Catheterization and Cardiovascular Interventions 87:E54–E61 (2016)

# Angiographic Appearance of Spontaneous Coronary Artery Dissection With Intramural Hematoma Proven on Intracoronary Imaging

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**Background: The pathognomonic appearance of multiple radiolucent lumen on angiography is used to diagnose spontaneous coronary artery dissection (SCAD). However, this finding is absent in >70% of SCAD, in which case optical coherence tomography (OCT) or intravascular ultrasound (IVUS) is useful to assess arterial wall integrity. Methods: We report the angiographic appearance of SCAD that were proven on intracoronary imaging with OCT or IVUS. Our angiographic classification and algorithm for SCAD diagnosis was previously reported. Patients with type 1 SCAD (multiple radiolucent lumen) do not require OCT/IVUS, whereas, it was recommended for those with suspected type 2 (diffuse stenosis) or 3 (mimic atherosclerosis) SCAD. Results: Twenty-two consecutive patients with non-type 1 angiographic SCAD in 25 coronary arteries (22 OCT and 4 IVUS) were studied. Mean age was 52.9 ± 9.9 years, 89.5% were women, and 16/22 (72.7%) had underlying fibromuscular dysplasia. Sixteen SCAD arteries were type 2 SCAD, and nine were type 3. All 25 SCAD arteries had intramural hematoma and intimomedial membrane separation with double lumen on OCT or IVUS. The mean visual angiographic stenosis was 74.6 ± 17.5% (range 40–100%). Dissected segments were long with mean qualitative coronary analysis (QCA) length 45.2 ± 29.2 mm, especially in patients with type 2 SCAD (mean QCA length 58.3 ± 29.0 mm). The mean QCA length in type 3 SCAD lesions was 22.1 ± 5.7 mm. Conclusions: Intracoronary imaging confirms that SCAD may appear angiographically without multiple radiolucent lumen. Angiographers should be familiar with angiographic SCAD variants to improve SCAD diagnosis, and utilize intracoronary imaging when the diagnosis is uncertain.** © 2015 Wiley Periodicals, Inc.

**Key words: spontaneous coronary artery dissection (SCAD); fibromuscular dysplasia (FMD); optical coherence tomography (OCT); intravascular ultrasound (IVUS)**

## INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a non-iatrogenic and non-traumatic separation of the coronary arterial wall with intramural hematoma, occurring with or without an inciting intimal tear, and can affect any coronary artery. The diagnosis of SCAD has traditionally hinged upon coronary angiography; however, this technology is imperfect as it is a luminogram that does not image the arterial wall. Thus, intracoronary imaging with optical coherence tomography (OCT) or intravascular ultrasounds (IVUS) is the true

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Conflict of interest: Nothing to report. No conflicts of interest with regards to content of this paper. Dr Saw had received research grants for SCAD research (Canadian Institutes of Health Research, University of British Columbia Division of Cardiology, AstraZeneca, Abbott Vascular, St Jude Medical, and Servier), and speaker honararium for SCAD (AstraZeneca, St Jude Medical, Sunovion).

Received 26 November 2014; Revision accepted 18 April 2015

DOI: 10.1002/ccd.26022
Published online 21 July 2015 in Wiley Online Library (wileyonlinelibrary.com)

gold-standard for SCAD diagnosis. The diagnosis of SCAD on coronary angiography is particularly challenging in the absence of pathognomonic multiple radiolucent lumen with contrast staining of the arterial wall. Many angiographers are not familiar with non-pathognomonic angiographic variants of SCAD and this contributes importantly to the under-diagnosis of SCAD.

We have previously described a simple classification of angiographic SCAD and an algorithm to aid SCAD diagnosis [1]. Type 1 angiographic SCAD describes those with pathognomonic multiple radiolucent lumen with contrast wall staining. Type 2 angiographic SCAD describes a long diffuse (typically >20 mm) stenosis that varies in severity from mild to complete occlusion. This appearance commonly involves the mid segment and often extends to distal segment and tip of the arteries. There is typically an appreciable but subtle demarcation of change in arterial caliber from normal diameter to diffuse narrowing. Type 3 angiographic SCAD describes focal or tubular stenosis that mimics atherosclerosis, and intracoronary imaging is central to SCAD diagnosis for this appearance.

We report our consecutive series of angiographic SCAD proven on intracoronary imaging with detailed angiographic descriptions and images, to help raise the awareness of non-pathognomonic angiographic variants of SCAD and to improve clinical angiographic diagnosis of SCAD.

