Complications In Pregnancy 
Part II: Hypertensive 
Disorders Of Pregnancy 
And Vaginal Bleeding

It is Sunday morning and you are working in a quiet ED. You hear a code called up in the neonatal ICU and wonder what it is. You will soon find out! A few minutes later the nursing supervisor notifies you that a visitor had a seizure and is being transported to the ED by 2 NICU nurses. Upon arrival, the patient appears postictal. She is brought into the resuscitation room and placed on a monitor. As you note that her blood pressure is slightly elevated, the patient begins to have a second generalized seizure. You initiate your standard seizure protocol and give an order to the nurse for lorazepam IV but wonder if you are missing something else...

Recognition and management of life-threatening disorders of pregnancy are integral parts of emergency medicine practice. This issue of Emergency Medicine Practice focuses on hypertensive disorders of pregnancy and vaginal bleeding. More information about complications in early pregnancy, specifically ectopic pregnancies, can be found in the June 2007 issue of Emergency Medicine Practice.

Critical Appraisal Of The Literature

A literature search was initiated using Ovid MEDLINE, Cochrane, and PubMed for pregnancy-related emergencies between 1966 and 2007.

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CME Objectives
Upon completion of this article, you should be able to:
1. Identify the etiology, pathophysiology, and management of hypertensive disorders of pregnancy.
2. Practice proper management of vaginal bleeding during pregnancy including placental abruption and placenta previa.

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2006. The search was limited to English-language and human studies and included key words such as pregnancy, placenta, previa, abruptio, eclampsia, preeclampsia, hypertension, vaginal bleeding, Rh immunization, and ultrasound. In addition, articles were also identified from the bibliographies of the identified articles in selected cases. A search of the Cochrane Database of systemic reviews for late pregnancy-related complications included many reviews on pregnancy-related hypertension and its management.\(^{54,79,87,91,93}\)

Well-designed scientific articles on clinical trials, prospective cohort studies, and aggregate studies including meta-analyses were weighed as most credible. Retrospective studies, case-controlled studies, and other meta-analyses provided secondary support for analysis followed by panel consensus, cross-sectional studies, and case reports. The American College of Emergency Physicians (ACEP) has not published clinical policies relevant to the care of late pregnancy emergencies. The American College of Obstetrics and Gynecology (ACOG) has published multiple guidelines related to the care of emergency in the latter half of pregnancy, but these policies do not specifically address ED management.

**Terminology**

**Hypertensive Disorders In Pregnancy**

Approximately 6% to 8% of patients will be diagnosed with hypertension at some point during their pregnancy.\(^2,3\) The National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy defines hypertensive disorders of pregnancy into several categories.\(^2,3\) See Table 1 for the categories, their symptoms, and time of onset.

The International Society For The Study Of Hypertension In Pregnancy defines hypertension as “a diastolic blood pressure of 90 mm Hg or higher on 2 consecutive occasions at least 4 hours apart or a single diastolic blood pressure greater than 110 mm Hg.”\(^3\) Systolic blood pressure readings above 140 mm Hg are often used as criteria as well.\(^5\) Relative changes in blood pressure are no longer included in the definition of gestational hypertension due to the lack of evidence of any effect on outcome.\(^3,5\) Chronic hypertension is present before pregnancy or diagnosed within the first 20 weeks of gestation. Gestational hypertension occurs only after the 20th week of pregnancy and resolves during the postpartum period. Chronic hypertension is also diagnosed if gestational hypertension persists for more than 6 weeks postpartum.\(^47\)

**Preeclampsia**

Preeclampsia is defined as “gestational hypertension with proteinuria of greater than 300 mg per 24-hours typically correlated with a 30 mg/dL (+1 urine dipstick).”\(^3,7\) It is a common complication of late pregnancy and occurs in 6% to 8% of pregnancies in developed nations.\(^6,7\) Due to discrepancies between random urine protein and 24-hour samples, it is generally advised that the diagnosis be made based on a 24-hour sample.\(^3,7\) Since pregnancy is normally associated with excess extracellular fluid and dependent edema, this is no longer used as a criterion for diagnosing preeclampsia. Preeclampsia may be superimposed upon chronic hypertension as well. Although rare, if a patient appears to be preeclamptic prior to 20 weeks gestation, a hydatidiform mole, hydropic degeneration of the placenta, or choriocarcinoma must be considered.\(^11-13\) Table 2 differentiates between mild and severe preeclampsia and Table 3 lists signs of severe preeclampsia.\(^3,7\)

**HELLP Syndrome**

HELLP syndrome is a life-threatening obstetric complication usually considered to be a variant of preeclampsia. HELLP is an acronym based on the major symptoms of the syndrome: hemolytic anemia, elevated liver enzymes, and low platelet count. Both HELLP syndrome and preeclampsia can occur during the later stages of pregnancy or up to 4 weeks postpartum.

**Eclampsia**

Eclampsia is the occurrence of seizures in the patient with signs of preeclampsia. Progression of preeclampsia to eclampsia is unpredictable and can occur rapidly. Eclampsia can occur several days after delivery and is diagnosed when seizures occur in the patient with signs of preeclampsia. The term “late
The incidence of eclampsia has progressively declined but is still one of the leading causes of maternal mortality. According to the Centers for Disease Control and Prevention, approximately 20% of maternal deaths in the U.S. were attributable to preeclampsia-eclampsia, with a mortality rate of 1.5 deaths per 100,000 live births. Preeclampsia-eclampsia is also a major cause of neonatal morbidity and mortality. The World Health Organization (WHO) estimates that nearly 4.2 million premature births worldwide are attributable to hypertensive disorders of pregnancy. Causes of neonatal and intrauterine death include placental infarcts, intrauterine growth retardation, and placental abruption. In addition, fetal hypoxia from maternal seizures and the complications of premature delivery mandated by the maternal condition contribute significantly to fetal mortality and morbidity.

Eclampsia may occur at any point during the puerperium period but is most likely to occur during the intrapartum period or within 48 hours after delivery. In multiple retrospective studies of more than 100 patients, the reported frequency of antepartum eclampsia has ranged from 38% to 53% in most studies. Almost all cases of eclampsia (91%) occur after 28 weeks gestation. The remaining cases occur at 21 to 27 weeks gestation (7.5%) and prior to 20 weeks gestation (1.5%). Postpartum and intrapartum eclampsia rates range from 11% to 44% and 18% to 36%, respectively.

Delivery may be necessary in patients with severe preeclampsia despite the associated neonatal morbidity and mortality. Although the overall incidence of eclampsia is decreasing, the rate of late postpartum eclampsia is increasing relatively. Rates of late eclampsia range from 5% to greater than 20% for an overall incidence of approximately 0.04% to 0.1%. Risk factors for developing preeclampsia-eclampsia are listed in Table 4.

**Vaginal Bleeding**

**Placental Abruption**

Placental abruption or *abruptio placentae* is the premature separation of the placenta from the uterine wall.

**Placenta Previa**

Placenta previa is defined as the implantation of the placenta in the lower uterine segment.

**Epidemiology**

**Hypertensive Disorders**

**Preeclampsia And Eclampsia**

Traditionally, preeclampsia has been considered a disease of the young primigravida, but family history, multiple gestations, and diabetes are also common risk factors. In the past, race was thought to be a risk factor for the development of preeclampsia. However, a secondary analysis from the NICHD’s (National Institute Of Child Health And Human Development) Maternal-Fetal Medicine Units Network’s low-risk aspirin prevention trial failed to find any association between race and the development of preeclampsia.

