Coronary artery dissection is a rare, sometimes fatal traumatic condition, with 80% of cases affecting women. The coronary artery develops a tear, causing blood to flow between the layers, which force them apart.

We describe the case of a 35-year-old multiparous woman with acute myocardial infarction secondary to right coronary artery dissection at 32 weeks’ gestation. The patient underwent successful primary percutaneous coronary intervention with the placement of 4 coronary stents. She subsequently experienced preterm labor and preeclampsia 11 days following the procedure. Elective cesarean delivery under general anesthesia was performed uneventfully at 35 weeks’ gestation.

Keywords: Coronary dissection, general anesthesia, myocardial infarction, pregnancy.

Pregnancy-associated acute myocardial infarction (AMI) is rare but is associated with a high incidence of morbidity and mortality of both the mother and fetus.1 The estimated incidence of AMI during pregnancy is 3 to 10 cases per 100,000 deliveries with an overall maternal mortality rate of 21% and fetal mortality rate of 13%.3

Pregnancy-associated changes in the cardiovascular system and coagulation system increase the risk of myocardial ischemia by approximately 3- to 4-fold.1 The most common underlying mechanisms implicated in AMI during pregnancy are atherosclerotic coronary artery disease, coronary thrombosis with hypercoagulability, and coronary artery dissection. Spontaneous coronary artery dissection is a rare cause of infarction in the general population, but a common cause of myocardial infarction during pregnancy, and especially in the 3 months following delivery.2 The cause for dissection may be attributed to progesterone-mediated biochemical and histologic changes that occur in the arterial wall during gestation.3-5

We present the case of a multiparous parturient who suffered an AMI with right coronary artery dissection at 32 weeks’ gestation. The patient subsequently experienced preeclampsia and preterm labor. She delivered by cesarean delivery under general anesthesia at 35 weeks’ gestation. This case explores the medical, obstetric, and anesthetic care of this patient.

Case Summary
A 35-year-old gravida 5, para 4 woman, 170 cm tall and 60 kg in weight, with a body mass index of 29 kg/m² at 33 weeks’ gestation arrived at the Emergency Department by emergency medical services (EMS) transfer. She arrived approximately 45 minutes after the acute onset of left-sided chest pain radiating down her left arm with associated dizziness, dyspnea, and nausea. The patient denied previous episodes of chest pain. No history of cardiovascular risk factors, such as diabetes mellitus, hypertension, smoking, or family history of coronary artery disease existed. There was no history of coagulopathy or antiphospholipid antibody syndrome, which are associated with a thrombotic tendency. The patient’s medical history was unremarkable, with the exception of a previous pregnancy 4 years earlier that was complicated by eclampsia and required an emergent cesarean delivery at 33 weeks’ gestation. In the current pregnancy the patient was receiving prenatal care, and her pregnancy had been uncomplicated to this point. Home medications included only prenatal vitamins.

The patient was in distress but alert and oriented. Her temperature was 36.7°C, blood pressure was 159/102 mm Hg in both arms, pulse was 82/min, respiratory rate was 20/min, and oxygen saturation measured with pulse oximetry was 98% on room air. The breath sounds were normal bilaterally. Findings of the cardiac examination revealed no obvious murmurs, gallops, or pericardial rub. The electrocardiogram (ECG) demonstrated normal sinus rhythm, ST-segment elevation in leads II, III and aVF, V₅, and V₆ with T-wave inversion in leads V₁ and V₂. Laboratory results were as follows: troponin I, 0.045 ng/mL; creatinine kinase (CK)-MB fraction, 0.84 ng/mL; total CK, 53 U/L; lactate dehydrogenase, 432 U/L; triglycerides, 275 mg/dL; total cholesterol, 219 mg/dL; high-density-lipoprotein cholesterol, 78 mg/dL; and low-density-lipoprotein cholesterol, 123 mg/dL.

The patient was placed on oxygen therapy and was treated with aspirin, clopidogrel, unfractionated heparin, metoprolol, morphine, and intravenous nitrates. She was urgently transported to the cardiac catheterization labora-
tory, where coronary angiography revealed a completely dissected right coronary artery. At that time, 4 bare metal stents were placed. Immediately on completion of the procedure the patient experienced reperfusion ventricular fibrillation and required defibrillation approximately 10 times until converted to sinus rhythm. Following the procedure, she was transferred to the cardiac intensive care unit (ICU) and started on a regimen of carvedilol, 12.5 mg; aspirin, 325 mg; ferrous sulfate, 325 mg; hydralazine, 1 mg; clopidogrel, 75 mg; simvastatin, 20 mg; and prenatal vitamins. The patient remained stable following the procedure and on postoperative day 3 she was transferred to telemetry. She had no recurrent episodes of chest discomfort and improved greatly.

