Spontaneous Coronary Artery Dissection: A Review of Pathogenesis, Presentations, Treatment, and Outcomes

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Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndromes and sudden cardiac death. The epidemiology, pathogenesis, and optimal approaches to diagnosis and management are poorly understood. Additionally, SCAD as a syndrome is commonly under-recognized and its prognosis is not well studied. Guidelines on management of SCAD have not yet been established. We present three cases of SCAD that varied in their clinical presentation and describe the different management strategies utilized. This is followed by a review of the clinical features, epidemiology, prognosis, and potential treatment strategies for patients presenting with SCAD.

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KEY WORDS

Spontaneous coronary artery dissection • Fibromuscular dysplasia

Spontaneous coronary artery dissection (SCAD) can present with a wide spectrum of clinical presentations, ranging from chest pain symptoms alone to ST-elevation myocardial infarction (STEMI), ventricular fibrillation, and sudden cardiac death (SCD).[1] Overall, it remains a relatively rare cause of acute coronary syndromes (ACS) and tends to affect the young, healthy population, with preponderance in women. SCAD has been more commonly observed in individuals with a history

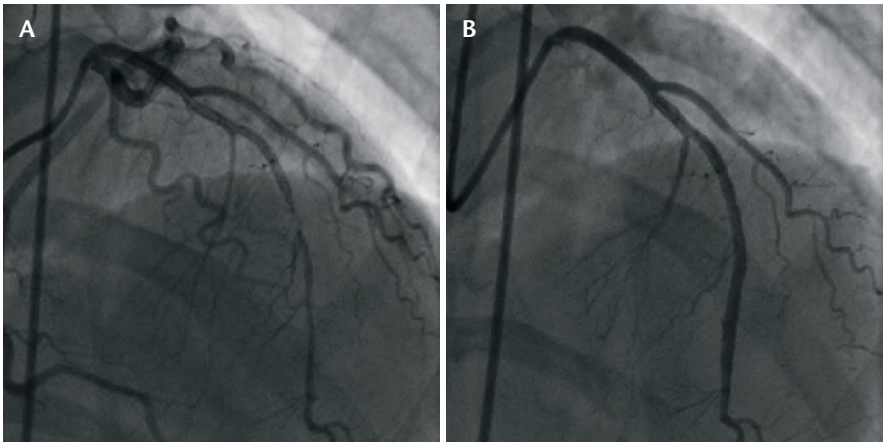

Figure 1. **(A)** Angiogram before PCI showing a 90% narrowing of the mid LAD with dissection. **(B)** Angiogram after PCI showing successful drug-eluting stent placement in the mid LAD. LAD, left anterior descending artery; PCI, percutaneous coronary intervention.

of atherosclerotic disease, and in peripartum and early postpartum women.[2] There are also several reported cases of idiopathic SCAD with no identifiable risk factors or evidence of atherosclerosis at presentation. We report three cases of SCAD in young, previously healthy individuals and review epidemiology, clinical features, and different management strategies for this group.

Case 1

A 43-year-old white woman with past medical history of hypertension presented to the emergency department with constant, nonradiating, substernal chest pain. She had presented to the emergency department a few days earlier with intermittent chest pain and was sent home after results of a brief cardiac workup were negative.

During evaluation, she denied any other associated symptoms. Her vital signs were stable. Pertinent physical examination findings included a grade 1/6 systolic ejection murmur at the right upper sternal border, with no jugular venous distention. She denied any home medications and reported only occasional alcohol use. She denied ever taking oral contraceptive pills and reported having regular periods since menarche at age 16. Her family history was positive for abdominal aortic aneurysm, atrial fibrillation, and hypertension in her father, and hypertension in a sister. An electrocardiogram (ECG) performed upon admission showed anteroapical ST segment changes consistent with ischemia or infarction.

On presentation to the emergency department, the chest pain had resolved. Her echocardiogram showed normal left ventricular size with a left ventricular ejection fraction (LVEF) of 60%, no wall motion abnormality, and grade I diastolic dysfunction. Pertinent laboratory results included a troponin elevation of 1.3 ng/mL, which subsequently increased to 2.89 ng/mL.

