Case Report

Spontaneous Coronary Artery Dissection: A Case Series

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Abstract

Spontaneous coronary artery dissection (SCAD) is a rare, infrequent & often missed clinical entity in patients presenting with the acute coronary syndrome (ACS). Patients of SCAD can present with different clinical presentation ranging from myocardial ischemia to myocardial infarction, different types of fatal arrhythmias and even death. Frequently missed due to lack of angiographic recognition, documentations are the major causes of under-diagnosis. With the advancement of diagnostic modalities including intracoronary imaging, there has been an improved diagnosis of SCAD. These series aim to showcase this rare clinical entity as well as make awareness among the Cardiologists to diagnose & further management of the patients to reduce morbidities & mortality among the patients with acute coronary syndrome.

Keywords: *Spontaneous coronary artery dissection (SCAD), acute coronary syndrome, myocardial infarction, coronary angiogram*

Case Summary

Case 1:

Mr. X, 38 years hypertensive, diabetic & current smoker patient was evaluated for a coronary angiogram. He sustained acute anterior wall STEMI 01 month back, with it he got himself admitted to a local hospital. There he was diagnosed as a case of acute STEMI (Anterior) with late presentation and was treated with anti-platelets, statin, other anti-ischaemic drugs & sub-cutaneous low molecular weight heparin. After stabilization, he was discharged with the advice of a coronary angiogram.

In our center, he was at first clinically evaluated in the outpatient department. There he gave complaints of exertional SOB (NYHA class II/III) since his myocardial infarction (MI). His vitals showed, Pulse: 88/ min, regular; BP: 130/80 mmHg without any postural drop, Heart: S1 & S2 were audible without any added sound, Lungs: Clear without any added sound, Respiratory Rate: 20/ min, Temperature: 98°F, SPO2: 94% in room air. He was evaluated for CoVid19. His RT-PCR for CoVid was found Negative. His ECG showed Old anterior wall MI. He was advised for a chest X-ray P/A view & Echo screening. His chest X-ray showed mild cardiomegaly without any other remarkable abnormality. His echo screening showed dilated LA & LV, with regional wall motion abnormality, severe LV systolic dysfunction (LVEF: 25-30%) & Mild MR. Then he got himself admitted for coronary evaluation by coronary angiogram.

His initial blood picture showed,
Hb: 15.3 gm/dl,
TLC: 12.19x10^9/L,
Platelet: 373x10^9/L.
Serum creatinine: 0.91 mg/dl,
urea: 30 mg/dl,
sodium: 138 mmol/L,
potassium: 4.3 mmol/L,
chloride: 103 mmol/L,
bicarbonate: 32 mmol/L.
His viral markers were found negative.

