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CASE REPORT

Recurrent Spontaneous Coronary Artery Dissection: A Case of Triple Trouble

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Abstract

SCAD (Spontaneous Coronary Artery Dissection) is a rare disorder which rarely recurs. It is increasingly diagnosed as a cause for acute coronary syndrome (ACS) with limited insight into its pathophysiology and treatment. Lack of randomized trials and consensus guidelines make this a unique and challenging disease to manage. We describe a complex case of recurrent idiopathic SCAD with prior history of NSTE-ACS (Non-ST elevation Acute Coronary Syndrome) and discuss its management based on current clinical practices.

Keywords: Spontaneous coronary artery dissection, Ventricular fibrillation, Acute coronary syndrome, Fibromuscular dysplasia

1. Introduction

SCAD is a rare, life-threatening coronary disorder characterized by the separation of the coronary arterial wall with a risk of myocardial infarction.[1,2] It is a poorly understood disorder with limited etiological and pathological mechanisms knowledge. It is more prevalent among females and has known associations with diseases disrupting coronary microvasculature. Recent data have suggested a lower rate of recurrent SCAD.[9]

2. Case presentation

A 55-year-old female presented to the emergency department with three hours of acute, retrosternal, non-radiating chest pain with associated diaphoresis that was not relieved with rest. She denied significant physical exertion or emotional stress prior to onset of symptoms. On presentation she was hypertensive with a blood pressure of 153/93 mmHg, but otherwise afebrile, with a preserved heart rate (96 beats/minute) and saturation on room air. Her cardiovascular and pulmonary examinations were unremarkable. Electrocardiogram demonstrated a left anterior fascicular block with lateral ischemic changes (Fig. 1). Laboratory diagnostics included an unremarkable complete blood count, metabolic panel, and negative urine toxicology. High sensitivity troponin peaked at 4710 ng/L.

She was hospitalized 6 months prior for an ST-elevation myocardial infarction secondary to SCAD involving the mid to distal left anterior descending artery (LAD). Her clinical course was complicated by a ventricular fibrillation arrest due to distal propagation of the dissection and complete occlusion of the LAD (Fig. 2). She was managed conservatively due to lack of percutaneous coronary intervention (PCI) or surgical revascularization options with placement of an intra-aortic balloon pump for hemodynamic recovery and ultimately ICD (implantable cardiac defibrillator) implantation was performed 3 months later. She had a negative

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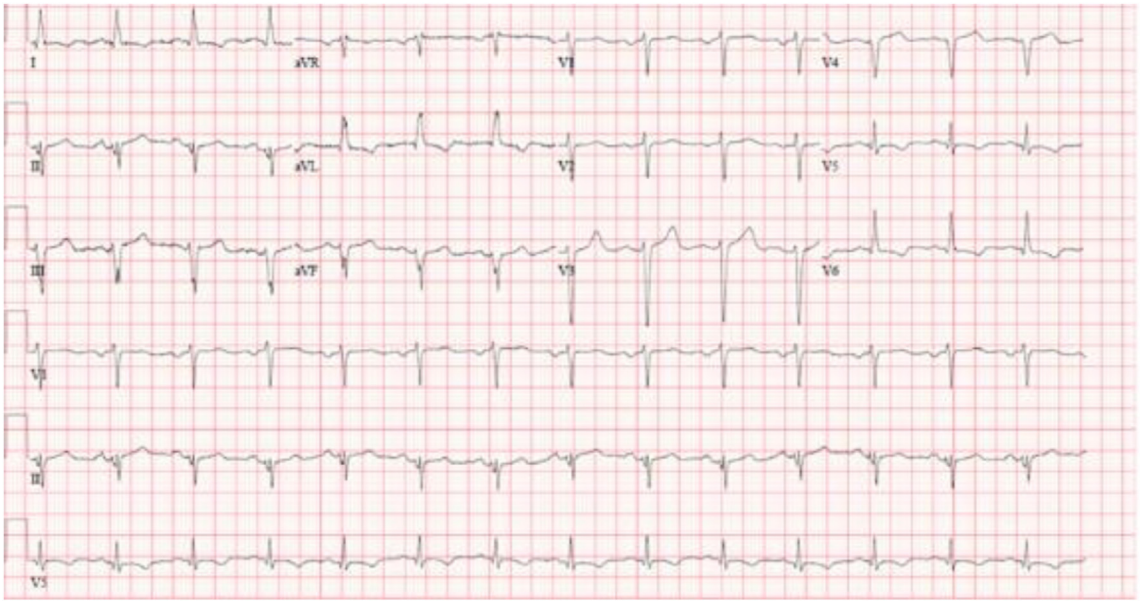

