Obstetrics: Hypertension in Pregnancy, Preeclampsia, and Eclampsia

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Peer Review Status: Externally Peer Reviewed by Mosby
University of Iowa Family Practice Handbook, 3rd Edition, Chapter 8

I. Pregnancy-Induced Hypertension (PIH)

A. **Definition.** Hypertension in pregnancy is present when diastolic BP >90 mm Hg, systolic BP >140; systolic BP rises at least 30 mm Hg over baseline value or diastolic BP rises at least 15 mm Hg over baseline value.

B. **Risk factors for PIH.** First pregnancy, multiple gestation, polyhydramnios, hydatidiform mole, malnutrition, positive family history of PIH, underlying vascular disease. Molar pregnancy should be expected if PIH occurs early in gestation.

II. Preeclampsia and Eclampsia

A. **Preeclampsia.** Defined as the presence of hypertension or PIH accompanied by proteinuria, edema, or both. Preeclampsia is divided into mild and severe forms.

1. **Criteria for mild preeclampsia.**

a. Hypertension as defined above but not meeting the criteria for severe preeclampsia.
b. Proteinuria >300 mg/24 hours.
c. Mild edema, signaled by weight gain >2 lb/week or >6 lb/month.
d. Urine output >500 ml/24 hours.

2. **Criteria for severe preeclampsia.**

a. The presence of any of the systemic symptoms noted below categorizes the patient as having severe preeclampsia regardless of the blood pressure.
b. BP >160/110 on 2 occasions at least 6 hours apart with patient on bed rest.
c. Systolic BP rise >60 mm Hg over baseline value.
d. Diastolic BP rise >30 mm Hg over baseline value.
e. Proteinuria >5 g/24 hours or 3+ or 4+ on urine dipstick.
f. Massive edema.
g. Oliguria <400 ml/24 hours.
h. Systemic symptoms including pulmonary edema, headaches, visual changes, right upper quadrant pain, elevated liver enzymes, or thrombocytopenia.
i. Presence of IUGR in fetus.

B. **Eclampsia. Occurrence of a seizure that is not attributable to other causes in a preeclamptic patient.**

III. Evaluation of Pregnancy-Induced Hypertension and Preeclampsia

A. **History.** Document risk factors and any symptoms outlined above.

B. **Physical.** Look for evidence of edema (particularly of the hands and face), BP changes, retinal changes, hyperreflexia, clonus, and RUQ tenderness.

C. **Initial laboratory studies.**

1. **Blood.** CBC, electrolytes, BUN and creatinine, uric acid, liver function tests (AST, ALT, LDH), and coagulation studies (PT, PTT, and fibrinogen degradation products). If patient is in labor, send a blood type and screen.
2. **Urine.** 24-hour collection for protein and creatinine clearance.
3. **HELLP syndrome.** Hemolyis; elevated liver function tests; low platelet count.
4. **Lab test results that may be abnormal. Uric acid (>5.5 ng/dl) may be elevated before there are other signs or symptoms of preeclampsia,** elevated or normal hemoglobin, creatinine >1.0 ng/dl, BUN >10 ng/dl, or decreased platelet count, hypoalbuminemia, increased LDH or AST, elevated fibrin degradation products, prolonged PT/PTT, schistocytes or helmet cells on peripheral smear (hemolysis).

D. **Complications of preeclampsia.** Eclamptic seizures, HELLP syndrome, hepatic rupture, DIC, pulmonary edema, acute renal failure, placental abruption, intrauterine fetal demise (IUFD), cerebral hemorrhage, cortical blindness, retinal detachment.

IV. **Management of PIH/Preeclampsia**

A. **Ambulatory management.** For pregnancy-induced hypertension without significant proteinuria, home bed rest is recommended. Home blood pressure monitoring, weight, and urine protein checks are helpful. It is generally recommended that antepartum surveillance (NST) begin early. Ultrasound exams should be performed periodically to ensure adequate amniotic fluid and to monitor for intrauterine growth retardation (IUGR).

