Am J Cardiovasc Dis 2026;16(3):142-155
www.AJCD.us /ISSN:2160-200X/AJCD0162760

Original Article

Clinical characteristics, medication, and outcomes of spontaneous coronary artery dissection patients with an initial strategy of percutaneous coronary intervention or conservative therapy

Kang-Zheng Yuan[1*], Jian Wang[2*], Min Lu[3], Fang-Mei Liu[1], Min Deng[1], Sai-Nan Chang[1], Qing-Chi Liao[1]

[1]Department of Cardiology, Northern Jiangsu People's Hospital, Yangzhou 225000, Jiangsu, China; [2]Department of Cardiology, The Affiliated Taizhou People's Hospital of Nanjing Medical University, Taizhou 225300, Jiangsu, China; [3]Department of Anesthesiology, Yangzhou Hospital of Traditional Chinese Medicine, Yangzhou 225000, Jiangsu, China. [*]Equal contributors.

Received December 18, 2024; Accepted April 21, 2026; Epub June 15, 2026; Published June 30, 2026

Abstract: Objectives: Spontaneous Coronary Artery Dissection (SCAD) is a rare non-obstructive acute coronary syndrome. Although previously characterized by a lack of consensus on optimal management, treatment guidelines for SCAD have now been established to inform clinical practice. However, long-term follow-up data specifically for SCAD patients in China are lacking. The present study aimed to assess the clinical and angiographic outcomes of a Chinese cohort managed with initial Percutaneous Coronary Intervention (PCI) or conservative therapy. Methods: The study enrolled 33 patients from the Northern Jiangsu People's Hospital, affiliated with Yangzhou University, whose initial diagnosis of SCAD was made from April 1, 2014 to June 30, 2020. The study recommended a follow-up coronary angiography for all patients 6 months after the initial onset. Clinical characteristics, angiographic findings, initial treatment strategies, discharge medications, and outcomes were recorded for all patients. Results: Among the 33 patients with SCAD (mean age 51.9±9.7 years, 69.7% female), myocardial infarction was the predominant presentation (75.7%), with Non-ST-Segment Elevation Myocardial Infarction (NSTEMI) accounting for the majority of cases (51.5%). The left anterior descending artery (LAD) was the most frequently involved vessel (63.6%). According to the Saw classification, type 2b lesions predominated (65.7%). Compared to the conservative group, patients in the PCI group presented with a significantly higher degree of stenosis [100% (IQR, 98-100) vs. 70% (IQR, 70-80), P < 0.001] and a higher prevalence of baseline TIMI 0-1 flow (81.8% vs. 0%, P < 0.001). PCI was technically successful (TIMI 2-3 flow post-procedure) in all patients. During the initial hospitalization, the overall MACE rate was 9.1% (3/33). Over a median follow-up of 27.5 months (IQR, 15.3-51.3), the MACE rate was 9.4% (3/32), including one cardiac death. Recurrent Spontaneous Coronary Artery Dissection (R-SCAD) was confirmed in 3 patients (9.4%). No significant difference was observed in MACE-free survival between the PCI and conservative groups (59.3% vs. 85.7%, P=0.126). Follow-up coronary angiography (CAG) performed in 16 patients (50%) demonstrated angiographic healing in 15 patients (93.8%). Notably, 2 of the 3 patients who experienced late MACE had previously demonstrated complete lesion healing on 6-month follow-up CAG. Patients who underwent 6-month follow-up CAG exhibited a numerically lower MACE-free survival rate compared to those who did not, although this difference did not reach statistical significance (62.9% vs. 100.0%, P=0.10). Conclusion: This study reported that the outcomes of patients subjected to an initial conservative strategy were not inferior as compared to those treated with PCI. In patients who underwent follow-up CAG, healing was commonly observed. Therefore, guiding antiplatelet therapy based on angiographic findings and comorbidities appears rational.

Keywords: Spontaneous coronary artery dissection, percutaneous coronary intervention, conservative strategy, myocardial infarction

Introduction

Spontaneous Coronary Artery Dissection (SCAD) is a rare non-atherosclerotic Acute Coronary Syndrome (ACS) and is frequently underdiagnosed. Two primary pathological mechanisms have been described for SCAD. The first mechanism involves an intimal tear that initiates

https://doi.org/10.62347/FDZD1794

a medial dissection and hemorrhage, leading to the formation of a false lumen. The second mechanism is thought to be initiated by rupture of the vasa vasorum, resulting in spontaneous bleeding into the arterial wall and the formation of an Intramural Hematoma (IMH) [1]. SCAD was diagnosed per 2018 American Heart Association (AHA) criteria [2], requiring either (1) definitive angiographic evidence (IMH, double lumen, or diffuse stenosis without atherosclerosis) or (2) intracoronary imaging confirmation of intimal tear/false lumen. Secondary causes (atherosclerosis, trauma) were excluded via clinical, laboratory, and imaging assessments. Based on the Saw classification [3], four distinct angiographic appearances and patterns have been described for SCAD [4], namely type 1 (evident arterial wall stain), type 2 (diffuse stenosis of varying severity), type 3 (mimic atherosclerosis), and type 4 (abrupt vessel occlusion). The incidence of SCAD is underestimated, primarily because its classic angiographic hallmarks are lacking in >70% of patients and may be discovered exclusively with intravascular imaging [5, 6]. Among all patients presenting with ACS, SCAD was identified in 1.0-4.0%, using Coronary Angiography (CAG) [2, 7]. Buccheri et al. proposed a clinical-angiographic score, suggesting that patients with a score of at least 3 - based on angiographic suspicion - should undergo endovascular imaging [8]. Significant advancements have been made in the diagnosis of SCAD in recent years, driven by the widespread adoption of intracoronary imaging techniques, notably Optical Coherence Tomography (OCT) and Intravascular Ultrasound (IVUS). Despite an increase in awareness and diagnosis of SCAD, optimal treatment strategies remain controversial. Current AHA guidelines advocate conservative management as the cornerstone of SCAD therapy, reserving Percutaneous Coronary Intervention (PCI) for high-risk anatomical or clinical scenarios [9]. Over 70-97% of conservatively managed patients exhibit angiographic healing within 1-3 months [10]. In contrast, the success rates of PCI vary considerably (ranging from 36.4% to 72.5%), which is likely attributable to significant heterogeneity in the definition of PCI success in SCAD. Nevertheless, all reported success rates are markedly lower than those observed in control subjects with ACS [2]. Compared with patients who have atherosclerotic acute myocardial infarction (AMI), those with SCAD when treated with PCI are at higher risk of periprocedural complications [2, 11], particularly iatrogenic dissection and acute vessel occlusion [12]. Due to variations in definitions, study designs, and follow-up durations, reported recurrence rates for SCAD differ across existing studies, ranging from 10% to 30% [10]. In contrast, the overall mortality rate is lower than that of atherosclerotic acute myocardial infarction, with a multicenter cohort study reporting a rate of 0.8% over a median follow-up of 3 years [13]. This study focuses on two key issues in the clinical management of SCAD: (1) the long-term clinical outcomes of percutaneous coronary intervention versus conservative treatment strategies after the acute phase, and (2) antiplatelet therapy adjustment strategies based on angiographic healing patterns. Through systematic serial IVUS imaging and standardized repeat coronary angiography at six months, this study aims to generate evidence-based guidance for clinical decision-making in SCAD. Building upon existing long-term follow-up studies on SCAD conducted in North America [13], this study is specifically designed to focus on the Asian (Chinese) population. It provides important data and a comparative perspective on the clinical characteristics and management strategies of this disease within this demographic.