She was started on aspirin, high-dose atorvastatin, clopidogrel, carvedilol, amlodipine, and intravenous heparin infusion at 12 U/kg/h. As presentation was concerning for a non–ST-elevation myocardial infarction (NSTEMI), a coronary angiogram was performed. Left ventriculography revealed an LVEF of 50% with mild apical hypokinesis and left ventricular end-diastolic pressure of 16 mm Hg; coronary angiography found a patent left main artery, right coronary artery, and left circumflex artery, with 90% narrowing of the mid left anterior descending artery (LAD) with diminished Thrombolysis In Myocardial Infarction 1 flow (Figure 1). The lesion looked atherosclerotic initially; however, when the wire was threaded down, a false lumen was identified. A renal angiogram was also performed at this time, which showed 50% stenosis and fibromuscular dysplasia of the left renal artery (Figure 2). There was no angiographic evidence of atherosclerosis.

A drug-eluting stent was placed in the mid LAD and a balloon angioplasty was performed for the left renal fibromuscular dysplasia. Postprocedure, the patient was monitored for 24 hours and was sent home on aspirin, 81 mg once daily, clopidogrel, 75 mg once daily, pravastatin, 80 mg once nightly, carvedilol, 6.25 mg twice daily, and famotidine, 20 mg once daily, and instructed to follow up with an outpatient cardiologist. At her subsequent clinic visit, she remained free of chest pain. She was enrolled in cardiac rehabilitation after discharge, which she successfully completed.

Case 2

A 40-year-old white woman with no significant past medical history presented to the emergency department with sudden-onset chest pain that started while she was exercising. She described the pain as substernal, and pressure similar in quality. The patient mentioned a 1-week history of chest uneasiness and denied any home medications. On presentation the patient had ongoing severe chest pain (10/10 on a pain scale), and her ECG showed ST elevation in the anterior leads.

She was taken emergently for a left heart catheterization; left ventriculography showed an LVEF of

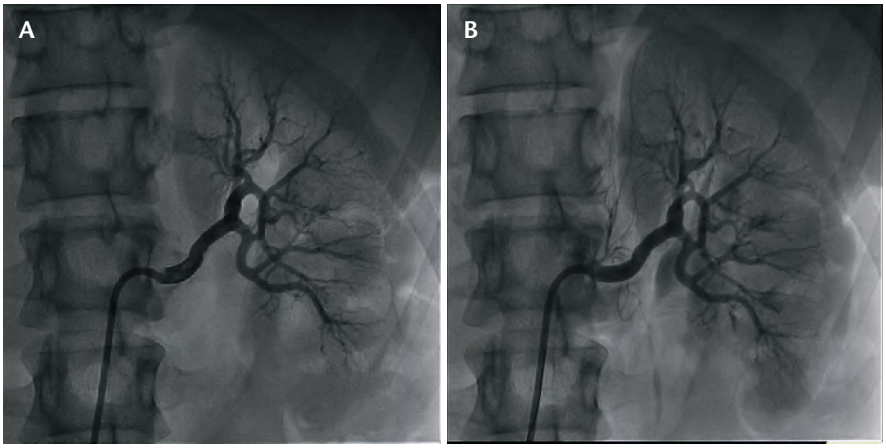

Figure 2. (A) Renal angiogram showing 50% stenosis and fibromuscular dysplasia of the left renal artery. **(B)** Renal angiogram after balloon angioplasty of fibromuscular dysplasia.

40% with a left ventricular end-diastolic pressure of 12 mm Hg and a hypokinetic distal anterior wall. Coronary angiography showed a right dominant heart with 10% stenosis of the right coronary artery (atherosclerotic lesion). There was a dissection of the left main coronary artery extending into the LAD, with an 80% obstruction of the distal LAD (Figure 3). This was confirmed by intravascular ultrasound (IVUS) (Figure 4). An intra-aortic balloon pump (IABP) was inserted and was placed on 1:1 augmentation.

Due to the location of the dissection, it was not deemed amenable to percutaneous coronary intervention (PCI), and a cardiothoracic surgeon was consulted. An emergent coronary artery bypass was performed. She received a left internal mammary artery bypass to the LAD and a saphenous vein graft to the obtuse marginal artery.

She did well postoperatively and the IABP pump was discontinued on the second postoperative day. The patient was sent home on aspirin, 81 mg once daily, carvedilol, 3.125 mg twice daily, and pravastatin, 40 mg once nightly, and instructed to follow-up with an outpatient cardiologist. At 1-year follow-up she was doing well, with complete recovery of her cardiac function, and no clinical limitation.

