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Case Report Open Access

Spontaneous Coronary Artery Dissection Causing Cardiac Arrest in the Early Postpartum Period: A Case Report

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Abstract

Introduction: Spontaneous coronary artery dissection (SCAD) is an uncommon but important cause of acute coronary syndrome (ACS), predominantly affecting young women without traditional cardiovascular risk factors. The postpartum period represents a particularly high-risk phase, during which pregnancy-associated SCAD often manifests with severe clinical features such as left main coronary involvement, ventricular dysfunction, or sudden cardiac arrest.

Case Presentation: Twelve days after cesarean delivery, a 39-year-old lady developed acute chest discomfort, loss of consciousness, and cardiac arrest. Electrocardiography showed ST-segment elevation in leads V1–V4 with Q waves in V1–V3 after CPR. Anteroseptal hypokinesia and lowered left ventricular ejection fraction by 25–35% on transthoracic echocardiography. Coronary angiography indicated substantial ostial and proximal left anterior descending artery constriction and mild left main coronary artery involvement, confirming type 2A SCAD. The patient was conservatively treated with dual antiplatelet therapy, beta-blocker, guideline-directed heart failure medication, and vigilant monitoring. Repeat angiography showed near-complete spontaneous coronary dissection repair. The patient was clinically stable and improved left ventricular systolic function over 6 months. The patient was advised to do cardiology follow-up every 6–12 months for evaluation.

Conclusion: This case underscores the potential severity of postpartum SCAD while highlighting favorable outcomes with conservative management. Early recognition of SCAD in young postpartum women presenting with ACS is crucial to avoid misdiagnosis and guide appropriate therapy. The patient's meaningful recovery in cardiac function (EF rising from 25% to 45%) at follow-up supports conservative treatment and follow up strategies in appropriately selected cases.

Keywords: Spontaneous coronary artery dissection, Acute coronary syndrome, Postpartum period, Myocardial infarction, Cardiac arrest, Coronary angiography.

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Introduction

Spontaneous coronary artery dissection (SCAD), although infrequent, is a growingly acknowledged etiology of acute coronary syndrome (ACS), especially among young, otherwise healthy women lacking conventional atherosclerotic risk factors.[1] SCAD is defined by the development of an intramural hematoma or an intimal tear within the coronary artery wall, which subsequently compresses the arterial lumen externally, thereby precipitating myocardial ischemia or infarction.[2] Historically, SCAD has been underdiagnosed; nevertheless, increased awareness and improvements in intracoronary imaging have substantially augmented the number of reported instances in the last decade. Recent findings indicate that SCAD constitutes 1–4% of all ACS cases, and 22–43% of ACS cases in women under 50 years of age.[3]

The risk of SCAD is increased by factors including fibromuscular dysplasia (FMD), connective tissue disorders, systemic inflammatory diseases, pregnancy, and the postpartum period.[4] Pregnancy-

associated SCAD (P-SCAD) frequently manifests within the initial month following childbirth and is generally more severe than SCAD not linked to pregnancy.[5] P-SCAD is correlated with elevated incidences of multivessel involvement, left main coronary artery disease, ventricular dysfunction, and hemodynamic instability.[6]

SCAD often presents as either ST-segment elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI). Symptoms may encompass chest discomfort, cardiogenic shock, or sudden cardiac arrest.[7]

Coronary angiography is frequently used to confirm the diagnosis.[8] Most cases of SCAD resolve spontaneously, and conservative treatment is typically the preferred approach. However, revascularization may be required in some cases.[9]

This report presents a 39-year-old postpartum woman who suffered cardiac arrest and anterior STEMI, ultimately diagnosed with SCAD involving the left main coronary artery (LMCA) and left anterior descending artery (LAD). This case underscores the importance of early recognition of pregnancy-associated SCAD in postpartum women presenting with acute coronary syndrome and cardiac arrest. Additionally, it highlights the diagnostic challenges, management considerations, and favorable outcomes achievable with conservative therapy in appropriately selected patients.

Presentation

A 39-year-old woman from Tehran was admitted to Baqiatallah Hospital on May 3rd, 2023, at 17:58 after losing consciousness. Earlier that day, she had chest pain that went down her left arm and shoulder, which continued even when she was resting (FC 4). The patient lost consciousness around 5:00 p.m., which led to a call to emergency medical services (EMS). When EMS arrived, the patient had no pulse, so cardiopulmonary resuscitation (CPR) was started right away. The defibrillator showed ventricular fibrillation, and two 100-J defibrillation shocks were given, which restored her heartbeat. After she regained consciousness, the patient was confused and was then taken to the emergency department.