Methods

Study population and diagnostic criteria

The study population consisted of inpatients diagnosed with Spontaneous Coronary Artery Dissection (SCAD) who presented to Yangzhou University Affiliated Northern Jiangsu People's Hospital between April 1, 2014, and June 30, 2020. The diagnosis of SCAD was established by the presence of a double-lumen morphology or an image of intramural hematoma by Coronary Angiography (CAG). Intravascular Ultrasound (IVUS) was applied to ensure the accuracy of diagnosis for a suspected SCAD on coronary angiography, with commercially available mechanical systems (Atlantis SR Pro, 40-MHz catheter; Boston Scientific). The coronary angiographic pattern was grouped based on the Saw et al. classification [3]. A complete concordance was noted between 2 independent cardiologists in the diagnosis of SCAD for every individual. Coronary Artery Territory Involvement: The involved coronary artery territories were recorded according to the segmental definitions of the American College of Cardiology/American Heart Association/Society for Cardiovascular Angiography and Interventions [14]. This included documenting the major epicardial vessels (left main, left anterior descending, left circumflex, and right coronary arteries) and secondary branches (e.g., diagonal, obtuse marginal) where SCAD was present. Multivessel dissection was defined as the simultaneous presence of SCAD in two or more major epicardial arteries. Saw Classification [3]: SCAD lesions were classified according to the angiographic classification proposed by Saw et al. The specific types were defined as follows: Type 1 (visible arterial wall staining or an intimal flap), Type 2 (diffuse stenosis, typically extending >20 mm in length, with subtype 2a denoting a tapering lumen and subtype 2b denoting an abrupt lumen caliber change), Type 3 (focal or tubular stenosis, usually <20 mm in length, mimicking atherosclerosis), and Type 4 (total vessel occlusion where the distal vessel anatomy is not visualized). Degree of Stenosis: The percent luminal diameter stenosis was quantified using visual estimation by the interventional cardiologist. The assessment was made in the projection that revealed the most severe narrowing. For lesions with total occlusion, the stenosis degree was recorded as 100%. Baseline Flow: Coronary blood flow at baseline was assessed using the Thrombolysis in Myocardial Infarction (TIMI) flow grading system: grade 0 (no perfusion), grade 1 (penetration without perfusion), grade 2 (partial perfusion), and grade 3 (complete perfusion).

Population selection criteria and extracted variables

Inclusion criteria: Patients aged over 18 years diagnosed with SCAD by CAG or IVUS. Exclusion criteria: Patients with secondary or iatrogenic coronary artery dissection; those complicated with malignant tumors or chronic kidney disease stage 5; lost to follow-up or with incomplete clinical data.

Patient grouping

This was an observational study in which patients were not randomized to treatment groups. The decision to pursue an initial conservative management strategy or PCI was made by the attending physician based on a comprehensive assessment of the patient's clinical presentation, hemodynamic status, and angiographic findings. The specific criteria for group assignment were as follows: PCI Group: This strategy was employed for patients with ongoing symptoms of myocardial ischemia, a large area of myocardium at risk (e.g., due to involvement of the left main coronary artery or proximal left anterior descending artery), compromised coronary blood flow (TIMI flow grade 0-1), or hemodynamic instability. Conservative Management Group: This strategy was selected for patients who were hemodynamically stable, without persistent ischemic symptoms, and had preserved coronary blood flow (TIMI flow grade 2-3).

Treatment strategy

Conservative Treatment: The conservative management strategy primarily involved pharmacological therapy aimed at alleviating symptoms, reducing myocardial oxygen consumption, preventing thrombus formation, and controlling arterial blood pressure to facilitate dissection healing. The specific regimen included the following components: Antiplatelet Therapy: Patients received either aspirin or clopidogrel monotherapy, or Dual Antiplatelet Therapy (DAPT), as clinically indicated. Beta-Blockers: Administered to control heart rate and blood pressure, thereby reducing shear stress on the coronary artery wall. Angiotensin-Converting Enzyme Inhibitors/Angiotensin II Receptor Blockers (ACEI/ARB): Used for blood pressure management and cardioprotection. Statins: Prescribed for lipid management in applicable patients. Additionally, antianginal medications such as nitrates and trimetazidine could be administered based on individual patient presentation. All patients undergoing conservative management received close clinical monitoring.

PCI Treatment: The specific PCI approach was determined based on intraprocedural findings, with priority given to restoring coronary flow rather than achieving an optimal anatomical result. The technical maneuvers included: Plain Old Balloon Angioplasty (POBA): Low-pressure, gentle balloon dilatation was applied to the dissected segment to restore true lumen flow and avoid stent implantation. Cutting Balloon Angioplasty: This technique was utilized in select cases for more precise lesion expansion. Stent Implantation: This was reserved for scenarios where balloon angioplasty failed to achieve satisfactory flow restoration, or in cases of flow-limiting dissection or acute vessel occlusion. Notably, the vast majority of patients in this study cohort underwent balloon angioplasty alone. Procedural success was defined as the restoration of TIMI flow grade 2-3 upon procedure completion.

Follow-up

We accordingly recommended a repeat angiography to all patients at 6 months after the initial onset. The follow-up tests included a return visit, telephone inquiry, and medical record review. The end-of-study date was set to June 30, 2021 and all participants were followed for at least 12 months after the SCAD diagnosis.

Data collection and outcomes

Clinical characteristics (such as demographic, comorbidities, and clinical presentation), angiographic findings (coronary artery territory involved, Saw Classification [3], degree of stenosis, and the baseline flow), initial treatment strategies, discharge medication, outcomes during initial hospitalization, follow-up, and treatment effect (such as healing and chest pain improvement) of all patients were recorded. The Major Adverse Cardiac Events (MACEs) were defined as death, recurrent SCAD, congestive heart failure, and myocardial infarction.

Ethical statement: The study was approved by the Ethics Committee of Yangzhou University Affiliated Northern Jiangsu People's Hospital (Approval No. 2022ky167), and written informed consent was obtained from all patients.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data, and as median [interquartile range (IQR)] for non-normally distributed data. Comparisons between groups were performed using the Student's t-test for normally distributed continuous variables and the Mann-Whitney U test for non-normally distributed continuous variables. Discrete variables were expressed as frequencies and percentages, and comparisons were made using Fisher's exact test. The Kaplan-Meier method was used to estimate survival curves for major adverse cardiovascular events (MACE) during follow-up, and differences between curves were assessed using the log-rank test. A two-tailed P value < 0.05 was considered statistically significant. All statistical analyses were performed using SPSS version 19.0 (IBM Corp., Armonk, NY, USA).

Results

Clinical characteristics (demographic, comorbidities, and diagnosis at the time of admission)

The present study enrolled 33 patients, of whom 10 underwent percutaneous coronary intervention (PCI) and 23 received conservative management. Clinical characteristics, including demographics, comorbidities, and admission diagnoses, are summarized in **Table 1** for both groups. The mean age of the patients was 51.9±9.7 years, and 69.7% were female. The mean initial left ventricular ejection fraction (LVEF) was 56.7% ± 6.3% for the entire cohort. Regarding comorbidities, the incidences of atherosclerotic risk factors were as follows: hypertension in 42.4%, diabetes in 9.1%, hypercholesterolemia in 18.2%, and smoking in 15.2% of patients. Coronary artery atherosclerosis was present in six patients (18.2%). No significant differences were observed between the two groups for any of these variables. Other notable conditions included superior mesenteric artery dissection in one patient (3.0%), connective tissue disease in one patient (3.0%) who had been taking hydroxyurea, a history of cytotoxic drug treatment in two patients (6.1%), and a history of leiomyoma in seven female patients (30.4%). The distribution of admission diagnoses was similar between the groups. Of the 25 patients (75.7%) admitted for myocardial infarction, 8 (24.2%) presented with ST-segment elevation myocardial infarction (STEMI) and 17 (51.5%) with non-ST-segment elevation myocardial infarction (NSTEMI). The remaining patients presented with unstable angina (UA, n=5, 15.2%) or stable angina (SA, n=3, 9.1%).

