Spontaneous Coronary Artery Dissection: Insights From Histology and Optical Coherence Tomography

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Spontaneous coronary artery dissection (SCAD) is a well-known but infrequent cause of acute coronary syndrome (ACS), and often goes unrecognized. Although management of SCAD is, at times, controversial, when a patient presents with ACS, percutaneous coronary intervention (PCI) is frequently necessary. We present a patient with ST-segment elevation myocardial infarction (STEMI) with SCAD that illustrates two important points: use of intracoronary optical coherence tomography to guide PCI, and histologic assessment to provide a unique insight into the etiology of SCAD. Following the case, we briefly review the important aspects of the pathophysiology, epidemiology, diagnosis, and interventional management of SCAD.

[Rev Cardiovasc Med. *2017;18(1):44-52 doi: 10.3909/ricm0856*]

KEY WORDS

Coronary • Intervention • Myocardial infarction • Optical coherence tomography • STEMI

Spontaneous coronary artery dissection (SCAD) is a condition resulting from an increase in shear stress that causes a tear in the intimal lining.[1] SCAD is noted in 0.1% to 1.1% of patients referred for coronary angiogram.[2,3] However, this may be an underrepresentation of the true prevalence of SCAD, as the diagnosis may be missed at angiography and some patients with self-limiting symptoms may not undergo angiography. Diagnosis is challenging with coronary angiography alone, and management remains controversial. We present a unique case highlighting the use of optical coherence tomography (OCT) to confirm the diagnosis and also to aid in the management of a patient

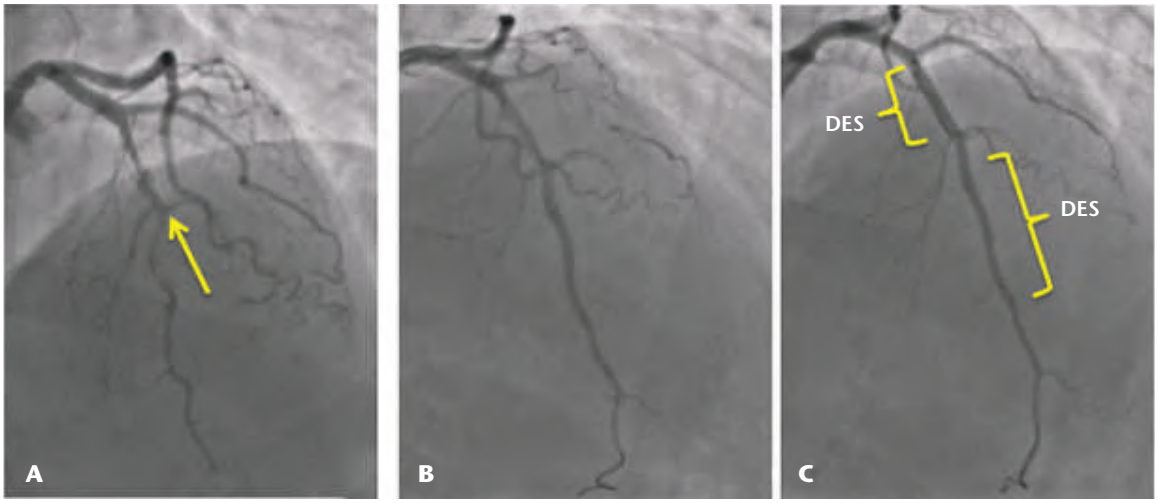

Figure 1. Initial angiographic image showing mid left anterior descending (LAD) coronary dissection with thrombus with limited compromise of the second diagonal branch (*arrow*) and thrombolysis in myocardial infarction (TIMI) 2 flow. TIMI 3 flow is restored post aspiration thrombectomy with residual visible dissection. Excellent angiographic outcome after deployment of stents in both the proximal and distal LAD (*brackets*).

presenting with ST-segment elevation myocardial infarction (STEMI) secondary to SCAD. In addition, aspirated plaque and thrombus provide histologic findings, which lend insight into the etiology of SCAD in this case. Although OCT has been reported to facilitate the diagnosis and management of SCAD,[4-9] the experience with STEMI is limited to fewer than 15 patients[4,8] and there are no reported histologic findings from a living patient.

