Jikeikai Med J 2016 ; 63 : 71-6

Case Report

A Case of Recoiling of Everolimus-eluting Stent at the Ostium of the Right Coronary Artery by Out-stent Plaque Progression

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ABSTRACT

In a 76-year-old man with acute heart failure, 75% stenosis of the proximal right coronary artery (RCA) was treated with everolimus-eluting stent stent (3.5 × 16 mm), the proximal end of which was placed 2 mm from the orifice. However, 4 months later, a 90% restenosis had occurred at the proximal end of the stent. The intravascular ultrasonographic examination revealed substantial recoil of the proximal end of the stent at the ostium of the RCA, with out-stent plaque growth but without intimal growth, vessel remodeling, or stent fracture. Thus, the present early in-stent restenosis at the proximal end of the stent occurred owing to the recoil with an out-stent plaque progression at the ostium of the RCA for 4 months despite additional metal connector wires at its proximal end to preclude recoil and deformation. (Jikeikai Med J 2016 ; 63 : 71-6)

Key words : everolimus-eluting stent, stent recoil, ostium, remodeling, out-stent plaque

INTRODUCTION

The ostium of the right coronary artery (RCA), which is the part of the RCA 3 mm or less from its origin from the aorta, is a target lesion ineffectively dealt with by percutaneous coronary intervention (PCI). The rate of target lesion revascularization of the RCA ostium with a sirolimus-eluting stent has been 13.5%[1]. Although the revascularization rate of the RCA ostium with an everolimus-eluting stent (EES) has been even lower (7.6%)[2], it is still 3% higher than the mean revascularization rate with an EES[3]. Thus, the mechanisms that cause target lesion revascularization because of in-stent restenosis (ISR)[4] after EES placement must be studied in cases in which revascularization of the RCA ostium is required. Recently, out-stent plaque progression, which is related to chronic vessel remodeling, was reported to be involved as a mechanism of ISR of drug-eluting stents (DESs) rather than as a mechanism of neointimal hyperplasia[5].

We report a case of early ISR at the proximal end of an EES which occurred with the stent recoil at the ostium of the RCA for 4 months. According to intravascular images, the present case shows that an out-stent plaque[5] without vessel remodeling was a mechanism of early ISR after placement of an EES to an RCA ostium.

CASE REPORT

A 76-year-old man was considered to be at risk for coronary artery disease because he had hypertension, dyslipidemia, and diabetes mellitus and was an ex-smoker. In January 2011, the patient was admitted to the hospital be-

Received for publication, June 4, 2016
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cause of acute myocardial infarction with cardiogenic shock. An emergent coronary angiographic examination revealed that he had 3-vessel disease. Everolimus-eluting stents (EES) (Xience V, Abbott Vascular, Santa Clara, CA, USA) had been inserted from the proximal segment of the left anterior descending coronary artery to the left main coronary artery, and from the proximal to the middle segments of the left circumflex coronary artery. After the stents had been inserted, the left ventricular ejection fraction was approximately 30%, and the patient continued to undergo secondary preventive chemotherapy against in-stent thrombosis.

