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Spontaneous Coronary Artery Dissection: A Case Series of 8 Patients with Literature Review

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Abstract **Original Research Article**

Introduction*:** Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndromes, affecting a mainly female population with few cardiovascular risk factors, with an incidence of 0.07–0.2% of all angiograms and 2–4% of angiograms performed for acute coronary syndrome (ACS) .It is defined by a spontaneous, non-traumatic and non-iatrogenic separation of the coronary wall, in the absence of an associated atheromatous lesion. The pathophysiology is heterogeneous, including hormonal influences, arteriopathies, and precipitating intense physical or emotional stressors. *Methods and results*: Between 2018 and 2020, 8 cases of SCAD were collected from a database our cardiology unit. The mean age was 46 years with a female exclusive and 47% of patients were under 50 years old. Three patients (37.5%) had no cardiovascular risk factor (CVRF). One patient had SCAD in a peripartum setting with a personal and a family history of fibromuscular dysplasia. All our patients were admitted for ACS, the half of them presented a ST-segment elevation myocardial infarction (STEMI), and the other half presented non-STEMI. In all de cases the diagnosis was confirmed by angiography alone. All patients were managed with conservative approach in the early and late phases. The anterior interventricular artery (AIV) was most frequently involved in 62.5% of cases. No endocoronary imaging was required; 37.5% were classified as Type 1, 37.5% as Type 2 and 50% as type 4. No lesions were classified as Type 3. The cardiac computed tomography angiography (CCTA) control was performed in 62.5% between 1 and 6 months. The extracoronary workup was performed in 37.5% of the cases and revealed lesions in 66% of the cases. ***Conclusion: The data from this study are consistent with the literature, and involvement of the left coronary artery and left main coronary artery were highly prevalent. Clinicians must be aware of angiographic appearances of SCAD for prompt diagnosis and management in these patients.

Keywords: Spontaneous coronary artery dissection, acute coronary syndrome, myocardial infarction, fibromuscular dysplasia, diagnosis, management.

INTRODUCTION

Spontaneous Coronary Artery Dissection (SCAD) is an underdiagnosed form of acute coronary syndrome (ACS) affecting a mainly female population with few cardiovascular risk factors [1, 2].

Until the early 2000s, SCAD was reported only through isolated cases or short series [3]. It was described as a rare pathology and a fatal cause of ACS and was wrongly considered to affect mainly young women in the peripartum period and to be responsible for sudden cardiac death in them. The therapeutic management was empirical with compassionate recourse to bypass surgery for the most severe forms.

Since the first description of SCAD by Pretty in 1931 at autopsy, our understanding has progressed over the last 8 decades, especially over the last 5 years.

Currently, this pathology is classified as MINOCA (Myocardial Infarction with Nonobstructive Coronary Arteries) and, according to the DISCO study, represents 36% of ACS in women under 60 years of age without or with a cardiovascular risk factor[4] This is due to the almost systematic use of coronary angiography in the case of chest pain with troponin elevation and to the use of endocoronary imaging in case of diagnostic ambiguity.

It is defined by a spontaneous, non-traumatic and non-iatrogenic separation of the coronary wall, classically in the absence of an associated atheromatous lesion. It can present in two forms: with or without intimal rupture (the most frequent form) (Figure 1) [5].

Intimal rupture, call the 'inside-out' model (Figure 1B), where the causal event is the development

of an endothelial and intimal discontinuity or 'tear', allowing blood to cross the internal elastic lamina and accumulate in the media easily accessible to angiographic diagnostic

Without intimal rupture: the 'outside-in' mechanism (Figure 1C) where the causal event is the primary disruption of a vasa vasorum micro-vessel leading to hemorrhage directly into the tunica media. Conversely this mechanism represents a challenge diagnostic.

In both cases, the presence of true luminal compression by the hematoma can lead to myocardial ischemia and infarction.

