



Revascularization in Patients With Spontaneous Coronary Artery Dissection and ST-Segment Elevation Myocardial Infarction

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ABSTRACT

BACKGROUND Spontaneous coronary artery dissection (SCAD) is an increasingly recognized cause of myocardial infarction (MI) in younger women, often treated conservatively due to revascularization risks. Revascularization outcomes are largely unknown in SCAD presenting with ST-segment elevation myocardial infarction (STEMI).

OBJECTIVES The purpose of this study was to compare revascularization strategies and outcomes of STEMI-SCAD with STEMI atherosclerosis (STEMI-ATH).

METHODS Consecutive STEMI patients were retrospectively analyzed (2003 to 2017) at 2 regional STEMI programs (Minneapolis Heart Institute and Cedars-Sinai Smidt Heart Institute) with 3-year outcomes.

RESULTS Among 5,208 STEMI patients, SCAD was present in 53 (1%; 93% female). SCAD prevalence was 19% in female STEMI patients age \leq 50 years. Compared with STEMI-ATH, STEMI-SCAD patients were younger (age 49 ± 10 years vs. 63 ± 13 years), were more often female (93% vs. 27%), and had more frequent cardiogenic shock (19% vs. 9%); all $p \leq 0.03$. In STEMI-SCAD, the culprit artery was more commonly left main (13% vs. 1%) or left anterior descending (47% vs. 38%); both $p = 0.003$. Acute revascularization was lower in STEMI-SCAD (70% vs. 97%); $p < 0.001$. In STEMI-SCAD, acute revascularization included percutaneous coronary intervention (PCI), $n = 33$ (62%), or bypass grafting, $n = 4$ (8%); PCI success was 91%. Those with revascularization were more likely to have shock, left main culprit, proximal dissection, and initial TIMI (Thrombolysis In Myocardial Infarction) flow grade 0 to 1. The 3-year survival was 98% for STEMI-SCAD versus 84% for STEMI-ATH; $p < 0.001$.

CONCLUSIONS STEMI-SCAD represents an important STEMI subset, particularly among younger women, characterized by significantly greater frequency of left main or left anterior descending culprit and cardiogenic shock than STEMI-ATH. Primary PCI is successful in most STEMI-SCAD patients, with low 3-year mortality. (J Am Coll Cardiol 2019;74:1290-300)
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Spontaneous coronary artery dissection (SCAD) is an increasingly recognized cause of acute myocardial infarction, often afflicting younger women without coronary atherosclerosis (1–5). In SCAD patients, the reported frequency of ST-segment elevation myocardial infarction (STEMI) at presentation is quite variable (26% to 87%) and is largely based on registries and referral networks that may not represent SCAD encountered in real-world practice (1–14).

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Most STEMI events are related to atherosclerotic plaque disruption (STEMI-ATH), in which setting timely reperfusion with primary percutaneous coronary intervention (PCI) is highly effective (15). Systems of care have evolved to provide timely and successful emergent PCI revascularization for STEMI patients (16). However, SCAD represents a different pathophysiological process in which PCI is challenging due to concern for false lumen entry, dissection extension, iatrogenic dissection, and propagation of intramural hematoma (1–5,17–20). PCI failure has been reported in 30% to 40% of SCAD patients (6,8,12), and conservative management of hemodynamically stable patients without ongoing ischemia is associated with favorable outcome (6,12,14,21).

Nonetheless, STEMI-SCAD represents a unique patient population, often with ongoing chest pain and ST-segment elevation, sometimes with hemodynamic instability, in whom a revascularization management decision must be made immediately. In this context, knowledge regarding the role of emergent revascularization for STEMI-SCAD is limited (1–5). Therefore, to further understand the potential role for emergent revascularization in STEMI-SCAD, we compared clinical characteristics, revascularization strategies, and outcomes of STEMI-SCAD with STEMI-ATH among consecutive patients gathered from 2 large U.S. regional STEMI networks.

METHODS

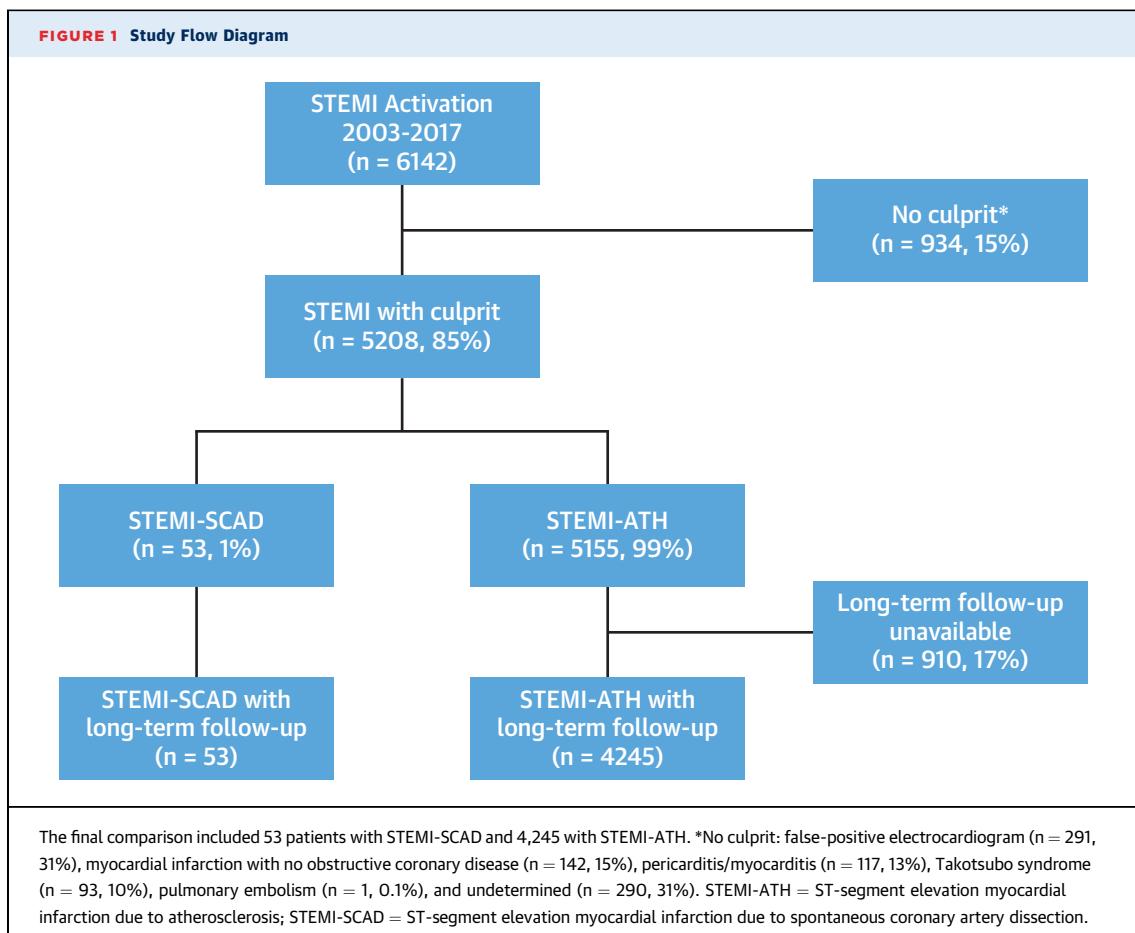
PATIENT POPULATION. From 2003 to 2017, we queried the comprehensive databases of 2 established regional STEMI programs (Minneapolis Heart Institute, Minneapolis, Minnesota; and Cedars-Sinai Smidt Heart Institute, Los Angeles, California) to identify consecutive STEMI patients within 24 h of symptom onset who presented for emergency primary PCI treatment. Clinical and angiographic characteristics, revascularization strategies, and outcomes were compared for consecutive patients with STEMI-SCAD and STEMI-ATH.

