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Case Series

Spontaneous Coronary Artery Dissection (SCAD): Report of Five Cases and Literature Review

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Abstract

Spontaneous Coronary Artery Dissection (SCAD) is an infrequent and often missed diagnosis among patients presenting with Acute Coronary Syndrome (ACS) and is typically affecting a younger, otherwise healthy population. Its pathogenesis remains unclear and various mechanisms have been postulated. The clinical presentation of SCAD includes angina, ACS, heart failure, arrhythmias, sudden death and depends on the extent and the flow limiting severity of the coronary artery dissection. The diagnosis is usually confirmed by coronary angiography. There are no guidelines on the treatment of SCAD, which can be conservative or interventional with PCI or surgery. We present five cases of SCAD identified with coronary angiography and successfully treated in our institution between June 2015 and June 2016, along with a short review of the literature.

Keywords: *Coronary Artery Dissection; Coronary Artery Disease; Acute Coronary Syndrome*

Introduction

Spontaneous Coronary Artery Dissection (SCAD) is an infrequent and often missed diagnosis among patients presenting with Acute Coronary Syndrome (ACS). It's an extremely rare condition that has a variety of presentations including angina, ACS, heart failure, arrhythmias and, in some cases, sudden death [1-4]. It is typically affecting a younger, otherwise healthy population and it should be suspected in healthy middle-aged patients without risk factors for CAD presenting with ACS [5]. The most common presentation of SCAD is the acute onset of severe chest pain associated with autonomic symptoms such as diaphoresis, palpitations, and either hyper- or hypotension [6]. The incident case in a 42-year-old woman was diagnosed at autopsy in 1931 [7].

Case Presentation

We present five cases of SCAD identified with coronary angiography and successfully treated in our institution between June 2015 and April 2016.

Case 1: A 49 year old menstruating, female with a history of dyslipidemia presented to the emergency department with an acute onset chest pain. The patient had no family history of premature coronary artery disease (CAD) and no other predisposing factors. Her electrocardiogram (ECG) on admission (Figure 1) was in sinus rhythm with ST elevation in leads II, III, aVF and ST depression in I and aVL. She was treated with aspirin, unfractionated heparin and a loading dose of ticagrelor and taken to the cath lab for an urgent coronary angiography which revealed a very distal subtotal occlusion of the second (distal) OM with TIMI 3 flow due to a type 2 spontaneous dissection (Figure 2). The patient was decided to be treated medically and was transferred to the Coronary Care Unit (CCU). The clinical course during hospital stay was uneventful, while echocardiography revealed no regional wall motion abnormalities and only a small pericardial effusion. She was discharged 6 days after admission on dual antiplatelet therapy, statins and b blockers. Follow up with echocardiography at one month showed resolution of the pericardial effusion and the patient was still asymptomatic.

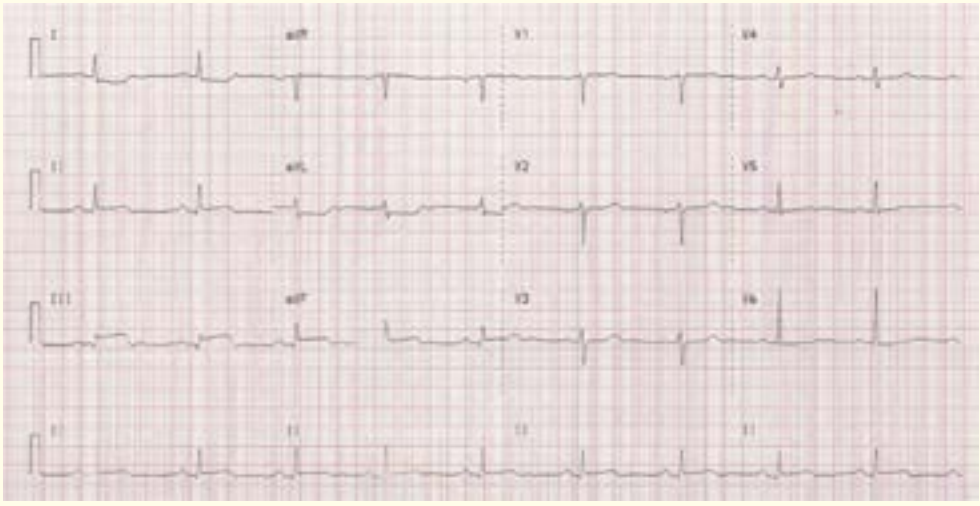

Figure 1: *ST elevation in leads II, III and aVF, ST depression in I and aVL (case 1).*