## METHODS

Patients in our SCAD registry at Vancouver General Hospital who had intracoronary imaging (OCT or IVUS) proven SCAD were included in this report. Patients with type 1 angiographic SCAD (multiple radiolucent lumen) do not require intracoronary imaging confirmation, and thus OCT or IVUS is not routinely performed [1], whereas, intracoronary imaging was recommended for those with suspected type 2 (diffuse stenosis) or 3 (mimic atherosclerosis) SCAD. We routinely perform OCT or IVUS for patients with suspected type 3 SCAD. For patients with type 2 angiographic SCAD, we performed OCT or IVUS earlier in our experience if technically feasible, until we became familiar with such angiographic appearance at our center. More recently, we have transitioned to repeat coronary angiography in 4–6 weeks to confirm SCAD diagnosis and healing, especially for those where intracoronary imaging is not technically feasible. We also categorized type 2 angiographic SCAD into those with dissections extending to the absolute distal tip of the artery (classic type 2 appearance), and those with clear delineation of normal distal non-dissected coronary artery.

Clinical features that raise the index of suspicion for SCAD prompting intracoronary imaging include myocardial infarction (MI) in young women, absence of cardiovascular risk factors, peripartum state, presence of fibromuscular dysplasia (FMD) or relevant connective tissue or systemic inflammatory disorders, and intensive exercise or emotional stressors [2,3].

The baseline patient demographics, angiographic, and noninvasive imaging characteristics were recorded. The coronary angiograms were reviewed by two experienced interventional cardiologists (JS and EA) and categorized according to our classification [1]. We have additionally categorized type 2 angiographic appearance into variant A (normal arterial segments proximal and distal to dissection) or variant B (dissection extends to the distal tip of the artery without discernable normal segment distally). Angiographic characteristics of dissection segment location (as per Bypass Angioplasty Revascularization Investigation classification) [4], length (by quantitative coronary angiography analysis), visual stenosis severity, and thrombolysis in myocardial infarction (TIMI) flow were recorded. Patients were consented and enrolled in our NACAD (non-atherosclerotic coronary artery disease) registry for SCAD patients, and followed prospectively.

Intracoronary imaging was performed with either OCT or IVUS, according to operator preference. Intracoronary 100–200 μg of nitroglycerin was administered routinely prior to image acquisition. OCT imaging was acquired using the second-generation frequency domain system (C7-XR/ILUMIEN$^{TM}$, St. Jude Medical/LightLab Imaging, Westford, MA), with automated pullback at 20 mm/sec. Blood in the vessel was displaced using contrast media by manual or power injections. IVUS imaging was performed by mechanical transducer with the Atlantis® SR Pro 40-MHz catheter (Boston Scientific, Santa Clara, CA), with motorized pullback at 0.5 mm/sec. The diagnosis of SCAD on intracoronary imaging required visualization of intramural hematoma and/or separation of the intimomedial membrane creating a double-lumen [5,6]. The length of lesion imaged with the intracoronary imaging is at the operator discretion; some may not advance the imaging catheter throughout the entire length of the lesion for concern of occlusiveness by the catheter, or extension of dissection.

We routinely screen SCAD patients for non-coronary FMD in the renal and iliac arteries with catheter angiography (done during the index coronary angiogram if possible) or CT angiography. Cerebrovascular FMD is screened with head and neck CT angiography.

Patients who underwent revascularization and their outcomes were recorded. The definitions of percutaneous coronary intervention (PCI) outcomes were

**TABLE I. Background Characteristics According to Angiographic Types**

| Mean ± SD, or *n* (%) | Type 2 ($n=14$) | Type 3 ($n=7$) | *P* value |
|---|---|---|---|
| Age | 52.6 ± 11.3 | 51.1 ± 6.8 | NS |
| Sex (women) | 11 (78.6%) | 7 (100%) | NS |
| Height | 166.6 ± 8.3 | 163.7 ± 4.8 | NS |
| Weight | 74.1 ± 18.4 | 62.3 ± 6.0 | NS |
| BMI | 26.2 ± 6.2 | 23.3 ± 2.9 | NS |
| Diabetes mellitus | 0 (0%) | 0 (0%) | NS |
| Dyslipidemia | 4 (28.6%) | 1 (14.3%) | NS |
| Hypertension | 5 (42.9%) | 0 (0%) | NS |
| Current smoker | 2 (14.3%) | 0 (0%) | NS |
| Previous MI | 0 (0%) | 0 (0%) | NS |
| Hypothyroid | 2 (14.3%) | 0 (0%) | NS |
| Postmenopausal | 9 (64.3%) | 3 (42.9%) | NS |
| Migraines | 1 (7.1%) | 4 (57.1%) | 0.025 |
| ST elevation MI | 2 (14.9%) | 2 (28.6%) | NS |
| NSTEMI | 12 (85.1%) | 5 (71.4%) | NS |
| Emotional stress | 6 (42.9%) | 3 (42.9%) | NS |
| Physical stress | 5 (35.7%) | 4 (57.1%) | NS |
| FMD | 9 (75.0%) | 7 (100.0%) | NS |
| EF (mean ± SD) | 51.5 ± 8.9 | 59.7 ± 3.1 | NS |
| QCA length | 61.1 ± 29.9 | 22.0 ± 6.6 | 0.0058 |
| QCA diameter | 2.6 ± 0.7 | 2.7 ± 0.6 | NS |
| Stenosis severity | 74.6 ± 19.5 | 75.6 ± 12.4 | NS |