Fig. 1. Initial electrocardiogram on presentation. Electrocardiogram showed left anterior fascicular block with T wave inversion in the lateral leads.

diagnostic workup for fibromuscular dysplasia, cardiac sarcoidosis, and autoimmune vasculitis. She has a history of cardiomyopathy first diagnosed 9 years ago following an episode of NSTE-ACS and her coronary angiogram showed minimal coronary artery disease. Subsequent non-ischemic workup for her cardiomyopathy was negative. She had triple negative infiltrating ductal carcinoma of the right breast 13 years ago managed with mastectomy (negative margins) and 6 cycles of doxorubicin, cyclophosphamide, and docetaxel. Personal history was negative for multiple pregnancies, migraine headaches, or hormonal replacement therapy. Additional medical history is notable for hypertension, hyperlipidemia, and pre-diabetes. She is a lifelong abstainer of tobacco and illicit substances with infrequent alcohol use. Family history is significant for breast cancer in a paternal aunt and hypertension in mother. Medications at admission included amiodarone, aspirin, rivaroxaban, carvedilol, dapagliflozin, sacubitril-valsartan and spironolactone; she reported good compliance.

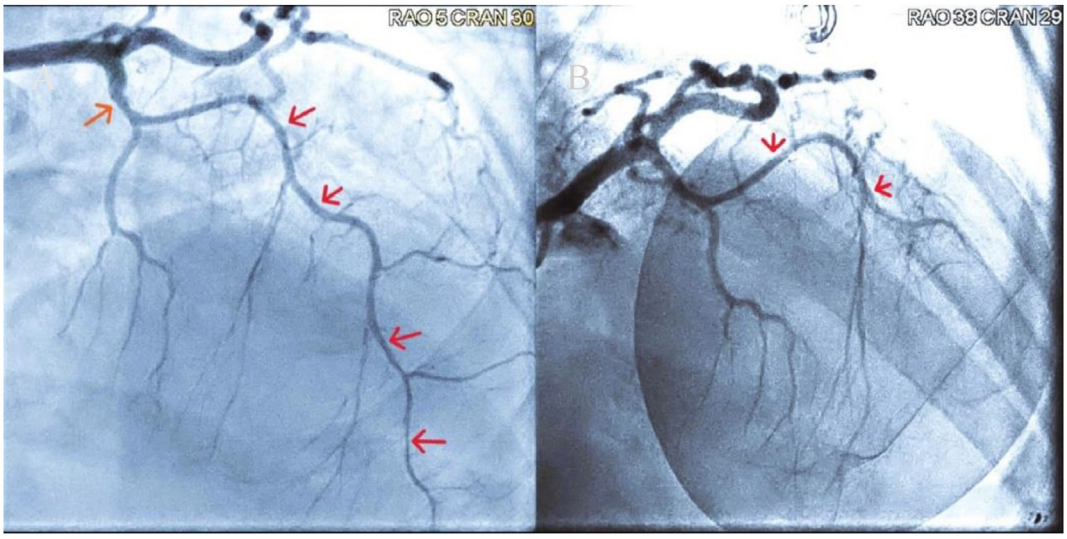

Fig. 2. Coronary angiogram 6 months prior to presentation. Image A shows coronary angiogram showing LAD dissection with proximal entry point (orange arrow) with distal to apical extension (red arrows). Image B shows complete LAD obstruction with no distal collaterals (red arrows) 24 h post initial presentation.

