Post-partum Spontaneous Coronary Artery Dissection: A Case Report

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Abstract

Spontaneous coronary artery dissection (SCAD) is a rare illness often misdiagnosed, that accounts for up to 4% of cases of acute coronary syndrome in young women. Interestingly, SCAD is the most common cause of myocardial infarction related to pregnancy. Here, we present a case of a 35-year old postpartum patient who presented in cardiac arrest due to ventricular fibrillation and was found to have a ST-segment elevation myocardial infarction. Cardiac catheterization revealed significant coronary vasospasm and dissection of the right circumflex and distal left anterior descending artery. We discuss clinical diagnosis and management of spontaneous coronary dissection along with literature review.

Keywords: spontaneous coronary artery dissection, post-partum, ventricular fibrillation

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1. Introduction

Coronary artery disease (CAD), due to atherosclerotic coronary disease, is associated with high morbidity and mortality rates [1]. Other reasons of coronary artery obstruction are emboli, spasm and dissection. Spontaneous coronary artery dissection (SCAD) is defined as dissection of the epicardial coronary vessels not linked to atherosclerosis, trauma or related to cardiac catheterization and is commonly related to connective tissue diseases [2]. SCAD has an incidence of less than 5%, however it is found to occur related to pregnancy and post-partum [3]. We present a case of young woman in the postpartum period who presented with cardiac arrest secondary to ventricular fibrillation. ST elevation myocardial infarction was identified and she underwent immediate cardiac catheterization which revealed dissection in the right circumflex and distal left anterior descending artery (LAD).

2. Case Presentation

A 35-year old African American female, with no significant past medical history, was brought to our institution in cardiac arrest. Approximately thirteen days prior, she had an uneventful spontaneous normal vaginal delivery. Five days prior to the presentation, the patient had experienced intermittent episodes of chest and upper epigastric pain which lasted 5-10 minutes and subsided by rest. On the day of presentation, the patient had chest pain. Blood pressure, was 165/120 and heart rate of 94 beats per minute. On the way to the hospital, the patient became unresponsive with agonal breathing and went pulseless. Cardiopulmonary resuscitation (CPR) was initiated. Cardiac monitor from EMS revealed ventricular fibrillation, and therefore, unsynchronized cardioversion was given with immediate return of spontaneous circulation (ROSC).

In the emergency department, patient was intubated to protect the airway. EKG showed ST-segment elevation myocardial infarction (STEMI) in leads II, III, aVF (Figure 1). Patient was transferred immediately for cardiac catheterization which revealed significant coronary vasospasm and dissection in the right circumflex and distal left anterior descending artery (LAD) (Image 1, Image 2). Percutaneous coronary intervention was not performed due to the presence of extensive dissection and TIMI 3 flow was found in all major epicardial vessels. Her estimated ejection fraction by left ventriculography was 33% and the left ventricle end-diastolic pressure (LVEDP) was 33 mmHg.

2-D echocardiogram post-catheterization revealed inferolateral wall hypokinesia. Cardiac biomarkers were elevated with troponin I of 6.71 ng/L and brain natriuretic peptide of 339 pg/mL. She was started on aspirin,
clopidogrel, nitroglycerin drip for vasospasm, enalapril and eptifibatide. She was successfully extubated by day 1.

A computerized tomography coronary angiography was ordered to better visualize the coronary dissection. Imaging revealed normal coronary arteries with a calcium score of zero.

Five days after admission, patient was discharged on aspirin, plavix, metoprolol succinate, spironolactone and enalapril. Given the history of ventricular fibrillation, cardiac arrest and low ejection fraction as per echocardiography and cardiac catheterization, the patient was fitted for a LifeVest. Patient was advised not to continue breastfeeding.

Repeat transthoracic echocardiography, two months after discharge, revealed a reduced ejection fraction of approximately 30% and inferoposterior, inferoapical, inferoseptal and posterolateral akinesis. The ventricle was mildly dilated with grade 2 diastolic dysfunction. Follow-up visits have focused on optimizing her medications, with plans to repeat the 2-D echocardiogram in 3 months to evaluate improvement in the ejection fraction.

