

**Mechanisms of recurrent coronary stenosis identified by optical coherence  
tomography and directional coronary atherectomy**

C-25      Daisuke Isomatsu, Tomoki Kokubun, Sachika Ishii, Shun Watanabe, Himika Oohara,  
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A 66-year-old man with hypertension and a history of smoking was diagnosed with acute coronary syndrome five years ago and underwent percutaneous coronary intervention (PCI) with drug-eluting stent (DES) implantation to #7. Three years ago, he underwent PCI with drug-coated balloon (DCB) for #6. Subsequently, he experienced recurrent restenosis in segment #6 and underwent PCI with DCB again. Two months later, he presented with chest pain and emergent coronary angiography (CAG) showed severe restenosis in #6 and coronary spastic angina (CSA) was suspected, as marked vasodilation was observed after isosorbide dinitrate administration. Optical coherence tomography (OCT) showed a layered plaque with a low-intensity area without attenuation. Directional coronary atherectomy (DCA) followed by DCB was performed. Pathology showed no plaque rupture, thrombus, or microvessels but marked neointimal hyperplasia composed predominantly of spindle-shaped cells. Because CSA was suspected as a trigger, amlodipine 5 mg was added.

Six months after the last PCI, he presented with chest pain again, and CAG showed restenosis in the left anterior descending artery. A DES was implanted to treat the recurrent restenosis after stent-less PCI with DCA and DCB. The patient remained symptom-free at six-month follow-up.

This case illustrates recurrent restenosis after stent-less PCI. The mechanisms were DCB-related neointimal hyperplasia and DCA-induced medial injury. If restenosis recurs after DCA and DCB, especially with medial injury, early restenosis is expected. When stent-less PCI with DCA and DCB involves medial injury, DES implantation is a reasonable option.