Caesarian Dissection
A Case of Chest Pain and Dyspnea During Delivery

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Forward

Information about a real patient is presented in stages (boldface type) to an expert clinician (Dr Nandita S. Scott), who responds to the information, sharing her reasoning with the reader (regular type). A discussion by the authors follows.

A 35-year-old woman, G2P1, with a history of Hashimoto’s thyroiditis, vitiligo, and gestational diabetes mellitus presented in the 30th week of pregnancy with premature labor. She was admitted to the obstetrics service for monitoring and tocolytic therapy. On hospital day 2, the patient complained of chest heaviness and left arm discomfort, but her assessment was confounded by pain secondary to frequent uterine contractions. On hospital day 3, fetal monitoring disclosed a nonreassuring fetal heart rate pattern, prompting transfer to the operating room for urgent Caesarian section. A viable female infant was delivered, but the immediate postoperative course was complicated by acute dyspnea, coughing, and hypoxia.

Dr Nandita S. Scott: Dyspnea is quite common during pregnancy as a result of physiological changes including weight gain, dilutional anemia of pregnancy, and progesterone-induced hyperventilation. This can make the distinction between pathology and normal dyspnea of pregnancy difficult. In contrast, hypoxia during pregnancy is never normal. The cardiac causes of hypoxia in the peripartum period can be broadly separated into 2 categories: congestive heart failure from preexisting cardiac conditions (eg, cardiomyopathy, valvular disease, or congenital heart disease) or congestive heart failure attributable to pregnancy-induced cardiac conditions (eg, peripartum cardiomyopathy or acute ischemic events). Pulmonary causes such as pulmonary embolism and anamniotic fluid embolism also need to be considered.

Pregnancy poses a large hemodynamic burden on the cardiovascular system. A 30% to 50% rise in blood volume and cardiac output, increase in heart rate, reduction in systemic vascular resistance resulting from the low-resistance placental unit, physiological anemia, and positional reduction in cardiac output attributable to compression of the inferior vena cava all contribute to stress the heart.1 Pregnancy may therefore unmask a preexisting cardiac condition that was unknown before conception. At delivery, this hemodynamic burden increases further as a result of the autotransfusion of uteroplacental blood, tachycardia from pain, and the loss of the low-resistance placental unit. Consequently, the cardiovascular adaptations of pregnancy remain clinically relevant after delivery of the fetus. Indeed, it may be months before the circulation fully returns to normal.

This patient had no preexisting cardiac conditions that we are aware of. On hospital day 2 she developed chest pain, which suggests a possible ischemic cause for her symptoms. Peripartum cardiomyopathy is a less likely explanation for her symptoms, because this patient was only 30 weeks pregnant and peripartum cardiomyopathy occurs most often in the last month of pregnancy and the first 5 months after delivery.

Intravenous furosemide was administered with adequate urine output and improvement in dyspnea. Cardiology was consulted. At the time of cardiology evaluation, the patient was afebrile with a blood pressure of 99/63 mm Hg, heart rate of 100 beats per minute, and oxygen saturation of 98% on 1 L of supplemental oxygen by nasal cannula. Physical examination was notable for bibasilar rales, estimated jugular venous pressure of 11 cm H2O, regular tachycardia without murmurs, rubs, or gallops, and a nondisplaced point of maximal impulse.

Dr Scott: Just as symptoms that would be considered abnormal in the nonpregnant state may be normal during pregnancy, it is important to recognize that certain notable physical examination findings may be considered normal during pregnancy. These include the following: prominent X and Y descents; distinct A and V waves of the jugular venous pulsation; full systemic arterial pulses with brisk collapse; a hyperdynamic left ventricular impulse; palpation of the right ventricle and pulmonary trunk; a loud S1; splitting of S1; a loud S2; flow murmurs; diastolic murmurs attributable to increased flow across the mitral and tricuspid valves; a venous hum over the right supraclavicular fossa; and mammary souffle.7 A prominent JVP is normal in pregnancy, but elevation of the JVP is not normal.

