

Surgery is an option in evolving myocardial infarction induced by spontaneous coronary artery dissection: a case report

Ghaitha Al Mahruqi ^{1*}, Hilal Alsabti², and Mirdavron Mukaddirov²

¹Oman Medical Specialty Board, Muscat, Oman; and ²Cardiothoracic Surgery Division, Department of Surgery, Sultan Qaboos University Hospital, Muscat, Oman

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Background

Spontaneous coronary artery dissection (SCAD) has emerged as one of the important yet rare causes of acute coronary syndrome that primarily affect young peripartum women without cardiovascular risk factors. Despite the recent improvements in diagnosis and recognition of the importance of SCAD, it remains poorly studied and there has been no consensus of opinion regarding its optimal management.

Case summary

A 29-year-old breastfeeding woman presented with 1-day history of severe chest pain radiating to the jaw and both shoulders. Cardiovascular examination, 12 leads electrocardiogram, and echocardiography were normal. Troponin levels were elevated; hence, coronary angiogram was done and showed type 2 SCAD of the left anterior descending artery (LAD). The patient was managed conservatively. The next day, she started again to complain of severe chest pain and her troponin levels continued to rise. Repeated coronary angiogram revealed progression of the previous LAD dissection. Another dissection was also noticed in the left circumflex artery. Chest pain recurred over the night and her troponin levels continued to rise. An emergency coronary artery bypass grafting (CABG) was performed. The patient was doing well postoperatively and was discharged home on Day 8.

Discussion

Our patient presented with acute ischaemic changes secondary to SCAD. The report illustrates the risk factors, pathogenesis, diagnostic work up, and the possible therapeutic options of SCAD, which include conservative management and CABG. The management varies depending on the clinical presentation and the extent of the coronary artery dissection.

Keywords

Coronary arteries • Dissection • Myocardial infarction • Coronary artery bypass grafting • Case report

Learning points

- A high index of suspicion is critically important when dealing with any postpartum young woman presenting with chest pain.
- The management modality of spontaneous coronary artery dissection varies depending on the patient's clinical condition and the extent of dissection.

* Corresponding author. Tel: +968 97709800, Email: almahruqighaitha@gmail.com

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Introduction

Spontaneous coronary artery dissection (SCAD) was considered a rare cause of acute coronary syndrome (ACS) in the past. However, nowadays SCAD is increasingly diagnosed and is recognized as a significant clinical entity especially in young peripartum women presenting with sudden chest pain.¹ Dr Pretty reported the first case of SCAD in 1931.² The true incidence of SCAD is not known as most of the evidence comes from isolated case reports and small series of patients.² In consonance with the fourth universal definition of myocardial infarction (2018), SCAD is defined as a spontaneous, non-traumatic, non-iatrogenic rupture of the coronary artery wall with a consequential formation of a false lumen and an intramural haematoma (IMH), which is capable of compressing the true lumen causing ACS.³

Timeline

Day of admission	The patient presented with angina symptoms; her cardiac enzymes were elevated, echocardiography (ECHO) was normal; therefore, coronary angiogram was done and it showed type two spontaneous coronary artery dissection of the left anterior descending artery (LAD). Patient was managed conservatively.
Day 2 of admission	Patient symptoms recurred so coronary angiogram was repeated and it revealed progression of the previous LAD dissection + dissection of the left circumflex artery.
Day 3 of admission	Non-ST-elevation myocardial infarction, troponin levels continued to rise so the patient was taken for emergency coronary artery bypass grafting surgery.
Day 8 of admission	ECHO was normal, patient was discharged home.

Case presentation

A 29-year-old Arabic woman presented to the emergency department (ED) with 1-day history of severe chest pain radiating to the jaw and both shoulders; the pain was not exertional and it lasted for 5 min. On examination, the patient was conscious and had normal vitals apart from 110 b.p.m. heart rate (HR). The patient was recently diagnosed with Wolff–Parkinson–White syndrome, not on any medication, had three miscarriages in the past, and is currently breastfeeding. Twelve lead electrocardiogram (ECG) was done in the ED and the result was normal. Bedside echocardiography (ECHO) showed good left ventricular systolic function with ejection fraction (EF) of >55%. The laboratory results showed troponin T of 169 ng/mL (<14 ng/mL). Rheumatic work up was normal. Coronary angiogram was done on the day of presentation and revealed a long-diffused narrowing of the left anterior descending artery (LAD) suggestive of

