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MINI-REVIEW

Revascularization in Patients With Spontaneous Coronary Artery Dissection: Where Are We Now?

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Key Words: percutaneous coronary intervention ■ revascularization ■ spontaneous coronary artery dissection

Spontaneous coronary artery dissection (SCAD) is a heterogeneous condition that often presents as an acute coronary syndrome in young patients with a paucity of cardiovascular risk factors. In most registries, >90% of patients with SCAD are women.[1] SCAD is primarily a non-atherosclerotic, non-calcified, non-iatrogenic dissection of the coronary artery resulting from either an intramural hematoma (IMH) alone, an intimal tear alone, or both an IMH and an intimal tear. Recent studies suggested that an IMH usually precedes the occurrence of an intimal tear.[2,3] Although underlying mechanisms remain unclear, fibromuscular dysplasia, pregnancy, as well as certain autoimmune and inflammatory disorders have been reported as possible predisposing factors in patients with SCAD.[1] To date, there are no randomized clinical trials comparing revascularization versus conservative management for SCAD; thus, SCAD management is primarily based on observational data and expert opinion[1,4] (Table 1).[5-22]

A retrospective analysis from the Mayo Clinic showed that in patients presenting with SCAD and low-risk features (clinically stable with TIMI [Thrombolysis in Myocardial Infarction] flow 2–3), both conservative management and revascularization were associated with low mortality (1 of 94 conservative versus 1 of 95 revascularization).[20] However, an earlier systematic review inclusive of 440 patients from a total of 381 reports identified in a Medline database search between 1931 and 2008 demonstrated that 21% of conservatively treated patients with SCAD required subsequent revascularization because of early SCAD progression and recurrent ischemia.[23] A recent study (n=750 patients) showed that 8.8% of conservatively treated patients with SCAD had major adverse cardiac events within 30 days of initial presentation.[24] Another recent study showed that SCAD-related ST-segment–elevation myocardial infarction (STEMI) had a higher frequency of left main or left anterior descending culprit lesions (13% versus 1%; *P*=0.003), and cardiogenic shock (19% versus 9%; *P*=0.026) compared with STEMI attributable to atherosclerotic plaque disruption.[9] We herein summarize the contemporary evidence for revascularization after SCAD and review the indications, techniques, and factors influencing its outcomes.

WHICH PATIENTS WITH SCAD SHOULD UNDERGO REVASCULARIZATION?

The decision to revascularize patients with SCAD is challenging, given the associated complications

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Table 1. Registries in Patients With SCAD Comparing Conservative Management Versus Revascularization

Author	Y	No.	Age, y	Women (%)	Follow-Up	STEMI (%)	NSTEMI (%)	ACS (%)	Mortality %	Medically Managed (%)	Revascularization (%)
Daoulah et al (G-SCAD Registry)[5]	2020	83	44 [37–55]	51	18.8 mo (9.06–40.1)	49.4	46.9	100	1.2	39.8	60.2
García-Guimaraes et al[6]	2020	318	53 [47–60]	88	4 y	39	53	94	1.3	78	22
Inoue et al[7]	2020	19	52.1±6.1	100	960 d (686–1504)	57.8	21	100	0	63	37
Liu et al[8]	2019	118	57±10	14	43 mo	23.7	23.7	74.6	6.8	72	28
Lobo et al[9]	2019	53	49	95	3 y	100	0	100	1.9	30	70
Abreu et al[10]	2018	27	56	82	20 mo	37	51.9	89	0	55	45
Cade et al[11] (P-SCAD)	2017	13	33.8±3.7	85	14 mo	46	46	92	7	54	46
Saw et al[12]	2017	327	52.5	91	3.1 y	25.7	74.3	100	0	83.2	18.7
Rogowski et al[13]	2017	64	53	94	4.5 y	30	69	100	N/A	88	12
Nakashima et al[14]	2016	63	46	94	34 mo	87	13	100	N/A	45	55
McGrath-Cadell et al[15]	2016	40	45	95	16 mo	30	65	100	N/A	67	33
Godinho et al[16]	2016	17	51±9	59	52 mo	59	41	100	0	77	23
Roura et al[17]	2016	34	47	95	4 mo	55	N/A	N/A	N/A	77	23
Lettieri et al[18]	2015	134	52	82	72 mo	40.3	49.2	93	2.2	59	41
Saw et al[19]	2014	168	52	93	6.9 y	26	74	100	0	80	20
Tweet et al[20]	2014	189	44	93	2.3 y	37	N/A	N/A	0.5	50	50
Buja et al[21]	2013	38	51.4±11.6	84.2	17 mo	50	29	79	0	48	52
Mortensen et al[22]	2009	22	48.7±8.9	78	3.6±2.9 y	72	N/A	100	N/A	31	69

