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CLINICAL INVESTIGATIONS

Changes in left ventricular function after spontaneous coronary artery dissection

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Background: Spontaneous healing of spontaneous coronary artery dissection (SCAD) and left ventricular ejection fraction (LVEF) recovery is frequently observed clinically. However, LVEF on presentation and follow-up imaging has not been described.

Hypothesis: We hypothesize that LV dysfunction improves at follow-up after initial SCAD presentation.

Methods: We included patients with nonatherosclerotic SCAD prospectively followed at Vancouver General Hospital, who had baseline assessment of LVEF and wall-motion abnormality (WMA) during their index presentation. A subset of these patients had repeat assessment of their ventricular function at follow-up. We compared the baseline LVEF and WMA with follow-up assessments and correlated to long-term cardiovascular outcomes.

Results: We included 277 SCAD patients who had baseline ventricular assessment performed. The average age was 52.4 $\pm$ 9.4 years, and 90.3% were female. All presented with myocardial infarction (24.2% STEMI, 75.8% NSTEMI). At baseline, the mean LVEF was 55.6% $\pm$ 9.1% and 72/277 (26.0%) had LVEF <50%. The presence of WMA was observed in 237/277 (85.6%) cases. Of 164 patients with repeat assessments, the baseline LVEF was 54.6% $\pm$ 9.2%, with improvement to 60.7% $\pm$ 7.2% at follow-up ($P < 0.001$). Baseline LVEF of <50% was observed in 29.9%, but only 6.7% had LVEF <50% at follow-up ($P < 0.001$). Baseline WMA was observed in 87.2% but decreased to 44.5% at follow-up ($P < 0.001$). Multivariable analysis showed that presentation with STEMI (odds ratio [OR]: 2.71, $P = 0.001$), troponin I >50 μg/L (OR: 1.02, $P = 0.005$), and SCAD involvement of the LAD (OR: 2.5, $P = 0.002$) were independent predictors of baseline LVEF <50%.

Conclusions: In our large, prospectively followed SCAD cohort, the majority of patients presented with WMA and had relatively normal LVEF. Over half had subsequent normalization of WMA and LVEF on follow-up assessment.

KEYWORDS

spontaneous coronary artery dissection, left ventricular function, wall motion abnormality

1 | INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a clinically challenging entity that is an important cause of acute coronary syndrome (ACS) in women.[1] With fewer than 1500 cases published in the medical literature, previous retrospective studies have reported SCAD in 0.07% to 1.1% of all coronary angiograms and as a causative factor in 0.1% to 0.4% of ACS and 0.4% of sudden cardiac death.[2] However, in series with careful review for angiographic features of SCAD, the reported prevalence is much higher, accounting for 8.7% of troponin (Tn)-positive ACS[3] and 24% of women age ≤50 years with myocardial infarction (MI).[4] Moreover, we recently described much higher rates of major adverse cardiac events (MACE) of 10% to 20% in our prospectively followed cohort of angiographically confirmed SCAD.[5] Taken together, these data suggest that SCAD is more prevalent than previously observed and represents a significant burden of cardiovascular risk. At present, however, the true population-based incidence of SCAD, as well as the natural history of this clinically important entity, remain poorly described.

SCAD is defined as a nontraumatic and noniatrogenic separation of the coronary arterial wall. The dissection plane can occur at the intima and result in the creation of a false lumen. Alternatively, spontaneous disruption of the adventitial vasa vasorum can occur, causing separation of the intimal-medial-adventitial interface with intramural hematoma.[6,7] The etiology of SCAD can be multifactorial, frequently with contribution of both a predisposing arteriopathy (resulting in vulnerable vessel-wall segments) as well as precipitating stressors. Predisposing arteriopathies can be broadly classified as atherosclerotic and nonatherosclerotic (NA-SCAD),[5] which affect different populations with distinct cardiovascular risks, angiographic profiles, and management strategies.

