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**CASE REPORT**
*Coronary intervention*

# Severe multifocal coronary artery spasms after cessation of vasodilators in a patient with a spontaneous coronary artery dissection: a case report

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**Background** Vasospastic angina (VSA) and spontaneous coronary artery dissection (SCAD) are challenging causes of non-atherosclerotic acute coronary syndromes (ACS). Here, we report a unique ACS case with coexisting VSA and SCAD, highlighting specific strategies in diagnosis and management of these poorly studied conditions.

**Case summary** A woman in her mid-60s with a history of suspected microvascular angina and no atherosclerosis in a previously performed coronary computed tomography angiography presented with worsening chest pain. Invasive coronary angiography revealed a focal SCAD with a resulting high-degree stenosis of the right coronary artery. Shortly after successful percutaneous coronary intervention with stent implantation and stopping her previous vasodilator therapy with nitroglycerine and molsidomine, the patient developed recurrent anterior non-ST-segment elevation myocardial infarction. Surprisingly, repeat coronary angiography revealed severe multifocal coronary artery spasms that were successfully treated with intracoronary nitroglycerine. Vasospastic angina was subsequently managed with diltiazem, molsidomine, and nitrates.

**Discussion** Our report underscores the challenges in diagnosing and managing SCAD and VSA in ACS. The possible interplay between SCAD and VSA highlights the need for careful vasodilator therapy management, as seen in our patient, where therapy discontinuation led to severe multifocal VSA. This emphasizes the need for a comprehensive approach for optimal outcomes in complex ACS cases.

**Keywords** Acute coronary syndrome • Case report • Coronary angiography • Coronary artery spasm • MINOCA • Spontaneous coronary artery dissection

**ESC curriculum** 3.1 Coronary artery disease • 3.2 Acute coronary syndrome • 3.4 Coronary angiography

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## Learning points

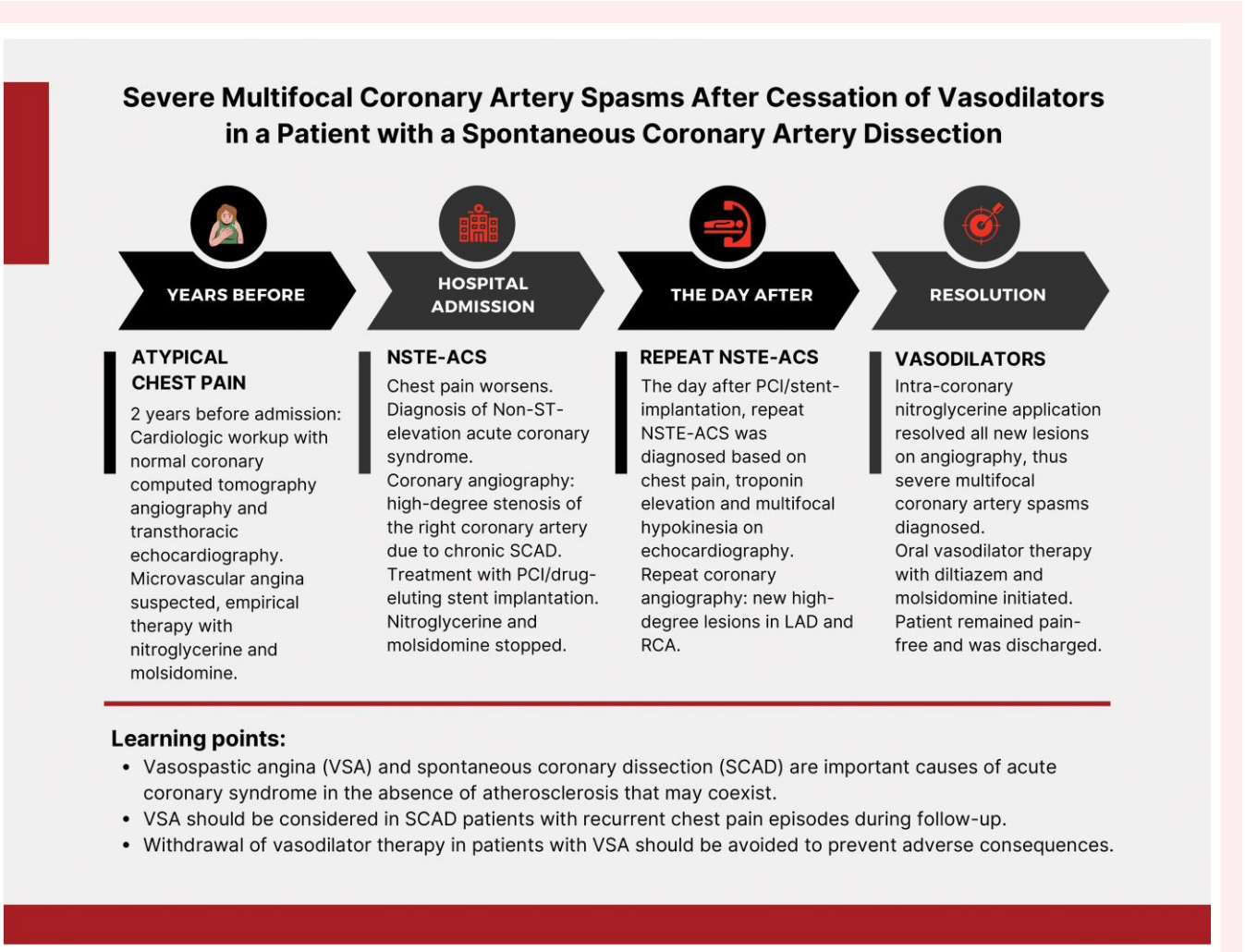
- Vasospastic angina (VSA) and spontaneous coronary dissection (SCAD) are important causes of acute coronary syndromes in the absence of atherosclerosis that may coexist.
- Vasospastic angina should be considered in SCAD patients with recurrent chest pain episodes during follow-up.
- Withdrawal of vasodilator therapy in patients with suspected VSA should be avoided to prevent adverse consequences.

