

CASE REPORT

INTERMEDIATE

CLINICAL CASE SERIES

# Atherosclerotic Spontaneous Coronary Artery Dissection



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## ABSTRACT

Spontaneous coronary artery dissection is a nontraumatic, noniatrogenic intimal tearing of the coronary artery in patients without atherosclerotic coronary disease. We present 3 unique cases of patients with spontaneous coronary artery dissection and atherosclerotic coronary artery disease. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2023;10:101732) © 2023 Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Spontaneous coronary artery dissection (SCAD) is defined as a nontraumatic, noniatrogenic separation of the coronary arterial wall and is an emerging cause of acute myocardial infarction (MI). The conventional definition of SCAD refers to nonatherosclerotic disease because coronary dissection in patients with atherosclerotic coronary artery disease (CAD) is mechanistically distinct in etiology and management.<sup>1</sup> Patients with plaque dissection of CAD are typically treated with revascularization. However, SCAD can also occur in patients with underlying atherosclerotic CAD in the nondiseased coronary segments, in which case the management is similar to conventional SCAD, with preference for conservative therapy. It can be challenging to distinguish between atherosclerotic plaque dissection and

conventional SCAD, especially if there is evidence of atherosclerotic disease in nondissected segments. We have excluded patients with atherosclerotic CAD in contemporary SCAD registries as to have a uniform cohort to define the natural history of conventional SCAD.<sup>2</sup> We hereby describe 3 cases of SCAD in patients with concomitant atherosclerotic CAD because there is clinical uncertainty on etiology and management.

## CASE 1

A 68-year-old man with hypertension, dyslipidemia, and paroxysmal atrial fibrillation presented with exertional chest pain. He had a history of non-ST-segment elevation myocardial infarction 9 years prior, with angiography showing occluded small diagonal, and minor left anterior descending (LAD) atherosclerosis that was treated medically (**Figure 1**).

On presentation, his heart rate was 60 beats/min and blood pressure was 115/60 mm Hg. Electrocardiography showed new anterolateral T-wave inversions. Peak high-sensitivity troponin was 120 ng/L (normal <76 ng/L). He underwent coronary angiography, which showed diffuse type 1 SCAD from

## LEARNING OBJECTIVES

- To understand pathophysiology and clinical presentation of patients with SCAD with atherosclerotic CAD.
- To discuss management and treatment strategies for SCAD with atherosclerotic CAD.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

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**ABBREVIATIONS  
AND ACRONYMS****CAD** = coronary artery disease**DAPT** = dual anti-platelet therapy**LAD** = left anterior descending coronary artery**MI** = myocardial infarction**PCI** = percutaneous coronary intervention**SCAD** = spontaneous coronary artery disease

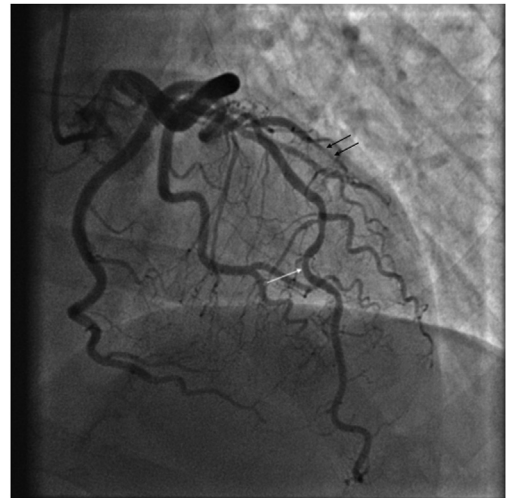
mid to distal LAD with subtotal occlusion (Figure 2, Video 1). The previously occluded diagonal artery in retrospect was a missed SCAD, which had healed on this repeat angiogram. Iliac and renal angiography showed no evidence of arteriopathy. Left ventricular ejection fraction was 60% with antero-apical hypokinesis on ventriculogram.

He was treated conservatively with bisoprolol, perindopril, and dual anti-platelet therapy (DAPT) because the LAD disease was thought to be SCAD. However, repeat coronary angiogram at 9 months for recurrent angina showed focal residual 90% and 60% mid-LAD stenosis that was due to atherosclerosis (Figure 3A). Fractional flow reserve was measured and was significant at 0.74; therefore, percutaneous coronary intervention (PCI) was performed with drug-eluting stent from proximal to mid LAD (Figure 3B).

