Percutaneous coronary intervention in recurrent spontaneous coronary artery dissection: a case report

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Background
Spontaneous coronary artery dissection (SCAD) is an infrequent but potentially life-threatening condition in patients with acute myocardial infarction. Conservative medical therapy is recommended in patients with SCAD. However, very little evidence exists in the management of recurrent SCAD when conservative medical therapy fails.

Case summary
A 48-year-old woman presented with non-ST-elevation myocardial infarction (NSTEMI) on a background of cigarette smoking. Her coronary angiogram showed the first diagonal artery (D1) and right marginal branch (RM) occlusion with angiographic appearance that is consistent with SCAD. She was medically managed. She represented 2 months later with another NSTEMI, and her coronary angiogram showed healing SCAD in the D1 and RM, but a new SCAD in the first obtuse marginal artery (OM1). She was managed medically. She represented 4 months later complaining of angina every 2 days. This time her coronary angiogram showed healed SCAD in OM1 and RM, but the recurrence of SCAD in D1. Given that she had recurrent events despite medical therapy, we decided to proceed with percutaneous coronary intervention (PCI) to D1. She presented with an atypical chest pain 10 months later and her coronary angiogram showed complete healing of all coronary arteries and a patent stent in D1. She has remained symptom free.

Discussion
The management of SCAD is contentious given the lack of randomized clinical trials to assess optimal treatment strategy. In most patients with SCAD, conservative medical therapy is recommended after the diagnosis is secured. We believe that PCI may be beneficial in patients with recurrent SCAD.

Keywords
Spontaneous coronary artery dissection • Percutaneous coronary intervention • Acute coronary syndrome • Case report

Introduction
Spontaneous coronary artery dissection (SCAD) is an infrequent but potentially life-threatening condition in patients with acute myocardial infarction. Pre-disposing factors for SCAD are fibromuscular dysplasia, postpartum status, multiparity, connective tissue disorders.
systemic inflammatory conditions, and hormonal therapy. Conservative medical therapy is preferred in most cases although there is a lack of prospective randomized data for the management of this condition. We believe revascularization may be indicated in certain scenarios, and the following case highlights our decision to perform percutaneous coronary intervention (PCI) in a patient with recurrent SCAD.

**Timeline**

<table>
<thead>
<tr>
<th>Timeline</th>
<th>Events</th>
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<tbody>
<tr>
<td>Day 1</td>
<td>First spontaneous coronary artery dissection (SCAD).</td>
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<tr>
<td></td>
<td>Crushing chest pain at rest with an elevated troponin I of 2.72 μg/mL (normal &lt;0.04 μg/mL).</td>
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<td></td>
<td>First diagonal artery (D1) and right marginal branch (RM) SCAD.</td>
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<td></td>
<td>Medically managed on dual antiplatelets and a beta-blocker.</td>
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<td>Two months after first presentation</td>
<td>Second SCAD.</td>
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<td></td>
<td>Crushing chest pain at rest with troponin elevation of 2500 ng/L (normal &lt;18 ng/L).</td>
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<td></td>
<td>Healing SCAD in the D1 and RM, but a new SCAD in the first obtuse marginal artery (OM1).</td>
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<tr>
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<td>Medically managed.</td>
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<tr>
<td>Six months after first presentation</td>
<td>Third SCAD.</td>
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<td></td>
<td>Frequent angina with no rise in troponin.</td>
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<td></td>
<td>Healed SCAD in OM1 and RM, but the recurrence of SCAD in D1.</td>
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<tr>
<td></td>
<td>Successful percutaneous coronary intervention to D1.</td>
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<tr>
<td>Sixteen months after first presentation</td>
<td>Atypical chest pain.</td>
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<td>Normal coronary angiogram. Healed SCAD in OM1 and RM, and patent stent in D1.</td>
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**Case presentation**

A 48-year-old woman presented with crushing chest pain at rest with an elevated troponin I of 2.72 μg/L (normal <0.04 μg/L) and a normal electrocardiogram (ECG). Her background history includes 30 pack years of cigarette smoking, obesity, gastric banding, fibromyalgia, and depression/anxiety. On examination, her heart rate was 70 b.p.m., her blood pressure was 127/82 mmHg, and her oxygen saturation was 98%. Cardiac and pulmonary auscultations were normal. She was given loading doses of aspirin and ticagrelor. She had a coronary angiogram which showed first diagonal artery (D1) and right marginal branch (RM) occlusion (Figure 1) with an angiographic appearance that is consistent with SCAD. It did not resolve with intracoronary nitrates. We performed a computed tomography aortogram to look for fibromuscular dysplasia in the carotid, renal, and iliac arteries and they were absent. She was medically managed on aspirin 100 mg and clopidogrel 75 mg daily, and metoprolol 25 mg twice daily.

She represented 2 months later with similar symptoms with a troponin elevation of 2500 ng/L (normal <18 ng/L) and a normal ECG. She had another coronary angiogram which showed healing SCAD in the D1 and RM, but a new SCAD in the first obtuse marginal artery (OM1) (Figure 2). She was managed conservatively on the same medications.