type 2 SCAD (Figure 1). The Patient was managed conservatively and was treated with bisoprolol 2.5 mg and clopidogrel 75 mg. However, her chest pain recurred the next day. The patient became tachycardic with HR of >120 b.p.m and troponin levels went up to 190 ng/mL (<14 ng/mL). Repeated coronary angiogram showed distal tapering of the left main coronary artery (40%) suggestive of extending IMH proximally. LAD showed an element of type 2 SCAD with 70–80% stenosis and there was a dissection of the proximal left circumflex artery (Figure 2). Six hours later, the patient was reassessed again, the pain settled and troponin level went down to 53 ng/mL. Taking into consideration her clinical status and the improvement in cardiac troponin level, we opted to continue with conservative management. The patient was kept on bisoprolol 5 mg, aspirin 100 mg once a day, clopidogrel was stopped, and she was closely monitored. Over the night, the patient started to complain of severe prolonged chest pain that occurs at rest. Her vitals were normal. Twelve lead ECG showed an element of ST depression in the anterolateral leads, and troponin level went up to 699 ng/mL. Hence, the decision was made to proceed with an emergency coronary artery bypass grafting (CABG). Intraoperative transoesophageal echocardiography showed decrease of left ventricular contractility with 30% EF. Intra-operatively we found that the left coronary arteries were completely dissected with visible intra-mural thrombosis. True lumens were identified after intramural thrombus removal and the following surgery was done: left internal mammary artery was anastomosed to LAD, reversed segments of saphenous vein grafts were anastomosed to diagonal artery and to the two obtuse marginal branches of circumflex artery (CxOM1 and CxOM2, Figure 3). Grafts' flowmetry was performed and showed an excellent flow in all four grafts. In the cardiac surgery intensive care unit, patient maintained normal vitals on noradrenalin 0.1 µg/kg/min and intra-aortic balloon pump (IABP). Patient was extubated on post-operative Day 2. IABP was removed on Day 3. ECHO was done on Day 6, and it showed a good left ventricular function with EF of 50%. On Day 8, patient was discharged on ivabradine 5 mg b.i.d., carvedilol 6.25 mg b.i.d., and clopidogrel 75 mg/day.

Discussion

SCAD is a rare cause of acute myocardial infarction and ACS, accounting for 0.1–0.4% of ACS cases.⁴ The exact mechanism of SCAD is not known, however, there are two hypotheses that have been proposed to explain its pathogenesis. The 'inside-out' hypothesis, the cause behind the development of the false lumen in SCAD is a disruption in the endothelial-intimal layer, which allows blood from the lumen to enter the vessel wall leading to the formation of IMH. Whereas, in 'outside-in' hypothesis, a bleeding episode within the coronary artery wall at the level of the vasa vasorum is the cause of acute coronary arteries dissection.⁵ Pregnancy and peripartum period have been linked to SCAD; and they account for about 5% of SCAD cases. The hormonal changes that occur during this period may weaken the wall of the arteries and lead to SCAD.⁶ Majority of SCAD patients present with signs and symptoms of ACS and elevated cardiac enzymes as in our patient.³ Once SCAD is suspected coronary angiography should be performed. The traditional angiographic finding of SCAD is the presence of multiple radiolucent lumens and extraluminal contrast staining.⁷ To simplify the diagnosis

