Case Report

Stent Fracture and Restenosis of a Paclitaxel-Eluting Stent

Michalis I. Hamilos, Michail I. Papafaklis, Jurgen M. Ligthart, Patrick W. Serruys, Georgios Sianos

Department of Interventional Cardiology, Thoraxcenter, Erasmus Medical Center, Rotterdam, The Netherlands

Key words:
Stent fracture, restenosis.

We describe the case of a patient with restenosis six months after stent implantation, at two points where stent fracture had occurred. Fracture is an unusual and probably underestimated cause of restenosis, which acquires special significance in this era of drug-eluting stents.

Manuscript received:
October 6, 2005;
Accepted:
October 25, 2005.

Address:
Georgios Sianos

Department of Interventional Cardiology Thoraxcenter, Erasmus Medical Center, Dr Molewaterplein 40, 3015 GD, Rotterdam, The Netherlands, e-mail:
g.sianos@erasmusmc.nl

The problem of restenosis is the Achilles' heel of all angioplasty procedures. Before the use of metal stents restenosis occurred in approximately 35-40% of procedures. The advent of stenting reduced this rate to 20-25%. The latest great revolution in the management of the restenosis problem has been the introduction of drug-eluting stents. Their use reduced restenosis rates to single figures, even in high risk cases such as diabetics, long lesions and vessels of small-diameter.

We describe a case with restenosis after the implantation of a paclitaxel-eluting stent, as a result of stent fracture.

Case description

A 66-year-old male, with hypertension and hyperlipidaemia, underwent coronary angiography four months after suffering an inferior myocardial infarction. During the intervening period he had experienced angina on effort (CCS Stage II). The angiogram revealed a 90% stenosis of the circumflex artery and complete occlusion of the right coronary artery (RCA) at its origin, with collateral filling from the left coronary artery. The patient initially underwent angioplasty of the circumflex artery and two overlapping Taxus (Boston Scientific, USA) stents (2.5 × 12 mm at 14 atm distally and 3 × 13 mm at 12 atm proximally) were implanted with a good angiographic result. Fifteen days later the patient underwent angioplasty of the completely occluded RCA. The RCA was engaged using a 7F guiding catheter (Mach1 FR4) and a straight guidewire (RF 0.014", 275 cm) and two balloons (Maverick 1.5 × 15 mm and 2.5 × 20 mm) were used for predilatation. The vessel was then treated with implantation of three overlapping Taxus stents, a 3 × 32 mm distally, a 3 × 20 mm in the middle part and a 3 × 12 mm proximally. All stents were deployed at 20 atm with a very good final result (Figure 1A). Six months later the patient underwent a control coronary angiographic examination, which showed two focal in-stent restenotic lesions (Figure 1B). Further investigation with intracoronary ultrasound (IVUS Atlantis SR, 40 MHz, Boston Scientific, USA) showed significant focal neointimal hyperplasia at the locations of angiographic stenosis (Figure 2). It should be noted that although the stents appeared to have been deployed satisfactorily throughout their length with no signs of intimal hyperplasia, stent struts were not visible at the two restenosis sites, a finding which is compatible with stent fracture in these regions. The stent fracture, as confirmed by IVUS, was located in

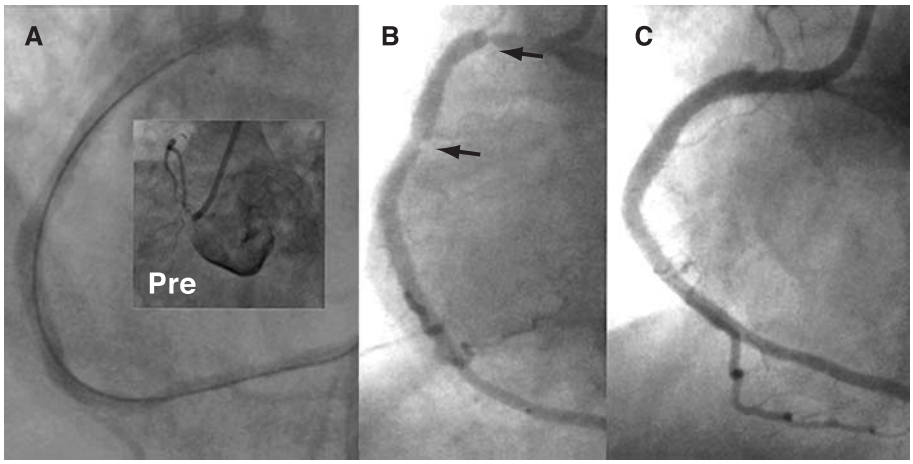

Figure 1. The insert in A shows the totally occluded right coronary artery (Pre). A: Final result after recanalisation and implantation of Taxus stents. B: Control angiographic examination at six months demonstrating two focal restenotic lesions (arrows). C: Result after further implantation of Taxus stents.

the middle stent, adjacent to the regions of overlap between the distal/medial and medial/proximal stents. The patient was finally treated with implantation of two Taxus stents, a 3.5 × 12 mm (20 atm) at the distal and a 3.5 × 8 mm (26 atm) at the proximal restenosis sites with good result (Figure 1C).

