



CASE REPORT

Multiple coronary micro-aneurysm formation after drug-eluting stent implantation



Apostolos H. Christou*, Emmanouil C. Papadakis, Nikolaos I. Nikolaou, Dionysios G. Antonatos, Athanasios V. Anadiotis, Sotirios P. Patsilinakos

Cardiology Department, Konstantopoulio General Hospital, Athens, Greece

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Abstract Although there are limited data regarding the formation of coronary artery aneurysms (CAAs) after drug-eluting stent (DES) implantation, CAAs appear to be a rare complication of coronary stenting. The exact mechanism of CAA formation is unknown, but several hypotheses have been proposed. As the use of DES increases, the clinical significance of these findings will become clearer. We report on a patient who developed multiple CAAs in 2 different locations after sirolimus-eluting stent implantation.

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1. Introduction

Recently, with the advent of the implantation of drug-eluting stents (DES), there have been increasing reports suggesting that stents cause coronary aneurysm (CAA) months or years after the procedure.¹ Although there are

limited data, CAA formation appears to be a rare complication of coronary stenting.² Two studies with a large number of patients and late angiographic evaluation have demonstrated CAA incidences after DES of 1.25% and 0.76%, respectively.³⁻⁴

We now report on a patient who developed multiple coronary artery micro-aneurysms after sirolimus-eluting stent implantation, which developed in 2 different locations at different times.

2. Case presentation

A 47-year-old man was referred to the cath-lab for primary percutaneous coronary intervention because of an inferior

* Corresponding author. Apostolos H. Christou, 20 Argiroupoleos str, GR-14231 Nea Ionia, Athens, Greece. Tel.: +30 210 2715415; fax: +30 210 2779509.

E-mail address: christouapostolos@yahoo.com (A.H. Christou).

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ST elevation acute myocardial infarction. Cardiac catheterization revealed a completely occluded right coronary artery (RCA), which was considered to be the culprit artery (Figure 1A). The proximal left anterior descending (LAD) artery was aneurysmatic (5-mm diameter), and the remainder was atheromatic with a stenosis of approximately 50%. After an initial balloon pre-dilatation, a sirolimus-eluting stent (3×28 mm) was delivered to the occluded right coronary artery. Post dilatation was performed with a 3.5×15 -mm non-compliant balloon in 14 atm; at the end of the procedure, there was no significant residual stenosis. (Figure 1B)

At a routine follow-up, 12 months after the original stent insertion, a myocardial perfusion study showed ante-septal and apical ischemia. A new coronary angiogram revealed a mid LAD 99% stenosis, which was stented with a 3×13 mm sirolimus-eluting stent. Post dilatation was performed with a 2.75×10 mm non-compliant balloon at 16 atm and had optimal immediate results. (Figure 2A–B) The catheterization also revealed multiple, small saccular aneurysms at the location of the stent in the right coronary artery that were not present on the original diagnostic angiogram or in the subsequent interventional procedure (Figure 1C).

One year later (2 years after the first stent was placed in the RCA), the patient remained asymptomatic, but he was still smoking. A routine coronary angiogram was performed, which confirmed the existence of coronary aneurysms in

the RCA stent site (Figure 1D) as well as revealed multiple saccular micro-aneurysms at the location of DES implantation in the LAD. (Figure 2C) The aneurysms were located in the proximal and mid segments of the LAD stent and in the mid segment of the RCA stent. Optical coherence tomography (OCT) imaging examination demonstrated good stent apposition of both stents in the vascular wall, and all of the struts appeared to be endothelialized. The aneurysm entry sites were located between the stents' struts (Figure 2D).

No further intervention was performed and medical treatment was chosen for this patient.

3. Discussion

The patient in this report developed coronary micro-aneurysms at the location of 2 stents, which were implanted in different coronary arteries at different times.

Dissection and deep arterial wall injury could be the cause of CAA formation after coronary intervention. These CAA types are mostly pseudo-aneurysms instead of true aneurysms. Additionally, delayed re-endothelialization, inflammatory changes in the medial wall and hypersensitivity reactions have been associated with CAA after DES implantation, which is caused by the antiproliferative and antimetabolite effects of the eluted drug or by hypersensitivity reactions to the drug/polymer.² There is some evidence that the use of bioresorbable stents could provide an answer for this type of impaired vascular healing.⁵ Vessel