![Figure 1. EKG showing STEMI in leads II, III, aVF](image1.png)

**Image 1.** Severe narrowing with likely vasospasm in distal LAD, severe narrowing and probable dissection in distal LCX and 1st obtuse marginal
3. Discussion

CAD is associated with some of the highest morbidity and mortality rates in the United States. One-third of adults above the age of 35 die due to coronary artery disease [1]. Usually, coronary arteries are mostly obstructed due to atherosclerosis. Other reasons of obstruction include emboli, spasm and dissection.

Coronary dissection is either caused by trauma, iatrogenia or spontaneous. Spontaneous coronary artery dissection (SCAD) is defined as dissection of the epicardial coronary vessels not linked to atherosclerosis, trauma or related to cardiac catheterization [2]. The coronary arteries are built of 3 layers from inward: tunica intima, tunica media and tunica adventitia. The blood supply of the coronary arteries comes from the vasa vasorum, small vessels within the wall of coronary arteries. Coronary dissection happens if either the vasa vasorum vessels bleed within the coronary artery or with tunica intima laceration leading to the formation of a false lumen and ultimately, coronary obstruction [3].

Spontaneous coronary artery dissection (SCAD) is fairly underestimated due to misdiagnosis. It was reported to be accountable for up to 4% of acute coronary syndrome (ACS), especially in young adults, predominantly in women. SCAD typically can occur in all coronary territory, however can be most commonly seen in the left anterior descending artery [4]. It is usually presented as a case of acute coronary syndrome in younger populations.

Multiple conditions were reported to cause SCAD. These include fibromuscular dysplasia, connective tissue diseases, inflammatory diseases (i.e. vasculitis), and coronary artery vasospasm. It can be precipitated by stressful events, such as labor and delivery, strenuous exercise or emotion, or recreational drugs [5].

Spontaneous coronary artery dissection (SCAD) is the most common cause of myocardial infarction related to pregnancy. However, pregnancy related SCAD is only less than 5% of all reported SCAD [3]. This includes SCAD cases presented during pregnancy and during lactation. The association between SCAD and pregnancy is not well-understood. It was postulated that it is related to hormonal changes during pregnancy and the changes in the connective tissue and arterial walls related to it [6].

Early identification of SCAD is crucial. The management of SCAD differs from atherosclerosis related ACS. It should be suspected in women of childbearing years, particularly if they have a history of connective tissue disease, vasculitis or pregnancy. Once suspected, coronary angiography should be considered. By angiography, SCAD is categorized into 3 types using by the Saw classification: Type 1 is characterized by multiple radiolucent lumens, Type 2 (the most common type) is described as intramural hematoma and diffuse stenosis, and Type 3 mimics atherosclerosis [7].

The management of SCAD depends of multiple factors such as the site and extension of the lesion, number of vessels affected, level of expertise of the interventional cardiologist, availability of service and hemodynamic status. Clinically stable patients with low risk coronary lesions are advised to have a conservative approach with clinical monitoring. When multiple coronary vessels or the left main coronary artery are involved, coronary artery bypass grafting (CABG) is recommended. However, hemodynamically unstable patients should be sent for urgent CABG or percutaneous coronary angiography (PCI) according to the availability [2].
Patients with SCAD tend to have favorable outcomes in regards to prognosis, especially if diagnosed and managed properly. Unfortunately, SCAD can recur with a frequency of 27% within five years from the first event. Surprisingly, the recurrent lesions are usually reported in previously healthy vessels, which implies that SCAD is a disease that can affect all coronary vessels [8].

In our case, the patient was a young postpartum woman with no significant cardiovascular risk factors. Her chief complaint was chest pain for five days and she subsequently developed ventricular fibrillation, which reverted by unsynchronized cardioversion and achieved ROSC. Her EKG showed STEMI and she went for urgent cardiac catheterization which revealed diffuse coronary spasm and dissection. A decision was made to pursue conservative management giving the pathophysiology of her condition.

Once the diagnosis of SCDA has been confirmed by angiography, the management depends on the patient’s hemodynamic stability and extension of the coronary dissection. Our patient became hemodynamically stable after successful resuscitation and the extension of the dissection was limited to the right circumflex and distal left anterior descending arteries. Therefore, the decision was made to provide medical management and to schedule her for regular follow up to assess the progression or stabilization of coronary dissection.

4. Conclusion

SCAD is a poorly understood, underdiagnosed subtype of ACS. However, it has an excellent prognosis if managed appropriately. It is common in young women with history of recent pregnancy or connective tissue disease with a recent stressor. Early suspicion, recognition and management are important to achieve a successful outcome.

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