

# **Annals of Case Reports & Reviews**

# **Case Report**

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# Successful Treatment of Severe Spontaneous Coronary Artery Dissection with Veno-arterial Extracorporeal Membrane Oxygenation: A Case Report

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## Abstract

**Background:** Severe spontaneous coronary artery dissection (SCAD) concomitant acute cardiogenic shock has not been well studied. Neither the treatment strategy for spontaneous coronary dissection evolving severe ischemia has been established.

**Case Summary:** A 40-year-old woman presented with recurrent chest pain to the emergency department. Her electrocardiogram revealed ST elevation and depression in lateral and inferior leads, respectively. Her transthoracic echocardiography demonstrated a left ventricular ejection fraction of 40% with hypokinetic left ventricular wall motion in the anterior, lateral, and posterior segments. In addition, her emergent coronary angiogram revealed 99% stenosis of the proximal part of the left circumflex branch and 75% stenosis of the first diagonal branch. Intravascular ultrasonography revealed that the left anterior descending artery and left circumflex branch were involved in the dissection, and an entry lesion was identified in the left main trunk. In the absence of any option, a drug-eluting stent was implanted in the left main trunk; however, the fenestration technique for making re-entry by a cutting balloon was not successful. The hemodynamics remained unstable after the percutaneous coronary intervention (PCI); thus, veno-arterial extracorporeal membrane oxygenation was successfully removed. Finally, the patient was discharged without residual symptoms. She was re-admitted because of SCAD recurrence in the right coronary artery at the 5-month follow-up and was successfully managed conservatively. She was event-free in the outpatient clinic after the second admission.

**Discussion:** SCAD is known to predominantly affect middle-aged women. The pathophysiologic hypothesis of SCAD is that the intramural hematoma is caused by spontaneous hemorrhage in the vasa vasorum. Intravascular ultrasonography or optical coherence tomography could help diagnose SCAD and determine its therapeutic strategy. As most of the reported SCAD cases are self-healing, it should be conservatively managed. Patients with progressing ischemia or persistent ischemic symptoms should be managed invasively, such as with PCI or coronary artery bypass graft. The PCI outcomes for SCAD are less predictable and have higher rates of complications and suboptimal outcomes than the atherosclerotic acute myocardial infarction. Coronary artery fenestration by cutting or scoring balloons might be a promising technique, particularly in the case of long and wide hematoma without rupture.

*Keywords:* Spontaneous coronary artery dissection, Percutaneous coronary intervention, Extracorporeal membrane oxygenation, Case report.

#### Introduction

Spontaneous coronary artery dissection (SCAD) disease has diagnostic challenges and it should be treated conservatively <sup>(2)</sup>; however, in the case of evolving ischemia, the treatment strategy becomes debatable. Besides, only a few studies have reported SCAD requiring mechanical cardiopulmonary support. Herein, we presented a successful case of the treatment of SCAD with cardiogenic shock.

#### **Case presentation**

A 40-year-old female patient was worried for her son had to be admitted to the emergency room 4 days before her admission. She had a history of obsessive-compulsive disorder and epilepsy. Her medications included zolpidem

tartrate at 10 mg and lemborexant at 5 mg. Afterward, she presented to the emergency department due to acute chest pain. Electrocardiogram (ECG) (Figure 1A) and transthoracic echocardiogram were expected, but laboratory findings revealed elevated troponin levels (1.52 ng/mL). She was suspected of vasospastic angina. Cardiac computed tomography was performed 3 days before admission, revealing no significant stenosis, but slight regional wall motion abnormality in the anterior free wall. Atypical Takotsubo syndrome was considered for diagnosis. She presented to the emergency department complaining of recurrent chest pain 2 days later. She did not have a family history of genetic disease, cardiac disease, or sudden death. Vital signs on initial evaluation revealed a blood pressure of 102/75 mmHg, a heart rate of 97 beats/min, arterial oxygen saturation of 92% (ambient air), and a body temperature of 36.2°C. Physical examination revealed no abnormal findings. An ECG revealed ST elevation in lateral leads and ST depression in inferior leads with a wide QRS duration of 169

ms (Figure 1B). A transthoracic echocardiography demonstrated a left ventricular ejection fraction of 40%, visually assessed, with hypokinetic left ventricular wall motion in the anterior, lateral, and posterior segments. The differential diagnosis included ST-segment elevation myocardial infarction (including atherosclerotic stenosis or spasm attack) and catecholamine-induced severe cardiomyopathy (including Takotsubo cardiomyopathy) given the initial presentation, ECG, and transthoracic echocardiogram findings. A coronary angiogram (CAG) revealed 99% stenosis of the proximal part of the left circumflex (LCX) branch and 75% stenosis of the first diagonal branch (Figure 2A, 2B). LCX was a flow-limiting lesion and likely to be the culprit lesion of myocardial infarction. Evolving myocardial ischemia was definitive, considering the ECG and CAG findings. First, we tried to inject isosorbide dinitrate into the left coronary artery, but stenosis was not recovered. Therefore, we diagnosed it as SCAD.

