

# Concomitant presentation of spontaneous coronary artery dissection with Takotsubo syndrome: a case report

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## Background

Spontaneous coronary artery dissection (SCAD) is still an underdiagnosed condition that requires a detailed assessment of angiographic signs. It also shares similar clinical presentations with Takotsubo syndrome (TTS). The concomitant presentation of SCAD with TTS is a possible occurrence, making it difficult for clinicians to treat and manage.

## Case summary

This study included a 49-year-old woman with retrosternal chest pain who was admitted to the emergency department. Coronary angiography indicated Type 2A SCAD involving the middle part of the left anterior descending artery, while the left ventriculography indicated a typical left ventricular apical ballooning compatible with TTS. A conservative approach to the management of SCAD was observed. After a 3-month follow-up, the control coronary angiography showed a complete angiographic resolution. The results of the transthoracic echocardiogram (TTE) and cardiac magnetic resonance revealed a complete normalization of the pathological features. The patient remained asymptomatic and showed no recurrence of chest pain.

## Discussion

Although TTS and SCAD are commonly observed in patients who share certain characteristics (women, without atheromatous terrain, stress-related factors), it is difficult to establish a pathophysiological link between them. This observation confirms the non-random association of two rare entities of myocardial infarction with no obstructive coronary arteries. Although TTS can be easily diagnosed via non-invasive imaging, the diagnosis of SCAD is more difficult. The findings of this study suggest a concomitant presentation between SCAD and TTS. Although the treatment approach to SCAD is usually conservative, severe forms of this disease require early diagnosis and appropriate treatment.

## Keywords

Case report • Spontaneous coronary artery dissection • Takotsubo syndrome • Acute coronary syndrome • Left ventricular apical ballooning

## ESC Curriculum

2.1 Imaging modalities • 2.2 Echocardiography • 2.3 Cardiac magnetic resonance • 3.4 Coronary angiography

## Learning points

- The presence of Takotsubo syndrome may be concomitant with spontaneous coronary artery dissection (SCAD)
- SCAD remains an underdiagnosed condition that requires a detailed assessment of angiographic signs.

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## Introduction

Spontaneous coronary artery dissection (SCAD) and Takotsubo syndrome (TTS) are two cardiovascular syndromes with similar clinical presentations that predominantly affect women, often occurring after a stressful emotional or physical event, and are transient with a reversion ad integrum.<sup>1</sup> It is also likely that these conditions might co-occur in some patients. In contrast, SCAD is usually common in young subjects<sup>2</sup> (mean age: 50 years), while TTS is common in women with a mean age of 67–70 years. Clinical features associated with SCAD tend to be extracoronary vascular abnormalities, such as fibromuscular dysplasia, pregnancy or postpartum, or connective tissue diseases, whereas TTS is associated with conditions of catecholamine excess<sup>3</sup> (pheochromocytoma and central nervous system disorders) and endothelial dysfunction or abnormal vasomotor function, such as in postmenopausal patients or those with migraine or Raynaud's phenomenon. The SCAD remains an underdiagnosed<sup>4,5</sup> condition that requires a detailed assessment of angiographic signs. It can be concomitant with TTS, making it difficult for clinicians to treat and manage. The aim of this case report is to review the case of a 49-year-old woman with a history of stress disorder who presented with acute chest pain and was admitted to our medical centre. Paraclinical examinations have also demonstrated the potential co-existence of TTS and SCAD.

