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Chest pain during a daily run unfolds an unfortunate diagnosis: A case report and review of the literature on spontaneous coronary artery dissection

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Abstract

Spontaneous coronary artery dissection (SCAD) is an uncommon but important cause of acute coronary syndrome. This condition is not linked to typical atherosclerotic risk factors, such as tobacco use, high blood pressure, diabetes, and hyperlipidemia. The majority of patients with SCAD experience chest pain and have some form of acute coronary syndrome with acute ST-elevation myocardial infarction (STEMI) being the most common. Early diagnosis is of utmost importance, as it can be fatal if management is delayed. Medical management is preferred over percutaneous intervention for hemodynamically stable cases. A case is presented of a 51-year-old female patient with no significant comorbidities who presented with exertional chest pain and STEMI, which unfolded a diagnosis of SCAD.

Keywords: chest pain, SCAD, spontaneous coronary artery dissection, coronary angiogram

Introduction

Spontaneous coronary artery dissection (SCAD) is defined as a dissection/tear of a coronary artery that is not related to atherosclerosis, trauma, or iatrogenic causes [1,2]. It is a rare cause of acute coronary syndrome (ACS) and commonly occurs in young healthy women aged 40-50 [3,4]. In SCAD, ACS is caused by blockages in the coronary arteries resulting from either intramural hematoma or intimal disruption, as opposed to atherosclerotic plaque [5]. Due to the frequent utilization of coronary angiography, there has been increased recognition of SCAD in patients presenting with ACS, with an overall incidence ranging from 0.28% to 1.1% [6]. Patients with SCAD experience chest pain and present with some form of ACS, with STEMI being the most common presentation. Coronary angiography is often the diagnostic modality used for SCAD, although intravascular ultrasound or coronary tomography has been used in rare conditions [5]. Management of SCAD is controversial and depends on the patient's characteristics and presentation. Most of the time, SCAD is managed medically without immediate revascularization. Nevertheless, percutaneous intervention may be required if the patient is hemodynamically unstable or has recurrent chest pain.

We report a case of a middle-aged healthy woman who presented with acute ST-segment elevation myocardial infarction (STEMI) and was found to have SCAD.

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Case presentation

A 51-year-old Caucasian healthy female presented to the emergency department with exertional substernal chest pain. She reported that the pain started when she was jogging. She described the experience as a throbbing pain in her mid-chest, non-radiating, and rated it ten out of ten in intensity, which slightly improved with rest. It is associated with lightheadedness and dizziness. She denied any chest pain before the episode. There was no reported history of hormonal contraceptive use, diabetes mellitus, hypertension, dyslipidemia, smoking, or hypothyroidism. Both parents had coronary artery disease at the age of 60 but there was no family history of SCAD, fibromuscular dysplasia (FMD), autoimmune disease, or malignancy.

The patient experienced moderate distress owing to chest pain. Her blood pressure was 140/90 mmHg in the right and 136/86 mmHg in the left arm. She had a normal lung and cardiac examination. An initial electrocardiogram (ECG) showed ST-segment elevation in the inferior leads, as shown in Figure 1. The initial troponin T level was 0.17 ng/ml (normal <0.03 ng/ml). The patient was quickly administered a loading dose of aspirin 325 mg, ticagrelor 180 mg, heparin 4000 U, and sublingual nitrate. Given the evidence of inferior wall STEMI and refractory chest pain, emergent coronary angiography was done, which revealed SCAD with 99% luminal stenosis in the mid-right coronary artery (RCA), as shown in Figure 2, with normal flow in the left anterior descending and left circumflex arteries. The patient had a successful stent placement on her mid-RCA with a resumption of TIMI-3 flow (Thrombolysis in Myocardial Infarction-3) (Figure 3). There was no ST-segment elevation on the EKG that was performed after cardiac catheterization (Figure 4).

