Available online at www.rajournals.in

RA JOURNAL OF APPLIED RESEARCH
ISSN: 2394-6709
DOI:10.31142/rajar/v5i7.03
Volume: 05 Issue: 07 July-2019

International Open Access
ICV- 74.25
Impact Factor :5.985
Page no.- 2488-2494

The Spontaneous Dissection of Coronary Arteries: An Entire Still Unusual!

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ARTICLE INFO

Publication Online:
09 July 2019

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SUMMARY

Hematomas and coronary dissections are rare and little-known features of acute coronary syndrome (ACS) or even sudden death, especially in young women. Relatively large series have been published over the past five years to highlight this underestimated condition. Thus, the diagnostic and therapeutic modalities are better and better understood. We report a clinical case of coronary dissection in a 52-year-old patient due to compressional wall hematoma demonstrated in coronary angiography. The evolution was favorable under medical treatment combining platelet antiaggregants and beta blockers. Our case illustrates an approach to this particular entity while reviewing its clinical and paraclinical characteristics, its therapeutic management and its evolution.

KEYWORDS: spontaneous dissection of coronary arteries

INTRODUCTION

Spontaneous dissection of the coronary arteries known as SCAD (spontaneous coronary artery dissection) encompasses the concepts of hematoma and dissection and generally implies a non-atheromatous etiology.

Dissection is defined by the non-traumatic and non-iatrogenic appearance of arterial wall cleavage secondary to intra-mural hemorrhage with or without intimal tear and creating a false channel [1]. This separation can sit between the intima and the media or between the media and the adventitious [1]. The intraparietal hematoma, thus created, progresses and cleaves the different parietal layers. Myocardial ischemia is then the consequence of the compression of the true light by the hematoma (or the false light) without intraluminal thrombus [2].

OBSERVATION

We report the case of a 54-year-old patient admitted to the emergency room for prolonged acute chest pain. It is a constrictive retrosternal pain radiating to the back and upper left limb. She has no significant cardiovascular risk factor apart from android obesity. The patient has no particular pathological history. She weighs 96 kg and measures 1.56 m, ie a BMI of 39.5 kg / m2.

The cardiovascular clinical examination found a Blood pressure at 120/70 mmHg with a heart rate at 72bpm. Cardiac auscultation regains rhythmic beats with no added noise or audible murmur, especially in the course of large vascular axes. Peripheral pulses are present and symmetrical. Pulmonary auscultation does not aim at rattles. The abdominal examination does not find hepato-splenomegaly. The rest of the somatic examination is without particularity.

The electrocardiogram found a regular sinus rhythm, a frequency at 72 bpm, a left axis with negative anterior T-waves in anterior-septo-apical and lateral low with lower biphasic T waves, without rhythm or conduction disturbances associated.

