Very Late Development of Coronary Artery Aneurysm with Total Occlusion following Sirolimus-Eluting Stent Implantation

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Abstract

Keywords

- coronary artery aneurysm
- coronary artery bypass
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- percutaneous coronary intervention

A 41-year-old African American woman presented with chest pain and was found to have non-ST segment elevation myocardial infarction with a peak cardiac troponin I of 28.5. Elective cardiac catheterization revealed a 70% ostial left anterior descending (LAD) artery stenosis. The patient underwent percutaneous coronary intervention and a sirolimus-eluting stent (Cypher, Miami, FL, 3.5 × 8 mm) was successfully deployed. Three years after stent implantation, the patient presented with recurrent angina. Repeat coronary angiography revealed a large aneurysm involving the proximal portion of the stent with a total occlusion at the mid to distal portion of the stent with collaterals to LAD from left circumflex artery. The patient underwent coronary artery bypass surgery with left internal mammary artery graft to LAD and ligation of LAD at its origin proximal to the aneurysm.

Coronary artery aneurysm (CAA) is defined as a dilation of the coronary artery that exceeds 1.5 times the reference diameter of the adjacent coronary segments which are angiographically normal.¹ Meta-analysis by Hakeem et al² reported that the incidence of CAA with drug-eluting stent (DES) was 0.6 to 1.9% at a mean follow-up of 9 months when compared with pre-DES era which was 1.4 to 4.9%.

Aneurysm formation has been observed from 1 week to 4 years after DES implantation, and up to 9 years after bare-metal stent implantation.³ By using intravascular ultrasound (IVUS), Alfonso et al identified that 1.25% of patients developed CAA after DES implantation in the culprit vessel in acute myocardial infarction.⁴ Nakagawa reported that CAA was identified in 1.7% of patients by coronary angiography (CAG) mostly 1 year after the implantation of DES.⁵ However, the incidence and natural history of CAAs after percutaneous coronary intervention (PCI) are not well known by CAG alone.⁶ Aneurysm formation is more commonly a result of traumatic rupture or deep-vessel injury of an artery. We

described a case of angiographic CAA formation 3 years after a sirolimus-eluting stent (SES) implantation accompanied by chronic total occlusion of the stent.

Case Report

A 41-year-old African American woman with past medical history of hypertension, hyperlipidemia, and diabetes mellitus type 2 presented with chest pain and was found to have non-ST segment elevation myocardial infarction with a peak cardiac troponin I of 28.5. The patient underwent CAG in December 2007. The angiogram revealed a 70% ostial left anterior descending (LAD) artery stenosis (►Fig. 1A), and a 10 to 20% proximal stenosis of left circumflex artery (LCx) and a normal right coronary artery. The patient underwent PCI. A SES (Cypher, Miami, FL, 3.5 × 8 mm, maximal inflating pressure of 11 atm) was successfully deployed (►Fig. 1B) in ostial LAD.

After 3 years (December 2010), the patient presented to cardiology clinic with a 6-month history of chronic stable

Fig. 1 Coronary artery aneurysm with a total occlusion following drug-eluting stent implantation. (A) Angiogram showing a 70% ostial LAD artery stenosis. (B) A sirolimus-eluting stent deployed in ostial LAD. (C) Repeat angiogram showing a large aneurysm involving the proximal portion of the stent with a total occlusion of the mid to distal portion of the stent and collaterals filling of distal LAD from the LCx. LAD, left anterior descending; LCx, left circumflex artery.

angina. Echocardiogram showed normal left ventricular (LV) systolic function with normal wall motion. Repeat CAG revealed a large aneurysm involving the proximal portion of the stent with a total occlusion of the mid to distal portion of the stent (►Fig. 1C) and collaterals filling of distal LAD from the LCx. There was a 50 to 60% proximal LCx stenosis. She underwent coronary artery bypass surgery with left internal mammary artery graft to LAD. The very proximal LAD was exposed and then the surgeon was able to pass a right-angle clamp just beyond the takeoff of the LCx. The LAD was ligated proximal to the origin of the aneurysm using a single 2–0 silk tie so that the aneurysm was no longer subjected to systemic pressure. A saphenous vein graft to the obtuse marginal artery was also performed. Since the LAD was totally obstructed just distal to the stent and aneurysm, this procedure is assumed to have effectively isolated the aneurysm from the arterial circulation and prevent further enlargement and the risk of late rupture. As the aneurysm was not excised, the type of aneurysm is unknown. However, it was thought to be most likely pseudoaneurysm. The bypass surgery was uneventful and the patient had a good recovery.

