In-Stent Neoatherosclerosis

A Cause of Late Stent Thrombosis in a Patient With “Full Metal Jacket” 15 Years After Implantation: Insights From Optical Coherence Tomography

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A 62-year-old man presented with inferior ST-segment elevation myocardial infarction for primary percutaneous coronary intervention. He had a history of myocardial infarction 15 years ago and had a bare-metal stent implanted in the right coronary artery. Upon presentation, coronary angiography revealed total occlusion of the right coronary artery, which was covered at its full length with stents (Fig. 1A). We performed thrombus aspiration with partial restoration of antegrade flow (Fig. 1B) and proceeded with optical coherence tomography imaging of the vessel.

Optical coherence tomography (Fig. 2, Online Video 1) revealed a highly heterogeneous tissue coverage, presenting with several features that resemble native atherosclerosis, such as calcific depositions and thin-cap fibroatheroma (1,2). A rupture was detected in the neointima with mural thrombus at the rupture site protruding into the lumen. The neointima had high variability across the stent demonstrating different patterns of coverage (3), as well as a peristrut low-intensity area, reported to correspond to areas of fibrin accumulation (4). Although, there have been pathological reports demonstrating growth of de novo

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atheromatous tissue inside stents (1), now with optical coherence tomography, it is feasible to unravel the complexity of this entity in vivo, as well as the mechanisms of thrombus formations in such cases.

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