Table 1. Clinical characteristics (Demographic, comorbidities and admitting diagnosis)

Variables	Total (n=33)	PCI (n=10)	Conservative (n=23)	P
Demographic				
Age (mean ± SD), years	51.9±9.7	55.0±10.1	50.5±9.5	0.23
Female, n (%)	23 (69.7)	9 (90.0)	14 (60.9)	0.113
Comorbidities				
Atherosclerosis risk factors				
Hypertension, n (%)	14 (42.4)	2 (20.0)	12 (52.2)	0.131
Diabetes, n (%)	3 (9.1)	0	3 (13.0)	0.536
Hypercholesterolemia, n (%)	6 (18.2)	1 (10.0)	5 (21.7)	0.640
Smoking, n (%)	5 (15.2)	0	5 (21.7)	0.291
Coronary artery atherosclerosis, n (%)	6 (18.2)	1 (10.0)	5 (21.7)	0.640
Other conditions				
Connective tissue diseases, n (%)	1 (3.0)	0	1 (4.3)	1.000
Other vessel dissection, n (%)	1 (3.0)	0	1 (4.3)	1.000
Cytotoxic drug, n (%)	2 (6.1)	0	2 (8.7)	1.000
Leiomyoma (female), n (%)	7 (30.4)	2 (22.2)	5 (35.7)	0.657
Admitting Diagnosis				0.534
STEMI, n (%)	8 (24.2)	4 (40.0)	4 (17.4)	
NSTEMI, n (%)	17 (51.5)	5 (50.0)	12 (52.2)	
UA, n (%)	5 (15.2)	1 (10.0)	4 (17.4)	
SA, n (%)	3 (9.1)	0	3 (13.0)	

Note: *P* values are for comparison between the PCI and conservative groups. Abbreviations: SD, Standard Deviation; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; UA, unstable angina; SA, stable angina.

Angiographic findings

A total of 35 vessel lesions were identified in 33 patients (**Table 2a** and **2b**). Intravascular ultrasound (IVUS) was used to confirm the diagnosis in 20 patients (60.6%), with no significant difference between the PCI and conservative groups (60.0% vs. 60.9%, P=1.000). The left anterior descending artery (LAD) was the most frequently involved territory, affected in 21 patients (63.6%), followed by the left circumflex artery (LCX) in 7 patients (21.2%) and the right coronary artery (RCA) in 7 patients (21.2%). Secondary branch involvement was observed in 6 patients (18.2%), and multivessel disease was present in 2 patients (6.1%). No significant differences were observed between the two groups in terms of coronary artery territory involvement (all P > 0.05). According to the Saw classification, the majority of lesions were classified as type 2 [31 lesions (88.6%)], of which 23 (65.7%) were type 2b and 8 (22.9%) were type 2a. Type 1 and type 3 lesions accounted for 1 (2.9%) and 3 (8.6%) lesions, respectively. **Figure 1** shows the angiographic features based on the Saw classification. The overall distribution of Saw classification was similar between the PCI and conservative groups (P=0.241). Patients in the PCI group had a significantly higher degree of stenosis [100% (IQR, 98.0-100.0) vs. 70% (IQR, 70.0-80.0), P < 0.001] and poorer baseline TIMI flow compared to the conservative group. Specifically, 9 of 11 lesions (81.8%) in the PCI group presented with TIMI grade 0-1 flow, whereas all 24 lesions (100%) in the conservative group had TIMI grade 2-3 flow at baseline (p < 0.001).

Patient management

Overall, 10 patients underwent PCI. In this study, achievement of TIMI 2-3 flow after PCI was considered a technical success, and coronary angiography (CAG) confirmed TIMI 2-3 flow in all patients post-procedure. Emergent PCI was performed within 2 hours of admission in 8 of these 10 patients. Specifically, 7 patients underwent plain balloon angioplasty, while one patient received cutting balloon an-

Table 2a. Angiographic findings (per patient)

Variables	Total (n=33)	PCI (n=10)	Conservative (n=23)	P
Coronary artery territory involved				-
LM	0	0	0	-
LAD, n (%)	21 (63.6)	6 (60.0)	15 (65.2)	0.537
LCX, n (%)	7 (21.2)	1 (10.0)	6 (26.1)	0.397
RCA, n (%)	7 (21.2)	4 (40.0)	3 (13.0)	0.161
Secondary branches (D/OM), n (%)	6 (18.2)	2 (20.0)	4 (17.4)	1.000
Multivessel disease, n (%)	2 (6.1)	1 (10.0)	1 (4.3)	0.521
Confirmation by IVUS, n (%)	20 (60.6)	6 (60.0)	14 (60.9)	1.000

Note: Percentages may sum to >100% due to some patients having multiple vessels involved. Multivessel disease was defined as involvement of at least two major epicardial arteries. Abbreviations: LM, left main coronary artery; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; D, diagonal branch; OM, obtuse marginal branch; IVUS, intravascular ultrasound.

Table 2b. Angiographic findings (per involved vessel lesions)

Variables	Total (n=35)	PCI (n=11)	Conservative (n=24)	P
Saw classification, n (%)				0.241
Type 1	1 (2.9)	0	1 (4.2)	
Type 2	31 (88.6)	11 (100)	20 (83.3)	
2a	8 (22.9)	1 (9.1)	7 (29.2)	
2b	23 (65.7)	10 (90.9)	13 (54.2)	
Type 3	3 (8.6)	0	3 (12.5)	
Percent stenosis (visual assessment), median (IQR), %	80.0 (70.0-98.3)	100.0 (98.0-100.0)	70.0 (70.0-80.0)	<0.001
Baseline TIMI flow, n (%)				<0.001
0-1	9 (25.7)	9 (81.8)	0	
2-3	26 (74.3)	2 (18.2)	24 (100.0)	

Note: *P* value for comparison of overall distribution of Saw classification (Type 1, Type 2, and Type 3) between the PCI and conservative groups was calculated using Fisher's exact test (due to expected cell frequencies <5). Subcategories (2a and 2b) are presented for descriptive purposes only. Abbreviations: PCI, percutaneous coronary intervention; IQR, interquartile range; TIMI, Thrombolysis in Myocardial Infarction.

gioplasty with a 2.5 × 10-mm cutting balloon, followed by balloon angioplasty with a 2.5 × 20-mm balloon. All patients had restored TIMI 2-3 flow after the procedure. Two patients underwent CAG due to worsening symptoms during hospitalization. One patient underwent CAG on Day 3 after admission because of progressive troponin elevation; CAG revealed occlusion of the posterior descending artery, which was restored to TIMI 3 flow after simple balloon angioplasty. The other patient presented with transient syncope, and electrocardiography (ECG) showed ST-segment elevation in leads II, III, and aVF. This patient was immediately transferred to the cardiac catheterization laboratory. CAG with intravascular ultrasound (IVUS) revealed an intramural hematoma (IMH) extending from the proximal to the distal segment of the right coronary artery (RCA). Four stents (2.5 × 33 mm, 3.0 × 33 mm, 3.5 × 36 mm, and 4.0 × 15 mm) were implanted in the RCA from the proximal posterior descending branch to the ostium of the RCA, with the stents overlapping by approximately 2 mm. Repeat CAG confirmed restoration of TIMI 3 flow in the RCA. No complications such as propagation of dissection flap or intramural hematoma, failure to cross into the distal true lumen with a wire, or failure to cross the lesion with a balloon were observed during PCI. Coronary artery bypass grafting (CABG) was not recommended for any patient due to stable hemodynamics.

