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Rupture of a stenotic thin-cap fibroatheroma in an area of low endothelial shear stress

Implication for mechanism of acute coronary syndromes

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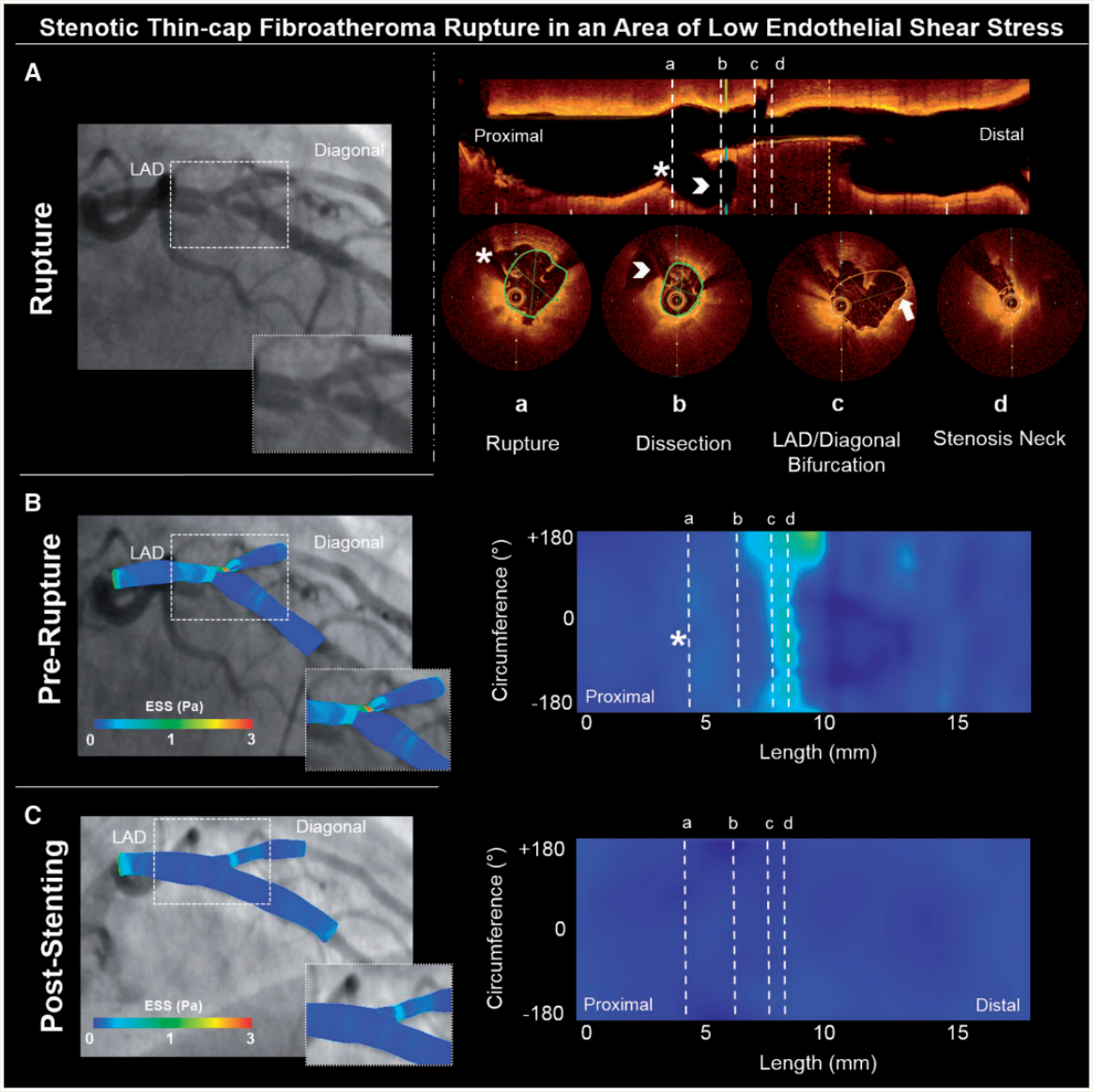
A 56-year-old man with history of hypertension, hyperlipidaemia, smoking, and HIV on highly active antiretroviral therapy was admitted to our hospital with intermediate risk non-ST-elevation myocardial infarction. Urgent coronary angiography revealed a culprit complex bifurcation lesion at the proximal left anterior descending (LAD) artery involving the ostial first diagonal branch (*Panel A*). Optical coherence tomography showed an ulcerated stenotic thin-cap fibroatheroma (TCFA) with presence of thrombus, extending distally as dissection [*Panel A*: rupture site denoted by asterisk (*A*) and dissection (double lumen) by arrowhead (*B*)]. Using 3D quantitative coronary angiography (QCA 3D CAAS 7.5; Pie Medical Imaging BV, Maastricht, The Netherlands), the bifurcation prior to plaque rupture was 3D reconstructed



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and coronary blood flow and local endothelial shear stress (ESS) were calculated using computational fluid dynamics (*Panel B*). Combined plaque morphological and local haemodynamic assessment showed that the rupture of the complex bifurcation lesion occurred at the upstream part, an area exposed to low ESS (*Panel B*, asterisk). The bifurcation stenosis was successfully treated with a 3.5 × 18 mm drug eluting stent following the provisional stenting technique. Flow simulation post-intervention showed normalization of the ESS in the ruptured TCFA area (*Panel C*). The patient was discharged home on antiretroviral therapy, dual-antiplatelet therapy, statin, and β-blocker.

This case provides important advanced morphological and haemodynamic imaging of one of the mechanisms of acute coronary syndrome: TCFAs evolve to stenotic plaques and undergo rupture and precipitation of acute atherothrombosis in the upstream part, an area exposed to low ESS that promotes inflammation and plaque instability.

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