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IMAGES IN INTERVENTION

Cyphering the Mechanism of Late Failure of Bioresorbable Vascular Scaffolds in Percutaneous Coronary Intervention of the Left Main Coronary Artery

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A 48-year-old woman with history of multiple percutaneous coronary interventions and bypass grafting on the left anterior descending coronary artery presented with unstable angina. Coronary angiography showed a patent left internal mammary artery and de novo critical stenoses of the mid-shaft left main and the unprotected left circumflex coronary artery (LCX). Both lesions were treated with 2 nonoverlapping bioresorbable vascular scaffolds (BVS) (Absorb, Abbott Vascular, Santa Clara, California) 3.5 × 12 mm at 16 atm and 3.0 × 18 mm at 12 atm, respectively. Optical coherence tomography (OCT) confirmed good expansion and apposition of the BVS (**Figure 1**). A short rim of fracture was detected in the proximal portion of the left main BVS, which landed in healthy coronary segments both proximally and distally. No further action was undertaken.

Thirteen months later, the patient presented with unstable angina. The coronary angiography showed a critical focal restenosis of the BVS on the left main. The OCT demonstrated a severe multilayered pattern of neointimal proliferation with multiple areas of light attenuation suggestive for neoatherosclerosis (the largest one presenting with a partially disrupted cap). The previously detected rim of fracture was embedded in the neointimal tissue. After lesion pre-dilation, a zotarolimus-eluting stent 3.5 × 9 mm was implanted in-scaffold with a good final OCT result (**Figure 1**).

Mechanisms of restenosis of metallic stents include underexpansion, geographical missing, fracture, and drug resistance (1). Assuming similar etiologies for BVS, underexpansion and geographical missing were reasonably excluded by OCT at post-implantation (**Figure 1**). A causal role of the observed scaffold fracture (located 2.1 mm proximal to the site of restenosis) or drug resistance cannot be excluded. Also, the presence of neoatherosclerosis with disrupted cap could have played a causing role.

Management of BVS failure is not standardized. Balloon angioplasty or drug-eluting balloon were excluded, given the uncertain consequences inside a BVS with partially lost integrity as a result of the ongoing bioresorption process (2). Therefore, implantation of a metallic drug-eluting stent was preferred. A zotarolimus-eluting stent was chosen because of the previously observed failures of paclitaxel-, sirolimus-, and everolimus-eluting platforms.

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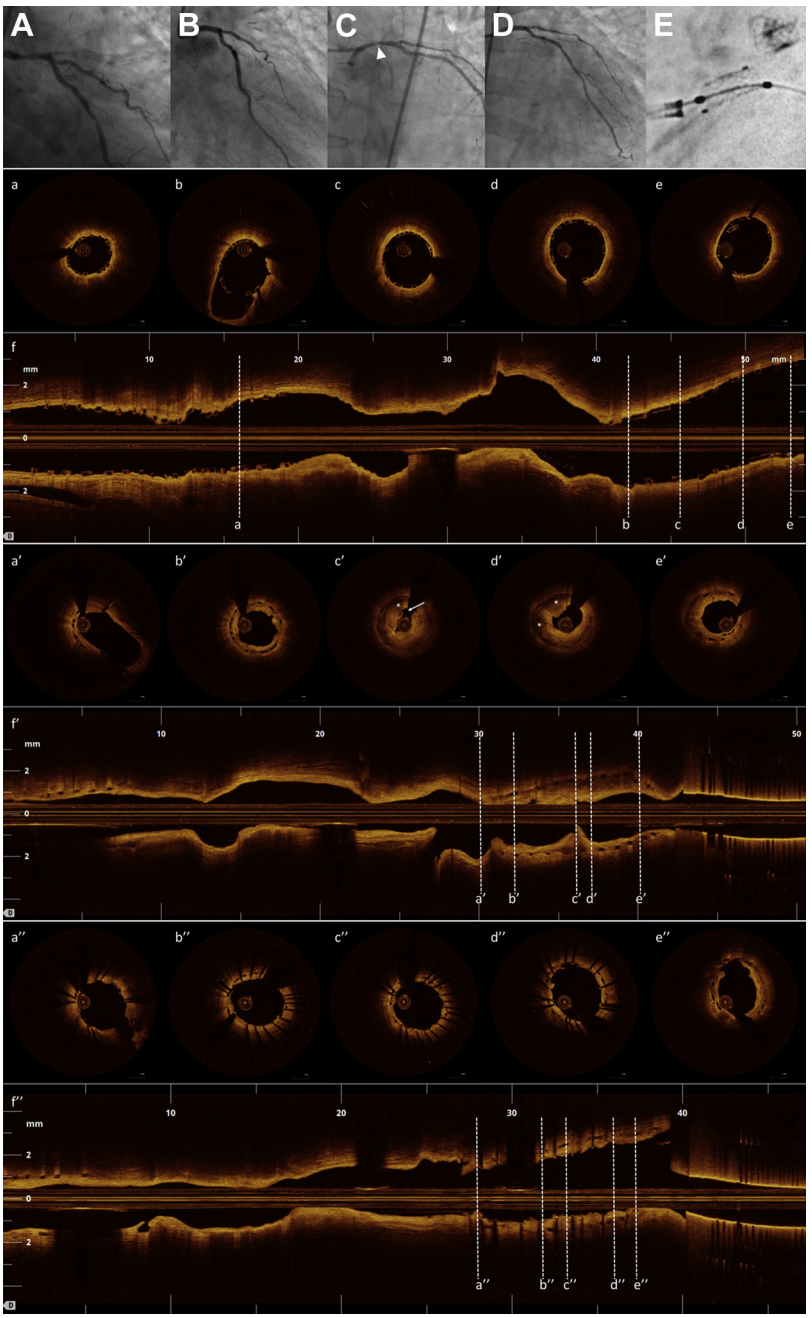
FIGURE 1 Case Illustration



(A to E) Coronary angiograms at baseline **(A)**, post-BVS treatment of the LCX and the left main **(B)**, at follow-up **(C)**, with the **white arrowhead** showing the restenosis site, and post-treatment of the left main BVS restenosis **(D)**, with stent enhancement magnification after in-scaffold stent implantation **(E)**. **(a to e)** Selected OCT cross sections and reference longitudinal view **(f)** post-BVS treatment of the LCX and the left main documenting good expansion and apposition of the scaffolds, with evidence of a short rim of fracture in the proximal portion of the left main BVS **(e)**. **(a′ to e′)** Selected OCT cross sections and reference longitudinal view **(f′)** at follow-up with evidence of severe focal left main in-scaffold restenosis **(c′)** not involving the site of fracture **(e′)**, with multilayered pattern of neointimal proliferation with multiple areas of light attenuation suggestive for neoatherosclerosis **(asterisk)**, the largest with a partially disrupted cap **(white arrow)**. **(a″ to e″)** Selected OCT cross sections and reference longitudinal view **(f″)** after treatment of the BVS failure documenting good expansion and apposition of the newly implanted zotarolimus-eluting stent). BVS = bioresorbable vascular scaffold; LCX = left circumflex coronary artery; OCT = optical coherence tomography.

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