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Natural History of Spontaneous Coronary Artery Dissection With Spontaneous Angiographic Healing

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ABSTRACT

OBJECTIVES Given the uncertainty regarding the degree and prevalence of spontaneous healing following spontaneous coronary artery dissection (SCAD), the aim of this study was to assess the angiographic characteristics of the dissected segments in a large cohort of patients with SCAD who underwent subsequent repeat coronary angiography.

BACKGROUND SCAD is an uncommon yet important cause of myocardial infarction in women. Very little is known about the characteristics of healing of dissected arteries.

METHODS Patients with nonatherosclerotic SCAD followed prospectively at Vancouver General Hospital who underwent repeat angiography were included in this study. Those who underwent percutaneous coronary intervention for SCAD were excluded. Baseline patient demographics and in-hospital and long-term cardiovascular events were recorded. Angiographic characteristics of the SCAD artery at index and repeat angiography were assessed by 2 experienced angiographers. Criteria for angiographic healing were as follows: 1) improvement of stenosis severity from index event; 2) residual stenosis <50%; and 3) TIMI (Thrombolysis In Myocardial Infarction) flow grade 3.

RESULTS One hundred fifty-six patients with 182 noncontiguous SCAD lesions were included. The mean age was 51.5 ± 8.7 years, 88.5% were women, 83.3% were Caucasian, and 75.6% had fibromuscular dysplasia. All patients presented with myocardial infarction. At index angiography, type 2 SCAD was most commonly observed, in 126 of 182 lesions (69.2%); TIMI flow grade <3 was present in 85 of 182 (46.7%); and median lesion stenosis was 79.0% (interquartile range: 56.0% to 100%). Median time to repeat angiography was 154 days (interquartile range: 70 to 604 days), with median residual lesion stenosis improving to 25.5% (interquartile range: 12.0 to 38.8 days), and TIMI flow grade <3 observed in 10 of 182 lesions (5.5%). Angiographic healing occurred in 157 of 182 lesions (86.3%). Of repeat angiography performed ≥30 days post-SCAD, 152 of 160 (95%) showed spontaneous angiographic healing.

CONCLUSIONS The majority of coronary arteries affected by SCAD heal spontaneously on repeat angiography, with apparent time dependency, with the vast majority having complete healing after 30 days from the SCAD event. (J Am Coll Cardiol Intv 2019;■:■–■) © 2019 Published by Elsevier on behalf of the American College of Cardiology Foundation.

Spontaneous coronary artery dissection (SCAD) is an important cause of acute coronary syndrome, especially in young to middle-aged women. There is a predilection for women in a 9:1 ratio compared with men because of underlying predisposing causes such as fibromuscular dysplasia (for which there is also a predilection in women), pregnancy, and hormonal therapy (estrogen, progesterone)

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ABBREVIATIONS AND ACRONYMS

IQR = interquartile range

MACE = major adverse cardiac event(s)

MI = myocardial infarction

PCI = percutaneous coronary intervention

SCAD = spontaneous coronary artery dissection

(1). The true prevalence of the condition is unknown because of underdiagnosis, but it has been reported in 0.1% to 4.0% of patients presenting with acute coronary syndrome (1). In a large National Inpatient Sample analysis from 2009 to 2014, among about 750,000 women who presented with myocardial infarction (MI) who underwent coronary angiography, about 1% were reported to be due to SCAD (2). However, in contemporary studies with improved recognition of SCAD on angiography, this disease was reported to cause 24% to 35% of MI in women <60 years of age (3).

SCAD is defined as a nontraumatic and noniatrogenic separation of the coronary arterial wall by intramural hemorrhage creating a false lumen, with or without an intimal tear. The separation can occur between the intima and media or between the media and adventitia (1,4). The dissection may originate from an intimal tear leading to dissection into the arterial wall or may result from spontaneous bleeding from ruptured vasa vasorum without intimal tear (4). The definition of SCAD has evolved over the years, with the first case report by Pretty in 1931 describing a dissected coronary artery atherosclerotic aneurysm (3,5,6). However, modern use of the term "SCAD" excludes atherosclerotic causes. The pathophysiology of this disease remains incompletely understood, but there appears to be an underlying arteriopathy that weakens the arterial wall in the majority of cases. Furthermore, SCAD is commonly triggered by physical, emotional, or hormonal stressors (7).

Following the acute SCAD event, conservative therapy is currently the recommended first-line therapy, except in the setting of ongoing ischemia, hemodynamic instability, or left main dissection (8,9). The major reasons for conservative therapy include poor outcomes associated with percutaneous coronary intervention (PCI) and the natural tendency for the dissected arteries to heal spontaneously. Previous small series reported spontaneous healing in 73% to 97% of cases in which repeat angiography was performed (1). However, these studies were small, retrospective, and nonuniform in the definition of angiographic healing.

Given the uncertainty of the degree and prevalence of spontaneous healing following SCAD, we sought to assess the angiographic characteristics of the dissected segments in a large cohort of patients with SCAD who underwent subsequent repeat coronary angiography.