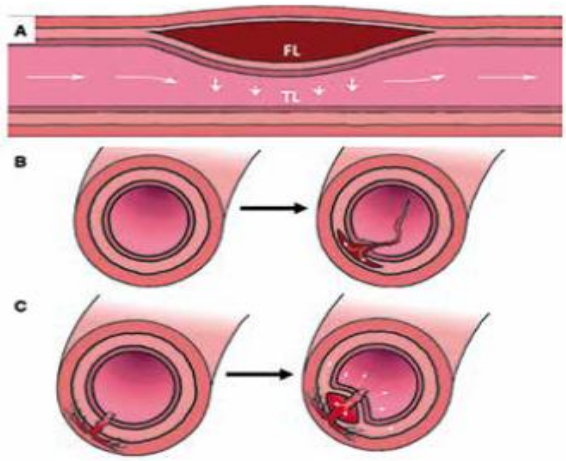

Figure 1: Schematic illustration of spontaneous coronary artery dissection. Accumulation and axial propagation of blood forms a false lumen in the outer third of the tunica media leading to external compression of the true lumen (A). Blood may enter through an endothelial-intimal disruption or 'tear' (B) or as a result of bleeding from a microvessel within the vessel wall (C) leading to an expanding and compressing false lumen (dotted arrows).

The pathophysiology of SCAD remains poorly understood. Constitutional or acquired vascular wall fragility associated with mechanical stress has been suggested to explain the occurrence of such an abnormality 2. 2 Hormonal influence has been suggested in view of a high female predominance (approximately 90% of women) and an average age at diagnosis ranging from 44 to 53 years [2, 4, 6-10].

The association with peripartum would represent only a minority of cases, accounting for approximately 5 to 10% of all SCAD [9-11]. Other contributing factors have been described, such as physical exercise or intense emotional stress [7, 11] the use of drugs (cocaine), and coronary spasm could be responsible for an increase in stress and/or parietal tension. Finally, Valsalva maneuvers with significant increases in intrathoracic pressure are also sometimes the cause of SCAD.

Among the pathologies favoring the occurrence of SCAD, connective tissue diseases such as Marfan's syndrome or Ehlers-Danlos disease have been exceptionally reported12,13 ; on the other hand, the recent work of Saw et al. highlights a strong association with fibromuscular dysplasia (FMD), between 50 and 86% [6, 12, 13] depending on the registers.

This article describes 8 patients with SCAD. It further highlights that physicians should be particularly vigilant for atypical presentations of this disease. It should be included among differentials, especially in young females presenting with ACS, in the absence of notable risk factors for cardiovascular illness.

METHODS

The cases presented here were retrospectively collected from a database of 1 secondary hospitals in which patients developed SCAD in the context of ASC, between 2018 and 2020. Angiographic diagnosis of SCAD was established by agreement of two experienced interventional cardiologists.

Inclusion Criteria

All patients older than 18 years with a diagnosis of SCAD in the setting of ACS were included in the study. The diagnosis of SCAD was based on the occurrence of an ACS defined according to the usual recommendations, with the presence of angiographic signs suggestive of SCAD on the initial coronary angiography and categorized according to the classification of SAW et al , possibly assisted by intracoronary imaging (OCT). The coronary angiography was performed urgently in case of ST-segment elevation ACS, or within a time defined by the risk stratification in case of non-ST-segment elevation ACS.

Exclusion criteria for the study included

Age less than 18 years and patients with coronary dissection of iatrogenic (by coronary catheterization), traumatic, or atherosclerotic (atherosclerotic plaque dissection) origin.

RESULTS

Table 1 summarizes the patient demographic and procedural characteristics. The mean age of patients was 46 years, with a female exclusive. 47% of patients were under 50 years old. 3 patients (37.5%) had no cardiovascular risk factor (CVRF) and 37.5% had less than 2 CVRF.