# Introduction

Recently, non-atherosclerotic causes of acute coronary syndromes (ACS), such as vasospastic angina (VSA) and spontaneous coronary dissection (SCAD), have gained greater awareness. Although they comprise 5–10% of ACS cases[1] and are often overlooked in younger ACS patients without traditional risk factors,[1,2] the pathophysiology and management of VSA and SCAD remain poorly studied. However, increased use of enhanced diagnostic methods, including invasive functional coronary assessment and imaging techniques such as optical coherence tomography (OCT),[3] intravascular ultrasound (IVUS),[4] and coronary computed tomography angiography (CCTA)[5,6] may improve diagnostic accuracy particularly in patients with ambiguous culprit lesions.[7]

Increasing evidence indicates that VSA and SCAD share identical triggers such as cocaine, amphetamines, 5-fluorouracil, sumatriptan, and other prescription drugs, and indeed, coronary spasms may even provoke SCAD.[8–10] We present a case of a woman with severe multivessel coronary artery VSA following treatment of an ACS due to SCAD, illustrating the coexistence and the challenges in diagnosing and managing these conditions.

# Summary figure



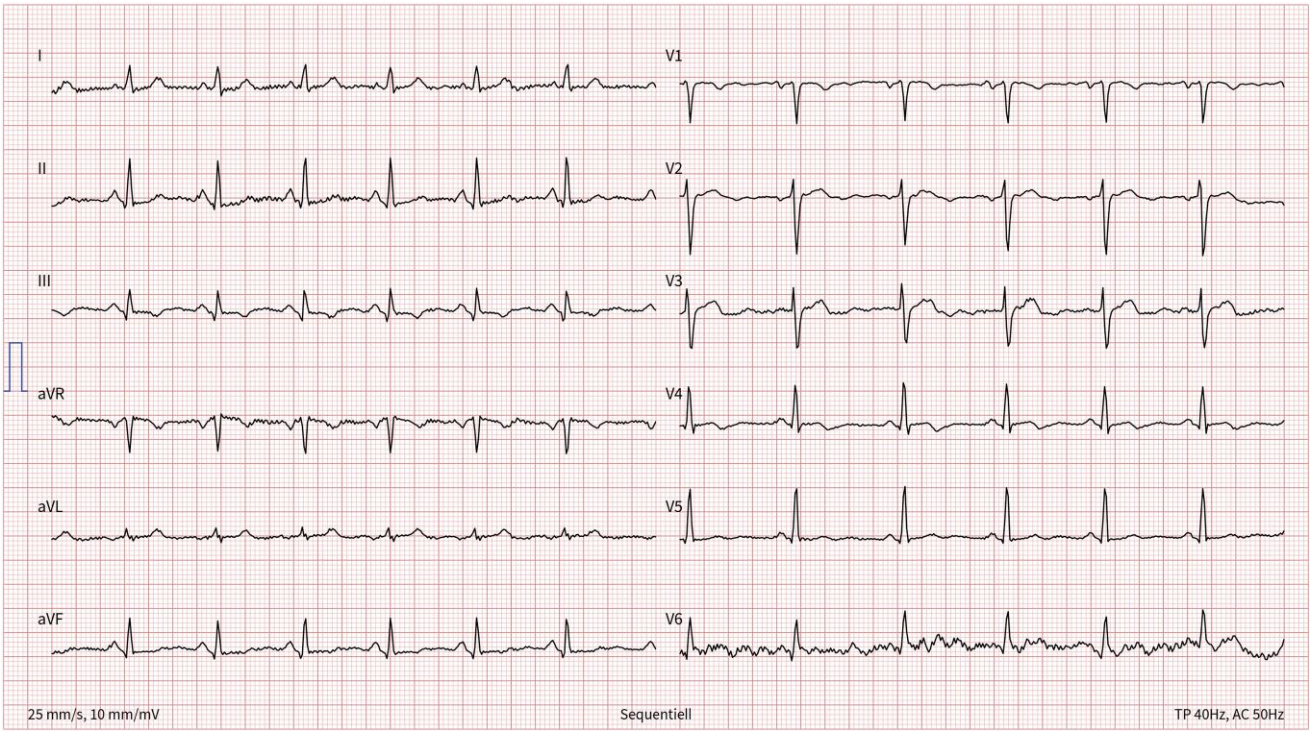



**Figure 1** Twelve-lead electrocardiogram at presentation, showing normal sinus rhythm and subtle ST-segment changes in the inferior leads.

## Case presentation

A woman in her mid-60s with no traditional cardiovascular risk factors presented to the emergency department with worsening chest pressure radiating to her left arm and nausea. She reported intermittent non-exertional chest pain for years, and a CCTA 2 years prior excluded coronary atherosclerosis. Suspected of having microvascular angina, she was treated with transdermal nitroglycerine 2.5 mg q.d., and molsidomine 2 mg q.d. was added in the past 3 months due to worsening symptoms. Her medical history included Sjögren's syndrome and endometrial carcinoma, and she had a first-degree relative with VSA.