Case 3

A 22-year-old white man with past medical history of attention deficit

TABLE 1

Side-by-side Comparison of Clinical Presentation and Management of Patients

	Case 1	Case 2	Case 3
Age (y)	43	40	22
Sex	Female	Female	Male
Clinical presentation	Substernal chest pain	Substernal pressure like-chest pain	Chest pain with radiation to left arm and neck
ECG at presentation	Anteroapical ST-segment elevation	ST-segment elevation	No change
Initial troponin (ng/mL)	1.31	23.80	0.62
Diagnosis	LHC showing LAD dissection	LHC showing left main coronary dissection with LAD involvement	CTCA concerning for dissection with LHC showing proximal LAD dissection
Management	DES in mid LAD	IABP placement and CABG	Medical management with aspirin, carvedilol, clopidogrel, atorvastatin, and ramipril
Outcome	Discharged postoperatively	Discharged postoperatively	Discharged on medical therapy

CABG, coronary artery bypass graft; CTCA, computed tomography coronary angiogram; ECG, electrocardiogram; DES, drug-eluting stent; IABP, intra-aortic balloon pump; LAD, left anterior descending artery; LHC, left heart catheterization.

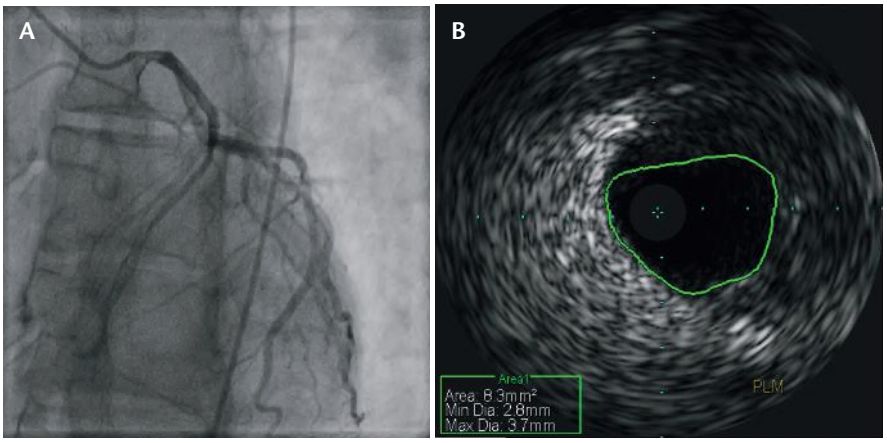

Figure 3. **(A)** Dissection of the left main coronary, artery extending into the left anterior descending artery, with an 80% obstruction of the distal left anterior descending artery. **(B)** Dissection of the left main coronary artery confirmed via intravascular ultrasound.

