JACC: CASE REPORTS VOL. 30, NO. 2, 2025

ISCHEMIC HEART DISEASE

CLINICAL CASE

Pregnancy-Associated Spontaneous Coronary Dissection in a 32-Year-Old During the Third Trimester

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ABSTRACT

We report a case of spontaneous coronary dissection (SCAD) in a 32-year-old pregnant patient during the seventh month of her second pregnancy. A 32-year-old pregnant woman in the 28th week of gestation was referred to our intensive care unit because of angina as well as elevated troponin levels. The initial electrocardiogram and transthoracic echocardiogram (TTE) were normal. Four hours after admission, the patient experienced angina with ST-segment elevation, and the TTE showed de novo apical hypokinesia. The episode lasted approximately 10 minutes, with subsequent resolution of the ST-segment elevation. An emergency coronary angiogram revealed dissection of the left anterior descending artery. A conservative approach with aspirin monotherapy was chosen. Follow-up TTE at 3 months revealed full recovery of left ventricular function. A multidisciplinary approach is crucial in pregnancy-associated SCAD. Conservative management is generally recommended because of the potential for angiographic healing, with percutaneous coronary intervention reserved for severe cases. (JACC Case Rep. 2025;30:102769) © 2025 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Spontaneous coronary artery dissection (SCAD) and pregnancy-associated SCAD (P-SCAD) have been increasingly identified over the past decades as a significant cause of acute myocardial infarction, heart failure, and sudden cardiac death. In this case report, we highlight a particularly rare case of P-SCAD in an otherwise healthy woman during her seventh month of pregnancy that manifested as an ST-segment elevation myocardial infarction (STEMI).

TAKE-HOME MESSAGES

- P-SCAD should be suspected in any patient with peripartum or postpartum angina and should preferably be treated in a hospital with maximal capacities by a multidisciplinary team.
- Individualized decision making regarding PCI should be prioritized.

HISTORY OF PRESENTATION

A 32-year-old pregnant woman, in the 28th week of her second pregnancy, was referred to our cardiology intensive care unit (ICU; University Hospital Giessen, Hesse, Germany) from a peripheral hospital for suspected myocarditis requiring further monitoring,

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received August 16, 2024; revised manuscript received September 18, 2024, accepted October 3, 2024.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2024.102769

ABBREVIATIONS AND ACRONYMS

DAPT = dual antiplatelet therapy

ECG = electrocardiogram

ICU = intensive care unit

LAD = left anterior descending (artery)

LM = left main (coronary artery)

PCI = percutaneous coronary intervention

P-SCAD = pregnancy-associated spontaneous coronary artery dissection

SAPT = single antiplatelet therapy

SCAD = spontaneous coronary artery dissection

STEMI = ST-segment elevation myocardial infarction

TTE = transthoracic echocardiogram

diagnostic evaluation, and treatment. She had presented to the external emergency department with sudden onset chest pain, lasting up to 15 minutes, which subsequently resolved. The symptoms were isolated, without pain radiation.

PAST MEDICAL HISTORY

The patient had no previous history of coronary artery disease, SCAD, aortic dissection, pulmonary embolism, or myocarditis. She denied experiencing palpitations or syncope and had no family history of coronary artery disease, SCAD, or P-SCAD. Previous medication consisted of L-thyroxine, 112 μg.

PHYSICAL EXAMINATION

The results of a physical examination were unremarkable, with a blood pressure of 137/93 mm Hg and a heart rate of 76 beats/min; her oxygen saturation was normal at 96%. No cardiac murmurs, dilated jugular veins, or leg edema were noted. Lung auscultation was normal. A gynecologic examination, a cardiotocogram, and an ultrasound scan ruled out any pregnancy complications.

