INTERVENTIONAL CARDIOLOGY PERSPECTIVES

Inter Cardio Per

DOI: 10.4274/intercardiopers.2026.2025-11-1

CASE REPORT

Spontaneous Coronary Artery Dissection Mimicking Stress Cardiomyopathy (Takotsubo Syndrome): A Case Report

Şahhan Kılıç, Süha Asal

Clinic of Cardiology, Çorlu State Hospital, Tekirdağ, Türkiye

ABSTRACT

Spontaneous coronary artery dissection (SCAD) is a rare but increasingly recognized cause of acute coronary syndrome, particularly in middle-aged women. Its clinical presentation can be highly variable, occasionally mimicking other conditions such as stress cardiomyopathy [Takotsubo syndrome (TTS)]. We report the case of a 59-year-old woman with a history of hypertension who presented with chest pain and echocardiographic findings suggestive of Takotsubo cardiomyopathy. Coronary angiography, however, revealed SCAD in the mid-left anterior descending artery. Given the preserved coronary flow, conservative medical management was pursued. This case underscores the diagnostic challenges and overlapping features of SCAD and TTS and highlights the importance of invasive imaging for accurate diagnosis.

Keywords: Spontaneous coronary artery dissection, Takotsubo cardiomyopathy, acute coronary syndrome, women's heart health, MINOCA

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is an uncommon but important cause of acute myocardial ischemia, accounting for approximately 1–4% of all acute coronary syndrome (ACS) cases worldwide.[1] It predominantly affects women, especially those in their fourth and fifth decades, making it a critical consideration in the differential diagnosis of chest pain in this population.[1] SCAD is defined by the spontaneous separation of the coronary arterial wall layers, resulting in an intramural hematoma or false lumen that can compromise the true coronary lumen, disrupt blood flow, and potentially lead to myocardial infarction.[2] Unlike conventional ACS, which is usually caused by atherosclerotic plaque rupture, SCAD occurs in the absence of trauma, iatrogenic injury, or significant atherosclerosis, distinguishing it as a unique pathological entity.[3] Its pathophysiology is multifactorial, with predisposing factors including fibromuscular dysplasia (FMD), connective tissue disorders such as Marfan syndrome and Ehlers–Danlos syndrome, hormonal influences, and exposure to extreme physical or emotional stress.[4] These stressors may exacerbate underlying arterial fragility, particularly in the coronary vasculature, triggering or propagating the dissection.[5]

In parallel, stress cardiomyopathy, commonly known as Takotsubo syndrome (TTS), is another rare but increasingly recognized form of ACS that shares clinical and demographic features with SCAD.[6] First described by Sato et al.[7] in Japan over three decades ago, TTS is named after the Japanese "Takotsubo" (octopus trap) because of the characteristic apical ballooning observed on ventriculography during systole. TTS is characterized by transient left ventricular dysfunction, typically triggered by acute emotional or physical stress, which induces a catecholamine surge leading to coronary microvascular dysfunction and direct myocardial injury.[8] The condition predominantly affects postmenopausal women, with up to 90% of cases occurring in females, and is often associated with reversible wall motion abnormalities that extend beyond a single coronary artery distribution.[9] Despite the absence of obstructive epicardial coronary artery disease in most cases, TTS presents with chest pain, electrocardiographic changes, and elevated cardiac biomarkers that closely resemble ACS, complicating early diagnosis.[10] The diagnostic overlap between SCAD and TTS stems from their shared clinical features, female predominance, and potential association with stress-induced triggers.[6] However, while TTS is primarily a microvascular phenomenon, SCAD involves epicardial coronary artery pathology, which can occasionally mimic the regional ventricular dysfunction seen in TTS.[11] This overlap presents a significant diagnostic challenge, as misdiagnosis can lead to inappropriate management. For example, echocardiographic findings of apical ballooning in patients with SCAD may initially suggest TTS, potentially delaying the angiographic evaluation necessary to identify dissection.[12]

