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# Simultaneous spontaneous arterial dissections in a 38-year-old woman presenting during the covid-19 pandemic: a case report

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## Background

Spontaneous coronary artery dissection (SCAD) is an increasingly recognized and important cause of acute myocardial infarction, particularly in women under 50, often with minimal risk factors. Many patients have underlying arteriopathy, most commonly in the form of fibromuscular dysplasia.

## Case summary

A 38-year-old woman presented to the hospital with chest pain and elevated high-sensitivity Troponin. Invasive coronary angiography demonstrated SCAD of the left anterior descending artery. The same day the patient developed a severe progressive headache and subsequent imaging revealed a left vertebral artery dissection. She was managed conservatively with optimal medical therapy and was successfully discharged from hospital on Day 7.

## Discussion

To our knowledge, this is the first case report of simultaneous spontaneous coronary and vertebral artery dissections not related to pregnancy. It highlights not only the importance of recognizing and accurately diagnosing SCAD, but also of appreciating the possibility of underlying arteriopathy: this is paramount to ensuring appropriate investigations, follow-up and assessment of any unexplained symptoms in this patient group.

## Keywords

Spontaneous coronary artery dissection • Vertebral artery dissection • Fibromuscular dysplasia • Coronary angiography • Acute coronary syndrome • Case report

## Learning points

- Spontaneous coronary artery dissection is an important cause of acute myocardial infarction, particularly in women <50 years.
- It is commonly associated with Fibromuscular Dysplasia, with non-coronary vessel abnormalities found in a high proportion of those investigated.
- While simultaneous coronary and non-coronary events are rare, additional symptoms should always be taken seriously, with appropriate consideration of further investigation.

This case is a competition winner from the British Junior Cardiologists' Association's Starter Member Case Competition 2020 and underwent review by a judging panel from that organization

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## Introduction

Spontaneous coronary artery dissection (SCAD) is an important cause of acute myocardial infarction (MI). It is frequently associated with underlying arteriopathy, most commonly fibromuscular dysplasia (FMD).<sup>1</sup> While these patients are known to be at risk of recurrent SCAD and other dissections, concurrent events at coronary and non-coronary sites are rare.<sup>1,2</sup> We present a case of simultaneous spontaneous coronary and vertebral artery dissections: to our knowledge the first reported case not associated with pregnancy.<sup>3–5</sup>

## Timeline

Day	Event
0	Presentation with central chest pain and elevated troponin T.
1	Invasive coronary angiography demonstrated spontaneous dissection of the left anterior descending artery. Post-procedure the patient complained of a left-sided headache.
2–3	Computed tomography angiogram imaging, performed for persistent headache on Day 3, revealed left vertebral artery dissection. Magnetic resonance imaging revealed no acute intracranial infarct.
4–6	Conservative management and multidisciplinary (MDT) involvement.
7	Discharge from hospital.
Follow-up	Outpatient MDT follow-up. Computed tomography angiography screening: renal and iliac vessels normal.

## Case presentation

A 38-year-old woman presented to hospital with severe central chest pain, radiating to the jaw, lasting 1.5 hours. She was a non-smoker with no past medical history and took only the combined oral contraceptive pill (COCP). She had two children, aged 4 and 6, with uncomplicated pregnancies. Clinical examination was unremarkable with no abnormal findings, she was haemodynamically stable and pain free. Electrocardiogram (ECG) demonstrated sinus rhythm with T wave inversion in leads V2–6, II, III, and aVF (Figure 1). Blood tests revealed elevated high-sensitivity troponin T (84 ng/L at presentation to 513 ng/L at 6 h, ref value <14 ng/L). Chest X-ray was normal. Admission screen for SARS-CoV-2-RNA was negative.

Treatment for the acute coronary syndrome (ACS) was commenced: Aspirin, 300 mg stat then 75 mg o.d.; Clopidogrel, 300 mg stat then 75 mg o.d.; Fondaparinux 2.5 mg o.d. and Bisoprolol 2.5 mg o.d. (subsequently up-titrated). Invasive coronary angiography,

performed approximately 15 h after the presentation, demonstrated mid to distal vessel irregularity in the left anterior descending artery (LAD), consistent with a diagnosis of SCAD (Figure 2). No intervention was performed as the distal coronary flow was reasonable. The other coronary arteries were normal. An echocardiogram revealed good left ventricular (LV) systolic function with apical hypokinesia.

