Emergency Repair of Aortic Dissection in a 37-week Parturient: A Case Report

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Aortic dissection is a life-threatening condition with a 50% mortality rate in the first 48 hours and a 3-month mortality rate of 90% in untreated patients. Aortic dissection is a rare complication of pregnancy, but there is significant morbidity and mortality for the mother and infant.

A 43-year-old woman with a 37-week intrauterine pregnancy was admitted to the emergency department 6 hours after the onset of tightness in her throat and neck pain. She described the pain as similar to pain she experienced with a myocardial infarction 2 years previously. Other pertinent history included hypertension, gestational diabetes, coronary artery disease, and a family history of aortic dissection. The patient was initially misdiagnosed. Transthoracic echocardiogram and computed tomography scan revealed a type A thoracic aortic dissection extending into the abdominal aorta. An emergency aortic repair and cesarean section were successfully performed.

Recognition of aortic dissection and an evidence-based, collaborative approach to optimize treatment and recovery are vital to the patient's survival. The purpose of this article is to highlight successful management of aortic dissection in a parturient and to broaden the body of literature on the topic.

Keywords: Aortic, anesthesia, dissection, pregnancy, thoracic.

ortic dissection is a life-threatening condition with a 50% mortality rate in the first 48 hours and a 3-month mortality rate of 90% in untreated patients.[1] Most commonly, patients die of subsequent aortic rupture and cardiac tamponade.[2] Aortic dissection is a rare complication of pregnancy, but there is significant morbidity and mortality for the mother and infant. Outcome is poor, with maternal mortality as high as 25% and fetal mortality even higher.[3,4] The pregnant state accounts for more than 50% of dissections occurring in women younger than 40 years.[5] This article describes a report of an aortic dissection that occurred late in pregnancy in which the patient underwent cesarean section and then immediate dissection repair.

Case Summary

A 43-year-old woman with a 37-week intrauterine pregnancy, gravida 2, para 0, was admitted to the emergency department in the evening, 6 hours after the onset of tightness in her throat and neck pain. The patient stated that she was concerned because the pain was similar to what she experienced with a myocardial infarction (MI) 2 years previously. Her vital signs were stable: blood pressure, 137/57 mmHg; heart rate, 84/min; respiratory rate, 20/min; oxygen saturation 97%; and temperature, 98.4°F. She weighed 91.8 kg and was 165 cm tall. Abdominal ultrasound and fetal heart tones were normal, and the patient was not having contractions. An electrocardiogram revealed normal sinus rhythm at 80/min. Samples were drawn for laboratory testing, including complete blood cell count, chemistry, serial cardiac enzymes, and a blood type and screen. The patient's medical history included chronic hypertension, coronary artery disease, an MI 2 years previously with left anterior descending coronary artery stenting, a negative stress test during the past 12 months, and gestational diabetes. There was no history of Marfan syndrome or connective tissue disorders. The patient's surgical history also included tonsillectomy. Before pregnancy, the patient was taking metoprolol, atorvastatin, and lisinopril but had only been taking prenatal vitamins, insulin, and aspirin during pregnancy. The patient had an allergy to penicillin and denied use of alcohol, tobacco, and drugs. Her obstetrical history included an abortion at 19 years and intrauterine insemination for the current pregnancy. Her family history was significant for lethal descending aortic dissection in her mother at age 54 years and paternal grandmother at 65 years.

Soon after admission to the emergency department, the patient developed epigastric pain and vomited stomach contents. A gastrointestinal consult was ordered. A "gastrointestinal cocktail" of aluminum/magnesium concentrate and viscous lidocaine was given along with intravenous ondansetron, 8 mg; meperidine, 25 mg; and pantoprazole, 40 mg. The gastroenterologist reported that the patient's pain had decreased after the meperidine but that the patient had some substernal discomfort. Review of systems and physical examination were reported to be normal. Abdominal ultrasound revealed multiple gallstones and a contracted gallbladder; the aorta was not well visualized. Laboratory values were normal: hemo-

globin, 13.8 g/dL, white blood cell count, 9,500/µL; platelet count, 246 × 10³/µL; troponin I, less than 0.04 ng/mL; and creatine phosphokinase, 54 U/L. The initial diagnosis was cholecystitis, and the plan was to start sliding scale insulin, admit the patient to labor and delivery, and observe her overnight for return of symptoms.