BMI, body mass index; EF, ejection fraction; FMD, fibromuscular dysplasia; MI, myocardial infarction; NS, not significant; NSTEMI, non-ST elevation MI; QCA, quantitative coronary angiography.

previously reported by our group [7]. In short, successful PCI was defined as angioplasty or stenting of the dissected segment with no residual dissection and with final TIMI 3 flow. Partial success was defined as residual dissection or stenosis $\leq$50%, and with final TIMI 3 or improved flow. Unsuccessful PCI was defined as residual dissection or stenosis $>$50%, or worsened TIMI flow compared with baseline pre-PCI, or extension of dissection requiring bailout coronary artery bypass grafting (CABG).

### Statistical Analysis

Descriptive statistics were used to describe the baseline characteristics of patients. Continuous variables were summarized as mean ± standard deviation. Categorical variables were summarized as frequency and percentage. Categorical variables were compared using a chi-square test or Fisher exact test, and continuous variables using the Student *t* test, with two-sided *P* value of $<$0.05 considered significant. Statistical analyses were performed with the SPSS software (IBM SPSS version 20, New York).

## RESULTS

Among our cohort of patients with non-atherosclerotic SCAD, we performed intracoronary imaging in 22 patients with SCAD involving 25 coronary arteries (one patient had four simultaneously dissected arteries imaged on OCT). OCT was performed in 22 SCAD arteries, and IVUS was performed in 4 SCAD arteries (1 artery was imaged with both OCT and IVUS). The mean age of the patients was 52.9 ± 9.9 years, 89.5% were women, and all presented with myocardial infarction (13.6% had ST elevation). Non-coronary FMD was observed in 16/22 (72.7%) patients. Nine patients (40.9%) reported significant emotional stress prior to their SCAD event. And ten patients (45.4%) reported participating in physical exercises within the week prior to their SCAD, including two (9.1%) reporting heavy isometric weight lifting. One patient had dissections of both type 2 and 3 SCAD angiographic types, the remainder 21 patients' background characteristics are reported in Table I. Patients with type 2 SCAD had greater dissection lengths, but lower incidence of migraines.

Of the 25 SCAD coronary arteries proven on intracoronary imaging, 16 had type 2 angiographic SCAD appearance and 9 had type 3 angiographic SCAD appearance. None had pathognomonic multiple radiolucent lumen as per our protocol. All 25 SCAD arteries had evidence of intramural hematoma and intimomedial membrane separation with double lumen on OCT or IVUS; in two cases, the intracoronary imaging was performed 7 weeks after the SCAD event during follow-up angiography.

The angiographic characteristics of each SCAD artery are detailed in Table II. Examples of SCAD artery and their corresponding intracoronary images (OCT or IVUS) confirming SCAD are shown in Figs. 1–3. The mean visual angiographic stenosis of the 25 SCAD lesions was 74.6 ± 17.5%, varying from mild stenosis to complete occlusion (ranging from 40 to 100%). Dissected segments were long with mean qualitative coronary analysis (QCA) length 45.2 ± 29.2 mm, especially in patients with type 2 SCAD (mean QCA length 58.3 ± 29.0 mm). The mean QCA length in type 3 SCAD lesions was 22.1 ± 5.7 mm. TIMI flow was 3 in 19/25 SCAD arteries, 3 had TIMI 2 flow, 1 had TIMI 1 flow, and 2 had TIMI 0 flow.

For the intracoronary imaging proven type 2 angiographic SCAD, 10/16 cases had variant A appearance with normal arterial segment distal to the dissection (Fig. 1), and 6/16 had variant B appearance with dissections that extended to the distal tip of the arteries (Fig. 2).