**Table 2. Preeclampsia Severity**

<table>
<thead>
<tr>
<th>Mild</th>
<th>Severe</th>
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<tbody>
<tr>
<td>Systolic blood pressure between 140 and 160 mm Hg</td>
<td>Systolic blood pressure &gt;160 mm Hg</td>
</tr>
<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>
</tr>
</tbody>
</table>

**Table 3. Signs and Symptoms Suggestive Of Severe Preeclampsia**

- Headache
- Visual disturbances
- Confusion
- Right upper quadrant pain
- Epigastric pain
- Oligohydramnios
- Impaired renal function
- oliguria (< 500 mL/24hr)
- Pulmonary edema
- Microangiopathic hemolytic anemia
- Thrombocytopenia (<100,000)
- Fetal impaired liver function

**Table 4. Risk Factors Associated With The Development Of Preeclampsia-Eclampsia**

- Prior preeclampsia-eclampsia
- Family history of preeclampsia
- Primigravida
- Young and older maternal age
- Multiple gestations
- Pre-existing hypertension
- Chronic renal disease
- Thrombophilia
- Collagen vascular diseases
- Hydropsy
- Hydatidiform mole
- Diabetes

The incidence of eclampsia has progressively declined but is still one of the leading causes of maternal mortality. According to the Centers for Disease Control and Prevention, approximately 20% of maternal deaths in the U.S. were attributable to preeclampsia-eclampsia, with a mortality rate of 1.5 deaths per 100,000 live births. Preeclampsia-eclampsia is also a major cause of neonatal morbidity and mortality. The World Health Organization (WHO) estimates that nearly 4.2 million premature births worldwide are attributable to hypertensive disorders of pregnancy. Causes of neonatal and intrauterine death include placental infarcts, intrauterine growth retardation, and placental abruption. In addition, fetal hypoxia from maternal seizures and the complications of premature delivery mandated by the maternal condition contribute significantly to fetal mortality and morbidity.
oxygen flow. The risk of fetal death increases in proportion to both the percentage of placental surface involved and the rapidity of separation. Maternal death can result, usually from coagulopathy or exsanguination. Feto-maternal transfusion occurs in a significant minority of patients. Placental separation also predisposes the mother to amniotic fluid embolism.

Vaginal bleeding during the third trimester complicates 4% of all pregnancies, with the most common causes being due to placental issues and less commonly due to infection or polyps. Table 5 lists additional causes of third trimester bleeding.

Placental Abruption
Placental abruption is the premature separation of the placenta from the uterine wall. It accounts for approximately one-third of vaginal bleeding episodes during the second half of pregnancy. Small subclinical or marginal separations may go undetected until the placenta is examined at delivery. These separations probably account for many of the other self-limited episodes of bleeding for which no diagnosis is made. Recent large epidemiologic studies report an incidence ranging from 5.5 to 5.8 per 1000 singleton births to 12.2 per 1000 twin births. Interestingly, the incidence of abruption peaks at 24 to 26 weeks gestation and declines as the pregnancy progresses.

Risk factors associated with the development of a placental abruption are listed in Table 6. However, in a significant minority of cases of pathology-confirmed abruption, many patients had an unremarkable obstetric history. Perhaps the largest single risk factor for developing a placental abruption is a history of prior abruption. Ananth et al found that severe preeclampsia increased the relative risk of placental abruption 3.8 fold. However, in an evaluation of 445 patients with severe preeclampsia or eclampsia, the degree of blood pressure evaluation or proteinuria did not correlate directly with risk of placental abruption. Cocaine and tobacco use are also clearly associated with an increased risk of abruption. Placental separation can also be associated with blunt trauma to the abdomen. In these cases, shearing forces on the non-elastic placenta cause separation from the elastic uterine wall at impact. Women who reported physical violence during pregnancy were twice as likely to have an abruption as women who did not report any violence.

Placenta Previa
Placenta previa is defined as the implantation of the placenta in the lower uterine segment. It is responsible for potentially life-threatening antenatal and postpartum hemorrhage. Placenta previa may be categorized based on the proportion of the cervix it covers. A complete previa covers the cervix entirely. Partial previa covers only part of the cervix, while a marginal previa abuts the cervix. (See Figure 1.) A placenta previa identified early in the pregnancy may resolve as the pregnancy progresses. As gestational age increases, placental migration occurs due to differential growth of the lower uterine segment compared with the rest of the myometrium. Placenta previa diagnosed prior to the 20th week of gestation will resolve in more than 90% of the cases. Complete placenta previa diagnosed in the 2nd trimester will persist into the third trimester in approximately 26% of cases, whereas only 2.5% of cases of partial and marginal placenta previa persist. Becker et al scanned 8650 women at 20 to 23 weeks gestational age and demonstrated that unless the placenta was overlying the internal os no women suffered vaginal bleeding or required a caesarean section for placenta previa.

Although placenta previa is the most common cause of clinically significant third trimester uterine bleeding, it is a relatively rare condition, developing in only 2 to 5 of 1000 pregnancies. At one hospital, the incidence was 0.26 percent (1 in 390) for more than 169,000 deliveries over 12 years. A common risk factor for placenta previa is a prior caesarean section, which quadruples the risk of placenta previa in subsequent pregnancies (15-20 per 1000 pregnancies). After 4 or more cesarean sections, the risk of placenta previa increases almost 20 fold to 100 per 1000 live births. Other risk factors include multiparity, maternal age greater than 35, tobacco use, leiomyomata, maternal anemia, previous abortions, uterine infections, and cocaine use.

Table 5. Possible Causes Of Vaginal Bleeding In Gestations > 20 Weeks

| • Placenta previa |
| • Placental abruption |
| • Vasa Previa |
| • Premature or term labor |
| • Genitourinary lesions or lacerations |
| • Genitourinary infections |
| • Congenital or acquired bleeding disorders |

Table 6. Risk Factors Associated With Development Of Placental Abruption

- Gestational hypertensive disease
- Maternal age
- Increasing parity
- Multiple gestations
- Polyhydramnios
- Chorioamnionitis
- Prolonged rupture of membranes
- Trauma
- Thrombophilias
- Maternal cocaine use
- Tobacco use
Pathophysiology

Hypertensive Disorders

Several mechanisms have been implicated as potential causes of preeclampsia and eclampsia. Common mechanisms include an increase in oxidative stress, diffuse endothelial dysfunction, alterations in inflammatory mediators, and abnormalities in the renin-angiotensin system. Although many of these theories have seemed promising, none completely describe the disease process. Since preeclampsia typically disappears after delivery, the placental fetal unit is the most likely cause. Evidence is quite compelling that the presence of the placenta is required for development of this disorder.

A recent study found increased fms-like tyrosine kinase-1 and reduced levels of placental and vascular endogenous growth factor in women prone to developing preeclampsia. In addition, the infusion of the fms-like tyrosine kinase-1 and the cofactor endoglin in rats induced a preeclampsia-like syndrome with hypertension and proteinuria. However, a recent review of the pathogenesis of preeclampsia found insufficient evidence to recommend using sFlt or other endothelial growth factors as early markers of preeclampsia. The renin-angiotensin system has also been implicated in the development of preeclampsia. In a normal pregnancy, the placenta produces renin, which leads to increased activity in the renin-angiotensin system. It was recently discovered in preeclamptic patients that an IgG autoantibody interacts with the angiotensin type one (AT1) receptor leading to activation. Not only does this increase blood pressure, it also mediates coagulation via tissue factor and the fibrinolytic system, induces reactive oxygen species, and influences fms-like tyrosine kinase -1 secretion. Prostaglandin aberrations have also been found in preeclamptic patients. During normal pregnancy, there is an increase in the ratio of endothelial cell-produced prostacyclin and platelet-produced thromboxane, resulting in a vasodilatory state. In preeclampsia this ratio is reversed, promoting vasoconstriction and a proaggregatory state. Studies of urinary metabolites have found that a decrease in prostacyclin production is the main cause of the reversed ratio.

