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Case Report

Inadvertent consequences of percutaneous coronary intervention to treat unstable spontaneous coronary artery dissection

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ABSTRACT

We present two cases of spontaneous coronary artery dissection (SCAD), which were diagnosed and treated with emergent percutaneous coronary intervention (PCI). Patients with ongoing ischemia due to SCAD need emergent coronary revascularization with PCI or coronary artery bypass grafting. We discuss the difficulties of PCI to bail out unstable SCAD regardless of the modern techniques and modalities. Brief reviews of the literature with relevance are included.

<**Learning objective:** We should probably pay more careful attention to perform PCI for unstable SCAD because of the fragileness of the dissected vessels.>

Introduction

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome or sudden cardiac death. There is no specific guideline on how to manage patients with SCAD. If a pronounced dissection persists in a major vessel with ongoing ischemia, percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) should be considered. We report a case series of two unstable SCAD patients with inadvertent clinical events during PCI. The aim of this case series is to heighten the awareness of arterial weakening or fragility. We think that these two cases suggest a mechanism and provide some direction for further investigation.

Case reports

Case 1

A 47-year-old woman with frequent resting angina was transferred to our tertiary center. She suffered a transient ST-segment elevation myocardial infarction (STEMI), characterized by an elevated troponin I and anterior ischemic electrocardiographic (ECG) changes (Fig. 1A). Seven years previously, she was treated with a bare-metal stent in the right coronary artery (RCA) due to SCAD at another hospital. Her current medical treatment consisted of daily aspirin, a calcium channel blocker, a nitrate, a statin, and an angiotensin receptor blocker.

A resting transthoracic echocardiogram demonstrated mild impaired left ventricular function (LVEF 55%) with an inferior region wall motion abnormality. A 6-Fr sheath was introduced into the right femoral artery. Emergent diagnostic coronary angiography revealed 99% compressive diffuse stenosis with thrombolysis in myocardial infarction (TIMI)-2 flow in the left anterior descending artery (LAD) (Fig. 2A and B). The second diagonal branch had 99% stenosis. We cannulated the left main trunk with a 6F JL3.5 guiding catheter. We performed PCI after crossing the LAD lesion with a floppy guidewire. Intravascular ultrasound (IVUS) and optical coherence tomography (OCT) were used to clarify the culprit lesions (Fig. 2C). We could not identify the intimal tear despite OCT guidance. The strategy for this SCAD was to deploy long stents to maintain coronary flow. We used two bare-metal stents at segments 7 to 8 (4.0 × 32 mm, 3.0 × 32 mm). However, a few minutes later, the proximal LAD showed gradual haziness (Fig. 2D). To treat this lesion, we deployed another stent at segment 6 (4.0 × 28 mm). After that, the coronary dissection propagated to the left circumflex artery (LCx) instantly with flow limitation (Fig. 2E). We reinserted the guidewire into the obtuse marginal

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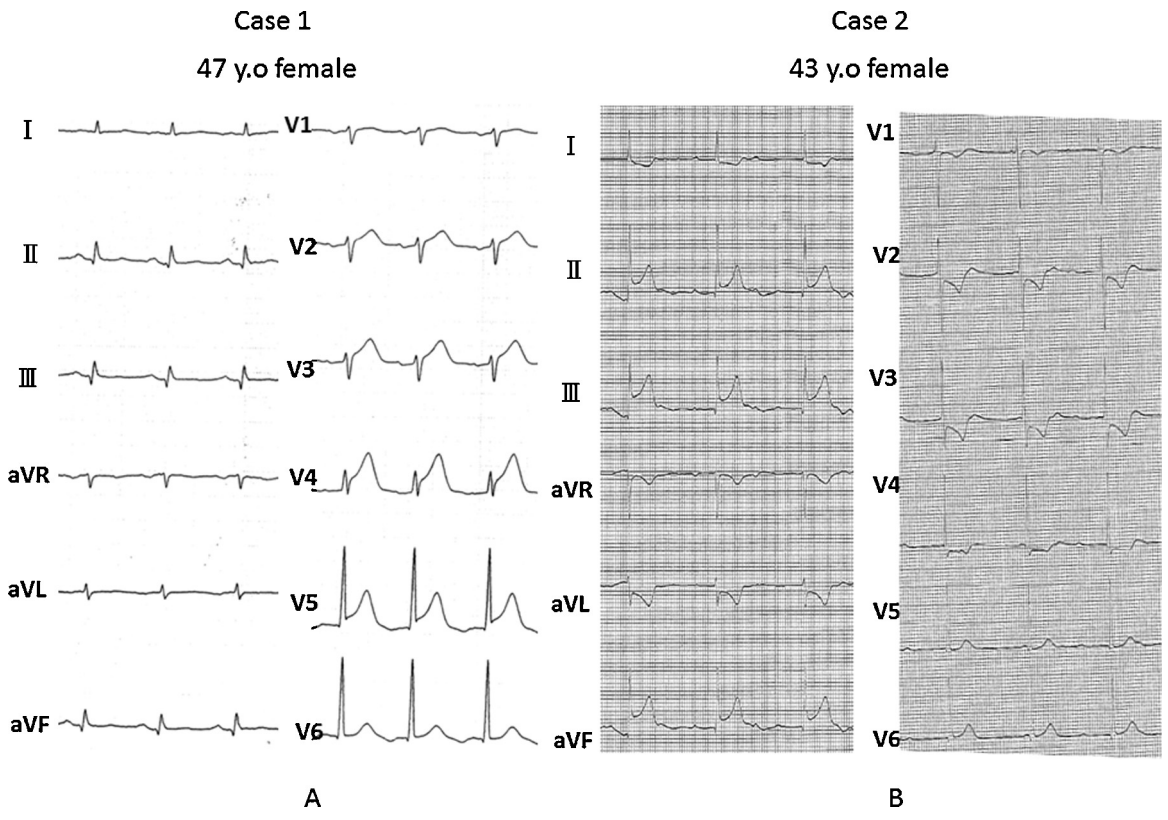