In October 2014, acute heart failure led to the patient

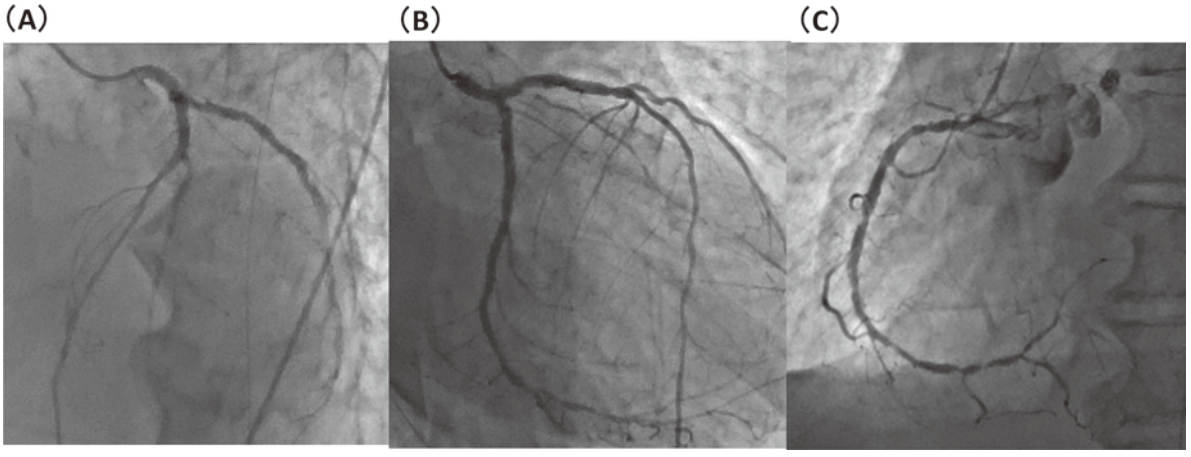

Fig. 1. Coronary angiogram upon initial admission in October 2014.
(A) Left anterior oblique cranial view of the left coronary artery. Two years interval after the last coronary angiogram, there was no in-stent restenosis or de novo stenotic lesions in either the left anterior descending artery or the left circumflex artery. Two Xience V® stents (3.5 × 23 mm and 3.0 × 23 mm) were deployed from the proximal segment of the left anterior descending artery to the ostium of the left main coronary artery, and 2 Xience V stents (3.0 × 18 mm and 2.5 × 18 mm) were deployed from the middle to the proximal segment of the left circumflex artery. (B) Right anterior oblique caudal view of the left coronary artery. Diffuse lesions in the distal left circumflex artery (#14) were similar to those depicted in the angiogram performed 2 years earlier. (C) The left anterior oblique view of the right coronary artery. Several de novo stenotic lesions were evident in the proximal, middle, and distal segments of the right coronary artery.

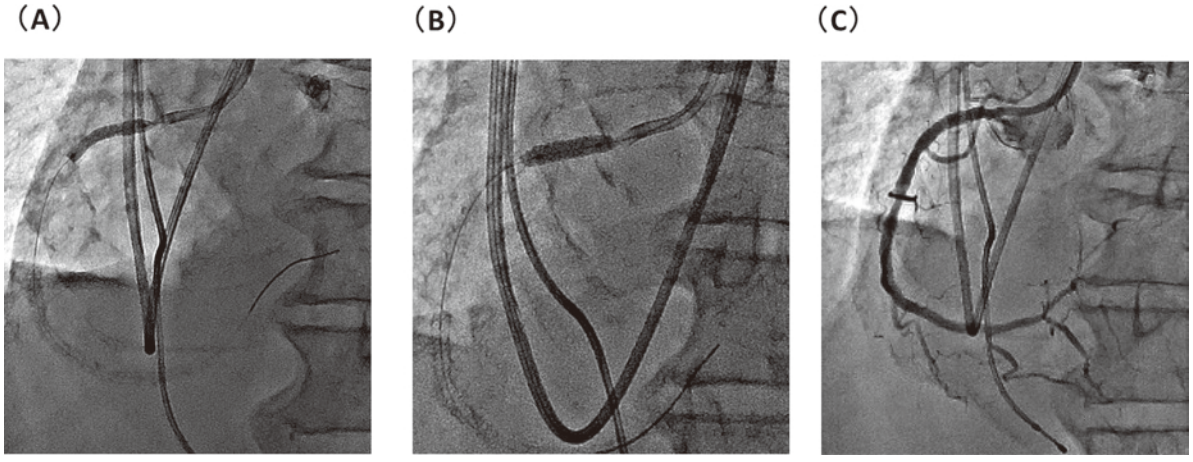

Fig. 2. Percutaneous coronary intervention in the right coronary artery in October 2014.
(A) Left anterior oblique view of the Promus Premier® (3.5 mm × 16 mm) placed in the proximal right coronary artery (RCA). The proximal end of the stent was located 2 mm inside the RCA near its ostium by intravascular ultrasonography image (Figures 4A and B). (B) The Promus Premier® was expanded using a 3.5-mm noncompliant balloon inflated to 22 atm. (C) Final left anterior oblique view of the RCA after 3 Promus Premier® stents were deployed at the proximal, middle, and distal segments of the RCA. Due to worsening hemodynamics, a Swan-Ganz catheter and a temporary pacemaker were inserted.