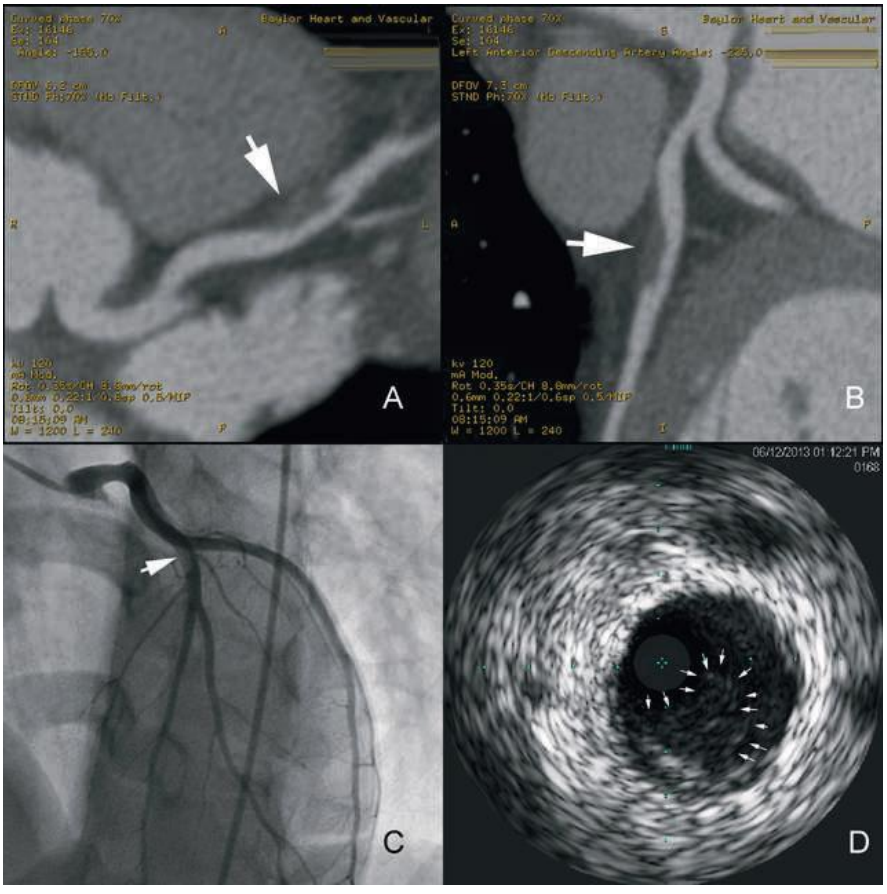

Figure 4. **(A, B)** Computed tomography coronary angiogram demonstrating an area of decreased density in the proximal LAD concerning for dissection and intramural hematoma (*arrows*). **(C)** Coronary angiogram with hazy area in the proximal LAD, concerning for dissection with thrombus (*arrow*). **(D)** Intravascular ultrasound confirming a proximal LAD dissection (*arrows*). LAD, left anterior descending coronary artery.

hyperactivity disorder (ADHD) presented to the emergency department with complaints of substernal, pressure-like chest pain (8/10 on a pain scale) with radiation to the left arm and neck that started while he was bicycling. It was associated with shortness of breath, nausea, and vomiting. However, the pain had resolved by the time he got to the emergency department. His only home medication was lisdexamfetamine, which he had been taking for approximately 5 months prior to presentation.

His social history was negative for drug use, and drug screen results were negative for illicit substances (but positive for amphetamines given his prescribed medication). His family history was positive for myocardial infarction in his father at age 45, with subsequent four-vessel coronary artery bypass graft (CABG) many years later.

The patient's presenting ECG result was negative for any acute changes and results of an initial troponin check were negative. An echocardiogram showed septal hypokinesis with a normal ejection fraction. The second troponin concentration was 2.06 ng/ mL 3 hours later, and ultimately peaked at 26 ng/mL. The patient was started on intravenous heparin at 12 U/kg/h, aspirin, 81 mg once daily, carvedilol, 3.125 mg twice daily, and clopidogrel, 75 mg once daily, and was evaluated with a gated computed tomography coronary angiogram (CTCA), given the thought that significant coronary pathology was unlikely. Surprisingly, the CTCA showed a density within the proximal LAD that was concerning for a dissection with intramural hematoma. He was subsequently taken for a left heart catheterization, which showed a right dominant heart with patent left main coronary artery, circumflex artery, and right coronary artery; IVUS confirmed dissection, with a hematoma present in the wall of the proximal LAD. There was no evidence on coronary computed tomography angiography or coronary angiography of atherosclerosis (eg, absence of calcification, irregular lesions).

No PCI was performed, and the patient was started on aggressive medical management. The patient remained free of chest pain and was observed for 24 hours after catheterization. He was discharged on aspirin, 81 mg once daily, carvedilol, 3.125 mg twice daily, clopidogrel,

75 mg once daily, atorvastatin, 10 mg once nightly, and ramipril, 2.5 mg once daily, and was referred for cardiac rehabilitation and follow-up at an outpatient cardiology facility. At 1 year, he was doing well and was exercising regularly with no symptoms. He was taken off his ADHD medication, and was not started on any similar medications. Follow-up CTCA at 6 months showed complete resolution of both the dissection and the hematoma. An overview and side-by-side comparison of the three patients presented and the management strategies used is outlined in Table 1.

Discussion

SCAD is defined as a nontraumatic and noniatrogenic separation of a coronary arterial wall by intramural hemorrhage resulting in a false lumen with or without an intimal tear. Separation can occur between the intima and media or between the media and adventitia. Often, the greatest length of propagation is in the central media. The formation of an intramural hematoma leads to the compression of the arterial lumen, which, in turn, compromises antegrade blood flow.[3] Exit through the adventitia is rare but can lead to epicardial hematoma or pericardial tamponade.

