Case Report

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# Myocardial Infarction in a Postpartum Female: Integral Role of Multimodality Imaging

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## Introduction

Spontaneous coronary artery dissection (SCAD) is a rare antecedent to myocardial ischemia and subsequent myocardial infarction in the general population [1]. SCAD has increasingly been recognized as a cause of acute coronary syndrome in young women, with a prevalence as high as 25% of those <50 years old who present with myocardial infarction (MI) [2]. Patients without risk factors for coronary artery disease more often present with non-atherosclerotic SCAD (NA-SCAD), which has a predilection for young women, and can occur during pregnancy [3]. Plain coronary angiography may not reveal the dissection plane, often necessitating the use of other intravascular technologies or non-invasive imaging to ascertain the diagnosis. We report a case of suspected spontaneous coronary artery dissection in a postpartum patient which was not appreciated on traditional 2-dimensional coronary angiography. Subsequent confirmation of myocardial infarction occurred with the use of cardiac resonance imaging (CMR).

## Case Presentation

A 38 years old female with previous medical history notable only for anemia and anxiety presented to the emergency room 6days post-partum with complaints of new cough, orthopnea, dyspnea and lower extremity swelling. She denied chest pain. She reported an unremarkable pre-partum course all the way into delivery. Initial ECG in the ER revealed sinus tachycardia with Q waves in V3 and V4 with T wave inversions seen throughout the precordial leads. Computed tomography scan of the chest with contrast was performed which was negative for pulmonary embolism but showed findings consistent with congestive heart failure. Basic natriuretic peptide was elevated at 754 pg/ml and initial troponin I was elevated 19.70 pg/ml (normal troponin cut off <0.04). She denied chest pain, endorsing chest pressure, orthopnea and paroxysmal nocturnal dyspnea. Vital signs were stable. Shortness of breath improved with the use of intravenous diuretics and she was admitted to telemetry for further management of congestive heart failure and work-up for elevated troponin.

On arrival to our facility she underwent transthoracic echocardiogram which showed preserved right and left ventricular function with evidence of a new wall motion abnormality extending from the mid-distal anteroseptal wall of the left ventricle. She was subsequently started on medical therapy for non-ST elevated myocardial infarction (NSTEMI) with aspirin, heparin and clopidogrel. Second troponin I returned at 13.64 pg/ml and then increased to 17.11 pg/ml. Left heart catheterization revealed a left ventricular end diastolic pressure of 38 mmHg. Coronary angiography revealed a subtle defect involving the mid portion of the LAD **(Figure 1)**, but was not felt to be convincing evidence for significant atherosclerosis or arterial dissection.

While in cardiac rehab she underwent cardiopulmonary stress testing which revealed ST depressions. She subsequently underwent repeat coronary angiography (3 months' post infarction) with optical coherence tomography and coronary flow reserve-all of which were normal.

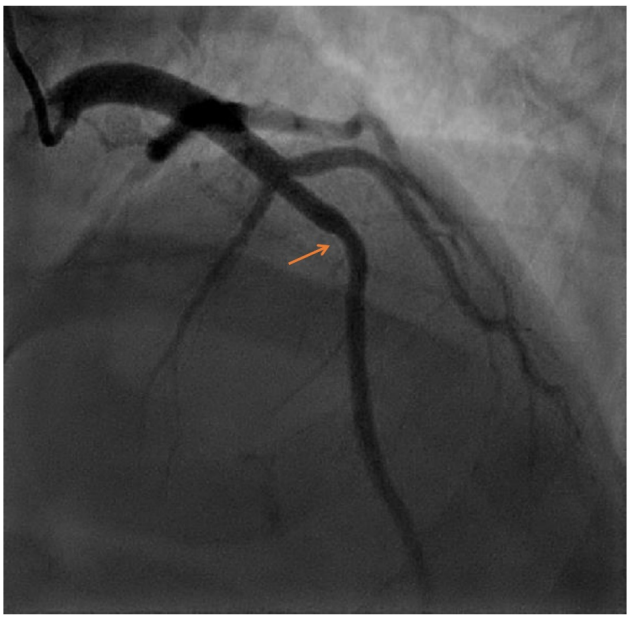

**Figure 1** Coronary angiography portraying subtle lesion (arrow) involving the mid left anterior descending artery taken in the LAO-Cranial projection

