

IMAGES IN INTERVENTION

# Erosion of Thin-Cap Fibroatheroma in an Area of Low Endothelial Shear Stress



## Anatomy and Local Hemodynamic Environment Dictate Outcomes

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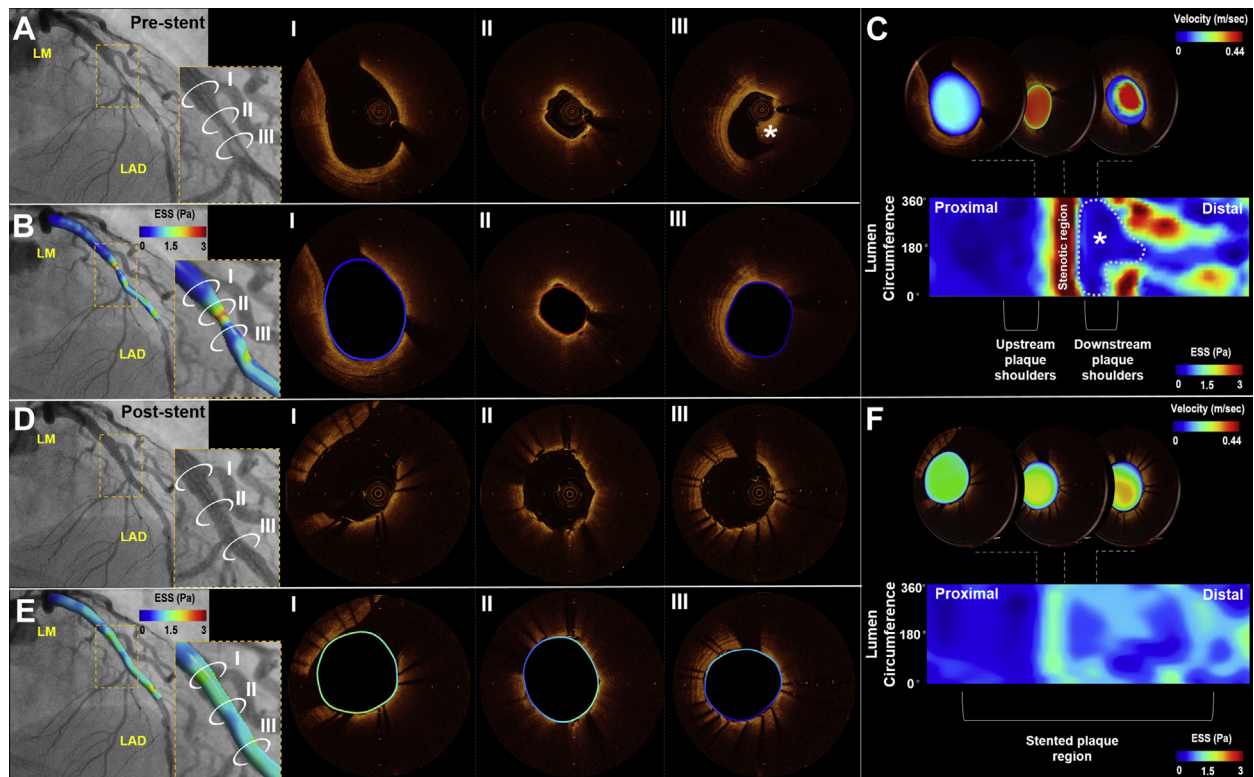
A 52-year-old man was admitted with an anterior ST-segment elevation myocardial infarction for primary percutaneous coronary intervention (Figure 1A). The diagnostic coronary angiogram showed a hazy filling defect in the midsegment of the left anterior descending artery. Optical coherence tomography (OCT) showed a significantly stenotic thin-cap fibroatheroma (TCFA) associated with erosion of the fibrous cap (no sign of rupture) and thrombus formation downstream of the plaque (asterisk in Figure 1A). Blood flow across the lesion was simulated using 3-dimensional OCT and computational fluid dynamics. Pre-erosion flow simulation revealed an area of low endothelial shear stress (ESS) at the upstream plaque shoulder (Figure 1B, section I), high ESS at the neck of the plaque (Figure 1B, section II), and low ESS and blood velocity at the downstream shoulder (Figures 1B, section III and Figure 1C), colocalizing with the site of erosion and thrombus. The lesion was treated with an everolimus-eluting stent (Promus Premier 3.0 × 16 mm, Boston Scientific, Marlborough, Massachusetts) and was further evaluated with OCT (Figure 1D). Post-stenting blood flow simulation demonstrated normal ESS and blood flow patterns across the plaque (Figure 1E, sections I, II, III and Figure 1F).

The patient was discharged on double antiplatelet therapy (aspirin and ticagrelor), statin, angiotensin converting enzyme inhibitor, and beta-blocker.

Erosion commonly occurs over thick-capped fibroatheromas, yet in our case the underlying plaque was a TCFA (1). This case shows evidently a stenotic TCFA, eroded at the downstream plaque shoulder, an area exposed to low ESS, which in turn may lead to endothelial cell apoptosis, the suggested pathobiological mechanism of erosion (2-4). Although management of eroded plaques is not well elucidated, local and systemic therapies are of value. Local therapies with drug-eluting stents aim to alleviate the stenotic burden and normalize the hemodynamic milieu across the plaque, thus minimizing the proinflammatory, prothrombotic, and proapoptotic effects of low ESS. Systemic therapies, primarily with statins, stabilize the highly inflamed coronary artery microenvironment, exerting a beneficial effect in the long term.

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**FIGURE 1** Anatomic and Local Hemodynamic Plaque Assessment

(A) Coronary angiography showing a mid-left anterior descending artery (LAD) stenosis. Optical coherence tomography (OCT) reveals an eroded thin-cap fibroatheroma (I = the upstream plaque shoulder; II = the neck of the plaque; and III = downstream plaque shoulder, erosion site.) (B) Circumferential endothelial shear stress (ESS) distribution in the respective OCT frames. (C) Blood velocity (embedded in the corresponding OCT frames) and ESS distribution (reshaped into a 2-dimensional map) across the lesion. Asterisk depicts erosion and thrombus site. (D) Post-stenting coronary angiogram and OCT frames at the same location (I, II, III). (E) Post-stenting circumferential ESS distribution in the respective OCT frames. (F) Post-stenting blood velocity pattern and ESS 2-dimensional map. (Insets in A, B, D, and E zoom in on the lesion and demonstrate the OCT cross-sectional images location.) LM = left main; Pa = Pascal.

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