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CORONARY ARTERY DISEASE

CASE REPORT: CLINICAL CASE

Spontaneous Coronary Artery Dissection With Concomitant Coronary Vasospasm

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ABSTRACT

We herein describe a case of acute myocardial infarction due to spontaneous coronary artery dissection complicated by vasospastic angina. Given the need for different clinical management strategies of these cardiac manifestations, clinicians should consider the possibility of concomitant vasospasm in cases of spontaneous coronary artery dissection. (J Am Coll Cardiol Case Rep 2024;29:102281) © 2024 The Authors. 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/).

HISTORY OF PRESENTATION

A 59-year-old woman was transferred to the emergency department from another hospital because of sudden-onset strong chest pain with ST-segment elevation in leads V_1-V_6 on electrocardiography. She had experienced mental distress for 1 week and was a current smoker with a history of 36 pack-years. Her blood pressure was 138/87 mm Hg, heart rate was 60 beats/min, and oxygen saturation was 98%. Physical examination revealed no cardiac murmur or abnormal lung sounds.

LEARNING OBJECTIVES

- To recognize the possibility of coexisting VSA in cases of SCAD with intracoronary imaging.
- To understand the utility of provocative coronary vasospasm testing for diagnosing concomitant VSA in post-SCAD patients.
- To learn an optimal therapy for preventing recurrence in patients with SCAD and VSA.

MEDICAL HISTORY

The patient had no medical history.

DIFFERENTIAL DIAGNOSIS

The initial differential diagnoses included acute coronary syndrome, acute aortic dissection, acute myocarditis/pericarditis, and Takotsubo cardiomyopathy.

INVESTIGATIONS

Electrocardiography showed ST-segment elevation in leads V_1-V_6. Laboratory examination demonstrated an elevated troponin T concentration of 0.150 ng/mL (reference range: ≤0.014 ng/mL) and normal creatine kinase concentration (142 U/L). Chest radiographs showed no abnormalities. Transthoracic echocardiography exhibited apical hypokinesis with a preserved left ventricular ejection fraction. These abnormalities supported a working diagnosis of acute coronary syndrome, and emergency cardiac catheterization was performed.

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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

LAD = left anterior descending artery

OFDI = optical frequency domain imaging

PCI = percutaneous coronary intervention

SCAD = spontaneous coronary artery dissection

STEMI = ST-segment elevation myocardial infarction

VSA = vasospastic angina

MANAGEMENT

Emergency coronary angiography after intracoronary administration of isosorbide dinitrate revealed an occlusion of the mid left anterior descending artery (LAD) (**Figure 1A**, Video 1). No significant stenosis was present in the right coronary artery. The patient was diagnosed with ST-segment elevation myocardial infarction (STEMI) and immediately underwent percutaneous coronary intervention (PCI). A guidewire with a microcatheter was smoothly advanced into the distal LAD, and coronary blood flow was restored to thrombolysis in myocardial infarction grade 2 (Videos 2 and 3). Optical frequency domain imaging (OFDI) revealed an intimal tear in the distal LAD, vasa vasorum proliferation in the mid-to-distal LAD, and medial dissection in the mid LAD (**Figure 1B to 1G**, Video 4). There was no atherosclerotic plaque in the mid-to-proximal LAD (**Figure 1H**, Video 4), suggesting that the etiology of STEMI was type 4 spontaneous coronary artery dissection (SCAD) due to a total occlusion at the initial angiography. Given the disturbed coronary flow, a drug-eluting stent was deployed to cover the severely distorted segment (**Figure 2A**). The intimal tear was treated conservatively because of the small and tortuous vessel. OFDI displayed the stent covering almost part of the medial dissection, and the residual uncovered dissection did not require additional stent deployment due to thrombolysis in myocardial infarction grade 3 blood flow (**Figure 2B**, Videos 5 and 6). The maximum postprocedural creatine kinase concentration was 780 U/L, and intravenous administration of heparin and nicorandil was continued. On hospital day 4, chest pain with mild ST-segment elevation suddenly occurred after the discontinuation of nicorandil and was completely relieved immediately after sublingual administration of nitroglycerin. This episode suggested a complication of vasospastic angina (VSA), and, after the initiation of a calcium channel blocker (benidipine 4 mg), the chest pain did not recur. Cardiac computed tomography on hospital day 11 revealed preserved coronary flow in the LAD, and the patient was discharged on hospital day 13. She remained asymptomatic with cessation of smoking and treatment with calcium channel blocker. Three months after the initial hospitalization, repeat coronary angiography performed to investigate VSA showed partial restoration of the SCAD in the LAD (**Figure 3A**, Video 7). An acetylcholine provocation test also revealed diffuse stenosis of the mid-to-distal LAD with chest pain and terminal T-wave inversion in leads II, III, aVF, and V_3-V_6 (**Figure 3B**, Video 8), which was completely restored after intracoronary isosorbide dinitrate (**Figure 3C**, Video 9). OFDI in the LAD displayed resolution of the intimomedial dissection and medial thickening, mild stent malapposition, and mild tissue growth in the proximal edge of the stent (**Figure 4A to 4C**, Video 10). The patient remained asymptomatic more than 1 year after the initial hospitalization.

