Think SCAD (Spontaneous Coronary Artery Dissection) for MINOCA (Myocardial Infarction in Patients With Unobstructed Coronary Arteries)

Dr. Morton Kern with contributions from Drs. Steve Ramee, New Orleans, Louisiana; Jacqueline Saw, Vancouver, British Columbia; Jon Tobis, Los Angeles, California; Barry Uretsky, Little Rock, Arkansas.

Although some of the causes of myocardial infarction with no coronary artery obstruction (MINOCA) are often unknown, spontaneous coronary artery dissection (SCAD) in MINOCA should always be considered early on as one of the major etiologies of the MINOCA diagnosis. Dr. Barry Uretsky from the University of Arkansas prompted this discussion on SCAD. I thought this column would be a good time to address Dr. Uretsky's questions and provide a brief review of SCAD for the cath lab.

Dr. Uretsky describes 2 patients about whom he was consulted:

Patient 1 is a woman in her 50s, with chronic back pain, hypertension, and remote history of a transient ischemic attack (TIA). She presented to the emergency department with chest pain and elevated high sensitivity troponins >80,000 and was taken to the cath lab. She was found to have type II spontaneous coronary artery dissection in the mid to distal left anterior descending (LAD) coronary artery (Figures 1-2; Video 1 online). The right coronary artery (RCA) was normal. Of note, the patient had a similar presentation more than 1 year ago with high sensitivity troponins >16,000. The angiogram was normal (Figure 3; Video 2 online).

Patient 2 is a woman in her 40s who is 4 weeks postpartum and presented with chest pain, accelerated hypertension, and elevated high sensitivity troponins >32,000. In the cath lab, she was found to

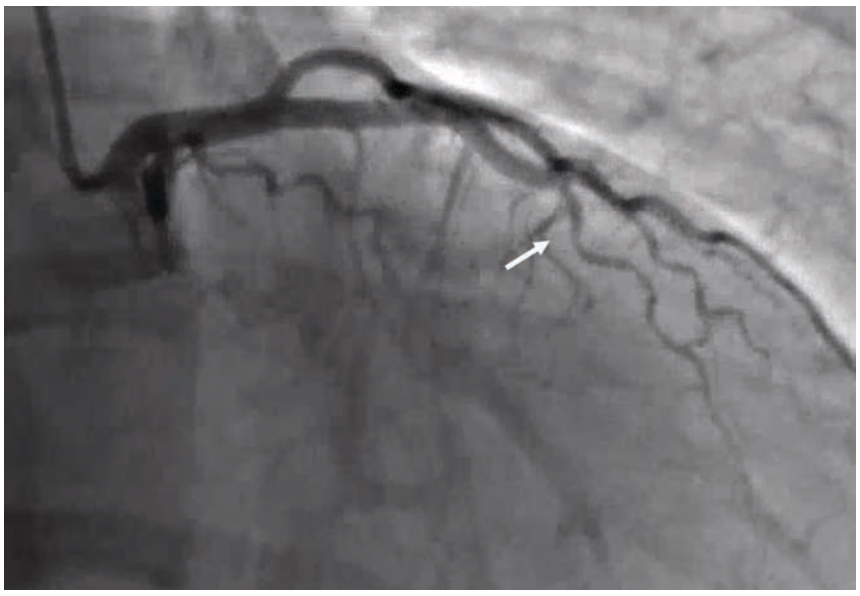

Figure 1. Patient #1 (Video #1). Cine frame from 2024 angiogram. Arrow indicates spontaneous coronary artery dissection (SCAD) in the left anterior descending (LAD) coronary artery.