Wall motion abnormalities (hypokinesis or akinesis) corresponding to the dissected arteries were observed in all but one case (Table II). Eight SCAD patients were evaluated with repeat coronary angiogram at least 4 weeks after their SCAD event (mean 190 days later), and all 11 dissected arteries spontaneously healed

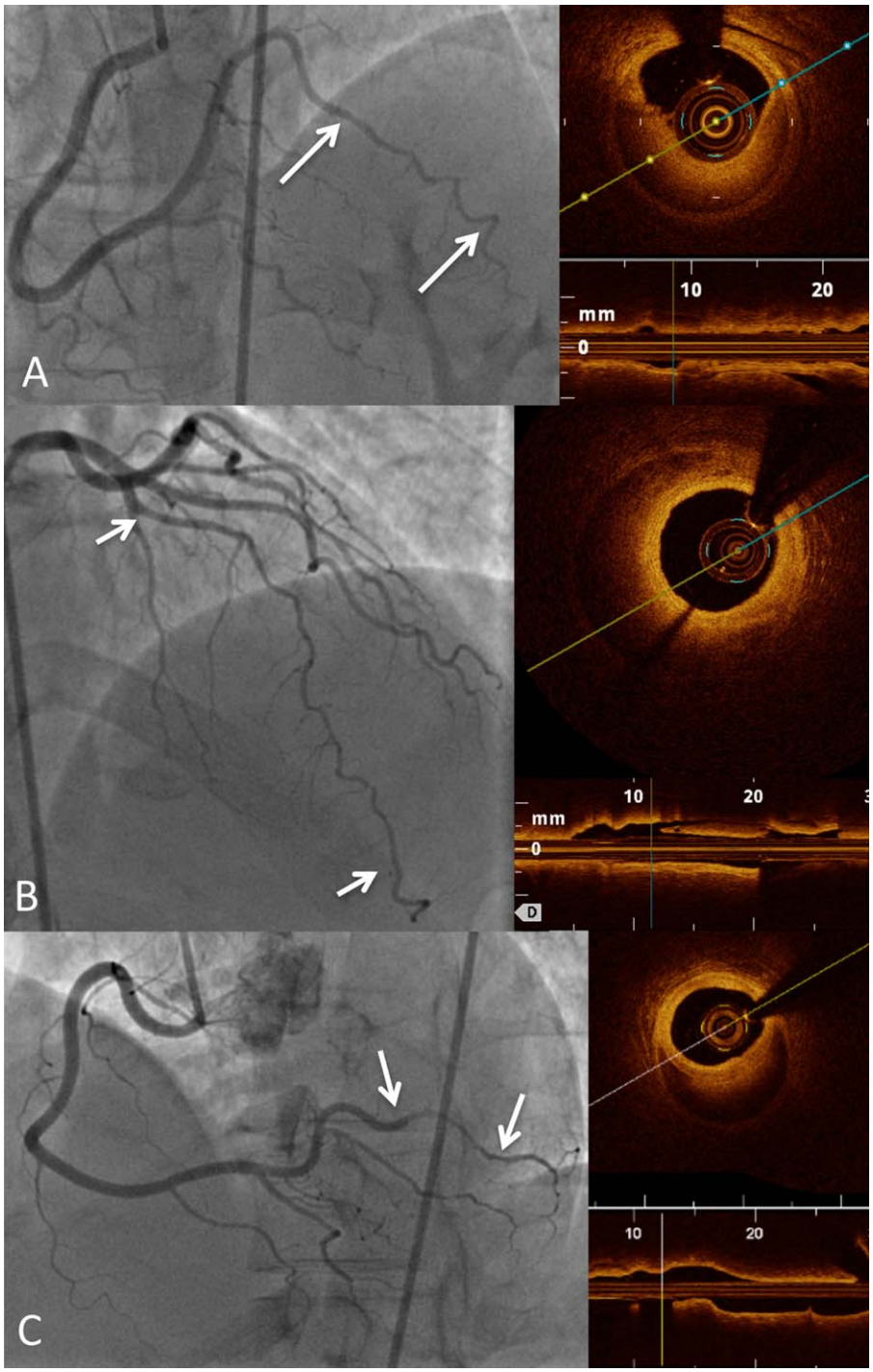


**Fig. 1. Type 2 angiographic SCAD, variant A. A: SCAD artery 11: Diffuse SCAD of the RPL with diffuse moderate 60% stenosis (between arrows), and corresponding OCT showing intramural hematoma. B: SCAD artery 5: Extensive SCAD involving the mid to apical LAD (between arrows), and corresponding intramural hematoma on OCT. C: SCAD artery 2: SCAD affecting the right posterolateral (RPL) branch (between the arrows), and corresponding OCT showing residual intramural hematoma 7 weeks later.**