In this patient, the bibasilar rales and significantly elevated JVP suggest volume overload. The absence of a murmur argues against valvular disease.
A 12-lead ECG demonstrated sinus tachycardia, small Q waves in III, aVF, and V3-V6, poor R-wave progression, and 0.5-mm anteroapical ST elevations with biphasic T-waves (Figure 1). Troponin-T was 2.80 ng/mL. N-terminal probrain natriuretic peptide (NT-proBNP) was 1525 pg/mL. The white blood cell count was 21.0 cells per high-power field, hemoglobin 10.2 g/dL, hematocrit 31.0%, and platelet count 415 thrombocytes per high-power field. A basic metabolic panel was within normal limits. Chest radiography demonstrated patchy opacities at the bases bilaterally with perihilar fullness and indistinct pulmonary vasculature with peribronchial cuffing, consistent with pulmonary edema (Figure 2).

Dr Scott: The patient’s BNP and troponin were elevated. BNP levels during pregnancy may rise but generally appear to stay within the normal range. The rise is greater in those with gestational hypertension and pre-eclampsia. A BNP <100 pg/mL in the pregnant patient with preexisting heart disease has a 100% negative predictive value for adverse cardiac events.

Troponin levels have also been studied during pregnancy and are generally felt to remain in the normal range, but they may rise to the upper limit of normal. Like BNP, troponin levels are higher in women with hypertensive disorders of pregnancy, particularly pre-eclampsia.

The elevated troponin and BNP values in this patient, coupled with ST segment elevation and abnormal chest x-ray findings, suggest acute myocardial injury with congestive heart failure. The presence of Q-waves suggests that the precipitating event may have occurred some time before the cardiology team was consulted.

While planning the next diagnostic steps, therapeutic measures are indicated. Treatment for presumed acute coronary syndrome (ACS), including heparin, aspirin, and high-dose statin, is reasonable. Administration of a loop diuretic is indicated for treatment of dyspnea and hypoxia. However, β-blocker administration is relatively contraindicated given the evidence of acute heart failure. Though guidelines would support administration of a thienopyridine, many institutions prefer to withhold clopidogrel loading until the coronary anatomy is clear in the event that coronary artery bypass graft (CABG) surgery is needed. Use of an intravenous glycoprotein IIb/IIIa inhibitor would be reasonable, but the bleeding risks would be significant in this patient so soon after delivery and Caesarian section.

Unfractioned intravenous heparin, atorvastatin 80 mg, and aspirin 325 mg were administered, and intravenous furosemide was continued. Given the diagnostic uncertainty at this point, clopidogrel was not administered. The patient was transferred to the cardiac stepdown unit for further management. At this point she was clinically stable, responding to diuresis, and pain-free. Because she was without pain and Q-waves were evident on electrocardiography, bedside echocardiography was performed to identify regional wall motion abnormalities and assess the left ventricular ejection fraction. This disclosed apical akinesis, anterior and septal wall motion abnormalities, and a left ventricular ejection fraction of 47%, with normal chamber sizes, normal valvular function, and no pericardial effusion (Movie I in the online-only Data Supplement).

Dr Scott: The bedside echocardiogram was valuable in this situation to identify mild left ventricular systolic dysfunction and regional wall motion abnormalities. These findings are more consistent with ACS than a peripartum or stress.
cardiomyopathy. Though the patient’s presentation mandates cardiac catheterization regardless of echocardiographic results, the finding of regional wall motion abnormalities is a valuable diagnostic clue.

The differential diagnosis includes ACS from plaque rupture, coronary arterial embolism (invoking the possibility of a patent foramen ovale or right to left shunt), coronary vasospasm, microvascular ischemia, and spontaneous coronary artery dissection (SCAD). The next step is to evaluate the coronary arteries. It was elected to proceed to coronary angiography. This patient was planning on breastfeeding, and the use of contrast was felt to be safe by the obstetrics team.