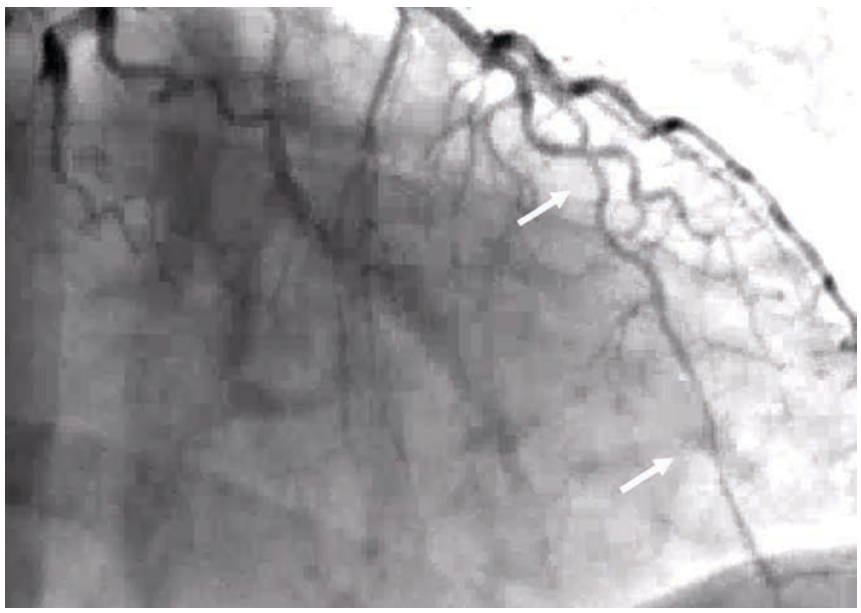

Figure 2. Patient #1. Later cine frame from 2024 angiogram. Arrows indicates SCAD in the LAD.

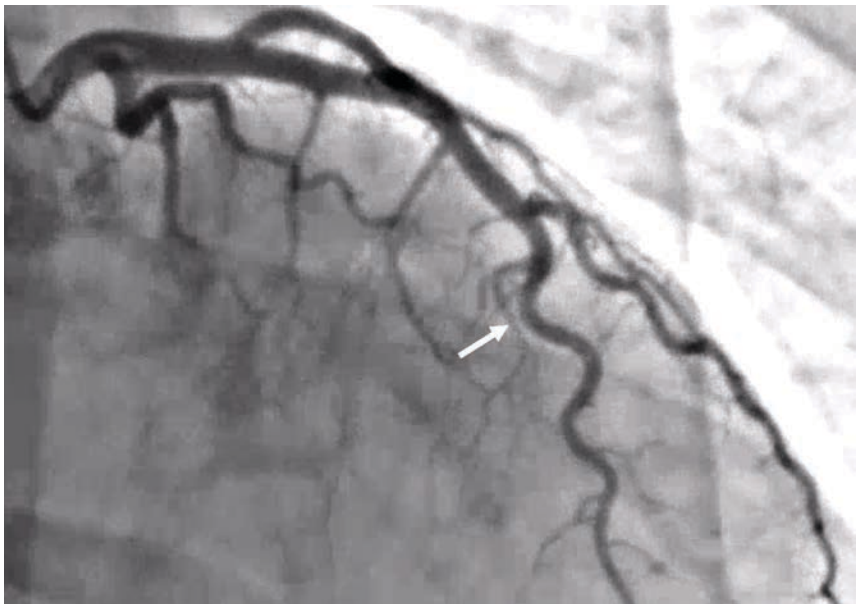

Figure 3. Patient #1 (Video #2). Cine frame from 2022 angiogram. Note caliber of LAD (arrow).

