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CASE REPORT

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Late giant coronary aneurysm associated with a fracture of sirolimus eluting stent: A case report

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KEYWORDS

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Summary A 73-year-old female underwent percutaneous coronary intervention (PCI) because of stable angina. An elective PCI for the RCA lesion was first performed with deploying sirolimus eluting stents (SES). Three weeks later, PCI was also provided in the residual LAD lesion. Eight months later, she presented with new angina. CAG revealed an in-stent restenosis in the mid LAD and a large eccentric saccular coronary aneurysm (17 mm × 9 mm) at the proximal RCA. Intravascular ultrasound (IVUS) showed absence of stent struts around the orifice of aneurysm, which suggested a fracture of SES stent. The entry of coronary aneurysm was finally sealed with a polytetrafluoroethylene-covered stent. This report documented a rare case of late giant coronary artery aneurysm associated with a fracture of SES.

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Introduction

Sirolimus eluting stents (SES) have dramatically reduced the occurrence of angiographic and clinical restenosis compared to bare-metal stents [1]. Despite this overall benefit, clinical restenosis occurs in up to 4–5% of unselected patients who receive SES [2,3], and late stent thrombosis has been reported to occur in about 1.0% of the patients

treated with drug-eluting stents [3,4]. Recently, it has been reported that stent fracture may be one of the causes of in-stent restenosis or stent thrombosis after SES implantation [5–8]. Stent fracture of SES is more likely to occur than that after bare-metal stent implantation [9–11].

The formation of coronary artery aneurysm (CAA) is one of the critical complications after percutaneous coronary intervention (PCI). It has been reported that after PCI, the aneurysm occurs with a frequency of 2–10% [12]. Recently, some cases of development of CAA after implantation of a

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drug-eluting stent have been reported [13–16]. However, the main mechanism of CAA formation after drug-eluting stent is not yet clarified. This case report described a rare case of late giant coronary aneurysm which was probably associated with a fracture of SES.

Case report

A 73-year-old female with a history of hypertension, hypercholesterolemia and diabetes mellitus underwent coronary angiography (CAG), because of stable angina (CCS class III). She received an AAI pacemaker implantation due to sick sinus syndrome ten years ago. Her height was 157.5 cm, and weight 75 kg. Blood pressure was 110/60 mmHg, pulse 50/min, regular. Blood chemistry showed total cholesterol was 188 mg/dl, triglyceride 116 mg/dl,

high-density lipoprotein cholesterol 31 mg/dl, and LDL-C 134 mg/dl, fasting blood sugar 124 mg/dl, HbA1c 5.6% under the medication. Her coronary risk factors were hypertension, hypercholesterolemia, diabetes mellitus and obesity. In electrocardiogram, complete right bundle branch block and abnormal Q waves with T wave inversions were noticed in III, and precordial leads. An echocardiogram revealed left ventricular end-diastolic dimension was 58 mm, ejection fraction 65% and mild left ventricular hypertrophy. The inferior wall of left ventricle showed normal contraction, but hypokinesis of mid-septum and apex wall was noticed. CAG revealed chronic total occlusion at the middle portion of the right coronary artery (RCA) with a diffuse lesion proximal to the occluded segment (Fig. 1A). A severe stenosis at the middle portion of the left anterior descending artery (LAD) was also revealed. There were grade 3 collaterals

