Case Report



Late Restenosis Related to Cypher Stent Fracture

Michel Pereira Cadore², Henrique Pereira Abelin¹, Romualdo Bolzani dos Santos¹, Paulo Ricardo Avancini Caramori² Instituto Cardiovascular¹, Santa Maria, RS; Hospital São Lucas da PUCRS², Porto Alegre, RS - Brazil

Drug-eluting stents represent a significant evolution in the therapy of coronary artery disease. Recently, restenosis and thrombosis related to drug-eluting stent fractures have been described. This work reports a case of fracture of a drug-eluting stent 18 months after implantation, associated with restenosis.

Introduction

In spite of their strong inhibitory effect on intimal hyperplasia, drug eluting stents (DES) are susceptible to restenosis and thrombosis, which represent relevant clinical problems. Not only have stent fractures been reported, but it has also been suggested that they have a role in the induction of restenosis and thrombosis. Recent studies have shown that the incidence of DES fracture varies from 0.84% to 7.7%¹⁻⁵. The frequency of clinically relevant events in patients with stent fractures may reach 70%². We report a case of restenosis of bare metal stent treated with implantation of drug eluting stent, in which late restenosis related to stent fracture was observed.

Case report

A 53-year old male patient, hypertensive, dyslipidemic, had been submitted to surgical myocardial revascularization with grafting of the left mammary artery on the anterior descending artery and grafting of the right mammary artery on the circumflex artery, in 1988. In August 2003, the patient presented with progressive angina (class III). He was under the following medication: AAS 100 mg/day, Atorvastatina 20 mg/day, Atenolol 100 mg/day, Enalapril 40 mg/day and Anlodipina 10 mg/day. The patient was submitted to coronary angiography, that revealed pervious grafts of mammary artery, severe proximal stenosis and significantly tortuosity of proximal and middle segments of the right coronary artery (RCA). A percutaneous coronary intervention (PCI) was performed and a Bx-Sonic stent (Cordis, Jonhson & Jonhson), 3.0x23 mm, was chosen for implantation. The stent was deployed at 16

Key words

Drug-eluting stents; Sirolimus-eluting stents; Restenosis; Stent fracture.

Mailing address: Michel Pereira Cadore•

Rua Tomaz Flores, 45 / 21, Independência – 90035201 - Porto Alegre,

 $E\text{-mail: michel_cadore@hotmail.com}$

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atm, with good angiographic result, but with rectification of the tortuosity of the vessel (Fig 1).

After six months without symptoms, the patient present recurrent angina (class II). A new coronary angiography showed intra-stent restenosis and recurrence of the original tortuosity of the vessel which suggested stent fracture. A PCI was performed and a 3.0x33 mm Cypher stent (Cordis, Jonhson e Jonhson) was deployed, with good angiographic result, but with significant rectification of the RCA proximal segment, and kinking distal to the stent (Figure 1).

Eighteen months later (August 2005), the angina symptoms returned. A new coronary angiography showed the presence of two points of focal intra-stent restenosis, with recurrence of the original RCA path and aspect suggestive of stent fracture in two points, associated to restenosis. Two 3.0x12 mm Taxus stents (Boston Scientific) were implanted at 18 atm on the restenosis areas. Angiographic success was obtained, and the RCA tortuosity was maintained (Figure 2). The use of clopidogrel for 12 months after the procedure was recommended. The patient is under regular clinical follow-up, free from angina symptoms and without evidence of myocardial ischemia until the most recent evaluation (March 2009).

Discussion

Cases of fracture of coronary stents have been recently described. Fractures have been observed on stents implanted in the three main coronaries, in any segment of the vessel, and with variable presentation periods, ranging from acute fractures to fractures detected two years after implantation⁶⁻⁸.

The inhibition of intimal hyperplasia resulting from DES demands that the stent is adequately apposed to the vessel wall. On the fractured site the drug distribution tends to be less uniform, resulting in areas of excessive neointimal hyperplasia with predominance of focal restenosis. Furthermore, stent fracture may represent a mechanical stimulation for the vessel wall, inducing inflammation, intimal hyperplasia and exposure of subendothelial components which result in higher risk of restenosis and thrombosis.