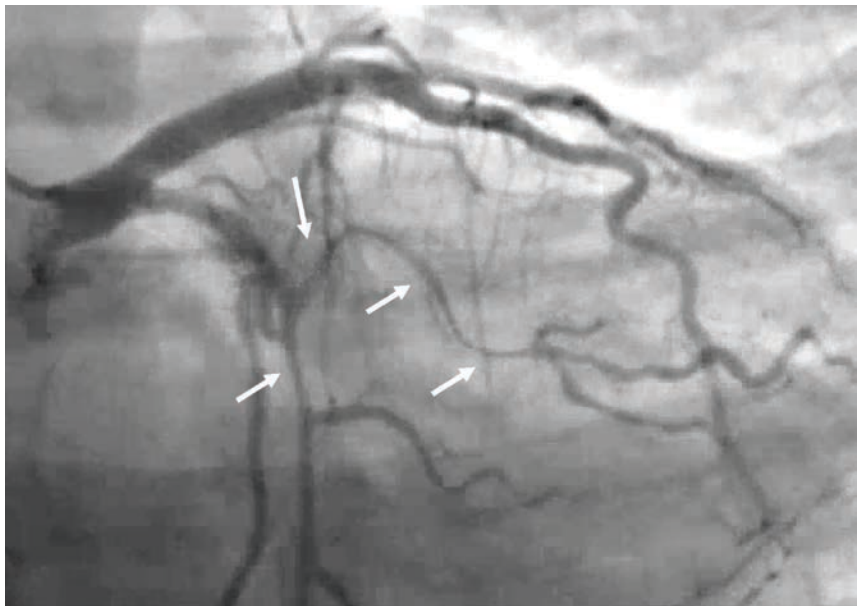

Figure 4. Patient #1. Cine angiogram frame showing diffusely dissected obtuse marginal 1 (arrows) (Video #3).

Although a "conservative as possible" approach is generally preferred in acute SCAD, more severe presentation, ongoing symptoms, and other high-risk features may warrant revascularization (Figure 6).

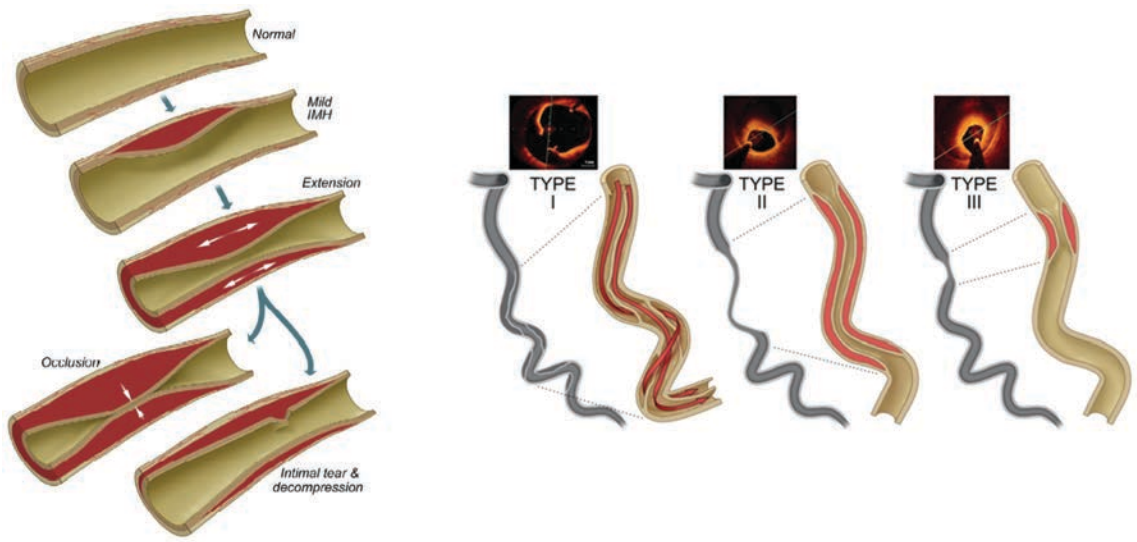

Figure 5. (Left) An intramural hematoma forms and most commonly reabsorbs and heals. Early hematoma extension can result in vessel occlusion or develop an intimal tear resulting in decompression and restoration of flow. (Right) Angiographic, anatomic, and optical coherence tomographic (OCT) representation of spontaneous coronary artery dissection types 1 to 3. Type 1 has contrast dye stasis or slow clearing of dye within the intimal tear. OCT shows an intimal tear and/or an intramural hematoma. Type 2 SCAD shows no intimal tear and appears as a long (>20mm) diffusely narrowed artery, often normal on each end of dissected area (type 2a) or involves the entire length of artery (type 2b). It is the most common type of SCAD, occurring in 60 to 75% of patients. Type 3 SCAD has a narrowed lumen by compression by an intramural hematoma, usually <20 mm. Intracoronary imaging is often necessary to make the diagnosis.

