

CASE REPORT

Two-VesselSpontaneousCoronaryArteryDissection Requiring LAD Stents Placement

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Abstract

Introduction: Spontaneous coronary artery dissection is a rare cause of acute coronary syndrome, most often seen in young to middle aged women without cardiac risk factors. It is often fatal due to it's sudden onset and rapid progression. Clinical signs and symptoms of SCAD may range widely, from minor symptoms and stable angina to myocardial infarction, cardiogenic shock, and arrhythmias. Risk factors divided into four categories including, Genetics, Atherosclerotic and vasculitis, Peripartum, and Idiopathic.

Case Presentation: A 47-year-old woman presented to the emergency room via EMS due to chest pain. ECG showed significant ST elevation in anterolateral leads. Code STEMI was activated prior to patient's arrival in the ED. The patient was taken directly to the cardiac catheterization lab on arrival. Review of coronary angiograms appeared to be consistent with spontaneous coronary artery dissection (SCAD) Angiography showed 99% occlusion in mid- LAD. Intracardiac nitroglycerin was given for possibility of coronary vasospasm with no improvement. Due to persistent ST elevations with ongoing chest pressure, it was decided to proceed with PCI. Few days later she was eventually discharged home.

Conclusion: Given the high mortality rate associated with SCAD, it is crucial to diagnose and treat this disorder as soon as possible. Observational studies have indicated that 70-97% of patients receiving conservative therapy showed angiographic repair of SCAD lesions when repeat angiography was done.

Results: As a result, a cautious approach to therapy is usually advised, which calls for a prolonged inpatient observation period of 3-5 days. Although conservative therapy is indicated most of the times, few times stent might be needed. We present the following case who presented with anterolateral ST elevation myocardial infarction secondary to SCAD that required a stent placement which ended with a favorable outcome.

Keywords:

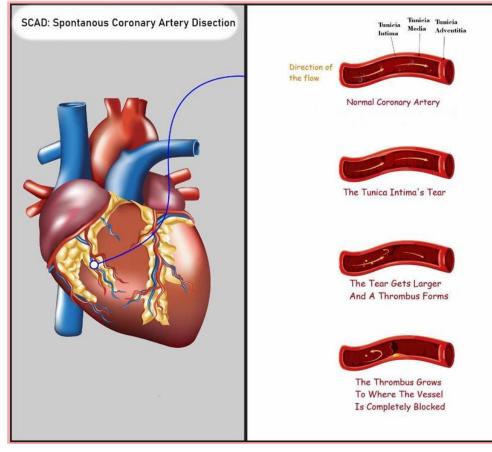
- Coronary Artery Dissection
- SCAD
- 🔸 MI

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Introduction

A spontaneous separation of the coronary arterial wall caused by intramural hemorrhage that is neither traumatic, iatrogenic, or atherosclerotic is known as a Spontaneous Coronary Artery Dissection (SCAD). (Khan et al., 2017) Either an intimal rip or a spontaneous hemorrhage from the vasa vasorum is the cause. As a consequence, an Intramural Hemorrhage (IMH) forms, whichmay constrict the actual artery lumen and cause myocardial ischemia and even infarction. Acute Coronary Syndrome (ACS) is now more often thought to be caused by SCAD (Figure 1), especially in young to middle-aged women without cardiovascular risk factors (Seecheran et al., 2019). SCAD accounted for 24% of cases in one series of women with Myocardial Infarction (MI) who were under 50 years old. SCAD may be fatal because of its abrupt start, rapid development, and diagnostic conundrum as a non-atherosclerotic etiology of ACS. occur more often in the first few weeks after delivery. By inducing aberrant cell growth in artery walls, Fibromuscular Dysplasia (FMD) may weaken arterial walls, leading to blockages, dissections, or aneurysms. Furthermore, it might lead to a stroke, high blood pressure, and tears in other blood vessels. Women are more likely than men toget FMD (Naderi, 2017). SCAD has also been connected to conditions that cause blood vessel inflammation, such as lupus and polyarteritis nodosa. Genetic disorders that impact the body's connective tissues, such as vascular Ehlers-Danlos syndrome and Marfan syndrome, have been linked to persons with SCAD.

SCAD's pathophysiology is still debatable, and many hypotheses have been put forward. According to the theory, a spontaneous intimal rip causes bleeding into the coronary artery wall, which then promotes the growth of hematomas and blocks the real arterial lumen. 2 In contrast, it's thought that a first vasa vasorum rupture inside the coronary arterial media may cause bleeding and hematoma development later on without any link to the artery



lumen. Following compression of the coronary artery wall, myocardial ischemia occurs (Ghani et al., 2018).

Additionally, a limited involvement for genetics in the pathophysiology of SCAD has been suggested, along with an element of recessive inheritance patterns in sporadic instances. However, the pathophysiology remains imprecise, necessitating more research.

