

Spontaneous Coronary Artery Dissection in a Young Female: A Complex Case of Diagnosis and Treatment Dilemma

A Fouseki¹, N Hadjigeorgiou¹, E Leonidou², K Andreou³, TE Plakomyti¹, V Giannakopoulos¹, V Dimou¹ and A Mouzarou^{1*}

Abstract

Spontaneous coronary artery dissection (SCAD) represents a rare, yet critical condition characterized by the spontaneous tearing of coronary artery walls, predominantly affecting younger populations, particularly women. This case report elucidates the complexities of diagnosis and management of SCAD in a 34-year-old female presenting with acute chest pain and ST elevation on electrocardiogram (ECG), highlighting the challenges encountered in selecting the optimal treatment strategy.

The patient underwent a comprehensive diagnostic workup, including coronary angiography, confirming the diagnosis of SCAD. Continuing management posed a therapeutic dilemma, necessitating a complex approach to address the patient's unique clinical presentation and potential complications. This case emphasizes the importance of individualized care, multidisciplinary collaboration, and long-term follow-up in optimizing patient outcomes and decision-making in managing SCAD.

This case report aims to contribute to the existing literature by providing valuable insights into the diagnostic and therapeutic challenges associated with SCAD, supporting the need for further research to clarify optimal management strategies and improve patient care.

Keywords: Spontaneous coronary artery dissection; Non-atherosclerotic coronary artery disease; Acute chest pain; ST elevation on ECG; Diagnostic workup; Treatment dilemma; Multidisciplinary management.

Abbreviation: SCAD: Spontaneous Coronary Artery Dissection; ECG: Electrocardiogram; LAD: Left Anterior Descending; TTE: Transthoracic Echocardiogram.

¹Department of Cardiology, General Hospital of Paphos, State Health Organization Services, Cyprus

²Department of Cardiology, General Hospital of Limassol, State Health Organization Services Cyprus

³STGBC Paphos Interventional Cardiology Center, Paphos, Cyprus

*Corresponding Author: Mouzarou A, MD, FESC, MSc, MScHM, PhDc, Department of Cardiology, General Hospital of Paphos, State Health Organization Services, Cyprus.

Received Date: 04-02-2024

Accepted Date: 04-16-2024

Published Date: 04-30-2024

Copyright© 2024 by Fouseki A, et al. All rights reserved. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Introduction

Spontaneous coronary artery dissection (SCAD) is an infrequent but serious condition that has gathered increasing attention in the field of cardiovascular medicine. Classified among the non-atherosclerotic causes of acute coronary syndrome, SCAD is characterized by the spontaneous dissection of coronary artery layers, creating an intimal flap and/or a false lumen with intramural hematoma which will cause restriction of blood flow and consequently myocardial ischemia. SCAD poses significant diagnostic and therapeutic challenges despite its rarity due to its diverse clinical manifestations and the absence of clearly identifiable causes. Lately, the frequency of the diagnosis has increased due to higher clinical suspicion and early angiographic investigation of acute coronary syndrome. Although the symptoms closely resemble those of other coronary syndromes, significant differences exist in characteristics such as pathophysiology, risk factors, age group, and angiographic appearance. It is predominantly observed in women, particularly those below the age of 50, SCAD often occurs during or after pregnancy [1].

The clinical presentation of SCAD can be highly variable, encompassing a spectrum of symptoms ranging from acute chest pain and dyspnea to more subtle manifestations such as fatigue or palpitations. This variability, coupled with the overlapping symptomatology with other coronary syndromes, might complicate the timely and accurate diagnosis of SCAD [2]. Furthermore, the management of SCAD remains a subject of ongoing debate and controversy within the medical community. The lack of consensus

guidelines and the limited evidence base surrounding optimal treatment strategies contribute to the therapeutic dilemma encountered in clinical practice [3].

This case report aims to clarify the complexities associated with diagnosing and managing SCAD, highlighting the importance of a multidisciplinary approach and individualized care in optimizing patient outcomes. By shedding light on the nuances of SCAD, this case report seeks to contribute to the existing literature and offer a deeper understanding of this intriguing yet challenging cardiovascular condition.