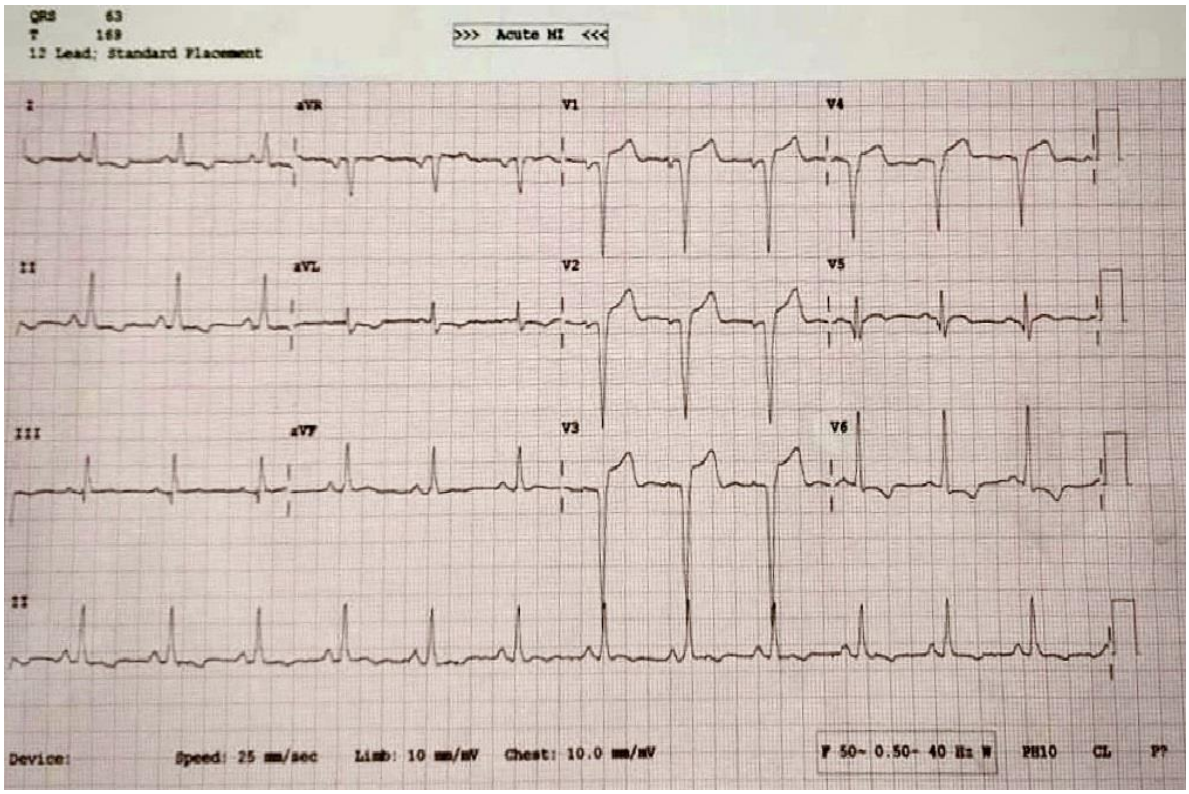

Figure 1: Electrocardiograph of the patient showing old anterior MI.

His coronary angiogram was done through the right radial approach under local anesthesia with a 5F Tiger diagnostic catheter & it showed: spontaneous spiral dissection from proximal to mid segment of type IV LAD involving second diagonal branch which was occluded from its proximal segment. In LCx there was a 95% stenosis in the mid-segment with a good size first obtuse marginal branch having 50% of ostial stenosis. His RCA was also totally occluded from its mid-segment which was a dominant vessel. So, coronary angiographically, a triple vessel coronary artery disease was made & considering lesion morphology, character & co-morbidities revascularization with CABG was advised.

Figure 2: Coronary angiogram of the patient showing spontaneous coronary artery dissection.

Case 2:

Mr. Y, 54 years normotensive, non-diabetic & known COPD patient was evaluated for a coronary angiogram. He has a history of positive TMT and was treated with anti-platelets, statin & other anti-ischaemic drugs.

In our center, he was at first clinically evaluated in the outpatient department. There he gave complaints of exertional angina (CCS class II/III). His vitals showed, Pulse: 80/ min, regular; BP: 130/70 mmHg without any postural drop, Heart: S1 & S2 were audible without any added sound,

Lungs: Clear without any added sound, Respiratory Rate: 17/ min, Temperature: 98°F, SPO2: 97% in room air. He was evaluated for CoVid19. His RT-PCR for CoVid was found Negative. His ECG was within the normal limit. He was advised for a chest X-ray P/A view, Echo screening & TMT. His chest X-ray was unremarkable. His echo screening showed no regional wall motion abnormality, good LV & RV systolic function (LVEF: 65%) & DRA (Grade-I). Then he got himself admitted for coronary evaluation by coronary angiogram.

His initial blood picture showed, Hb: 16.1 gm/dl, TLC: 10.88x10^9/L, Platelet: 287x10^9/L. Serum creatinine: 1.17 mg/dl, sodium: 138 mmol/L, potassium: 3.9 mmol/L, chloride: 99 mmol/L, bicarbonate: 30 mmol/L. His viral markers were found negative.

