Clinical Case Report

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# Spontaneous coronary artery dissection in a middle-aged woman with acute anterior myocardial infarction

## A case report

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**Abstract**

**Rationale:** Spontaneous coronary artery dissection is a highly unusual cause of acute coronary disease. It is a result of a hematoma formation within the outer third of the tunica media,with subsequent expansion leading to compression of the true lumen and resultant myocardial ischemia.

**Patient concerns:** We present a case of a middle-aged woman presenting with chest pain with acute anterior myocardial infarction, who did not reveal any of the cardiovascular risk factors. Finally,when pressed further about her past history, the patient revealed she had been taking oral contraceptives for the past 2 years.

**Diagnoses:** The diagnosis is usually confirmed by coronary angiography, but it has some limitations. If necessary, intravascular ultrasound (IVUS) may help in further examinations to reduce the incidence of erroneous diagnosis or missed diagnosis.

**Interventions:** There is no guiding suggestion for the treatment of SCAD. The main treatment methods are 3 aspects: conservative internal medical treatment, stent implantation, coronary artery bypass grafting. The treatment strategy depends mainly on the patienst' clinical symptoms, the hemodynamic state, and the position and quantity of the dissection. In this case,we used stent implantation treatment originally and then we resorted to medical treatment.

**Outcomes:** After careful review and analysis, coronary arteriography results was able to prove the existence of the spiral shaped dissection. We then resorted to medical treatment and her symptoms were gradually relieved.

**Lessons:** SCAD should be suspected in young to middle-aged women with chest pain symptoms, particularly during the perinatal period or for subjects who use oral contraceptives. Doctors should be able to obtain a detailed past history and analyze coronary angiography results carefully.

**Abbreviations:** ECG = electrocardiogram, IVUS = intravascular ultrasound, LAD = left anterior descending artery, RCA = right coronary artery, SCAD = spontaneous coronary artery dissection, TIMI = thrombolysis in myocardial Infarction.

**Keywords:** acute myocardial infarction, middle-aged woman, spontaneous coronary artery dissection

### 1. Introduction

Spontaneous coronary artery dissection (SCAD) is known to be a rare but often fatal cause of acute coronary syndrome (ACS).[1] It has been observed in patients with coronary disease, and women in their perinatal period or with the use of oral contraceptives who were easily misdiagnosed.[2] The severity may range from unstable angina to acute myocardial infarction even cardiogenic shock.[3] The diagnosis is usually confirmed by coronary angiography, but it has some limitations.[4] If necessary, intravascular ultrasound (IVUS) may help in further examinations to reduce the incidence of erroneous diagnosis or missed diagnosis.[5] We present this case of a middle-aged woman with acute anterior myocardial infarction.

### 2. Case report

This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Dongzhimen Hospital Affiliated to Beijing University of Chinese Medicine. Written informed consent was obtained from the patient.

A 49-year-old woman was admitted to the Emergency Department. The patient had 5 hours of chest pain following intense physical activity. There was no past history of coronary heart disease, hypertension, diabetes mellitus, or hyperlipidemia. She also denied smoking and drinking, and had a negative family history. Her blood pressure and heart rate on admission were 131/72 mmHg and 59 beats per minute (bpm), respectively.

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*Ethic statement: complied with ethical standards.*