Upon arrival, the patient said she had typical left-sided chest pain that went to her shoulder, along with nausea, dyspnea (worsening in the supine position), and cold sweating.

The patient's medical history included a recent elective cesarean section performed twelve days earlier. There was no significant family or personal medical history.

The physical exam showed a conscious but agitated patient. Vital signs were: blood pressure 121/69 mmHg, pulse rate 94 bpm, and oxygen saturation 86%. Chest wall movements were normal, and listening to the heart revealed no abnormal sounds. An electrocardiogram (ECG) showed ST-segment elevation in leads V1-V4 and Q waves in leads V1-V3 (Figure 1). Initial and later laboratory results are summarized in Table 1.

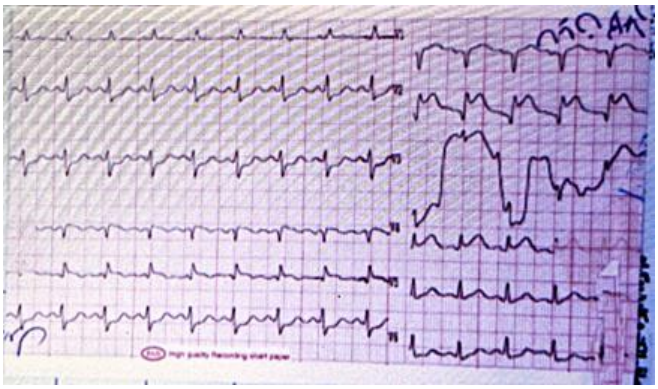

Figure 1. Electrocardiogram showing ST-segment elevation in leads V1–V4 with Q waves in leads V1–V3, consistent with an acute anteroseptal myocardial infarction in the setting of spontaneous coronary artery dissection.

Table 1. The initial lab tests and the course of lab tests

LAB TEST/ Date & Time	(Admission) 02/13 , 18:29	(Discharge) 02/20 , 21:45
WBC(3.9-11.1)	13.1	6.5
HB(11.6-15.3)	12.9	12.0
PLT(155-440)	356	202
BS(<200)	192	106
Cr(0.6-1.1)	1.4	1.1
SGOT(<31)	343	52
SGPT(<34)	328	91
ALK.P(<270)	232	185
CPK(26-140)	135	-
LDH(207-414)	928	-
PT(<14.5)	15.0	13.9
INR(0.8-1.2)	1.12	1.1
PTT(30-40)	38.0	46.2
D-DIMER(<1.0)	3.04	
ESR(<20)	40	
CRP(<5)	6.6	
VBG	Normal	
Blood Culture	Negative	

Troponin-I serum levels and it's course

Date	02/13 –	02/13	02/13
Time	18:29	20:41	22:05
Troponin I	0.128	>50	50.0

Transthoracic echocardiography showed an ejection fraction (EF) of 25-35%, with reduced movement in the septum and anteroseptal wall. Given the significant increase in troponin levels (Table 1), indicating heart muscle damage, the patient received 180 mg Ticagrelor, 320 mg Aspirin, 40 mg Pantoprazole, 40 mg Atorvastatin, and subcutaneous Heparin. She was then transferred for primary percutaneous coronary intervention (PCI). Coronary angiography, done on May 3rd, 2023, at 19:10, showed moderate narrowing in the left main coronary artery (LMCA) and significant narrowing in the left anterior descending artery (LAD). The patient was diagnosed with Spontaneous Coronary Artery Dissection (SCAD) Type 2A and was moved to the coronary care unit (CCU) for monitoring and treatment.

Later, the following medications were ordered: daily Aspirin (20mg), Plavix(75mg), Pantoprazole(40mg), Atorvastatin(20mg), Bisoprolol(2.5 mg BiD), and a heparin drip. The patient received Lasix (20mg BiD) and Ondansetron(4mg PRN) to manage nausea, and serial troponin assessments and electrocardiograms were performed. Subsequent to consultations with gynecology and rheumatology, all diagnostic findings were within normal limits.