Medication at the time of discharge

Medication prescribed at the time of discharge is shown in **Table 3**. For antiplatelet therapy, 29 (87.9%) patients were administered dual antiplatelet therapy (DAPT) (aspirin + clopido-

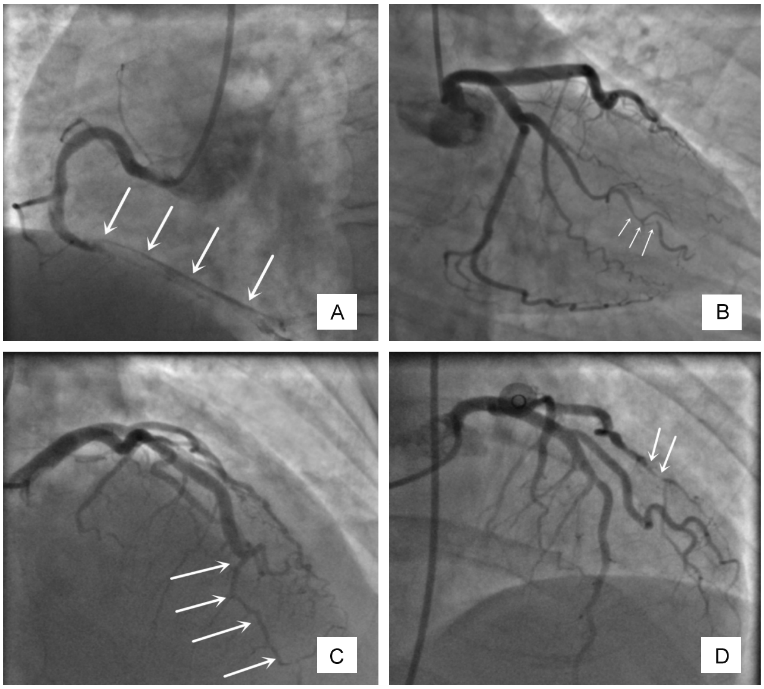

Figure 1. Coronary angiographic appearances of the four Saw classification types of spontaneous coronary artery dissection. The arrows indicate (A) a type-1 SCAD on RCA; (B) a type-2a SCAD on OM; (C) a type-2b SCAD on LAD; and (D) a type-3 SCAD on LCX. Abbreviations: SCAD, Spontaneous Coronary Artery Dissection; OM, Obtuse Marginal branch; LCX, Left Circumflex Artery.

Table 3. Medication at the time of discharge

Medications	Total (n=33)	PCI (n=10)	Conservative (n=23)
Antiplatelet			
aspirin + clopidogrel, n (%)	29 (87.9)	10 (100.0)	19 (82.6)
aspirin, n (%)	3 (9.1)	0	3 (13.0)
clopidogrel, n (%)	1 (3.0)	0	1 (4.3)
ACEI/ARB and Beta blocker			
ACEI/ARB, n (%)	13 (39.4)	5 (50.0)	8 (34.8)
Beta blocker, n (%)	25 (75.8)	9 (90.0)	16 (69.6)
Statin, n (%)	28 (84.8)	10 (100.0)	18 (78.3)
Anti anginal			
Nitroglycerin/nitrate Esters, n (%)	5 (15.2)	1 (10.0)	4 (17.4)
trimetazidine, n (%)	11 (33.3)	0	11 (47.8)
CCB, n (%)	3 (9.1)	0	3 (13.0)

Abbreviations: PCI, percutaneous coronary intervention; ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin II receptor blockers; CCB, calcium channel blockers.

grel), 3 (9.1%) patients were provided aspirin, while one (3.0%) patient was given clopidogrel for single antiplatelet therapy (SAPT). Beta-blockers were prescribed for 25 (75.8%) patients, and 13 (39.4%) patients were given angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers (ACEI/ARB). Importantly, 28 (84.8%) patients were given statins. Nitroglycerin/nitrate esters, trimetazidine, and calcium channel blockers (CCB) were administered to 5 (15.2%), 11 (33.3%), and 3 (9.1%) patients, respectively.

Initial hospitalization

Major adverse cardiovascular event (MACE) during initial hospitalization was defined as a composite of all-cause death, recurrent spontaneous coronary artery dissection (R-SCAD), recurrent myocardial infarction (RMI), and congestive heart failure. During initial hospitalization, one patient (3.0%) died of ventricular fibrillation, and three patients (9.1%) (including the patient who died) experienced congestive heart failure. All these patients received initial conservative treatment (**Table 4**). R-SCAD and RMI were not observed during initial hospitalization. The overall incidence of MACE was 9.1% (3/33).

Long-term follow-up

MACE during long-term follow-up: One patient in the conservative therapy group died during initial hospitalization; thus, a total of 32 patients (33 initially enrolled minus 1 deceased) were included in the long-term follow-up. MACE during long-term follow-up was defined as a composite of all-cause death, R-SCAD, RMI, and congestive heart failure. Over a median follow-up of 27.5 months (IQR, 15.3-51.3) (**Table 5**), three patients experienced five MACE at 18, 36, and 42 months, respectively. Notably, one patient (3.1%) was readmitted for RMI and died (she was also

Table 4. MACE during initial hospitalization

Variables	Total patient (n=33)	PCI (n=10)	Conservative (n=23)	P
MACE	3 (9.1)	0	3 (13.0)	0.536
all-caused death, n (%)	1 (3.0)	0	1 (4.3)	1.000
R-SCAD, n (%)	0	0	0	-
CHF, n (%)	3 (9.1)	0	3 (13.0)	0.536
RMI, n (%)	0	0	0	-

Note: MACE during initial hospitalization was defined as a composite of all-cause death, R-SCAD, RMI and CHF. Abbreviations: MACE, major adverse cardiovascular events; PCI, percutaneous coronary intervention; R-SCAD, recurrent spontaneous coronary artery dissection; CHF, congestive heart failure; RMI, recurrent myocardial infarction.

Table 5. Long-term follow-up outcomes

Variables	Total patient (n=32)	PCI (n=10)	Conservative (n=22)	P
follow-up duration, median (IQR), months	27.5 (15.3-51.3)	36.0 (18.3-46.0)	26.0 (15.0-60.0)	0.501
follow-up CAG, n (%)	16 (50.0)	9 (90.0)	7 (31.8)	0.003
MACE	3 (9.4)	2 (20.0)	1 (4.5)	0.224
all-cause death, n (%)	1 (3.1)	1 (10.0)	0	0.313
R-SCAD, n (%)	3 (9.4)	2 (20.0)	1 (4.5)	0.224
CHF, n (%)	0	0	0	-
RMI, n (%)	1 (3.0)	1 (10.0)	0	0.521
Treatment effect				-
Healed (6 months CAG), n (%)	15 (93.8)	9 (100.0)	6 (85.7)	0.438
Chest pain with CCS class I or lower, n (%)	30 (93.8)	8 (80.0)	22 (100.0)	0.091

Note: MACE during long-term follow-up was defined as a composite of all-cause death, R-SCAD, RMI and CHF. Abbreviations: PCI, percutaneous coronary intervention; IQR, interquartile range; CAG, coronary angiography; MACE, major adverse cardiovascular events; R-SCAD, recurrent spontaneous coronary artery dissection; CHF, congestive heart failure; RMI, recurrent myocardial infarction; CCS, Canadian Cardiovascular Society classification.

confirmed to have R-SCAD), while three patients (9.4%) (including the patient who died) were diagnosed with recurrent SCAD by repeat CAG. The overall MACE rate was 9.4% (3/32). No significant differences were observed in MACE-free survival between the two groups (59.3% vs. 85.7%, P=0.126) (**Figure 2**).