Retrospective review of the electronic medical records and STEMI databases at each institution (2003 to 2017) identified 6,142 consecutive STEMI activations. After exclusion of patients without culprit artery and with unavailable long-term follow-up, we identified 4,245 patients with STEMI-ATH and 53 patients with STEMI-SCAD (Figure 1). The diagnoses of STEMI-ATH and STEMI-SCAD (without coexisting coronary atherosclerosis) were made by an experienced interventional cardiologist performing the procedure. In angiographically uncertain cases, intracoronary imaging with optical coherence tomography or intravascular ultrasound was performed (n = 16) at the discretion of the interventional cardiologist. Patients with iatrogenic coronary dissection or admission to the STEMI center >24 h after the acute event were excluded. Coronary angiograms were reviewed by 2 cardiologists (T.D.H., S.W.S.) to confirm SCAD and classified using established angiographic criteria (4,22) as follows: type 1 (retained contrast with evidence of double lumen), type 2 (long diffuse stenosis of variable severity), or type 3 (focal atherosclerosis mimic). Patients not fitting this classification scheme (e.g., occluded distal or side branch vessel with TIMI (Thrombolysis In Myocardial Infarction) flow grade 0 to 1 without double lumen) were considered type 1.

OUTCOMES. Outcomes assessment included all-cause mortality, PCI success, cardiogenic shock, cardiac arrest, nonfatal reinfarction, repeat revascularization, and stroke occurrence in-hospital, at 30 days, and 1 year. Follow-up was conducted by scheduled telephone interview and/or review of electronic medical records. Survival assessment was carried to 3 years, whereas nonfatal reinfarction, repeat revascularization, and stroke assessment was limited to 1 year. PCI success for STEMI-SCAD was defined as improvement in baseline TIMI flow by at least 1 grade or maintenance/improvement of TIMI flow grade 2 to 3 and residual stenosis <50% (12,13). PCI success for STEMI-ATH was defined as final TIMI flow grade 3 and residual stenosis ≤20%. Cardiogenic shock was defined as systolic blood pressure <90 mm Hg requiring vasopressors with clinical hypoperfusion or need for mechanical circulatory support. Repeat revascularization was defined as PCI or coronary artery bypass grafting (CABG) within 1 year of the initial event. Stroke was defined as focal neurological deficit lasting >24 h with imaging evidence of cerebral infarction or intracerebral hemorrhage.

ABBREVIATIONS AND ACRONYMS

CABG	= coronary artery bypass graft
ICD	= implantable cardioverter-defibrillator
LAD	= left anterior descending
PCI	= percutaneous coronary intervention
SCAD	= spontaneous coronary artery dissection
STEMI	= ST-segment elevation myocardial infarction
STEMI-ATH	= ST-segment elevation myocardial infarction due to atherosclerosis
STEMI-SCAD	= ST-segment elevation myocardial infarction due to spontaneous coronary artery dissection
TIMI	= Thrombolysis In Myocardial Infarction



In the STEMI-SCAD subset we included additional endpoints: SCAD progression, SCAD recurrence, stent thrombosis, stent restenosis, need for implantable cardioverter-defibrillator (ICD), and heart transplantation. SCAD progression was defined as a new myocardial ischemic event within 14 days of initial presentation supported by repeat angiography demonstrating progression of the initial dissection to a new critical coronary obstruction, i.e., worsening stenosis with residual $\geq 90\%$ and any reduction in TIMI grade blood flow (12,13). Recurrent SCAD was defined as a new SCAD event with evidence of acute myocardial ischemia and biomarker increase not involving progression of the initial dissection (13,14).

In addition, for long-term survival comparison, we identified 53 patients from the STEMI-ATH population matched for age and sex with the STEMI-SCAD population.

This study was approved by the Institutional Review Boards at Cedars-Sinai Medical Center and the Minneapolis Heart Institute.

STATISTICAL ANALYSES. Descriptive statistics are displayed as mean \pm SD for continuous variables;

number and percentage with characteristic for categorical variables. In the event of skewed distribution of continuous variables, values are reported as median (25th, 75th percentile). Categorical variables were analyzed using Pearson's chi-square or Fisher exact tests, and continuous variables were analyzed using Student's *t*-test or Kruskal-Wallis tests. Time-to-event outcomes were performed using the Kaplan-Meier analysis and compared using log-rank tests. Statistical significance was considered as $p < 0.05$; p values are 2-sided where possible. Statistical calculations and plots were performed with Stata version 14.1 (StataCorp, College Station, Texas).