The underrepresentation of women in traditional ACS clinical trials further complicates recognition, as sex-specific differences in pathophysiology and presentation remain poorly understood,

Address for Correspondence: Şahhan Kılıç MD, Clinic of Cardiology, Çorlu State Hospital, Tekirdağ, Türkiye
E-mail: drsahhankilic@gmail.com **ORCID ID:** orcid.org/0000-0002-3524-5396

Cite as: Kılıç Ş, Asal S. Spontaneous coronary artery dissection mimicking stress cardiomyopathy (Takotsubo syndrome): a case report. *Inter Cardio Pers.* [Epub Ahead of Print]

Received: 18.11.2025
Accepted: 02.01.2026
Epub: 21.01.2026

potentially resulting in suboptimal therapeutic approaches.[6] This case report describes an instance of SCAD masquerading as Takotsubo cardiomyopathy in a hypertensive 59-year-old woman, highlighting the importance of maintaining a high index of clinical suspicion and the pivotal role of coronary angiography in resolving diagnostic uncertainty.[13] By examining this case, we aim to contribute to the growing body of evidence on these rare ACS variants, emphasizing the need for tailored diagnostic and therapeutic strategies to optimize patient outcomes in this complex clinical scenario.

CASE REPORT

A 59-year-old woman with a history of well-controlled hypertension presented to the emergency department with acute-onset substernal chest pain radiating to the left arm, accompanied by diaphoresis and shortness of breath. The symptoms began suddenly, without identifiable emotional or physical triggers. On admission, her vital signs were: blood pressure 150/90 mmHg, heart rate 65 beats per minute, respiratory rate 14 breaths per minute, and oxygen saturation 96% on room air. Physical examination was unremarkable, except for mild distress.

Initial electrocardiography showed sinus rhythm with T-wave inversions in leads V4–V6, suggestive of anterolateral ischemia. Serum troponin I was markedly elevated at >50,000 ng/mL (reference: 0.016 ng/mL). Other laboratory parameters, including complete blood count, renal function, and electrolytes, were within normal limits.

Transthoracic echocardiography revealed apical akinesia with ballooning of the left ventricular apex, hypercontractility of the basal segments, and a reduced ejection fraction of approximately 40%, findings highly suggestive of Takotsubo cardiomyopathy (Figure 1).

Given the persistent chest pain and markedly elevated troponin, urgent coronary angiography was performed. This demonstrated a smooth, tapered narrowing in the mid-left anterior descending (LAD) artery, consistent with type 2b SCAD (diffuse and smooth stenosis due to intramural hematoma), without evidence of atherosclerotic plaque or thrombosis (Figure 2). Distal coronary flow was preserved with thrombolysis in myocardial infarction (TIMI) grade 3 perfusion. No percutaneous interventions were required, and the patient was managed conservatively with dual antiplatelet therapy (aspirin and clopidogrel), beta-blockers, and angiotensin-converting enzyme inhibitors for blood pressure control.[1]

The patient's symptoms resolved within 48 hours. Follow-up echocardiography at one week demonstrated normalization of left ventricular function, with an ejection fraction of 60%. She was discharged on medical therapy, along with recommendations for risk factor modification and avoidance of extreme stressors. At three-month follow-up, she remained asymptomatic, with no recurrence of events. Given her complete symptom resolution and absence of recurrent events, repeat vascular imaging, such as coronary computed tomography angiography, was deferred to avoid unnecessary radiation and contrast exposure.