**CASE 2**

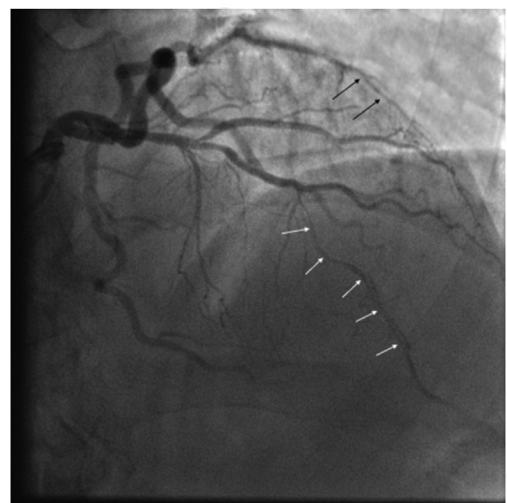
A 70-year-old woman with hypertension and dyslipidemia presented with witnessed out-of-hospital cardiac arrest after exercise at the gymnasium. She had ventricular fibrillation and required cardiopulmonary resuscitation with defibrillations and return of spontaneous circulation at 6 minutes. Peak high-sensitivity troponin was 654 ng/L (normal <51 ng/L). Coronary angiography showed type 2b SCAD in the second obtuse marginal branch with poor distal flow, and a moderate lesion in proximal LAD (Figure 4A and 4B, Video 2). Intravascular ultrasound showed the LAD lesion to be atherosclerotic with no evidence of plaque rupture or dissection (Figure 5).

A cardiac magnetic resonance imaging was performed showing left ventricular ejection fraction of 54% and mid to apical lateral wall hypokinesis. There was late subendocardial myocardial enhancement of the antero- and inferolateral wall with >50% myocardial thickness involvement and transmural late gadolinium enhancement involving the apical lateral segment. Computerized tomography scan showed fibromuscular dysplasia with beading and irregularity of the mid- and distal cervical internal carotid arteries bilaterally. She was treated conservatively with DAPT, bisoprolol, and statin and with insertion of implantable cardioverter-defibrillator. Repeat coronary angiography at 9 months showed spontaneous healing of the second obtuse marginal and right posterior descending artery, and unchanged LAD disease

**FIGURE 1** Case 1 Presentation 9 Years Prior

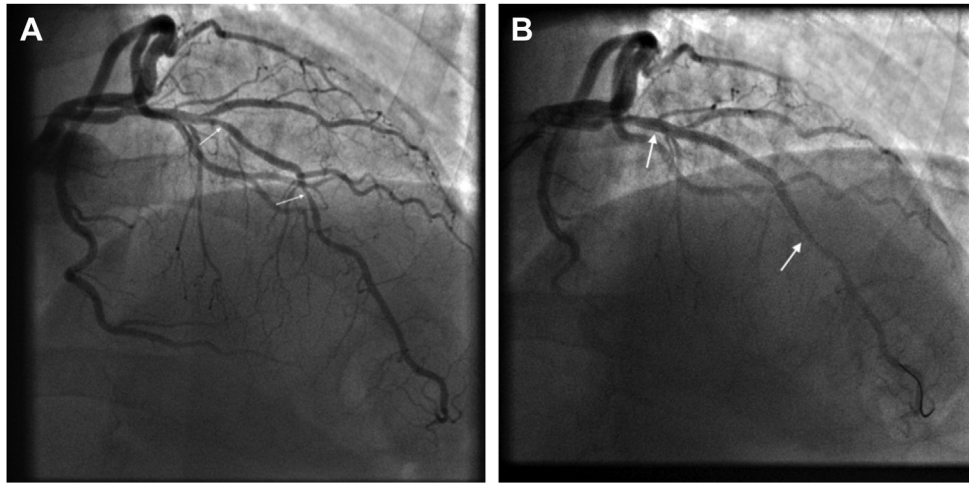
Occluded diagonal artery (black arrows). Minor LAD disease (white arrow). LAD = left anterior descending.

(Figure 6A and 6B). Renal angiogram showed right renal fibromuscular dysplasia with distal dissection and aneurysm (Figure 7, Video 3). She remained clinically well on medical therapy.

**FIGURE 2** Case 1, Second Presentation

Diffuse Type 1 SCAD from mid to distal LAD (white arrows). Healed diagonal artery (black arrows). SCAD = spontaneous coronary artery disease; other abbreviation as in Figure 1.