The patient was admitted, and the cardiology service was consulted the following morning because of the patient's history of MI and the family history of aortic dissection. The patient was asymptomatic during the consult with the following vital signs: blood pressure, 105/40 mm Hg; heart rate, 70/min; and respiratory rate, 18/min; and she was afebrile. The cardiologist's physical examination noted a grade 2-3/6 diastolic murmur at the left upper sternal border. The electrocardiogram was repeated and showed normal sinus rhythm with a rate of 68/min. No additional laboratory results were available. A transthoracic echocardiogram was done that showed normal left ventricular systolic function (ejection fraction, 65%), normal wall motion, and moderate aortic regurgitation. A dissecting flap in the ascending aorta/aortic root, characteristic of a type A aortic dissection, was also visualized. At this point, the cardiovascular surgery team was notified, the patient was transferred to the intensive care unit, and an urgent computed tomography (CT) scan was done to confirm the dissection. Beta-blockade was initiated, and a toxicology screen for cocaine was done, the results of which were negative. The CT scan confirmed the diagnosis and revealed that the dissecting aortic aneurysm continued into the abdominal aorta. The thoracic portion extended into the innominate artery, and pericardial and pleural effusions were present. Surgery was then discussed with the patient, and she was immediately taken to the operating room for emergency cesarean section and repair of the ascending aortic dissection.

At 20 hours after the onset of symptoms, the patient entered the operating room for cesarean section and type A aortic dissection repair. She was without premedication and was anxious and tearful. She was positioned supine with mild left uterine displacement; vital signs were stable with a blood pressure of 120/48 mm Hg, a heart rate of 92/min, respiratory rate of 24/min and oxygen saturation was 100% with oxygen by face mask. The fetal heart rates were 150 to 160/min. Two units of typed and cross-matched packed red blood cells were available in the room. A radial arterial line and a 20-gauge peripheral intravenous line were in place. The patient was prepped and draped, and then rapid-sequence induction was performed with midazolam (2 mg), fentanyl (150 µg), rocuronium (5 mg), etomidate (20 mg), and succinylcholine (200 mg). Propofol (200 mg) was given during induction in divided doses. The patient was then intubated without difficulty while cricoid pressure was maintained. The blood pressure was 104/52 mm Hg with a heart rate of 90/min after induction. During intubation, the cesarean section was started and the infant was delivered 2 minutes after skin incision. A classical (midline longitudinal) uterine incision was made secondary to abnormal venous plexuses located on both broad ligaments and the need for hemostasis. A 6-lb, 6-oz girl was born with 1- and 5-minute Apgar scores of 0 and 7, respectively. The neonate was mask-ventilated and intubated by an anesthesiologist. She was then taken to the neonatal intensive care unit where she was extubated that evening and eventually weaned to room air.

Following delivery, propofol (40 µg/kg per minute) and sufentanil (0.3 µg/kg per hour) infusions were initiated and titrated to effect. Muscle relaxation was maintained throughout the case with vecuronium (total, 18 mg), and anesthesia was maintained with isoflurane throughout the case (end-tidal concentration, 0.6%-1.0%). Metoprolol (1 mg) and furosemide (20 mg) were also given immediately after delivery, oxytocin (20 U/L) was started and infused at 125 mL/h, and vancomycin (1 g) was given slowly. Misoprostol (1,000 mg) was given rectally. A peritoneal drain was placed to monitor for increased bleeding after heparinization for cardiopulmonary bypass (CPB). During closure of the abdominal incision, an introducer sheath and quadruple-lumen catheter were inserted with a double stick of the right internal jugular vein. A pulmonary artery catheter was inserted to 47 cm; the pulmonary artery pressure was 41/27 mm Hg, the central venous pressure was 15 mm Hg, and mixed venous saturation, 52%. Transesophageal echocardiography (TEE) was also performed and was used throughout the aortic repair and for weaning from CPB.