RESULTS

CLINICAL AND ANGIOGRAPHIC CHARACTERISTICS:

STEMI-SCAD VERSUS STEMI-ATH. Between 2003 and 2017, we identified 5,208 consecutive STEMI patients with culprit artery, of whom 53 (1%, 93% female) presented with STEMI-SCAD and 5,155 (99%) with STEMI-ATH (Figure 1). Long-term follow-up was available for all STEMI-SCAD patients and 4,245 (85%) STEMI-ATH patients (Figure 1). The temporal

prevalence of STEMI-SCAD versus STEMI-ATH was: 2003 to 2007 (4 vs. 1,871; 0.2%), 2008 to 2012 (27 vs. 1,769, 1.5%), 2013 to 2017 (22 vs. 1,515; 1.5%).

Among female STEMI patients age ≤ 50 years, SCAD was present in 19%. Baseline characteristics of the groups are compared in **Table 1**. Patients with STEMI-SCAD were younger, were more commonly female, had lower body mass index, and had fewer coronary risk factors. Among STEMI-SCAD patients, 28 of 53 (53%) were women ≤ 50 years of age. The culprit coronary artery was more commonly the left main or left anterior descending (LAD) coronary artery in STEMI-SCAD versus STEMI-ATH, 60% versus 39% respectively; both $p = 0.003$ (**Table 1**). Initial TIMI flow grade 0 to 1 was present in 53% of STEMI-SCAD patients versus 58% for STEMI-ATH; $p = 0.29$. Dissection origin was proximal or midvessel in $>75\%$ of STEMI-SCAD; noncontiguous multivessel dissection was present in 4 (7.5%) patients. In STEMI-SCAD, the initial ejection fraction was $<50\%$ in 21 (40%) and $<35\%$ in 7 (13%). Cardiogenic shock was present in 19% of those with STEMI-SCAD, twice the frequency observed with STEMI-ATH, whereas cardiac arrest prevalence was similar (**Table 1**). Hospital length of stay was significantly longer for STEMI-SCAD, median 4 days (interquartile range: 2 to 5 days) versus 3 days (interquartile range: 2 to 4 days) for STEMI-ATH; $p = 0.037$. STEMI-SCAD length of stay was influenced by 3 patients with pregnancy-associated left main dissection (length of stay 17, 29, and 48 days, respectively).

Thrombolytic therapy was administered to 4 (7.5%) STEMI-SCAD patients and 829 (19.5%) STEMI-ATH patients as part of a pharmacoinvasive strategy for patients emergently transferred from distant non-PCI facilities (**16**).

ACUTE REVASCULARIZATION: STEMI-SCAD VERSUS STEMI-ATH. The revascularization rate was significantly lower in STEMI-SCAD versus STEMI-ATH: 70% versus 97%, respectively; $p < 0.001$. In STEMI-SCAD, acute treatment included PCI stent ($n = 31$; 58%), PCI balloon angioplasty ($n = 3$; 6%), CABG ($n = 3$; 6%), PCI stent attempt followed by CABG ($n = 1$; 2%), and medical management ($n = 16$; 30%) (**Figure 2**). In 13 patients, revascularization was performed in the presence of initial TIMI flow grade 2 or 3 (**Table 2**). The decision to perform emergent revascularization in this subset was influenced by the presence of ongoing chest pain and/or hemodynamic instability with ST-segment elevation. Additional factors included culprit stenosis $> 90\%$ ($n = 8$), culprit left main with hemodynamic instability treated with CABG ($n = 1$), culprit left main with extensive intimal flap treated with CABG ($n = 1$), culprit LAD with long stenosis