All our patients were admitted for ACS with elevated troponins, the half of them presented a ST-segment elevation myocardial infarction (STEMI), and the other half presented non-STEMI. Left ventricle ejection fraction (LVEF) was preserved in all patients. Intense emotional stress before the SCAD episode was reported in one patient and heavy exercise in also one

case. It should be noted that in our series we did not find any consumption of toxic substances: cannabis, cocaine, etc. However one patient had SCAD in a peripartum setting with a personal and a family history of fibromuscular dysplasia and SCAD. Her mother had a post-partum type A aortic dissection treated by mechanical Bentall on fibromuscular dysplasia.

None of the patients had associated connective tissue disease or inflammatory disease. And two patients presented with a recurrence of SCAD.

Diagnosis was possible by coronary angiography alone for all cases. There were 62.5% of SCADs involved a single coronary vessel, 37.5% of SCADs were multifocal (involving >1 vessel). The anterior interventricular artery (AIV) was most frequently involved in 62.5% of cases.

No endocoronary imaging was required; 37.5% were classified as Type 1 and 37.5% as Type 2.and 50% as type 4. No lesions were classified as Type 3. All patients were managed with conservative approach in the early and late phases.

None of the patient underwent percutaneous or surgical revascularization. The acute occlusions occurred in the distal coronary arteries and were managed conservatively. The anticoagulation used was per procedural only and stopped after that. At discharge from the initial hospitalization, 100% of patients were receiving antiplatelet therapy with aspirin and 12.5% with aspirin and clopidogrel. All patients were prescribed beta-blockers and half of the cases received statins and renin angiotensin system blockers. In our series, one patient presented a ventricular hyper-excitability that resolved after beta-blocker treatment. The cardiac computed tomography angiography (CCTA) control was performed in 62.5% between 1 and 6 months after the acute event, the rest of the patients did not come to their appointment and all the CCTA revealed a healing of the coronary dissection lesions seen previously by coronary angiography.

The extracoronary workup was performed in 37.5% of the cases and revealed lesions in 66% of the cases, including a dissection of the internal carotid artery in one patient and a dissection of the sinus of Valsalva in another patient. In 2/3 of the cases the screening of these lesions was performed by Doppler ultrasound of the supra aortic trunks, abdominal and lower limbs, and in 1/3 of the cases by computed tomography angiography of the abdominal and lower limb

Table 1: Demographic and procedural characteristics of patients with SCAD

Patients	Age	Risk factor	Precipitating factor	Clinical presentation	Vessel	Type of lesions	Treatment	Aspirin	Clopidogrel	statins	Betablockers	Screening ECL
1	26	none	delivery	STEMI	LAD, PI, LRV	Type 4	Medical	yes	No	No	Yes	Yes
2	36	Smoker, DLP	none	STEMI	LM, LAD lCX	Type 4 and 1	Medical	Yes	No	Yes	Yes	Yes
3	47	HT, DM obesity, smoker	Emotional stress	NSTEMI	LAD	Type 2	Medical	Yes	No	Yes	Yes	No
4	52	Obesity	Intense exercise	STEMI	Mg	Type 4	Medical	Yes	No	No	Yes	No
5	48	None	none	NSTEMI	LAD	Type 4	Medical	Yes	No	No	Yes	No
6	40	Over weight	none	NSTEMI	IP	Type 2	Medical	Yes	No	No	Yes	No
7	49	DLP	none	STEMI	LAD, lCX	Type 1 and 4	Medical	Yes	No	Yes	Yes	Yes
8	74	HT, obesity	none	NSTEMI	LAD	Type 1	Medical	Yes	yes	Yes	Yes	No

DM: diabetes millitus; DLP, dyslipidaemia; STEMI: ST-elevation myocardial infarction; NSTEMI: non-ST elevation myocardial infarction, LM,: l eft main coronary artery; LAD: left anterior descending artery; Cx: circumflex coronary artery; RCA: right coronary artery; Mg: marginal; IP: posterior interventricular, LRV: left retro ventricular, ECL extra coronary lesion.