Before and during sternotomy, 3 boluses of sufentanil (20 µg each) were given, with a total of 140 µg given during the entire case, including the infusion. Midazolam (2-mg boluses) was given every hour before and after CPB, and fentanyl (100-mg boluses) was given before CPB with totals of 10 and 250 mg given, respectively. Sodium nitroprusside, nitroglycerin, and phenylephrine were titrated throughout the case to maintain hemodynamic stability. An insulin infusion was titrated, and blood glucose level ranged from 94 to 183 mg/dL. Amiodarone (150-mg bolus followed by an infusion at 1 mg/min) and aminocaproic acid (5-g loading dose followed by a 2-g/h infusion) were also maintained. Femoral cannulas were used for CPB after the patient was fully heparinized with 65,000 U. The patient's head was wrapped in ice, and she was cooled to 11°C for circulatory arrest. The CPB period was uneventful with unremarkable hemodynamic alterations. Etomidate (20-mg boluses) was given every 30 minutes during CPB. The patient had an intimal tear just above the sinotubular junction with moderate non–coronary sinus leaflet prolapse. Her heart was moderately enlarged but contracted symmetrically. The aortic valve was resuspended, and the aortic arch was repaired with a 24-mm tube graft. Total circulatory arrest was 37 minutes

with antegrade cerebral perfusion of 21 minutes. After the repair, TEE revealed only trace aortic insufficiency. The patient was weaned off of CPB with 1 internal defibrillation and required no additional inotropic agents or vasoactive infusions. Her cardiac index ranged from 2.0 to 2.6 after weaning. The entire case lasted approximately 5.5 hours with no significant intraoperative events. The total urine output was 826 mL, and blood loss during the cesarean was 750 to 1,000 mL. The patient received 956 mL of cell-saver blood, 4 L of lactated Ringer's solution, 1,250 mL of albumin, 1 U of packed red blood cells, 2 U of fresh frozen plasma, and 6 U of platelets.

The patient was transported to the cardiovascular intensive care unit, intubated and in stable condition with insulin (3.1 U/h), oxytocin (62.5 mL/h), propofol (50 μg/kg per minute), amiodarone (1 mg/min), nitroprusside (0.2 μg/kg per minute), and aminocaproic acid (2 g/h) infusions. Morphine (3 mg) was given before transport to the intensive care unit. The patient was extubated 13 hours after surgery and was weaned from all continuous intravenous infusions within 36 hours. Oxytocin was continued until postoperative day 2. Postoperative issues included blood glucose monitoring, abdominal pain, a urinary tract infection, and lower extremity edema. Three days after surgery, her temperature peaked at 102.4°F; urine cultures were sent to the laboratory, and levofloxacin was started for a urinary tract infection. The patient also had a genetics consult to evaluate for connective tissue disorders that might have contributed to the dissection. The consult revealed no significant findings. It was suggested that this was most likely a familial thoracic aneurysm/dissection in which 20% of family members can have aneurysms elsewhere. The specialist suggested DNA sequencing of the transforming growth factor, beta receptor I and II genes to evaluate for familial thoracic aneurysm, which was done and did not reveal any mutation. Patients with mutations of these genes are predisposed to widespread and aggressive vascular disease.[6] It was recommended that the patient and her immediate living relatives undergo further genetic counseling.

On postoperative day 5, the patient was transferred to the step-down unit. She had a transthoracic echocardiogram that confirmed dissection in the descending portion of the aorta that would be medically managed until surgical intervention became indicated. A gastrointestinal consult was also done for right lower quadrant abdominal pain, which revealed normal findings. The pain resolved as soon as the patient's bowel habits were normalized.