The risk of cerebrovascular, cardiovascular, and renal compromise is proportional to the degree of blood pressure elevation. Central nervous system effects commonly include headache or visual disturbances. Other complications of severe preeclampsia include spontaneous hepatic and splenic hemorrhage as well as placental abruption. The most dangerous complication of preeclampsia is eclampsia. Certain features increase the likelihood of progression to eclampsia, but there is no sign, symptom, or test that can reliably predict who will end up with eclampsia.

Vaginal Bleeding

Placental Abruption

In cases of nontraumatic placental abruption, spontaneous hemorrhage from uterine arterial vessels into the decidua basalis occurs, causing separation and compression of the adjacent placenta. Small amounts of bleeding may be asymptomatic and remain undetected until delivery. In other instances, the hematoma expands and extends the dissection; bleeding may be concealed or may be clinically apparent if dissection occurs along the uterine wall to the cervix. (See Figure 2.) Placental abruption can be an acute or an indolent problem throughout late pregnancy.

Although the exact cause of placental abruption is not known, theories generally originate around vascular and placental abnormalities. Increased vessel fragility, vascular malformations, and abnormalities of placentaion have all been described. Cytotrophoblastic invasion of the spiral arteries could be one culprit. The lack of cytrophoblastic

Figure 1. Placenta Previa Categories

![Figure 1](image1.png)


Figure 2. Revealed Versus Concealed Placental Abruption

![Figure 2](image2.png)

invasion of the spiral arteries leads to decreased placental blood flow and an abnormal response to vasoactive substances. The abnormal placental vessels lead to ischemia and predispose to vessel rupture.\textsuperscript{52}

**Placenta Previa**

In placenta previa, bleeding occurs when marginal placental vessels implanted in the lower uterine segment are torn, either as the lower uterine wall elongates or with cervical dilation near the time of delivery. Since the myometrium of the lower uterine segment is thin, it is unable to contract and control the bleeding. Early bleeding episodes tend to be self-limited unless separation of the placental margin is aggravated by iatrogenic cervical probing or onset of labor.\textsuperscript{32}

**Prehospital Care**

Prehospital care of the pregnant patient is focused on supportive measures, resuscitation, and rapid transport to an appropriate facility. Much of the literature on pregnancy and prehospital care is based on consensus opinion. No prospective studies that address this issue could be found.

As with any patient, vigilantly assess and initiate treatment using the ABCs. Support the patient’s airway and apply supplemental oxygen if indicated. Obtain intravenous access if needed. If the patient is hypotensive, initiate aggressive fluid resuscitation with an isotonic solution through a large bore intravenous catheter. If the patient is in the third trimester, consider the left lateral decubitus position to prevent the gravid uterus from compressing the inferior vena cava and reducing venous return to the heart. If spinal immobilization is required, secure the patient in a modified 45 degree angle on their left side. In addition, placing the patient in a Trendelenburg position can also improve venous return.

A patient who presents with seizures due to suspected eclampsia may be a challenge for the paramedics. Moderate or severe preeclampsia associated with edema and hyperactive reflexes requires no prehospital treatment other than prophylactic venous access. In cases of eclampsia, there may be no historian available and if the patient is less than 30 weeks gestation, pregnancy may not be recognized. Initiate the ABCs and establish IV access. Measure blood glucose to exclude hypoglycemia as a cause of the seizure. Benzodiazepines are typically the first agent of choice in any patient with active seizures, but if the diagnosis of eclampsia is suspected, give IV magnesium sulfate. In some regions, magnesium sulfate is available to prehospital providers. Benzodiazepines can be administered as an anticonvulsant, although studies have suggested that it is not as effective as magnesium in eclampsia.\textsuperscript{54}

Transport the patient to an appropriate facility with high-risk obstetric capabilities.\textsuperscript{1} If the patient is unstable in the field, the patient should be taken to the closest emergency medical facility to stabilize their condition prior to transfer to an appropriate facility.

**ED Evaluation**

When a patient in the latter half of pregnancy presents to the emergency department, a careful history and physical examination is required. The history should concentrate on the presenting symptom, which will guide further workup. Obtain information on any risk factors for common complications of late pregnancy. Assess fetal well-being. Consider using ultrasound to assess for movement, heart rate, and amount of amniotic fluid. Continuous fetal monitoring may be appropriate. Utilize serial measurements of the fetal heart rate. A high or low heart rate may indicate fetal distress and the need for urgent resuscitation of the mother and/or delivery.

**Preeclampsia And Eclampsia**

While one study evaluating the risk factors that lead from preeclampsia to eclampsia determined that hypertension may be absent in up to 16% of cases at the time of BP measurement in the ED,\textsuperscript{55} hypertension remains the hallmark of both preeclampsia and eclampsia. The risk of end-organ compromise is proportional to the degree of blood pressure elevation. In severe preeclampsia, diastolic BP can exceed 110 mm Hg, proteinuria worsens, and there is evidence of vasospastic end-organ effects. Signs of preeclampsia include headache and visual disturbances, spontaneous hepatic and splenic hemorrhage, as well as placental abruption.

The most dangerous complication of preeclampsia is eclampsia. Eclampsia may occur at any point during the puerperium, but is most likely to occur intrapartum or within 48 hours of delivery.\textsuperscript{55,57,58} In multiple retrospective studies of more than 100 patients with preeclampsia, the reported frequency of antepartum eclampsia ranged from 38% to 53% with 91% of cases occurring after 28 weeks gestation.\textsuperscript{55} Postpartum and intrapartum eclampsia evolving from preeclampsia occurs in 11% to 44% and 18% to 36%, respectively.\textsuperscript{56} Certain features increased the likelihood of progression, but there is no sign, symptom or test that can reliably predict the development of eclampsia. One study of 399 patients found that 58% of patients with antepartum eclampsia and 71% of patients with eclampsia prior to 32 weeks gestation have severe hypertension\textsuperscript{59} (defined by the American Heart Association as “a blood pressure reading of 180 to 210/110 to 120 mm Hg”). Fifty-nine to seventy-five percent of eclamptic patients have at least one of the following symptoms, headache (most common), right upper quadrant pain, and visual disturbances.\textsuperscript{57,58} In a retrospective review of
Placental Abruption And Placenta Previa

The classic presentation of placental abruption includes vaginal bleeding, abdominal pain, uterine tenderness, and uterine irritability. (See Table 7.) Though variable, the blood is characteristically dark and low volume, although the mother may have hemodynamic evidence of significant blood loss. Up to 20% of women will have no pain or vaginal bleeding. In a prospective study of patients with confirmed abruptions, vaginal bleeding was present in 78%, uterine tenderness in 66%, and uterine contractions in 34%. Placental abruption can be broadly classified into grades based on coagulation parameters and signs of fetal distress. Grade I placental abruptions (40%) are characterized by slight vaginal bleeding and some uterine irritability. Maternal blood pressure is stable and fetal heart rate monitoring is normal. Coagulopathy is not present. Grade II placental abruptions (45%) are associated with mild to moderate vaginal bleeding; additionally, uterine contractions, postural hypotension or an elevated maternal heart rate, and signs of fetal distress may be present. Finally, grade III placental abruptions (15%) present with severe hemorrhage (although it may be concealed) and a tender tectic uterus. Fetal distress and possibly death may occur and maternal uncompensated shock is usually present. At this stage, disseminated intravascular coagulation (DIC) is often clinically apparent.