Post-angiogram the patient complained of a left-sided headache which was thought to be a migraine. The neurological exam was normal and this was treated with simple analgesia (Paracetamol 1 g QDS and Codeine Phosphate 30–60 mg as required). However, the headache progressed over 36 h, prompting further investigation with a CT angiogram on Day 3. This revealed a left vertebral artery dissection (Figure 3). There was also additional vessel abnormality proximally with a small aneurysm and focal stenosis at the levels of C4 and 5, respectively (Figure 4). Magnetic resonance imaging (MRI) head revealed small areas of established infarction in both cerebellar and the left cerebral hemispheres, but no acute infarct.

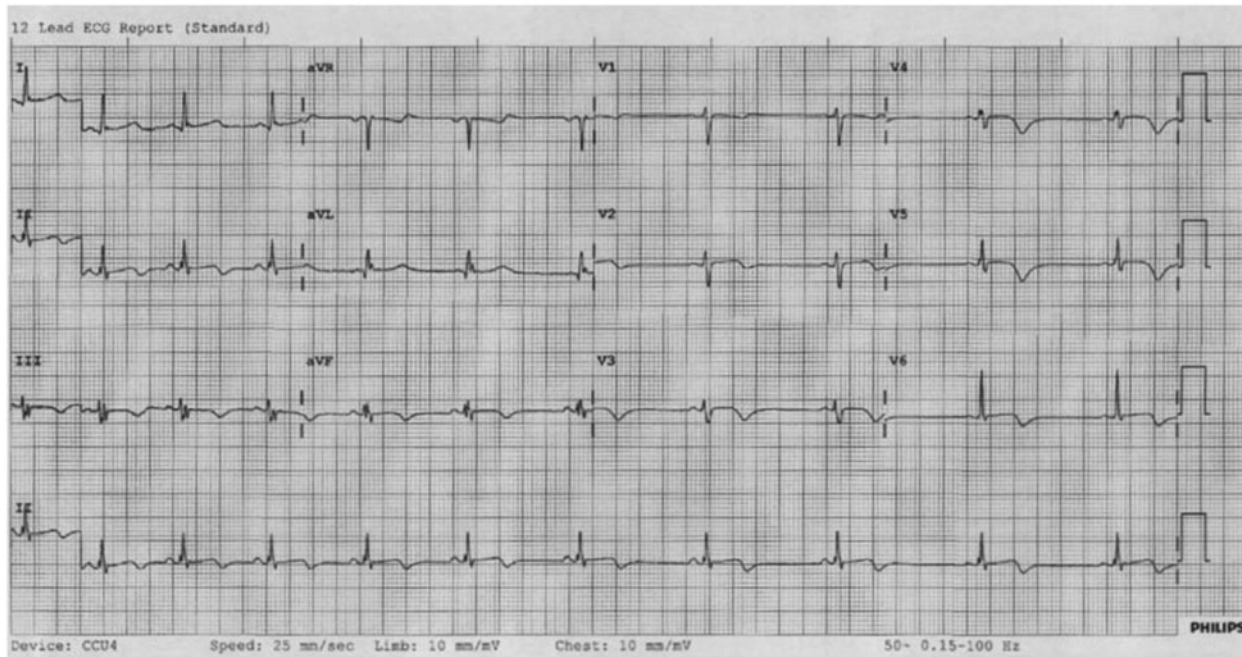
Following multidisciplinary discussion and input from speciality teams, there was a consensus for conservative management. A statin was commenced (total cholesterol 5.0 mmol/L), and the COCP was stopped. A full autoimmune screen was negative.

The patient was counselled regarding the diagnoses, possible underlying FMD, and risk of recurrence. Multidisciplinary follow-up was arranged, including referral to genetic services and the regional specialist in SCAD. She was discharged from the hospital on Day 7. Outpatient CT angiography demonstrated normal major vessels. A CT coronary angiogram, to assess for resolution of SCAD, is awaited. The patient has not yet undergone long-term follow-up but is believed to be well.