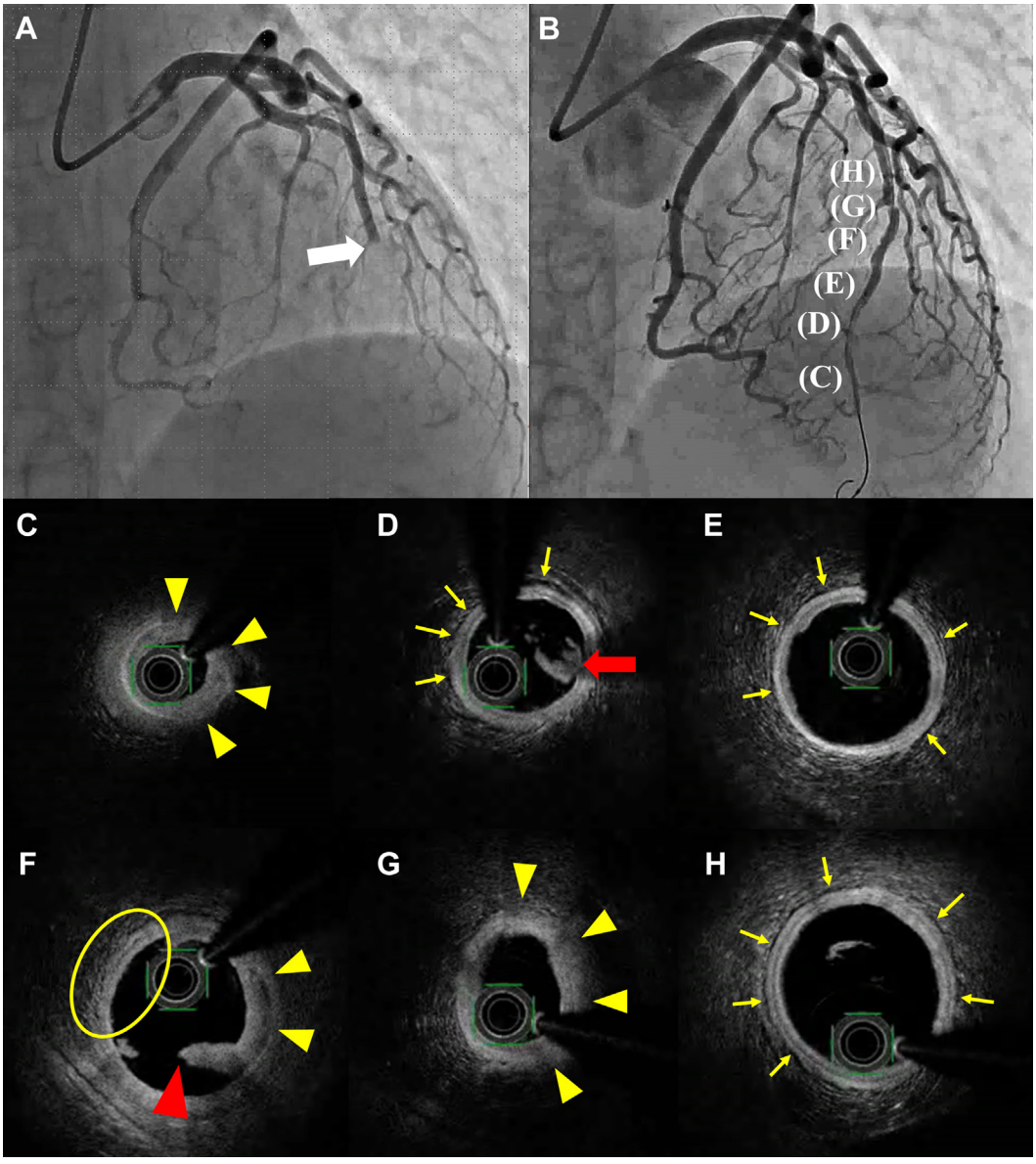
DISCUSSION

SCAD is a rare cause of myocardial infarction and mainly affects women.[1] According to multicenter registries, 26.8% of SCAD patients are type 4, characterized by complete occlusion due to intramural hematoma and predominantly presenting with STEMI,[2] which is challenging to diagnose without intracoronary imaging. Most patients are treated conservatively, whereas patients with ongoing ischemia undergo PCI,[1] as in the present case.

VSA is characterized by hyper-reactivity of coronary vascular smooth muscle cells and endothelial dysfunction.[3] The acetylcholine provocation test is the gold standard for diagnosing VSA.[3] However, recent technical advancements in intracoronary imaging have revealed VSA-specific morphologic findings including enhanced adventitial vasa vasorum formation and intraplaque neovessels,[4] medial thickening, and layered plaque,[3] which could help identify episodes of VSA. In this case, OFDI during the primary PCI demonstrated coronary artery dissection with