The patient recovered well, and her major concerns during hospitalization centered around visiting and breastfeeding her daughter. Her daughter's bilirubin level peaked at 8.4 mg/dL, but did not require treatment, and she had an uneventful hospital course otherwise. It was proposed that the newborn's depressed respiratory status at birth was due to general anesthesia, but the neonate did not receive any reversal agents and was quickly extubated as previously mentioned. One week after surgery, the patient and her newborn were discharged. The patient's discharge medications included aspirin, metoprolol, prenatal vitamins, polyethylene glycol, propoxyphene-acetaminophen combination, levofloxacin, and 2 weeks' worth of furosemide. Her blood glucose level had normalized, and she was instructed to continue monitoring her glucose levels until postoperative follow-up.

Discussion

Aortic dissection occurs when an intimal tear allows blood to flow into the aortic media and create a false lumen filled with blood that is separated from the true lumen by an intimal flap.[7] Although no single disorder is responsible for aortic dissection and the mechanism by which dissection actually occurs is unclear, several risk factors have been identified that can damage the aortic wall and lead to dissection. These include direct mechanical forces on the aortic wall and forces that affect the composition of the vessel.[7] Direct forces include hypertension, hypervolemia, and derangements of aortic flow, such as a bicuspid aortic valve, aortic root dilation greater than 4 cm, and coarctation of the aorta.[7,8] Several studies have found that the presence of a bicuspid aortic valve disrupts normal aortic flow, and histologic studies have revealed diminished aortic elasticity.[9-11] The aortic media is made up of concentric smooth muscle with proteins such as elastin, collagen, and fibrillin within a ground substance. People with deterioration of medial components are at increased risk for development of aortic dissection; this includes connective tissue disorders such as Marfan, Turner, and Ehlers-Danlos syndromes.[7,12] More subtle forms of inherited metabolic disorders are postulated to have a more important contribution than previously considered.[13] Chemical destruction of the media, which accompanies smoking, is implicated as well. Atherosclerosis is only a risk factor in preexisting aneurysms or atherosclerotic ulceration. Iatrogenic trauma caused by catheterization procedures, aortic cannulation, or intra-aortic balloon pumps has also been reported to be related to dissection.[7,12,14] The reported relevance of cocaine use as a risk factor has varied with a very high correlation in one urban study (37% of dissections), but a low incidence (0.5%) was found as part of the International Registry for Aortic Dissection (IRAD).[15,16] Diabetes has also been proposed to contribute to aortic dissection, although the exact mechanism is unknown.[14]

During pregnancy, hypervolemia, increased outflow resistance, and an abnormal hormonal milieu exist. Blood volume, heart rate, cardiac output, left ventricular wall mass, and end-diastolic dimensions increase as pregnancy progresses, and the risk of dissection increases with an increasing gestational period.[14] Outflow resistance is also

increased due to compression of the aorta by the gravid uterus.[14,17] Although the exact role of hormones and receptors in dissection has been debated, estrogen receptors have been found in aortic tissue, and increasing estrogen levels may alter the normal elastic properties of the aorta.[14,18] Several authors have suggested that these physiologic, cardiovascular, and hormonal changes predispose a normal pregnant patient to aortic dissection.[2,4,19] Patients who have additional risk factors such as connective tissue disorders, smoking, and hypertension are at the highest risk for aortic dissection during pregnancy. Hypertension is associated with 25% to 50% of cases.[14] Dissection most commonly occurs during the third trimester or during the first stage of labor, with the majority occurring during the third trimester.[14,20] In addition, there are reports of postpartum aortic dissection.[21]

Two major classification systems are used to describe the involvement of an aortic dissection. DeBakey type I and Stanford type A include dissections that involve the proximal aorta, arch, and descending thoracic aorta. DeBakey type II involves only the ascending aorta and is included in the Stanford type A classification. DeBakey type III and Stanford type B include dissections that originate in the descending thoracic and thoracoabdominal aorta regardless of any retrograde involvement of the arch. These are subdivided into IIIa and IIIb depending on abdominal aortic involvement; type IIIb extends into the abdominal aorta.[7] Pregnancy-associated aortic dissections most commonly occur in the proximal aorta, with an intimal tear originating within 2 cm of the aortic valve in 75% of cases.[14]