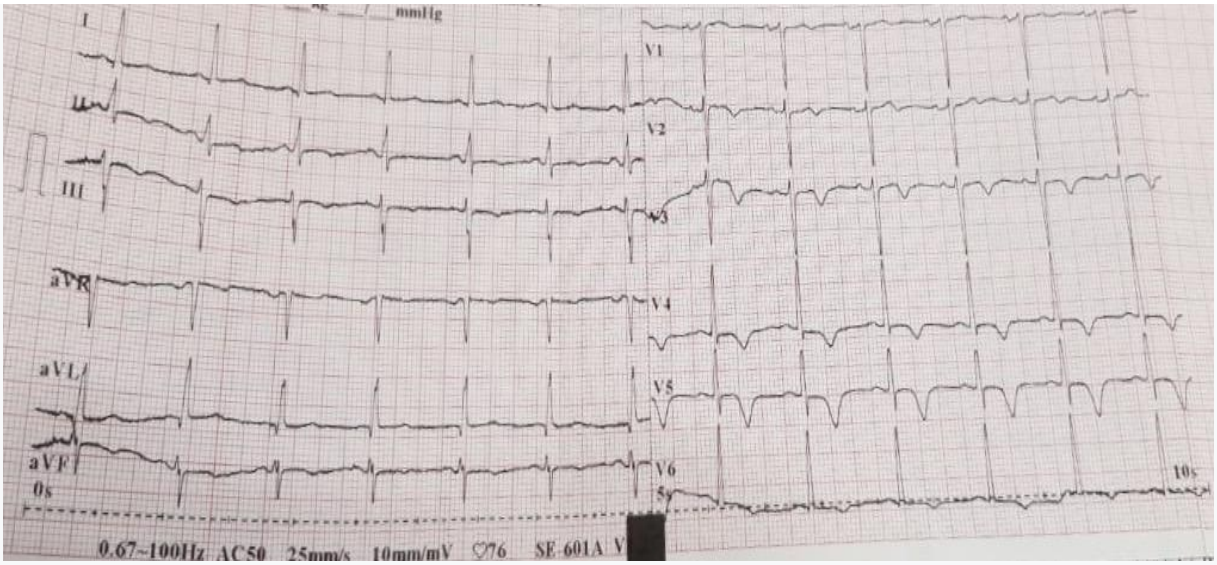

Figure n°1: Electrocardiogram of the patient showing negative antero-apical and lateral lateral T waves with lower biphasic T waves

Chest X-ray does not involve cardiomegaly or pulmonary parenchymal abnormality

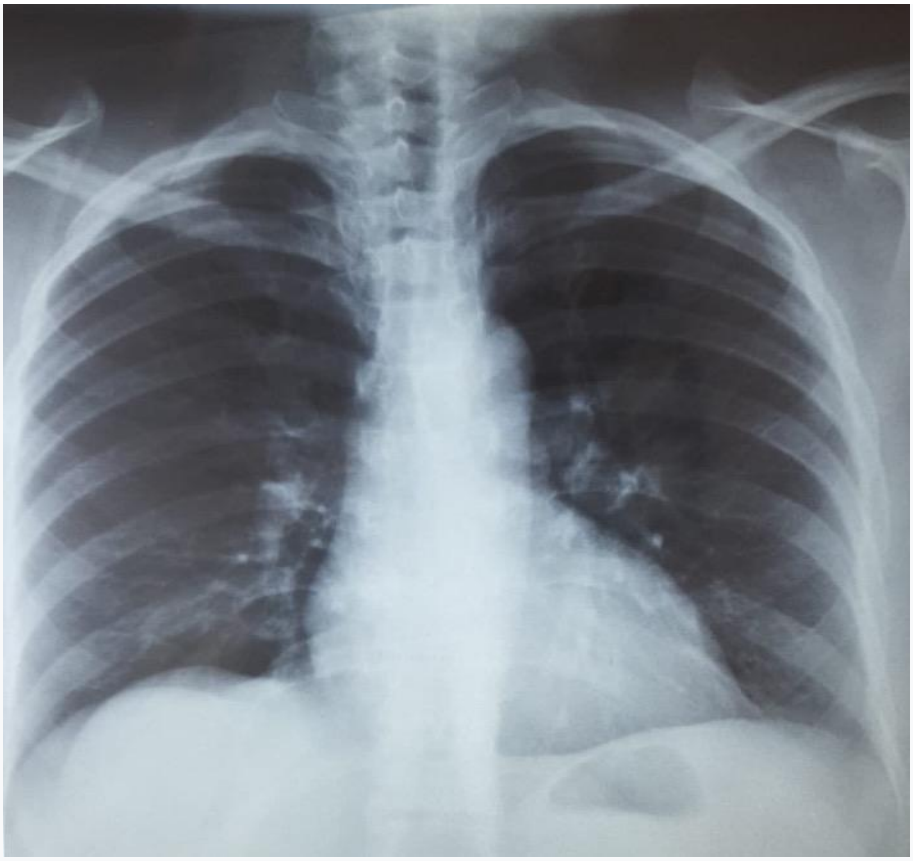

Figure n°2: Patient's lung radio showing a normal sized heart shape with healthy lung parenchyma

At the hemogram; microcytic anemia with a hemoglobin level of 10.3 g / dl and a VGM of 78.2%, white blood cells of 8200 elements / mm3, platelets of 272000 elements / mm3, sedimentation rate of 35 mm the first hour .

