OPEN ACCESS

Received: 11/11/25
Accepted: 02/01/26
Online: 21/01/26

CASE REPORT

Spontaneous coronary artery dissection as a cause of ST-segment elevation myocardial infarction: a clinical case report

Daniel Botero Cubides[1,a], Laura Salinas Muñoz[1,a], Edgar Fabian Manrique Hernández[2,b], Gianmarco Camelo Pardo[1,b], Karol Macias Quiñonez[2,b]

[1] Fundación Cardiovascular, Bucaramanga, Colombia.
[2] Hospital Internacional, Bucaramanga, Colombia.
[a] MD.
[b] Master's degree in Epidemiology.

Keywords:
cardiomyopathies; myocardial infarction; myocardium; ST elevation myocardial infarction; coronary circulation (source: MeSH-NLM).

ABSTRACT

Spontaneous coronary artery dissection is an uncommon cause of acute coronary syndrome, accounting for 1% to 4% of cases; it mainly affects young women without traditional coronary risk factors. This case highlights the importance of considering it as a differential diagnosis in patients with acute ST-segment elevation myocardial infarction. After informed consent was obtained, we report the case of a 45-year-old woman with no history of cardiovascular disease who presented to the Emergency Department with pressure-like chest pain radiating to the jaw. The electrocardiogram showed ST-segment elevation in the anterolateral leads. Cardiac biomarkers revealed significantly elevated troponin and CK-MB levels. Coronary angiography showed proximal dissection of the left anterior descending artery with thrombus in the proximal and mid segments, confirming the diagnosis. Distal TIMI grade III flow was documented; therefore, conservative management was chosen with single antiplatelet therapy (ASA 100 mg), a beta-blocker (metoprolol 50 mg), and statin therapy (atorvastatin 80 mg). The patient had a favorable clinical course after 10 days of hospitalization. Spontaneous coronary artery dissection should be suspected in young women with acute myocardial infarction and no traditional coronary risk factors. Early diagnosis and appropriate management are essential to reduce complications and improve prognosis.

Disección coronaria espontánea como causa de infarto con elevación del segmento ST: reporte de caso clínico

Palabras clave:
cardiomiopatías; infarto del miocardio; miocardio; infarto del miocardio con elevación del ST; circulación coronaria (fuente: DeCs-BIREME).

RESUMEN

La disección coronaria espontánea es una causa infrecuente de síndrome coronario agudo, representando entre el 1 % y el 4 % de los casos; afecta principalmente a mujeres jóvenes sin factores de riesgo coronario tradicionales. Este caso resalta la importancia de considerarla como diagnóstico diferencial en pacientes con infarto agudo de miocardio con elevación del segmento ST. Se presenta el caso de una mujer de 45 años con previo consentimiento informado, sin antecedentes cardiovasculares, que acudió al Servicio de Urgencias por dolor torácico opresivo irradiado a la mandíbula. El electrocardiograma mostró elevación del segmento ST en derivaciones anterolaterales. Los biomarcadores cardíacos evidenciaron elevación significativa de troponina y CK-MB. La angiografía coronaria reveló disección proximal de la arteria descendente anterior izquierda con trombo en segmentos proximal y medio, confirmando el diagnóstico. Se documentó flujo TIMI III distal, por lo que se decidió manejo conservador con antiagregación simple (ASA 100 mg), betabloqueador (Metoprolol 50 mg) y estatinas (Atorvastatina 80 mg). La paciente evolucionó favorablemente tras 10 días de hospitalización. La disección coronaria espontánea debe sospecharse en mujeres jóvenes con infarto agudo de miocardio sin factores de riesgo coronario tradicionales. El diagnóstico temprano y el manejo adecuado son fundamentales para reducir complicaciones y mejorar el pronóstico.

