Neoatherosclerosis After Paclitaxel-Coated Balloon Angioplasty for In-Stent Restenosis

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In-stent neoatherosclerosis (NA) has been reported after drug-eluting stent (DES) and bare-metal stent implantation. NA is not only more frequent but also occurs earlier in patients undergoing DES implantation compared with those treated with bare-metal stents. This phenomenon has major potential implications because complicated NA (mainly from rupture of a thin-cap fibroatheroma) may result in very late stent thrombosis. Although NA can be visualized using intravascular ultrasound (IVUS), optical coherence tomography (OCT), because of its unique resolution (15 μm), appears ideally suited to detect this phenomenon in vivo. We report on a patient treated with a drug-coated balloon (DCB) for recurrent in-stent restenosis (ISR) who at late follow-up developed NA as depicted by OCT and IVUS.

A 52-year-old man required a paclitaxel-eluting stent implantation on the proximal left anterior descending coronary artery 2 years ago for unstable angina. Ten months later, an everolimus-eluting stent was implanted for ISR and, eventually, 4 months later surgery was required for recurrent ISR with a left internal mammary artery sequentially anastomosed to the left anterior descending coronary artery and to a diagonal branch. Presently, he was admitted for unstable angina, and coronary angiography revealed an occluded left internal mammary artery with just a string sign and faint filling of the diagonal branch. A critical ISR of the proximal left anterior descending coronary artery was demonstrated. After lesion predilation, IVUS and OCT revealed the presence of residual neointimal tissue with a classical homogeneous pattern, hyperechogenic on IVUS (Figure 1A) and uniformly bright on OCT (Figure 1B), and moderate DES underexpansion. Subsequently, a DCB (SeQuent Please; B. Braun Melsungen) was used (60 seconds) with a satisfactory final angiographic result. At 9 months follow-up, the patient was completely asymptomatic and the scheduled coronary angiography showed a mild lumen narrowing (30% diameter stenosis) at the same DES site. At this location, IVUS disclosed a hyperechogenic tissue (consistent with a black hole; Figure 2A and 2B) immediately distal to an area of hyperechogenic neointima with a layered pattern (Figure 2C and 2D). OCT revealed that this dark tissue actually had a speckled pattern with brighter tiny spots (Figure 3B and 3C), whereas a glistening bright tissue, with dorsal attenuation and marked shadowing of the stent struts, was depicted at the proximal site (Figure 3D). Minimal lumen area was 3 mm². The remaining DES segments showed just mild uniformly bright neointimal proliferation. The patient was discharged on medical therapy, including intensive lipid-lowering treatment.

DCB are highly effective for patients presenting with ISR. In this setting, DCB are clearly superior to conventional balloon angioplasty and similar to first-generation DES. DCB elicit a dramatic inhibition of late neointimal response. However, in rare cases, DCB may induce adverse local effects at the vessel wall. Indeed, the occurrence of late acquired malapposition with aneurysm formation on IVUS and a black-hole phenomenon on OCT² have been reported as exceedingly rare incidental findings at late follow-up after DCB treatment. However, to our knowledge, the presence of NA after DCB therapy has not been reported. Notably, in our patient, the initial ISR tissue had a homogeneous bright pattern characteristic of classical neointimal proliferation on IVUS and OCT. However, late after DCB therapy, the novel intrastent tissue had a distinct, markedly heterogeneous pattern. Dark areas mimicking a black-hole phenomenon on IVUS and OCT may correspond to proteoglycan- or fibrin-rich tissue. However, in our patient, OCT revealed that these areas were actually infiltrated with tiny, bright, speckled structures, highly suggestive of macrophage accumulation. Furthermore, a strikingly different tissue was recognized at the proximal site, consisting of a glistening bright neointima with marked shadowing of the underlying stent struts. This pattern is highly suggestive of lipid-laden/infiltrated neointima, the hallmark of NA. Interestingly, the patient was asymptomatic, and these intravascular imaging findings were angiographically silent. Notably, no ruptured plaques or associated luminal thrombi were identified; thus, complicated NA was ruled out. Additional studies are warranted to determine the incidence and the potential long-term clinical implications of NA developing after DCB treatment for ISR.

Disclosures

None.
References


Figure 1. Residual homogeneous neointima showing an echogenic pattern on IVUS (arrows) and a bright uniform appearance on OCT. *denotes wire artifact.

Figure 2. Intravascular ultrasound findings within the stent. A and B, Semilunar, echolucent tissue (arrows; mimicking a black hole) contrasting with the relatively hyperechogenic lumen. C and D, Mild eccentric tissue proliferation displaying an echogenic and layered appearance (arrows).
Figure 3. Optical coherence tomography images. 

A, Mild, concentric, and uniformly bright, neointimal proliferation (notice that the stent struts are clearly depicted along the entire vessel circumference). 

B, Well-delineated, eccentric, dark tissue (arrows). 

C, This relatively dark tissue included a faint speckled pattern with tiny bright spots (yellow arrows) with localized dorsal shadowing.

D, Glistening bright neo-intima progressively fading off into dark areas (+) suggestive of lipid accumulation. Notice that, as a result of severe shadowing, only 2 stent struts (at 6 and 7 o’clock) are visualized. * denotes wire artifact.
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