The troponin-hypersensitive is positive at 300 times normal. The level of CRP is 41 mg / l, urea at 0.23 g / l, the creatinine at 5 mg / l. Lipid and thyroid balance are normal.

The initial diagnosis was that of a ACS without positive troponin ST segment elevation and the patient was treated with a combination of platelet antiaggregant and anticoagulant after receiving nitrate sublingual derivatives.

Echocardiography initially performed in the acute phase found hypokinesia of the apex and apical segments of the inferoseptal and anterolateral wall with a left ventricular

ejection fraction (LVEF) at 55%. She had no pericardial effusion or valvulopathy.

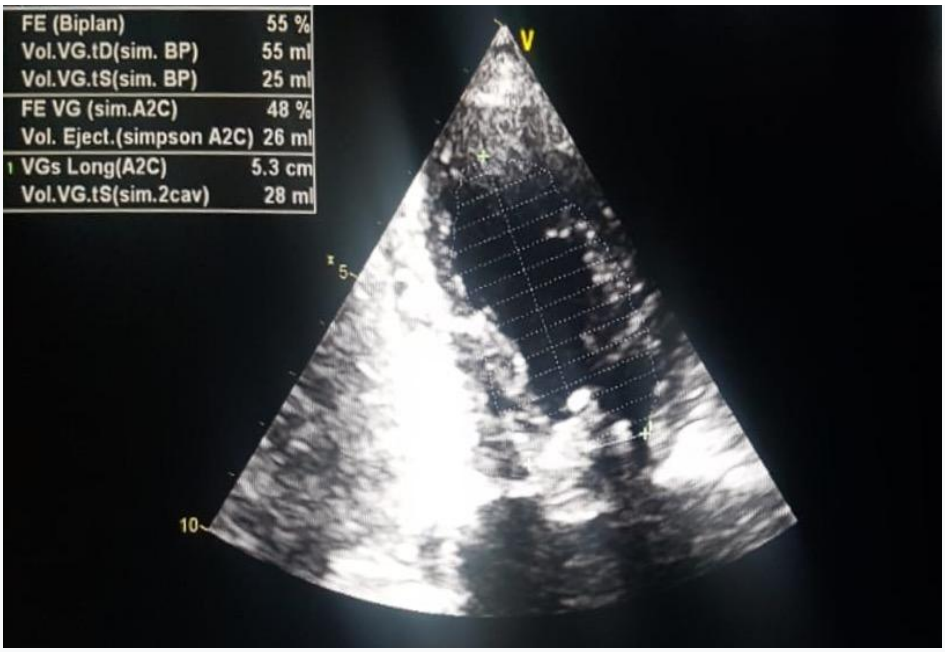

Figure n°3: ETT of admission showing a LVEF at 55%

Right femoral coronary angiography showed a sharp reduction in inter-ventricular artery (IVA) caliber in the tail of radishes, mimicking a vasospasm, very evocative of a spontaneous coronary dissection by hematoma and parietal.

Faced with this evocative aspect of a spontaneous compressive parietal hematoma that has evolved secondarily into coronal dissection with distal intimal rupture, we have decided on an initial conservative management with surveillance and medical treatment associating salicylates and Betabloquant. The evolution was favorable; the patient remained hemodynamically and respiratory stable, she did not represent angina recurrence or rhythm disturbances during her hospitalization. The therapeutic strategy has not been changed in this non-painful patient.

