POST-PARTUM MALIGNANT HYPERTENSION IN A PATIENT WITH PREECLAMPSIA AND ABRUPTIO PLACENTA

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Introduction

There are many physiologic changes and potential risks of pregnancy. Though there are numerous proposed mechanisms related to the etiology of preeclampsia, it is well known that this condition can potentially result in morbidity and mortality to both the mother and child. Preeclampsia is a condition commonly encountered by obstetric anesthesiologists and uncommonly complicated by placental abruption.

As a review, preeclampsia is characterized by new onset hypertension occurring beyond 20 weeks gestation and can predispose organ failure, seizure, and stroke to the mother. Diagnosis is made with two different blood pressure measurements of greater than 140/90 and a 24-hour urine sample with 300 mg of protein or more[1,2]. Severe preeclampsia is seen with blood pressures greater than 160/110, proteinuria worsens to 5g in 24 hours, and other associated symptoms and signs. HELLP syndrome is a variant of severe preeclampsia, with the acronym HELLP referencing the clinical triad of hemolysis, elevated liver enzymes, and low platelets [3]. Typical treatment regimens for elevated blood pressure at present include magnesium, and the antihypertensive medications labetalol, nifedipine, and hydralazine. Ultimate treatment for preeclampsia is delivery of the fetus.

Keywords: Malignant hypertension, abruptio placenta, anesthesia, nitroglycerin, preeclampsia, vaginal bleeding.

The following case describes a unique situation where a normotensive parturient presented with placental abruption and subsequently developed hypotensive shock, requiring vasopressor support. Soon after delivery, the patient became profoundly hypertensive. Further investigation revealed a history of severe preeclampsia with a prior pregnancy. Both cases tragically resulted in fetal demise.

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Case Presentation

A 24 year old G4P2 at 24 weeks gestation presented to the Emergency Department (ED) with cramping, abdominal pain and mild "spotty" vaginal bleeding. Her medical history was significant for a previous cesarean section 3 years earlier. Review of systems was positive for abdominal pain, pelvic pain, and vaginal bleeding. Physical exam was significant, abdominal distention consistent with a gravid uterus without tenderness. Laboratory values were within normal limits other than 2+ red blood cells and 1+ leukocytes on urinalysis. A renal ultrasound was conducted in the ED and the summary was, "normal anatomy" and "bilateral perinephric fluid" by the reviewing radiologist. Her kidneys were somewhat echogenic, and acute renal failure/acute tubular necrosis could not be excluded. The patient was discharged from the Emergency Department with a diagnosis of abdominal pain in pregnancy and pyuria. The patient was prescribed a regimen of oral antibiotics.

Four hours later the patient represented to the ED via ambulance with diffuse vaginal hemorrhage and altered mental status. A STAT Cesarean section was called while the patient was in route to the hospital. The anesthesia team first encountered the now somnolent patient in the OR. Immediately upon arrival, preoxygenation of the patient with 100% O2 began, two large bore intravenous lines was obtained, standard ASA monitoring were placed, and an arterial line was inserted in her right radial artery within five minutes of presentation. A rapid sequence induction consisting of etomidate, in a dose of 12 mg iv, fentanyl, in a dose of 100 ug iv, lidocaine, in a dose of 50 mg iv, and succinylcholine, in a dose of 120 mg iv, which was administered to facilitate intubation. The initial blood pressure was 102/54 mmHg. The time to incision was 8 minutes after arrival, and the baby was delivered 3 minutes after incision. The diagnosis of abruptio placenta was made intraoperatively. The newborn child required intubation, and was transferred to the NICU. The patient had an estimated 3L of intraoperative blood loss and received 4 units of PRBCs intraoperatively. Her vital signs remained relatively stable throughout the case. Mean arterial blood pressure readings on induction were approximately 65-70 mmHg and pulses were approximately 110 beats per minute at the start of the case, requiring multiple doses of vasopressors and eventually a phenylephrine infusion to maintain mean arterial pressure of 60-80 mmHg intraoperatively. She was successfully extubated at the end of the case after meeting standard criteria. Upon arrival at the ICU, the patient had a blood pressure of 146/65 mmHg and a pulse of 100 beats per minute. The vasopressor infusion was stopped and the blood pressure measurement repeated, with a subsequent BP reading noted to be 311/109 mmHg. This value was confirmed with a manual reading from the opposite arm, and radial artery measurement. The patient remained asymptomatic and intravenous nitroglycerin was administered with stabilization of her blood pressure over 10 minutes. After 3 hours and 20 minutes of intravenous nitroglycerin delivery postoperatively in the ICU, which included titration to a mean arterial pressure of 100 mgHg, the patient stabilized with cessation of antihypertensive medication. Unfortunately, the neonate died several hours later in the NICU from complications of pulmonary hemorrhage. The patient later reported she vaguely recalled high blood pressure with a previous pregnancy which also resulted in fetal demise.