being admitted to the hospital because of cardiogenic shock. Emergent coronary angiography revealed no ISR in the left main coronary artery, left anterior descending coronary artery, or left circumflex coronary artery (Fig. 1A and B). However, noted were 75% stenosis of the proximal segment of the RCA and 90% stenosis of the RCA from the middle to the distal segment (Fig. 1C). The PCI of the RCA was performed on day 3 of hospitalization. Three EESs of a different type (Promus Premier®, Boston Scientific Corp., Marlborough, MA, USA) were inserted at 16 atmospheres (atm) in the RCA's distal segment (2.25 × 32 mm), middle segment (3.0 × 24 mm), and proximal segment (3.5 × 16 mm) (Fig. 2A). Intravascular ultrasonography (IVUS) (Fig. 3A and B) revealed mild calcification in the proximal segment of the RCA, and postdilatation ballooning was performed with a 3.5-mm balloon at 22 atm (Fig. 2B). According to final IVUS, a Promus Premier® stent was inserted into the RCA with its proximal end 2 mm from the ostium (Fig. 3A and B).

In February 2015, approximately 4 months after the PCI, the patient was admitted to the hospital because of cardiogenic shock, ventricular fibrillation, and pulseless electrical activity. The findings of IVUS in the left coronary artery were unchanged from those of October 2014. However, severe restenosis was observed in the RCA ostium (quantitative coronary angiography in the left anterior

