In-stent Neoatherosclerosis 10 Years after Bare Metal Stent Implantation Observed by Coronary Angioscopy

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Previous studies showed that intimal atherosclerotic change over stent was found, which is the so-called in-stent neoatherosclerosis. Previous studies with coronary imaging devices such as intravascular ultrasound and optical coherence tomography demonstrated that in-stent neoatherosclerosis was observed several years after stent implantation. However, a few cases of in-stent neoatherosclerosis observed by coronary angioscopy (CAS) have been reported. A 50-year-old male was treated with bare metal stent (BMS) implantation at proximal right coronary artery (RCA) for first acute myocardial infarction (AMI). Ten years later, he suffered from a second AMI. Emergency coronary angiography showed a de novo lesion of severe stenosis at mid RCA and mild restenosis of BMS which had been implanted at proximal RCA 10 years ago. Severe stenosis at mid RCA seemed to be the culprit lesion. At mid RCA of the culprit lesions, CAS showed that red thrombus was observed on yellow plaque without plaque rupture. Intravascular ultrasound (IVUS) showed luminal narrowing with attenuated plaque, with neither plaque rupture nor thrombus. Plaque erosion was the mechanism of occurrence of acute coronary syndrome. At mid and proximal portions in BMS, several yellow plaques were observed whose morphology was complex with irregular surface. Red thrombus stuck on yellow plaque stickily, not sticking out of the lumen. In BMS segment, IVUS showed neointimal proliferation with dissection over BMS, which part was low echoic plaque. CAS revealed yellow plaque and silent stent thrombus in the BMS segment implanted 10 years ago. These findings suggested neoatherosclerosis with high thrombogenity formed in neointima after BMS implantation.

Key words: coronary angioscopy, neoatherosclerosis, bare metal stents
arrival, automated external defibrillator showed ventricular fibrillation. By two times of defibrillation, sinus rhythm was obtained. He was urgently transported to our hospital. On arrival, electrocardiogram showed ST elevation of the inferior leads. Abnormal contraction of the inferior wall was observed by echocardiogram. We diagnosed him as having a re-attack of inferior AMI. Emergent coronary angiography showed a de novo lesion of severe stenosis at mid RCA and mild restenosis of BMS which had been implanted at proximal RCA 10 years ago (Fig. 1). We thought that the severe stenosis at mid RCA was the culprit lesion and performed thrombectomy, which aspirated the white and red thrombus. After thrombectomy, we performed CAS and IVUS. At mid RCA of the culprit lesions, CAS showed that red thrombus was observed on yellow color plaque. We did not find any plaque rupture. IVUS showed luminal narrowing with attenuated plaque. IVUS detected neither plaque rupture nor thrombus. Plaque erosion was the mechanism for the occurrence of acute coronary syndrome. Uchida et al. defined plaque erosion as shallow superficial defect with or without mural thrombus.6)

At distal portion of the BMS, a part of metallic stent was visible although 10 years had passed after stent implantation (b, yellow arrow). Red thrombus was also found. At mid and proximal portions of the BMS, several yellow plaques were observed whose morphology was complex with irregular surface. Red thrombus stuck on yellow plaque stickily, not sticking out of the lumen (c and d, yellow arrow). In BMS segment, IVUS showed neointimal proliferation with dissection over BMS, which part was low echoic plaque, and thrombus was not apparently detected in BMS.

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**Fig. 1** CAS and IVUS findings
CAS showed plaque erosion (a, red arrow), red and white thrombus adhering to yellow color plaque at mid RCA (culprit lesion) (a, yellow arrow). IVUS showed severe stenosis with attenuated plaque at culprit lesion (a). At distal portion of BMS, a part of metallic stent was visible although 10 years had passed after stent implantation (b, yellow arrow). Red thrombus was also found. At mid and proximal portions of the BMS, several yellow plaques were observed whose morphology was complex with irregular surface. Red thrombus stuck on yellow plaque stickily, not sticking out of the lumen (c and d, yellow arrow). In BMS segment, IVUS showed neointimal proliferation with dissection over BMS, which part was low echoic plaque, and thrombus was not apparently detected in BMS.
was added, because angiographic haziness in BMS appeared after percutaneous coronary intervention procedure.

In the present case, CAS revealed yellow plaque and silent stent thrombus in BMS segment implanted 10 years ago. These findings suggested neoatherosclerosis with high thrombogenicity formed in neointima after BMS implantation. Previous angiographic study demonstrated that BMS showed a triphasic luminal response characterized by an early narrowing phase over 6 months, a medium-term regression phase from 6 months to 3 years, and a late narrowing phase beyond 4 years.7,8 In medium-term angioscopic follow-up study, Asakura et al demonstrated that in-stent neointimal thickness in QCA analysis decreased, and nontransparent neointima on angioscopy became partially transparent between 6 months and 3 years.9 A long-term angioscopic follow-up study by Yokoyama et al revealed that a new formation of vulnerable atherosclerotic plaque with thrombus occurred between 6 months and 8 years after BMS implantation.9 One of the possible mechanisms of a medium-term regression was a decrease in cell number and modification of histological composition caused by apoptosis, resulting in changes of neointima such as neointimal remodeling with thinning and transparency. A pathological study demonstrated that prominent infiltration by lipid-laden macrophages into the neointima, and adherent thrombus, were found on disrupted lumen ≥4 years after BMS implantation.10 Nakazawa et al demonstrated that it had taken a duration of more than 4 years to develop neoatherosclerosis after BMS implantation than DES implantation. Neoatherosclerosis after BMS implantation promotes late restenosis and later stent thrombosis.11

At distal portion of BMS, a part of stent struts was transparent, different from majority of stent struts. This mechanism of occurrence of partial transparent struts might be neointimal remodeling with thinning and transparency or a result of rupture of neoatherosclerosis over stent struts.

Disclosure

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References