pregnancies associated with severe gestational hypertension appear to be at increased risk of maternal and perinatal morbidity [3-5,12,20,49,50]. These pregnancies have rates of preterm delivery, small for gestational age (SGA) infants, and abruptio placentae significantly higher than the rates in the general obstetric population, and similar to rates reported for women with severe features of preeclampsia. In a study that compared selected outcomes in women who developed severe preeclampsia (n = 45), severe gestational hypertension (n = 24), and women who remained normotensive or developed mild gestational hypertension (n = 467), the rates of delivery <35 weeks were 36, 25, and 8 percent, respectively [4]. The rates of delivery of a SGA infant were 11, 21, and 7 percent, respectively. The small number of patients with severe gestational hypertension or severe preeclampsia limits the generalizability of these results.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Hypertensive disorders of pregnancy".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: High blood pressure and pregnancy (The Basics)")

SUMMARY AND RECOMMENDATIONS

Definition and diagnosis

- Gestational hypertension is the most common cause of hypertension during pregnancy. It is defined as the new onset of hypertension (defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg) after the 20th week of pregnancy in the absence of proteinuria or new signs of end-organ dysfunction.

  Depending on its course during pregnancy and postpartum, this provisional diagnosis may change to a final diagnosis of preeclampsia (table 1), chronic hypertension (gestational hypertension that persists longer than 12 weeks postpartum), or transient hypertension of pregnancy (gestational hypertension that returns to normal blood pressures by 12 weeks postpartum). (See 'Definition and provisional diagnosis' above and 'Final diagnosis' above and 'Prevalence' above.)

Evaluation and management

- The main goals in the evaluation and management of women with gestational hypertension are to distinguish this disorder from preeclampsia (table 1), which has a different prognosis and management, and to identify those with severe hypertension (systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥110 mmHg), which affects management and outcome. Therefore, we measure blood pressure once or twice weekly and measure urine protein, platelets, and liver enzymes weekly. Ten to 50 percent of women initially diagnosed with gestational hypertension go on to develop preeclampsia within one to five weeks. (See 'Maternal blood pressure and laboratory monitoring' above and 'Blood pressure greater than 160/110 mmHg' above and "Preeclampsia: Management and prognosis" and 'Risk of progression to preeclampsia' above.)

  Starting at 32 weeks of gestation, we order either a nonstress test with sonographic estimation of the amniotic fluid index or a biophysical profile weekly and perform serial ultrasound examinations to monitor fetal growth every three to four weeks. (See 'Fetal assessment' above.)

- Antihypertensive drugs and antenatal corticosteroids are administered for standard indications. (See 'Antihypertensive therapy' above and 'Antenatal corticosteroids' above.)

Timing of delivery

- For pregnancies with gestational hypertension characterized by systolic blood pressure ≥160 mmHg and/or diastolic blood pressure ≥110 mmHg, management is the same as that for preeclampsia with severe features. Delivery at ≥34 weeks is advised after maternal stabilization. (See 'Blood pressure greater than 160/110 mmHg' above.)

- For pregnancies with gestational hypertension characterized by frequent blood pressures ≥140/90 mmHg but <160/110 mmHg, comorbidities, or other risk factors for adverse outcome, we suggest delivery at 37+0 weeks of gestation (**Grade 2C**).

- For uncomplicated pregnancies (no comorbidities or other risk factors for adverse outcome) with gestational hypertension in which a substantial proportion of blood pressures are <140/90 mmHg and a few blood pressures ≥140/90 mmHg but <160/110 mmHg, we suggest delivery at 38+0 to 39+0 weeks (**Grade 2C**). Neonatal morbidity decreases with advancing gestational age, and maternal morbidity is unlikely to be increased with only occasional episodes of nonsevere blood pressure elevation. (See 'Timing of delivery' above.)

Recurrence

- Hypertension recurs in approximately 22 percent of subsequent pregnancies and is associated with development of hypertension later in life. (See 'Maternal prognosis' above.)