Presentation

A 49-year-old perimenopausal Asian woman with a history of hypothyroidism and dyslipidemia was transferred from a non-percutaneous coronary intervention (CPI) capable hospital with electrocardiographic changes consistent with an anterolateral STEMI. Her chest pain began 5 hours prior to her initial presentation and was preceded by a 1-week history of flu-like symptoms. Her initial troponin level was 0.4 mg/dL but had risen to 38 mg/dL at the time of transfer. Her history, physical examination, and laboratory findings were otherwise unremarkable.

Emergent cardiac catheterization (Figure 1) revealed a subtotal occlusion with contrast staining and a string-like lucency in the mid and distal left anterior descending coronary artery (LAD) after the takeoff of a large diagonal branch (Figure 1A), consistent with SCAD. Once a 6F guiding catheter was engaged in the left main, a guide wire was negotiated into the true lumen of the distal LAD. Thrombus and tissue-like particles were aspirated using an aspiration catheter, and thrombolysis in myocardial infarction (TIMI) 3 flow was established (Figure 1B). During aspiration, small pieces of tissue were removed in addition to the thrombus, and were subsequently sent for histologic examination (Figure 2).

A catheter was advanced into the LAD to determine the extent of the spiral dissection (Figure 3) which extended from the proximal diagonal across the large second diagonal into the distal vessel. A 2.75 × 38 mm drug-eluting stent (DES) was deployed first to cover the distal edge of the dissection. The entry tear was located across from the second diagonal branch, with retrograde extension into the proximal LAD and antegrade spiral dissection into the distal LAD. In order to spare the large second diagonal branch, we covered the flow-limiting proximal lesion (proximal LAD and inciting tear) with a 3.5 × 20 mm DES using length and diameter dimensions obtained from the OCT images. The OCT catheter was reinserted to confirm adequate stent apposition, expansion of the stented segments, and full coverage of dissection. A final angiogram revealed excellent angiographic results with TIMI-3 flow (Figure 1C). Postprocedure,

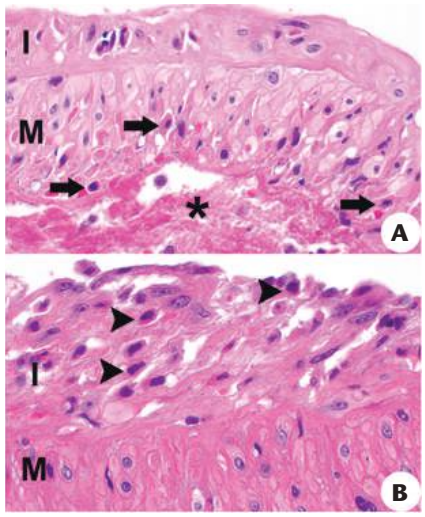

Figure 2. Histologic sections of aspirated wall tissue. Necrosis is visible in the medial layer of the arterial wall. Inflammatory cells infiltrate the intimal layer of the arterial wall. I, tunica intima; M, tunica media. *Necrosis. *Arrows* = pyknotic cells with condensed cytoplasm in the tunica media. *Arrowheads* = inflammatory cells in the tunica intima