She presented pain-free, with normal vitals and unremarkable clinical examination. The electrocardiogram (ECG) showed subtle ST-segment changes in the inferior leads (*Figure 1*). Mildly elevated high-sensitive cardiac troponin T (hsTnT) levels were noted without dynamic change after 5 h (20.7 and 22.8 ng/L; normal < 14 ng/L). Other laboratory studies were unremarkable. A diagnosis of ACS was made.

Coronary angiography revealed a normal left coronary artery (*Figure 2A* and Supplementary material online, *Video S1*) and a 95% stenosis in the right coronary artery (RCA; *Figure 2B* and Supplementary material online, *Video S1*), with partial collateralization from the left anterior descending (LAD) coronary artery. The focal lesion appeared double contoured, with no resolution after intracoronary nitroglycerine application. In the absence of coronary atherosclerosis, we considered a chronic type 1 SCAD as the most likely diagnosis. Given the characteristic double-contoured lesion appearance, we diagnosed SCAD foregoing intracoronary imaging, which may be considered in unclear cases, but may also jeopardize vessel patency. Although revascularization is avoided in acute SCAD in the absence of ongoing ischaemia due to high complication rates and frequent spontaneous healing,[8] the assumed chronicity with already established collateralization, the focal appearance of the lesion, and the patient's recurrent symptoms prompted us to proceed with revascularization. We performed successful percutaneous coronary intervention (PCI) with implantation of one everolimus-eluting stent in the RCA's middle segment (*Figure 2C* and second half of Supplementary material online, *Video S1*). Dual antiplatelet therapy was started (aspirin 100 mg q.d. and prasugrel 10 mg q.d.), while nitroglycerine and molsidomine were discontinued. No beta-blockers were given.