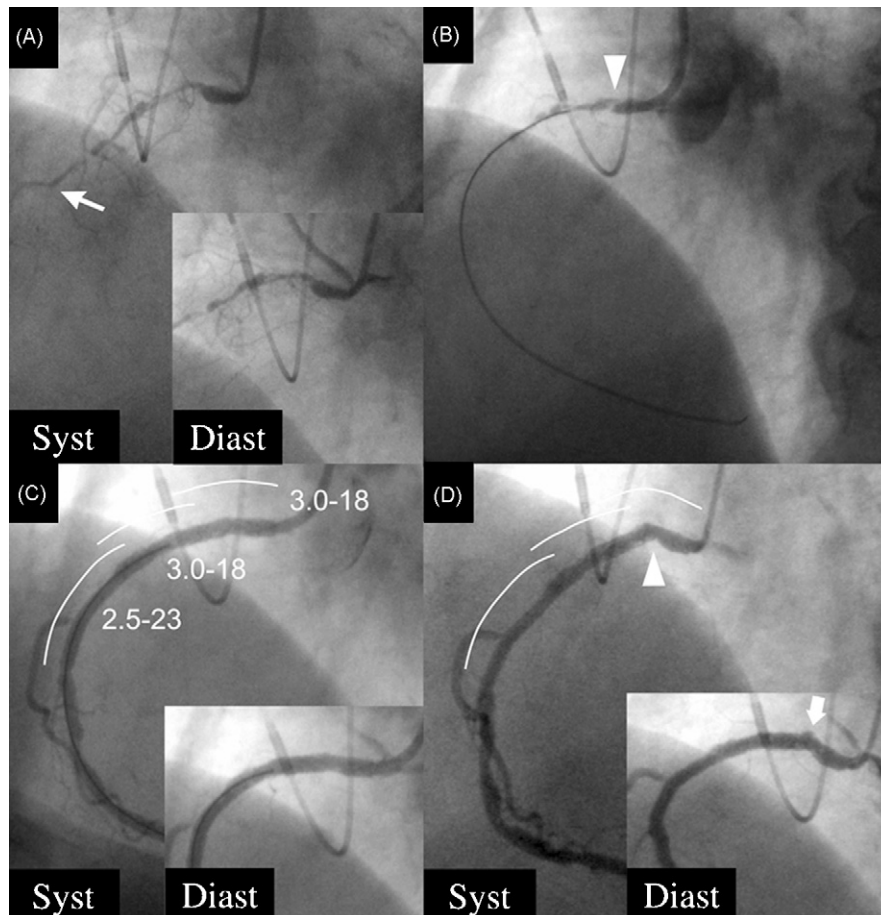


Figure 1 Angiograms of RCA on the first admission. (A) Pre PCI. RCA was totally occluded at the middle portion (arrow), and a diffuse severe stenosis could be seen in the proximal segment. (B) An arterial dissection (arrow) was seen after pre-dilatation. (C) Post PCI. Three Cypher™ stents were deployed from the middle segment to the ostium of RCA having some overlaps between stents. (D) Two weeks later after PCI. RCA angiography showed the pulsatile distortion in geometry of the proximal stent during the heart cycle (triangular arrow). There is a residual dissection (white arrow).