The initial challenge for providers is to recognize patients who are at risk for aortic dissection with a thorough history and physical examination because diagnosis requires a high level of suspicion.[7] As was the case with this patient, up to 30% of patients are initially misdiagnosed. For patients with chest pain, differential diagnoses include MI, aortic dissection, pulmonary embolism, musculoskeletal pain, pericarditis, pneumonia, pneumothorax, and acute aortic regurgitation. When severe pain involves the abdomen in a pregnant patient, the practitioner should consider aortic dissection, gallbladder disease, ectopic pregnancy, uterine rupture, abruptio placenta, placenta previa, and liver rupture, among other common diagnoses.[7,22] Most commonly, patients with aortic dissection have sudden, severe, unrelenting chest pain that is not relieved by narcotics. This classic picture occurs in approximately 80% of cases.[19,23] Of the large sample of patients from the IRAD (n = 464), 76% had chest pain as an initial symptom, 55% had back pain, and 18% had migrating pain.[23] The location of maximum pain varies depending on the location of the dissection and changes as the dissection extends.[15] Pain may be substernal, interscapular, or even epigastric and may radiate, similar to an acute MI.[15] Historically, it has been taught that patients

often describe the pain as ripping or tearing, but the IRAD found that patients more often describe the pain as sharp.[15] Painless dissections have been reported but are usually in patients with chronic aneurysmal pain.[7,19,24]

Patients with aortic dissection may have signs and symptoms related to decreased perfusion of the brain, limbs, or visceral organs, which may distract the practitioner away from diagnosis of dissection. Patients usually appear ill and may have nonspecific signs and symptoms such as tachycardia, dyspnea, hypertension, abnormal peripheral vascular examination findings, murmur, S3 heart sound, jugular vein distention, pleural effusion, confusion, rigid abdomen, nausea and vomiting, hemiparesis, hemiplegia, and even MI or stroke.[7,19,24]

Once a high level of suspicion exists, aortic dissection must be confirmed with diagnostic testing, such as aortography or magnetic resonance imaging (MRI). History and physical examination findings may be suggestive but are highly variable and inconclusive. Diagnostic testing may prove difficult because common tests such as chest radiography and 12-lead electrocardiogram may be normal. The IRAD found that 12.4% of patients had normal chest radiographs and that the electrocardiogram most frequently showed nonspecific abnormalities. Electrocardiogram results were normal for 31.3% of the patients with dissection.[15] Aortography is the "gold standard" for diagnosis but is rarely used due to its invasive nature. In addition, evidence suggests that a combination of noninvasive testing yields sensitive and specific results on which to base management of the patient. A study by Nienaber et al[25] found that MRI and TEE more reliably detected aortic dissection than transthoracic methods. In this study, MRI provided more superior anatomical mapping of type A and B dissections and more detailed information on the site of entry and thrombus formation than TEE.[25]

A more recent study found that the sensitivity and specificity (95% and 91%, respectively) of combined transthoracic echocardiogram and CT evaluation were not different from those for TEE or MRI (100% and 96%, respectively). They also found that thrombus formation, side-branch involvement, aortic regurgitation, pericardial effusion, and mediastinal hematoma were also detected with similar sensitivities and specificities.[26] The decision of which noninvasive test to use—MRI, TEE, or a combination of transthoracic echocardiogram and CT—primarily depends on availability.[26] The effects of intravenous contrast during CT and aortography and radiation doses during aortography should be considered.[1,2] The CT scan is currently the most widely used diagnostic tool for aortic dissection, followed by TEE.[7]

