Case Report
Stent Fracture after Everolimus-Eluting Stent Implantation

Ali S. Almasood, Xavier Freixa, Sohail Q. Khan, Peter H. Seidelin, and Vladimír Džavík

Interventional Cardiology Program, Division of Cardiology, Peter Munk Cardiac Centre, University Health Network, University of Toronto, 6–246 EN, Toronto General Hospital, 200 Elizabeth Street, Toronto, ON, Canada M5G 2C4

Correspondence should be addressed to Vladimír Džavík, vlad.dzavik@uhn.on.ca

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Compared with bare-metal stents, drug-eluting stents (DES) have greatly reduced the risk of in-stent restenosis (ISR) by inhibiting neointimal growth. Nevertheless, DES are still prone to device failure, which may lead to cardiac events. Recently, stent fracture (SF) has emerged as a potential mechanism of DES failure that is associated with ISR. Stent fracture is strongly related to stent type, and prior reports suggest that deployment of sirolimus eluting stents (SES) may be associated with a higher risk of SF compared to other DES. Everolimus eluting stents (EES) represent a new generation of DES with promising results. The occurrence of SF with EES has not been well established. The present paper describes two cases of EES fracture associated with ISR.

1. Introduction

The occurrence of stent fractures (SF) is recognized as a potential complication of stent deployment. Its incidence varies markedly in published reports, ranging from less than 1% to more than 16% [1–3]. Little is known about the precise incidence of SF in the "real world". However, SF is likely to be underrecognized due to difficulty in diagnosis and the lack of standardized definitions. Stent fracture has currently become an important concern after drug-eluting stent (DES) implantation due to its potential association with in-stent restenosis (ISR) and stent thrombosis [4]. Sirolimus-eluting stents (SES), as compared with paclitaxel (PES) or zotarolimus-eluting stents (ZES), are considered the DES with the highest risk of SF [5, 6].

The everolimus eluting stent (EES; Multilink Vision platform, Abbott Vascular, Temecula, CA), is a new DES with promising long-term results [7–9]. As compared with other DES, EES provides the thinnest available strut profile (0.08 mm) [10]. The incidence of SF with EES has never been assessed. The present paper describes two cases of EES fracture associated with ISR.

2. Case Reports

2.1. Case-1. This patient was a 72-year-old lady with a chief complaint of prolonged chest pain. Her medical history included hypertension, hyperlipidemia, family history of coronary artery disease, and cutaneous lupus erythematosus. In May 2009, she experienced exertional chest pain and underwent coronary angiography which revealed a calcific 90% mid- and 80% distal RCA stenosis. The distal lesion was treated with rotational atherectomy using 1.25 mm burr following which a 2.25 × 23 mm Xience V stent (Abbott Vascular Devices, Santa Clara, CA) was deployed at 16 atmospheres (ATM). A 2.75 × 28 mm Xience V stent was then deployed at 16 ATM in the mid-segment, and postdilated with a 3.25 × 10 mm noncompliant balloon up to 24 ATM. An excellent result was achieved, and she was discharged home in a stable condition (Figure 1). Four months later she presented to the emergency department with a prolonged episode of chest pain associated with T wave inversion in the inferior leads and an elevated troponin I. Coronary angiography revealed focal in-stent restenosis (ISR) in the mid-RCA (Figure 2(a)). Multiple fluoroscopic
images revealed a gap in the middle of the EES stent indicating a complete stent fracture (Figure 2(b)) probably related to the severe calcification and angulation of the artery. We elected to cover this with a further stent. Predilation was performed with a 2.5 × 15 mm balloon at 18 ATM and a 3.0 × 13 mm Cypher Select Plus stent was delivered at 24 ATM, with an excellent final result. No further angiograms were performed as the patient remained free of symptoms.