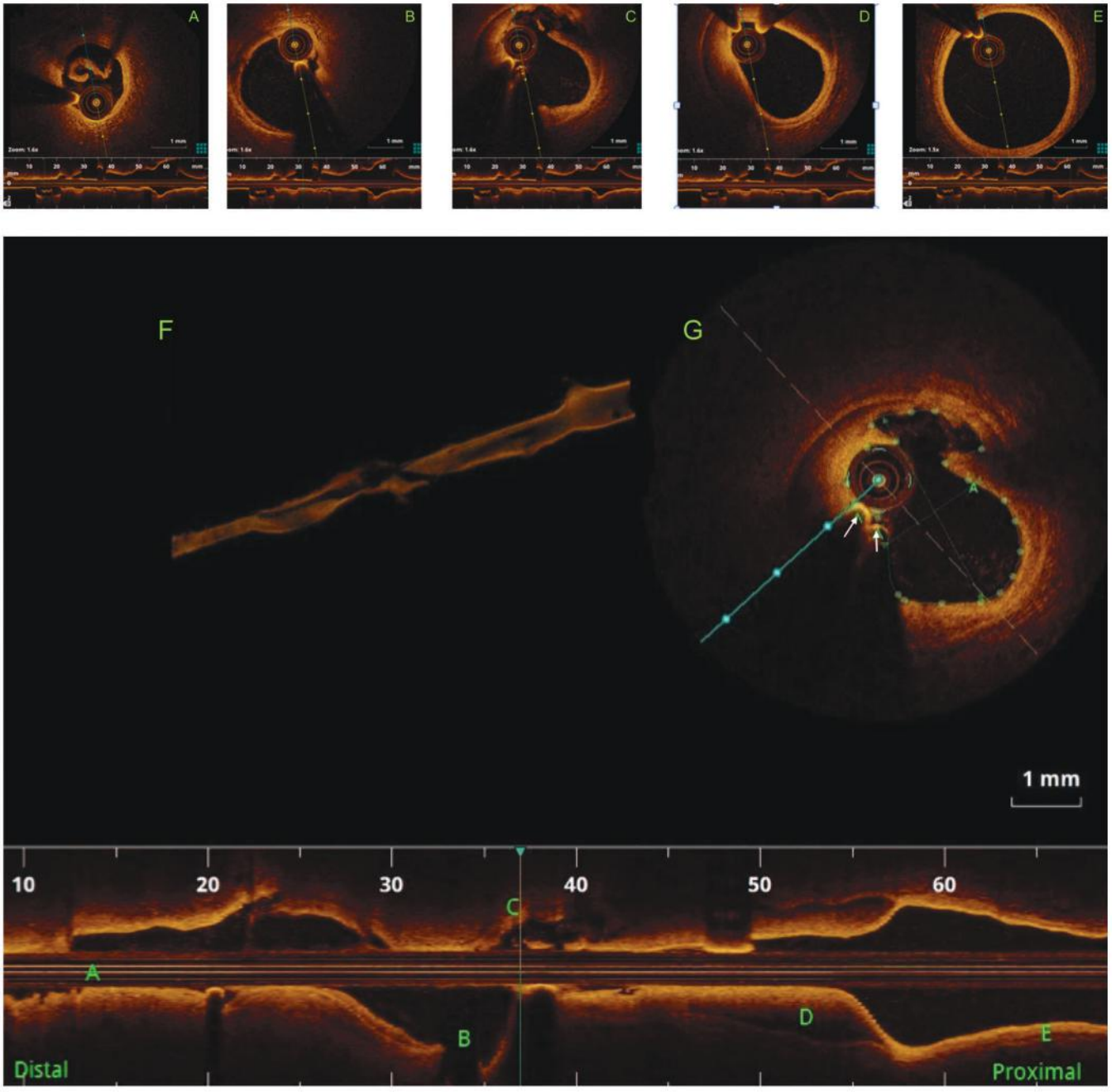

Figure 3. Longitudinal and corresponding axial displays are marked. Dissection flap in distal left anterior descending artery (LAD). Vessel near the second diagonal branch showing no dissection; thus no stent was placed here. Primary tear in mid LAD. Mid portion of the LAD showing the retrograde extension of dissection. Image demonstrates a complete separation of intima/media vessel layer with hematoma formation. Proximal LAD showing normal, healthy vessel wall. Three-dimensional luminal reconstruction of the LAD showing spiral dissection. Cross-section of the mid LAD showing primary tear. *White arrows* indicate the LAD and second diagonal wires. Longitudinal display of the pullback of patient's LAD prior to stent placement.

the patient had an ejection fraction of 40% with anterior wall hypokinesis. Pertinent laboratory findings included mild elevation in her erythrocyte sedimentation rate (17 mm/h), and a normal rheumatologic evaluation including C-reactive protein.

At 6 months, her echocardiogram revealed a left ventricular ejection fraction of 60% with normal wall motion. She remains asymptomatic on dual antiplatelet therapy and a statin 2 years after STEMI.