Fig. 1. Electrocardiography (ECG). (A) Case 1: An ECG on arrival to the emergency room showing ST-segment elevation in precordial leads (V2 through V6). (B) Case 2: Standard 12-lead ECG at time of presentation showing ST elevation in leads II, III and aVF.

branches promptly, and performed IVUS (Fig. 2F). After recognition of the true lumen, another stent (3.5 × 12 mm) was deployed at the proximal site of the LCx. TIMI-3 flow at the end of PCI was obtained without hemodynamic compromise (Fig. 2G). The peak serum creatine kinase level was 542 IU/l. The patient's recovery was uneventful and she was discharged on hospital day 14 after undergoing cardiac rehabilitation. She had no symptoms during 8 months of follow-up. Follow-up coronary angiography at 8 months showed good flow in the LAD and LCx with no evidence of restenosis (Fig. 2H).

Case 2

A 43-year-old woman was admitted with persistent central chest oppression. The initial ECG revealed ST-segment elevation in the inferior leads (Fig. 1B). We diagnosed inferior STEMI with complete atrioventricular block. She had no history of a myocardial infarction. Her only coronary risk factor was a history of past smoking. Treatment with aspirin and unfractionated heparin was initiated. A 7-Fr sheath was introduced into the right femoral artery. Emergent coronary angiography with temporary pacing disclosed a 99% complex double-barrel severe stenosis with TIMI-1 flow in the RCA (Fig. 3A). Subsequently, aortography was performed to exclude aortic dissection with coronary involvement. We cannulated the RCA with a 7F JR4.0 guiding catheter and performed PCI in the RCA under IVUS guidance. IVUS confirmed that the second guidewire was in the true lumen. IVUS on the first guidewire showed that it was in the false lumen (Fig. 3B). We placed a stent (3.5 × 20 mm) with 16 atmospheres pressure to seal the entry point of dissection. Moreover, we deployed an additional stent (3.5 × 20 mm) with 14 atmospheres pressure in the distal part of the RCA. The final angiogram showed TIMI-3 flow (Fig. 3C). The patient's peak serum creatine kinase level was 3021 IU/l. The next day, she was diagnosed with cardiac tamponade based on her vital signs and echocardiography. Emergent enhanced CT was performed, but aortic dissection could not be identified. Cardiac tamponade due to hemopericardium was released by pericardiocentesis with no recurrence. About 250 ml of bloody pericardial fluid was drained. Thereafter, she was discharged on hospital day 15 after undergoing cardiac rehabilitation. At 7 months of follow-up, the patient remained symptom free; however, repeat angiography demonstrated the lack of healing of the previously dissected RCA with spiral double barrel (Fig. 3D). A repeat PCI was performed in the RCA with stenting (3.5 × 30 mm, 3.5 × 30 mm, 3.0 × 15 mm). The final angiogram showed better coronary flow with additional coronary stenting (Fig. 3E). After 8 months of follow-up, the angiogram showed no restenosis with good flow (Fig. 3F).

Discussion

SCAD is defined as a spontaneous tear in the coronary arterial wall that is not related to trauma or medical instrumentation. SCAD affects mostly young women without typical coronary risk factors [1]. Moreover, patients with SCAD typically have an underlying arterial disease (connective tissue disorders, spasm, idiopathic) and often have a precipitating stress event (intense exercise, intense emotional stress, drugs, intensive bearing-down activities) [2]. In our two young female cases, we could not identify the underlying disease and precipitating events.

Generally, the dissection is defined as an intimal tear in the vessel wall. Many reports describe luminal narrowing with intimal tear as typical SCAD [3]. However, an intimal tear is seldom observed [4]. Some reports describe cases with only pericardial