Discussion

The use of rapamycin and paclitaxel drug-eluting stents (DES) has dramatically reduced restenosis rates after coronary angioplasty.[1-4]

One very interesting and inadequately studied mechanism of DES restenosis is stent fracture. Fracture has been reported in various cases of implantation of metal stents in the oesophagus, the biliary ducts, the urinary system, peripheral vessels, and in pulmonary arteries.[5-11] The principal mechanism is mechanical fatigue of the metallic stent, either from excessive movement or from the pressure to which it is subjected (e.g. in the case of an extraluminal oesophageal tumour[6]).

Fracture of an intracoronary stent has been reported in a few isolated cases.[12-15] The potential mechanism was mechanical fatigue due to stent placement in regions of twists and curves, where the motion of the vessel during cardiac diastole and systole exerts strong forces at certain locations, which thus become vulnerable to fracture. This is probably more common in longer stents.

It is notable that almost all reports in the literature refer to the RCA, whose course is strongly curved and whose motion during cardiac systole and diastole is thereby more forceful, resulting in greater stent fatigue. In addition, it is likely that overexpansion of the stent, as occurred in our case, could weaken its struts and, in combination with other factors, render it susceptible to fracture.[15] In our patient both fractures occurred around areas of increased rigidity due to metal overlapping, which may have acted as a fulcrum for metal deformation due to vessel movement.

The mechanism through which stent fracture causes focal restenosis is a dual one. The broken struts cause local mechanical stimulation of the vessel wall, resulting in inflammation and mobilisation of the intimal hyperplasia mechanism. In addition, the fracture leads to destruction of the stent architecture locally, so that the drug, which was distributed evenly along its length, is no longer present at a particular location. In consequence, extremely localised restenosis develops in an otherwise patent stent.

The case presented here, as far as we are aware, is the first in the literature to involve fracture of a paclitaxel-eluting stent. Previous reports referred either to conventional or to sirolimus-eluting stents. The incidence of this restenosis mechanism has not been adequately studied and reports tend to be sporadic. Conventional stents undoubtedly also sustain fractures, but these go unnoticed because of the diffuse nature of the restenosis in such cases.

To conclude, given the ever-increasing use of DES and the single-figure restenosis rates that are associated with them, fracture may represent an underestimated mechanism of restenosis. Further studies with