Discussion

SCAD was initially suspected based on clinical presentation and angiographic appearance; however, coronary angiography is often inadequate to visualize the extent of dissection[1] and OCT was a useful tool to evaluate the vascular wall.[8] The management of SCAD continues to be controversial. SCAD may be managed conservatively in stable patients using antiplatelet, antithrombotic, and antianginal therapy, with reports

of complete healing and excellent prognosis.[10] However, patients with STEMI and ongoing symptoms and/or hemodynamic compromise may require PCI. Primary PCI facilitated by OCT led to an excellent short- and long-term outcome in our patient.

OCT Determination of SCAD

Key features of the OCT on distal to proximal pullback include a spiral-type dissection beyond the primary tear distally (Figure 3A); a dissection-free portion of the mid LAD near the takeoff of the second diagonal branch (Figure 3B); a primary (entry) tear starting in the mid LAD extending across a large diagonal branch (Figure 3C); a complete separation of the intima and media layer with a large hematoma in the proximal LAD (Figure 3D); and healthy proximal LAD without atherosclerosis (Figure 3E). The complete preintervention pullback is seen in Figure 3F and the video file of the pullback is seen in Figure 3G.

The OCT results led to the decision to place two separate stents—a distal stent to cover the flow-limiting dissection and a proximal stent to cover the hematoma and entry tear. Evidence suggests stenting the "entry-door" leads to positive outcomes in SCAD by helping stabilize the coronary artery and facilitate natural healing of the remaining dissection.[11] The intentional short gap in between the two stents allowed unobstructed flow into the unaffected second diagonal branch and prevented "jailing" of the branch (an area relatively unaffected by the dissection). The use of OCT was integral to the precise placement of the two stents.

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Histopathology of Intima-Media Fragment

During the aspiration procedure, small tissue pieces were removed in addition to the thrombus. Histologic evaluation disclosed the layers of arterial wall with necrosis of the tunica media (Figure 2A), and polymorphonuclear and mononuclear leukocytes transmigrating through the tunica intima (Figure 2B) without evidence of atherosclerosis, vasculitis, cystic degeneration, or eosinophilic inflammation.

This unique histopathology may provide some insight into this patient's clinical presentation. Microscopic examination of SCAD has been exclusively restricted to autopsy material, and the most frequently cited histologic finding is a mixed, eosinophil-predominant inflammatory infiltrate in the adventitia, with minimal inflammation of the intimal and medial layers.[12]

Although the adventitial layer was unavailable, the finding of an inflammatory infiltrate in the intima is atypical in the reported cases of SCAD seen at autopsy. This patient's viral prodrome suggests that acute inflammation may have contributed to weakening of the intima and medial layers prior to the SCAD.

Review

Pathophysiology

Symptoms in SCAD result from compression of the arterial lumen via an intramural hematoma, which compromises blood flow, leading to myocardial ischemia or infarction.[1,13] This "primary" type of dissection is spontaneous in nature, meaning that it is not associated with iatrogenic or traumatic events.

SCAD is believed to result from two different mechanisms. One mechanism (Figure 4B and 4B') occurs via the accumulation of eosinophilic infiltrates and subsequent rupture of the vasa vasorum. This intramural bleeding creates an intramural hematoma that may lead to subsequent dissection of the intimal layer. It should be noted that an intimal tear might not be present in this type of SCAD. SCAD without an intimal entry tear may present as a nonatherosclerotic, healthy-appearing artery on angiogram. A second mechanism (Figure 4C and 4C') is an intimal tear that allows blood from the lumen to enter the subintimal plane and form an intramural hematoma or a false lumen.[1,14,15] Imaging modalities such as OCT can be helpful in the identification of the entry tear and extent of the dissection as noted in this case.[16]

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Epidemiology

SCAD has been reported in 0.1% to 1.1% of patients undergoing coronary angiography.[17] Due to inadequate imaging, unfamiliarity with SCAD's hallmarks, and the wide variety of symptoms on presentation, SCAD is frequently misdiagnosed.[17] Additionally, a high correlation between sudden cardiac death and SCAD may contribute to underestimation of the true incidence.[18,19] A SCAD registry is in development to provide more information about its true prevalence and natural history.[20]