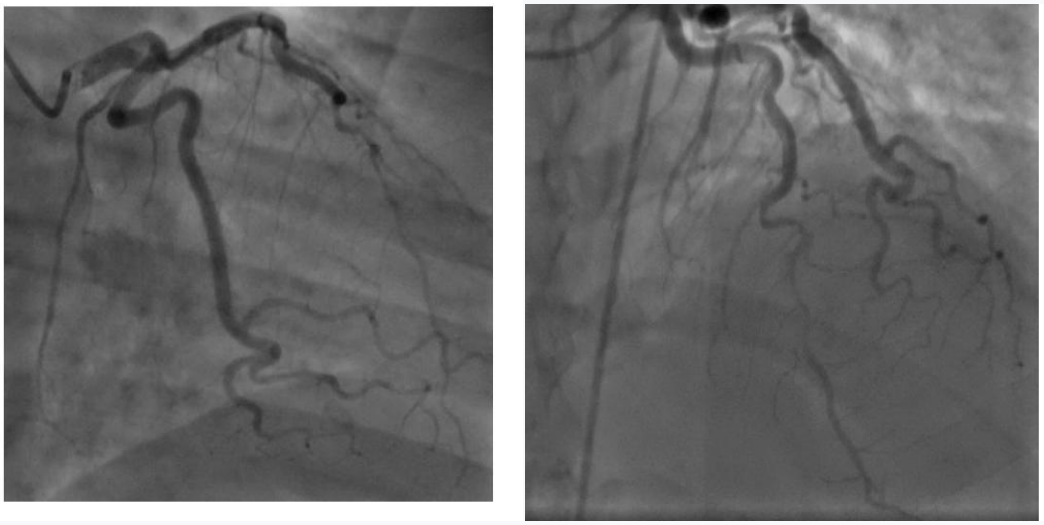

Figure n°4: Angiography of the anterior descending artery in anterior right angle 10 ° cranial angle 40 °, showing dissection of the distal anterior interventricular artery

Ten days after the cardiac event, the patient presented with inflammatory-like polyarthralgia affecting large joints associated with an evanescent generalized skin reaction made of confluent and pruritic, poorly defined, extensive macules. The patient was later admitted to the internal medicine department for aetiological assessment of her symptomatology that occurred a few days after the cardiac event. At the service, his clinical examination revealed arthralgia without inflammatory signs in relation to the joints, with a limitation of the mobility. She had no hepato-splenomegaly, no lymphadenopathy, or other signs pointing to an inflammatory or dysimmune disease.

Biologically, the patient did not have an inflammatory syndrome. Her lipid status and TSH and thyroid hormone

measurements were normal. The ASAT rate was 11 IU / l and ALAT 10 IU / l. Renal function was normal, urinary creatinine was 2282 mg / l and proteinuria was 0.28 g / l. An immunological report including antinuclear antibodies, native anti-DNA, anti-SSA, antiphospholipid antibodies, rheumatoid factor, ANCA was returned to normal. A viral screening essentially comprising HIV, HVB, HVC was negative. The dosage of ferritin and its glycosylated fraction was normal.

Morphologically, Doppler ultrasonography of the renal arteries and vessels of the neck was unremarkable.

The patient has evolved well under symptomatic treatment of short duration, associating analgesics and anti-inflammatories.

The etiological diagnosis of coronary dissection is idiopathic Two months after the initial event, the patient had undergone coronary examination; echocardiography showed an improvement in kinetic disorders with left ventricular ejection fraction increased from 55% to 62%.

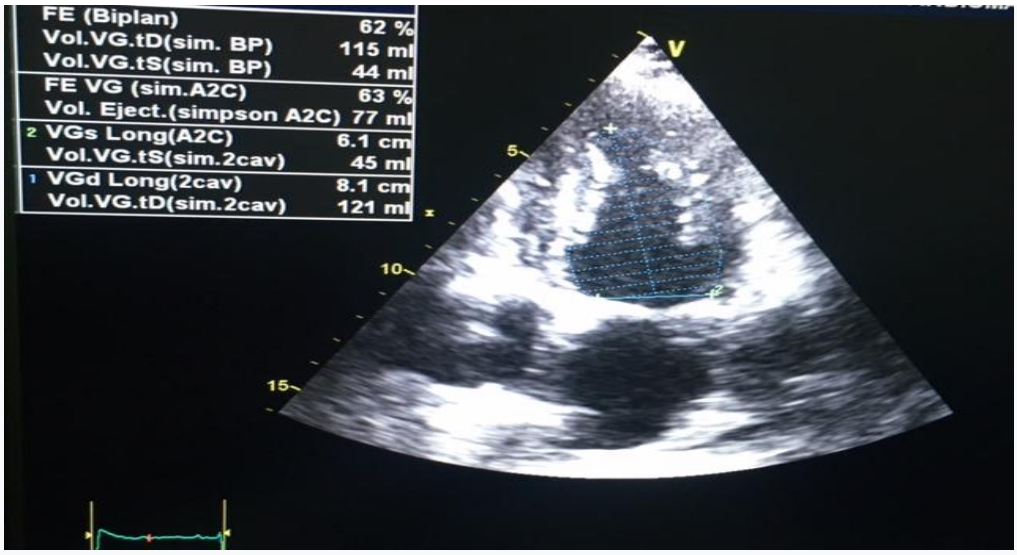

Figure n°5: Controlling echocardiography showing a 62% left ventricular ejection fraction

