Percutaneous treatment of spontaneous left main coronary artery dissection extending to the left anterior descending and circumflex arteries possibly triggered by thrombolytic therapy

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Spontaneous coronary artery dissection is a rare cause of myocardial infarction and the role of thrombolytic therapy in this setting is not

Spontaneous coronary artery dissection (SCAD) is a rare cause of myocardial infarction and sudden cardiac death. Approximately 150 cases have been described since 1931 (1,2).

The true incidence appears to be underestimated because the majority of cases are diagnosed at autopsy (1,3). Two-thirds of patients are women mostly presenting during pregnancy or in the peripartum period. Most SCADs in men involve the right coronary artery. In women, SCADs tend to involve the left main coronary (LM) and left anterior descending (LAD) arteries (1).

The etiology of SCAD is not known, but some factors such as middle age, female sex, peri- or postpartum state, oral contraceptive use, presence of atherosclerotic coronary artery disease, intense physical exertion, connective tissue disorders, cocaine abuse and high-impact blunt chest trauma have been identified as major important risk factors (4,5). We describe a case of SCAD in which thrombolytic therapy may have been a significant contributing factor. Optimal treatment of this condition is controversial. In the present case, percutaneous coronary intervention (PCI) was successful in treating this life-threatening condition.

CASE PRESENTATION

A 58-year-old woman without any risk factors for coronary artery disease presented to the hospital with sudden-onset

known. A case of acute ST elevation myocardial infarction is presented, with initial positive response to thrombolytic therapy and subsequent marked worsening of ST elevation due to extensive dissection, possibly triggered by thrombolytic therapy, which was successfully treated with percutaneous coronary intervention.

Key Words: Angioplasty; Coronary intervention; Dissection; Infarction; LAD; LM; MI; PCI; Stent; Thrombolytic

chest pain that lasted for 2 h. Her electrocardiogram (ECG) on admission revealed ST elevation in leads I, II, aVL, aVF, V1 and V6 (Figure 1). The patient was treated with tenecteplase for ST elevation myocardial infarction and anti-ischemic therapy. Initially, her chest pain resolved and a second ECG 1 h after tenecteplase administration showed ST segment resolution without Q waves.

Three hours after the initiation of thrombolytic therapy, her chest pain recurred. A repeat ECG showed extensive ST elevation in leads I, II, V1 and V6 (Figure 2). The patient was urgently taken to the cardiac catheterization laboratory. Her angiogram revealed extensive dissection of the LM artery extending into the LAD and circumflex (LCX) arteries (Figure 3). The right coronary artery was spared. An intravascular ultrasound (IVUS) study confirmed that the LM artery dissection extended into both the LAD and LCX coronary arteries (Figure 4). Based on the limited IVUS data, there was no significant atherosclerotic plaque in the LM, LAD or LCX arteries. The cardiothoracic surgery department did not consider coronary artery bypass grafting due to the extensive dissection involving distal coronary vasculature. Therefore, the decision was made to pursue treatment with PCI.

Initially, it was decided to stent the proximal LM and distal LAD arteries to seal the entry and exit part of the dissection flap. After wiring of the LAD and LCX arteries (Figure 5), the distal LAD artery was initially stented using a 2 mm × 23 mm

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Figure 1) Electrocardiogram on admission

Figure 2) Repeat electrocardiogram 3 h after thrombolytic therapy with recurrent chest pain

Mini Vision stent (Guidant Corporation, USA). The LM artery was then stented using a 3.5 mm × 28 mm Cypher stent (Cordis Corporation, USA) extending into the LAD artery (Figure 6). However, the coronary flow remained significantly compromised, requiring stenting of the entire LAD artery all the way back to the LM artery. Therefore, two Xience V stents (Abbott Laboratories, USA), one 2.75 mm × 28 mm and one 2.5 mm × 28 mm, as well as a 2.5 mm × 18 mm Vision stent (Guidant Corporation), were placed in the LAD artery with high-pressure postballoon dilation using noncompliant balloons inflated to 24 atm. A final angiogram revealed excellent results (Figure 7). Due to the presence of Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow, the LCX artery was not stented. The patient recovered and was discharged one

Figure 3) Angiogram demonstrating a dissection (arrows) involving the left main coronary, left anterior descending and circumflex arteries

Figure 4) Intravascular ultrasound confirming an extensive dissection (arrow) causing lumen narrowing

week later without angina. Her echocardiography revealed only a mildly reduced ejection fraction.

DISCUSSION

SCAD is a rare cause of ischemic heart disease. It occurs predominantly in young women. The LAD artery is involved in 80% of cases (6,7). Approximately 15% of dissections involve the LM artery (8,9).

At autopsy or during bypass surgery, the principle finding of SCAD has been described as displacement or distortion, and usually complete compression of the lumen by a large hematoma. The dissection plane runs in the outer one-third of the media, or between the media and adventitia (10-12).

In the present case, dissection extended from the LM artery into both the LAD and LCX arteries, which is extremely rare (9). Thrombolytic therapy could restore flow by thrombolysis of the false lumen thrombus, allowing the true lumen to expand, thus restoring coronary blood flow (7,13). However, in our case, thrombolysis may have triggered extensive dissection.

Figure 5) Intracoronary wire can be seen (arrow) in the true lumen of the left anterior descending artery

Figure 6) Stent positioning in the left main coronary artery before deployment (arrow)

If the diagnosis of spontaneous dissection is made during coronary angiography, thrombolysis is not recommended because it may propagate the dissection and expand

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Figure 7) Final angiographic result

the intramural hematoma (14,15). In our case, thrombolytic therapy may have extended the initial spontaneous dissection based on the second ECG, which showed extensive myocardial infarction. This rare occurrence has been reported previously (14,16). In the setting of acute myocardial infarction, PCI is generally preferred over thrombolytic therapy (15). The present case is unique because we have IVUS images documenting the extensive dissection of the coronary arteries with successful percutaneous treatment.

The prognosis of patients with SCAD is not known. It is believed that asymptomatic patients with a nonocclusive coronary vessel dissection and good distal flow can be managed conservatively. Symptomatic patients with a single coronary vessel dissection should be treated by primary stenting of the entry site. Patients with LM artery or multivessel dissection involving a large territory with an adequate distal target should undergo bypass surgery if feasible. However, finding the true lumen during bypass surgery could be technically challenging, and extensive dissection involving distal vessels may preclude surgery due to a poor distal target, as was the case in our patient (16,17).

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