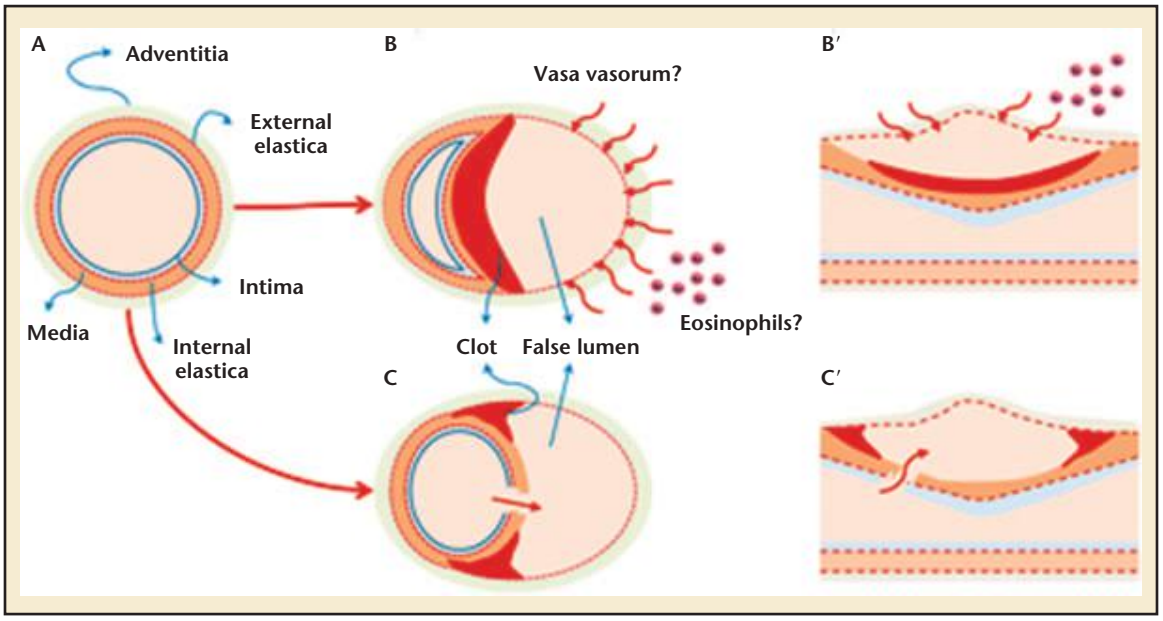

Figure 4. Two proposed mechanisms of spontaneous coronary artery dissection. **(A)** Cross-section of a healthy coronary artery. **(B)** Cross-sectional and longitudinal **(B′)** views: rupture of vasa vasorum related to eosinophilic infiltrates causing an intramural hematoma without an intimal entry door tear. **(C)** Cross-sectional and longitudinal **(C′)**: separation of the intimal layer creating a subintimal hematoma and compression of the true lumen. Adapted from Vrints CJ.[1]

From a database of 5 years of angiograms and over 11,000 patients, Vanzetto and colleagues[3] noted that the average age of a patient with SCAD is 30 to 45 years old.[21,22] SCAD affects more women than men (70% vs 30%), and women present earlier than men do, on average (41 vs 45 y). SCAD in women occurs in a bimodal distribution: 30% of women present in the peripartum period and the remainder a decade later (presumably with underlying atherosclerotic disease, hormonal shifts due to menopause, or immune-mediated disease).[21,23-25] This patient falls into the latter half of the bimodal curve; therefore, it is possible that hormonal shifts related to the patient's perimenopausal state predisposed her to SCAD.

