CLINICAL VIGNETTE

Using IVUS to Discriminate Between SCAD and Atherosclerotic Stenosis

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Case Report

A 35-year-old woman with hypertension and prediabetes presented to the emergency room with three weeks of substernal chest discomfort which radiated to her back and both hands. The chest pain initially occurred with moderate activity such as walking for 15 minutes. It worsened an in the days prior to presentation occurred making her bed or with simply walking a few steps. The chest pain resolved after 2 to 4 minutes of rest.

In the Emergency Department the patient's initial blood pressure was 153/107 mmHg with a heart rate of 92 beats per minute. She was afebrile with an oxygen saturation of 98% on room air. On physical exam she appeared comfortable with unremarkable cardiac and pulmonary findings. Her electrocardiogram (ECG) demonstrated normal sinus rhythm at 78 beats per minute with nonspecific T-wave abnormalities in leads V3-V6. Labs were notable for an initial mildly elevated high sensitivity troponin of 45ng/L followed by 58 ng/L (>12 ng/L considered elevated in females). The erythrocyte sedimentation rate and C-reactive protein were mildly elevated at 20 mm/hr and 1.8 mg/dL. Chest x-ray showed only mild peribronchial thickening. A CT angiogram of the chest showed no aortic aneurysm or dissection. Echocardiography demonstrated normal left ventricular size with normal wall motion and a preserved left ventricular ejection fraction. CBC and chemistries were normal. Coronary CT angiogram showed a 13 mm long high-grade (70-99%) stenosis of the proximal LAD. The remaining coronary arteries were widely patent. Radiology suggested the etiology of the coronary stenosis was most consistent with a spontaneous coronary artery dissection (SCAD) with intramural thrombus. However, noncalcified plaque could not be excluded. SCAD was the most probable diagnosis, in the context of the patient's clinical presentation. After discussion with interventional cardiology, a decision was made to not perform cardiac catheterization given the risk of catheter induced propagation of the dissection plane. The patient was treated medically with heparin drip, aspirin, and clopidogrel. Her high sensitivity troponins peaked at 399 ng/dL. By hospital day number 3 she was ambulating without chest discomfort and was discharged on hospital day number 4.

Post discharge the patient was treated with aspirin, clopidogrel, atorvastatin, and metoprolol tartrate. She continued her prior antihypertensive therapy with amlodipine and losartan. For the first 3 weeks post discharge she was able to ambulate without angina but intermittent episodes of chest pain at rest that were relieved with nitroglycerin. Three weeks post discharge she represented to the ED with more intense chest pain radiating to her jaw, which was not relieved with nitroglycerin. She was hypertensive with a blood pressure of 165/109. ECG demonstrated diffuse deep ST depressions concerning for global ischemia. She was placed on a nitroprusside drip with relief of her chest pain. Telemetry showed recurrent runs of nonsustained ventricular tachycardia. Echocardiography showed a decline in her left ventricular ejection fraction to 45% with anterior wall motion abnormalities localized to the LAD territory. Repeat coronary CT angiography showed similar findings to the prior CT with an unchanged 13 mm long segment high-grade (70-99%) stenosis of the proximal LAD from presumed thrombosed dissection flap in the setting of coronary dissection. Because of the patient's hemodynamic instability, she was taken to the cardiac catheterization lab where her angiogram confirmed a focal 90% stenosis in the proximal LAD that could be consistent with SCAD or plaque. Given proximity of the dissected LAD to the left main, the patient was transferred to a quaternary care center with surgical back up in the event extracorporeal membrane oxygenation (ECMO) was required. On repeat angiography a wire was used to cross the lesion and intravascular ultrasound (IVUS) demonstrated no evidence of a dissection flap with significant atherosclerotic plaque burden in the proximal LAD. The LAD stenosis was successfully stented with a 3.5 x 33mm Xience Skypoint drug-eluting stent, post-dilated to 4.0 mm. The high sensitivity troponin peaked at 82 ng/L.