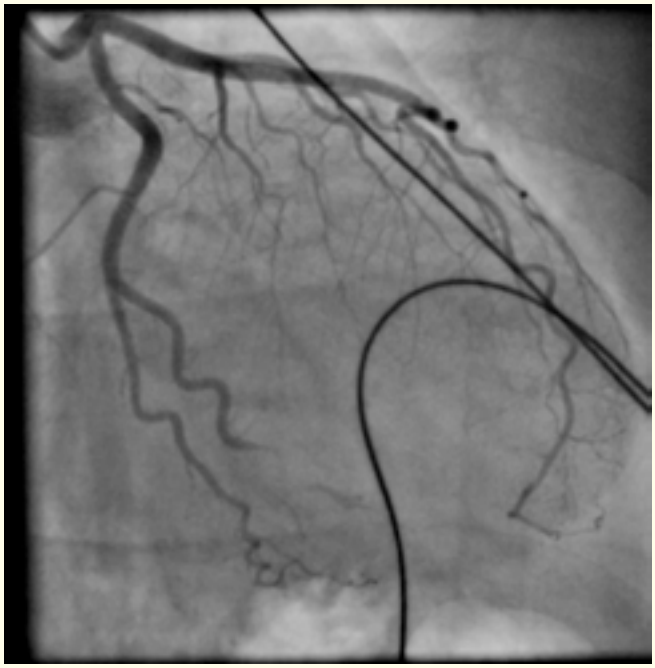

Figure 2: *Type 2 spontaneous dissection and subtotal occlusion of the second OM (case 1).*

Case 2: A 37 year old male with a history of dyslipidemia presented to the emergency department with a twelve hour history of acute onset chest pain. The patient had no family history of premature CAD and no other predisposing factors. His ECG on admission (Figure 3) was in sinus rhythm with ventricular bigeminy and Q waves in leads III, aVF. The patient was treated for ACS with antiplatelets, anticoagulation, nitrates and beta blockers and admitted to CCU. Coronary angiography revealed a dominant RCA, dissected and subtotally occluded from the mid segment with a heavy load of thrombus. The dissection (type 1) and a large false lumen was starting from the mid segment and extending beyond the crux and both PLB and PDA were involved and occluded. PDA was filling retrogradely from the LCA (Figure 4a). Despite difficulty both the PDA and PLB were wired and thrombus aspirations were performed. PCI was done using drug eluting stents (DES) in a culotte technique (Figure 4b). Echocardiography revealed moderate inferior wall hypokinesis with an ejection fraction of 55 - 60%. The clinical course during hospital stay was uneventful and the patient was discharged 6 days after admission. Discharge treatment included dual antiplatelet therapy, statins, beta blockers and ACE inhibitors.