Discussion

Hypertensive disorders of pregnancy are a leading cause of peripartum morbidity and mortality, and complicate 8-12% of pregnancies[4,5]. Placental abruption is a major complication of hypertensive disorders of pregnancy leading to adverse outcomes. Placental abruption is the premature detachment of the placenta from the uterus, either partially or fully. The mechanism of this complication is not completely understood, but it is an obstetric emergency with high rates of maternal and fetal morbidity and mortality[6]. A study by Nankali et. al., demonstrated a 7.7% risk of placental abruption in severe preeclampsia, while others have reported an incidence as high as 15%[7]. Risk factors for placental abruption include maternal hypertensive disorders, smoking, addictive behaviors, maternal age greater than 35, multiparity, multiple gestations, and premature rupture of membranes. Of note, previous placental abruption may increase the risk of placental abruption 10-30 fold[6]. This may be

attributed to an individual parturient's predisposition for abnormal uterine vascularization during trophoblast migration after implantation of the embryo. It is widely accepted that this abnormal angiogenisis is closely associated with hypertensive disorders of pregnancy. Many studies have demonstrated plasma angiogenic factor abnormalities in patients with preeclampsia. It is presumed that these factor abnormalities are originating from the placenta itself, and are associated with improper vascularization of the placenta. Patients with preeclampsia have significantly more placental vascular lesions including placental abruption[8].

The diagnosis of placental abruption is typically first made on a clinical presentation of abdominal pain and vaginal bleeding associated with abnormal fetal heart tones. Management is guided by fetal condition and maternal well-being. If the fetus is viable, emergent cesarean section should be performed unless vaginal delivery is eminent. With a bradycardic fetus, delivery within 20 minutes significantly reduces neonatal mortality and the incidence of cerebral palsy. Maternal mortality with placental abruption has been greatly reduced in the last 100 years from 8% to 1%. However, maternal complication of severe hemorrhage and resulting DIC are still common. Neonatal mortality associated with placental abruption is typically related to prematurity. However, after 32 weeks gestation, placental abruption is an independent risk factor for neonatal mortality. Placental abruption is also a major independent risk factor for intrauterine fetal demise[6].

Though preeclampsia may often be considered a disorder associated with primigravida, it also is common with subsequent gestations. The risk of preeclampsia in a subsequent pregnancy after having preeclampsia in a previous pregnancy has been demonstrated to be 40% in multiple studies[7]. A study by Melamed et. al., regarding risk factors for adverse outcomes in pregnancy demonstrated that preeclampsia with a previous pregnancy is an independent risk factor for preeclampsia and placental abruption in subsequent pregnancies. Furthermore, they concluded that women who had been preeclamptic with the complication of placental abruption in a previous pregnancy were at the highest risk for an adverse outcome in a subsequent pregnancy. Similarly, placental abruption in a previous pregnancy was the strongest predictor of preeclampsia in a subsequent pregnancy of all the risk factors they examined[9].

Knowing the increased risk of morbidity and mortality associated with a previous pregnancy complicated by preeclampsia or placental abruption should alert the clinician that an increased level of vigilance may be warranted in these parturients. Maintaining a high index of suspicion in emergent cesarean sections to preeclampsia, despite a hypotensive presentation, could prevent hypertensive urgency/emergency once intravascular volume had been repleted. In this patient's case, that was not achieved until ICU transfer, at which the presentation of preeclampsia became clearer, with a consistent medical history. In addition administration of IM corticosteroids at her initial presentation may have helped promote lung maturity in the fetus, possibly increasing the likelihood of survival.

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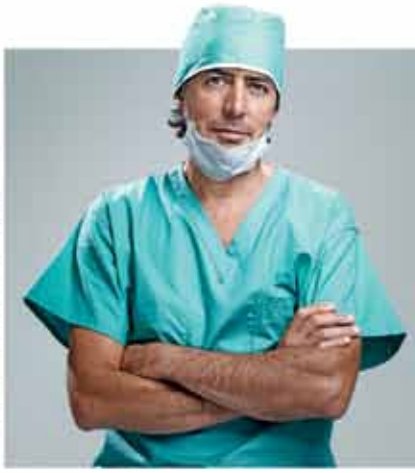

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* Train-of-four
† Post-tetanic counts
‡ Second twitch

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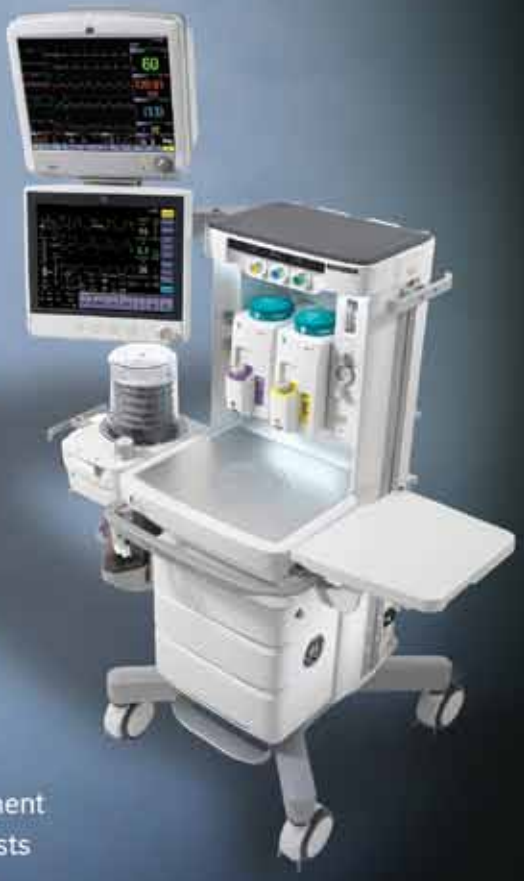

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