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Topic 6805 Version 62.0

GRAPHICS

Criteria for the diagnosis of preeclampsia

Systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg on at least 2 occasions at least 4 hours apart after 20 weeks of gestation in a previously normotensive patient AND the new onset of 1 or more of the following*:
▪ Proteinuria ≥0.3 g in a 24-hour urine specimen or protein/creatinine ratio ≥0.3 (mg/mg) (30 mg/mmol) in a random urine specimen or dipstick ≥2+ if a quantitative measurement is unavailable
▪ Platelet count <100,000/microL
▪ Serum creatinine >1.1 mg/dL (97.2 micromol/L) or doubling of the creatinine concentration in the absence of other renal disease
▪ Liver transaminases at least twice the upper limit of the normal concentrations for the local laboratory
▪ Pulmonary edema
▪ New-onset and persistent headache not accounted for by alternative diagnoses and not responding to usual doses of analgesics¶
▪ Visual symptoms (eg, blurred vision, flashing lights or sparks, scotomata)

Preeclampsia is considered superimposed when it occurs in a woman with chronic hypertension. It is characterized by worsening or resistant hypertension (especially acutely), the new onset of proteinuria or a sudden increase in proteinuria, and/or significant new end-organ dysfunction after 20 weeks of gestation in a woman with chronic hypertension.

* If systolic blood pressure is ≥160 mmHg or diastolic blood pressure is ≥110 mmHg, confirmation within minutes is sufficient.
¶ Response to analgesia does not exclude the possibility of preeclampsia.

Adapted from: American College of Obstetricians and Gynecologists (ACOG) Practice Bulletin No. 222: Gestational Hypertension and Preeclampsia. Obstet Gynecol 2020; 135:e237.

Graphic 79977 Version 36.0

Features of severe disease in a woman with a pregnancy-related hypertensive disorder

Systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥110 mmHg, or both (on two separate occasions)
Symptoms of central nervous system dysfunction:
New-onset cerebral or visual disturbance, such as: ▪ Photopsia, scotomata, cortical blindness, retinal vasospasm ▪ Severe headache (ie, incapacitating, "the worst headache I've ever had") or headache that persists and progresses despite analgesic therapy
Hepatic abnormality:
Severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by an alternative diagnosis or serum transaminase concentration ≥2 times the upper limit of the normal range, or both
Thrombocytopenia:
<100,000 platelets/microL
Renal abnormality:
Progressive renal insufficiency (serum creatinine >1.1 mg/dL [97.2 micromol/L] or doubling of serum creatinine concentration in the absence of other renal disease)

Adapted from ACOG Practice Bulletin No. 222: Gestational Hypertension and Preeclampsia. Obstet Gynecol 2020; 135:e237.

Graphic 111086 Version 4.0

Clinical screening tools for substance use disorders during pregnancy

4 Ps[1]
Parents: Did any of your parents have a problem with alcohol or other drug use?
Partner: Does your partner have a problem with alcohol or drug use?
Past: In the past, have you had difficulties in your life because of alcohol or other drugs, including prescription medications?
Present: In the past month, have you drunk any alcohol or used other drugs?
Scoring: Any "yes" should trigger further questions.
NIDA Quick Screen[2]
Screen your patients
Step 1. Ask patient about past year drug use – the **NIDA Quick Screen**
Step 2. Begin the **NIDA-Modified ASSIST**
Step 3. Determine risk level
Conduct a brief intervention
Step 4. **A**dvise, **A**ssess, **A**ssist, and **A**rrange
CRAFFT – Substance Abuse Screen for Adolescents and Young Adults[3]
C Have you ever ridden in a **CAR** driven by someone (including yourself) who was high or had been using alcohol or drugs?
R Do you ever use alcohol or drugs to **RELAX**, feel better about yourself, or fit in?
A Do you ever use alcohol or drugs while you are by yourself or **ALONE**?
F Do you ever **FORGET** things you did while using alcohol or drugs?
F Do your **FAMILY** or friends ever tell you that you should cut down on your drinking or drug use?
T Have you ever gotten in **TROUBLE** while you were using alcohol or drugs?
Scoring: Two or more positive items indicate the need for further assessment.

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