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CASE REPORT

Cardiology

Spontaneous coronary artery dissection: An uncommon cause of cardiac arrest in the young

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Abstract

Spontaneous coronary artery dissection (SCAD) is a cause of acute coronary syndrome that frequently goes undiagnosed due to its rarity and variable presentation. Additionally, patients with SCAD are frequently young and relatively healthy; factors that may inadvertently lower clinical suspicion of serious pathology, thereby causing delayed or missed diagnosis and inadequate management. Our case report describes a young female who presents after cardiac arrest with inconclusive initial labs and diagnostic tests who was ultimately diagnosed with SCAD. Additionally, we briefly review the pathogenesis and risk factors, as well as the diagnostic and management recommendations for SCAD.

1 | INTRODUCTION

Acute coronary syndrome (ACS) is estimated to result in the hospitalization of over 1 million patients in the United States each year, emphasizing the importance of appropriate diagnosis and management in the emergency department (ED).[1] Spontaneous coronary artery dissection (SCAD), a relatively uncommon cause of ACS, results in reduced coronary blood flow from either a spontaneous tear in a coronary artery wall or compression of the coronary artery by an intramural hematoma. Patients suffering from SCAD present with a spectrum of symptoms, ranging from mild chest pain to cardiac arrest. The rarity of the condition, combined with our inability to confirm the diagnosis in the ED, makes the identification and emergency management of patients with SCAD difficult. The following case describes a patient who presented to the ED with the return of spontaneous circulation (ROSC) after cardiac arrest, who was eventually taken for a coronary angiogram and diagnosed with SCAD.

2 | CASE

A 45-year-old female with no known past medical history presented to the ED with sustained ROSC after suffering a cardiac arrest at home. Per reports from the emergency medical services (EMS) medics, the patient complained of fatigue and some chest pain associated with bilateral arm tingling while taking her dog for a walk. On returning to her home, the patient became unconscious and was found to be pulseless by her husband. EMS reports the husband began chest compressions within 1 minute of the patient's collapse. EMS arrived soon thereafter, continuing cardiopulmonary resuscitation (CPR), and applying an automated external defibrillator, which administered 1 shock, after which the patient regained pulses but remained unresponsive. Total CPR time was reported as 10 minutes.

On arrival at the ED, the patient was found to have a Glasgow Coma Scale score of 8 and was intubated. Prearrival electrocardiogram (ECG) reported atrial fibrillation with uncontrolled ventricular response without any evidence of ST-segment elevation myocardial infarction (STEMI). Initial ECG in the ED showed sinus tachycardia, similarly without any evidence of STEMI (Figure 1). A bedside

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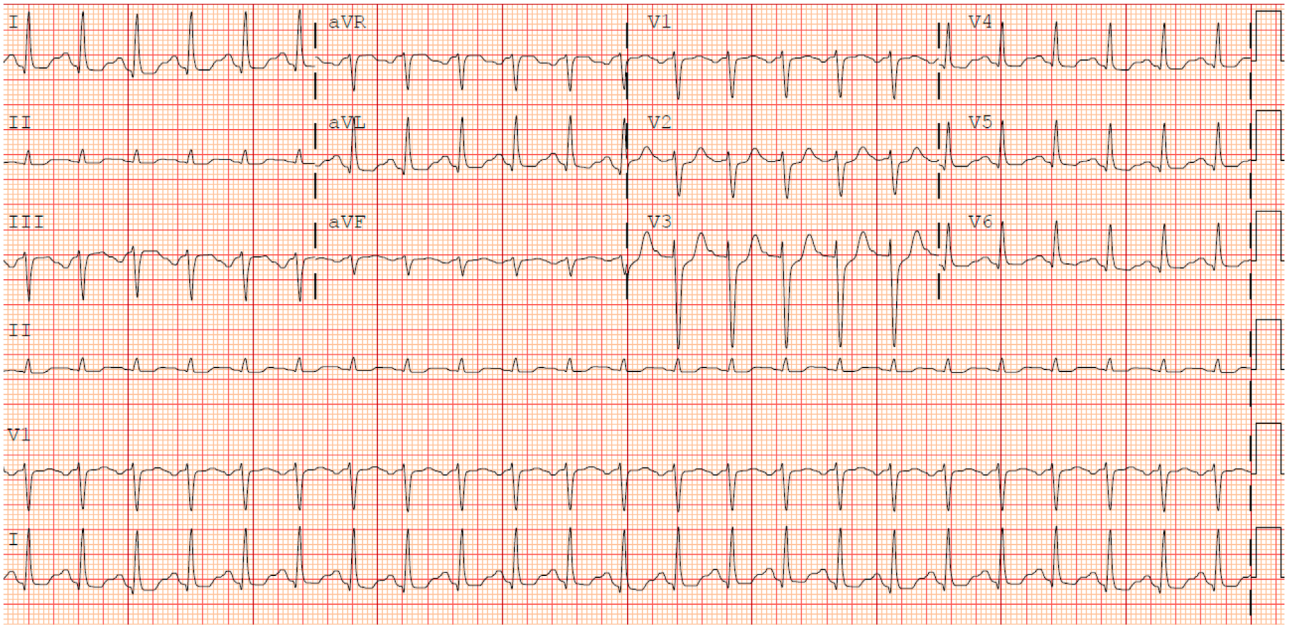