In our recent report of our 168-patient prospectively followed NA-SCAD cohort, spontaneous angiographic arterial healing was observed in 79 of 79 patients with angiographic follow-up who were managed conservatively.[5] This observation may reflect the natural history of coronary dissection and could provide a rationale for a conservative management strategy in stable patients. We also observed that left ventricular (LV) function and wall-motion abnormality (WMA) tended to improve after the acute SCAD event on repeat imaging, which has not been previously described. We hypothesize that myocardial stunning is a major cause of LV dysfunction in patients presenting with SCAD, which can subsequently normalize after vessel healing. Therefore, we sought to evaluate the change in LV function and WMA at baseline presentation of SCAD and on repeat follow-up imaging in our cohort of prospectively followed SCAD patients.

2 | METHODS

2.1 | Study population

We included NA-SCAD patients prospectively followed at Vancouver General Hospital, which is a quaternary referral center for prospectively and retrospectively identified SCAD patients in British Columbia. Patients judged to have atherosclerosis as the underlying condition causing SCAD were excluded. All patients provided informed consent as approved by the institutional review board and in accordance with the Declaration of Helsinki. All patients with LV function assessment during the acute SCAD presentation comprised the study cohort (N = 277). Thirteen patients with repeat LV assessment were excluded from the main cohort comparison: 10 because of interval repeat MI prior to their reassessment and 3 because of revascularization complications, including iatrogenic guide catheter-induced coronary dissection. The remaining 164 patients were included in the analysis comparing baseline to follow-up left ventricular ejection fraction (LVEF) and WMA.

2.2 | Baseline characteristics

Patient data were obtained from a combination of patient interviews and patient-completed questionnaires and were extracted from hospital admission records and relevant clinical source documents from physician offices. Baseline characteristics, cardiovascular risk factors, medications, hospital presentation, electrocardiogram changes, angiographic and noninvasive imaging characteristics, and in-hospital and follow-up cardiovascular events were recorded.

2.3 | Angiographic SCAD diagnosis and classification

All coronary angiograms and SCAD diagnoses were confirmed by 2 experienced cardiologists. Our diagnostic SCAD criteria on angiography and classification (type 1, 2, or 3) were previously described.[8] The diagnosis of SCAD on optical coherence tomography or intravascular ultrasound required visualization of intramural hematoma and/or separation of the intimomedial membrane creating a double lumen.[9] Coronary segments were defined by the Bypass Angioplasty Revascularization Investigation (BARI) classification.[10] The number of dissected coronary artery segments, location, lesion characteristics (stenosis severity and lesion length), and corresponding WMAs were recorded. Repeat coronary angiography after the index event was performed at the discretion of the treating physicians, and results from repeat coronary angiography or intracoronary imaging were recorded.

2.4 | Assessment of ventricular function

All patients included in this study cohort had an index assessment of LV function at their SCAD presentation by either catheter ventriculography or echocardiography using standard techniques. For catheter ventriculograms, LVEF was calculated using the Simpson method with manual tracing of end-systolic and end-diastolic volumes. WMAs were reported by the invasive cardiologists as normal, hypokinetic, akinetic, or dyskinetic, and the segments involved in the right anterior oblique projections were anterobasal, anterolateral, apical, inferior, and posterobasal. Echocardiograms were reviewed by echocardiographers, and LVEF was assessed both visually and using the Simpson biplane method. WMAs on echocardiograms were assessed using a 16-segment model. The lowest LVEF was recorded for the purpose of this study. Repeat assessment of LVEF and WMA were performed according to the treating physicians. We compared the baseline and follow-up LVEF and WMA in the cohort of patients who had repeat LV assessment performed.

2.5 | Cardiovascular events

In-hospital MACE of all-cause mortality (cardiac mortality), stroke, reinfarction, 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 (cardiac mortality), stroke, recurrent MI (including recurrent dissection), congestive heart failure admission, and revascularization were recorded.