In contrast, a classic sign of placenta previa is painless bright red vaginal bleeding after the 24th week of gestation. Often the bleeding is spontaneous and without warning. In about 20% of cases, some degree of uterine irritability is present, though it is generally minor. The bleeding typically is self-limited but may recur.

The physical examination often reveals no specific findings other than bright red vaginal bleeding. The degree of bleeding is often proportional to the degree of hemodynamic compromise. In cases of significant blood loss, signs of hemorrhagic shock must first be addressed, but in cases of mild bleeding, the vital signs may be completely normal. Digital or instrumental probing of the cervix should never be performed during the second half of pregnancy, because it can precipitate severe hemorrhage in the patient with an asymptomatic or minimally symptomatic placenta previa. In the emergency department, speculum examination of the vagina and cervix should be performed only in those settings where obstetric consultation is not readily available. In these cases, it should be limited to an atraumatic, partial speculum insertion to identify other causes of potential bleeding.

Diagnostic Studies

The ancillary studies utilized to help elucidate the late emergencies of pregnancy depend on the differential diagnosis which generally falls into 2 categories: hypertensive disorders of pregnancy and vaginal bleeding. In addition, pregnant patients presenting with a viable gestation require assessment of fetal well-being utilizing ultrasound and/or fetal monitoring.

Laboratory Testing - Hypertensive Disorders

In hypertensive diseases of pregnancy, the diagnosis of preeclampsia is based on hypertension and the presence of greater than 300 mg of protein from a 24-hour urine specimen. On a random urine sample in the ED, 30 mg/dL or +1 on a urine dipstick can estimate a positive 24-hour specimen for proteinuria. However, proteinuria may be variable at any given time and may not be detectable in a random urine specimen. The laboratory workup for a patient with suspected preeclampsia focuses on excluding end-organ dysfunction. A peripheral smear of the blood may also identify signs of microangiopathic hemolysis such as schistocytes and burr cells. An electrolyte panel including renal function should be obtained. Blood urea nitrogen (BUN) and creatinine levels may prove helpful in identifying end-organ kidney dysfunction. If a magnesium infusion is anticipated, a baseline magnesium level should be obtained but should not delay therapy initiation.

Uric acid has also been routinely measured in hypertensive disorders of pregnancy. Serum uric acid is increased once a patient develops preeclampsia and the degree of elevation has been found to correlate with the severity of illness. However, uric acid is not useful as a screening tool to predict which patients may develop preeclampsia or the presence of significant proteinuria.

Table 7. Clinical Features Associated With Placental Abruption

<table>
<thead>
<tr>
<th>Classic Symptoms And Signs</th>
<th>Other Signs And Symptoms</th>
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<tbody>
<tr>
<td>Vaginal bleeding</td>
<td>Back pain (especially with posterior placentas)</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>Idiopathic preterm labor</td>
</tr>
<tr>
<td>Uterine irritability or hypercontractile state</td>
<td>Fetal distress or fetal unrecognized fetal demise</td>
</tr>
<tr>
<td></td>
<td>Coagulopathy or diffuse disseminated coagulation</td>
</tr>
<tr>
<td></td>
<td>Shock – hypovolemic or septic</td>
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eclampsia. In addition, uric acid has also been found to be a poor predictor of maternal and fetal adverse events. Prothrombin time, partial thromboplastin time, and fibrinogen levels are usually normal but if coagulation times are prolonged and fibrinogen levels are decreased, disseminated intravascular coagulation must be considered and a type and screen should be obtained. In rare instances, spontaneous liver hematoma or spleen rupture may require immediate transfusion of packed red cells. Elevated total leukocyte count, creatinine, and aspartate aminotransferase are also predictive of increased morbidity for the patient with severe preeclampsia. HELLP syndrome develops in 5% to 10% of all preeclamptic patients. The diagnosis of HELLP syndrome is based entirely on laboratory parameters in the setting of preeclampsia. Laboratory evaluation in patients with suspected HELLP includes a complete blood count (CBC) with platelet count, peripheral smear, coagulation studies, liver function studies, and electrolyte panel including measures of renal function and lactate dehydrogenase. Generally accepted laboratory values for HELLP syndrome are defined in Table 8.

**Laboratory Testing - Vaginal Bleeding**

A baseline hemoglobin level should be obtained, and blood should be sent for type and crossmatch in all women with late trimester vaginal bleeding. Other recommended studies include baseline coagulation studies (including platelet count and prothrombin time) and partial thromboplastin, fibrinogen level, and fibrin split products. Normal fibrinogen levels in pregnancy are 400 to 450 mg/dL; values below 300 mg/dL indicate significant consumption of coagulation factors.

Traditionally, the Kleihauer-Betke test (KBT) has been recommended to rule out feto-maternal transfusion. Unfortunately, the test is difficult to perform, not immediately available in most emergency laboratories, and only sensitive enough to detect 5 mL of fetal cells in the maternal circulation. Furthermore, its utility has been questioned in late pregnancy. A retrospective case control study of 100 women with low-risk pregnancies and 151 women being evaluated for abdominal trauma found that the incidence of a positive KBT was similar in both groups. This finding did not demonstrate definitively the utility of a positive KBT. In addition, there was no association between abruption and a positive KBT. Therefore, the KBT has limited utility in the diagnosis of placental abruption. No studies were found addressing the role of the KBT with placenta previa either. However, if a positive KBT is found, it will help quantify the amount of feto-maternal transfusion and possibly guide Rh-immune globulin in Rh-negative women. Other tests for detecting feto-maternal hemorrhage are also available, but none have been studied as extensively as the KBT. In general, it is suggested that patients who are Rh-negative and at risk of feto-maternal transfusion receive RhoGAM therapy.

**Diagnostic Studies - Hypertensive Disorders**

### Neuroimaging

Seizures are the hallmarks of eclampsia. As in all seizure patients, hypoglycemia, drug overdose, and other causes of seizures should be excluded using appropriate tests. A non-contrast head CT is indicated for focal neurologic findings or refractory seizures to evaluate for intracranial hemorrhages and venous sinus thrombosis. Patchy hemorrhages and micro-infarcts of the cortex are characteristic and may be due to loss of cerebral autoregulation in patients with severe pregnancy-related hypertension. Diffuse cerebral edema can also be seen.

### Diagnostic Studies - Vaginal Bleeding

#### Ultrasonography

Ultrasonography is the diagnostic imaging modality of choice for localizing the placenta in any patient who presents with vaginal bleeding during the latter half of pregnancy. The placental location in relationship to the cervical os is vital in making the diagnosis of placenta previa. By the 28th week of pregnancy, the placenta can typically be located with ultrasound in 99% of pregnancies. The exact location of the placenta is not as accurate. Diagnostic accuracy in the second and third trimester has been reported to be approximately 90% to 95%, with a false positive rate of 5% to 10%. A transvaginal ultrasound is more accurate than a transabdominal ultrasound in identifying placenta previa, in particular with posterior and low lying placentas. In a study comparing the ability to detect a posterior placenta previa by ultrasound, the transvaginal ultrasound had a positive predic-

**Table 8. Laboratory Diagnostic Criteria for HELLP Syndrome**

<table>
<thead>
<tr>
<th>Hemolysis</th>
<th>Lactate dehydrogenase &gt;600 U/L</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Peripheral smear with burr cells, schistocytes, or other abnormal RBC forms</td>
</tr>
<tr>
<td></td>
<td>Elevated bilirubin</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>Platelet count &lt; 100,000/mm³</td>
</tr>
<tr>
<td>Elevated liver enzymes</td>
<td>AST &gt; 70 U/L or 2 times above the normal standard deviation of lab</td>
</tr>
<tr>
<td></td>
<td>Lactate dehydrogenase &gt; 600U/L</td>
</tr>
</tbody>
</table>

and sonographic evidence of placental hemorrhage is found in only 50% of abruptions. If complete escape of the blood occurs, ultrasound examination of the placenta may be completely normal. In addition, an acute abruption may appear similar to other soft tissue abnormalities of the uterus such as leiomyomata. In these cases, Doppler color flow analysis may prove helpful. A contained acute hemorrhage will appear hypoechoic or isoechoic when compared to the placenta.

