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Spontaneous Coronary Dissection: Practical Management about 3 Cases

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Abstract | **Case Series**

Spontaneous coronary artery dissection (SCAD) is an acute coronary event related to the development of a hematoma in the media, leading to separation of the intima or intima-media complex from the underlying vessel, thus compressing the true lumen, causing ischemia and myocardial infarction. It usually occurs in middle-aged women with few or no cardiovascular risk factors. Coronary angiography can make the diagnosis in most cases, and endocoronary imaging is reserved for ambiguous cases. There is currently no randomized controlled trial comparing the different management strategies for spontaneous coronary dissections (conservative approach, coronary revascularization strategies including percutaneous coronary intervention and coronary artery bypass grafting). Current practice is therefore based on case observations and registries on clinical experience and on extrapolation (when applicable) of guidelines for the treatment of acute coronary syndrome.

Keywords: Spontaneous Coronary Dissection, intima-media complex, myocardial infarction.

INTRODUCTION

Spontaneous coronary dissection, considered as rare from a long time ago, is currently recognized as one of the main causes of non-atheromatous acute coronary syndrome (ACS) and non-traumatic in young patients, with no or few cardiovascular risk factors.

This is related to a more frequent use of coronary angiography and endocoronary imaging techniques. We present through three observations the clinical particularities of this entity and the methods of its management.

First Case

This is a 35 years old woman, multiparous (5 living and healthy children) on oral contraception, without cardiovascular risk factors.

Received in the cathlab for the management of an acute coronary syndrome with anterior ST segment elevation, two hours from the onset of pain.

The coronary angiogram showed an appearance of a spontaneous coronary dissection type I in the ostio-proximal portion of the left anterior descending artery (LAD): significant ostio-proximal lesion with an intimal flap and TIMI Flow 2. Furthermore the rest of the coronary arteries were angiographically free from significant lesions (Figure 1).

This pathognomonic aspect of a spontaneous coronary dissection didn't need an endocoronary imaging to confirm the diagnosis.

The decision to undergo angioplasty of the LAD was taken due to the persistence of symptoms and the extent of ischemia, we opted for a femoral access, EBU 3.5 guiding catheter and BMW guide wire.

When crossing the lesion, the appearance of the guide wire mimed a crossing throught the false lumen, (Figure 2) the patient's condition deteriorates with an exacerbation of pain and increased ST elevation on the ECG, we quickly took a second wire BMW while leaving the first on site (buddy wire technique). Which hopefully passed through the true lumen. A drug eluting stent (DES) has been placed at the ostio proximal portion of the LAD, with a propagation of the hematoma at the middle LAD in the angiographic control, thus requiring the placement of a second DES, with a satisfying final result (Figure 3).

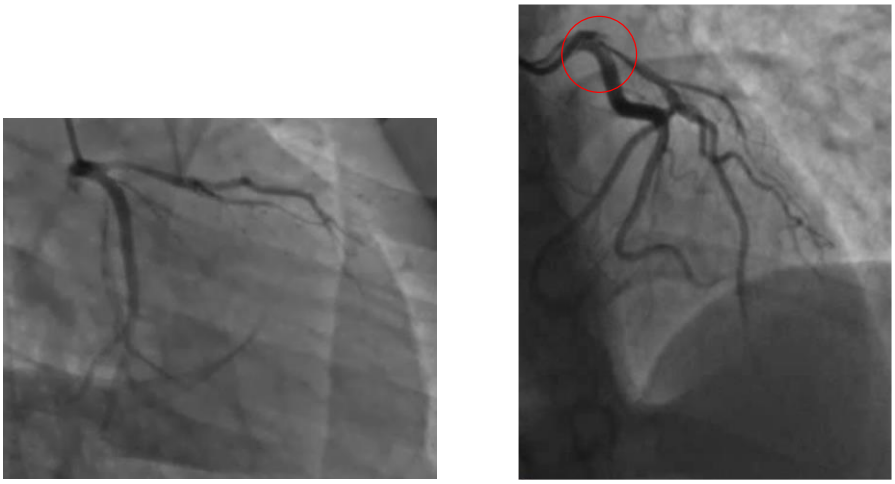

Figure 1: appearance of a spontaneous coronary dissection type I in the ostio-proximal portion of the left anterior descending artery , with an intimal flap and TIMI 2 Flow