*All authors have no conflicts of interest to disclose.*

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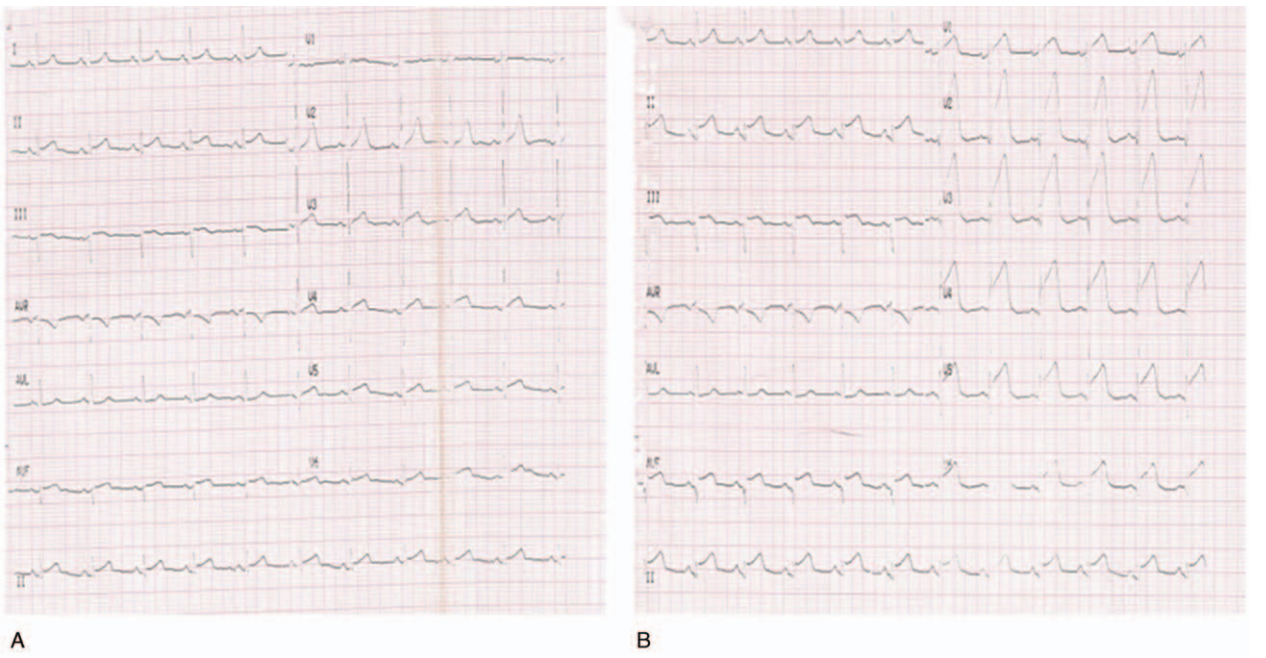

**Figure 1.** A. Admission electrocardiogram revealed arched ST-segment elevation of 0.2 mv in leads II, III, aVF and 0.05 to 0.2 mv in leads V5 and V6. B. Electrocardiogram during the patient's recurrence of chest pain showed arched ST-segment elevation of 0.3 mv in leads II, III, aVF; ST-segment showed arched elevation of 0.05 to 0.2 mv and T-wave appeared sharp and high in leads V1 to V6.

Electrocardiogram (ECG) results revealed an elevated arched ST-segment in leads II, III, aVF, and V5 to V6 (Fig. 1A). Elevated cardiac biomarkers were as follows: cardiac troponin I level was 0.684 ng/mL (normal range, 0–0.03 ng/mL), myoglobin was 143.1 ng/mL (normal range, 0–100 ng/mL), and creatine kinase-MB was 18.8 ng/mL (normal range, 0.6–6.3 ng/mL). Emergency coronary angiography revealed irregular vascular wall and plaque from the ostium to the first diagonal proximal part of the left anterior descending artery (LAD), which also demonstrated a 50% localized stenosis of the LAD at its ostium with a thrombolysis in myocardial infarction (TIMI) grade 3 flow. There was no obvious stenosis of the right coronary artery with TIMI