Post-discharge the patient did well. Her chest pain resolved. She participated in cardiac rehab program and improved her exercise tolerance. Repeat echocardiography showed resolution of her previously seen wall motion abnormality with normal ejection fraction.

Discussion

Spontaneous coronary artery dissection (SCAD) is defined as the spontaneous (i.e. nontraumatic, nonatherosclerotic, and noniatrogenic) separation of the coronary artery wall. It is most commonly seen in younger women.[1] SCAD registries report mean age ranging from 43 to 52 years[2] with women accounting for 82-92 percent of cases.[3] The pathophysiology of SCAD is poorly understood. Potential mechanisms include inflammation, tearing of the vasovasorum, and vessel tortuosity. Risk factors include fibromuscular dysplasia,[3] pregnancy,[4] multiparity, the postpartum state, other hormonal causes (including

IVF, hormonal therapy, oral contraception, and hormone replacement therapy), and connective tissue disorders such as Marfan's or Ehlers-Danlos.[5] Fibromuscular dysplasia has the strongest association with SCAD and has been found in 72% of SCAD patients upon subsequent screening. Potential acute triggers include emotional stress and physical stress. Presenting symptoms are similar to those of a myocardial infarction (MI), with the most common symptom by far being chest discomfort. Other symptoms include arm pain, nausea or vomiting, diaphoresis, dyspnea, and back pain.[6] Ischemic ST and T-wave abnormalities are typically found on ECG, including ST elevations.[7] Diagnosis is typically confirmed by coronary angiography. There are three types of angiographic appearance with SCAD. Type I is pathognomonic dye staining of the arterial wall with a radiolucent lumen. Type II is a diffuse long and smooth stenosis. Type III is a focal or tubular stenosis that can often mimic atherosclerosis (as with our patient). In this last type, optical coherence tomography (OCT) or intravascular ultrasound (IVUS) may be necessary to differentiate between SCAD and atherosclerotic stenosis.

SCAD cases are typically managed conservatively. Procedural failure rates with percutaneous coronary intervention (PCI) are high at 53%. In observational studies comparing conservative medical management versus PCI, vessel occlusion occurs more often with PCI at a rate of 46% vs 19% with conservative care.[8] One meta-analysis of patients with SCAD found that conservative management was associated with similar clinical outcomes and lower target vessel revascularization when compared with an invasive strategy.[9] In another study of 134 patients who were treated conservatively, only 3 required revascularization for SCAD extension. Seventy-nine patients had repeat angiography more than 4 weeks later and all were found to have spontaneous healing.[3] PCI in SCAD is often technically challenging. Wiring and stenting of the vessel risks propagating the false lumen and expanding the dissection plane. Dissections with SCAD can often be extensive, requiring a substantial stent burden to cover the affected area. Dissections can also often affect smaller caliber vessels. The potential for extensive stenting compounded with the stenting of smaller caliber vessels also increases the risk of future in-stent thrombosis or restenosis. Therefore, in stable patients the standard of care is to treat conservatively with dual antiplatelet therapy, beta blockade, and statins. However, if patients are unstable with findings of hemodynamic instability, ongoing ischemic symptoms, or left main dissection then an invasive strategy with PCI or coronary bypass graft surgery is warranted and appropriate.

In our patient an initial conservative strategy was followed given the presumptive diagnosis of SCAD based on the clinical characteristics and coronary CT angiogram findings. Subsequently, the patient became hemodynamically and electrically unstable with a decline in her left ventricular ejection fraction and frequent runs of ventricular tachycardia. Because of this clinical deterioration a coronary angiogram was performed with subsequent IVUS which revealed that the patient did not have a type III spontaneous coronary dissection but rather a severe stenosis due to atherosclerotic disease. In retrospect, earlier use of IVUS may have resulted in earlier adoption of an invasive treatment strategy. Fortunately, this patient had a favorable outcome with resolution of her symptoms and normalization of left ventricular systolic function. Unlike type I or type II SCAD angiographic findings, type III SCAD is often difficult to discern from atherosclerosis with angiography alone. In these cases, IVUS or OC are often required to make an accurate diagnosis and help decide on appropriate treatment.

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