Case Report

Spontaneous Coronary Artery Dissection Associated with Pulmonary Hemorrhage: A Case Report

Rudaynah A. Alali[1,2]

[1]Department of Internal Medicine, Cardiology Unit, King Fahd Hospital of the University, Al Khobar, [2]College of Medicine, Imam Abdulrahman Bin Faisal University, Dammam 31441, Saudi Arabia

Abstract

Spontaneous coronary artery dissection is becoming an important cause of acute coronary syndrome, particularly among young women. Its association with female gender, pregnancy, and postpartum period and emotional stress differentiate it from atherosclerotic heart disease. In recent years, there has been more awareness and improved diagnostic and management capabilities, which in turn has increased the diagnostic yield, although knowledge gaps remain. In the present case, a 36-year-old female, who was at 1-month postpartum period, presented with ventricular fibrillation and cardiac arrest. The clinical course was associated with pulmonary hemorrhage. The patient had no current atherosclerotic risk factors, only a family history of sudden cardiac arrest in her mother and sister. She underwent a coronary angiogram, which revealed spontaneous coronary artery dissection (SCAD) in both the left anterior descending and left circumflex artery. Percutaneous coronary intervention was performed but the clinical course was associated with pulmonary hemorrhage. Bronchopulmonary lavage was performed as a diagnostic and therapeutic intervention, and she was discharged in good health. SCAD is an important differential diagnosis in young females presenting with acute coronary syndrome or cardiac arrest. Early recognition and diagnosis are important to decrease the high mortality rate of this disease.

Keywords: Acute coronary syndrome, cardiac arrest, coronary angiography, coronary vessel anomalies, postpartum, pregnancy, pulmonary hemorrhage, spontaneous coronary artery dissection

Address for correspondence: Dr. Rudaynah A. Alali, Department of Internal Medicine, Cardiology Unit, King Fahd Hospital of the University, Al Khobar, Saudi Arabia.
E-mail: raali@iau.edu.sa

Submitted: 14-Oct-2023 **Revised:** 28-Dec-2023 **Accepted:** 27-Feb-2024 **Published:** 05-Apr-2024

INTRODUCTION

Spontaneous coronary artery dissection (SCAD), an emergency ischemic event, forms a false lumen that compresses the true lumen of the coronary vessel, resulting in cardiac ischemia and acute coronary syndrome.[1,2] In the pre- and postpartum periods, patients with SCAD are likely to present with an ST-elevation myocardial infarction (STEMI). SCAD is significantly correlated with multiparity, infertility treatment, and pre-eclampsia as well as connective tissue diseases.[3,4] A prospective observational study from Canada found that 69.9% of SCAD patients did not have STEMI, 29.7% had STEMI, 8.1% had a life-threatening ventricular tachycardia or fibrillation, and <3% presented with cardiogenic shock. Chest discomfort was the most common presenting symptom.[5]

Access this article online	
Quick Response Code:	Website: https://journals.lww.com/sjmm
	DOI: 10.4103/sjmms.sjmms_471_23

How to cite this article: Alali RA. Spontaneous coronary artery dissection associated with pulmonary hemorrhage: A case report. Saudi J Med Med Sci 2024;12:194-7.

The gold standard for diagnosing SCAD is invasive coronary angiography with intra-coronary imaging, which increases the yield of diagnosis.[6] Both optical coherence tomography and intravascular ultrasound (IVUS) assess the presence of intramural hematoma or a double lumen.[7,8]

Here, to the best of the author's knowledge, the first case of SCAD associated with pulmonary hemorrhage is reported.

CASE REPORT

A 36-year-old female, who was at 1-month postpartum, experienced sudden chest pain and collapsed. Return of spontaneous circulation (ROSC) was achieved after 20 minutes of cardiopulmonary resuscitation with an initial shockable rhythm of ventricular fibrillation. The results of post-ROSC electrocardiogram showed sinus rhythm with T-wave inversion in V1 and V2. A transthoracic echocardiogram (TTE) showed severe global left ventricular hypokinesis with regional variability and a left ventricular ejection fraction (LVEF) of 29%. The patient had a family history of sudden cardiac death in her sister and mother.