During current presentation, she was ruled in for NSTE-ACS with ongoing chest pain and a TIMI (Thrombolysis in MI) risk score of 5. Emergency cardiac catheterization showed a Type II SCAD involving the large, bifurcating OM1 (obtuse marginal) branch with distal TIMI 0 flow, and a prior healed LAD dissection with distal TIMI III flow (Fig. 3). Conservative management was instituted for recurrent SCAD given the diffuse coronary dissection involving multiple branches and not amenable to PCI, lack of hemodynamic or electrical instability, and no ongoing chest pain. OCT (Optical Coherence Tomography) and/or IVUS (Intravascular Ultrasonography) imaging was deferred due to risk of dissection propagation and iatrogenic complications. She was managed with antiplatelet agents (aspirin, clopidogrel) and her home dose rivaroxaban for a planned one-year period with subsequent de-escalation to aspirin and rivaroxaban.

A transthoracic echo (TTE) showed normal left ventricular (LV) cavity size with an ejection fraction (EF) of 35%–40% with apical hypokinesia (unchanged from a recent study 2 weeks ago). She was reinitiated on GDMT (Guideline-directed medical therapy) with carvedilol, dapagliflozin, valsartan/sacubitril and spironolactone. She remained symptom-free and was discharged in a hemodynamically stable condition. Our patient was doing well at one month follow up with no chest pain and has been advised to complete cardiac rehabilitation with planned further re-imaging or coronary angiogram as indicated.

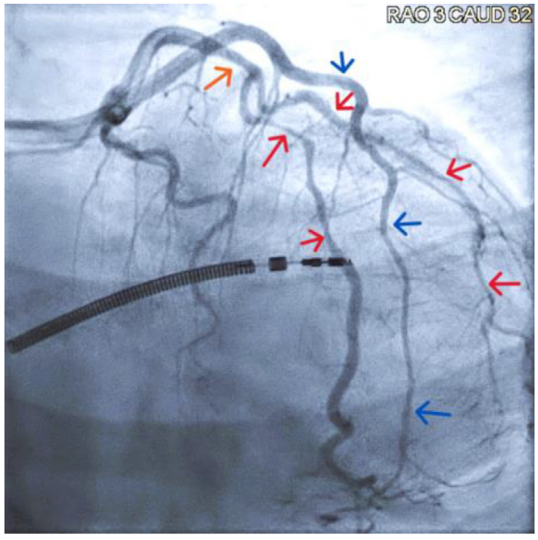

Fig. 3. Coronary angiogram during current presentation. Coronary angiogram demonstrating LCx (orange arrow) with bifurcating OM1 showing a Type II SCAD (red arrows) and healed LAD with distal atherosclerotic disease (blue arrows).

3. Discussion and conclusion

SCAD is a non-traumatic, non-atherosclerotic separation of the coronary artery tunica intima and media, leading to compression of the true lumen.[1] SCAD is a cause of ACS (most common symptom being chest pain) in up to 4% of cases, average age of presentation is 53 years and females comprise 89% cases. It may present as ventricular arrhythmias, cardiogenic shock, and even sudden cardiac death.[1,2] The etiopathogenesis involves the "outside-in" hypothesis (de novo hematoma within the tunica media) and the "inside-out" hypothesis (luminal flap development followed by sub-intimal blood collection). OCT (Optical Coherence Tomography) studies favor the former and hematomas may arise from the vasa vasorum undergoing shear stress.[1,3] The role of emotional and/or physical stressors in the setting of predisposing factors like inherited arteriopathies and connective tissue disorders, change in sex hormones, migraine, pregnancy, multiparity, and infertility treatments has been shown to be a contributing factor. Fibromuscular dysplasia (FMD) is seen in >50% cases and head-to-toe angiographic screening is recommended.[1,2] Studies have also found no increased prevalence of autoimmune disorders in SCAD patients.[9]