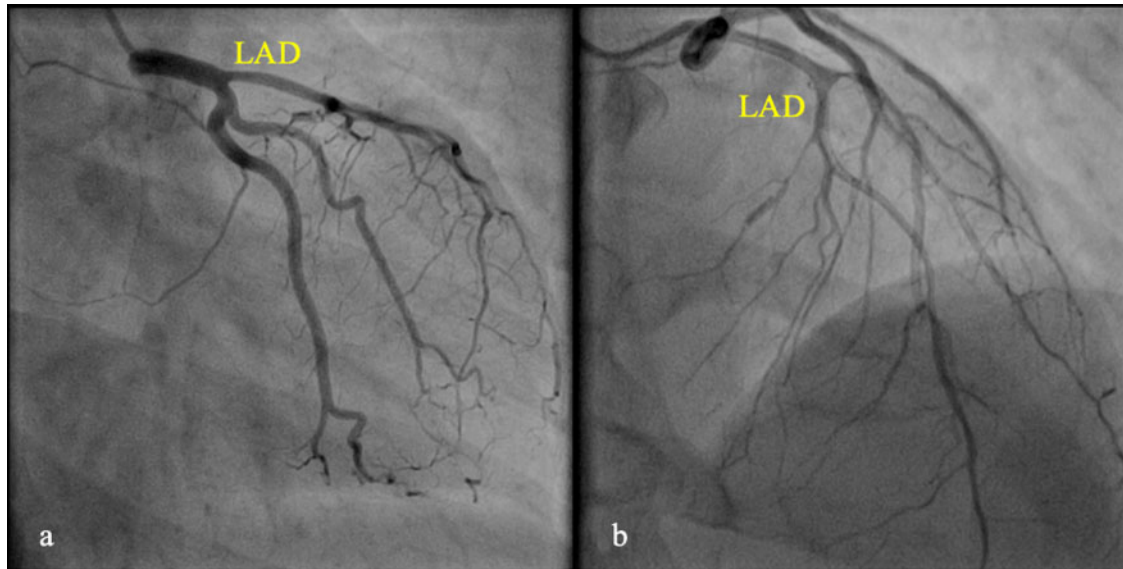


Figure 1 (A and B) Coronary angiogram showing spontaneous coronary artery dissection