Development of a Coronary Artery Aneurysm
Following Stent Implantation in a Chronic, Totally Occluded Lesion

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Coronary artery aneurysm formation is an uncommon finding after coronary stent implantation and is possibly related to deep vessel injury, plaque regression and positive remodeling. We report herein a 31-year-old patient who developed an aneurysm of a coronary artery four months after successful stent implantation to relieve a chronic, totally occluded lesion. The aneurysm occurred within the stented segment of the artery. Intravascular ultrasound image revealed good stent apposition without visible vessel dissection. The aneurysm regressed spontaneously after the ensuing five months.

Key Words: Coronary artery aneurysm • Stent • Positive remodeling • Chronic total occlusion

INTRODUCTION

Coronary artery aneurysm (CAA) is an uncommon finding after coronary balloon angioplasty or stent implantation. The mechanism of transcatheter therapy-induced CAA formation include: deep vessel injury, plaque regression, and vessel positive remodeling. The development of a CAA following stent implantation in a chronic, totally occluded (CTO) lesion has not been previously reported.

CASE REPORT

A 31-year-old man was hospitalized for chronic stable angina, in Canadian Cardiovascular Society (CCS) class 3. The major risk factor was hyperlipidemia. Stress myocardial thallium scintigraphy demonstrated a perfusion defect in the apex and anterior wall of the left ventricle. Coronary angiography showed 100% occlusion of the proximal right coronary (RCA) and the proximal left anterior descending (LAD) artery, with an intra-coronary collateral from the left circumflex artery (LCX) to the distal LAD and RCA.

Standard percutaneous transluminal angioplasty (PTCA) was performed for the CTO lesion of the RCA (level III, bridging collateral) after administration of 10,000 IU heparin intravenously (Figure 1). An AL1 7F Cordis guiding catheter and two 0.014″ guidewires (Asahi Conquest, 12 g) with one microcatheter (Boston Excelsior) were used. The parallel wire successfully penetrated the distal cap. A 1.25 mm × 15 mm Terumo Ryujin monorail balloon and a 2.5 mm × 20 mm Boston Maverick monorail balloon were used to pre-dilate the lesion, with maximal pressure to 12 atm. Two overlapping bare-metal stents (Medtronic Driver, 3.0 mm × 30 mm and Orbus R stent, 3.0 mm × 33 mm) were then placed at the proximal-to-mid RCA, with a pressure to 14 atm. Stent placement appeared satisfactory on angiography, with normal TIMI flow and without evidence of dissec-
tion (Figure 1). Four months later, the patient underwent coronary angiography for the CTO lesion of the LAD. RCA follow-up angiography revealed a large CAA involving the proximal stented segment (Figure 2). Intravascular ultrasound (IVUS) analysis of the RCA demonstrated a symmetric stent expansion and complete apposition to the vessel wall at the distal stent, with large plaque and media area. In the region of the aneurysm, there was no contact between the stent strut and the vessel wall (Figure 2A). The proximal stent inlet and the distal stent outlet showed complete stent apposition to the vessel wall. There was no significant intimal hyperplasia. No further intervention was performed for the aneurysm.

Five months later, the patient underwent coronary angiography again for the CTO of the LAD lesion. RCA follow-up angiography revealed complete regression of the CAA (Figure 2B). IVUS analysis revealed a residual aneurysm, but the size was much smaller as compared with the previous study (Figure 2d). PTCA was performed for the CTO lesion of the LAD successfully with kissing wire technique.

DISCUSSION

Before the advent of arteriography, CAA was an incidental finding in 1.4% of autopsy patients.\(^1\) By definition, a characteristic coronary aneurysm is greater than 1.5 times the diameter of the normal artery. The incidence of CAA after coronary intervention is unknown. Vassanelli et al. found five CAAs in four patients among 80 patients (5%) who underwent elective coronary angiography six months after PTCA.\(^2\) Elective stent implantation did not increase the risk for CAA formation, as compared with conventional PTCA, in the Slota et al. study.\(^3\) In the case presented here, CAA was observed after 4 months of stenting a CTO lesion. The potential

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**Figure 1.** A, RCA CTO lesion with bridging collateral. B, distal Medtronic Driver stent 3.0 × 33 mm location (arrow). C, proximal Orbus R stent 3.0 × 33 mm location (arrow). D, final result, normal TIMI flow and no angiographically visible dissection.

**Figure 2.** Follow-up angiography with IVUS imaging 4 (panel A) and 9 (panel B) months later. In A, angiogram shows a large CAA in the proximal stented RCA segment, where IVUS images (a-c) shows no contact between stent strut and vessel wall in about 200° of vessel circumference. The stent strut is regular without break. In B, angiogram shows regression of the RCA CAA, where IVUS image (d) shows that the vascular cross-section area is much smaller, compared to the IVUS images in A. Note that each of the IVUS images a-d are taken from the corresponding locations a-d of the angiograms.
mechanisms of CAA formation include: dissection or deep vessel injury, plaque displacement of regression, and coronary artery positive remodeling.

