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CARDIOVASCULAR FLASHLIGHT
Cardiac imaging

Very late stent thrombosis caused by the rupture of in-stent neoatherosclerosis 15 years after bare nitinol stent implantation in superficial femoral artery visualized with multimodality imaging

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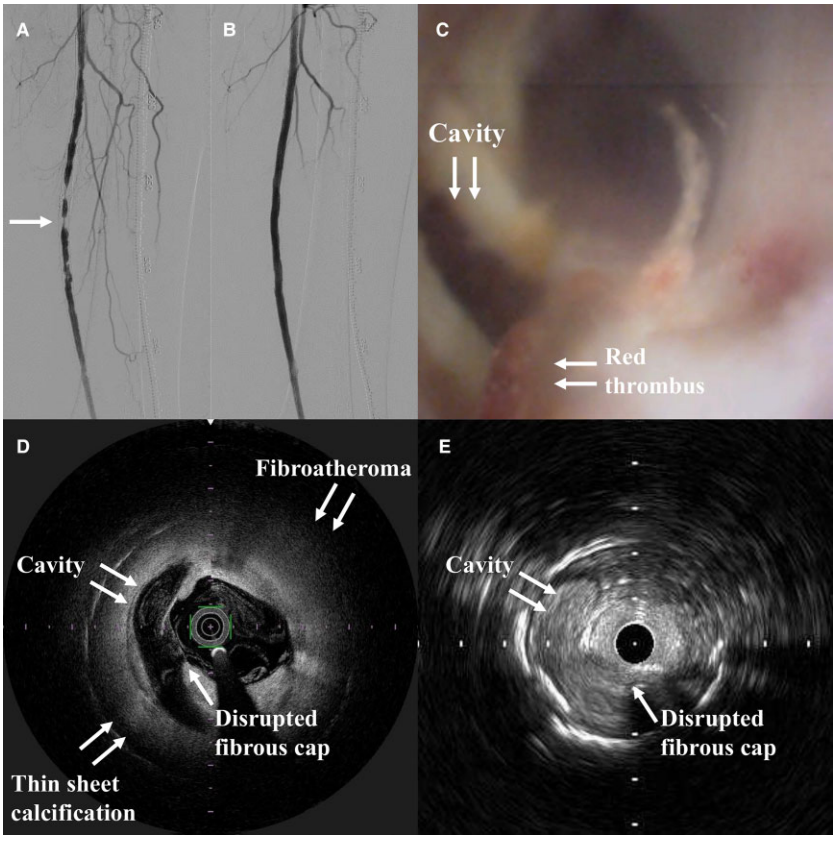

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A 77-year-old man complained of intractable rest pain in his left leg, which occurred 1 week before hospital admission. His past medical history included diabetes mellitus with insulin therapy and history of smoking. He was prescribed cilostazol for treatment of peripheral vascular disease. He underwent endovascular therapy 15 years earlier with a nitinol stent (BNS, S.M.A.R.T ®: Cordis Corp, Santa Clara, CA, USA) implantation in his left superficial femoral artery (SFA), and no restenosis was detected by annual duplex ultrasound follow-up until this session. Duplex ultrasound observed restenosis with 14.8 peak systolic velocity ratio (PSVR), and initial angiography (*Panel A*, white arrow) revealed subtotal occlusion at the stented lesion. Intravascular angioscopy (*Panel C*; Supplementary material, Video S1) demonstrated disrupted cavity with red thrombus attached. Optical frequency domain imaging (*Panel D*, Supplementary material, Video S2) and intravascular ultrasound (*Panel E*) demonstrated fibrous cap disruption with a clear cavity formed inside the plaque (6–11 o'clock in Panel D and E), as well as thin sheet calcification. The other side (11–6 o'clock) shows a signal-rich region with high attenuation that indicates fibroatheroma. The lesion was diagnosed as very late stent thrombosis (VLST) because multimodality images revealed rupture of in-stent neoatherosclerosis with red thrombus attached. After successful vessel preparation with a high-pressure 6 mm balloon (SHIDEN HP, Kaneka Medix Corporation, Osaka, Japan), 6 mm drug-coated balloons (IN.PACT Admiral, Medtronic, Minneapolis, MN, USA) were used. In Japan, stent used was not reimbursed for the treatment of femoropopliteal (FP) in-stent restenosis (ISR), and drug-coated balloon used was the optimal treatment of FP-ISR covered by medical insurance. The completion angiogram revealed an acceptable result with neither residual stenosis nor flow-limiting dissection (*Panel B*). Neoatherosclerosis has been reported as one of the major causes of VLST in the coronary arteries; however, it has rarely been reported in the peripheral arteries.[1] Okuno *et al.* reported histopathological findings with disruption of in-stent neoatherosclerosis from retrieved thrombi in very late intrastent thrombotic occlusion after BNS implantation in the SFA.[2] However, this case illustrates valuable direct visualization with multimodality imaging of a case with VLST caused by rupture of in-stent neoatherosclerosis 15 years after BNS implantation, and the value of multimodality imaging for comprehensive evaluation to diagnose the mechanism of VLST in SFA.

Supplementary material

Supplementary material is available at the *European Heart Journal – Case Reports* online.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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