A follow-up transthoracic echocardiogram revealed mild to moderate left ventricular dilation, accompanied by an ejection fraction of 25%. Consequently, the Lasix dosage was decreased, and Aldactone was initiated. Furthermore, heparin was discontinued in the following days due to the occurrence of vaginal bleeding. Repeat angiography performed on May 10, 2023, at 14:20, demonstrated substantial resolution of the dissection, with the stenosis in both the LMCA and LAD having resolved.

The patient's clinical status improved, and she was subsequently discharged on a regimen of aspirin, clopidogrel, bisoprolol, spironolactone, pantoprazole, and short-term atorvastatin therapy, with explicit instructions for routine follow-up appointments.

The patient was followed at 1, 3, and 6 months post-discharge and remained clinically stable without recurrent chest pain or syncope. Serial transthoracic echocardiography showed gradual improvement in left ventricular systolic function, with an ejection fraction of approximately 40% at 6 months. Overall, the patient demonstrated favorable recovery with conservative medical management and was advised to continue cardiology follow-up every 6–12 months.

Discussion

This example underscores significant characteristics of spontaneous coronary artery dissection, particularly in the postpartum setting. A 39-year-old lady, 12 days after a cesarean section, came in with chest pain, then lost consciousness, and then went into asystole, which required CPR and defibrillation. When she got there, she had typical ischemia symptoms, such as ST-segment elevation in leads V1–V4 with Q waves and a very low left ventricular ejection percent. Coronary angiography revealed significant proximal stenosis in the LAD and LMCA, heightening suspicion for SCAD. A subsequent angiography revealed signs of spontaneous repair, consistent with the natural development of the illness.

1. Pregnancy-associated SCAD and vulnerability in the postpartum period

The postpartum period is an acknowledged risk factor for SCAD and its complications. Changes in hormones, higher heart output during pregnancy, and changes in the structure of collagen and the vascular media make coronary arteries more likely to get damaged.[10] The patient's presentation, occurring 12 days postpartum and worsened by cardiac arrest involving the LAD and LMCA, corresponds with the recognized high-risk characteristics of P-SCAD. The patient's history of repeated miscarriages and the utilization of prophylactic anticoagulation during pregnancy indicate possible underlying vascular fragility or prothrombotic tendencies. Rheumatologic testing and subsequent evaluations did not reveal a conclusive systemic connective tissue condition or inflammatory origin.

2. Sudden cardiac arrest and a big first sign

Cardiac arrest, however infrequent, has been documented as a presenting symptom of SCAD. Recent registry data show that 8–14% of those with SCAD have ventricular arrhythmias or sudden cardiac arrest.[11]. The process is probably linked to acute ischemia caused by dynamic luminal blockage from an intramural hematoma.[4] The patient's rapid restoration of spontaneous circulation and subsequent neurological recovery align with other studies indicating that prompt resuscitation can yield favorable outcomes.

3. Angiographic Features and Diagnosis

Coronary angiography is the main way to diagnose SCAD. Common angiographic findings include lengthy, even stenosis, many radiolucent lumens, or evidence of an intramural hematoma.[12] The SCAD angiographic classification system identifies three separate categories. Type 1 is characterized by several radiolucent lumens and contrast staining of the artery wall, sometimes associated with an intimal flap. Type 2, the most common variant, is defined by extensive, gradual, and uniform constriction due to an intramural hematoma; within this classification, Type 2A displays normal proximal and distal segments,

whereas Type 2B is marked by diffuse narrowing that extends to the distal vessel. Type 3 is defined by localized or tubular stenosis, typically less than 20 mm, and is sometimes erroneously classified as atherosclerosis, requiring advanced imaging techniques like optical coherence tomography (OCT) or intravascular ultrasonography (IVUS) for precise diagnosis.[13]

Although representative coronary angiographic images were unavailable for inclusion, the angiographic findings aligned with Saw type 2A spontaneous coronary artery dissection. This type is defined by a prolonged, diffuse constriction of the artery, while the distal segment of the vessel remains unaffected. The classification relied on the angiographic characteristics and the observed spontaneous healing in subsequent angiographic evaluations. This report is limited by the absence of stored images.

Medical Treatment is the best course of action for most SCAD patients, except when the LMCA is the culprit artery or when ischemia is still happening[3]. P-SCAD has been associated with elevated failure rates of percutaneous coronary intervention (PCI) owing to the vulnerability of artery walls and the risk of dissection propagation.[10] Consequently, the decision to eschew stenting in favor of medicinal treatment was warranted, particularly considering the angiographic results and the patient's subsequent clinical improvement.