Case presentation

A 34-year-old female presented to the emergency department with acute onset chest pain that began approximately 15 minutes before admission. Notably, the patient had no significant past medical history, was a non-smoker, and maintained a healthy lifestyle. The patient was a mother of two children with no recent history of pregnancy. The chest pain was described as acute and sharp, radiating to the back between the shoulder blades. It started during exercise, specifically while engaging in jumping exercises. The patient denied experiencing diaphoresis, dizziness, or other associated symptoms.

Upon evaluation in the emergency department, the patient was hemodynamically stable (non-invasive blood pressure 115/70mmHg, pulses 89, oxygen saturation 98%(on air) and a temperature of 37.20 °C. An ECG revealed ST-segment elevation in leads I and AVL, accompanied by ST-segment depression in leads V2-V5 (Figure 1).

Given the persistent nature of the chest pain, a transthoracic echocardiogram (TTE) was performed, demonstrating hypokinesia of the apical and apical-lateral segments of the left ventricle. Notably, the aortic root and ascending aorta appeared normal in dimensions, with no evidence of an intimal flap or pericardial effusion. Given the clinical suspicion of a coronary event, despite initially negative Troponin I (TNI) levels, intravenous nitrate infusion was initiated to alleviate the persistent chest pain. Clinical suspicion of SCAD was reinforced by a combination of factors. The patient was a young woman without known risk factors contributing to an atherosclerosis-related myocardial infarction event. The patient also experienced acute chest pain during intense exercise despite maintaining a regular exercise routine. Subsequently, the patient was transferred to the catheterization laboratory for a primary coronary angiography. Catheterization of the

left anterior descending (LAD) coronary artery revealed the presence of a dissection (Figure 2), posing significant challenges during the procedure, as each infusion of contrast agent carried the potential risk of exacerbating the dissection. Patient management overview:

In the presented case, the treating invasive cardiologist adopted a conservative therapeutic approach, electing to manage the coronary artery dissection through pharmacological interventions. The patient was initiated on a regimen comprising aspirin and beta-blockers, aimed at mitigating thrombotic risks, and stabilizing cardiac function.

Subsequent monitoring over a three-day period revealed no immediate adverse effects, with the patient maintaining hemodynamic stability and reporting alleviation of chest pain symptoms.