these VSA-specific features (**Figures 1C to 1H**). Although these imaging characteristics, particularly medial thickening, should be cautiously interpreted in the acute phase of SCAD due to some biases generated from the primary parietal bleeding in the adventitia and media, intracoronary imaging in this case suggested coexisting VSA in the culprit LAD and highlighted the need for vasoreactivity study.

Several cases of SCAD complicated by VSA have been reported.[5,6] In fact, SCAD and VSA may share certain risk factors, including middle age, emotional stress, and endothelial dysfunction.[7] However, few studies have confirmed vasospasm in the culprit vessel of SCAD by vasoreactivity test. In a previous study involving the ergonovine provocation test in 11 patients with a history of SCAD, only 1 patient had a positive vasospasm response in the culprit vessel.[5] Another study demonstrated that the coronary

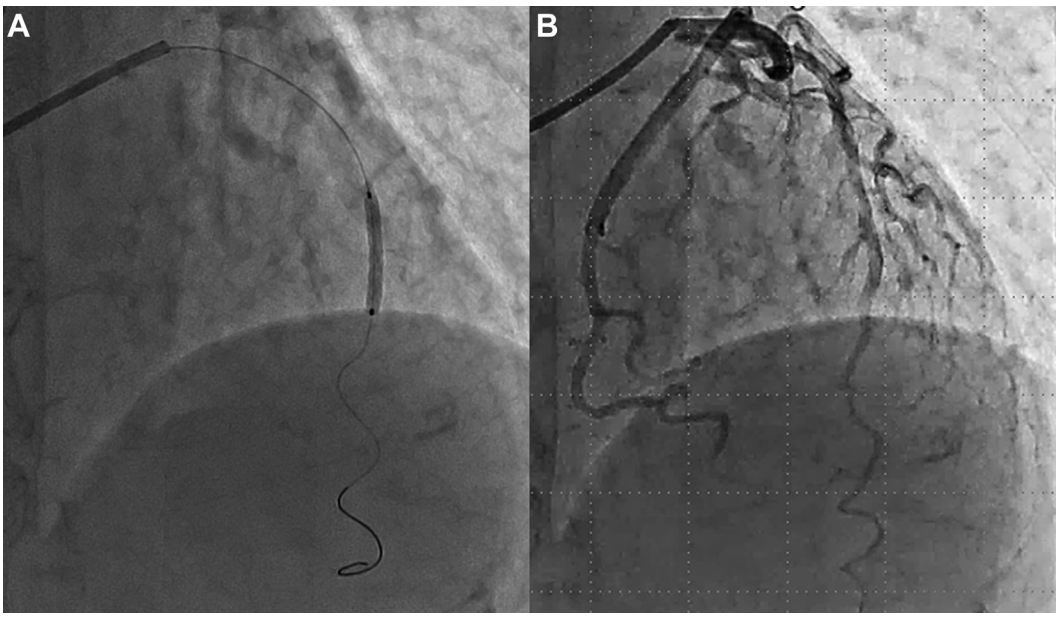
FIGURE 1 **Emergency Left Coronary Angiography and OFDI of LAD at Initial Admission**



