Case Report

Acute coronary syndrome secondary to in-stent plaque rupture occurred at 9 years after deployment of bare metal stent

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ABSTRACT

We report a case with acute coronary syndrome due to in-stent plaque rupture that occurred 9 years after bare metal stent deployment. From the results of intravascular imaging and pathological findings, we diagnosed this case as acute coronary syndrome caused by in-stent plaque rupture. In-stent neoatherosclerosis is an important mechanism of very late stent thrombosis, and the intravascular imaging is helpful to identify this and to decide clinical judgment.

Introduction

Recent data have shown that in-stent neoatherosclerosis could develop long after stent implantation and cause subsequent plaque rupture leading to acute coronary syndrome [1–3]. We report a case of acute coronary syndrome associated with in-stent plaque rupture, which occurred 9 years after coronary stenting and was visualized clearly using intravascular ultrasound (IVUS) and optical coherence tomographic (OCT) imaging. We also compared findings of IVUS and OCT with pathological observations of aspirated materials.

Case report

The case concerns a woman in her 80s with hypertension and dyslipidemia. In February 2002, she had undergone emergent percutaneous coronary intervention (PCI) to the right coronary artery (RCA) due to acute myocardial infarction. Bare metal stents (BMS), Multi-Link Tristar (Guidant, Santa Clara, CA, USA) 4.0 mm × 13 mm and S670 3.5 mm × 18 mm, were deployed in segments #1 to #2. In August 2002, she had undergone elective PCI to the left circumflex artery (LCX) due to effort angina. BMS were deployed in 90% stenosis of segment #13. Successful coronary stenting had been performed in both RCA and LCX. Follow-up coronary arteriography (CAG) was performed in March 2003, and there was no in-stent restenosis in either RCA or LCX. Then, she visited our outpatient clinic regularly without any symptoms for about 8 years.

In January 2011, 9 years after the prior coronary stenting, she had sudden chest pain and was transferred to our hospital. On arrival, she still had severe chest pain (10/10). Her pulse rate was 52 bpm, and blood pressure was 202/86 mmHg. There were no rales and no murmur on chest auscultation. Chest X-ray showed cardiomegaly (cardio-thoracic ratio 64%) and slight pulmonary congestion (Fig. 1). Electrocardiogram (ECG) showed ST elevation in leads II, III, and aVF and ST depression in leads I, aVL, V2 through V6. Echocardiographic finding was severe hypokinesis of the inferior wall. Laboratory data on admission showed a slight elevation of high-sensitive troponin I (0.13 ng/mL) with no changes in levels of cardiac enzymes. Emergent CAG revealed total occlusion at two previous bare metal stent-sites in RCA (in S670 stent) and LCX (Fig. 2). We found the haziness of RCA total occlusion site, suggesting the involvement of fresh thrombus. From these findings of ECG, echocardiography, and CAG, we judged that...
the culprit lesion was RCA and decided to perform emergent PCI to RCA. LCX was thought to be chronic total occlusion lesion.

Prior to PCI to RCA, a temporary pacing lead was inserted into the right ventricle as shown in Fig. 3. Because thrombolysis in myocardial infarction (TIMI) II flow was achieved after the guide wire crossed to RCA, examinations with IVUS and OCT were performed. After thrombus aspiration, drug-eluting stent was deployed directly in the previous BMS site. Finally TIMI III flow was
achieved after post-dilatation (Fig. 3). ST elevation in II, III, aVF leads and ST depression in I, aVL, V<sub>4</sub>–V<sub>6</sub> leads observed before PCI was resolved to baseline immediately after PCI. Peak creatinine kinase level was 486 U/L at 7 h after PCI.

Direct comparisons of IVUS and OCT images are shown in Fig. 4. IVUS and OCT were pulled back from the same distal edge of the previously implanted BMS. IVUS showed thickness of low-echoic intima at the distal edge of stent, and OCT showed homogenous high signal structure, which indicated fibrous plaque. After pull back to the proximal region, OCT showed high signal linear structure with backward signal-attenuation, which indicated the cluster of foamy macrophages (Fig. 4A). In more proximal sites, IVUS and OCT showed spotty calcification and thrombus. In Fig. 4B, there were findings of in-stent plaque rupture by IVUS and OCT, and intra-intimal neovascularization by OCT. A lot of thrombus formations within vessel were observed. In more proximal sites, IVUS showed attenuated plaques which indicated pathological necrotic core, and there was lipidic plaque associated with thin cap fibro-atheroma (TCFA) by OCT (Fig. 4C). In the proximal edge of previous BMS, TCFA-like intima was observed by OCT as shown in Fig. 4D. Taken together, IVUS demonstrated thick neointima, spotty calcified plaque, thrombus formation, plaque rupture, and necrotic core in BMS. OCT revealed fibro-fatty intima, cluster of foamy macrophage, neovascularization in neointima, plaque rupture, thrombus formation, and TCFA-like intima. These were findings of in-stent neatherosclerosis and in-stent plaque rupture, and there was no finding of incomplete stent apposition.