The controle angiographique confirms this diagnosis with very probable resolution of the hematoma and one finds the normal aspect of the caliber of the interventricular artery without plate of atheroma nor thrombus.

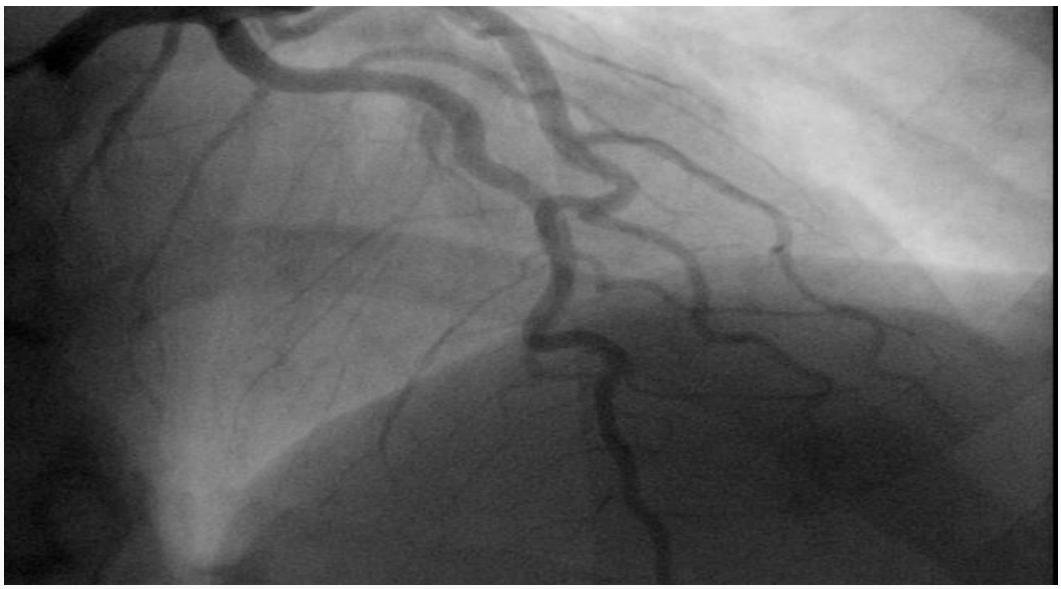

Figure N ° 6: Coronarography control

DISCUSSION

Undiagnosed and long considered rare cause of ACS, the first case of SCAD was reported in 1931, in a 42-year-old woman after completion of the autopsy [3]. However the coronary dissection is experiencing a revival of interest in the last 5 years. It is a special entity that does not usually have many cardiovascular risk factors [4] [5]. The diagnosis will be evoked on the ground more than on the clinical presentation; The average age of discovery of a SCAD is 44 -55 years, affecting the woman [6-9]. The age of our patient is 54 years similar to what is described in the literature.

The lower prevalence in the current registers only betrays the under-diagnosis of an unknown pathology. In the literature, higher incidences have been reported in young women: 8.7% of acute coronary syndrome women under 50 years and even up to 10.8% for ST + SCA [4].

Dissection and hematoma are the likely expression of the same pathophysiological entity; 2 mechanisms are thus evoked:

-The hypothesis of a primitive intimal breach creating a point of entry into the arterial wall

- The hypothesis of an initial rupture of the vasa vasorum: as illustrated by our case where the intraparietal hematoma diagnosed initially evolves into secondary dissection with intimal breach "inverted"; related to the increase in intramural pressure [13].

SCAD is almost always a acute coronary syndrome with troponin elevation. In the literature the most common symptom is chest pain (95, 9%) [1]; The electrocardiographic presentation is variable without any specificity, but also simulating and in the majority of the cases a acute coronary syndrome [1] [2].

