Preeclampsia

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Case

A 26-year-old G1P0 parturient at 32 weeks gestation presents to the labor and delivery floor with a severe headache and onset of contractions.

Physical Exam:
Visibly uncomfortable from labor pain
Mild crackles bilaterally on auscultation
Regular rate and rhythm

Vital Signs:
BP: 190/112 mmHg
HR: 115/min
RR: 26/min
O_2: 99% on room air

Labs:
Platelet count: 85,000/mm^3
2 + proteinuria on urinalysis

Questions:

What is preeclampsia?

Preeclampsia is a clinical syndrome whose primary distinguishing features include new onset hypertension and proteinuria after 20 weeks' gestation. The disease can be categorized as either mild preeclampsia (BP ≥ 140/90 mmHg, proteinuria 300 mg/24 h) or severe preeclampsia (BP ≥ 160/110 mmHg, proteinuria 5 g/24 h). Other symptoms include headache, visual disturbances, epigastric or right upper quadrant pain, as well as fetal symptoms (e.g., intra-uterine growth restriction) [1].

Is proteinuria necessary for a diagnosis of preeclampsia?

No. The Task Force Report on Hypertension in Pregnancy by The American College of Obstetricians and Gynecologists published in November 2013 reported that the absence of proteinuria should not exclude a diagnosis of preeclampsia. Parturients without proteinuria who present with persistent epigastric or right upper quadrant pain, persistent cerebral symptoms, fetal growth restriction, thrombocytopenia, and elevated serum liver enzyme concentrations should be evaluated for possible preeclampsia. Waiting for development of proteinuria may delay delivery of optimal care [2].

What is HELLP syndrome?

The acronym of HELLP stands for: Hemolysis, Elevated Liver enzymes, and Low Platelets. Hemolysis can be recognized by abnormal peripheral blood smear and anemia. Laboratory studies reveal AST ≥ 70 IU/L and platelet count less than 100,000/mm^3. Parturients with HELLP syndrome are at an increased risk for peripartum morbidity, including DIC, placental abruption, and preterm delivery [1].

What are risk factors for developing preeclampsia?

Risk factors for developing preeclampsia include obesity, chronic hypertension, diabetes mellitus, and metabolic syndrome. The risk of preeclampsia doubles with each 5 to 7 kg/m^2 increase in body mass index from pre-pregnancy values. The presence of preexisting chronic hypertension triples the risk of developing preeclampsia. Diabetes mellitus is associated with a twofold increased risk of developing preeclampsia [3].

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What might be a good prophylactic medication for preeclampsia?

Preeclampsia is associated with relatively excessive levels of thromboxane compared to prostacyclin. Thromboxane plays an important role in vasoconstriction and one hypothesis suggests that preeclampsia may be caused by excessive placental thromboxane production. It has been suggested that aspirin could prevent preeclampsia because aspirin inhibits synthesis of thromboxane A_2.

Nevertheless, randomized controlled trials have not found aspirin to be superior to placebo in the prevention of preeclampsia [1].

What are your considerations for placing an epidural for labor analgesia?

Since preeclampsia can be associated with thrombocytopenia, it is important to consider the risks and benefits of placing a labor epidural. Mild preeclampsia in the setting of normal platelet count is not a contraindication for neuraxial labor analgesia and early epidural placement may improve uteroplacental perfusion. Women with severe preeclampsia and thrombocytopenia are at an increased risk for epidural or spinal hematoma. It is important to consider, however, that they are also at an increased risk for emergency Cesarean delivery. The decision to place a labor epidural will ultimately have to be based on each individual patient's clinical situation [1].

Would you place a combined spinal–epidural (CSE) for labor analgesia in this patient?

There are many advantages and disadvantages of a CSE in a laboring parturient with preeclampsia. The main advantage of a CSE is rapid onset of analgesia, which can instantaneously help to decrease the hypertensive response to pain. However, the primary disadvantage of a CSE is the inability to evaluate epidural catheter function after resolution of spinal analgesia. Given the increased risk of emergency Cesarean delivery in the preeclamptic patient, a standard epidural technique may be more favorable because it can be immediately verified and provides a means for rapid induction of epidural anesthesia [1].

What is the minimum platelet count for performing a neuraxial technique in the preeclamptic patient?

Simply considering a "minimum" platelet count for performing a neuraxial technique in the preeclamptic parturient is inadequate for assessing the risk of epidural hematoma. Generally speaking, parturients with mild preeclampsia and a platelet count greater than 100,000/mm^3 do not need further evaluation prior to neuraxial technique. Women with a platelet count less than 100,000/mm^3 may require additional coagulation studies, including PT, PTT, and fibrinogen levels. The trend in platelet count is more important than the absolute platelet count. With a rapid decline, the nadir in platelet count is difficult to predict and may complicate the neuraxial technique. Regardless, parturients with HELLP syndrome and a platelet count less than 50,000/mm^3 are at an increased risk of bleeding and general anesthesia may be required [1, 4].