Cardiac catheterization via the right radial artery disclosed a normal left main coronary artery and a normal, dominant left circumflex artery with 2 obtuse marginal branches. However, the left anterior descending (LAD) artery was totally occluded in its mid-third with distal reconstitution from left-to-left collaterals (Figure 3, Movie II in the online-only Data Supplement). Before additional diagnostic or therapeutic maneuvers could be undertaken, the case was complicated by severe spasm of the right radial artery, which precluded wire exchange and right coronary angiography.

**Dr Scott:** At this point, it is clear that the patient’s symptoms are secondary to an acute coronary event involving the LAD artery. This vessel is occluded, and the cause is still not clear. The lack of visualized plaque in the other vessels argues against plaque rupture attributable to atherosclerosis. Intravascular ultrasound (IVUS) or optical coherence tomography (OCT) may have been helpful in determining the cause of the LAD occlusion, but this initial procedure was limited by vasospasm of the radial artery.

The second troponin T level was 1.96. At this time the patient was pain-free and no longer in decompensated heart failure. It was therefore decided to defer the transition to femoral access and instead manage the patient medically and review options for further care. A trial of conservative management—consisting of intravenous unfractionated heparin, aspirin, metoprolol, and low-dose nitrates and verapamil for vasospasm—was undertaken. Over the next 24 hours, troponin levels declined. However, the patient began to experience recurrent chest pain. Repeat coronary angiography was therefore performed via the right femoral artery. Though a nondominant right coronary artery was confirmed to be normal, significant arterial spasm was noted on engagement of the right coronary ostium. Left coronary angiography confirmed the previous findings of an occluded LAD artery. IVUS demonstrated extensive intramural hematoma throughout the length of the LAD consistent with coronary artery dissection. 2.5×33 and 3.0×12-mm overlapping Xience stents were deployed (Movie III in the online-only Data Supplement). After stent deployment, intracoronary OCT was performed to evaluate stent placement and expansion. OCT revealed intimal disruption proximal to the origin of the first diagonal branch as well as proximal to the first stent, with evidence of dissection and intramural hematoma extending into the stented segment (Figure 4, Movie IV in the online-only Data Supplement). Given TIMI-3 flow in the LAD, additional proximal stenting was not undertaken at this time, although the possibility of a relook angiogram in 6 to 10 weeks to confirm stent expansion after hematoma reabsorption was discussed.

**Dr Scott:** Based on the angiographic and OCT findings, the unifying diagnosis is pregnancy-associated SCAD with mild left ventricular systolic dysfunction. The increased blood volume associated with pregnancy and the autotransfusion of uteroplacental blood at time of delivery contributed to the development of pulmonary edema.

Spontaneous coronary dissections occur predominantly in young women and are associated with pregnancy as a result of progesterone-mediated changes in the vasculature and

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**Figure 3.** Cardiac catheterization demonstrated total midvessel occlusion of the left anterior descending artery (arrow).

**Figure 4.** Optical coherence tomography demonstrated an intimal disruption (arrow) and intramural hematoma (arrowhead) extending from the proximal left anterior descending artery into the stented area.
The dissection flap may be missed by coronary angiography. Hence, this case illustrates the advantages of IVUS and OCT in confirming the diagnosis. Vessel manipulation can propagate the dissection flap. Therefore, it is often advisable to minimize percutaneous interventions. It was appropriate in this situation to stop intervening once TIMI-3 flow was restored.

In the absence of randomized data or clear guidelines, medical therapy for SCAD is empirical. Because the pathophysiology of this disorder does not involve acute plaque rupture with thrombus formation, the role of thienopyridines and IIb/IIIa inhibitors is less defined than in traditional ACS. Given the increased bleeding risk in a patient in the immediate postoperative period, we did not feel that these agents were indicated before stenting.

