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Spontaneous Coronary Artery Dissection Masquerading as Coronary Artery Stenosis in a Young Patient

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 34
Final Diagnosis: Spontaneous coronary artery dissection
Symptoms: Chest pain
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Rare disease

Background: Spontaneous coronary artery dissection (SCAD) is primarily found in females. SCAD can have many precipitating factors such as exercise, trauma, pregnancy, drugs, and connective tissue disease. Prognosis is poor for left main stem, left anterior descending (LAD) artery, and multivessel involvement, especially for females.

Case Report: We present a case of young African American male with sickle cell disease who presented with chest pain associated with shortness of breath. He was found to have non-ST elevation myocardial infarction (NSTEMI). He was diagnosed with SCAD during catheterization with the help of intravascular ultrasound imaging. Three drug-eluting stents were placed to cover the proximal LAD vessel along its whole length until resolution of the lesion. The patients' hospital course was complicated by an additional finding of left ventricular thrombus, possibly a complication of NSTEMI, which was treated with anticoagulation to complete resolution.

Conclusions: SCAD is fatal, it can proceed to cause myocardial infarction as in this particular patient's case, and sudden death if not recognized early. It can be missed on angiography alone; further intracoronary imaging such as intravascular ultrasound and optical computed tomography should be used to confirm the diagnosis of SCAD so that early and appropriate treatment can ensue.

MeSH Keywords: Coronary Artery Disease • Dissection • Myocardial Infarction

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/913522

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Background

Spontaneous coronary artery dissection (SCAD) is an infrequent cause of acute myocardial infarction. Earlier, diagnosis of SCAD was mostly established on postmortem examination [1,2]. Coronary dissection has been reported to be associated with several factors, including pregnancy [3], hypertension [4], cocaine use [5], connective tissue and vasculitic disorder [6,7], coronary spasm [8], blunt chest trauma, and physical exercise [9]. Newer techniques have been able to establish diagnosis of SCAD more accurately in young populations like optical computed tomography and intravascular ultrasound.

Case Report

We present a case of a 34-year-old African American male with a history of sickle cell disease who presented to our emergency department with complaints of left sided chest pain radiating towards shoulder associated with shortness of breath. The patient was admitted by Cardiology Service as his troponin was positive in the emergency department. The patient was administered morphine, nitroglycerin, and started on intravenous heparin to be treated as non-ST elevation myocardial infarction (NSTEMI). Hospitalist service was consulted as the patient was suspected to also have concurrent sickle cell crisis. The patient's chest x-ray was without any signs of infiltrates, the ECG showed T-wave inversion in v4-v6 leads and right bundle branch block, troponins were trended which peaked at 2.7 ng/mL.

An echocardiogram was ordered but the patient was taken to the catherization laboratory before results were interpreted. During the catheterization, the patient had radiolucency in the proximal left anterior descending (LAD) artery which was identified by the angiographer and looked suspicious for SCAD (Figure 1). Intravascular ultrasound was performed which demonstrated the false lumen and intramural hemorrhage and possible thrombus/plaque formation within it (Figure 2). As the patient had NSTEMI, percutaneous coronary revascularization was performed, and 3 drug-eluting stents placed in the vessel to cover the entire length of lesion (Figure 3).

Furthermore, the patient's echocardiogram showed a left ventricular thrombus (Figure 4), therefore intravenous heparin was resumed after percutaneous coronary revascularization and bridged to coumadin. During the succeeding days, repeat echocardiogram showed clearance of left ventricular thrombus. Hypercoagulable workup was performed but negative. It was believed that the patient developed left ventricular thrombus possibly from NSTEMI. The patient was discharged in stable condition on coumadin and dual antiplatelets (aspirin and Plavix) with no recurrent chest pain.

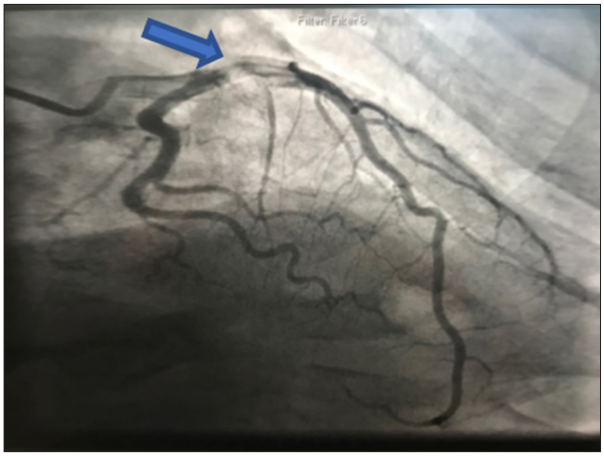

Figure 1. Angiographic haziness (arrow) on invasive angiography.