An urgent coronary angiogram was performed while the patient was on mechanical ventilation. She had been loaded with aspirin and clopidogrel as dual anti-platelet therapy. Femoral access was achieved, and the left coronary system was engaged with no ventricularization of the pressure waves. It showed a dissection from the left main (LM) artery, extending into both bifurcations of the left anterior descending (LAD) and left circumflex (LCx) artery [Figure 1]. The right coronary artery was normal. An intra-aortic balloon pump (IABP) was inserted. An IVUS confirmed that the wire was in the true lumen of the LAD and the dissection extended into the proximal LAD. The Culotte technique was used to perform bifurcation percutaneous coronary intervention to LM/LAD/LCx [Figures 2 and 3]. A repeat IVUS of LAD showed well-apposed stent struts in the LM and proximal LAD with a hematoma compressing the non-stented midsegment of the LAD.

On the following day, the IABP was successfully removed with good hemostasis and the therapeutic heparin infusion was discontinued. On the subsequent day, the patient started to desaturate to <80% while still on mechanical ventilation, and oxygen requirements increased to the maximum. Her complete blood count showed a 4 g/dL drop in hemoglobin (from 12 g/dL to 8 g/dL) over a 24-hour period and there were blood-streaked excretions from the endotracheal tube. A chest X-ray showed bilateral

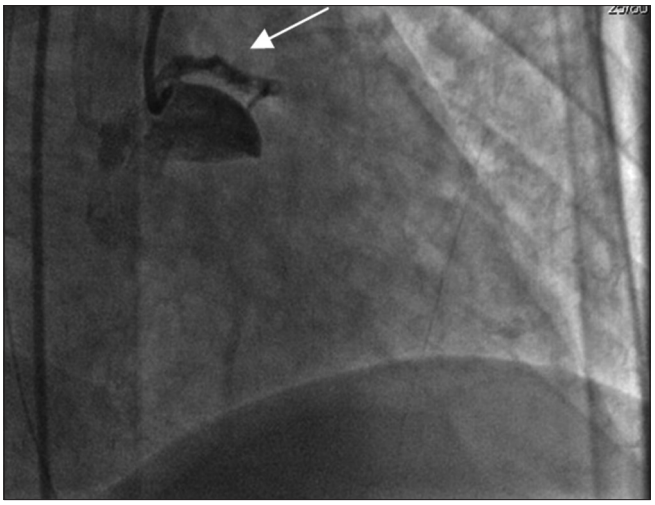

Figure 1: Initial coronary angiography showing dissection extending from the distal left main into the left anterior descending and left circumflex arteries (white arrow)

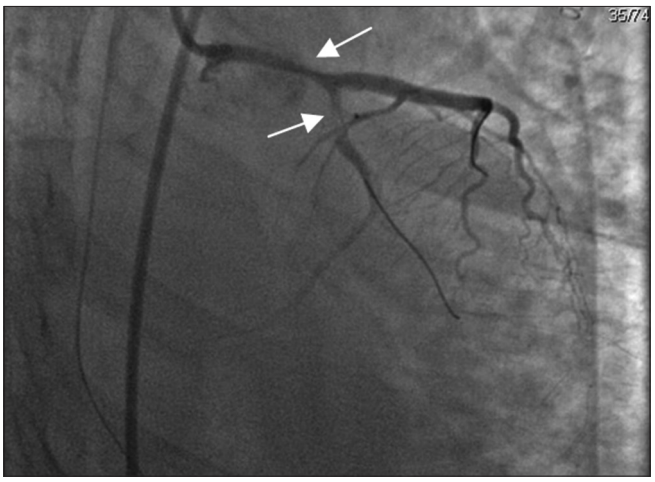

Figure 2: Postballoon dilatation coronary angiography showing return of TIMI II flow after balloon dilatation of left main/left anterior descending (white arrows)