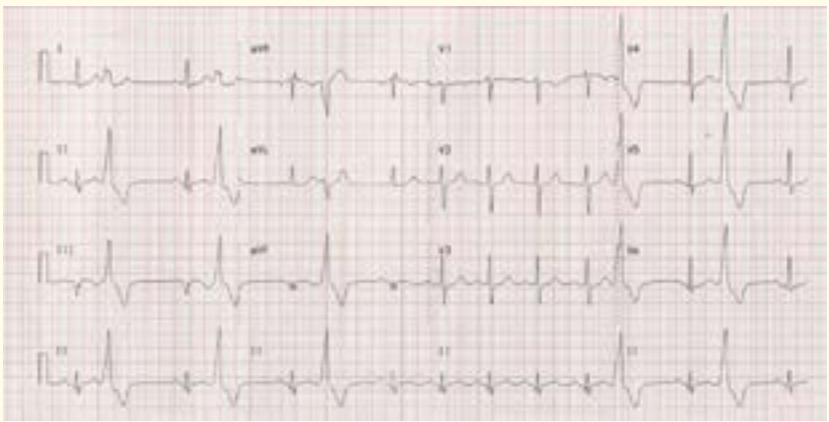

Figure 3: *Ventricular bigeminy and Q waves in leads III, aVF (case 2).*

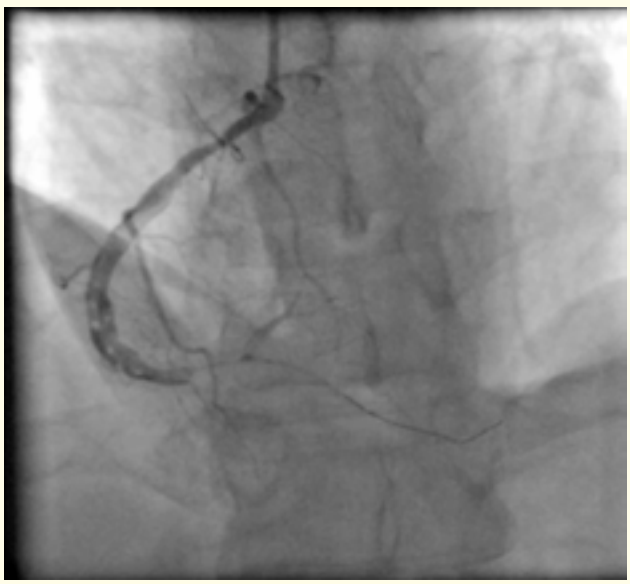

Figure 4a: *Type 1 dissection and subtotal occlusion of the RCA with large false lumen and heavy load of thrombus (case 2).*

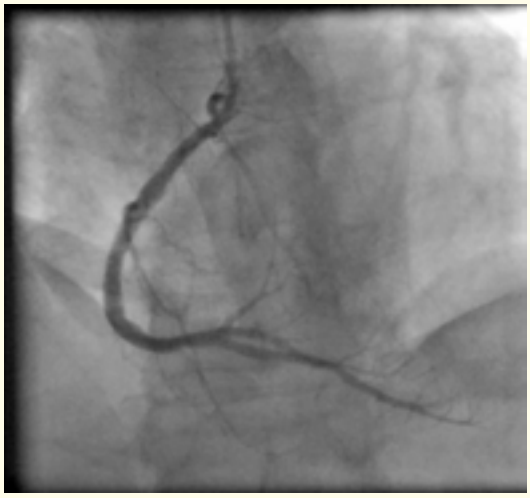

Figure 4b: *PCI to RCA with DES in a culotte technique (case 2).*

Case 3: A 24 year old male with history of dyslipidemia was referred for a coronary angiogram due to ACS. He had no previous history of CAD or family history of premature CAD. His ECG on admission (Figure 5) was in sinus rhythm with a non-persistent ST-segment elevation in leads II, III and aVF which was followed by ST depression and T wave inversion in leads III, aVF. The patient was treated with morphine, dual antiplatelet therapy, statins, b blockers and low molecular weight heparin. Echocardiography revealed anterior wall hypokinesis with an ejection fraction of 45-50%. The coronary angiogram revealed an occluded LAD at the mid segment with distal retrograde filling (Figure 6a). Predilatations revealed the presence of thrombus which were aspirated and a type 1 dissection which was treated with a DES (Figure 6b). The patient was discharged with dual antiplatelet therapy, statins, beta blockers and ACE inhibitors.

