International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

The Hidden Fissure: A Case of Spontaneous Coronary Artery Dissection

Dr. Samantha Aster Pinto[1], Dr. Joel Piedade Quadros[2]

[1]Junior Resident, M.B.B.S, Goa Medical College and Hospital, Department of Cardiology
Corresponding Author Email: *aster.pinto3099[at]gmail.com*
ORCID ID- 0009-0001-4106-476X

[2]Assistant Professor, Department of Cardiology, MBBS, MD Medicine, DM Cardiology, MRCP, Goa Medical College and Hospital, Bambolim
Email: *joelquadros11[at]gmail.com*

Abstract: ***Spontaneous coronary artery dissection (SCAD) is an uncommon, non-atherosclerotic cause of acute coronary syndrome, characterized by a sudden tear in the coronary arterial wall with subsequent accumulation of blood between its layers. [(1)] Separation of these layers leads to the formation of an intramural hematoma, which may extend under pressure, creating a true and a false lumen. This process compromises coronary blood flow and can result in myocardial infarction, arrhythmias, or sudden cardiac death. [(2,3)] We report the case of a young male who presented with an acute coronary event and was angiographically diagnosed with Type III SCAD. He was managed conservatively with medical therapy. The underlying etiology of his presentation remains under investigation.***

Keywords: Spontaneous Coronary Artery Dissection, Hyperhomocystinemia, Acute Coronary Syndrome

1. Introduction

This case emphasizes spontaneous coronary artery dissection (SCAD) as a rare cause of acute coronary syndrome. It highlights key aspects including pathophysiology, clinical presentation, and diagnosis, focusing on coronary angiography and adjunctive imaging. The report discusses limitations of percutaneous intervention, supports conservative management in stable cases, and outlines indications for surgery. It stresses identifying risk factors like hyperhomocysteinemia, recognizing recurrence risk, and applying appropriate follow-up for improved SCAD management and prognosis.

2. Case Report

A 36-year-old man with a significant history of chronic alcohol use, former tobacco smoking, and long-standing hyperhomocysteinemia (29.2 µmol/L) presented with acute non–ST-elevation myocardial infarction (NSTEMI). His past medical history was notable for portal vein thrombosis in 2013, managed with long-term warfarin therapy targeting an INR of 2–3, and a jejunal perforation in 2019 associated with markedly elevated D-dimer levels (1266.98; normal <500).

Coronary angiography revealed an extensive spontaneous coronary artery dissection (SCAD) involving the left main coronary artery with propagation into both the left anterior descending (LAD) and left circumflex (LCX) arteries (Figures 1 and 2). Despite the severity of the dissection, the patient remained hemodynamically stable throughout his hospitalization. Given his stable condition over the subsequent 48 hours, acute surgical intervention was not pursued, and conservative management was chosen.

At one-month follow-up, a treadmill exercise stress test was performed to assess for inducible ischemia, which was negative. The patient continues on guideline-directed medical therapy and is planned for annual follow-up, including repeat stress testing.

The underlying cause of the SCAD remains uncertain. However, the patient's longstanding hyperhomocysteinemia, previous thrombotic episode, and prior surgical complications suggest a possible underlying systemic vascular vulnerability. A comprehensive thrombophilia workup was unremarkable. Evaluation for Marfan syndrome was not undertaken, as he exhibited no clinical stigmata of connective tissue disorders. Transthoracic echocardiography demonstrated normal aortic valve morphology, preserved left ventricular systolic function (ejection fraction 60%), and no phenotypic features suggestive of syndromic pathology.

3. Methodology

This case report was developed using a retrospective review of the patient's clinical presentation, medical history, diagnostic investigations, treatment course, and follow-up data obtained from hospital records. Diagnostic information was derived from laboratory results, electrocardiography, transthoracic echocardiography, and coronary angiography. Follow-up evaluation included a treadmill stress test performed one month after discharge. All data were anonymized to ensure patient confidentiality.

4. Discussion and Conclusion

Spontaneous coronary artery dissection (SCAD) accounts for approximately 0.1–0.4% of all acute coronary syndrome (ACS) presentations [(3)]. Nearly 90% of cases occur in women aged 30–60 years [(1)]. While plaque rupture remains the predominant cause of ACS in the general population, SCAD is the leading etiology of ACS in young women without traditional cardiovascular risk factors, responsible for up to 25% of cases in women under 50 years [(3)]. SCAD in men is uncommon, and data regarding presentation and outcomes remain limited. Reported mortality is low (1–2%), though

recurrence rates approach 18%. Patients with recurrent events benefit from close in-hospital monitoring for 3–5 days, with vigilant follow-up thereafter [4].