of the left anterior descending artery.

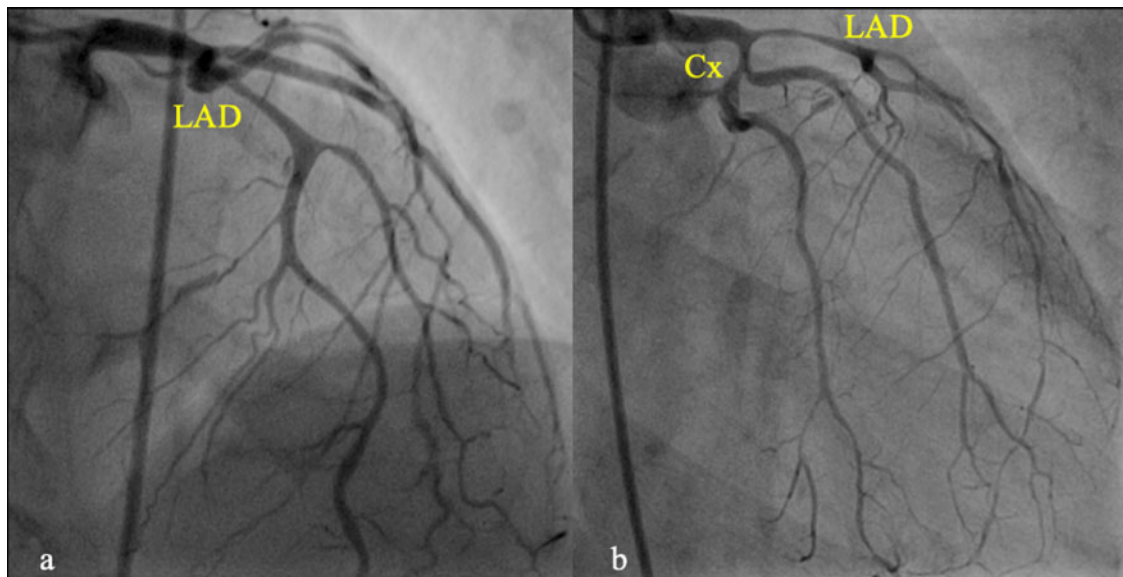
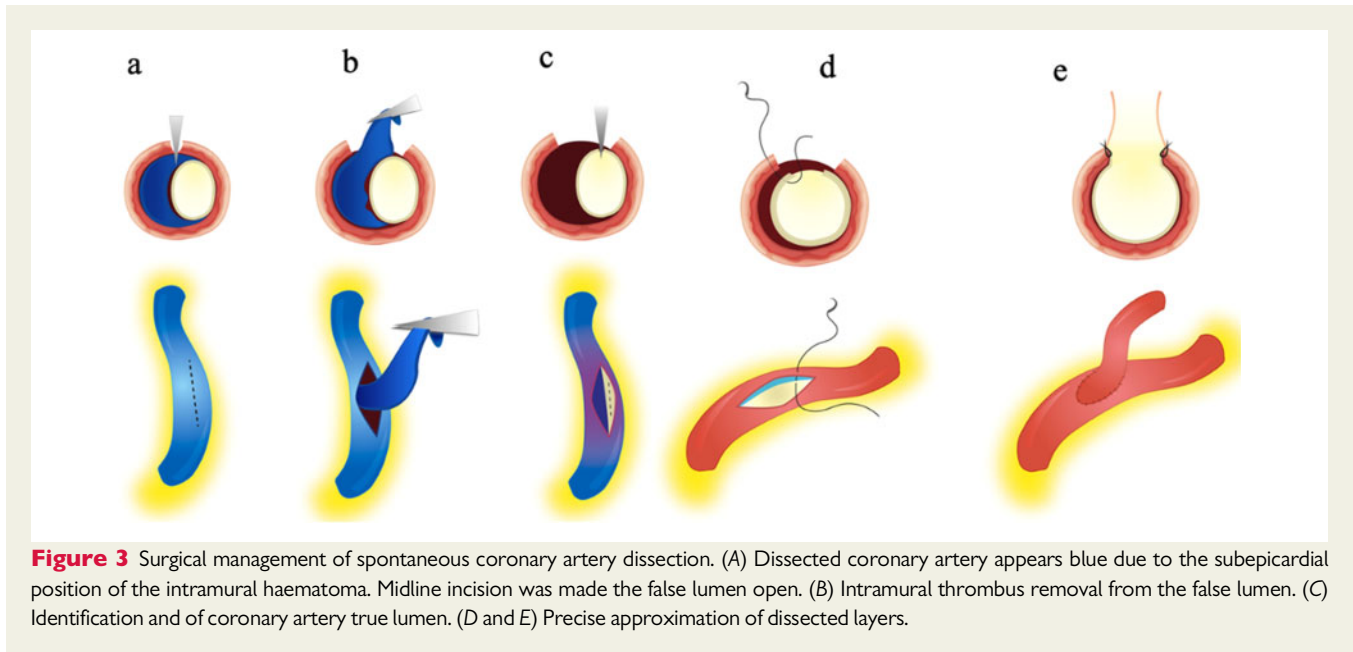