The following day, a recurrent non-ST-elevation ACS was diagnosed, based on recurrent worsening chest pain, new dynamic anterior ST-segment changes on ECG (*Figure 3*), elevated hsTnT (330.4 ng/L, normal < 14 ng/L), and new severe hypokinesia in the inferior and septal apical as well as in the inferoseptal and anteroseptal midventricular segments with a reduced left ventricular ejection fraction of 45% on transthoracic echocardiography (*Figure 4* and Supplementary material online, *Video S2*). Surprisingly, repeat angiography revealed a new total occlusion of the distal LAD, ostial LAD stenosis, and subtotal stenosis of the RCA just distal to the stent implanted the day before (*Figure 5A and B*; Supplementary material online, *Video S3*). Initially, multifocal SCAD progression was feared, but repeated intracoronary nitroglycerine administration (800 µg in total) resolved all new stenoses, identifying severe coronary artery spasms (*Figures 5C and D*; Supplementary material online, *Video S4*).

Diltiazem 120 mg b.i.d. and molsidomine 8 mg b.i.d. were prescribed. Four days later, echocardiography showed improved left ventricular function (see Supplementary material online, *Video S5*). In the absence of arterial hypertension, no vascular screening for fibromuscular dysplasia was performed at that time. The patient remained pain free and was discharged. Still, finding the optimal pharmacotherapy during regular

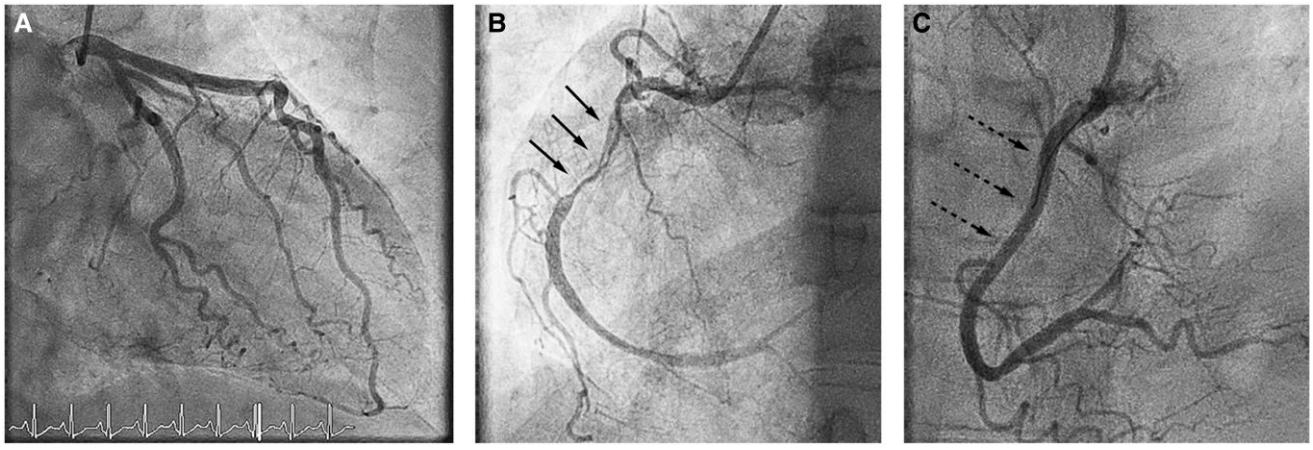



**Figure 2** Coronary angiography demonstrating a normal appearing left coronary artery (*A*). The right coronary artery shows a high-degree, double-contoured lesion in the middle segment (*B*, arrows) with peripheral collateralization from the left coronary artery. The middle segment was successfully revascularized with percutaneous coronary intervention and implantation of one everolimus-eluting stent (*C*, dashed arrows).

