



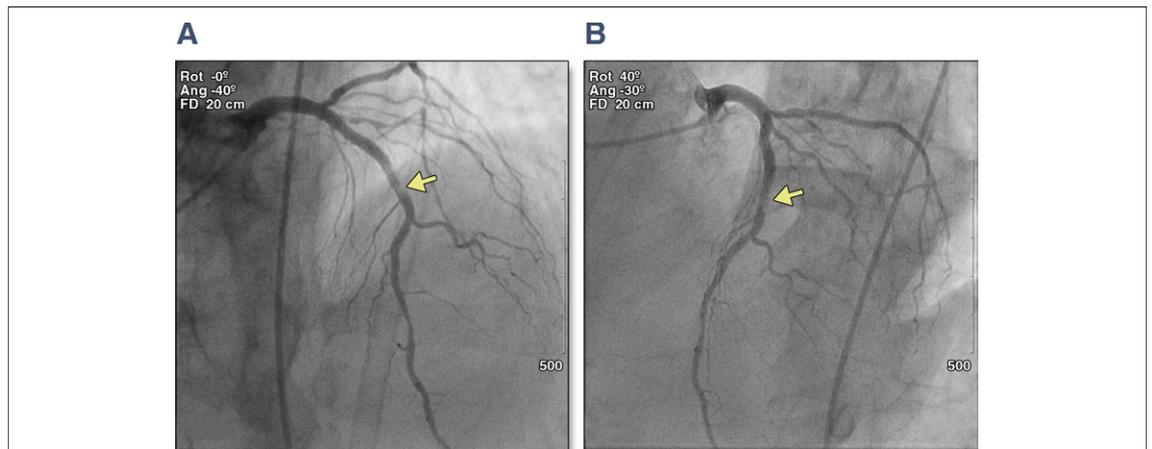
# iPIX

IMAGING VIGNETTE

## Vulnerable Plaque Inside Stent

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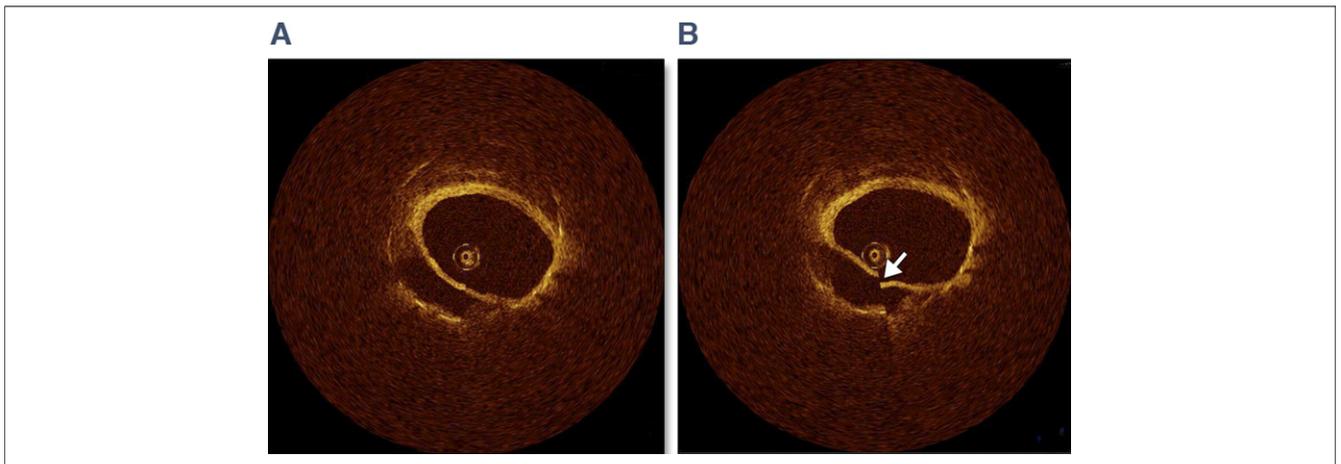
**IT HAS BEEN PROPOSED THAT NONHEALING ENDOTHELIUM** after percutaneous coronary intervention may allow for insudation of lipids superficial to the stent strut level, especially when the underlying plaque carried a large necrotic core. This may result in formation of a lipid-rich neointima, and the lipid core so created is referred to as *nouveau* atherosclerosis; the neointimal lipid accumulation is physically not connected to the original (pre-interventional) lipid core (1). The likelihood of development of *nouveau* atherosclerosis is substantially enhanced in the presence of drug-eluting stents, which further delay the endothelial healing. Furthermore, new atherosclerosis occurs much earlier in drug-eluting stent-based percutaneous coronary intervention compared to bare-metal stent-based interventions. Since endothelial lining is not mature over these atherosclerotic lipid cores, evolving fibrous caps remain predominantly attenuated (1,2). Thin fibrous caps are susceptible to rupture and may contribute to possible late stent thrombosis. Although tacitly believed, an actual plaque rupture has not been documented in such a setting.



**Figure 1. Coronary Angiograms of Left Coronary Artery**

Coronary angiogram of the left coronary artery on the anteroposterior (A) and left anterior oblique (B) view with cranial angulation. A mild focal in-stent restenosis with some intraluminal haziness is visualized in the distal left anterior descending (arrow). Also see Online Video 1.

A 74-year-old man with a history of sirolimus-eluting stent (Cypher, Cordis Corp., Miami Lakes, Florida) placement for an acute myocardial infarction 2 years prior presented to the emergency department with new onset chest pain and diaphoresis. Electrocardiogram showed new anterior wall changes. His serum creatine kinase-MB and Troponin I were elevated at 7.0 ng/ml and 2.39 ng/ml, respectively. He underwent coronary angiography, which revealed a focal hazy area (Fig. 1, white arrow) inside the stent (Online Video 1). Optical coherence tomography (LightLab Imaging, Westford, Massachusetts) of the lesion demonstrated a large, empty space covered by a thin layer (80  $\mu\text{m}$ ) (Fig. 2, Online Video 2). The membrane was disrupted at one focal area (Fig. 2, white arrow) with some residual material inside the space: the features that are similar to “thin cap fibroatheroma.”



**Figure 2. OCT of the Culprit Lesion in the LAD**

Sequential optical coherence tomography (OCT) images of the distal left anterior descending (LAD) in-stent restenosis lesion demonstrate a large empty space with intact layer on the luminal side with high signal inside of the stent (A). The layer becomes thinner proximally and ruptured into the lumen (arrow, B). The morphology is similar to that of ruptured vulnerable plaque. A lipid-rich plaque with a thin fibrous cap has developed inside the stent, which was disrupted with extrusion of thrombogenic substrates leading to occlusive thrombus formation. The “vulnerable plaque” inside a stent may be one possible mechanism for very late stent thrombosis. Also see Online Video 2.

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2. Higo T, Ueda Y, Oyabu J, et al. Atherosclerotic and thrombogenic neointima formed over sirolimus drug-eluting stent: an angioscopic study. *J Am Coll Cardiol Img* 2009;2: 616–24.