Discussion

Although there are several underlying mechanisms for the formation of CAA, the exact mechanism is still not clearly defined.⁵ The pathologic mechanism of CAA is most likely multifactorial. This may be due to the following: atherosclerosis, positive vessel remodeling, and hypersensitivity reaction to the polymer and/or drug, with a local inflammatory response. Factors such as drug toxicity, superimposed infection, and mechanical factors such as residual dissection, injury to arterial wall caused by oversized balloons and stents, high-pressure inflations, atherectomy, and perforations may also be associated with aneurysm formation after PCI.^{3,4}

Inhibition of normal healing process of the vessel wall by DES resulting in delayed endothelialization might be contributed to the expansion of the weakened areas of intima and

media secondary to the trauma of PCI and further influence the remodeling process leading to stent malposition, and finally aneurysm formation.⁷ Lastly, it has been reported that DES-related CAA might predispose to DES thrombosis which is a potentially fatal event due to acute vessel closure.⁴

Although development of CAA following placement of a SES was not uncommon, our report is significant for the following reasons. First, CAA is a rare complication after DES and the size of the aneurysm in this case is very large. Second, the clinical presentation of CAA in this case is very late and was found to occur almost 3 years after stenting. This has rarely been reported in the literature.⁸ Furthermore, different portions of the stent involve different pathologies. The aneurysm formed around the proximal portion and a total occlusion occurred at the mid portion of the stent. In our patient, sirolimus-induced positive remodeling of the artery was possible since there was no angiographic evidence of stent-induced dissection or perforation at the time of the procedure. In our patient, surgical option was the correct decision, especially since the aneurysm involved the left main and encroached upon the takeoff of the LCx. The distal left main involvement and thrombosis prevented use of a covered stent. Although rupture of coronary aneurysm is quite rare, follow-up coronary computed tomography might be helpful.

Due to the inherent limitations of CAG, pseudoaneurysms that form after PCI are usually overlooked or misdiagnosed as true aneurysms.⁹ Moreover, it is difficult to determine whether the aneurysm resolves, persists, or if any changes in morphology occur over time due to the necessity for repeat invasive coronary angiogram.

There are no clear guidelines for the management of post-stenting CAA, as the aneurysms have varied clinical outcome. Aoki et al³ recommended that treatment depends on the size of the aneurysm, expansion history, pathophysiology, and symptoms of the patient. In addition, enlarging aneurysms, infected aneurysms, and large, chronic aneurysms with symptoms should be treated aggressively. Covered stents or surgery may be indicated in the case of very large or enlarging aneurysms. From our case, we recommend that use of DES is

the best choice in a young diabetic female with normal left ventricular ejection fraction and single LAD disease. However, if the patient had multivessel disease or LV systolic dysfunction, coronary artery bypass graft would be the first choice.

The long-term follow-up of patients with DES implantation will help to identify patients developing aneurysm formation and treat patients to prevent complications of aneurysm formation. More prospective and randomized studies with late and very late follow-up are required in patients receiving DES to more accurately assess the risk of CAA and to clearly define the treatment guidelines.

One of the limitations of our case report was that no repeat CAG was done after stent placement until the CAG study 3 years later showing total occlusion of LAD and the aneurysm. Another limitation was the lack of either pathology report or IVUS report to clarify the morphology of CAA, or whether this was a pseudoaneurysm. The diagnosis of aneurysm was made by CAG and we did not excise the aneurysm.

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