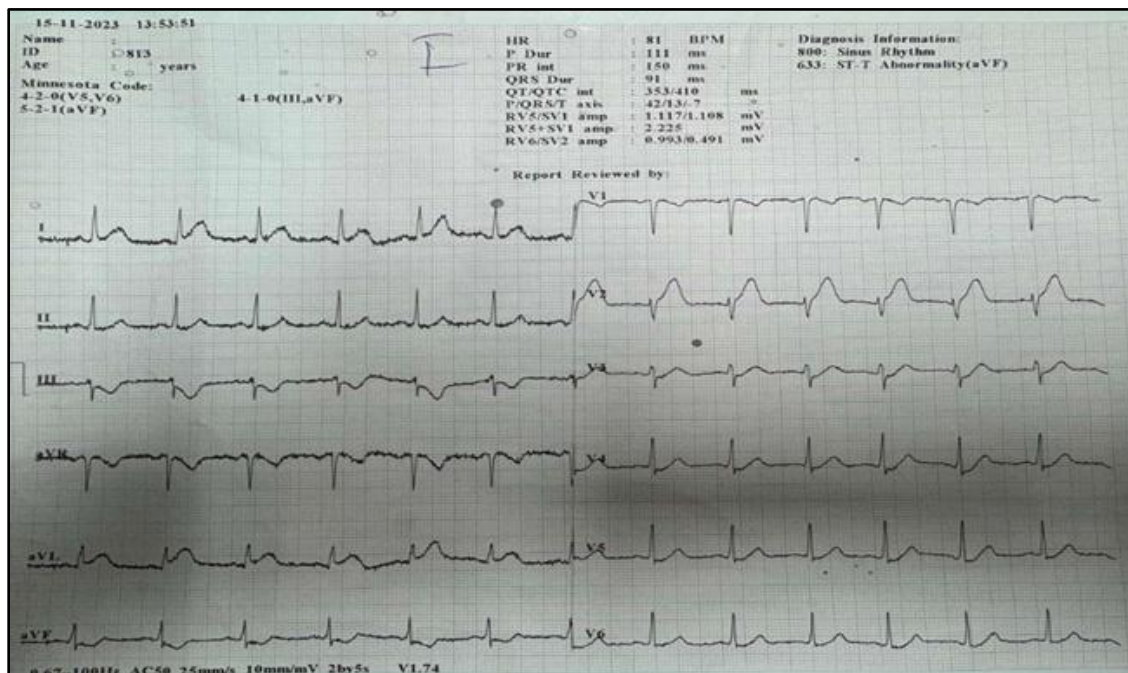


Figure 1: Electrocardiogram (ECG): Acute ST-segment elevation observed during patient's episode.

The comprehensive evaluation included hormonal assays and systemic disease screenings to elucidate potential underlying etiologies or contributing factors. Upon achieving symptomatic relief and hemodynamic stability, the patient was discharged with a structured follow-up plan encompassing cardiac assessments and lifestyle modifications. Specific recommendations were delineated, encompassing a temporary cessation of vigorous exercise, gradual resumption with

reduced intensity, stress reduction strategies, and potential psychological support avenues. Given the patient's demographic profile and reproductive considerations, the potential implications of pregnancy on cardiovascular health were considered. Although the patient has not expressed immediate intentions for pregnancy, this aspect warrants ongoing dialogue and consideration in future consultations, ensuring informed decision-making aligned with the patients' health priorities and lifestyle commitments.



Figure 2: Coronary angiogram: Extensive dissection and diffuse blockage of the left anterior descending artery.

SCAD literature review

Definition-pathophysiology

SCAD, or spontaneous coronary artery dissection, involves the sudden and non-traumatic tearing of the coronary wall, leading to reduced blood flow in the true lumen. There are two suggested mechanisms for SCAD. The first involves an intimal tear that results in bleeding into the media,

leading to medial dissection and the formation of a false lumen. This may be visualized angiographically by the distinctive presence of multiple radiolucent lumens, contrast dye stains in the arterial wall, or by the slow clearance of contrast dye within the lumen [4]. The second mechanism is less understood and is proposed to begin with the rupture of vasa vasorum, causing spontaneous bleeding into the arterial wall

and generating an intramural hematoma (IMH). This might only manifest as luminal compression, and this angiographic appearance is more commonly observed than the classic arterial wall stains. Both mechanisms create a separation between the artery layers, forming a false lumen. The entry of blood into this false lumen can result in hematoma formation, compressing the true lumen and causing ischemia, ultimately presenting symptoms similar to a myocardial infarction [5].

Epidemiology

SCAD is a rare contributor to acute coronary symptoms, accounting for only 1-4% of myocardial infarction cases. The percentage has shown an increase in recent years, reflecting heightened awareness, suspicion, and more frequent diagnoses of this condition [6]. The typical patient affected by SCAD is a young woman, usually in the age range of 20 to 40, with occurrences becoming more common during or after pregnancy. Recent studies indicate that older women, who are postmenopausal and under the age of 65, could also experience SCAD [1,7].

Causes

The etiology of SCAD is not consistently evident, and when associated with atherosclerosis, it manifests as a distinct condition that predominantly impacts men. Discussions within the medical community regarding SCAD primarily focus on its non-atherosclerotic form. In over 80% of patients, there is an association between SCAD and fibromuscular dysplasia [8]. Additional factors include connective tissue disorders (examples: Marfan syndrome, alpha-1 antitrypsin deficiency, polycystic kidney

disease, polycystic ovarian syndrome), hormonal disturbances, and vascular abnormalities, systemic inflammatory disease (such as systemic lupus erythematosus, Crohn's disease, sarcoidosis). SCAD may be triggered by stressful incidents such as intense emotional pressure, physical strains (such as lifting heavy weights or engaging in vigorous aerobic exercises), substance use (including cocaine, methamphetamines, β -human chorionic gonadotropin), and exertion from bearing-down activities (such as retching, vomiting, coughing). Additionally, labor or the process of delivering neonate could also be a contributing factor [9].

