

## Spontaneous Coronary Artery Dissection: What Do We Know? A Case Report

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### Abstract

### Case Report

Spontaneous coronary artery dissection (SCAD) has long been recognized as a cause of acute coronary syndromes (ACS) with very low prevalence (3% in most cohorts). It predominantly occurs in young to middle-aged women. SCAD patients have fewer traditional cardiovascular risk factors for ischaemic heart disease than patients with atherosclerotic coronary artery disease, however, many patients do have some risk factors for ischaemic heart disease including hypertension, smoking, and dyslipidaemia, although there is no evidence these contribute directly to the risk of SCAD. We report in this case an observation of spontaneous coronary dissection, in order to discuss its pathogenesis, diagnosis and management.

**Keywords:** SCAD, acute coronary syndrome, Conservative therapy.

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## INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is defined as an epicardial coronary artery dissection that is not associated with atherosclerosis or trauma and not iatrogenic.

Since the advances in coronary angiography and endocoronary imaging techniques, this entity is now recognized as a non-negligible cause of acute coronary syndrome in young subjects, especially females, with few or no traditional risk factors.

Except the atypical risk population, the clinical presentation is similar to an acute coronary syndrome, the long-term prognosis is generally good, a few rare cases of sudden death have been reported in the literature [1].

In stable patients, management should be as conservative as possible, however in case of coronary occlusion, hemodynamic instability or threatening arrhythmia, revascularization is required. The success rate of revascularization techniques remains low (around 50%) [1].

We expose through this case report the clinical peculiarities of this entity and the management modalities.

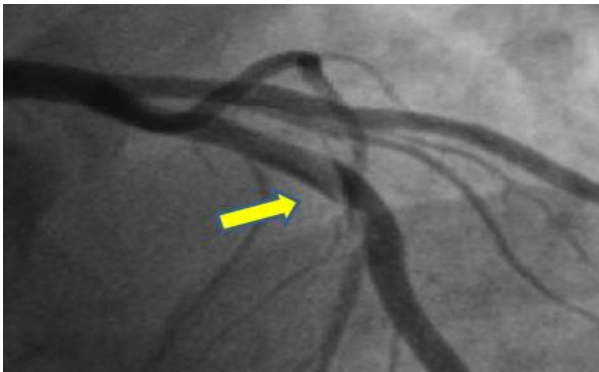
## CASE REPORT

We present the case of a 57-year-old patient, non-insulin-dependent diabetic, active chronic tobacco user at a rate of 40 PY, with no particular medical history, and no previous angina symptoms; who was admitted to emergency department at H6 of a typical infarctoid pain without any signs of hemodynamic instability. His ECG showed sinus rhythm with antero-septo-apical ST elevation. The transthoracic echocardiography showed concordant segmental left ventricular hypokinesis with ECG findings.

A coronary angiography (Figure 1 & 2) was performed and showed a dissection in the proximal segment of the left anterior descending artery (LAD 1). 5 criteria confirmed the diagnosis of a spontaneous dissection with a TIMI 3 flow:

1. Absence of atherosclerotic plaques
2. Visualization of an endoluminal flap indicating an intimal rupture
3. Contrast medium stagnation in the false lumen
4. Smooth reduction of the luminal diameter suggesting an extrinsic compression by the hematoma
5. The limitation of the dissection at the origins of side branches: the bifurcation zones are more solid and prevent the longitudinal extension of the hematoma or the dissection.

