Neointimal Proliferation Is Associated With Clinical Restenosis 2 Years After Fully Bioresorbable Vascular Scaffold Implantation

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Drug-eluting stents, introduced to reduce neointimal proliferation and the subsequent clinical restenosis, represent the current mainstay for the treatment of coronary artery stenosis. However, this benefit is paid back with an increased incidence of late stent thrombosis. In addition, the presence of a permanent metallic cage within the arterial wall prevents a full restoration of normal vessel physiology. In the past years, fully bioresorbable vascular scaffolds (BVSs) have emerged as a novel promising approach to treat coronary stenosis by providing transient vessel support with drug delivery capability, without the long-term limitations associated with vessel caging. This technology has the potential to overcome many of the safety concerns associated with drug-eluting stents, with possible clinical benefits.

We report the first comprehensive intracoronary imaging study of late clinically driven BVS restenosis, including 3D optical coherence tomography.

A 72-year-old man with a 70% de novo left circumflex stenosis (Figure 1A, 1C, and 1D) was treated with a 3.0×18 mm BVS (ABSORB, Abbott Laboratories), deployed through a standard stepwise balloon dilation (2 atm every 5° up to a 30° inflation at 16 atm), after predilation with a 3.0×9 mm Maverick balloon (Boston Scientific). A good angiographic result was achieved after postdilation with 3.25×12 mm non-compliant Quantum balloon (Boston Scientific; Figure 1B). At the time of BVS implantation, the patient was on the following pharmacological therapy: (1) aspirin lysine 75 mg QD; (2) clopidogrel sulfate 75 mg/die; (3) bisoprolol 5 mg QD; (4) irbesartan 150 mg QD; (5) fenofibrate 145 mg QD; and (6) omeprazole 20 mg QD. The pharmacological therapy changed following pharmacological therapy: (1) aspirin lysine 75 mg QD; (2) clopidogrel sulfate 75 mg/die; (3) bisoprolol 5 mg QD; (4) irbesartan 150 mg QD; (5) atorvastatin 20 mg QD; and (6) ranitidine 150 mg BID.

Twenty-eight months later, a coronary angiography performed because of a novel effort angina showed a 70% intrascaffold restenosis (Figure 2A and 2B). Intracoronary imaging with 3D optical coherence tomography (Illumien Optis, St. Jude Medical) confirmed a critical restenosis because of extensive finely textured neointimal thickening, with a high backscatter in most areas, whereas a heterogeneous and layered aspect in some areas could suggest ongoing evolution to neointimal hyperplasia (Figure 2C and 2D). Surprisingly, most of the strut remnants still showed the open box appearance (Figure 2C and 2D). However, some struts showed a more advanced degree of degradation (dissolved black box) at the level of major neointimal hyperplasia (Figure 2C–2D). Because the dissolved black box remnants colocalized with the area of maximal neointimal thickening, we cannot exclude that this morphological pattern might be related to the occurrence of restenosis.

Drug-eluting stents were proven highly effective in preventing neointimal proliferation. As an emerging and attractive breakthrough technology, BVS is expected to maintain a similar efficacy toward prevention of restenosis while allowing restoration of vessel physiology. Although a 6% 2-year target lesion revascularization (TLR) rate has been recently reported, no conclusive data on the underlying mechanism(s) are yet available.

To the best of our knowledge, this is the first comprehensive intracoronary imaging report of BVS restenosis, including 3D optical coherence tomography, showing ominous neointimal proliferation causing significant restenosis.

Disclosures

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References


**Key Words:** coronary restenosis • tissue scaffold • tomography, optical coherence

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**Figure 1.** Baseline imaging data. A, Baseline coronary angiography showing a critical stenosis of the left circumflex (LCX). B, Final angiographic results after bioresorbable vascular scaffold (BVS) implantation (the arrows indicate the 2 BVS radio-opaque markers). C, Baseline intravascular ultrasound (IVUS) of the LCX lesion, showing a significant stenosis with an irregular texture, compatible with a fibro-fatty plaque with calcification spots. D, Same lesion visualized in the Virtual Histology (VH) mode. Video clips are available as supplementary on-line material.
Figure 2. Imaging data of bioresorbable vascular scaffold (BVS) after restenosis. A and B, Coronary angiograms at 28 months after BVS implantation, showing a critical restenosis (indicated by the white arrow) within the proximal portion of the previously implanted BVS. C and D, Cross-sectional aspect of the finely textured neointimal thickening, with a high backscatter, responsible for the restenosis (the green line indicates the lumen profile). The magnified boxes with the * symbols show the particular of strut remnants with the open box appearance. The magnified boxes with the # symbols show the particular of strut remnants with the dissolved black box appearance. A video clip of the PCT imaging is available as supplementary on-line material. E, 3D optical coherence tomographic reconstruction of the scaffolded segment shows neointimal proliferation above the BVS struts in the proximal portion of the scaffold (right). F and G, Longitudinal aspect of the restenotic lesion in the lumen profile mode (F) and in the classical L mode (G). It is evident that the reduction of the internal lumen area is related to in-scaffold neointimal proliferation.
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