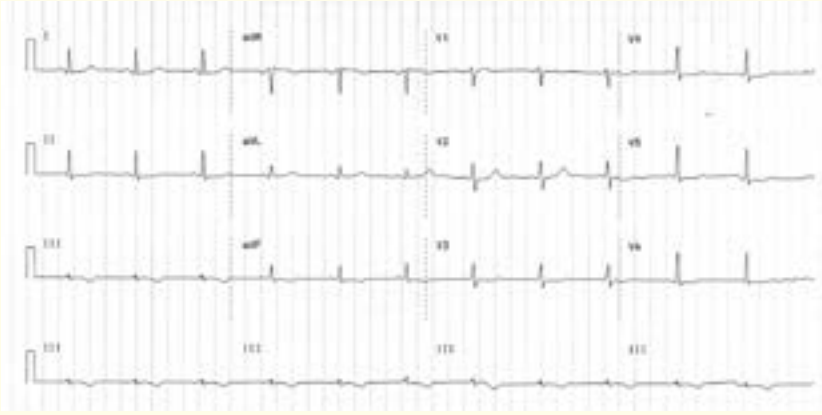

Figure 5: *ST depression and T wave inversion in leads III, aVF (case3).*

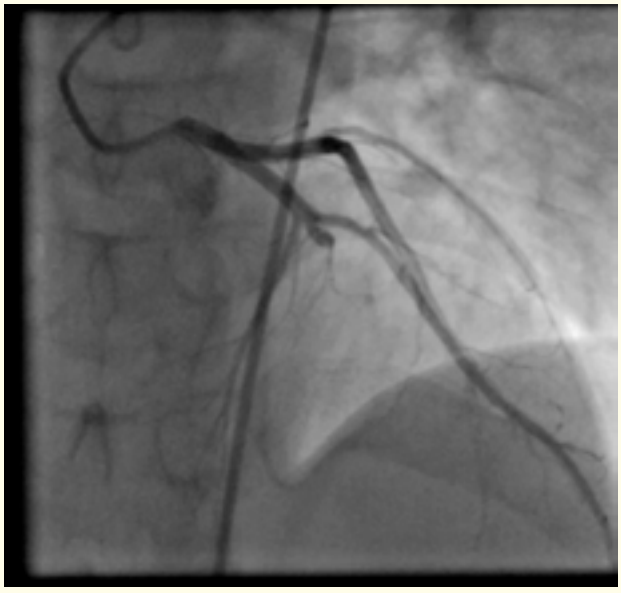

Figure 6a: *Occluded LAD at the mid segment with distal retrograde filling (case 3).*

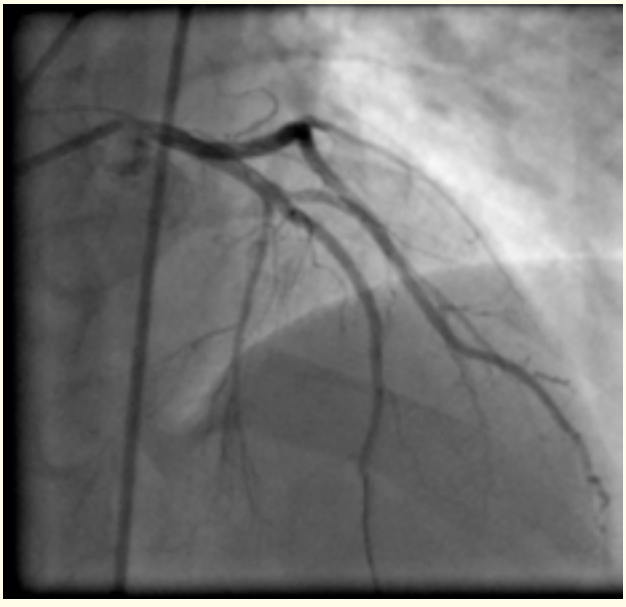

Figure 6b: *PCI to LAD with DES (case 3).*

Case 4: A 46 year old menstruating, female with no previous medical history was referred because of ACS. She was a smoker with no previous history of CAD or family history of premature CAD. Her ECG on admission (Figure 7) was in sinus rhythm with non-persistent ST-segment elevation in leads V1-V4. The patient was treated with morphine, dual antiplatelet therapy, statins, beta blockers and low molecular weight heparin for ACS. Echocardiography revealed no regional wall motion abnormalities. Coronary angiogram revealed an LCx with diffuse distal stenosis, extending to the third OM due to a type 2 spontaneous dissection (Figure 8). The patient was asymptomatic, and in the absence of ongoing ischemia, she was decided to be treated conservatively. The clinical course during hospital stay was uneventful and the patient was discharged 5 days after admission with dual antiplatelet therapy, statins and b blockers.