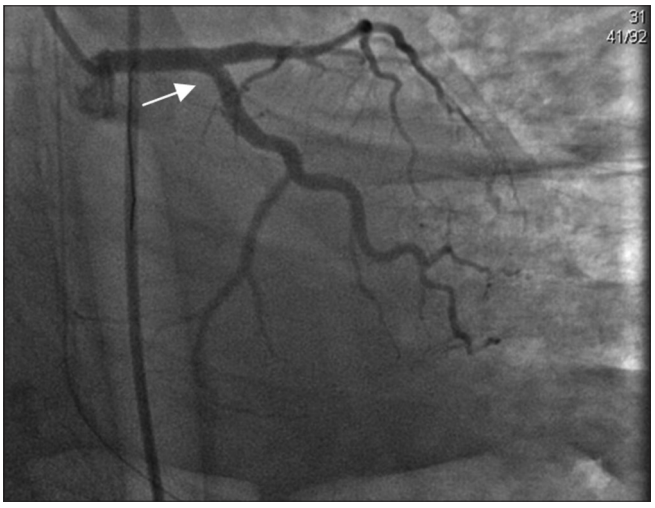

Figure 3: Post-percutaneous coronary intervention angiography after performing the Coulotte technique (white arrow)

infiltrations. A computed tomography (CT) scan of her chest revealed bilateral patchy ground glass opacities giving the appearance of intralobular septal thickening and peribronchial consolidations. Based on the findings of the CT scan and the acute drop in hemoglobin levels, a pulmonary hemorrhage was suspected. She was transfused with two units of packed red blood cells and started on intravenous methylprednisolone. A bronchoscopy was performed, which showed obvious bleeding but no clot. A bronchopulmonary lavage was performed with >50% return. At the end of the procedure, there was no active bleeding and the patient's oxygen saturation increased to 84% on 100% oxygen through mechanical ventilation.

The patient's hemoglobin stabilized without further blood transfusion, and she was extubated 2 days later with no further complications. The patient's dual anti-platelet therapy was continued due to the high risk of thrombosis in the recently deployed complex coronary bifurcation stenting. A repeat TTE after the extubation revealed normalized LVEF to 64% with no regional wall motion abnormalities. She was discharged on dual anti-platelet, beta blocker, and statin therapy. All the autoimmune blood investigations that were performed during her admission returned negative. She was advised to repeat all the autoimmune work up 6–8 weeks after discharge to confirm if there are any underlying auto-immune disorders.

The patient was being regularly followed up in the cardiology clinic for the past 1 year at the time of reporting this case, and her clinical condition had been stable.

DISCUSSION

The patient in this case presented with cardiac arrest and SCAD was diagnosed in the left coronary system in the first month postpartum, which was associated with pulmonary hemorrhage. There are no randomized trials to compare management strategies for SCAD; therefore, many of the recommendations are from observational studies and expert opinions. Unless the patient has high-risk features such as left main dissection, continued ischemia, and electrical or hemodynamic instability, conservative management is the preferred strategy. In the present case, the high-risk features included a strong family history of sudden cardiac death in her sister and mother, involvement of the LM artery dissection, and hemodynamic instability.[2,9]

Upon literature review and to the best of the author's knowledge, there have been no case reports of patients with SCAD associated with pulmonary hemorrhage. The very strong family history of sudden cardiac death indicated the involvement of possible genetic influences. SCAD associated with pulmonary hemorrhage is likely due to medial hemorrhage as an underlying pathophysiology of SCAD caused by either primary thrombosis or rupture of the vasa vasorum, with consequent hemorrhage into the vessel wall. This may well occur in the coronary arterial tree and manifest as SCAD and may also occur in the pulmonary vasculature and present as pulmonary hemorrhage, as in this case.[10]