TABLE 1 Characteristics of STEMI-SCAD Versus STEMI-ATH

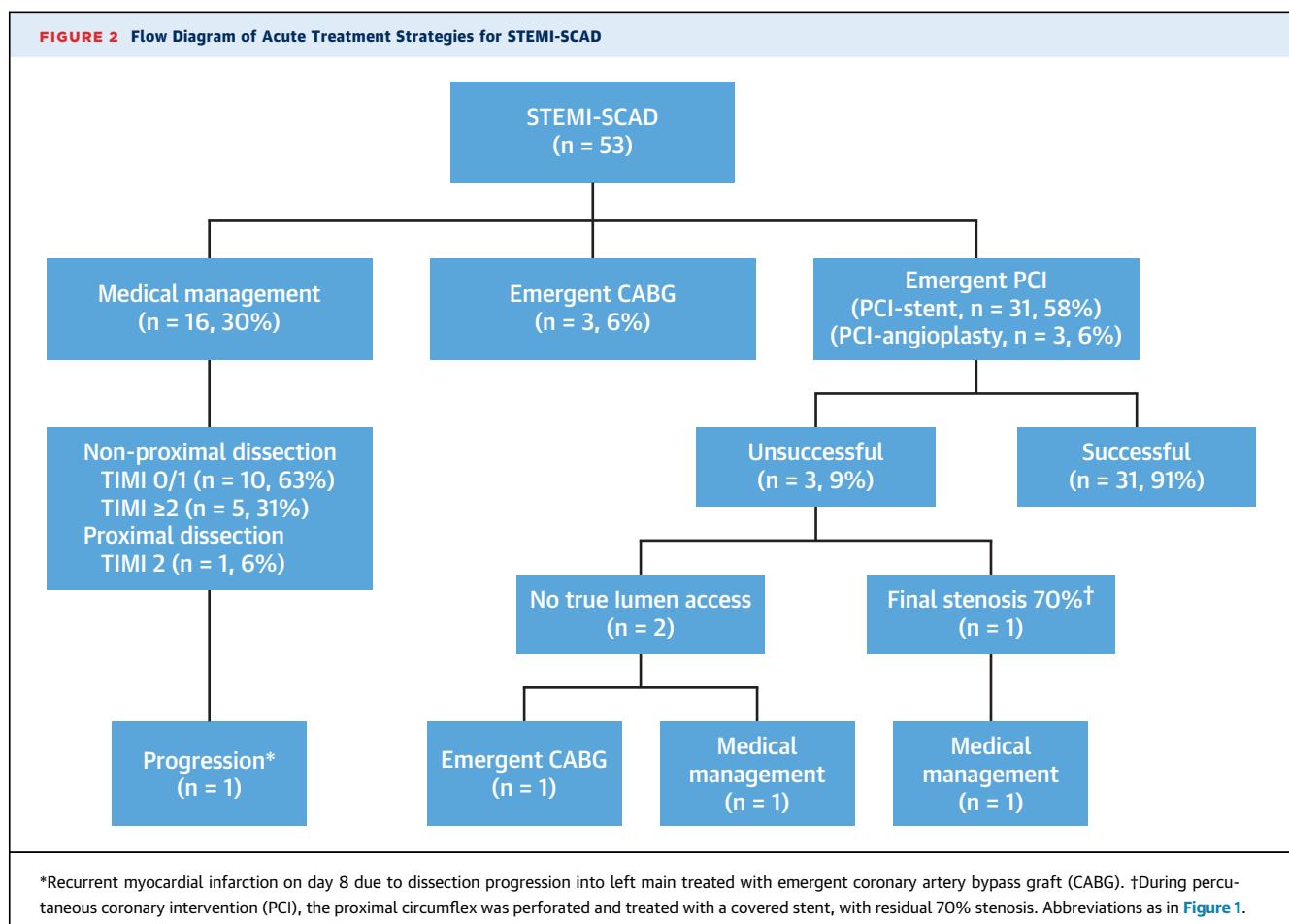
	STEMI-SCAD (n = 53)	STEMI-ATH (n = 4,245)	p Value
Age, yrs	49.4 \pm 10.2	63.3 \pm 13.4	<0.001
Female	49 (92.5)	1,124 (26.5)	<0.001
BMI, kg/m ²	26.6 \pm 5.3	29.2 \pm 5.8	0.001
Hypertension	20 (37.7)	2,462 (58.3)	0.003
Hyperlipidemia	17 (32.1)	2,333 (56.2)	<0.001
Diabetes	3 (5.7)	771 (18.3)	0.018
Tobacco use	22 (41.5)	2,642 (62.7)	0.002
Family history	17 (32.7)	1,803 (46.3)	0.050
Presentation location			0.006
PCI facility	25 (47.2)	1,215 (28.6)	
Referral facility	28 (53.8)	3,030 (71.4)	
Culprit artery			<0.001
Left main	7 (13.2)	49 (1.2)	
Left anterior descending	25 (47.2)	1,608 (37.9)	
Right coronary artery	11 (20.8)	1,837 (43.4)	
Circumflex	10 (18.9)	630 (14.9)	
Bypass graft	0 (0.0)	114 (2.7)	
Dissection location*		NA	NA
Proximal	17 (32.1)		
Mid	23 (43.4)		
Distal	13 (24.5)		
SCAD class		NA	NA
I	29 (54.7)		
II	20 (37.7)		
III	4 (7.5)		
Initial ejection fraction, %	50.3 \pm 13.5	47.8 \pm 13.0	0.17
Initial TIMI flow grade			0.29
0/1	28 (52.8)	2,442 (57.5)	
2	12 (22.6)	839 (20.0)	
3	13 (24.5)	922 (21.9)	
Cardiogenic shock	10 (18.9)	385 (9.1)	0.026
Circulatory support†	6 (11.3)	378 (9.5)	0.66
Cardiac arrest	8 (15.1)	484 (11.4)	0.40
Revascularization	37 (69.8)	4,131 (97.3)	<0.001
PCI/PTCA	33 (62.3)	4,032 (94.9)	
CABG	4 (7.5)	99 (2.3)	

Values are mean \pm SD or n (%). *The most proximal dissection location was reported. †Circulatory support: intra-aortic balloon pump, extracorporeal membrane oxygenation, or catheter left ventricle assist device.

BMI = body mass index; CABG = coronary artery bypass grafting; PCI = percutaneous coronary intervention; PTCA = percutaneous transluminal coronary angioplasty; SCAD = spontaneous coronary artery dissection; STEMI-ATH = ST-segment elevation myocardial infarction due to atherosclerosis; STEMI-SCAD = ST-segment elevation myocardial infarction due to spontaneous coronary artery dissection; TIMI = Thrombolysis In Myocardial Infarction.

($n = 2$), and culprit proximal circumflex with long stenosis and extensive intimal flap ($n = 1$). Among PCI-treated patients, successful revascularization was achieved in 31 patients (91%) while PCI was unsuccessful in 3 (9%) due to failure to access true lumen ($n = 2$) or residual stenosis $> 50\%$ ($n = 1$) (**Figure 2**). Final TIMI flow grade 3 was achieved in 91% of PCI-treated STEMI-SCAD versus 98% of STEMI-ATH; $p = 0.016$ (**Figure 3**).

The number of stents per patient for STEMI-SCAD versus STEMI-ATH was: 1 stent (16.7% vs. 63%), 2 stents (36.7% vs. 25.5%), and ≥ 3 stents (46.7% vs.



*Recurrent myocardial infarction on day 8 due to dissection progression into left main treated with emergent coronary artery bypass graft (CABG). †During percutaneous coronary intervention (PCI), the proximal circumflex was perforated and treated with a covered stent, with residual 70% stenosis. Abbreviations as in Figure 1.

11.5%), respectively; $p < 0.001$. In STEMI-SCAD, mean stent length was 62 ± 37 mm (range 12 to 140 mm). STEMI-SCAD patients with attempted revascularization were more likely to have cardiogenic shock, left main or LAD culprit artery, proximal dissection location, and initial TIMI flow grade 0 to 1 (Table 2). Acute revascularization was not attempted in 16 (30%) of STEMI-SCAD patients most commonly due to nonproximal dissection in hemodynamically stable patients (Figure 2).

LEFT MAIN CULPRIT IN STEMI-SCAD. The left main was the culprit coronary artery in 7 (13%) STEMI-SCAD patients, ages 32 to 53 years, each of whom developed cardiogenic shock. Revascularization was attempted in each patient, 4 with PCI stent (3, 1, 6, and 5 stents, respectively) and 3 with CABG. Among PCI stent-treated patients, there was a single revascularization failure due to hematoma propagation from left main into circumflex and LAD with concomitant circumflex perforation (Figure 2).