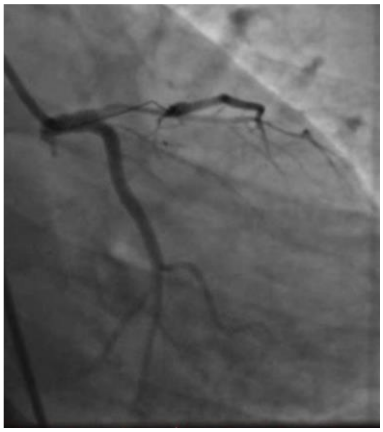

Figure 2: the guidewire shape was an indicator of a false lumen passage

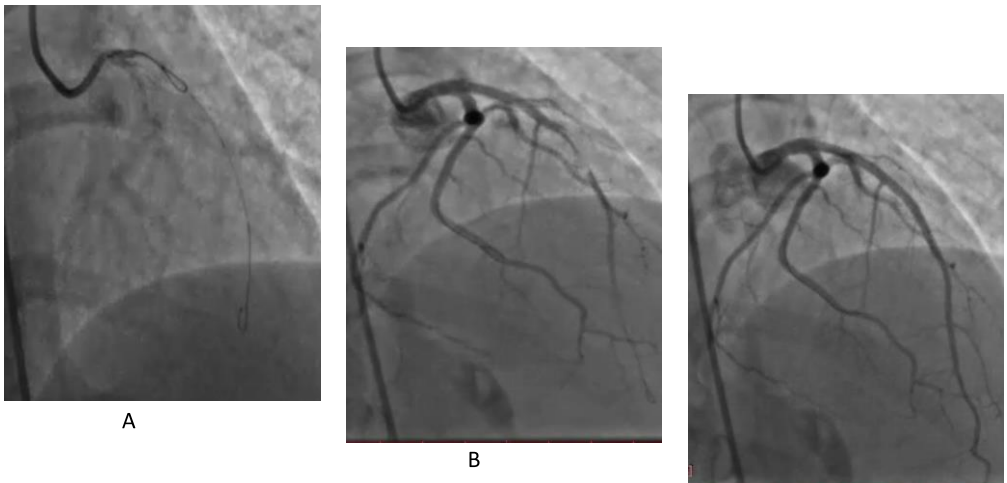

Figure 3:
A: positioning of the second guidwire in the true lumen
B: progression of the hematoma at the middle LAD,after implantation of the stent
C: Implantation of the second stent at the middle of the LAD

Second Case

This is a 36 years old young woman, without cardiovascular risk factors, admitted for the management of an acute coronary syndrome without ST segment elevation.

A coronary angiogram performed within 24 hours, revealed a suggestive appearance of a type II coronary dissection: homogeneous and long narrowing of the LAD and begins and ends at the birth of a collateral, on a smooth coronary network (Figure 4).

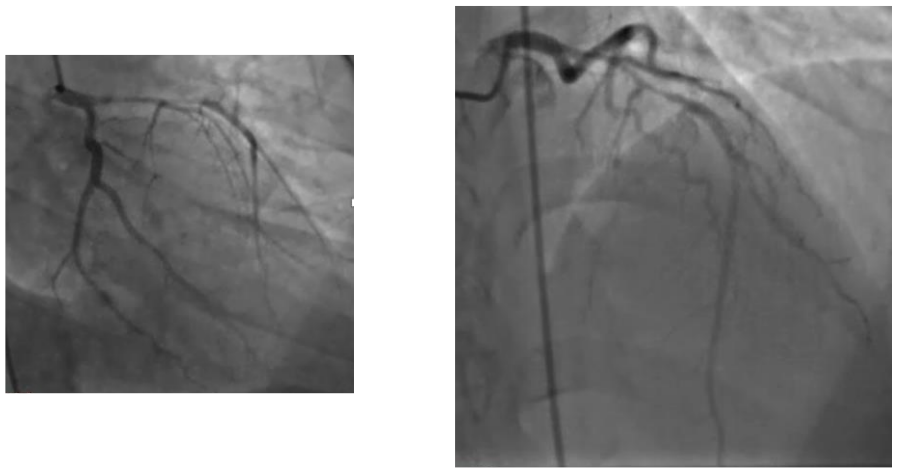

Figure 4: appearance of a type II coronary dissection of the LAD

Given the absence of anginal recurrence and a TIMI flow 3, we decided to adopt a conservative strategy with treatment with mono anti-platelet aggregation (aspirin 100 mg/day) and beta-blocker. The anticoagulant treatment was stopped. The clinical evolution was simple with an angiographic control done 30 days later showing the reestablishment of a normal LAD architecture (Figure 5).

