Coronary Artery Aneurysm Caused by a Stent Fracture

A Case Report and Management Overview

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Summary

Coronary stent fracture (SF) is rare as a complication of percutaneous coronary intervention (PCI), and its adverse events are increasingly being recognized with the development in devices of PCI. The major adverse events caused by SFs are in-stent restenosis due to neointimal overgrowth caused by poor drug delivery.^{1,2)} A coronary artery aneurysm (CAA) is a rare complication of SF, but may lead to lethal events such as acute coronary syndrome or rupture of the CAA further leading to cardiac tamponade.³⁻⁵⁾ However, the management of CAAs is controversial with or without SF.⁶⁾ Herein, we report a case of a CAA caused by an SF and discuss the management of CAA complicated with SF, along with a literature review. We suggest that surgical treatment should be considered the higher-priority strategy in the cases of CAA with SF as compared to CAA without SF.

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Key words: Coronary artery disease, Drug-eluting stent, In-stent restenosis

Although the use of drug-eluting stents (DESs) has significantly improved its long-term outcomes after percutaneous coronary intervention (PCI), in-stent restenosis (ISR) still occurs in some cases and target vascular revascularization is required.^{7,8)} Coronary stent fracture (SF) is increasingly being recognized as one of the causes of ISR.¹⁾ SF can be classified into several types, and complete and severe fracture types rarely lead to coronary artery aneurysm (CAA).^{9,10)} Thus far, the established treatment and management for CAAs with SF are unclear.

Case Report

A 69-year-old man with multiple coronary risk factors, including hypertension, hyperlipidemia, type-2 diabetes mellitus, smoking history, and chronic kidney disease, presented to our hospital for a follow-up coronary angiography (CAG) 6 months after PCI. He had undergone CAG and PCI several times as described below.

In 2003, an electrocardiogram showed ST segment depression, and a cardiac exercise stress test and radioisotope revealed ischemic changes in the anterior and inferior segments of the left ventricle; therefore, CAG was performed for the first time. CAG demonstrated 90% stenosis in the proximal right coronary artery (RCA) and 75% stenosis in the proximal left anterior descending artery (LAD) with mild stenosis in the left circumflex artery (LCX). PCI was successfully performed, and bare-metal stents (BMSs; 4.0 × 13 mm and 3.5 × 20 mm) were im-

planted in the ostium of the RCA and proximal LAD, respectively. Six months later, in 2004, a follow-up CAG revealed no in-stent restenosis (ISR), and dual antiplatelet therapy (DAPT) was changed to single antiplatelet therapy (SAPT). In 2014, the patient was diagnosed with early-stage gastric cancer, and CAG was performed before the gastric cancer surgery. CAG revealed ISR in the RCA and progression of the stenosis in the LCX. A week before the surgery, SAPT was changed to heparin, and DAPT was started after it. Following the surgery, PCI was performed and a zotarolimus-eluting stent (Resolute Integridy® 2.5 × 22 mm) was implanted in the LCX, and a second-generation biolimus-eluting stent (BES; Nobori® 3.5 × 24 mm) was implanted in the ostium of the RCA partially overlapping the first stent. The stent in RCA was dilated with a 3.5 mm high-pressure balloon (inflation pressure, 18 atmospheric pressures). A follow-up CAG was performed 6 months after the second PCI in 2015, and ISR was detected again in the RCA. Therefore, third PCI to RCA was performed with a drug-coated balloon without stent implantation. We dilated the lesion by a 4.0-mm high-pressure balloon up to 18 atmospheric pressures. Final optical coherence tomography showed inadequate stent dilation due to severe calcification, though stent fracture was not observed (Figure A). Several months later, CAG was performed again before surgery for newly detected renal cell carcinoma, and it revealed 100% ISR in the overlapped-stent area in the RCA (Figure B). After the renal surgery, 4th PCI to RCA was performed, and first intravascular ultrasound (IVUS) showed severe stenosis in

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Figure. **A:** Final optical coherence tomography at 3rd percutaneous coronary intervention (PCI) shows inadequate stent dilation due to severe calcification, though stent fracture was not observed. **B:** Coronary angiography (CAG) performed before the last PCI shows complete occlusion in the stent in proximal right coronary artery (RCA). **C:** First intravascular ultrasound (IVUS) at 4th PCI shows severe stenosis in the previous stent. **D:** PCI was successfully performed with the implantation of a third-generation drug eluting stent. Re-canalization was achieved, although adequate dilatation was achieved only partially. **E:** Final IVUS at 4th PCI shows mildly dilated stent, and stent fracture was not observed. **F:** CAG performed after the last PCI shows 99% in-stent restenosis and aneurysm formation distal to the stenosis. **G, H:** Contrast-enhanced coronary computed tomography shows the complete fracture of stent in the RCA, and a coronary artery aneurysm (CAA) around the fracture. **I, J:** Intraoperative photograph and its schematic illustration show CAA of RCA after the plication. The area number 1 means exposed CAA wall and number 2 means arterial wall covered with surrounding tissue after running suture to obtain hemostasis. Dotted lines show RCA under surrounding tissue. RA indicates right atrium.