## Timeline

Time	Events
Day 1 <i>Emergency department</i>	<p>Patient with history of smoking and very intense stress disorder admitted for retrosternal chest pain radiating to the left arm.</p> <p>The electrocardiogram (ECG) indicated sinus tachycardia with no ST-T change (elevated hs-Tn level of 5883.4 ng/L; upper normal limit &lt;15.6).</p>
<i>Cardiac catheterization laboratory</i>	<p>The coronary angiography indicated Type 2A spontaneous coronary artery dissection (SCAD) involving the middle part of the left anterior descending artery, while the left ventriculography showed the typical left ventricular apical ballooning and a hypercontractile base compatible with a Takotsubo syndrome (TTS).</p>
Day 2 <i>Cardiovascular intensive care unit</i>	<p>The cardiac magnetic resonance (CMR) showed transmural myocardial oedema in T2-weighted sequence and the absence of myocardial necrosis in the late gadolinium enhancement images.</p> <p>Management included medical therapy with the administration of bisoprolol and aspirin.</p>
Day 4	Hospital discharge.
Three months after discharge	The patient remained asymptomatic and showed no reoccurrence of chest pain.

The coronary angiography showed normal coronaries. CMR and echocardiography revealed complete resolution.

## Case summary

A 49-year-old Caucasian woman with no history of cardiovascular disease was admitted to the emergency department presenting with retrosternal chest pain radiating to the left arm. A history of smoking is one of the risk factors for cardiovascular disease. No chronic diseases were recorded. Anamnestic questioning revealed a very intense stress disorder during the weeks preceding the acute event.

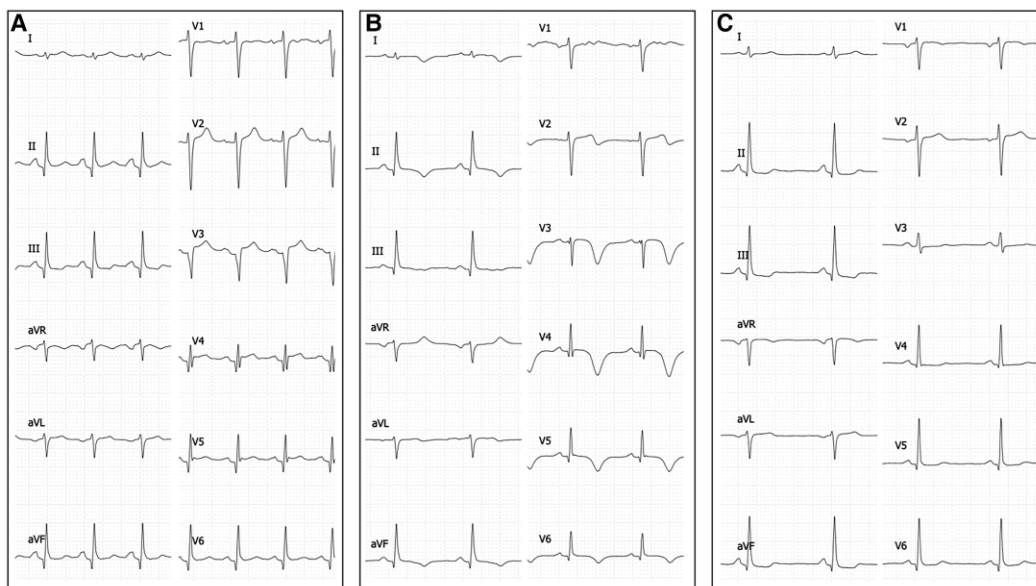
Upon admission, the patient was afebrile and tachycardic with a heart rate of 110 b.p.m. and a blood pressure of 130/87 mmHg. The results of the physical examination were insignificant. Cardiovascular examination revealed a regular rate and rhythm with clear S1 and S2 and no murmurs or rubs. The lungs were clear to auscultation and the lower extremities were warm without oedema bilaterally. A 12-lead electrocardiogram (ECG) revealed sinus tachycardia, normal axis, no ST-T change, and a normal QTc interval (*Figure 1*). The laboratory results showed elevated cardiac enzyme values, including a hs-Tn I level of 5883.4 ng/L (upper normal limit <15.6). The NT-proBNP (45 pg/mL; upper normal limit <450) and D-dimer (138 ng/mL; upper normal limit <500) levels were within normal limits. The InterTAK Diagnostic Score<sup>6</sup> was 61 out of 100 with a TTS probability of 58.6%. The items checked were 'female sex,' 'emotional stress,' and 'no ST-segment depression.'

