Letter to the Editor

Neoatherosclerosis 16 years following bare-metal stent implantation: Different tissue components in different underlying lesions observed with optical coherence tomography

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A 61 years old male with a history of multiple treatments of percutaneous coronary intervention (PCI) with bare-metal stents (BMS) was admitted for the treatment of acute coronary syndrome (ACS). Sixteen years prior to this admission when he was 45 years old, he experienced inferior ST-elevation myocardial infarction. He received one Palmaz-Schatz Stent (PS) for the treatment of lesion in distal right coronary artery (RCA). During the hospitalization, he received one PS for the treatment of stable lesion in proximal left anterior descending artery (LAD) (Fig. 1). Routine 3-year follow up angiography revealed no restenosis. The controls of other coronary risk factors were good except for obesity (BMI = 36). Sixteen years after PS implantation, he felt sudden chest pain suggestive of ACS, although there was no significant ST-T change in electrocardiography at rest. The angiography revealed focal severe stenoses in both the PS at the RCA and LAD. Optical coherence tomography (OCT) was performed to decide which lesion was the culprit. OCT findings within both lesion demonstrated intimal hyperplasia over the BMSs. In the RCA stent area. Within the stable neointima, microvessels with tubular structures differentiated from any side branches were observed (Fig. 2, Panel B1, B2). In this LAD lesion, thin cap fibroatheroma was not observed, which is compatible with a stable plaque. PCI was performed for in-stent neatherosclerotic lesions in both distal RCA and proximal LAD with zotarolimus-eluting stents.

In the present case, OCT revealed typical unstable plaque morphology in the RCA in-stent lesion, which was the culprit of acute myocardial infarction treated 16 years prior to the event. Also, OCT found stable plaque morphology in the LAD in-stent segment whose underlying segment was the stable lesion. Therefore, the types of neoatherosclerosis were different according to the underlying plaque, although the BMS implantation was performed 16 years ago.

PCI with stenting is the most widely performed procedure for the treatment of ischemic heart disease, and this device improved the clinical performance as well [1]. In addition to in-stent restenosis (ISR) and late stent thrombosis, neoatherosclerosis within the stent has been recently brought into focus [2,3]. Traditionally, intimal hyperplasia after BMS implantation has been considered stable, with an early peak between 6 months and 1 year at late quiescent period thereafter [4]. However, Kimura et al. demonstrated that late luminal re-narrowing was common beyond 4 years following BMS implantation [5]. Hasegawa et al. reported that atherosclerotic progression occurred in the in-stent intima of BMS more than 5 years after stent deployment based on pathological analysis [6]. OCT has demonstrated its potential capacity to accurately characterize or evaluate the responses after stent deployment. Habara et al. reported that the qualitative OCT findings of BMS restenosis tissue between very-late-ISR and early-ISR are significantly different [6]. Based on their OCT analysis, the restenosis lesions > 5 years after BMS implantation were found with a high incidence (90.7%) accompanied with heterogeneous OCT appearance with low-density area, whereas lesions < 1 year after BMS implantation showed extremely lower incidence (17.9%) of neoatherosclerosis [7]. Nakazawa et al. reported that an underlying unstable lesion, which included ruptured plaque and thin-cap fibroatheroma, is an independent risk factor for neoatherosclerosis [3]. It has been reported that the quality of underlying plaque has influenced the incidence of neatherosclerosis after stenting [3], however, it is not known whether the quality of the underlying plaque affects neoatherosclerotic process. In the present case, based on OCT analysis, the observed neointima demonstrated very similar...
Fig. 1. Coronary intervention 16 years prior to this admission. A: Coronary angiography demonstrated severe stenoses in the distal right coronary artery (RCA) and proximal left anterior descending artery (LAD). B: Staged percutaneous coronary intervention was performed. First, Palmaz-Schatz Stent (PS) was implanted at the distal RCA lesion. After a week, PS was placed at the proximal LAD lesion.

Fig. 2. OCT findings in neoatherosclerosis. OCT findings at the RCA lesion shown in Panel A1 and A2. A1: Disruption of thin fibrous cap with intraluminal thrombus. A2: Diffusely thickened neointima that exhibited lower signal density with attenuation, partially with layered tissue pattern. OCT findings at the LAD lesion shown in Panel B1 and B2. B1, B2: Homogeneous concentric high backscatter structure with calcification in all of the stent area. All of stent struts were covered neointima. Microvessels with tubular structures were observed.
These findings may suggest that neoatherosclerotic progression after stenting is influenced by original atherosclerotic composition.

References