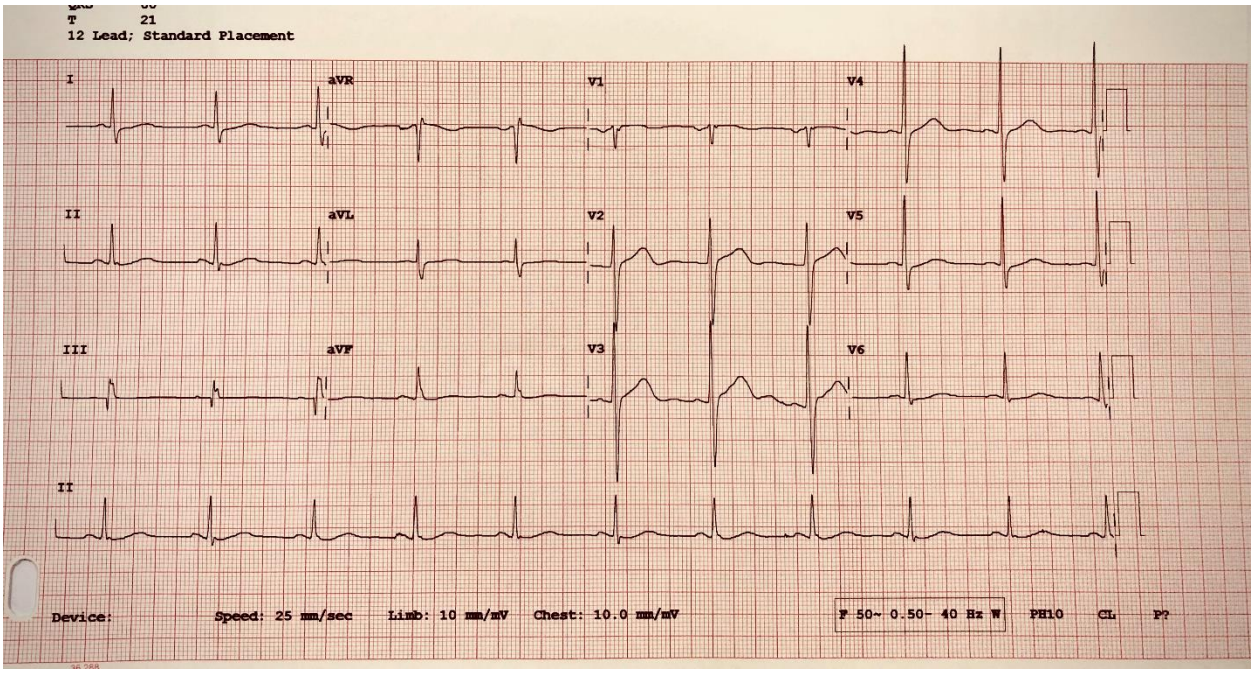

Figure 3: Electrocardiograph of the patient showing normal findings.

His coronary angiogram was done through the right radial approach under local anesthesia with a 5F Tiger diagnostic catheter & it showed: spontaneous spiral dissection from proximal to mid segment of dominant RCA. In LCx there was a mild narrowing in the proximal segment of the first obtuse marginal branch & 50% narrowing in the proximal segment of a good-sized second obtuse marginal branch. His LAD was a type IV vessel without any disease. So, coronary angiographically, a single vessel coronary artery disease was made & considering lesion morphology, character & co-morbidities revascularization with PCI was advised.