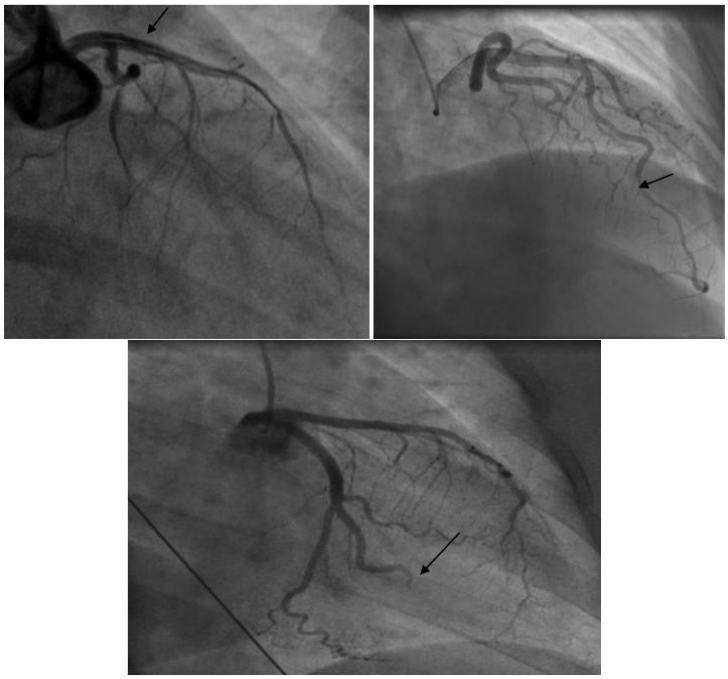

Figure 2: A: Right anterior oblique caudal image of left main a d proximal left anterior descending artery showing SCAD type 1 (patient 2). B: Right anterior oblique cranial image of Left anterior descending artery showing SCAD type 2 (patient 3) Right anterior oblique (RAO) caudal image of left coronary angiography showing SCAD type 4 of de second marginal artery (arrow) (Patient 4)

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.

The true prevalence of SCAD remains uncertain, primarily because it is an underdiagnosed condition. Historically, SCAD was considered very rare but contemporary angiographic series report SCAD diagnosis rates of 0.07–0.2% of all angiograms and 2–4% of angiograms performed for ACS [14-16]. 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.

Previously considered primarily a disease of young adults, SCAD has now been described in patients aged 18–84 years [17, 18] with the mean age in large contemporary series ranging from 44 to 53 years, with a female predominance (90%). In this study, the mean age for female patients was 46 years with women's exclusive. The predilection of SCAD for female patients and the association with pregnancy suggest a pathophysiological role for female sex hormones.

The relationship between SCAD and gestational and puerperal period varies among populations, ranging from 0 to 18% among all women with SCAD [16, 19]. In the largest and most recent cohort of patients thus far, among 168 patients with SCAD, 92.3% were female, and 2.4% occurred during the gestational period [17]. It is estimated that 1 in 16 000 pregnancies is complicated by acute myocardial infarctions, and up to 40% of such events can be attributed to pregnancy-associated SCAD (P-SCAD) [20, 21] occurring pre- dominantly in late pregnancy or the early postpartum period [22].

The risk factors for SCAD have been recently studied in detail. It is associated with atherosclerotic and several nonatherosclerotic risk factors, such as connective tissue disorders, including FMD, postpartum status, breastfeeding, coronary artery spasm, hormonal therapy, intense emotional stress, an intense exercise, inflammatory conditions like lupus erythematosus, inflammatory bowel disease, polyarteritis nodosa, sarcoidosis, celiac disease.

However, many patients have risk factors for ischemic heart disease, including hypertension, smoking, and dyslipidemia, although there is no evidence that these factors contribute directly to the risk of SCAD.

For example, the mean blood LDL concentration in the Rogowski et al series [23] was 3.3 mmol/L, and some cases of SCAD have been reported

in patients with severe hypertension. In our study 63.5% of patient had at least one CVR factor.

Fibromuscular dysplasia is the most frequent disease associated with various predisposing arteriopathies. Since the first case series reported in 2012 associating SCAD with extracoronary FMD involvement, FMD has been found in 11-86% of SCAD cases.