The mechanism responsible for the fracture is not well understood, but seems to be related to stent overexpansion and mechanical fatigue. Stent overexpansion may lead to stretching, deformation and weakening of the structure, so that fractures are more probable to happen. Mechanical fatigue of the stent may occur in areas of tortuosity, due to factors such as stretching, compression, torsion, kinking, elongation, and shearing of the stent structure during the cardiac cycle.

Fractures of stents implanted in the RCA and in saphenous

Case Report

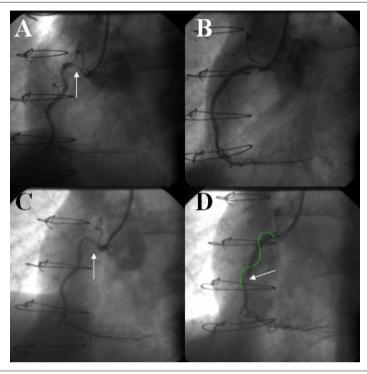


Figure 1 - A - RCA with proximal stenosis and B - after implantation of a Bx-Sonic stent; C - Intra-stent restenosis and recurrence of the original tortuosity of the vessel; D - Modification of the conformation of the proximal segment of the RCA after implantation of a Cypher stent (the line shows the original conformation of the vessel) and kinking distal to the stent during systole (arrow).

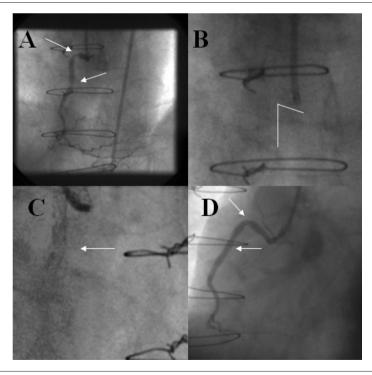


Figure 2 - A - Restenosis on the fracture areas of the Cypher stent; B - Fracture on the proximal segment of the Cypher stent (outlined by the white line); C - Fracture on the distal segment of the Cypher stent; D - After implantation of two Taxus stents.

Case Report

vein grafts have been reported⁹. The fracture may be induced by a more dynamic movement of the RCA during the cardiac cycle. The present case illustrates a tortuous RCA before stenting, rectification of the vessel after stent implantation, and kinking distal to the stent, associated to increased mobility of the artery during cardiac cycle.

The use of overlapping stents represents another factor leading to decreased flexibility of the vessel and susceptibility to deformation and fracture of the stents⁹.

Bare metal stents are more susceptible to restenosis of the diffuse type, making the detection of fractures more difficult. In the present study, rectification of the RCA proximal segment was observed after implantation of the first stent. The posterior cinecoronariography showed restenosis and recurrence of the original tortuosity of the vessel, suggesting the occurrence of a fracture, but the pattern of diffuse restenosis makes difficult the recognition of this possible fracture.

Most of the fracture cases reported are related to Cypher Sirolimus-eluting stents. Different from the paclitaxel-eluting Taxus stents, the Cypher stent is designed as a closed cell. Furthermore, it seems to be less conformable. When implanted in tortuous vessels, it may induce excessive rectification of the coronary, originating points of tension on the stent.

Intracoronary ultrasound and fluoroscopy have been efficient in the diagnosis of fractures. In a few patients, the fracture is complete and might be observed by coronary angiography.

There is no consensus about the best therapy for restenosis related to DES fracture. The implantation of another stent represents a controversial alternative, due to the possible recurrence of the fracture. In the present case, we decided

to treat the second restenosis by implanting a stent with different conformability and drug, although there is limited evidence for this approach. The duration of the antiplatelet therapy is also a controversial issue, due to the potential risk for thrombosis associated to the use of overlapping stents and drugs with different mechanisms. In this case, double antiplatelet therapy was indicated for a period of 12 months. Currently, the patient is under clinical follow-up, with no evidence of complications.

The present case shows that stent fracture may determine relevant outcomes, with sequential restenosis and the need for repeated interventions. The main factors predisposing to DES fracture are implantation in tortuous vessels, the use of overlapping stents and stent overexpansion. The lesser conformability of a particular type of stent seems to determine a greater probability of fracture. The actual incidence of fractures in coronary stents is difficult to assess, but it probably increases the chances of restenosis and thrombosis, followed by relevant clinical events.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any post-graduation program.

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