Hypertensive Disorders of Pregnancy

by Sue Prophet, RRA, CCS, director of classification and coding

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Hypertension complicates at least 10 percent of all pregnancies. The hypertensive disorder of pregnancy most likely to result in significant adverse clinical outcomes is preeclampsia.

Preeclampsia
Preeclampsia is unique to pregnancy and is characterized by elevated blood pressure, proteinuria, and generalized edema. Coagulation (particularly thrombocytopenia) and/or liver function abnormalities may also occur. Preeclampsia is defined as maternal hypertension that develops between the 20th week of gestation and the end of the first week postpartum. It develops in 5 percent of pregnant women and occurs primarily in primigravidas. The cause of preeclampsia is unknown. It occurs more frequently in women with preexisting hypertension or vascular disease. A variety of abnormalities are apparent early in pregnancy in women destined to develop preeclampsia. Placental changes are evident by the 20th gestational week, and alterations in pressor responsiveness are noted soon after, each preceding overt disease by weeks to months. These observations, and the multisystem abnormalities suggestive of generalized endothelial cell dysfunction, underlie a working hypothesis whose central theme is that preeclampsia is the result of primary placental pathology. Then relative uteroplacental ischemia, production of circulating substances toxic to endothelium, increased vascular reactivity, coagulation changes, and glomerular endotheliosis follow. The cause of the placental disease is uncertain. The possibility of genetic or immunologic influences has been investigated. There are two hypotheses for a familial basis for preeclampsia. It has been suggested that it is due to a maternal recessive gene or the interaction between maternal and fetal recessive genes. It has also been suggested that susceptibility to the disease may be associated with a specific polymorphism in the angiotensinogen gene. Immune hypotheses include a breakdown of the usual adaptations in the immune system required to sustain a normal gestation. Immune-mediated injury would most likely occur in the placenta and/or the uteroplacental circulation. Support for this hypothesis derives from the observations that preeclampsia occurs primarily in nulliparous women, whose subsequent gestations tend to be uncomplicated, unless there is a change in paternity. Evidence suggests that the fetuses of preeclamptic mothers may be more genetically compatible with their mothers than fetuses in normal pregnancies. It has also been observed that the trophoblast and kidneys share some antigenic determinants that have been linked to proteinuric hypertensive disorders in animals and that certain pathologic changes in the placental vasculature resemble those of allograft rejection.

Compared with transient (nonproteinuric) hypertension of pregnancy, preeclampsia is associated with a higher incidence of adverse maternal/fetal outcomes, such as fetal growth retardation and maternal or fetal death. Preeclampsia is characterized by vasospasm which may lead to reduced perfusion of multiple organs including uterus, placenta, kidney, brain, and liver. The presentation of preeclampsia varies widely in severity from mild blood pressure elevation to malignant hypertension, which is associated with marked coagulopathy, convulsions, and liver and renal failure. Blood pressure elevations after midpregnancy in previously normotensive women are often the initial clue of impending preeclampsia. Women with mildly elevated blood pressure and even minimal proteinuria can rapidly progress to eclampsia. Thus even when the disorder is considered mild, close scrutiny and possibly even hospitalization is warranted.

The standard criteria for diagnosing hypertension in pregnancy are:

- Increases in systolic blood pressure of greater or equal to 30 mm Hg
- Increases in diastolic levels of greater or equal to 15 mm Hg compared to mean values recorded prior to 20 weeks gestation

The diagnosis of preeclampsia is uncertain in the absence of proteinuria. Abnormal protein excretion is defined as greater or equal to 300 mg/24 hr. However, proteinuria may develop as a late manifestation. Mild preeclampsia develops as borderline hypertension, unresponsive edema, or albuminuria.

Since edema frequently accompanies normal gestation, its presence alone does not necessarily indicate preeclampsia. Sudden and rapid weight gain does often precede overt manifestation of the disease. However, severe disease can occur even in the absence of edema.

Abruptio placentae is a potential complication of preeclampsia.