Cite as: Botero-Cubides D, Salinas-Muñoz L, Manrique-Hernández EF, Camelo-Pardo G, Macias-Quiñonez K. Spontaneous coronary artery dissection as a cause of ST-segment elevation myocardial infarction: a clinical case report. Rev Peru Cienc Salud. 2026;8(1):89-93. doi: https://doi.org/10.37711/rpcs.2026.8.1.8

Correspondence:
Karol Macias Quiñonez
kmacias@unab.edu.co

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is currently an uncommon but increasingly recognized cause of acute coronary syndrome (ACS), accounting for 1% to 4% of cases, with a marked predominance among young women without traditional coronary risk factors [1,2]. Its clinical importance lies in the fact that, unlike atherosclerotic disease, SCAD results from nontraumatic and noniatrogenic separation of the layers of the coronary arterial wall, generating a false lumen or an intramural hematoma that compromises blood flow and causes myocardial ischemia [1,2].

The pathophysiology may involve an initial intimal tear that allows blood to enter the subintimal space or, more commonly, bleeding from the *vasa vasorum* within the arterial media, producing a hematoma that compresses the true lumen [3]. In both cases, progression of the hematoma may lead to severe stenosis or complete occlusion [1].

Several predisposing conditions have been associated with SCAD, including fibromuscular dysplasia, autoimmune diseases, connective tissue disorders, migraine, hormonal states such as pregnancy and the puerperium, as well as physical or emotional triggers such as strenuous exercise, Valsalva maneuvers, and intense emotional stress [3]. Clinically, SCAD may present with a broad spectrum ranging from ST-segment elevation acute myocardial infarction and non-ST-segment elevation acute coronary syndrome to electrical instability with ventricular tachyarrhythmias, acute heart failure, and even sudden death [4,5].

Because SCAD frequently occurs in young patients without atherosclerosis, diagnosis requires a high index of clinical suspicion and is confirmed by coronary angiography, which may reveal typical patterns of dissection or luminal compression by hematoma. In cases of diagnostic uncertainty, intracoronary techniques such as intravascular ultrasound (IVUS) and optical coherence tomography (OCT) allow precise characterization of arterial wall involvement [3].

Regarding management, the preferred strategy in hemodynamically stable patients is conservative treatment, because most dissections heal spontaneously within days or weeks, whereas percutaneous interventions are associated with high rates of technical complications due to coronary wall fragility and the extent of the intramural hematoma [2]. Percutaneous coronary intervention or coronary artery bypass grafting is reserved for cases with persistent ischemia, left main coronary artery involvement, or malignant arrhythmias. Medical therapy includes beta-blockers and single antiplatelet therapy, although the available evidence remains limited [2]. In the long term, patients with SCAD require close follow-up because recurrence rates are estimated at 10% to 20%, in addition to systematic evaluation to rule out associated vasculopathies such as fibromuscular dysplasia [4,6].

Overall, SCAD represents a diagnostic and therapeutic challenge that requires timely recognition, individualized management, and an understanding of its pathophysiological features in order to avoid unnecessary interventions and optimize prognosis in affected patients.

The objective of this case report was to present a female patient with acute coronary syndrome in whom coronary artery dissection was identified, a condition that represents a challenge both diagnostically and therapeutically because of its uncommon clinical presentation.

CASE PRESENTATION

A 45-year-old patient with no history of cardiovascular disease presented to the Emergency Department with sudden-onset pressure-like chest pain radiating to the jaw, lasting approximately 30 minutes. During the episode, the patient experienced profuse diaphoresis and a sensation of anxiety. Acute coronary syndrome (ACS) was suspected, and an electrocardiogram was performed, showing sinus rhythm with ST-segment elevation in leads I, V3, and V4 (see Figure 1). These electrocardiographic findings supported the diagnosis of ACS, and percutaneous coronary intervention (PCI) was indicated.