SCAD has multiple risk factors, the most common being atherosclerotic plaque, implicated in 8.5% to 28% of SCAD.[16,23] Underlying atherosclerosis with plaque rupture leads to a disrupted subintimal space (Figure 4C and 4C') providing a conduit for blood to enter and create an intramural hematoma that may propagate and create a false lumen. Other risk factors include perimenopausal hormonal shifts, oral contraceptive use, presence of fibromuscular dysplasia, connective tissue disorders, and pregnancy.[26,27]

Based on data from small registries and case series with at least five patients, SCAD occurs most frequently in the LAD (60%), followed by the right coronary (26%), circumflex (19%), and left main artery (9%)—this holds true for both men and women.[21,27,28] SCAD affecting multiple vessels occurs in approximately 20% of cases, with women having a higher propensity of two- and three-vessel presentations.[16]

Our patient had SCAD in her LAD. Her etiology remains unclear, but, based on the aspirated segment of vessel wall, our patient had an underlying inflammatory process in her tunica intima (a form of vasculitis), which may have predisposed her to SCAD. Inflammation of the intimal vascular layer is not reported in the literature.

Diagnosis of SCAD

Coronary angiography has historically been the diagnostic test of choice. The false lumen fills at a slower rate than the true lumen, allowing accumulation of dye within the subintimal plane long after contrast washout in the true lumen of the vessel. Contrast injection may also reveal haziness that correlates with the presence of thrombus and/or intramural hematoma. Coronary computed tomography angiography may be an effective noninvasive diagnostic tool.[29]

When there is no visible intimal flap, angiographic diagnosis may be challenging. Extensive hematoma may be present and may completely fill a false lumen that does not communicate with the true lumen, and hence, does not opacify with contrast injections. Misdiagnosis of SCAD is common and the angiographic appearance may mimic that of a vessel with stenosis due to a routine atherosclerotic plaque or spasm. Angiography in SCAD must be performed with

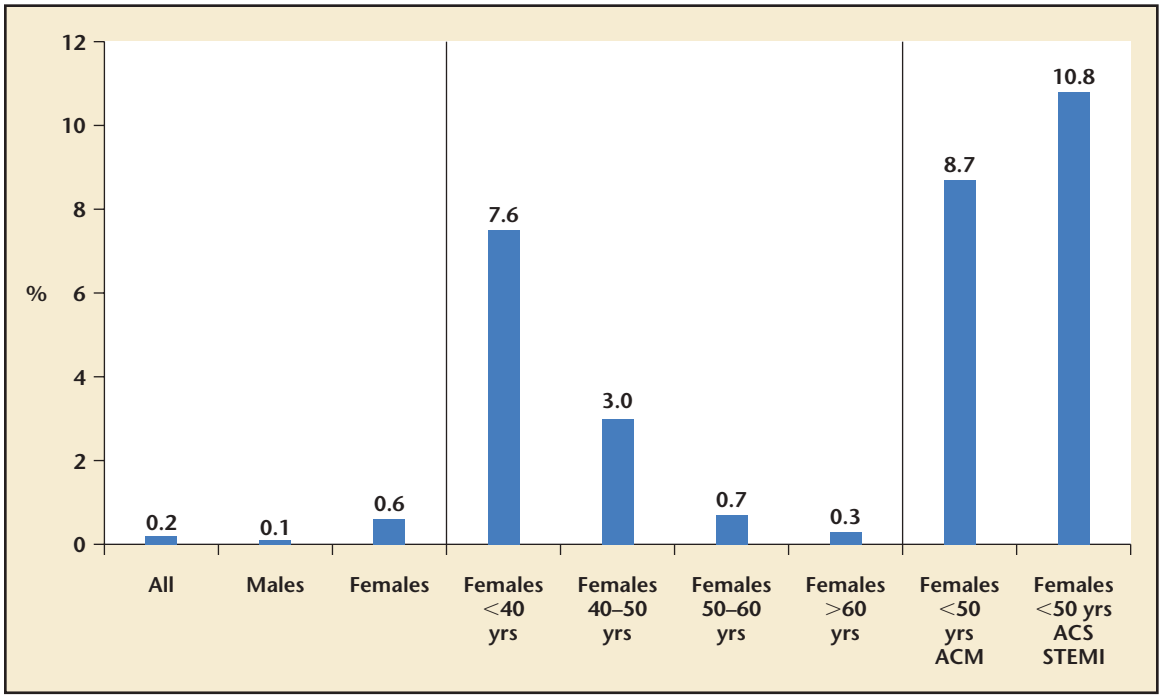

Figure 5. Prevalence of spontaneous coronary artery dissection (SCAD): a possible bimodal distribution in women. According to data from Vanzetto G et al[3] from a catheterization laboratory database in France compiled over 5 years including 11,605 angiograms and over 20 cases of SCAD.