SCAD is predominantly seen in women, with some studies reporting 80% of cases in women. The mean age at presentation is approximately 35 to 40 years, and it typically presents in the absence of coronary risk factors or atherosclerosis.[2] SCAD is thought to be an under-diagnosed cause of acute myocardial infarctions and SCD.

Retrospective studies have shown that SCAD is detected in 0.07% to 1.1% of all coronary angiograms performed.[1] It is estimated to account for 0.1% to 4% of all ACS and can be responsible for up to 25% of ACS in women under age 50 years.[3] In a retrospective study done by Tweet and colleagues,[1] 87 patients with SCAD were identified, of whom 44% presented with NSTEMI, 49% presented with STEMI, and 7% presented with unstable angina. The LAD is the most commonly affected vessel, with multivessel coronary dissection seen in 23% of patients.[1] In another study, roughly 8% of patients had two separate dissected coronary arteries and 4% had extension of dissection involving more than 1 coronary artery.[4] In the study published by Saw and associates,[4] 40% of SCAD affected the LAD, 34% affected the circumflex artery or its branches, and 32% affected the right coronary artery or its branches. Multivessel disease is seen more commonly in women.[2] In one series, men had right coronary artery dissection in 73% of cases, whereas women had left coronary system dissection in 88% of cases.[5]

There are two proposed mechanisms of SCAD. One is initiation of a medial dissection and hemorrhage by an intimal tear and creation of a false lumen. The second is intramedial hemorrhage without an intimal tear leading to medial dissection, possibly caused by rupture of vasa vasorum.[3] On coronary angiography, intramural hematoma compression can mimic atherosclerosis, which can lead to under-diagnosis of SCAD.[1] Adjunctive imaging with IVUS or optical coherence tomography can help make a definitive diagnosis.

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There are several etiologies of SCAD. Often, there is a predisposing arteriopathy, which can be atherosclerotic or nonatherosclerotic in origin. Peripartum, postpartum, or antepartum status, connective tissue disease such as Marfan syndrome, systemic inflammatory diseases such as systemic lupus erythematosus, vasculitides such as polyarteritis nodosa, inflammatory bowel diseases, and coronary spasms are some examples of nonatherosclerotic etiologies.[6] Physical, emotional, or drug-induced stress can also be a trigger for SCAD. Drugs such as cocaine, oral contraceptives, and cyclosporine have been documented to increase the risk of SCAD.[5] There are isolated reports of methamphetamines causing SCAD.[6] Extreme exertions, such as vigorous exercise or bicycling at SCAD onset, were more frequent in men.[7]

Atherosclerotic causes of coronary artery dissections have been variably included in prior studies evaluating incidence of SCAD. However, it seems more likely that nonatherosclerotic SCAD represents a distinct clinical entity and should be evaluated separately from dissection secondary to plaque disruption.[1]

There have been several reports of association between SCAD and fibromuscular dysplasia (FMD). In the study by Saw and associates,[4] FMD was identified in at least one noncoronary vessel either in

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renal iliac, or cerebrovascular territory in 86% of patients. FMD is a segmental nonatherosclerotic, noninflammatory vasculopathy, which usually affects small- to medium-sized arteries. FMD can affect intima, media, or adventitia.[8] The exact prevalence of FMD is unknown; however, in one study, renal artery FMD was identified in 3.8% of renal donors.[8] There are several types of FMD and are classified as intimal fibroplasia, medial fibroplasia, medial hyperplasia, perimedial fibroplasia, and adventitial fibroplasia. Medial fibroplasia is most common (80%-90% of FMD) and appears as a "string-of-beads" on imaging.[8] Discovery of FMD should alert the clinician to the risk of multiple vascular dissections in the future.

There are no prospective randomized data on the management of SCAD, given its sporadic and low rates of occurrence. The lack of firm recommendations on treatment of SCAD makes therapeutic decisions controversial. Most clinical decisions are currently based on personal experience and literature reports.[6] Typically, therapy depends on several factors, such as the site of dissection, single- versus multivessel involvement, and hemodynamic status.[2] Early diagnosis of SCAD is essential, as it can potentially avoid use of unnecessary procedures and pharmacologic treatments, and potentially avoid complications.