(A) Total occlusion of the mid LAD (white arrow). (B) OFDI of the LAD after recanalization, demonstrating (C) medial thickening at the far distal site (yellow arrowhead), (D) an intimal tear at the distal LAD (red arrow) with enhanced vasa vasorum formation (yellow arrows), (E) vasa vasorum proliferation (yellow arrows) in the mid-to-distal LAD, (F) medial dissection (red arrow) with concomitant medial thickening (yellow arrowheads) and increased microvessels (yellow circle) in the mid LAD, (G) medial thickening at the proximal site of the dissection (yellow arrowheads), and (H) a healthy coronary artery with enhanced vasa vasorum formation (yellow arrows) in the mid-to-proximal LAD. LAD = left anterior descending artery; OFDI = optical frequency domain imaging.

epicardial and microvascular responses to acetylcholine, adenosine, and nitroglycerine were comparable between patients with and without SCAD.[8] These results suggest that coronary epicardial and microvascular vasomotor dysfunction may not be a predominant feature of SCAD; however, a complication of VSA could manifest as a phenotype of SCAD. Such patients might ideally be treated using calcium

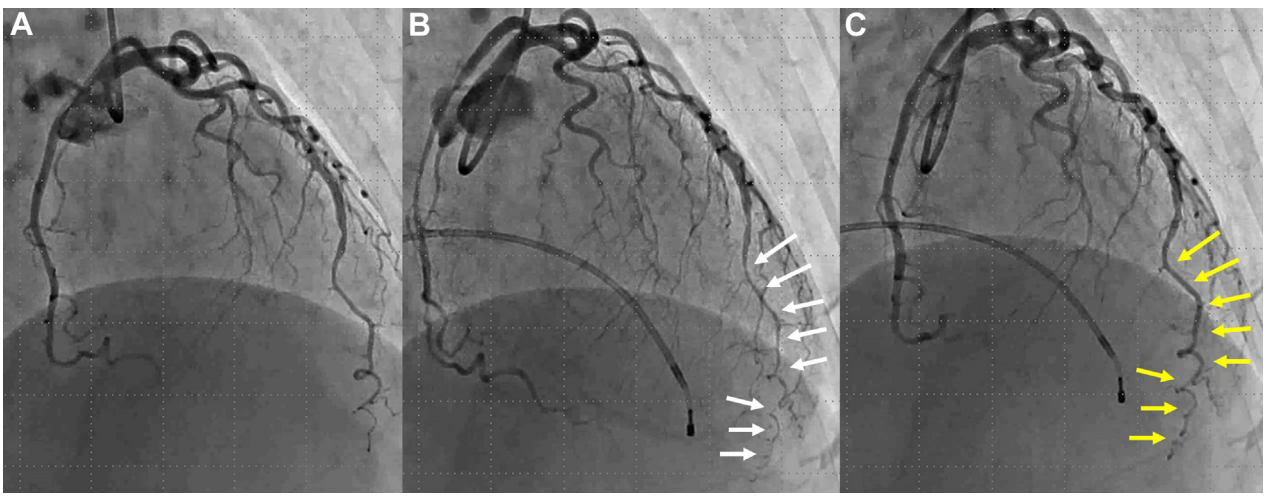
FIGURE 2 Emergency Revascularization of Culprit Lesion



(A) A drug-eluting stent was deployed to cover the severely distorted segment. (B) Final angiography revealed restoration of coronary blood flow to the distal LAD. Abbreviation as in Figure 1.