4. Conservative management and self-resolution

The patient's progress shows the typical healing phase of SCAD: the stenosis that was seen in follow-up angiography going away, which usually happens between weeks to months. Studies show that more than 70% of patients who are treated conservatively heal on their own[3]. The healing of lesions in both the LMCA and LAD in this case shows how beneficial it is to avoid needless PCI in stable individuals.

Medical therapy usually includes beta-blockers, antiplatelet drugs, nitrates, and treating heart failure at the same time. Observational studies have shown that beta-blockers are associated with a lower incidence of recurrence[14]. Anticoagulation is usually stopped when SCAD is diagnosed to stop the growth of intramural hematoma. In this case, heparin was stopped correctly because of bleeding and confirmation of the diagnosis. Although statins are not routinely recommended in SCAD in the absence of atherosclerosis, they may be temporarily prescribed during the acute phase of myocardial infarction before the diagnosis is confirmed.[15] In this case Atorvastatin was commenced as part of the acute coronary syndrome protocol before a definitive angiographic diagnosis was established, and it was subsequently maintained for a brief period in the setting of myocardial infarction and left ventricular systolic dysfunction.

5. Heart failure management and postpartum considerations

This patient had an ejection fraction of only 25–35% and major problems with the motility of the front wall. Therapeutic therapies for heart failure, including ACE inhibitors, beta-blockers, aldosterone antagonists, and diuretics, were initiated in accordance with recognized protocols.[16] The amelioration of symptoms and stabilization prior to discharge underscores the potential for substantial myocardial healing; nonetheless, many SCAD patients demonstrate persistent left ventricular dysfunction, requiring extended follow-up. [17]

Managing anticoagulation and figuring out how often it is for someone to bleed are really hard tasks for new mothers. The patient had vaginal bleeding that required an obstetric examination, which led to the decision to stop heparin and showed how important it is to have tailored postpartum care.

6. Follow-up and recurrence

Recurrence of SCAD happens in 10–30% of patients, usually months to years after the first occurrence [18]. This risk underscores the imperative for prolonged monitoring, enhancement of beta-blocker treatment, and patient education on the identification of ischemia symptoms. The discharge plan that says to follow up with a cardiologist once a month and keep an eye on chest pain that comes back is in line with current guidelines. Screening for associated arteriopathies is strongly recommended once the patient is stabilized, using CT angiography from head to pelvis to assess for fibromuscular dysplasia (FMD), renal, carotid, and vertebral artery abnormalities, as well as cerebral aneurysms.[19]

Conclusion

This case shows the importance of considering spontaneous coronary artery dissection in postpartum women who present with acute coronary syndrome, even without common cardiovascular risk factors. The patient had a severe presentation with cardiac arrest and involvement of the left main coronary artery, but she was successfully treated with conservative medical therapy. The improvement of symptoms and spontaneous healing seen in repeat angiography support current recommendations for individualized management of SCAD. Early diagnosis, careful treatment selection, and regular follow-up are very important to achieve good outcomes and to reduce the risk of recurrence in these patients.

Highlights

What Is Already Known?

SCAD is a rare but important cause of acute coronary syndrome, particularly in young women. Pregnancy and the postpartum period increase the risk, and cases may present with severe complications such as myocardial infarction, ventricular dysfunction, or cardiac arrest. Conservative management is preferred in clinically stable patients because spontaneous healing is common.

What Does This Study Add?

This case highlights a severe early postpartum SCAD presenting with cardiac arrest, anterior STEMI, left main and LAD involvement, and severe ventricular dysfunction. Despite the severity, conservative treatment was followed by spontaneous coronary healing and improvement in cardiac function, emphasizing the importance of early recognition and individualized management.

Consent For Publication

all authors agree to the publication of the article

Ethics approval

there was no need for a code of ethic

The extent of AI use

Artificial intelligence tools were used solely for language editing and grammatical improvement of the manuscript. The study design, clinical data collection, interpretation, and scientific conclusions were performed by the authors.

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Conflicts of Interest Disclosures

The authors declare no conflict of interest.

Authors' Contributions

Emad Dadgar and Seyed Hossein Sharoubandi Conceptualized the case report, Emad Dadgar collected patient data, and wrote the manuscript.

Zahra Hajiakhoundian: Assisted in reviewing the literature and interpreting the clinical findings.

Seyed Hossein Sharoubandi: Provided expert advice on the diagnosis and treatment strategy, and reviewed the manuscript

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