2.2. Case-2. This patient was a 67-year-old lady with recurrent chest pain associated with dyspnea. Her medical history included former smoking, hypertension, hyperlipidemia, diabetes mellitus, and chronic obstructive pulmonary disease. She underwent quadruple bypass surgery in 2004 with a left internal thoracic artery (LITA) to the LAD and 3 venous grafts, to the RCA, diagonal, and obtuse marginal arteries. She required a dual-chamber pacemaker 2 days after bypass surgery. In July 2005, she developed stable class III angina, and coronary angiography revealed a stenosis where the LITA was inserted into the LAD. This was treated with a bare metal stent (BMS). She was stable until June 2009 when she developed recurrent angina and was found to have ISR within the BMS stent. On this occasion, we elected to perform PCI and stenting of the native LAD across the distal anastomosis of the LITA graft. A total of three overlapping everolimus eluting stents (EESs) were deployed. A 2.5 × 18 mm and 2.5 × 25 mm Xience V stents and 2.25 × 15 mm Promus stent (Boston Scientific Corp., Natick, MA) were implanted from distal to proximal with good angiographic results (Figure 3). Five months later, she again presented with progressive angina. Repeat coronary angiography demonstrated focal in-stent restenosis in two stents (the 2.5 × 18 mm Xience V and the 2.25 × 15 mm Promus). Further angiographic evaluation revealed stent strut separation in the two stents at the focal ISR site indicating a complete stent fracture (Figure 4). Predilatation of both sites was performed with a 2.5 × 15 mm balloon. We deployed a 2.5 × 14 mm Endeavor stent (Medtronic CardioVascular, Santa Rosa, CA) covering the distal stent fracture position at 18 ATM. Considerable stent movement was visible with each cardiac cycle. We felt that additional stenting and structural support would be required for success. Therefore a second Endeavor 3.0 × 30 mm stent was deployed to cover this segment, extending back through
Figure 3: Angiographic images of the left anterior descending artery after two Xience V and one Promus stent deployment showing the absence of stent strut separation (a) and the presence of a good angiographic result (b).

Figure 4: Focal in-stent restenosis of the mid- and distal left anterior descending artery (a). Double stent fracture with complete strut separation in two different locations (b).

the ISR in the mid-LAD. The mid-portion of the stented segment was then postdilated with a $3.0 \times 15\text{mm}$ non compliant balloon to a maximum of 22 ATM. An excellent angiographic result was obtained (Figure 5). Subsequently, she represented in July 2009 again with troponin-negative progressive angina. Repeat coronary angiography revealed that she had a fracture within her Endeavor $3.0 \times 30\text{mm}$ stent (Figure 6) and had developed occlusive ISR within her LAD. On this occasion, she had repeat PCI to her LITA into LAD with further deployment of an Endeavor $2.5 \times 30\text{mm}$ with a good angiographic result.

3. Discussion

Stent fracture, currently an important concern after DES implantation, has been associated with a higher potential risk of ISR and stent thrombosis [4]. The mechanism of ISR in stent fracture is probably related to lower drug delivery at the fracture site and higher mechanical irritation by the fractured struts causing smooth muscle proliferation and impaired re-endothelialization [11].

Stent fracture has been linked to longer stent length, stent overlap, stent overexpansion, lesion calcification, severe angulation and dynamic flexure [5, 12, 13]. Sirolimus-eluting stent deployment is also considered a risk factor for SF [5, 6]. Nonetheless, it remains unclear if the relatively high incidence of SF is related to its strut structure, component materials, the type of antiproliferative drug, or just because it is underdetected as compared with other stents [6].

The risk of SF in EES has never been assessed, perhaps due to the relatively new introduction of this stent. EES uses cobalt chromium technology and confers a very unique architecture with the thinnest strut profile [10]. It remains unknown if this design and the cobalt chromium composition might modify the risk of SF compared to the stainless steel DES generation (SES and PES). In our case reports, SF was associated with a focal ISR pattern, which is very frequently seen with SES fractures [14]. The fracture sites were all at hinge movement points that occur in areas of curves and twists leading to increased shear forces on the vessel wall and stent. The repetitive cardiac contractions may ultimately lead to metal fatigue and fracture. Both patients
presented with an acute coronary syndrome secondary to ISR without evidence of stent thrombosis.

The treatment of SF is a challenging situation due to the absence of formal recommendations. In our cases, we used a different stent platform and polymer coating to treat the stent fractures. In the first case, we used an SES, and for the second case, a zotarolimus eluting stent. Although SF appears to be more frequent after SES than other DES implantation, in lesions with high angulations, especially those in the right coronary artery, SF can occur in any type of stent. While no clinical trials have showed any advantage in changing the type of DES, this strategy has never been evaluated specifically in patients with SF.

In conclusion, this report illustrates two cases of EES restenosis secondary to stent fracture. Further studies should be performed to clarify whether this is an important issue or a sporadic observation.

References


