



DETERMINATION OF RISK FACTORS OF HYPERTENSIVE DISORDERS IN PREGNANT WOMEN

Ataxodjayeva F.A.¹, Kayumova D.T², Nabiyeva R.M.³

¹t.f.d, dotsent.

TTA №-2 Obstetrics and Gynecology

²t.f.d., dotsent.,

TTA №-2 Obstetrics and Gynecology

³Tashkent Medical Academy

№-2 Department of Obstetrics and Gynecology

1st year master's student

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ABSTRACT

In this article determination of dangerous groups of hypertensive disorders in pregnant women. Hypertension is the most common medical problem encountered during pregnancy, complicating 2-3% of pregnancies. Pregnancy: 1) chronic hypertension, 2) preeclampsia-eclampsia, 3) preeclampsia superimposed on chronic hypertension, and 4) gestational hypertension (transient hypertension of pregnancy or chronic hypertension identified in the latter half of pregnancy) (1). This terminology is preferred over the older but widely used term pregnancy-induced hypertension (PIH) because it is more precise.

Introduction

Worldwide there is disagreement about many aspects of the classification, diagnosis, and management of the hypertensive disorders of pregnancy. This lack of consensus hampers our ability to study not only the immediate rates of adverse maternal and fetal outcomes for the various hypertensive disorders in pregnancy, particularly preeclampsia, but also the long-term health outcomes of women and babies who survive this condition. It also impacts on research into the pathophysiology of this condition and has almost certainly delayed the development of effective screening tests and treatments, leading to poorer pregnancy outcomes.

Classification

1. Hypertension in pregnancy may be chronic (predating pregnancy or diagnosed before 20 weeks of pregnancy) or de novo (either preeclampsia or gestational hypertension).

2. Chronic hypertension is associated with adverse maternal and fetal outcomes and is best managed by tightly controlling maternal blood pressure (BP, 110–140/85 mm Hg), monitoring fetal growth, and repeatedly assessing for the development of preeclampsia and maternal complications. This can be done in an outpatient setting.

3. White-coat hypertension refers to elevated office/clinic ($\geq 140/90$ mm Hg) BP, but normal BP measured at home or work



(<135/85 mm Hg); it is not an entirely benign condition and conveys an increased risk for preeclampsia.

4. Masked hypertension is another form of hypertension, more difficult to diagnose, characterized by BP that is normal at a clinic or office visit but elevated at other times, most typically diagnosed by 24-hour ambulatory BP monitoring (ABPM) or automated home BP monitoring.

5. Gestational hypertension is hypertension arising *de novo* after 20 weeks' gestation in the absence of proteinuria and without biochemical or hematological abnormalities. It is usually not accompanied by fetal growth restriction. Outcomes in pregnancies complicated by gestational hypertension are normally good, but about a quarter of women with gestational hypertension (particularly those who present at <34 weeks) will progress to preeclampsia and have poorer outcomes.

6. Preeclampsia is a complex medical disorder; worldwide, each year, it is responsible for >500 000 fetal and neonatal deaths and >70 000 maternal deaths. Preeclampsia can deteriorate rapidly and without warning; we do not recommend classifying it as mild or severe.

7. Proteinuria is not mandatory for a diagnosis of preeclampsia. Rather, this is diagnosed by the presence of *de novo* hypertension after 20 weeks' gestation accompanied by proteinuria and/or evidence of maternal acute kidney injury (AKI), liver dysfunction, neurological features, hemolysis or thrombocytopenia, or fetal growth restriction. Preeclampsia may develop or be recognized for the first time intrapartum or early postpartum in some cases.

8. The hemolysis, elevated liver enzymes, low platelets syndrome is a (serious) manifestation of preeclampsia and not a separate disorder.