INVESTIGATIONS

Laboratory test results revealed that cardiac biomarker levels were elevated: high-sensitivity-troponin I, 2,194 ng/L, peaking at 17,410 ng/L; creatine kinase, 762 U/L; and creatine kinase-myocardial band, 88 U/L. The initial electrocardiogram (ECG) was normal, without signs of ischemia or accelerated heart rhythm (**Figure 1**). A transthoracic echocardiogram (TTE) showed normal left ventricular function without abnormalities. Four hours after admission, the patient experienced another episode of sudden onset chest pain. The ECG showed hyperacute ST-segment elevation in leads I, II, aVL, and V_4 to V_6, as well as concordant ST-segment depression in leads aVR and V_1 (**Figure 2**). A TTE indicated good left ventricular function overall, with apical hypokinesia. The episode lasted approximately 10 minutes, and a subsequent ECG showed resolution of the ST-segment elevation, albeit with residual discrete elevation in lead V_6 as well as negative T waves in leads V_5 and V_6 (**Figure 3**).

MANAGEMENT

A multidisciplinary team consisting of cardiologists, anesthesiologists, obstetrician/gynecologists, and neonatologists was convened for further diagnostic and therapeutic planning. The consensus was to perform a diagnostic coronary angiogram, with readiness to perform an emergency cesarean delivery contingent on the findings. The patient was hemodynamically stable at the point of the coronary angiogram. The angiogram revealed a slight illumination with delayed coronary flow in the left anterior descending (LAD) artery; the remainder of the vessels appeared intact (**Figures 4 and 5**, Videos 1 and 2). The finding in the LAD artery was suggestive of a spontaneous dissection (Yip-Saw classification type 2), with coronary flow in the distal part of the artery still present. Intracoronary imaging was not performed because of the obvious angiographic features of the lesion and to avoid further radiation exposure and reduce procedural risk during the preterm pregnancy. Additionally, we decided against percutaneous coronary intervention (PCI) on the basis of the position of the lesion (mediodistal) and to avoid complications such as vessel occlusion and hematoma propagation. Because of the high bleeding risk during the planned repeat cesarean operation, a conservative approach with permanent aspirin therapy was initiated. The procedure ended without any complications. Subsequently, aspirin monotherapy was initiated.

OUTCOME AND FOLLOW-UP

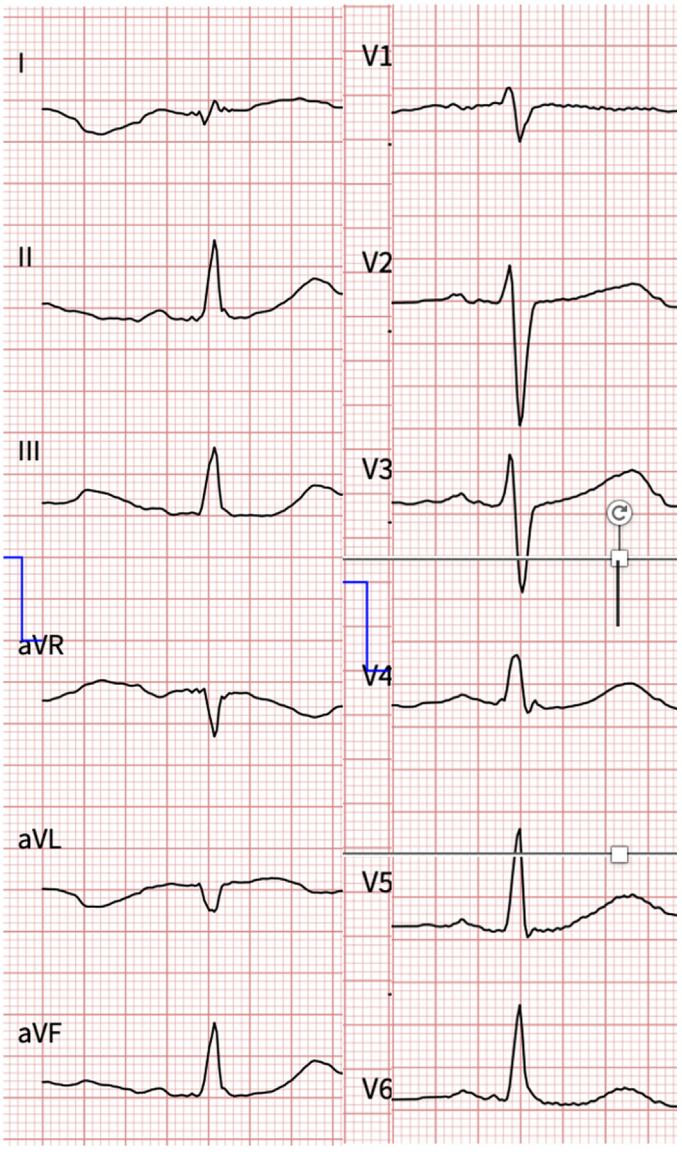
The patient was monitored in the ICU for an additional 10 days without further incidents. An early follow-up TTE 2 weeks later showed complete recovery of the apical wall contraction. The delivery was uncomplicated and occurred 8 weeks later. No peripartum or postpartum cardiac events were noted. Follow-up TTE and ECG at 3 months revealed full recovery of left ventricular function without signs of apical hypokinesia.