In some cases of acute hemorrhage, the placenta may only appear thickened or spherical in appearance. (See Figure 3.) Ultrasound may be helpful in identifying the size and location of an abruption, which can have prognostic significance. There are 3 predominant locations: subchorionic (which is associated with less than 10% fetal mortality), retroplacental (which is associated with greater than 50% fetal mortality), and preplacental.

**Figure 3. Normal Placenta Versus Placental Abruption**

A. Normal placenta: Note the retroplacental hypoechoic space. (p) It is important not to mistake this for abruption. B. Large, retroplacental abruption (a) between the placenta (p) and the uterus. The fetus (f) can be seen. This hypoechoic area is the typical appearance of abruption. C. Large, extensive ultrasonographic preplacental collection (c) beneath the chorionic plate, amniotic fluid (f), and placenta (p). D. Thickened placenta (p) with heterogenous appearance. The arrowheads point to areas of hemorrhage. (Reprinted with permission from Yeo L, Ananth CV, Vintzileos AM. Placental abruption. In: Sciarra J, editor. Gynecology and obstetrics. Vol 2. Hagerstown (MD). Lippincott, Williams & Wilkins; 2003. © 2003 Lippincott Williams & Wilkins. and Oyelese. Placental Abruption. Obstet Gynecol 2006. From: Oyelese: Obstet Gynecol, Volume 108(4), October 2006:1005-1016 and Jaffe MH. Sonography of Abruptio Placentae. AJR. 1981;137:1049.)

Unlike placenta previa, abruption cannot be ruled out using ultrasound. Placental abruption is primarily a clinical diagnosis. Ultrasound is purely an adjunct in the diagnosis and helps exclude other causes of vaginal bleeding such as placenta previa. Symptomatic or even fetus-threatening abruption can occur in the presence of a negative sonogram, and sonographic evidence of placental hemorrhage is found in only 50% of abruptions. If complete escape of the blood occurs, ultrasound examination of the placenta may be completely normal. In addition, an acute abruption may appear similar to other soft tissue abnormalities of the uterus such as leiomyomata. In these cases, Doppler color flow analysis may prove helpful. A contained acute hemorrhage will appear hypoechoic or isoechoic when compared to the placenta. In some cases of acute hemorrhage, the placenta may only appear thickened or spherical in appearance. (See Figure 3.) Ultrasound may be helpful in identifying the size and location of an abruption, which can have prognostic significance. There are 3 predominant locations: subchorionic (which is associated with less than 10% fetal mortality), retroplacental (which is associated with greater than 50% fetal mortality), and preplacental.

**Treatment**

**Hypertensive Disorders**

For any patient who presents with a hypertensive disorder of pregnancy, treatment is guided by the degree of blood pressure elevation, signs of end-organ dysfunction, and the presence of severe preeclampsia or eclampsia. Accurate determination of gestational age by ultrasonography is needed to help direct management if symptoms progress. Definitive treatment is delivery of the fetus, but it must be weighed against the risks of prematurity to the fetus.
Chronic hypertension without superimposed preeclampsia triples the risk for perinatal mortality, doubles the risk for placental abruption and increases the risk for fetal growth retardation.79

In the Cochrane Database of Systemic Reviews, the role of antihypertensive treatment for mild to moderate hypertension has been reviewed.79 Forty-six trials with over 4000 women were included. Antihypertensive therapy results in a 50% risk reduction of developing severe hypertension during pregnancy but no reduction in the risk of developing preeclampsia. There also was no reduction in the risk of fetal mortality, small for gestational age infants, or preterm delivery. Among anti-hypertensive agents, β-blockers appeared to be better than methyldopa for reducing the risk of severe hypertension, but no differences were found among any of the drugs studied in the risk of developing preeclampsia. The authors concluded that it is unclear whether antihypertensive therapy should be initiated for mild to moderate hypertension during pregnancy. The authors did suggest that a moderate but clinically important reduction in the risk of progression to preeclampsia may not have been detected. The Cochrane review specifically addresses outpatient treatment with oral BP medications and gestational hypertension. It does not address severely elevated blood pressure and the need for acute treatment. Decisions about initiating antihypertensive therapy for individual patients should include a discussion with their obstetrician, as this is outside the usual scope of ED practice.

For patients with chronic hypertension and a systolic blood pressure greater than 180 mm Hg or a diastolic blood pressure greater than 110 mm Hg, there is general consensus that treatment is warranted, even in the absence of preeclampsia.6,7,86 The objective of treatment is to minimize the risk of end-organ damage without compromising cerebral and uteroplacental blood flow. Systolic and diastolic blood pressure should be maintained below 160 and 105 mm Hg respectively.6,7

IV labetalol and hydralazine have been used as first-line treatment for severely elevated blood pressure in pregnancy. (See Table 9.) Oral nifedipine may be an alternative if immediate intravenous access cannot be established. Blood pressure is often controlled with these regimens, negating the need for more aggressive therapy with nitroprusside or nitroglycerin IV. Although there is no direct evidence against their use, diuretics are generally avoided unless pulmonary edema is present.89

Treatment of very high blood pressure in pregnancy was reviewed in the Cochrane Database with the most recent in 2007.87 (See Table 10 for list of medications.) All drugs reviewed reduced blood pressure, but magnesium sulfate and ketaserin appeared to be the least effective of those studied. Diazoxide and nimodipine also had disadvantages. Diazoxide given in 75 mg IV boluses was associated with a precipitous drop of blood pressure that required treatment. Nifedipine was less effective in controlling blood pressure and was found to be associated with an increased risk of preeclampsia. Other than the medications listed above, the authors concluded that there is insufficient evidence to recommend using one drug over another. Picking a drug should be based on the patient, risks to the fetus, and side effect profile. The Cochrane authors also suggested that severely elevated blood pressure should be treated with IV medications.