The patient was transferred to the cardiac catheterization laboratory due to refractory chest pain despite medical treatment with nitroglycerin and P2Y12 inhibition. Coronary angiography indicated Type 2A SCAD involving the middle part of the left anterior descending (LAD) artery with a diffuse smooth stenosis of 26 mm. The thrombolysis in myocardial infarction (TIMI) flow was Grade 3 (*Figure 2* and *Supplementary material online, Figure S1*). Left ventriculography demonstrated a typical left ventricular apical ballooning and a hypercontractile base compatible with a TTS (*Supplementary material online, Video 1*). Given the typical appearance of SCAD, we did not use intracoronary imaging and opted for a conservative approach in accordance with the current guidelines.<sup>7</sup>

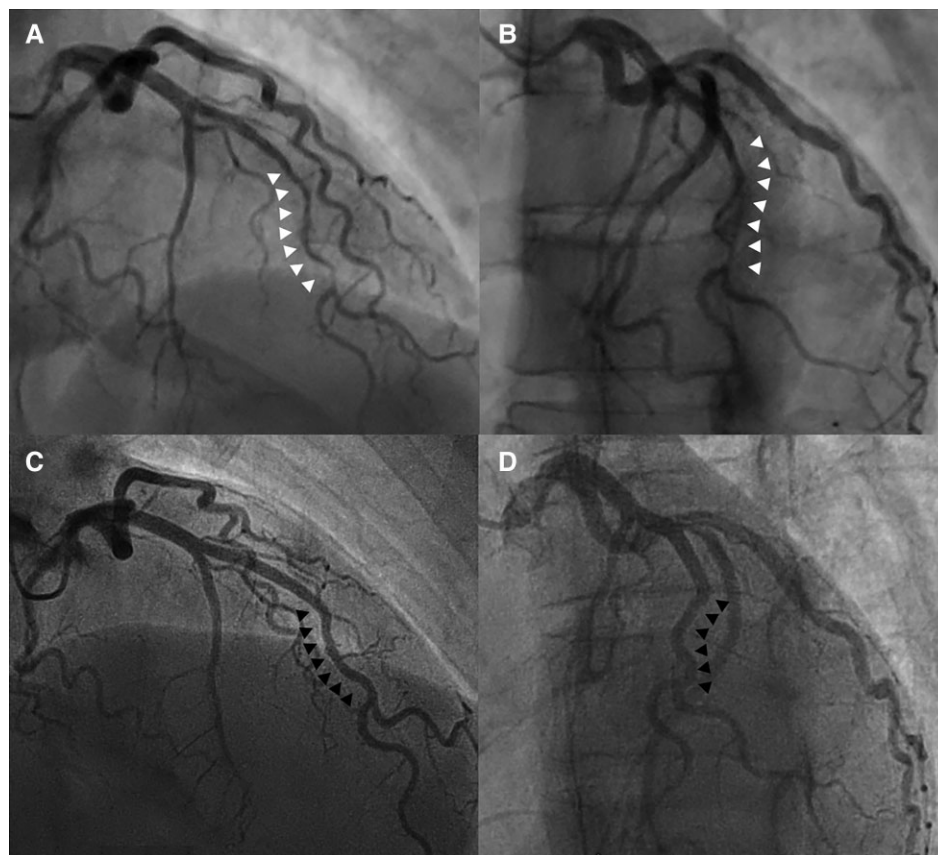
The patient was admitted to the cardiovascular intensive care unit. Management included medical therapy with the administration of beta-blockers (bisoprolol 2.5 mg daily) and antiplatelet agents (aspirin 75 mg daily).