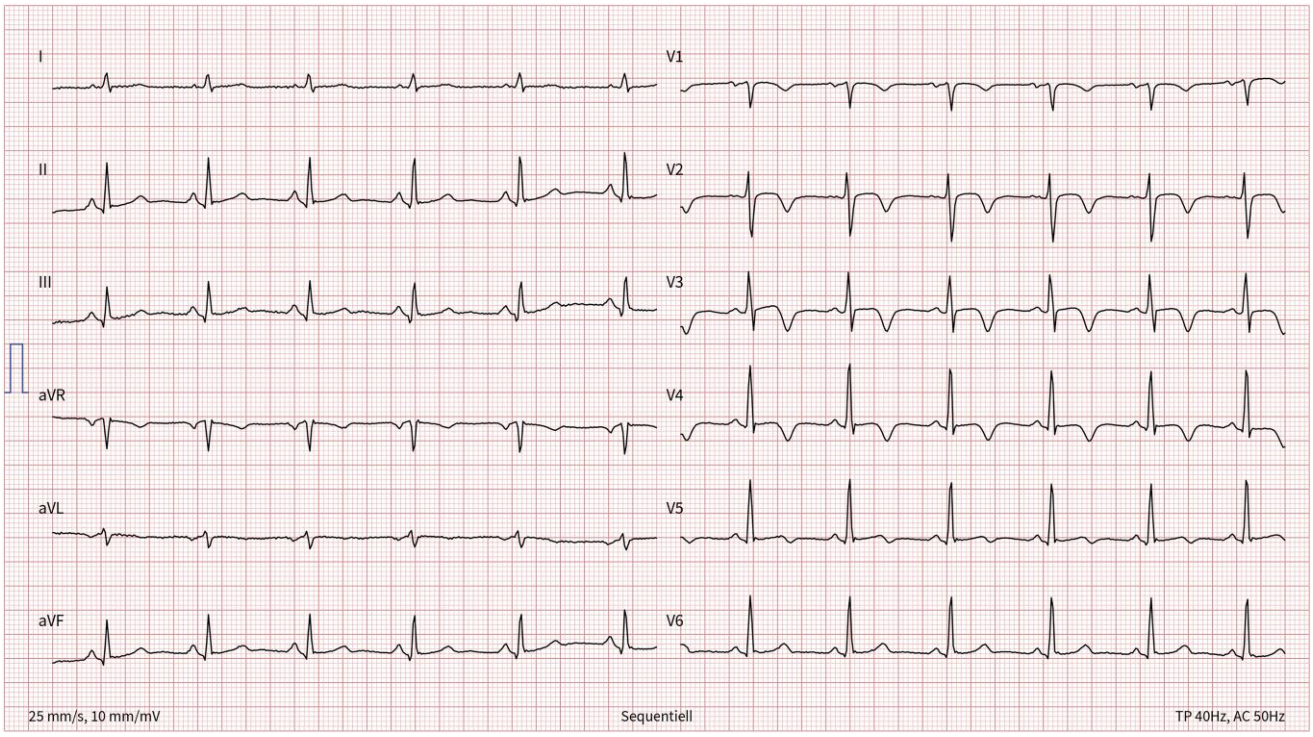



**Figure 3** During a recurrent chest pain attack on the day after percutaneous coronary intervention, a 12-lead electrocardiogram revealed dynamic ST-segment changes with T wave inversions in the anterior leads (V2–V5).

follow-up was challenging. Diltiazem was switched to sustained-release verapamil 120 mg q.d. due to recurrent VSA symptoms. During a hospitalization 5 months later due to another ACS, believed to be breakthrough VSA (with no significant ECG changes but hsTnT rise from 26 ng/L on admission to 56 ng/L 1 h later), transdermal nitroglycerine patch 5 mg q.d. was added, and molsidomine modified to 4 mg b.i.d., with symptoms resolution without performing repeat coronary angiography.