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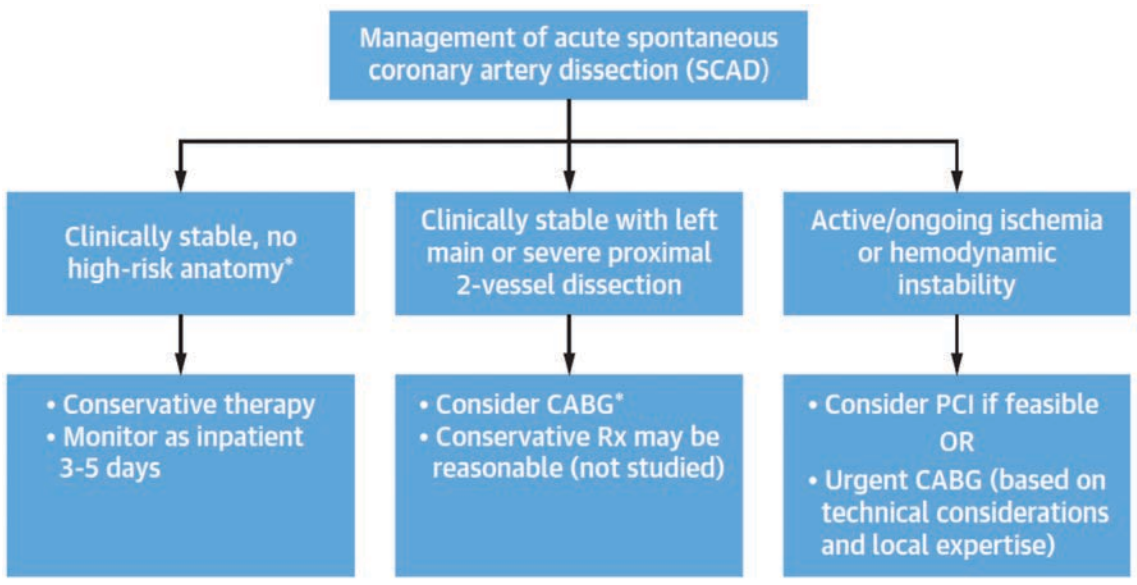

Figure 6. A proposed management algorithm for SCAD. Although a "conservative as possible" approach is generally preferred in acute SCAD, more severe presentation, ongoing symptoms, and other high-risk features may warrant revascularization. *Left main or proximal 2-vessel coronary artery dissection. *CABG = coronary artery bypass grafting; PCI = percutaneous coronary intervention; Rx = management.*

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have type II spontaneous coronary artery dissection of an obtuse marginal (OM) branch (Figure 4; Video 3 online) with a small branch cutoff.

Dr. Uretsky was asked the following questions, which he shared with our expert group for their responses:

What is your approach for acute inpatient management?

1) Heparin or no heparin? Rationale?
2) Dual antiplatelet therapy (DAPT) or no DAPT? If yes, for how long?
3) What additional therapies, apart from beta blockers and blood pressure control, would you recommend?
4) What is the role of novel oral anticoagulants (NOAC) (eg, apixaban [Eliquis, Bristol-Myers Squibb and Pfizer]) in both the short- and long-term management?

Mort Kern, Long Beach, California: Barry, great questions on the management of SCAD. While at the VA we do not see many women, let alone women with SCAD, although I have seen a couple. My understanding is that unless there is active ischemia/infarction, a conservative approach of beta blockers and other antihypertensives can be effective. The major question of when to intervene with stenting is a tough one. Stenting for ongoing symptoms not responding to the conservative approach seems to be a right answer but not an attractive one (see discussion below). I am uncertain but disinclined to use DAPT and/or

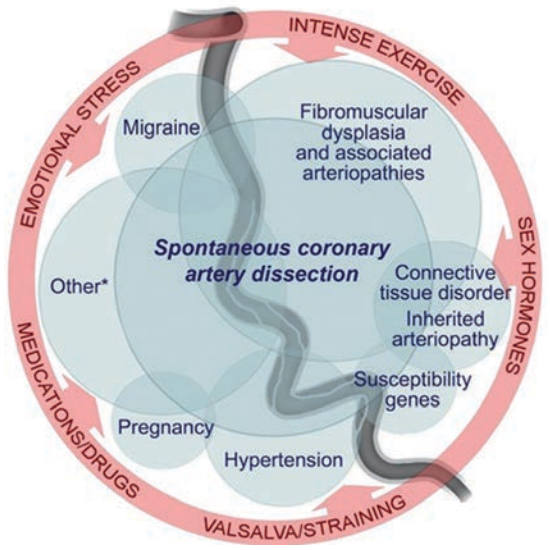

Figure 7. SCAD and associated conditions. SCAD often occurs in the setting of 1 or more overlapping conditions (blue circles), often with a precipitant or trigger (red outer ring) which includes idiopathic, systemic inflammatory conditions, coronary vasospasm, and other less common associated conditions.