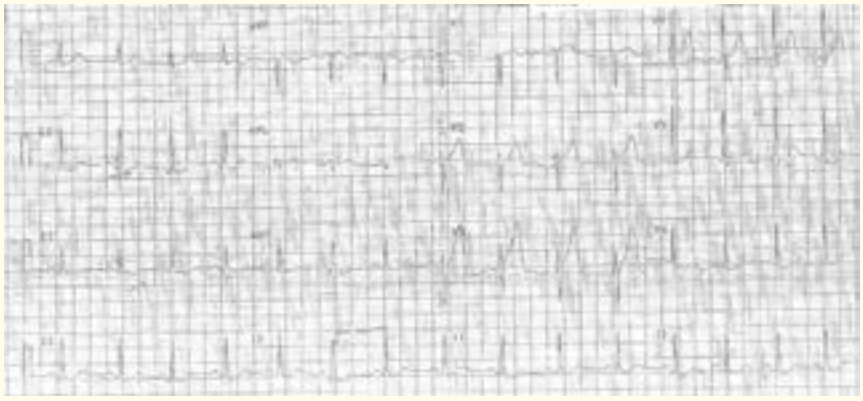

Figure 7: *ST elevation MI in V1-V4 (case 4).*

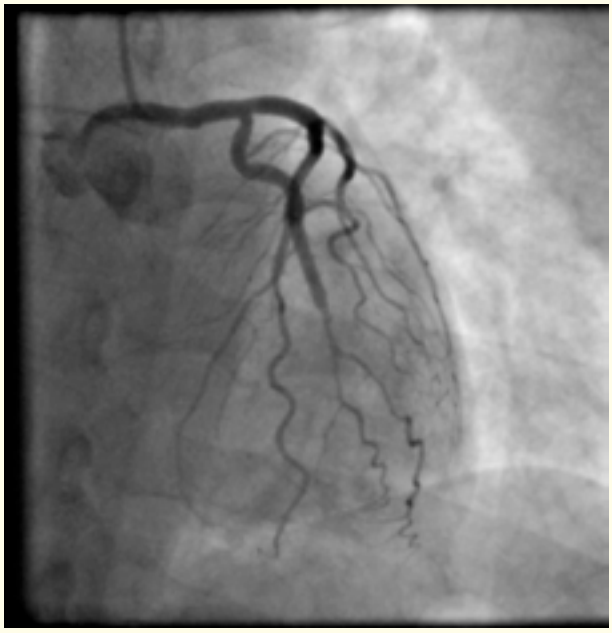

Figure 8: *Type 2 spontaneous dissection of the LCx (case 4).*

Case 5: A 49 year old menstruating, female with no previous medical history was admitted because of acute chest pain and troponin rise (Non STEMI). She had no ECG changes (Figure 9) and no wall motion abnormalities on echo. Coronary angiography revealed a type 2 spontaneous dissection of the distal LCx-OM (Figure 10) which was decided to be treated medically with dual antiplatelet treatment, b blockers, nitrates and statins. Atypical symptoms of chest pain persistent for a few weeks more which were controlled with oral nitrates. Three months later was free of symptoms without need for nitrates.

