Drug-Eluting Stent Used to Treat a Case of Recurrent Right Coronary Artery In-Stent Restenoses often Accompanied by Acute Inferior Wall Myocardial Infarction

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ABSTRACT

We successfully treated a case of in-stent restenosis, which presented on 6 occasions, resulting in frequent acute inferior wall myocardial infarctions, using a drug-eluting stent. This case demonstrates that drug-eluting stents offer the promise of an effective treatment for frequent in-stent restenosis, and importantly, that the underexpansion of a drug-eluting stent heightens the risk of acute or subacute stent thrombosis. (Korean Circulation J 2005;35:480–483)

KEY WORDS : Coronary restenosis : Myocardial infarction : Thrombosis.

Introduction

Although dramatic reductions in restenosis have been attributed to the use of drug-eluting stents (DES), it remains unknown as to whether DES are effective for treating in-stent restenosis (ISR). The effectiveness of DES as a therapeutic tool for ISR might be evident when the results of the SECURE (Sirolimus-Eluting BX Velocity Balloon-Expandable Stent in a Compassionate Use Registry) study are fully known. The described case highlights the use of DES as a promising treatment for recurrent ISR, and the importance of optimal stent expansion, even in the DES era.

Case

A 67-year-old man first presented in June 1999 with unstable angina. The patient was a smoker, with hyperlipidemia, but without a history of diabetes or hypertension. Coronary angiography (CAG) showed a discrete 95% stenosis of the mid right coronary artery (RCA) (Fig. 1A). After predilating the mid RCA lesion using a balloon catheter (3.0 × 20 mm, 12 atm), a 3.0 × 16 mm Nir stent was implanted using a non-compliant balloon catheter (3.0 × 10 mm, 11 atm) at the mid RCA (Fig. 1B). Nine months later, the patient developed an inferior acute myocardial infarction (AMI). CAG demonstrated near total occlusion of the Nir stent and newly developed 80% discrete stenosis at the proximal RCA. Significant stenoses were noted at the proximal and distal edges of the Nir stent after opening of the total occlusion (Fig. 1C). A 3.0 × 17 mm MAC stent was electively implanted at the proximal RCA using a non-compliant balloon catheter (3.0 × 10 mm, 12 atm) after successful ballooning of the Nir stent (Fig. 1D). A follow-up CAG, 11 months later, showed a 50% diameter of stenosis at the proximal edge of the Nir stent of the mid RCA (Fig. 1E). Because the patient was asymptomatic, intervention was not performed. However, less than 1 month after the follow-up CAG, an inferior AMI occurred. CAG revealed total occlusion at the proximal edge, with 70% stenosis at the distal edge of the Nir stent (Fig. 1F). A 3.0 × 17 mm MAC stent was implanted at the mid RCA, distal to the Nir stent, including the distal edge of the Nir stent after opening of the total occlusion (Fig. 1G). Eight months later, an inferior AMI developed. CAG showed 95% stenosis at the proximal edge, 90% stenosis at the distal overlapped site of the Nir stent and 50% stenosis at distal edge of the distal MAC stent (Fig. 1H). Intervention was performed, using a cutting balloon catheter (3.25 × 10 mm, 6 atm) at the stenotic sites, resulting in a patent RCA, but with some irregula-
Fig. 1. Right coronary angiography (CAG) showing repeated in-stent stenoses, frequently accompanied by acute myocardial infarction over a 5-year period. A: a discrete 95% stenosis of the mid right coronary artery (RCA), B: after percutaneous coronary intervention (PCI) (June, 1999), C: stenosis at the proximal and distal edges of the Nir stent, D: after PCI (2000-3-13), E: 50% stenosis at the proximal edge of the Nir stent of the mid RCA (2001-116), F: total occlusion at the proximal edge and 70% stenosis at the distal edge of the Nir stent, G: after PCI (2001-2-12), H: 95% stenosis at the proximal edge and 90% stenosis at the distal overlapping site of the Nir stent and 50% stenosis at the distal edge of the distal MAC stent, I: PCI (2001-10-15), J: 60% stenoses at the proximal edge and distal overlapping site of the Nir stent and 40% stenosis at the distal edge of the distal MAC stent, K: after PCI (2003-12-30), L: total occlusion of the proximal RCA, M: after PCI (2004-1-6), N: Diagram of three bare metal stents and a Taxus stent within the bare metal stents in the RCA. RCA: right coronary artery, PCI: percutaneous coronary intervention, CAG: coronary artery angiography.
with a thrombolytic agent. He was discharged without riddles (Fig. 1I). Two years and seven months later, the patient experienced an inferior AMI, which was treated with a CAG. Seven months later, the patient complained of chest pain, and CAG demonstrated total occlusion of the proximal MAC stent with grade II collaterals from the first RV wall branch, 60% stenoses at the proximal edge and the distal overlapping site of the Nir stent, and 40% stenosis at the distal edge of the distal MAC stent (Fig. 1J). Intervention was performed at the site, using a 3.0 × 32 mm Taxus under 14 atm, within three bare metal stents from the proximal RCA to the distal RCA (Fig. 1K, 1N). Seven days later, an inferior AMI developed. CAG showed total occlusion of the proximal RCA (Fig. 1L). The intravascular ultrasound (IVUS) findings revealed underexpansion of the stents: a stent area of 4.9 mm² and a minimal luminal diameter (MLD) of 2.3 mm at the proximal MAC stent; a stent area of 3.3 mm² and MLD of 2.0 mm² at the proximal edge of the Nir stent, and a stent area of 5.3 mm² and an MLD of 2.5 mm at the distal edge of the distal MAC stent. Intervention was successfully performed using a non-compliant balloon catheter (3.0 × 30 mm, 23 atm) at the proximal edge of the Nir stent (Fig. 1M); the final minimal stent area was 5.1 mm². The patient was prescribed low molecular heparin (LMH) for 4 weeks, at home. Antiplatelets, including aspirin, clopidogrel and cilostazol, were also prescribed. The patient declined a follow-up CAG because he remained asymptomatic throughout the 15 month follow-up period. A thallium scan showed normal findings, without perfusion defects. We concluded that his more than 5-year old RCA problem may have been resolved.