The patient did well and was discharged home on aspirin, clopidogrel, metoprolol succinate, and sustained release verapamil for presumed superimposed vasospasm attributable to vessel injury. She enrolled in cardiac rehabilitation and underwent an exercise stress test, which was notable for a functional capacity of 11 metabolic equivalents without angina. She was advised that additional pregnancies would pose a significant risk, and her partner underwent vasectomy. No further events have occurred. Her baby was discharged from neonatal intensive care unit and ultimately home, and has also done well.

Dr Scott: The data for optimal treatment for spontaneous coronary dissection are limited, and treatment is therefore empirical. This patient received drug-eluting stents, and was therefore appropriately prescribed aspirin and clopidogrel. Metoprolol was used because of the presence of left ventricular wall motion abnormalities and to reduce wall shear stress. Verapamil was used for treatment of vasospasm that was felt to be secondary to vessel injury and pregnancy and will likely not be required long-term. The benefit of HMG-CoA reductase inhibitors (statins) in this setting is much less clear. This patient does not have an ACS from the typical plaque rupture but does have acute vessel injury. Statins are considered category X during pregnancy because of the risk of congenital anomalies and may be unsafe while breastfeeding.1 This patient did elect to breastfeed her premature neonate, and statins were therefore withheld after evaluation of the risks and benefits of therapy.

Discussion

Our patient initially presented to the attention of the cardiology service with symptoms of congestive heart failure, primarily dyspnea, hypoxia, and pulmonary infiltrates. The differential diagnosis of acute heart failure during pregnancy in women without preexisting cardiac conditions includes the following: peripartum cardiomyopathy and ACS, which may be consequent to plaque rupture and thrombosis; coronary artery vasospasm; or SCAD. In this case, the patient’s symptoms proved to be the result of the latter.

Though rare, with an estimated incidence of 3 to 6 episodes per 100000 pregnancies,12-14 ACS during pregnancy is a potentially devastating event. In addition to traditional risk factors, such as hypertension, dyslipidemia, age, and family history, physiological changes of pregnancy may contribute to the development of ACS and SCAD. As reviewed by Appleby et al,11 postulated contributory mechanisms include elevated cardiac output, increasing concentrations of procoagulant factors, changes in the hormonal milieu, and perturbations in vascular collagen content.

The diagnosis and management of ACS in pregnancy is particularly challenging because of concerns regarding in utero radiation and medication exposure. Though our patient had already delivered at the time of diagnosis, mitigating concerns of teratogenicity from medications and of fetal irradiation during diagnostic testing, care had to be taken with regards to medication use in the context of a breastfeeding mother.

The use of anticoagulant and antiplatelet medications, including aspirin, heparin, and low-molecular-weight heparins, was reviewed by the American College of Chest Physicians and found to be safe; warfarin, however, is a known teratogen and should be avoided.15 Of note, thrombolytics are not contraindicated in pregnancy but are relatively contraindicated in SCAD.11 The safety of thienopyridine and other GPIIb/IIIa inhibitors is uncertain. With regard to adjunctive therapies, angiotensin-converting enzyme–inhibitors and angiotensin receptor blockers are known teratogens but are thought to be compatible with breastfeeding, particularly benazapril, captopril, and enalapril.1 β-blockers, particularly atenolol, have been associated with intrauterine growth retardation but are felt to be safe during breast feeding.1 The safety of calcium channel blockers and nitrates during pregnancy is uncertain, but these agents are also not contraindicated during breastfeeding.1 In contrast, diltiazem has possible teratogenic effects. Statins are considered category X during pregnancy because of the risk of congenital anomalies and may also be unsafe while breastfeeding, and should therefore be used judiciously.1