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TABLE 1. Associations with spontaneous coronary artery dissection (SCAD).
Intimal tearing and bleeding of vasa vasorum
Inflammation
Tortuosity
Pregnancy and postpartum, other hormonal causes
Genetic predisposition
Disease associations – migraine headache, fibromuscular hyperplasia, connective tissue disorders, autoimmune disorders
Trigger associations – emotional stress, exercise, hormonal cycles.

a NOAC in the short-term or long-term management of SCAD. I would probably start short-term heparin (<48 hours), beta blocker, and low dose aspirin (ASA). While I don't have strong data to support heparin, it might minimize acute thrombosis progression, something to be balanced against the chance of enlarging an intimal hematoma. With this mindset, patient 2 seems like she might benefit from stenting, but even with some active ischemia, I would prefer not to put wires in the marginal branch for what might become a complicated vessel closure and with relatively little myocardial salvage. I mentioned recently at an interventionalists meeting that for OM branch interventions, "marginal percutaneous coronary intervention (PCI) is often marginal" (meaning of marginal benefit). As for long-term management, I think beta blocker, low dose ASA, and diabetes mellitus control are the only recommendations I have.

Jacqueline Saw, University of British Columbia, Vancouver, British Columbia: For patient 1, I urge you to relook at the high 1st diagonal artery on the 2022 angiogram. From the one angiogram projection that was provided, it looks like there is diffuse narrowing (type 2) SCAD of this high diagonal artery. Please look at other cranial projections to hunt for it. Also look for wall motion abnormality on the left ventriculogram. I assume there will be lateral hypokinesis corresponding to the high troponin >16,000. In fact, when you compared to the right anterior oblique (RAO) cranial angiogram of 2024, this high 1st diagonal is of larger caliber (healed).

To your questions, we generally do not continue heparin post-SCAD diagnosis on cath. As to DAPT, it is a good question. We just obtained a Canadian Ischemic Heart Research grant (CIHR) grant to randomize acute SCAD patients to D(dual)APT versus S(single)APT. For now, my standard practice is DAPT for about a month, then aspirin long term. For beta blocker and blood pressure control, we strongly recommend beta blocker long term, with optimal blood pressure control. Lastly, we do not use DOAC post SCAD. Unless, obviously, there is atrial fibrillation or another indication for DOAC.

Steve Ramee, Ochsner Clinic, New Orleans, Louisiana: I have treated many patients with SCAD over the years. Before we knew what SCAD was, we would intervene [with PCI], usually with bad outcomes (perforation, persistent occlusion, myocardial infarction [MI]) and leaving an occluded vessel. For the past 10 years I have recommended and managed SCAD with conservative therapy (DAP +/- OAC) with a relook angiogram at 2-3 months. Usually, the vessels heal and appear normal on repeat angiogram. The initial MI is unavoidable, but intervention will probably make matters worse.

Jon Tobis, Los Angeles, California: I agree with Steve Ramee that most of the time, conservative therapy is best for SCAD. I have performed PCI when the patient had a left main (LM) dissection or is near cardiogenic shock. I always use intravascular ultrasound (IVUS) after I pass the wire and that tells me whether the guidewire is in the true lumen. If the wire is in the false lumen, I use a second wire. Once I am convinced from IVUS that the wire is in the true lumen, then I stent the vessel with good results. IVUS-guided stenting is the appropriate method if the patient is unstable.