2.6 | Statistical analysis

Descriptive statistics were used to report the patient baseline characteristics. Continuous variables were summarized as mean $\pm$ SD or median and interquartile range (IQR). Categorical variables were summarized as frequency and percentage. Comparisons between independent groups of categorical data were made with the χ^2 or Fisher exact tests, and the McNemar test for paired data. Continuous data were

TABLE 1 Patient characteristics (N = 277)

Baseline Characteristics	
Age, y	52.4 ± 9.4
Female sex	250 (90.3)
BMI, kg/m^2	25.5 ± 5.7
Race	
Caucasian	228 (82.3)
East Asian	28 (10.1)
South Asian	15 (5.4)
African Canadian	3 (1.1)
First Nations	1 (0.4)
Other	1 (0.4)
DM	14 (5.1)
Dyslipidemia	64 (23.1)
HTN	101 (36.5)
Current smoker	28 (10.1)
Family history of CAD	92 (33.2)
Previous MI	3 (1.1)
Cerebrovascular disease	9 (3.2)
Hypothyroidism	36 (13.0)
Postmenopausal	169/250 (67.6)
Depression	61 (22.0)
Extracardiac FMD present	176 (63.5)
Hospital characteristics	
STEMI	67 (24.2)
NSTEMI	210 (75.8)
VT/VF	20 (7.2)
SCAD-involved artery	
LAD or branch	135 (48.7)
Circumflex or branch	98 (35.4)
RCA or branch	65 (23.5)
>1 coronary artery dissected	37 (13.4)
Treated with PCI	42 (15.2)
Treated with CABG	7 (2.5)

Abbreviations: BMI, body mass index; CABG, coronary artery bypass surgery; CAD, coronary artery disease; DM, diabetes mellitus; FMD, fibromuscular dysplasia; HTN; hypertension; LAD, left anterior descending artery; MI, myocardial infarction; NSTEMI, non–ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; RCA, right coronary artery; SCAD, spontaneous coronary artery dissection; STEMI, ST-segment elevation myocardial infarction; SD, standard deviation; VF, ventricular fibrillation; VT, ventricular tachycardia.

Data are presented as n (%) or mean ± SD.

compared using the Student *t* test. Logistic regression was used to assess predictors of LV dysfunction. Statistical analyses were performed with SPSS software version 23 (IBM Corp., Armonk, New York).

3 | RESULTS

We included 277 patients who had LV function assessed during their acute SCAD presentation. Baseline characteristics of our patient cohort are presented in Table 1. The mean age was 52.4 ± 9.4 years, and the majority (90.3%) were women. All presented with MI, with 24.2% as ST-segment elevation myocardial infarction (STEMI) and 75.8% as non–ST-segment elevation myocardial infarction (NSTEMI). Overall, 15.2% underwent percutaneous coronary intervention and 2.5% underwent coronary artery bypass surgery. A subset of 177 patients had repeat LV function assessment on follow-up at median of 4.4 months (IQR, 2.2–14.9 months).

At baseline, all patients had LVEF assessment during their index event (92.4% by contrast ventriculography and 7.6% by echocardiography). The mean LVEF was 55.6% ± 9.1%, and 72 of 277 patients (26.0%) had an LVEF <50% (Table 2). Moderate to severe LV dysfunction after SCAD with LVEF <40% was observed in only 14 of 277 cases (5.1%). The presence of WMA was observed in 237 of 277 patients (85.6%), which corresponded to the arterial distribution of their SCAD. Focal hypokinesis was reported in 60.3% of cases, akinesis in 20.9%, and dyskinesis in 4.3%. Normal wall motion was observed in only 14.4% of cases.

Univariate analysis revealed that patients with baseline LVEF <50% were more likely to present with STEMI (38.9% vs 19.0%; $P < 0.001$), had peak troponin I (TnI) levels >50 μg/L (16.6% vs 5.2%; $P = 0.005$), and SCAD involving the left anterior descending artery (LAD; 65.3% vs 42.9%, $P < 0.002$; Table 3). Among patients with baseline LVEF <40%, there was a higher incidence of STEMI presentation (50.0% vs 22.8%; $P = 0.047$), SCAD involvement of LAD (100% vs 46.0%; $P < 0.001$), and percutaneous coronary intervention for SCAD (35.7% vs 14.1%; $P < 0.044$).

