Spontaneous Coronary Artery Dissection Treated with Direct Coronary Stenting

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Patient: Female, 39-year-old
Final Diagnosis: Spontaneous coronary artery dissection
Symptoms: Chest pain • dyspnea
Medication: —
Clinical Procedure: Percutaneous coronary intervention
Specialty: Cardiology

Objective: Rare disease
Background: Spontaneous coronary artery dissection (SCAD) is a rare medical emergency characterized by non-traumatic and non-iatrogenic tearing of the intima of a coronary artery, with an estimated incidence of 1-4%.
Case Report: A 39-year-old woman with no known cardiac risk factors or recent trauma presented with acute chest pain, electrocardiographic (ECG) changes consistent with ST-elevation acute coronary syndrome, and elevated cardiac enzymes. Coronary angiography revealed near-complete stenosis of the distal left anterior descending (LAD) coronary artery with findings consistent with coronary artery dissection. Due to ongoing chest pain refractory to medical therapy, she underwent successful complex intervention on the distal LAD lesion with a 2.0×30 mm Onyx drug-eluting stent that was post-dilated to high pressure with a 2.5 noncompliant balloon, reducing the 99% stenosis to a 0% residual. She recovered fully and was discharged on aggressive risk factor modification with dual antiplatelet therapy (aspirin and clopidogrel) and high-intensity statin.

Conclusions: Spontaneous coronary artery dissection (SCAD) is a rare condition that can present with ECG changes and ischemic symptoms identical to ST-elevation transmural myocardial infarction secondary to plaque rupture. Coronary angiography is required to evaluate patients, and, depending on the catheterization findings, the patient’s hemodynamic profile, and severity of ischemic symptoms, complex interventions such as direct coronary stenting can best treat patients such as ours, while medical management might be considered for others.

MeSH Keywords: Coronary Artery Disease • Dissection • Stents

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**Background**

Spontaneous coronary artery dissection (SCAD) is a rare emergency condition characterized by a non-traumatic and non-iatrogenic tear of the intima of a coronary artery, with an estimated incidence of 1–4% [1]. Most cases occur in females and there appears to be a predilection for the left coronary vasculature in females compared to right coronary vasculature in males [2–5]. Although its pathogenesis is not fully understood, an intimal tear or bleeding of the vasa vasorum has been proposed [6]. Risk factors include connective tissue disorders such as Ehlers-Danlos syndrome and Marfan syndrome, as well as physical stress, emotional stress, or hormonal states [7]. Symptoms depend on the severity of the dissection and may range from exertional chest pain to sudden cardiac death.

Because definitive diagnosis is often made during coronary angiography, it is believed that its incidence is underestimated [8,9]. Diagnosis can also be made with intravascular ultrasound and optical coherence tomography, but coronary angiography remains first line for diagnosis [1]. We present the case of a 39-year-old healthy woman who presented to the Emergency Department with severe anginal chest pain and dyspnea with electrocardiographic and biochemical evidence of acute coronary syndrome. She had emergent left heart catheterization, which revealed left anterior descending coronary artery dissection, and was treated with stent placement, dual antiplatelet therapy, beta-blockers, and high-intensity statin, which led to complete recovery.

**Case Report**

A 39-year-old otherwise healthy woman presented to the Emergency Department with one-day history of left-sided chest pain while at work. She described her pain as being intermittent, worse with activity, and relieved by rest. She had associated dyspnea but denied any cough, nausea, vomiting, or palpitations. She neither smoked cigarettes nor used recreational drugs. Her family history was unremarkable for cardiac disease and she was not on any prescribed medications. Vital signs showed mild hypertension with a blood pressure of 149/98 mmHg, pulse rate 80 beats/min, respiratory rate of 20/min, temperature of 36.5°C with oxygen saturation of 97% on ambient air. On physical examination, she was diaphoretic and in discomfort. Heart sounds were normal and lungs were clear to auscultation. The remainder of the physical examination was unremarkable. The chest pain worsened in the Emergency Department to become persistent and was described by the patient as “10/10 discomfort”. The pain was refractory to sublingual nitroglycerin and morphine boluses.