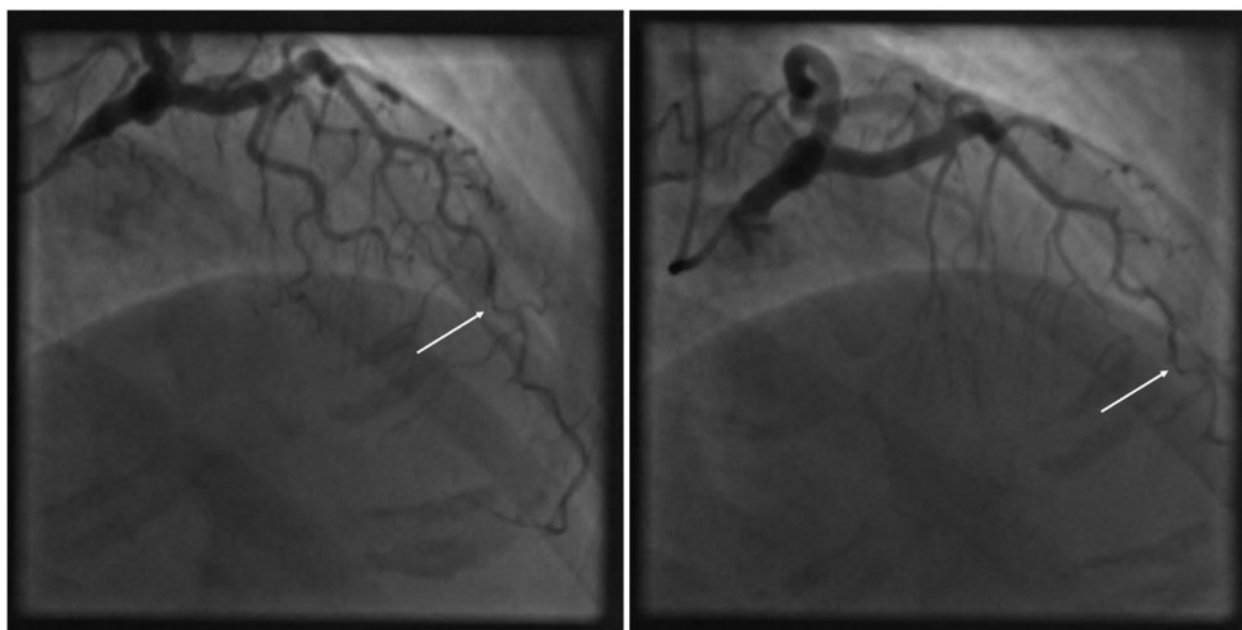
## Discussion

Spontaneous coronary artery dissection accounts for 1–4% of all ACS presentations and 25% of those in women <50 years.<sup>1</sup> It is defined by the spontaneous separation of the coronary arterial wall, creating a false lumen that is neither atherosclerotic or iatrogenic in origin. Resulting haematoma leads to compression or obstruction of the true lumen with ensuing ischaemia or infarction.<sup>6</sup> The vast majority of cases occur in women, with cardiovascular risk factors often absent. Spontaneous coronary artery dissection is also the most common cause of acute MI in pregnancy, though only 2.4–9% of presentations occur in this cohort.<sup>1</sup>

Spontaneous coronary artery dissection is thought to result from predisposing factors combined with acute stressors, most often physical or emotional. The association with fibromuscular dysplasia (FMD) was first reported in 2005 and non-coronary FMD is now recognized in up to 86% of SCAD patients investigated.<sup>2,4,7</sup> Fibromuscular dysplasia is idiopathic and most common in females aged 20–60. Evidence to suggest it is inherited is scarce.<sup>1</sup> It affects medium-sized arteries—typically renal, iliac, and carotid—making them susceptible to dissection. Medial fibroplasia and abnormal thickening of the vessel wall lead to stenosis, aneurysm formation, and a classical 'string in bead' appearance, which can be identified on imaging.<sup>1,7</sup> Arterial dissection, most frequently carotid, is seen in a quarter of patients with FMD: headache in SCAD patients should therefore always raise suspicion. However, concomitant dissections in