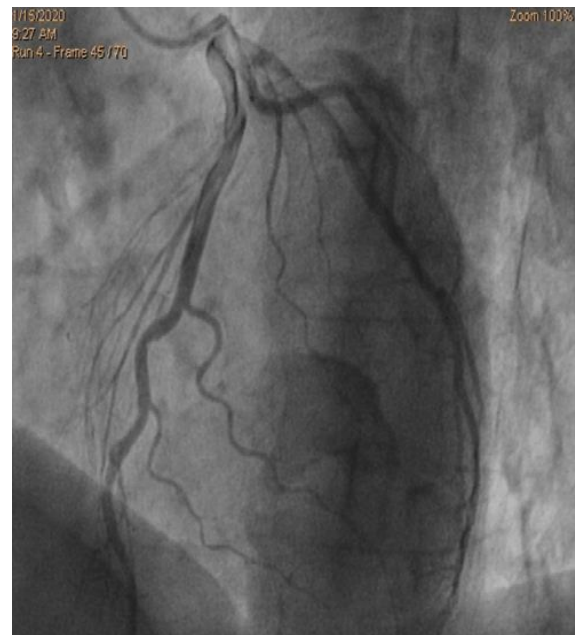
Given the absence of chest pain recurrence and the low angiographic risk, we opted for a conservative strategy: medical treatment with aspirin and beta-blockers and strict monitoring in intensive care unit for one week. Supra-aortic trunk echodoppler and renal arteries doppler were performed as part of the fibromuscular dysplasia (FMD) investigations and turned normal. Clinical follow-up was normal; The angiographic control (Figure 3) performed after 6 weeks showed complete spontaneous healing and disappearance of the dissection line.



**Fig 1: Reduction of luminal diameter and visualization of the false lumen and**



**Fig 2: Contrast medium stagnation and limitation of the dissection at the bifurcation with the first side branch**



**Fig 3: Angiographic control: disappearance of the dissection line**

## DISCUSSION

Spontaneous coronary artery dissection (SCAD) is defined as an epicardial coronary artery dissection that is not associated with atherosclerosis or trauma and not iatrogenic. The predominant mechanism of myocardial injury occurring as a result of SCAD is coronary artery obstruction caused by formation of an intramural hematoma (IMH) or intimal disruption rather than atherosclerotic plaque rupture or intraluminal thrombus.

SCAD is an underdiagnosed condition. Missed diagnoses are driven by a low suspicion of ACS in young women even in the presence of classic presenting symptoms, limitations of current coronary angiographic techniques, and lack of clinician familiarity with the condition. SCAD most commonly occurs in patients with few or no traditional cardiovascular risk factors. First studies suggested a prevalence of 0.2- 1% of ACS. Recent series using careful diagnostic criteria that exclude iatrogenic, traumatic, and atherosclerotic dissection suggest that SCAD may be a cause of up to 1% to 4% of ACS cases overall [1, 2].

The incidence of SCAD in young women has been more explored in late decades. It typically affects women between 45 and 55 years old; In Canadian and Japanese ACS registers involving women under 50 year old, the prevalence were respectively 24% and 35% [3].

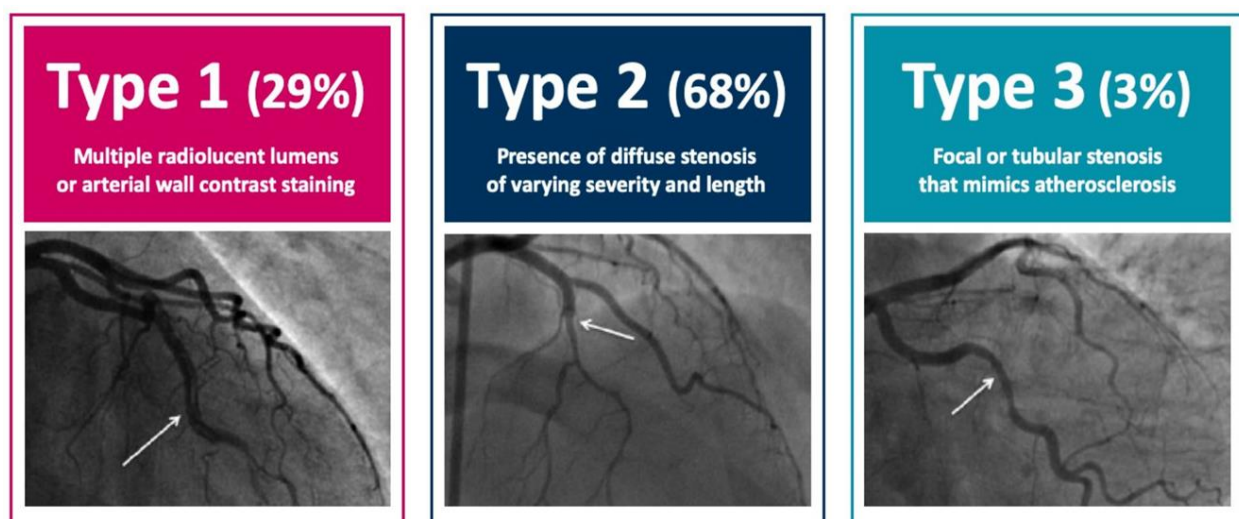
Pregnancy is a special condition that should be emphasized: in the first published series, up to 40% of SCADs occurred during pregnancy or in the immediate postpartum period. However, it now seems that this prevalence was greatly overestimated: In recent series, SCAD directly linked to pregnancy would represent 5% of overall cases [4].