**Investigations**

An electrocardiogram (ECG) revealed anterior precordial ST-segment elevation and T wave inversions in the anterior, inferior, and lateral leads (Figure 1). Cardiac enzymes revealed troponin I of 1.18 (normal <0.06 ng/ml), CK-MB of 8.8 ng/ml (normal 0.6–6.8 ng/ml), and elevated percentage MB relative index of 4.6% (normal 0–2.5%). Complete blood count, lipid panel, and comprehensive metabolic panel were essentially normal.

**Differential diagnosis**

Based on the characteristic angina-type chest pain and ischemic findings on ECG, our initial diagnosis was ST-elevation myocardial infarction (STEMI). Myopericarditis and takotsubo myopathy were felt to be less likely due to lack of supporting data from the patient’s history. We considered aortic dissection, but equal blood pressure in both arms and the lack of an enlarged mediastinum on x-ray made this less likely. Despite no known history of connective tissue disorder, recent excess emotional or physical stress, given the presentation of a young woman with no cardiac risk factors presenting with acute chest syndrome, SCAD was considered.

**Treatment**

The patient had emergent coronary angiography, which demonstrated 99% stenosis of the distal third of the left anterior descending (LAD) coronary artery, with a diffuse, long, smooth tubular lesion (thought to be due to intramural hematoma) with no visible dissection plane (Figure 2). There was TIMI grade 1 flow in the distal LAD coronary artery. The remainder of the coronary vessels were completely normal. Given that the patient had continued symptoms despite maximal anti-anginal therapy with TIMI grade 1 flow noted, the decision was made to perform percutaneous coronary intervention. A 2.0×30 mm Onyx drug-eluting stent was placed with 0% residual stenosis and restoration of TIMI 3 blood flow (Figure 3). She was started on beta-blocker, high-intensity statin, and dual antiplatelet therapy with aspirin and prasugrel.

**Outcome and follow-up**

A post-percutaneous coronary intervention (PCI) echocardiogram revealed normal left ventricular systolic function and no regional wall motion abnormality. The patient subsequently recovered fully and was discharged on dual antiplatelet therapy (aspirin and clopidogrel) and statin therapy. Follow-up ECG demonstrated normalization of ST segments, without evidence of myocardial infarct pattern (lack of the development of q-waves) (Figure 4).
Figure 1. ECG showing anterior precordial ST elevation and T wave inversion in the anterior, inferior, and lateral leads in a 39-year-old woman admitted with chest pain and elevated troponins.

Figure 2. Coronary angiogram in the LAO projection. The red arrow shows a type 2 SCAD involving the distal third of left anterior descending coronary artery, resulting in 99% occlusion. It shows a diffuse, long, smooth tubular lesion (thought to be due to intramural hematoma), with no visible dissection plane.

Figure 3. Coronary angiogram in the LAO projection after coronary stent implantation. The red arrow demonstrates 0% residual stenosis in the dissected area.
SCAD is an uncommon etiology of acute coronary syndrome, with symptoms ranging from angina, dyspnea, and diaphoresis to sudden cardiac death, depending on the extent of the dissection [9]. Its incidence accounts for about 1–4% of cases of acute coronary syndrome, particularly among young women with little or no risk factors for atherosclerosis, as was the case in this patient [1]. It is responsible for up to 35% of all cases in women <50 years of age presenting with acute coronary syndrome, according to the American Heart Association scientific statement in 2018 [1]. Our patient exhibited findings consistent with type 2 coronary artery dissection with diffuse, long, smooth tubular lesions as a result of intramural hematoma, without a visible dissection plane. This is the most common type of SCAD, occurring in approximately 67% of cases. Type 1, which occurs in 29% of cases, usually appears as a longitudinal filling defect in which contrast stains the arterial wall, giving the appearance of a double lumen. Type 3, which is least common, mimics atherosclerosis, giving it the appearance of multiple focal tubular lesions from an intramural hematoma [10]. The diagnosis can be easily missed or delayed, as young patients without cardiac risk factors are often classified as low risk using risk stratification tools such as Heart Score [11,12]. The pathogenesis is poorly understood, but there seems to be an association with abnormal shear forces and changes in the arterial wall [6]. Several factors have also been associated with SCAD, including use of oral contraceptive pills, heavy exercise, fibromuscular dysplasia, blunt chest trauma, cocaine use, connective tissue disorders such as Marfan syndrome and Ehlers-Danlos syndrome, and some inflammatory diseases such as Kawasaki disease, sarcoidosis, and systemic lupus erythematosus [7]. The diagnosis of SCAD is based on coronary angiography and intracoronary imaging, including optical coherence tomography (OCT) and intravascular ultrasound (IVUS), which can image the arterial wall layers [7]. However, OCT and IVUS are not readily available and are associated with other risks and costs. Therefore, coronary angiography is still the mainstay of diagnosis despite its limitation in imaging the arterial wall. Treatment options include conservative management or emergent revascularization with PCI and CABG. A conservative approach can be considered in patients with non-critical luminal obstruction, TIMI grade 3 flow, and stable clinical symptoms and hemodynamic findings [3,8]. Some studies have shown that PCI is associated with high failure rates and complications, and does not protect against recurrent SCAD [3,8]. In a prospective study by Saw et al., during which 327 patients with SCAD were followed up for a mean period of 3.1 years, the recurrence rate was 10.4%; of note, adequate blood pressure control and use of beta-blocker seemed to be protective in this cohort of patients [13]. However, PCI may be necessary for those presenting acutely with symptoms of refractory ischemia and hemodynamic compromise.
Conclusions