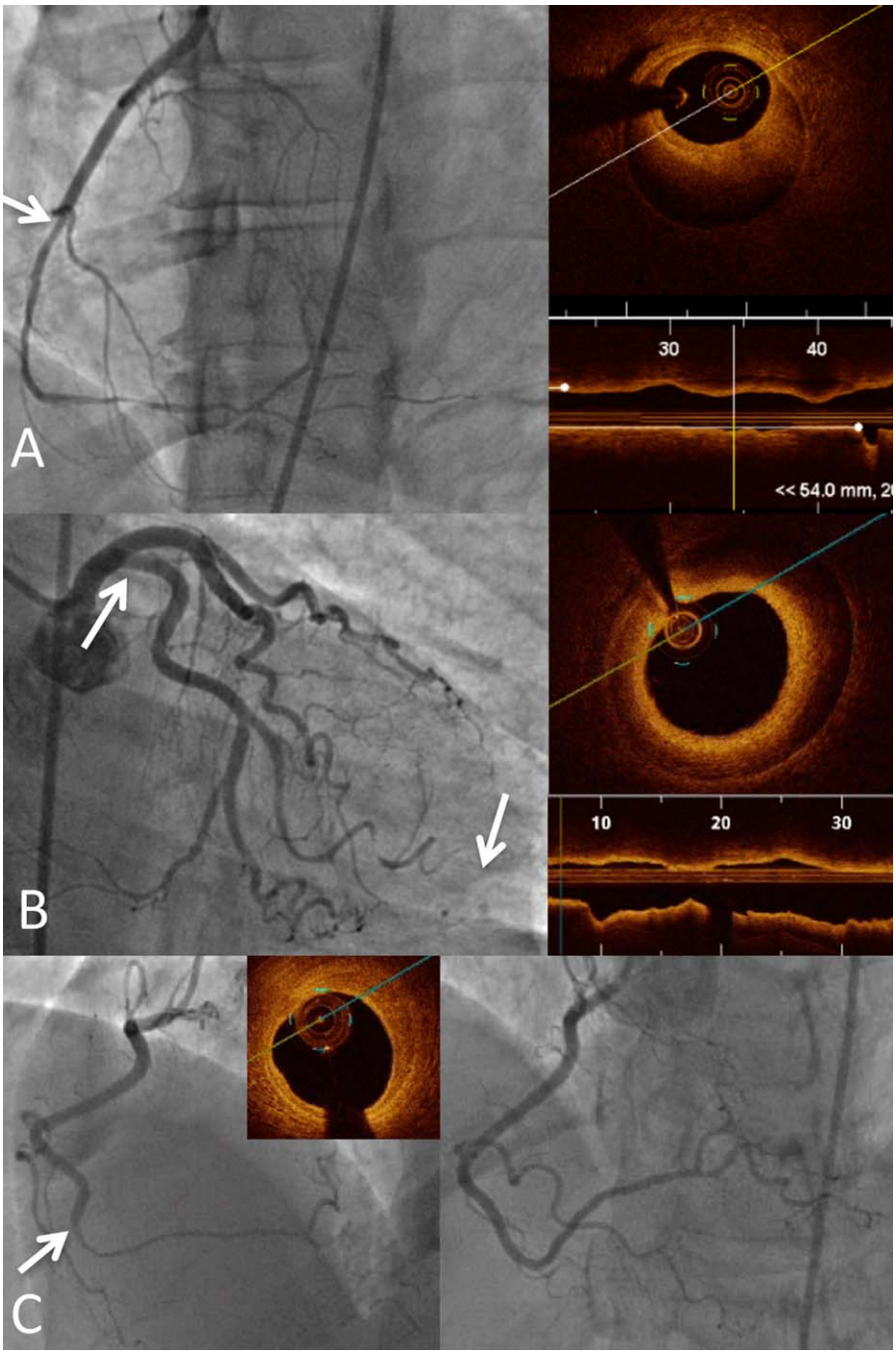


**Fig. 2. Type 2 angiographic SCAD, variant B. A: SCAD artery 1: Extensive SCAD from the mid right coronary artery (RCA) (arrow) to the distal tip of the right posterior descending artery (RPDA), and corresponding OCT showing intramural hematoma. B: SCAD artery 6: Extensive SCAD involving the proximal circumflex artery extending to the distal tip of the 2nd obtuse marginal (OM) branch (between arrows), and corresponding OCT showing intramural hematoma. C: SCAD artery 8: Extensive SCAD starting from the distal RCA to the distal tip of the RPDA (starting from the arrow), with repeat angiography 7 weeks later (right image) showing angiographic healing but OCT (middle image) showing mild residual partially healed intramural hematoma.**

angiographically. Two patients also had OCT done during their repeat angiogram at 7 weeks, which showed minor residual resolving intramural hematoma (Figs. 1C and 2C).