ACS indicates acute coronary syndrome; NSTEMI, non–ST-segment–elevation myocardial infarction; SCAD, spontaneous coronary artery dissection; and STEMI, ST-segment–elevation myocardial infarction.

and technical difficulties. An assessment of the clinical presentation and angiographic features is critical when determining whether, when, and how to revascularize patients with SCAD. Invasive coronary angiography is the diagnostic modality of choice for SCAD, which according to the Saw classification, can be angiographically classified into 3 types.[25] (Figure) The angiographic characteristics of type 1 SCAD include a dissection flap, false lumen appearance, contrast staining of the arterial wall, and late contrast clearing. Type 2 SCAD consists of a long diffuse (usually >30 mm) lesion with abrupt changes in the arterial caliber from its normal diameter to diffuse smooth narrowing. Type 2 SCAD can also be classified into type 2A (diffuse narrowing bordered by normal artery segments) and 2B (diffuse narrowing extending to the apical tip of the artery). Similar to atherosclerosis, the angiographic characteristics of type 3 SCAD include single or multiple focal stenoses attributable to intramural hematoma. In patients with high clinical suspicion for SCAD (eg, young women with AMI and no cardiovascular risk factors), SCAD should be initially evaluated by prompt coronary angiography, with particular emphasis on identifying type 1 and 2 SCAD. Intracoronary imaging such as optical coherence tomography (OCT) or intravascular ultrasound (IVUS) may be further required to distinguish between specific SCAD types, especially between type 3 SCAD and coronary atherosclerotic disease. The aforementioned angiographic classification assists with angiographic recognition of SCAD as an entity but does not direct the choice of therapy and has not been shown to impact outcomes. Patients with SCAD with completely occluded coronary arteries subtending a large area of jeopardized myocardium, cardiogenic shock, active/ongoing ischemia with persistent compromise in coronary blood flow, hemodynamic instability, ventricular arrhythmias, STEMI, and high-risk anatomy (eg, TIMI flow 0–1, left main dissection and proximal dissection) should be considered for revascularization.[1] In addition, the classification of SCAD by the presence or absence of IMH, presence of additional intimal dissections, and length/stenosis parameters can inform the clinician as to the patient's risk of early serious SCAD extension. In a retrospective study of 240 patients with SCAD, Waterbury et al found that the angiographic finding of isolated IMH (ie, absence of intimal tear) was associated with a higher risk of early clinically important extension of SCAD in patients treated with conservative management.[2] Lesion length and stenosis severity were additional parameters that conferred a higher risk of acute extension. Angiographic features may be dynamic, as recurrent SCAD often involves coronary segments not affected previously and may manifest as a different angiographic type than that of the initial presentation. However, revascularization is associated with suboptimal procedural success rates and high rates of