METHODS

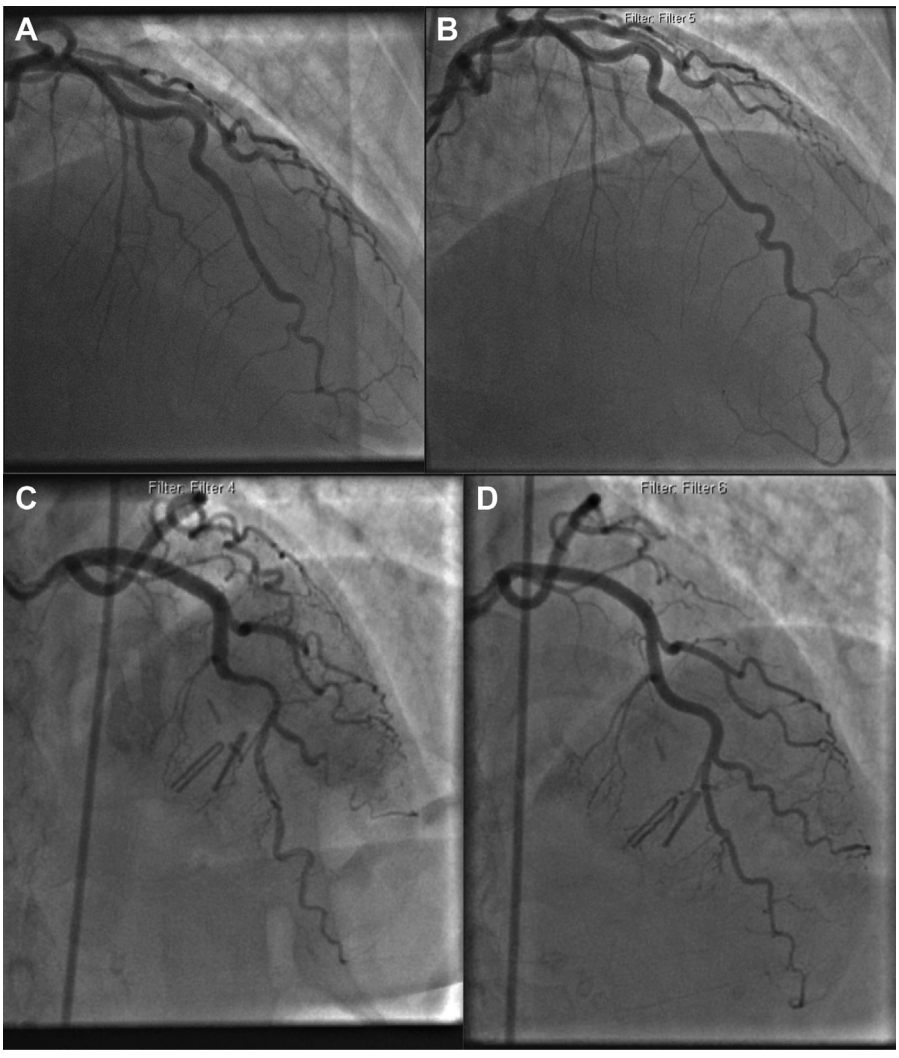
We retrospectively reviewed our cohort of patients with nonatherosclerotic SCAD who were prospectively followed at Vancouver General Hospital and who underwent repeat coronary angiography following the index SCAD event. Patients were referred primarily from hospitals in British Columbia but also from surrounding cities and provinces. All patients provided informed consent for our SCAD registries (the Non-Atherosclerotic Coronary Artery Disease registry or the Canadian SCAD study), approved by the University of British Columbia research ethics board. Detailed medical history, baseline demographics, laboratory results, and angiographic findings of index and repeat studies were recorded. In-hospital and long-term cardiovascular events were collected. All patients were followed at least annually at our SCAD clinic or by telephone follow-up.

Coronary angiograms were reviewed by 2 experienced cardiologists for SCAD diagnosis and were classified as previously described (8,10,11). In brief, type 1 angiographic SCAD required the classic appearance of contrast dye staining of the arterial wall with multiple radiolucent lumen. Type 2 angiographic SCAD was defined as diffuse smooth narrowing that could vary in severity; variant 2A involved normal arterial segments proximal and distal to the dissection, whereas variant 2B involved dissection that extended to the distal tip of the artery. Type 3 angiographic SCAD appeared as focal or tubular stenosis that mimicked atherosclerosis and typically required optical coherence tomography (OCT) or intravascular ultrasound to prove intramural hematoma or double lumen. The SCAD coronary segment involved was defined by the BARI (Bypass Angioplasty Revascularization Investigation) classification (12,13). Angiographic stenosis by visual estimation, lesion length, and TIMI (Thrombolysis In Myocardial Infarction) flow grade was recorded.

In this substudy, we excluded patients who underwent PCI of the SCAD lesion to evaluate for spontaneous healing of the dissected segments. The decision to perform repeat coronary angiography was at the discretion of the treating physicians. We defined spontaneous angiographic healing (**Figure 1**) when all the following criteria were met: 1) improvement of stenosis severity from index event; 2) residual stenosis <50%; and 3) TIMI flow grade 3.

In-hospital major adverse cardiac events (MACE), including all-cause mortality, stroke, reinfarction,

FIGURE 1 **Examples of Spontaneous Angiographic Healing**



(A) Distal left anterior descending coronary artery (LAD) type 2 spontaneous coronary artery dissection (SCAD) with **(B)** subsequent angiographic healing. **(C)** Mid-LAD type 1 SCAD with **(D)** subsequent angiographic healing.

