World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(RESEARCH ARTICLE)

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Two case reports of spontaneous coronary dissection

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World Journal of Advanced Research and Reviews, 2024, 22(01), 1809–1814

Publication history: Received on 15 March 2024; revised on 21 April 2024; accepted on 24 April 2024

Article DOI: https://doi.org/10.30574/wjarr.2024.22.1.1256

Abstract

Spontaneous coronary artery dissection (SCAD) has nowadays been described as a non-negligible etiology of acute coronary syndromes (ACS). Its prevalence approximates 3% in most cohorts. It mainly affects women in the young to middle-aged range. Individuals with SCAD typically exhibit fewer conventional cardiovascular risk factors associated with ischemic heart disease compared to those with atherosclerotic coronary artery disease. Nevertheless, some SCAD patients may present with risk factors such as hypertension, smoking, and dyslipidemia, although their direct contribution to SCAD risk remains uncertain.

We present two cases of spontaneous coronary dissection to explore its pathogenesis, diagnosis, and treatment options.

Learning objectives

- Recognize spontaneous coronary artery dissection as the possible cause of ACS in females even with cardiovascular risk factors.
- Learn the importance of angiographic control after a conservative therapy that may lead to a more invasive approach.

Keywords: CAD; SCAD; IVUS; ACS; PCI

1. Introduction

Spontaneous coronary artery dissection (SCAD) is characterized by an epicardial coronary artery dissection that occurs independently of atherosclerosis, trauma, or iatrogenic causes.

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 since the advances in coronary angiography and endocoronary imaging technique.

SCAD can manifest in any coronary artery, with the left anterior descending artery (LAD) being the most commonly affected, followed by the right coronary artery (RCA) and its branches. It seldom occurs in the left main coronary artery (LMCA).

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].

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

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

2. Case reports

2.1. Case 1

We report the case of 57 year old female, active chronic tobacco user with history of hypertension, and no previous angina symtoms; presented with sudden-onset chest pain.14 hours after the pain intensified, she was admitted to emergencies. Her ECG showed sinus rhythm with antero-lateral ST depression and negative T waves. The transthoracic echocardiography showed a preserved ejection fraction with no abnormal wall motion and no pericardiac effusion. The troponin levels were significantly high: 240 ng/ml. A coronary angiogram was performed and showed a 90% stenosis of proximal LAD (LAD 1). We performed an intra-coronary ultrasound imagine (IVUS) (Figure 1). Five criteria confirmed the diagnosis of a spontaneous dissection with a TIMI 3 flow

- Absence of atherosclerotic plaques
- Smooth reduction of the luminal diameter suggesting an extrinsic compression by the hematoma
- Visualization of an endoluminal flap indicating an intimal rupture
- Presence of an intramural hematoma in the outer third of the media compressing the true lumen.
- 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, 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. However, repeat coronary angiogram with IVUS at 3 months for recurrent angina showed focal residual 80% proximal-LAD stenosis that was due to the persistence of the hematoma (figure 2).

Therefore, percutaneous coronary intervention (PCI) was performed with drug-eluting stent (4mm*15mm) in the proximal LAD (figure 2). She remained clinically well on medical therapy with DAPT, betablokers and statins.

2.2. Case 2

We report the case of 40-year-old female with history of hypertension, and no previous angina symptoms. She was admitted to emergencies 2 hours after sudden-onset chest pain. Her ECG showed sinus rhythm with septo-apical ST elevation and negative T waves. The transthoracic echocardiography showed a preserved ejection fraction with antero-septal hypokinesia. The troponin levels were significantly high: 76 ng/ml. The coronary angiogram (Figure 3) revealed spontaneous dissection of the proximal left anterior descending artery (LAD), involving a great portion of the artery.

Otherwise, the patient had angiographically normal coronary arteries. The patient received conservative therapy with no stenting. She was discharged home and was prescribed bisoprolol 5 mg and aspirin 75 mg daily. At the 1-week and 1-month follow-up, she reported no symptoms. We performed an angiographic control after 1 month that showed a complete spontaneous healing of the proximal LAD (figure 3).