DISCUSSION

P-SCAD is an extremely rare cause of acute myocardial infarction of unknown origin that can occur at any time during pregnancy, although it most commonly occurs post partum, primarily within the first week.[1] Current data suggest a prevalence of 1.81 per 100.000 pregnancies,[2] and P-SCAD comprises 5% to 17%[3-5] of all SCAD cases. Approximately 14.5% to 43%[6,7] of P-SCAD cases manifest as acute myocardial infarction. Multiparity, fertility hormones, and preeclampsia have been reported as the main risk factors.[8,9] Previous data have linked P-SCAD to conditions that are more severe than SCAD, in particular STEMI and occlusion of the proximal coronary arteries, as well as reduced ejection fraction in P-SCAD survivors.[2,10] Other identified risk factors for SCAD, such as fibromuscular dysplasia, inflammatory and/or connective tissue disorders, atherosclerosis, genetic predisposition, and mechanical and emotional stressors, may play a role in the pathogenesis of P-SCAD, albeit data on this remain scarce.[11] Moreover, P-SCAD is frequently associated with left main (LM) coronary artery and multivessel dissections, cardiogenic shock, and impaired left ventricular ejection fraction during the acute phase of the dissection.[10]

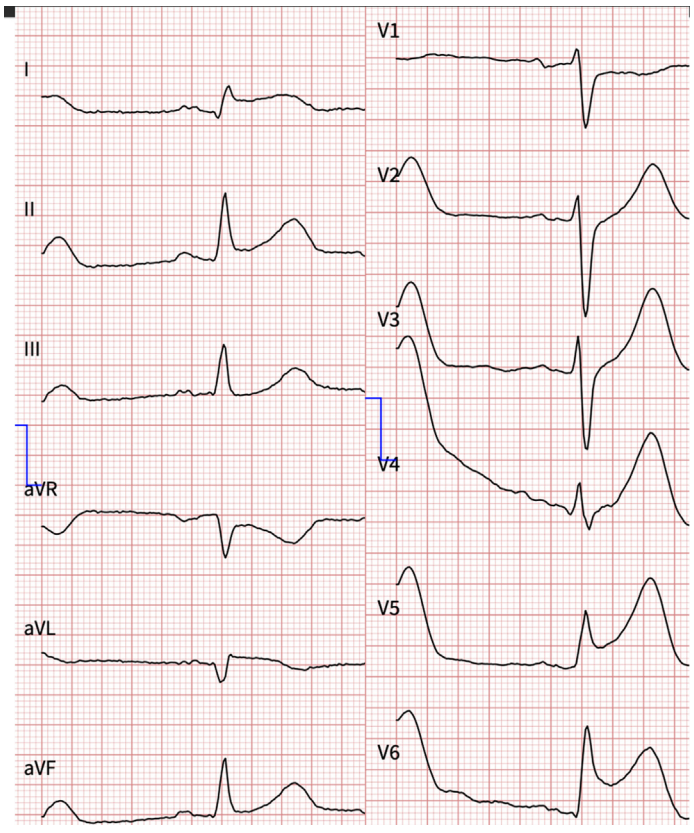
SCAD or P-SCAD typically manifests with symptoms similar to those of acute coronary syndrome. The main difference is the phenotype because the typical patient with SCAD or P-SCAD is a young or middle-aged woman during or after pregnancy with few or no cardiovascular risk factors. The primary diagnostic tool is coronary angiography. The Yip-Saw classification[12] and morphologic characteristics such as the absence of intraluminal thrombus and vessel tortuosity are key factors in correctly diagnosing SCAD and differentiating it from "typical," plaque rupture-mediated coronary dissection. PCI is associated with an elevated risk of complications and adverse outcomes. Data suggest that especially distal lesions with preserved flow should be managed

FIGURE 1 Baseline Electrocardiogram



Initial electrocardiogram without any signs of acute coronary ischemia.