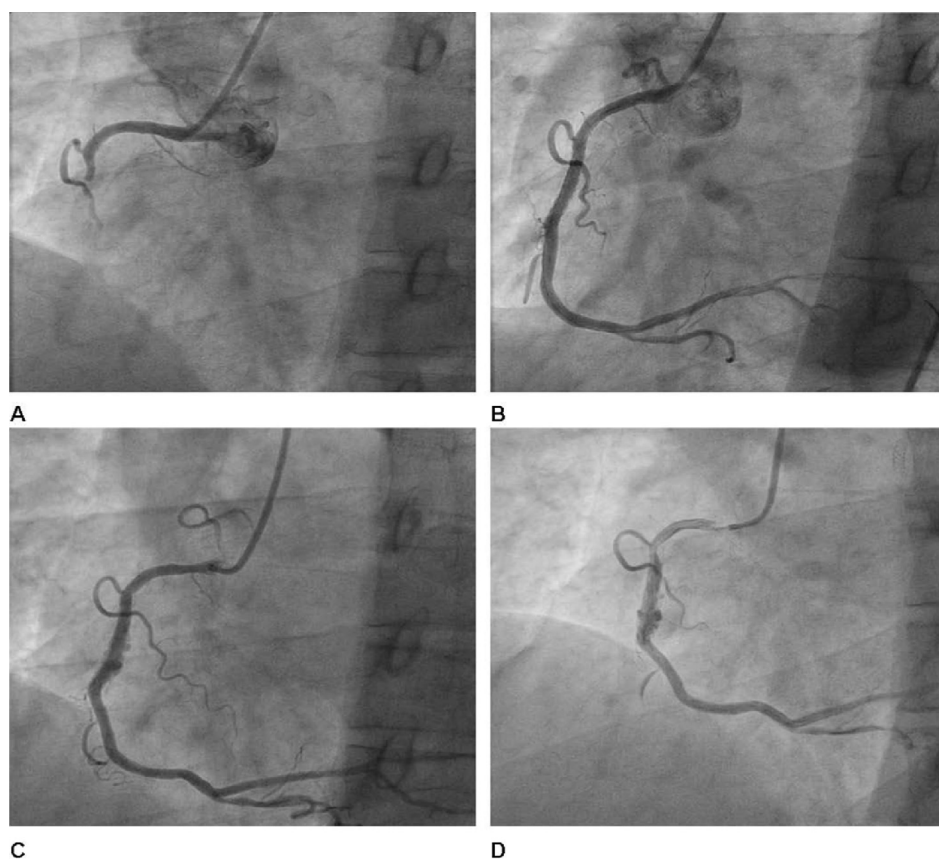


Figure 1 A: Total occlusion of RCA. B: Immediate result after stenting in RCA. C–D: Small coronary aneurysms in the location of the stent in RCA 1 (C) and 2 (D) years after DES implantation. RCA: Right coronary artery, DES: Drug eluting stent.

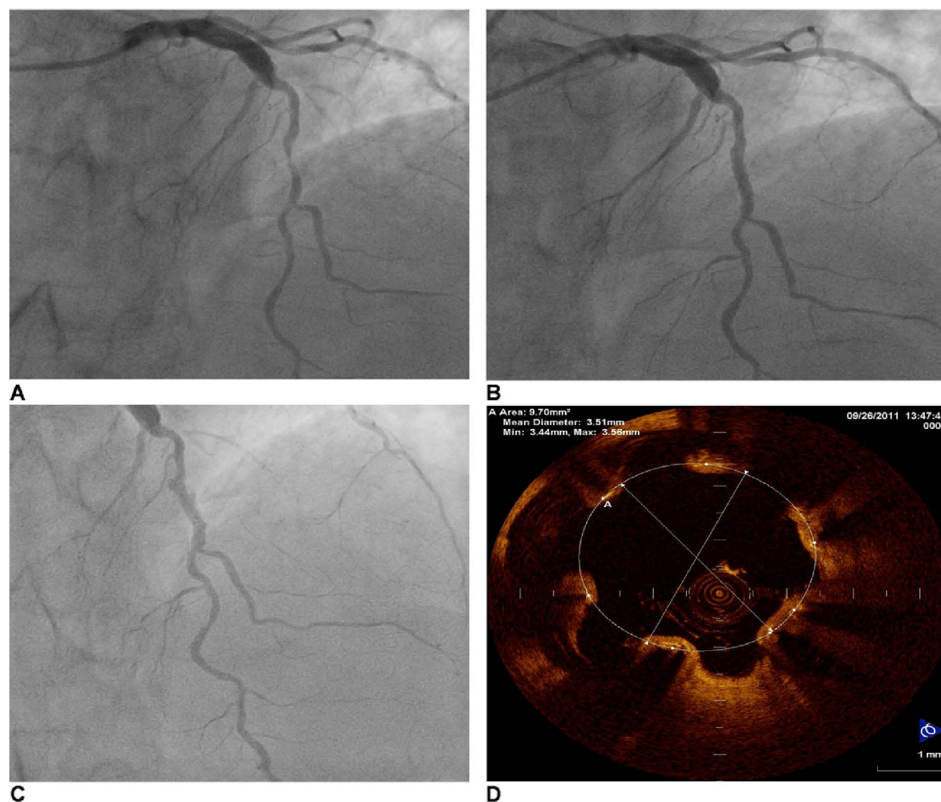


Figure 2 A: Mid LAD 95% stenosis 1 year after the first stent implantation in RCA. Aneurysm in proximal LAD. B Immediate result after stenting in LAD C: Multiple coronary aneurysms in the location of the stent in LAD 1 year later. D: Optical coherence tomography revealed struts endothelialisation and aneurysms' entry sites. LAD: Left anterior descending, RCA: Right coronary artery.

remodeling and DES malapposition have also been recognized as a possible mechanism for CAA formation. Predictors of late-acquired malapposition include DES implantation in acute coronary syndromes, long lesions, and chronic occlusions.³

In our patient, OCT revealed that all of the struts of both stents were covered by endothelium as well as the existence of adventitial straining. (Figure 2d) Given that the size of the aneurysms did not exceed the penetration ability of OCT (20 μm), the method reported here was chosen for its ability to accurately show neointimal coverage and stent endothelialization, detect aneurysms entry sites and visualize stent apposition in the vascular wall.⁶ However, it cannot be excluded that aneurysmal formation could be from incomplete stent apposition, but there is no clear evidence supporting such a hypothesis.

The formation of multiple aneurysms in 2 different locations in the coronaries following stent implantation is unusual. Although the stent in the RCA was implanted during primary percutaneous coronary intervention, which is a predictor of late-acquired malapposition,³ the lesion in the LAD was selectively stented. As there is no clear etiology, we hypothesize that the combination of physical trauma from oversizing the stents and aggressive post-dilation, with poorly understood biological reactions

following DES implantation, might contribute to our patient's tendency for CAA formation.

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