CASE REPORT DOI: https://doi.org/10.18502/fem.v9i3.20023

Spontaneous coronary artery dissection (SCAD) as a rare but life-threatening emergency department presentation: a case report

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Published online: 2025-10-05

Abstract: Spontaneous coronary artery dissection (SCAD) is a rare but life-threatening cause of acute coronary syndrome, particularly in young women with few traditional risk factors for atherosclerosis. SCAD is often underrecognized due to its atypical presentation which can lead to misdiagnosis or delayed diagnosis in emergency settings. We present a case of a 40-year-old female who presented to the emergency department with acute chest pain and was found to have ST-elevation myocardial infarction (STEMI) due to SCAD. This case highlights the importance of considering SCAD in the differential diagnosis of acute coronary syndrome in young patients, especially females, and the need for increased awareness among emergency physicians to optimize patient outcomes and reduce recurrence risks. This case also emphasizes the importance of recognizing SCAD as a distinct entity from atherosclerotic ACS, as it requires a different management approach, particularly in the initial steps of care.

Keywords: Acute Coronary Syndrome; Emergency Medicine; Spontaneous Coronary Artery Dissection

Cite this article as: Pupulin A, Vairavanathan R, Hayre J. Spontaneous coronary artery dissection (SCAD) as a rare but life-threatening emergency department presentation: a case report. Front Emerg Med. 2025;9(3):e27.

1. Introduction

Spontaneous coronary artery dissection (SCAD) is an uncommon cause of acute coronary syndrome (ACS) that is not associated with atherosclerosis or trauma but can be life-threatening if not addressed. It is characterized by the separation of the coronary artery wall, leading to the formation of an intramural hematoma or intimal disruption, which can cause coronary artery obstruction and subsequent myocardial injury (1). Historically, SCAD was considered a rare and often fatal condition, but recent advances in diagnostic techniques and increased awareness have led to more frequent recognition and better understanding of this condition (2).

SCAD predominantly affects women, especially those of reproductive age, and is often associated with hormonal changes, emotional or physical stress, and certain genetic or connective tissue disorders (3). The clinical presentation can mimic that of atherosclerotic ACS, making diagnosis challenging. However, the management differs significantly, with conservative treatment being the preferred approach in many cases due to the risk of complications with percutaneous coronary intervention (PCI) (4).

This case report presents a typical case of SCAD in a young female patient. It discusses the diagnostic and management considerations, with a focus on the role of emergency physicians in identifying and initiating appropriate care, particularly addressing why intervention was chosen in this instance despite the usual conservative approach.

2. Case presentation

A 40-year-old female with no significant past medical history presented to the emergency department with a 2-hour history of sudden onset midsternal chest pain and diaphoresis. The pain was described as a pressure-like sensation, non-radiating, and associated with mild anxiety but no shortness of breath, dizziness, or nausea. She had recently traveled extensively for work, involving multiple flights to various cities, and reported significant work-related stress. She was a non-smoker with minimal alcohol intake and had a history of seasonal allergies managed with over-the-counter antihistamines.

Upon arrival, her vital signs were as follows: temperature, 35.3°C; heart rate, 79 beats per minute; blood pressure, 180/102 mmHg; respiratory rate, 18 breaths per minute; and oxygen saturation, 99% on room air. Physical examination revealed a well-appearing, but anxious patient with clear lung sounds and no signs of heart failure or peripheral vascular disease.

An initial electrocardiogram (ECG) showed ST elevations in leads V2, V3, and V4 with reciprocal changes in lead III and aVF, and large T waves in V3-V5, consistent with an acute anterior ST-elevation myocardial infarction (STEMI) (Figure 1). A 15-lead ECG confirmed the initial findings. She was im-

