Spontaneous coronary artery dissection (SCAD), which typically affects premenopausal women who are otherwise healthy, is caused by a nonatherosclerotic coronary event. Because SCAD is rare, its association with acute coronary syndrome (ACS) as a cause of myocardial infarction may go unrecognized. In searching the medical literature, one group of authors found more than 1,200 reports of SCAD, 63 of recurrent SCAD, and only 3 of multiple episodes that involved different vascular territories. We report the case of a patient who presented 4 times with SCAD in different vascular territories, and we discuss some of what is known about the condition and its treatment.

**Case Report**

In 2007, a 30-year-old woman with chest pain presented at our university teaching hospital. Her medical history included hyperlipidemia and irritable bowel syndrome; she had been adopted, so her family medical history was unknown. Examination revealed no hypermobile joints or other indications of Marfan syndrome. An electrocardiogram (ECG) showed sinus rhythm with T-wave inversion in leads V1 through V3. The patient was treated initially with aspirin, intravenous heparin, nitrate therapy, β-blockers, and morphine. The dynamic ECG changes and a cardiac troponin level that peaked at 42 ng/mL prompted cardiac catheterization. Angiograms showed 75% diffuse stenosis of the first obtuse marginal branch (OM1) and suggested spontaneous dissection (Fig. 1). Of note, the coronary vessels were generally tortuous; however, no disease was obvious beyond the OM1. Because of the preserved flow, the small caliber of the affected vessel, and the patient’s hemodynamic stability, we prescribed only medical therapy.

In 2014, the patient presented with recurrent chest pain and cardiac troponin elevation (peak level, 7 ng/mL); the diagnosis was non-ST-segment-elevation myocardial infarction. Shortly after arriving at the cardiology catheterization laboratory, she became hemodynamically unstable and had a ventricular fibrillation cardiac arrest. Resuscitation efforts included one shock, and spontaneous circulation resumed within 5 minutes. A coronary angiogram showed 99% stenosis of the mid left anterior descending coronary artery (LAD) and possible coronary artery dissection (Fig. 2A). The patient underwent percutaneous coronary intervention (PCI), during which a drug-eluting stent was deployed in her mid LAD with excellent results (Fig. 2B). Screening results were negative for scleroderma, rheumatoid arthritis, lupus erythematosus, and Marfan syndrome.
In October 2016, the patient presented for the 3rd time with chest pain. An ECG revealed T-wave inversions in leads V1 through V3 that were stable in comparison with those on her previous ECG, and her cardiac troponin level peaked at 36 ng/mL. An angiogram showed a patent mid-LAD stent, but a new dissection involved the proximal LAD and extended to the left main coronary artery (Fig. 3). The patient's symptoms soon resolved; she was also hemodynamically stable and had Thrombolysis in Myocardial Infarction (TIMI) III flow, so we decided to pursue conservative management and to defer revascularization. We advised the patient to continue β-blocker therapy and recommended that she and her children avoid contact sports.

One week after discharge from the hospital, the patient presented at the emergency department, reporting severe substernal chest pain. An ECG revealed new 1-mm ST-segment elevation in leads V1 through V3. An angiogram showed propagation of her prior proximal LAD dissection, 99% stenosis of the LAD, and TIMI II flow (Fig. 4A). The remaining coronary circulation appeared to be unchanged.

Given the patient’s ST-segment-elevation myocardial infarction and compromised LAD flow, we and our hospital’s cardiac surgeons discussed the merits of PCI versus left internal mammary artery (LIMA)-to-LAD coronary artery bypass grafting (CABG). We decided to deploy a proximal LAD stent, which resulted in proximal left circumflex coronary artery (LCx) impingement and the consequent addition of a stent in the LCx ostium. An angiogram showed patency of both stents and TIMI III flow (Fig. 4B). The patient’s symptoms and ST-segment elevation rapidly resolved. We admitted the patient for observation, with the plan to perform urgent coronary angiography and surgical revascularization if her symptoms returned.

The patient indeed had recurrent chest pain despite antianginal therapy, so she underwent double-vessel CABG (LIMA–LAD and a saphenous vein graft to the OM1), without complications. She was discharged from the hospital 5 days later. As of March 2018, she was doing well on prescribed antianginal therapy, with no restrictions.

**Discussion**

The prevalence of SCAD (0.1%–0.2% in patients undergoing cardiac angiography) is probably underestimated because of the condition’s infrequent association with clinical ACS. One identification system (not yet widely adopted) proposes 3 groups of patients...
with SCAD: those who have underlying atherosclerosis; those with childbirth-associated SCAD; and those without identifiable risk factors, like our patient.

Because SCAD is rare and heterogeneous, its pathophysiology is poorly understood. Possible causes are increased density of the vasa vasorum, intimal injury, and cystic medial necrosis associated with an underlying connective-tissue disease such as fibromuscular dysplasia. The reported pattern of arterial involvement resembles that of atherosclerotic disease: 66% of instances affected the LAD, 25% the right coronary artery, and 7% the LCx; few reported cases involved multivessel dissection. Our patient had tortuous cardiac vessels, particularly the LAD. Coronary artery tortuosity is often present in patients with SCAD. Furthermore, 80% of recurrent dissections were reported within tortuous segments. It is undetermined whether tortuosity predisposes patients to arterial fragility or if it is a marker of SCAD.

The prognosis in SCAD is unknown, and there is little evidence to guide therapeutic strategies. The relevant literature consists mainly of case reports, not large controlled studies. In hemodynamically stable patients with SCAD who do not have ongoing symptoms, medical management (antiplatelet therapy, heparin and nitrate therapy, and β-blockade) has been recommended. Thrombolytic therapy is contraindicated because of possible dissection propagation. In patients with single-vessel SCAD and ongoing ischemia, hemodynamic instability, or both (as in our patient's 2nd and 4th presentations), PCI has been recommended. In patients with multivessel or left main coronary artery disease, or with SCAD refractory to other management, CABG is recommended, if feasible, and this was ultimately performed in our patient.

Our patient's case has several noteworthy features. First, she had what we consider to be 4 separate dissection events. Second, these events involved different vascular territories, which is highly unusual. Third, there were no clear predisposing risk factors, implying recurrent idiopathic SCAD. We regret our inability to obtain intracoronary images consequent to the patient's tenuous presentations and our concern about dissection propagation; furthermore, we had no access to images at the time of CABG.

On the basis of this experience, we concur with prevailing opinion: PCI is indicated in symptomatic, isch-
emic patients with recurrent SCAD, and CABG should be performed when recurrent dissection is unresponsive to other options.

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