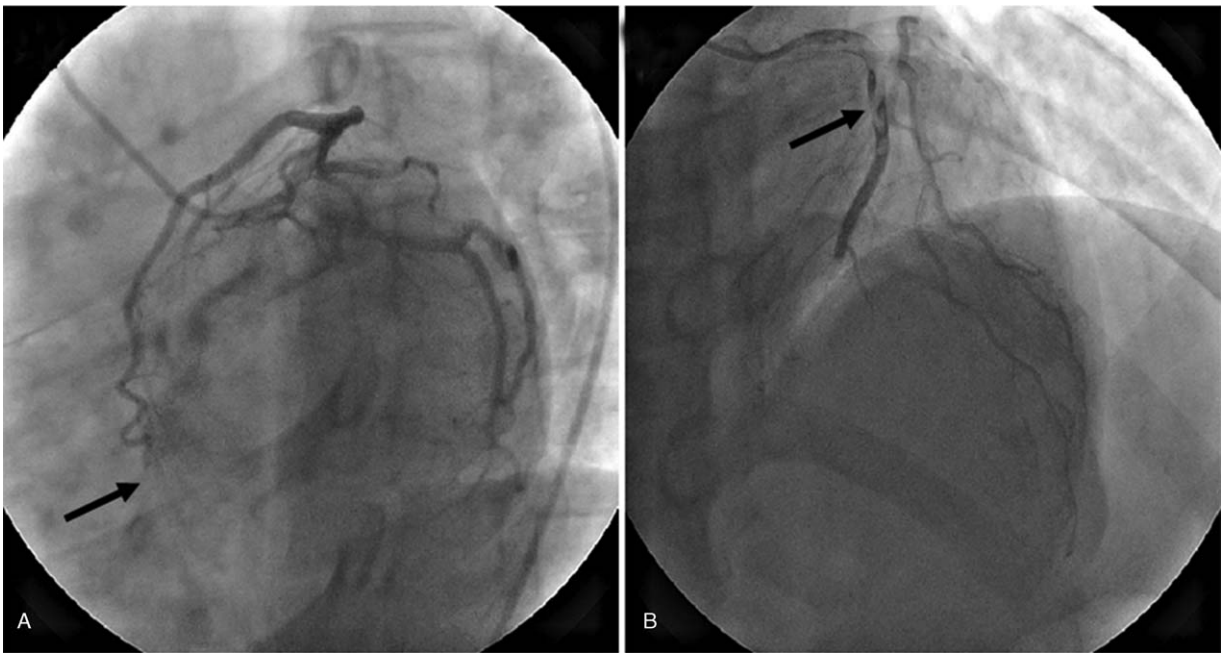

**Figure 2.** A. Emergency coronary angiography revealed irregular vascular wall and plaque from the ostium to the first diagonal proximal part of the LAD, which also demonstrated a 50% localized stenosis of the LAD at its ostium with TIMI Grade 3 flow (as the arrow can be seen the dissection which we diagnosed). B. Second coronary angiography showed total occlusion of the middle LAD with TIMI Grade 0 flow (as the arrow can be seen the spiral shaped dissection of the proximal and middle LAD). LAD = left anterior descending artery, TIMI = thrombolysis in myocardial infarction.

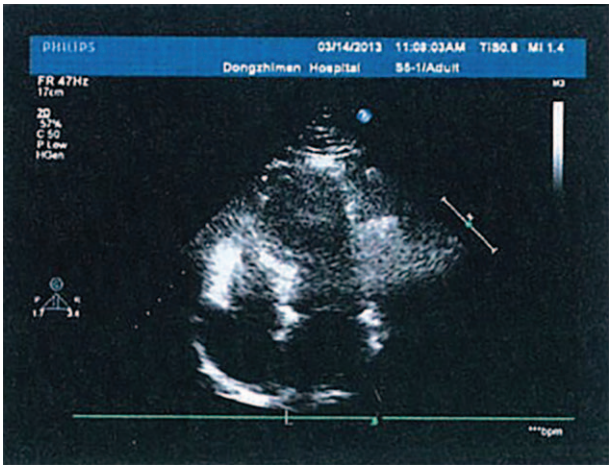


**Figure 3.** Echocardiogram revealed left ventricular hypertrophy, segmental wall motion abnormalities (interventricular septum, lateral wall, inferior and posterior wall motion disappearance; anterior wall motion abatement), a left ventricular aneurysm was present (occupied 30% left ventricular area) with left ventricular ejection fraction 45%.