The patient underwent cardiac catheterization, which revealed a proximal dissection flap and thrombus in the proximal and mid segments of the left anterior descending artery (see Figure 2). The presence of intracoronary thrombus raised the differential diagnosis of pure SCAD versus SCAD with secondary thrombosis; the angiographic morphology (absence of atherosclerotic plaques, linear dissection pattern, and longitudinal extension) was more suggestive of type 1 SCAD according to the Saw classification. OCT/IVUS was not performed because of clinical stability and TIMI III flow. Cardiac biomarkers showed significant elevation of troponin and CK-MB.

 https://doi.org/10.37711/rpcs.2026.8.1.8

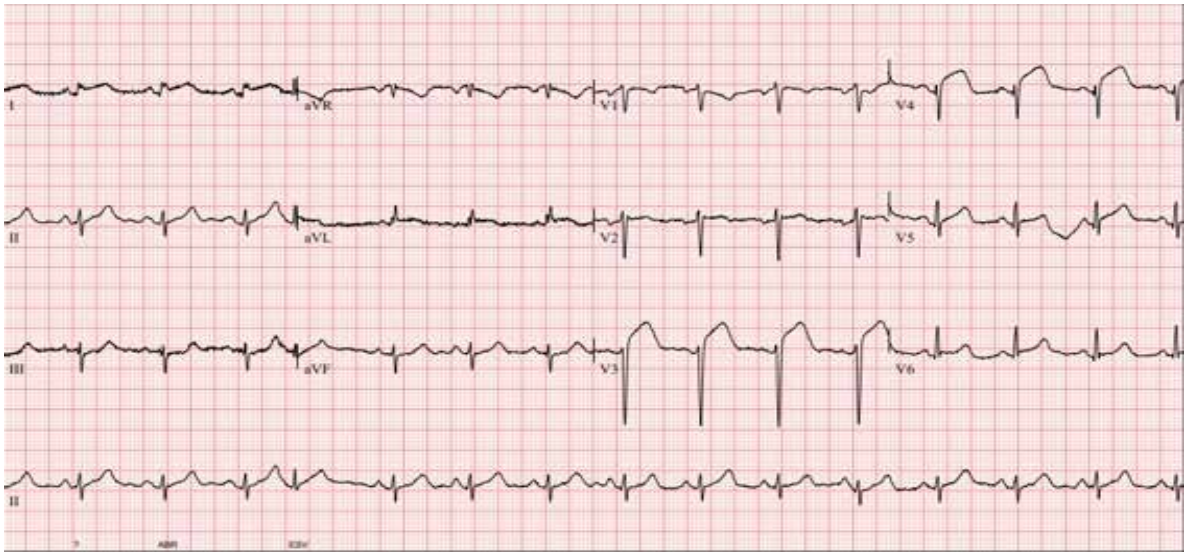

Figure 1. Twelve-lead electrocardiogram showing sinus rhythm with ST-segment elevation in leads I, V3, and V4, consistent with acute ischemia affecting the anterolateral region of the heart.

A subsequent transthoracic echocardiogram showed a normal-sized left ventricle with segmental wall motion abnormalities and akinesia of the true apex, in addition to involvement of all four apical segments. The left ventricular ejection fraction was estimated at 45%, while the right ventricle was normal in size (see Figure 3).

A second diagnostic and therapeutic coronary angiography was performed (left anterior oblique projection with caudal angulation), showing a normal left main coronary artery and circumflex artery, with no angiographic lesions. The left anterior descending artery showed a proximal dissection with good distal flow, and the right coronary artery showed no lesions (see Figure 4).

The patient required continuous monitoring in the Intensive Care Unit because of the risk of hemodynamic instability associated with her clinical condition. During her ICU stay, strict monitoring protocols were implemented to assess vital signs and hemodynamic parameters, allowing timely detection of any fluctuation suggestive of deterioration. Adequate pain management was instituted, with medication adjusted according to clinical needs to ensure patient comfort and support recovery. During hospitalization, she had no recurrence of chest pain or hemodynamic instability. After 10 days of comprehensive medical care, the patient was discharged in satisfactory clinical condition, with recommendations for outpatient follow-up and post-discharge management. Cardiology follow-up

