Neoatherosclerosis in the Iliac Artery Stent
— Insights From Optical Coherence Tomography and Intravascular Ultrasound —

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Figure. (A) Angiogram of very late in-stent restenosis of the Wallstent in the right iliac artery. (B) Signal-poor region with diffuse border within the neointima on optical coherence tomography (OCT; white arrows), indicating lipid accumulation or fibrin deposition, corresponding to the echoluent area on intravascular ultrasound (IVUS; orange arrows). (C,D) Protruding mass with signal attenuation suggesting red thrombus on OCT (white arrows). IVUS also indicates the presence of thrombus (orange arrows). The deeper neointima exhibits signal attenuation with diffuse border on OCT, indicating the presence of lipid accumulation or fibrin deposition, where a focal potential microvessel (arrowhead) is also observed. (E) Homogeneous, signal-rich neointima on OCT (white arrows) suggesting the presence of dense collagen within the neointima. IVUS shows iso- to low-echoic neointima (orange arrows). (F) Signal-rich confluent region with signal attenuation on the luminal surface on OCT (white arrow) indicating superficial macrophage accumulation within the neo-intima, which could not be detected on IVUS.
There has been increasing attention on neoatherosclerosis in coronary arteries following implantation of bare-metal and drug-eluting stents. Neoatherosclerosis might be a concern even in the field of peripheral artery disease (PAD).

A 75-year-old man with hypertension, diabetes mellitus, dyslipidemia and a history of right iliac artery stenting with a Wallstent 12 years previously, was referred to hospital for treatment of recurrent/progressive intermittent claudication (Rutherford category 3) that had started 6 months earlier. Ankle brachial index (ABI) was 0.51 on the right. Diagnostic angiography showed significant in-stent restenosis in the right iliac artery stent (Figure A). Intravascular optical coherence tomography at the site of in-stent restenosis showed red thrombus, in the area where the underlying neointima contained a signal-poor region with a diffuse border, suggesting lipid accumulation or fibrin deposition (Figure B–D). The corresponding intravascular ultrasound also showed intra-luminal thrombus with underlying low-echoic neointima (Figure B–D). The non-restenotic region showed thin neointimal formation consisting of dense collagen (Figure E), but superficial foamy macrophages were also focally identified (Figure F). This suggests that the very late in-stent restenosis was attributable to in-stent plaque rupture or erosion with thrombus formation secondary to the development of neoatherosclerosis. Primary stenting was successfully performed with the stent-in-stent technique using a nitinol bare-metal self-expanding stent. The symptom resolved, with an increase of ABI to 0.75.

Since the first endovascular stent was implanted approximately 3 decades ago, iliac artery stenting has been recognized as a reliable method for improving the results of angioplasty for iliac artery disease. These intravascular imaging findings, however, suggest that even iliac artery bare-metal stents could incur very late stent failure due to neoatherosclerosis. An increasing awareness of the potential for peripheral neoatherosclerosis is essential for the management of PAD.

**Disclosures**

The authors declare no conflict of interest.

**References**