Chest pain during follow-up: This study assessed chest pain in 32 patients at the last follow-up. According to the Canadian Cardiovascular Society (CCS) classification, 30 patients (93.8%) presented with CCS class I or lower, one patient with class II, and one patient was readmitted with class IV.

Six-month coronary angiography: In the present study, 16 patients (50%) underwent follow-up coronary angiography (CAG), with or without intravascular ultrasound (IVUS). Of the 10 patients in the PCI group, 9 (90%) underwent follow-up CAG, compared with 7 of 23 (30.4%) in the conservative group (P=0.002). CAG showed healing of SCAD lesions in 15 of these 16 patients. Notably, CAG confirmed healing in the remaining patient at the 42-month follow-up (**Figure 3**). The 6-month follow-up CAG demonstrated healing, which allowed discontinuation of antiplatelet therapy in 11 patients and de-escalation from dual antiplatelet therapy (DAPT) to single antiplatelet therapy (SAPT) in three patients. For the unhealed patient, aspirin was continued for three years until the lesion was completely healed. Patients who did not undergo follow-up CAG received DAPT for 6 months to 1 year and were then switched to long-term SAPT. Among the three patients who experienced recurrent SCAD, one received DAPT for 1 year and then switched to prolonged SAPT, another received SAPT for 6 months until repeat CAG confirmed complete healing, and one died after the recurrent SCAD. Three of the patients who underwent 6-month follow-up CAG experienced MACE at 18, 36, and 42 months, respectively. Two of these three

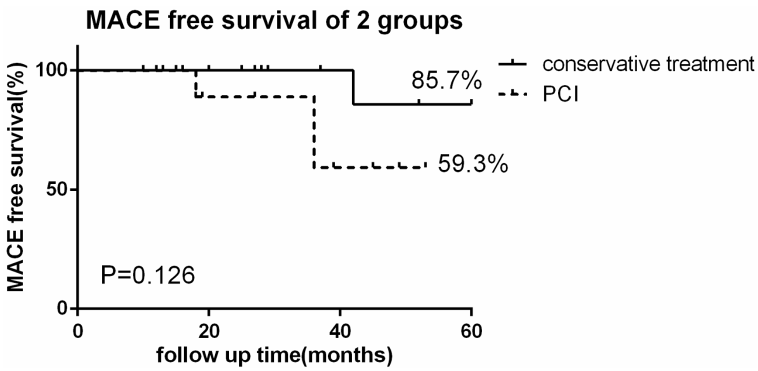

Figure 2. Comparison of MACE-free survival between patients in the two groups. No significant differences were recorded for MACE-free survival for the patients in the two groups (59.3% vs. 85.7%, P=0.126). Abbreviations: MACE, major adverse cardiovascular event; PCI, percutaneous coronary intervention. MACE was defined as a composite of all-cause death, recurrent SCAD, congestive heart failure, or myocardial infarction.

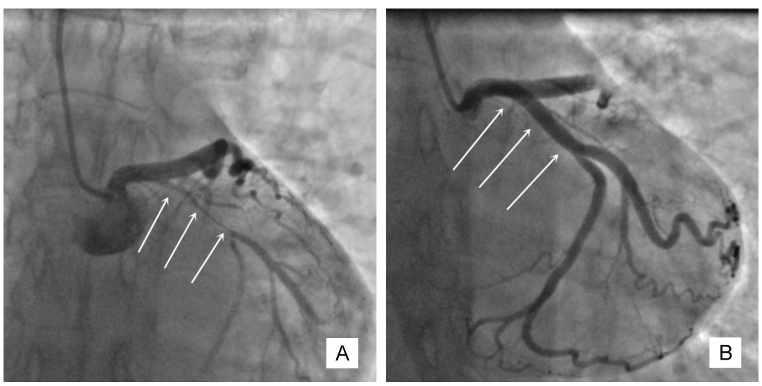

Figure 3. Follow-up coronary angiography at 42 months after the initial admission confirmed lesion healing in one patient. The arrows indicate a type-2a SCAD on LCX (A); The arrows indicate the original SCAD healed at 42 months (B). Abbreviations: SCAD, Spontaneous Coronary Artery Dissection; LCX, Left Circumflex Artery.

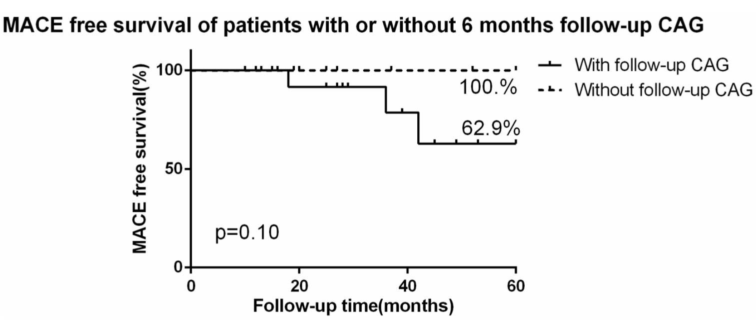

Figure 4. Comparison of MACE-free survival according to whether patients underwent 6-month follow-up coronary angiography. Patients who underwent 6-month follow-up CAG showed a lower survival rate of MACE compared to those without follow-up CAG, although the difference was not statistically significant (62.9% vs. 100.0%, P=0.10). Abbreviations: MACE, major adverse cardiovascular event; CAG, Coronary Angiography. MACE was defined as a composite of all-cause death, recurrent SCAD, congestive heart failure, or myocardial infarction.

patients had shown complete healing of the initial lesions at the 6-month follow-up CAG. Patients who underwent 6-month follow-up CAG had a lower MACE-free survival rate compared with those without follow-up CAG, although the difference was not statistically significant (62.9% vs. 100.0%, P=0.10) (**Figure 4**).

Discussion

The epidemiology and associated conditions observed in this study were consistent with those reported in previous literature [9, 15-17]. No pregnancy-associated Spontaneous Coronary Artery Dissection (SCAD) cases were included in the present series. Fibromuscular dysplasia (FMD) is a well-established dominant risk factor for SCAD [2, 18]. Other relevant risk factors include female sex, pregnancy or the peripartum period, young age, absence of classical cardiovascular risk factors, severe physical or emotional stress, and systemic connective tissue disorders [19]. In the present study, only one patient was found to have a superior mesenteric artery dissection, which may be attributable to the low screening rate for extracoronary vascular abnormalities. Our results indicated that myocardial infarction (MI) was the most common clinical presentation of SCAD, and the left anterior descending artery (LAD) was the most frequently involved vessel. Notably, most lesions were classified as type 2. These findings are consistent with those previously reported from other centers [20-24].