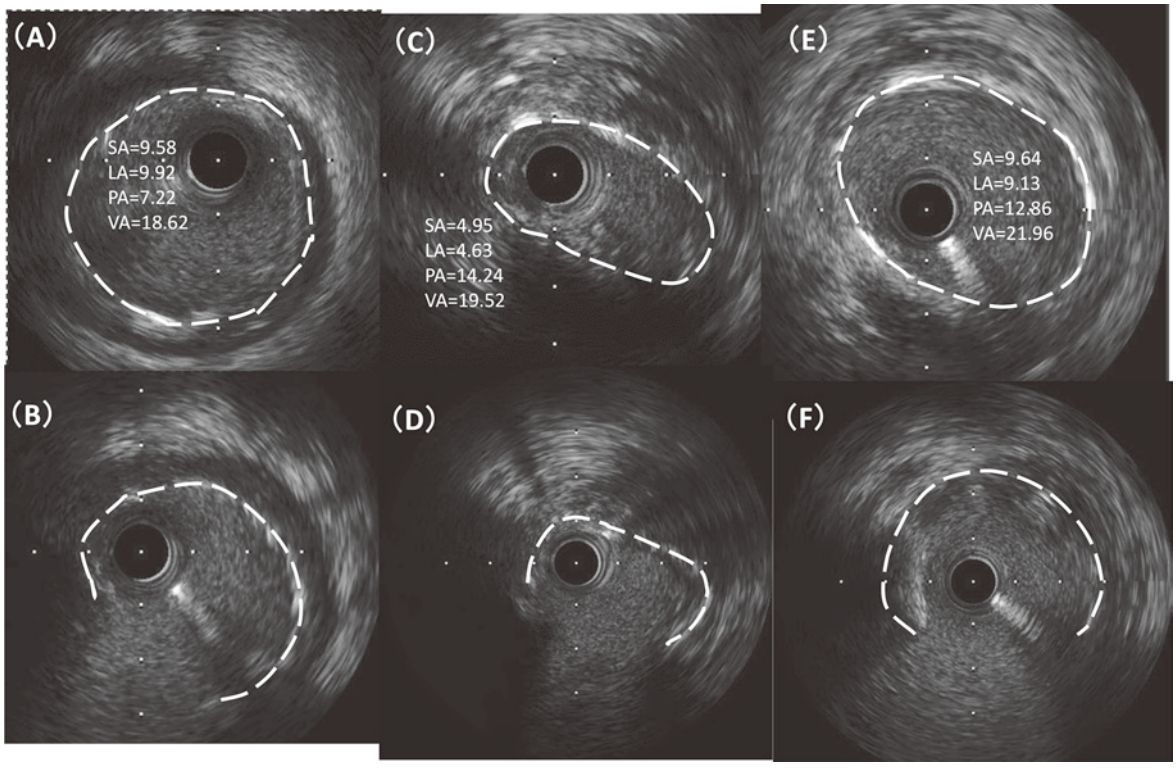

Fig. 3. Intravascular images at 3 times
Intravascular images from the Atlantis SR Pro (Boston Scientific Corp., pull back speed ; 10 mm/s, 40 MHz) at 3 times. (A) Final image of the proximal end of the Promus Premier® (2 mm from the ostium of the right coronary artery [RCA]) in October 2014. The final stent area (SA), the final lumen area (LA), the final plaque area (PA), and the final vessel area (VA) were described in the Figure. White dashed thin lines showed the stent struts. (B) Image of the orifice of the RCA after placement of the Promus Premier®. Struts of the Promus Premier® were evident along approximately half of the circumference of the vessel of RCA ostium. (C) Image of the Promus Premier® depicting the site in (A) after dilatation with a 2.0-mm balloon during emergency percutaneous coronary intervention in February 2015. The Promus Premier® was elliptically compressed by approximately twice as much plaque although the RCA had the same VA, suggesting that out-stent plaque caused mechanical recoil of the Promus Premier® in the 4 months while it was deployed. (D) Image of the orifice of the RCA after dilatation with a 2.0-mm balloon. Like in (C), out-stent plaque caused a decrease in the SA. (E) Final image of the area depicted by an angiogram in Figure 3 (D) after dilatation with a 4.5-mm balloon. The recoiled Promus Premier® shown in (C) clearly expanded as the VA increased. (F) Final image of the orifice of the RCA. The Promus Premier® is shown as in (E).