The complete blood count, complete metabolic panel (CMP), lipid panel, thyroid function test, and glycated hemoglobin (HbA1C) levels were unremarkable. The urine pregnancy test result was negative. The laboratory work is shown in Table 1. Transthoracic echocardiography (TTE) revealed a left ventricular ejection fraction of 60% with normal left ventricular wall motion and normal valves. Computed tomography angiography (CTA) of the head and neck revealed subtle areas of bead appearance in the mid to distal cervical internal carotid artery, suggestive of FMD (Figure 5). Abdomen/Pelvis CTA showed superior left main renal artery with possible slight beading of the vessel, which was suspicious for FMD without frank aneurysm or stenosis. No other abnormalities were observed in the visceral vessels. Serologic tests were also performed to rule out secondary causes of SCAD. These included tests for rheumatoid factors, cyclic citrullinated C peptide antibody, antinuclear antibody screening, anti-double-stranded DNA antibody, and U1-ribonucleoprotein antibody, which were all negative.

The patient was discharged with aspirin, ticagrelor, atorvastatin, metoprolol succinate, and nitroglycerin. It was recommended that she follow up with a primary care physician, cardiologist, and cardiac rehabilitation.

After hospitalization, the patient had one admission and two emergency visits within the next two months. She presented with chest tightness associated with shortness of breath and palpitations. EKG and troponin levels were unremarkable during each visit. A cardiology team was consulted during each visit. Her symptoms resolved after extensive counseling and treatment for her anxiety. The patient has been doing well for the last six months and has regularly followed up with cardiology examinations.

Figure 1: ECG on admission showing ST-segment elevations in lead II, III, and aVF.

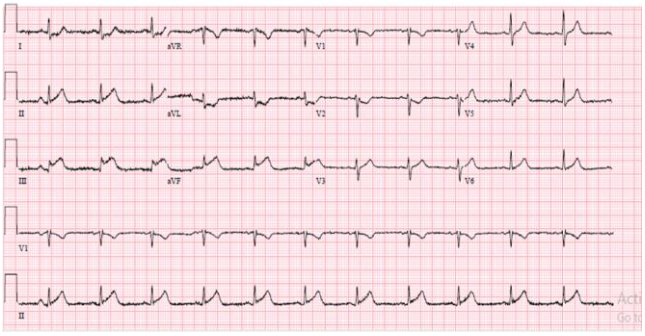

Figure 2: Coronary angiogram showing SCAD in mid-RCA

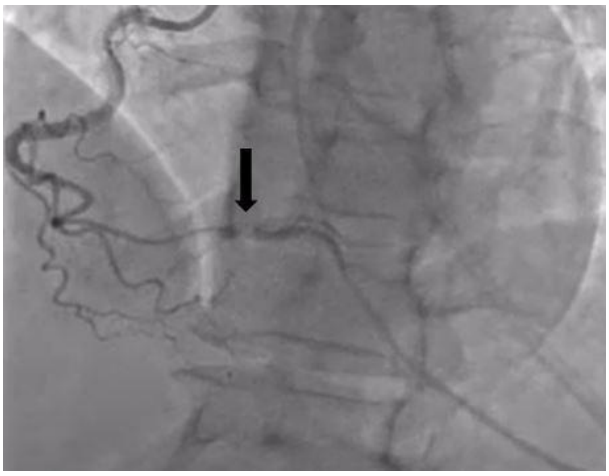

Figure 3: Coronary angiogram showing normal flow in the stenotic vessels after placement of stent

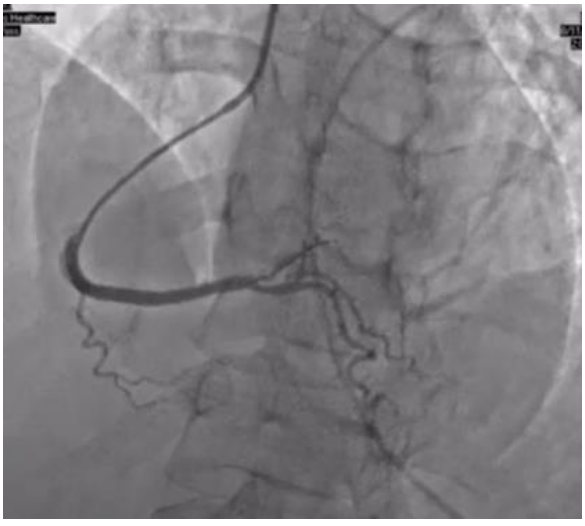

Figure 4: Repeat EKG after successful stent placement in mid-RCA showed no more ST-segment elevations in inferior leads.