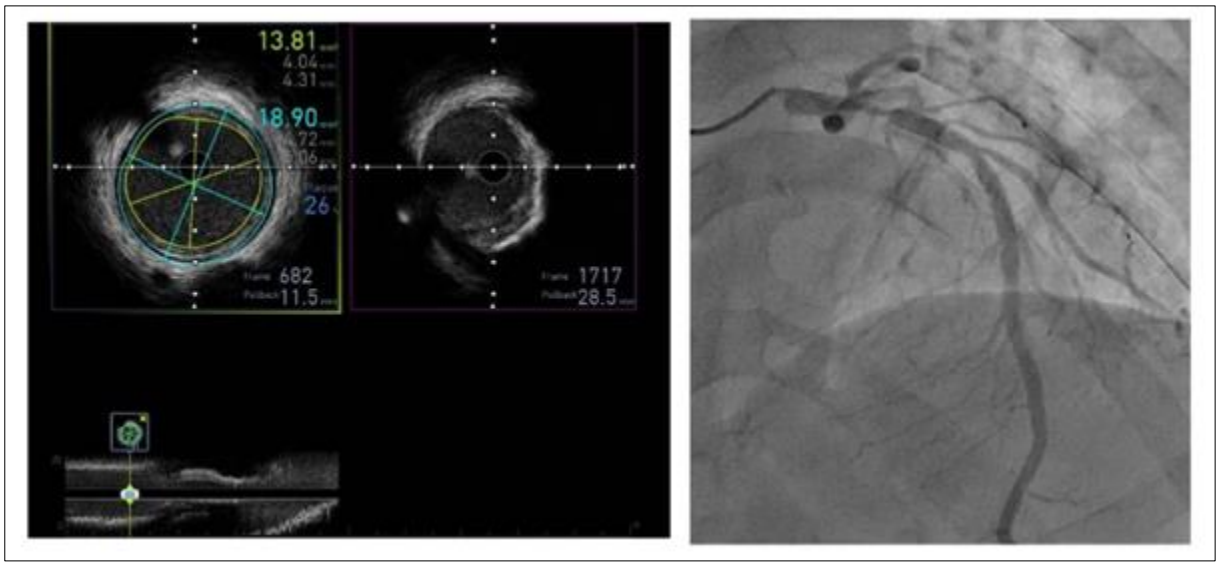

Figure 1 Coronary angiogram and IVUS showing 90% stenosis of proximal LAD with an intramural hematoma in the outer third of the media compressing the true lumen spanning from 1 to 8 o'clock