FIGURE 2 ST-Segment Elevation Myocardial Infarction Electrocardiogram



Hyperacute ST-segment elevation in leads I, II, aVL, and V_4 to V_6 during a sudden onset angina episode.

conservatively because angiographic healing has been demonstrated in repeat angiograms.[4,13] However, the treatment of SCAD or P-SCAD affecting the LM coronary artery, the proximal LAD artery, or more than 1 major vessel, or in patients with hemodynamic deterioration or malignant arrhythmias, remains even more challenging and more complex, with the potential negative impact of the hemodynamic status. In these cases, PCI should be performed in patients with SCAD or P-SCAD, even when taking into account slow or low flow in the target lesion.

In a retrospective study comparing patients with SCAD who underwent PCI with patients who were treated conservatively, there were more in-hospital complications and recurrent myocardial infarctions in the PCI cohort.[14]Additionally, in a case report of a patient with P-SCAD of the LM artery with subsequent cardiogenic shock, a conservative approach was chosen and the patient recovered completely, without requiring further angiographies or PCI.[15] Nevertheless, the present case was more difficult in terms of decision making because the patient was pregnant and a cesarean delivery was scheduled in the near future.

Given the paucity of data on the appropriate medical management of SCAD/P-SCAD, recommendations are generally varied. There is consensus around the necessity of dual antiplatelet therapy (DAPT) in patients undergoing PCI.[16] However, there is still debate on the use of DAPT in conservatively treated patients, the dose and duration of DAPT, or the use of single antiplatelet therapy (SAPT).[17] Data

from the DISCO (DIssezioni Spontanee COronariche [Spontaneous Coronary Dissection]) registry reported a more than 2-fold increased risk of major adverse cardiovascular events in conservatively treated patients with SCAD who were discharged on DAPT compared with patients receiving SAPT.[18] Most current data on medical therapy is based on the general group of patients with SCAD because data on the optimal treatment in patients with P-SCAD is scarce.

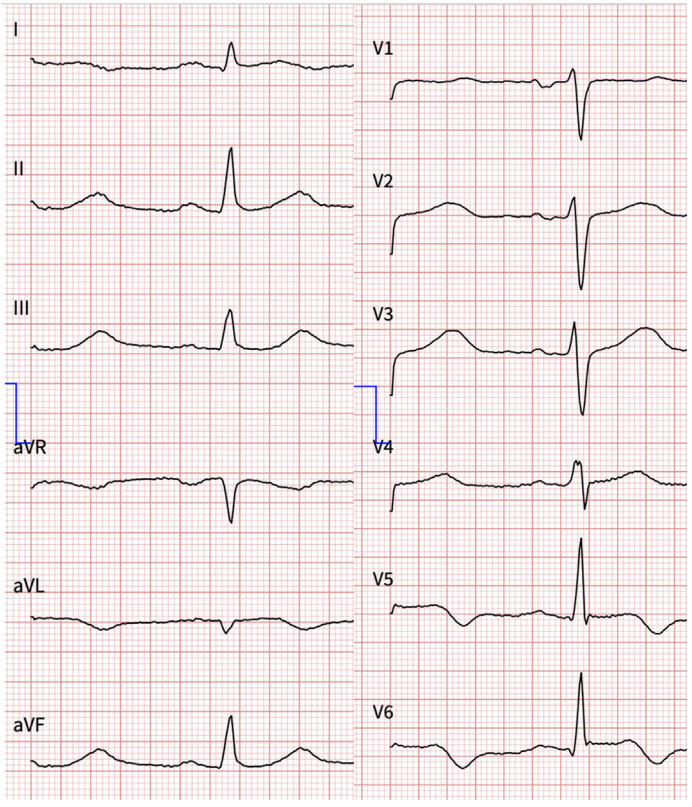
The present case highlights several key factors in patient management and decision making. First, patients with suspected P-SCAD should be referred to a hospital with facilities to allow for a multidisciplinary diagnostic and therapeutic strategy that will safeguard against complications for both the patient and the infant. Second, intracoronary imaging should be used only in cases where the diagnosis cannot be established by standard coronary angiography, to avoid unnecessary complications. Additionally, individual decision making regarding the use of PCI, apart from the region of the culprit lesion, should also take into account immediate and future adverse effects. Iatrogenic injury of the vessel, as well as long-term bleeding complications, should be considered before PCI in undertaken. Moreover, especially in patients with P-SCAD while still pregnant, complications from perioperative bleeding during childbirth should be carefully considered. Finally, this case highlights the paroxysmal nature of the ST-segment elevation with which patients with P-SCAD can present.

