Fetal and neonatal complications of pregnancy induced hypertension

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Abstract

Most pregnancy lead to a successful outcome without complication, however, many factors can interfere with the normal process of pregnancy and may threaten the health of the mother or the development of the fetus. These factors can be detected at the beginning of pregnancy by reviewing the danger signs that may indicate complication. Pre eclampsia (PE) is a multi-system disorder of the mother that affects the fetus because of utero-placental insufficiency. In consequence, these children are at risk for intra-uterine growth restriction and may be delivered prematurely. They may also suffer from the consequences of high rate of operative deliveries and the adverse effects of maternal drugs. These neonates may also have a spectrum of hematological changes which may add to the existing morbidity in them. This can cause uncontrollable bleeding and be life-threatening for both mother and baby. Another complication is Eclampsia (Pre-eclampsia plus seizures) that happens when PE is uncontrolled. This is associated with maternal mortality. Fetal and newborn complications of hypertensive disorders of pregnancy include growth restriction, prematurity, and stillbirth. In addition, there is evidence that the intrauterine milieu in a hypertensive pregnancy may, by mechanisms related to the failure of the fetus to exercise full growth potential, confer increased risk of cardiovascular events in adult life.

Keywords: pregnancy induced hypertension, fetal & neonatal complications.

Introduction

The hypertensive syndromes that occur during pregnancy, especially PE result in real risk and significant impact on indicators related to maternal and child health. These syndromes are causal factors related to maternal and perinatal death, and they cause definitive limitations to maternal health and serious problems resulting from associated elective prematurity. [1]

Preeclampsia is a clinical syndrome, defined as the new onset of hypertension and proteinuria during the 2nd half of
Fetal & neonatal complications of pregnancy induced hypertension

pregnancy. [1] It afflicts 3.0% to 5.0% of pregnant women. It is considered a leading cause of maternal mortality, especially in developing countries. [2,3] Since the only known remedy is delivery of the placenta, in developed countries preeclampsia is an important cause of premature delivery usually medically indicated for the benefit of the mother. This results in infant morbidity and substantial excess health care expenditure. [4] Despite the considerable morbidity and mortality, the cause of preeclampsia has remained enigmatic.

There is no accurate information on the incidence of preeclampsia worldwide, but it is estimated to occur in 3.0% to 5.0% of pregnancies. A systematic review identified an incidence of 1.5% for PE and of 0.6% for eclampsia. [5]

Classification of hypertension [6]

There are several classifications described for hypertensive disorders in pregnancy. In 2014, the classification and diagnosis of the hypertensive disorders of pregnancy: statement from the International Society for the Study of Hypertension in Pregnancy (ISSHP) reviewed the classification of hypertensive disorders during pregnancy. [7]

A. Chronic hypertension

Chronic arterial hypertension in pregnancy is the occurrence of systemic arterial hypertension (SAH) preceding pregnancy. SAH is considered chronic when observed in the 1st trimester of gestation or, at most, up to the 20th week. Chronic hypertension refers to essential hypertension, that is usually associated with a family history of hypertension, and often accompanied by overweight or obesity. Essential HTN is also called primary HTN and has no known etiology, what you are referring to is secondary HTN which can be linked to other factors such as genetics and/or obesity. [7]

B. Gestational hypertension

Gestational hypertension is defined as arterial hypertension arising for the 1st time after the 20th week of gestation without being accompanied by any signs, symptoms or laboratory abnormality that characterizes preeclampsia. [7]

C. White coat hypertension

About 25% of people with increased BP measurements in medical consultations have white coat hypertension. The diagnosis can be confirmed by serial measurements (preferably taken by nurses) or ambulatory BP monitoring (ABPM). There are few studies on the repercussion of this type of disorder in pregnancy, some suggesting that up to 50% of these cases evolve to gestational hypertension or PE. [8,9]

Maternal and fetal complications of preeclampsia

Approximately 20% of women with Hemolysis, Elevated liver enzymes, Low platelets (HELLP) syndrome develop disseminated intra vascular coagulation, which carries a poor prognosis for both mother and fetus. [7] Placental abruption, ascites, hepatic infarction, hepatic rupture, intra-abdominal bleeding, pulmonary edema, and acute renal failure are all severe clinical manifestations associated with preeclampsia which can result in maternal death. [10] Perhaps the most feared complication of PE is eclampsia itself, defined by the presence of seizures, for which women with severe PE are often treated with magnesium sulfate prophylaxis. [1] The brain injury in eclampsia is associated with cerebral edema and characteristic white matter changes of reversible posterior leukoencephalopathy syndrome (RPLS), that is similar to what is noted in hypertensive encephalopathy and with cytotoxic immunosuppressive therapies. [11] Cerebrovascular complications, including stroke and cerebral hemorrhage, are responsible for the majority of eclampsia related deaths. [12] Complications affecting the developing fetus include indicated prematurity, intrauterine fetal growth restriction (IUGR), oligohydramnios, bronchopulmonary dysplasia, and increased risk of perinatal death. [13-15]

Feto-maternal complications of SAH in pregnancy [16-18]

Systemic arterial hypertension during pregnancy can generate several complications that will invariably require careful evaluation and management by the medical staff.

1. Cardiovascular system: Severe systemic arterial hypertension (SAH), vascular accidents, severe uncontrolled hypertension (for a 12-hour period despite maximum doses of hypertensive agents), myocardial ischemia or infarction. Hematological: Hemolysis, thrombocytopenia, Leukocytosis, disseminated intravascular coagulation (DIC), thrombocytopenia, coagulopathy, high INR PTT, Platelets< 50,000/dl (need for transfusion of any blood product).

2. Respiratory system: SO2< 90%, need for O2> 50% for > 1 hour, intubation, support with vasoactive drugs, pulmonary edema, pulmonary embolism.

3. Central nervous system (Neurological): Headache, visual symptoms, Retinal detachment, cerebral edema, Eclampsia, stroke, cortical blindness, posterior reversible encephalopathy syndrome (PRES), Glasgow scale<13, transient ischemic attack (TIA), reversible neurological deficit (RND)

4. Urinary system: Oliguria, elevated creatinine & uric acid, renal insufficiency, acute renal failure (ARF) (creatinine> 1.5 mg/dL without previous renal disease); need for dialysis (without previous CRF).

5. Gastrointestinal system (Hepatic): nausea; vomiting epigastralgia, upper right quadrant of the abdomen (URQ) pain; serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), lactate dehydrogenase...
(LDH), elevated bilirubin; low plasma albuminemia, Hepatic impairment (INR > 2 in absence of DIC or use of warfarin), hepatic dysfunction, hepatic hematoma (with or without rupture), and capsular rupture.


7. Fetoplacental: Non reactive cardiotocography (CTG), Oligohydramnios, intrauterine growth restriction (IUGR), Doppler of umbilical artery with absent or reversed diastolic flow

Management of Preeclampsia

Regardless of the severity of the clinical picture, every patient diagnosed with PE should be hospitalized for follow-up in a high-risk gestational unit. Any patient with PE apparently with a benign condition may suddenly develop complications severe enough to result in maternal and/or fetal death. The fetuses of mothers with PE who remain hospitalized have half the risk of death compared with fetuses of mothers who are not hospitalized. In addition, hospital-based patients with PE have newborns with more advanced GA at delivery and greater birthweight. [19]

References


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