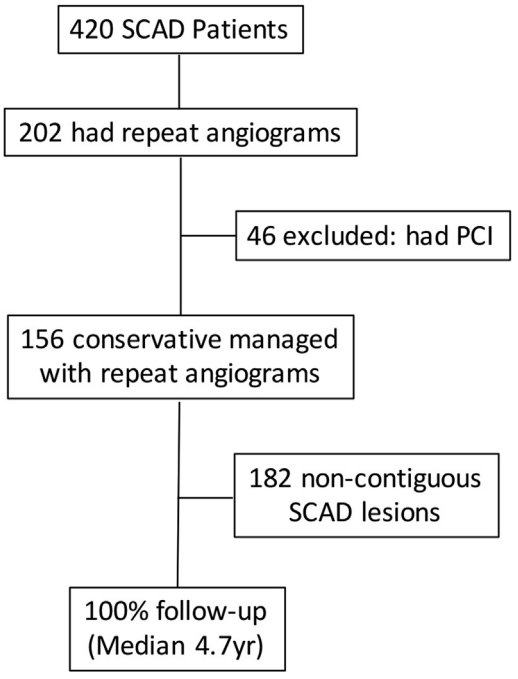
cardiogenic shock, congestive heart failure, severe ventricular arrhythmia, revascularization, repeat or unplanned revascularization, and cardiac transplantation, were recorded. Long-term MACE of all-cause mortality, stroke, recurrent MI (including recurrent dissection), congestive heart failure admission, and revascularization were recorded.

STATISTICAL ANALYSIS. Descriptive statistics were used to summarize the baseline characteristics. Continuous variables are reported as mean ± SD or as median (interquartile range [IQR]). Categorical variables are summarized as frequencies and percentages. Comparisons between categorical data were made using the chi-square or Fisher exact test. Continuous data were compared using the Student's *t*-test or Mann-Whitney *U* test. Two-sided p values of <0.05 were considered to indicate statistical significance. Statistical analyses were performed using SPSS version 23 (IBM, Armonk, New York).

RESULTS

Two hundred two patients underwent repeat coronary angiography after the index SCAD event. Of these, 46 underwent PCI of the SCAD artery and were excluded from this analysis (**Figure 2**). The remaining

FIGURE 2 Flow Diagram of Study



Of 420 patients with spontaneous coronary artery dissection (SCAD), 202 underwent repeat angiography, and 156 treated conservatively were included in this substudy. PCI = percutaneous coronary intervention.

TABLE 1 Baseline Patient Characteristics (N = 156)

Age (yrs)	51.5 ± 8.7
Female	138 (88.5)
Ethnicity	
Caucasian	130 (83.3)
East Asian	17 (10.9)
South Asian	7 (4.5)
First Nations	1 (0.6)
African Canadian	1 (0.6)
Cardiac risk factors	
Family history of heart disease	55 (35.3)
Diabetes mellitus	8 (5.1)
Hypertension	70 (44.9)
Dyslipidemia	40 (25.6)
Cigarette smoking	19 (12.2)
Clinical presentation	
NSTEMI	121 (77.6)
STEMI	35 (22.4)
Presence of extracoronary FMD	118 (75.6)

Values are mean ± SD or n (%).
FMD = fibromuscular dysplasia; NSTEMI = non-ST-segment elevation myocardial infarction; STEMI = ST-segment elevation myocardial infarction.

156 patients who were treated conservatively were included. Baseline characteristics of the patients are shown in Table 1. The mean age was 51.5 ± 8.7 years, 88.5% were women, 83.3% were Caucasian, and 75.6% had extracoronary fibromuscular dysplasia. All patients presented with MI; 77.6% had non-ST-segment elevation MI, and 22.4% had ST-segment elevation MI. There was a low prevalence of cardiovascular risk factors: diabetes mellitus in 5.1%, smoking in 12.2%, and dyslipidemia in 25.6%.

There were 182 noncontiguous SCAD lesions on index angiography, and the baseline angiographic characteristics are shown in Table 2. The left anterior descending coronary artery was the most commonly affected artery in 48.9%, followed by the left circumflex coronary artery (28.6%), and the right coronary artery (22.5%). Type 2 angiographic SCAD was the most common pattern observed on index angiography, affecting 126 of 182 lesions (69.2%): type 2B in 78 (42.9%) and type 2A in 48 (26.4%). Type 1 SCAD occurred in 47 of 182 lesions (25.8%) and type 3 SCAD in 9 of 182 lesions (4.9%). TIMI flow grade <3 occurred in 85 of 182 lesions (46.7%), with 40 (22.0%) having TIMI flow grade 0. The median lesion angiographic stenosis severity was 79.0% (IQR: 56.0% to 100%).

The median time to repeat angiography was 154 days (IQR: 70 to 604 days), and the results are summarized in Table 2. Of the 182 SCAD lesions, repeat angiography was performed after <30 days for 22 lesions and after ≥30 days for 160 lesions. On follow-up angiography, the median residual lesion stenosis improved to 25.5% (IQR: 12.0% to 38.0%) (Figure 3), and TIMI flow grade <3 was observed in only 10 of 182 lesions (5.5%), with only 4 (2.2%) having TIMI flow grade 0 (Figure 4). Of the 40 lesions with TIMI flow grade 0 at baseline, only 3 had TIMI flow grade 0 on follow-up angiography, and 2 had TIMI flow grade 2; the remainder (35 of 40 [87.5%]) improved to TIMI flow grade 3.