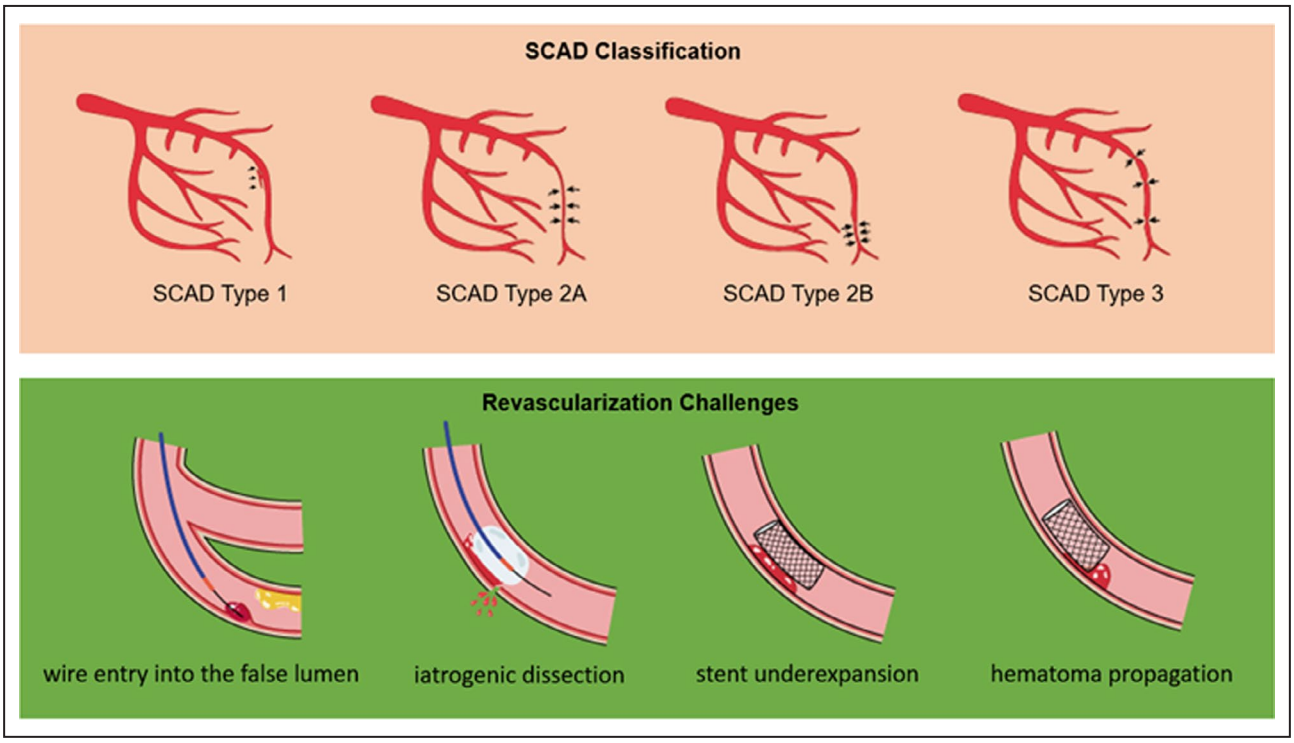

Figure. Spontaneous coronary artery dissection classification and revascularization challenges.
SCAD indicates spontaneous coronary artery dissection.

complications despite preserved coronary flow.[19,20,26] Most importantly, revascularization strategies may not protect against future target vessel revascularization or recurrent SCAD.[20] Overall, revascularization should be avoided, particularly in patients with low-risk features (eg, no active ischemia, coronary dissection in an arterial segment subtending a small territory), because the majority of dissections in patients with SCAD will heal spontaneously. In a study of 156 patients with SCAD (182 lesions) who underwent repeat coronary angiography with a median time of 154 days (interquartile range, 70–604 days) for a variety of indications, 95% of SCAD lesions were healed when angiography was performed ≥30 days after the acute SCAD event.[27]

REVASCULARIZATION STRATEGIES AND TECHNIQUES

Prompt coronary revascularization is a cornerstone of SCAD management in the context of major coronary occlusion. However, SCAD has a different pathophysiological process involving a dissected artery, and percutaneous coronary intervention (PCI) for SCAD is associated with lower rates of procedural success, higher rates of disease-specific technical challenges, and increased rates of complications compared with PCI for atherosclerotic acute coronary syndrome. Challenges include coronary artery fragility, iatrogenic dissection, wire entry into a false lumen, abrupt vessel occlusion, dissection extension, late strut mal-apposition, and hematoma propagation with stent placement requiring additional unplanned stents[19,26] (Figure). Several small observational studies showed lower success rates and higher rates of complications in PCI for SCAD.[13,14,18] In a study of 34 patients with SCAD who underwent PCI, 8 patients (24%) were complicated by propagation of the dissection flap, and in 2 patients there was an inability to pass the wire distally into the true lumen, leading to dissection with a lower final TIMI flow grade ultimately requiring emergent CABG.[14] In a study of 56 patients with SCAD who were treated with revascularization (51 for PCI and 5 for CABG), 3 patients (5.8%) treated with PCI were switched to urgent CABG for procedural failure; one died from retrograde aortic dissection after PCI on the proximal LAD, and another patient died after emergency CABG for STEMI and multivessel SCAD complicated by cardiogenic shock.[18] Among PCI-treated patients, 5 had repeated percutaneous revascularization; one for late stent thrombosis, 2 for recurrent spontaneous dissection in other vessels, one for progression of dissection distal to the implanted stent, and one for restenosis.

