


The Conundrum of Anticoagulation and Antiplatelet Therapy in Spontaneous Coronary Artery Dissection

Journal of Investigative Medicine High
Impact Case Reports
Volume 10: 1–4
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Medical Research
DOI: 10.1177/23247096221097541
journals.sagepub.com/home/hic


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Abstract

Spontaneous coronary artery dissection (SCAD) is a relatively rare and frequently misdiagnosed disease. The current knowledge of its pathophysiology and management is limited and based mostly on hypotheses. We present a patient with recurrent SCAD whose condition worsened soon after discontinuation of anticoagulation, prompting us to question the current management and review the evidence about pathophysiology, anticoagulation, and antiplatelet therapy.

Keywords

spontaneous coronary artery dissection, therapy, anticoagulation

Introduction

Spontaneous coronary artery dissection (SCAD) is defined as epicardial coronary artery dissection that is not associated with atherosclerosis, trauma, or iatrogenic.¹ It is an underdiagnosed, under-recognized condition that might represent up to 4% of all myocardial infarctions (MIs).^{2,3} Spontaneous coronary artery dissection is mostly seen in woman⁴ and might represent up to 45% of myocardial infarction in those younger than 50 years old.³ Spontaneous coronary artery dissection is associated with extra-coronary abnormalities, especially fibromuscular dysplasia.¹ Precipitating factors are associated up to 57% of the time. The most common are emotional stress (40.5%), exercise (24.4%), and maneuvers increasing intrathoracic pressure, such as vomiting and retching (2.4%).⁵ Spontaneous coronary artery dissection recurrence can be seen in up to one-third of patients. The best approach for prevention appears to be trigger avoidance, hypertension control, and beta-blocker use.

Management consists of a conservative approach, avoiding percutaneous coronary intervention, and thrombolytics when possible.^{6–8} Anticoagulation and antiplatelet therapy in SCAD have not been studied in randomized clinical trials. Current consensus is to stop anticoagulation once SCAD has been diagnosed and start dual antiplatelet therapy (DAPT) for 30 days followed by 1 year of aspirin (ASA).

We present a patient with recurrent SCAD whose condition worsened soon after discontinuation of anticoagulation and improved with its reinstatement. Prompting us to

question the current management and review the evidence about pathophysiology, anticoagulation, and antiplatelet therapy.

Case

A 54-year-old female without identifiable risk factors for coronary artery disease and with past medical history significant for mixed connective tissue disease, supraventricular tachycardia, and SCAD which was diagnosed 3 years ago with lesions in the left anterior descending (LAD) artery and obtuse marginal (OM) artery that was treated with 2 stents to the LAD (Figure 1). The patient came to the emergency department complaining of substernal and epigastric pressure of approximately 1-day duration. Her chest pain started earlier in the day with an intensity 5/10 and was partially relieved after sublingual nitroglycerin. She went to sleep and awoke hours later with severe 10/10

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Received March 10, 2022. Revised April 5, 2022. Accepted April 11, 2022.

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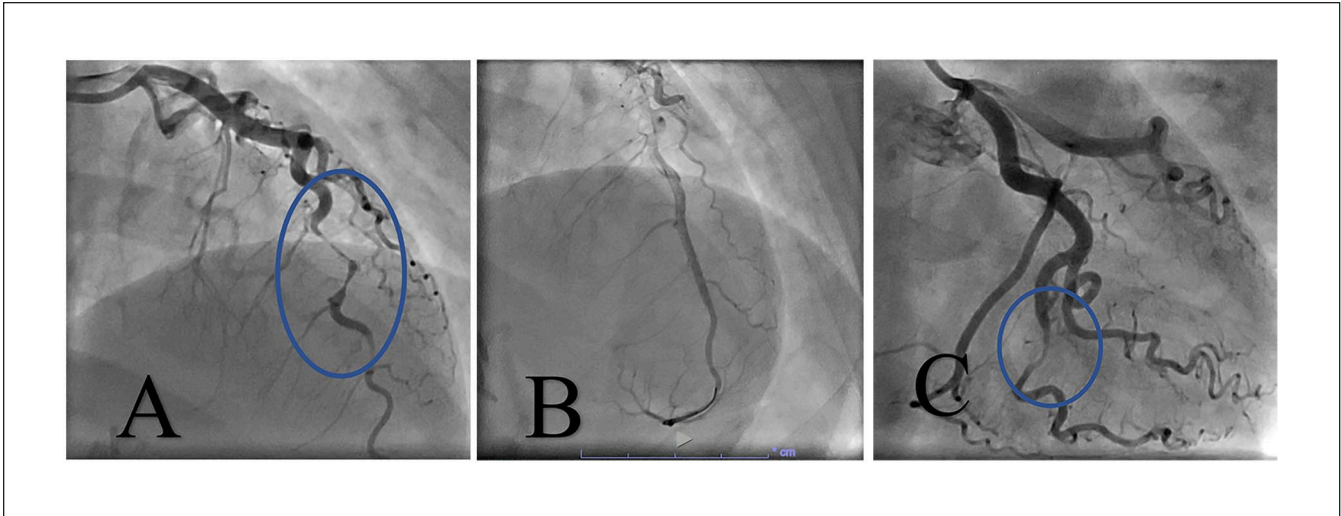