Angiographic classification

There are three distinct angiographic patterns of SCAD [10]:

- Type 1 (evident arterial wall stain): This represents the pathognomonic angiographic appearance of SCAD, characterized by contrast dye staining of the arterial wall and the presence of multiple radiolucent lumens.
- Type 2 (diffuse stenosis of varying severity): This pattern frequently indicates the presence of an intramural hematoma with no discernible dissection plane that can result in complete vessel occlusion. The lesions commonly exceed a length of 30 mm, exhibiting a sudden alteration in vessel diameter between healthy and affected segments. There is an absence of response to intracoronary nitrates, and no atherosclerotic lesions are observed in other coronary segments.
- Type 3 (mimic atherosclerosis): Distinguishing this presentation from

atherosclerosis is particularly challenging and is susceptible to misdiagnosis. Angiographic characteristics supporting SCAD diagnosis include (a) the lack of atherosclerotic alterations in other coronary arteries, (b) extended lesions (11-20 mm), (c) hazy stenosis, and (d) linear stenosis.

Clinical presentation and complications

Most patients experience symptoms indicative of acute coronary syndrome (ACS). Nearly all cases involve chest pain or discomfort, often accompanied by radiation to the left arm or neck, back or epigastric pain, nausea, vomiting, diaphoresis, or dyspnea. Elevated levels of cardiac enzymes are detected in over 95% of cases, with reported precipitating stress factors such as emotional tension or previous excessive physical activity. Clinicians must exercise heightened vigilance when young individuals, especially women with few or no predisposing risk factors for atherosclerotic cardiovascular disease, present with symptoms indicative of ACS.

SCAD can result in diverse complications, including [11]:

- Myocardial infarction, where the pathophysiology was previously described
- Heart failure, arising from limitations in myocardial blood supply
- Arrhythmias, due to potential disruptions in the heart's electrical pathways
- Cardiogenic shock, stemming from inadequate supply to vital organs

- Recurrent SCAD, often associated with underlying causes
- Cardiac arrest, particularly linked to ventricular arrhythmias
- Psychological impact, especially in young patients experiencing such cardiac events.

Management-treatment

Considering the distinctive pathophysiology and etiology of SCAD, which differs significantly from other causes of coronary artery disease, is crucial for implementing appropriate management strategies.

The treatment options were to either proceed with invasive management with stent implantation or revascularization with coronary artery bypass grafting (CABG) surgery. A decision shall be made of those 3 options or a combination of them, considering that all options have their advocates and detractors, supported by studies [7].

According to CanSCAD (Canadian SCAD cohort study), a multicenter prospective observational study on spontaneous coronary artery dissection (SCAD) from 2014 to 2018, involving 750 patients, the mean dissection length was 33.2 mm, and medial stenosis severity averaged 79.0%. Initially, 86.4% of patients were managed conservatively, with only 2.0% requiring subsequent percutaneous coronary intervention (PCI) and 0.3% needing CABG. For those opting for revascularization, reasons included ongoing ischemic symptoms (39.1%), ongoing ischemic changes on the ECG (34.5%), and severe luminal stenosis (31.8%). Overall, 14.7% of patients underwent revascularization (only 0.7% CABG). The technical success rate

of PCI was discouraging, with only 29.1% achieving successful outcomes [12].

Moreover, according to the European society of cardiology (ESC), in hemodynamically stable patients with SCAD, the preferred treatment is medical therapy, although there is a lack of supporting studies. Revascularization is indicated in cases of complete vessel occlusion with thrombolysis in myocardial infarction (TIMI) 0 flow, unlikely to resolve with medical treatment alone; left main stem involvement; ongoing ischemia; recurrent chest pain; hemodynamic instability; and sustained ventricular tachyarrhythmias.