B. **Hospital management.**

1. **Indications.** For women with pregnancy-induced hypertension and 2+ or greater proteinuria and for those who fail outpatient management. Bed rest with bathroom privileges is allowed. The goal of IV fluids in severe cases is to replace urine output and insensible losses.

2. **Laboratory evaluation and weights.** Performed daily to every other day. Antepartum surveillance including daily fetal movement count, daily NSTs, and weekly amniotic fluid determinations by ultrasonography is essential. Monitor symptoms such as headache, visual disturbances, epigastric pain.

3. **Delivery is treatment of choice.** Delivery should be accomplished when the fetus is mature but may be required early if maternal health is in danger or if there is evidence of fetal distress. Delivery is indicated when the patient meets criteria for severe preeclampsia. Betamethasone 12.5 mg IM given twice 24 hours apart to stimulate fetal lung maturation can be repeated weekly if pregnancy is prolonged. Electronic FHR monitoring during labor is indicated.

C. **Antihypertensive therapy.**

1. Indicated only if BP persistently >160/110.

2. Aim for a diastolic BP 90 to 100 mm Hg. Avoid overcorrection because normal blood pressures can result in placental hypoperfusion.

3. Diuretics are never indicated. These patients are already hypovolemic. ACE inhibitors are also not to be used during pregnancy.

4. Long-term medications (if the fetus is immature) include methyldopa (Aldomet), atenolol, and labetalol.

D. **Anticonvulsive therapy.**

1. **Seizure prophylaxis** is indicated in all preeclamptic patients during labor and delivery and for a minimum of 24 hours post partum. Some perinatologists continue IV magnesium therapy in preeclamptic patients until the patient begins to diurese. Seizures may occur in the absence of hyperreflexia, and increased DTRs may be present in the normal population; therefore, hyperreflexia is not a useful predictor of risk.

2. **Magnesium sulfate** is the drug of choice.

a. Loading dose for seizure prophylaxis is 4 to 6 g of magnesium sulfate IV over 20 minutes and continued at 2 g/hour.

b. **Treatment of seizures.** Magnesium sulfate 1 g/min IV until seizure controlled up to 4 to 6 g maximum. If this fails, see Chapter 1 for management of status epilepticus.

c. **Serum levels.** Therapeutic level is 4 mEq/L (takes 12 to 18 hours to equilibrate). Serum levels of magnesium sulfate are of dubious value, since infusions rarely go longer than 2 days. Monitor urine output (100 ml in 4 hours), presence of deep tendon reflexes.

d. Magnesium toxicity may be signaled by excessive drowsiness and absence of patellar reflexes. At levels of 10 to 12 mEq/L and above, muscle weakness, respiratory paralysis, and cardiac depression can occur. 10 ml of 10% calcium gluconate (or calcium chloride) may be administered IV push in the event of magnesium toxicity, or the infusion can be turned off for 1 to 2 hours.

e. Continue magnesium sulfate therapy at least 24 hours post partum. In 25% of the patients postpartum eclampsia can occur. Monitor urine output and can stop therapy if urine output is >200 ml/hour for 4 consecutive hours. Watch for postpartum hemorrhage because magnesium sulfate can relax the uterus.

E. **Prevention.** Daily aspirin 81 mg can be given after the first trimester in women with chronic hypertension, previous history or preeclampsia, DM, and SLE. However, the efficacy of aspirin for this indication has been called into question. Several studies show that aspirin makes no difference in maternal or fetal outcomes. A recent meta-analysis indicates that calcium supplementation during pregnancy may reduce the risk of preeclampsia and hypertension during pregnancy. These results will need to be confirmed. However calcium supplementation is innocuous and should be considered.