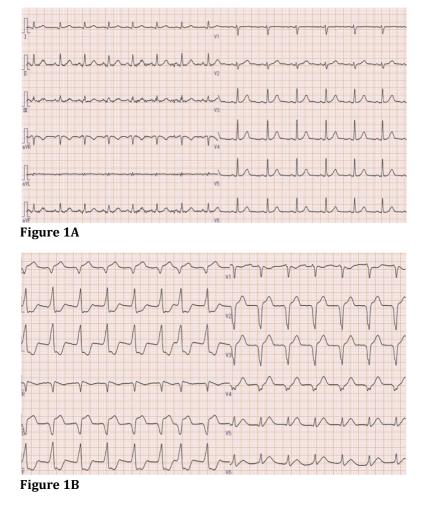


Figure 1: Initial electrocardiogram at the emergency department.

The first electrocardiogram revealed no significant findings (Figure 1A).

The second electrocardiogram demonstrated ST elevation in lateral leads and ST depression in inferior leads (Figure 1B).

Next, we performed percutaneous coronary intervention (PCI). Wiring through the left anterior descending artery

(LAD) and LCX was easy to perform. Intravascular ultrasound (IVUS) findings revealed that both the LAD and the LCX were involved with dissection (Figure 2A') and an entry lesion was recognized in the left main trunk (LMT) (Figure 2B', Figure 2B, yellow arrows).

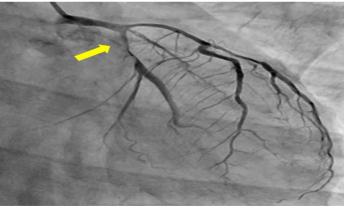


Figure 2A

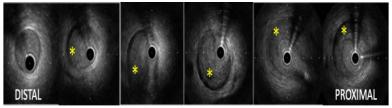


Figure 2A', asterisks

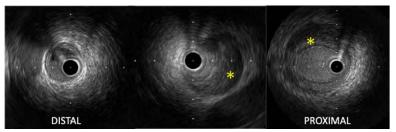


Figure 2B, yellow arrows

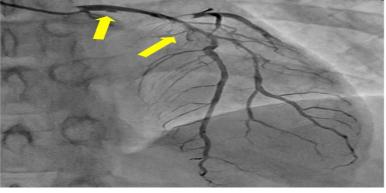


Figure 2B', asterisks

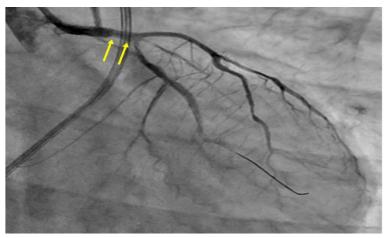


Figure 2C'

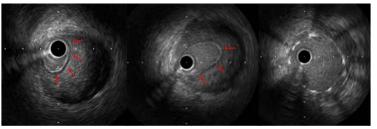


Figure 2C

#### Figure 2: Coronary angiogram and IVUS findings.

- (A) The coronary angiogram revealed significant stenosis (yellow arrows) in the proximal left circumflex branch (Figure 2A). Intravascular ultrasound imaging confirmed spontaneous coronary artery dissection in the left circumflex branch. The true lumen was compressed by intramural hematoma (Figure 2A', asterisks). The hematoma continued to the left main trunk.
- (B) The left anterior descending artery and the left main were involved with dissection in the coronary angiogram (Figure 2B, yellow arrows). Intravascular ultrasound imaging demonstrated a mildly compressed true lumen by intramural hematoma (Figure 2B', asterisks). The right coronary artery was angiographically normal.
- (C) Final result after LMT-stenting and plain old balloon angioplasty by cutting balloon (Figure 2C). Intravascular ultrasound imaging revealed a wellapposed stent but unsuccessful fenestration (no intima cleavage, red arrows, Figure 2C').

