Five-Year Optical Coherence Tomography Follow-Up of an Everolimus-Eluting Bioresorbable Vascular Scaffold
Changing the Paradigm of Coronary Stenting?

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A 58-year-old man, included in cohort A of the Bioabsorbable Vascular Solutions First in Man Clinical Investigation: A Clinical Evaluation of the Bioabsorbable Vascular Solutions Everolimus Eluting Coronary Stent System in the Treatment of Patients With Single de Novo Native Coronary Artery Lesions (ABSORB) trial, had undergone implantation of a bioresorbable everolimus-eluting scaffold (BVS; Abbott Vascular, Santa Clara, CA) in June 2006 because of stable angina class III with a positive exercise test (Figure 1). He had a positive family history for coronary artery disease. At implantation, his total cholesterol was 207 mg/dL, his low-density lipoprotein level was 130 mg/dL, and his high-density lipoprotein level was 71 mg/dL. Coronary angiography revealed a single lesion at the middle left anterior descending artery that was treated with a 3.0×12 mm BVS, and he was discharged on a treatment regimen of dual antiplatelet therapy (aspirin clopidogrel) for 6 months and atorvastatin 20 mg. His lipid profile 2 years after implantation showed a total cholesterol of 144.3 mg/dL, a low-density lipoprotein level of 65 mg/dL, and a high-density lipoprotein level of 60 mg/dL.

The patient was asymptomatic with a negative exercise test at a 5-year follow-up in March 2012. Repeat coronary angiography as part of a local study protocol did not demon-
strate any significant lesions at the site of the previous BVS implantation in the left anterior descending (Figure 2C and 2D). Optical coherence tomography evaluation of the target segment did not reveal any struts at the vascular wall (Movie I in the online-only Data Supplement). Minimal lumen area inside the scaffold had increased by 22.1% (4.51 to 5.51 mm²) from 6 months to 5 years (5.31 mm² [A] versus 7.45 mm² [D]; 5.09 mm² [C] versus 6.43 mm² [F]). Note the complete disappearance of the scaffold struts at the 5-year follow-up, as well as the regular contour of the lumen as opposed to the 6-month follow-up.

at 5 years that had showed an open box or bright box appearance at 6 months’ follow-up, whereas the thickness of the intimal layer shielding the calcified tissue from the lumen had significantly increased (baseline, 10 μm; 6 months, 260 μm; 5 years, 200 μm) (Figure 4). Using dedicated online software (qAngioOCT, Medis Medical Imaging Systems bv, Leiden, Netherlands), 3-dimensional optical coherence tomography rendering illustrates the patent, smooth lumen surface throughout the treated vessel segment (Figure 5 and Movie II in the online-only Data Supplement).
This is the first human case demonstrating the complete resorption in vivo of a BVS scaffold 5 years after implantation. Using optical coherence tomography, we observed the complete absence of struts in the vascular wall together with an increase of the minimal lumen area by 22.1%. This is in line with previous human studies that have demonstrated the temporal course of the polymer degradation at 6 months and 2 years, as well as an increase in luminal area due to a decrease in plaque size without change in vessel size, whereas studies in a porcine experimental model have demonstrated complete resorption of the polymer 4 years after implantation.

Importantly, we observed in our patient the development of a homogenous, bright, signal-intense layer shielding the persistent signal-poor, sharply delineated calcified plaque toward the lumen. Conceptually, such tissue growth could be protective because it potentially could diminish the risk of plaque erosion or rupture and prevent thrombogenic plaque components (such as calcified nodules, as in our patient, or necrotic core) from coming in direct contact with the blood stream. Of note, this effect on the vascular wall did not come at the cost of lumen narrowing in our patient; luminal dimensions in the target segment increased from 6 months to 5 years. Such potentially favorable tissue response, as witnessed in this case, might hold promise for the future, evoking the utopia of a passivation of potentially unstable plaques by local therapeutic intervention.

Disclosures
None.

References
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