In a preliminary report from the Vancouver General Hospital SCAD registry, of all patients with SCAD with stents, 6.9% required bail-out surgery, 1.4% had stents placed into the false lumen, 9.7% had iatrogenic dissection, and 2.8% suffered stent thrombosis.[28] Compared with atherothrombotic STEMI, emergent PCI for SCAD-related STEMI was associated with lower rates of achieving TIMI grade 3 flow (91% versus 98%, P=0.016) and a 9% failure rate (mostly related to the inability to access the true lumen or to residual stenosis >50%), with more and longer stents (mean stent length was 62±37 mm; range 12–140 mm) required for SCAD lesions.[9] Thrombolytic therapy for SCAD-related STEMI should be avoided because of the conceptual hazard of extension of the dissection or hematoma.

In theory, compared with radial access, femoral access could be associated with more complications and bleeding given SCAD's propensity to affect young women and its association with fibromuscular dysplasia/arteriopathies. However, radial access may result in noncoaxial engagement in the coronary ostium, deep catheter engagement, and frequent need for more aggressive catheter manipulation.[29] The American Heart Association Scientific Statement on SCAD recommends femoral access over radial access, and that extra caution be taken with radial catheterization if required. The study reported guide-induced iatrogenic dissections occurred in 3 of 42 (7.1%) radial-approach angiograms, including 2 extensive LM dissections requiring emergent CABG.[30] In the Vancouver General Hospital SCAD registry, cases with iatrogenic coronary artery dissection had an increased proportion of radial access (50.0%) than non-iatrogenic coronary artery dissection cases (16.4%; P=0.009), compared with femoral access.[29]

CABG can be considered for unstable patients in certain specific scenarios (eg, left main dissections with ongoing ischemia/infarction, severe proximal 2-vessel dissection, or in the case of PCI failure). CABG should also be considered when PCI is technically challenging or has been attempted and unsuccessful.[31] One small study of patients with SCAD (5 left main dissections and 1 right coronary artery dissection) showed that all 5 patients with left main SCAD had favorable outcomes following CABG, while one patient with SCAD of the RCA died on the 30th postoperative day.[32] Tweet et al demonstrated good early outcomes with CABG (n=20 patients with SCAD) and comparable 5-year event rates to those treated conservatively.[20] Although significant late graft occlusion occurred (11 of 16), perhaps because of restored flow from spontaneous healing over time, there was no further increase in mortality at 5 years.[20] Notably, there is no evidence that CABG protects against recurrent SCAD, and conduit failure may occur because of poor distal targets affected by

the SCAD. Clearly these studies are confounded by selection bias and the findings should be interpreted with caution. In addition, fragile and dissected coronary artery tissue may be more prone to anastomosis complications during CABG in patients with SCAD.[31] Moreover, vein grafts should be considered to preserve arterial conduits for future use, if needed, in light of the high incidence of late graft failure attributable to expected native vessel healing.[1]

A key concept in the revascularization of patients with SCAD is to reestablish coronary blood flow rather than restore normal coronary architecture (Table 2). In the absence of data from randomized controlled trials, most of the described revascularization techniques in patients with SCAD are based on clinical experience and expert opinion. For example, undersized balloon angioplasty may be considered to restore flow in focal and distal lesions, while fenestration of the intramural hematoma by regular or cutting balloon angioplasty may be considered to reduce the true lumen's compression[12] in long and diffuse lesions. Some studies suggested that a hybrid approach using cutting balloon angioplasty and stenting may be considered for the compressive intramural hematoma to prevent late mal apposition once the hematoma is resorbed.[33] Some consider the use of a non-hydrophilic wire preferable to avoid extending the dissection by entering the false lumen.[34] Some operators suggest that starting with a floppy wire and then escalating to a hydrophilic wire or a stiff wire, if needed, leads to a high rate of PCI success (up to 71.4%).[34] IVUS and OCT may be considered to confirm the diagnosis, ensure proper positioning of the guidewire, confirm true lumen entry, and optimize stent parameters. IVUS can identify the false lumen in detail and reduce the use of contrast agents. On the other hand, OCT has a high resolution for confirming guidewire position, intramural hematoma location, and optimal stenting, but requires filling of the coronary artery with contrast media and thus carries a risk of hydraulic extension of the SCAD.[25] Overall, IVUS and OCT should be encouraged for PCI in patients with SCAD as complications from these advanced imaging techniques in SCAD (eg, dissection of the imaged vessel and stent deformation) are rare. Undersized stents may increase the risk of restenosis and stent thrombosis once the hematoma has resolved, while oversized stents may cause an extension of the dissection flap. When treating long lesions, a multistep approach of stenting may be considered, for example stenting the distal edge followed by the proximal edge, and then finally stenting the middle portion of the dissected artery to avoid hematoma propagation. Only 1 study compared stent types in patients with SCAD. A non-significant trend towards a lower rate of major adverse cardiovascular events occurrence in the drug-eluting stent group was observed after a median follow-up time of 3.3 years (17% versus 31%, P=0.11) compared with the bare metal stent group,