The optimal treatment strategies for SCAD remain contro-

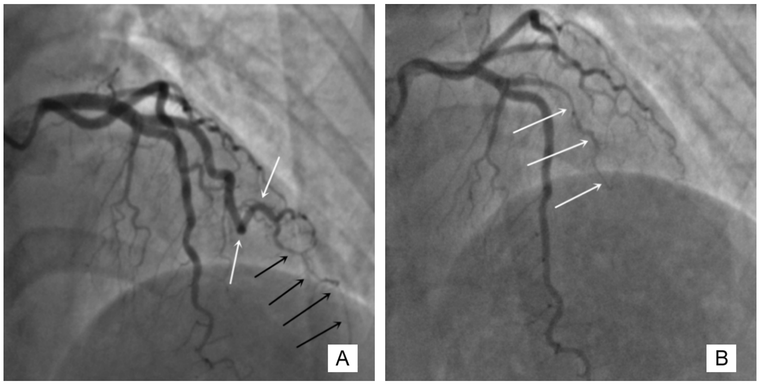

Figure 5. Follow-up angiography two years after the initial admission confirmed retrograde extension of the lesion in the diagonal branch of one patient. The white arrows indicate that this patient had two consecutive curvatures (>90°) on the diagon branch, black arrows indicate a type-2b SCAD on the diagon branch (A); The arrows indicate a retrograde extension of the original SCAD on the diagon branch on CAG after 2 years (B). Abbreviations: CAG, Coronary Angiography; SCAD, Spontaneous Coronary Artery Dissection.

versial. A prospective registry study from Spain reported excellent in-hospital survival in most SCAD patients initially managed conservatively [21]. In contrast, initial management with Percutaneous Coronary Intervention (PCI) and an angiographic presentation of intramural hematoma were associated with a higher incidence of in-hospital adverse events. Accordingly, García-Guimaraes et al. suggested that PCI be reserved as first-line therapy primarily for patients with ongoing ischemia or evidence of compromised distal flow at the time of procedure [21]. Patients with left main SCAD exhibit a poorer prognosis compared to those with non-left main SCAD [17], whereas a more aggressive revascularization strategy is warranted in this population, with PCI demonstrating greater clinical benefit [25]. Another study found that ST-Segment Elevation Myocardial Infarction (STEMI), Thrombolysis In Myocardial Infarction (TIMI) flow grade 0-1, and the severity of luminal narrowing were independent predictors favoring the choice of PCI treatment [26]. Buccheri et al. collected 402 survey responses from authors who had published on SCAD via PubMed, finding that medical therapy was most frequently preferred as first-line treatment [19]. Consistent with this tendency, the majority of patients in the present study were managed conservatively following a SCAD diagnosis. Notably, only 10 patients required revascularization via PCI, all of whom presented with severe stenosis, vessel occlusion, or impaired flow. Of these, just one patient received stents, while the other nine were treated with balloon angioplasty alone. Despite previous reports of high PCI failure rates in SCAD patients [15, 22], all patients in the present study achieved restored TIMI flow grade 2-3 post-procedure, with no in-hospital Major Adverse Cardiovascular Events (MACE) observed. The findings of this study support prior recommendations for the initial management of SCAD, which favor a conservative approach in the absence of severe hemodynamic compromise [2, 11, 21].

Previous research reports a poorer prognosis for SCAD involving the left main coronary artery [25], yet shows similar MACE rates whether initial management is PCI or conservative [26]. Consistent with the need for ongoing vigilance, the present study found that MACE often occurred long after discharge, emphasizing the importance of long-term follow-up in SCAD care. In line with earlier reports associating severe tortuosity with SCAD recurrence [27], three patients in our study experienced recurrence. One illustrative case involved a diagonal branch with two consecutive >90° curvatures; follow-up coronary angiography (CAG) two years later confirmed retrograde extension of the original SCAD in this branch (**Figure 5**). Patients undergoing PCI more frequently presented with occluded arteries and experienced a higher incidence of MACE during follow-up, although the difference was not statistically significant. Notably, the majority of patients reported an improvement in chest pain symptoms. Moreover, all patients who underwent repeat coronary angiography showed spontaneous healing of the initial SCAD, a finding consistent with the study by Alfonso et al., which posits that spontaneous vessel healing constitutes part of the natural history of SCAD [24]. In another study with a median follow-up of 270 days, the healing rate reached 80% among patients who underwent repeat coronary angiography [26].

In terms of medication, while the present study included patients on various drug regimens, the following discussion will focus specifically on antiplatelet therapy, given its central role in management and the detailed data available for analysis. The 2020 American Heart Association/American College of Cardiology (AHA/ACC) guidelines recommend Dual Antiplatelet Therapy (DAPT) for patients undergoing PCI [28], and its consideration is also extended to the acute phase of SCAD [11], typically for up to one year in medically managed patients. However, robust clinical trial data to definitively guide the optimal antiplatelet strategy in SCAD remain lacking. Spontaneous Coronary Intramural Hematoma (SCIH), a subtype of SCAD, is characterized by spontaneous hematoma formation between the coronary intima and media. SCIH carries a low thrombotic risk; however, antiplatelet therapy may promote hematoma progression or delay its absorption [16]. Consequently, for patients with SCIH, the potential harms of antiplatelet treatment may outweigh its benefits [16]. Data from the DISCO multicentre international registry indicate that patients treated with DAPT had a significantly higher incidence of MACE (a composite of all-cause death, non-fatal MI, and any unplanned PCI) at one year compared to those receiving Single Antiplatelet Therapy (SAPT) [29]. To further clarify optimal treatment duration, a prospective, multicenter, pragmatic randomized clinical trial is underway, directly comparing 1-month SAPT with 1-year DAPT; its results are expected to inform future management of SCAD [30]. Collectively, these findings suggest that intensive or prolonged antiplatelet therapy may not benefit all patients. Experts from the Association of Cardiovascular Interventions of the Polish Cardiac Society propose a pragmatic strategy: asymptomatic patients may be treated with DAPT for a minimum of 3 months. Therapy can be discontinued earlier than initially planned if follow-up imaging with Coronary Computed Tomography Angiography or invasive angiography confirms vessel healing [31]. In our cohort, all patients received antiplatelet therapy at discharge (predominantly DAPT). In light of bleeding risk, therapy was uniformly discontinued at 12 months, save for seven cases with atherosclerosis or stents. Furthermore, the antiplatelet strategy was modified based on 6-month repeat CAG, and no bleeding events [Bleeding Academic Research Consortium (BARC) >3a] [32] were reported during follow-up. Follow-up CAG at 6 months revealed a high rate of spontaneous healing. In view of this finding, formulating an individualized antiplatelet strategy based on angiographic results and comorbidities appears rational. Given the high risk of iatrogenic dissection, the procedure must be conducted very carefully. In the present study, no procedural complication was observed during repeat CAG. Although not statistically significant, a higher MACE rate was observed among patients who completed the 6-month follow-up. This may be explained by the higher follow-up adherence in the PCI subgroup, coupled with the fact that two of the three patients who experienced MACE had initially been treated with PCI.

Limitations

This study has several limitations, primarily its small sample size and the low number of observed events, which consequently limit the conclusions that can be drawn regarding Major Adverse Cardiovascular Events (MACE) during long-term follow-up. Additionally, the study included patients with concomitant coronary artery atherosclerosis. Although enrolled for Spontaneous Coronary Artery Dissection (SCAD), their outcomes may have been influenced more by atherosclerotic disease progression than by SCAD-specific healing or recurrence. Optical Coherence Tomography (OCT) is known to be superior for visualization of intimal tears, intraluminal thrombosis, false lumens, and Intramural Hematoma (IMH). However, Intravascular Ultrasound (IVUS) also exhibits adequate resolution to visualize IMH and false lumens, which are used for the diagnosis of SCAD. Considering the potential risk of hydraulic extension with OCT contrast injection and higher experience of the physicians in operating IVUS at the center involved in the present study, IVUS rather than OCT was recommended, owing to safety concerns. The present study was not a randomized controlled trial comparing conservative therapy with PCI for SCAD, which limits the ability to directly compare outcomes between these strategies. Consequently, the findings may not be generalizable to the broader SCAD population.