Table 1 Summary of key laboratory investigations related to chest pain workup

Test	Result	Units / Notes
Blood glucose	6.3	mmol/L
Sodium	140	mmol/L
Potassium	4.0	mmol/L
Chloride	101	mmol/L
Total CO_2 (Bicarb)	23	mmol/L
Creatinine	70	µmol/L
eGFR	95	mL/min/1.73m^2
CK	85	U/L
Lactate	2.4	mmol/L
VBG – pH	7.30	
VBG – pCO_2	54	mmHg
VBG – HCO_3	25	mmol/L
WBC	8.3	x10^9/L
RBC	5.18	x10^{12}/L
Hemoglobin	162	g/L
Platelets	331	x10^9/L
INR	1.0	
aPTT	27	seconds
β-HCG	Negative	
Troponin – Initial	19	ng/L
Troponin – 9 hrs	872	ng/L
Troponin – 18 hrs	885	ng/L
Troponin – Post-procedure	666	ng/L
D-dimer	Not performed	

GFR: Glomerular filtration rate; CK: Creatine kinase; VGB: Venous blood gas; WBC: White blood cell; RBC: Red blood cell; INR: International normalized ratio; PTT: Prothrombin time

mediately started on aspirin 162 mg, ticagrelor, and heparin and was then transferred to a tertiary care center for consideration of urgent cardiac catheterization. Initial laboratory investigations revealed an elevated troponin level of 19 ng/L, which rose to 872 ng/L at 9 hours and 885 ng/L at 18 hours at the tertiary center, consistent with acute myocardial injury. Other laboratory tests, including complete blood count, electrolytes, renal function, and coagulation profile, were within normal limits (Table 1).

At the tertiary center, coronary angiography was performed and revealed a mid to distal left anterior descending (LAD) artery occlusion with appearances suggestive of SCAD, specifically a 100% occlusion with TIMI 0 flow, indicating no blood flow through the artery. Given symptoms refractory to initial conservative management and coronary angiography revealing lack of flow, the interventional cardiologist elected to proceed with revascularization, resulting in TIMI 2 flow. Intravascular ultrasound (IVUS) confirmed the presence of an intramural hematoma.

Post-procedure, she was admitted to the coronary care unit and started on aspirin, ticagrelor, atorvastatin, and metoprolol. Her chest pain improved, and serial ECGs showed resolution of ST elevations. Cardiac biomarkers trended downward, and transthoracic echocardiography demonstrated preserved left ventricular function with apical hypokinesis.

She was discharged on day 3 with a diagnosis of SCAD, prescribed aspirin, metoprolol, and ramipril, and referred to a SCAD clinic for follow-up.

3. Discussion

Spontaneous coronary artery dissection (SCAD) remains an underrecognized cause of acute coronary syndrome, particularly in young women without traditional cardiovascular risk factors, presenting significant diagnostic and therapeutic challenges. Emergency physicians play a critical role in the early recognition and management of SCAD, where timely diagnosis and intervention can impact patient outcomes.

3.1. Diagnosis

The diagnosis of SCAD is primarily based on coronary angiography, which may show a dissection flap, intraluminal filling defects, or a "double-barrel" appearance (5). Intravascular imaging techniques like IVUS or optical coherence tomography (OCT) can confirm the presence of an intramural hematoma or dissection (6).

In the emergency department, emergency physicians must have a high index of suspicion for SCAD in young women presenting with chest pain and ECG changes suggestive of STEMI, but without typical risk factors (SCAD risk factors listed in table 2). In this case, the patient's age (40 years), lack of risk factors, and angiographic findings, including the intramural hematoma seen on IVUS, were consistent with a diagnosis of SCAD. Notably, her recent extensive travel and work-related stress may have contributed, aligning with known