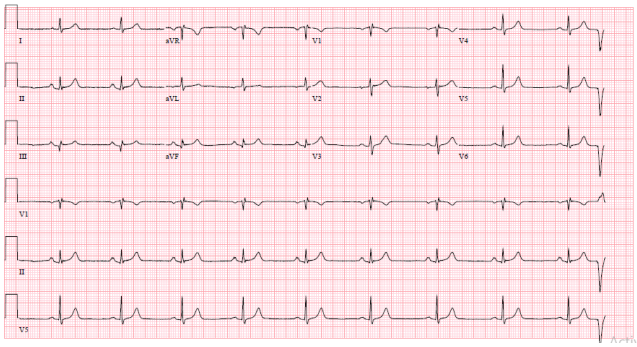

Figure 5: CTA of head and neck showing subtle areas of bead appearance of mid to distal cervical ICA.

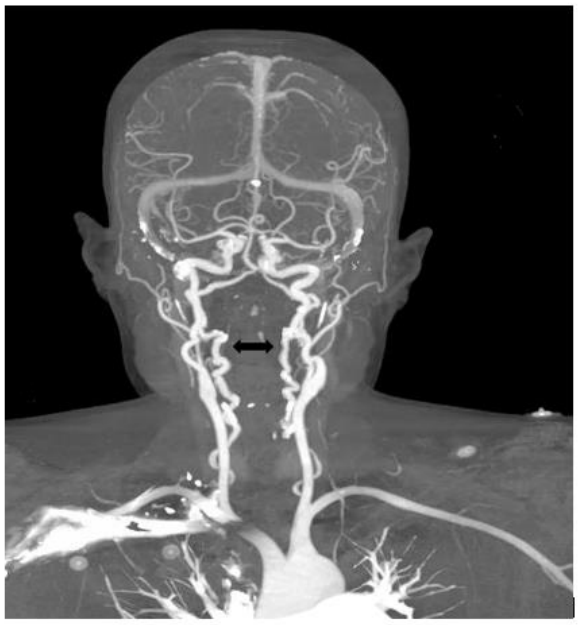

Table 1: Lab values along with their reference range

Labs	Values	Normal references values
WBC	5.6 K/µL	4-11 K/µL
Hemoglobin	12.5 g/dl	12-15 g/dl
Platelets	169 K/µL	150-400 K/µL
Sodium	140 mEq/L	135-145 mEq/L
Potassium	3.9 mEq/L	3.5-5 mEq/L
Blood Urea Nitrogen	16 mg/dl	8-21 mg/dl
Creatinine	0.9 mg/dl	0.6-1.2 mg/dl
HbA1C	4.7%	3.6-5.6 %
LDL	103 mg/dl	<100 mg/dl
HDL	66 mg/dl	>50 mg/dl
Total Cholesterol	179 mg/dl	<200 mg/dl
Triglycerides	31 mg/dl	<150 mg/dl

K/µL: thousand/microliter, g/dl: gram/deciliter, mEq/L: milliequivalent/liter, mg/dl: milligram/deciliter

Discussion

The coronary arteries, which supply blood to the heart muscles, have three layers: tunica intima, tunica media, and tunica adventitia [7]. Coronary artery dissection occurs when there is a separation between any of these adjacent layers. The exact etiopathogenesis of SCAD is still not fully understood. Further research is needed to definitively determine the primary initiating event in the development of SCAD. Two hypotheses have explained the pathophysiological process of coronary artery dissection. According to the “inside-out” hypothesis, an endothelial intimal disruption or "flap" allows blood to enter the subintimal space from the true lumen. This leads to the formation of an intramural hematoma that dissects the arterial wall layers. In the “outside-in” hypothesis, the hematoma arises *de novo* in the media, possibly from disruption of traversing microvessels within the arterial wall. This then leads to dissection of the layers of the arterial wall [8].