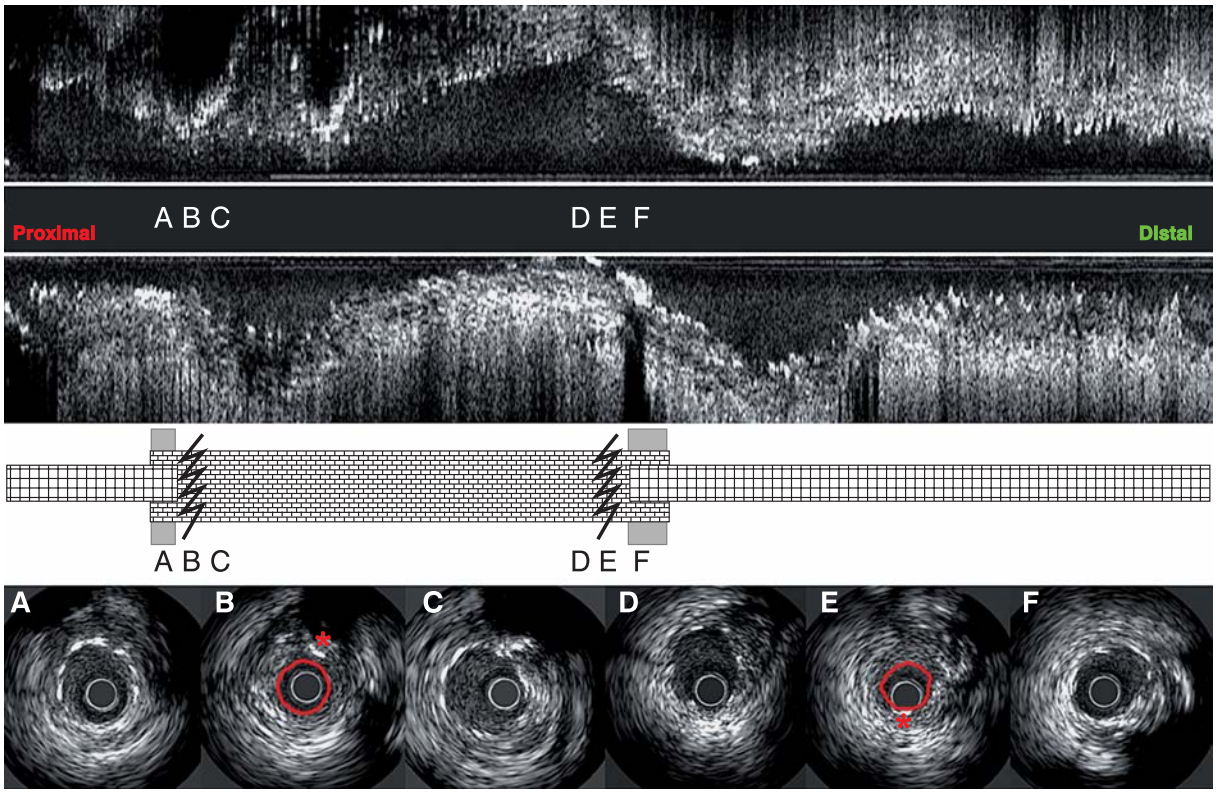

Figure 2. Top: Longitudinal intravascular ultrasound (IVUS) reconstruction of the right coronary artery.
Middle: Schematic drawing indicating the anatomical relationship of the three overlapping stents and the location of the two stent breaks adjacent to the overlapping segments.
Bottom: Cross-sectional IVUS images corresponding to the location indicated by the capital letters (A to F) on the longitudinal reconstruction. (A) Overlap of the proximal with the middle stent (2 stent strut layers are visible). (B) Proximal stent break of the middle stent with pronounced neointimal formation and only one visible stent strut (asterisk). The red line delineates the lumen plaque interface. (C) Distal to the proximal stent break one stent strut layer is visible. (D) Proximal to the distal stent break one stent strut layer is visible. (E) Distal stent break of the middle stent with pronounced neointimal formation and only one visible stent strut (asterisk). The red line delineates the lumen plaque interface. (F) Overlap of the middle with the distal stent (2 stent strut layers are visible).

intravascular ultrasound should be performed to clarify whether this is an important issue or a sporadic observation.

References

1. Serruys PW, de Jaegere P, Kiemeneij F, et al: A comparison of balloon-expandable-stent implantation with balloon angioplasty in patients with coronary artery disease: Benestent study group. N Engl J Med 1994; 331: 489-495.
2. Serruys PW, Kay IP, Disco C, Deshpande NV, de Feyter PJ: Periprocedural quantitative coronary angiography after Palmaz-Schatz stent implantation predicts the restenosis rate at six months: results of a meta-analysis of the Belgian Netherlands Stent study (BENESTENT) I, BENESTENT II Pilot, BENESTENT II and MUSIC trials. J Am Coll Cardiol 1999; 34: 1067-1074.
3. Morice MC, Serruys PW, Sousa JE, et al: A randomized comparison of a sirolimus-eluting stent with a standard stent for coronary revascularization. N Engl J Med 2002; 346: 1773-1780.
4. Stone GW, Ellis SG, Cox DA, et al: A polymer-based paclitaxel-eluting stent in patients with coronary artery disease. N Engl J Med 2004; 350: 221-231.
5. Donahue DG, Saltzman JR, Krims P: Stent fracture in malignant biliary obstruction. Gastrointest Endosc 1993; 39: 864-865.
6. Schoefl R, Winkelbauer F, Haefner M, Poetzi R, Gangl A, Lammer J: Two cases of fractured esophageal nitinol stents. Endoscopy 1996; 28: 518-520.
7. Grimley CE, Bowling TE: Oesophageal metallic stent dysfunction: first reported case of stent fracture and separation. Endoscopy 1999; 31:S45.
8. Sacks BA, Miller A, Gottlieb M: Fracture of an iliac artery Palmaz stent. J Vasc Interv Radiol 1996; 7: 53-55.
9. Phipp LH, Scott DJ, Kessel D, Robertson I: Subclavian stents and stent-grafts: cause for concern? J Endovasc Surg 1999; 6: 223-226.
10. Knirsch W, Haas NA, Lewin MA, Uhlemann F: Longitudinal stent fracture 11 months after implantation in the left pulmo-

nary artery and successful management by a stent-in-stent maneuver. Catheter Cardiovasc Interv 2003; 58: 116-118.
11. Duda SH, Pusich B, Richter G, et al: Sirolimus-eluting stents for the treatment of obstructive superficial femoral artery disease: six-month results. Circulation 2002; 106: 1505-1509.
12. Chowdhury PS, Ramos RG: Images in clinical medicine: coronary-stent fracture. N Engl J Med 2002; 347:581.
13. Sianos G, Hofma S, Ligthart J, et al: Stent fracture and restenosis in the drug-eluting stent era. Catheter Cardiovasc Interv 2004; 61: 111-116.
14. Shite J, Matsumoto D, Yokoyama M: Sirolimus-eluting stent fracture with thrombus, visualization by optical coherence tomography. Eur Heart J 2005; [Epub ahead of print].
15. Halkin A, Carlier S, Leon M: Late incomplete lesion coverage following Cypher stent deployment for diffuse right coronary artery stenosis. Heart 2004; 90:e45.