SCAD causes ischemia when a plane of dissection or intramural hematoma within the tunica media impinges on the true arterial lumen. Among pregnant women with ACS, the pathogenic lesion is SCAD in an estimated 27% of cases.12 Even aside from pregnancy, SCAD affects predominantly young women, with 1 recent study reporting a mean age of 42.6 years and 82% female predominance.16 In addition to postpartum status, reported associations with SCAD include cocaine use, extreme physical exertion, Ehlers-Danlos syndrome, fibrillin gene variants, fibromuscular dysplasia, hormonal therapy, vasculitis, and polycystic kidney disease.16 The presentation of SCAD can be variable, but in the largest published series of patients with SCAD 49% of cases presented with ST-elevation myocardial infarction (most often involving the LAD territory), and 23% had multi-vessel involvement. Though ACS during pregnancy has been associated with a case fatality rate of 5.1%,14 early series reported 38% mortality with SCAD.17 However, more recent data suggest that a more favorable 99% of patients survive to discharge,16 albeit with an estimated 10-year mortality of 7.7%, recurrence rate of 21%, and major adverse event rate of 47%.16

Various treatment strategies for SCAD have been documented, including conservative management, percutaneous coronary intervention (PCI) with stenting, and CABG. Though evidence is not sufficient to uniformly recommend one strategy over others, recent case series have helped to
clarify management based on individual patient characteristics. Though SCAD can present with complete vessel occlusion, as in this case, PCI may propagate dissection and intramural hematoma and must therefore be undertaken with caution. Indeed, in the largest published series of patients with SCAD, PCI was associated with a higher rate of complications than conservative management or CABG.16 Hence, it may be reasonable to reserve PCI for cases in which ischemia persists with conservative therapy. Encouraging outcomes with CABG have been reported in cases involving the left main coronary artery or multiple vessels.11,16,18 In our patient, ongoing chest pain made conservative management an unattractive option, whereas single-vessel involvement argued against CABG.

The diagnosis of SCAD can be challenging, as the false lumen or dissection flap are often not visualized at coronary angiography. Hence, there is an emerging role for intravascular imaging technologies such as IVUS and OCT in the diagnosis and treatment of SCAD.

IVUS uses a catheter with a miniaturized ultrasound probe to interrogate the structure and composition of the coronary arteries. In addition to established uses in assessment of intracoronary plaque and during PCI, IVUS is capable of detecting angiographically silent SCAD.19–21 In this context, IVUS is useful for identifying intramural hematoma and intimal tears, identifying the true lumen and guiding correct wire placement in the event of PCI, and guiding stenting.

OCT is an emerging technology which, deployed in a coronary catheter, uses interferometry in the near-infrared spectrum to acquire high-resolution intracoronary images. Case reports and series have described the utility of OCT in confirming and assessing the extent of SCAD.22–24 The most extensive of these series reported the results of OCT for confirming the presence of a double lumen or intramural hematoma in 11 patients; of these, only 3 patients had an angiographically visible intimal flap. Thus, OCT was diagnostic in 8 of 11 (73%) cases.24 Furthermore, OCT ruled out SCAD as a diagnostic consideration in 6 cases. Hence, OCT may play a useful role when SCAD is included in the differential diagnosis. As with IVUS, OCT can also help to guide PCI by ensuring that the true lumen is engaged during wiring. However, aside from case reports,17 comparison of OCT and IVUS for evaluation of SCAD has not been undertaken.

This patient was counseled against future pregnancies because of the high risk of recurrent dissection. The modified World Health Organization (WHO) classification risk stratifies cardiovascular conditions during pregnancy (Table).25 Pregnancy is contraindicated in WHO 4 conditions because of high maternal mortality or severe morbidity. Though SCAD is not formally included in this classification, it is likely that a history of SCAD significantly increases the risk for future pregnancies. Contraceptive planning is therefore indicated. In this case, the patient’s husband underwent vasectomy.

Though the relative rarity of SCAD prohibits systematic, prospective evaluation of treatment strategies, the recent development of comprehensive registries with long-term follow-up has started to unravel the epidemiology, prognosis, and optimal approach for this disorder. Furthermore, technologies such as IVUS and OCT offer additional methods for investigating the pathophysiology of SCAD and for improving diagnostic accuracy in suspected cases. In the coming years, we can therefore look forward to greatly improved understanding and treatment of this challenging clinical scenario.

Disclosures

None.

References


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