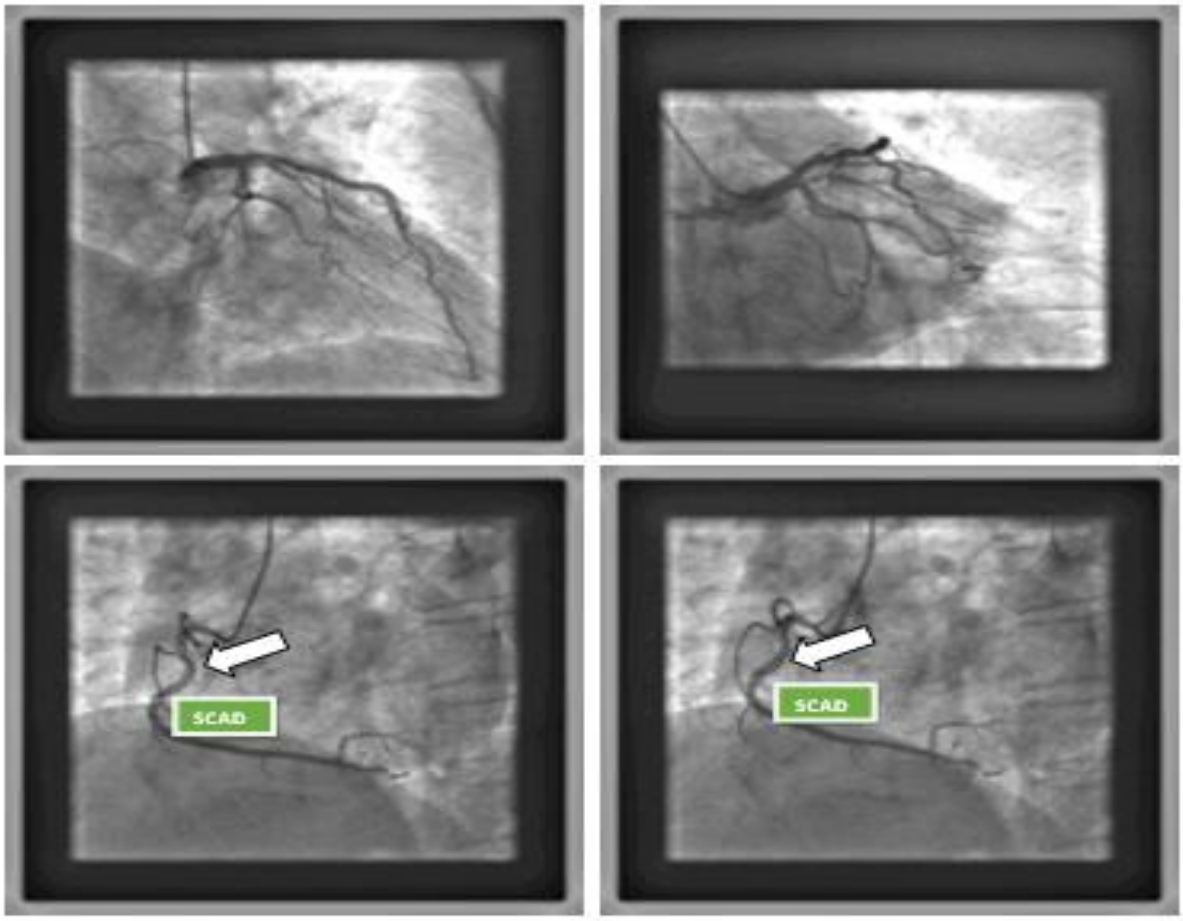

Figure 4: Coronary angiogram of the patient showing spontaneous coronary artery dissection.

Discussion

SCAD is defined as a non-traumatic and non-iatrogenic separation of the coronary arterial walls, creating a false lumen [1]. This separation can occur between the intima and media or between the media and adventitia, with intramural hematoma (IMH) formation within the arterial wall that compresses the arterial lumen, decreasing ante-grade blood flow and subsequent myocardial ischemia or infarction [1,2]. Spontaneous coronary artery dissection, also termed as dissecting aneurysm, intramural hemorrhage, or hematoma, is an uncommon clinical entity.[3-7]

The sign & symptoms that raise the possibility of SCAD are as follows:

Clinical features that raises suspicion of SCAD
Myocardial infarction in young women (especially age ≤50)
Absence of traditional cardiovascular risk factors
Little or no evidence of typical atherosclerotic lesions in coronary arteries
Peripartum state
History of fibromuscular dysplasia
History of relevant connective tissue disorder: Marfan's syndrome, Ehler Danlos syndrome, Cystic Medial Necrosis, Fibromuscular Dysplasia
History of relevant systemic inflammation: Systemic Lupus Erythematosus, Crohn's disease, Ulcerative colitis, Polyarteritis Nodosa, Sarcoidosis
Precipitating stress events, either emotional or physical (intensive exercise)