Spontaneous angiographic healing (achieving the 3 criteria outlined earlier) was observed in 157 of 182 lesions (86.3%). The remaining 25 (13.7%) did not meet criteria for angiographic healing, and in 17 of these cases (68.0%), repeat angiography was performed early during the course of follow-up (<30 days after the index SCAD event). Of these 25 unhealed lesions, angiography was performed in 10 for recurrent MI, in 6 for recurrent symptoms, and in 9 to confirm SCAD diagnosis. There was no significant difference in baseline angiographic characteristics between lesions that healed and those that did not (Table 2). The only significant differences were time to repeat angiography (shorter for lesions that did not heal; median 4 days vs. 212 days; $p < 0.001$) and

worse ejection fraction for lesions that did not heal (47.5% vs. 60.0%; p = 0.018).

Among the 157 healed lesions, 152 (96.8%) angiograms were obtained ≥30 days post-SCAD (median 212 days; IQR: 106 to 960 days); only 5 were obtained <30 days from the index event. Of the repeat angiograms obtained ≥30 days post-SCAD, 95% (152 of 160) showed spontaneous angiographic healing, whereas only 5 of 22 lesions (22.7%) healed when angiography was repeated <30 days post-SCAD; these patients without healing on early repeat angiography were continued on conservative management without PCI.

OCT was performed in 3 patients on follow-up angiography (2 for cases that had healed angiographically and 1 for a nonhealed lesion). Examples of appearance on OCT of intramural hematoma in various stages of healing and resorption at 31 days post-SCAD are shown in **Figure 5**.

The median time of follow-up in this cohort was 4.7 years (IQR: 3.1 to 7.2 years). The overall rate of MACE was 37.8% (**Table 3**), including mortality in 1.3%, recurrent MI in 35.9%, revascularization in 6.4%, and stroke in 1.9%. There was no significant difference in the occurrence of MACE between those with angiographic healing and those without (log-rank p = 0.086).

TABLE 2 Angiographic Lesion Characteristics and Comparison Between Healed and Unhealed Lesions

	Overall Lesions (N = 182)	Healed Lesions (n = 157)	Unhealed Lesions (n = 25)	p Value*
Angiographic healing	157 (86.3)	157 (100)	0 (0)	
Coronary territory affected				
LAD	89 (48.9)	75 (47.8)	14 (56.0)	0.52
LCx	52 (28.6)	44 (28.0)	8 (32.0)	0.64
RCA	41 (22.5)	38 (24.2)	3 (12.0)	0.21
SCAD type				
1	47 (25.8)	40 (25.5)	7 (28.0)	0.81
2	126 (69.2)	110 (70.1)	16 (64.0)	0.64
2A	48 (26.4)	41 (26.1)	7 (28.0)	0.81
2B	78 (42.9)	69 (43.9)	9 (36.0)	0.52
3	9 (4.9)	7 (4.5)	2 (8.0)	0.36
TIMI flow grade				
0	40 (22.0)	35 (22.3)	5 (20.0)	0.80
1	18 (9.9)	14 (8.9)	4 (16.0)	0.28
2	27 (14.8)	27 (17.2)	0 (0)	0.029
3	97 (53.3)	81 (51.6)	16 (64.0)	0.29
Stenosis severity (%)	79.0 (56.0–100)	69.9 (54.0–90.0)	57.6 (45.0–72.0)	0.94
Stenosis length (mm)	32.4 (20.9–47.0)	34.7 (21.9–47.4)	25.1 (19.1–38.0)	0.32
Multiple segments involved	46 (25.3)	36 (22.9)	10 (40.0)	0.084
Multivessel SCAD	33 (18.1)	25 (15.9)	8 (32.0)	0.088
Time to repeat angiography (days)	154 (70–604)	212 (106–960)	4 (1.7–11.2)	<0.001
Angiography performed at <30 days	22 (12.1)	5 (3.2)	17 (68.0)	<0.001
Follow-up stenosis severity (%)	25.5 (12.0–38.8)	22.0 (10.7–33.0)	73.0 (50.0–100)	<0.001
Baseline EF (%)	58.0 (50.0–64.0)	60.0 (51.0–65.0)	47.5 (45.0–61.0)	0.018

Values are n (%) or median (interquartile range). *p value comparing healed versus unhealed lesions.

EF = ejection fraction; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; RCA = right coronary artery; SCAD = spontaneous coronary artery dissection; TIMI = Thrombolysis In Myocardial Infarction.