### Table 9. Common Protocols For Antihypertensive Agents In Patients With Severe Hypertension In Pregnancy88

<table>
<thead>
<tr>
<th>Agent</th>
<th>Protocol</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labetalol</td>
<td>20 to 40 mg IV every 10 minutes until goal blood pressure achieved Or 20 mg IV doubled every 10 minutes to max of 80 mg IV until goal blood pressure achieved</td>
<td>Flushing, light headedness, palpitations, scalp tingling</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>5 mg IV every 10 minutes, May increase to 20 mg IV until goal blood pressure achieved Or 10 mg IM followed by 10 mg increases every 15-20 minutes to a maximum of 30 mg IM</td>
<td>Headache, flushing, lightheadedness, nausea, palpitations</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>10 to 20 mg orally every 30 minutes for maximum dose of 50 mg in one hour or goal blood pressure achieved</td>
<td>Flushing, nausea, vomiting</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>0.25 mcg/kg/min IV titrated every 3 to 5 minutes for desired BP to a maximum dose of 5 mcg/kg/min</td>
<td>Cyanide production</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>5 mcg/min IV titrated every 3 to 5 minutes for desired blood pressure to a maximum dose of 100 mcg/min.</td>
<td>Headache, tachycardia, methemoglobinemia, increased intracranial pressure</td>
</tr>
</tbody>
</table>

**Preeclampsia**
Once the diagnosis of preeclampsia is made, the severity of blood pressure elevation, degree of end-organ dysfunction, presence of eclampsia, and fetal well-being will guide management. Severe or fulminant preeclampsia with marked blood pressure elevation (>160/110 mm Hg) is managed in the same way as eclampsia. The goal of therapy is prevention of seizures and permanent damage to maternal organs. Severely elevated blood pressure is often treated with IV hydralazine and labetalol with a goal of maintaining blood pressure below 160/100 mm Hg. Bolus therapy is often adequate and IV drips are rarely needed.

Magnesium administered as a loading dose of 4 to 6 g IV followed by 2 g IV/hour is recommended. Magnesium administration should be accompanied by clinical observation for loss of reflexes (which occurs at a serum level of about 10 mg/dL) or respiratory depression (which occurs at levels over 12 mg/dL). In the setting of normal renal function, magnesium infusions are considered safe and levels can be reliably followed clinically, making routine magnesium serum level monitoring unnecessary. The infusion should be stopped if signs of hypermagnesemia are seen; such patients may require assisted ventilation. Calcium gluconate (1 gram given slowly into a secure vein) will reverse the adverse effects of hypermagnesemia.10

**HELLP Syndrome**
The diagnosis of HELLP syndrome mandates aggressive management to minimize the associated fetal and maternal morbidity and mortality.67 Assess the central nervous system and respiratory status, since both are at risk for complications. Start maintenance IV fluid and monitor urine output. Give IV magnesium sulfate as prophylaxis for eclamptic seizures. Treat severe hypertension in a similar fashion to severe preeclampsia. Transfuse blood products for profound thrombocytopenia (<20,000/mm3), marked anemia, or symptomatic coagulopathy.90 Intravenous steroids should also be considered, but data showing efficacy in HELLP syndrome is conflicting. If the pregnancy is <34 weeks, steroid administration may augment fetal lung maturity and reduce the risk of neonatal intracerebral hemorrhage due to prematurity.91 A Cochrane review found that dexamethasone was no better than placebo in reducing maternal mortality or morbidity associated with placental abruption, pulmonary edema, and maternal hematoma or rupture.91 In addition, no differences were found in fetal mortality or morbidity associated with respiratory distress syndrome, need for ventilatory support, intracerebral hemorrhage, necrotizing enterocolitis, or a 5-minute Apgar of less than 7. However, there was a statically significant decrease in the number of hospitalized days and an increase in the mean interval hours to delivery. There was also a trend towards an increased platelet count at 48 hours.

**Eclampsia**
The seizures of eclampsia are typically self-limited.100 Maintain the airway and support vital signs as needed. Perform frequent neurologic evaluations.

---

**Table 10. Benefits And Harms of Antihypertensive Agents During Pregnancy**

<table>
<thead>
<tr>
<th>Agent/Class</th>
<th>Benefits</th>
<th>Harms</th>
<th>Clinical Experience</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methyl dopa</td>
<td>Insufficient evidence to conclude effect on maternal/fetal morbidity or mortality.</td>
<td>Evidence of no major fetal adverse events. Risk of maternal hepatitis.</td>
<td>Large</td>
</tr>
<tr>
<td>β-blockers</td>
<td>Insufficient evidence to conclude effect on maternal/fetal morbidity or mortality.</td>
<td>Limited evidence of possible fetal growth restriction with atenolol in early pregnancy. Evidence of no major maternal adverse events.</td>
<td>Large</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Insufficient evidence to conclude effect on maternal/fetal morbidity or mortality.</td>
<td>Evidence of no major fetal or maternal adverse events.</td>
<td>Large</td>
</tr>
<tr>
<td>α-β Blockers</td>
<td>Insufficient evidence to conclude effect on maternal/fetal morbidity or mortality.</td>
<td>Limited evidence of no major fetal or maternal adverse events.</td>
<td>Small</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>Insufficient evidence to conclude effect on maternal/fetal morbidity or mortality.</td>
<td>Limited evidence of no major fetal or maternal adverse events.</td>
<td>Small</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Insufficient evidence to conclude effect on maternal/fetal morbidity or mortality.</td>
<td>Evidence of no major fetal or maternal adverse events.</td>
<td>Moderate</td>
</tr>
<tr>
<td>Angiotensin converting enzyme inhibitors</td>
<td>Insufficient evidence to conclude effect on maternal/fetal morbidity or mortality.</td>
<td>Limited evidence of no major maternal adverse events. Risk of fetal renal failure if used in the latter half of pregnancy.</td>
<td>Small</td>
</tr>
<tr>
<td>Angiotensin receptor blockers</td>
<td>No evidence.</td>
<td>No evidence.</td>
<td>None</td>
</tr>
</tbody>
</table>

Clinical Pathway For Management of Hypertensive Disorders Of Pregnancy

Patient presents with hypertension during the second half of pregnancy (after 20 weeks gestation).

Is proteinuria present to confirm the diagnosis of preeclampsia? (A single dip is not sufficient. A 24-hour urine sample is the most reliable means of documenting proteinuria.)

YES

Is the BP > 160/110 mm Hg?

NO

If asymptomatic, discharge home with close obstetric follow up in 1-2 days.

YES

Admit for observation and monitoring to obstetric unit.

Is there evidence of new end-organ dysfunction or fetal distress?

YES

Treat with IV medications and admit to the obstetric unit.

NO

Treat BP with oral agents and monitor.

Is BP controlled with intervention?

YES

Discharge home with close obstetric follow up in 1-2 days.

NO

If the BP > 160/110 mm Hg?

NO

If asymptomatic, discharge home with close obstetric follow up in 1-2 days.

YES

Admit to obstetric unit for further care.

Is there evidence of new end-organ dysfunction or fetal distress?

These clinical pathways are intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with these pathways does not represent a breach of the standard of care.

If seizures persist after the recommended doses of magnesium sulfate have been administered, give further anticonvulsant therapy in conjunction with obstetric consultation and a careful search for other causes (eg, hypoglycemia, intracranial bleed). If recurrent seizures do occur, consider adjunctive agents. Although phenytoin has been commonly used as an adjunctive agent for eclamptic seizures, consider fosphenytoin due to its better safety profile.

The occurrence of eclamptic seizures is reduced by magnesium prophylaxis in severe preeclampsia. 92,93 Multiple studies and reviews have evaluated the effectiveness of magnesium sulfate as prophylaxis against developing eclampsia in severe preeclampsia. 94-97 In the largest prospective multi-center placebo controlled study to date, the MagPie investigators found a significant reduction in the rate of eclampsia in women assigned to the magnesium sulfate group. In a Cochrane review of magnesium sulfate use for preventing eclampsia, the researchers concluded also that magnesium sulfate provided a 50% risk reduction of developing eclampsia and a reduced the risk of ma-
Excessive intravenous fluids increase extravascular fluid stores that are difficult to mobilize postpartum, resulting in a higher incidence of pulmonary edema in patients treated aggressively with fluid therapy. Invasive pulmonary artery pressure monitoring may be required for accurate fluid management in the eclamptic patient.