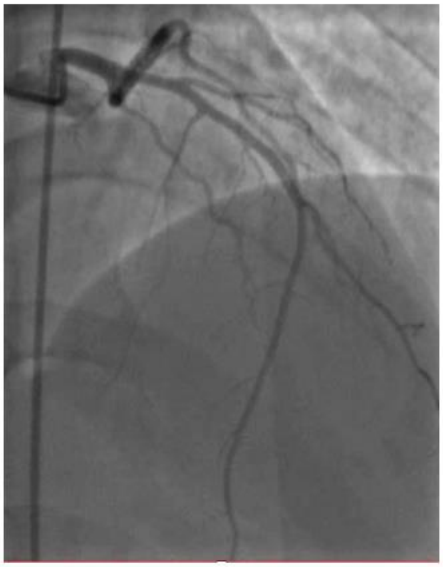

Figure 5: reestablishment of a normal LAD architecture

Third Case

This is a 30 years old young man, without cardiovascular risk factors, received in the cathelab for an acute coronary syndrome with a posterior ST segment elevation at 7 hours from the onset of pain, which occurred during intense physical effort. Primary angioplasty performed on admission visualized a short ostioproximal lesion of the circumflex with TIMI flow 3, on a coronary angiogram free of atheromatous lesion, suggestive of a type 3 spontaneous coronary dissection (Figure 6).

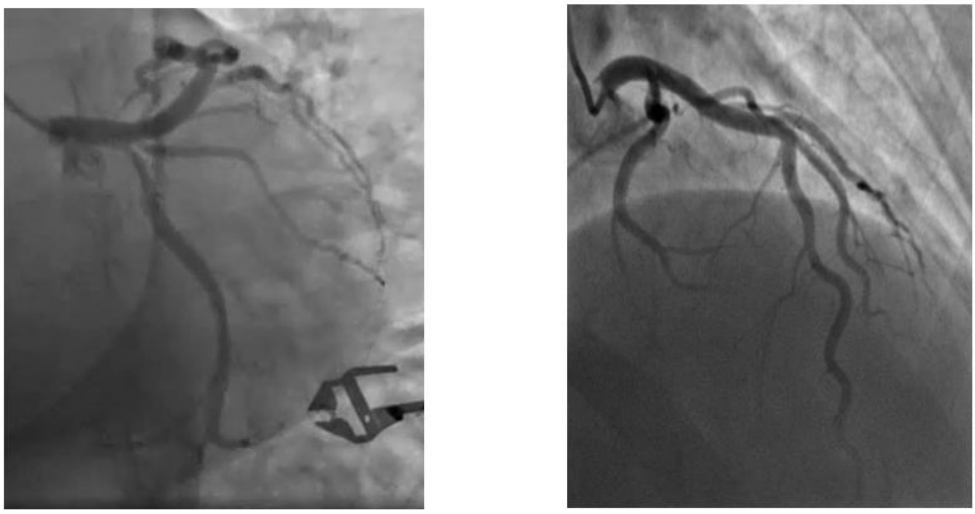

Figure 6 : short ostioproximal lesion of the circumflex with TIMI flow 3, on a coronary angiogram free of atheromatous lesion, suggestive of a type 3 spontaneous coronary dissection

But faced with doubtful diagnosis with an atheromatous lesion, we used IVUS (*Intravascular UltraSound*) which confirmed the diagnosis with the visualization of the intraparietal hematoma (homogeneous hyposignal with blunt edges, splitting the media) (Figure 7).

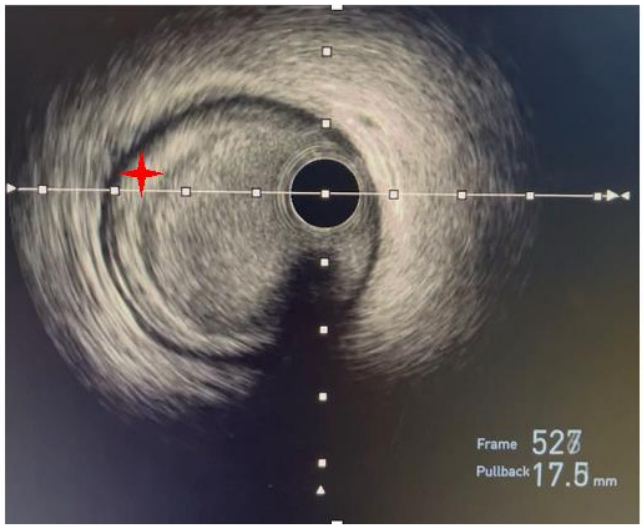

Figure 7:intraparietal hematoma :homogeneous hyposignal with blunt edges, splitting the media.