CARDIOGENIC SHOCK AND CARDIAC ARREST IN STEMI-SCAD. Cardiogenic shock was present in 10 (19%) STEMI-SCAD patients, among whom the culprit

coronary artery was the left main (n = 7), circumflex (n = 1), LAD (n = 1), and right coronary artery (n = 1). Initial flow grades were TIMI flow grade 0 to 1 (n = 6), TIMI flow grade 2 (n = 2), and TIMI flow grade 3 (n = 2). Revascularization was attempted in each patient: PCI stent (n = 6), PCI balloon angioplasty (n = 1), and CABG (n = 3). Mechanical circulatory support was necessary in 4 patients, and included intra-aortic balloon pump (n = 2), combined intra-aortic balloon pump and left ventricular assist device (n = 1), and combined intra-aortic balloon pump, Impella device (Abiomed, Danvers, Massachusetts), and extracorporeal membrane oxygenation (n = 1).

Cardiac arrest occurred in 8 (15%) patients and was due to ventricular fibrillation (n = 5) and/or ventricular tachycardia (n = 3). Cardiac arrest location was field (n = 1), ambulance (n = 2), referring hospital emergency department (n = 4), and catheterization laboratory (n = 1).

PREGNANCY-ASSOCIATED STEMI-SCAD. Pregnancy associated STEMI-SCAD occurred in 7 (13%) patients, mean age 36 years (range 29 to 41 years), at 6.6 ± 10.2 weeks (range 1 to 28 weeks) postpartum. The

culprit vessel was the left main in 4 of 7 (57%), treated successfully with PCI stent in 2 and CABG in 2. The culprit vessel in the remaining 3 patients was mid-LAD (successful PCI stent), proximal LAD (successful PCI stent), and LAD/RCA, that is, multivessel SCAD (conservative treatment). Hospital length of stay was 16.1 ± 17 days (range 1 to 48 days). At follow-up, significant LV dysfunction was present in 4 patients (ejection fraction 36%, 33%, 32%, and 30%, respectively), 3 of whom had initial left main dissection.

EARLY AND LATE OUTCOMES IN STEMI-SCAD.

Revascularization after hospital discharge was necessary during the first year in 5 (9%) patients, 4 of whom were treated with initial PCI stent and 1 of whom was initially treated conservatively (mid-LAD dissection, TIMI flow grade 3) and experienced recurrent myocardial infarction 8 days after presentation due to dissection progression into the left main requiring CABG (**Figure 2**).

Stent thrombosis occurred in 1 (2%) patient during the first year, involving left main and left anterior descending drug-eluting stents while receiving aspirin and clopidogrel, and presenting as unstable angina. This event was attributed to stent malaposition caused by coronary intramural hematoma resorption, confirmed by intravascular ultrasound. At 1 year, in-stent restenosis occurred in 5 (9%) patients, 2 of whom had recurrent restenosis.

Among all STEMI-SCAD patients, recurrent myocardial infarction occurred in 2 (3.7%) patients during the first year, due to early dissection progression (noted in the previous text) and presumed vasospasm, respectively. No patient had recurrent SCAD during the first year. Stroke occurred in 2 (3.7%) patients within the first year.

In STEMI-SCAD, ejection fraction improved from $50 \pm 14\%$ to $55 \pm 14\%$ at follow-up (mean 6 months) and was $\geq 55\%$ in 35 (65%) patients and $< 35\%$ in 6 (11%) patients. A primary prevention ICD was used in 8 (15%) STEMI-SCAD patients, 3 of whom were treated with PCI, 3 with CABG, and 2 without revascularization. Cardiac transplantation was performed because of post-infarction heart failure in 2 patients, a 51-year-old woman and 38-year-old man, both with culprit left main dissection.

Survival at 3 years was significantly greater in STEMI-SCAD (98%) versus STEMI-ATH (84%) versus age- and sex-matched STEMI-ATH (72%); $p < 0.001$ (**Figure 4**). In the STEMI-SCAD population there was 1 (1.9%) in-hospital death, with culprit proximal right coronary artery dissection, cardiogenic shock, and recurrent ventricular fibrillation in the catheterization laboratory. This patient (female, age 64 without significant comorbidity) had TIMI flow grade 1 after

TABLE 2 STEMI-SCAD Characteristics With Versus Without Attempted Revascularization

	STEMI-SCAD Attempted Revascularization (n = 37)	STEMI-SCAD Medical Management (n = 16)	p Value
Age, yrs	49.6 ± 10.3	49.0 ± 10.3	0.85
Culprit artery			0.29
Left main	7 (18.9)	0 (0.0)	
Left anterior descending	17 (46.0)	8 (50.0)	
Right coronary artery	7 (18.9)	4 (25.0)	
Circumflex	6 (16.2)	4 (25.0)	
Dissection location*			0.027
Proximal	16 (43.2)	1 (6.3)	
Mid	14 (37.8)	9 (56.3)	
Distal	7 (18.9)	6 (37.5)	
Initial TIMI flow			0.032
0/1	24 (64.8)	5 (31.3)	
2	8 (21.6)	4 (25.0)	
3	5 (13.5)	7 (43.8)	
Initial ejection fraction, %	47.3 ± 14.5	57.2 ± 8.0	0.014
Cardiogenic shock	10 (27.0)	0 (0.0)	0.023
Cardiac arrest	6 (16.2)	2 (12.5)	0.73
PCI stent	30 (81.1)	NA	NA
PCI angioplasty	3 (8.1)		
CABG	4 (10.8)		