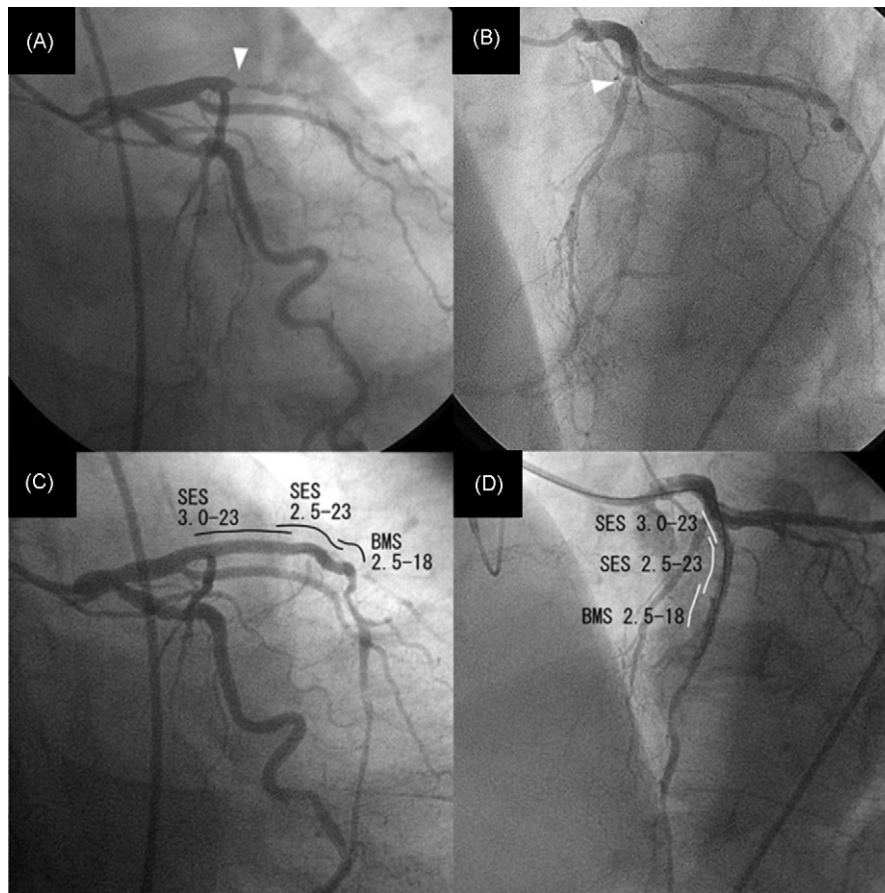


Figure 2 Angiograms of LCA on the first admission. (A and B) Pre PCI. A severe stenosis at the middle portion of LAD (triangular arrow) could be seen. (C and D) Immediately after PCI. Two Cypher™ stents (SES) and a Multi-Link Zeta™ stent (BMS) were deployed.

from the left circumflex artery (LCx) to RCA (Fig. 2A and B).

An elective PCI for the RCA lesion was first performed. A 0.014-in. floppy guide wire and even Miracle 3-gram guide wire (Asahi Intec Corp.) could

not cross the lesion. A stiffer Miracle 6-g guide wire (Asahi Intec Corp.) was then used to cross the lesion. After pre-dilatation using 2.0 mm balloon of this lesion, CAG revealed coronary dissection at the proximal RCA (Fig. 1B). Three SESs (2.5/28 mm,

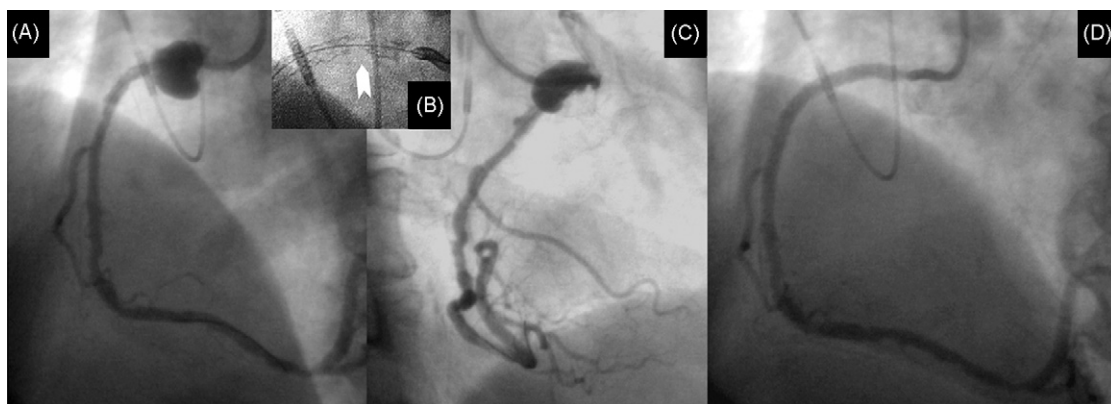


Figure 3 Angiograms of RCA at 8-months follow-up. (A and C) A giant aneurysm formation was revealed at the proximal segment of RCA (size: 17 mm × 9 mm). (B) Stent struts are separated at a distance of about 1 or 2 mm (arrow). (D) A PTFE-covered stent graft of 3.0 mm × 14 mm was implanted to fully cover the aneurysm.

