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First Evidence of Complete Resorption 4 Years After Bioresorbable Scaffold Implantation in the Setting of ST-Segment Elevation Myocardial Infarction

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In December 2012, a 76-year-old woman with a history of hypertension underwent coronary angiography for ST-segment elevation myocardial infarction. A subocclusive lesion in the proximal left anterior descending coronary artery (Figure 1A) was pre-dilated with a 2.5 × 15-mm balloon and a 3.0 × 18-mm coronary bioresorbable scaffold (Absorb, Abbott Vascular, Abbott Park, Illinois) was implanted (Figure 1B). Discharge medication included prasugrel 10 mg and aspirin 100 mg.

In December 2013, the patient underwent scheduled control angiography and optical coherence tomography assessment, which demonstrated patency of the scaffold with <10% angiographic restenosis (Figures 1C, 1D, 2A, and 2B), good apposition and expansion of the scaffold struts. On optical coherence tomography imaging, the remnants of the culprit plaque were covered by a 130-μm layer of fibrous neointima (Figure 2A). In the 3-dimensional reconstruction (Figure 2B), the pattern of the scaffold struts was still visible. The maximal dilation to intracoronary acetylcholine and nitroglycerin 200 μg were 7.8% and 9.1%, respectively.

Repeat coronary angiography performed in September 2016 showed the persistence of an optimal result. Quantitative coronary angiography showed evidence of late luminal gain (from 2.3 to 2.78 mm), invasive imaging demonstrated the complete resorption of the scaffold struts ("golden tube") (Figures 1D, 1E, 2C, and 2D), and the plaque presented characteristics of stability, including a 330 μm-thick fibrous cap, no evidence of microvessels or macrophages, and a calcific pattern (Figure 2C). The maximal dilation to intracoronary acetylcholine and nitroglycerin were 6.1% and 7.2%, respectively.

This is, to our knowledge, the first report of a "golden tube" after bioresorbable scaffold implantation for the treatment of an ST-segment elevation myocardial infarction. The persistence of vasomotion and anatomic stabilization of the plaque (already shown at 1 year) (1) support this therapeutic concept.