The transthoracic echocardiogram (TTE) revealed broad, non-systematized hypokinesia of the left ventricular apex with typical apical ballooning with hypercontractile basal segments and no dynamic intraventricular obstruction. Left ventricular systolic function was impaired, with an ejection fraction of 45%. No valvular abnormalities or pericardial effusion was observed (*Supplementary material online, Video 2*).

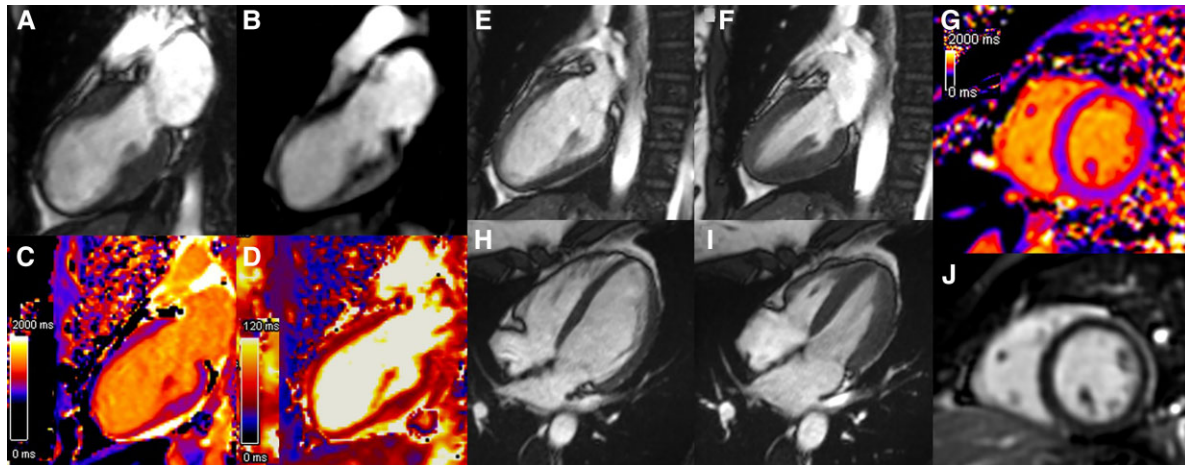
Cardiac magnetic resonance (CMR) imaging on Day 1 showed typical apical ballooning with transmural myocardial oedema in



**Figure 1** Twelve-lead electrocardiogram performed upon admission (A) indicated sinus tachycardia, normal axis, and no ST-T change. (B) 24 h post-admission: T-wave inversions in lead V2–V6 with QTc prolongation. (C) At 3 months.



**Figure 2** (A and B) Coronary angiography in the right anterior oblique cranial view indicated Type 2A spontaneous coronary artery dissection based on the classification system published by Saw *et al.*<sup>9</sup> involving the middle portion of the left anterior descending artery with a 'stick insect' appearance bordered by normal artery segment (white arrows) (C and D). Control angiography at 3M showed complete resolution of the previous pathological findings (black arrows). (A and C) Face cranial view. (B and D) Left anterior oblique cranial view.



**Figure 3** (A) Cine cardiac magnetic resonance two-chamber showed typical apical ballooning in end-systole. (B) Late gadolinium enhancement excluded myocardial necrosis in the dyskinetic anterior wall. (C) T1 mapping revealed a focal elevation in the T1 values of the distal anterior, apical, and distal inferior walls (T1 1300 ms vs. 1000 ms in other myocardial regions). (D) T2 mapping showed an elevation in the T2 values of the apical wall (60 ms vs. 50 ms in the other territories) indicating myocardial oedema. At follow-up, Cine cardiac magnetic resonance imaging in the vertical long axis (E and F) and four-chamber view (H and I) showed a normalization of global left ventricular systolic function. (G) T1 cartography in the short axis showed a homogeneous aspect with normal T1 at 1030 ms. (J) Late gadolinium enhancement-cardiac magnetic resonance imaging could not detect significant late gadolinium enhancement.