By hospital day 5 she was deemed stable for discharge to home. Predischarge echocardiogram showed an ejection fraction (EF) of 35% to 40% with mild tricuspid regurgitation. However, the patient was free of shortness of breath, chest pain, or ectopy on ECG.

Six days following her discharge, the patient was readmitted because of complaints of contractions that were increasing in frequency and intensity. She also complained of a headache that began that morning. She denied visual changes, right upper quadrant pain, nausea, vomiting, chest pain, or dyspnea. Blood pressure at the time of admission was 155/108 mm Hg. She was alert and oriented, lungs were clear to auscultation, and she had a regular heart rate and rhythm with a grade 2/6 systolic ejection murmur at the left sternal border, with no gallops or rubs. Extremities had no edema or clonus. Deep tendon reflexes were 2+. Results of her laboratory results (magnesium level, complete blood cell count, basic metabolic profile, and coagulation studies), blood pressure, and cardiac function remained stable. After multiple discussions with the anesthesia, neonatology, perinatology, and cardiology departments, the decision was made to proceed with a cesarean delivery at 35 weeks’ gestation, 17 days following her AMI. Because of her anticoagulation with aspirin and clopidogrel, regional anesthesia was not an option, and cesarean delivery under general anesthesia was agreed on by obstetrics, cardiology, and anesthesiology.

On hospital day 6, the patient was brought to the operating room, where monitors including ECG, noninvasive blood pressure cuff, pulse oximeter, and fetal heart rate monitor were placed. She was positioned supine with left uterine displacement. Preinduction vital signs were as follows: blood pressure, 130/58 mm Hg (mean arterial pressure, 106 mm Hg); heart rate, 68/min; respiratory rate, 16/min; and arterial oxygen saturation (SaO2), 98% on 10 L of oxygen via face mask. Midazolam, 1 mg, was given intravenously (IV) to promote relaxation. A cordis was inserted into the right internal jugular vein for monitoring of central venous pressure (10 mm Hg), and a left radial arterial line was placed. A Vigileo monitor with FloTrac sensor (Edwards Lifesciences, Irvine, California) was used to measure continuous cardiac output and index before and during induction. FloTrac/Vigileo technology allows the cardiac output to be determined continuously using pulse wave analysis without external calibration. It samples pressure wave signals using a standard arterial line. The standard deviation of pulse pressure is empirically correlated to the stroke volume based on patient characteristic after automatic adjustment for actual vascular compliance and is displayed as continuous cardiac output.

Rapid-sequence induction with tricoid pressure was initiated. Propofol, 200 mg, followed immediately by succinylcholine, 80 mg, was given IV. A 7-mL internal-diameter endotracheal tube was easily placed, verified, and secured. Administration of sevoflurane at less than 1 minimal alveolar concentration was implemented. An additional 1 mg of midazolam, 150 μg of fentanyl (Sublimaze), and rocuronium (Zemuron), 30 mg, were administered IV.

The cardiologist was present at induction and placed a transesophageal echocardiogram (TEE) probe to assess ventricular wall motion and valvular function. Fetal heart tones and the patient’s vital signs remained stable throughout induction and surgery. Intraoperative TEE showed a left ventricular EF of 20% to 29% with global hypokinesis. The cardiologist elected to initiate a dopamine infusion at 3 μg/kg/min to improve cardiac contractility. A 1,982-g female infant was delivered with Apgar scores of 8 and 9 at 1 and 5 minutes. Total blood loss was noted to be 400 mL, and 1,500 mL of crystalloid solution was given. The neuromuscular blockade was reversed with neostigmine, 5 mg, and glycopyrrolate, 1 mg. The human side effects. She had responded well to magnesium sulfate and had a decrease in contractions without substantial side effects. She was continued on her home medication regimen. She was given IV. She was given IV. A 7-mL internal-diameter endotracheal tube was easily placed, verified, and secured. Administration of sevoflurane at less than 1 minimal alveolar concentration was implemented. An additional 1 mg of midazolam, 150 μg of fentanyl (Sublimaze), and rocuronium (Zemuron), 30 mg, were administered IV.