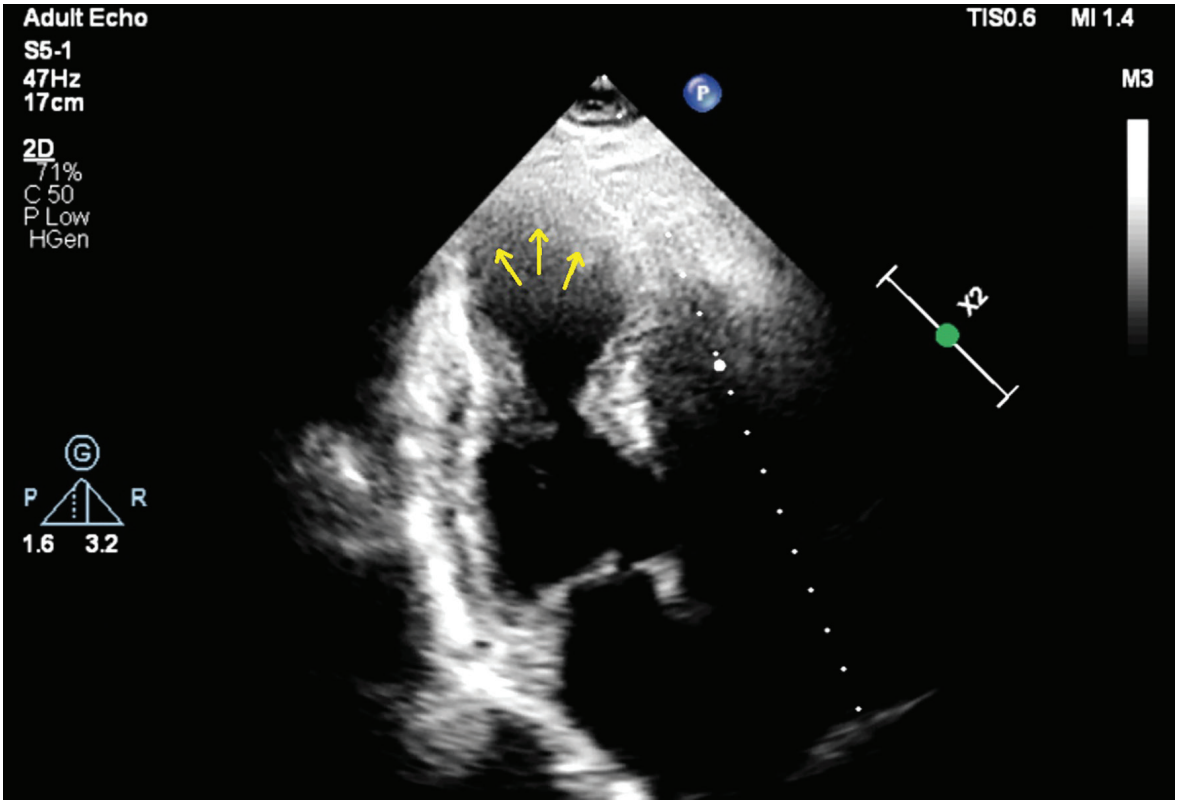

Figure 1. Transthoracic echocardiographic apical four-chamber view showing apical ballooning with akinesia of the apex and hyperkinesis of the basal segments, consistent with a Takotsubo-like pattern

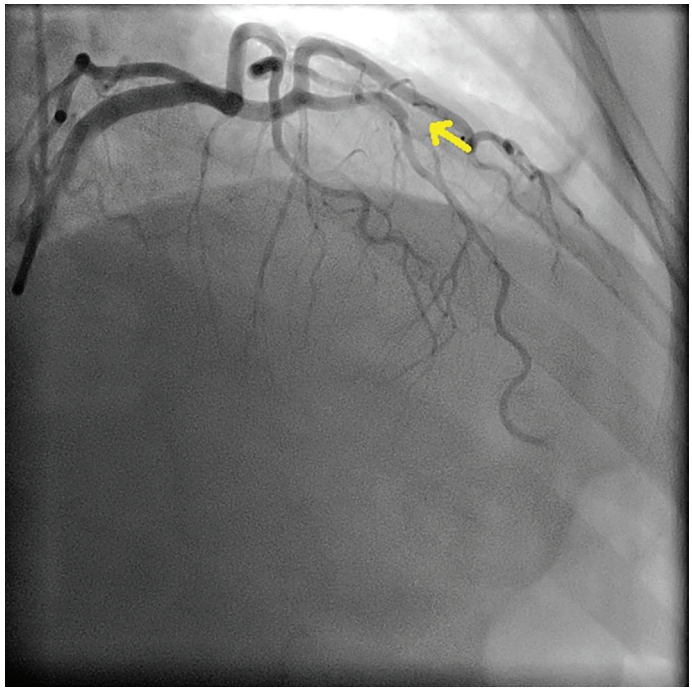

Figure 2. Coronary angiographic views demonstrating smooth narrowing of the mid-left anterior descending artery, indicative of spontaneous coronary artery dissection with preserved distal flow

