Case Report

SCAD, as Rare Unique Cause of Non-ST Elevation Myocardial Infarction in Middle Aged Male

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Introduction

Spontaneous Coronary Artery Dissection (SCAD) is a rare but important clinical entity. It is an important cause of myocardial infarction as well as sudden cardiac death. Its etiopathogenesis is still unclear. The main mechanism postulated is spontaneous, non-traumatic disruption of coronary arterial wall layers without underlying atherosclerosis. This results in intramural hematoma that may compromise blood flow causing myocardial injury and even life-threatening arrhythmias. The final mechanism of this unique condition is due to the weakness of the wall structural integrity. Associated conditions according to the available literature are genetically predisposing conditions like collagen vascular connective tissue disease, hormonal therapy, and some immune-mediated conditions. **(**1**)** Published case series have established this condition is predominantly in young/middle-aged females where SCAD results from a non-traumatic separation of the coronary artery wall without underlying atherosclerosis. **(**1**)**

The proposed mechanism of myocardial injury is a coronary artery lumen compromise due to intramural hematoma or intimal disruption, compromising true lumen at dissection site. **(**2**)**

Earlier studies have implicated SCAD causing acute coronary syndrome in 0.1% to 4% of cases, but more recent studies show SCAD to cause MI in 22-43% of women <50 years. **(**3,4**)** Despite

progress, a clear understanding of SCAD remains illusionary. This hampers a good proportion of patients being properly diagnosed and treated.

Here we describe a unique case of SCAD in a 56 years old Asian male treated at our tertiary care private hospital, in Qatar. He was not having any underlying predisposing clinical conditions as mentioned above like collagen vascular disease or inherited genetic disease features. We decided to go for conservative treatment after coronary angiography, which showed no coronary arterial lumen compromise. Coronary angiography, was done as he had a subacute presentation of exertional and non-exertional chest pain of 4 days duration and troponin- I was elevated. We evaluated him as a case of the acute coronary syndrome as pre-investigation suggested, only to be surprised with SCAD findings confirmed on IVUS. He made a good progressive recovery with a conservative approach.

The clinical and biological background of the patient

A 56 years male patient. He presented to us at an outpatient clinic with recurrent chest tightness lasting 20 -30 minutes with sweating from the last 4 days even without exertion or effort; describes as tightness with severity 6-7 on a scale of 10. He also had breathless on exertion class 2 of a similar duration. Clinically, his vital signs were stable with BP of 130/80 mm Hg and a regular heart rate of 89/min. He appeared worried but showed no signs of cardiac decompensation.

Saturation was 99% at room air and temperature was 37.4 degrees Celsius. He had no radio-radial or radio-femoral delays and had no obvious sign of valvular heart disease. He was obese with a BMI of 35.

He had no marfanoid or marfans clinical features. Inquiring about the past, he had diabetes and hypertension for the last 2 yrs. He was on Telmisartan 40 mg once daily and diet control with exercise for Diabetes. He took regular aerobic exercise of 30 minutes, 5 days/week. He smoked 5-6 cigarettes/day for the last 5 years (1 pack-year). He however had severe stress for the past 2-3 months at his work and personal life. There was no history of recent trauma to the chest or any heavy exertion. He was advised to get an ECG, as the first investigation, which showed poor R wave progression, loss of septal R waves with left axis deviation and normal sinus rhythm. His troponin I was elevated, 0.288(normal – 0.040) ng/ml and hence he was admitted at CCU for further evaluation and treatment.

Investigations done

Serial ECG did not evolve poor R wave progression persisted as also loss of R waves in septal leads with left axis deviation and normal sinus rhythm. Echocardiographs done twice were essentially normal, done on admission and post coronary angiography and IVUS. It showed no RWMA or valvular pathologies. Left ventricular systolic function was preserved, only mild diastolic dysfunction was stage I impaired relaxation. The patient had also normal blood workup except mild triglyceride elevation of 2.5 mmol/dl (normal 1.95 mmol/dl). The thyroid screen was normal. D dimer was elevated mildly (1.5 mcg/ml), against a normal of 0.50 mcg/ml. HBA1C was 6.8 %.

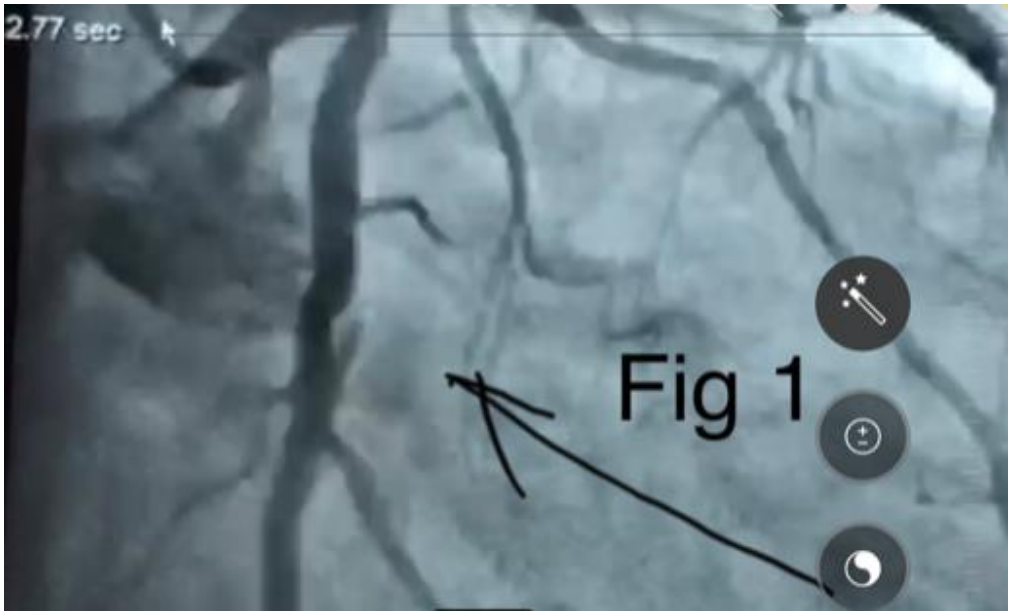

Figure 1

His coronary angiography showed (Figure 1):

CAG- Left main / Left anterior descending artery / Diagonals and ramus were normal.