Given the stable hemodynamic state of the patient, the TIMI flow 3, a conservative strategy was favored based on a mono anti-platelet aggregation (aspirin 100 mg/day) and beta-blocker.

DISCUSSION

Spontaneous coronary artery dissection is a rare cause of acute coronary syndromes and sudden death, with a prevalence that varies according to studies between 1 and 4% [1, 2].

This acute coronary event related to the development of hematoma in the media, leading to separation of the intima or intima-media complex from the underlying wall and compressing the true lumen, causing ischemia and myocardial infarction. Two hypotheses have been proposed to explain the pathophysiological process: the "inside-out" hypothesis suggests that blood enters the subintimal space from the true lumen after the development of an endothelial-intimal rupture or a “flap”; and in the "outside-in" hypothesis, the hematoma appears de novo in the media, following a rupture of the microvessels of the vasa vasorum [3, 4].

Women account for 87% to 95% of cases of spontaneous coronary artery dissection with a mean age of presentation between 44 and 53 years [5, 6]. Although patients with spontaneous coronary artery dissection have lower rates of traditional cardiovascular risk factors such as hypertension, dyslipidemia and smoking than patients with atherosclerotic myocardial infarction [5-8], the prevalence of hypertension ((32% to 37%)) and dyslipidemia ((20% to 35%)) in patients with spontaneous coronary dissection [5, 6, 9] is similar to those of the general population depending on age and sex [10].

The pathophysiology of spontaneous coronary dissection remains unknown. It is likely that a combination of predisposing factors increases susceptibility such that a relatively minor triggering event is sufficient to precipitate coronary dissection; the main predisposing factors found in the series are fibromuscular dysplasia, pregnancy and female sex [11]. Among patients with spontaneous coronary dissection who reported precipitating factors, emotional stressors appeared to be more common in women, while physical stressors were more often reported in men.

Coronary imaging helps to establish the diagnosis. Coronary angiography may be sufficient to make the diagnosis in typical cases like the one reported in our first observation.

Several classifications have been proposed to facilitate the angiographic diagnosis of spontaneous coronary dissections, the most used is the Canadian classification of JAW [14], the Type 1 angiographic SCAD appears as the classic contrast dye staining of arterial wall with multiple radiolucent lumen, with or without the presence of dye hang-up or slow contrast clearing from the lumen. Type 2 angiographic SCAD appears as a diffuse (typically 20 to 30 mm) and smooth narrowing that can vary in severity. Type 3 angiographic SCAD mimics atherosclerosis with focal or tubular stenosis that typically requires optical coherence tomography or intravascular ultrasound to prove the presence of intramural hematoma or double lumen (Figure 8) [14].