In 2013, the team of Jacqueline Saw at the University of Vancouver in Canada published a work resulting from a multicenter prospective series collecting spontaneous dissection cases; this series gives us valuable information on the contributing and predisposing factors of SCAD: in the 750 cases studied, more than 50% were under psychological stress and 30% performed intense physical exercise such as heavy

load lifting [5]. Although fibromuscular dysplasia was not systematically investigated, 30% were diagnosed with confirmed fibromuscular dysplasia. Furthermore, systemic inflammatory diseases and connective tissue disease were found in respectively 5% and 4% of the study population [5].

Coronary angiography remains the first-line diagnostic tool; Dedicated intracoronary imaging methods, including intravascular ultrasonography and optical coherence tomography, provide detailed visualization of the arterial wall that aids the diagnosis of SCAD. However, these tools have additional risks and costs, and they are not readily available in all catheterization laboratories. Thus, availability, competence, and expertise can vary widely with these technologies. As a consequence, conventional coronary angiography remains instrumental in diagnosing SCAD, and cardiologists should become proficient at recognizing its various angiographic patterns. A normal CT scan should not eliminate a coronary dissection but can be an interesting option for the follow-up of medically treated patients [6].

The most commonly used angiographic classification is that proposed by Jacqueline Saw and her team (Fig 4): Type 1 corresponds to the pathognomonic aspect of intimal rupture; type 2, the most frequent, corresponds to diffuse stenosis with abrupt changes in the arterial caliber; and type 3 which describes focal stenosis mimicking atherosclerosis often requiring the use of intracoronary imaging to confirm the diagnosis. Predominant involvement of the left anterior descending artery and its branches has been reported in most series [7].



**Fig 4: Jacqueline Saw *et al.*, classification of SCAD**

What are the therapeutic options? When should we opt for a conservative approach? What kind of monitoring can we adopt for SCAD patients?

Current recommendations are based on expert consensus from series of observations.

According to the current data, with the exception of high risk profiles, the initial management should be as conservative as possible. Current studies suggest that spontaneous coronary healing has been recorded in 90% of medically treated patients [8].

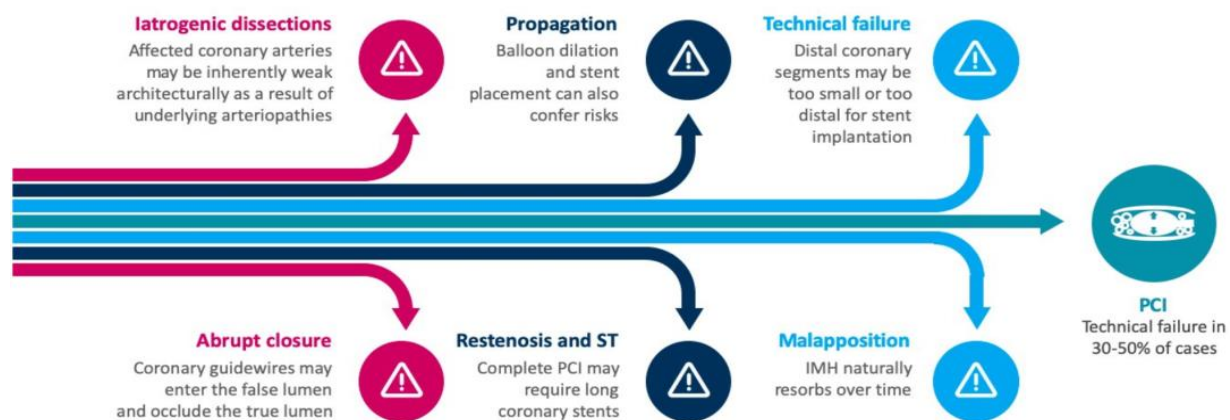
The appropriate timing of the angiographic check-up also remains a point of discord: given that the healing rate is significantly higher in the groups where the check-up was performed after 5 weeks compared to the groups where the check-up coronary angiography were performed in the first 3 weeks; it seems more logical to wait at least 1 month before the angiographic control.