**FIGURE 3** Case 1, Repeat Angiogram 9 Months After 2nd Presentation



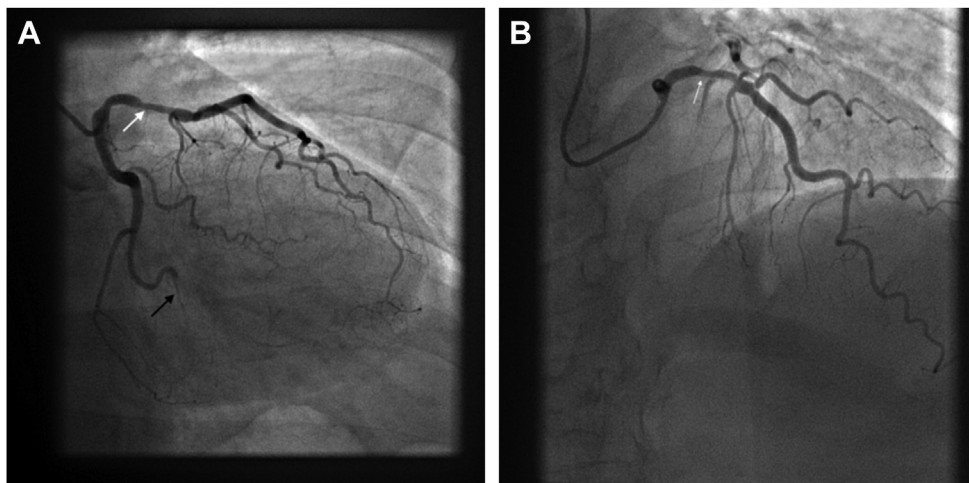
(A) Significant LAD stenosis from atherosclerotic disease (white arrows). (B) Post PCI and drug-eluting stent to proximal to mid LAD (white arrows). PCI = percutaneous coronary intervention; other abbreviation as in Figure 1.

### CASE 3

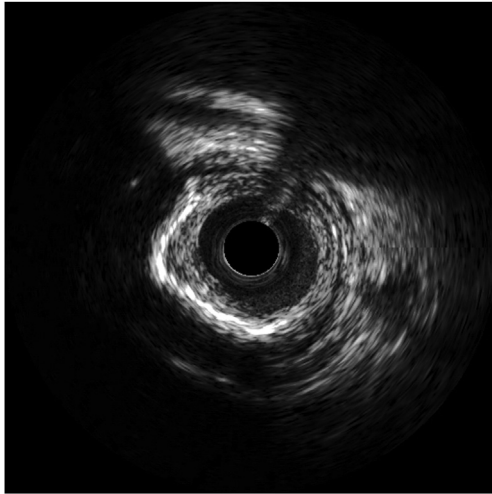
A 64-year-old physically active woman with hypertension presented with 15 hours of intermittent substernal chest pain to a community hospital. Initial

vitals were blood pressure 206/100 mm Hg, heart rate 77 beats/min, oxygen saturation of 98% on ambient air, and afebrile at 36.4 °C. Electrocardiography showed subtle anterior ST-segment elevation with peak troponin-I of 11.7 µg/L (<0.05 µg/L). Coronary

**FIGURE 4** Case 2 Angiogram



(A) Type 2b SCAD in second obtuse marginal branch (black arrow) and LAD lesion (white arrow). (B) Moderate proximal LAD lesion (white arrow). Abbreviations as in Figures 1 and 2.

**FIGURE 5 Case 2 Intravascular Ultrasound**

Cross-section of LAD lesion using intravascular ultrasound showing calcification indicated by high-intensity signal in the media from 5-11 o'clock. Abbreviation as in [Figure 1](#).

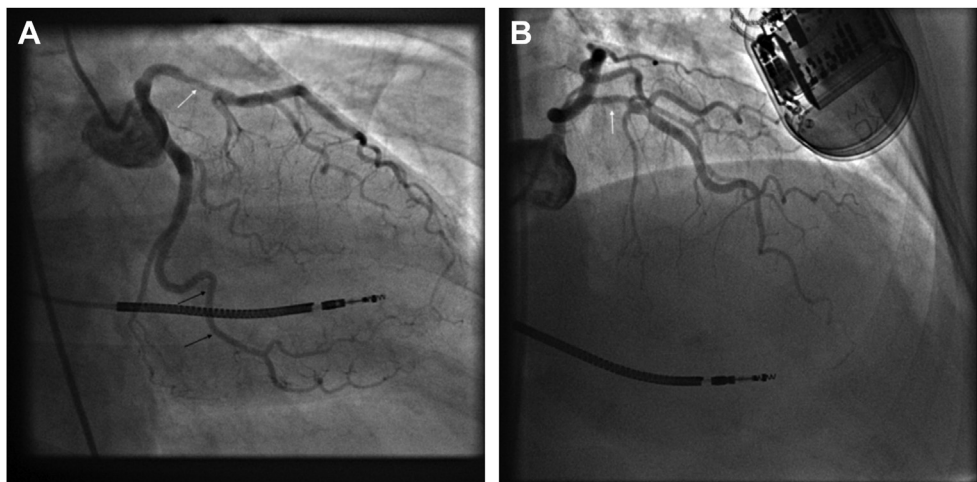
angiogram showed type 2b dissection and occlusion of apical LAD ([Figure 8](#)). There was also a separate 60% stenosis in the proximal LAD with optical coherence tomography showing atherosclerosis ([Figure 9](#)), which was treated with PCI. The apical LAD SCAD was treated conservatively with

DAPT for 1 year, along with bisoprolol, candesartan, and statin.