CONCLUSIONS

This case highlights the complex nature of P-SCAD and the demanding management required for pregnant patients with this condition, and it adds to the relatively small data pool of these cases. A multidisciplinary approach is necessary to achieve the best possible outcome.

ACKNOWLEDGMENTS The authors would like to thank Professor B. Unsöld, Professor B. Aßmus, Dr M. Arsalan, and Dr G. Ibrahimi for their valuable contribution in this case.

FIGURE 3 Electrocardiogram After Angina Episode



Resolution of ST-segment elevation, residual elevation in lead V_6, negative T waves in leads V_5 and V_6.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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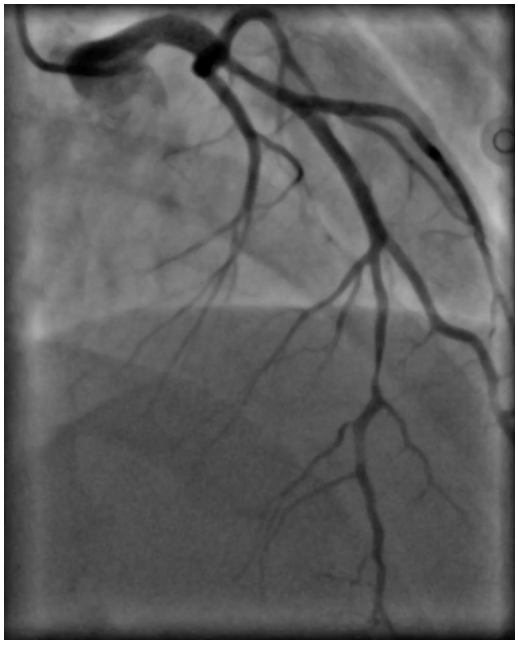
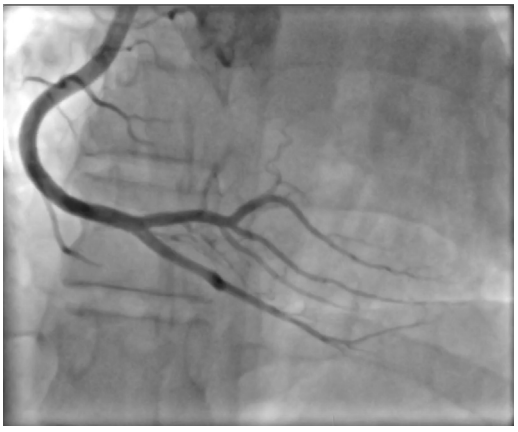
FIGURE 4 Emergency Coronary Angiogram (Left Anterior Oblique Projection)



Spontaneous dissection in the mediodistal part of the left anterior descending artery.

FIGURE 5 Emergency Coronary Angiogram (Right Anterior Oblique Projection)



Normal right coronary artery.

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KEY WORDS **acute coronary syndrome, coronary intervention, pregnancy-associated coronary artery dissection (P-SCAD), spontaneous coronary artery dissection (SCAD), ST-segment elevation myocardial infarction**

APPENDIX **For supplemental videos, please see the online version of this paper.**