A Brief Review of SCAD: Definition, Mechanisms, Associations, and Management

SCAD occurs because of a separation of the layers of the epicardial coronary artery wall by intramural hemorrhage, with or without an intimal tear.[1-4] SCAD is not associated with atherosclerosis, iatrogenic injury, or trauma. The typical presentation of SCAD is a middle-aged woman with acute coronary syndrome, often ST-elevation MI (STEMI). The angiogram appears mostly normal except for evidence of a dissection or narrowing out of proportion to the adjacent angiographic anatomy (see types of SCAD below). Intravascular imaging with ultrasound (IVUS) or optical coherence tomography (OCT) has provided support that medial dissection or rupture of the vasa vasorum results in intramural hemorrhage and formation of an intramural hematoma with arterial narrowing or occlusion.

At first glance, the SCAD patient may have entirely normal-appearing vessels by angiography and scrutiny is warranted. Some operators recommend cautious coronary contrast injections once the diagnosis of a dissection is made.

Angiographic Types of SCAD

There are 3 types of angiographic findings related to SCAD (Figure 5):

- **Type 1** has contrast dye stasis or slow clearing of dye within the intimal tear. OCT shows an intimal tear and/or an intramural hematoma.
- **Type 2**: No intimal tear and appears as a long (>20 mm), diffusely narrowed artery, often normal on each end of dissected area (type 2a) or involves the entire length of artery (type 2b). Type 2 is the most common type of SCAD, occurring in 60 to 75% of patients.
- **Type 3** has a narrowed lumen by compression from an intramural hematoma, usually <20 mm or less. Intracoronary imaging is often necessary to make the diagnosis.

Prevalence of SCAD

The incidence of SCAD is 0.5 to 0.7% among patients with acute coronary syndrome (ACS) referred for angiography. SCAD occurs most in women. Approximately 90% of patients with SCAD are women who present between 45-55 years old. Angiographic studies reviewing SCAD patients note that about 25-30% of myocardial infarctions in women <50 years of age are caused by SCAD. SCAD also accounts for approximately 15-20% of myocardial infarctions during pregnancy or the peripartum period.[5]

Men account for <10-15% of SCAD cases. However, males are more likely to have a different reason for coronary dissection, related to atherosclerosis rather than a medial flap or tearing common to SCAD. Men also present at a younger age (<50 years) than women.

Diagnostic Approach

As with most STEMI patients, the history, exam, electrocardiogram, chest x-ray, and troponins are integral to the diagnosis. At first glance, the SCAD patient may have entirely normal-appearing vessels by angiography and scrutiny is warranted. Some operators recommend cautious coronary contrast injections once the diagnosis of a dissection is made. Treatment is mostly conservative (see management algorithm [Figure 6]). Beyond angiography, it may be necessary to perform intravascular imaging to establish the angiographic type of SCAD that is occurring. Coronary computed tomographic imaging is not very good for SCAD, but cardiac magnetic resonance imaging has been used to make the diagnosis.

Clinical Associations

Table 1 lists several of the clinical associations and triggers of SCAD. Interestingly, SCAD associated with atherosclerosis is typically limited in extent by medial atrophy and scarring from atherosclerotic degeneration. Figure 7 summarizes the associations and causes of SCAD.

Management of SCAD

Most experts agree that a conservative approach is the preferred strategy for most patients. Clinically stable patients benefit from blood pressure control and reduction of contractility via beta blockers (Figure 6).

In patients presenting with acute MI and/or those having ischemic symptoms, hemodynamic compromise, or left main artery dissection, revascularization with PCI or coronary artery bypass grafting is recommended.

It is worth noting that interventional PCI outcomes are generally poor. Acute PCI success rates are reported at most at 50-70% of cases.[3,4,6] Long-term success with PCI without complication was noted in only 30% of cases.[4] The two-year major adverse event rate with PCI was higher than usually expected at 10-17%, with a recurrent dissection rate of 13%.[4]

For long-term management of SCAD patients, antiplatelet drugs and beta blockers are recommended with continuation of other coronary artery disease risk factor medications specific to the patient.

The Bottom Line

SCAD is uncommon but should be high on the differential list for patients with acute coronary syndrome later found to have MINOCA, particularly middle-aged or younger women and a few young men. Conservative medical management is the preferred first approach unless significant ischemic sequelae appear. Bear in mind that PCI success is well below that of routine acute coronary syndrome PCI procedures. As Dr. Jacqueline Saw is one of the premiere experts on SCAD, I want to thank her for contributing personally to this editor's page and hope all labs will benefit from this discussion. ■

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