Vaginal Bleeding

Patients who experience vaginal bleeding during late pregnancy require immediate obstetric consultation. Make arrangements for safe transfer to an appropriate obstetric facility if a benign source of bleeding cannot be identified. Emergency department management consists of maternal stabilization, with establishment of 2 large-bore intravenous lines and fluid resuscitation as well as continuous fetal monitoring if available.

Blood loss requiring transfusions can occur in patients with placenta previa or placental abruption. Fresh frozen plasma or fresh whole blood may be needed because of the coagulopathy associated with significant placental abruption.
Clinical Pathway For Treatment Of Eclamptic Seizures During Pregnancy

Patient presents with generalized tonic-clonic seizure during the latter half of pregnancy (after 20 weeks gestation).

Support patient’s ABCs and protect from further harm. Check blood glucose.

Administer one ampule of D50W IV and monitor response.

Is blood glucose > 60 mg/dL?

YES → Initiate magnesium sulfate therapy (4-6 gm IV bolus over 10-20 minutes followed by a drip at 2 gm/hr).

NO → Continue close monitoring for magnesium toxicity.

Do seizures recur?

YES → • Consider second line agent such as valium or phenytoin.
      • Initiate further workup for other causes of seizures.

NO →

Because only 0.1 mL of fetal cells is required to sensitize the mother, routine immunoglobulin administration has been recommended in situations likely to result in sensitization. Patients with third-trimester bleeding are not at increased risk of sensitization compared with patients with normal pregnancy; this suggests that RhoGAM need only be administered if the patient did not receive a prophylactic dose at 28 weeks.104 The standard dose (300 µg) is sufficient to prevent maternal immunization for fetal transfusions of up to 15 mL of red blood cells or 30 mL of whole blood.105 If transfer to another hospital is required, a high-risk transfer team should be used if bleeding is significant or if the fetus is in distress. Although the bleeding source may not be identified or may be relatively benign, assessment is best accomplished by obstetricians who are accustomed to evaluating late-pregnancy complications and who can perform emergent cesarean section if needed.

In cases of term or near-term placental abruptions and no evidence of fetal or maternal instability, prompt delivery is indicated. Delivery is often most expeditious by the vaginal route, but determination must be made if vaginal delivery can occur without risk of significant maternal or fetal morbidity or mortality. If the decision to attempt vaginal delivery is made, close observation is needed to identify a change in fetal or maternal status that would necessitate an immediate cesarean section. In cases of fetal or maternal compromise when delivery is not imminent, cesarean section is indicated. At gestational ages prior to 34 weeks where the mother and fetus are stable, the patient should be managed conservatively. Administer steroids to enhance fetal lung maturity and consider tocolysis. The risk of prematurity must be discussed with the family. If the mother or fetus becomes unstable, immediate delivery is indicated.
Disposition

The disposition of a patient who presents with a complication of late pregnancy is determined by the suspected diagnosis, estimated gestational age, and the documentation of fetal well-being. If the pregnancy is near term, consider admission and delivery in almost all cases. If there are contraindications to vaginal delivery or an urgent delivery is required due to maternal or fetal instability, a caesarean delivery is typically indicated. If the pregnancy is less than 24 weeks and the mother is clinically stable, close observation and conservative management is the most appropriate course of action.

The disposition of patients with hypertensive disorders of pregnancy depends on the degree of blood pressure elevation. Patients with mild elevations of blood pressure and no evidence of proteinuria may be managed as outpatients with close follow up. Patients with severely elevated blood pressure with no evidence of preeclampsia, no evidence of end-organ dysfunction, and close follow up may be discharged home if their blood pressure can be reduced and maintained to minimally elevated levels. Once the diagnosis of preeclampsia is made, the patient should be admitted to the hospital for further stabilization and management. Patients with eclampsia are typically delivered after a period 24 to 36 hours for stabilization. If severe preeclampsia develops, management is based on gestational age. If the pregnancy is < 34 weeks, management is based on whether the blood pressure can be controlled and fetal well-being established. If fetal well-being or maternal instability exists, expedient delivery is the typical course of action.

For cases of severe preeclampsia, HELLP syndrome, or eclampsia, preparations for admission should be made to a high-risk obstetric unit. Delivery via cesarean section is often mandated if fetal compromise is identified. If a higher level of care is required, stabilize the patient to the capabilities of the facility and arrange for transportation to a tertiary care center.

Every patient with placenta previa who presents with symptomatic vaginal bleeding should be admitted for a period of observation and/or delivery. In patients between 24 and 36 weeks gestation, conservative management in the hospital consisting of bed rest, possible tocolysis, and steroid administration is warranted if the bleeding is mild or resolves, fetal well-being is documented, and maternal stability is ensured. If any of the above cannot be confirmed, delivery via cesarean section must be considered. Despite the fact that approximately 60% of patients between 24 weeks and 36 weeks gestation with placenta previa will have recurrent bleeding, many may qualify for a trial of outpatient management but only after a period of inpatient stabilization.106-109

Any patient with a suspected placental abruption based on clinical presentation should be hospitalized. In any patient less than 34 weeks gestation with maternal stability and reassuring fetal monitoring, the pregnancy may be managed conservatively in the hospital.110,111 Preterm birth is the

Risk Management Pitfalls

1. New onset seizures that occur between 48 hours and 4 weeks after delivery should be considered late onset eclampsia until proven otherwise.
2. Although a patient with new onset preeclampsia may look well, there is no way to adequately predict who will progress to eclampsia, thus they all should be considered for admission.
3. If a pregnant patient in the latter half of pregnancy presents with hypertension, a normal urinalysis does not rule out the development of preeclampsia. A 24-hour urine sample is the most reliable means of documenting proteinuria.
4. Do not rely on a normal ultrasound to rule out a placental abruption. An ultrasound is insufficient to diagnosis abruption. Therefore, placental abruption is a clinical diagnosis.
5. Know the capabilities of your obstetric facilities. If you are unable to provide care, stabilize the patient to the ability of the facility and transfer the patient to a facility with capability of providing high-risk obstetric care.
6. In any patient with severe preeclampsia or eclampsia, magnesium is the drug of choice to prevent first time and recurrent seizures.
7. Not all placental abruptions present with vaginal bleeding. A patient with a concealed abruption may present with fetal distress, abdominal pain, and in severe cases maternal shock.
8. Be wary of HELLP syndrome…patients may appear clinically well, but any patient who presents with preeclampsia in the latter half of pregnancy should be screened with labs to exclude HELLP syndrome.
9. Work with obstetric services. Often critically ill patient will end up in the ED prior to transfer to an obstetric unit. Having clearly defined protocols and expectations will improve care in this critically ill population.
leading cause of neonatal morbidity and mortality in patients with an abruption, thus prolonging the pregnancy is desirable. The patient should be admitted for tocolysis and steroid administration to promote fetal lung maturity. Outpatient management is not generally recommended for placental abruption, but a small subset in a study of 72 women with an abruption had a trial of outpatient management. Under strict criteria, they found that outpatient management was safe and allowed time for fetal lung maturity and advancement of gestational age.

Summary

The complications of late pregnancy are varied. ED physicians must be vigilant in their assessment of any patient who presents to the emergency department in late pregnancy or in the early postpartum period. Consultation with an obstetrician is required, and admission may be necessary. The emergency physician must always consider the mother as well as the fetus when making any management decision. Successful treatment outcomes are maximized with a thorough evaluation, an understanding of the management options, good communication, and coordinated care with the obstetrician.