Of the subgroup of patients who had repeat assessment of LV function (n = 164), imaging modalities used were predominantly catheter ventriculogram (50.0%) or echocardiography (46.3%). Only in 6 (3.7%) cases was LV function was reassessed using a nuclear imaging. In this subgroup, the baseline LVEF was 54.6% ± 9.2%, and there was improvement of LVEF to 60.7% ± 7.2% at follow-up ($P < 0.001$; Table 2). Baseline LVEF of <50% was observed in 29.9%, but only 6.7% had LVEF <50% at follow-up ($P < 0.001$; see Supporting Information, Figure 1, in the online version of this article). Baseline WMA was observed in 87.2%, but in only 44.5% at follow-up reassessment ($P < 0.001$). In fact, 70 of 143 (49.0%) of patients with WMA at baseline had normalization of wall motion by the time of reassessment (see Supporting Information, Figure 2, in the online version of this article).

We further analyzed the subgroup of patients who had the same imaging modality used for LV function assessment at baseline and follow-up (n = 86; 77 with catheter ventriculogram and 9 with echocardiography). We observed similar results, with improvement of LVEF from 54.2% ± 9.1% at baseline to 62.2% ± 6.3% at follow-up ($P < 0.001$) and WMA observed in 90.7% at baseline vs 57.0% at follow-up ($P < 0.001$).

Among patients with baseline LVEF <50% and repeat assessment, 38 of 49 patients (77.6%) improved their LVEF to ≥50% at follow-up imaging. Of patients with persistent LVEF <50% (n = 11), there were higher incidences of presentation with STEMI (63.6% vs 24.2%; $P = 0.009$) and peak TnI >50 μg/L (33.3% vs 3.9%; $P = 0.009$), compared with those with LVEF ≥50% at follow-up. Among patients with moderate to severe LV dysfunction (<40%), 10 of 14 had repeat assessment of LV function, and 7 of the 10 (70.0%) had improvement in their LVEF to ≥50% at follow-up.

In our multivariable analysis, presentation with STEMI (odds ratio [OR]: 2.44, 95% confidence interval [CI]: 1.18-5.03, $P = 0.016$), SCAD

TABLE 2 LV function and WMA characteristics

LV Function	Overall Cohort Baseline LV Function, N = 277	Repeat LV Function Assessment Performed, n = 164		*P* Value
		Baseline	Follow-up	
LVEF, %	55.6 ± 9.1	54.6 ± 9.2	60.7 ± 7.2	<0.001
LVEF <50%	72 (26.0)	49 (29.9)	11 (6.7)	<0.001
WMA				
Any WMA	237 (85.6)	143 (87.2)	73 (44.5)	<0.001
Normal wall motion	40 (14.4)	21 (12.8)	91 (55.5)	
Hypokinesis	167 (60.3)	95 (57.9)	58 (35.4)	
Akinesis	58 (20.9)	40 (24.4)	11 (6.7)	
Dyskinesis	12 (4.3)	8 (4.9)	4 (2.4)	

Abbreviations: LV, left ventricular; LVEF, left ventricular ejection fraction; SD, standard deviation; WMA, wall-motion abnormality.

Data are presented as n (%) or mean ± SD.

TABLE 3 Univariate predictors of low LVEF at baseline

	Baseline LVEF <50%, n = 72	Baseline LVEF ≥50%, n = 205	OR (95% CI)	*P* Value[1]
Connective-tissue disorder	7 (9.7)	2 (1.0)	10.93 (2.216-53.59)	0.003
STEMI presentation	28 (38.9)	39 (19.0)	2.71 (1.504-4.878)	0.001
SCAD in LAD territory	47 (65.3)	88 (42.9)	2.50 (1.430-4.369)	0.002
Revascularization for SCAD	20 (27.8)	31 (15.1)	2.16 (1.136-4.102)	0.017
>1 artery segment dissected	13 (18.1)	21 (10.2)	1.93 (0.911-4.092)	0.082
Type 2 SCAD is present	58 (80.6)	137 (66.8)	2.06 (1.071-3.947)	0.030
Peak TnI (>50 μg/L)	16.6 (3.1–27.2)	5.2 (1.7–11.7)	1.02 (1.006-1.032)	0.005
SCAD segment length (per 1 mm)	57.2 ± 28.5	45.2 ± 22.4	1.02 (1.006-1.032)	0.003
SCAD segment stenosis (per 1%)	84.4 ± 14.9	78.9 ± 17.9	1.02 (1.001-1.039)	0.035