Pathological findings of aspirated materials are shown in Fig. 5. Platelet-rich thrombus (Fig. 5A), necrotic core with cholesterol crystals (Fig. 5B), and the cluster of foamy macrophages were observed in hematoxylin–eosin staining (Fig. 5C) and MAC387 anti-macrophage antibody-immunostaining (Fig. 5D). These were typical findings of atherosclerosis and vulnerable plaque, which were consistent with IVUS and OCT images.

**Discussion**

We experienced a case with acute coronary syndrome due to in-stent plaque rupture occurring at 9 years after BMS implantation. To our knowledge, this is the first paper demonstrating the direct comparisons of IVUS and OCT images with pathological findings in the same lesion. In this case, IVUS and OCT imaging showed thrombus formation, TCFA-like intima, plaque rupture, and neovascularization of neointima, which were considered as vulnerable plaque and in-stent neatherosclerosis. The pathological findings of aspirated materials showing platelet-rich thrombus, necrotic core with cholesterol crystals, and cluster of foamy macrophages, which are commonly observed in atherosclerotic lesions, further support our observations with intracoronary imaging modalities. Therefore, we diagnosed this case with acute coronary syndrome caused by in-stent plaque rupture secondary to progression of neatherosclerosis.

It has been reported that the cumulative incidence of very late stent thrombosis (VLST) is 2.0% at 10 years after BMS implantation [1]. VLST of BMS occurs with relatively high frequency at 5–17 years after coronary stenting [2,3]. According to IVUS examination of VLST, incomplete stent apposition is unique in DES-related VLST, whereas disease progression with plaque rupture appears to be relatively common in BMS-related VLST [4]. OCT examination beyond 5 years after coronary stenting shows lipid-laden intima, intimal disruption, neovascularization of neointima, and thrombus formation within BMS [5]. The appearance of fragments of atherosclerotic plaque harvested from patients with BMS-related VLST and those with acute coronary syndrome unrelated to stent thrombosis histologically resemble each other [6–8], as we demonstrated in this case.

In this case, we carefully used the aspiration catheter only in the occluded in-stent segment. Thus, we thought that the aspirated materials were from the occluded in-stent lesion and consistent
Fig. 4. Comparisons of intravascular ultrasound (IVUS) and optical coherence tomographic (OCT) findings. IVUS and OCT were pulled back from the same distal edge of the previously deployed bare metal stents (BMS). Upper images are IVUS and lower panels show OCT images. Thin cap fibro-atheroma (TCFA) indicates 45 μm thickness. (A) OCT showed high signal linear structure with backward signal-attenuation, which indicated the cluster of foamy macrophages. (B) Plaque rupture by IVUS and OCT, and intra-intimal neovascularization by OCT. (C) IVUS showed attenuated plaques which indicated pathological necrotic core, there was lipidic plaque with TCFA by OCT. (D) In the proximal edge of previous BMS, TCFA-like intima was observed by OCT.

Fig. 5. Pathological findings of aspirated materials are shown. Sections A, B, and C were stained with hematoxylin–eosin, and D was CD68-immunostaining. Platelet-rich thrombus (A), necrotic core with cholesterol crystals (B), and the cluster of foamy macrophages were clearly observed (C and D).
with IVUS images. However, of note, we could not completely exclude the possibility that the finding of IVUS or OCT might be different from the aspirated materials.

Conclusions

We experienced a case with acute coronary syndrome due to in-stent plaque rupture occurring 9 years after BMS deployment. From the result of intravascular imaging and pathological findings, we diagnosed this case as acute coronary syndrome caused by in-stent plaque rupture secondary to progression of neoatherosclerosis.

Conflict of interest

None.

References