The LAD and its branches are involved in >50% cases, followed by the left circumflex, right coronary, and the left main arteries.[2] Angiographically, SCAD is classified as: Type 1 showing multiple radiolucent vessel lumens; Type 2 showing long segment narrowing (>20 mm); and Type 3 showing a focal narrowing of the vessel lumen (mimicking atherosclerotic process). Type 2 SCAD is the most common type and seen in 70% of cases, as seen in our case.[1,4] Index SCAD involves a single coronary artery in 87% cases. Multivessel index involvement has been reported in 9%–23% of cases with non-contiguous disease in 5%–10% of cases.[2,5] Recurrence is defined as new dissection, excluding extension of a prior hematoma, presenting as new acute coronary syndrome with enzyme elevation and new data suggest 2.4% recurrence in 1.9% cases after 3 years and this is attributed to increased diagnosis of milder forms of SCAD.[9] Studies show that uncontrolled hypertension increases the recurrence risk and beta blocker use lowers the risk, suggesting a role for reducing sheer arterial stress in prevention.[1]

It is interesting to consider that our patient received chemotherapy for breast cancer and case reports of SCAD occurring acutely following chemotherapy have been published.[10,11] Chemotherapeutic agents of multiple classes are known to induce oxidative stress and cause direct endothelial damage resulting in an increased risk of heart failure and CAD (Coronary artery disease) in the long term.[12] Chemotherapy-related ACS mechanisms include diffuse coronary vasospasm due to hyperreactivity, accelerated fibrous intimal proliferation and prothrombotic states, leading to plaque formation.[13] Vasospasm has been considered a precipitating event for SCAD in patients with prolactinoma treated with cabergoline.[14] However, studies have failed to show any association between positive provocative coronary vasospastic response and SCAD.[15]

Spontaneous healing occurs in 95% of cases of SCAD after >30 days, suggesting hematoma resorption and healing of the intimal flap in a time-dependent manner.[4] Most cases of recurrent ischemia or occlusion occur within one day and are rare after 6 days. Patients should be observed in hospital for a few days following the initial event. MACE (Major adverse cardiovascular events) events are reported in 14% at 3 years follow-up.[6,9] Studies show higher rates of catheterization complications, including iatrogenic dissections (3.4% in SCAD vs <0.2% for non-SCAD patients), high rates of PCI failure in up to 53% cases, and a higher risk of vessel occlusion in revascularization (0.46%) vs conservative (0.16%) management.[7,8]

Revascularization is reserved for proximal vessel occlusions, unstable rhythms, hemodynamic compromise, and failure of initial conservative management. CABG (Coronary artery bypass grafting) is performed for failed or high-risk PCI, such as left main dissections with ongoing ischemia, although graft failure rates are high.[1] Recent data suggests the uncertain role of Antiplatelet agents or beta-blockers in reducing 3-year MACE risk.[9] A lack of randomized trials limits providers to using established guidelines for atherosclerotic ACS and heart failure, in the management of SCAD. Anti-platelets are continued if PCI is performed, although in conservatively managed patients, decisions should be based on comorbidities and bleeding risk. Hypertension management and use of beta blockers, if tolerated, may confer a lower risk of recurrence. Routine lipid lowering therapy is not recommended due to differing pathophysiology from atherosclerotic disease unless other indications exist.[1]

In conclusion, SCAD remains an under-investigated phenomenon, with further research needed to fully define its natural course, risk factors, and adequate management strategies. However, recent data suggests low recurrence rates in the setting of increased recognition of index events by intervention cardiologists and is reassuring for patients. There is a definite need for further clinical trials to formulate consensus guidelines for the management of SCAD.

Disclaimer

The current manuscript is not under review in any other publications and has not been presented at a conference or a meeting.

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Conflict of interest

No potential conflicts of interest exist for the current manuscript.

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