T2-weighted sequence and the absence of myocardial necrosis based on the late gadolinium enhancement images (Figure 3A–D and Supplementary material online, Figure S2). Additional ECG on Day 1 showed T-wave inversions in lead V2–V6 with QTc prolongation to 464 ms. The patient did not experience a recurrence of chest pain during hospitalization and was discharged on the fourth day after the procedure.

The control coronary angiography performed 3 months after the acute event showed a complete angiographic resolution (Figure 2C and D and Supplementary material online, Figure S3). The TTE revealed complete normalization of the left ventricular function and a global longitudinal strain score (–22.4%). The CMR revealed a complete resolution of the pathological features described above (Figure 3E–J and Supplementary material online, Figure S4). The patient remained asymptomatic and had no recurrence of chest pain.

## Discussion

As described in the revised Mayo Clinic Diagnostic Criteria (2008), the presence of coronary artery disease may be concomitant with TTS.<sup>3</sup> Chou et al.<sup>8</sup> found that in up to 8% of patients with diagnosed or suspected TTS, SCAD is misdiagnosed. SCAD remains an underdiagnosed condition that requires a detailed assessment of angiographic signs. The treatment and management of SCAD remains a challenge for clinicians as the therapeutic approach to this disease varies greatly depending on the diagnosis. Since the classification of Saw et al.<sup>9</sup> published in 2017, new angiographic signs have been described<sup>5</sup> as for the onset and/or termination of the angiographic ambiguity on a side branch, narrowing of the lumen diameter mimicking a ‘radish’ aspect, or a ‘stick insect’ as in this case.

Although TTS and SCAD are commonly observed in patients who share certain characteristics (women, without atheromatous terrain, stress-related factors), it is difficult to establish a pathophysiological link. TTS is caused by myocardial sideration after catecholamine release, which explains cases complicating endocrinopathies, such as pheochromocytoma. SCAD is known to be caused by mechanical stress on constitutional arterial fragility, hence the association with fibromuscular dysplasia found in almost 50% of cases.<sup>2</sup> This mechanical stress can be promoted not only by exertion but also by emotion, leading to the release of catecholamines, affecting the inotropism and blood pressure levels, as encountered in TTS. Hyperkinesia in some parts of the myocardium and apical ballooning could also contribute to the mechanical stress of epicardial coronaries as hypothesized by Madias et al.<sup>10</sup> Conversely, during SCAD, the sudden onset of pain in a young patient with no risk factors may be complicated by emotional shock, a panic attack similar to that described as a risk factor for TTS. Finally, a third hypothesis cannot be ruled out, which would assume that a catecholaminergic discharge could simultaneously or almost simultaneously lead to both stress cardiomyopathy and SCAD.

In this case, it was difficult to determine the risk factors of the patient’s condition. In this study, the evidence gathered is insufficient. The kinetics of biology showing elevated and decreased levels of troponin upon admission, while NT-proBNP increased only secondarily, might suggest that SCAD was the initial component complicated by TTS.

## Conclusions

This observation confirms the non-random association of two rare entities of myocardial infarction with no obstructive coronary arteries. Once the acute phase is over, the evolution of both is usually spontaneously favourable. While TTS can be easily diagnosed via

non-invasive imaging (CMR, TTE), the diagnosis of SCAD is more difficult. The findings of this study suggest a concomitant presentation between SCAD and TTS and the important role of diagnostic angiography in diagnosing these conditions. If needed, endocoronary imaging can also be used. Although the treatment approach to SCAD is usually conservative,<sup>2,11</sup> severe forms of this disease require early diagnosis and appropriate treatment.

## Lead author biography



Simon Fitouchi is a cardiologist at the University Hospital of Strasbourg (France). He graduated from the University of Strasbourg in Cardiovascular Medicine in 2021 and is currently training in interventional rhythmology.

## 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 the submission and publication of this case report including images and associated text has been obtained from the patient in accordance with the COPE guidelines.

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