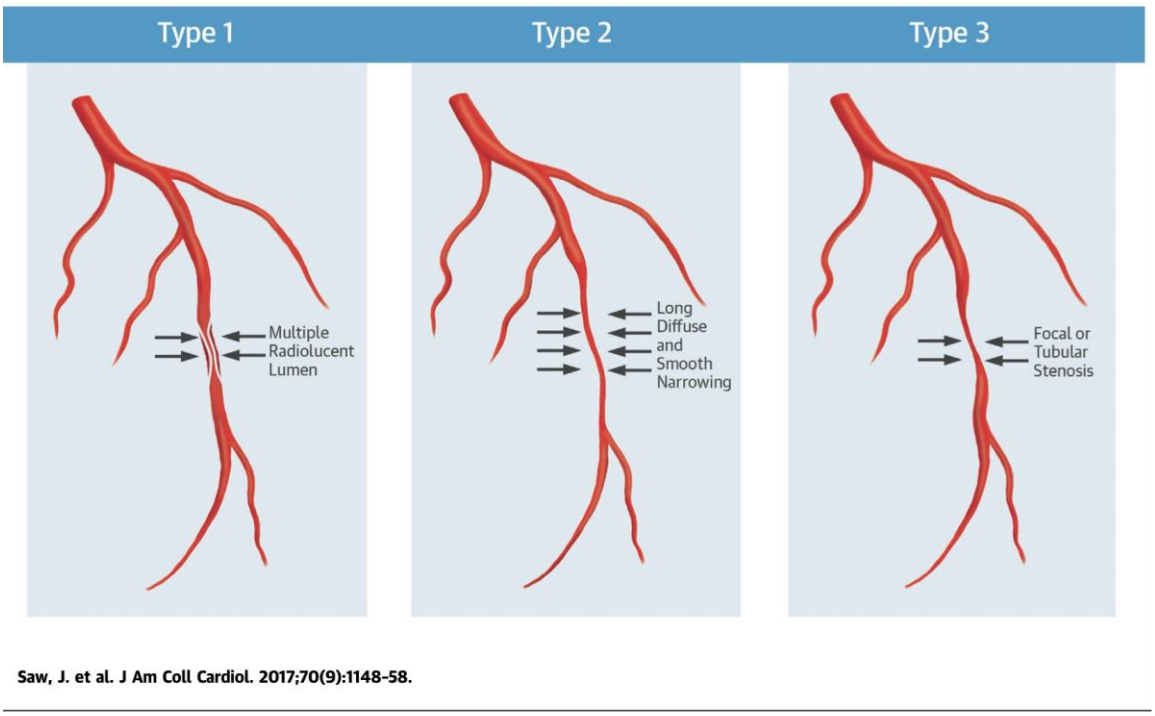

Figure 8: the Canadian classification of spontaneous coronary dissections

Until this day there is only a little scientific data of great force on the support of SCAD. Current European and American recommendations are based on expert consensus from observation series [15, 16].

In the absence of hemodynamic instability, ongoing ischemia or anatomy at risk, the best attitude is a conservative treatment.

Antiplatelet agents reduce the thrombotic risk linked to intimal rupture, and are widely recommended on expert advice [15, 16], In addition to their anti-ischemic and antiarrhythmic effect and by analogy with the aortic dissection, beta-blockers can theoretically reduce the extension of the dissection by reduction of the *shear stress*, and are the only treatment that has reduced recurrences. In a series of 327 patients recently reported by Vancouver, with a median follow-up duration of 3.1 years, the use of β-blockers was associated with a hazard ratio of 0.36 for SCAD recurrence in the multivariable analysis [17], which reinforces the practice of administering β-blockers after spontaneous coronary dissection. Anticoagulants are not recommended in order to avoid increasing the intramural hematoma.

Statin treatment is not routinely recommended after spontaneous coronary dissection. In a first retrospective cohort of 87 patients, statins were associated with recurrence of coronary dissection; however, this result may have been influenced by sample size, date of index event, and medication use started after discharge [18]. In a more recent cohort of 327 patients with SCAD, no association was found between statin use and SCAD recurrence [17].

The results of different revascularization techniques are less convincing than those obtained in atheromatous coronary artery disease.

The procedural success rate of angioplasty in this situation is only around 70% [19]. The difficulties are linked to the risk of the coronary guide wire crossing through the false channel, the frequent need for long stenting, the risk of propagation of the hematoma during deployment of the stent and the possibility of late malapposition after resorption of the intramural hematoma and hence an excess risk of stent thrombosis [20].

The results of coronary artery bypass grafting are disappointing with a graft occlusion rate estimated at 73% in a small series of 12 operated patients [21].

For these reasons, revascularization of spontaneous coronary dissections is reserved in cases of hemodynamic instability or ongoing ischemia.

In patients who survived to a spontaneous coronary dissection, long-term mortality is low. In the American Mayo Clinic series, 10-year survival according to Kaplan-Meier estimates is 92% [18]. Similarly, an Italian series reported 94.4% survival at 6years [19]. However, these figures mask a significant morbidity [22]. The overall rate of major adverse cardiac events (MACE) in patients with SCAD is significant but varies considerably between published series (47.4% MACE over 10 years based on Kaplan-Meier estimates in the American series [18]; the MACE rate in the Canadian prospective series was 19.9% during a median follow-up of 3.1 years [22], the MACE at 5 years in the Japanese series was 37%; 8 in the Italian series the MACE at 6 years was 14.6%). This is mainly due to recurrent dissections and a high rate of target vessel failure in patients undergoing PCI [19].

CONCLUSION

Spontaneous coronary dissection is a probably an underestimated cause of acute coronary syndromes in young subjects; the use of coronary angiogram and intracoronary imaging has allowed a better sensitivity of the diagnosis.

Angiographic diagnosis can be easy in certain situations, notably type I, but for type II and especially type III, intracoronary imaging finds its place in distinguishing it from an atheromatous lesion.

Management is not codified and it is based on expert opinions, but a conservative strategy is widely recommended in the absence of ischemic recurrence or alteration of coronary flow.

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