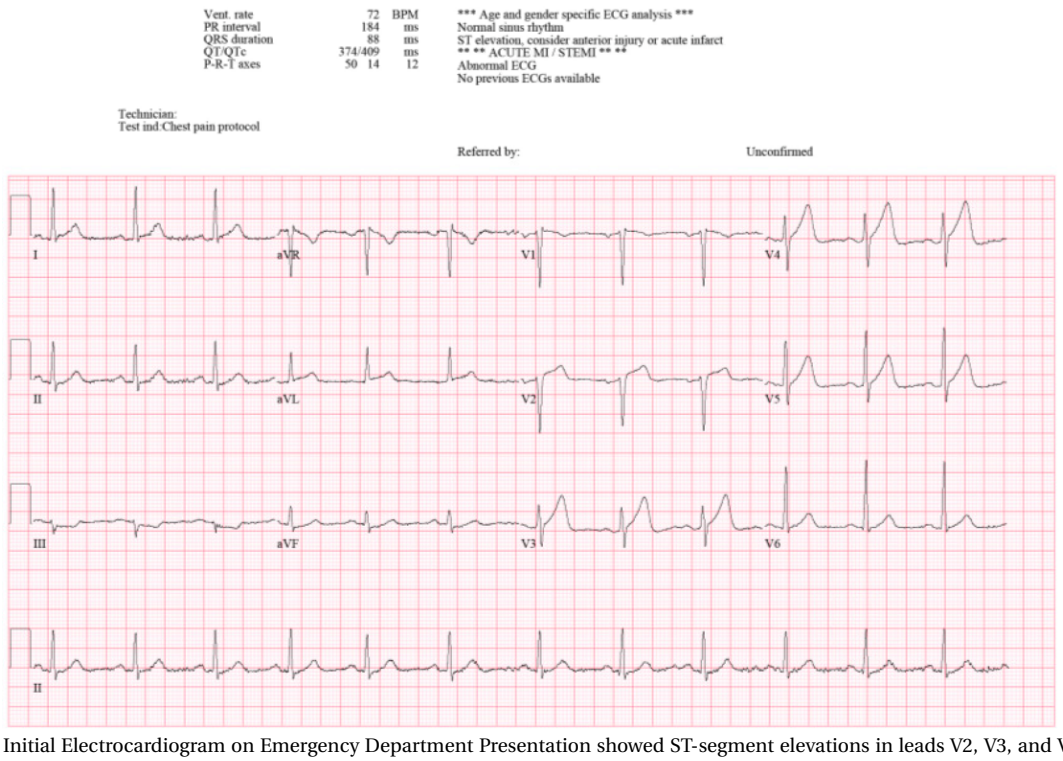

Figure 1 Initial Electrocardiogram on Emergency Department Presentation showed ST-segment elevations in leads V2, V3, and V4 with reciprocal changes in leads III and aVF, as well as large T waves in leads V3–V5. These findings are consistent with an acute anterior STEMI.

Table 2 Risk factors for spontaneous coronary artery dissection (SCAD)

Category	Risk factors
Demographic factors	Female sex, reproductive age (particularly peripartum period)
Hormonal factors	Menstrual cycle fluctuations, postmenopause
Genetic and connective tissue disorders	Fibromuscular dysplasia (FMD), Marfan syndrome, Ehlers-Danlos syndrome
Environmental and lifestyle factors	Emotional stress, physical stress (e.g., heavy lifting, intense exercise)
Vascular factors	Hypertension, no or few traditional cardiovascular risk factors (smoking, diabetes, hyperlipidemia)

Table 3 Differences between SCAD and atherosclerotic acute coronary syndrome (ACS)

Characteristic	SCAD	Atherosclerotic ACS
Age	Younger (mean age around 40-50 years)	Older (mean age around 60-70 years)
Sex	Predominantly female	More common in males
Risk factors	Few traditional risk factors; associated with FMD, hormonal factors, stress	Hypertension, hyperlipidemia, diabetes, smoking
Presentation	Often with ST-elevation MI	Variable, including ST-elevation MI, non-ST elevation MI, unstable angina
Angiographic findings	Dissection flap, intramural hematoma, "double-barrel" appearance	Atherosclerotic plaques, stenoses
Management	Conservative (medications) preferred; selective revascularization	Revascularization (PCI or CABG) commonly performed

precipitating factors such as emotional stress (7).

Consideration of SCAD is crucial in the emergency department, as prompt recognition and appropriate management of SCAD can significantly impact patient outcomes. In this case, early recognition of ACS facilitated prompt transfer to a tertiary centre, where coronary angiography could confirm suspicions of SCAD and allow for appropriate intervention.