DISCUSSION

This case illustrates a rare presentation of SCAD mimicking Takotsubo cardiomyopathy (also known as stress cardiomyopathy or TTS), highlighting a diagnostic challenge that can potentially delay optimal management.[13] SCAD is an underdiagnosed cause of ACS, predominantly affecting women aged 40–60 years, and accounts for up to 35% of ACS cases in women under 50.[14] In contrast, TTS is characterized by transient left ventricular apical ballooning in the absence of obstructive coronary artery disease, often triggered by emotional or physical stress, with a similar female predominance (up to 90% of cases).[3,7] The overlap between these conditions may arise from SCAD-induced ischemia triggering TTS-like ventricular dysfunction, as observed in our patient, who exhibited apical ballooning on echocardiography despite mid-LAD artery involvement.[6]

The apical ballooning in this patient is somewhat atypical for isolated mid-LAD SCAD, which typically affects mid-to-distal segments and produces regional wall motion abnormalities rather than the classic TTS pattern.[15] Nevertheless, ischemic insult from SCAD can induce catecholamine-mediated myocardial stunning, mimicking TTS—particularly in patients with hypertension, which may exacerbate microvascular dysfunction and shear stress on arterial walls.[2] A systematic review of SCAD-associated TTS cases reported this coexistence in various scenarios, including the postpartum period, with shared pathophysiological mechanisms such as hormonal fluctuations, inflammatory responses, and vascular fragility (e.g., FMD).[13] Approximately 10–23% of TTS cases undergoing detailed imaging were found to have underlying SCAD, emphasizing the need for diagnostic vigilance.[8] Although cardiac magnetic resonance imaging can differentiate myocarditis from TTS, it was not performed in this case because coronary angiography provided a definitive SCAD diagnosis explaining the wall motion abnormalities.

Differentiating SCAD from TTS is critical because management strategies differ significantly. SCAD often requires antiplatelet therapy, beta-blockers, and monitoring for dissection extension or arrhythmias, whereas TTS generally resolves spontaneously with supportive care.[3] Coronary angiography remains the gold standard for diagnosis, while intravascular imaging modalities such as optical coherence tomography or intravascular ultrasound can provide definitive evidence of intramural hematoma in ambiguous cases.[16] In this patient, preserved TIMI 3 flow supported a conservative approach, consistent with American Heart Association and European Society of Cardiology guidelines, which recommend medical management for hemodynamically stable SCAD without ongoing ischemia, given high spontaneous healing rates (up to 90% at follow-up).[14] Percutaneous coronary intervention is reserved for cases with compromised flow or persistent symptoms due to the risk of dissection propagation.[12]

The association with hypertension in our patient is noteworthy, as it is an established modifiable risk factor for SCAD, potentially mediated by increased arterial wall stress and endothelial dysfunction.[2,17] Other predisposing factors include pregnancy, extreme exercise, and connective tissue disorders, which were absent in this case.[1] The prognosis for SCAD-TTS overlap is generally favorable with early recognition; however, recurrence rates for SCAD can reach 10–30% within 5–10 years, highlighting the need for long-term follow-up and optimization of risk factors.[5] Clinicians should maintain a low threshold for invasive evaluation in middle-aged women presenting with chest pain and TTS-like echocardiographic features, particularly when ACS biomarkers are elevated or traditional risk factors, such as

hypertension, are present.[4,7] Future research, including prospective registries, is necessary to further elucidate the interplay between SCAD and TTS and to refine diagnostic algorithms.[9,13]

CONCLUSION

SCAD can closely mimic Takotsubo cardiomyopathy, posing significant diagnostic challenges. This case underscores the value of coronary angiography in identifying such overlaps and guiding appropriate therapy. Early recognition and conservative management can lead to excellent outcomes, as demonstrated in this patient.