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The patient was stable and asymptomatic following delivery. She had another echocardiogram, which demonstrated an EF of 30% to 35% with 3+ tricuspid and 3+ mitral regurgitation and moderate pulmonary hypertension. Although no substantial ECG changes were appreciated, she had a slight increase in troponin levels postoperatively, which was thought to be due to ischemia secondary to anemia (hemoglobin, 8.7 g/dL, and hematocrit, 27%) following the delivery. She received 2 U of packed red blood cells, which increased her hemoglobin and hematocrit concentrations to 10.6 g/dL and 31%, respectively, and normalized her troponin levels. The dopamine infusion was discontinued, and a milrinone infusion was initiated at 0.375 μg/kg/min once she arrived at the ICU. The milrinone infusion was discontinued after 2 days.

On postoperative day 1, she was restarted on a regimen of aspirin, 325 mg/d; clopidogrel, 75 mg/d; hydralazine, 25 mg/d; and carvedilol, 3.125 mg twice daily, which was titrated up to 6.25 mg twice daily to maintain a heart rate of 60/min. She was also started on lisinopril therapy, 5 mg/d. She remained stable and was discharged home 5 days following delivery, with instructions to follow up with cardiology as an outpatient in 2 weeks. At the time of discharge, the baby was doing well but remained hospitalized secondary to feeding concerns.

Discussion

The first documented report of myocardial infarction (MI) during pregnancy was in 1922.1,4,5 Myocardial infarction during pregnancy is a rare complication with an estimated occurrence of 1 in 10,000 deliveries.1,4,5 This estimate is expected to rise with the trend of older women conceiving in conjunction with other risk factors such as smoking or increased cholesterol.1 In a study performed by Jolly et al,6 the authors suggest that other risk factors, including diabetes mellitus and hypertension, that plague older parturients can lead to myocardial infarction, still births, premature labor, low birth weights, and fatal congenital anomalies. Although MI can occur in any stage of labor, it is most common in the third trimester of multigravida women who are older than 33 years.7 The overall maternal mortality rate is slightly above 20%, with the greatest mortality occurring in those patients sustaining an MI in late pregnancy.1 Hankins et al9 and Hands et al8 emphasized that most maternal deaths occurred either at the time of infarction or within 2 weeks of infarction, usually in relation to the onset of labor and delivery. Increased workload of the maternal heart that occurs during the late stages of pregnancy and delivery may contribute to the overall poor prognosis of patients who have experienced an MI.

Management of an acute infarct in the pregnant patient is the same as in a nonpregnant patient. However, the pregnant patient may pose more difficulties in treatment related to the physiological changes that occur during pregnancy and the presence of a fetus. Early consultation among the cardiology, obstetric, and anesthesia teams is imperative to have the best possible outcome for mother and baby. The consultation among the teams provides for careful and coordinated planning of the elective labor or, alternatively, the optimal management of an unexpected, premature labor, and for a prompt and effective rescue of the fetus in the event of sudden maternal demise. The pregnant patient should be transferred to an ICU capable of continuous fetal monitoring and continuous ECG monitoring. Initial therapy includes bed rest, supplemental oxygen, pain relief, and anticoagulation therapy to prevent deep vein thrombosis and occlusion to bare metal stents if placed. Therapy with β-blockers is initiated to prevent further episodes of myocardial ischemia. The patient in our case report was started on carvedilol treatment at a dosage of 6.125 mg twice daily. This medication is classified as category C because it has known teratogenic effects in animal models. There is no evidence that β-blockers cause fetal abnormalities, but fewer fetal and neonatal abnormalities have been reported with the use of more selective β1-receptor blockers than with nonsselective β-blockers.9,10

Cardiac failure should be treated promptly. The combination of supplemental oxygen, inotropes, chronotropic agents, rest, salt restriction, and diuretics usually is implemented. Diuretics, however, should be given cautiously because of the risk of electrolyte disturbances in the mother and fetus. In the preeclamptic patient, they are contraindicated because of the low plasma volume of the patient.

Vasodilators such as milrinone may be introduced to decrease afterload and/or preload in the patient with cardiac failure. Hydralazine, 25 mg, which was part of our patient’s daily medication regimen, has been widely used for decades during pregnancy without any adverse effects on the mother or fetus.4

Anticoagulation therapy usually is begun to prevent deep vein thrombosis in the patient on bed rest. However, it was also used to prevent occlusion of her bare metal stents. It is unknown why the cardiologist chose to employ bare metal stents over drug-eluting stents. However, it can be extrapolated that bare metal stents were used because drug-eluting stents are cytotoxic. It is also recommended that antiplatelet therapy be continued for 12 to 24 months after intervention, as opposed to 6 weeks for bare metal stents before any elective surgical procedure.11 Warfarin is contraindicated, as it crosses the placenta and causes intracranial bleeding of the fetus.12 Heparin is usually the anticoagulant of choice during pregnancy because it does not cross the placenta.
However, it can cause thrombocytopenia, which may be of concern in a preeclamptic patient with decreased platelet counts. Research supports that heparin is safe and efficacious when used in preeclamptic patients considering that HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count) has been excluded, systolic blood pressure is less than 180 mm Hg, and diastolic blood pressure is less than 110 mm Hg.\(^\text{13}\)