FIGURE 1 Initial ECG on patient arrival to emergency department. Later read by cardiology as sinus tachycardia with heart rate 138, mild upsloping ST depressions in V2–V3. Abbreviation: ECG, electrocardiogram.

echocardiogram (ECHO) done by an emergency physician showed a normal left ventricular ejection fraction (LVEF), small right ventricle, and collapsible inferior vena cava. The patient remained normotensive on arrival to the ED.

Initial labs were significant for a pH of 7.10, pCO2 of 73.5, and lactate of 6.91. Complete blood count (CBC) and metabolic panel demonstrated slight leukocytosis, anemia, and elevated creatinine. The initial high-sensitivity troponin was 9 ng/L, falling within the "indeterminate" range of the ED's institutional guidelines. Computed tomography (CT) of the head and CT angiography of the chest, abdomen, and pelvis were significant only for signs of aspiration pneumonia, likely in the setting of cardiac arrest and CPR.

Interventional cardiology was consulted from the ED and initially recommended a nonurgent coronary angiogram given the lack of evidence of ischemia on ECG and reassuring ECHO. The patient was admitted to the medical intensive care unit for post-ROSC care including targeted temperature management and subsequently had serial ECHOs showing worsening LVEF. Formal ECHO showed a markedly reduced systolic function with an estimated LVEF of 34%. High-sensitivity troponin drawn 4 hours after ED arrival showed significant elevation to 3258 ng/L. Given the changes in the patient's ECHO and elevated troponin level, the patient was taken for a diagnostic coronary angiogram and was found to have a Type 2 SCAD (Figure 2). The interventional cardiology team decided not to intervene in the lesion, and the patient was started on aspirin and clopidogrel. The patient's course was complicated by cardiogenic shock requiring vasopressors and aspiration pneumonia, presumably from cardiac arrest. During the hospital stay the patient was successfully extubated with a baseline neurologic exam and LVEF improved to 55%. She was additionally started on carvedilol once off vasopressors and discharged home 10 days after the initial presentation.

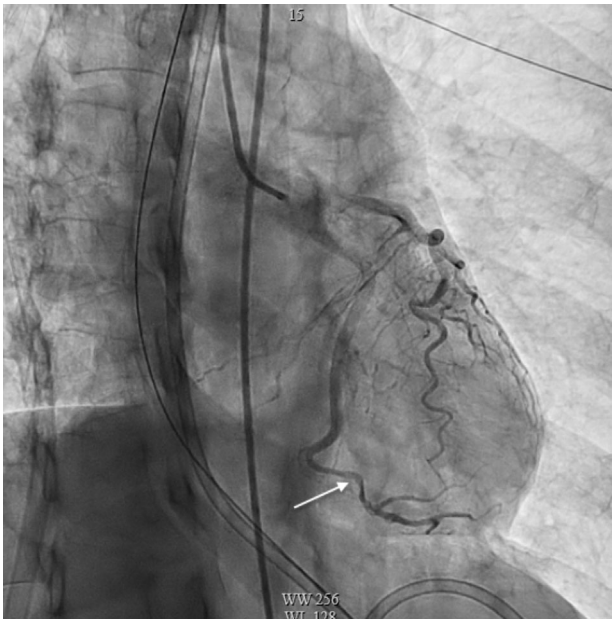

FIGURE 2 Coronary angiogram showing Type 2 spontaneous coronary artery dissection of the mid-to-distal left circumflex with 40% luminal stenosis (seen as slight narrowing of vessel compared to distal vessel).

