Lethal aortic dissection in a 33-week parturient: A case report

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Aortic dissection is a rare and potentially lethal complication associated with pregnancy. Although well reported in obstetric, medical, and cardiothoracic literature, it is poorly reported in the anesthesia literature. A 33-week parturient with chronic hypertension was admitted to an urban medical center with severe epigastric and chest pain. During a 24-hour admission, the patient's condition deteriorated, and she required emergency intubation and sedation for transesophageal echocardiography (TEE) that revealed a type I (DeBakey) aortic dissection. This discovery, along with worsening fetal vital signs, necessitated an emergency cesarean section with delivery of a critically ill infant. Unfortunately, grossly necrotic bowel was found on inspection of the peritoneum, and dissection of the superior mesenteric artery was suspected. After consultation with general and thoracic surgeons, this injury was determined to be terminal and the findings were discussed with the patient's family. Comfort measures only were instituted, and the patient died approximately 24 hours after admission. Because the nurse anesthetist may often be the primary or sole anesthesia provider, it is imperative that he or she become familiar with the signs, symptoms, predisposing factors, and treatment of this potentially lethal complication of pregnancy. It is our desire to expand the body of nurse anesthesia knowledge through review of this tragic case.

Key words: Anesthesia, aortic dissection, parturient, pregnancy, thoracic.

Case summary
A 37-year-old, gravida 4, para 3, 33-week gestation, white parturient was admitted to our labor and delivery unit at approximately 4:30 am complaining of crushing chest pain and abdominal pain that radiated into her back. She denied contractions but stated she was nauseated. No contractions were detected via tocodynamometry, and fetal heart tones showed good variability at 130 to 140 beats per minute (bpm) via fetal heart monitoring. The woman's vitalsigns were as follows: blood pressure, 159/55 mm Hg; heart rate (HR), 101 bpm; and respiratory rate (RR), 22 breaths per minute. Urinalysis revealed 3+ protein. An initial diagnosis of preeclampsia was made, and the patient was admitted to the antenatal unit.

Medical records, which were not available to the nurse anesthetist premonstet, revealed a 10-year history of hypertension, a previous hospital admission for congestive heart failure, obstructive sleep apnea, obesity with a pregravid body mass index of 43, and a 20 pack-year history of smoking. The surgical history was significant for 2 previous cesarean deliveries, both under regional anesthesia without complication. The obstetric history consisted of a vaginal birth after cesarean delivery and preeclampsia. The patient entered prenatal care at 7 to 8 weeks of gestation and was immediately referred to cardiology for evaluation because of her history of congestive heart failure. Before entering prenatal care, her hypertension was medically managed using furosemide with a potassium supplement. On entry into obstetric care, this was discontinued and a titrated dose of methyldopa and, eventually, metoprolol was instituted. An echocardiogram performed at 10 weeks of gestation was unremarkable,
with an aortic root measuring 3.5 cm and normal left ventricular function.

Postmortem review of the medical record revealed that the patient had been transferred to the antenatal unit where a 12-lead electrocardiogram (ECG) and cardiac enzymes were obtained, all of which were unremarkable. Fourteen hours after admission, at approximately 6:00 PM, a drastic change in the patient’s mental status was observed. The patient was noted by the nursing staff to be acutely confused and walking around her room naked. This acute change in mental status was interpreted as psychosis; the patient was medicated with lorazepam, and a psychiatric consultation was ordered by the obstetrics service. The patient's vital signs were as follows: blood pressure, 202/104 mm Hg; HR, 125 bpm; and RR, 33 breaths per minute. Repeated cardiac enzymes drawn around this time were not reported until 8:00 PM, at which time an acute increase of the troponin T level was noted, to 3.94 µg/L, and the creatine kinase-myocardial bound was 255 µg/L. This acute change precipitated the patient's immediate transfer to the labor and delivery suite. It was at this time that we had our initial exposure to the patient.

The patient was combative and required multiple staff to restrain her to obtain patient and fetal vital signs and a 12-lead ECG. The initial vital signs were as follows: blood pressure, 202/85 mm Hg; HR, 133 bpm; and RR, 33 breaths per minute. The 12-lead ECG revealed inverted T waves and pathologic Q waves in leads II and III, and augmented voltage foot consistent with an inferior infarct. Her speech was slurred, and she was oriented only to name. We acted on a diagnosis of acute myocardial infarction (AMI), because these were the only data available to us at the time. The cardiology team was immediately called, and 2 inches of nitroglycerine ointment was applied to the patient. Before the arrival of the anesthesia team, it was reported that the patient had been given 181 mg of aspirin by mouth and 3 mg of morphine sulfate intravenously. The patient was transferred to the operating room because of a potential need for an emergency cesarean section. On arrival in the operating room, the patient remained combative, making it impossible to obtain fetal vital signs. After consultation with obstetrics and cardiology physicians, a decision was made to chemically restrain the patient so that further evaluation could proceed without physical threat to staff or the patient.