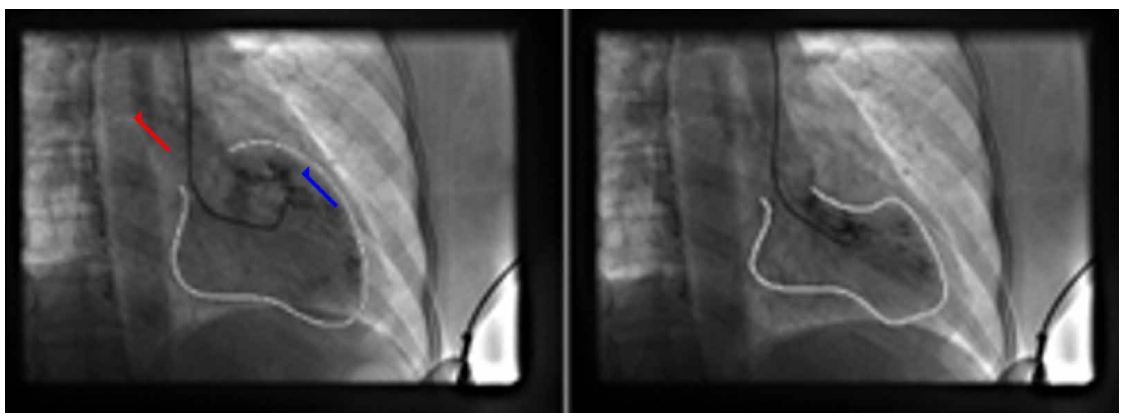

Figure 2. Cardiac catheterization showing a proximal dissection flap in the left anterior descending artery (red marker) with thrombus in the proximal and mid segments of the artery (blue marker).

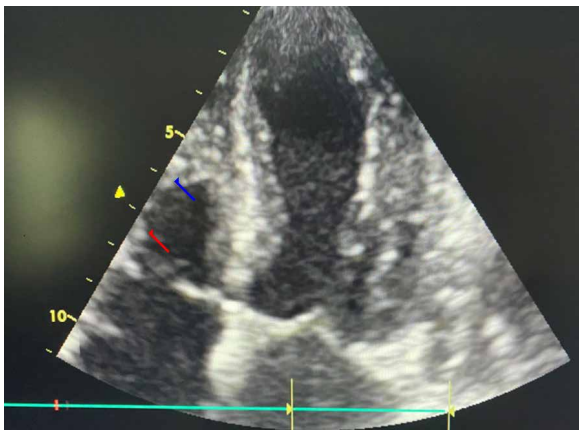

Figure 3. Transthoracic echocardiogram showing segmental wall motion abnormalities, akinesia of the true apex (red marker), and involvement of all four apical segments (blue marker).

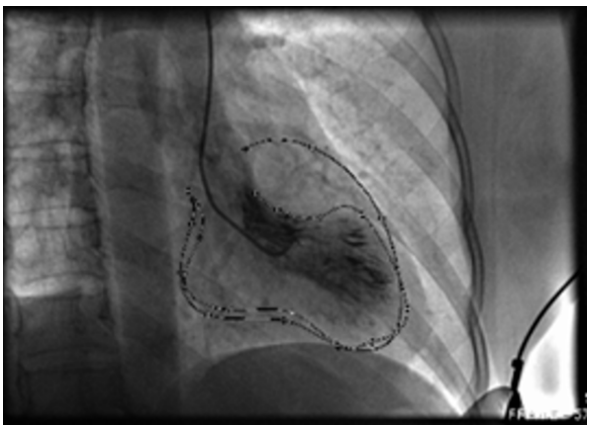

Figure 4. Second diagnostic and therapeutic coronary angiography.

was performed 1 month after discharge, at which time she was in favorable condition and cardiovascularly asymptomatic, with no evidence of new ischemic events.

