Successful Management of Intractable Coronary Spasm With a Coronary Stent

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Although the long-term survival of patients suffering from coronary spasm is usually excellent, serious complications can develop, such as disabling pain, myocardial infarction, ventricular tachyarrhythmias, atrioventricular block and sudden cardiac death. A 40-year-old man who had intractable chest pain from coronary artery spasm suffered ventricular fibrillation and an acute anterior myocardial infarction upon first admission. The patient underwent a coronary angiogram, which revealed a spontaneous focal spasm at the proximal left anterior descending coronary artery (LAD). He was treated by the combination of nitrate and calcium channel blocker, but continued to complain of severe chest pain despite intensive medical therapy and he had to be treated in the emergency room 5 times during an 8-month follow-up period. An ergonovine coronary angiogram was performed and an intracoronary ultrasound examination, which revealed a focal spasm at the same site of the proximal LAD with a small amount of localized eccentric atheromatous plaque. A coronary artery stent was placed in the proximal LAD and his symptoms resolved. A follow-up coronary angiogram was performed 3 years after stenting and the stent remained patent without any in-stent restenosis or spasm. (Jpn Circ J 2000; 64: 897–900)

Key Words: Coronary spasm; Stent

Variant angina (VA) was described by Prinzmetal et al. and is characterized by transient spastic narrowing of the coronary artery, angina at rest and an ST elevation on ECG. On diagnostic coronary angiograms, various observations have been documented, from normal coronary arteries to multivessel lesions, but the coronary spasm usually occurs in an artery with atheromatous plaque. The main treatment for VA is nitrate and calcium channel blocker, but a lack of response to conventional anti-anginal drugs may indicate that coronary intervention or bypass surgery is required, especially if associated with underlying fixed lesion.

We report a patient who developed acute myocardial infarction and ventricular fibrillation as a result of coronary artery spasm and who did not respond to intensive medical therapy, yet was successfully managed with a coronary stent.

Case Report

A 40-year-old male was admitted to the emergency room complaining of cold sweating, dyspnea and chest pains at 04.00h. His past and family history were not specific; he was a smoker with a history of smoking 10 packs per year. Physical examination at the emergency room revealed his blood pressure to be 130/100 mmHg, body temperature 36.8°C, pulse rate 64 beats/min, respiratory rate 20/min. He appeared acutely ill, but his heart and lung sounds were normal. Marked ST segment elevation was noted in leads V1–V4 (Fig 1A), he complained of aggravating chest pain and ventricular fibrillation developed, which was converted into sinus rhythm by DC version (Fig 1B, C). Double bolus injections of 1.5 million units of urokinase with intravenous heparin and nitrate were given and his chest pain decreased after this emergency care. Cardiac enzyme examination revealed 2,225 U/L creatinine kinase (CK), 66 μg/ml CK-MB and 736 U/L lactate dehydrogenase. Two-dimensional echocardiogram demonstrated anterior and lateral hypokinesia, and reversible perfusion defects in the anterolateral wall were shown on dipyridamole stress single photon emission computed tomography.

On a diagnostic coronary angiogram taken 3 days after admission, a spontaneous spasm was documented in the proximal left anterior descending artery (LAD) and an ejection fraction of 51% with anteropapillary hypokinesia was calculated from the left ventriculogram. Calcium channel blocker (15 mg of amlodipine) and long-acting nitrate (360 mg of isosorbide binitrate) were administered. Additional calcium channel blocker (540 mg of diltiazem), 15 mg of nicorandil, α-blocker (4 mg of doxazosin) and prostacyclin (200 μg of ejelatin) with a nitrate patch were given to the patient during outpatient clinic treatment because of his frequent complaints of disabling angina. However, he had to be treated in the emergency room 5 times during the following 8 months in spite of his intensive medical treatment. On his sixth visit he complained of severe chest pain lasting more than 20 min, so a coronary angiogram was performed the next day. A 95% coronary spasm was induced by intracoronary ergonovine (30 μg) at the same site of the proximal

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LAD where the spontaneous spasm had been observed 8 months previously, with ST segment elevation in leads V1-4 (Fig 2). An intracoronary ultrasound examination revealed focal eccentric atheromatous plaque within the proximal LAD (Fig 3A). A GFX stent (3.0x24 mm, Advanced Vascular Engineering) was placed in the proximal LAD covering the entire lesion site with the small plaque. The minimal luminal diameter was increased from 2.8 mm to 3.0 mm after stenting and the stenosis diameter decreased by 10% (Fig 3B).