Table 2. Summary of Proposed Invasive Strategies and Post-Revascularization Care

Invasive Strategies	Post-Revascularization Care
IVUS or OCT guided wire and stent placement should be considered for identifying the compressed true lumen and stent optimization	Repeat angiography should be considered in patients with indication for angiography (eg, recurrent or ongoing chest pain) and revascularization is felt necessary for ongoing, unstable ischemia (or in the context of recurrent SCAD)
Start with a floppy wire, particularly in patients with SCAD with TIMI 0/1, and then escalate to a hydrophilic wire or stiff wire if needed	Follow-up computerized tomography coronary angiography may also be considered medically managed patients, particularly with type 1 dissections, while follow-up echocardiography should be considered if left ventricular systolic dysfunction
To reduce the risk for catheter-induced dissection, avoid deep catheter engagement, keep coaxial non-deep catheter intubation, and limit the force of injection	Beta blockers are recommended in all patients with SCAD
If possible, direct stenting without pre- and post-dilatation to reduce risk of hematoma extension is preferred. If balloon angioplasty needed, a hybrid approach with cutting balloon to fenestrate the intimal flap before stent implantation should be considered	Angiotensin-converting enzyme inhibitor/angiotensin-receptor blocker should be considered if evidence of left ventricular systolic dysfunction
Three-stent technique or multi-stent approach should be considered by stenting the distal segment first followed by the proximal segment and finally the mid-part to prevent hematoma extension	Cardiac rehabilitation should be considered in all patients with SCAD
Long stents covering additional 5–10 mm on both proximal and distal edges beyond the margins of the dissection should be considered	Colchicine is not routinely recommended but may be considered in inflammatory conditions related to SCAD (eg, eosinophilic coronary periarteritis)
Avoid 1:1 vessel:stent sizing and optimal apposition and self-expanding stents should be considered	Referral to a specialist in medical genetics may be considered (eg, genetic testing is often directed at identification of an underlying systemic arteriopathy or connective tissue disorder)

ACS indicates acute coronary syndrome; IVUS, intravascular ultrasound; OCT, optical coherence tomography; SCAD, spontaneous coronary artery dissection; and TIMI, Thrombolysis in Myocardial Infarction.

a difference that was mainly driven by target-vessel revascularization.[35] Overall, IVUS and OCT are generally safe in patients with SCAD, and it is important to remember that stents do not prevent SCAD recurrence and may increase rates of repeat revascularization, especially when long stents are placed.[20] Bioresorbable stents have been proposed because of their ability to preserve tissue biomechanics and recover the endothelial function, but data on their use in patients with SCAD are limited.[36]

MECHANICAL CIRCULATORY SUPPORT

Current literature on SCAD and cardiogenic shock with/without mechanical circulatory support is limited. In a systematic review of pregnancy-associated SCAD, 29 patients with SCAD developed cardiogenic shock requiring placement of an intra-aortic balloon pump and subsequently revascularization or heart transplant.[37] In a case series of 4 patients with SCAD and cardiogenic shock, Impella provided valuable procedural support in those patients with cardiogenic shock, especially in cases without evidence of ongoing ischemia.[38] In theory, mechanical circulatory support should be considered in SCAD patients with cardiogenic shock without evidence of ongoing ischemia (no need for revascularization) to temporarily support the myocardium while allowing SCAD vessels to heal independently. In addition, some cases demonstrate the beneficial use of mechanical circulatory support devices as a bridge to recovery or heart transplant in the setting of refractory cardiogenic shock because of SCAD.[39] Several case reports demonstrate the feasibility of mechanical circulatory support using extracorporeal membrane oxygenation.[40,41] These cases provide additional data on the safety of venoarterial extracorporeal membrane oxygenation in the management of cardiogenic shock secondary to SCAD.

