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Case Report

Spontaneous coronary artery dissection: a rare diagnosis in a postmenopausal woman

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We report a case of a 50-year-old ex-smoker, postmenopausal woman presenting with an acute myocardial infarction caused by a spontaneous coronary artery dissection of a long 'wrap-around' left anterior descending coronary artery. After the diagnostic coronary angiography, she was treated medically with subsequent improvement.

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute myocardial infarction. SCAD typically occurs more frequently in patients with underlying atherosclerosis and women in the peripartum postpartum period. So far, there is not enough data to guide the management which is often individualized depending on the hemodynamics and the anatomy.

CASE REPORT

A 50-year-old African-American postmenopausal woman presented to the hospital with a 1-h history of mild chest discomfort associated with nausea and diaphoresis. An electrocardiogram showed 2-mm inferoapical ST segment elevations (Fig. 1). The Emergency Medical Service was called and she received aspirin 325 mg. Upon presentation to our emergency department, she had improved symptoms. She was initially in mild distress and pale in appearance with a blood pressure of 179/103 mmHg and a respiratory rate of 12/min. Her medical history was notable only for depression. However, she had no prior history of hypertension or hyperlipidemia. The patient had recently stopped smoking, drank wine occasionally and denied any illicit drug use. She only took bupropion for depression and smoking cessation. Her first Troponin I was 0.073 ng/ml (normal range: 0.0–0.034 ng/ml). She was taken for urgent coronary angiography, which revealed a large 'wrap-around' left anterior descending artery (LAD) with a long dissection extending from its mid to distal vessel (Fig. 2) without any atherosclerotic changes in the right or left coronary arteries. The patient remained hemodynamically stable without worsening of her symptoms, laboratory or electrocardiogram findings. The decision was made to manage conservatively with close monitoring. She was started on aspirin, unfractionated heparin, atorvastatin, metoprolol tartrate, lisinopril and intravenous nitroglycerin to help control her blood pressure.

During the patient's 6-day hospitalization, she remained asymptomatic. Troponin I peaked at 7.6 ng/ml and rheumatologic work-up including anti-citrullinated protein antibodies, antinuclear antibody and anti-neutrophil cytoplasmic antibodies was negative.

An electrocardiogram prior to discharge revealed persistent ST elevation in the previously involved leads (Fig. 3), whereas her echocardiogram showed akinesis in the apical region with an ejection fraction of 61%. On outpatient follow-up, the patient was clinically asymptomatic and no repeat angiography was done.

DISCUSSION

SCAD results from a separation of the coronary artery intimal and medial wall followed by hemorrhage and subsequent dissection. In contrast to our case, SCAD occurs more frequently in young women under 50 years of age, especially in the peripartum period, and those who have associated atherosclerotic risk factors [1, 2]. SCAD represents 0.1–4% of all causes of acute coronary syndrome (ACS), whereas in women under 50

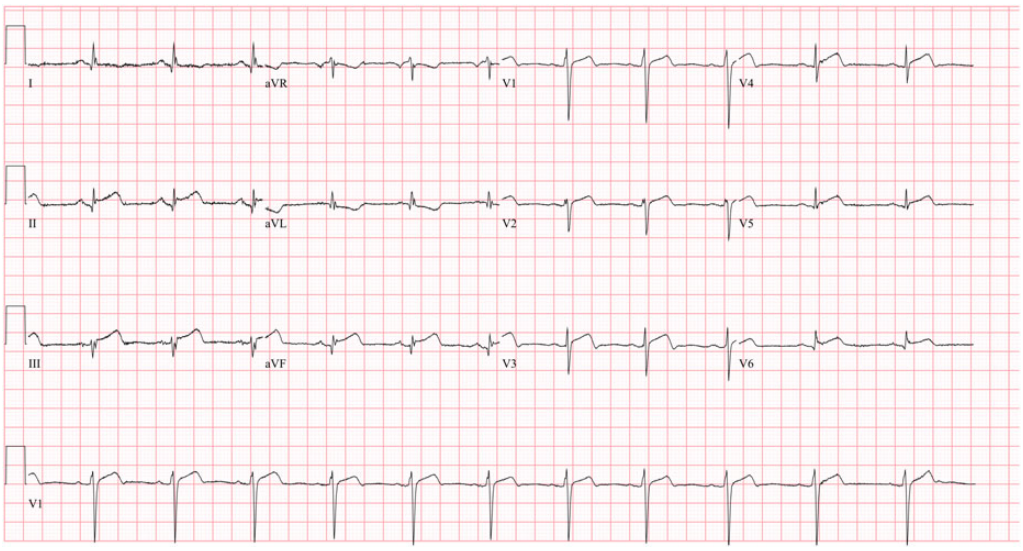

Figure 1: Electrocardiogram on Day 1.

