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Neoatherosclerosis as the Cause of Late Failure of a Bioresorbable Vascular Scaffold

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A 48-year-old man who underwent uneventful 3.0 × 28 mm bioresorbable vascular scaffold (BVS) implantation on the mid left anterior descending coronary artery for stable angina, presented 15 months later with unstable angina. Coronary angiography showed a critical in-scaffold restenosis with Thrombolysis In Myocardial Infarction flow grade 2 (**Figures 1A and 1B**). Optical coherence tomography assessment (Ilumien, St. Jude Medical, St. Paul, Minnesota) was performed to elucidate the mechanism of BVS failure, which revealed heterogeneous tissue coverage within the scaffold (**Figure 1C**, cross sections D to G), as follows: 1) neointimal rupture with mural white thrombus at the rupture site (**Figures 1D and 1E**); 2) a low signal-intensity scaffold coverage pattern with dorsal attenuation and marked shadowing of the scaffold struts (**Figures 1E and 1F**); and 3) a normal pattern of neointimal hyperplasia (i.e., high signal intensity) (**Figure 1G**). The minimal lumen area was 1.3 mm^2. An excellent angiographic result was obtained after plain old balloon angioplasty and abciximab infusion.

Metallic stents fail to fully protect the vessel from late plaque progression or neoatherosclerosis. BVS eliminate permanent vessel caging and promote late lumen enlargement; in addition, they are supposed to promote plaque stability and passivation of vulnerable plaques by providing a uniform homogeneous neointimal layer (1). However, BVS have not eliminated the early- and mid-term presence of polymer and antiproliferative drugs, both previously associated with a pro-inflammatory milieu. The time course of neoatherosclerosis in the present case (i.e., 15 months after the index procedure) resembles that of metallic drug-eluting stents (2). Although it might be a rare event, neoatherosclerosis could be considered a potential cause of late failure even with BVS. Replication of this finding in BVS populations is warranted to determine the incidence and the potential long-term clinical implications of neoatherosclerosis in this setting.

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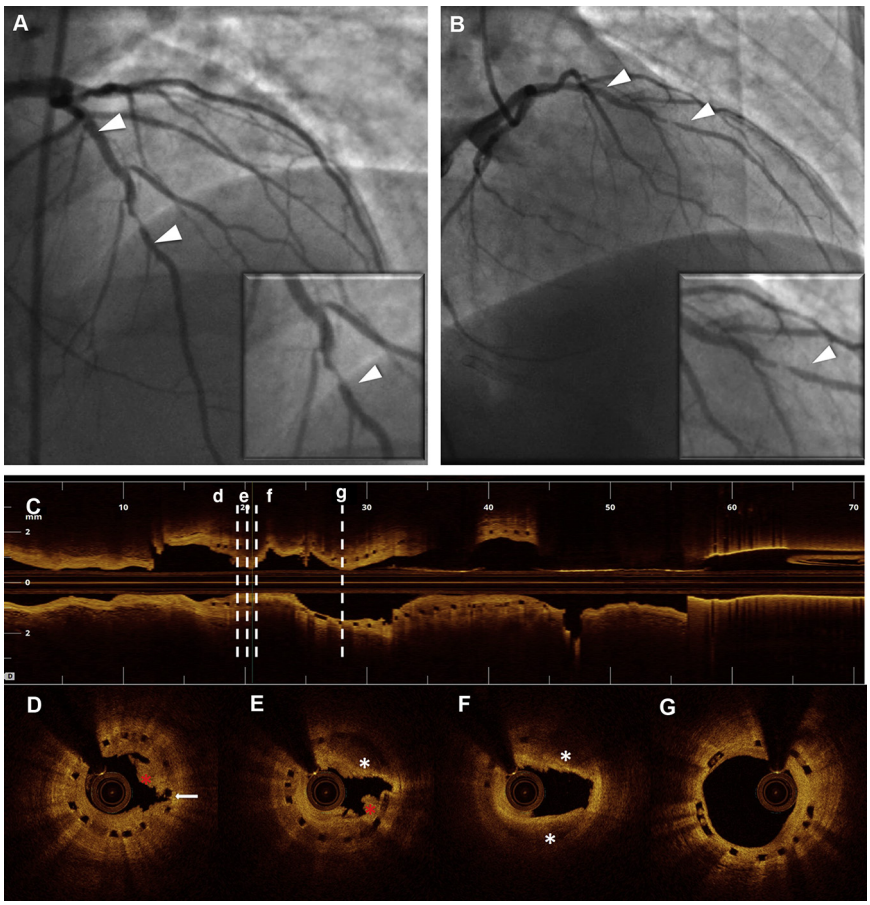
FIGURE 1 Angiograms and OCT Images of Scaffold Failure at 15 Months



(A and B) Angiograms demonstrate focal in-scaffold restenosis. **White arrowheads** show the position of the platinum markers of the bioresorbable vascular scaffold. **(C)** The **dashed lines** in the longitudinal optical coherence tomography (OCT) view correspond to the respective cross sections **(D to G)**. **(D)** Neointimal rupture **(white arrow)** with mural thrombus **(red asterisk)** at the rupture site protruding into the lumen. **(E)** Mural thrombus **(red asterisk)** with highly attenuating area **(white asterisk)**. **(F)** Low signal-intensity scaffold strut coverage pattern with dorsal attenuation **(white asterisks)** and marked shadowing of the scaffold struts (note that only a few struts are depicted). **(G)** Normal pattern of neointimal hyperplasia (i.e., high signal intensity) (note that the scaffold struts are clearly visible along the entire vessel circumference).

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