Table 1: Depicting the clinical features that raise suspicion of SCAD

It usually tends to involve the outer media and causes luminal occlusion by pushing the inner media against the opposing wall. Clotted blood filling the false lumen may simulate coronary thrombosis at the naked eye, masking the dissection. Thus, the accurate scenario of this clinical condition may be underestimated. A review of published reports showed that 69% of the cases were diagnosed at necropsy. [7] Among them 80% of the cases were women, and more than 25% of these were in the peripartum period. [8-22] Involvement of the right coronary artery seems to be more common in men, whereas left anterior descending coronary artery involvement is frequent in women. Coronary artery dissection affects young adults; in a series of cases that were diagnosed before death. According to De Maio et al [6], the mean age was 46 years in males and 38 years in females. The clinical presentation of this includes the entire spectrum of the acute coronary syndrome and is primarily related to the extent of the dissection and the vessel involved. Nonetheless, sudden death without pre- evidence of myocardial infarction is much more frequent. [23] Survival is possible if obstruction of the lumen is incomplete, or an uncomplicated myocardial infarction. [24-27] Spontaneous healing of a coronary dissection has also been shown to occur, on both clinical and histological evidence.[13,26] When secondary causes are excluded, [11, 15, 24, 28-36] the etiology of it remains uncertain. Among the risk factors, hypertension has rarely been reported. [11] Pregnancy or oral contraceptives induced arterial wall changes have been well documented [37] hormonal and hemodynamic factors also may contribute to the weakening of the

tunica media, thereby correlate well with a higher incidence of spontaneous coronary dissection in the puerperium[8-16,38,39] .A true pattern of cystic medial necrosis has been described in the involved coronary artery as the cause of spontaneous coronary artery dissection. [40]

In many published cases, a fairly diffuse adventitial and periadventitial inflammatory reaction consisting mainly of eosinophils was observed.[4,29,30] Because of the presence of these "periarteritis-like" adventitial change, it was proposed that the dissection is a result of the lytic action of protease released from eosinophils. [30] The in vivo diagnosis of spontaneous coronary artery dissection by selective angiography depends on the visualization of two lumina separated by a radiolucent intimal flap. [7] This procedure entails the risk of cardiac arrest by injection of the hypertonic medium into the false lumen, thus aggravating the dissection. Some cases may not be recognized if an intimal tear does not occur or if the true lumen is severely narrowed or if the false lumen is occluded by a clot.

The prognosis for these patients is poor. In a survey of 123 cases, Benham and Tillinghast found that 67% of the patients died and 33% survived, treated either surgically or medically. [41] Since sudden death is the most frequent clinical presentation, the clinician is left without the time of any potential therapeutic option in the majority of cases. Medical treatment may play a palliative role. Although the outcome for patients with spontaneous coronary dissection is considered grim, with a high mortality rate, aggressive surgical treatment in a recent series of 10 consecutive patients resulted in 100% survival. [7] Thayer et al [5] recommended coronary artery bypass grafting for all patients with spontaneous dissection, whereas De Maio et al [6] advocated this intervention only in cases of left main disease, three-vessel disease, few or recurrent ischemia.

Conclusion

SCAD is an infrequent condition that is under-diagnosed among patients presenting with ACS. Risk factors for SCAD are multifold, including young women, fibromuscular dysplasia, systemic inflammation, connective tissue disorders and pregnancy, and often compounded by precipitating stressors. The long-term outcome of patients who survived their initial SCAD presentation is good; however, recurrent events are frequent and these patients should be followed closely by cardiovascular specialists. Treatments typically entail conservative medical management for stable patients with ischemia resolution; however, revascularization with PCI or CABG may be necessary for a small proportion of patients.