Informed Consent: The patient was informed that the procedures performed would be included in a publication and written consent was obtained.

Authorship Contributions: Concept: Ş.K., S.A., Design: Ş.K., S.A., Data Collection or Processing: Ş.K., Literature Search: Ş.K., S.A., Writing: Ş.K., S.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

REFERENCES

1. Hayes SN, Kim ESH, Saw J, et al.; American Heart Association Council on peripheral vascular disease; council on clinical cardiology; council on cardiovascular and stroke nursing; council on genomic and precision medicine; and stroke council. Spontaneous coronary artery dissection: current state of the science: a scientific statement from the American Heart Association. *Circulation.* 2018;137:e523-e557.
2. Adlam D, Alfonso F, Maas A, Vrints C; Writing Committee. European Society of Cardiology, acute cardiovascular care association, SCAD study group: a position paper on spontaneous coronary artery dissection. *Eur Heart J.* 2018;39:3353-3368.
3. Tweet MS, Gulati R, Aase LA, Hayes SN. Spontaneous coronary artery dissection: a disease-specific, social networking community-initiated study. *Mayo Clin Proc.* 2011;86:845-850.
4. Kim ESH. Spontaneous coronary-artery dissection. *N Engl J Med.* 2020;383:2358-2370.
5. Tweet MS, Hayes SN, Pitta SR, et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. *Circulation.* 2012;126:579-588.
6. Di Vece D, Templin C, Ghadri JR. Takotsubo syndrome and spontaneous coronary artery dissection. *Cardiovasc Med.* 2023;26:160-167.
7. Sato H, Tateishi H, Uchida T, et al. Takotsubo-type cardiomyopathy due to multivessel spasm. In: Kodama K, Haze K, Hori M, editors. Clinical aspect of myocardial injury: from ischemia to heart failure. Tokyo (Japan): Kagakuhyoronsha Publishing Co; 1990. p.56-64.
8. Lyon AR, Bossone E, Schneider B, et al. Current state of knowledge on Takotsubo syndrome: a position statement from the taskforce on Takotsubo syndrome of the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail.* 2016;18:8-27.
9. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. *Am Heart J.* 2008;155:408-417.
10. Y-Hassan S. Spontaneous coronary artery dissection and takotsubo syndrome: an often overlooked association; review. *Cardiovasc Revasc Med.* 2018;19:717-723.
11. Ryan B, Yendamuri K, Yendamuri S. Anatomical considerations in bronchoscopy. *J Thorac Dis.* 2017;9:S1123-S1127.
12. Hausvater A, Smilowitz NR, Saw J, et al. Spontaneous coronary artery dissection in patients with a provisional diagnosis of Takotsubo syndrome. *J Am Heart Assoc.* 2019;8:e013581.
13. Bhanushali A, Kohli M, Prakash A, et al. Spontaneous coronary artery dissection-associated takotsubo syndrome: a systematic review of case reports. *World J Cardiol.* 2023;15:406-414.
14. Saw J, Aymong E, Sedlak T, et al. Spontaneous coronary artery dissection: association with predisposing arteriopathies and precipitating stressors and cardiovascular outcomes. *Circ Cardiovasc Interv.* 2014;7:645-655.
15. Saw J. Coronary angiogram classification of spontaneous coronary artery dissection. *Catheter Cardiovasc Interv.* 2014;84:1115-1122.
16. Kim Y, Han X, Ahn Y, et al. Clinical characteristics of spontaneous coronary artery dissection in young female patients with acute myocardial infarction in Korea. *Korean J Intern Med.* 2021;36:106-113.
17. Templin C, Ghadri JR, Diekmann J, et al. Clinical features and outcomes of Takotsubo (stress) cardiomyopathy. *N Engl J Med.* 2015;373:929-938.