Periprocedural stent thrombosis after percutaneous coronary intervention for a bifurcation lipid-rich plaque

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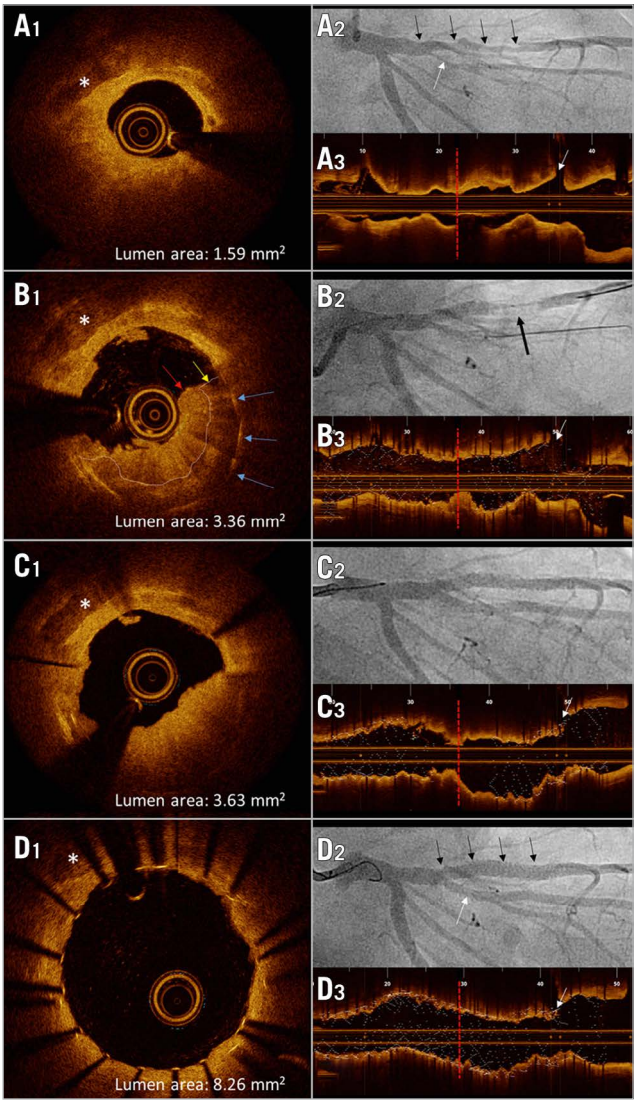

Figure 1. *Stent thrombosis due to atheromatous protrusion after stenting. Left: cross-sectional OCT images (A1-D1; *=calcification) at four stages. Right: corresponding coronary angiographies (A2-D2; black arrows=LAD; white arrows=diagonal branch) and longitudinal OCT views of LAD (A3-D3; red vertical bars indicate site of stent thrombosis and correspond to site of OCT cross-sections; white arrows=diagonal branch). A1-A3. Pre-interventional OCT/angiography: circumferential, predominantly lipid-rich plaque (A1). B1-B3. OCT/angiography of stent thrombosis (arrow in B2): OCT (B1) shows thrombus (red arrow) attached to atheromatous protrusion (yellow arrow), blue arrows=buried stent struts. C1-C3. OCT/angiography two days later: residual stenosis due to protruding mass (C1). D1-D3. Final OCT/angiography: protruding material successfully compressed by additional stent, achieving good lumen area.*

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Preprocedural optical coherence tomography (OCT) of the left anterior descending artery (LAD) in a 58-year-old man with unstable angina due to a stenosis involving the LAD and first diagonal branch detected underlying mixed plaque, partially containing lipid in a circumferential distribution distal to the first diagonal branch **(Figure 1A1-Figure 1A3)**. Bifurcational percutaneous coronary intervention (PCI) with side branch stenting (double-kissing mini-crush: 2.0×18 mm zotarolimus-eluting-stent [ZES] in diagonal branch, 2.25×38 mm ZES in the LAD with post-dilation using 3.0x20 mm and 3.5×8 mm non-compliant balloons) was performed after loading with ticagrelor (180 mg) and cumulative administration of 15,000 IU of heparin to achieve an activated clotting time >250 seconds. At the end of the procedure, acute stent thrombosis (ST) occurred in the LAD stent distal to the bifurcation **(Figure 1B1-Figure 1B3)**. OCT excluded underexpansion or malapposition as possible causes for ST but showed fresh thrombus on top of protruding signal-rich tissue, suggestive of atheromatous material at the site of maximal preprocedural lipid burden **(Figure 1B1)**. Acute ST was treated with post-dilation and administration of a GP IIb/IIIa inhibitor for 48 hours. Planned re-angiography with OCT two days later showed persisting protrusion with a consequently insufficient lumen area of 3.63 mm^2 **(Figure 1C1-Figure 1C3)**. Strategies to treat this finding were discussed. We proceeded with implantation of an additional stent (3.0×12 mm ZES) within the previously implanted stent, which resulted in excellent compression of the protruding material, achieving a lumen area of 8.26 mm^2 **(Figure 1D1-Figure 1D3)**.

OCT-detected irregular protrusion is more likely to be found after stenting of lesions with high lipid content and is an independent predictor for device-oriented clinical endpoints. Strategies to avoid acute ST include early administration of GP IIb/IIIa inhibitors or cangrelor, if bleeding risk allows. Implantation of a second stent to compress the protruding material to prevent further distal embolisation and in-stent restenosis has not yet been described but may be helpful in selected cases with extensive protrusion.

Conflict of interest statement

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