Conclusion

This study reported that the outcomes of patients subjected to an initial conservative strategy were not inferior as compared to those treated with Percutaneous Coronary Intervention (PCI). In patients who underwent follow-up coronary angiography (CAG), healing was commonly observed. Therefore, guiding antiplatelet therapy based on angiographic findings and comorbidities appears rational.

Acknowledgements

The authors would like to thank all the reviewers who participated in the review and MJEditor (www.mjeditor.com) for its linguistic assistance during the preparation of this manuscript.

Disclosure of conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Abbreviations

ACEI/ARB, Angiotensin-Converting Enzyme Inhibitors/Angiotensin II Receptor Blockers; ACS, Acute Coronary Syndrome; AHA/ACC, American Heart Association/American College of Cardiology; CABG, Coronary Artery Bypass Grafting; CAG, Coronary Angiography; CCB, Calcium Channel Blockers; CCS, Canadian Cardiovascular Society Classification; CHF, Congestive Heart Failure; DAPT, Dual Antiplatelet Therapy; ECG, Electrocardiogram; FMD, Fibromuscular dysplasia; IQR, Interquartile Range; IMH, Intramural Hematoma; IVUS, Intravascular Ultrasound; LAD, Left Anterior Descending artery; LCX, Left Circumflex Artery; LVEF, Left Ventricular Ejection Fraction; MACE, Major Adverse Cardiovascular Events; MI, Myocardial Infarction; NSTEMI, Non-ST-Segment Elevation Myocardial Infarction; OCT, Optical Coherence Tomography; OM, Obtuse Marginal branch; PCI, Percutaneous Coronary Intervention; POBA, Plain Old Balloon Angioplasty; RCA, Right Coronary Artery; RMI, Recurrent Myocardial Infarction; R-SCAD, Recurrent Spontaneous Coronary Artery Dissection; SA, Stable Angina; SAPT, Single Antiplatelet Therapy; SCAD, Spontaneous Coronary Artery Dissection; SCIH, Spontaneous Coronary Intramural Hematoma; SD, Standard Deviation; STEMI, ST-Segment Elevation Myocardial Infarction; TIMI, Thrombolysis in Myocardial Infarction; UA, Unstable Angina.

Address correspondence to: Qing-Chi Liao, Department of Cardiology, Northern Jiangsu People's Hospital, No. 98 Nantong West Road, Guangling District, Yangzhou 225000, Jiangsu, China. E-mail: lqchyl@163.com

References

[1] Maehara A, Mintz GS, Castagna MT, Pichard AD, Satler LF, Waksman R, Suddath WO, Kent KM and Weissman NJ. Intravascular ultrasound assessment of spontaneous coronary artery dissection. Am J Cardiol 2002; 89: 466-468.

[2] Hayes SN, Kim ESH, Saw J, Adlam D, Arslanian-Engoren C, Economy KE, Ganesh SK, Gulati R, Lindsay ME, Mieres JH, Naderi S, Shah S, Thaler DE, Tweet MS and Wood MJ; American Heart Association Council on Peripheral Vascular Disease; Council on Clinical Cardiology; Council on Cardiovascular and Stroke Nursing; Council on Genomic and Precision Medicine; and Stroke Council. Spontaneous coronary artery dissection: current state of the science: a scientific statement from the American Heart Association. Circulation 2018; 137: e523-e557.

[3] Saw J. Coronary angiogram classification of spontaneous coronary artery dissection. Catheter Cardiovasc Interv 2014; 84: 1115-1122.

[4] Al-Hussaini A and Adlam D. Spontaneous coronary artery dissection. Heart 2017; 103: 1043-1051.

[5] Alfonso F, Paulo M, Gonzalo N, Dutary J, Jimenez-Quevedo P, Lennie V, Escaned J, Bañuelos C, Hernandez R and Macaya C. Diagnosis of spontaneous coronary artery dissection by optical coherence tomography. J Am Coll Cardiol 2012; 59: 1073-1079.

[6] Saw J, Mancini GB, Humphries K, Fung A, Boone R, Starovoytov A and Aymong E. Angiographic appearance of spontaneous coronary artery dissection with intramural hematoma proven on intracoronary imaging. Catheter Cardiovasc Interv 2016; 87: E54-E61.

[7] Mahmoud AN, Taduru SS, Mentias A, Mahtta D, Barakat AF, Saad M, Elgendy AY, Mojadidi MK, Omer M, Abuzaid A, Agarwal N, Elgendy IY, Anderson RD and Saw J. Trends of incidence, clinical presentation, and in-hospital mortality among women with acute myocardial infarction with or without spontaneous coronary artery dissection: a population-based analysis. JACC Cardiovasc Interv 2018; 11: 80-90.

[8] Buccheri D and Zambelli G. Focusing on spontaneous coronary artery dissection: actuality and future perspectives. J Thorac Dis 2016; 8: E1784-E1786.

[9] Krittanawong C, Kumar A, Wang Z, Johnson KW, Baber U, Palazzo A, Mehran R and Bhatt DL. Clinical features and prognosis of patients with spontaneous coronary artery dissection. Int J Cardiol 2020; 312: 33-36.

[10] Hayes SN, Tweet MS, Adlam D, Kim ESH, Gulati R, Price JE and Rose CH. Spontaneous coronary artery dissection: JACC state-of-the-art review. J Am Coll Cardiol 2020; 76: 961-984.

[11] Adlam D, Alfonso F, Maas A and Vrints C; Writing Committee. European Society of Cardiology, acute cardiovascular care association, SCAD study group: a position paper on spontaneous coronary artery dissection. Eur Heart J 2018; 39: 3353-3368.

[12] Prakash R, Starovoytov A, Heydari M, Mancini GB and Saw J. Catheter-induced iatrogenic coronary artery dissection in patients with spontaneous coronary artery dissection. JACC Cardiovasc Interv 2016; 9: 1851-1853.

[13] Saw J, Starovoytov A, Aymong E, Inohara T, Alfadhel M, McAlister C, Samuel R, Grewal T, Parolis JA, Sheth T, So D, Minhas K, Brass N, Lavoie A, Bishop H, Lavi S, Pearce C, Renner S, Madan M, Welsh RC, McGrath BM, Vijayaraghavan R, Har B, Ibrahim R, Chaudhary P, Ganesh SK, Graham J, Matteau A, Martucci G, Ko DT, Humphries K and Mancini GJ. Canadian spontaneous coronary artery dissection cohort study: 3-year outcomes. J Am Coll Cardiol 2022; 80: 1585-1597.

[14] Writing Committee Members; Lawton JS, Tamis-Holland JE, Bangalore S, Bates ER, Beckie TM, Bischoff JM, Bittl JA, Cohen MG, DiMaio JM, Don CW, Fremes SE, Gaudino MF, Goldberger ZD, Grant MC, Jaswal JB, Kurlansky PA, Mehran R, Metkus TS Jr, Nnacheta LC, Rao SV, Sellke FW, Sharma G, Yong CM and Zwischenberger BA. 2021 ACC/AHA/SCAI guideline for coronary artery revascularization: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol 2022; 79: e21-e129.