## DISCUSSION

SCAD with concomitant atherosclerotic disease is distinct from atherosclerotic plaque dissection because plaque dissections are typically limited in length due to medial atrophy and scarring. The underlying mechanism is not fully understood; it has been suggested that vessels with developing atheroma are prone to disruption of the vasa vasorum leading to intramural hemorrhage and dissection.<sup>3</sup> A recent retrospective observational study showed that patients with atherosclerotic coronary plaque dissections were predominantly male and more likely to have multiple cardiovascular risk factors compared with conventional patients with SCAD.<sup>4</sup> Plaque dissections are considered a variant of atherosclerotic acute coronary syndrome and should be treated with early revascularization therapy.<sup>5</sup>

However, patients with atherosclerotic CAD can also have SCAD as a cause of MI and the acute management of these patients should be similar to standard SCAD patients without atherosclerotic CAD. The majority of patients with SCAD heal spontaneously and do not require revascularization. Despite limited evidence, aspirin and beta-blockers are the mainstay of long-term treatment in SCAD.<sup>2</sup> Coronary revascularization is offered to SCAD patients with

**FIGURE 6 Case 2 Repeat Angiogram 9 Months Later**

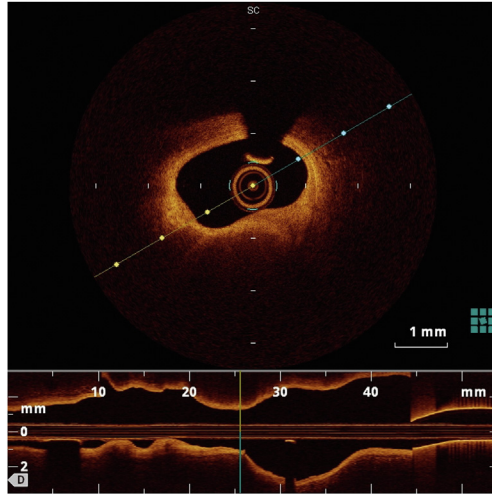
(A) Spontaneous healing of OM2 (**black arrows**), LAD disease (**white arrow**). (B) Unchanged LAD disease on repeat coronary angiogram (**white arrow**). OM2 = second obtuse marginal artery; other abbreviation as in [Figure 1](#).

**FIGURE 7** Case 2 Renal Angiogram



Renal angiogram showing FMD of right renal artery with dissection and aneurysm (**black arrow**). FMD = fibromuscular dysplasia.

**FIGURE 9** Case 3 Optical Coherence Tomography

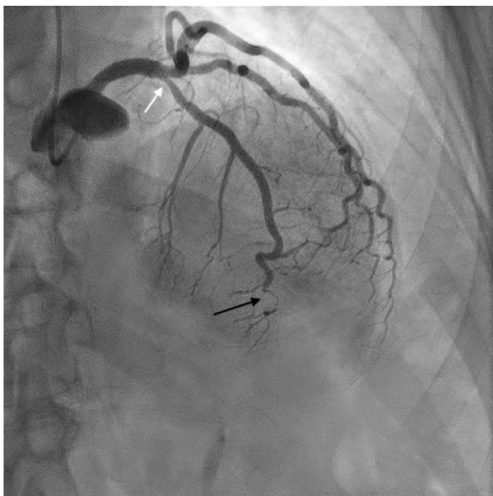


OCT showing atheroma in the proximal LAD lesion.  
OCT = optical coherence tomography; other abbreviation as in [Figure 1](#).

PCI favorable lesions and ongoing ischemia, ST-segment elevation, or hemodynamic instability.<sup>6</sup> DAPT is required for patients who underwent PCI as per guidelines.<sup>7,8</sup> There is no clear evidence for

P2Y12 inhibitors in nonrevascularized patients with SCAD. The area of intimal tear is prothrombotic, therefore, antiplatelet agents may theoretically reduce false lumen burden. Short duration DAPT with clopidogrel for 1-3 months, followed by long-term aspirin is the standard of practice at our center.<sup>9</sup> Statin administration is recommended for patients with underlying atherosclerotic CAD and pre-existing dyslipidemia.

**FIGURE 8** Case 3 Coronary Angiogram



Type 2b apical LAD SCAD (**black arrow**) and 60% stenosis of proximal LAD (**white arrow**). Abbreviations as in [Figures 1 and 2](#).

## CONCLUSIONS

Patients with atherosclerotic CAD can also present with SCAD. It is important to differentiate the etiology of the dissection because management differs. Intracoronary imaging may be required to clarify the etiology if coronary angiography is inconclusive.

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**KEY WORDS** acute coronary syndrome, atherosclerosis, dissection

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 **APPENDIX** For supplemental videos, please see the online version of this paper