Spontaneous coronary artery dissection with left ventricular thrombus

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ABSTRACT

Spontaneous coronary artery dissection (SCAD) is a rare, nonatherosclerotic cause of acute coronary syndrome. The etiology is unclear, and optimal treatment for SCAD remains undefined. We describe a patient with significant cardiovascular risk factors who presented with SCAD resulting in anterior wall acute myocardial infarction with left ventricular thrombus. The patient was managed conservatively with anticoagulant and antiplatelet therapy.

KEYWORDS Acute coronary syndrome; medical management; spontaneous coronary artery dissection

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome (ACS). Patients with SCAD present differently than typical patients with ACS, because they are often young women without significant cardiovascular risk factors. The etiology remains unclear and treatment results have been suboptimal.

CASE DESCRIPTION

A 64-year-old woman with morbid obesity (body mass index 45 kg/m²) with previous hypertension and hyperlipidemia presented with substernal chest pain with radiation to her right arm. Electrocardiogram showed normal sinus rhythm with T wave inversion in the precordial leads, lead III, and lead aVF. Her troponin level was 2.02 ng/mL. Treatment with intravenous heparin, a beta-blocker, an antiplatelet, and a statin eliminated her symptoms.

Echocardiography showed a left ventricular ejection fraction of 35% with anteroapical akinesis and left ventricle (LV) apical thrombus (*Figure 1a*). Coronary angiogram revealed a diffuse, moderate to severely stenotic long tubular lesion in the mid left anterior descending artery >100 mm in length, consistent with SCAD (*Figure 2a*). There was normal flow into the apical left anterior descending artery, and the narrowing persisted after administration of 400 mcg

intracoronary injection of nitroglycerin. No other stenoses were noted. Clopidogrel, warfarin, and beta-blockade therapy was continued, and she remained stable at discharge.

Follow-up coronary angiogram 4 months later showed complete resolution of the coronary artery dissection (*Figure 2b*). Echocardiogram nearly a year after the SCAD diagnosis showed normalization of her LV function and complete resolution of the LV apical thrombus (*Figure 1b*).

DISCUSSION

SCAD is estimated to be responsible for about 2% of ACS cases, with a prevalence of <1% in angiographic studies.¹ It is notable for its preponderance in younger women and is responsible for up to 24% of ACS cases in women <50 years of age.^{2,3} An association between SCAD and fibromuscular dysplasia has been described, but the etiology and pathogenesis of SCAD are still uncertain.⁴

The optimal treatment for SCAD remains undefined. Possible interventions include conservative medical therapy and invasive therapies such as percutaneous coronary intervention (PCI) and coronary artery bypass grafting. Medical management is preferred in stable patients with normal flow in the affected coronary artery, because most dissected segments tend to heal spontaneously. Medical therapy is similar to that used for ACS patients, with dual antiplatelet therapy,

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Figure 1. Echocardiogram (systole and diastole) showing apical views of the left ventricle (a) at presentation and (b) at 1-year follow-up.

Figure 2. Coronary angiogram of the left coronary artery system (a) at presentation and (b) at 4-month follow-up.

heparin, and beta-blockers. Studies have shown that most patients treated with conservative management experience a benign in-hospital course with angiographic resolution on follow-up studies.⁵ PCI has been associated with significant challenges such as propagation of the dissection and persistent vessel occlusion, with a reported success rate of <50%. Coronary artery bypass grafting is considered when PCI has been unsuccessful or is not feasible or when there is left main coronary artery dissection. Due to reported success rates of <32% at 3.5 years, PCI is generally preferred if revascularization is indicated.^{6,7}

This case is important for several reasons. First, it highlights coronary dissection as an unusual cause of an anterior wall myocardial infarction with LV thrombus. Second, the resolution of symptoms with medical therapy was crucial in avoiding PCI. Third, the patient healed with anticoagulant and antiplatelet therapy. Consistent with studies that suggest excellent in-hospital and long-term prognosis in most cases

of SCAD, our patient has remained asymptomatic for over a year since admission.⁷

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