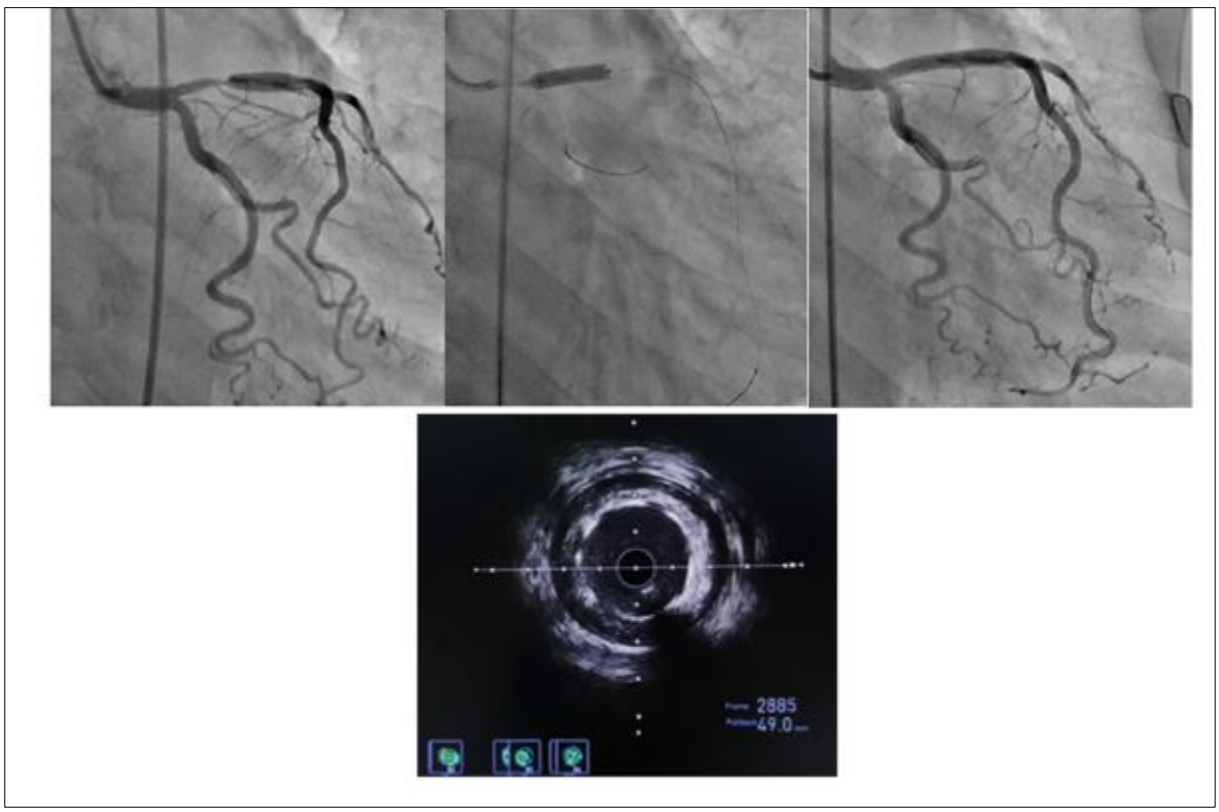

Figure 2 Coronary angiogram showing the persistance of proximal LAD stenosis after 3 months treated with direct stenting of proximal LAD with drug eluting stent ULTIMASTER Tensei 4*15 mm, IVUS control after stent implantation showing good struts apposition

Figure 3 Coronary angiogram showing a type 2 SCAD of the proximal LAD, follow-up coronary angiogram showing spontaneous healing of the proximal LAD

3. Discussion

The primary mechanism of myocardial injury in SCAD is attributed to coronary artery obstruction, typically arising from the formation of an intramural hematoma (IMH) or intimal disruption, rather than conventional atherosclerotic plaque rupture or intraluminal thrombus. SCAD predominantly affects individuals with minimal or no traditional cardiovascular risk factors. Recent studies employing stringent diagnostic criteria, which exclude iatrogenic, traumatic, and atherosclerotic dissections, indicate that SCAD may account for approximately 1% to 4% of cases of acute coronary syndrome (ACS) overall [1-2].

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

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 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 continues to serve as the primary diagnostic modality for SCAD. Specialized intracoronary imaging techniques such as intravascular ultrasonography and optical coherence tomography offer precise visualization of the arterial wall, thereby confirming the diagnosis of SCAD. However, the use of these tools entails additional risks and costs, and their availability is limited in some medical centers. CT scan can be an interesting option for the follow-up of patients treated medically [6].

The most commonly used angiographic classification is that proposed by Jacqueline Saw and her team: 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].

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 unclear: 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.

Bypass surgery is also difficult to perform due to the difficulties of anastomosis on fragile and dissected walls. | 10].

Thrombolysis can lead to extension of dissection, coronary rupture or even tamponade. Therefore, current data indicate that thrombolysis is contraindicated [1]. Surgical revascularization is best reserved for the left main, multi-vessel disease, or refractory ischemia despite aggressive medial therapy. In the case of a well-localized symptomatic single coronary dissection not involving the left main, percutaneous coronary intervention with stenting is possible [8]. 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].

4. Conclusion

The accessibility of intravascular imaging techniques and advancements in SCAD-specific angiographic classification suggest that SCAD may be more prevalent than previously understood, particularly among young women.

Although a conservative approach is mostly recommended, some patients with high risk angiographic features, may require an interventional or surgical revascularization.

PCI should be very cautious, by trying to cover the whole dissection without extending it. A good evaluation of the true diameter and the length of the dissection with intra-vascular imaging is highly recommended.

Compliance with ethical standards

Disclosure of conflict of interest

The authors have no conflicts of interest to declare.

Statement of ethical approval

All subjects gave their informed consent for inclusion in these case reports.

Statement of informed consent

The authors confirm that written informed consent has been obtained from the involved patients.

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