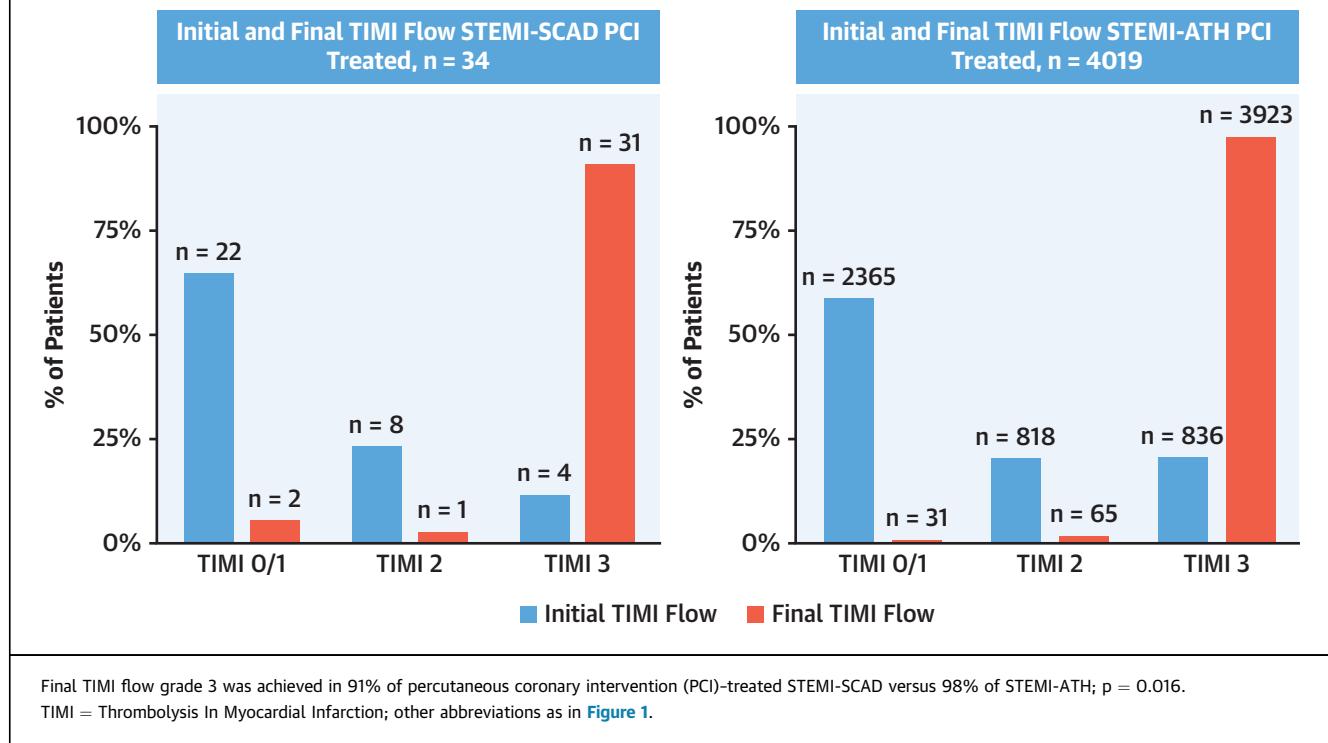
Values are mean \pm SD or n (%). *The most proximal dissection location was reported.

Abbreviations as in Table 1.

PCI stent and subsequently died at 24 h from refractory cardiogenic shock. No STEMI-SCAD deaths occurred after hospital discharge to 3 years.

DISCUSSION

SCAD represents a unique and increasingly recognized cause of acute myocardial infarction with predilection for younger women (1–5,13,23). To our knowledge, this is the largest study to compare revascularization strategies and outcomes in STEMI-SCAD with STEMI-ATH (**Central Illustration**). The results, gathered from consecutive patients with initial presentation at 2 large regional STEMI centers, document SCAD prevalence of 1% among all STEMI patients and 19% among STEMI women ≤ 50 years of age. During the latter years of this study, STEMI-SCAD prevalence approached 2% of all STEMI cases due to increased recognition of this condition. Patients with STEMI-SCAD were younger and more commonly female (53% women age ≤ 50 years), with fewer coronary atherosclerosis risk factors than those with STEMI-ATH, a clinical profile that should heighten concern for presence of SCAD at initial STEMI presentation. The reported STEMI prevalence in SCAD ranges from 26% to 87% (4,9), although in the largest registries, STEMI prevalence was 25% to 40% (6,12,14,21). In the Vancouver experience, SCAD was

FIGURE 3 Initial and Final TIMI Flow for STEMI-SCAD Versus STEMI-ATH

Final TIMI flow grade 3 was achieved in 91% of percutaneous coronary intervention (PCI)-treated STEMI-SCAD versus 98% of STEMI-ATH; $p = 0.016$.
 TIMI = Thrombolysis In Myocardial Infarction; other abbreviations as in Figure 1.

