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.
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: TCFA 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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