Drug-eluting stent (DES) thrombosis remains a rare but feared complication in patients undergoing coronary interventions. Mechanical factors (stent underexpansion, malapposition, inflow-outflow disease, residual dissections) have been suggested to play a potential role in this setting.1,2 These predisposing problems can be readily identified with the use of intravascular ultrasound or optical coherence tomography (OCT).1–4 Furthermore, lack of complete endothelialization appears to be implicated in cases with very late DES thrombosis, in which OCT might allow a more precise diagnosis.3,4 We present a patient with very late DES thrombosis in whom OCT unraveled a unique, previously unreported, underlying mechanism.

A 49-year-old man with a large anterior myocardial infarction was referred to our center for rescue angioplasty after failed thrombolysis. Six years previously, he presented with unstable angina and coronary angiography revealed a severe lesion in the proximal left anterior descending coronary artery (LAD), diffuse disease in the diagonal branch, and an occluded left circumflex coronary artery. A DES (Cypher, sirolimus-eluting stent, 3.5×23 mm, 18 bar) was directly implanted in the LAD with excellent results, although mild irregularities, distal to the stented segment, were noticed (Figure 1A). An attempt to recanalize the left circumflex coronary artery was unsuccessful. Nine months later, a scheduled angiographic evaluation confirmed the persistence of the optimal result of the LAD DES and absence of disease progression in other coronary segments.

On admission, the patient had persistent chest pain and the ECG showed V2-V3 ST-segment elevation. A total occlusion of the LAD was visualized on angiography with an image highly suggestive of thrombus precisely located in the mid portion of the DES (Figure 1B). Intracoronary abciximab was administered, and Thrombolysis In Myocardial Infarction (TIMI) 2 coronary flow was obtained after guide wire advancement. OCT imaging (Light Lab Imaging, Inc) revealed a ruptured plaque 10 mm distal to the stent and a large associated red thrombus that retrogradely extended into the proximal DES. The plaque had a large lipid pool and a ruptured thin fibrous cap (Figure 2A and 2B). Interestingly, on repeated OCT imaging, the proximal DES (Figure 2C and 2D) showed an excellent appearance without malapposition, edge-disease/dissection, or residual thrombus and presented a homogeneous pattern of mild neointimal proliferation along its length. A normal vessel wall was visualized between the 2 DES. An optimal deployment of the new DES was also confirmed. Subsequent clinical course was uneventful.

Our findings suggest that rupture of a vulnerable plaque “adjacent” to a previously implanted stent may result in a
unraveled the culprit distal plaque rupture but also the adequate tissue coverage of the proximal DES that showed a reassuring mild, uniform, neointimal hyperplasia. Identification of this rare underlying etiology may have major practical implications. Indeed, treatment of the culprit lesion alone should be indicated in this scenario, simplifying the procedure and avoiding repeated treatment of the previous DES. On OCT, red thrombus induces distal shadowing, which may prevent a comprehensive DES appraisal before intervention. However, the unique spatial resolution provided by this technique (15 µm) might be very useful to identify causative substrates at the DES edges and therefore appears particularly valuable in the clinical decision-making process involved in the treatment of patients with this complication.

Disclosures
None.

References

KEY WORDS: intravascular imaging ■ stent ■ thrombosis

Figure 2. Optical coherence tomography. A, Complicated lesion with associated red thrombus (shadowing) (arrows) in the mid left anterior descending coronary artery. B, Ruptured (arrow) thin cap fibroatheroma (yellow asterisk). C, Healthy coronary segment proximal to the ruptured plaque but immediately distal to the drug-eluting stent (DES). D, Proximal DES showing uniform strut coverage (small arrows) by mild neointimal proliferation. Asterisk indicates wire artifact; plus sign, calcified plaque. A and B are magnified images, compared with C and D.
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