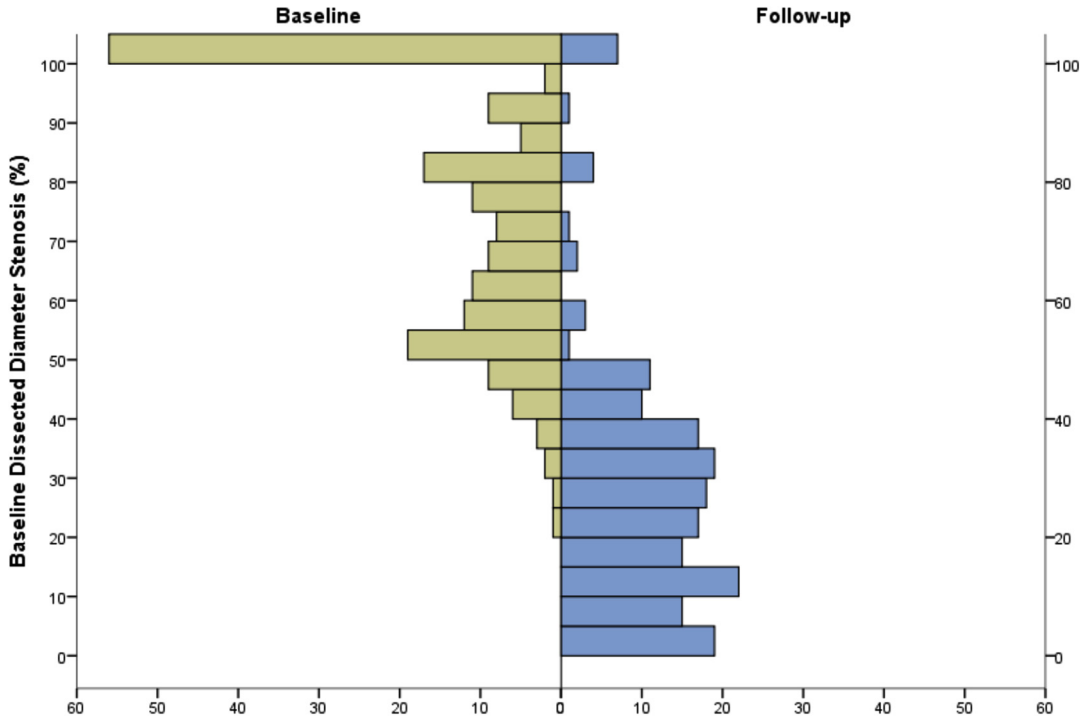
DISCUSSION

We performed a retrospective analysis of angiographic healing after an acute SCAD event. Our study is the largest angiographic series with repeat coronary angiography after SCAD, involving 156 patients (182 noncontiguous lesions), and showed that the majority of SCAD lesions (86.3%) treated conservatively healed spontaneously. There appears to be a time dependency to angiographic healing, whereby 95% of SCAD lesions were healed when angiography was performed ≥30 days after the acute SCAD event.

Although SCAD is relatively uncommon, it is now increasingly recognized as an important cause of acute coronary syndrome among young and middle-aged women. The pathophysiology of MI with SCAD is related to compression of the true lumen by intramural hematoma in the false lumen or by intimal dissection flaps that encroach against the true lumen. The majority of SCAD lesions are due to intramural hematoma without the presence of intimal dissection, as shown by the fact that >75% of angiographic SCAD lesions are type 2 (long diffuse narrowing) and/or type 3 (14). In the absence of instrumentation with angioplasty or stenting, the natural history of SCAD appears to be spontaneous healing with gradual resorption of the intramural hematoma and tacking up of the intimal flap against the deeper arterial walls. There appears to be a time dependency for arterial healing to occur. Early resorption of intramural hematoma had been described to start within days of the dissection on OCT (9). Full resorption of intramural hematoma will likely require weeks to occur, depending on the volume of hematoma within the false lumen. We previously reported that angiographic healing appeared to be complete 26 days post-SCAD in 79 cases (8).

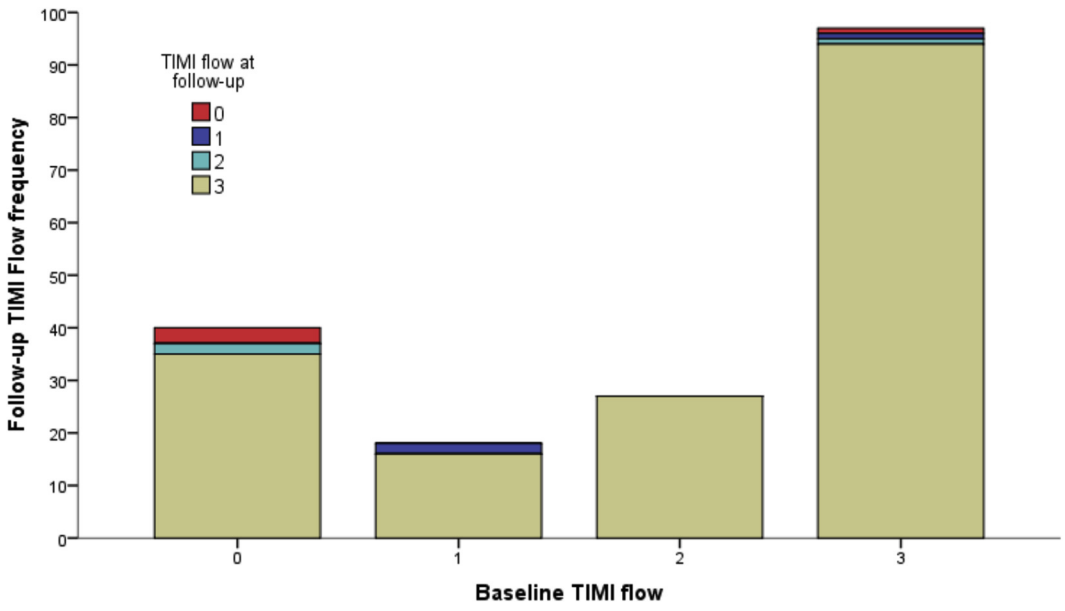
In our present study, we retrospectively reviewed 156 conservatively treated patients (182 SCAD lesions) who underwent repeat coronary angiography to assess the angiographic healing pattern and resorption of intramural hematoma. We confirmed our previous observation that most SCAD lesions heal spontaneously and that there is a time dependency for healing to occur. In this study, the largest repeat angiographic series to our knowledge, we objectively reviewed each repeat angiogram for angiographic