Rapid-sequence induction, including cricoid pressure, was performed using 30 mg of etomidate and 200 mg of succinylocholine intravenously. The trachea was intubated on the first attempt using a 7.0 endotracheal tube. After confirmation of end-tidal carbon dioxide and bilateral breath sounds, the tube was secured and mechanical ventilation was started. Sevoflurane was administered at 1.2% with 100% oxygen, and vecuronium, 10 mg, was administered. Initial fetal heart tones were reassuring after induction at 130 to 140 bpm, with good variability. Maternal vital signs also improved and were as follows: blood pressure, 161/82 mm Hg; and HR, 110 bpm. In light of the improved maternal vital signs and the reassuring fetal vital signs, the obstetrics team believed that emergency cesarean section was not immediately necessary. In addition, the cardiology team believed that the added stress of the operation could potentially exacerbate the patient’s evolving AMI to a terminal point. After consultation among all medical teams involved, the consensus was that the best hope for survival of the patient and fetus would be further evaluation and management of the AMI by the cardiology team in the cardiac intensive care unit (CICU) with close monitoring by the obstetrics team. General anesthesia was discontinued, and by orders of cardiology and obstetrics teams, metoprolol, 10 mg, and midazolam, 4 mg, were administered intravenously. The intubated and sedated patient was transferred to the CICU at approximately 11:30 PM.

On arrival in the CICU, a left radial arterial line was placed and 2 large-bore peripheral intravenous lines were inserted. Simultaneous placement of the transesophageal echocardiography (TEE) probe was performed by the cardiology staff, and an immediate diagnosis of aortic dissection was made. The dissection extended from the aortic root to as far as the probe could be passed distally. Obstetrics, cardiothoracic, and general surgery teams were alerted to the diagnosis. Fetal vital signs were reevaluated and found to be critical with a HR of 60 to 80 bpm. An immediate bedside cesarean section was undertaken by the obstetrics team, using local anesthesia, with delivery of a male infant with Apgar scores of 0 and 2. Inspection of the peritoneum revealed grossly necrotic bowel. The general surgery team was consulted intraoperatively, and it was the consensus of the teams involved that the dissection likely involved the superior mesenteric artery. The degree of bowel necrosis present indicated a terminal injury in opinion of the surgical team. The uterus and surgical incisions were closed. Consultation among the obstetric, surgical, and cardiothoracic teams, along with the patient's family, was conducted, at which time the decision was made to discontinue life-prolonging treatment. The patient died at approximately 5:30 AM on day 2 of admission. Despite aggressive resuscita-
tion in the neonatal intensive care unit, the infant died 2 days later.

Discussion

Seminal studies in the area of aortic dissection have estimated that 50% of all aortic dissections that occur in women younger than 40 years occur during pregnancy, often during the last trimester. Additional investigation in 2003 further narrowed the average age to 30 years with an average gestation of 32 weeks. Also of note was a 43% average incidence of systemic disease associated with the dissection. These findings should come as no surprise because the third trimester has been documented to be the period of maximal hemodynamic stress. Outflow resistance is increased in the lower body due to compression of the aorta and iliac vessels by the gravid uterus. This compression, along with the increased ejection of blood into the aorta, secondary to an increased total blood volume, may predispose the parturient to an intimal tear.

Parturients with a history of bicuspid aortic disease have a strong predisposition to aortic dissection. Although subject to some debate, the hormonal influences on the aorta may predispose the parturient to dissection. Specifically, estrogen receptors have been documented in aortic tissue, and increasing estrogen levels have been implicated in fragmentation of the elastic fibers, which are scleroproteins found within the connective tissues of the lymphatic system. This diminishes the amount of acid mucopolysaccharides and favors the loss of the normal corrugation of the elastic fibers. The risk for aortic dissection is significantly increased in patients with Marfan and Ehlers-Danlos syndromes. Accordingly, patients with these syndromes are routinely counseled on the need for prepartum aortic root replacement or to consider medical sterilization. The presence of systemic diseases such as hypertension and diabetes contributes to an increased incidence of aortic dissection, although the exact contribution is not quantifiable.

A classic symptom of aortic dissection is the sudden onset of excruciating pain, usually in the anterior part of the chest, which radiates to the back and travels caudad as the dissection progresses. This intense pain often is confused with symptoms of AMI. It is not uncommon for aortic dissections to involve the coronary arteries, which would increase the risk for myocardial ischemia and infarction. It is important to remember that the pain associated with AMI rarely radiates into the abdomen, lumbar area, or legs.

As in this case, abdominal pain in the parturient with aortic dissection only serves to further confuse the situation. Careful observation should be given to the presence or absence of uterine contractions. Severe abdominal pain in the absence of contractions may be associated with aortic dissection and is often related to mesenteric dissection and subsequent bowel ischemia. This pain rarely involves spasm, rebound tenderness, or guarding, but often produces a rigid abdomen and nausea and vomiting. As evidenced by our patient, confusion and combative behavior and an impending sense of doom are not uncommon in the later stages of dissection, often related to profound metabolic acidosis. Hemiparesis or hemiplegia may manifest secondary to involvement of a single carotid artery. In addition, complete paraplegia may result if there is significant occlusion of spinal cord blood flow.