SCAD is a rare condition that can present with severe complications such as myocardial infarction, congestive heart failure, and sudden cardiac death. It often presents with clinical symptoms and electrocardiographic and laboratory findings identical to a transmural myocardial infarction related to plaque rupture. Percutaneous coronary intervention may be considered in patients with hemodynamically unstable, compromised coronary artery TIMI flow grade, or those with persistent ischemia despite maximal medical therapy, provided there is suitable coronary anatomy.

References:

1. Hayes SN, Kim ES, Saw J et al: Spontaneous coronary artery dissection: Current state of the science: A scientific statement from the American Heart Association. Circulation, 2018; 137(19): e523–57
2. Nishiguchi T, Tanaka A, Ozaki Y et al: Prevalence of spontaneous coronary artery dissection in patients with acute coronary syndrome. Eur Heart J, 2016; 5(3): 263–70
3. Tweet Marysia S, Hayes Sharonne N, Pitta Sridevi R et al: Clinical features, management, and prognosis of spontaneous coronary artery dissection. Circulation, 2012; 126(5): 579–88
4. Saw J: Spontaneous coronary artery dissection. Interventional cardiology (London, England), 2015; 10(3): 142–43
5. DeMaio SJ Jr, Kinsella SH, Silverman ME: Clinical course and long-term prognosis of spontaneous coronary artery dissection. Am J Cardiol, 1989; 64(8): 471–74
6. Kamineni R, Sadhu A, Alpert JS: Spontaneous coronary artery dissection: Report of two cases and a 50-year review of the literature. Cardiol Rev, 2002; 10(5): 279–84
7. Saw J, Mancini GB, Humphries KH: Contemporary review on spontaneous coronary artery dissection. J Am Coll Cardiol, 2016; 68(3): 297–312
8. Tweet MS, Eleid MF, Best PIM et al: Spontaneous coronary artery dissection. Circulation: Cardiovascular Interventions, 2014; 7(6): 777–86
9. Tanis W, Stella PR, Kirkels JH et al: Spontaneous coronary artery dissection: Current insights and therapy. Neth Heart J, 2008; 16(10): 344–49
10. Aziz S: Spontaneous coronary artery dissection. E-Journal of Cardiology Practice, 2017; 14(38) https://www.escardio.org/Journals/E-Journal-of-Cardiology-Practice/Volume-14/spontaneous-coronary-artery-dissection
11. Lindor RA, Tweet MS, Goyal KA et al: Emergency Department presentation of patients with spontaneous coronary artery dissection. Emerg Med, 2017; 52(3): 286–91
12. Than M, Flaws D, Sanders S et al: Development and validation of the Emergency Department assessment of chest pain score and 2 h accelerated diagnostic protocol. Emerg Med Australas, 2014; 26(1): 34–44
13. Saw J, Humphries K, Aymong E et al: Spontaneous coronary artery dissection: Clinical outcomes and risk of recurrence. J Am Coll Cardiol, 2017; 70(9): 1148–58

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Conflict of interest

None.