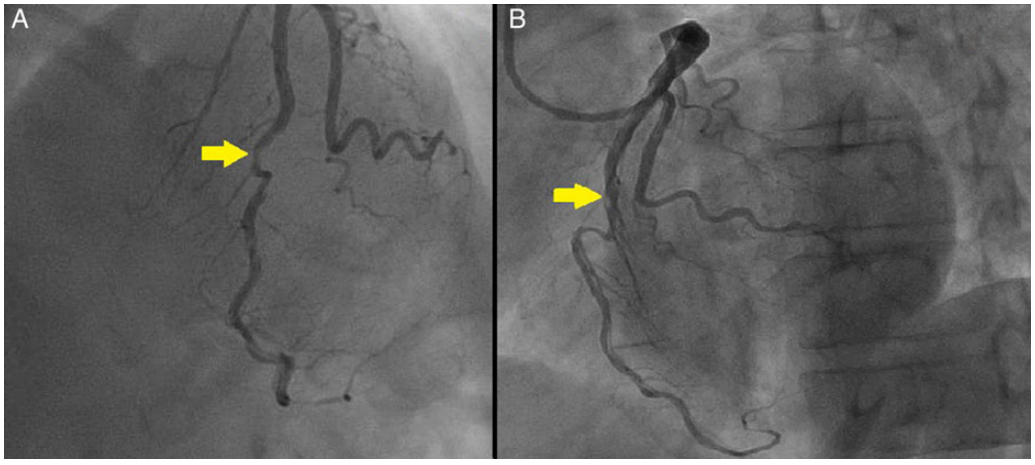

Figure 2: (**A** and **B**) Left anterior oblique cine angiographic view showing a large 'wrap-around' LAD with a long dissection extending from its mid (arrow) to distal segment.

years old with ACS, the prevalence of SCAD increases to 8.7–10.8% [3]. The most frequently involved vessel in SCAD is typically the LAD (60%) [3, 4]. SCAD is usually seen in three settings: patients with underlying coronary atherosclerotic disease, women in the third trimester of pregnancy and those in the early postpartum period. There are some associated conditions such as Marfan syndrome, cystic medial necrosis, sarcoidosis, oral contraceptive therapy and cocaine use [2].

The clinical presentation of SCAD is highly variable and depends on its location, extent and severity. The clinical spectrum includes unstable angina, acute myocardial infarction, heart failure, cardiogenic shock, cardiac tamponade, ventricular arrhythmias and sudden cardiac death. Rarely, it can be asymptomatic and an incidental finding on coronary angiography [5].

The pathogenesis of SCAD remains uncertain. Two major mechanisms have been proposed: the first is the result of an intramural hematoma in the medial layer of the arterial wall creating a false lumen. The expansion of the false lumen by accumulationof the hematoma leads to compression of the true lumen, which results in myocardial ischemia. The second, more frequent, mechanism originates from an intimal or medial tear, which is often complicated by a superimposed thrombus [4].

In our patient, use of bupropion could have contributed to the dissection. However, it was difficult to prove a temporal relationship between the time of starting this medicine and the dissection since the patient was on bupropion intermittently.

The gold standard for SCAD detection remains coronary angiography [2, 4]. Angiography of SCAD with an intimal tear typically shows two lumens separated by a radiolucent flap. However, in cases without an intimal tear, angiograms can be silent, non-diagnostic or misinterpreted as showing atherosclerosis. A high index of suspicion of SCAD in such cases may warrant intravascular ultrasound or optical coherence tomography [2, 4, 6].

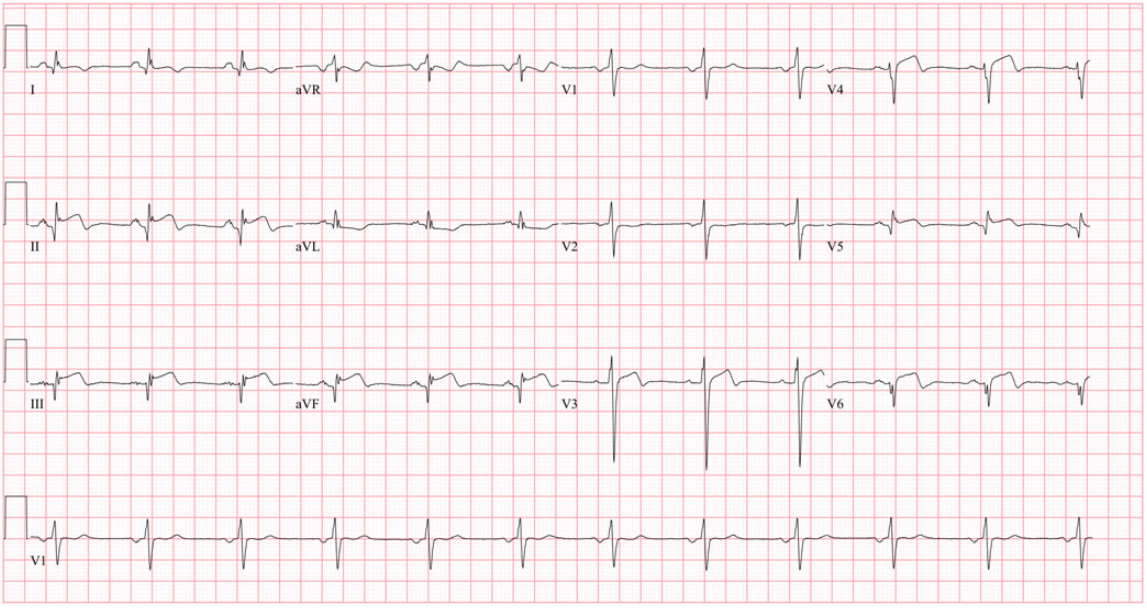

Figure 3: Electrocardiogram on Day 6.

