Spontaneous Coronary Artery Dissection as Acute ST Elevation Myocardial Infarction Receiving Primary Coronary Intervention

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Spontaneous coronary artery dissection (SCAD) represents a rare cause of acute coronary syndromes. A 38-year-old female without traditional cardiovascular risk factors, admitted for an acute anterior wall ST-elevation myocardial infarction, received primary coronary intervention, in addition to repeated cardiac catheterization for coronary stenting over the middle left anterior descending artery due to the initially obscure culprit lesion during admission. SCAD also occurred in the left circumflex artery. She was finally discharged uneventfully. Six months after discharge, coronary angiography for the angina symptoms showed a new SCAD just below the original stenting site, plus coronary stenting performed. She has remained asymptomatic for more than one year till now.

Key Words: Primary coronary intervention • Spontaneous coronary artery dissection

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute ischemic heart disease with complex pathophysiology.1 It typically affects young healthy women, particularly peripartum or oral contraceptive associated. Its clinical presentation relates to the extent and rate of dissection as well as the degree of myocardial ischemia.2 This young woman with no conventional risk factors for coronary artery disease underwent primary coronary intervention for an acute anterior wall ST elevation myocardial infarction caused by SCAD of the middle left anterior descending (LAD-M) coronary artery, in addition to two repeated cardiac catheterizations for the obscure culprit lesion. She finally had an uneventful clinical course.

CASE REPORT

A 38-year-old woman who suffered from sudden onset severe chest pain with cold sweating around 4 a.m was sent to our emergency department (ER) about 5 a.m. The electrocardiogram evinced ST-elevation in leads V1-V5 accompanying acute anterior wall myocardial infarction. Two cardioversions were exerted for ventricular tachycardia (VT) attack at first the ER and then in the catheterization room when primary coronary intervention was performed. After coronary angiography, the right and left circumflex coronary (LCX) arteries were both patent. The LAD-M tapered its caliber with vessel irregularity (Figure 1A). The occlusion site was supposed in the middle portion; this small vessel might be its branch. But after repeated trials of wiring, no true route of the anterior descending (LAD) coronary artery was observed. So the procedure was stopped, and the heparinization and glycoprotein IIb/IIIa inhibitor Tirofiban infusion were started at the coronary care unit. After repeated catheterization 3 days later, the angiogram assured the dissection lesion over the LAD-M with sluggish distal flow and the new LCX dissection over
the distal part without flow being compromised (Figure 1B). Despite a 3.0 × 20 mm bare metal stent over the LAD-M, the flow could only be improved to TIMI grade 1 to 2 even after adenosine infusion (Figure 1C). Heparinization and nitroglycerin infusion were continued; the third catheterization 3 days later was conducted. The angiogram averred the LAD flow improved to TIMI grade 3, but the SCAD of the LCX still stayed without flow compromised (Figure 1D). The patient was then transferred to the ordinary ward.

She had two healthy children, with no conventional cardiovascular risk factor for coronary artery disease and with an unremarkable past medical history; she denied using any vasoconstricting or recreational drugs; she did not smoke, drink or take oral contraceptives.

The cardiac enzyme was elevated with the peak total creatine kinase (CK) 1762 IU/L, peak CK-MB 155 IU/L and troponin I 35.2 μg/L (normal < 0.05). The electrolytes and full blood examination were normal.

After the uneventful clinical course, the patient was discharged on a β-blocker, aspirin, clopidogrel and an ACE inhibitor.

Before the coronary angiography follow-up 6 months later, mild chest tightness with short duration and relief by rest for 2 to 3 weeks existed. The angiogram showed a new dissection just below the original lesion where the stent was, but the SCAD over the LCX had disappeared (Figure 2A). Afterwards, the positioning of another 2.75 × 24 mm bare metal stent went quite well (Figure 2B).

The patient has remained asymptomatic for more than one year after last admission; the echocardiogram at one year affirmed only mild anteroseptal wall hypokinesis with the acceptable ejection fraction estimated to be about 65%.

**DISCUSSION**

SCAD is a rare condition, predominantly found in
young and otherwise healthy women. Patients may present with acute coronary syndrome, cardiogenic shock, or sudden death. Clearly, the mortality rate depends upon the extent and location of the dissection. SCAD is in light of hematoma formation within the outer third of the tunica media, with subsequent expansion leading to true lumen compression and myocardial ischemia. Combining hemodynamic factors and changes in the integrity of the coronary vessel contributes to SCAD, often if atherosclerotic disease is absent.

The classic angiographic appearance of SCAD is with contrast media in the two lumens separated by a radiolucent intimal flap, with persisting contrast in the false lumen after the remainder of the vessel has been washed out. An intimal tear may or may not be present, and the dissection may be obscured by significant narrowing of the true lumen with vessel irregularity. In this case with the initial angiogram-ascertained only greater narrowing of the true lumen, but no intimal tear was noted. It appeared 3 days later after heparin and glycoprotein IIb/IIIa inhibitor infusion which might limit hematoma formation and subsequent luminal compression. When angiography is ambiguous, intravascular ultrasound (IVUS) or computed tomography (CT) coronary angiography can distinguish dissection from atherosclerosis and determine the dissection morphology.

The treatment options for SCAD include medical therapy and revascularization procedures using percutaneous coronary intervention or coronary artery bypass surgery. Conservative treatment with aspirin, other antiplatelet medications, antithrombin agents, nitrates, and beta blockers may work for asymptomatic, stable patients with limited dissections. Thrombolytic therapy is relatively contraindicated in SCAD due to the potential worsening of the dissection. The data are limited on utilizing newer antiplatelet agents such as glycoprotein IIb/IIIa inhibitors in SCAD. Percutaneous coronary balloon angioplasty alone has been reported, however, intracoronary stenting may be the preferred percutaneous treatment modality for patients with single-vessel SCAD, not involving the left main coronary artery (LMCA), and for those with acute coronary syndromes or recurrent ischemia, thanks to the potential for the stent to obliterate the false lumen. Surgical revascularization is indicated with SCAD with multivessel involvement, LMCA involvement, dissection evolution and/or lumen narrowing, or refractory recurrent ischemia.

SCAD complications comprise dissection extension, new dissections, and recurrent infarctions. This patient developed the new dissection over the LCX in the second angiogram and that over the LAD in the 6-month follow-up angiogram. In general, the SCAD long-term prognosis is considered favorable if the patients survive the acute phase. The prognosis is poorer with LMCA, LAD and multivessel involvement, all of which typically result in extensive myocardial infarction or sudden death.

Figure 2. (A) Coronary angiogram 6 months later shows a new dissection below original stenting site but with good distal flow and the SCAD over LCX now disappeared. (B) A new bare metal stent was deployed with good final result.
In conclusion, the timely and correct diagnosis of SCAD as well as its management greatly challenges clinicians. We believe that an acute coronary event in an otherwise healthy woman without traditional risk factors for coronary heart disease can cause a SCAD. If the initial angiogram is obscure, IVUS or CT angiography may aid the differential diagnosis.

REFERENCES