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The decision to proceed with medical therapy versus revascularization is dependent on patients' clinical status and the affected coronary segments.[3] In patients who are hemodynamically stable and do not have severe chest pain, a conservative approach with medical management is generally preferred. Conversely, patients with ST elevation or hemodynamic instability should undergo emergent PCI, especially when it is suspected that one of the major arteries is compromised. Patients with left main, proximal LAD, circumflex, or right coronary artery disease should be intervened upon if possible.[3,4]

Early mortality appears to be low, regardless of the initial treatment modality, but PCI is associated with higher rates of complications and procedural failure.

However, Tweet and colleagues[1] reported a technical success rate of only 65% in patients undergoing PCI. Early mortality appears to be low, regardless of the initial treatment modality, but PCI is associated with higher rates of complications and procedural failure.[1] One of the reasons for the high procedural failure rate is the challenge of advancing the guidewire into the true distal lumen.[3] Commonly, dissection can involve distal segments, which are too small to stent. In light of risk of complications, Saw and colleagues[3] recommend reserving PCI only for patients with stentable lesions in the setting of active chest pain or hemodynamic instability. We were able to utilize an IABP for hemodynamic support in one of our patients during the acute phase. For more extensive dissections, patients should be evaluated for CABG surgery.

There is debate regarding appropriate medical therapy for patients with SCAD. It is not clear if standard ACS medical management is beneficial for patients with SCAD.[3] However, given the benefits of aspirin in ACS and its relatively low-risk side-effect profile, it is reasonable to proceed with treatment with aspirin. The use of dual antiplatelet therapy has not been studied. It has been hypothesized to benefit patients without stents to reduce false lumen thrombus burden, thus reducing true lumen compression. The use of glycoprotein IIb/IIIa inhibitors has not been evaluated but is generally avoided due to the potential risk of extending dissection.[5] The use of heparin has not been extensively evaluated either. Heparin can increase the risk of extending the dissection but can also provide benefit by resolving the commonly observed overlying thrombus. It is reasonable to treat these patients like ACS patients and give heparin until a diagnosis of SCAD is made via angiography. Patients are typically started on high-dose statin drugs as well, but their benefit has not been evaluated. β-blockers have been routinely used in aortic dissection and are recommended for SCAD as well, because they reduce arterial shear stress. Angiotensin-converting enzyme inhibitors are the cornerstone of postmyocardial infarction treatment, particularly in patients with depressed LVEF; they are used in the treatment of SCAD as well.[3] There are case reports in which SCAD has been seen with coronary artery spasm. If vasomotion

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is suspected, calcium channel blockers may be used.[9]

Intense exercises with increased cardiocirculatory stresses and shear force on the coronary wall has been reported to precipitate SCAD in patients with or without predisposing conditions.[4] In a recent review by Saw and colleagues,[4] 2 of the 50 patients evaluated reported vigorous exercise prior to SCAD. There are limited data regarding the role of cardiac rehabilitation and exercise after an episode of SCAD. It has been observed that many physicians are hesitant in recommending resumption of regular physical activity after SCAD and tend to discourage patients from lifting more than 5 lbs. Silber and colleagues[10] recently reviewed data on patients with SCAD undergoing a cardiac rehabilitation program. Nine patients underwent an average of 28 cardiac rehabilitation sessions, which were well tolerated. Patients did not report cardiac symptoms and no adverse symptoms were reported during exercise testing or training. Patients showed an increase in peak oxygen uptake on average of 18%, with an increase in 6-minute walk distance by 22%.[10] A recent study by Chou and coworkers[11] designed a SCAD-specific cardiac rehabilitation protocol in which the exercise component was altered to reduce arterial shear stress. At the start, target exercise heart rate was aimed at 50% to 70% of the heart rate capacity, and resistance training focused more on increasing muscle strength using repetitions with lighter weights rather than increasing weight lifted. The study showed improvement in exercise duration with no major adverse events during the program. These studies showed that cardiac rehabilitation can be implemented safely in patients with SCAD; however, further research is needed to determine the role of cardiac rehabilitation in SCAD.

