Case Report

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Postpartum Spontaneous Coronary Artery Dissection in a Young Woman: A Case Report

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ABSTRACT
Spontaneous coronary artery dissection (SCAD) is a rare but potentially life-threatening condition, particularly in the postpartum period. It is the potential cause of acute coronary syndrome in the population. We report a 32-year-old woman who presented with chest pain, diaphoresis, and shortness of breath 12 hours after an uncomplicated vaginal delivery. ECG demonstrated anterior ST-segment elevation, and coronary angiography revealed a mid-to-distal LAD dissection extending into the first diagonal branch. She was managed conservatively with medical therapy and remained clinically stable. This case highlights the importance of considering SCAD in postpartum patients with chest pain, as its management differs significantly from typical myocardial infarction.

Keywords: Spontaneous Coronary Artery Dissection, Postpartum, Acute Coronary Syndrome

Introduction

Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome (ACS), occurring in about 0.1% to 4% of all patients with ACS [1]. It is characterized by the separation of the coronary artery wall layers, which leads to impaired blood flow [2]. Although, SCAD is rare in the general population, it is reported to account for approximately 24% to 35% of pregnancy-related myocardial infarctions in women less than 50 years of age [1]. SCAD during pregnancy typically occurs in the third trimester and up to 6 weeks postpartum [1, 3-4].

The exact etiology of SCAD during pregnancy is poorly understood. Studies have shown this is be due to hormonal and hemodynamic changes that weaken coronary artery walls, increasing the risk of dissection [1, 4-5]. Consequences can include myocardial infarction, heart failure, cardiogenic shock, or death [6]. Some studies suggest that estrogen can have cardioprotective effects [6]. However, the concomitant increase in progesterone weakens arterial walls by disrupting normal corrugation, leading to a loss of structural support due to changes in elastic fibers and medial wall collagen [6].

Pregnant women with SCAD do not have typical risk factors that would raise suspicion for coronary artery or cardiac disease [3]. The EM physician should therefore be increasingly cognizant of this possibility in this specific population with chest pain.

Case Presentation

A 32-year-old woman, G2P2002 discharged 12 hours earlier from the hospital after an uncomplicated spontaneous vaginal delivery and uneventful hospital stay, presented to the emergency department (ED) complaining of chest pain radiating to her left arm, diaphoresis, and shortness of breath. She stated that her symptoms began 90 minutes prior to her arrival. She had no significant medical history except for mild gestational hypertension, which was well-controlled with lifestyle modifications.

On examination, the patient appeared to be in acute distress with a heart rate of 110 beats per minute, respiratory rate of 20 breaths per minute, blood pressure of 140/90 mmHg, and oxygen saturation of 96% on room air. She was alert and oriented, speaking in full sentences, and moving all her extremities freely and without difficulty. Pulmonary exam revealed clear breath sounds, no retractions, and normal work of breathing. Cardiovascular examination revealed normal heart

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sounds without murmurs, gallops or rubs, no jugular venous distention. Pulses were intact in all extremities. Abdomen was soft, nontender, and not distended.

An initial electrocardiogram (ECG) revealed ST-segment elevation in leads V2-V4, T-wave inversion in lead aVL, and no reciprocal changes [Figure 1]. Given the clinical presentation and ECG findings, an initial diagnosis of acute myocardial infarction (AMI) was suspected along with potential preeclampsia. Cardiology was emergently consulted, and the patient was immediately started on dual antiplatelet therapy with aspirin and clopidogrel and subsequently underwent emergent coronary angiography. Oral labetalol was administered for management indicated of both SCAD and pre-eclampsia, which was also a concern given the patient's initial blood pressure of 140/90 post-partum.

Coronary angiography revealed a focal, long-segment dissection involving the mid to distal left anterior descending (LAD) artery extending into the first diagonal branch [Figure 2]. The remainder of the coronary arteries appeared normal. Given the distal extent of the dissection and the risk of extending the dissection, conservative management with medical therapy was preferred over percutaneous coronary intervention (PCI). The patient was started on clopidogrel 75 mg, amlodipine 10 mg, aspirin 81mg, metoprolol 50 mg, and losartan 25 mg as home medications. Cardiac biomarkers were notable for a peak troponin I level of 0.35 ng/mL while in the ED (reference range: 0.00-0.30 ng/mL). Transthoracic echocardiography (TTE) post catheterization demonstrated preserved left ventricular function without regional wall motion abnormalities.

In the cardiac critical care unit (CCU) troponin-I values continued to downtrend and the patient remained hemodynamically stable. The obstetrics service was consulted and recommended systolic blood pressure goals less than 120 mmHg and rest of management as per cardiology. The patient was discharged on hospital day 4 to outpatient follow up with the cardiology and obstetrics services. Genetic testing for connective tissue disorders was planned as an outpatient. At the one-month cardiology follow-up visit, the patient remained asymptomatic without evidence of recurrent chest pain or adverse cardiovascular events. The patient was lost to follow up thereafter, prior to planned repeat angiogram or echocardiogram.

