Spontaneous coronary artery dissection presenting with chest pain: a case report.

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Introduction

Spontaneous Coronary Artery Dissection (SCAD) is a nonatherosclerotic, nontraumatic cause of acute coronary syndrome. SCAD has been considered a rare cause of Acute Myocardial Infarction (AMI), accounting for 1-4% of presentations [1], but is the cause of 24-30% of AMI in women younger than 50 years [2] as well as 15-20% of AMI during pregnancy [3]. Clinically, SCAD presents like atherosclerotic ACS, but diagnosis can be challenging due to a lack of typical risk factors. Management also differs from atherosclerotic ACS; thus, it is important to keep a strong index of suspicion for timely accurate diagnosis and management. Treatment options depend on hemodynamic stability and the location of the dissection. More distal lesions are treated more conservatively, and proximal lesions are treated with Percutaneous Coronary Intervention (PCI) or Coronary Artery Bypass Graft (CABG). Although SCAD is uncommon, it makes up a significant number of AMI cases in women although remains under-recognized and underreported. We present a common SCAD scenario with our patients' clinical course underlining the uncertainties and challenges with optimal management.

Case Presentation

A 50-year-old Hispanic woman presented to the Emergency Department (ED) with a sudden onset of chest pain occurring while walking up the stairs carrying heavy objects at work. She described the sensation as starting as a large amount of gas rising in her chest followed by extreme pressure and pain radiating to bilateral arms. The entire episode lasted for about 20 minutes and was alleviated after receiving Nitroglycerin spray, aspirin, and ticagrelor (Brilinta) per EMS while en route to the Emergency Department. She denied any similar previous episodes. Her past medical history includes hypertension, active tobacco use, and a stable liver hemangioma. She reported smoking about 4 cigarettes daily for the past 30 years. She denied any history or family history of heart disease. The patient was not taking any medications and reported no known drug allergies.

In the Emergency Department, her blood pressure was 140/85 mm Hg, and her other vitals were normal. The physical exam was unremarkable. Labs were only significant for initial troponin elevated to 0.111 ng/ml. CXR was unremarkable.

Initial EKG showed sinus bradycardia with 1st-degree AV block with non-specific, non-ischemic T wave changes. A transthoracic echocardiogram performed showed an LVEF of 61%, bi-atrial enlargement, moderate tricuspid and aortic regurgitation, aortic root/ascending aortic dilation (4.3 mm) and a moderate size interatrial septal aneurysm. No regional wall motion abnormalities were seen.

She underwent a CT Angiography of chest which did not demonstrate aortic dilatation or dissection or any evidence of pulmonary embolism. The patient reported no symptoms of chest pain during the examination. She was started on aspirin, ticagrelor, and heparin drip and was transferred to a tertiary care facility for cardiac catheterization and coronary angiography.

At the tertiary care hospital, the patient remained hemodynamically stable and reported mild chest discomfort. Her high-sensitivity troponin increased and peaked at 2,153.56. The lipid panel revealed a total cholesterol of 218 mg/dL, LDL of 106 mg/dL, HDL of 102 mg/dl, and Hemoglobin A1c of 5.4. Cardiac catheterization revealed pre-intervention TIMI flow grade 3 (Type A lesion with pre-intervention TIMI-3.), 70% stenosis of the second right posterolateral branch with a dissection flap in the distal vessel concerning Spontaneous Coronary Artery Dissection (SCAD). The left main left anterior descending, and left circumflex vessels were angiographically normal. No further intervention was carried out.

The patient was started on aspirin 81 mg, clopidogrel (Plavix) 75 mg, Metoprolol succinate 12.5 mg, and Nitroglycerin PRN. During outpatient follow-up, the patient underwent CT Angiography of the Abdomen/Pelvis and Head/Neck, which did not show any evidence of fibromuscular dysplasia. Given an ASCVD score, she was not started on statin therapy. (Figure 1-4).

Second right posterolateral coronary artery with small dissection flap concerning for spontaneous coronary artery dissection.