POST-REVASCULARIZATION CARE

Several ongoing clinical trials and registries in patients with SCAD are currently underway (Table 3). Although a paucity of data on re-imaging in patients with SCAD exists, repeat angiography should be considered in patients with an indication for angiography (eg, recurrent or ongoing chest pain), and revascularization is necessary for ongoing, unstable ischemia (or in the context of recurrent SCAD).[1,31] It takes time for dissected vessels to heal, and evidence of residual dissection in the absence of clinical ischemia would not be an indication for revascularization.[1] Follow-up computerized tomography coronary angiography can also be considered in medically managed patients, particularly those with type 1 dissections. No clinical trial data exist to guide the need and timeline for repeat imaging, the duration of DAPT, or the merits of other pharmacotherapies in patients with SCAD. The American Heart Association Scientific Statement on SCAD suggests using DAPT as one would base on current guidelines for non-SCAD PCI.[1] In individuals at higher risk of bleeding events, per the expert consensus, consideration of recommending DAPT for at least 2 to 4 weeks after SCAD and low-dose aspirin alone is reasonable. Long-term DAPT may be considered in cases of severe late-acquired stent mal-apposition observed at follow-up.[31] Aspirin and clopidogrel are generally used, given the higher risk of bleeding with the newer generation $P2Y_{12}$ receptor inhibitors. In general, some experts recommended lifelong low-dose aspirin DAPT for 1 year following the current guideline-based therapy for acute coronary syndrome.[32] In fact, the duration of DAPT therapy remains controversial and depends on the location of SCAD, revascularization modality, and the number and size of stents used. For example, there is a theoretical concern that antiplatelet therapy in patients with SCAD with IMH could potentially cause bleeding or dissection extension, even with low-dose aspirin monotherapy. Further study of antiplatelet therapy in patients with SCAD is needed. Beta-blockers may be considered in all patients with SCAD, as they likely help mitigate SCAD recurrence, possibly by reducing coronary arterial shear stress and reversing catecholamine-mediated cardiac dysfunction.[12] Angiotensin-converting enzyme inhibitor/angiotensin-receptor blocker and follow-up echocardiography should be considered if evidence of left ventricular systolic dysfunction is present. Statin use in patients with SCAD remains controversial but should be considered in patients with elevated atherosclerotic risk. Cardiac rehabilitation may be considered in all patients with SCAD to expedite their recovery and improve quality of life.[19]

CONCLUSIONS

Ascertaining the appropriateness of revascularization, choosing the safest modality, and using optimal techniques are critically important in the treatment of patients with SCAD. Patients presenting with STEMI, hemodynamic instability, active/ongoing ischemia, sustained ventricular arrhythmias, cardiac arrest, proximal coronary occlusions, and those who progress to occlusion after initial conservative management should be considered for coronary revascularization. PCI is the most common revascularization strategy for SCAD. However, CABG may be considered in those with high-risk features (eg, left main dissections with ongoing