Coronary artery dissection can result from chest trauma, coronary angiography, or extension of aortic dissection. SCAD is the dissection of the coronary arteries without any obvious etiology, as listed above [1,2]. SCAD leads to the blockage of the coronary artery due to the narrowing of its lumen, which can be caused by either a dissection flap or the spread of an intramural hematoma [9]. SCAD was first reported in 1931 in the autopsy of a 42-year-old woman [10]. According to angiographic evaluations, the incidence of SCAD is approximated to be between 0.28% and 1.1%. [6]. Literature review shows that almost 90% of patients with SCAD are young, healthy females between the ages of 47 and 53 [11].

SCAD has been observed to occur before or during a woman's menstrual cycle. This has been reported in women who are using hormonal contraceptives, postmenopausal hormone therapy, or receiving infertility treatment due to a history of infertility. In addition, pregnancy-associated SCAD (P-SCAD) can occur at any stage during pregnancy or up to 12 weeks postpartum, but the majority of cases occur in the early postpartum, usually in the first week [8].

SCAD has been associated with triggers, such as emotional and physical stress including the Valsalva maneuver, vomiting, retching, and pregnancy [12]. Several instances have been recorded in case reports linking SCAD with inflammatory and autoimmune conditions, such as systemic lupus erythematosus, inflammatory bowel disease, sarcoidosis, and celiac disease [3,4,12-14]. Many studies have also reported an association of SCAD with fibromuscular dysplasia. It has been hypothesized that SCAD might be an initial manifestation of fibromuscular dysplasia [14-16].

Approximately 90% of people presenting with SCAD have some form of ACS, of which 20 to 50% present with ST elevated myocardial infarction [4,17]. Other presentations include ventricular arrhythmias, cardiogenic shock, or sudden cardiac death [18,19]. The left anterior descending artery is the most commonly affected artery, which accounts for 32% to 46% of all cases, followed by the circumflex and the right coronary artery. [14,18,19]. Chest pain is the most common symptom of SCAD, which prompts the patient to visit the emergency room [20].

SCAD is typically diagnosed through coronary angiography. In some cases, additional imaging tests, including intravascular ultrasonography, optical cardiac tomography, or coronary computed tomographic angiography (CCTA) may be used to further delineate the SCAD lesion and visualize features like dissection flaps and intramural hematomas [8,21-23]. In a prospective study of 327 SCAD patients, research found that hypertension was more than two times likely to be associated with an increased risk of recurrent SCAD. Conversely, the use of beta-blocker medications was found to be significantly associated with a reduced risk of recurrent SCAD by almost two-thirds [14]. According to this study, the majority of patients (83.1%) received initial medical treatment, while only 16.5% or 2.2% underwent in-hospital percutaneous coronary intervention or coronary artery bypass graft surgery, respectively.

Early recognition is of key importance in the management of SCAD. The decision to attempt revascularization or medically treat acute myocardial infarction due to SCAD depends on the severity of the disease and various other factors. No randomized clinical trials have compared medical management with immediate revascularisation in SCAD. Medical management should be based on each individual, including the use of statins, beta-blockers, antihypertensive medications, and antiplatelet medications [10]. Medical management is generally preferred over percutaneous intervention/stent placement, mainly if the patient is hemodynamically stable [8,21]. The risk of hematoma propagation, iatrogenic injury, wire placement in the false lumen, and the need for multiple stents are some of the reasons why PCI is a challenging option in SCAD. Studies have

indicated that most SCAD lesions that are medically treated tend to heal with time. These lesions generally show an improvement in blood flow and a reduction in severity, which can be observed through angiography [18,22].

Research has also shown that SCAD survivors have significant rates of psychological distress, including anxiety, depression, and posttraumatic stress disorder [24]. Our patient presented to the hospital three times after her diagnosis of SCAD, including one admission and two ER visits, during which no abnormalities were found. Extensive psychological support was given, together with a referral to the SCAD survivors community group.

Conclusion

SCAD is often misdiagnosed and managed as atherosclerotic acute coronary syndrome (ACS), which can be catastrophic for the patient. Accurate and timely diagnosis is crucial, as it not only provides appropriate supportive care but also helps stratify patients and ensure that percutaneous coronary intervention (PCI) is performed selectively in appropriate patient groups with SCAD. This is important because PCI for SCAD is associated with higher rates of complications and a lower success rate in comparison to PCI for atherosclerotic coronary artery disease.

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