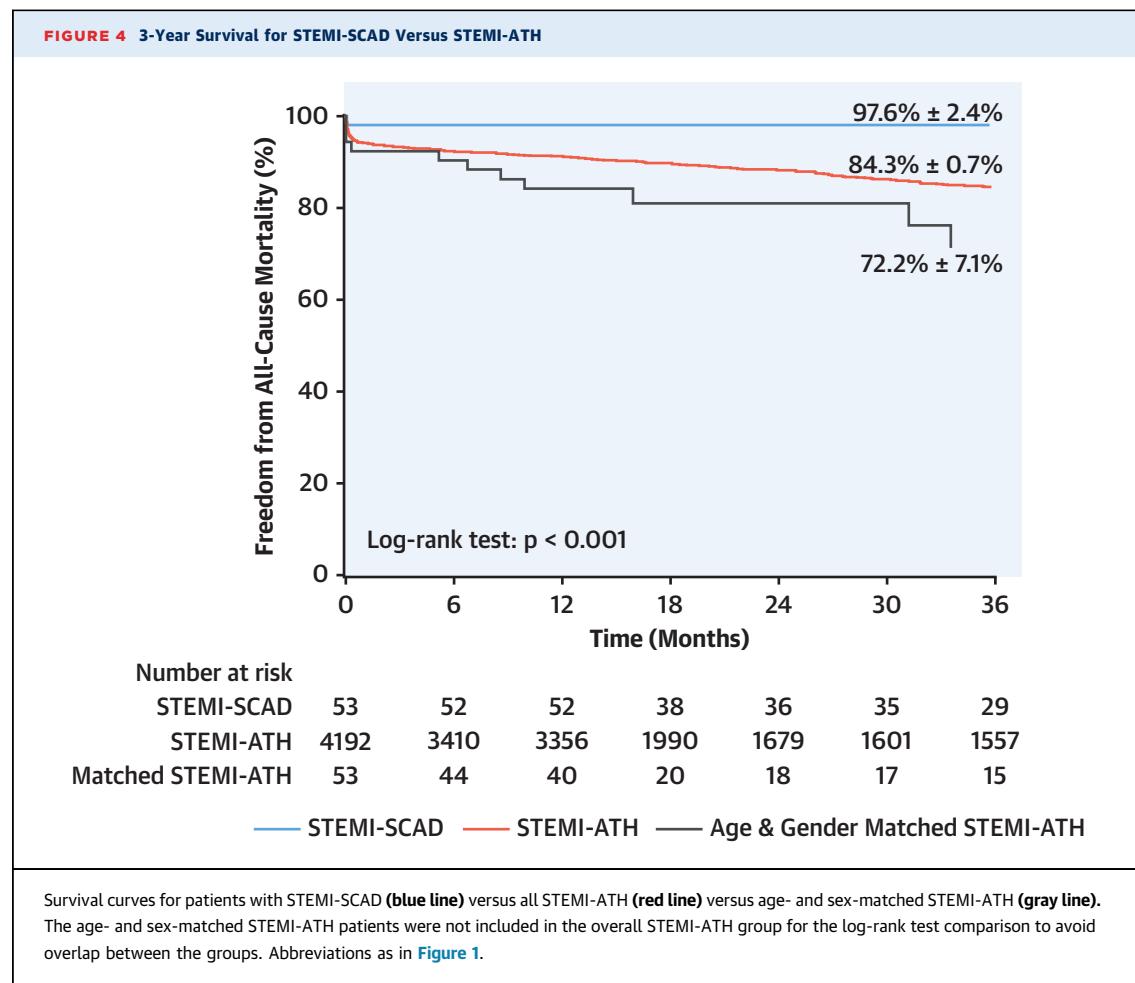
the cause of acute myocardial infarction (STEMI and non-STEMI) in 24% of women age ≤ 50 years undergoing angiography for myocardial infarction (23). These figures likely represent an underestimate given the diagnostic challenges of identifying SCAD among patients with acute myocardial infarction. Several findings from our study are relevant for physicians participating in the care of STEMI patients.

REVASCULARIZATION STRATEGY. Revascularization rates were significantly lower in STEMI-SCAD (70%) than STEMI-ATH (97%), likely representing the prevailing view favoring conservative PCI use in hemodynamically stable SCAD with distal dissection or perceived risk of further vessel injury. Among STEMI patients treated with revascularization, PCI stent was the dominant method used in both STEMI-SCAD and STEMI-ATH, although stent number was significantly greater in STEMI-SCAD. Whether the increased stent burden in STEMI-SCAD carries risk of stent thrombosis or restenosis beyond 1 year in these younger patients requires further study (24).

Achievement of post-PCI TIMI flow grade 3 was significantly greater in STEMI-ATH than in STEMI-SCAD. Contemporary guidelines for PCI success may not be appropriate for STEMI-SCAD (12,13). Indeed, some have proposed different criteria for PCI success

in SCAD including improved TIMI flow grade > 1 when initial TIMI flow grade is 0 to 1, or maintenance of TIMI flow grade 2 to 3 with $< 50\%$ residual stenosis, whereas unsuccessful PCI has been defined as worsened TIMI flow grade, progression of dissection plane requiring placement of > 2 additional stents, or conversion to urgent CABG (12,13).

LEFT MAIN DISSECTION. In our study, STEMI-SCAD was characterized by a substantial frequency of left main dissection (13%). Patients were treated with a left main PCI stent beginning in 2009, reflecting the relatively recent emergence of PCI stent as treatment for left main stenosis/occlusion. Each patient with left main dissection developed cardiogenic shock. In previous SCAD studies (involving patients with both STEMI and non-STEMI), the frequency of left main involvement was quite variable, ranging from 0.6% to 7% (12-14,25). Left main dissection presents a formidable management challenge requiring execution of both emergent hemodynamic support and successful revascularization. Experience with left main SCAD PCI stent revascularization is limited to a few case reports (26-28). Left main PCI stent carries the risk of dissection propagation into the LAD and circumflex coronary arteries and, in some circumstances, may not be technically feasible, thereby necessitating



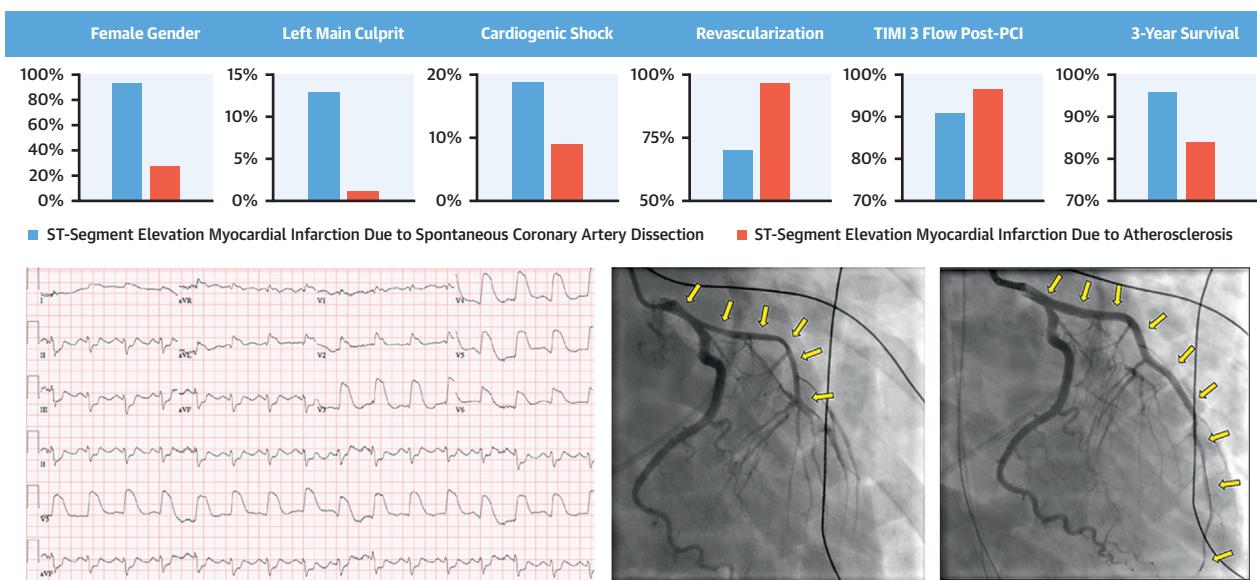
emergent CABG. Our study demonstrated successful left main revascularization with PCI stent in 3 of 7 patients, although mechanical circulatory support was often necessary during this complex intervention. The appropriate revascularization strategy for left main coronary dissection requires patient-specific consideration of hemodynamic status, dissection extent, initial TIMI flow grade, and risk of antiplatelet agents in the event of CABG.