The echocardiography could possibly find kinetic disorders which can, here too, wander the diagnosis and make it confused with an acute coronary syndrome, these disorders are generally localized and seem to be concordant with the lesions found in the coronarography. Left ventricular function is usually conserved or moderately impaired. However, the repeated control echocardiography during the course of the evolution can be practically normal, which is in line with the results of the echocardiography of our patient.

Coronary angiography is the gold standard for diagnosis, but it does not perform well in the evaluation and evaluation of certain types of SCAD because it does not allow analysis of the arterial wall; she is not luminogram. Coronary artery diagnosis was essentially based on the visualization of a pathognomonic aspect: the radio-transparent "flap" separating the two lights, sometimes associated with a stagnation of contrast material in the false light.

A French team has recently identified 5 angiographic criteria which, in case of evocative context, could make it possible to make the diagnosis in angiography (more than 3 criteria) and to reserve endocoronary imaging only in doubtful cases: absence of atheromatous lesion, flapendoluminal, stagnation of contrast medium, lesion limited by the birth of collateral, linear or more diffuse smooth caliper reduction mimicking a stick or tail of radish. The existence of a long stenos is of variable severity biconcave appearance often framed by division branches not changing after injection of risordantracoronaire should challenge the interventional cardiologist, especially if the clinical context is suggestive (ACS of the young woman) [5]. The diagnosis of coronary dissection in our patient was retained on the presence of 3/5 of the aforementioned criteria

Recently, an angigraphic classification has been proposed [10]:

SCAD type 1: "classic" aspect of dissection with "flap" and stagnation of contrast medium.

SCAD type 2: long and diffuse stenosis (> 20 mm) of variable severity with abrupt reduction of size. These long size reductions can concern the middle segments of the arteries or extend until distal,

SCAD type 3: more focal tubular stenosis mimicking atherosclerotic lesions (<20 mm) requiring con firmation in endocoronary imaging. The coronary hematoma is most often distributed on a single artery. The involvement can be pluritroncular (9 to 19% of cases) by progression of the hematoma by contiguity; More rarely, non-contiguous plurifocal lesions are observed (5 to 10% of cases)

Our patient was classified stage 2.

Currently, intra-coronary imaging, including IVUS (Intra-vascular Ultrasound) and optical coherence tomography (Optical Coherence Tomography OCT), thanks to an accurate analysis of the different layers of the coronary artery wall, presents a better sensitivity to estimate the prevalence of SCAD in ACS at 4% [9]. Alfonso et al demonstrated the added value of OCT in the diagnosis of SCAD: 17 patients with a clinical and angiographic suspicion of SCAD were prospectively included. All benefited in addition to the coronarography of an OCT. The diagnosis of SCAD was confirmed in OCT by visualization of a double lumen or an intraparietal hematoma in 11 cases, whereas only 3 patients of them had a flap appearance in angiography [11]. The OCT allows to highlight an intimal entrance, to measure the length and the thickness of the intimomedial membrane, the surfaces of the real and false lights [11]. In addition to its diagnostic value, OCT also has a therapeutic interest in guiding rescue angioplasty [11] [12]. Unfortunately, this imaging technique is not yet available in our training.

The etiologies are essentially represented by pathological or physiological conditions that weaken the arterial wall and predispose to dissection, notably: atherosclerosis, fibromuscular dysplasia, pregnancy and postpartum, certain connective tissue diseases (Marfan, Ehler-Danlos, etc.).), systemic inflammatory diseases (lupus, Crohn ...), toxic causes (cocaine), coronary spasm. While idiopathic coronary dissections remain the most frequent [14].

Recent data support the hypothesis of a strong association between fibromuscular dysplasia and spontaneous coronary dissection. The coronary dysplastic arteries are more likely to dissect [15] [16]. To validate this hypothesis, the team of Saw et al. took over 50 cases of SCAD between 2006 and 2012, with search for fibromuscular dysplasia in the territories of the renal, iliac and supra-aortic trunks in Forty-seven of these patients (angiography or angioscan or angio-MRI). 86% of patients had at least one affected territory [16].