Discussion

If we could have used DES under IVUS guidance from the onset in 1999, this patient would probably not have experienced a 5-year history of RCA. However, DES was not available in Korea until early 2003. If the bare metal stent (BMS) had initially been more fully available in 1999, and with the guidance of IVUS, could the ISR have been avoided? Possibly yes. In this patient, the bare metal stents appeared to be underestimated at the proximal edge of the stent at the mid RCA, and this may have contributed to the ISR. The final stent area has been reported to be a predictor of the outcome, but with inconsistent results. Therefore, is cutting balloon angioplasty better than conventional balloon angioplasty for ISR treatment? Although cutting balloon angioplasty is associated with some procedural advantages and a lower incidence of balloon slippage, it does not reduce recurrent ISR or major adverse cardiac events compared to conventional balloon angioplasty.

This case highlights DES as a promising tool for the treatment of recurrent ISR, and the importance of optimal stent expansion. Whether DES are effective at treating ISR is uncertain. Recently; however, the In-Stent Restenosis Registry produced favorable data at the 1 year follow-up, namely, one case of restenosis among 25 patients, without stent thrombosis (Brazilian data), and conversely, 2 deaths, 1 late thrombosis, 1 vessel occlusion and 2 in-lesion restenoses among 16 patients with more complex lesions, including a previous brachytherapy (Dutch data). These results imply that focal, less complex ISR is more responsive to DES than diffuse ISR. Our case showed multiple IRS, but focal lesions. The SECURE study after 6 months also demonstrated good interim results for DES in ISR, i.e., a low target lesion revascularization rate (11.6% in the radiation failure group vs. 5.4% in the non radiation failure group), with infrequent stent thrombosis (presented at TCT 2003).

Optimal stent high pressure expansion, under IVUS guidance, warrants special attention for the treatment of ISR, even in the era of DES. Recent data has shown that stent underexpansion is associated with failure of the Sirolimus-eluting stent implantation for the treatment of ISR: 9 of 11 recurrences occurred in lesions with a mean stent area of <3.0 mm², despite the use of a high inflation pressure (18 ± 4 atm). As demonstrated by our case, even higher pressure may be needed in ISR lesions to achieve an adequate stent area. Then, why did our patient show recurrent AMI? We assume that severe ISR, or residual stenosis, contributed to his developing AMI after the implantation of the BMS or DES. Finally, another key lesson is that adjunctive pharmacotherapy (unfractionated heparin, GPIIb/IIIa inhibitors, and extended combination of antiplatelet therapy) should be optimized to prevent stent thrombosis in cases with complex/multiple stents and a high thrombus burden. We utilized low molecular heparin for 4 weeks, even after successful DES dilation.

REFERENCES