Abbreviations: CI, confidence interval; IQR, interquartile range; LAD, left anterior descending artery; LVEF, left ventricular ejection fraction; OR, odds ratio; SCAD, spontaneous coronary artery dissection; SD, standard deviation; STEMI, ST-segment elevation myocardial infarction; TnI, troponin I.

Data are presented as n (%), mean ± SD, or median (IQR).

[1] *P* value for logistic regression.

segment length (per mm; OR: 1.02, 95% CI: 1.02-1.03, *P* = 0.019), and SCAD involvement of the LAD (OR: 1.99, 95% CI: 1.02-3.91, *P* = 0.045) were independent predictors of presenting LVEF <50%.

Patients were followed prospectively with median of 2.49 (IQR, 1.02–4.84) years. In-hospital events included recurrent MI in 5.1%, repeat or unplanned revascularization in 4.0%, stroke in 1.4%, and overall MACE of 7.2%. Postdischarge events included mortality in 1.8%, recurrent MI in 19.1%, revascularization in 6.9%, and stroke in 1.4%, with overall MACE of 22.0%. There was no difference between cardiovascular events among patients who presented with LVEF <50% compared with those with LVEF ≥50% (Table 4).

4 | DISCUSSION

The natural history of LV dysfunction in patients with SCAD has not been previously described. In the current study, we report the largest series of baseline LV function, WMA assessment, and clinical follow-up of SCAD patients to date. We found that although 85.6% of SCAD patients had WMA at baseline, most (74%) had baseline LVEF ≥50%. The overall LVEF improved (absolute ~5%) in the cohort of patients with repeat assessment, with 49% (70/143) having WMA resolution, and 77.6% (38/49) had follow-up LVEF >50%. There was no difference in cardiovascular outcomes among those with abnormal baseline LVEF <50% vs those with LVEF ≥50%.

The improvement in LV function is interesting and may reflect resolution of myocardial stunning after spontaneous arterial healing with SCAD. In our previously published SCAD series, we found that 79 of 79 patients who had repeat coronary angiography (≥4 weeks after dissection), when treated conservatively, had spontaneous arterial healing. In a separate series of patients with SCAD who were misdiagnosed as Takotsubo syndrome, we also noted that LV dysfunction and WMA normalized on repeat imaging.[11] Therefore, we hypothesize that SCAD can result in prolonged myocardial ischemia in the territory subtended by the dissected artery, potentially causing myocardial stunning to a greater degree than MI, which may consequently lead to improvement in LV function with vessel healing. Only 6.0% of patients had very high TnI elevation >50 μg/L, and such high Tn elevation was a predictor of both baseline and persistent LVEF <50%. Thus, a larger degree of myocardial damage appears to be required for persistent LV dysfunction in SCAD patients. Conversely, the majority of SCAD patients with a lesser degree of myocardial damage appeared to have baseline LV dysfunction related to stunning, which subsequently improved with vessel healing.

It is possible that the resolution of WMAs could be due to other factors aside from spontaneous healing of the dissected arteries. Overall improvement of stunned myocardium, even in the absence of healing of

TABLE 4 In-hospital and postdischarge CV events in patients with baseline LVEF <50% compared with those with LVEF ≥50%