Thirteen patients were treated conservatively (59.1%) and 9 patients underwent PCI (none required coronary artery bypass surgery). Of those who underwent PCI, four were successful (44.4%) (although two

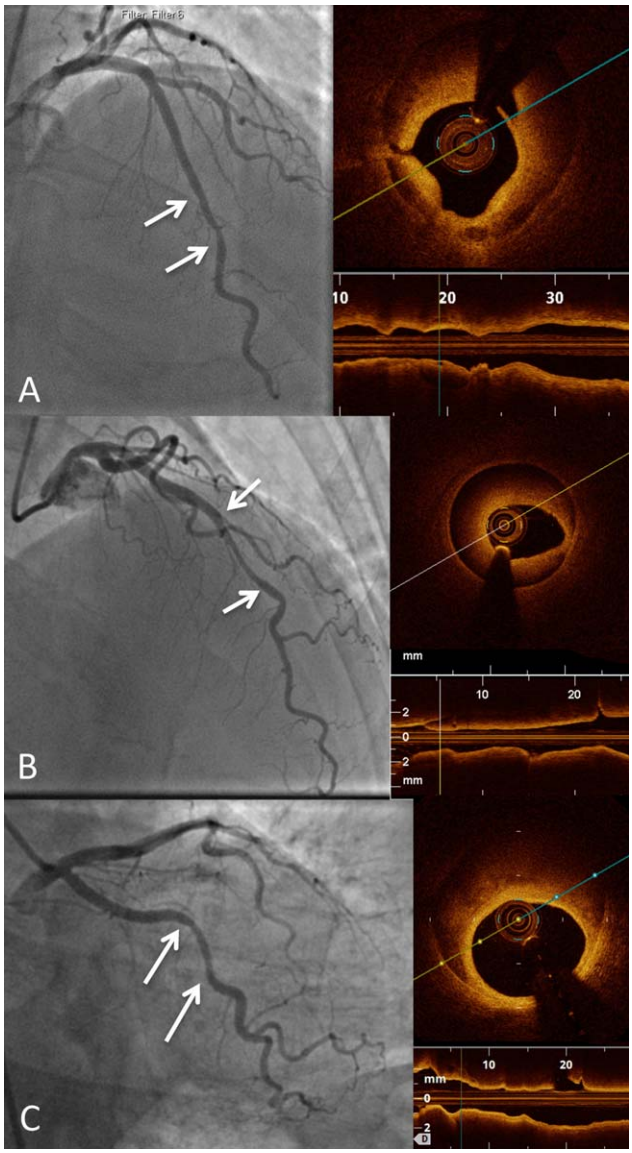


**Fig. 3. Type 3 angiographic SCAD: (A) SCAD artery 18: SCAD of the mid LAD with mild tubular stenosis (between arrows), and corresponding OCT showing intramural hematoma. B: SCAD artery 19: SCAD of the mid LAD with tubular stenosis (between arrows), and corresponding OCT showing intramural hematoma and the external elastic membrane. C: SCAD artery 25: SCAD of the mid circumflex artery (between arrows), and corresponding OCT showing intramural hematoma.**

had extension of dissection), three (33.3%) were partially successful (2 had subsequent in-stent restenosis), and two (22.2%) were unsuccessful.

## DISCUSSIONS

Angiographic SCAD appearance without the pathognomonic multiple radiolucent lumen with contrast wall stain (i.e., type 2 and 3 angiographic SCAD) is often not recognized by cardiologists. Sole reliance on the presence of a type 1 angiographic appearance to make a diagnosis of SCAD contributes significantly to the under-diagnosis of SCAD. We previously reported that only <30% of SCAD has angiographic type 1 appearance [7], which corroborated the findings by Alfonso et al. [5]. Therefore, it is important for angiographers to improve their recognition of other angiographic variants of SCAD. In this report, we described and presented the angiographic appearances of 25 dissected coronary arteries (non-type 1) with proven intramural hematoma and intimomedial separation on intracoronary imaging.