Interventional vessel trauma is a known reason for vessel remodeling of aneurysm. Injury or dissection of the media can lead to dilatation or thinning of the vessel wall, and increasing stress can ultimately result in an aneurysm. Different transcatheter therapies at the site of subsequent aneurysm formation have been reported, such as balloon angioplasty, directional coronary atherectomy, excimer laser angioplasty, and stenting. The continued presence of a small dissection after stent implantation may be a cause of CAA. Among the reported CAAs related to deep vessel injury, the size of the aneurysm enlarged or remained stationary during follow-up. No regression had previously been reported. In our case, detailed information about vessel wall structure and the apposition of the stent to the vessel was obtained by IVUS at follow-up angiography. No dissection or deep injury was found. Moreover, the CAA in our case resolved five months later. Dissection or a deep vessel injury did not appear to be causal.

CAA formation can occur even after an uncomplicated coronary stent implantation. Explanations include a decrease in plaque volume and vessel wall positive remodeling. Reasons for a decrease in plaque include clot lysis, plaque regression, or apoptosis. Okura et al. used IVUS to examine the serial geometric changes to stented coronary vessels after exposure to intravascular brachytherapy. They found the cross-sectional area of the plaque plus media tended to decrease during follow-up in stent late incomplete apposition group. In a CTO lesion, there is heavy plaque burden, and plaque regression after intervention may contribute to the formation of an aneurysm.

Vessel positive remodeling refers to an increase in the cross-sectional area of the external elastic membrane (EEM). In effect, the vessel grows and the vessel wall pulls away from the stent. Using serial IVUS, a number of investigators have reported positive remodeling associated with a peri-stent plaque increase after BMS implantation. However, an EEM increase is not necessarily a response to peri-stent intimal hyperplasia, but can occur independently. An increase in the EEM greater than the increase in plaque, leading to an increase in lumen dimensions, has been reported in 10% of non-stent interventions and in early atherosclerosis. A primary signal for arterial remodeling is shear stress. A macroscopic increase in blood flow increases local shear stress and stimulates arterial expansion until shear stress has been restored to baseline. In our RCA CTO lesion, vessel restructuring during the acute procedure and continuous vessel expansion (positive remodeling due to large increased local shear stress) in the subsequent months combined to cause aneurysm formation. Some predisposing effects can augment the vessel remodeling. Rab et al. reported predisposing effects of cortisone and colchicine treatment in patients with stent implantation for bailout purposes, and 32% of patients (6 of 19 patients) developed an aneurysm. The positive remodeling in response to increased flow is largely dependent on shear-responsive endothelial production of nitric oxide and the gelatinase matrix metalloproteinases (MMPs) MMP-2 and MMP-9. Once the shear stress is restored to baseline, positive remodeling stopped, and negative remodeling may occur. Negative remodeling occurred predominantly between 1 and 6 months after angioplasty, possibly due to adventitial cicatrisation and low shear stimulation of platelet-derived growth factor and transforming growth factor-β expression. Kimura et al. used serial IVUS measurements for analysis of vessel remodeling after directional coronary atherectomy or balloon angioplasty. The study described an increase in EEM cross-sectional area, or positive remodeling, between 24 h and 1 month after intervention, and a decrease in EEM cross-sectional area, or negative remodeling, between 1 and 6 months after intervention. However, negative remodeling was not found in every CAA, and the major determinants of CAA regression are still unclear.

Little is known about the course and prognosis of intervention-related CAA. Two major events are relevant to prognosis, however: the rupture of the CAA and thrombosis with distal embolization. The time period of CAA progression or rupture is poorly defined. There have been case reports that CAA formation after stent implantation enlarged progressively in a span of 8 months, with concern of vessel rupture. Patients can be symptomatic with ischemia in the area supplied by the aneurysm secondary to an embolism, thrombus, or dissection with lumen narrowing, and the frequency is unknown. Left ventricular hypokinesia in an area supplied by a coronary artery without significant obstruction.
but with an aneurysm has been reported in 6 patients, and a possible explanation could be distal embolization. However, Slota et al. described that angiographic re-stenosis and clinical outcome were not affected by the development of a CAA after PTCA or stent placement. Nevertheless, with the increasing size of the aneurysm, symptoms, and the documentation of ischemia, treatment would have still been required. Beneficial treatments include surgical intervention, such as bypass grafting, resection, ligation in the atherosclerotic aneurysms, or percutaneous intervention. Obliteration of CAA by percutaneous intervention with covered stent (vein-coated stent or PTFE-covered stent) has become a new treatment of CAAs in recent years. In our case, we chose medical therapy due to the absence of existing ischemic symptoms and no uncovered dissection being visualized by IVUS. The aneurysm regressed five months later. Awaiting vessel negative remodeling and spontaneous regression of aneurysm are also considered to be choices for treatment.

CONCLUSION

CAA can occur after an coronary artery stent implantation, and also can regress spontaneously without further intervention. The most likely mechanism is shear stress inducing vessel positive remodeling after an intervention procedure, with subsequent negative remodeling when shear stress returns to baseline.

REFERENCE

冠狀動脈置放支架後產生冠狀動脈瘤是不常見的，且可能和深層管壁損傷、粥狀斑塊消退、和血管壁正向塑型有關。我們在這裡報告一位 31 歲病患於慢性完全阻塞冠狀動脈成功放置支架後的四個月產生了冠狀動脈瘤。動脈瘤發生於支架置放段的血管。血管內超音波影像分析顯示支架和管壁貼附良好，沒有可見的血管壁剝離。冠狀動脈瘤在五個月後自然消退。

關鍵詞：冠狀動脈瘤、支架、正向塑型、慢性完全阻塞。