However, fibromuscular dysplasia was not a cause for our patient for several reasons: clinically, she has no history of stroke or transient ischemic attack, she did not present a elevation of blood pressure during hospital stay with a near normal clinical examination. Renal function is normal, 24-hour proteinuria is negative. The vascular assessment performed by including echo-Doppler renal arteries and supra-aortic trunks is negative.

A trigger factor seems essential, such as intense physical exercise, emotional stress or Valsalva-type maneuver [8], [17]. Consumption of cocaine or amphetamines [2] [17] could be responsible for increased stress and parietal stress. In the case of our patient, the interrogation had found a psychic stress triggered by a marital conflict occurring just before the onset of her cardiac symptomatology.

Due to the lack of data from randomized studies, there are no "guidelines" for the management of SCAD. Any suspicious case of SCAD must benefit from an early coronarography in order to confirm the diagnosis and implement a suitable therapeutic strategy.

On the basis of expert consensus, conservative medical treatment is the first-line treatment in the management of stable SCAD, associated with close monitoring of +/- 5 days in ICUI [16] [18-21]. This strategy is all the more justified because spontaneous parietal scarring is observed in most cases on late angiographic tests (≥ 30 days) [20] [16].

In the Vancouver series 134/168 patients benefited from a conservative initial strategy; only 3 (2.3%) required revascularization for dissection extension [2]. The 79 one-month control coronary angiograms found complete resolution of the lesions after medical treatment alone [2].

Because of the risk of progression of the hematoma and the risk of bleeding weighed against the potential benefit of the thrombogenic nature of the dissection, it is preferable to put the patients under salicylate monotherapy which would appear to reduce the compression on the true arterial light [14]. Beta-blockers may reduce parietal stress similar to what is observed in aortic dissection.

ACE inhibitors are prescribed for cardioprotective purposes, especially for ventricular dysfunction. There is no consensus on the value of statins in this indication [14]. They are empirically prescribed [6]

Revascularization has never been randomized; this is necessary in complicated cases: recurrent myocardial ischemia, hemodynamic instability, ventricular rhythm disorder or dissection of the common core [5].

The majority of patients in the Mayo Clinic series (189 patients) were conservatively treated and of the 90 non-revascularized patients initially, only 9 (10%) required subsequent revascularization (mean delay of 4 days) for pain. recurrent thoracic and / or progression of dissection [19].

On the basis of these data, it was decided to treat our patient conservatively without resorting to initial revascularization.

Revascularization by coronary angioplasty is the treatment of choice in the presence of a favorable coronary anatomy. In the opposite case, coronary bypass grafting will be preferred. Technically, angioplasty is difficult to perform. The failure rate varies from 27% to 53% [2] [22]. This angioplasty would be more relevant if guided by OCT in a rescue situation [23] [12]. Bridging surgery is also difficult because of the fragility of the arterial wall and the difficulty of anastomosis on dissected walls. Moreover, its long-term results are disappointing,> 70% of grafts found occluded or involuted during follow-up [18]. The natural evolution of the dissection towards the spontaneous cicatrization explains these disappointing results.

The prognosis is better in ACS [18] [29]. Cardiac markers are low and left ventricular ejection fraction is readily preserved [18] [18] [21]. Recent series report a relatively low hospital mortality of 0 to 4% with excellent long-term prognosis for patients surviving in the acute phase [4] [16] [24]

In prospective cohorts, in which patients underwent control coronary angiography during follow-up, there was spontaneous healing of ADSD in 73-97% of cases [2] [19] [25]. This cure rate can reach 100% of cases when coronary angiography is performed in a period greater than 26 days post-dissection [26]. This same finding was found in our patient we observed a spontaneous healing of the dissection.

The risk of recurrence of coronary dissection justifies attentive cardiovascular follow-up. In the Tweet et al. Registry, the recurrence rate was 17% (15/87) during a follow-up of 47 months and 12 of these 15 recurrences affected another coronary than the one initially involved [18] [22].].

CONCLUSION

SCAD is an infrequent cause of ACS, which is increasingly being tested with better diagnostic means. In Morocco, the recent introduction of interventional cardiology will help identify this rare cause of ACS in our context and improve the management of our patients

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