Although PCI is the preferred revascularization strategy, its reported success rate is less than 50% [13]. This is often attributed to challenges such as navigating the guidewire into the true lumen, managing dissection or hematoma extension, and addressing side branch occlusion. Additionally, stent placement may contribute to hematoma propagation and the subsequent loss of vessel flow. Finally, CABG is recommended for patients with left main stem dissections or in cases where PCI has proven unsuccessful or is not technically feasible [14]. The incidence of emergency CABG following PCI failure is notable, ranging between 10% and 13% in reported series [15,16]. As far as we are concerned, there are no substantial studies indicating the optimal and appropriate treatment for SCAD. Conservative management of SCAD is recommended for hemodynamically stable patients [17], demonstrating effectiveness in most instances. Beta blockers are the preferred initial pharmacological intervention due to their potential to

decrease dissection recurrence. Additionally, blood pressure reduction is warranted in all cases [18]. The use of antiplatelet agents and their timing of administration remain controversial, as they may exacerbate dissection [19].

PCI becomes more relevant when the patient is hemodynamically unstable, particularly in cases of ST-segment elevation or LAD dissection [20]. As the suspicion of coronary artery dissection increases and the diagnosis becomes more frequent, additional studies must be conducted to establish the optimal treatment choice and document the potential adverse effects associated with each option.

Conclusion

In the rapidly evolving domain of cardiovascular medicine, the precise diagnosis and adept management of SCAD remain paramount. This case underscores the criticality of heightened clinical vigilance, particularly in young women devoid of conventional atherosclerotic risk factors, to facilitate timely recognition and intervention. The judicious integration of advanced imaging modalities, multidisciplinary collaboration, and individualized therapeutic strategies emerges as a cornerstone in optimizing patient outcomes. Interdisciplinary synergy, encompassing cardiologists, interventional radiologists, and cardiothoracic surgeons, underscores the complexity and multifaceted nature of SCAD management. The patient's experience shows the importance of a comprehensive approach that includes ongoing care, lifestyle changes, and strong emotional support to fully address SCAD treatment. Through integrating clinical insight with patient-centered empowerment and interdisciplinary teamwork, this case

outlines a model for optimizing SCAD care. It underscores the significance of personalized treatment and collaborative strategies in managing SCAD complexities, leading to

enhanced patient outcomes. As SCAD gains attention in cardiology, this case emphasizes the ongoing necessity for research, advocacy, and innovative therapeutic approaches.