The general guidelines for management of dissection in the parturient vary with gestation and location of dissection. Before 28 weeks' gestation, it is recommended that a type A aortic dissection be repaired and the pregnancy continued. After 32 weeks' gestation, the recom-

mendation is a cesarean section followed immediately by dissection repair. Between 28 and 32 weeks' gestation, aortic dissection repair is recommended and delivery at this time is reserved for cases of fetal distress.[4] Immer et al[14] recommend that fetal lung maturation be stimulated at 26 weeks' gestation in patients at risk for dissection and that hospitalization of high-risk patients be considered between 28 and 32 weeks. In addition, they recommend that acute dissections be repaired immediately before 30 weeks of gestation and that repair be preceded by cesarean section after 30 weeks' gestation.[14] Performance of CPB during pregnancy is not without risk, and reports have suggested a 2% to 6% maternal mortality rate and a 20% to 30% fetal mortality rate.[4,27-29] Fetal hypothermia associated with CPB may be detrimental because, although it decreases oxygen consumption, significant fetal hypoxia may result from constriction of uterine blood flow. Warming after hypothermia may also result in contractions and premature labor. It is therefore recommended to use high-flow, high-pressure normothermic CPB.[4,27,29] The use of CPB in the first trimester is associated with congenital malformations.[30]

Type B dissections can be managed medically in the absence of rupture and signs of malperfusion.[31] Medical management of dissection in a pregnant patient includes prevention of hypertensive episodes and frequent monitoring of the aorta by using echocardiograms. Beta-blockers are the drug of choice, although effects on the fetus may include hypoglycemia, bradycardia, and interference with fetal growth. Sodium nitroprusside may also be considered, although a risk of fetal cyanide toxicity exists.[14,19,32(p732-736)] Intravenous nitroglycerin may cause loss of fetal heart rate beat-to-beat variability but showed no effect on acid-base status in the study by Cotton et al.[33] Hydralazine has been used extensively with no detrimental fetal effects.[32(p420-422)] The study by Vigil-De Gracia et al[34] found that labetalol and hydralazine both met the criteria for antihypertensive treatment in pregnant patients, although neonatal bradycardia and hypotension occurred more frequently with labetalol.

When delivery is undertaken in a parturient with aortic dissection, anesthesia should be planned and implemented carefully. The risk of hemodynamic stress secondary to rapid-sequence induction, laryngoscopy and intubation, and regional anesthesia should be considered with the goal of minimizing lability to avoid progressive dissection and aortic rupture. Along with control of maternal hemodynamic status, fetal exposure to cardiorespiratory and central nervous system depressants should also be minimized.[35-37] Successful management of parturients with aortic dissection using general or regional anesthesia is reported, and there is no consensus regarding the superiority of either approach.[37,38] Epidural anesthesia decreases vessel wall shearing forces and tension, although a patient with Marfan syndrome would be more prone to epidural

hematoma due to the associated increased epidural vein fragility.[30] Patients with Marfan syndrome, aortic root enlargement, or other risk factors in the absence of dissection should also be managed carefully during delivery to prevent dissection; management may include invasive monitoring, pharmacologic treatment, and intraoperative TEE. A multidisciplinary evaluation of the risks and benefits of vaginal vs cesarean delivery must also take place because both are controversial. Vaginal delivery results in less blood loss and fewer postoperative complications; however, it is highly unpredictable and may be associated with greater hemodynamic stress. Cesarean section provides more control over delivery but is coupled with increased bleeding and the consequences of anesthesia.[38]

Conclusion

Aortic dissections in pregnant patients are underreported in anesthesia literature. One year before the present case, a report was published in the anesthesia literature about a fatal aortic dissection in a 33-week parturient.[39] The case reported in this article represents successful management and a favorable outcome in a similar situation. Although aortic dissection in pregnant patients presents very complex issues with the potential for a doubly tragic outcome, a multidisciplinary, calculated, and vigilant approach gives anesthesia providers the most likely opportunity for accurate diagnosis and a successful outcome. The purpose of this case report is to further enhance nurse anesthetists' knowledge and to bolster confidence in managing similar cases. It is also intended to further expand the limited body of anesthesia literature on this topic.

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