the previous stent (Figure C). A third-generation sirolimus-eluting stent (SES; Ultimaster[®] 3.5 × 38 mm) was implanted successfully without wiring to the false lumen, followed by a 4.0-mm high-pressure balloon inflation (14 atmospheric pressures), and as a result 3 stents were partially overlapped in the proximal RCA (Figure D). Final IVUS showed a mildly dilated stent, and stent fracture was not observed (Figure E). From the gastric cancer surgery to the 4th PCI to RCA, DAPT was continued, except for a week before the renal cancer surgery. In

this week, heparin was administered instead of DAPT.

At the present admission in 2016, follow-up CAG performed 6 months after the last PCI revealed 99% stenosis in the proximal RCA with aneurysm formation at the level of the area where the 3 stents partially overlapped and 75% stenosis in the proximal LAD (Figure F). Contrast-enhanced coronary computed tomography (CT) demonstrated a complete SF with a diameter of 7 × 13 mm CAA in the area of the fracture (Figure G, H). We considered that PCI was no longer effective, and coronary

Table. Review of Reported Cases of Coronary Aneurysm Complicated with Stent Fracture

Authors	Age	Gender	Type of stent	Fracture type	Vessel	Number of PCI*	Treatment
Kawai Y, <i>et al.</i>	48	male	PES	2 or 3	RCA	1	covered stent
D'Amico M, <i>et al.</i>	49	male	SES	4	RCA	1	CABG
Nakao F, <i>et al.</i>	61	male	PES + SES + EES	unknown	RCA	3	CABG + ligation
Daneault B, <i>et al.</i>	42	female	SES	4	LCX	2	thrombectomy + balloon angioplasty
Kim S, <i>et al.</i>	55	male	BES	4	LAD	1	TAPT
Pontone G, <i>et al.</i>	65	male	Unknown	unknown	RCA	1	CABG + stent removal
Del Trigo M, <i>et al.</i>	72	male	PES	unknown	RCA	1	CABG + stent removal
Sandhu PS, <i>et al.</i>	72	male	SES	4	LCX	2	coiling
Balghith M	unknown	unknown	SES	4	RCA	1	PCI
Loke KS	69	male	DES	3 or 4	LAD	1	debridement + stent removal
Choi EY, <i>et al.</i>	7	male	SES	3 or 4	LITA	1	observation
Manola S, <i>et al.</i>	63	female	BMS + BMS	4	LAD	1	observation
Stribling WK, <i>et al.</i>	83	male	PES	unknown	SVG	1	covered stent
Stambunk K, <i>et al.</i>	62	female	SES + SES	4	LAD	1	covered stent
Kim SH, <i>et al.</i>	55	male	SES	4	RCA	1	CABG + stent removal
Lee KH, <i>et al.</i>	67	female	SES	4	LCX	1	POBA
Choi JH, <i>et al.</i>	73	male	DES	4	RCA	1	CABG + aneurysm removal
Okamura T, <i>et al.</i>	73	male	SES	unknown	LAD	1	covered stent
Aizadehranjbar K, <i>et al.</i>	67	male	SES + SES	3 or 4	LAD	2	CABG
Harish A, <i>et al.</i>	56	male	SES	unknown	LAD	1	observation

PES indicates paclitaxel-eluting stent; SES, sirolimus-eluting stent; EES, everolimus-eluting stent; DES, drug-eluting stent; BES, biolimus-eluting stent; BMS, baremetal stent; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; LITA, left internal thoracic artery; SVG, saphenous vein graft; CABG, coronary artery bypass grafting; TAPT, triple antiplatelet therapy; PCI, percutaneous coronary intervention; and POBA, plain old balloon angioplasty. *number of PCI means number of past PCI to the aneurysm-related lesion before the aneurysm detection.

artery bypass grafting (CABG) was recommended because of two-vessel disease involving the proximal of the LAD with repeated ISR and CAA in the RCA.