Case Conclusion

Before the PICU nurses leave, you ask if they know this patient. They say that the patient was visiting her 2-week-old daughter in the neonatal step down unit. Hearing this information, you quickly cancel the order for IV lorazepam and instead order 5 grams of IV magnesium. You order a complete blood count, an electrolyte panel, a coagulation panel, liver function studies, a urinalysis, a urine drug screen, and consider a head CT. After the IV magnesium sulfate is administered, the patient slowly wakes up from her seizure and her blood pressure improves. A repeat neurologic examination shows no focal findings, but she clearly has hyperactive reflexes. The patient’s labs show a creatinine of 1.3, +3 protein in the urine, and a mild anemia with normal platelets. A diagnosis of postpartum eclampsia is made and the patient is subsequently admitted to the obstetric unit where she had an uncomplicated course.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trials should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available.


81. Joint letter of Bristol-Myers Squibb Company; Ciba-Geigy Corporation, Pharmaceutical Division; Hoechst-Roussel Pharmaceuticals Inc; ICI Pharmaceutical Group, ICI Americas Inc; Meck Human Health Division. (Systemic Review)


86. Duley L, Henderson-Smart DJ, Meher S. Drugs for Treatment of Very High Blood Pressure During Pregnancy. The Cochrane Database of Systemic Reviews. 2007. (Systemic Review)


3. Which of the following statements regarding placenta previa is TRUE?
   a. A transabdominal ultrasound is more accurate that a transvaginal ultrasound for identifying placenta previa.
   b. Placenta previa is the premature separation of the placenta from the uterine wall.
   c. Placenta previa identified early in the pregnancy may resolve as the pregnancy progresses
   d. Previous cesarean sections do not increase the risk for placenta previa in subsequent pregnancies.

4. Which of the following statements regarding placental abruption is FALSE?
   a. Placental abruption accounts for approximately one-third of vaginal bleeding episodes during the second half of pregnancy.
   b. Maternal age is a risk factor for development of a placental abruption.
   c. The largest single risk factor for development of placental abruption is a history of prior abruption.
   d. Marginal separations of the placenta may go undetected until the placenta is examined at delivery.
   e. Ultrasound can be used to rule out abruption.

5. For the patient who presents to the ED with suspected placental abruption based on clinical presentation, which of the following is TRUE?
   a. All patients can be safely discharged since it is a relatively minor complication.
   b. If fetal heart tones are within normal limits, no ultrasound is needed and the patient may be discharged.
   c. If an ultrasound is normal, placental abruption can be completely ruled out.
   d. Not all placental abruptions present with vaginal bleeding.
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Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NHHS, and ACEP; and evaluation of prior activities for emergency physicians.
Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.
Goals & Objectives: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medical problems for each topic covered.
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In compliance with all ACCME Essentials, Standards, and Guidelines, all faculty for this CME activity were asked to complete a full disclosure statement. The information received is as follows: Dr. Keadey, Dr. Houry, Dr. Hahn, Dr. Lukens and their related parties report no significant financial interest or other relationship with the manufacturer(s) of any commercial product(s) discussed in this educational presentation.
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Complications In Pregnancy Part II: Hypertensive Disorders Of Pregnancy And Vaginal Bleeding
Keadey M, Houry D. May 2009; Volume 11, Number 5

This issue of Emergency Medicine Practice focuses on hypertensive disorders of pregnancy and vaginal bleeding. For a more detailed discussion of this topic, including figures and tables, critical appraisal of the literature, and risk management pitfalls, please see the complete issue at www.EBmedicine.net.

### EVIDENCE-BASED CLINICAL RECOMMENDATIONS FOR PRACTICE

<table>
<thead>
<tr>
<th>Hypertensive Disorders Of Pregnancy</th>
<th>References*</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>If a pregnant patient in the latter half of pregnancy presents with hypertension, a single sample, normal urinalysis does not rule out the development of pre eclampsia.</td>
<td>3-7</td>
<td>Due to discrepancies between random urine protein and 24-hour samples, it is generally advised that the diagnosis be made based on a 24-hour sample. Any patient with newly diagnosed preeclampsia should be admitted to the hospital for further management and stabilization.</td>
</tr>
<tr>
<td>Severe preeclampsia is characterized by systolic blood pressure &gt;160 mm Hg, diastolic blood pressure &gt; 110 mm Hg, and proteinuria &gt; 5 gm on a 24-hour sample.</td>
<td>3-7</td>
<td>Delivery may be necessary in patients with severe preeclampsia despite the associated neonatal morbidity and mortality.</td>
</tr>
<tr>
<td>HELLP syndrome is a life-threatening obstetric complication usually considered to be a variant of preeclampsia.</td>
<td>66</td>
<td>HELLP syndrome develops in 5% to 10% of all preeclamptic patients.</td>
</tr>
<tr>
<td>Eclampsia is the occurrence of seizures in the patient with signs of preeclampsia.</td>
<td>55,57,58</td>
<td>Eclampsia may occur at any point during the puerperium period but is most likely to occur during the intrapartum period or within 48 hours after delivery.</td>
</tr>
<tr>
<td>Magnesium sulfate is the most effective treatment for prophylaxis and prevention of recurrent eclamptic seizures.</td>
<td>87</td>
<td>Magnesium sulfate is ineffective in controlling blood pressure that is significantly elevated in severe preeclampsia. Hydralazine and labetalol are 2 common agents that are safe and effective for control of severely elevated blood pressure in pregnancy.</td>
</tr>
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<table>
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<tr>
<th>Vaginal Bleeding During Pregnancy</th>
<th>References*</th>
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<tbody>
<tr>
<td>Placental abruption is the premature separation of the placenta from the uterine wall. It accounts for approximately one-third of vaginal bleeding episodes during the second half of pregnancy.</td>
<td>16,19</td>
<td>Small subclinical or marginal separations may go undetected until the placenta is examined at delivery. These separations probably account for many of the other self-limited episodes of bleeding for which no diagnosis is made.</td>
</tr>
<tr>
<td>The classic presentation of placental abruption includes vaginal bleeding, abdominal pain, uterine tenderness, and uterine irritability.</td>
<td>25,62</td>
<td>In a prospective study of patients with confirmed abruptions, vaginal bleeding was present in 78%, uterine tenderness in 66%, and uterine contractions in 34%.</td>
</tr>
<tr>
<td>Placenta previa is defined as the implantation of the placenta in the lower uterine segment and is responsible for potentially life-threatening antenatal and postpartum hemorrhage.</td>
<td>26,27,30,31</td>
<td>Placenta previa diagnosed prior to the 20th week of gestation will resolve in more than 90% of the cases. Although placenta previa is the most common cause of clinically significant third trimester uterine bleeding, it is a relatively rare condition, developing in only 2 to 5 of 1000 pregnancies.</td>
</tr>
<tr>
<td>The placental location in relationship to the cervical os is vital in making the diagnosis of placenta previa.</td>
<td>57</td>
<td>Ultrasonography is the diagnostic imaging modality of choice for localizing the placenta in any patient who presents with vaginal bleeding during the latter half of pregnancy.</td>
</tr>
<tr>
<td>Patients who experience vaginal bleeding during late pregnancy require immediate obstetric consultation.</td>
<td></td>
<td>Emergency department management consists of maternal stabilization, with establishment of 2 large-bore intravenous lines and fluid resuscitation as well as continuous fetal monitoring if available.</td>
</tr>
</tbody>
</table>

*See reverse side for reference citations.*
REFERENCES

These references are excerpted from the original manuscript. For additional references and information on this topic, see the full text article at ebmedicine.net.


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