In the Canadian SCAD study, patients were frequently discharged on aspirin (93.7%), P2Y12 inhibitor (67.4%), beta-blocker (84.8%), ACEI or ARB, and statin. The survival rate was excellent; however, 8.8% of the patients had a major adverse cardiovascular event (MACE), which included: recurrent myocardial infarction (MI), ventricular arrhythmias, cardiogenic shock, and unplanned revascularization and resulted in the death of one patient while in hospital. In addition, the median duration of hospital stay was 4 days, and connective tissue disorders and peripartum SCAD were found to be independent predictors of 30-day MACE.[5]

Routine genetic testing is not currently advised, especially in patients who do not show any sign of an inherited systemic arteriopathy or connective tissue disease.[11] If there is suspicion of an underlying or inherited cause for SCAD, these patients should be referred to a medical geneticist, as should also have been the case in the current reported case.[7,12]

CONCLUSIONS

SCAD is an important cause of MI that predominately affects young to middle-aged women. To the best of the author's knowledge, this is the first reported case of SCAD associated with pulmonary hemorrhage. Bronchoscopic lavage in diagnosing and treating the pulmonary hemorrhage may have allowed the continuation of the dual antiplatelet therapy in this acute ischemic event.

Declaration of patient consent

The author certifies that all appropriate patient consent forms have been obtained. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published, and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Ethical considerations

The Institutional Review Board of Imam Abdulrahman Bin Faisal University provided ethical approval for reporting this case (Ref. no.: IRB-2024-01-144).

Peer review

This case report was peer-reviewed by three independent and anonymous reviewers.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Waterbury TM, Tweet MS, Hayes SN, Eleid MF, Bell MR, Lerman A, *et al*. Early natural history of spontaneous coronary artery dissection. Circ Cardiovasc Interv 2018;11:e006772.
2. Igbokwe N, Gomersall J, Ugwoke SP, Esmonde S. Pregnancy-associated spontaneous coronary artery dissection: Multidisciplinary management, challenges and literature review. BMJ Case Rep 2021;14:e244851.
3. Tweet MS, Hayes SN, Codsi E, Gulati R, Rose CH, Best PJ. Spontaneous coronary artery dissection associated with pregnancy. J Am Coll Cardiol 2017;70:426-35.
4. Saw J, Ricci D, Starovoytov A, Fox R, Buller CE. Spontaneous coronary artery dissection: Prevalence of predisposing conditions including fibromuscular dysplasia in a tertiary center cohort. JACC Cardiovasc Interv 2013;6:44-52.
5. Saw J, Starovoytov A, Humphries K, Sheth T, So D, Minhas K, *et al*. Canadian spontaneous coronary artery dissection cohort study: in-hospital and 30-day outcomes. Eur Heart J. 2019;40(15):1188-97
6. Prakash R, Starovoytov A, Heydari M, Mancini GB, Saw J. Catheter-induced iatrogenic coronary artery dissection in patients with spontaneous coronary artery dissection. JACC Cardiovasc Interv 2016;9:1851-3.
7. 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;89:466-8.
8. Alfonso F, Paulo M, Gonzalo N, Dutary J, Jimenez-Quevedo P, Lennie V, *et al*. Diagnosis of spontaneous coronary artery dissection by optical coherence tomography. J Am Coll Cardiol 2012;59:1073-9.
9. Zupan I, Noc M, Trinkaus D, Popovic M. Double vessel extension of spontaneous left main coronary artery dissection in young women treated with thrombolytics. Catheter Cardiovasc Interv 2001;52:226-30.
10. Henkin S, Negrotto SM, Tweet MS, Kirmani S, Deyle DR, Gulati R, *et al*. Spontaneous coronary artery dissection and its association with heritable connective tissue disorders. Heart 2016;102:876-81.
11. Hayes SN, Kim ES, Saw J, Adlam D, Arslanian-Engoren C, Economy KE, *et al*. Spontaneous coronary artery dissection: Current state of the science: A scientific statement from the American Heart Association. Circulation 2018;137:e523-57.
12. Kaadan MI, MacDonald C, Ponzini F, Duran J, Newell K, Pitler L, *et al*. Prospective cardiovascular genetics evaluation in spontaneous coronary artery dissection. Circ Genom Precis Med 2018;11:e001933.