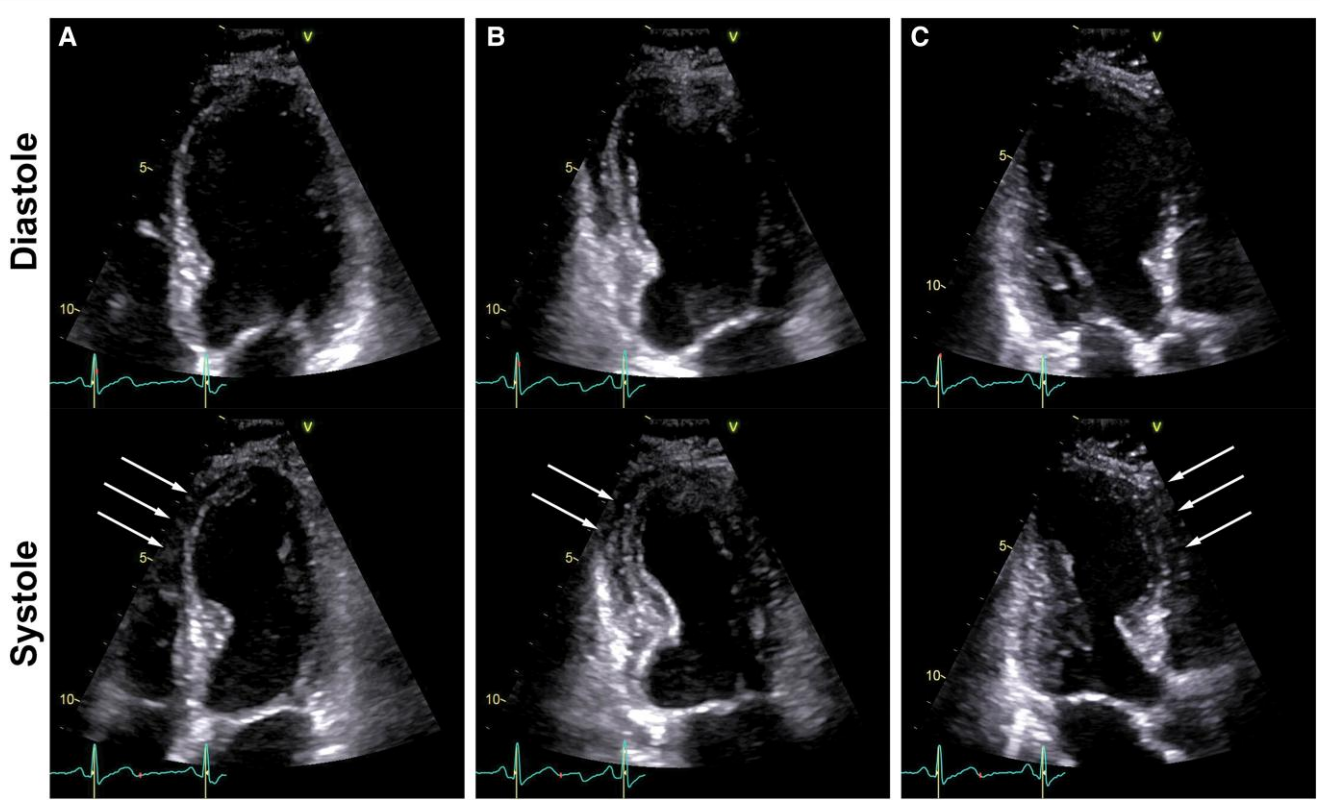



**Figure 4** Transthoracic echocardiography demonstrating severe hypokinesia (arrows) in the apical to mid-inferoseptal (*A*, apical four-chamber view), apical inferior (*B*, two-chamber view), and apical-septal to mid-anteroseptal segments (*C*, apical three-chamber view).

## Discussion

Spontaneous coronary artery dissection and VSA are significant causes of ACS, arrhythmia and sudden cardiac death, particularly in younger to middle-aged individuals without previously diagnosed ischaemic heart disease or traditional cardiovascular risk factors.[2] Our case illustrates several unique diagnostic and therapeutic challenges in these conditions.

Vasospastic angina should be strongly considered in ACS patients with a previous chronic pattern of recurrent, nitrate-responsive chest pain at rest, particularly if coronary atherosclerosis has been excluded in a prior CCTA as in our patient. Invasive functional assessment with intracoronary nitroglycerine administration is important in the VSA diagnostic algorithm, with resolution of non-catheter-induced stenosis being a diagnostic hallmark as in our patient. If VSA is suspected but unconfirmed, invasive pharmacologic provocative testing can confirm the diagnosis in experienced centers.[11]

Recently, interventional cardiologists became more aware of SCAD's angiographic characteristics. According to the Saw classification,[12] Types 1 and 2 SCAD are often identifiable angiographically, avoiding the risk of propagating vessel dissection after coronary instrumentation. In Type 3 SCAD or cases of diagnostic uncertainty, intracoronary OCT or IVUS imaging may be used.[7,13] We avoided intracoronary imaging, considering the lesion's characteristic angiographic appearance and no atherosclerosis on recent CCTA. Despite the general preference for conservative SCAD treatment, which is the preferred approach for acute SCAD in our institution, the experienced operator performing the procedure opted for PCI in this case under the impression of a chronic dissection with symptoms of unstable angina, fearing the patient would otherwise remain symptomatic. However, we acknowledge that this clinical situation might have been managed differently by other operators.

Importantly, our case illustrates the potential coexistence of SCAD and VSA in ACS, with each potentially triggering the other,[8–10,14] highlighting a possible pathophysiological link. This interplay demands cautious vasodilator management to prevent complications like re-infarction due to therapy withdrawal. In retrospect, VSA was likely the primary aetiology of our patient's symptoms as well as a potential trigger for her previous SCAD. Focusing on treatment of the high-degree RCA stenosis without considering potential other causes led to discontinuation of vasodilators post-PCI, resulting in a hazardous multi-vessel coronary vasospasm that ultimately had to be resolved by high-dose intracoronary nitrate administration.