As a result of the dearth of randomized studies contrasting medical treatment with revascularization techniques, there is a lack of information on the best care of SCAD. Observational studies have indicated that 70-97% of patients receiving conservative therapy showed angiographic repair of SCAD lesions when repeat angiography was done. 5,6 As a result, a cautious approach to therapy is usually advised, which calls for a prolonged inpatient observation period of 3-5 days. (Del Rio-Pertuz et al., 2022) The foundation of long-term medical

Figure 1: The pathophysiology of SCAD

Patients with SCAD may have symptoms such as chest pain, a fluttering or rapid heartbeat, discomfort in the arm, shoulder, back, or jaw, trouble breathing, and dizziness. Both men andwomen may be affected by SCAD, however often women are affected more than men are.

Recently, few SCAD sufferers have given birth. SCAD may occur at any time throughout pregnancy, however it has been shown to treatment for SCAD is a beta-blocker and aspirin cocktail. Because beta-blocker usagehas been linked to a decreased risk of SCAD recurrence (hazard ratio: 0.36), it is a crucial component of longterm treatment at our facility. In a newly released review, the topic ofmedical treatment in SCAD is covered in great depth.

Case Presentation

A 47-year-old woman with past medical history of tobacco dependence and family history of MIpresented to the emergency room via EMS due to chest pain. She described it as a 10/10 non-

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radiating chest pressure. EMS performed an ECG in the field showing significant ST elevation in anterolateral leads. EMS administered 324 mg Aspirin, 2 Nitroglycerin tablets, and 50 mg of Fentanyl. Code STEMI was activated prior to patient's arrival in the ED. Repeat EKG in the emergency department showed persistent anterior ST elevations with reciprocal changes in the inferior leads (Figure 2).

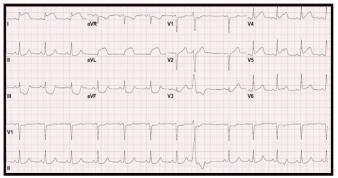


Figure 2: Initial Electrocardiography

The patient was taken directly to the cardiac catheterization lab on arrival. Review of coronaryangiograms appeared to be consistent with $SCAD^1$, likely the cause of the patient's MI. Angiography showed 99% occlusion in mid-LAD (Figure 3).



Figure 3: Initial Coronary Arteries Catheterization

Intracardiac nitroglycerin was given for possibility of coronary vasospasm with no improvement.Due to persistent ST elevations with ongoing chest pressure, it was decided to proceed with PCI (Figure 4).



Figure 4: Left Anterior Descending Artery (LAD) Stent Placement

Patient underwent IVUS guided percutaneous transluminal coronary angioplasty of the proximal to mid-LAD using two overlapping synergy everolimus drug eluting stents. The ostial second diagonal branch showed 71% stenosis secondary to jailing from the stent placement. It was decided to treat this medically given patient's clinical improvement after placement of LAD stents. Patient was admitted to ICU for medical management of STEMI secondary to SCAD andstarted on Aspirin, Brillinta, Crestor and Metoprolol. An echocardiogram was obtained that showed a left ventricular ejection fraction of 28% secondary to ischemic cardiomyopathy with severe hypokinesis of the apical wall.

Throughout the night following catheterization patient continued to experience chest pain refractory to nitroglycerin drip and multiple rounds of IV morphine. This waned over the course of the day, however, recurred overnight. Another EKG which is showed in (Figure 5) was performed showing new ST changes in V2-V4.

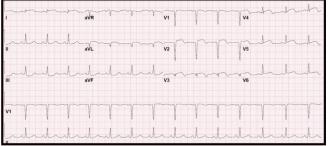


Figure 5: Repeated Electrocardiography

Repeat EKG 20 minutes later following morphine administration showed persistence of ST changes. Patient was taken to catheterization lab for evaluation of stent patency. Proximal and mid-LAD stents remained patent (Figure 6). Angiography showed unchanged compromise of diagonal branch but no new stenosis. It was determined that this was like the cause of the patient's continued chest discomfort.

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Figure 6: Coronary Arteries Catheterization showing the remained patent LAD post Stent

However, the patient was chest pain free at the time of the procedure thus, continued medical management was recommended. Risk stratification for sudden cardiac death was performed and life vest was ordered for three months. The next day, patient's chest pain improved, and she was deemed stable for transfer to general medical floor. Patient was monitored on the general medical floor for the next 3 days. Chest x ray showed pulmonary vascular congestion with smallbilateral pleural effusions likely secondary to acute systolic congestive heart failure following myocardial infarction. Furosemide was given once intravenously. She was eventually discharged on Aspirin, Atorvastatin, Furosemide, Isosorbide Mononitrate, Lisinopril, Metoprolol and Ticagrelor.

Discussion

SCAD was originally documented in an autopsy of a 42-year-old woman in 1931. 3 Based on angiographic evaluations, the total incidence of SCAD is predicted to vary from 0.28 to 1.1%. As a result of the significant number of spontaneous dissections that present as sudden death, it seems that the true incidence is greater (Mokhberi et al., 2015). About 70% of the patients are young women (male to female ratio: 2:1), and 30% of these instances are connected to the peripartum period (Camacho Freire et al., 2019).

