Aneurysm Formation after Paclitaxel-Eluting Coronary Stent Implantation

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Formation of coronary aneurysm is a rare complication after implantation of bare metal stents (BMS). However, in the drug-eluting stent (DES) era, the incidence of such complication is still unknown. We herein report a 67-year-old man who had a chronic total occlusion lesion in the left anterior descending artery and underwent percutaneous coronary intervention with deployment of 3 stents, including 2 polymer-based paclitaxel-eluting stents. Fourteen months post intervention, a large aneurysm developed within the distal DES. Intravascular ultrasound demonstrated a true aneurysm about 5.6 mm in diameter. The patient remained asymptomatic. We review the literature and discuss the etiology as well as treatment of coronary artery aneurysms within DES.

Key Words: Coronary artery aneurysm • Drug-eluting stent • Paclitaxel

INTRODUCTION

Formation of coronary aneurysm is a rare complication after implantation of bare metal stents (BMS). However, in the drug-eluting stent (DES) era, the incidence of such complication is still unknown. Experimental studies had shown that in animals implanted with the paclitaxel-eluting stent, under certain circumstances, the lumen progressively dilated over time, leading to aneurysm formation.1 In humans, to our knowledge, less than 5 cases of coronary aneurysms after the deployment of paclitaxel-coated stents have so far been reported.

CASE REPORT

A 67-year-old man with acute ST segment elevation myocardial infarction underwent primary percutaneous coronary intervention (PCI) for the right coronary artery (RCA), the infarct-related artery. The coronary angiogram at that time also showed a chronic total occlusion (CTO) lesion in the middle portion of left anterior descending artery (LAD) (Figures 1a and 1b). Second catheterization was scheduled for two months later. In that second catheterization, the CTO lesion was successfully opened and a bare-metal stent (Express 2, 4.0 × 20 mm, Boston Scientific Scimed, Ireland) and 2 drug-eluting stents (Taxus, 3.5 × 32 mm and 2.75 × 32 mm, Boston Scientific Scimed) were deployed sequentially after intravascular ultrasound assessment with maximum inflation pressures 14, 12, and 16 atm, respectively, from proximal to distal segments of the LAD (Figures 2a through 2d).

The patient was given aspirin 100 mg and clopidogrel 75 mg daily after the initial attack of acute myocardial infarction, but clopidogrel was cancelled 4 months after the deployment of DES. He denied any discomforts after the deployment of DES. Seven months post infarct, treadmill test showed no evidence of myocardial ischemia. Sixteen months post infarct, the patient underwent repeat coronary angiography to evaluate the vessels and an ectasic area around the position of the distal DES
in the LAD was found (Figures 3a and 3b). Assessment of the ectasic area using intravascular ultrasound (IVUS, 40 MHz, Boston Scientific Scimed) demonstrated a true aneurysm about 5.6 mm in diameter (Figure 3c). Since the patient was free of symptoms, we decided to add clopidogrel 75 mg daily and continue the dual anti-

Figure 1. Coronary angiograms taken before the PCI for the CTO lesion. a, AP-cranial view and b, LAO-caudal view show a chronic total occlusion in the middle portion of the LAD.

Figure 2. Coronary angiograms taken during the PCI for the CTO lesion. A, AP-cranial view and B, LAO-caudal view show no dissection or thrombus formation after the LAD (arrows) was opened. C, AP-cranial view and D, LAO-caudal view show that 1 BMS and 2 DES were deployed sequentially from proximal to distal segments of the LAD (arrows). See text for details.
platelet agents therapy. At the submission of this report, he was still free of symptoms.

**DISCUSSION**

Coronary arterial aneurysms (CAAs) are defined as dilated arterial segments with diameters exceeding 1.5 times those of the adjacent reference segments. In infants, a diameter of more than 8 mm is considered as “giant CAA”, but in adults, the data varies from 10 to 50 mm.\(^2\)\(^3\) Most CAAs are found incidentally during coronary angiographic examination or at necropsy. The incidence varies from 0.9% to 4.9%, with male dominance and a predilection for the right coronary artery.\(^4\) Atherosclerosis is the most common etiology of CAAs, accounting for 50% of cases diagnosed in adults, followed by Kawasaki’s disease and congenital aneurysms. Regarding the incidence of CAA after endovascular procedures, CAA has been reported to exist in approximately 4% of patients post percutaneous transluminal coronary angioplasty (PTCA)\(^5\) and up to 10% after directional coronary atherectomy.\(^6\)