In conclusion, our case emphasizes the need for high clinical awareness in diagnosing VSA and SCAD as underlying causes of ACS, particularly in individuals without evident epicardial coronary atherosclerosis. This requires a comprehensive diagnostic approach, incorporating functional coronary assessment and selected coronary imaging techniques in unclear cases. Moreover, the case exemplifies the critical importance of judicious management of vasodilator therapy in patients with VSA to avoid severe complications, as well as awareness of a potential coexistence of VSA in patients with SCAD.

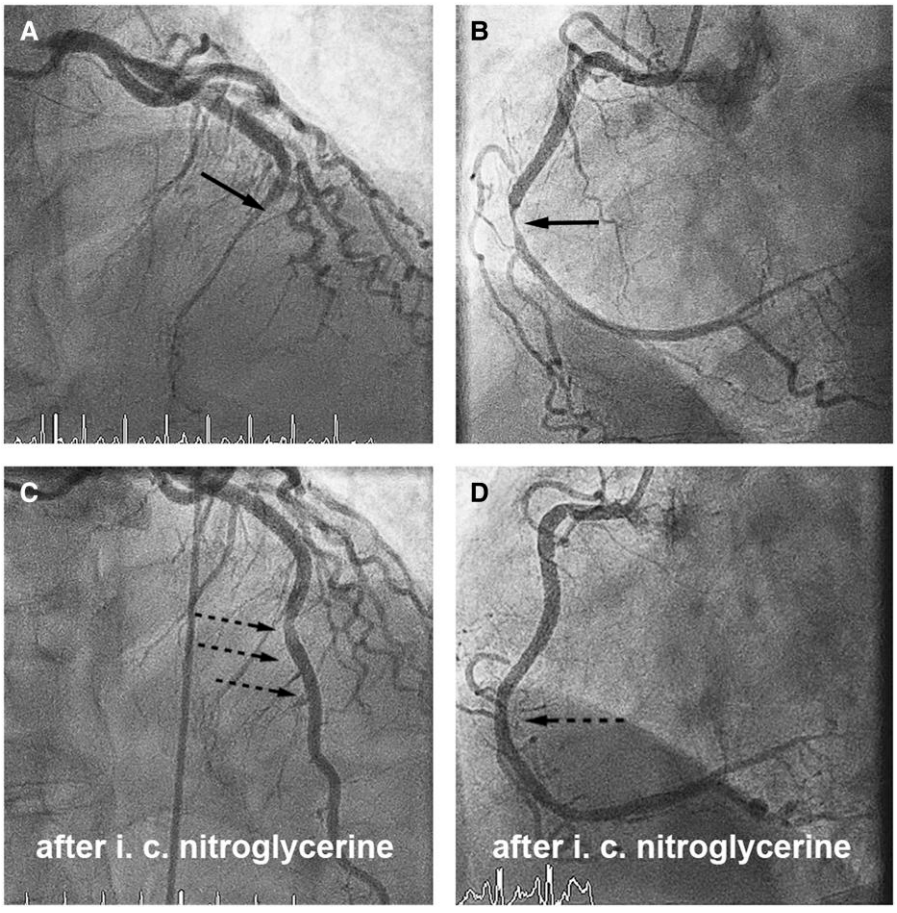



**Figure 5** Repeat coronary angiogram demonstrating a new occlusion of the distal left anterior descending artery (*A*, arrow). The right coronary artery showed a high-degree stenosis (*B*, arrow), just distal of the stent implanted the day before. After intracoronary (i.c.) application of nitroglycerine, these lesions resolved completely (*C* and *D*, dashed arrows).

## Lead author biography

Zdenek Steffek is currently working as a general cardiologist at Kantonsspital St. Gallen, Switzerland. He obtained his medical degree at the Charles University in Prague and received the majority of his clinical training in cardiology and internal medicine at the University Hospital Zurich and at the Triemli City Hospital Zurich. He aims to specialize in advanced cardiac imaging, his primary area of interest.

## Supplementary material

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

**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 accordance with COPE guidance.

**Conflict of interest:** None declared.

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## Data availability

The data underlying this article are available in the article and/or in its online Supplementary material.

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