Delivery is the definitive cure for preeclampsia, although occasionally the disease appears to develop in the first postpartum week or to transiently worsen in the immediate postpartum period.

HELLP Syndrome

A severe complication of preeclampsia is HELLP syndrome, which stands for Hemolysis, Elevated Liver Enzymes, and Low Platelet Count. Patients with this disorder often present with right upper quadrant and epigastric pain and a peripheral blood smear consistent with a microangiopathic hemolytic anemia. There may be decreases in platelet counts and increases in transaminase and/or lactic acid dehydrogenase enzymes. This is a life-threatening emergency which requires prompt termination of the pregnancy. At delivery, platelets, fresh frozen plasma, and packed red blood cell transfusions may be required.

Infants born to patients with HELLP syndrome may have thrombocytopenia. Death rates of infants whose mothers have HELLP have been reported to be as high as 60 percent.

Chronic Hypertension

Chronic hypertension refers to high blood pressure present before pregnancy or diagnosed before the 20th week of gestation. It is usually due to essential hypertension. Generally, the maternal and fetal prognosis is good in cases of chronic hypertension. Women with chronic hypertension are at increased risk for the development of superimposed preeclampsia. Antihypertensive therapy cannot prevent the development of preeclampsia in a woman with chronic hypertension.

Chronic Hypertension with Superimposed Preeclampsia

The risk of superimposed preeclampsia in women with chronic hypertension is estimated to be 10 to 20 percent. Generally, increments of 30 mm Hg or more in systolic pressure and 15 mm Hg or more in diastolic pressure, along with the development of significant proteinuria, are enough to establish a clinical diagnosis. However, sometimes a diagnosis of preeclampsia is erroneous.

Women with chronic hypertension and superimposed preeclampsia are prone to experience increased morbidity, and even mortality, compared to "pure" preeclamptics, or those who were normotensive prior to conception.

Transient Hypertension in Pregnancy

Transient hypertension is the elevation of blood pressure (>140/90mm Hg) during pregnancy or in the first 24 hours postpartum without other signs of preeclampsia or preexisting hypertension. The hypertension usually resolves in days to weeks after delivery. Rarely, women will become persistently hypertensive following a pregnancy complicated by transient hypertension. This condition is thought to be a precursor to the development of essential hypertension later in life.

Eclampsia

Eclampsia is the convulsive form of preeclampsia. If untreated, preeclampsia will suddenly progress to eclampsia. Eclampsia is usually fatal if it is not treated.

Treatment for Preeclampsia and Eclampsia

The primary treatment for preeclampsia and eclampsia is delivery. Most physicians would choose to terminate the pregnancy in the presence of persistent severe hypertension (diastolic blood pressure greater or equal to 110 mm Hg), abnormal liver function tests, and low platelet count. Under these circumstances, conservative management is usually ineffective in improving fetal outcome and may result in serious maternal morbidity.

If the woman is a long way from term and has mild to moderate hypertension and no signs of deteriorating renal or hepatic function or coagulopathy, conservative treatment is generally attempted. This treatment may include antihypertensive therapy.

Diuretics are not used in the treatment of preeclampsia because they further upset the existing electrolyte imbalance and reduce both renal and uteroplacental perfusion. A low-salt diet is also of no benefit in the treatment of preeclampsia. Patients

require normal salt intake and increased water intake. Bed rest and lying on the left side increase urinary output and reduce intravascular dehydration and hemoconcentration. Severe preeclampsia is treated with intravenous magnesium sulfate, which decreases the risk of convulsions and lowers the blood pressure. Infusion of a balanced salt solution helps to increase urinary output and lessen edema. If the magnesium sulfate does not lower the blood pressure, intravenous hydralazine may be administered. If urinary output does not improve, furosemide may be administered intravenously to produce diuresis.