[15] Tweet MS, Eleid MF, Best PJ, Lennon RJ, Lerman A, Rihal CS, Holmes DR Jr, Hayes SN and Gulati R. Spontaneous coronary artery dissection: revascularization versus conservative therapy. Circ Cardiovasc Interv 2014; 7: 777-786.

[16] Chang S, Dai Y, Song X, Li C, Lu H, Huang Z, Ma J, Qian J and Ge J. Characteristics, management, and prognosis of spontaneous coronary intramural hematoma. Angiology 2022; 73: 374-379.

[17] Morosato M, Gaspardone C, Romagnolo D, Pagnesi M, Baldetti L, Dormio S, Federico F, Scandroglio AM, Chieffo A, Godino C, Margonato A, Adamo M, Metra M, Tchetche D, Dumonteil N, Tweet MS, Saw J and Beneduce A. Left main spontaneous coronary artery dissection: clinical features, management, and outcomes. JACC Cardiovasc Interv 2025; 18: 975-983.

[18] Saw J. Spontaneous coronary artery dissection. Can J Cardiol 2013; 29: 1027-1033.

[19] Buccheri D, Zambelli G, Alfonso F and Cortese B. Pulse on spontaneous coronary artery dissections: experience-based survey. JACC Cardiovasc Interv 2017; 10: 1469-1471.

[20] Tweet MS, Hayes SN, Pitta SR, Simari RD, Lerman A, Lennon RJ, Gersh BJ, Khambatta S, Best PJ, Rihal CS and Gulati R. Clinical features, management, and prognosis of spontaneous coronary artery dissection. Circulation 2012; 126: 579-588.

[21] García-Guimaraes M, Bastante T, Macaya F, Roura G, Sanz R, Barahona Alvarado JC, Tizón H, Flores-Ríos X, Moreu J, Ojeda S, Nogales JM, Veiga G, Masotti M, Camacho-Freire SJ, Jiménez-Valero S, Jiménez-Kockar M, Lozano Í, González-Ferreiro R, Velázquez M, Avanzas P, Rivero F and Alfonso F. Spontaneous coronary artery dissection in Spain: clinical and angiographic characteristics, management, and in-hospital events. Rev Esp Cardiol (Engl Ed) 2021; 74: 15-23.

[22] Saw J, Starovoytov A, Humphries K, Sheth T, So D, Minhas K, Brass N, Lavoie A, Bishop H, Lavi S, Pearce C, Renner S, Madan M, Welsh RC, Lutchmedial S, Vijayaraghavan R, Aymong E, Har B, Ibrahim R, Gornik HL, Ganesh S, Buller C, Matteau A, Martucci G, Ko D and Mancini GBJ. Canadian spontaneous coronary artery dissection cohort study: in-hospital and 30-day outcomes. Eur Heart J 2019; 40: 1188-1197.

[23] Lettieri C, Zavalloni D, Rossini R, Morici N, Ettori F, Leonzi O, Latib A, Ferlini M, Trabattoni D, Colombo P, Galli M, Tarantini G, Napodano M, Piccaluga E, Passamonti E, Sganzerla P, Ielasi A, Coccato M, Martinoni A, Musumeci G, Zanini R and Castiglioni B. Management and long-term prognosis of spontaneous coronary artery dissection. Am J Cardiol 2015; 116: 66-73.

[24] Alfonso F, Paulo M, Lennie V, Dutary J, Bernardo E, Jiménez-Quevedo P, Gonzalo N, Escaned J, Bañuelos C, Pérez-Vizcayno MJ, Hernández R and Macaya C. Spontaneous coronary artery dissection: long-term follow-up of a large series of patients prospectively managed with a “conservative” therapeutic strategy. JACC Cardiovasc Interv 2012; 5: 1062-1070.

[25] Samuel R, Alfadhel M, McAlister C, Nestelberger T, Starovoytov A, Parolis JA, Grewal T, Aymong E and Saw J. De novo left main spontaneous coronary artery dissection: a case se-

ries. JACC Cardiovasc Interv 2022; 15: 2349-2351.
[26] Benenati S, Giacobbe F, Zingarelli A, Macaya F, Biolè C, Rossi A, Pavani M, Quadri G, Barbero U, Erriquez A, Aranzulla T, Cavallino C, Buccheri D, Rolfo C, Patti G, Gonzalo N, Chinaglia A, Musumeci G, Escaned J, Varbella F, Cerrato E and Porto I; DISCO Collaborators. Interventional versus conservative strategy in patients with spontaneous coronary artery dissections: insights from DISCO registry. Circ Cardiovasc Interv 2023; 16: e012780.
[27] Eleid MF, Guddeti RR, Tweet MS, Lerman A, Singh M, Best PJ, Vrtiska TJ, Prasad M, Rihal CS, Hayes SN and Gulati R. Coronary artery tortuosity in spontaneous coronary artery dissection: angiographic characteristics and clinical implications. Circ Cardiovasc Interv 2014; 7: 656-662.
[28] Collet JP, Thiele H, Barbato E, Barthélémy O, Bauersachs J, Bhatt DL, Dendale P, Dorobantu M, Edvardsen T, Folliguet T, Gale CP, Gilard M, Jobs A, Jüni P, Lambrinou E, Lewis BS, Mehilli J, Meliga E, Merkely B, Mueller C, Roffi M, Rutten FH, Sibbing D and Siontis GCM; ESC Scientific Document Group. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J 2021; 42: 1289-1367.
[29] Cerrato E, Giacobbe F, Quadri G, Macaya F, Bianco M, Mori R, Biolè CA, Boi A, Bettari L, Rolfo C, Ferrari F, Annibali G, Scappaticci M, Pavani M, Barbero U, Buccheri D, Cavallino C, Lombardi P, Bernelli C, D'Ascenzo F, Infantino V, Gambino A, Cinconze S, Rognoni A, Montagna L, Porto I, Musumeci G, Escaned J and Varbella F; DISCO Collaborators. Antiplatelet therapy in patients with conservatively managed spontaneous coronary artery dissection from the multicentre DISCO registry. Eur Heart J 2021; 42: 3161-3171.
[30] Alfonso F, de la Torre Hernández JM, Ibáñez B, Sabaté M, Pan M, Gulati R, Saw J, Angiolillo DJ, Adlam D and Sánchez-Madrid F. Rationale and design of the BA-SCAD (Beta-blockers and antiplatelet agents in patients with spontaneous coronary artery dissection) randomized clinical trial. Rev Esp Cardiol (Engl Ed) 2022; 75: 515-522.
[31] Kądziela J, Kochman J, Grygier M, Michałowska I, Tomaniak M, Wojakowski W, Araszkiewicz A, Dąbrowski M, Hawranek M, Huczek Z, Kralisz P, Kusa J, Roleder T, Januszewicz A, Witkowski A, Adlam D and Bartuś S. The diagnosis and management of spontaneous coronary artery dissection - expert opinion of the Association of Cardiovascular Interventions (ACVI) of Polish Cardiac Society. Kardiol Pol 2021; 79: 930-943.
[32] Tzikas A, Holmes DR Jr, Gafoor S, Ruiz CE, Blomström-Lundqvist C, Diener HC, Cappato R, Kar S, Lee RJ, Byrne RA, Ibrahim R, Lakkireddy D, Soliman OI, Nabauer M, Schneider S, Brachmann J, Saver JL, Tiemann K, Sievert H, Camm AJ and Lewalter T. Percutaneous left atrial appendage occlusion: the Munich consensus document on definitions, endpoints, and data collection requirements for clinical studies. Europace 2017; 19: 4-15.