The LAD accounts for 60% of coronary dissections, making it the most common location of dissection. The left main artery is the most frequent location, followed by the right coronary artery (more often in men). (Paratz et al., 2018) Dissection may also happen between the media and the adventitia, although it often happens between the intima and media. 1 The two most frequent causes of SCAD are atherosclerosis and the peripartum period. Patients with SCAD are often divided into four categories. One category of patients includes those with genetic connective tissue diseases linked to a weak artery wall, such as Marfan and Ehlers-Danlos syndromes. Patients with underlying atherosclerosis, particularly males with an average age of 55, make up a different group (Tajrishi et al., 2019). (It is thought

that vasculitis and atherosclerosis contribute to lupus patients having a higher incidence rate of SCAD). Women who are peripartum make up the third category. (Ninety-four percent of instances happen between one day and three months after giving birth. This occurrence is thought to have been caused by increased blood flow, shear stress, and variations in the blood levels of relaxin and estrogen (Tweet et al., 2017). The patients in the last group had idiopathic SCAD. It is importantto remember that SCAD has also been linked to extreme physical activity, chest injuries, and the use of certain pharmaceuticals such cocaine, cyclosporine, 5fluorouracil, oral contraceptives, and fenfluramine (Hayes et al., 2020).

Coronary angiography, optical computed tomography, and intravascular ultrasonography are used to make the diagnosis. Based on angiographic results, four forms of SCAD are described: Kind 1 is described as having a link between the true and false lumen; Type 2 is the most prevalent type of SCAD and has a dissection that is longer than 20 mm without a connection between the true and false lumen. Type 3 lacks a link between the true and false lumens and hasa dissection of less than 20 mm. Type 4 resembles an embolus because it has a dissection and restriction to blood flow at the end of the coronary arteries (Antonutti et al., 2021). In example 1, coronary angiography was the only procedure used since both the real and spurious lumens couldbe clearly seen. In instance 2, IVUS was used to identify and categorize SCAD since coronary angiography was uncertain.

Troponins are often increased, and an EKG reveals ischemia or infraction symptoms similar to ACS, both of which assist with diagnosis and provide information on the development and course of the condition (Tweet et al., 2018). In some ambiguous circumstances, cardiac magnetic resonance may be employed to enhance results and early diagnosis.

Clinical signs and symptoms of SCAD may range widely, from minor symptoms and stable angina to myocardial infarction, cardiogenic shock, and arrhythmias. The primary presentation is acute coronary syndrome. All young women who come with acute coronary syndrome should beevaluated for SCAD, especially during the peripartum period (Kim, 2020).

The primary diagnostic method is coronary angiography. In the event of hazy angiographic images, imaging modalities like intravascular ultrasonography (IVUS) and optical coherence tomography (OCT) provide additional information regarding the morphology and intramural location of the lesions. The use of IVUS is beneficial for therapy monitoring (Tweet et al., 2014).Due to the limited frequency of SCAD, the extant literature does not include any randomised clinical trials on the treatment of SCAD outside of a few publications and data collections.

Surgery, percutaneous coronary intervention, and medication therapy are all part of the therapeutic care of SCAD. Surgery or percutaneous coronary intervention is often used to treat patients with severe dissections that result in chronic ischemia, although instances with modest involvement may be managed conservatively with medicines (Ravipati et al., 2021). Anticoagulants, aspirin, clopidogrel, beta blockers, nitrates, and sometimes calcium channel blockers are used to treat SCAD in a manner similar to how acute coronary syndrome is treated.Although GP2b-3a inhibitors (which potentially enhance hematoma development) are often administered to patients with acute coronary syndrome before coronary angiography, there is a dearth of research on their use in SCAD. Additionally, administering fibrinolytic drugs is not advised (Mokhberi et al., 2015).

In addition to traditional medical care, Koller et al., 4 reported that a woman with postpartum SCAD recovered after using prednisone and cyclophosphamide. After proper identification of false and genuine lumens in minor lesions needing intervention, coronary stent implantation maybe performed; however, surgical therapy is preferable in multi-vessel involvement or big lesions, especially those affecting the left main artery (Paratz et al., 2018).

Due to her ongoing ischemia and dynamic alterations in her ECGs, our patient had coronary angiography, and due to the substantial LAD involvement, she underwent CABG. Beta blockers, aspirin, and cholesterol-lowering medications must be prescribed for individuals with acute coronary syndrome throughout the observation period, as well as calcium channel blockers and nitrates in the event of long-term coronary artery spasm. The prognosis for treating SCAD is generally positive (Naderi, 2017). The risk of coronary artery dissection increases with advancing age in individuals with a history of peripartum SCAD. Patients with SCAD are not advised to have routine follow-up angiography, however it is permissible to undertake nuclear perfusion scans when monitoring patients who have substantial vascular involvement.

Conclusion

SCAD is a rare condition that affects young women. Acute coronary syndrome is how the majority of patients arrive. Particularly in younger individuals, the differential diagnosis of chestdiscomfort should include the diagnosis of SCAD. Conservative treatment may be regarded as the cornerstone of illness care in patients with hemodynamically stable patients. PCI or CABG may eventually be used to treat unstable individuals. Given the high mortality rate associated with SCAD, it is crucial to diagnose and treat this disorder as soon as possible.

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Ethical Consideration: None

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