Evidence suggests that percutaneous coronary intervention (PCI) in SCAD is associated with high procedural failure rates, up to 53%, due to technical challenges such as intramural hematoma propagation and guidewire entry into the false lumen [5]. Conversely, conservative management has been associated with favorable in-hospital outcomes, with approximately 90% of patients recovering uneventfully, though a minority may experience early progression necessitating revascularization [5]. Coronary artery bypass grafting (CABG) is generally reserved for left main involvement [3], failed PCI, proximal dissections, or persistent ischemia despite medical therapy [4]. While immediate outcomes of CABG are excellent, with a reported mortality of <2% [3], long-term studies report a high rate of graft failure due to competitive flow with healed native vessels [4].

In the present case, the patient had left main coronary artery dissection with extension into the left anterior descending and circumflex arteries. Given the patient's hemodynamic stability, conservative management was pursued, and the patient remains clinically well. In the absence of conventional risk factors, elevated homocysteine levels may represent a potential contributing factor, as hyperhomocysteinemia is a recognized prothrombotic state associated with premature coronary artery disease and SCAD [6].

Recurrence of SCAD, defined as a new dissection event often at a different coronary site, occurs in approximately 20% of cases [1]. Beta-blocker therapy has been associated with reduced recurrence risk [3]. Patients should be counseled to avoid isometric or high-intensity exercise and prolonged Valsalva maneuvers. Spontaneous arterial healing typically occurs within one month in conservatively managed cases. Follow-up imaging can be performed using coronary CT angiography (CCTA), while repeat invasive angiography is generally reserved for high-risk anatomy or ischemia on functional studies [3]. Non-invasive stress testing remains a useful modality for assessing residual ischemia following a SCAD event [7].

Patient permission/ Consent statement- N/A

Conflict of Interest- There is no conflict of Interest.

Figure Legend's:

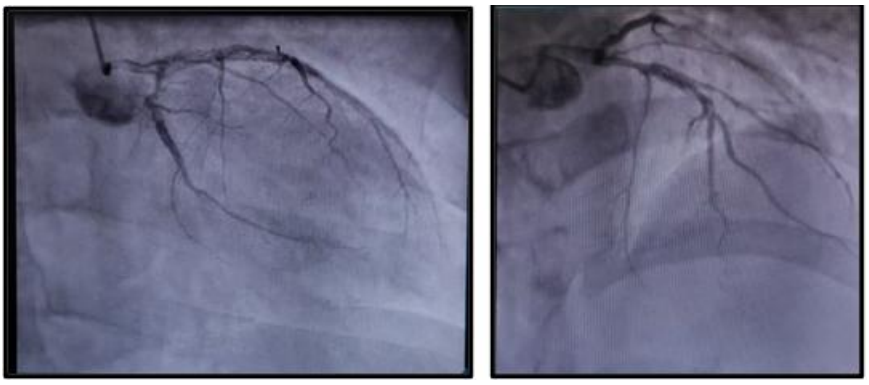

Figure 1 & 2: Spontaneous Coronary Artery Dissection of the left main coronary artery extending to the left anterior descending artery (LAD) and the left circumflex artery (LCX).

References

[1] Heart and Stroke Foundation of Canada. Spontaneous coronary artery dissection (SCAD). Toronto: Heart and Stroke Foundation of Canada; 2025
[2] St. Vincent's Heart health. Spontaneous Coronary artery dissection (SCAD). Sydney-2025.
[3] Dr. Shahid Aziz. Spontaneous Coronary Artery Dissection-European Society of Cardiology. Volume 14, No 38- 22 Feb 2017.
[4] Nepal S, Chauhan S, Bishop MA. Spontaneous Coronary Artery Dissection. (Updated 2023 Jun 21). In: StatPearls (Internet). Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK582143/.
[5] Tweet MS, Eleid MF, Best PJ, Lennon RJ, Lerman A, Rihal CS, Holmes DR, Haynes SN, Gulati R. Spontaneous coronary artery dissection: revascularisation versus conservative therapy. Circ Cardiovasc Interv. 2014 Dec;7(6):777-86. doi: 10.1161/CIRCINTERVENTIONS.114.001659. Epub 2014 Nov 18. PMID: 25406203.
[6] Katsouras CS, Papafalkis MI, Lakkas L, Rammos A, Michalis LK. Three vessel spontaneous coronary artery dissection in a patient with homocysteinemia. J Cardiol Cases. 2021 Sep 21; 25(3): 182-184. soi:10.1016/j.jccase.2021.09.003. PMID: 35261707; PMCID: PMC8888715
[7] Jacqueline Saw, Tara Sedlak, Santhi K. Ganesh, Saul Isserow, et al. Spontaneous Coronary Artery Dissection (SCAD) Jan 6, 2015- American Heart Journal. Copyright 2015, Wolters Kluwer Health.