FIGURE 3 Percentage Diameter Stenosis of Spontaneous Coronary Artery Dissection Lesions at Baseline and Follow-Up



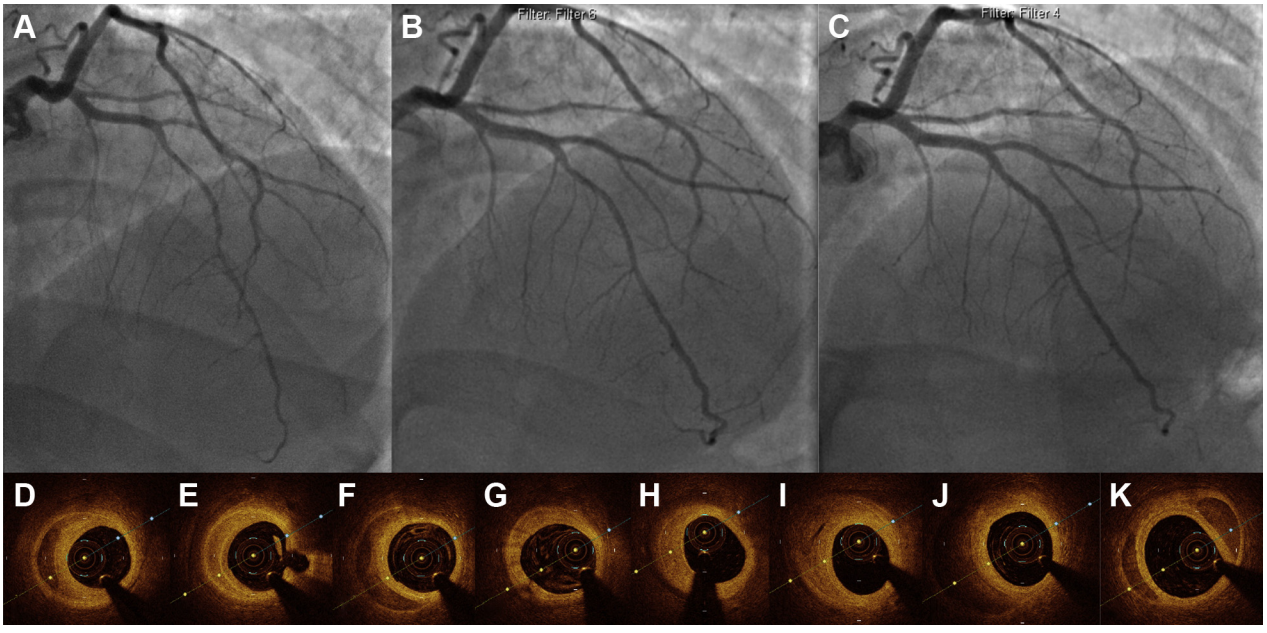
(Left) At baseline. **(Right)** On follow-up repeat angiography.

FIGURE 4 Baseline and Follow-Up Thrombolysis In Myocardial Infarction Flow of 182 Spontaneous Coronary Artery Dissection Lesions



(Horizontal axis) Baseline TIMI (Thrombolysis In Myocardial Infarction) flow. **(Vertical axis)** Follow-up TIMI flow on repeat angiography.

FIGURE 5 Angiographic and Optical Coherence Tomographic Case Example of Healing



(A) Index angiogram showing type 2 spontaneous coronary artery dissection (SCAD) of the mid-left anterior descending coronary artery (LAD). **(B)** Repeat angiogram at 31 days showing partial healed mid-LAD SCAD. **(C)** Subsequent angiogram at 19 months showing angiographic healing of mid-LAD SCAD. **(D to K)** Optical coherence tomographic images obtained (from distal to proximal) at 31 days of the mid-LAD SCAD lesion.

criteria of healing, which required meeting all 3 criteria of 1) improvement of angiographic stenosis; 2) residual stenosis <50%; and 3) normal TIMI flow grade 3. Indeed, 86.3% of 182 SCAD lesions met the criteria of angiographic healing at follow-up angiography. For the remaining 11.5% that did not fulfill the healing criteria, many angiograms were obtained early after the SCAD event, of which 73% were for recurrent MI or recurrent symptoms. Indeed, of the repeat angiograms obtained <30 days from the SCAD event, only about 23% showed angiographic healing, whereas 95% of angiograms performed at ≥30 days showed angiographic healing. We did not identify angiographic characteristics (such as lesion length, stenosis severity, or SCAD angiographic subtype) that predicted spontaneous healing of SCAD lesions. The only baseline clinical differences were worse ejection fraction and early repeat angiography for patients with unhealed lesions.

TABLE 3 Follow-Up Clinical Event Rates in Patients With Spontaneous Coronary Artery Dissection

	Overall (N = 156)	Healed (n = 134)	Unhealed (n = 22)	p Value*
Death	2 (1.3)	2 (1.5)	0 (0)	0.74
Myocardial infarction	56 (35.9)	47 (35.1)	9 (40.9)	0.64
Revascularization	10 (6.4)	9 (6.7)	1 (4.5)	0.57
Stroke	3 (1.9)	3 (2.2)	0 (0)	0.64
MACE	59 (37.8)	50 (37.3)	9 (40.9)	0.81

Values are n (%). *p value comparing patients with healed versus unhealed lesions.
MACE = major adverse cardiac event(s).

