

Acute coronary syndrome, not always what you think.

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Introduction

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome, more common in young women without risk factors for, or a history of, coronary artery disease and usually occurs in the peripartum period.

Case Presentation

38-year-old obese, nonsmoking, active woman who presented to the emergency department (ED) with recurrent episodes of heartburn following a meal. On the day of presentation, her heartburn was persistent, more severe, and was not relieved by calcium carbonate tablets as the symptom normally did. At presentation, she was afebrile, with normal pulse and respiratory rates, a blood pressure of 130/78 mmHg. She had no signs of poor perfusion or volume overload. Her ECG was normal however, her troponin was elevated at 0.20 ng/ml and trended up to a peak of 2.03 ng/ml. Her transthoracic echocardiography was only significant for mild concentric left ventricular hypertrophy with an EF of 55% to 65%. Hence cardiology was consulted. Cardiac catheterization revealed SCAD of the distal left anterior descending artery, which is a type 2 dissection. Given her TIMI-3 flow and absence of typical chest pain. We decided to treat her medically. She was started on metoprolol, aspirin, and atorvastatin. Given her young age and family history of early death due to brain aneurysm, we conducted further workup for vasculitis, autosomal dominant polycystic kidney disease, and fibromuscular dysplasia (FMD) the results of which were all unremarkable. She was discharged on metoprolol, aspirin, and atorvastatin with a scheduled follow-up evaluation with a cardiologist.



Figure 1: electrocardiogram at presentation showing a normal sinus rhythm. It also meets criteria for low voltage in the precordial leads but that is not the scope of this presentation

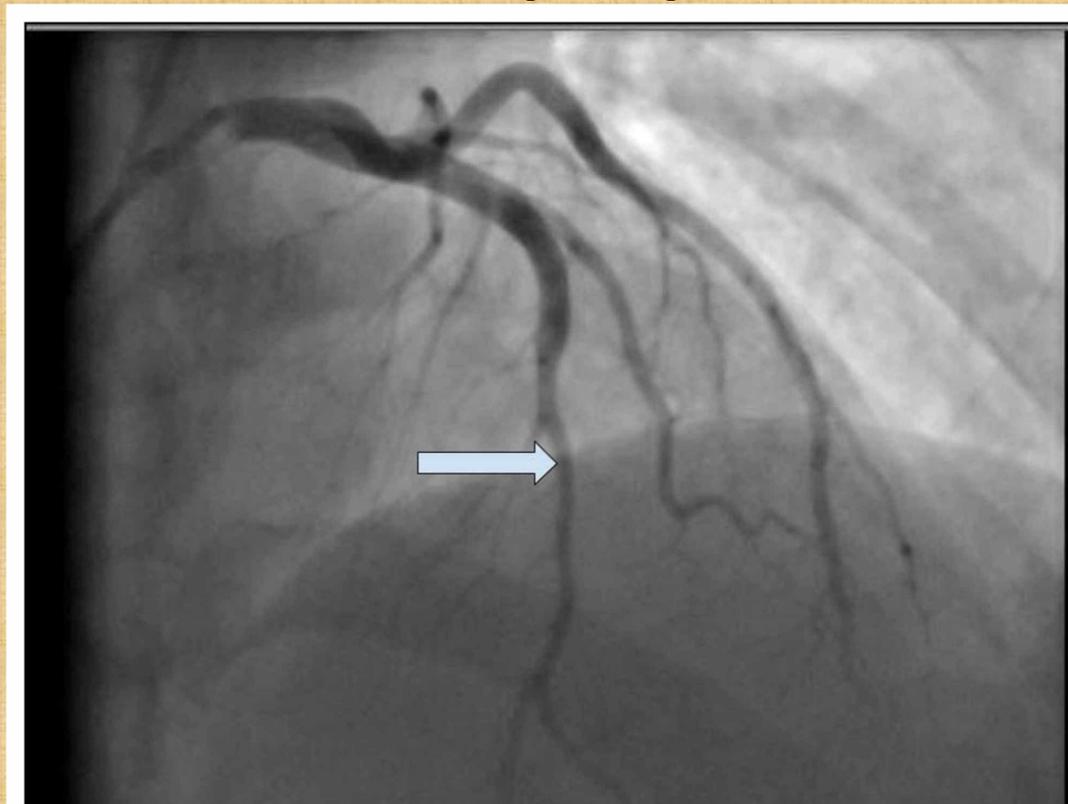


Figure 2 : Type 2 dissection of the distal left anterior descending.

Discussion

SCAD is a non traumatic and non iatrogenic separation of the coronary arterial wall and is a rare cause of acute myocardial infarction and ACS. SCAD is more common in young women in the peripartum period, usually without cardiovascular risk factors.

Weakening of the arterial wall due to hormonal changes, cystic medial necrosis, and proteolytic enzymes from periadventitial eosinophils in susceptible patients can lead to an underlying vulnerable coronary wall. Underlying factors could potentially predispose a patient to SCAD are FMD, postpartum status, multi parity, connective tissue disorders, systemic inflammatory conditions, and hormonal therapy. Stressors such as intense exercise or emotional stress, labor and delivery, intense Valsalva-type activities, and recreational drug use can precipitate SCAD. SCAD can present with a variety of features including arrhythmias, unstable angina, acute myocardial infarction, cardiogenic shock, pericardial effusion, tamponade, and even sudden cardiac death.

Chest pain is the most common symptom presentation in >96% of cases; less common symptoms include arm pain, neck pain, nausea or vomiting, diaphoresis, dyspnea, and back pain. In most patients, in the absence of prior trauma, the diagnosis of SCAD is made at the time of coronary angiography.

In most SCAD patients, conservative management with medical therapy is the preferred strategy as was the case with our patient. PCI can be used for patients needing emergent revascularization. Other treatment options include coronary artery bypass grafting, fibrinolytic therapy (with or without subsequent PCI), mechanical hemodynamic support, and cardiac transplantation, the selection of which should be based on patient presentation and unique characteristics .

Conclusion

Our case highlights the need to also consider the diagnosis of SCAD as a cause of ACS in young patients presenting to the ED with nonspecific symptoms such as heartburn, and not just those with the more typical presentation of chest pain.

References

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