Is delivery of the child a "cure" for preeclampsia?

Although delivery of the child and placenta are often called the "cure" for preeclampsia, the risks of preeclampsia continue for several days postpartum. These risks include pulmonary edema, cerebrovascular accident, venous thromboembolism, and eclampsia. In fact, pulmonary edema is more likely to occur during the postpartum period because of the marked fluid shifts that occur with delivery of the child. Furthermore, approximately 5% of parturients are found to have postpartum onset of preeclampsia. Therefore, preeclamptic patients need to be closely monitored during the postpartum period before discharging them from the hospital [5].

Would you place an arterial catheter in this patient?

Parturients who present with mild preeclampsia may not need an arterial catheter, but these patients should be closely monitored for progression of the disease. Preeclamptic parturients who have poorly controlled blood pressure and require rapid titration of vasodilators will likely require arterial line placement. Parturients with severe preeclampsia, who require induction of general anesthesia for Cesarean delivery or frequent blood gas measurements, may also require invasive blood pressure monitoring [1].

The patient requires a general anesthetic for STAT Cesarean delivery. What are your concerns with laryngoscopy?

Cerebrovascular accident is the leading cause of death in preeclamptic parturients. The risk of intracranial hemorrhage is particularly high during laryngoscopy and intubation due to the hypertensive response to the procedure. Severely hypertensive parturients will require an arterial catheter for close hemodynamic monitoring prior to induction of general anesthesia. Labetalol should be administered prior to induction until the blood pressure is approximately 140/90. If the hemodynamic goal cannot be attained with labetalol

alone, additional antihypertensive medications, including infusions of nitroprusside and/or nitroglycerin, should be strongly considered [1].

The patient has been on magnesium sulfate infusion for seizure prophylaxis. What are your concerns?

Because magnesium sulfate decreases the sensitivity of the neuromuscular junction for acetylcholine, one of the primary concerns is prolonged duration of nondepolarizing muscle relaxants. If general anesthesia is required, doses should be decreased and a peripheral nerve stimulator should be used to carefully monitor return of twitches. On the other hand, the duration of action of succinylcholine is not increased and standard doses should be used for intubation [6].

The obstetrician states the uterus is boggy and requests uterotonics. Which medications are contraindicated in preeclampsia?

The preeclamptic patient may exhibit significant peripartum hemorrhage secondary to uterine atony. Methylergonovine maleate (i.e., methergine, an ergot alkaloid) is an excellent medication for uterine contractility. However, methergine can also cause significant increases in both pulmonary and systemic vascular resistance, leading to pulmonary and systemic hypertensive crisis due to its effects on serotonergic, dopaminergic, and alpha-adrenergic receptors. Therefore, methergine is generally contraindicated in preeclampsia [1].

A preeclamptic parturient with HELLP syndrome presents with acute right upper quadrant abdominal pain and severe hypotension. What are your concerns?

Patients with HELLP syndrome are at an increased risk for rupture of subcapsular hematoma of the liver. Ultrasonography can confirm the diagnosis and emergent surgical intervention is required. Since the most common causes of death include coagulopathy and exsanguination, this patient will require transfusions of both red blood cells and fresh frozen plasma. Emergency laparotomy is necessary to save the life of the mother, but the mortality rate is still very high [1].

What is placental abruption?

Placental abruption is an abnormal separation of the placenta from the uterus prior to delivery of the child. Bleeding is a major complication of placental abruption. It is important to remember that the bleeding may be concealed in the uterus, leading to underestimation of blood loss and delayed diagnosis. Placental abruption is also complicated by coagulopathy, further increasing blood loss. Parturients with abruption will require careful monitoring and early transfusion [7].

Does a patient with preeclampsia have an increased risk of placental abruption compared to an otherwise healthy parturient?

The risk of placental abruption in preeclamptic parturients is approximately triple compared to healthy parturients. Given that placental abruption is associated with a higher risk of DIC and that preeclampsia is already associated with coagulopathy, these parturients are at a particularly high risk for large blood loss during the peripartum period [8].

What may be the cause of cerebral edema in a parturient with preeclampsia?

Cerebral edema is a complication of preeclampsia that may be caused by loss of cerebral autoregulation. In the setting of excessive hypertension, endothelial dysfunction results in hyperperfusion and formation of interstitial edema. Intravenous fluids should be minimized to decrease the risk of exacerbating the cerebral edema [1].

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