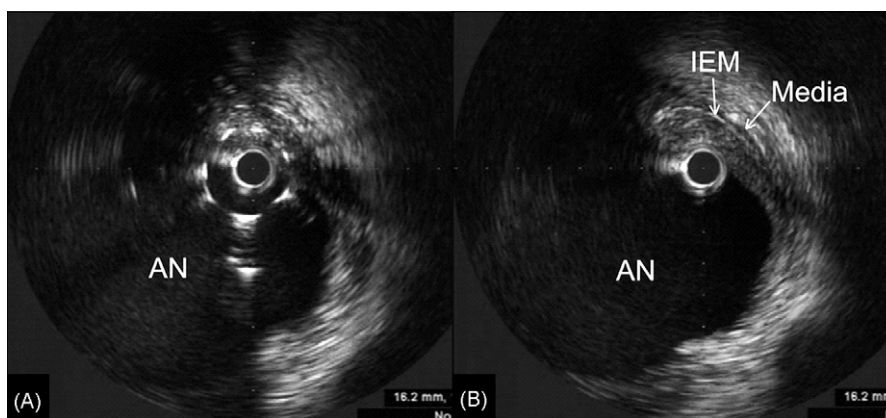


Figure 4 IVUS images using a negative contrast injection at the site of the aneurysm. (A) Stent struts could be visible. An aneurysm was revealed behind the struts (AN). (B) Just proximal to the site of Fig. 4A. Stent struts were absent. The wall of aneurysm did not provide a three-layer appearance, suggesting that this was a pseudo-aneurysm. IEM: internal elastic membrane.

2.5/18 mm and 3.0/18 mm, Cypher™, Johnson & Johnson, Miami, Florida, USA) were then deployed from the distal RCA to the RCA ostium with an overlapping between each stents. Post-dilatation was performed for all of the stents with a 3.0 mm non-compliant balloon up to 20 atm with a good angiographic result (Fig. 1C). Intravascular ultrasound (IVUS) was not performed then. Three weeks later, PCI was also provided in the LAD lesion. Two SESs were deployed in LAD and one bare-metal stent (Multi-Link Zeta™ stent, Guidant Corp., USA.) were used for the bail-out situation to treat the distal-edge dissection. Final angiography showed a good angiographic result (Fig. 2C and D). CAG of RCA at the time revealed no in-stent restenosis but a pulsatile deformation in geometry of the SES (Fig. 1D). She had no angina after the intervention, and was discharged with an administration of 100 mg aspirin and 200 mg ticlopidine.

Eight months later after the PCIs, she was admitted to our hospital with new angina. CAG revealed a focal and tight in-stent restenosis (90% stenosis) at the site of bare-metal stent in the middle portion of LAD. CAG also revealed a large eccentric saccular coronary aneurysm (17 mm × 9 mm) at the proximal RCA (Fig. 3A–C). The middle portion of LAD was retreated with a balloon dilatation, which led to a good angiographic result. IVUS showed absence of stent struts around the orifice of aneurysm of RCA, which suggested a fracture of SES (Fig. 4). This segment was corresponded to a portion that showed a marked pulsatile deformation at the time of the deployment of stent. Significant in-stent neointimal hyperplasia could not be observed in any part of the three SESs. It was suggested that the aneurysm might be a pseudoaneurysm without any thrombus formation, because IVUS did not show a three-layer

appearance of arterial wall. Considering the potential risk of coronary rupture, the entry of coronary aneurysm was finally sealed with a polytetrafluoroethylene (PTFE)-covered stent (Fig. 3D).

Discussion

This is the first case report of a late giant coronary aneurysm associated with a fracture of sirolimus eluting stent, which was clearly depicted by both CAG and IVUS. Although the mechanism of aneurysmal formation is still unclear, the stent fracture might be the main cause for this aneurysm through a plaque dissection (pseudoaneurysm) in this case.

The actual incidence of coronary arterial aneurysm (CAA) formation after SES implantation is not yet thoroughly surveyed. Reported incidence of CAA ranges from 3.9% to 5% after balloon angioplasty [17,18] and up to 10% after directional coronary atherectomy [19]. According to the study by Slota et al., the incidence of CAA was 3.9% in the bare-metal stent group at 6-month angiographic follow-up and 7% in the balloon angioplasty group [20].