References

1. Vrints CJ. Spontaneous Coronary Artery Dissection. *Heart*. 2010;96(10):801-8. [PubMed](#) | [CrossRef](#)
2. Tweet MS, Hayes SN, Pitta SR, Simari RD, Lerman A, Lennon RJ, et al. Clinical features, Management, and Prognosis of Spontaneous Coronary Artery Dissection. *Circulation*. 2012;126(5):579-88. [PubMed](#) | [CrossRef](#)
3. Kim ESH. Spontaneous Coronary-artery Dissection. *N Engl J Med*. 2020;383(24):2358-2370. [PubMed](#) | [CrossRef](#)
4. Maehara A, Mintz GS, Castagna MT, Pichard AD, Satler LF, Waksman R, et al. Intravascular Ultrasound Assessment of Spontaneous Coronary Artery Dissection. *Am J Cardiol*. 2002;89(4):466-8. [PubMed](#) | [CrossRef](#)
5. Hayes SN, Tweet MS, Adlam D, Kim ESH, Gulati R, Price JE, et al. Spontaneous Coronary Artery Dissection: JACC State-of-the-Art Review. *J Am Coll Cardiol*. 2020;76(8):961-984. [PubMed](#) | [CrossRef](#)
6. Khiatah B, Jazayeri S, Yamamoto N, Burt T, Frugoli A, Brooks DL. Cardiovascular Disease in Women: A Review of Spontaneous Coronary Artery Dissection. *Medicine (Baltimore)*. 2022;101(38):e30433. [PubMed](#) | [CrossRef](#)
7. Matta A, Levai L, Elbaz M, Nader V, Parada FC, Carrié D, et al. Spontaneous Coronary Artery Dissection: A Review of Epidemiology, Pathophysiology and Principles of Management. *Curr Probl Cardiol*. 2023;48(7):101682. [PubMed](#) | [CrossRef](#)
8. Iismaa SE, Hesselson S, McGrath-Cadell L, Muller DW, Fatkin D, Giannoulatou E, et al. Spontaneous Coronary Artery Dissection and Fibromuscular Dysplasia: Vasculopathies with a Predilection for Women. *Heart Lung Circ*. 2021;30(1):27-35. [PubMed](#) | [CrossRef](#)
9. Zeven K. Pregnancy-associated Spontaneous Coronary Artery Dissection in Women: A Literature Review. *Curr Ther Res Clin Exp*. 2023;98:100697. [PubMed](#) | [CrossRef](#)
10. Saw J. Coronary Angiogram Classification of Spontaneous Coronary Artery Dissection. *Catheter Cardiovasc Interv*. 2014;84(7):1115-22. [PubMed](#) | [CrossRef](#)
11. Al Emam AR, Almomani A, Gilani SA, Khalife WI. Spontaneous Coronary Artery Dissection: One Disease, Variable Presentations, and Different Management Approaches. *Int J Angiol*. 2016;25(3):139-47. [PubMed](#) | [CrossRef](#)
12. 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*. 2022;80(17):1585-97. [PubMed](#) | [CrossRef](#)
13. Hassan S, Samuel R, Starovoytov A, Lee C, Aymong E, Saw J. Outcomes of Percutaneous Coronary Intervention in Patients with Spontaneous Coronary Artery Dissection. *J Interv Cardiol*. 2021;2021:6686230. [PubMed](#) | [CrossRef](#)
14. Moghadam R, Rahman T, Reiss CK. Complicated Spontaneous Coronary Artery Dissection (SCAD) Culminating in Aneurysm Formation: Coronary Artery Bypass Graft Surgery is Preferable Over Percutaneous Coronary Intervention in Peripartum SCAD. *Cureus*. 2021;13(3):e14145. [PubMed](#) | [CrossRef](#)
15. Saw J, Aymong E, Sedlak T, Buller CE, Starovoytov A, Ricci D, et al. Spontaneous Coronary Artery Dissection: Association with Predisposing Arteriopathies and Precipitating Stressors and Cardiovascular Outcomes. *Circ Cardiovasc Interv*. 2014;7(5):645-55. [PubMed](#) | [CrossRef](#)
16. Tweet MS, Eleid MF, Best PJ, Lennon RJ, Lerman A, Rihal CS, et al. Spontaneous Coronary Artery Dissection: Revascularization Versus Conservative Therapy. *Circ Cardiovasc Interv*. 2014;7(6):777-86. [PubMed](#) | [CrossRef](#)

17. Gad MM, Mahmoud AN, Saad AM, Bazarbashi N, Ahuja KR, Karrthik AK, et al. Incidence, Clinical Presentation, and Causes of 30-day Readmission Following Hospitalization with Spontaneous Coronary Artery Dissection. *JACC Cardiovasc Interv.* 2020;13(8):921-32. [PubMed](#) | [CrossRef](#)
18. Saw J, Humphries K, Aymong E, Sedlak T, Prakash R, Starovoytov A, et al. Spontaneous Coronary Artery Dissection: Clinical Outcomes and Risk of Recurrence. *J Am Coll Cardiol.* 2017;70(9):1148-1158. [PubMed](#) | [CrossRef](#)
19. Aliyary S, Mariani MA, Verhorst PM, Hartmann M, Stoel MG, Von Birgelen C. Staged Therapeutic Approach in Spontaneous Coronary Dissection. *Ann Thorac Surg.* 2007;83(5):1879-81. [PubMed](#) | [CrossRef](#)
20. Yip A, Saw J. Spontaneous Coronary Artery Dissection-A Review. *Cardiovasc Diagn Ther.* 2015;5(1):37-48. [PubMed](#) | [CrossRef](#)