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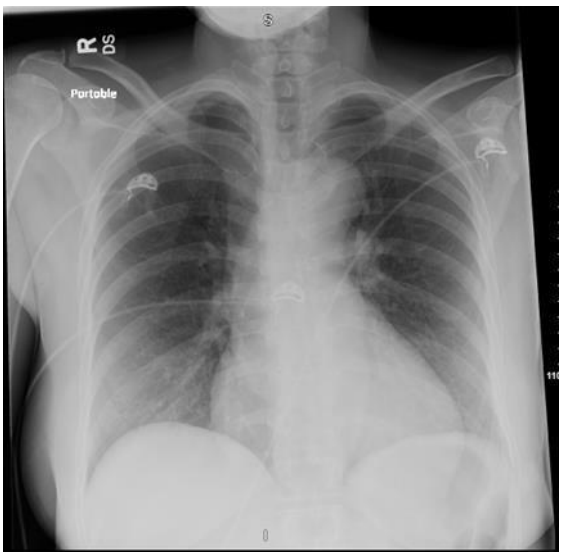

Figure 1. *Normal Chest X-ray.*

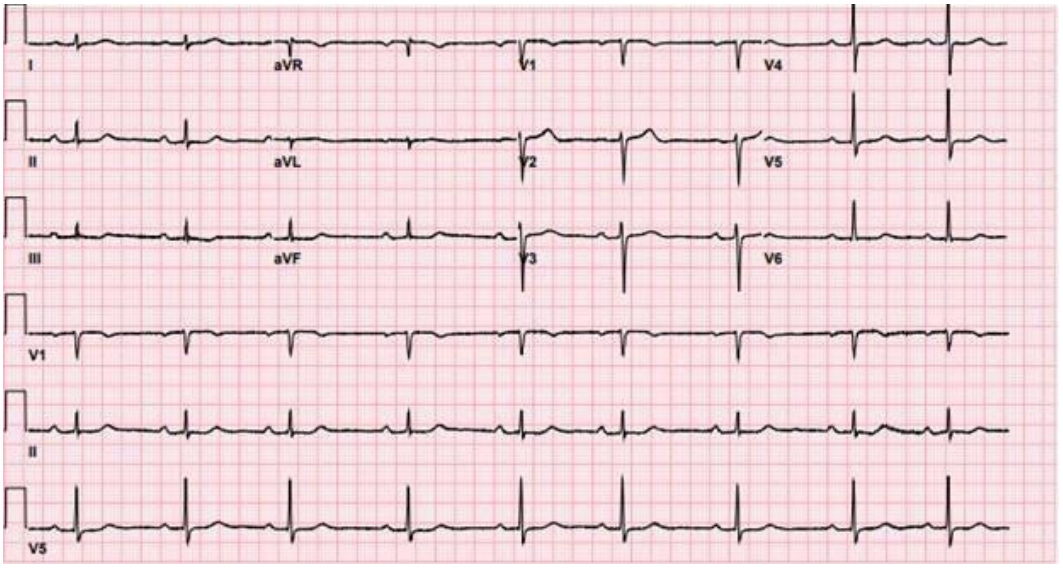

Figure 2. *Sinus Bradycardia with 1st Degree AV block.*

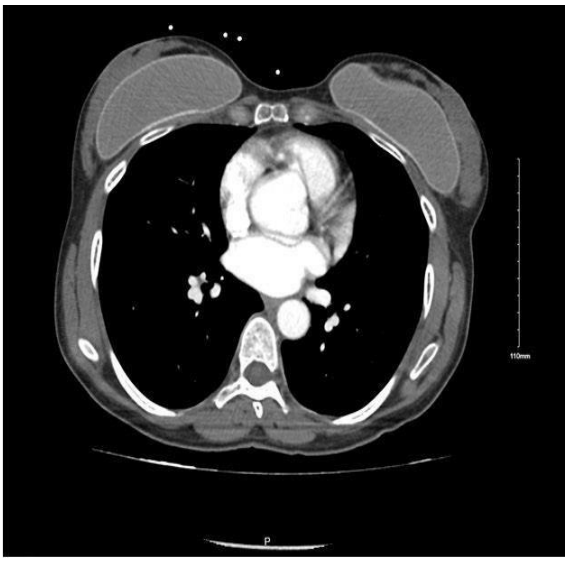

Figure 3. *CT chest Angiography.*

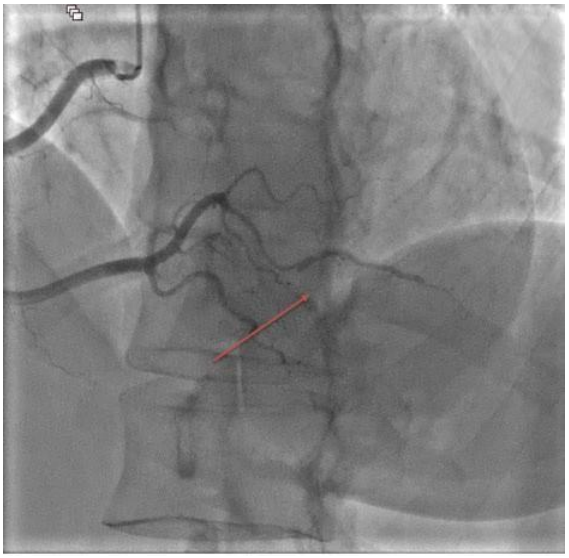

Figure 4. *Coronary catheterization imaging.*

Discussion

The pathophysiology of SCAD involves the development of hematoma within tunica media leading to intimal or intima-medial separation and compression of the true lumen, causing AMI. Two hypothesized mechanisms are . endothelial-intimal disruption causing blood from the true lumen to enter the subintimal space or 2. a de novo hematoma in the medial space due to microvascular disruption. [4]

The cause of SCAD remains unknown, but risk factors include pregnancy, hormone use, FibroMuscular Dysplasia (FMD), connective tissue, and inflammatory disorders, physical or emotional stress, the use of stimulant medications, illicit drugs, and genetic factors. [5] The incidence of SCAD is predominantly in women >90% of cases with a mean age of 44-53. This supports the hormone hypothesis of etiology although the magnitude and mechanism remain unknown. Our patient's risk factors were more typical for atherosclerotic ACS and included tobacco use and hypertension. She underwent CTA which ruled out FMD but did not undergo further workup like genetic testing for which indications are unclear.