It has been described that the cause of new CAA formation after PCI may be an excessive use of oversized balloons or high-pressure inflation of the balloon resulting in intimal and medial tearing with continuous weakening and stretching of the artery [21–23]. These mechanical consequences may finally lead to the perforation of the media without penetration of blood through the adventitia. In this case, wall dissections were identified both RCA and LAD during each procedure. Both lesions were diffuse and calcified. Immediately after SES deployment at the LAD, a minor dissection

occurred at the distal edge of the stent probably due to size mismatch with distal vessel size, which was bailed out using the bare-metal stent. In contrast, a coronary dissection at the proximal RCA after pre-dilatation might provide an initial trigger of this aneurysmal formation. Post-dilatation with high-pressure balloon inflations was used to optimize the expansion of SESs, and then the dissection was sealed off angiographically.

One of the remarkable features revealed in this case was the SES fracture demonstrated by IVUS. IVUS also showed absence of stent struts around the aneurysm. It has been documented that stent fracture may occur in 1.9–2.6% of cases with drug-eluting stents, and that the in-stent restenosis at the fractured lesion is up to 70% [6,7]. However, the incidence of stent fracture may be underestimated, because not all of the stented patients underwent the follow-up angiography and IVUS. Sianos et al. [9] have described that in tortuous vessels or calcified lesions longer stents with a high pressure are likely to be used, which then increase the possibility of stent fracture. In this case, calcification could be frequently observed around the aneurysm. Repeated excessive movement at the site of maximum bending of the stent might also provoke stent fracture through extreme flexion of the vessel, excessive strut stretching, compression, torsion, kinking, elongation, and overloading of shear stress [9,10,24]. Actually, the fracture in this case occurred at the maximum bending and flexing site. It can be speculated that the edges of fractured stent strut might injure the arterial wall. Moreover, it is also possible that pulsatile stress concentration to the dissected media might break down the structure of arterial wall mechanically.

Another possible mechanism may be related to the characteristic property of SES materials. SES consists of three components that are stainless steel metal, the polymer and the drug. Recently, Gupta et al. reported two cases of CAA after SES implantation, which needed a surgical repair [15]. The pathological findings in both cases showed an inflammation at the site of aneurysm formation. Virmani et al. reported that localized inflammation by polymer fragments of SES was speculated as a cause of late stent thrombosis [25]. Inflammations against the polymer may be one of the causes of CAA formation after SES implantation. On the other hand, anti-healing mechanism by the eluted drugs might relate to CAA formation. Rab et al. [26] reported CAA in 32% of patients who received glucocorticoid following bailout stenting. They suggested that steroid-mediated impairment of vascular healing might have led to weakening of the arterial wall and aneurysm formation. The inhi-

bition of the healing process by sirolimus, which has anti-inflammatory properties, could have contributed to the expansion of the weakened parts of vessel wall due to the trauma of PCI. Therefore, arterial wall stress by the edge of fractured stent strut coupled with an inflammatory response set up by the polymer/drug combination, might have reduced the arterial wall tonus and developed CAA [15].

The long-term prognosis of balloon angioplasty induced CAA was as well as spontaneous CAA [17]. However, the appropriate management of CAA after SES implantation is unknown. The PCI using PTFE- or autologous venous covered stent, the surgical repair with CABG or conservative management under continuing dual-antiplatelet therapy were considerable strategy. PTFE-covered stent might be more convenient than autologous covered stent [12]. From any reasons, CAA can lead to serious sudden cardiovascular events such as thrombosis, embolization and rupture [27,28], which would be fatal. However, in this case, CAA was successfully sealed with PTFE-covered stent. Such types of CAA which has a wide orifice, a large branch or a difficulty in stent delivery are not favorable for treatment by covered stent. Although Gupta et al. recommended that surgery should be favored because hypersensitivity reaction to the drug maybe persistent even after sealing the aneurysm [15], it was controversial. Alternatively, if CAA is small and maybe has a lower risk for rupture, conservative management may be more appropriate. It is considered that multi-detector row computed tomography is useful in assessing the size of CAA. This case therefore might warn that the patients stented with SESs should be meticulously followed even if they are asymptomatic.

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