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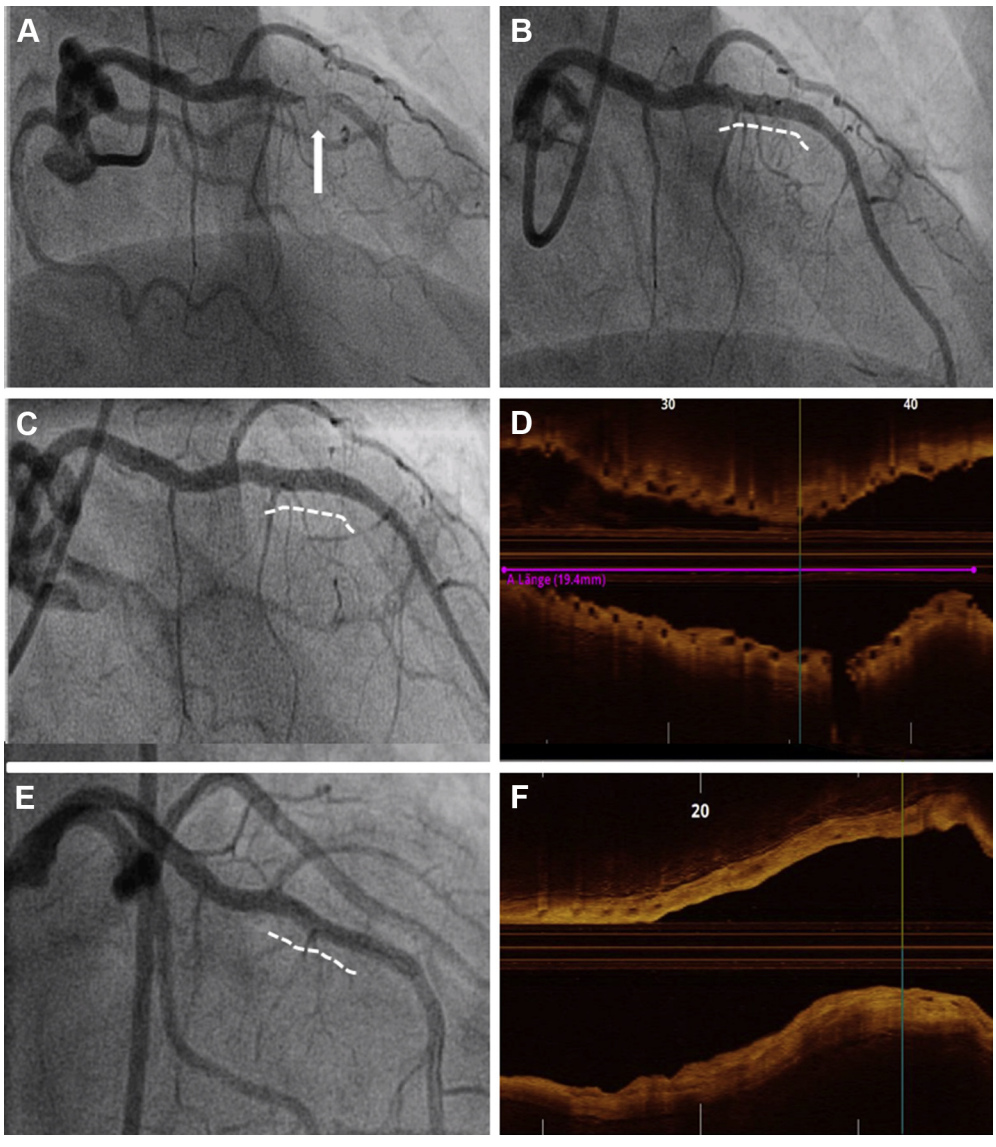
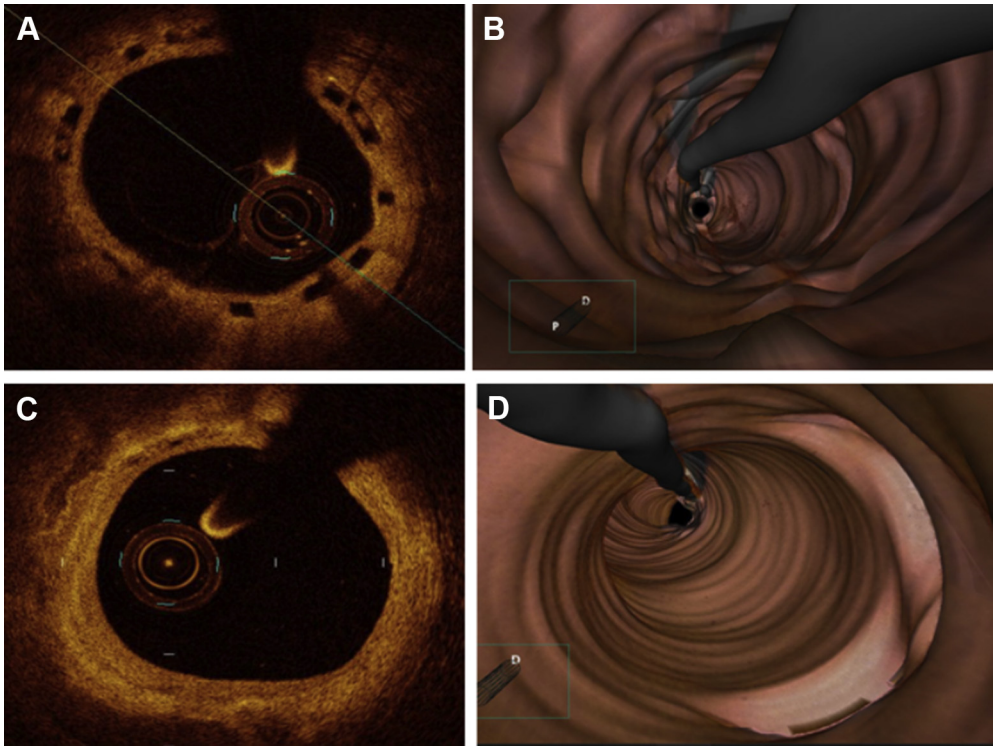
FIGURE 1 **Angiography and Optical Coherence Tomography Images at Index, 1 Year, and 4 Years Follow-Up**



(A) Right cranial view of the proximal left anterior descending coronary artery showing a subocclusive lesion compatible with electrocardiogram evidence of anterior myocardial infarction. **(B)** After implantation of a 3.0 × 18 mm bioresorbable scaffold. **(C)** 12-months follow-up. **(D)** 12-months optical coherence tomography follow-up demonstrating the persistence of the scaffold struts. **(E)** 4-year result at angiography. **(F)** 4-year optical coherence tomography showing complete strut resorption.

FIGURE 2 Optical Coherence Tomography Images



(A) A 1-year optical coherence tomography control. Cross-sectional image confirming good strut apposition and the presence of a neointimal cap (130 μm thick) on the remnants of the culprit plaque. **(B)** Fly-through 3-dimensional reconstruction of optical coherence tomography images. The strut pattern is still visible 12 months after implantation. **(C)** The control at 4 years. On cross-sectional analysis, the plaque seems to have acquired characteristics compatible with calcification (well-defined border, poor attenuation). The neointimal cap increased to 330 μm without lumen loss. **(D)** Three-dimensional reconstruction. The strut pattern has disappeared.

REFERENCE

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