Objective data may be difficult to obtain and misleading. The ECG may be benign but often shows signs of left ventricular hypertrophy, which is associated with a history of hypertension. Consistent with the objective data for our patient, a pattern of ischemia or pericarditis may be present, which is associated with coronary occlusion or hemopericardium secondary to dissection. The classic finding in chest radiography is a widened mediastinum with disparate ascending-to-descending diameter.

Laboratory data may also be of little use in the parturient because the natural drop in hemoglobin and hematocrit values may mask the pathologic drop seen with aortic dissection. However, as with this case, an elevation in cardiac enzyme levels is an ominous finding because it is likely a sign of coronary occlusion. As the dissection progresses, acute elevations in the blood urea nitrogen and creatinine levels may indicate renal artery occlusion. Profound metabolic acidosis observed in arterial blood gas levels is likely secondary to low cardiac output and/or bowel ischemia secondary to mesenteric artery dissection, as was the case in our patient.

Angiography remains the "gold standard" for determining the severity and extent of aortic dissection. It can be carried out with parturients given stable hemodynamics, proper shielding, and patient cooperation. Magnetic resonance imaging (MRI) may also be an alternative source of imaging. An MRI provides a high degree of sensitivity and specificity and is a noninvasive method of imaging. However, MRI is frequently not performed in parturients primarily because the safety of MRI imaging in regard to the fetus is not well established. In addition, the long duration of the imaging and the risk of aortocaval compression from the supine positioning for the examination may also preclude the use of MRI.
In cases such as ours in which the patient is admitted in extremis, the preferred imaging test is TEE, which is considered as sensitive as MRI and, “in the hands of a skilled sonographer,” can be completed in as little as 15 minutes. Pulsed- and color-flow Doppler aid in the diagnosis of the dissection, which will appear as a mobile intimal flap within the aorta. In addition, TEE can quickly diagnose the presence of pericardial effusion and cardiac tamponade and give a rapid assessment of left ventricular function, thereby guiding further intervention.9

Management of parturients with acute aortic dissection is complex. Management must be based on the location of dissection. Ascending dissections (DeBakey types I and II) require immediate surgical intervention, whereas descending dissection (DeBakey type III) may often be medically managed. However, universal to all types is the requirement for immediate hemodynamic control, consisting of decreased ventricular ejection velocity and decreased systemic mean arterial pressure. Historically, this has involved the use of nitroprusside and propranalol.11 It should be noted that nitroprusside has been implicated in fetal cyanide toxicity and that the placental transfer of beta blockers has been shown to induce fetal bradycardia and hypoglycemia.12,13 Intravenous nitroglycerin has been used successfully but has been associated with loss of beat-to-beat variability in the fetus.14 Labetalol, nicardipine, and hydralazine represent the agents with the greatest efficacy and safety profile of those currently available to anesthesia providers in the United States. Although nicardipine has not been used as extensively in parturients as have labetalol and hydralazine, several studies have shown it to be safe and as effective at lowering mean arterial pressure as labetalol. In addition, as a dihydropyridine calcium channel blocker, nicardipine provides a certain degree of tocolytic effect.15

After initial control of hemodynamics is accomplished, selection of an anesthetic plan can proceed. Anesthetic choice for delivery of the fetus is heavily influenced by the type of dissection, the patient’s physiologic condition, and the need for emergency aortic repair or replacement after delivery. General and regional techniques have been described in the literature, and no consensus exists on which is most appropriate.

Standard preparation for the patient should include ECG, insertion of 2 large-bore intravenous catheters, an arterial catheter, and, if possible, central venous and pulmonary artery catheters. It is important to remember that if the dissection is type I or II, the femoral artery is the preferred cannulation site for monitoring and eventual cardiopulmonary bypass.9 For induction of general anesthesia, a balance must be struck between the need to rapidly secure the airway and the need to prevent wide swings in hemodynamics. Recommendations have been made for a “modified” rapid-sequence induction that allows for cricoid pressure with manual ventilation and titration of anesthetic agents, which provides for some airway protection and limited hemodynamic stimulation.9 Alternatively, the successful use of extradural anesthesia for a parturient with a type III dissection has been reported using a slowly titrated dose of 2% lidocaine without a fluid preload.16 No matter which approach is used by the anesthetist, a multispecialty approach has been recommended by several sources. Therefore, consultation among obstetrics, cardiothoracic, cardiology, and anesthesia teams should be considered key to a successful outcome for the mother and fetus.

Certified Registered Nurse Anesthetists administer approximately 65% of all anesthetics given each year in the United States and are the sole anesthesia providers in approximately two thirds of all rural hospitals in the United States as estimated by the AANA.17 Many of these providers may be the only experienced providers of critical care at their respective institutions. Anesthesia-oriented journals are often relied on heavily for continuing education and proficiency in the practice of anesthesia. After an extensive review of the currently available medical journal databases, a lack of information on aortic dissection in parturients was identified within the US anesthesia literature. The condition has been well documented in obstetric, medical, and cardiothoracic literature; however, limited time and monetary resources may preclude many anesthesia providers from reviewing such materials. It is our desire to expand the body of nurse anesthesia knowledge through review of this very tragic case.

REFERENCES


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