Stent Deformity Caused by Coronary Artery Spasm

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Previous studies have shown that coronary stents have radial strength above the pressure induced by coronary artery spasm. This case report describes a stent deformity caused by coronary artery spasm during percutaneous coronary intervention. (Circ J 2006; 70: 800–801)

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Despite full medical treatment with calcium channel blockers and nitrates, some patients with coronary artery spasm continue to have recurrent episodes of angina and myocardial infarction, and arrhythmic sudden death might occur.1–6 Previous reports demonstrate the usefulness of coronary stents to prevent vasospasm refractory to medical therapy.7,8 However, coronary stent implantation is sometimes ineffective in patients with multivessel spasm.9 This case report describes another limitation of coronary stent implantation to prevent coronary artery spasm.

Case Report

A 53-year-old man had been well until he had severe chest pain caused by acute myocardial infarction. He was referred 2 weeks after the onset of acute myocardial infarction. Coronary angiography revealed a 90% stenosis in the proximal obtuse marginal artery (Fig 1A). There was no significant stenosis in the left anterior descending coronary artery and right coronary artery. A 0.014-inch Skipper guidewire (Asahi Intecc, Nagoya, Japan) was placed across the lesion into the distal obtuse marginal artery. Predilation was performed by using a 3.0-mm OMNIPASS balloon catheter (Cordis, Miami, FL, USA) inflated to 6 atm. Intravascular ultrasound (IVUS) imaging was performed in the obtuse marginal artery using a 30-MHz 3.2F Ultracross catheter (Boston Scientific, Natick, MA, USA). The IVUS image showed a significant stenosis with fibrofatty plaque. A 25 mm NIR stent premounted on a 3.0-mm balloon catheter (Medinol, Tel Aviv, Israel) was deployed in the proximal obtuse marginal artery using an inflation pressure of 14 atm. Angiography and IVUS showed a good result (Figs 1B, 2A). After the guidewire was withdrawn, the patient complained of chest pain; electrocardiogram demonstrated ST-segment elevation in lead I and aV1, and the systolic blood pressure dropped from 140 to 80 mmHg. Angiography demonstrated coronary artery spasm at the proximal stented segment and distal reference (Fig 1C).

Thereafter, stent deformity was observed (Fig 2B). Intravenous norepinephrine (0.2 mg) and intracoronary nitroglycerine (200 μg) were administered. The systolic blood pressure increased to 100 mm Hg and the coronary artery spasm was relieved (Fig 1D). Further coronary intervention was not performed because there was no flow disturbance (TIMI 3) in the obtuse marginal artery. The patient received oral aspirin (100 mg daily), ticlopidine (100 mg twice daily for 4 weeks), diltiazem (100 mg daily), and isosorbide mononitrate (20 mg twice daily). There was no in-hospital event. During follow-up, no adverse event was observed. Six months later, follow-up angiography was performed. The deformed stent (Fig 2C) and a 25% stenosis at the distal stented segment (Fig 1E) were observed.

Discussion

Calcium antagonists and nitrates are effective in preventing coronary artery spasm in most cases. However, coronary artery spasm refractory to the treatment with these drugs is observed in some cases.1–6 Alpha-1 blocking agents2 magnesium3, benzhexol hydrochloride4, denopamine5 and nicorandil6 have been reported as alternatives. Previous reports demonstrated the efficacy of coronary stenting in patients with clinically severe coronary artery spasm refractory to aggressive pharmacologic management.7–9 Gaspardone et al evaluated the usefulness of coronary stent placement in 9 patients with vasospastic angina refractory to medical treatment.10 The NIR stent was used in 6 patients. During follow-up, 3 patients developed recurrent episodes of angina at rest. Holter monitoring demonstrated ST-segment elevation associated with angina. Repeat coronary angiography showed coronary artery spasm after the administration of methylergometrine in these patients. Coronary artery spasm occurred proximally to the previously implanted stent in 2 patients and in other coronary arteries in 1 patient.

Coronary artery spasm is sometimes observed during percutaneous coronary intervention. It is usually relieved by the intracoronary administration of nitroglycerin. Balloon inflation at a low pressure may be used to treat it. Agrawal et al calculated the minimum acceptable collapse pressure for stents using arterial strain caused by experimentally induced artery spasm.11 They reported 0.4 atm as the minimum acceptable limit for collapse pressure. Almost all coronary stents have more radial strength12,13 The NIR stent is one of the stents with strong radial strength12 An in vitro study reported that the NIR stent expanded to 3 mm.
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Collapsed at a compressive strength of 1.05 atm. This case report demonstrates unusually severe coronary artery spasm because the NIR stent was deformed. Coronary stenting may be ineffective in some patients with severe coronary artery spasm as well as in those with multivessel spasm. These are the limitations of stent implantation for coronary artery spasm. Thus, alternative medical treatment such as β1 blocking agents, magnesium, benzhexol hydrochloride, denopamine and nicorandil should be tried for coronary artery spasm refractory to calcium antagonists and nitrates before stent implantation is considered. Stent implantation would be the last resort. In some patients, stent implantation for refractory coronary artery spasm might be performed. However, intensive medical treatment should be continued in those patients even after stent implantation.

References

Fig 1. Coronary angiography showing a 90% stenosis in the proximal obtuse marginal artery (A). After deployment of a NIR stent, angiography demonstrates a good result (B). Arrowheads indicate the edges of the stent. Angiography demonstrates coronary artery spasm at the proximal stented segment and distal reference (C). After intracoronary administration of nitroglycerine, coronary artery spasm is relieved (D). Follow-up angiography shows a 25% stenosis at the distal stented segment (E).

Fig 2. Fluoroscopy demonstrates a fully expanded stent (A). After coronary artery spasm, stent deformity is observed (B). Fluoroscopy shows the deformed stent at follow up (C). Arrowheads indicate the edges of the stent.