The results of revascularization strategies make the conservative approach more reasonable. Published studies show an increased risk of iatrogenic complications linked to the technical difficulty of performing angioplasty or bypass surgery: In Canadian

series, successful angioplasty was obtained in 64% of patients and only 30% didn't present any clinical complications in long-term follow-up (ACS, Death) [9]. A Mayo Clinic study showed a success rate of angioplasty in only 57% of study group with a relatively high complication rate. In addition, the revascularization strategy has not shown any benefit in terms of recurrence and re-intervention [10].

Angioplasty can be complicated with iatrogenic dissection, extension of the intramural hematoma, passage of the guidewire in the false lumen, or malapposition of the stent after resorption of the hematoma, increasing the risk of restenosis and intrastent thrombosis (Figure 5).

Bypass surgery is also difficult to perform due to the difficulties of anastomosis on fragile and dissected walls.



**Fig 5: Risks and complications of revascularization in SCAD**

Several algorithms have been proposed for the management of SCAD; the most recent guidelines are published by the American College of Cardiology in 2019 and the European Society of Cardiology in 2020 [8, 11].

Both American and European algorithms recommend that the decision to treat medically or with revascularization must be individualized and based on clinical and angiographic criteria.

Thrombolysis can lead to extension of dissection, coronary rupture or even tamponade. Therefore, current data indicate that thrombolysis is contraindicated [1, 12]. The benefit of anticoagulant and antiplatelet therapy is controversial: Dual antiplatelet therapy is not recommended in the absence of revascularization, and Aspirin monotherapy is usually prescribed. The optimal duration remains

unknown, some authors recommend aspirin for life while others question this approach [1].

Anticoagulation has no place in conservative treatment and current data suggest that it should be stopped once the diagnosis of dissection has been made. Its administration should be limited to the procedure of revascularization [1].

There seems to be a consensus on the administration of beta-blockers: their prescription have shown benefits on limitation of initial extension and decreased risk of long-term recurrence [13]. On the other hand, there is no consensus on the benefit of ACE inhibitors or ARBs2 in coronary dissections.

What about statins? Apart from their benefits in atherosclerotic patients, their use did not show any benefit. A retrospective study of 87 patients found a higher recurrence rate in patients who were under statins [14].