	Baseline LVEF <50%, n = 72	Baseline LVEF ≥50%, n = 205	Overall Cohort, N = 277	*P* Value
In-hospital events				
Hospital stay >5 days	18 (25.0)	26 (12.7)	44 (15.9)	0.023
Recurrent MI	7 (9.7)	7 (3.4)	14 (5.1)	0.055
Repeat or unplanned revascularization	4 (5.6)	7 (3.4)	11 (4.0)	0.48
Stroke	1 (1.4)	3 (1.5)	4 (1.4)	0.96
In-hospital MACE	8 (11.1)	12 (5.9)	20 (7.2)	0.18
Postdischarge events				
Death	3 (4.2)	2 (1.0)	5 (1.8)	0.11
Recurrent MI	14 (19.4)	39 (19.0)	53 (19.1)	0.93
Revascularization	4 (5.6)	15 (7.3)	19 (6.9)	0.79
Stroke	0 (0)	4 (2.0)	4 (1.4)	0.58
Postdischarge MACE	17 (23.6)	44 (21.5)	61 (22.0)	0.74
Overall MACE	24 (33.3)	53 (25.9)	77 (27.8)	0.23

Abbreviations: CV, cardiovascular; LVEF, left ventricular ejection fraction; MACE, major adverse cardiac events; MI, myocardial infarction.
Data are presented as n (%).

the dissected arteries, may occur. Alternatively, natural establishment of collateral blood vessels to supply occluded segments may occur. Furthermore, medical therapy for SCAD, including β-blockers and angiotensin-converting enzyme inhibitors, may remodel the LV following infarction, subsequently improving LV function and WMA.

In our multivariable analysis, aside from TnI >50 μg/L, we also found that SCAD involving the LAD and STEMI were independent predictors of baseline LVEF <50%. However, presenting TnI >50 μg/L was the only independent predictor of persistent LVEF <50% in those with serial assessment. These data suggested that larger SCAD-related infarcts resulted in greater and even persistent LV dysfunction. However, we did not observe any significant difference in clinical outcomes in patients with baseline or persistent LVEF <50%, compared with those with normal function.

The cardiovascular outcome data presented in this cohort represent the largest dataset of prospective SCAD follow-up to date. Although there was numerically higher incidence of MACE events in patients with LVEF <50%, there was no statistically significant difference in clinical outcomes in patients with baseline or persistent LVEF <50%, compared with those with LVEF ≥50%. It may be that our study was not powered to detect the difference in clinical event rates, or that more extended clinical follow-up is required. Furthermore, LVEF may be a poor predictor of subsequent recurrent cardiovascular events such as recurrent SCAD, which may more likely be related to the underlying pathophysiologic process (ie, arteriopathy predisposing to dissection) than the degree of myocardial damage. Our findings of an overall MACE rate of 27% with a median prospective follow-up of 2.5 years are consistent with previous reports[5] and reinforce that clinical outcomes post-SCAD are not benign. Ongoing prospective studies are required to refine and identify factors associated with adverse clinical outcomes following SCAD and to test therapies that may alter the natural history of this complex clinical entity.

4.1 | Study limitations

Patients included in this study excluded SCAD patients who did not survive to hospital presentation. The assessment of ventricular function in this study was performed largely using catheter ventriculography or echocardiography. A proportion of repeat assessments were done using different modalities, each with its own limitations. Although this is a reflection of the "real-world" retrospective nature of our data, direct comparisons of the different imaging modalities may not be perfect. However, previous reports have established reasonably good correlation between catheter ventriculography and echocardiography in the assessment of LVEF, with reported correlation coefficients of 0.76 to 0.89.[12] Moreover, we observed similar improvements in LVEF and WMA in the subset of patients (n = 86) who had the same imaging modality at baseline and follow-up. In our study, repeat assessment of LV function was only performed in ~50% of patients. There may be a selection bias as to who was subjected to repeat assessment and when the repeat assessment was performed. Finally, a near-normal LVEF, as was seen in most of the patients, may be achieved by hypermobility and compensation of noninfarcted myocardial segments, and thus may not be the optimal measurement for the size of MI that corresponds to clinical outcomes.

5 | CONCLUSION

In our large SCAD cohort, the majority of patients presented with WMA, although the majority had relatively normal LVEF. More than half of our patients had normalization of WMA and LVEF on follow-up functional assessment. This supports our hypothesis that a significant proportion of SCAD patients had myocardial stunning as the etiology of their presenting LV dysfunction.

Conflicts of interest

The authors declare no potential conflicts of interest.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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