Slow-Flow Phenomenon After Stent Deployment in Lipid Rich Plaque Harboring Cholesterol Crystals

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Figure. (A,B) Two stenotic lesions in the (a,b) proximal and (c,d) middle segments of the right coronary artery (RCA), identified on coronary angiography. (a) Optical coherence tomography (OCT) showing lipid-rich plaque (L) containing cholesterol crystals (arrowheads). Minimum fibrous cap thickness of the proximal lesion was 70 μm, and the largest lipid arc was 360°. Intravascular ultrasound (IVUS) and near-infrared spectroscopy (NIRS) showed ultrasound attenuation (UA) and a nearly entirely circumferential yellow signal. (b) OCT showing another small cholesterol crystal (arrowhead) and rupture (dashed arrow) in the proximal lesion. This lesion was also accompanied by UA and yellow signal on NIRS/IVUS. (c) On OCT, the other lesion in the middle segment of the RCA exhibited a mixed plaque harboring calcific (C) and lipid (L) materials. Minimum fibrous cap thickness of the middle lesion was 94 μm and the largest lipid arc was 158°. The extent of yellow signal on NIRS was smaller compared with the proximal lesion. (d) Ulceration on OCT and IVUS (dashed arrow).
A 81-year-old man with angina pectoris was hospitalized to receive percutaneous coronary intervention (PCI) for 2 significant stenoses at the proximal and the middle segments of the right coronary artery (Figure A, B). Optical coherence tomography (OCT), gray-scale intravascular ultrasound (IVUS) and near-infrared spectroscopy (NIRS) were conducted prior to PCI. At the proximal lesion (Figure a, b), OCT showed ruptured lipid-rich plaque. Furthermore, cholesterol crystals were observed within lipid-rich atheroma with a fibrous cap thickness of 70 μm (Movie S1). Corresponding NIRS/IVUS identified ultrasound attenuation, indicating a high lipid core burden index (LCBI) of 879. The other lesion at the middle segment contained calcific and lipid materials on OCT (Figure c, d) and had an LCBI of 222. Distal protection device was used before stent implantation due to the high LCBI in the proximal lesion. Following the implantation of zotalimus-eluting stent (3.5×38 mm) from the proximal to the middle lesion of the right coronary artery, the patient complained of chest pain, and electrocardiography showed ST-segment elevation in the inferior leads. Coronary angiography confirmed slow-flow phenomenon. Coronary flow was recovered after the collection of the distal protection device and i.c. injection of nitroprusside. Final angiography confirmed an optimal result without delay of coronary flow.

Slow-flow phenomenon is one of the serious complications during PCI, and may lead to worse outcome. Embolization of the lipid content of plaque containing cholesterol crystals is one of the important causes of slow-flow phenomenon. In previous studies analyzing catheter-derived materials from patients who underwent PCI, a higher amount of cholesterol crystals was observed in patients with no-reflow phenomenon or in those with distal embolization. In addition, in an autopsy study of a patient who died from fatal no-reflow phenomenon, extensive cholesterol crystal emboli plugging the distal coronary artery were noted. Given that cholesterol crystals in the circulation have been shown to induce endothelial injury, these observations support the idea of cholesterol crystals as a potential contributor to deterioration of coronary flow after PCI. OCT is a high-resolution intravascular imaging modality that enables visualization of cholesterol crystal in vivo. In the current case, slow-flow phenomenon was observed after stent implantation at the lesion containing cholesterol crystal on OCT. Furthermore, crystalized-cholesterol atheroma had a substantially higher LCBI compared with another lesion without cholesterol crystal, consistent with the histological studies indicating a significant association between the size of the lipid pool and the risk of cholesterol crystallization. Given that high LCBI has been shown to predict periprocedural myocardial infarction after PCI, intravascular imaging in the present case indicates that cholesterol crystal may be an important contributor to deterioration of coronary flow following PCI. Further investigation is warranted to elucidate the association of cholesterol crystal with slow-flow phenomenon after PCI.

Disclosures

The authors declare no conflict of interest.

References


Supplementary Files

Supplementary File 1

Movie S1. Optical coherence tomography of the right coronary artery.

Please find supplementary file(s): http://dx.doi.org/10.1253/circj.CJ-17-0244