Major factors contributing to the formation of CAAs after PCI with BMS are an oversized balloon, an intensive procedure, and dissection of the vessel wall during PCI.\(^5\) Regarding the formation of CAA after DES deployment, apart from those factors mentioned above, the active drugs plus the polymer coating of DES can also be involved. In our patient, no angiographically detectable dissection was found before or after the DES deployment. In addition, the DES was not oversized, as revealed by IVUS. However, high inflation pressure (up to 16 atm) for the DES, the anti-migratory plus anti-proliferative effect of paclitaxel, and a marked inflammatory response induced by the polymer might work together to damage the vascular wall and weaken the repair mechanism of the vessel, thereby causing aneurysm formation.

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**Figure 3.** Coronary angiograms taken 14 months post opening of the CTO lesion. A, AP-cranial view and B, AP-caudal view show an ectasic segment (arrows) within the distal DES in the LAD. C, IVUS demonstrates a true aneurysm about 5.6 mm in diameter encircling the DES, the struts of which are indicated by arrows.
formation.5,7

Bal et al reported that CAA formation after PTCA appeared to be a relatively infrequent and harmless event, and carried the same good prognosis as aneurysmal coronary artery disease.5 But data from a recent retrospective study showed that CAA was an independent predictor of mortality, and overall 5-year survival in patients with CAA was only 71%.4 Moreover, there was no statistically significant association between aneurysm size and survival rate. In contrast, the type of the aneurysm is a major determinant. True aneurysm after PTCA is associated with low morbidity and mortality,5 while pseudoaneurysm has potential for progressive enlargement or eventual rupture. Unlike the wall of a true aneurysm, the fibrous wall of a pseudoaneurysm is not in continuity with the structure of the adjacent vessel wall and does not contain remnants of the original arterial wall. Although it is difficult to distinguish a true aneurysm from a pseudoaneurysm by angiography, IVUS has been reported to be useful for this purpose.8

Management of patients with CAA remains undetermined. The presence of CAA is not a surgical indication. But surgery has been recommended for large aneurysms in views of the risk for thrombosis and rupture, especially in the presence of saccular aneurysm.9 For atherosclerotic aneurysms, most authors agree that surgical treatment is warranted depending on the severity of associated coronary stenosis rather than the mere presence of aneurysm. There is also no standard therapy recommended for pseudoaneurysm, left main CAA or CAA with fistula, although outcome of operation was excellent.9,10 Saito et al successfully treated one patient with pseudoaneurysm by spring coils.11

In recent years, covered stents have become a new alternative to surgery in the treatment of CAAs. Autologous venous covered stents have shown promising results, but their implantation is technically demanding.12 Polytetrafluoroethylene (PTFE) graft covered stents are easy and rapid to deploy and have recently emerged as a new tool for the treatment of CAAs in several reports, even for left main CAA.13,14 But there are still major concerns after PTFE-covered stent implantation. First, the incidence of stent thrombosis observed with PTFE-covered stents is higher than that observed with conventional stents (5.5% vs. 3.6%).13,15 Second, side-branch occlusion rate may be as high as 18%.13 Third, the restenosis rate of PTFE-covered stents varies from 14–39%.13,14

To date, there is still no definite recommendation regarding the treatment of CAA in patients with DES. In our patient, we considered that PTFE-covered stent in LAD was prone to side-branch occlusion and the risks of stent thrombosis and restenosis after PTFE-covered stent might be higher than the adverse effects of a true aneurysm, therefore we decided to maintain dual anti-platelet agent therapy for this asymptomatic patient, and he was still free of symptoms after 6 months’ follow-up.

REFERENCES


冠狀動脈瘤形成於 Paclitaxel 塗藥支架置放術之後

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在置放一般金屬支架之後形成冠狀動脈瘤是一種罕見的併發症，然而，於塗藥支架的年代，此併發症的發生率仍然不清楚。我們在此提出一位 67 歲男性病患於冠狀動脈左前降支有慢性完全阻塞，接受經皮冠狀動脈介入性治療並置放 3 個支架，其中包含 2 個 paclitaxel-塗藥支架。介入性治療之後的 14 個月，在塗藥支架末端範圍形成一個大的冠狀動脈瘤。血管內超音波證實是一個直徑約 5.6 毫米的真動脈瘤。此病患無症狀，我們回顧文獻，討論在塗藥支架範圍形成冠狀動脈瘤的原因以及治療的方法。

關鍵詞：冠狀動脈瘤、塗藥支架、paclitaxel。