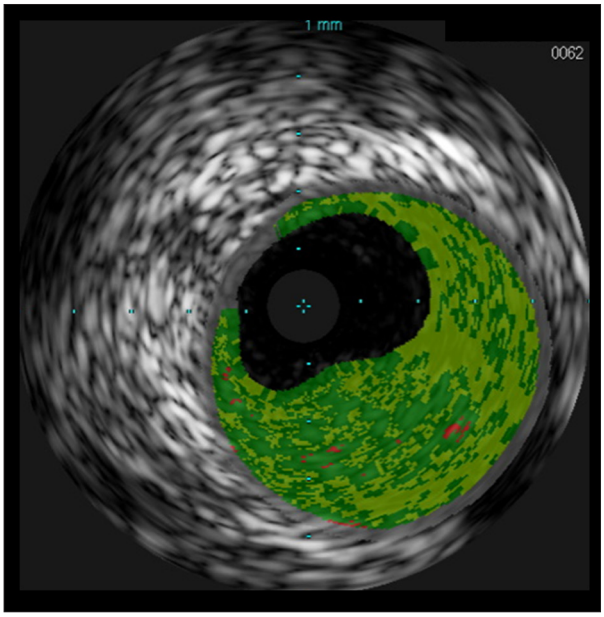

Figure 2. Intravascular ultrasound identifying false lumen (green) with intramural hematoma.

Discussion

SCAD is a rare condition described initially in 1931. Patients who have SCAD present with acute myocardial infarction or sudden death [10]. SCAD is reported in the literature to occur more frequently in females, age range 25 to 60 years, and related to pregnancy, the postpartum period [11], and oral contraceptive pills [12]. Our patient was a young male without any common risk factors to develop SCAD.

SCAD is an infrequent etiology of acute myocardial infarction with an incidence of 0.1% to 1.1% of all acute coronary syndromes [13,14]. Normally the pathophysiology of acute myocardial infarction involves a plaque acutely rupturing and forming a superimposed thrombus which leads to acute myocardial

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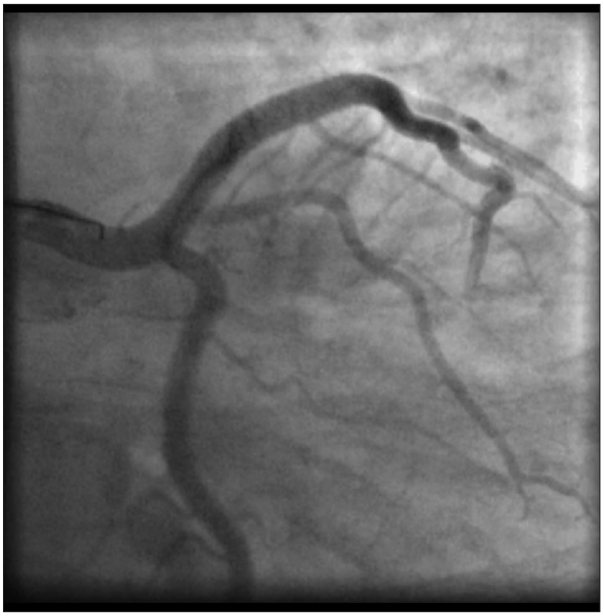

Figure 3. Angiogram showing post-stenting images of artery.

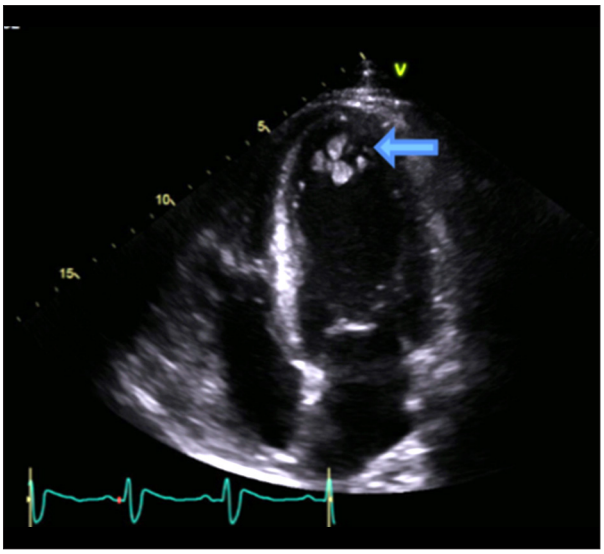

Figure 4. Echocardiogram showing left ventricular thrombus (arrow).

infarction [15], however, in SCAD it can mimic the appearance of atherosclerotic lesion in a non-atherosclerotic coronary artery.