She represented 4 months later complaining of angina every 2 days with a normal troponin and ECG. This time her coronary angiogram showed healed SCAD in OM1 and RM, but the recurrence of SCAD in D1. Given that she had recurrent events despite medical therapy, we decided to proceed with PCI. After cautious wiring and confirmation of guidewire placement in the true lumen with selective coronary contrast injection, we deployed a 2.25 × 15 mm Resolute Onyx (Medtronic, CA, USA) in D1, with excellent final result (Figure 3). She remained on the same medications. She presented with an atypical chest pain 10 months later and her coronary angiogram showed complete healing of all coronary arteries and a patent stent in D1. She has remained symptom free.

**Discussion**

Spontaneous coronary artery dissection is an uncommon cause of chest pain and acute myocardial infarction. It is defined as a non-traumatic and non-iatrogenic separation of the coronary arterial wall. This separation leads to intramural haematoma formation within the arterial wall which compresses the lumen, thereby reducing blood flow resulting in myocardial ischaemia or infarction. It frequently occurs in younger and relatively healthy females with limited traditional cardiovascular risk factors. Several pre-disposing factors have been found, including fibromuscular dysplasia, postpartum status, multiparity, connective tissue disorders, systemic inflammatory conditions, and hormonal therapy.

However, our patient’s presentation is unusual for a number of reasons. She has a few cardiovascular risk factors including smoking and obesity. She has had two pregnancies and two births and is not on any hormonal therapy. She did not have features of fibromuscular dysplasia on CT aortogram. Most cases of SCAD involve a single coronary artery, but she presented with a double vessel SCAD, followed by a recurrent presentation with SCAD in a de novo vessel. It is also worth noting that she had SCAD in branch vessels of all three vascular territories but not the main vessels.

There is a complex and not fully understood interplay between pre-disposing arteriopathies and precipitating stressors such as intense exercise, intense emotional stress, labour and delivery, retching, vomiting, coughing, bowel movement, and use of intense hormonal therapy and recreational drugs in SCAD. Men have been reported to have more physical stressors of intense exercise and methamphetamine use than women, while emotional stressors have more often been reported in women than men. Emotional stress has been postulated to lead to a catecholamine surge which can increase arterial shear stress causing intimal rupture. The first reported case of SCAD in 1931 was precipitated by retching and vomiting after a meal.
Coronary angiography remains the gold standard for the diagnosis of SCAD. Intracoronary imaging with intravascular ultrasound and optical coherence tomography could potentially be useful in patients whom the diagnosis is suspected but not confirmed with coronary angiography alone. Alternatively if the diagnosis is uncertain, a repeat coronary angiography may be pursued 4–6 weeks later to examine for spontaneous angiographic healing of the dissected segment. Although we did not utilize intracoronary imaging in the diagnosis of our patient, the evidence of healing dissection on the coronary angiography on repeat presentation 2 months later confirmed our initial diagnosis of SCAD. In fact, our serial coronary angiography demonstrated perfectly the progressive change, healing, and remodelling of coronary vessels in SCAD.

The management of SCAD is contentious given the lack of randomized clinical trials to assess optimal treatment strategy. In most patients with SCAD, conservative medical therapy is recommended after the diagnosis is secured. Patients with SCAD are usually managed on long-term aspirin, beta-blocker, and 1 year of clopidogrel. However, it is unclear whether the medications used in the management of acute coronary syndrome (ACS) have similar benefits in patients with SCAD. The rationale and potential risks of using standard therapy for ACS in patients with SCAD have been questioned by many investigators. Early administration of heparin may reduce thrombus burden but there are concerns about worsening intramural haematoma and extension of dissection in SCAD. Therefore, discontinuation of heparin should be considered once the diagnosis of SCAD is made. Our patient presented with chest pain and elevated troponin on the first two occasions, and therefore, was initially treated as ACS with...
heparin but was ceased once the diagnosis of SCAD was confirmed. Similar potential benefits of protection from thrombosis but increased bleeding risk exist in the use of dual antplatelets in patients who do not undergo PCI in SCAD. Most experts recommend long-term aspirin and judicious use of dual antplatelets in patients with SCAD.12 Revascularization with PCI or coronary artery bypass grafting may be indicated in patients with acute myocardial infarction with symptoms of ongoing ischaemia or haemodynamic compromise. We believe that revascularization may also be beneficial in patients with recurrent SCAD. However, revascularization with PCI is technically challenging and associated with high failure rates and complications, due to fragility of the vessel and difficulty in advancing coronary guide wires within the true lumen.12 Intracoronary imaging is useful in optimizing stent coverage and wall apposition. Long-term follow-up is essential given the increased risk of recurrence.

**Conclusion**

This case illustrates the challenge in the management of patients with SCAD due to the paucity of randomized clinical trials in this area. Medical management is preferred in most cases. However, revascularization may be beneficial in recurrent SCAD. There is a need for more prospective outcome studies to guide and standardize our management of SCAD.

**Supplementary material**

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

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**Figure 3** Third coronary angiogram. (A) Healed spontaneous coronary artery dissection in obtuse marginal artery; (B) Recurrent spontaneous coronary artery dissection in first diagonal artery; and (C) post-percutaneous coronary intervention in diagonal artery.

**Slide sets:** A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

**Consent:** The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** none declared.

**References**

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