At the surgical procedure, the ligation of the proximal and distal end of the aneurysm seemed difficult because the stents were implanted over 50 mm from the ostium, and microvascular development to the aneurysm might occur even after the ligation; therefore, off-pump CABG and wrapping of CAA with an autologous pericardial patch was scheduled. The aneurysmal wall was thick. During the trial to dissect and expose the wall from the surrounding fatty tissue, bleeding from the microvascular injury became difficult to control; therefore, simple plication of wall involving surrounding robust tissue enforced by fibrin glue was performed (Figure I, J). Before the plication, the left internal thoracic artery (LITA) graft was anastomosed to the distal LAD and the saphenous vein graft (SVG) was anastomosed to the distal RCA. After the plication, SVG was anastomosed to the ascending aorta. The surgery was successfully performed with anesthesia time of 371 minutes and surgical procedure time of 263 minutes. The postoperative course was uneventful, and CAG performed before discharge revealed patent bypass grafts and the CAA without leakage; the patient was discharged on the 15th postoperative day.

Discussion

With the development and improvement of coronary stents, particularly second-generation DESs, the long-term outcome after PCI has improved dramatically.^{7,8)} ISR was one of the most common adverse events and causes of

acute coronary syndrome (ACS), and the use of DESs significantly reduced the frequency of ISR and target lesion revascularization as compared to BMSs. Thus, apart from the necessity of prolonged dual antiplatelet therapy, DESs are preferred over BMSs.^{11,12)} However, ISR can still occur even after DES implantation, and the major cause is said to be poor control of coronary risk factors. Other possible mechanisms of ISR are biological factors such as drug resistance and hypersensitivity, technical factors such as stent gap and residual uncovered atherosclerotic plaques, and mechanical factors such as SF.⁷⁾

SF was first reported in 2002, and the incidence of SFs varies between 0.5% and 18.6%. Further, autopsy studies revealed the incidence to be 29%.^{1,13)} The incidence has reduced with the improvements in DES technology from the first-generation to second-generation stents, and the stent design and polymer technology is thought to contribute to the reduced incidence.¹⁾ However, SF sometimes occurs even with the second-generation DESs, and the risk factors include overlapped stents, tortuous or calcified lesions, stenting in the RCA, and aggressive post-dilatation.¹⁴⁾

SFs are classified into 5 types:⁹⁾ Type 1A, single strut fracture; type 1B, more than 2 strut fractures without deformation; type 2, incomplete transverse resulting in a V-shaped horizontal separation of the stent struts without discontinuity at one edge of the strut; type 3, complete transverse without displacement of the two components of the fractured stent over a distance of < 1 mm; and type 4, complete transverse with displacement of the two strut fragments over a distance of > 1 mm. Types 3 and 4 are complete fractures. SF is one of the major causes of ISR,

and target lesion revascularization is required in 24.8% of cases.⁹⁾ Micro SFs like type 1 SFs are subclinical, and patients with these fractures require only careful observation and medication. Clinically important adverse events such as ACS, ISR, and stent thrombosis mostly occur with type 2, 3, and 4 fractures. Complete fractures like type 3 or 4 SFs are risk factors for CAA, like in the case of our patient, although the incidence is quite low.^{9,10)}

CAA is defined as artery dilatation exceeding 50% of the reference vessel diameter; CAA has multiple etiologies and the prevalence ranges from 0.3% to 4.5%.^{6,15)} Atherosclerotic disease is the major cause of CAA, apart from autoimmune responses, such as Kawasaki disease and Behcet's disease, and connective tissue disorders, such as Ehlers-Danlos disease and Marfan syndrome.¹⁶⁻¹⁸⁾ PCI-related CAAs are rare and caused by chemical and mechanical factors.¹⁹⁻²¹⁾ Chemical factors include allergic reaction to metals, polymer-induced hypersensitivity, and infection, and the incidence of CAAs is higher after DES implantation than after BMS implantation. Mechanical factors include residual dissection and deep arterial wall injury due to wire insertion into the false lumen, excessive balloon or stent dilation, and SFs. In our case, the aneurysmal wall observed during the cardiac surgery was thick, and it would be reactive thicken change caused by continuous mechanical stress from the edge of fractured stent. Excessive balloon inflation would be also the cause of CAA. CAA complications, such as thrombosis, distal embolization, ACS, and rupture, can be fatal and therefore appropriate management of CAAs is important.³⁻⁵⁾

The management strategy for CAAs is controversial. Surgical approaches, percutaneous covered-stent implantation, coil embolization, and medications, such as antiplatelet and anticoagulant therapy, are used.^{6,22,23)} However, there are no guidelines for the management of CAAs. Herein, we discuss the management of CAAs by reviewing published reports on CAA with SF, as observed in our patient.