Eclampsia is also treated with magnesium sulfate to control the seizures. Studies have suggested that magnesium sulfate may work by inducing dilation of the intracranial vessels, and thus relieving vasospasm. If magnesium sulfate is unsuccessful in controlling the seizures, diazepam may also be given. Constant monitoring and attendance are required. Blood pressure, respirations, and reflexes are checked at regular intervals. The patient is regularly observed for complications such as blurring of vision, confusion, pain, vaginal bleeding, or loss of fetal heart tones.

There is evidence that populations with high dietary intake of calcium have a low incidence of preeclampsia and that preeclamptics demonstrate altered calcium regulation. Evidence also indicates that calcium loading lowers the blood pressure in normotensive pregnant women. Preliminary studies suggest that calcium supplementation may lower blood pressure in pregnant women and prevent preeclampsia. The developing fetus imposes a significant demand on maternal calcium homeostasis during a normal pregnancy, particularly late in gestation. Approximately 200 mg/day of calcium are deposited in the fetal skeleton during the third trimester, with a net accumulation of 25 to 30 g. In addition, urinary hypercalciuria of pregnancy is mostly due to a combination of increased gastrointestinal absorption of calcium and an increased glomerular filtration rate. Despite the increased transfer of calcium to the fetal skeleton, and increased urinary calcium excretion, there is little evidence that pregnant women suffer from skeletal demineralization. Maternal adjustments in calcium metabolism that occur during pregnancy compensate, at least in part, for fetal calcium demands and urinary losses.

Epidemiological evidence links dietary calcium deficiency to hypertensive disorders of pregnancy, and it has been observed that calcium deficiency may play a role in subgroups of patients with essential hypertension. Abnormalities in cellular calcium metabolism (for example, in platelets) have also been reported in women with preeclampsia.

A few small clinical trials have shown that low-dose aspirin (50 to 150 mg daily) administered early in pregnancy may prevent preeclampsia.

After delivery, the patient must be monitored carefully and frequently. Twenty-five percent of eclampsia occurs during the postpartum period, usually in the first two to four days. The blood pressure might remain elevated for as long as six to eight weeks, but if it remains elevated beyond this period of time, a possible diagnosis of hypertension should be considered.

Code Assignments

Hypertension complicating pregnancy, childbirth, and the puerperium is classified to category 642, Hypertension complicating pregnancy, childbirth, and the puerperium. No additional code from category 401 is necessary because the 642 codes adequately describe the specific form of hypertension. The appropriate fifth digit should be assigned to the 642 codes to indicate the episode of care.

Benign essential hypertension complicating pregnancy is assigned 642.0x, Benign essential hypertension complicating pregnancy, childbirth, and the puerperium. This code includes essential, chronic, or preexisting hypertension that is not specified as malignant.

Hypertension secondary to renal disease in pregnancy is assigned 642.1x, Hypertension secondary to renal disease, complicating pregnancy, childbirth, and the puerperium.

All other preexisting hypertension complicating pregnancy, including hypertensive heart disease, hypertensive renal disease, hypertensive heart and renal disease, and malignant hypertension, is assigned 642.2x, Other pre-existing hypertension complicating pregnancy, childbirth, and the puerperium.

Transient hypertension, or gestational hypertension, is assigned code 642.3x, Transient hypertension of pregnancy.

Preeclampsia is classified to codes 642.4x and 642.5x. Mild or unspecified preeclampsia is assigned code 642.4x. Severe preeclampsia and HELLP syndrome are assigned 642.5x, Severe preeclampsia. Eclampsia, or eclamptic toxemia, is classified to code 642.6x, Eclampsia.

Preeclampsia or eclampsia superimposed on preexisting hypertension is assigned 642.7x.

Hypertension unspecified as to type, but complicating pregnancy, childbirth, or the puerperium, is assigned 642.9x.

Note: The clinical information provided above is intended to advance understanding of the disease process of preeclampsia. It is the attending physician's responsibility to determine and document that a given patient has this diagnosis. If the medical record documentation is unclear as to the patient's diagnosis, query the physician.

References

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