Intravascular ultrasound or optical coherence tomography were not performed. Subsequent cardiac magnetic resonance imaging (CMR) was done due to high suspicion of spontaneous coronary artery dissection revealing T2 signal abnormality, regional mid-distal anteroseptal wall hypokinesis, and delayed myocardial enhancement involving the mid to distal anterior, septal and inferior walls extending to the left ventricular apex, consistent with a mid to distal left anterior descending artery (LAD) infarction **(Figure 2)**. The patient was diuresed until she reached a euvolemic state and she was discharged to home on metoprolol, hydrochlorothiazide and aspirin. She elected to forego breastfeeding while on medical therapy.

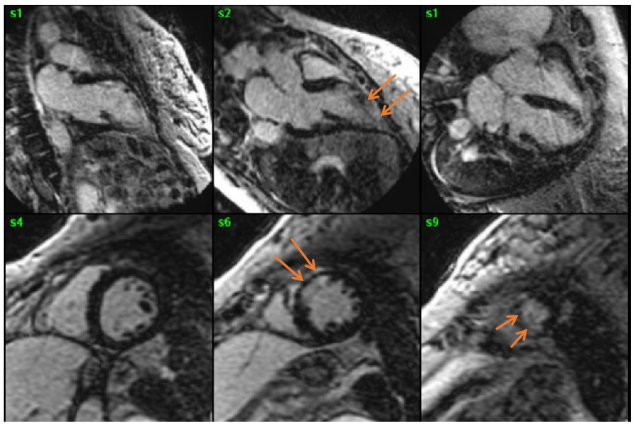


**Figure 2** Cardiac MRI revealing late gadolinium enhancement of the mid to distal anteroseptal wall (arrows). Still frames from horizontal long axis (top row) and short axis (bottom row) are consistent with mid to distal LAD infarction

## Discussion

Spontaneous coronary artery dissection is thought to result from an intimal tear or bleeding of the vasa vasorum with intramedial hemorrhage and typically affects young women without coronary risk factors [4]. In the peripartum period, the pathophysiology of SCAD can be considered two-fold: the increase in hemodynamic stress which is physiologic during pregnancy and delivery, coupled with the fragility of the coronary vessel walls secondary to hormonally mediated changes [5]. Diagnosis is often made at the time of coronary angiography which classically reveals a radiolucent intimal dissection flap and/or contrast staining presenting in one of three different phenotypic types [2]. Not uncommonly, a dissection plane is left unidentified; instead the lesion appears as luminal compression with a smooth appearance (type 2) and can vary from mild to total occlusion [6]. This phenotype is the most common in patients with NA-SCAD; lesions are often inconspicuous, thereby leading to under-recognition of SCAD in these patients [2]. Our patient had a subtle lesion involving the mid LAD which was of unclear significance initially. Subsequent performance of CMR then revealed mid to distal LAD infarction. Clinical presentation and angiography were not suggestive of vasospasm, embolic phenomena, microvascular disorders or other causes which comprise the syndrome of myocardial infarction with non-obstructive coronary arteries (MINOCA). It is believed, though not confirmed, that our patient presented with SCAD with a poorly visualized dissection lesion on initial angiography. This lesion likely healed, as suggested by a normal coronary lumen appearance on follow-up optical coherence tomography. Multimodal imaging in the form of CMR solidified the presence of MI with the cause most likely SCAD.

## Conclusion

Patients presenting with SCAD may have a normal or near normal coronary angiogram. Multimodal imaging is useful in obtaining a diagnosis of myocardial infarction in patients who present with normal or non-obstructive coronary artery disease.

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