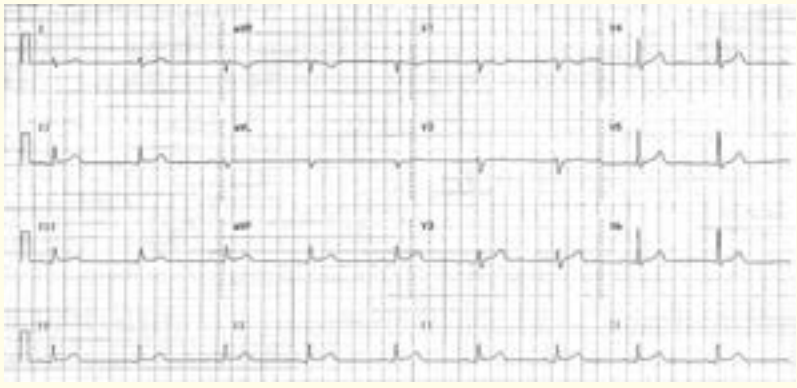

Figure 9: *No ECG changes (case 5).*

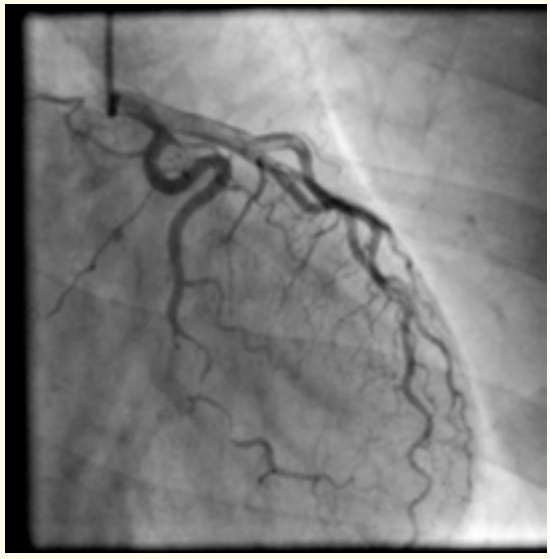

Figure 10: *Type 2 spontaneous dissection of the LCx-OM (case 5).*

Discussion

SCAD is defined as a non-traumatic and non-iatrogenic separation of the coronary arterial walls, creating a false lumen [1,4]. Coronary artery dissection is termed spontaneous once secondary causes like coronary interventions, cardiac surgery, trauma, and aortic dissection have been excluded [6]. Whilst SCAD is a recognized entity since it was described by Pretty in 1931 [7], its pathogenesis remains unclear and various mechanisms have been postulated even though no single etiology completely explains it.

It's difficult to estimate the true prevalence of SCAD due to under-diagnosis. In fact, earlier literature reports were mostly post-mortem [1,3]. The recent awareness of the disease and advances made in intracoronary imaging techniques such as intravascular ultrasound (IVUS) and optical coherence tomography (OCT) have led to several new insights, a better recognition of acute SCAD and an increased number of cases being reported ante mortem [1]. The population-based incidence of SCAD is unknown. Retrospective registry studies have reported SCAD detection in 0.07% to 1.1% of all coronary angiograms performed [5]. In our center the five cases in a year represent a 0.3% of all coronary angiograms. A Pubmed search yielded in excess of 500 articles, mostly case reports and case series [6]. An inherent difficulty in understanding the natural history and prognosis of the condition is the rarity of its occurrence [6]. The Discovery trial was initiated as the first prospective registry to look into the incidence, therapeutic options, and outcome analysis for SCAD [6,8]. The reported wide range of 0.07% to 1.1% relates primarily to the variable inclusion of atherosclerotic plaque dissection within study populations and it constitutes a limitation of prior studies, as it seems most likely that plaque dissection represents a variant of a typical ACS caused by underlying atherosclerosis, whereas true SCAD occurring outside the setting of atherosclerosis represents a distinct clinical entity [5,9-11].

The condition affects predominantly young adult patients with mean age of 30 - 45 years at presentation [12]. The mean age of presentation is 42 years old. At least 70% of SCAD cases are women and 26 - 38% of cases occur in late pregnancy, peripartum or postpartum. Nitroglycerin-resistant segmental narrowing in a young female without any cardiac risks can be a presumptive diagnosis for SCAD [6,13,14]. The reported incidence of involvement for the LAD is 57 - 75%; the RCA is 20 - 32%; the LCx 4 - 21% and the LMCA is 1 - 21% [5,12,13]. Multi-vessel SCAD is rare but has been reported [12]. The LAD is more common in women and the RCA in men.

Shamloo., *et al*. did a systematic analysis of all published cases and came to the following conclusions:

1. About 80% of cases were diagnosed by coronary angiogram and the rest 20% were diagnosed postmortem;
2. Isolated single vessel involvement was the most frequent lesion;
3. Early intervention (either stent or bypass graft surgery) strategy had a superior outcome compared to conservative management; and
4. Administration of thrombolytics (before diagnosis of SCAD) resulted in worsening of the condition [6,15].

