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Neoatherosclerosis as the Cause of Late Failure of a Bioresorbable Vascular Scaffold at 8 Months

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A 64-year-old man who underwent a successful 3.5 mm × 28 mm bioresorbable vascular scaffold (BVS) implantation on the mid left anterior descending artery for the treatment of stable angina presented with unstable angina 8 months later. Coronary angiography showed a critical in-scaffold restenosis with thrombolysis in myocardial infarction flow grade 2 (**Figures 1A and 1B**). Optical coherence tomography (Ilumien, St. Jude Medical, St. Paul, Minnesota) was performed to elucidate the mechanism of BVS failure. From the distal to mid portions of the BVS, the scaffolds were completely covered with neointima (**Figures 1C and 1D**). However, the proximal portion of the BVS had abundant neointima with a signal-poor region. At the minimal lumen area site, there was a large necrotic core with a fibrous cap thickness of <65 μm, suggesting the presence of thin-cap fibroatheroma (**Figures 1E and 1F**). An excellent angiographic result was obtained after implanting a zotarolimus-eluting stent (Resolute Onyx 4.0 × 18 mm, Medtronic Vascular, Minneapolis, Minnesota).

BVS eliminates permanent vessel caging and promotes late lumen enlargement. In addition, these scaffolds are supposed to promote plaque stabilization by providing a uniform homogeneous neointimal layer (1). Neoatherosclerosis, a time-dependent process, is an important mechanism for late or very late stent failure. Previous data suggested that thin-cap fibroatheroma–containing neointima and in-stent plaque rupture were identified within 2 years following the first-generation drug-eluting stent (DES) implantation and beyond 5 years after bare-metal stent placement (2). In this case, unstable intima was formed much earlier than in previously reported DES cases. Thus, even BVS implantations are not free from early and midterm effects of polymers and the toxic drugs associated with endothelial incompetence and proinflammatory action. Recent studies have reported a higher rate of target lesion failure after BVS implantation compared with the newer-generation DES (3,4). Longer-term follow-up studies are needed to clarify the incidence, time course, and clinical impact of neoatherosclerosis on late BVS failure.

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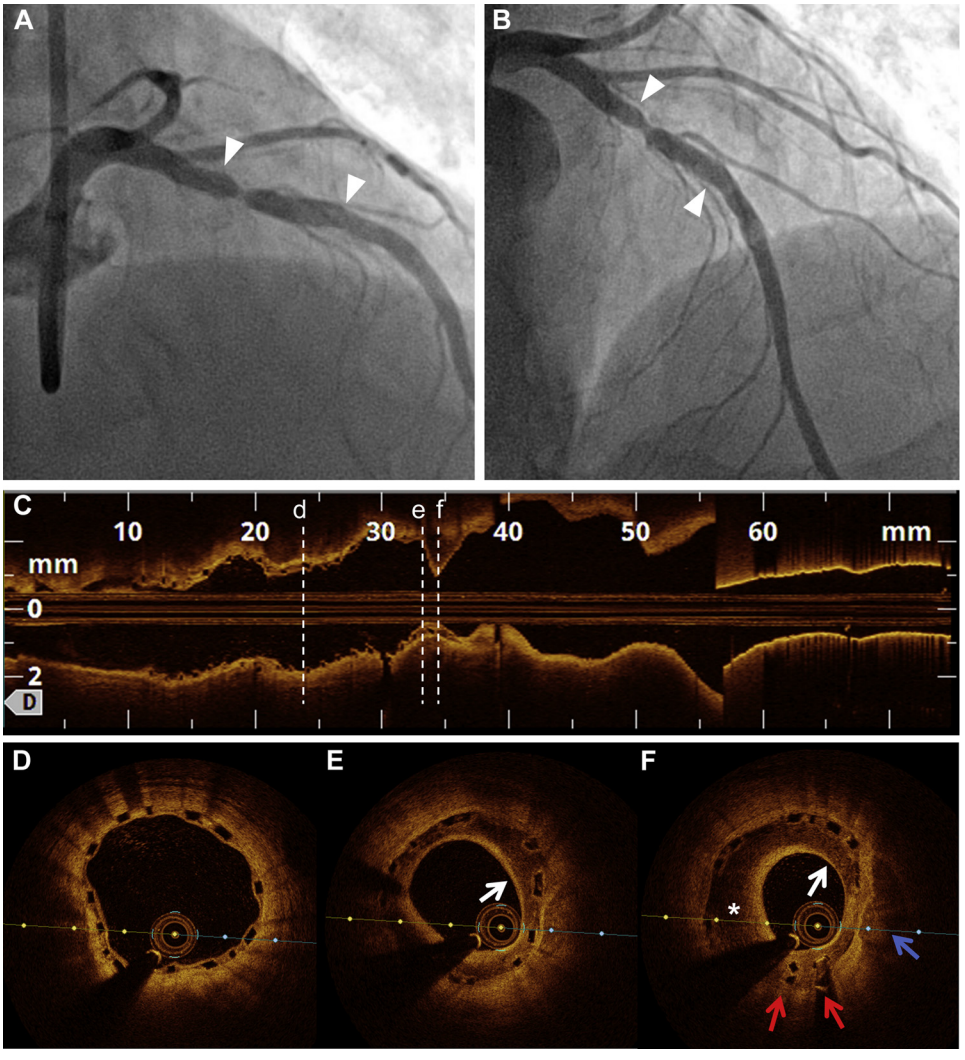
FIGURE 1 Angiograms and Optical Coherence Tomographic Findings



(A, B) Angiograms demonstrating 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 view correspond to the respective cross sections **(d to f)**. **(D)** The scaffolds are completely covered with neointimal tissue. **(E)** In-scaffold tissue contains a signal-poor region with an overlying thin fibrous cap (<65 μm, **white arrow**), which suggests the presence of thin-cap fibroatheroma. **(F)** At the minimal lumen area site, there is a thin-cap fibroatheroma with abundant intima **(asterisk)** and thin fibrous cap **(white arrow)**. At the shoulder of the periscaffold calcium **(blue arrow)**, there are stacked and protruded scaffolds, which is evidence of scaffold disruption **(red arrow)**.

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