

Spontaneous right coronary artery dissection

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DESCRIPTION

A 41-year-old gravida 1 para 1 female with a medical history of hypertension and panic attacks presented to the emergency room with crushing chest pain radiating to her back for 2–3 hours. There was associated shortness of breath and anxiety but no other complaints. Initial evaluation confirmed normal vital signs as well as unremarkable physical examination, ECG, chest X-ray, CT chest and echocardiogram. Troponin was elevated (0.89 ng/mL) and she was admitted for

cardiac monitoring, and was started on aspirin and metoprolol. She continued to have ongoing chest discomfort and rising cardiac enzymes along with new inferior lead T-wave inversion on ECG. Nitroglycerin, clopidogrel and fondaparinux were administered and ultimately the decision was made to perform cardiac catheterisation. Coronary angiography revealed 80% mid-right coronary artery stenosis secondary to spontaneous coronary artery dissection (Figure 1A, B) and a bare-metal stent was deployed with restoration of normal blood flow

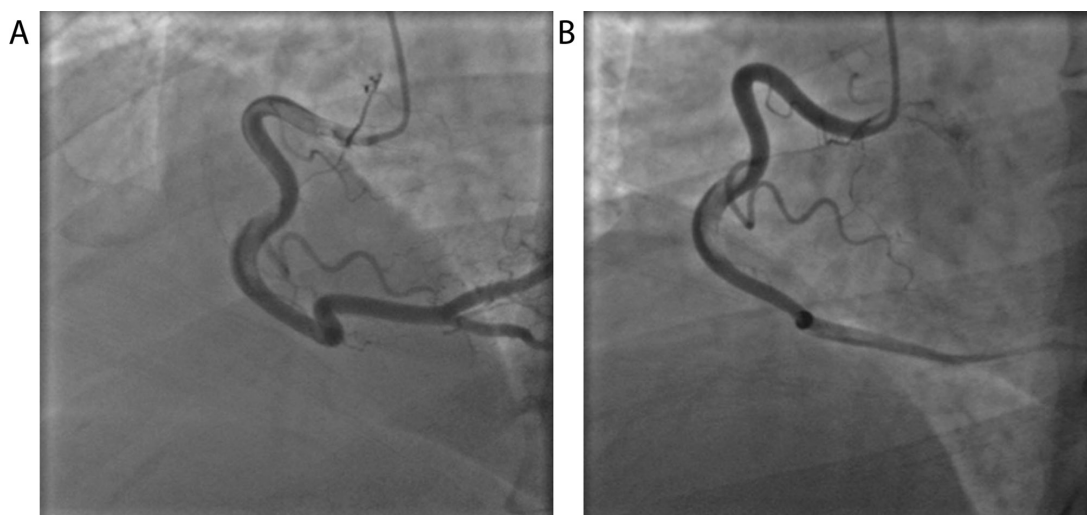


Figure 1 (A and B) Right coronary artery demonstrating a spiral 'barber shop pole' luminal filling defect.



Figure 2 Restoration of normal blood flow in the right coronary artery after stent deployment.

(Figure 2). She improved clinically soon thereafter and was discharged on aspirin, clopidogrel and metoprolol without further complications.

Contributors CH and AA: conception, design, literature search and data collection. NS: literature search and drafting of the manuscript.

Competing interests None declared.

Patient consent Obtained.

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Learning points

- ▶ Spontaneous coronary artery dissection (SCAD) is an uncommon cause of chest pain, myocardial infarction and sudden death. This entity frequently occurs in young relatively healthy females with limited atherosclerotic cardiovascular disease (ASCVD) risk factors.¹ Several predisposing factors have been identified, including multiparity, peripartum status, hormone therapy, connective tissue disorders, fibromuscular dysplasia and migraines.² Our patient's female gender and intermittent non-compliance with antihypertensive medication may have increased her risk of SCAD. While the mechanism is not fully understood, SCAD begins with an intimal tear or bleeding of the vasa vasorum resulting in intramural haemorrhage. With expansion of intramural haematoma, the lumen can become compromised, eventually leading to ischemia and infarction. The majority of cases present with non-ST-segment-elevation myocardial infarction, while a lesser number may present with ST-segment-elevation myocardial infarction or cardiac arrest.³
- ▶ SCAD is often limited to a single vessel, most commonly involving the left anterior descending artery. The diagnosis is typically confirmed with coronary angiogram by identification of contrast material staining within the arterial wall. In cases of intramural haematoma without a clear intimal dissection plane, intravascular ultrasound or optical coherence tomography may help make the diagnosis.⁴ SCAD accounts for less than 1% of acute myocardial infarction but should always be suspected in young female patients with chest pain and limited ASCVD risk factors.
- ▶ Due to the technically difficult nature and high complication rate of revascularisation owing to vessel fragility, conservative therapy is generally the preferred management strategy. Given the possibility of early dissection progression, a 5-day inpatient-monitoring period is prudent.⁵ Antiplatelet therapy and beta-blockers should be administered, with the addition of statins and angiotensin-converting enzyme inhibitors in cases of hyperlipidemia and left ventricular dysfunction, respectively.
- ▶ Revascularisation is reserved for unstable patients or persistent symptoms. Percutaneous coronary intervention (PCI) is associated with high rates of technical failure and residual stenosis, as well as a need for emergent coronary artery bypass grafting (CABG).⁶ It has been suggested but remains unclear whether CABG is superior to PCI in selected patients requiring revascularisation.¹ Long-term follow-up is essential given the increased risk of future events. Recurrence rates of 10.4%–13.1% have been reported.³ Furthermore, patients with SCAD frequently have concurrent extracoronary vascular pathology, including dissection, aneurysm and fibromuscular dysplasia. There is a role for non-invasive, comprehensive vascular imaging with CT angiogram, both for screening and post treatment surveillance.⁵ In addition, counselling regarding pregnancy and medical genetics is recommended.⁷ There exists a need for more clinical outcomes research, as there are currently no standardised protocols for optimal management. High clinical suspicion is of paramount importance given the potentially dire consequences of unrecognised SCAD.

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