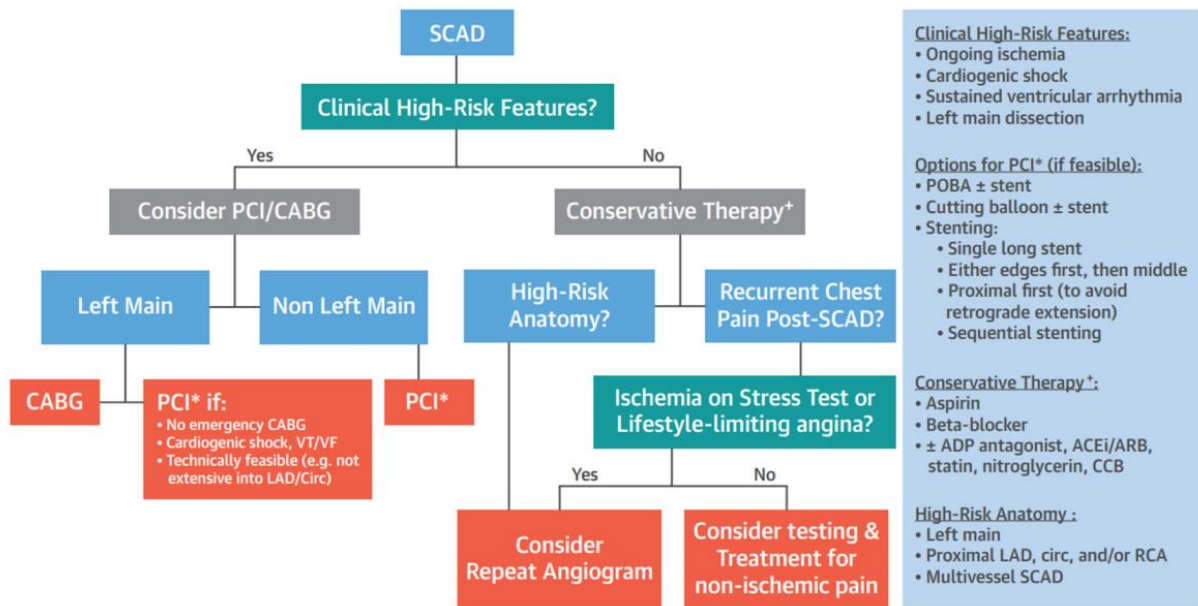


Fig 6: ACC algorithm for management of SCAD

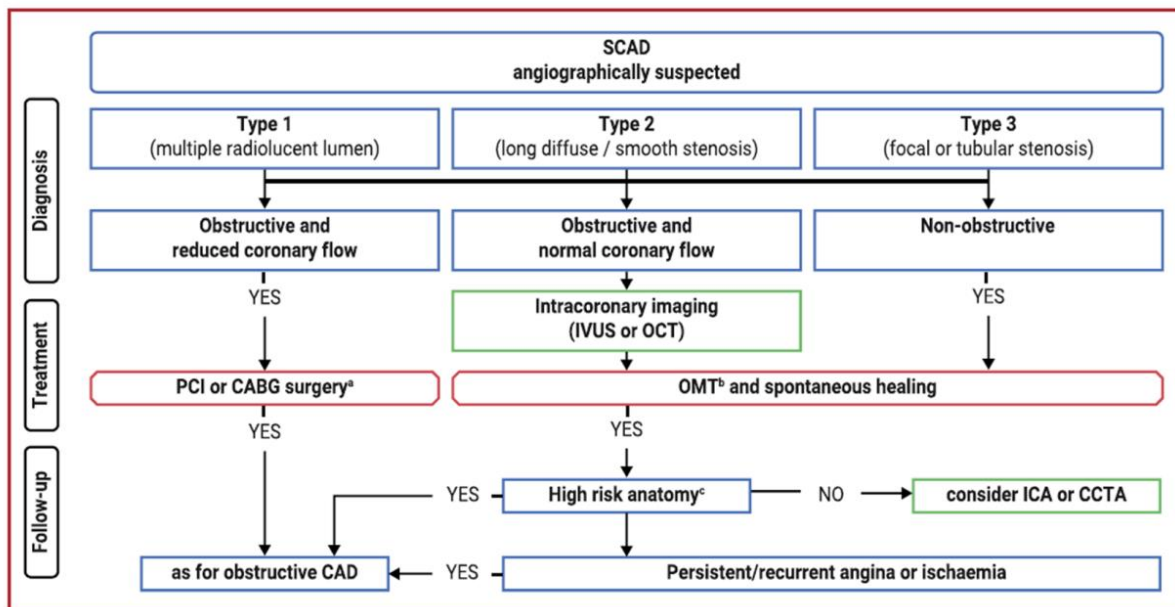


Fig 7: ESC algorithm for management of SCAD

## CONCLUSION

Advances in our understanding of the epidemiology of SCAD, the availability of intravascular imaging techniques, the development of SCAD-specific angiographic classification, heightened awareness among providers suggest that SCAD is far more common than previously thought, especially in young women. In addition, SCAD has unique risk factors and associated conditions and different diagnostic, therapeutic, and prognostic implications compared with atherosclerotic coronary disease.

Our case illustrates the role of conservative treatment in stable patients who do not present criteria of high clinical and angiographic risk.

When should we think about it? According to current data, it is necessary to evoke a coronary dissection in front of an acute coronary syndrome in young women or in period of peripartum, or with inflammatory disease, fibromuscular dysplasia or under physical or emotional stress in the absence of traditional risk factors.

What are the real predisposing factors? In the absence of clinical context, are there minimum investigations to be done? What optimal medical treatment can we prescribe? What is the appropriate control timing? Are there any predictor factors of recurrence? Several questions remain so far without clear answers.

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