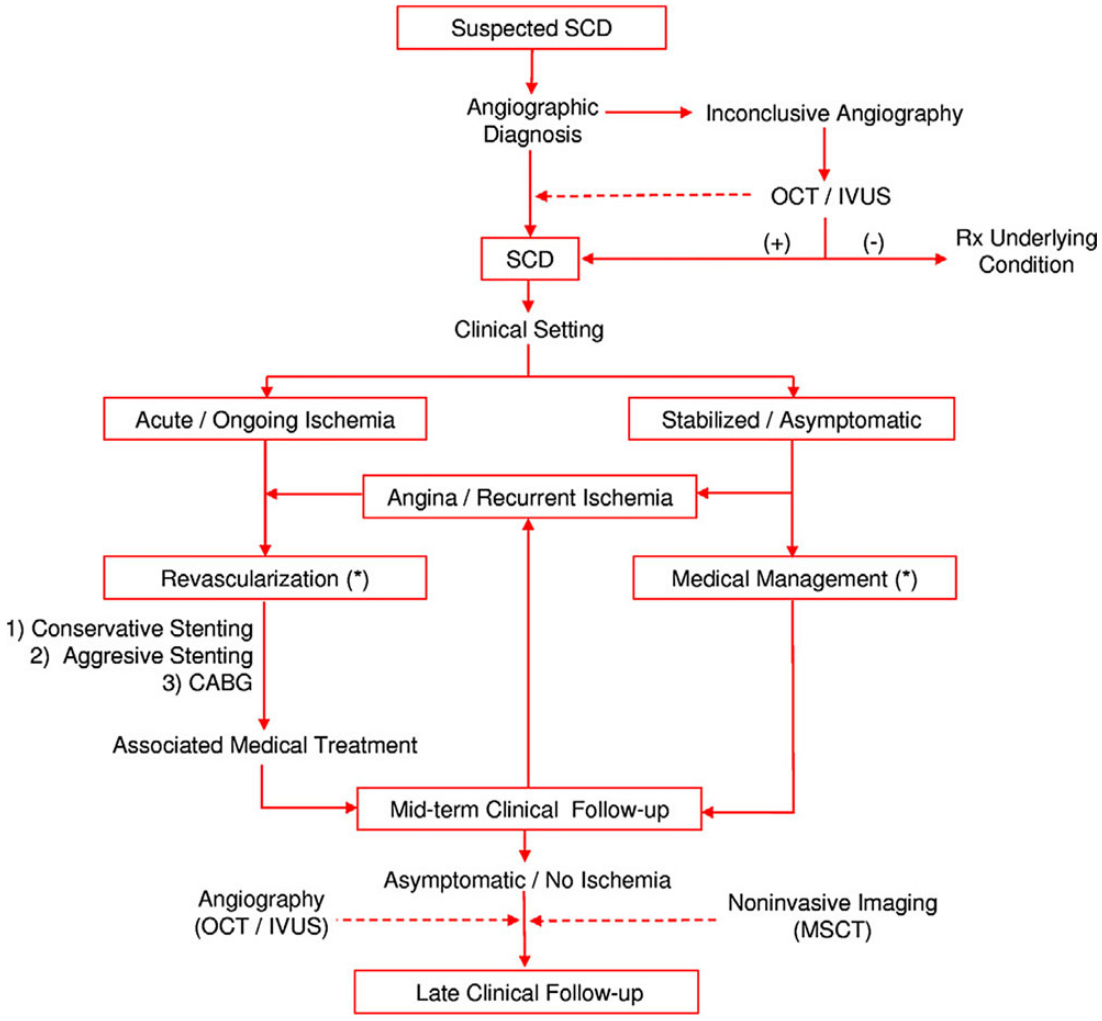

Figure 4: Algorithm for management of SCAD. Reprinted from [6], with permission from Elsevier.

The optimal treatment strategy for acute SCAD remains uncertain (Fig. 4). However, conservative management, defined as selecting revascularization only for patients with ongoing chest pain, ischemia, ST elevation or hemodynamic instability or recurrent ischemia, is associated with an excellent long-term prognosis [6, 7]. For patients presenting with an ACS, if the suspicion for coronary artery dissection is high, fibrinolytics should be avoided as they carry the risk of increasing flow into the false lumen and propagating the dissection [2, 4].

Long-term prognosis in patients who survive their initial SCAD is good, with 95% 2-year survival. Complete disappearance of the dissection image at follow-up is reported in patients with successful conservative management and those with residual (uncovered) dissections after stenting [4, 6].

CONFLICT OF INTEREST STATEMENT

None declared.

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