DISCUSSION

This report describes an adult patient with no relevant medical history who presented with chest pain and was diagnosed with ST-segment elevation myocardial infarction (STEMI). Coronary angiography revealed type 1 spontaneous coronary artery dissection (SCAD), according to the Saw angiographic classification, an uncommon cause of acute coronary syndrome characterized by the presence of an intimal flap. In this case, there was no clear evidence of contrast retention within the false lumen, which should be differentiated from other angiographic subtypes characterized by false-lumen contrast staining and arterial narrowing. Despite its rarity, SCAD should be considered in patients presenting with chest pain to facilitate timely diagnosis and appropriate intervention [4,5]. According to the Saw angiographic classification, SCAD is categorized into types 1, 2, and 3. No contrast retention was observed in the false lumen.

Management of SCAD is determined by several factors, including the location of the dissection, the degree of myocardial ischemia, and the accessibility of the affected vessel. In selected cases in which the dissection is limited and there is no significant hemodynamic compromise, a conservative approach may be preferred. This generally includes antiplatelet and anticoagulant therapy, together with control of hypertension and other risk factors, to prevent progression of the dissection and the development of myocardial ischemia. In this case, conservative management was justified by hemodynamic stability, TIMI III flow, and the absence of left main coronary artery involvement. Careful monitoring is essential to ensure that the patient does not develop additional symptoms or complications during conservative treatment [4-6].

By contrast, invasive management is required when there is significant impairment of coronary blood flow, as evidenced by severe myocardial ischemia or concerning electrocardiographic changes. In such cases, percutaneous revascularization becomes a critical option. This procedure may involve stent placement or, in more complex situations, coronary artery bypass surgery [7]. The decision to pursue an invasive approach depends on angiographic assessment of the anatomy of the affected vessel and myocardial viability [8–10].

Early diagnosis and prompt intervention are essential to minimize the risk of major adverse events. Through specialized care, the recovery process was optimized, the patient received the indicated treatment, and her prognosis and clinical course improved. This case highlights the importance of considering SCAD within the differential diagnosis of acute coronary syndrome, especially in patients without evident cardiovascular risk factors. Including this entity in the differential diagnosis allows earlier intervention, contributing to improved management and a reduction in potential complications in this patient population [11–13].

In addition, this case highlights the need to strengthen continuing medical education on spontaneous coronary artery dissection, because its clinical presentation may mimic more common causes of acute coronary syndrome. The implementation of protocols integrating clinical, electrocardiographic, and angiographic criteria may facilitate earlier and more accurate recognition. Likewise, including SCAD in cardiology and emergency medicine training programs could help reduce underdiagnosis and improve the selection of individualized therapeutic strategies. Promoting clinical research and strengthening international registries may also improve understanding of predisposing factors, pathophysiological mechanisms, and recurrence rates, thereby contributing to the development of more robust evidence-based guidelines for the comprehensive management of these patients.

Conclusion

Spontaneous coronary artery dissection is an uncommon but clinically relevant cause of acute myocardial infarction. Early recognition and conservative management in stable patients are essential to prevent major complications. A high index of clinical suspicion should be maintained in young women presenting with myocardial infarction in the absence of traditional coronary risk factors.

Informed consent

The patient provided written informed consent.