Table 3. Current Ongoing Clinical Trials and Registries in Patients With SCAD

Trial or Registry	No. (n)	Aims	Exclusion Criteria	Follow-Up	Status
The "Virtual" Multicenter SCAD Registry (NCT01429727)	Estimated 900 participants	Describe the clinical and physiologic characteristics of SCAD	Lack of angiographic confirmation of SCAD Iatrogenic dissection or an alternate diagnosis	10–15 y	Recruiting
Canadian SCAD Study (NCT02188069)	750 participants	In-hospital outcome, follow-up outcome of SCAD	Patients with troponin-negative ACS or typical atherosclerotic coronary artery disease with diameter stenosis ≥50%	3 y	Active, not recruiting
iSCAD Registry (NCT04496687)	Estimated 1000 participants	Describe the clinical and demographic characteristics of SCAD as well as clinical and psychological outcomes	Coronary dissection in association with atherosclerosis or as a result of iatrogenic injury	3 y	Recruiting
SAFER-SCAD (Statin and Angiotensin-converting Enzyme Inhibitor on Symptoms in Patients With SCAD) (NCT02008786)	40 participants	1) Rosuvastatin 10–20mg daily vs placebo 2) Ramipril vs placebo	Patients with glomerular filtration rate <50 mL/min; prior intolerance or allergy to rosuvastatin or Ramipril; coronary flow reserve >3.0; coronary artery disease (stenosis >50% in any artery) or residual dissection >50% with distal flow abnormalities		Recruiting
Angiographic and Psychosocial Evaluation of Peripartum vs Non: SCAD (NCT03390998)	241 participants	Determine differences in clinical and imaging presentation, in-hospital management and prognosis in peri-partum and non-peri-partum patients with SCAD	Male patients or patients with not a known or suspected diagnosis of SCAD	Cross-sectional	
Spontaneous Coronary Artery Dissection Registry (DIssezioni Spontanee COronariche ITalian-SPAnish) (DISCO-IT/SPA) (NCT04415762)	314 participants	Clinical characteristics, predisposing factors of SCAD, the incidence of SCAD recurrence The impact of a single antiplatelet therapy vs DAPT, impact of different angiographic SCAD type on outcome	Age <18 y or inability to provide informed consent	Retrospective	
Spontaneous Coronary Artery Dissection National Swiss Registry (SwissSCAD) (NCT04457544)	1000 participants	Demographic and procedural data in patients with SCAD	Patients with atherosclerotic or iatrogenic coronary dissection	5 y	Recruiting
The Study of the Prevalence Fibromuscular Dysplasia in Patient With Haematoma or Spontaneous Coronary Artery Dissection (DISCO) (NCT02799186)	200 participants	Prevalence of the FMD in SCAD and predisposing factors of SCAD and FMD	Coronary dissection with traumatic or iatrogenic origin	Interventional	
Spanish Registry on Spontaneous Coronary Artery Dissection (SR-SCAD) (NCT03607981)	300 participants	To assess clinical/angiographic characteristics, predisposing factors, associated conditions, risks of major adverse cardiovascular events and recurrent SCAD	Unable to provide informed consent.	3 y	Recruiting
Genetics of Spontaneous Coronary Artery Dissection (SCAD-INSPIRE Genetics) (NCT03876847)	100 participants	To determine genetic variants in patients with SCAD	Three cardiologists determine the coronary angiography was not a result of SCAD; poor quality images, heart, or bone marrow transplant	Cross-sectional	Enrolling by invitation
Spontaneous Coronary Artery Dissection anaLysIs of the Brazilian Updated Registry (SCALIBUR) (NCT03398850)	250 participants	Demographic and angiographic characteristics, type of treatment, long-term follow-up in SCAD.	Coronary dissections attributable to atherosclerotic plaque or trauma induced	10 y	Recruiting
SCAD and Autoimmunity (NCT03941184)	600 participants	To determine whether SCAD is associated with autoimmune diseases	Adults aged <18 y or >110 y	Case-control	Recruiting
Defining the Basis of Fibromuscular Dysplasia (DEFINE) (NCT01967511)	600 participants	The identification of regulatory gene networks, fibroblasts, DNA, plasma between FMD, SCAD, and CvAD	Patients with life expectancy to one year, organ transplantation, active autoimmune disease, Illicit drug use, HIV positive, prior malignancy, or PAD	Case-control	Recruiting

ACS indicates acute coronary syndrome; CvAD, cervical artery dissection; FMD, fibromuscular dysplasia; PAD, peripheral artery disease; and SCAD, spontaneous coronary artery dissection.

ischemia/infarction, severe proximal 2-vessel dissection, or in the case of PCI failure). Lifelong low-dose aspirin is recommended, and the duration of DAPT (preferably clopidogrel as a second antiplatelet agent) after stenting is tailored to the location of SCAD as well as the number and size of stents used. Beta-blockers and cardiac rehabilitation are preferred in all patients with SCAD, while statins and angiotensin-converting enzyme inhibitors/angiotensin-receptor blockers may be considered selectively when other clinical indications exist.

ARTICLE INFORMATION

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