Our results are reassuring and support the current recommendation for conservative therapy for low-risk patients with SCAD (i.e., in the absence of hemodynamic instability, ongoing ischemia, or critical anatomy such as left main dissection or multivessel proximal dissections) (8). The 2 key reasons supporting conservative management for SCAD are the challenges associated with PCI for SCAD lesions and the tendency for spontaneous healing of conservatively managed lesions. Retrospective observational studies have shown PCI in SCAD lesions to be associated with a high rate of complications and technical failure (15). These include challenges wiring into the true lumen, extending the intramural hematoma, stenting into false lumen, requirement for long stents, and stent thrombosis (8-10,16,17). However, patients with ongoing ischemia, hemodynamic instability, or critical SCAD anatomy should undergo revascularization if feasible, to maximize myocardial salvage.

TABLE 4 Summary of Studies With Repeat Coronary Angiography Assessing Spontaneous Coronary Artery Dissection Healing With Conservative Management

First Author/Study (Ref. #)	Year	Repeat Angiography	Timing of Repeat Angiography	Spontaneous Healing Results With Conservative Management
Maeder et al. (19)	2005	3 patients	Mean 5.9 mo	100% healed
Alfonso et al. (17)	2012	10 patients	Median 252 days	100% healed
Tweet et al. (20)	2012	13 patients, 17 lesions	Mean 40 mo	52.9% lesions healed 17.6% partially healed
Tweet et al. (10)	2014	59 patients	Median 2.4 yrs	72.9% healed
Saw et al. (8)	2014	88 patients	Early median 5 days Delayed median 161 days	89.8% healed (100% with delayed angiography ≥26 days, 0% with early angiography <20 days)
Rogowski et al. (11)	2015	30 patients	4-6 mo	96.7% healed
This study	2018	156 patients, 182 lesions	Median 154 days	86.3% lesions healed (95% with delayed angiography ≥30 days)

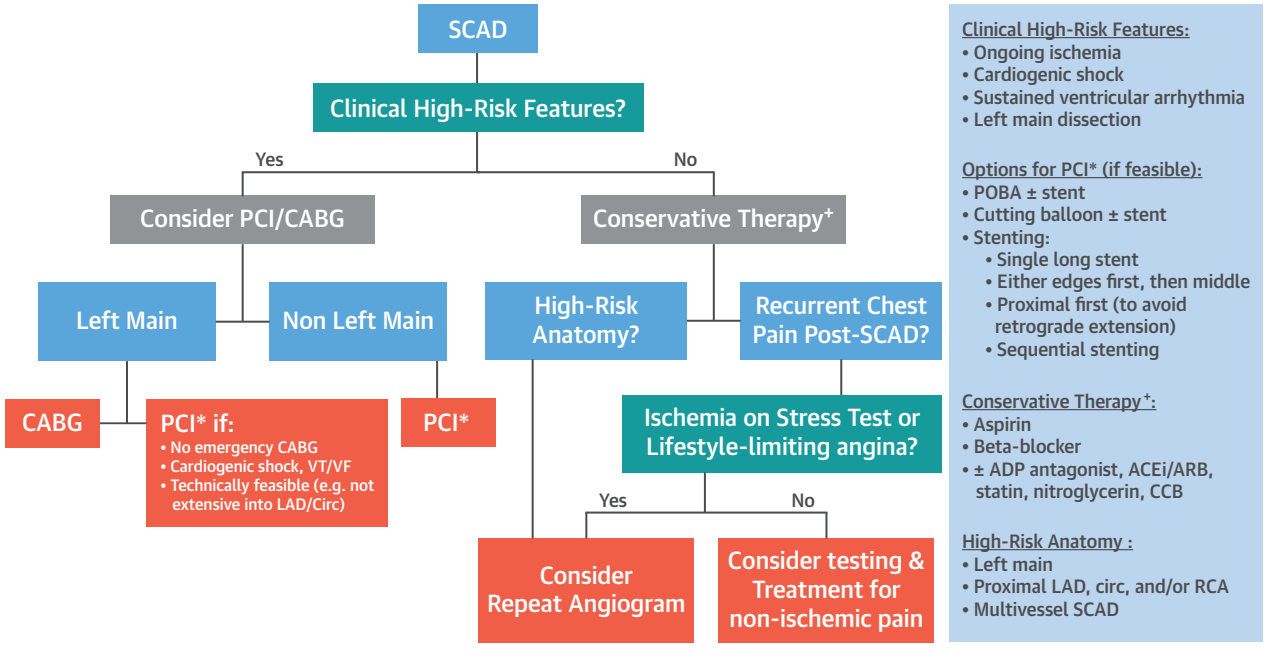
Prior published studies of conservatively managed SCAD lesions were limited to case reports and small retrospective observational studies (1,17-19) (**Table 4**). Maeder et al. (19) reported 5 patients with SCAD and repeat angiography (performed 5.9 months post-SCAD), of whom 1 underwent PCI and 1 underwent coronary artery bypass graft; the 3 treated medically had angiographic healing. In a 45-patient series by Alfonso et al. (17), 16 had revascularization, and repeat angiography was performed in 16 patients (6 were revascularized). Of the 10 patients treated conservatively, dissections healed in 4, improved in 1, progressed in 2 (both required late revascularization), and remained unchanged in 3. Overall, of the 13 patients with SCAD images at discharge (3 residual post-PCI and 10 conservatively treated), 7 (54%) had angiographic healing at late repeat angiography (median 262 days). Of note, patients with dissections due to coronary artery disease (40%) and to isolated SCAD (60%) were included in this series. Furthermore, the investigators included only patients with angiographic longitudinal radiolucent linear images (intimal flap), which excluded patients with type 2 and 3 Saw angiographic SCAD patterns of intramural hematoma. These factors may influence healing rates.