3.2. Differential diagnosis

The differential diagnosis for sudden onset chest pain in a young female is broad. It includes acute coronary syndrome, spontaneous coronary artery dissection, aortic dissection,

pulmonary embolism, pericarditis, myocarditis, esophageal disorders, musculoskeletal pain, and anxiety (8). In this case, the presence of ST-elevation on ECG and elevated troponins strongly suggested an acute coronary event. However, the patient's young age and absence of traditional risk factors raised suspicion for SCAD, which was subsequently confirmed by coronary angiography. In addition to SCAD, pulmonary embolism should be considered, especially given the patient's recent travel history (8).

3.3. Management

The management of SCAD has evolved, with a shift towards conservative treatment in stable patients due to the high risk of complications with PCI, such as propagation of the dissection or perforation (9). Conservative management typically involves medical therapy with antiplatelet agents, beta-blockers, and statins to reduce myocardial stress and prevent recurrence (10). However, in cases of ongoing ischemia or hemodynamic instability, revascularization may be necessary (2).

In our patient, despite initial medical management in the emergency department, her symptoms did not improve. Given the risk of further myocardial damage with 100% occlusion of the mid to distal left anterior descending (LAD) artery and TIMI 0 flow, the decision was made to restore flow. This aligns with literature suggesting that PCI can be considered in cases of ongoing chest pain or severe ischemia, though it is associated with higher risks compared with atherosclerotic disease (11). This approach was appropriate given the clinical context.

Emergency physicians must be cautious about the use of thrombolytics, as they are generally contraindicated in SCAD due to the risk of extending the dissection or causing hemorrhage (10), which could worsen the patient's condition. Instead, the focus should be on stabilizing the patient with antiplatelet therapy, such as aspirin and P2Y12 inhibitors like ticagrelor, and arranging urgent transfer to a center capable of performing coronary angiography, as was done in this case (12,13). This highlights the importance of early recognition and communication with cardiology teams to ensure appropriate management, particularly in settings where immediate angiography is not available.

In settings without immediate access to percutaneous coronary intervention (PCI), such as rural hospitals, the standard management for ST-elevation myocardial infarction (STEMI) often involves thrombolytic therapy if transfer time exceeds 90 minutes (14). However, in suspected SCAD, thrombolytics are generally contraindicated due to the risk of extending the dissection (10). This presents a diagnostic challenge, as definitive diagnosis requires coronary angiography. In such scenarios, careful clinical judgment is required, balancing the potential benefits of thrombolysis in possible atherosclerotic STEMI against the risks in SCAD. This case underscores the importance of considering SCAD in the differential diagnosis, even in rural settings with limited resources, to prevent inappropriate use of thrombolytics and ensure timely transfer to specialized centers.

3.4. Follow-up and prognosis

Patients with SCAD have a risk of recurrence, estimated at around 10-20% (10), necessitating long-term follow-up and risk factor management. From an emergency medicine standpoint, it is crucial to educate patients on warning signs for recurrence, such as recurrent chest pain, and to ensure they are referred to a SCAD clinic or outpatient cardiologist for ongoing care, as was done in this case.

3.5. Comparison with atherosclerotic ACS

SCAD differs significantly from atherosclerotic ACS, as shown in table 3, with younger age, female predominance, and different management strategies. This distinction is critical for emergency physicians to avoid misdiagnosis and inappropriate treatment (1).

4. Conclusion

SCAD is a significant cause of ACS in young women, often presenting without typical atherosclerotic risk factors. Emergency physicians play a crucial role in promptly identifying and managing this condition as early consideration of SCAD in the differential diagnosis of ACS can lead to appropriate interventions and improved patient outcomes. This case underscores the importance of emergency physicians maintaining a high level of awareness for SCAD, ensuring timely diagnosis and appropriate management in this patient population.

5. Declarations

5.1. Acknowledgement

None.

5.2. Authors' contribution

Conceptualization, data collection, initial manuscript draft and editing: AP; Manuscript Editing: JH, RV; Conceptualization, supervision: RV.

5.3. Conflict of interest

None.

5.4. Funding

None.

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