Nitrate and calcium channel blocker were administered after discharge, plus aspirin continuously and ticlopidine for 1 month. His 3-year clinical follow-up period was uneventful, although he did fracture his femur playing a soccer game. A follow-up coronary angiogram was performed before orthopedic surgery and it showed that the stented LAD was patent without any in-stent narrowing and no spasm was induced by the ergonovine test (Fig 4A, B).
Discussion

Variant angina can be induced in the proximity of atherosomatous plaque and in the artery because of endothelial dysfunction. The exact mechanism of coronary spasm is still unknown; an underlying lesion is frequently associated with coronary spasm and a spasm can be provoked by pharmacologic drugs. A spasm may be fatal in the case of ventricular tachyarrhythmia, and the sudden cardiac death rate is 2-16% in patients with VA. In these patients, the collateral circulation is usually not well-developed in comparison with patients suffering from chronic ischemic heart diseases. Thus acute ischemia in patients with VA can easily provoke serious arrhythmia.

In the present, unusual case, the patient’s clinical symptoms were not responsive to conventional medical therapy, the regional wall motion of the left ventricle was impaired by anterior myocardial infarction, and ventricular arrhythmia was associated with spastic events. Thus the decision was made to place a coronary stent at the site of the atherosomatous plaque as treatment for the recurrent intractable spasms. A previous report had shown that spasm could be treated by coronary intervention even in the case of mild fixed underlying spostesis. The incidence of acute complications after coronary intervention in patients with VA are not higher than in patients with other coronary artery diseases, but the rate of clinical and angiographic recurrence is higher in VA than in patients with fixed spasm. Repeated coronary spasm may play a role in the aggravation of coronary atherosclerosis. Severe coronary artery spasm can be managed by coronary artery bypass surgery, but the spasm can relapse even after such surgery. Therefore, coronary artery bypass surgery may not be beneficial in the treatment of VA. Recently, Kultursay et al reported that when a PAMAZ-Schatz stent was placed in the site of intractable spasm, the stent was patent at a 3-month follow-up coronary angiogram without any cardiac symptoms, and a stress scan revealed no perfusion defects. Lopez et al reported that when a Gianturco-Roubin Stent was inserted for the treatment of intractable spasm unresponsive to medical therapy, a 60% angiographic diameter stenosis was observed on a 3-month follow-up coronary angiogram, but the patient did not exhibit any cardiac symptoms and the myocardial perfusion scan was normal. Vadgernett et al reported 3 cases of coronary stenting for refractory multivesSEL spasm without significant organic coronary atherosclerosis. Those cases were complicated with myocardial infarction and ventricular arrhythmia, but after stenting all cases became asymptomatic with adjunctive calcium antagonist and nitrate. Gaspardone et al reported coronary artery stent placement in VA refractory to aggressive medical therapy and suggested that stents might be an attractive therapeutic option for the focal vasospastic lesion. Gupta et al and Kuppens et al reported stent implantation for refractory VA with beneficial clinical results. However, Muroya et al reported a case in which a stent is believed to have triggered contrast media-induced coronary spasm.

In the patient reported here, a GFX stent was placed at the site of the intractable spasm in the LAD, and the stented artery was patent without any in-stent restenosis or spasm on a follow-up coronary angiogram 3 years after stenting.

Conclusions

A coronary stent may be considered as one of the therapeutic modalities for the treatment of intractable focal spasm, especially in patients with severe cardiac symptoms, acute myocardial infarction, symptomatic ventricular arrhythmia or underlying focal atheromatous plaque. This may be the first report of successful long-term management of intractable coronary artery spasm by stenting of focal atheromatous plaque.

References