3 | DISCUSSION

SCAD is thought to occur through 2 mechanisms that lead to reduced coronary artery blood flow and thus signs and symptoms of ACS. The first mechanism is the formation of an intramural hematoma (IMH), most commonly through spontaneous rupture of the vasa vasorum, the small blood vessels that supply the larger coronary arteries. Formation

of an IMH compresses the lumen diameter and impedes blood flow, resulting in myocardial injury.[2,3] The other mechanism is identical to aortic dissection and involves the formation of an intimal tear of the coronary artery and the creation of a false lumen.[2]

Unlike other causes of ACS, SCAD is not associated with atherosclerosis and therefore not related to traditional cardiovascular risk factors, such as hypertension, hyperlipidemia, diabetes, smoking, and obesity.[2] Thus, SCAD is often underdiagnosed as it often presents in an otherwise "low-risk" group of patients including, most often, healthy females (who make up 90% of cases) < 50 years of age.[2,4] It is estimated that SCAD accounts for 0.1%–4% of all cases of ACS, and approximately 35% of women < 50 years old who are diagnosed with myocardial infarction (MI).[2,3] Additionally, SCAD is long known to be the most common cause of pregnancy-associated MI.[2]

Although the pathogenesis of SCAD is not completely understood, there is thought to be an interplay between arteriopathies or other systemic conditions, potentially interacting with extreme physiologic or emotional stressors.[2] Multifocal fibromuscular dysplasia, for example, is one of the most commonly reported underlying conditions in patients with SCAD.[2] Connective tissue disorders, systemic inflammatory diseases such as lupus, or hormonal alterations that occur due to either therapy or natural changes associated with pregnancy are believed to alter the architecture and potentially weaken the arterial wall.[2] Other precipitators, including strong emotional or physical triggers including recurrent Valsalva maneuver (retching, childbirth) or drug ingestions (cocaine, methamphetamines) are thought to potentially "trigger" SCAD in those who are predisposed. However, many people also have no identifiable underlying condition or trigger.

Similar to those with ACS, over 95% of patients present with chest pain.[2] ECG findings for patients with SCAD vary depending on the site of the lesion. Some studies have found that a majority of SCAD patients present with STEMI or non-STEMI.[2] However, in a small case series patients presented with normal ECGs (35%), nonspecific ST abnormalities (30%), and ST depressions (5%), in addition to ST elevation patterns.[5] Rarely, patients may present without initial ECG changes or elevated troponin levels, similar to the patient described here, further obscuring the diagnosis.[6,7] Frequent repeat troponin testing, serial ECGs, and cardiac ultrasounds are thus very helpful in managing these patients, especially as some may present as undifferentiated cardiac arrest. Any evidence of cardiac ischemia with repeat testing should prompt emergency physicians to consult interventional cardiology for possible cardiac catheterization.

Coronary angiogram is needed to secure the diagnosis of SCAD. Type 1 SCAD appears as multiple radiolucent lumens on the angiogram. Type 2 SCAD appears as diffuse luminal narrowing or stenosis (usually > 20 mm) whereas Type 3 SCAD is focal or tubular stenosis (usually < 20 mm) that mimics atherosclerosis. Thus, adjunctive imaging is often used to look at the vessel walls to distinguish between IMH and atherosclerosis.[3] This imaging includes intravascular ultrasound, and the technologically superior optical coherence tomography, either of which can be used if the diagnosis of SCAD is not clear on angiography.[3]

Most patients with SCAD are managed medically, and as many as 70%–97% of cases resolve on their own.[3] Although there is no clear consensus, many patients are started on dual-antiplatelet therapy and beta-blockers.[3] Although management may include percutaneous coronary intervention with angioplasty or stent placement, this is often associated with poor outcomes including iatrogenic dissection or worsening intramural hematoma.[3] Additionally, SCAD most commonly occurs in the distal coronary segments, which are often too small for intervention or involve large segments of vessels that are at high risk of stent thrombosis or restenosis if a stent is placed.[3,6] Coronary artery bypass grafting is rarely used in these patients due to poor outcomes. Inpatient monitoring is recommended as approximately 5%–10% of dissections may worsen over the first week, necessitating emergent revascularization.[3]

Given that the diagnosis of SCAD is confirmed only after an angiogram, the role of the emergency physician is to provide excellent supportive care in all patients with suspected ACS or cardiac arrest and to involve interventional cardiology early if concern for cardiac ischemia arises.

4 | CONCLUSION

This case highlights the importance of maintaining a broad differential when approaching a relatively healthy and young patient in cardiac arrest and including SCAD in this differential, especially if the patient is female. Additionally, as with our patient, not all patients with SCAD may present with the typical signs of ischemia on initial ECG or on troponin testing, thus emphasizing the importance of repeat testing, especially if any change in clinical status or suspicion occurs. We reviewed some of the "typical" risk factors for SCAD, emphasizing that these vary drastically from those we associate with ACS. Therefore, as the emergency physician deals frequently with patients complaining of chest pain, ranging from those well appearing to those in cardiac arrest, this is an important diagnosis to consider.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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