In a Mayo Clinic series of 87 patients with SCAD, 31 were treated conservatively, and 13 underwent repeat angiography (20). Of the 17 initially dissected vessels, 9 demonstrated resolution or near resolution of dissection (52.9%; mean time to repeat angiography 40 months). Partial resolution was seen in 3 of 17 (mean 27 months). This study also excluded type 2 and 3 angiographic SCAD cases of intramural hematoma. In their larger subsequent 189-patient series that included patients with intramural hematoma SCAD pattern, 94 patients were treated conservatively and 59 underwent repeat angiography (10). Of these, 43 (72.9%) had angiographic healing at a median of 2.4 years (IQR: 0.9 to 6.2 years). In a study by Rogowski et al. (11), of 64 patients with SCAD, 56 were treated conservatively, and 30 underwent repeat angiography 4 to 6 months after the index event. All except 1 patient had healed dissection at follow-up (96.7%). This study included patients with type 1 and 2 angiographic SCAD subtypes. Finally, in our prior publication of a 168-patient SCAD cohort, among conservatively managed patients who underwent early repeat coronary angiography <20 days after SCAD (n = 9; median 5 days; range 2 to 19 days), angiographic healing had not occurred yet. However, among conservatively managed patients who underwent elective repeat angiography ≥26 days later (coronary angiography in 74 and computed tomographic angiography in 5), all 79 showed spontaneous angiographic healing (median 161 days) (8).

SCAD affects a different patient population compared with atherosclerotic disease, and the pathophysiology and management are also different. As shown in our cohort, risk factors such as diabetes mellitus, smoking, and dyslipidemia are much lower than in patients with atherosclerotic disease (15). Whereas the latter group with obstructive disease are generally treated with PCI, patients with SCAD are preferentially treated conservatively (as discussed earlier). Furthermore, medical therapy for patients with SCAD typically comprises long-term aspirin and beta-blockers, without the need for statins (non-atherosclerotic disease) or angiotensin-converting enzyme inhibitors (typically preserved left ventricular function) (1). Given that the vast majority of SCAD arteries do heal spontaneously, the rationale for repeat imaging to assess for healing remains unclear. We do not routinely repeat angiography for surveillance post-SCAD; the decision to do so should be individualized. We may consider repeat angiography at our institution if patients have recurrent ischemia with symptoms documented on stress testing, life-style-limiting exertional angina, and/or SCAD involving high-risk anatomy (e.g., left main, proximal arteries, multivessel) with large myocardial jeopardy. Computed tomographic angiography may be considered to assess for healing of left main or large-vessel SCAD (>2.5 mm) instead of coronary angiography. Our suggested algorithm for management and repeat angiography is described in **Central Illustration**.

STUDY LIMITATIONS. Although this is the largest SCAD series with repeat coronary angiography, our

CENTRAL ILLUSTRATION **Suggested Algorithm for Management and Repeat Angiography**



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ACEi = angiotensin-converting enzyme inhibitor; ADP = adenosine diphosphate receptor; CABG = coronary artery bypass graft; CCB = calcium-channel blocker; Circ = circumflex artery; LAD = left anterior descending coronary artery; PCI = percutaneous coronary intervention; POBA = plain old balloon angioplasty; RCA = right coronary artery; VF = ventricular fibrillation; VT = ventricular tachycardia.

study limitations include being retrospective and observational. Repeat angiography was not routinely performed in all patients treated conservatively, but instead the decision to perform repeat coronary angiography was at the discretion of the treating physicians, which may include readmission with MI, recurrent chest pain, multivessel SCAD, or ominous geographic SCAD anatomy. This may result in bias as to the incidence of angiographic healing, because lower risk patients may not have been selected to undergo repeat angiography. Furthermore, performing repeat angiography early may not allow sufficient time for healing to occur (22 SCAD lesions were reassessed at <30 days in this study), which may complicate assessment of healing. Therefore, we segregated our analysis according to early (<30 days) and delayed (≥30 days) repeat angiography.

CONCLUSIONS

In our large series of patients with SCAD who underwent repeat coronary angiography after being treated conservatively, the majority demonstrated spontaneous angiographic healing, defined as improvement in angiographic stenosis to <50% and normal TIMI flow grade 3. There was a time dependency with healing, with the vast majority healed when angiography was performed after 30 days from the SCAD event. Further research should be done to understand why a small proportion of SCAD lesions do not heal spontaneously.

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PERSPECTIVES

WHAT IS KNOWN? SCAD is an important cause of MI in young to middle-aged women. However, very little is known about the timing and angiographic healing characteristics of SCAD.

WHAT IS NEW? In our retrospective cohort of 182 SCAD lesions assessed on repeat coronary angiography, most SCAD lesions (95%) healed after 30 days when treated conservatively. A small proportion remained unhealed.

WHAT IS NEXT? Our understanding of SCAD, although expanding, is still limited, particularly with regard to the factors contributing to poor outcomes, nonhealing, and recurrence. Further research should be done to enhance our knowledge in those regards.

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KEY WORDS acute coronary syndrome(s), angiographic healing, coronary angiography, fibromuscular dysplasia, spontaneous coronary artery dissection