Chronic Hypertension in pregnancy

Most studies have not found adverse pregnancy outcomes. Nonetheless, caution should be used in cases of impaired uteroplacental perfusion, such as preeclampsia or intrauterine growth restriction. Atenolol and other pure beta-blockers should be avoided: they have been associated with babies born small for their gestational age. Angiotensin-converting enzyme (ACE) inhibitors are contraindicated in the second and third trimester because they are associated with a myriad of congenital anomalies, including renal failure, oligohydramnios, renal dysgenesis, reduced ossification, pulmonary hypoplasia, and fetal and neonatal death. Patients presenting in the first trimester on an ACE inhibitor should either be taken off antihypertensive medications or switched to another agent. Exposure during this time is not an indication for pregnancy termination, however. Angiotensin II receptor antagonists are considered guilty by association because of their similarity to ACE inhibitors, but there are no data to confirm this. Chronic hypertension accounts for a disproportionate amount of maternal and perinatal morbidity and mortality, mostly because of an increased risk of superimposed preeclampsia. There is an increased risk of prematurity, birth of infants who are small for their gestational



age, intrauterine death, placental abruption, and cesarean delivery.

Complication rates are directly related to the severity and duration of elevated blood pressures. For instance, patients with severe hypertension in the first trimester have a greater than 50% risk of developing superimposed preeclampsia. All hypertensive patients should undergo increased surveillance, serial laboratory tests throughout pregnancy, serial ultrasound scans to follow growth, and antenatal testing. The baby should be delivered vaginally if possible.

Gestational Hypertension

Gestational hypertension, formerly known as pregnancy-induced hypertension or PIH, is the new onset of hypertension after 20 weeks of gestation. The diagnosis requires that the patient have:

- Elevated blood pressure (systolic ≥ 140 or diastolic ≥ 90 mm Hg, the latter measured using the fifth Korotkoff sound)
- Previously normal blood pressures
- No protein in the urine
- No manifestations of preeclampsia/eclampsia.

Also known as transient hypertension, gestational hypertension is actually diagnosed retrospectively when the patient does not develop preeclampsia and if blood pressure returns to normal by the 12-week postpartum visit. Fifty percent of women diagnosed with gestational hypertension between 24 and 35 weeks develop preeclampsia. The diagnosis of gestational hypertension mandates increased surveillance. Women who

progress to severe gestational hypertension based on the degree of blood pressure elevation have worse perinatal outcomes than do women with mild preeclampsia, and require management similar to those with severe preeclampsia.

Management of preeclampsia

Preeclampsia places both mother and fetus at risk. It is, however, a maternal disorder. The mainstay of treatment is early detection and managed delivery to minimize both maternal and fetal risks. If the pregnancy is at term, the decision is easy: the baby should be delivered. The decision to deliver involves balancing the risks of worsening preeclampsia against those of prematurity. Delivery is generally not indicated for women with mild preeclampsia until 37 to 38 weeks of gestation and should occur by 40 weeks. If remote from term, the mother should be admitted for evaluation. She will need:

1. Baseline and serial laboratory tests (complete blood cell count, BUN, creatinine, uric acid, ALT, AST).
2. Ultrasonography to measure fetal growth and amniotic fluid volume and Doppler ultrasonography. Umbilical artery systolic/diastolic ratios measured by Doppler ultrasonography may detect early uteroplacental insufficiency.
3. Antenatal testing (nonstress test or biophysical profile). The biophysical profile is an assessment of fetal well-being. Fetuses that are well oxygenated behave normally by twisting, squirming, flexing and extending extremities, and breathing. Fetuses that are hypoxic lie still, trying to conserve oxygen.



4. A 24-hour urine collection for protein. The goals of treatment are to prevent seizures, lower blood pressure to avoid maternal end-organ damage, and expedite delivery.

Magnesium sulfate is still the drug of choice for preventing and arresting eclamptic seizures. It has the additional benefit of reducing the incidence of placental abruption. Serum magnesium levels should be monitored in women with elevated serum creatinine levels, decreased urine output, or absent deep tendon reflexes.

Magnesium toxicity can lead to respiratory paralysis, central nervous system depression, and cardiac arrest. The antidote is calcium gluconate, 1 g infused intravenously over two minutes.

Antihypertensive medications are used solely to prevent maternal morbidity and have no effect on disease progression or preventing eclampsia. Medications must be given with caution: if blood pressure is lowered too fast, it can have a dramatic effect on uteroplacental perfusion and can cause an already compromised fetus to rapidly decompensate and become bradycardic. Preferred medications are hydralazine (5-10 mg intravenous bolus every 10-15 minutes), labetalol, nicardipine, and sodium nitroprusside. Intravenous labetalol and hydralazine are commonly used for the acute management of preeclampsia.