Saw [13] characterized SCAD angiographically into 3 types: type I presents as multiple radiolucent lumens during angiographic contrast staining with dye; type II is defined as a change in arterial caliber with demarcation from normal diameter to diffuse narrowing; and type III mimics atherosclerosis.

There are 2 proposed mechanisms of SCAD. First, an intimal tear leads to hemorrhage in the inner layer of media, therefore creating a false lumen. This mechanism relates to the radiolucent lumen identified angiographically. Second, it is proposed that there may be rupture of vaso vasorum that may cause intramural hemorrhage [16]. This can be related angiographically by identifying luminal compression [17].

Our patient case did present with an acute myocardial infarction, the patient had angiographic findings of radiolucent lumen leading to a suspicion of SCAD by the angiographer, therefore, an intravascular ultrasound was performed in order to confirm the diagnosis of SCAD. Intravascular ultrasound did confirm a false lumen with hemorrhage and organized thrombus. Optical computed tomography was not performed due to lack of availability at our institution. Although optical computed tomography does provide better visualization of dissection, it may propagate dissection in some cases due to forceful contrast injection and should be used with caution.

The incidence of catheter-induced coronary dissection was approximately 30% in the pre-stent area but decrease to 2% to 3% with availability of stents. The independent predictors for chronic ischemic cardiovascular disease (CICD) are female sex and percutaneous coronary revascularization in patients with a complex anatomy (chronic total occlusion, bifurcation, and left main trunk lesion). Reports have suggested that there is instability of plaque and endothelial cell dysfunction in females associated with changes in estrogen and progesterone levels which in turn increases the stress of vessel walls and risk of dissection [18]. Vigorous injection of contrast, selection of larger catheters, inappropriate positioning of catheters, especially in patients with ostial lesions and subintimal passage of guidewire, are also associated with risk of CICD [19]. In our case, the patient presented with NSTEMI, was hemodynamically stable, had left main disease but no ostial lesion identified, and had a fixed defect on angiography and intravascular ultrasound without any distal propagation even with repeated contrast injections, therefore our patient had SCAD rather than CICD.

SCAD is usually an underestimated diagnosis due to variable angiographic findings and dependent on skills of angiographer, therefore any suspicious finding on angiograph should be confirmed with additional imaging such as optical computed tomography and intravascular ultrasound. Optical computed tomography is better at identifying intimal tears, thrombi and intramural hemorrhage, however, intravascular ultrasound can identify false lumen, compression of true lumen, and intramural hemorrhage but is not very helpful in identifying intimal tears [13,20]. Some evidence suggests that SCAD can occur without intimal tears as reported by Maehara et al. [16] and Arnold et al. [21], which described 5 cases and 4 cases respectively that had SCAD, but intimal tear was not visualized connecting the true and false lumens. Optical computed tomography had a much lower resolution (15 um) and was

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considered ideal for identifying intimal tears, should an intervention be planned in order to seal the tear [22].

Management of SCAD is controversial. The final treatment does depend on patient's clinical condition, single vessel versus multivessel dissection and presence of ischemia [23]. Conservative therapy has been reported to have good outcomes [24]. It is described that a percutaneous coronary revascularization be performed if the patient is having symptoms of ischemia, if not, patient can be managed conservatively. In our case, the patient received 3 drug-eluting stents to cover the whole section of vessel involved with dissection. It is not clear the advantage of drug-eluting stents has over bare metal stent in SCAD, but it does have a lower risk of stent restenosis. Percutaneous coronary revascularization with drug-eluting stents does carry its own complication: 1) expansion of hemorrhage and hematoma requiring stenting of large lengths of artery, and 2) guidewire insertion into false lumen propagating the dissection [23,25].

Conclusions

SCAD is a rare entity and can present in young people with cardiac symptoms. It does take a high index of suspicion and attention to the angiographic findings of SCAD in order to confirm it with additional intracoronary imaging (optical computed tomography and intravascular ultrasound). SCAD is a fatal disease and earlier detection can help successfully manage it. Our patient did not have the common risk factors associated with SCAD, therefore clinicians should also be judicious of SCAD in patients without prominent risk factors as well.

Department and Institution where work was done

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Conflict of interests

None.

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