The study by Saw *et al*., reported a lower rate of 31% FMD increasing to 56.7% in patients with "full screening." In our study, one patient has FMD.

Coronary angiography is the "first line" examination for suspected ACS and remains the gold standard for the diagnosis of SCAD. The most commonly used angiographic classification of SCAD has been adapted from Saw *et al.,* [24, 25] From the Saw classification Type 1 represents the classical angiographic radiolucent 'flap' and linear double lumen often associated with contrast hold-. This reportedly occurs in 29– 48% of cases [8, 10, 23].

Type 2 pattern (52–67%) 4,7,8 characterized by a long diffuse and smooth stenosis predominantly located in mid-to-distal segments. Type 3 lesions are defined as angiographically indistinguishable from a focal atherosclerotic stenosis requiring diagnostic confirmation by intracoronary imaging. However, these account for a small minority of cases (0–3.9%) [8, 10, 23].

Type 4 SCAD is described as a total occlusion, usually of a distal vessel. In this uncommon circumstance, the diagnosis is particularly challenging and frequently can only be established during an ensuing coronary intervention once coronary flow is re-established or inferred by subsequent vessel healing and the exclusion of an embolic cause.

Intracoronary imaging like Optical Coherence Tomography (OCT) or Intravascular UltraSound (IVUS), has become instrumental to aid the diagnosis of SCAD in angiographically subtle or nondiagnostic cases. Fortunately, the need to use intracoronary imaging has diminished in recent years because of improved SCAD-pattern recognition on coronary angiography.

OCT allows to visualize an intimal entry portal, the intimal rupture, to measure the longitudinal extension of the hematoma and the surfaces and diameters of the true and false lumens, to measure the dimensions of the artery, to appreciate the presence of thrombi, to orientate the passage of the angioplasty guide in the good lumen if necessary, to choose the size of the stent, and to verify its good apposition at the end of the procedure.

The optimal treatment of SCAD remains poorly understood and is currently not based on any recommendation. Conservative management in a stable patient with preserved coronary flow has been associated with a low rate of in-hospital events and a good long-term prognosis.

If revascularization is required, percutaneous coronary intervention (PCI) is preferred to CABG. PCI has been associated with a high complication rate and a low success rate (approximately 30% failure rate) [8-10] because of the risk of iatrogenic dissection in fragile arteries [26], the risk of lesion extension, the spread of intramural hematoma on both sides of the stent with worsening of the coronary occlusion, and the risk of stent implantation in the false lumen.

The use of antiplatelet therapies and the duration of treatment in SCAD remains a controversial topic and a divergent practice. Furthermore, it seems wise to give anti-platelet aggregation since SCAD is sometimes associated with the presence of intraluminal thrombus and in view of the ACS context.

However, the modalities of administration of this anti-platelet aggregation are unclear, some experts recommending double anti-platelet aggregation (aspirin and clopidogrel) for an undefined duration, and others recommending simple anti-platelet aggregation.

Beta-blockers are frequently employed; they would reduce recurrent SCAD in hypertensive patients in the latest study by the Canadian Saw team. Angiotensin-converting enzyme inhibitors are used in patients with left ventricular dysfunction. The use of statins in SCAD is recommended in case of dyslipidemia or atheroma.

The prognosis of myocardial infarction following spontaneous coronary dissection is quite good, with a 10-year mortality rate of less than 10% in the literature,1 but is still associated with a high risk of cardiovascular events (myocardial infarction, heart failure, stroke, or secondary revascularization), and in particular of dissection recurrence.

CONCLUSION

SCAD, a particular form of ACS, remains a challenge in terms of diagnosis and management. It predominates in young women with few cardiovascular risk factors and is not specific to peripartum.

There are no clear-cut guidelines about management but data so far definitely suggest adopting a conservative approach. Angioplasty, risky and perilous, is justified when signs of ischemia persist or the flow of the artery is interrupted, while coronary bypass surgery remains a rescue solution.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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