Figure 1. Cardiac catheterization from 3 years before, showing (A) 80% mid-LAD lesion and (B) LAD poststenting (C) 80% OM lesion, no intervention was done in this artery.

pain, radiating to her left shoulder and left arm, this time not relieved by nitroglycerin.

On admission, she had a temperature of 37.1°C, heart rate 78 bpm, respiratory rate 18 r/min, blood pressure 152/101 mm Hg, and saturating 95% on room air. Her physical exam and initial blood work were unremarkable, including normal high-sensitivity (HS) troponin. Electrocardiogram (EKG) showed normal sinus rhythm with a heart rate of 78 and T-wave inversions in V2 and V3. Serial HS-troponins trended up significantly to 180 ng/L prompting the diagnosis of NSTEMI and cardiology evaluation. She was loaded with aspirin, clopidogrel, and a heparin drip was started. Cardiac catheterization showed patent stents in the LAD and 99% occlusion in a sub-branch of OM plus 40% stenosis in the mid to distal right coronary artery (RCA). The previous lesion in the OM seen 3 years ago was no longer there and the artery looked angiographically normal (Figure 2). Recurrent spontaneous coronary artery dissection was diagnosed, no coronary intervention was performed.

The heparin drip was discontinued after SCAD diagnosis, dual antiplatelet therapy, beta-blockers, and statin were continued. One day later, the chest pain recurred, and troponin rose to 250 ng/L after having decreased to 140 ng/L. The heparin drip was restarted and nitroglycerin plus ranolazine as antianginal therapy were added.

The echocardiogram showed normal left ventricular function with an ejection fraction of 60% and mild hypokinesis in the mid anteroseptal and mid lateral/inferolateral walls of the left ventricle that were present on the previous echocardiogram. On day 4 after admission, her chest pain had resolved and the HS-troponins were down trending. The decision to stop the heparin drip was made. She was kept for observation one more day and discharged on rosuvastatin, nitroglycerin, ranolazine, metoprolol, and dual antiplatelet therapy with

aspirin and clopidogrel. The patient declined screening for associated conditions.

Discussion

The clinical presentation of MI secondary to SCAD is similar to that secondary to atherosclerotic disease. The differentiation between the 2 of them has important management implications. Spontaneous coronary artery dissection is classified into 3 types. Type 1, when there is visible contrast in the arterial wall with multiple identifiable lumens, type-2 defined by diffuse stenosis, typically more than 20 mm in length, and type-3 which can mimic atherosclerotic disease, usually has long (11-20 mm), hazy, and linear lesions and is differentiated by the absence of atherosclerotic changes in other coronaries.⁹

The pathophysiology of SCAD is not completely understood. An intramural hematoma (IMH) is formed which causes separation of the intima and formation of a false lumen. The expansion of the IMH can cause occlusion of the true lumen leading to MI.⁹ There are 2 hypotheses regarding IMH formation, an endothelial tear allowing blood to enter the subintimal space and de novo IMH formation.¹ De novo IMH hypothesis was raised after the following observations: IMH can be seen before dissection¹⁰ most of the times SCAD does not appear to have communication between the true and false lumens, and the false lumen pressure, area, as well as the degree of stenosis are higher in the absence of identifiable lumen communication.¹¹

Anticoagulation and antiplatelet use in these patients are uncertain topics. The current approach to stop anticoagulation once SCAD is diagnosed¹ is based on the hypothesis that it may worsen IMH leading to extension of the dissection.⁹ However, pressures inside the vasa vasorum should be lower than those in the coronary arteries.¹² This significant pressure