Diuretics are usually contraindicated because of the already collapsed intravascular volume. However, if the pulmonary capillary wedge pressure is high, diuretics are necessary.

Intravenous hydration for oliguria must be given cautiously to avoid pulmonary edema, ascites and cardiopulmonary overload. If there is no evidence of pulmonary edema, a trial of fluid resuscitation (500 mL over an hour) should be given.

Prediction and Prevention of Preeclampsia and Associated Complications

1. No first or second trimester test or set of tests can reliably predict the development of all cases of preeclampsia; however, a combination of maternal risk factors, BP, placental growth factor (PIGF), and uterine artery Doppler can select women who may benefit from 150 mg/d of aspirin to prevent preterm (before 37 weeks gestation) but not term preeclampsia. ISSHP supports first trimester screening for risk of preeclampsia when this can be integrated into the local health system although the cost effectiveness of this approach remains to be established.

2. ISSHP recommends that women with established strong clinical risk factors for preeclampsia (ie, prior preeclampsia, chronic hypertension, pregestational diabetes mellitus, maternal body mass index >30 kg/m², antiphospholipid syndrome, and receipt of assisted reproduction) be treated, ideally before 16 weeks but definitely before 20 weeks, with low-dose aspirin (defined as 75-162 mg/d, as studied in randomized controlled trials).

3. We recommend at this stage against the routine clinical use of rule-in or rule-out tests (specifically PIGF or sFlt-1 [soluble fms-like tyrosine kinase-1]/PIGF



ratio) for preeclampsia, which should continue to be evaluated within the context of clinical trials.

General Recommendations

1. The recommendations described in this document are for an ideal setting. In some instances, it may not be possible to adopt all of these recommendations. Health systems in LMICs may have to consider the minimum required to reach as many women as possible.

2. It is recommended that there is ongoing review and update of national and facility clinical guidelines, preservice educational material, and in-service training materials to ensure that all documents reflect these ISSHP recommendations so as to improve outcomes for women and babies.

3. In circumstances where the documented goals of this guideline are not attainable in their entirety, physicians should work pragmatically toward them as far as the local resources allow.

4. It is the responsibility of managing physicians to advocate for the use of effective interventions whether they practice in well- or under-resourced settings.

5. The distances between community clinics and referral hospitals are often large, and transport problems exist. For this reason, patients diagnosed with preeclampsia should be referred as soon as possible to a center with an appropriate level of care and managed as inpatients.

6. The effectiveness of referral systems in many LMIC is less than optimal, and many rural areas are without centers

that can provide basic obstetric and neonatal services. Women diagnosed with preeclampsia in such settings should be advised to relocate immediately to areas with better healthcare services, especially where they have family members if possible.

7. Communities should put strategies in place for transport from clinics or primary healthcare centers to referral centers.

8. All healthcare facilities should regularly review and update facility and community health worker referral pathways for women with preeclampsia.

9. All women with a hypertensive disorder of pregnancy require delivery in a center that provides emergency obstetric and neonatal care while women with maternal complications require delivery in a center capable of providing maternal critical care. Those with pregnancies at the limit of viability require the highest available level of neonatal support.

10. Antihypertensive agents for treatment of moderate and severe hypertension and MgSO₄ to prevent or treat eclampsia must be available at community level centers and clinics so that patients can be stabilized and referred safely.

11. Women with preeclampsia in LMICs may have a limited comprehension of the nature and risks of the disease.

Conclusion

In conclusion, although many pregnant women with high blood pressure have healthy babies without serious problems, high blood pressure can be



dangerous for both the mother and the fetus. Women with pre-existing, or chronic, high blood pressure are more likely to have certain complications during pregnancy than those with normal blood pressure. However, some women develop high blood pressure while they are pregnant (often called gestational hypertension).

The effects of high blood pressure range from mild to severe. High blood pressure can harm the mother's kidneys and other organs, and it can cause low birth weight and early delivery. In the most serious cases, the mother develops preeclampsia-or "toxemia of pregnancy"- which can threaten the lives of both the mother and the fetus.

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