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IMAGES IN INTERVENTION

In-Scaffold Neovascularization 24 Months After Bioresorbable Vascular Scaffold Implantation in a Patient With ST-Segment Elevation Myocardial Infarction

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A 66-year-old man with hypertension, without diabetes, presenting with inferior ST-segment elevation myocardial infarction was treated with uncomplicated primary percutaneous coronary intervention with implantation of 2 overlapping Absorb bioresorbable vascular scaffolds (3.0 × 28 and 3.5 × 20 mm; Abbott Vascular, Abbott Park, Illinois) in the right coronary artery. Good expansion of the device was confirmed by optical coherence tomography (Ilumien, St. Jude Medical, St. Paul, Minnesota) (**Figure 1A**). Two-year clinical follow-up was uneventful. Optical coherence tomography confirmed vessel patency, with the scaffold clearly recognizable (**Figure 1B**). Strut coverage toward the lumen appeared concentric, homogeneous, and signal intense. The tissue around and on the abluminal side of the struts showed focally regions with signal-poor voids that were sharply delineated, could be followed in multiple contiguous frames, and communicated with the lumen (**Figures 1B and 1C**). These features were not apparent at baseline (**Figure 1A**).

Our case illustrates optical coherence tomographic findings consistent with the formation of microvessels (1) within the coverage layer after BVS implantation. This is of note, as neointimal neovascularization has been suggested as a trigger for in-stent neoatherosclerosis, restenosis, and subsequent plaque rupture in metallic stents (2). In contrast, the bioresorbable nature of the scaffold is expected to reduce such complications by preventing rupture and thrombosis due to the formation of a homogenous fibrotic neointimal layer that potentially can shield necrotic plaque components toward the lumen (3). In our patient, there were no clinical sequelae, the vessel was patent, the lumen was well preserved, and there was no restenosis. However, the clear documentation of relatively profuse neovascularization may point toward a need for a better understanding of such phenomenon (e.g., whether this finding represents physiological vessel healing response or, on the contrary, constitutes a sign of accelerated neointimal growth, potentially associated with pronounced inflammatory response, neoatherogenesis, and adverse clinical outcomes). As such, our observation might contribute to the understanding of causes for late scaffold failures (4), although its direct clinical implications have yet to be seen.

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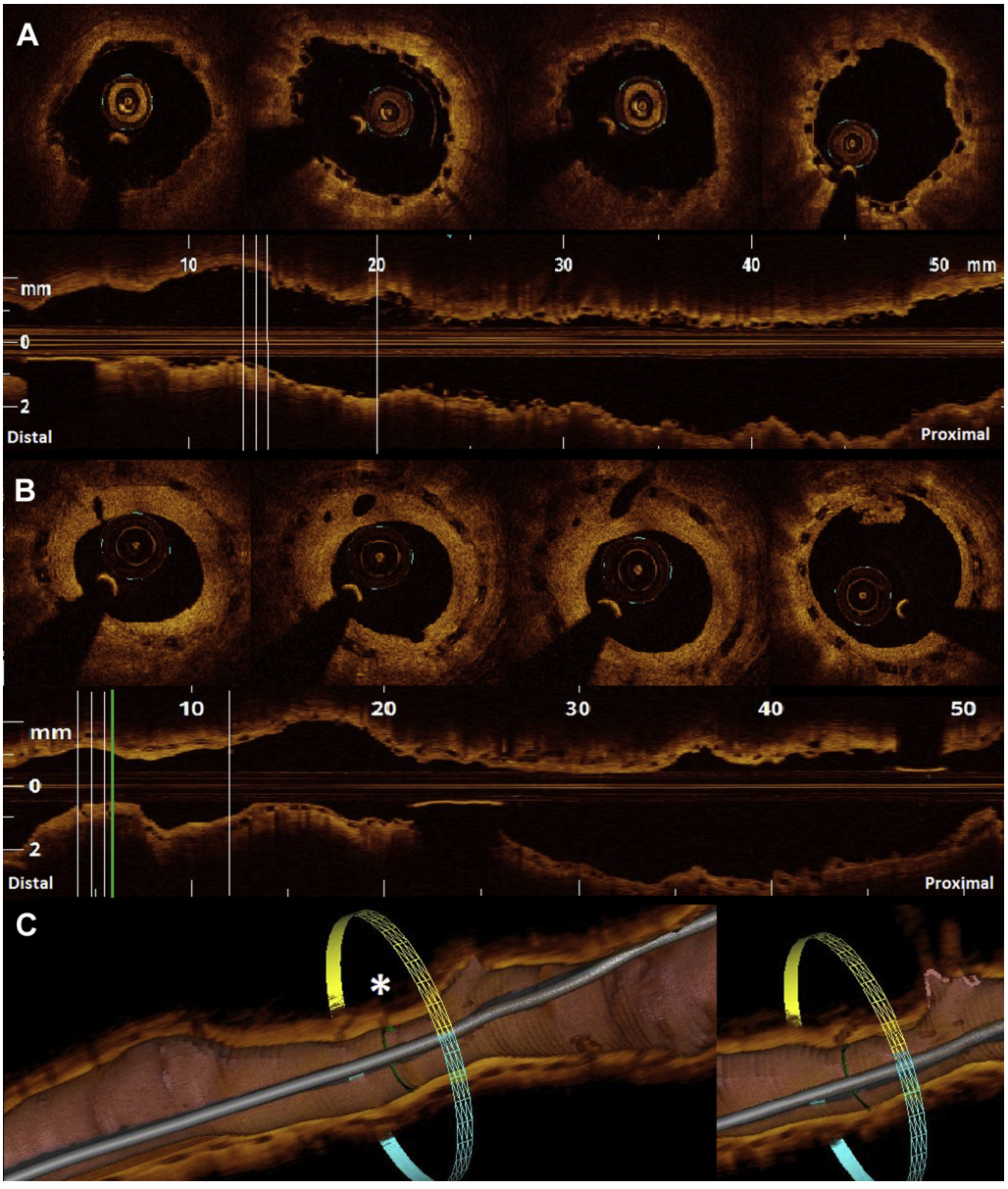
FIGURE 1 OCT Images of In-Scaffold Neovessels



(A) Post-procedural optical coherence tomography (OCT). OCT confirmed strut apposition, with no microvessels present within the non-dissected fibrous plaque covered by the distal scaffold portion. Some minor remnant intraluminal thrombi were observed. **(B)** Scheduled 2-year follow-up OCT. Neovascularization (focal black areas) within the neointima covering the 7-mm-long part of the distal scaffold was visualized. The vertical lines in the longitudinal mode relate to the respective cross sections. In addition, a small intraluminal thrombus was detected. **(C)** Three-dimensional reconstruction demonstrated neovessels with a heterogeneous intramural course, parallel or perpendicular to the lumen, with no clear communication with periadventitial vessels. The **green line** in the longitudinal mode **(B)** illustrates the respective cross section in the vessel reconstruction.

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