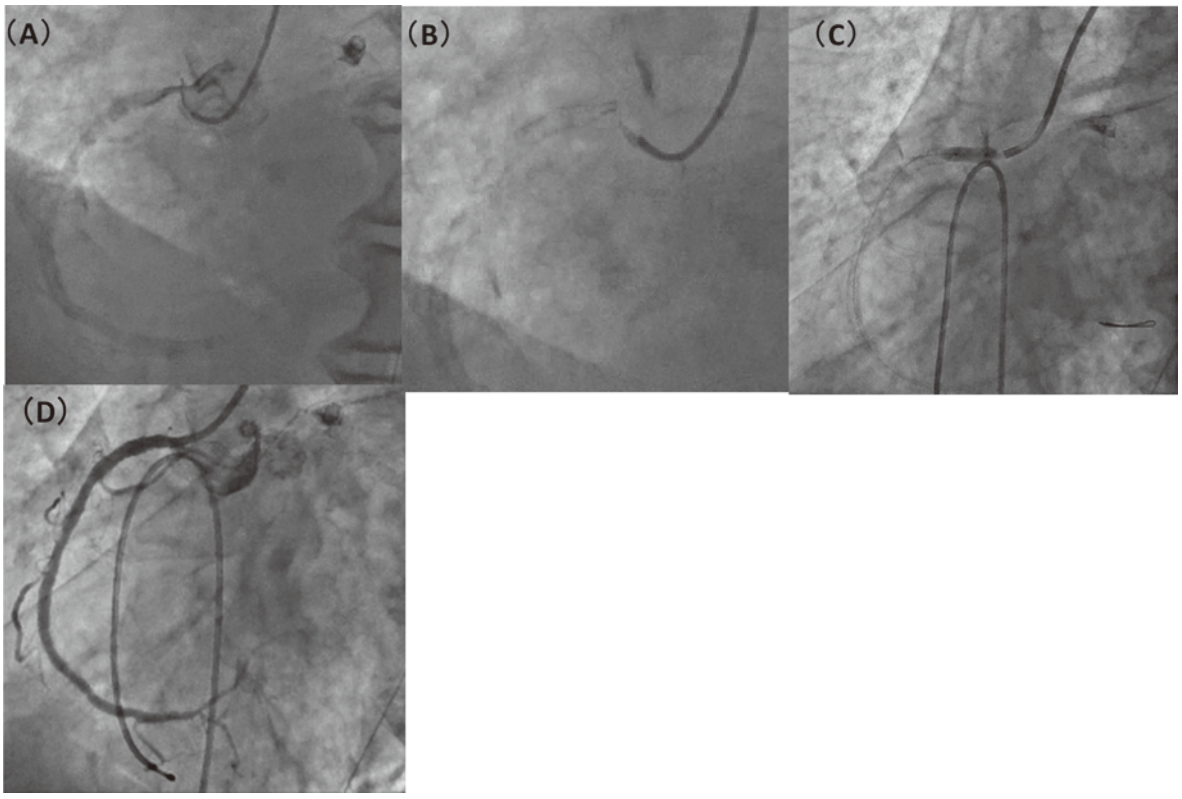

Fig. 4. Emergency percutaneous coronary intervention in February 2015
(A) An angiogram of the cusp of ascending aorta revealed in-stent stenosis of the proximal end of Promus Premier® at the ostium of the right coronary artery. (B) The left anterior oblique view of a transparent image of the Promus Premier® at the ostium of the right coronary artery. The compressed proximal end of the Promus Premier® was clearly evident. According to quantitative coronary angiography, the percent diameter stenosis was 62.76. (C) The Promus Premier® was expanded using a 4.5 × 10-mm non-compliant balloon inflated to 16 atm. (D) Final left anterior oblique view after balloon inflation.

oblique view : diameter stenosis, 62.8% ; minimal lumen diameter, 1.20 mm ; reference diameter, 3.23 mm ; and lesion length, 7.12 mm) (Fig. 4A). Initially, predilatation ballooning was performed with a 2 × 15-mm balloon. Angiography (Fig. 4A and B) and IVUS (Fig. 3C and D) indicated that the proximal end of the stent had flattened without stent fracture. Analysis of IVUS measurements (echoPlaque 4 software, INDEC Systems, Mountain View, CA, USA) indicated that the stent area (SA) had decreased 51.0% and that the plaque area (PA) had increased 97.2% ; the intima growth inside the stent (SA-lumen area [LA]) was 0.13 mm^2 at the proximal end of the stent (Table 1). These findings indicated that the stent had recoiled with the out-stent plaque progression[5]. Postdilatation ballooning was performed with a 4.5 × 10-mm balloon inflated to 16 atm (Fig. 4C). Stent expansion was satisfactory, and the procedure concluded (Fig. 4D, and 3E and 3F, and Table 1).

During stent placement, the vessel remodeling index up to the distal end of the stent was approximately 5% (Table 1). The lesion in the proximal segment of the RCA that was originally distal to the ostium might not have been what had caused positive remodeling of the vessel (Fig. 4), which had an usually large proportion of unstable plaque. Four months after stent placement, both the PA and the plaque burden had increased, but the remodeling index had not changed ; therefore, neither negative nor positive remodeling had occurred.

DISCUSSION

A lesion at the ostium of the RCA remains a complex lesion even if a second-generation DES is used[2,3]. After a DES is placed, ISR can be caused by 3 types of factor : (1) technical factors, (2) mechanical factors, and (3) biological

Table 1. Serial intravascular parameters

	October 2014, Final				February 2015, after balloon inflation and before stenting			February 2015, Final		
	In-stent RCA#1 ostium	In-stent RCA#1	In-stent RCA#1 Distal	Distal reference RCA#1	In-stent RCA#1 ostium	In-stent RCA#1	In-stent (Distal)	In-stent RCA#1 ostium	In-stent RCA#1	In-stent (Distal)
Lesion length (mm)		16.79				14.09			13.19	
Vessel area (mm^2)	18.62	19.80	19.65	19.88	19.52	20.10	18.60	21.96	23.88	19.85
Lumen area (mm^2)	11.39	9.67	10.60	11.13	5.29	9.56	9.62	9.13	10.00	8.64
Minimal lumen area (mm^2)	9.92	8.76	8.76	9.18	4.63			9.13	9.19	8.37
Stent area (mm^2)	11.07	9.70	11.10		5.42	10.18	10.65	9.64	12.10	10.01
Minimal stent area (mm^2)	9.85	8.98	8.98		4.95			9.64	10.14	9.70
Plaque area (mm^2)	7.22	10.20	9.05	8.75	14.24	10.54	8.98	12.83	13.88	11.21
Plaque burden (plaque area/vessel area)	0.39	0.51	0.46	0.44	0.73	0.52	0.48	0.58	0.58	0.57
Vessel volume (mm^2)	346.20				299.20			312.80		
Lumen volume (mm^2)	175.00				119.50			147.20		
Plaque volume (mm^2)	171.20				179.70			165.60		
Stent volume (mm^2)	178.20				134.40			165.00		
Stent area-lumen area (mm^2)	−0.32	0.03	0.50		0.13			0.51	2.10	1.37
Remodeling index (%)		6.3	5.5	6.8	4.8			17.9		