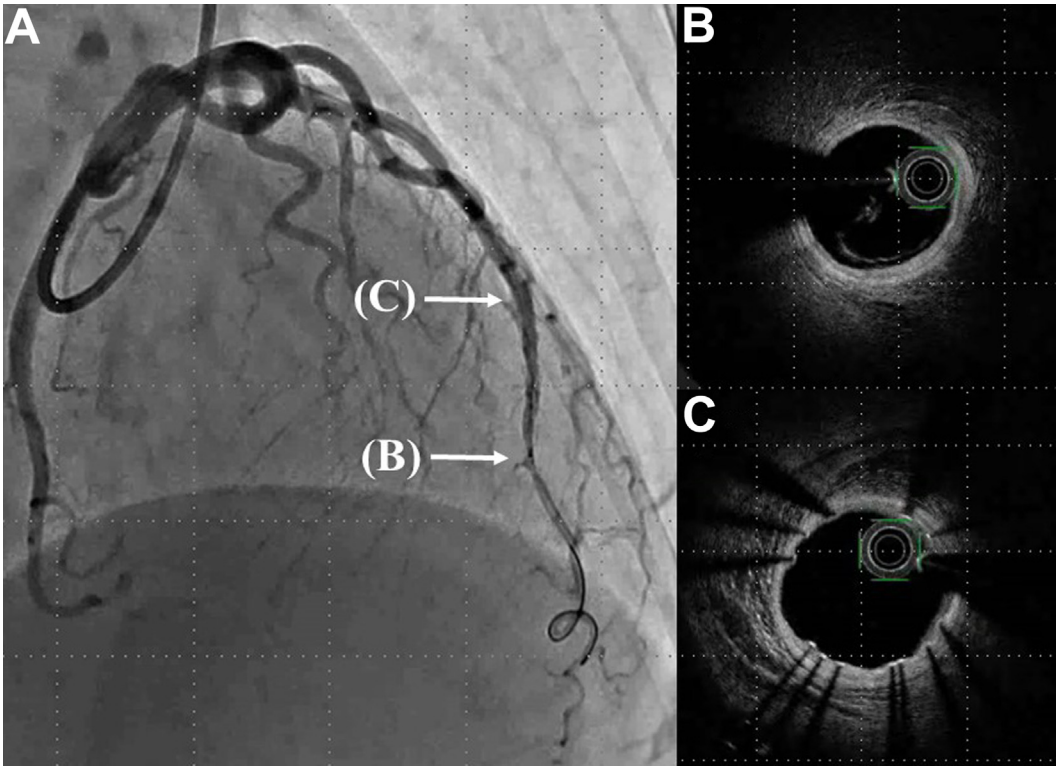
channel blockers because β blockers, predominantly prescribed in patients with SCAD,[1] may lead to worsening of coronary vasoconstriction in patients with VSA. Accordingly, clinicians should consider the possibility of coexisting VSA in patients with SCAD and investigate VSA using coronary vasospasm testing in suspicious cases, which requires further investigation.

FIGURE 3 Acetylcholine Provocation Test 3 Months After Prior Myocardial Infarction



(A) Left coronary angiography showing partial restoration of the dissection of the LAD. (B) Acetylcholine provocation test revealing diffuse stenosis of the mid-to-distal LAD (white arrows). (C) Complete restoration of vasospasm after intracoronary isosorbide dinitrate (yellow arrows). Abbreviation as in Figure 1.

FIGURE 4 **OFDI in LAD 3 Months After Initial Hospitalization**



(A) Follow-up OFDI in the LAD, displaying (B) resolution of the intimal tear and (C) the medial dissection covered with a stent. Abbreviations as in Figure 1.

FOLLOW-UP

The patient remained free of symptoms and cardiovascular events with long-term administration of calcium channel blocker for more than 1 year after the prior STEMI.

CONCLUSIONS

SCAD can coexist with VSA because of several shared risk factors. Given the need for different clinical management strategies, clinicians should recognize that SCAD can be complicated by VSA.

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KEY WORDS acetylcholine provocation test, optical frequency domain imaging, spontaneous coronary artery dissection, vasospastic angina

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