caution so as to not extend the dissection with contrast injections. This has been been reported in up to 25% of patients.[30]

Intravascular Imaging

Because angiography in SCAD patients can be unreliable, intravascular imaging, including both intravascular ultrasound (IVUS) and OCT, have been reported to help diagnose and manage SCAD. IVUS provides deep visual penetration into the vessel wall and allows for full visualization of the intramural hematoma. IVUS allows visualization of the dissection flap and thrombus, but is limited by its contrast and gray scale resolution, and the primary tear may be difficult to identify.

OCT has increased spatial resolution (15 μm) and superior contrast resolution that allows easier visualization of the false lumen, true lumen, intimal tear, and intramural hematoma.[4,7] The tradeoff with OCT as compared with IVUS is the depth of visualization, which may lead to an insufficient view of the full intramural hematoma. Additionally, OCT requires contrast injections for blood clearance during acquisition, which carries a risk of propagation of the dissection plane. There have been 24 cases published to date using OCT; the majority of the cases presented with acute coronary syndrome (ACS) and were treated with PCI (Table 1).[4,7,8,31-34]

Few publications have compared IVUS and OCT head-to-head in SCAD patients. In the largest, Paulo and colleagues[7] performed OCT and IVUS on eight SCAD patients presenting with ACS and reported contrasting features of OCT and IVUS. On their angiograms, all patients had diffuse, lengthy lesions; only one angiogram showed a radiolucent flap. Both intravascular imaging modalities clearly depicted the true and false lumens. However, OCT was superior to IVUS in the identification of intimal ruptures and intraluminal thrombi. OCT depicted the false lumen/intramural hematoma very well, but its full extension could not be measured in some areas because of residual blood swirl, or insufficient penetration. On IVUS, the lumen-intimal interface was not as sharply delineated, but the images allowed a complete estimation of the false lumen area because of its depth of penetration. Residual hematomas were well detected with both techniques, but stent features including malapposition of struts were more clearly visualized by OCT.

In March 2012, Alfonso and associates[32] sought to assess the diagnostic value of OCT in patients with suspected SCAD. They performed OCT in 17 consecutive patients with a clinical and angiographic suspicion of SCAD from 5002 patients undergoing coronary angiography. In six of those patients, OCT revealed atherosclerotic plaques and/or intracoronary thrombus; not SCAD. In 11 patients (age 48 ± 9 y, 9 women), OCT confirmed the presence of SCAD, with

TABLE 1

Reported Cases of Optical Coherence Tomography in Acute Coronary Syndrome Patients With Spontaneous Coronary Artery Dissection

	SCAD	Women, %	Age, Y (SD)	STEMI, %
Ishibashi K et al[33]	1	1 (100)	40 (NR)	0
Alfonso F et al[8]	1	1 (100)	50 (NR)	1 (100)
Poon K et al[4]	1	1 (100)	39 (NR)	1 (100)
Iglesias D et al[34]	1	1 (100)	42 (NR)	0
Alfonso F et al[32]	11	9 (82)	48 (9)	9 (82)
Paulo M et al[7]	8	6 (75)	50 (10)	NR
Malcles G et al[31]	1	1 (100)	49 (NR)	1 (100)
Totals	24	20		12

NR, not recorded or not applicable; SCAD, spontaneous coronary artery dissection; STEMI, ST-segment elevation myocardial infarction.

a double-lumen or intramural hematoma visualized in all cases. However, only three patients presented an intimal "flap" on angiography. In 7 of the 11 patients, OCT readily identified the intimal rupture site and the thickness and extent of the intima-media membrane, and the area of the true and false lumen. In nine patients, it identified associated intramural hematoma and thrombi in the true or false lumens. Most of these findings were not detected by angiography. Additionally, after stenting, OCT disclosed adequate stent coverage, expansion, and apposition, but also residual intramural hematoma at the stented site (abluminal) and in the distal vessel.[32]