For patients with type 2 angiographic SCAD, the length of the dissection can be extensive with mean length ~60 mm in our intracoronary imaging proven subset, and the stenosis severity varies from mild to complete occlusion (40–100%). We were initially not familiar with the type 2 forms of angiographic SCAD until our early experience with IVUS and OCT that showed intramural hematoma as the cause of the stenosis in such diffuse arterial narrowing. Since our early recognition, we subsequently observed that the majority of angiographic SCAD is of the type 2 variants in ~ 2/3 of SCAD cases in our series [7]. This angiographic appearance typically involves diffuse narrowing >20 mm in length, with relatively smooth arterial wall compression, and an appreciable (albeit subtle) abrupt change in arterial caliber from normal diameter to diffuse stenosis. The more common appearance of type 2 SCAD involves extension of the dissection from the proximal-mid segment of the artery to the absolute distal tip of the artery (type 2 variant B), which may lead to missed diagnosis of SCAD as angiographers may interpret the stenosis as a "normal tapering" of the coronary arteries. The type A variant of type 2 SCAD is the presence of a normal artery (non-dissected) distal to the dissected segment, which is also quite commonly observed.

Given our familiarity with the type 2 angiographic forms of SCAD, we now have lower propensity to perform intracoronary imaging in these cases, particularly if the dissected segments are of small caliber. Instead, we favor repeat coronary angiography in 4–6 weeks to assess for angiographic healing. For angiographers beginning to appreciate this angiographic appearance, we find that assessing for corresponding wall motion abnormalities is tremendously helpful, as this telltale sign is present in the vast majority of type 2 SCAD.

Type 3 angiographic SCAD is the most challenging to recognize on coronary angiograms. These dissections mimic atherosclerotic lesions; they are typically not very long, usually <30 mm in length (mean length

**TABLE II. Characteristics of Dissected Coronary Arteries**

| SCAD artery | Coronary segment dissected | SCAD type | Visual stenosis (%) | QCA length (mm) | TIMI flow | Wall motion abnormality | Non-coronary FMD |
|---|---|---|---|---|---|---|---|
| 1 | Mid-distal RCA | 2B | 90 | 106.3 | 2 | Yes | Yes |
| 2 | RPL | 2A | 80 | 28.8 | 3 | Yes | Yes |
| 3 | Mid circumflex | 2A | 40 | 35.1 | 3 | Yes | Yes |
| 4 | Mid-apical LAD | 2A | 70 | 74.1 | 0 | Yes | Not screened |
| 5 | Mid-apical LAD | 2A | 80 | 100.0 | 3 | Yes | Yes |
| 6 | Mid circumflex-OM2 | 2B | 90 | 117.2 | 2 | Yes | Yes |
| 7 | Mid circumflex | 2A | 60 | 42.2 | 3 | Yes | Yes |
| 8 | Distal RCA-RPDA | 2B | 90 | 77.3 | 3 | Yes | Yes |
| 9 | 1st Diagonal | 2B | 80 | 46.8 | 3 | Yes | Yes |
| 10 | RPDA | 2A | 80 | 30.4 | 3 | Yes | Yes |
| 11 | RPL | 2A | 60 | 34.1 | 3 | Yes | No |
| 12 | 3rd Diagonal | 2B | 90 | 41.3 | 3 | Yes | No |
| 13 | Mid-distal LAD | 2B | 100 | 28.1 | 0 | Yes | Incomplete screen |
| 14 | 1st Diagonal | 2A | 50 | 66.6 | 3 | Yes | Yes |
| 15 | Prox circumflex-OM2 | 2A | 95 | 54.1 | 2 | Yes | Incomplete screen |
| 16 | Mid LAD | 2A | 50 | 49.8 | 3 | Yes | Yes |
| 17 | OM1 | 3 | 90 | 22.4 | 3 | Yes | Yes |
| 18 | Mid LAD | 3 | 40 | 22.1 | 3 | Yes | Yes |
| 19 | Mid LAD | 3 | 70 | 22.0 | 3 | Yes | Yes |
| 20 | Mid LAD | 3 | 80 | 25.4 | 3 | Yes | Yes |
| 21 | Mid LAD | 3 | 70 | 30.7 | 3 | No | Yes |
| 22 | OM3 | 3 | 80 | 19.5 | 3 | Yes | Yes |
| 23 | OM2 | 3 | 70 | 24.3 | 3 | Yes | Yes |
| 24 | RPDA | 3 | 99 | 9.2 | 1 | Yes | Yes |
| 25 | Mid circumflex | 3 | 60 | 23.1 | 3 | Yes | Yes |