The clinical presentation of SCAD depends on the extent and the flow limiting severity of the coronary artery dissection [16]. The separation can occur between the intima and media or between the media and adventitia, with subsequent formation of intramural hematoma (IMH) within the arterial wall that compresses the arterial lumen thus compromising the antegrade blood flow leading to subsequent myocardial ischemia or infarction [1,4,17]. There are two proposed mechanisms for the formation of IMH with SCAD. The first involves an intimal tear resulting in blood from the endoluminal space entering the intimal space, creating a false lumen filled with blood. The second mechanism of IMH formation is thought to be due to rupture of the vasa vasorum and when such rupture occurs, blood can pool within the intramural space, creating a false lumen filled with hematoma [1]. The degree of luminal compression is variable, ranging from mild to complete occlusion, and can often be missed or misinterpreted, contributing to the underdiagnosis of SCAD [18].

SCAD constitutes a heterogeneous entity as several diseases and conditions have been associated with it [18]. Risk factors for SCAD include pregnancy and the peripartum period, autoimmune and collagen vascular diseases such as Ehlers-Danlos disease, Marfan's Syndrome, Fibromascular Dysplasia, Systemic Lupus Erythematosus and chest trauma [18-21]. Other factors related to SCAD include hyper-

tension [26,27], cocaine use [22,24], coronary spasm due to increased shear forces in the vessel [22,24], treatments such as cyclosporine [22,24] and coronary compressive forces secondary to intramyocardial pathways and exhausting physical exercise. Vessel wall weakness is an important substrate for the development of spontaneous coronary artery dissection, be it in pregnant states or as a part of systemic inflammation [22,25]. It is worth mentioning that reported familial groupings suggest the possibility of a hereditary factor at play [22,26].

Diagnosis: The diagnosis is usually confirmed by coronary angiography which is considered the primary tool for diagnosis of SCAD. Common angiographic findings include intimal flap, two separate communicating lumens, multiple dissecting lines, and coronary aneurysm communicating with the lumen [6].

Three distinct angiographic appearances and patterns of SCAD have been described in literature to aid diagnosis:

1. Type 1 refers to the pathognomonic angiographic appearance of SCAD with contrast dye staining of the arterial wall with multiple radiolucent lumens [1].
2. Type 2 refers to the appearance of a diffuse stenosis of varying severity. This angiographic appearance is often missed or misdiagnosed as it is not well appreciated. SCAD commonly involves the mid to distal segments of coronary arteries and it can extent to the distal tip. There is often an appreciable and subtle change to arterial caliber. This diffuse narrowing (typically > 20 mm) is usually smooth and can vary in severity from a mild stenosis to a complete occlusion [1].
3. Type 3 is the most challenging to differentiate from atherosclerosis and the most likely to be misdiagnosed as it mimics atherosclerotic lesions. Lack of atherosclerotic changes in other coronaries, long lesions, hazy and linear stenosis are some of the features that favor SCAD and in the case of high suspicion, the angiographer should employ intracoronary imaging techniques to reach a diagnosis [1].

The use of intracoronary imaging techniques such as IVUS and OCT, can provide detailed morphological information on coronary lesions and the location of dissection planes between the different layers of the arterial wall thus enabling a more detailed clinical assessment of SCAD [18]. These techniques should be utilized when angiographic assessment doesn't reveal high diagnostic accuracy and further information from additional imaging might change clinical decision making. Longitudinal follow-up of patients with SCAD could include the use of non-invasive coronary angiography by multidetector computer tomography (MDCT) which examines the extent and thickness of hematoma and documents healing during follow-up [6,18,27].

Management: There are no randomized trials on the treatment of coronary artery dissection as the literature consists of case reports and case series and therefore there is no consensus or guidelines about the right way of treatment including medical therapy, interventional treatment with PCI or surgery [6]. Different strategies of treatment have been discussed in the past years. Conservative management of patients with SCAD is a possible treatment strategy in stable patients [1]. Treatment is guided by the clinical symptoms, extent and location of the dissection, and the hemodynamic status of the patient [6]. In case of presentation with an acute myocardial infarction with ongoing ischaemia, the first objective should be to restore normal coronary flow [16]. The use of fibrin-specific thrombolytic drug therapy is discouraged because it may result in further propagation of the dissection due to progression of the intramural haematoma [16,28]. Primary percutaneous coronary intervention (PCI) remains the reperfusion strategy of choice as percutaneous coronary strategies to restore coronary perfusion and hemodynamic stability, are reasonable in acute cases with proximal dissection (of major epicardial vessels) and arterial occlusion [6,16]. No data exists regarding use of drug-eluting stents (DES) over bare metal stents, although there is a concern that DES may adversely affect arterial healing [16]. It should be noted that PCI was associated with elevated rates of technical failure relating to passage of coronary wire into the false lumen of the dissected vessel or loss of coronary flow through propagation of dissection and displacement of intramural hematoma by stent placement [5]. Cannulation of the false lumen and coronary perforation/occlusion are described as the potential complications of attempted intervention in SCAD [6].