OCT should be considered in selected patients in whom the angiogram appears suspicious for SCAD, but additional diagnostic data from intravascular imaging may help confirm the diagnosis. This information may help to determine the extent of vessel involved and help plan therapeutic strategies, as was demonstrated in this case. The primary operator should feel comfortable obtaining and interpreting the OCT images. The inherent risks of OCT imaging are small if it is used with caution.

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Management

The majority of patients with SCAD in the published literature are treated conservatively, despite the fact that most present with ACS. In a retrospective case identification study from the Western Denmark Heart Registry, SCAD was documented in 0.07% of 32,869 angiograms in the angiographic registry (22 patients); all 22 patients presented with ACS (100%), and 17 presented with STEMI (12 PCI, 2 coronary artery bypass graft [CABG], and 3 medical management).[21] In another large database analysis of 11,605 angiograms from France, 23 (0.2%) cases were identified as SCAD. Of those, 21 presented with ACS, including 7 with non-ST-segment elevation myocardial infarction (NSTEMI) and 14 with STEMI (7 PCI, 2 CABG, and 5 medical management).[3] Alfonso and associates[28] reported on 45 consecutive patients with SCAD over a 6-year period based on 16,813 angiograms (0.3%), including 16 with NSTEMI and 18 with STEMI. Although the authors support a conservative approach, 15 out of 45 patients (33.3%) required PCI.[28] Our patient demonstrated ongoing ischemia in the setting of STEMI and required urgent revascularization.

PCI in SCAD may be challenging; reported success rates approximate 65%, as the procedure is fraught with the potential for additional complications.[19] During angiography, passage of the coronary guide wire into the subintimal space or subsequent balloon or stent expansion of the dissected region, may squeeze the intramural hematoma and extend the dissection.[35] There are few data on the use of aspiration thrombectomy in SCAD patients, but from major randomized trials of aspiration thrombectomy in primary PCI patients, it appears that there is no increase in target vessel dissection from aspiration thrombectomy catheter use.[36]

PCI is recommended in SCAD patients with decreased TIMI flow on angiography, symptoms not responsive to medical therapy, or hemodynamic instability.[27] Stents are recommended as balloon angioplasty is usually insufficient to treat the SCAD.[37,38]

Conclusions

Our patient presented with a STEMI resulting from SCAD requiring PCI with excellent short- and long-term outcome. OCT

outlined key diagnostic features and contributed to the understanding of the dissected segment, which, in turn, allowed a more precise management of the patient using stent placement. Additionally, this is possibly the first report of histology from a live patient that provides insight into the etiology for SCAD, with inflammatory cells noted in the tunica intima. ■

The authors would like to recognize the contributions of Alex Zhu, BA, in the preparation of all portions of the manuscript. The manuscript would not have been possible without Brent Larson, DO, and Serguei Bannykh, MD, PhD, of the Cedars-Sinai Medical Center Department of Pathology, who provided Figure 2. The authors would also like to recognize Tara Rao in the preparation of Figure 3.

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MAIN POINTS

- Spontaneous coronary artery dissection (SCAD) is a condition resulting from an increase in shear stress that causes a tear in the intimal lining, and is a well-known but infrequent cause of acute coronary syndrome.
- Symptoms in SCAD result from compression of the arterial lumen via an intramural hematoma, which compromises blood flow, leading to myocardial ischemia or infarction.
- Coronary angiography has historically been the diagnostic test of choice. Because angiography in SCAD patients can be unreliable, intravascular imaging, including both intravascular ultrasound and optical coherence tomography, have been reported to help diagnose and manage SCAD.
- Percutaneous coronary intervention is recommended in SCAD patients with decreased thrombolysis in myocardial infarction flow on angiography, symptoms not responsive to medical therapy, or hemodynamic instability. Stents are recommended as balloon angioplasty is usually insufficient to treat the SCAD.

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