Grade 3 flow in the distal part (Fig. 2A). The chest pain symptom was slightly relieved after angiography, and the patient was treated with a loading dose of dual antiplatelet, anticoagulant, dilatation of the coronary artery, lipid-lowering, and plaque stabilization therapies. Three hours later, persistent chest pain recurred accompanied by nausea, vomiting, and sweating. Her blood pressure and heart rate were 149/72 mmHg and 68 bpm, respectively. ECG revealed an elevated arched ST-segment that greatly increased in leads II, III, and aVF, compared with the previous one. The ST-segment revealed an arched elevation, and T-wave appeared sharp and high in leads V1 to V6 (Fig. 1B). The following cardiac biomarkers were reviewed: cardiac troponin I level of 4.2 ng/mL (normal range, 0.010–0.023 ng/mL), myoglobin of 193 ng/mL (normal range, 23–112 ng/mL), and creatine kinase-MB of 244 ng/mL (normal range, 2.0–7.0 ng/mL). These biomarkers were significantly higher than before. Echocardiogram results revealed regional wall motion abnormalities, left ventricular hypertrophy, and left ventricular aneurysm was present with a left ventricular ejection fraction of 45% (Fig. 3). We took coronary artery occlusion into consideration and repeated coronary angiography, which revealed the total occlusion of the middle LAD with a TIMI Grade 0 flow (Fig. 2B). We attempted to open the culprit vessel, but no-reflow persisted when the guide-wire passed into the occlusion site of the middle LAD or with balloon dilatation. We initially attributed this to thrombosis overload. However, after careful review and analysis, 2 coronary arteriography results was able to prove the existence of the spiral shaped dissection of the proximal and middle LAD. This led to a thrombus aspiration therapy attempt, which was terminated when the guide wire passed into the false lumen. We then resorted to medical treatment with antiplatelet, anticoagulant, coronary artery dilatation, lipid-lowering, and plaque stabilization therapies after the operation. Her symptoms were gradually relieved. When pressed further about her past history, the patient revealed she had been taking oral contraceptives for the past 2 years.

## 3. Discussion

Spontaneous coronary artery dissection (SCAD) is a highly unusual cause of acute coronary disease.[6] It is a result of a hematoma formation within the outer third of the tunica media, with subsequent expansion leading to compression of the true lumen and resultant myocardial ischemia.[7] The most common presentation is acute coronary syndrome, but the severity may range from unstable angina to acute myocardial infarction even cardiogenic shock. This disease is associated with a low clinical detection rate and a series of ranges from 0.1% to 0.28%. Many patients are misdiagnosed or never diagnosed. As a result, SCAD has a high mortality rate if not identified and treated promptly.[8]

SCAD has been observed in patients with coronary disease, and in women especially in their perinatal period or with the use of oral contraceptives.[9] The incidence of SCAD in women is high (75%). It is related to the elevation of estrogen and progesterone.[10,11] The coronary artery walls are weakened through hormonal changes, which contributes to degeneration and dissection. LAD involvement is more common in women and right coronary artery (RCA) involvement is more common in men.[12] Our patient has the following features: a middle-aged woman who has been taking contraceptives and has irregular menstruation with menopausal indication in the recent 2 years. There was no past history of cardiovascular disease and relevant positive family history. Additionally, the onset of chest pain was sudden with intense physical activity, and coronary angiography revealed lesions of LAD progressing rapidly. Due to lack of experience on the diagnosis and treatment of this disease, the first angiography failed to detect the coronary artery dissection. The subsequent further expansion of the dissection and total occlusion of LAD led to the formation a left ventricular aneurysm.

The diagnosis is usually confirmed by coronary angiography, but it has some limitations including the precise localization of the dissection entry point and the identification of the true and false lumen.[13] This may require intravascular imaging modalities such as IVUS. IVUS can display the vascular cross-sectional images clearly and analyze quantitatively, providing a complementary role in the diagnosis of SCAD.

There is no guiding suggestion for the treatment of SCAD.[14] The treatment strategy depends mainly on the patienst' clinical symptoms, the hemodynamic state, and the position and quantity of the dissection.[15] The main treatment methods are 3 aspects. Firstly, conservative internal medical treatment: the commonly used drugs include aspirin, glycoprotein IIb/IIIa receptor antagonist, heparin, beta blocker, and angiotensin transferase inhibitor.[16] After medication, it can heal itself or exists for a long time. Secondly, stent implantation: it is the first and the most important treatment for SCAD. Percutaneous transluminal coronary angioplasty or direct stent implantation can be performed to cover the whole dissection in order to prevent the dissection enlargement and stabilize the vascular cavity.[17] Thirdly, coronary artery bypass grafting is mainly for patients with left main coronary artery dissection or complex lesions, especially those with hemodynamic instability.[18]

SCAD is a rare but serious cause of acute coronary syndrome. It should be suspected in young to middle-aged women presenting with chest pain, even in the absence of cardiovascular risk factors. One should be able to obtain a detailed past history and analyze coronary angiography results carefully. If necessary, IVUS may help in further examinations to reduce the incidence of erroneous diagnosis or missed diagnosis.[19–21]

## Author contributions

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