Serial intravascular variables were measured with echoPlaque 4 software (INDEC Systems, Mountain View, CA, USA). Variables were measured 3 times : (1) upon final assessment after stenting in October 2014 (October 2014, Final ; left-most columns), (2) upon first assessment after inflation of a 2-mm sized balloon prior to stenting in February 2015 (February 2015, after balloon inflation ; middle columns), and (3) upon final assessment after inflation of a 4.5-mm sized balloon in February 2015 (February 2015, Final ; right-most columns). Variables and abbreviations are described in the table. "Stent area-lumen area" represents the extent of intimal growth inside the stent. The remodeling index (%) was calculated as 100*(Vessel area.vessel area at #1os in October 2014 (18.62))/18.62. Abbreviations : RCA (Right Coronary Artery)

factors[4]. No biological factor was present, for the most part, in the present case, because there was little intimal growth 4 months after stent placement. In addition, the Xience V stents previously deployed in the left coronary artery did not undergo restenosis (Fig. 1A and B). The mean late lumen loss (LLL) has been reported to be 0.84 mm when a sirolimus-eluting stent is used in the RCA ostium[1]. According to a subanalysis of angiographic results from the Randomized Evaluation of Sirolimus-eluting versus Everolimus-eluting stent Trial[6], LLL remained stable for as long as 1 year after a sirolimus-eluting stent or an EES was inserted. Thus, use of even an EES would presumably result in a greater mean LLL than was noted in a Japan postmarketing surveillance study (mean LLL : 0.22 mm)[3]. Thus, biological factors were not of concern in the present case.

A metal EES recoils approximately 5% during deployment[7]. In the present case, final IVUS findings indicated that stent expansion was satisfactory (Fig. 3A). Postdilatation ballooning was performed with a 3.5-mm balloon inflated to 22 atm, providing close to the maximum SA, and the plaque burden at the RCA ostium was 0.39 (Table 1, Fig. 3A and B), and concluded PCI. The Promus Premier® stent, a second-generation DES, provides a substantial scaffolding effect and applies sufficient radial force to vessel walls. However, an increase in the out-stent plaque[5] volume caused recoil without substantial changes of the vessel area (VA). In addition, plaque growth was noted after 4 months in the portion of the RCA orifice that was not covered by the stent (Fig. 3B and D). The RCA ostium is likely to have facilitated the buildup of plaque replete with characteristic elastic fibers[8,9]. Thus, the PA 1 mm from the end of the stent (just the orifice of the RCA) may have caused restenosis at the end of the stent[10]. Furthermore, a technical factor relates the geometry where the proximal end of a stent might have played a role during PCI. One technique to prevent or lessen recoil involves deployment of a stent so that it protrudes slightly from the RCA into the aorta to completely cover the orifice. The reference vessel diameter is then measured with IVUS, and a balloon larger than 3.5 mm is inflated in the vessel, providing a greater SA. When a 4.5-mm balloon was ultimately inflated in the vessel during subsequent PCI, the VA increased 18% (Fig. 3E). A sub-

stantial acute gain and a greater scaffolding effect of the stent served to reduce flattening and recoil of the stent due to an increase in the PA (out-stent plaque growth). A study with integrated backscatter IVUS has found that out-stent plaque growth causes positive remodeling of a vessel[5]. In the present case, however, the aorta-orifice may not be conducive to vessel remodeling. Thus, in the present case, the VA changed little and plaque with elastic fiber elements extending from the coronary ostia (the proximal end)[8,9] resulted in an increase in the PA, as the chronic stent recoil established at RCA ostium[11], as the mechanical factor of ISR[4]. Thus, the early ISR in the present case was established owing to the recoil of the Promus Premier® stent within 4 months owing to the growth of out-stent plaque extending from the orifice of the RCA (proximal edge) without vessel positive remodeling, neointimal hyperplasia, postprocedural stent under-expansion, and stent fracture.

In summary, extensive growth of out-stent plaque caused early stent recoil within 4 months after Promus Premier® stents were placed at the ostium of the RCA, even if the stent had additional metal connector wires at its proximal ends to preclude recoil and deformation.

Acknowledgement : This study was supported by a Research Fund from Saitama Cardiovascular Respiratory Center under Grants 15EB to T.I.

Authors have no conflict of interest.

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