LCX- was a large vessel with a mid-aneurysmal segment with SCAD and normal distal flow.

OM- OM 1/ OM2/OM3, were small and normal

RCA/ PDA / PLV-normal.

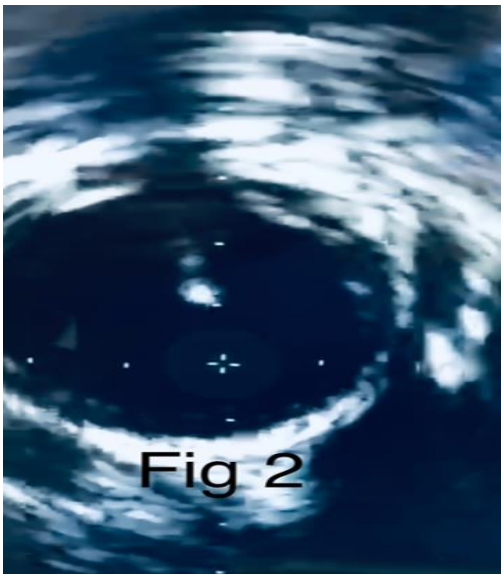

Figure-2

IVUS showed (Figure 2):

Mid-LCX intramural hematoma with healed dissection, the lumen was uncompromised, and the vessel did not show plaques.

CT-pulmonary angiography- done to rule out pulmonary embolism, as a patient after coronary angiography and appropriate treatment was still feeling exertional breathlessness and D-dimers were elevated; it showed pulmonary arteries were normal and thus ruled out pulmonary embolism.

CXR was normal.

Holter ECG showed 5-6 % PVC as bigeminy and trigeminal and couplets/triplets on the first day post angiography. (Holter ECG attached, to quantify arrhythmia burden, serial routine ECG done showed frequent mono-morphic PVC.

Repeat Holter after increased Metoprolol showed only isolated PVC, summing to 0.4%.

Treatment given

The patient has treated with Plavix loading 300 mg and Aspirin 300 mg before doing angiography. Injection Clexane 80 mg s/c stat given, as screening echocardiography was reassuring.

After coronary angiography, Injection Clexane was discontinued and the patient was started on Tablet Metoprolol XR 50 mg once daily, eventually made twice daily based on Holter ECG results. Crestor 10 MG was continued and tablet Fenofibrate 145 mg, daily added, as patient was very keen to start, on advice of relative for his only mildly elevated triglycerides. Sliding scale insulin

with sugar monitoring 6 hourly was replaced with Glucophage 48 hours after angiography. Tablet Telmisartan, discontinued, the original antihypertensive patient was on because it may increase shear stress and BP was well controlled on metoprolol.

Discussion

Spontaneous coronary artery dissection is an uncommon presentation for ACS seen mostly in younger women. There are a few reported cases in the medical literature of SCAD presenting with an association of stress cardiomyopathy. We present a case of SCAD in a middle-aged male associated with stress but had no feature of stress cardiomyopathy on echocardiography or coronary angiography, posting a significant management dilemma.

Based on angiographic characteristics conservative medical management is the mainstay of therapy in treating patients with SCAD who do not have features suggestive of invasive characteristics at coronary angiography. **(**3**)** A review of the literature suggests that the healing of SCAD is spontaneous. Chances of possible deterioration be kept in mind be anticipated with an observation of the recurrence of chest pain and arrhythmias.

For our patient, there was no recurrence of chest pain and the ventricular premature contractions nearly settled with optimized metoprolol dose. Intracoronary imaging with intravascular ultrasound was reassuring. No stent was deployed to LCX, as the lumen of culprit LCX was intact and invasive strategy clearly not supported here. Normal blood flow was maintained, prompting us to continue medical management. A second coronary angiography done at 6 weeks showed uncompromised findings of intact flow, supporting good healing.

The repeat angiography findings supported the resolution of the LCX SCAD, expected healing, thereby supporting our decision to pursue conservative management in the absence of flow limitations.

A bimodal approach to managing SCAD has been proposed, invasive with stent deployment versus conservative management. Some authors have suggested percutaneous coronary intervention (PCI) as a first-line treatment in the presence of ongoing ischemia and hemodynamic instability. **(**5**)** Nonetheless, PCI was found to be associated with significant complications including propagation of the dissection flap, intramural hematoma formation, and technical challenges limiting the placement of the wire into the true lumen. **(**1**)** There have been instances where coronary artery bypass surgery was done for the treatment of SCAD. **(**6**)**

Outcome

With an optimized dose of Metoprolol and other medications as mentioned above, the patient remained stable and pain-free after 3 months.

Future perspective

Management of recurrent SCAD continues to evolve and continues to be challenging, given the lack of specific guidelines or even expert consensus. Conservative management is a more supported modality of care for a first presentation. Managing recurrent SCAD is even more unclear. Further case series or multicentre coordinated registries are needed to evaluate outcomes in SCAD in long term and to formulate guidelines for management.

We treated the patient with optimal investigative strategy, and he responded to conservative management without stents. He was advised to have healthy lifestyle measures by stopping smoking and take care of his stress to protect from future recurrences. He is on regularly scheduled follow-ups in our cardiology clinic.

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