CARDIOGENIC SHOCK. Notably, cardiogenic shock was present in 19% of STEMI-SCAD patients in our study, a frequency twice that of STEMI-ATH, and most often associated with left main dissection. Mechanical circulatory support was necessary in 11% of SCAD patients. The prevalence of cardiogenic shock in SCAD (STEMI and non-STEMI) is largely unknown (4,5). Referral-type SCAD registries may not capture patients with cardiogenic shock who do not survive initial hospitalization. Management of SCAD-associated cardiogenic shock is limited to a few case

reports documenting successful use of intra-aortic balloon pump, extracorporeal membrane oxygenation, and left ventricular assist devices to support these patients during PCI or surgical revascularization or as bridge to cardiac transplantation (29–34). The use of mechanical circulatory support in the SCAD population carries the risk for iliac or femoral arterial injury in this largely female population (many with coexisting fibromuscular dysplasia or connective tissue disease), as well as potential for propagation of coronary artery dissection during diastolic pressure augmentation when using intra-aortic balloon pump (4). The results from our study demonstrate the need to consider the use of mechanical circulatory support when managing this unique STEMI population.

OUTCOMES. The aggressive revascularization strategy for STEMI-SCAD documented in our study was associated with favorable 3-year survival. Although urgent revascularization with primary PCI represents the standard of care for STEMI-ATH patients, this

CENTRAL ILLUSTRATION ST-Segment Elevation Myocardial Infarction Due to Spontaneous Coronary Artery Dissection Versus ST-Segment Elevation Myocardial Infarction Due to Atherosclerosis



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(Top) Characteristics of ST-segment elevation myocardial infarction due to spontaneous coronary artery dissection (STEMI-SCAD) versus ST-segment elevation myocardial infarction due to atherosclerosis (STEMI-ATH). **(Bottom left)** Initial electrocardiogram from a 35-year-old woman with anterior ST-segment elevation myocardial infarction due to left main and left anterior descending spontaneous coronary artery dissection. **(Bottom middle)** Initial left coronary angiogram demonstrates extensive coronary dissection (yellow arrows) beginning in the left main coronary and extending into proximal and mid-left anterior descending coronary artery. **(Bottom right)** Left coronary angiogram demonstrates restoration of TIMI flow grade 3 after placement of 3 drug-eluting stents into the left main and left anterior descending coronary artery. PCI = percutaneous coronary intervention; TIMI = Thrombolysis In Myocardial Infarction.

strategy has not been formally tested in STEMI-SCAD. Historically, PCI revascularization in SCAD is associated with technical failure in >30% (6,12), with risk for false lumen stenting, dissection extension, and intramural hematoma propagation, leading to current consensus for conservative management in the absence of ongoing ischemia or hemodynamic instability (2,4,5). However, most patients (60% to 70%) in these studies presented with non-STEMI (6,12). For example, in the recent Vancouver experience (n = 327), non-STEMI comprised 74% of patients and initial TIMI flow grade 3 was present in 67% (13), whereas initial TIMI flow grade 3 was present in only 25% of patients in our study. Therefore, patients with STEMI-SCAD represent a unique subset of the SCAD spectrum with ongoing ischemia, proximal or mid-vessel dissection, and frequent hemodynamic instability, in whom conservative management is an unsatisfactory alternative. Our study demonstrates that a traditional primary PCI strategy for STEMI-SCAD is effective in the substantial majority of patients, with technical success only modestly lower

than STEMI-ATH and infrequent need for urgent CABG. Despite the favorable STEMI-SCAD survival in our study, other adverse outcomes, including need for cardiac transplantation, residual LV dysfunction, stent thrombosis or restenosis, and need for ICD, demonstrate the significant risks imposed by STEMI-SCAD.

STUDY LIMITATIONS. The number of STEMI-SCAD patients in this study was small, the analysis was retrospective, and the treatment was non-randomized, thereby limiting generalization to the larger SCAD population. A number of interventionists were involved over the 15 years of this study with no standardized management strategy for STEMI-SCAD. During the more recent years of this study, awareness of SCAD as an important cause of acute myocardial infarction in younger women substantially broadened and the angiographic description of type 2 SCAD first emerged (6). We did not review the angiograms of patients classified as STEMI-ATH for presence of type 2 SCAD; therefore, the prevalence of STEMI-SCAD reported in this paper

likely underestimates the real-world incidence. Further, the current SCAD classification scheme (22) does not adequately reflect the full scope of angiographic findings observed in STEMI-SCAD. Distal side branch occlusions (diagonal, obtuse-marginal, or posterior descending artery) with no clear dissection plane were sometimes noted, an anatomic variant that does not easily conform with the current classification scheme.

CONCLUSIONS

STEMI-SCAD represents an important subset of the STEMI population, with notable predilection for younger women and characterized by significantly greater prevalence of left main or LAD culprit and cardiogenic shock than STEMI-ATH. Primary PCI is successful in most patients with favorable 3-year mortality, although the revascularization approach is complex and differs from STEMI-ATH. Regional STEMI centers should consider formulation of management plans that address the unique characteristics of STEMI-SCAD patients.

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