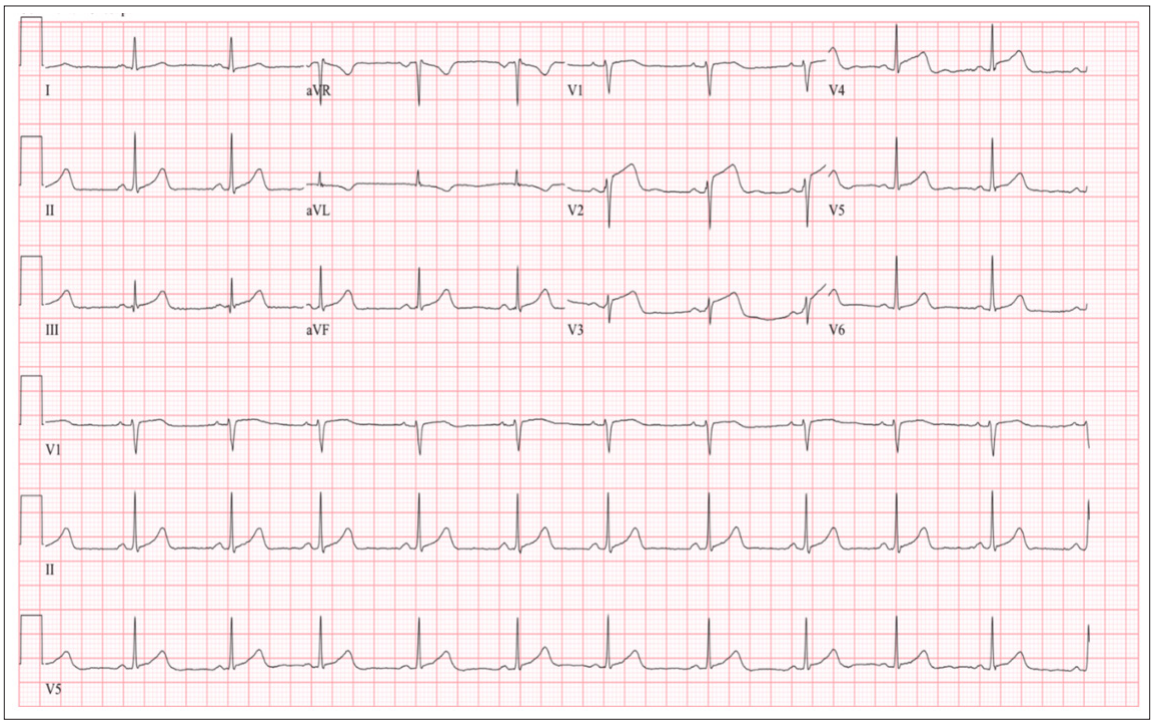

Figure 1: Initial ECG

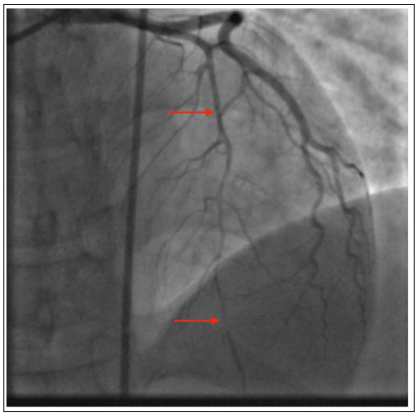

Figure 2: Cardiac Angiogram

Coronary angiography demonstrating a focal, long-segment dissection of the mid to distal left anterior descending artery extending into the first diagonal branch, indicated by the red arrow.

Discussion

Patients with SCAD typically present similarly to patients with ACS, with nonspecific symptoms including chest pain, shortness of breath, diaphoresis, and/or nausea [1,3-6]. However, treating SCAD as ACS may be detrimental as management differs significantly.

Diagnostic findings of SCAD include ECG changes, elevated troponins, regional wall motion abnormalities on ultrasound.

ECG findings and patterns can vary from normal to ST-segment elevations [3]. Although dissection can occur in any coronary artery, it is most common in the LAD or left circumflex artery (LCX), leading to precordial lead changes (V1-V6) and TWI in lead aVL [5,7].

Confirmation requires coronary angiography, which reveals the dissection flap or the presence of a false lumen [1,8]. In this case, coronary angiography revealed a dissection in the left anterior descending artery, consistent with SCAD, along with elevated troponins and ST-segment changes in V1-V4 and TWI in lead aVL.

The management of SCAD depends on the severity of the disease and may include conservative medical management, PCI with or without stents, coronary artery bypass graft surgery, or potentially transplantation [2]. Thrombolytic therapy, recommended in ACS or MI management, is not recommended in SCAD as it may worsen coronary dissection and increase the risk of coronary vasospasm [2]. Similarly, heparin and glycoprotein IIb/IIIa inhibitors are contraindicated as they may increase the size of the hematoma [4]. Additionally, controlling blood pressure and heart rate is critical to limit the shearing force on the affected vessels [2,4,5,8,9]. As in this case, conservative medical management is an option, which resulted in successful resolution of the patient's symptoms on follow-up.

The rarity and variation in management of SCAD compared to ACS or typical myocardial infarction makes recognition of this pathology in this population valuable for the emergency physician. Making the diagnosis of postpartum spontaneous coronary artery dissection challenging. Therefore, it is important to highlight the distinction, as treating this condition as typical ACS or myocardial infarction can lead to increased harm. Should SCAD be suspected, patient care can be optimized by starting a conversation early with cardiologists.

Conclusion

SCAD should be considered in the differential diagnosis of chest pain in young women, particularly during the peripartum and postpartum periods. Prompt recognition and appropriate management are essential for optimizing outcomes in these patients. Although SCAD represents a rare cause of ACS, maintaining a high index of suspicion and a low threshold for investigation is imperative in pregnant or postpartum females with no apparent risk factors who present with chest pain.

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