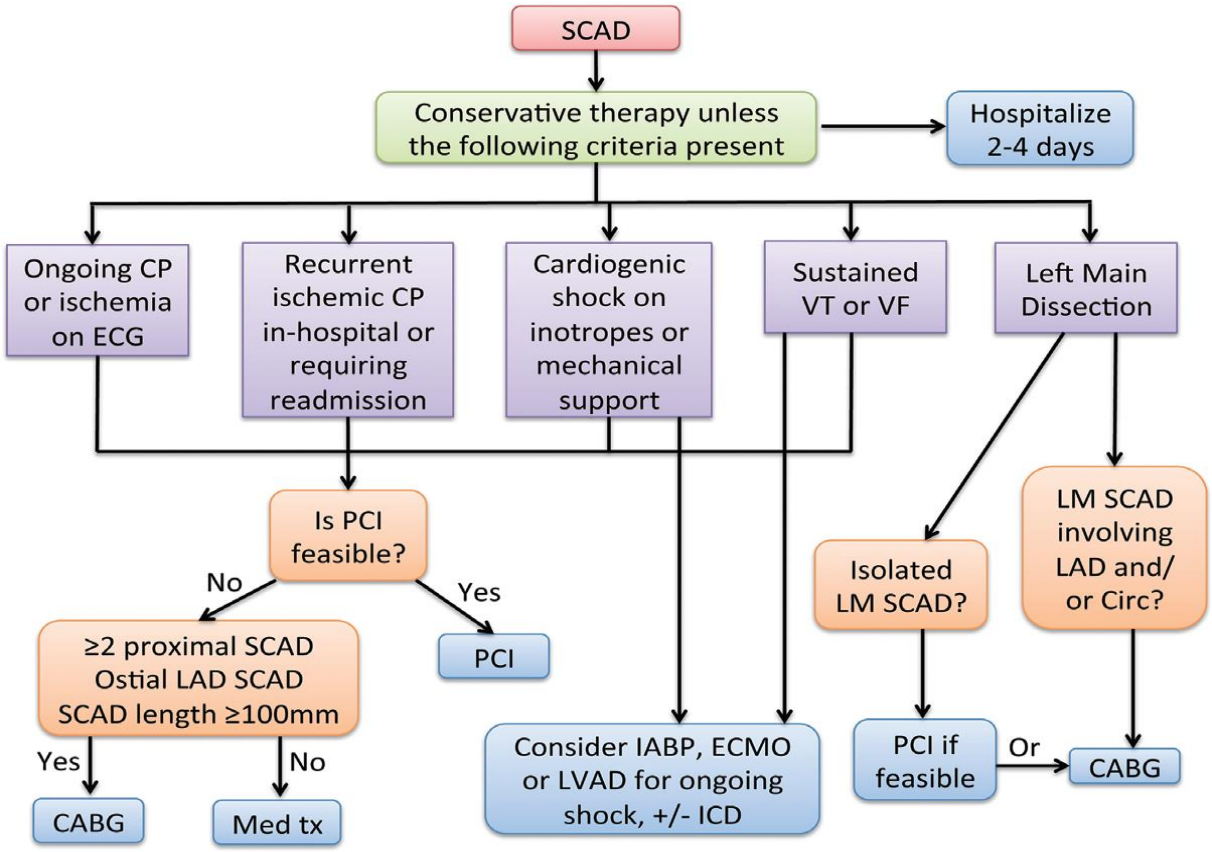

Figure 5: Management algorithm including revascularization for acute presentation of SCAD[42].

In conclusion, the possibility of a spontaneous coronary artery dissection should be considered in any young adult woman who presents with an unexpected circulatory collapse, myocardial ischemia, or infarction, without a previous history or risk factors, and not necessarily in the puerperium. Suspicion of the diagnosis may then lead to an emergency investigation of the coronary arteries, and surgical or interventional treatment. However, the high incidence of sudden death without premonitory symptoms casts doubt on the real possibility of prompt diagnosis and life-saving treatment in the majority of cases.

Disclosure

The research team did not receive any grant from any sources & declares no conflicts of interest.

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