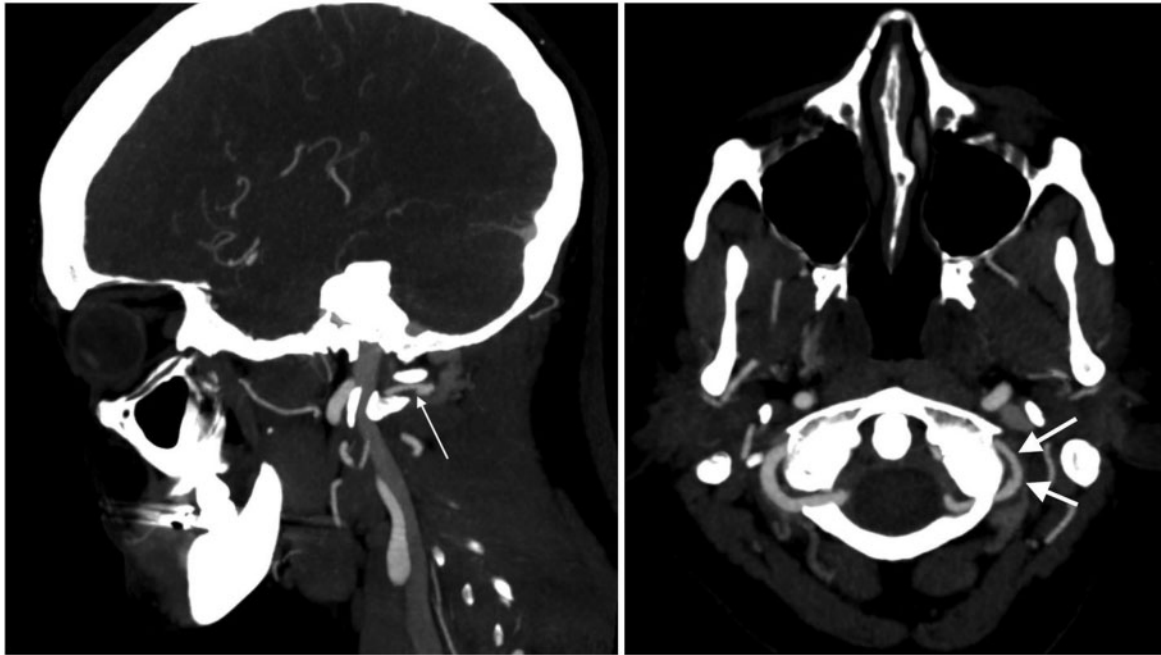


**Figure 1** Admission 12-lead electrocardiogram.



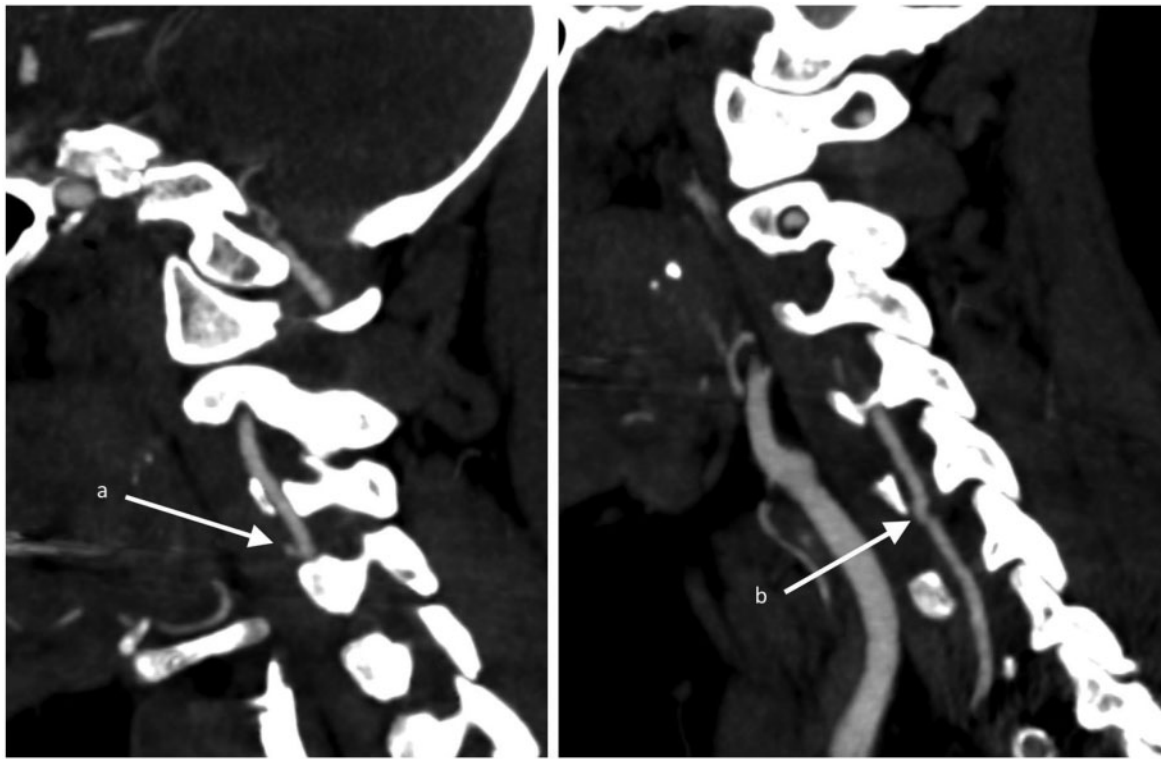
Pictures demonstrating SCAD of the LAD with abrupt change in arterial calibre.