Dissection of distal vessels of small caliber or with preserved blood flow without symptoms or ECG changes may occur [6]. If the vessel is open and the flow is normalised at the time of angiography, it is preferable to treat the dissection conservatively as good angiographic and clinical outcomes have been described with medical treatment only [11,16,30]. Many interventionists feel uncomfortable with conservative treatment alone, but this approach seems wise in small and medium-sized vessels with normal flow. With conservative measures, coronary artery dissections have even shown complete angiographic resolution after a year [11,16,31,32]. However, when there is a large epicardial vessel dissected, placement of a stent will often be the treatment of choice [16].

Reduction of vessel wall sheer stress with the use of beta blockers has been proposed as a therapeutic option [6,31]. The role of long-term antiplatelet treatment in patients with SCAD, not receiving stent, has never been examined in a clinical trial; however, there are anecdotal evidences of benefit [6,31]. Whether to use aspirin or clopidogrel or both in conservatively treated patients remains debatable [11,16]. Furthermore, ACE inhibitors or AT2 antagonists are of special interest, because the renin-angiotensin system has been shown to be involved in the regulation of matrix metallo-proteinases (MMPs) that stimulate the degradation of collagen and elastin and impede the structure and integrity of the vessel wall. ACE inhibitors and AT2 antagonists may inhibit the expression of MMPs and stabilize the vessel wall [16].

The phase following the occurrence of SCAD should not only be used for the recovery of the patient but also to evaluate the possible underlying cause of SCAD as it affects the treatment of choice which should be tailored to each patient [16]. Coronary angiography as well as IVUS will help to identify signs of coronary atherosclerosis [16]. In case of atherosclerosis, aggressive measures, including aggressive lipid lowering by statins, β-blockade, antihypertensive therapy and antiplatelet therapy, should be taken for the stabilization of atherosclerotic plaques. If there are no signs of atherosclerosis in the coronary arteries, statins are not indicated, although beta blockers and platelet inhibitors should be continued after discharge [16]. Pregnancy testing should always be performed in premenopausal women. If a pregnant patient is identified with SCAD, statins and ACE inhibitors should not be given due to their teratogenic effects [16]. A connective tissue disease should be suspected from a positive family history or abnormalities on physical examination as there is an association between SCAD and specific connective tissue disorders such as Marfan's syndrome and Ehlers-Danlos type IV [16].

Prognosis: A particularly poor prognosis is carried with the involvement of the left main coronary artery or the presence of multivessel lesions [6,33-36]. A better prognosis of SCAD is associated with male gender and atherosclerotic etiology [6,37]. Recurrent coronary dissection has been reported in 50% of patients mostly within 1 or 2 months [6]. Recurrent SCAD in subsequent pregnancies has not been reported yet [6,38].

The five cases we present from our institution are good examples of the distinct angiographic appearances and patterns of SCAD, the typical clinical presentation of acute coronary syndrome and the different treatment approach in each case. The decision to intervene in a dissected coronary artery will depend on the site of the occlusion, the size of the vessel, the extend of the dissection and the TIMI flow while the patient's clinical presentation, symptoms and hemodynamic status are very important. Investigations to exclude other diseases associated with SCAD and follow up for the adherence to medical treatment are essential. Even though 50% recurrence is reported usually in the first 2 months in our experience this seems to be less and was not observed to our five patients.

Conclusions

SCAD is an infrequent and often missed diagnosis among patients presenting with ACS. The diagnosis is usually confirmed by coronary angiography and identification of this entity will lead to the appropriate treatment, which can be conservative or interventional with PCI or surgery. We present five cases which represent different angiographic appearances, the variable clinical presentation and the different treatment options.

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