V. **Chronic Hypertension**

A. **Risks.**
1. **Maternal.** If no superimposed preeclampsia occurs, there is no additional maternal risk. In the presence of superimposed preeclampsia (20%), there is increased maternal mortality, frequently from intracranial hemorrhage.
2. **Fetal.** There is an increased incidence of perinatal death, IUGR, and fetal distress.
B. **Management.**
1. Treatment of chronic hypertension can decrease maternal and, to some extent, fetal morbidity but cannot reduce the risks of superimposed preeclampsia. Appropriate medications include methyldopa, hydralazine, and beta-blockers.
2. During pregnancy, it is not appropriate to use:
a. Sympathetic ganglion blockers (orthostatic hypotension)
b. Diuretics (aggravation of volume depletion)
c. ACE inhibitors (associated with fetal defects and neonatal renal failure)
3. Laboratory evaluation is performed early in pregnancy.
4. Obstetric visits are scheduled every other week at 24 weeks and weekly after 30 weeks.
5. Early ultrasonogram is obtained for dating, and repeated periodically to look for evidence of IUGR.
6. Antenatal surveillance (NSTs) should begin at 34 weeks.
7. The pregnancy should not be allowed to go beyond 40 weeks. Delivery may be required earlier if there is evidence of IUGR or fetal distress or if hypertension cannot be controlled by bed rest and medication.
8. Intrapartum monitoring is required during labor.
9. If there is evidence of IUGR, cesarean section is preferable to a prolonged induction.
10. Complicated cases or women with superimposed preeclampsia should be handled at an appropriate referral center.

• Incidence and predictors of severe obstetric morbidity: case-control study

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Objective: To estimate the incidence and predictors of severe obstetric morbidity.
Design: Development of definitions of severe obstetric morbidity by literature review. Case-control study from a defined delivery population with four randomly selected pregnant women as controls for every case.
Setting: All 19 maternity units within the South East Thames region and six neighbouring hospitals caring for pregnant women from the region between 1 March 1997 and 28 February 1998.
Participants: 48 865 women who delivered during the time frame.
Results: There were 588 cases of severe obstetric morbidity giving an incidence of 12.0/1000 deliveries (95% confidence interval 11.2 to 13.2). During the study there were five maternal deaths attributed to conditions studied. Disease specific morbidities per 1000 deliveries were 6.7 (6.0 to 7.5) for severe haemorrhage, 3.9 (3.3 to 4.5) for severe pre-eclampsia, 0.2 (0.1 to 0.4) for eclampsia, 0.5 (0.3 to 0.8) for HELLP (Haemolysis, Elevated Liver enzymes, and Low Platelets) syndrome, 0.4 (0.2 to 0.6) for severe sepsis, and 0.2 (0.1 to 0.4) for uterine rupture. Age over 34 years, non-white ethnic group, past or current hypertension, previous postpartum haemorrhage, delivery by emergency caesarean section, antenatal admission to hospital, multiple pregnancy, social exclusion, and taking iron or anti-depressants at antenatal booking were all independently associated with morbidity after adjustment.
Conclusion: Severe obstetric morbidity and its relation to mortality may be more sensitive measures of pregnancy outcome than mortality alone. Most events are related to obstetric haemorrhage and severe pre-eclampsia. Caesarean section quadruples the risk of morbidity. Development and evaluation of ways of predicting and reducing risk are required with particular emphasis paid on the management of haemorrhage and pre-eclampsia.

What is already known on this topic

Maternal mortality is used internationally as a measure of the quality of obstetric intervention, although it is now rare in the developed world
Hospital based series estimating the incidence of severe obstetric morbidity have used different definitions

Estimated incidence of severe obstetric morbidity ranges from 0.05 to 1.09

What this study adds
With clear definitions and population based estimates of some severe obstetric morbidities this study estimated the overall incidence of severe obstetric morbidity as 1.2 % of deliveries
Two thirds of the cases are related to severe haemorrhage, one third to hypertensive disorders

Risk factors for severe maternal morbidity include maternal age >34, social exclusion, non-white, hypertension, previous postpartum haemorrhage, induction of labour, and caesarean section