**Figure 2** Spontaneous coronary artery dissection of the left anterior descending artery.



Arrows depicting vertebral artery dissection in sagittal and axial planes.

**Figure 3** Left vertebral artery dissection.



a) Aneurysm at level of C4 and b) Focal stenosis at level of C5

**Figure 4** Additional left vertebral artery abnormalities.

coronary and non-coronary vascular beds are rare.<sup>2,4</sup> Of note, the presence of established infarcts on MRI head in this case may represent previous subclinical dissections.

Almost all patients with SCAD present with chest discomfort and elevated cardiac enzymes, with ST-segment elevation on ECG in up to 50%. Ventricular arrhythmia, cardiogenic shock, and sudden cardiac death can also occur.<sup>1</sup> Invasive coronary angiography is the investigation of choice, but is limited by lack of direct visualization of the arterial wall: dedicated intracoronary imaging with optical coherence tomography or intravascular ultrasound can be considered for further assessment, or if the diagnosis is uncertain.<sup>6</sup> Vasospasm should also be excluded by the administration of intracoronary vasodilators.

Angiographically, SCAD is categorized into three types: type 1 (pathognomonic) consists of a double lumen; type 2 is the most common (>60%) and consists of an abrupt change in vessel calibre with a long smooth stenosis; type 3 exhibits focal arterial stenosis, which must be distinguished from atherosclerotic disease.<sup>1</sup> Dissections are usually in mid to distal vessels, with <10% proximal, and are often long (mean length in one review 45 mm).<sup>6</sup> Single vessel dissection of the LAD is most common (>50% cases) and multi-vessel coronary involvement is rare.<sup>1,2</sup> Non-affected coronaries typically appear smooth and disease free. Left ventricular function is preserved or stunned and, in these cases, normally improves with vessel healing.<sup>6,7</sup>

Expert consensus is that conservative management is preferred and observational data have demonstrated spontaneous arterial healing in 70–97%: urgent revascularisation is required in up to 14% of patients.<sup>1,8</sup> However, percutaneous coronary intervention (PCI) carries risk of extending the dissection and often has poor success rates (36–72%).<sup>8</sup> It should therefore be reserved for high-risk circumstances, for example: left main dissection, ongoing ischaemia, and haemodynamic instability. Surgery should be considered in specific situations where PCI is not possible or has failed.<sup>6–8</sup> Medical therapy with single or dual antiplatelets is generally recommended, though the optimal duration of treatment is uncertain.<sup>8</sup> Beta-blockers are often advised and reduce arterial wall stress, though there are some concerns they may exacerbate vasospasm.<sup>8</sup> Angiotensin-converting enzyme inhibitors should be considered with impaired LV function or underlying FMD and Statins are advisable with dyslipidaemia.<sup>1</sup>

Overall, in-hospital mortality is low, at <5%, but the incidence of major adverse cardiovascular events at 2 years is significant (10–30%).<sup>6,8</sup> This is mostly driven by recurrence of SCAD, for which severe coronary tortuosity is a predictor.<sup>6</sup> Atypical chest pains, not accompanied by new angiographic findings, are also common. Patients should undergo cardiac rehabilitation, modification of any underlying risk factors, and be closely followed up by appropriate specialists. Hormonal therapy should ideally be avoided and any future pregnancies should be treated as high risk. Screening for FMD or predisposing arteriopathy should be performed: invasive or non-invasive imaging may be used; however magnetic resonance angiography may be preferable over CT imaging in young patients who often require repeated investigation. Routine cerebrovascular

imaging is not standard but should be considered due to the high prevalence of intracranial aneurysms in SCAD patients (14–20%), as is highlighted by this case.<sup>1,6</sup>

## Lead author biography



Lynsey Hewitson is a Cardiology Fellow at Southmead Hospital, Bristol. She graduated from the University of Edinburgh Medical School in 2013 and has since completed both Foundation and Core Medical Training in the Severn Deanery.

## Supplementary material

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

**Slide sets:** A fully edited slide set detailing these cases 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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