SCAD in the setting of an increased estrogen or progesterone state (such as pregnancy or use of oral contraceptives) has been documented in case reports, although the exact mechanism as to why this occurs is less clear. Possible mechanisms include increased arterial wall stress and impaired endovascular integrity during pregnancy and the peripartum period. Additionally, hormonally induced endovascular changes may contribute to a propensity for SCAD.[12] Due to the infrequent nature of SCAD with oral contraceptive use, there are no trials to determine the resumption of oral contraceptives in patients with SCAD secondary to pregnancy or oral contraceptive use. However, previous reports have recommended that further exogenous use of estrogen should be avoided in such patients by switching to nonhormonal contraception.[13]

The prognosis of SCAD patients is fairly uncertain. Kemenini and colleagues noted that up to 50% of patients with SCAD developed a recurrent dissection within the same or another vessel within 2 months.[7] However, it should be noted that the rate of recurrence likely varies with different management strategies. Such a high rate of recurrence has not been reported in other smaller studies. Tweet and colleagues[1] followed 87 patients for approximately 47 months, during which 17% of the patients experienced a SCAD recurrence. Based on Kaplan-Meier curve, an estimated 10-year SCAD recurrence rate was approximately 29.4%. Of note, in this study the recurrence was exclusively in women.[1] Although long-term survival after a SCAD episode is favorable, the unpredictability in timing and variable risk of recurrence points toward the need for close long-term follow-up. Recurrence can be monitored invasively with repeat left heart catheterization or CTCA. Alfonso and coworkers[14] followed 45 patients over a 6-year period to assess the long-term clinical outcomes of patients with SCAD. At 2-year follow-up, only 3 of the 45 patients presented with adverse events, and 2 of those patients required revascularization; 54% of the patients showing images of SCAD at discharge showed spontaneous resolution at follow-up.[14] In patients being treated with medical management of SCAD, it appears reasonable to perform noninvasive imaging with CTCA or use an invasive modality such as IVUS at 1- to 2-year follow-up to determine resolution of dissection. However, further data are needed to find the ideal time for follow-up with invasive or noninvasive imaging. In the event of recurrent chest pain in patients being managed medically, it is recommended to attempt revascularization if possible.[14]

Conclusions

SCAD is an uncommon cause of ACS. It is likely under-diagnosed, given the difficulty in differentiation from atherosclerosis-generated ACS. There is a recently reported association with FMD, which, when found, can alert the clinician to vascular dissection risk elsewhere in the body (aorta, and femoral, coronary, and carotid arteries). Because a large portion of women with SCAD have noncoronary FMD, further studies are needed to determine the utility of screening for FMD in women with a history of SCAD. The role of oral hormonal therapy and changes in hormonal status (pregnancy and menopause) on the development of this syndrome are worthy of further study. The use of PCI or referral for CABG

depends on clinical status and coronary anatomy. Most patients do well when they are treated conservatively with medical treatment, which predominantly involves antiplatelet therapy, angiotensin-converting enzyme inhibitors, and β-blockers. Revascularization (whether through PCI or surgery) is typically used as a "bail out" procedure in patients who have unstable or ongoing symptoms and cannot be treated medically. Because coronary intervention commonly provides incomplete revascularization, especially for side branches, follow-up testing for myocardial ischemia and management with anti-ischemic therapy is prudent. Further multicenter data collection is needed to determine specific guidelines for treatment of SCAD. ■

The authors report no real or apparent conflicts of interest.

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MAIN POINTS

- Spontaneous coronary artery dissection (SCAD) can present with a wide spectrum of clinical presentations, ranging from chest pain symptoms alone to ST-elevation myocardial infarction, ventricular fibrillation, and sudden cardiac death. Overall, it remains a relatively rare cause of acute coronary syndromes.
- On coronary angiography, intramural hematoma compression can mimic atherosclerosis, which can lead to under-diagnosis of SCAD. Adjunctive imaging with intravascular ultrasound or optical coherence tomography can help make a definitive diagnosis.
- The lack of firm recommendations on treatment of SCAD makes therapeutic decisions controversial. Typically, therapy depends on several factors, such as the site of dissection, single- versus multivessel involvement, and hemodynamic status. Early diagnosis of SCAD is essential, as it can potentially avoid use of unnecessary procedures and pharmacologic treatments, and potentially avoid complications.
- In patients who are hemodynamically stable and do not have severe chest pain, a conservative approach with medical management is generally preferred. Conversely, patients with ST elevation or hemodynamic instability should undergo emergent percutaneous coronary intervention (PCI), especially when it is suspected that one of the major arteries is compromised.
- The use of PCI or referral for coronary artery bypass graft depends on clinical status and coronary anatomy. Most patients do well when they are treated conservatively and medically, which predominantly involves antiplatelet therapy, angiotensin-converting enzyme inhibitors, and β-blockers.