Clinically, patients with SCAD most frequently present with chest pain or equivalent, elevated serum biomarkers, EKG findings consistent with (N)STEMI, or even ventricular arrhythmias, shock, and SCD. Our patient was presented with ongoing chest pain and positive biomarkers, prompting urgent left heart catheterization. She was found to have a 2nd Right posterolateral branch narrowing and a dissection flap in the distal vessel. Our patient was conservatively managed with DAPT.

There is currently no optimal treatment strategy for SCAD due to a lack of clinical trials comparing PCI with conservative management. Conservative strategy is generally preferred in hemodynamically stable patients without ongoing ischemia or left main involvement. [6] PCI in SCAD compared to atherosclerotic ACS has higher rates of complications including iatrogenic dissection, and hematoma propagation, furthermore, SCAD is known to have high rates of spontaneous angiographic healing. [7] Our patient was started on DAPT for 3 months plus metoprolol succinate and later continued treatment with Aspirin and metoprolol. Statins were not indicated due to low ASCVD score.

Medical therapy for SCAD also differs from atherosclerotic ACS. There are currently no randomized controlled trials regarding the choice of antiplatelets, and management differs unless the patient has undergone stenting. Aspirin use has been extrapolated to use in SCAD, but additional antiplatelet therapies are controversial. Anticoagulation studies are lacking as well and theoretically increase the risk of intramural bleeding and thus have no role in acute or chronic management of SCAD. [8]Saw et al reported clinical predictors for recurrence in 327 SCAD patients, showing a recurring rate of 10.4%. In their Cox regression multivariate analysis, Beta-blocker use was associated with decreased recurrence and hypertension with increased risk [9].

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