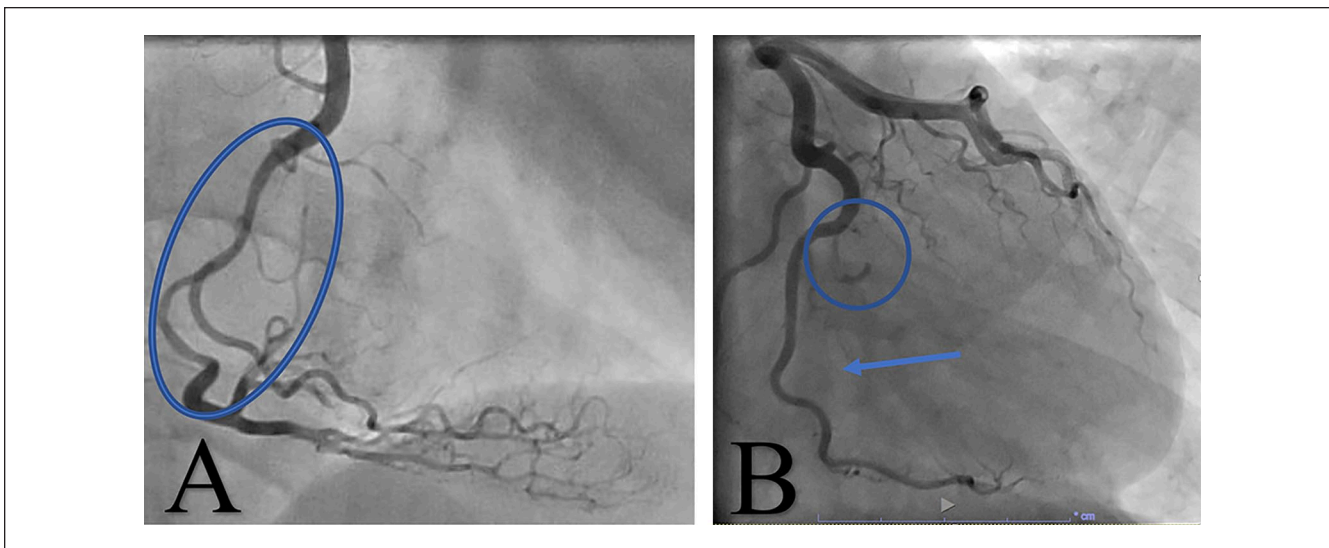


Figure 2. Cardiac catheterization from this hospitalization: (A) 40% stenosis in the mid to distal RCA, (B) patent stents in the LAD, and 99% lesion at a sub-branch OM (circle). The previous lesion in the OM seen 3 years ago is no longer there and appears angiographically normal (arrow).

difference should allow for the vessels and lymphatics to resorb the IMH and retard its expansion.¹³ Thus, it has been hypothesized that spontaneous hemorrhage should not be able to collapse the true lumen and an intramural thrombus would decrease the pressure inside the coronary artery making it easier for the IMH to expand. In this case, maintaining anticoagulation could be beneficial.¹³ There is not enough research to accept or deny any hypothesis.

There is no consensus regarding antiplatelet therapy. Dual antiplatelet therapy for 1 year and lifelong aspirin is the mainstay therapy for acute coronary syndrome (ACS). Spontaneous coronary artery dissection has different pathophysiology than atherosclerotic disease. Most of the patients with SCAD recover normal coronary anatomy in approximately 30 days.¹⁴ Cerrato et al¹⁵ found that patients with SCAD receiving DAPT had higher rates of major adverse cardiac outcomes at 1-year follow-up compared to single antiplatelet therapy. Dual antiplatelet therapy for up to 4 weeks followed by aspirin alone for 12 months is a reasonable approach. Each patient's risk factors and comorbidities should be taken into consideration for prolongation of antiplatelet therapy. The BA-SCAD trial (Identifier: NCT04850417) is a randomized control trial that is underway and aims to enroll 600 patients with SCAD to identify the differences between the use or not of beta-blockers and the use of a 1-month antiplatelet regimen versus 12 months DAPT.¹⁶

In our case, the patient had recurrence of chest pain and rising troponins soon after discontinuation of anticoagulation, leading us to restart the heparin drip. Her clinical picture improved significantly on anticoagulation. She was eventually discharged on DAPT. Although this is only one

case experience, it prompts us to review the current management and consider all alternatives regarding anticoagulation and antiplatelet management in SCAD.

Conclusion

Anticoagulation and antiplatelet management in patients with SCAD are based on hypothesis only. More research is required to confirm or deny the current management.

Author Contributions

A.C.S. contributed to conceptualizing and writing original draft. E.M. contributed to conceptualizing and writing original draft. D.A. contributed to supervision and review.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethics Approval

Our Institution does not require ethical approval for reporting individual cases.

Informed Consent

Verbal informed consent was obtained from the patient for their anonymized information to be published in this article.

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