Clopidogrel, 75 mg, was given daily for therapy in our patient. There are no current well-controlled studies in pregnant women that prove clopidogrel is teratogenic in human models. It was found to have no effect on fertility or fetotoxicity of male and female rats at oral doses up to 500 mg/kg/d (65 times the recommended human dose on a milligrams per meters squared basis).\(^\text{14}\)

The mother’s status and fetal maturity will ultimately affect the timing of delivery. If possible, delivery should be postponed for almost 2 weeks after infarction.\(^\text{4}\) The type of delivery should be individualized and chosen on the basis of which method will prevent the least hemodynamic instability. Close communication and collaboration among the cardiac, obstetric, and anesthesia teams are of the utmost importance. Although vaginal and cesarean deliveries both have advantages and disadvantages, cesarean delivery under general anesthesia was the safest option in our case. Cesarean delivery under regional anesthesia was discouraged because the patient was anticoagulated, and a vaginal delivery was put off because of previous cesarean delivery and an increased risk of uterine rupture.

During anesthesia, it is important to maintain adequate oxygenation and pain control to minimize reinfarction and myocardial oxygen demand and to optimize coronary and placental perfusion. The patient should be positioned with slight left uterine displacement to prevent aortocaval syndrome. Monitoring devices include use of continuous cardiac output, arterial line, TEE, ECG, pulse oximeter, and fetal heart rate.

Postpartum management should include a thorough assessment of cardiac function. This assessment should include echocardiogram, ECG, and laboratory workup. If the cause of MI is unknown, this assessment may provide some clues. It may also yield prognostic information that will help guide the cardiologists in future management. With this information, appropriate decisions can be made regarding exercise regimen, anticoagulation, and antianginal therapy, and regarding the advisability of undertaking a future pregnancy. The patient’s follow-up echocardiogram 2 weeks after discharge revealed a stable EF of 40% with mild mitral valve regurgitation and mild to normal pulmonary pressure.

Subsequent pregnancies after MI are not absolutely contraindicated. It depends on the amount of myocardial damage and ventricular function. Patients with deprived left ventricular function will have a poor prognosis. A bilateral tubal ligation was performed at our patient’s request after delivery of the fetus.

Anesthesia implications for this patient population include early recognition of MI by the anesthesia provider. Early discontinuation of antiplatelet therapy in a patient with cardiac stents can be problematic for the anesthesia provider. There is an increased risk of occlusion of the stents, which can lead to ischemic changes on the ECG. Early recognition of subtle changes in cardiac output and index that may indicate heart failure must be managed early and efficiently. Positive chronotropes and inotropes must be readily available. Effective communication between the cardiologist and anesthesia provider will ensure the appropriate time when these medications should be administered, while continually assessing ventricular function via TEE. Heart rate and contractility must be maintained at baseline or slightly higher because of the patient’s mitral valve regurgitation. Large-bore IV catheters or a central line is necessary to provide adequate fluid resuscitation in case of hemorrhage. A right internal jugular cordis was placed using ultrasound to decrease risk of bleeding. Close monitoring of blood loss is essential to prevent reinfarction caused by ischemia and hypovolemia. The anticoagulated patient having high-risk procedures should be typed and crossmatched and have blood readily available. Four units of packed red blood cells were available in the operating room, and 4 more units were on hold in the blood bank. No blood products were administered intraoperatively.

**Conclusion**

Spontaneous coronary artery dissection is a rare occurrence in the general population; however, it is the most common cause of MI in pregnant women. The medical team must always consider coronary artery dissection in a pregnant woman who complains of chest pain. Prompt diagnosis of this rare and severe condition contributes to a substantial improvement in the prognosis of both mother and fetus.\(^\text{15}\)

Anesthesia providers, when determining what type of anesthetic to deliver, must take every aspect of the patient’s health into consideration. This includes performing a thorough and efficient physical assessment as well as evaluating the results of all pertinent laboratory and diagnostic studies. Anesthesia providers are a crucial link to determine what type of outcome the patient will have. The goal is to provide the safest anesthetic for individuals in order to maximize a positive outcome.

**REFERENCES**


**AUTHORS**

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