Figure 2 Coronary angiogram showing complete dissection of left anterior descending artery, proximal circumflex with involvement of distal left main coronary artery.

of SCAD, three types of angiographic appearance were proposed: type 1 refers to the classic appearance of multiple radiolucent lumens; type 2 is the most common type, which refers to the presence of diffuse stenosis; and type 3 is focal or tubular stenosis that mimics atherosclerosis.³ Our patient presented initially with type 2 dissection of the LAD; however, after 24 h, the dissection continued to progress and it involved the left circumflex artery. The unusual feature of this case was complete dissection and intramural thrombosis

of all branches of left coronary artery including the stem. Other tools like intravascular ultrasonography and optical coherence tomography can be used to provide accurate diagnosis of SCAD.⁸ The optimal management of SCAD remains controversial. Based on the retrospective observational studies, conservative management is preferred in clinically stable patients as the false lumen and the IMH seems to diminish after few months.^{3,9} Treatment with antiplatelet therapy is controversial as antithrombotic drugs may prolong bleeding time in

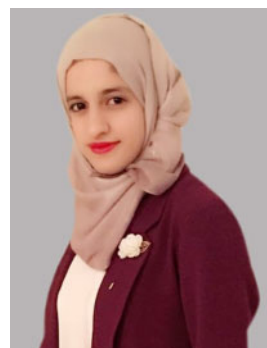


patients with intramural bleeding.¹⁰ Betablockers (BBs) are recommended in the short- and long-term management of SCAD as they reduce the coronary arterial shear stress, additional studies have found that BB lower the risk of SCAD recurrence.¹¹ Our patient was treated with clopidogrel and bisoprolol as soon as the diagnosis of SCAD was made, however, when the dissection progressed clopidogrel was stopped. Due to lack of sufficient data supporting coronary revascularization in SCAD, conservative management is suggested in the majority of cases. However, CABG and percutaneous intervention are indicated as a lifesaving procedure in patients with dissection of the left main coronary artery, ongoing ischaemic symptoms, haemodynamic instability, cardiogenic shock, or ventricular arrhythmias.³ CABG technique that was used in our case is not common, however, it can be applied in patients who present with progressive SCAD as in our case. Although there is no supportive data between CABG and percutaneous coronary intervention (PCI), observational studies have shown that PCI is associated with high rate of complications.¹² Mayo Clinic cohort study described a 53% rate of procedural PCI failure, in the prospective Canadian cohort, 30% of PCI procedures were unsuccessful.^{6,12} CABG remains an important treatment strategy in cases with extensive dissections involving proximal arteries including the left main, as a rescue strategy after failed PCI or when coronary anatomy is unfavourable for PCI.¹¹ One of the studies reported high success rates with CABG as an index treatment strategy ($n = 20$ patients, 32 conduits). Although these findings provide some reassurance for CABG as a viable revascularization option, caution should be taken as CABG is associated with high rate of graft failure and thrombosis (69% arterial and venous), secondary to spontaneous healing of SCAD and subsequent competitive flow; moreover, CABG does not provide any protection against recurrent SCAD.¹² Initially, our patient was managed conservatively; however, she was taken for CABG as soon as she started to manifest signs and symptoms of evolving myocardial infarction.

Conclusion

SCAD is an important cause of ACS in otherwise healthy young individuals, particularly peripartum women. Diagnosis and management of SCAD is challenging due to its heterogeneity, undefined mechanism, and lack of strong clinical evidence. Future larger-scale epidemiological studies will help in widening our knowledge about this condition and will allow more accurate prediction and ultimate prevention of recurrent SCAD.

Lead author biography



Ghaitha Al Mahruqi, graduated from Oman Medical college 2017, currently a surgical resident at Oman Medical Speciality Board.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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References

1. Ranke KB, Wong DTL, Baumann A, Nicholls SJ, Gulati R, Psaltis RJ. Current state-of-play in spontaneous coronary artery dissection. *Cardiovasc Diagn Ther* 2019;**9**:281–298.
2. Pretty H. Dissecting aneurysm of coronary artery in a woman aged 42. *BMJ* 1931;**1**:667.
3. Aristi B, Kassimis J, Zioutas D, Meletidou M, Mouselimis D, Tsarouchas A et al. Spontaneous coronary artery dissection (SCAD): case series and mini review. *Cardiovasc Revasc Med* 2020;**21**:1450–1456.
4. Okoroafor CD, Akram A, Ong A. Atypical presentation of spontaneous coronary artery dissection. *Cureus* 2020;**12**:e9543.
5. Saw J, Mancini GBJ, Humphries KH. Contemporary review on spontaneous coronary artery dissection. *J Am Coll Cardiol* 2016;**68**:297–312.
6. Saw J, Starovoytov A, Humphries K, Sheth T, So D, Minhas K et al. Canadian spontaneous coronary artery dissection cohort study: in-hospital and 30-day outcomes. *Eur Heart J* 2019;**40**:1188–1197.
7. Rogers JH, Lasala JM. Coronary artery dissection and perforation complicating percutaneous coronary intervention. *J Invasive Cardiol* 2004;**16**:493–499.
8. Krittanawong C, Saw J, Olin JW. Updates in spontaneous coronary artery dissection. *Curr Cardiol Rep* 2020;**22**:123.
9. Hassan S, Prakash R, Starovoytov A, Saw J. Natural history of spontaneous coronary artery dissection with spontaneous angiographic healing. *J Am Coll Cardiol Intv* 2019;**12**:518–527.
10. Adlam D, Alfonso F, Maas A, Vrints C, Al-Hussaini A, Bueno H et al.; Writing Committee. European Society of Cardiology, acute cardiovascular association, SCAD study group: a position paper on spontaneous coronary artery dissection. *Eur Heart J* 2018;**39**:3353–3368.
11. Gilhofer TS, Saw J. Spontaneous coronary artery dissection: a review of complications and management strategies. *Expert Rev Cardiovasc Ther* 2019;**17**:275–291.
12. Tweet MS, Eleid MF, Best PJM, Lennon RJ, Lerman A, Rihal CS et al. Spontaneous coronary artery dissection: revascularization versus conservative therapy. *Circ Cardiovasc Interv* 2014;**7**:777–786.