FMD, fibromuscular dysplasia; LAD, left anterior descending artery; OM, obtuse marginal; QCA, qualitative coronary analysis; RCA, right coronary artery; RPDA, right posterior descending artery; RPL, right posterolateral.

was 23 mm in our series), and the stenosis severity can vary from mild to severe. Other angiographic characteristics that may suggest type 3 SCAD in the appropriate clinical settings are: hazy-appearance of stenosis, lack of atherosclerotic changes in other coronary arteries, and unusual linear stenosis [1]. In such cases, the use of intracoronary imaging is necessary to make the definitive diagnosis.

The utility of IVUS and OCT in the diagnosis of SCAD has been well described by Alfonso's group [5,6]. In essence, both technologies provide complementary details on SCAD, with OCT having superior spatial resolution and thus better in visualizing intimal tears, intraluminal thrombi, false lumen, and intramural hematoma. However, shadowing and inadequate optical penetration may impede full visualization of the extent of intramural hematoma. Conversely, IVUS does not distinctly delineate the lumen–intimal interface (e.g., intimal tear) as well as OCT, but is adequate to visualize intramural hematoma and false lumen. Due to better ultrasound penetration, IVUS allows more complete and deeper vessel visualization, and better appreciation of the extent of intramural hematoma. For centers limited to only one technology, OCT is typically preferred due to the superiority in spatial resolution and clarity in visualizing intramural hematoma and intimal tear. Both technologies are relatively safe in the imaging of SCAD [5,6], although there are theoretical concerns of catheter-induced propagation of dissections, ischemia from an occlusive intracoronary catheter, and hydraulic initiation of dissections with OCT contrast injection. We have not experienced the latter complication, although we had a case of OCT catheter-tip induced worsening dissection of the proximal circumflex artery due to vessel tortuosity, which we anticipated would have similarly occurred with the IVUS catheter.

The mechanisms of appearance of intramural hematoma on both intracoronary imaging modalities should be explored. With IVUS, intramural hematoma can appear as a homogeneous, hyperechoic, crescent-shaped area of blood collection [8]. The echogenicity of the blood collection depends on the flow rate, red cell aggregation, and fibrin content [8,9]. With stagnant flow, the backscatter intensity of blood increased dramatically, likely due to red cell aggregation and increase in fibrin content [8]. Thus, with chronicity the blood collection can become more echogenic due to coagulating blood and eventual solid hematoma. A similar pathophysiologic explanation of time-dependent intramural hematoma appearance on OCT could be postulated, however, the appearance is dissimilar due to the difference with near-infrared light characteristics

on tissue reflection and backscattering. The appearance of blood collection as intramural hematoma on OCT is different from "normal blood" in the lumen of arteries. Normal flowing blood in arterial lumen causes dramatic light scattering, and thus contrast needs to be injected to clear the luminal blood for OCT imaging of arterial walls. Intramural hematoma, on the other hand, has a "dark appearance" (low reflectivity) and at times allows visualization of the external elastic membrane (Fig. 3B), implying low backscattering. We postulate that the "dark appearance" of intramural hematoma may be due to densely packed red blood cells with similar index of refraction in a relatively stagnant environment, causing low reflectivity and backscattering. With subacute SCAD, the "partially resolving" intramural hematoma takes on a higher reflectivity (higher intensity) as shown in Fig. 2C, which may represent digested red blood cells and progressive replacement by proteoglycans and other fibrotic tissues.

### Study Limitations

This is a retrospective analysis of a consecutive series of intracoronary imaging proven SCAD to describe the angiographic appearance of dissected coronary arteries. Only a small number of dissected coronary arteries are described here ($n = 25$) due to the relatively infrequent prevalence of disease, and the theoretical concerns of complications related to intracoronary imaging. The largest previously published series of SCAD intracoronary imaging by Alfonso et al. included only 11 dissected arteries. Thus, our study adds significantly to the limited available published data on SCAD imaging, and serves as an important educational tool to angiographers to improve the recognition of SCAD on coronary angiography.

## CONCLUSIONS

SCAD is under-diagnosed largely due to the challenges in angiographic diagnosis. Angiographers need to become familiar with and recognize non-pathognomonic angiographic forms of SCAD (types 2 and 3). Intracoronary imaging is instrumental in suspected SCAD cases, especially in type 3 forms, when angiography is inconclusive.

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