A 83-year-old man included in the ABSORB cohort B trial underwent successful percutaneous coronary intervention of the middle left anterior descending artery with a 3.0×18-mm bioresorbable scaffold (Absorb, Abbott Vascular, CA) that was postdilated with a 3.0-mm noncompliant balloon at 24 atm (Figure 1A and 1B). The 2-dimensional and 3-dimensional (3D) optical coherence tomography (OCT) confirmed the absence of structural discontinuity after the procedure (Figure 2B and Figure 3A’). At 6 months, the planned angiography showed the absence of restenosis but an ectasia in the scaffolded segment (Figure 1C). Intravascular ultrasound revealed a focal vessel and lumen enlargement (17.93 mm² [Δ+20.5%] and 6.99 mm² [Δ+9.6%], respectively, in the matched cross-section analysis; Figure 2C), whereas 3D OCT suggested a deformation of the scaffold in the 2-mm segment of the ectasia (Figure 3B’). At 18 months, the planned multislice computed tomography showed lumen dilatation in the scaffolded segment (Figure 1D). At 2 years, on angiography, the ectatic lesion in the scaffold became aneurysmal (50% increase compared with the adjacent reference vessel; Figure 1E). Intravascular imaging revealed the increase in the vessel area and lumen area (20.90 mm² [Δ+40.5%] on intravascular ultrasound and 10.91 mm² [Δ+35.7%] on OCT, respectively, from baseline; Figure 1E and 1F), whereas 3D OCT showed a focal cleavage of the scaffold rings and a bulge of the vessel in the segment free from the scaffold struts (Figure 3C and C’). Five years after implantation, angiography revealed that the aneurysm was still present but had become smaller compared with the previous time points (Figure 1F).

In general, aneurysm after drug-eluting device implantation is attributed to residual dissection and deep arterial wall injury and to inflammatory and allergic reactions to the drug, polymer, or device such as metal. In rare cases, a fully bioresorbable poly(L-lactide) acid prosthesis can cause inflammation.1 Further insight can be obtained from the 3D reconstructions of the OCT signal (Figure 3A’, 3B’, and 3C’), in which the pattern of the struts can qualitatively outline the time history of the aneurysmal expansion. From implantation to 6 months, the wall distended and displaced the strut pattern without an apparent change in intracrown angulations, indicating a wall distention that occurred while the strut material was still continuous and minimally degraded. Further expansion from 6 to 24 months occurred in part after substantial polymer degradation had already occurred, as evidenced by the widening of intracrown angulations or complete separation of strut segments, indicating that strut migration follows wall migration entirely and continuity of struts has diminished to subpattern levels. Cross-sectional and longitudinal reconstructions of these segments appear to show that, although diameter is substantially distended, the arterial wall thickness over and under the struts is uniform in nature, an appearance inconsistent with severe inflammatory reactions to polymer.

**Disclosures**

Dr Serruys is a member of Advisory Board of Abbott Vascular.

**References**


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Figure 1. Coronary angiography of the left anterior descending artery before (A) and after (B) intervention at baseline. At 6 months, the planned angiography showed an ectasia in the scaffolded segment (C). The planned multislice computed tomography showed lumen dilatation in the scaffolded segment at 18 months (D). Repeat angiography demonstrated that the ectatic lesion in the scaffold became aneurysmal at 2 years (E) and diminished at 5 years (F).
Figure 2. Intravascular ultrasound (IVUS) and optical coherence tomography (OCT) images from matched sites (aneurysm site and near proximal site) after the procedure (A and B), at 6 months (C and D), at 2 years (E and F), and at 5 years (G and H) after scaffold implantation. The white lines in the longitudinal view indicate the sites corresponding to the cross sections of A' to H". Postprocedural OCT showed some malapposed struts but confirmed the absence of structural discontinuity. At 6 months, IVUS revealed a focal vessel and lumen enlargement (C'). IVUS and OCT revealed the increase in the vessel and the lumen area at 2 years (E' and F') and the subsequent decrease in the aneurysm, with the scaffold becoming indiscernible on OCT at 5 years (G" and H""). FA indicates flow area; LA, lumen area; SA, scaffold area; and VA, vessel area.
Figure 3. Three-dimensional (3D) reconstruction of cross-sectional images corresponding to the scaffold segment. A’ through D’ are reconstructed for emphasizing the scaffold structure. 3D optical coherence tomography (OCT) confirmed the absence of structural discontinuity after the procedure. At 6 months, 3D OCT suggested a deformation of the scaffold in the 2-mm segment corresponding to the ectasia (B and B’). At 2 years, 3D OCT showed a focal cleavage of the scaffold rings and a bulge of the vessel in the segment free from the scaffold struts (C and C’). At 5 years, the aneurysm started to reduce, with the scaffold becoming indiscernible on OCT (D and D’). In A’ to D’, the yellow and green dots indicate the proximal and distal radiopaque makers. The blue struts indicate malapposed struts.
Development and Receding of a Coronary Artery Aneurysm After Implantation of a Fully Bioresorbable Scaffold

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