We considered a non-stenting strategy, i.e., fenestration by a cutting balloon. However, we had no choice but to implant a drug-eluting stent (Xience skypoint: 4.0/8 mm) due to hemodynamic instability. Then, we inserted intraballoon aortic pumping via the femoral artery. To make re-entry, the

fenestration technique by a cutting balloon (Wolverine: 3.0/10 mm) was not successful (Figure 2C'), but the TIMI grade was got to 3 from 2 (Figure 2C). The hemodynamic state was still unstable after the PCI procedure, thus we inserted veno-arterial extracorporeal membrane oxygenation (VA-ECMO). A massive hemorrhage in the respiratory tract was detected on day 2. VA-ECMO was successfully removed on day 5. Hemorrhage from the tracheal tube persisted after ECMO removal, so dual antiplatelet therapy was discontinued. The patient was extubated on day 14. She was transferred to the general ward from the cardiac care unit on day 20. She was discharged without residual symptoms on day 32.

Final result after LMT-stenting and plain old balloon angioplasty by cutting balloon (**Figure 2C**). Intravascular ultrasound imaging revealed a well-apposed stent but unsuccessful fenestration (no intima cleavage, **red arrows**, **Figure 2C'**).

The patient was admitted due to SCAD recurrence in the right coronary artery at the 5-month follow-up (**Figure 3**). She was successfully managed with a conservative strategy. She was event-free in the outpatient clinic after the second admission.

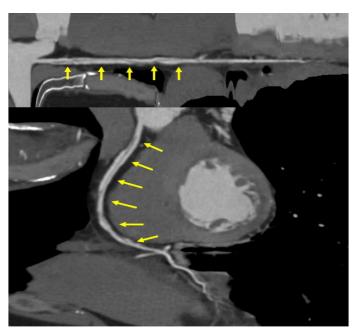


Figure 3: Coronary computed tomography.

The coronary computed tomography revealed a throughout coronary dissection (**yellow arrows**) in the right coronary artery.

#### Discussion

SCAD is a rare but important cause of acute coronary syndrome (ACS), consisting of <1% of all ACS cases (1). Patients with SCAD are predominantly female, mainly middle-aged (2). The pathophysiologic hypothesis of SCAD is that intramural hematoma is caused by spontaneous hemorrhage in the vasa vasorum (3) True lumen compression could cause myocardial infarction. SCAD is classified into three types based on the coronary angiogram (4). Intravascular ultrasound or optical coherence tomography could help diagnose SCAD and determine therapeutic strategy (5). The diagnostic and therapeutic strategy for ACS in the modern era has been established, but those for SCAD remain ambiguous (6) Most SCAD cases are reported to be self-healing (7). Therefore, SCAD should be conservatively managed.<sup>(2)</sup> Patients with progressing ischemia or persistent ischemic symptoms should be invasively managed, i.e., PCI or coronary artery bypass graft (CABG). The outcomes of PCI for SCAD are less predictable, with a higher rate of complication and suboptimal outcomes, compared to atherosclerotic acute myocardial infarction (8). Hematoma propagation frequently requires the use of multiple unplanned stents (9) CABG is less often performed for patients with SCAD with LMT involvement or after failed PCI. However, bypass grafts have poor long-term patency (9). Coronary artery fenestration by cutting or scoring balloon might be a promising technique, especially in case of long and wide hematoma without rupture (10). This technique was unsuccessful, but it is worth a try.

**Result:** We had a case of successful treatment for SCAD with hemodynamic collapse.

# Conclusion

SCAD is a rare cause of ACS in middle-aged females with scarce risk factors for atherosclerotic disease. Rapid diagnosis is often difficult even for experienced cardiologists. The treatment strategy for SCAD with LMT lesions remains controversial. Therefore, the optimal PCI strategy should be debated. Gaining hemodynamic stability is most important in the case of SCAD with hemodynamic instability.

# **Learning points**

Spontaneous coronary artery dissection has a diagnostic pitfall; thus, it must be considered a differential diagnosis for middle-aged women experiencing chest pain.

Hemodynamic stability is crucial in the case of ST-segment elevated myocardial infarction and hemodynamic instability. Therefore, an invasive strategy must be employed in the case of evolving ischemia.

# Consent

A written informed consent was obtained from the patient in line with the COPE guidelines for the submission and publication of this report, including images and associated text. **Patient perspective:** The patient was satisfied with the relief from symptoms following spontaneous coronary artery dissection with cardiogenic shock.

**Consent:** A written informed consent was obtained from the patient, in line with the COPE guidelines, for submission and publication of this case, including images and associated texts.

**Conflict of Interest:** None declared.

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