CLINICAL VIGNETTE

Spontaneous Coronary Artery Dissection (SCAD) in a 53-Year-Old Patient

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Case Presentation

A 53-year-old female without significant past medical history presented to the emergency department with epigastric pain radiating to the chest and shortness of breath. The patient stated that she was in her usual state of health until about 6 hours prior to presentation when she felt epigastric burning. She tried over the counter antacid medications which were not helpful in alleviating the pain. The pain worsened over the next several hours and became severe burning and heaviness in the epigastrium and over the left chest, 10/10 in severity and associated with diaphoresis and nausea and vomiting. A co-worker called 911 and she was brought in by ambulance to the Emergency Department.

Her medical history was without typical risk factors for coronary artery disease. She was a non-smoker, does not drink alcohol and does not use illicit drugs. She is multi-parous with six children, all of whom were vaginally delivered. She does not have a significant family history of coronary artery disease and she eats healthfully and exercises often. She recently lost her mother, which has been particularly stressful

In the Emergency Department, she had stable vital signs but appeared to be in distress with ongoing chest pain. Electrocardiogram showed biphasic T-waves in V2-V3 concerning for ischemia and echocardiogram showed mid to apical hypokinesis concerning of ischemia in LAD territory. Troponin was abnormal at 3.29 and she was taken for emergent coronary angiogram for further evaluation.

Emergent coronary angiography demonstrated acute diffuse tapering of the mid LAD without evidence of atherosclerosis in the LAD or other coronary arteries (Figure 1). There was no change in the LAD lumen with administration of nitroglycerin. Given morphologic appearance of the LAD in absence of coronary atherosclerosis, this was suspicious for spontaneous coronary artery dissection (SCAD). There was good flow in the LAD without further intracoronary imaging to avoid complication with potentially wiring of the false lumen. The patient was treated conservatively with medical therapy including aspirin, clopidogrel, metoprolol and statin therapy. CT coronary confirmed the presence of intramural hematoma in the LAD consistent with SCAD with absence of coronary atherosclerosis (CT calcium score of 0). She did well with medical therapy with resolution of her chest pain. She was able to ambulate without recurrence and was discharged on medical therapy with close follow up.

Discussion

Spontaneous coronary artery dissection (SCAD) is defined as a non-traumatic/non-iatrogenic separation of the coronary arterial wall resulting in dissection. It is an infrequent but important cause of acute chest pain and myocardial infarction. Timely identification and diagnosis is needed to prevent significant morbidity and mortality.[1]

SCAD is an important cause of myocardial infarction, especially in young women, accounting for approximately 0.2-1% of acute coronary syndrome cases.[1] The overall prevalence is thought to be due to underdiagnosis. It affects women in >90% of cases with a significant number of cases occurring in the postpartum period. SCAD can account for nearly a quarter of cases of ACS in women less than 50 years old.[1]

The underlying mechanism of non-atherosclerotic SCAD is thought to be secondary to an intimal tear or bleeding of the vaso vasorum with intramedial hemorrhage resulting in creation of a false lumen with intramural hematoma.[2,3] Expansion of the false lumen from an expanding intramural hematoma can encroach the arterial lumen leading to myocardial ischemia and infarction. Most patients presenting with SCAD do not have conventional risk factors for coronary artery disease. In women who are pregnant or early in the postpartum period, dissection may be secondary to increased physiological hemodynamic stresses or from hormonal effects weakening the coronary arterial wall.[4,5] Predisposing factors in patients presenting with SCAD include fibromuscular dysplasia (FMD), connective tissue disorders, postpartum status, multiparity (≥4 births), emotional stress and hormonal therapy.[1]

The diagnosis of SCAD is made usually at time of invasive coronary angiography. Angiographic findings that are suspicious for SCAD include the presence of a dissection plane with typical changes of radiolucent intimal flap and contrast staining in absence of coronary atherosclerosis. However, these "typical" changes are seen in <30% of SCAD cases.[6] The majority of SCAD cases reported in case series had long, diffuse luminal narrowing on coronary angiography due to intramural hematoma, which was frequently not diagnosed as SCAD.[6] Thus, it is key to maintain a high clinical suspicion in the right clinical context and pursue other diagnostic workup if needed. CT coronary angiography can confirm an intramural hematoma which can help further with diagnoses of SCAD. However, sensitivity of this modality is not high and SCAD cases can be missed. During invasive angiography, intravascu-

lar ultrasound (IVUS) or ocular coherence tomography (OCT) imaging can help to more definitively make the diagnosis of SCAD. These should be utilized with caution as wiring of the injured coronary artery can cause further propagation of the dissection plane.

In the majority of patients presenting with SCAD, the preferred strategy after diagnosis is conservative therapy given increased risk of significant complications with invasive intervention.[1,6] In patients who continue to have symptoms of ongoing ischemia presenting with acute myocardial infarction or hemodynamic instability, revascularization with PCI or coronary artery bypass grafting should be considered. However, revascularization in patients with SCAD is technically challenging and associated with higher failure rates or complications given the presence of dissection with a false lumen.[1,6] Medical therapy in patients presenting with SCAD includes long-term aspirin, short-term clopidogrel, beta blockers as well as statins in patients with dyslipidemia.[1,6]

Conclusion

While relatively uncommon, SCAD remains a very important cause of acute chest pain and myocardial infarction in patients without conventional coronary artery disease risk factors. It should be considered in any young patient, especially women, presenting with acute myocardial infarction as accurate and timely diagnosis is imperative to improve clinical outcomes.

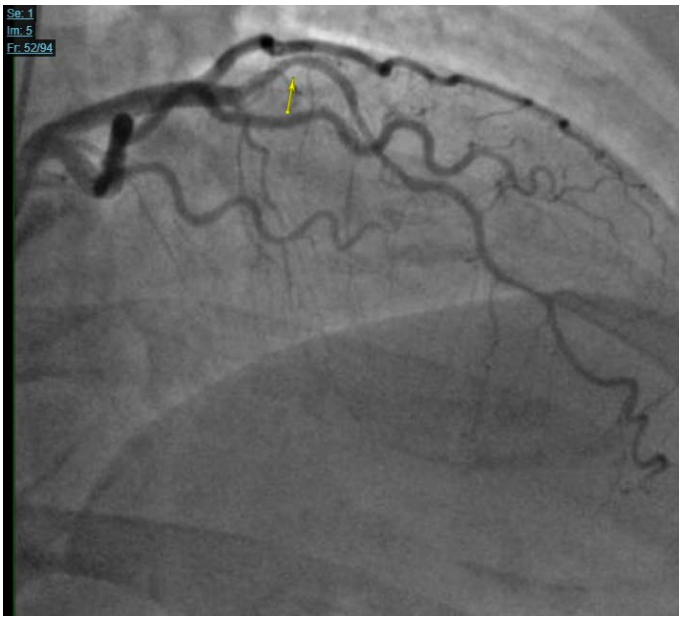

Figure 1: Invasive coronary angiography with yellow arrow point to sudden diffuse tapering of the mid to distal LAD suggestive of SCAD.

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