There are only 20 available English-language case reports on CAA with SF^{3-5,24-40)} (Table). In these case reports, the patient ages ranged from 7 to 73 years (mean, 59.9 years), and 15 males and 4 females were included; there was one case where the patient's age and gender were not specified. Partially overlapping stents were used in some cases, and the stent types were as follows: SES (12), paclitaxel-eluting stent (4), BMS (1), everolimus-eluting stent (1), and BES (1). The type of DES used was not specified in two cases and the type of stent was not specified in one case. Fracture types were mentioned only in a few reports, and we estimated the fracture types as far as possible by assessing the images in the report. Majority of the SFs were complete fractures (types 3 and 4). The locations were as follows: RCA (8), LAD (7), LCX (3), LITA (1), and SVG (1). The number of PCIs preformed for the aneurysm-related lesion was 1 in most cases. With regard to the management strategy, surgical treatment and PCI were performed in 8 cases, and 4 cases were managed with observation and triple antiplatelet therapy. Among the cases where surgery was performed, CABG was performed in 8 patients along with the following additional procedures in some cases: stent removal (3),

aneurysm ligation (1), aneurysm removal (1), and no mention of additional procedures (2). Debridement with stent removal without CABG was performed in 1 case. With regard to the PCIs, covered stents were implanted in 4 cases; thrombectomy, coil embolization, and only balloon dilation was performed in a case respectively; and the detailed procedure is not known in 1 case.

The use of first-generation DES is a known risk factor for SF.⁴¹⁾ The fracture types were mainly complete fractures, i.e., types 3 or 4, and this finding is consistent with previous reports.⁹⁾

In cases of micro-aneurysms, i.e., those without dilatation exceeding 50% of the reference vessel or those detected only with intravascular ultrasound or optical coherence tomography, PCI or surgical treatment can be avoided by careful follow-up with imaging modalities such as computed tomography. However, a CAA caused by an SF is most likely formed from disrupted vessel layers, i.e., a pseudo-aneurysm, and thus has a higher risk of rupture.^{3,5,25,27-29,31,35)} Antiplatelet or antithrombotic therapies can reduce thrombosis and distal embolization but cannot reduce the risk of rupture; therefore, aneurysm repair procedures are recommended. PCI, including coil embolization, covered-stent implantation, and CABG along with additional procedures are used.

Although the risk of perforation with coronary wire into the aneurysm is quite low in the absence of SFs, the risk of perforation is high in cases with SFs, especially those with complete fractures (type 3 or 4). Moreover, coil embolization involves a high risk of CAA rupture due to wire insertion into the aneurysm, and covered stent fractures might occur at the level of the previous SF due to the continuous mechanical stress. Therefore, a surgical approach might be more appropriate, although the management strategy chosen depends on the number of ISR-times, stenosis regions, clinical state including age, and the size of the aneurysm. In the cases reviewed, the number of PCIs for the aneurysm-related lesion was one; therefore, the number of prior PCIs may not be so important. Further, the aneurysm sizes were not reported in most cases, and therefore the size criterion for invasive treatment could not be established.

In our case, CABG with aneurysm plication was performed. Previously reported procedures for treating the aneurysm are stent removal; aneurysm ligation; ligation of the native coronary artery, including the aneurysmal lesion; plication; and wrapping. In the case of our patient, stent removal was difficult because the stents were implanted in the ostium of the RCA; therefore, aneurysm wrapping was scheduled. However, bleeding from microvascular connecting to the aneurysm occurred, and plication with fibrin glue reinforcement was performed. CAAs are rare, and their management is challenging because of the absence of treatment guidelines. Because CABG was performed, the flow through the CAA lesion might be less than pre-operation, the lesion might be spontaneously occluded, and careful follow-up by the modality such as CT is important. Herein, we present a case of CAA caused by an SF and attempt to clarify the management strategy by reviewing published literature.

CAA caused by SF is very likely to be a pseudo-

aneurysm and has a higher risk of rupture, and therefore the prevention of rupture is critical. In the case of CAA with SF, which has a higher risk of perforation on wire insertion and covered stent fracture, CABG with additional procedures such as ligation, plication, and wrapping will be the higher-priority strategy as compared to CAA without SF. However, the long-term outcome is unclear and number of same cases is too small. Studies with a larger number of patients will be needed to establish the best approach to managing CAA with SF.

Disclosures

Conflicts of interest: none.

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