REFERENCES BIBLIOGRAPHIC

1. Kim ESH, Saw J, Kadian-Dodov D, Wood M, Ganesh SK. FMD and SCAD: Sex-biased arterial diseases with clinical and genetic pleiotropy. Circ Res. [Internet]. 2021 [cited 2026 Feb 26];128(12):1958-72. doi: 10.1161/CIRCRESAHA.121.318300
2. Agrawal V, Kim ESH. Spontaneous coronary artery dissection: Cardiac manifestations of vascular disease. Prog Cardiovasc Dis. [Internet]. 2018 [cited 2026 Feb 26];60(6):629-34. https://doi.org/10.1016/j.pcad.2018.04.001
3. Kotecha D, Garcia-Guimaraes M, Premawardhana D, Pellegrini D, Oliver-Williams C, Bountzioka V, et al. Risks and benefits of percutaneous coronary intervention in spontaneous coronary artery dissection. Heart. [Internet]. 2021 [cited 2026 Feb 26];107(17):1398-406. doi: 10.1136/heartjnl-2020-318914
4. Museedi AS, Nashawi M, Ghali A, Hussein AA, Saca J. ST elevation myocardial infarction as presenting feature of C-ANCA vasculitis. Eur J Case Rep Intern Med. [Internet]. 2020 [cited 2026 Feb 26];7(8):001658. doi: 10.12890/2020_001658
5. Proença T, Martins Carvalho M, Alves Pinto R, Dias P, Macedo F. Spontaneous coronary artery dissection: Ten years' experience of a tertiary center. Rev Port Cardiol. [Internet]. 2023 [cited 2026 Feb 26];42(3):261-6. 10.1016/j.repc.2023.01.018.
6. Smirnova A, Aliberti F, Cavaliere C, Gatti I, Vilardo V, Giorgianni C, et al. Spontaneous coronary artery dissection: An unpredictable event. Eur Heart J Suppl. [Internet]. 2023 [cited 2026 Feb 26];25(Suppl B):B7-11. https://doi.org/10.1093/eurheartjsupp/suad059
7. Haas NA, Driscoll DJ, Rickert-Sperling S. Clinical presentation and therapy of coronary artery anomalies. Adv Exp Med Biol. [Internet]. 2024 [cited 2026 Feb 26];1441:799–809. 10.1007/978-3-031-44087-8_47
8. Saw J, Starovoytov A, Aymong E, Inohara T, Alfadhel M, McAlister C, et al. Canadian Spontaneous Coronary Artery Dissection Cohort Study: 3-year outcomes. J Am Coll Cardiol. [Internet]. 2022 [cited 2026 Feb 26];80(17):1585. 10.1016/j.jacc.2022.08.759
9. Gadelmawla A, Helmi A, Mohamed A, Ajaykumar Lakhani H, Khalid S, Bhatt K. et al. Comparative efficacy and safety of invasive approach versus conservative treatment in the management of spontaneous coronary artery dissection: an updated meta-analysis. JACC. [Internet]. 2025 [cited 2026 Feb 26];(12_Supplement) 885. https://doi.org/10.1016/S0735-1097(25)01369-5
10. Crousillat D, Sarma A, Wood M, Naderi S, Leon K, Gibson CM, et al. A. Spontaneous Coronary Artery Dissection: Current Knowledge, Research Gaps, and Innovative Research Initiatives: JACC Advances Expert Panel. JACC: Advances [Internet]. 2024 [cited 2026 Feb 26];3(12):101385. https://doi.org/10.1016/j.jacadv.2024.101385
11. Adlam D, Alfonso F, Maas A, Vrints C. European Society of Cardiology position paper on spontaneous coronary artery dissection. Eur Heart J. [Internet]. 2018 [cited 2026 Feb 26];39(36):3353–68. 10.1093/eurheartj/ehy080
12. Chen C. TCTAP C-105 A Twist in the Story: Iatrogenic Catheter-Induced Coronary Artery Dissection. JACC. [Internet]. 2024 [cited 2026 Feb 26];83(16_Supplement):S214-S215. https://doi.org/10.1016/j.jacc.2024.03.216
13. Kaddoura R, Aaysha Cader F, Ahmed A, Alasnag M. Spontaneous coronary artery dissection: an overview. Postg Med. J. [Internet]. 2023 [cited 2026 Feb 26];99:1226-1236. https://doi.org/10.1093/postmj/qgad086

Authorship contribution

DBC: Study protocol design and development, data collection, manuscript drafting, final review, and approval of the manuscript.

LSM: Study protocol design and development, data collection, manuscript drafting, final review, and approval of the manuscript.

EFMH: Study protocol design and development, data collection, manuscript drafting, final review, and approval of the manuscript.

GCP: Study protocol design and development, data collection, manuscript drafting, final review, and approval of the manuscript.

KMQ: Study protocol design and development, data collection, manuscript drafting, final review, and approval of the manuscript.

Funding sources

The study was self-funded.

Conflict of interest statement

The authors declare no conflicts of interest.