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LETTER TO THE EDITOR

Visualization of extensive intraplaque neovascularization by optical coherence tomography

Inflammation plays a key role in pathogenesis and progression of atherosclerosis.[1] Advent of novel intravascular imaging tools such as Optical Coherence Tomography (OCT) has led to visualization of coronary atherosclerotic plaque characteristics in vivo that were previously seen only on histology. Herein we discuss a case of extensive neovascularization, a feature that imparts vulnerability to atherosclerotic plaque in the coronary artery, as seen by two- and three-dimensional OCT images.

A 63-year-old female with history of diabetes mellitus and hypertension presented with troponin-positive chest pain. She underwent coronary angiography followed by drug-eluting stent implantation to the mid segment of right coronary artery (RCA). OCT was subsequently performed to assess the post-procedural result, showing a well-deployed stent and extensive network of microvessels in a thickened intima in the proximal segment of the RCA (Figure 1). Frame-by-frame 3D reconstruction of the microvessels revealed an extensive frond-like network in the proximal segment of RCA (Figure 2).

In most patients, acute coronary syndrome (ACS) is secondary to atherosclerotic plaque rupture[2] and resultant intracoronary thrombosis. Plaque expansion secondary to intra-plaque hemorrhage (IPH) is one of the factors that can destabilize the plaque.[3] IPH is thought to occur as a consequence of rupture of intra-plaque microvessels developed due to a process called neo-angiogenesis.

Human coronary artery is a tri-laminar structure and is lined by intima, media and adventitia from inside to the outside (Figure 1A and B). The normal human coronary artery derives blood supply from vasa-vasorum situated in the adventitious layer. A process called plaque neovascularization — which involves formation of neo-vessels (Figure 1C and D) takes place simultaneously, initially with intimal layer thickening and later during plaque expansion. These neo-vessels originating from the vasa-vasorum reach the plaque situated in the intima through the medial layer. It has been shown that these microvessels lack structural integrity and thus are fragile and might easily rupture. Following rupture of microvessels, deposition of free cholesterol, macrophage infiltration and accumulation of erythrocyte membranes result in the enlargement of the necrotic core within an existing atherosclerotic plaque. These factors increase the risk of plaque destabilization culminating in plaque rupture and acute event.

Owing to the advent of novel intravascular imaging tools such as OCT,[4,5] high-resolution pictures of atherosclerotic

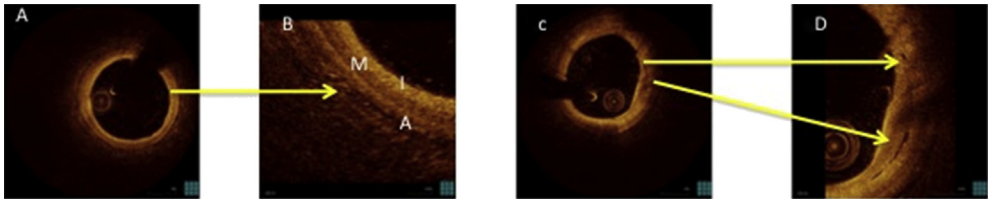

Figure 1 A: Normal OCT cross-section of a coronary artery, B: Magnified view demonstrating the three layers of normal coronary artery; I-Intima, M-Media, A-Adventitia, C: Cross-section showing microvessels, D: Magnified view showing microvessels.

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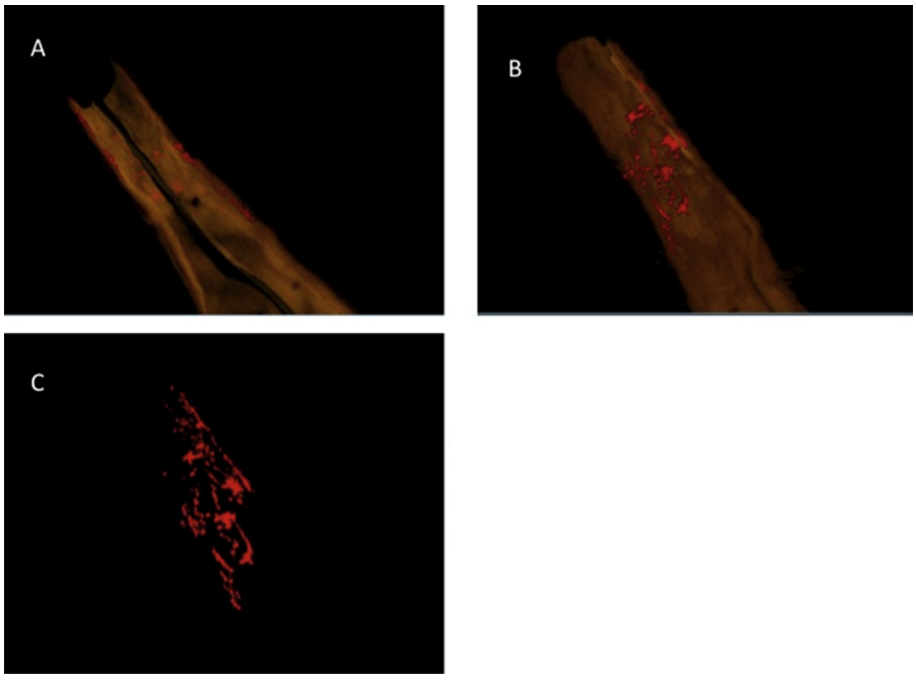

Figure 2 3D reconstruction showing microvessels from (in Red) A) Inside through cut open view, B) Outside and C) with out coronary artery.

plaque characteristics that were once assessed ex-vivo only can now be appreciated in vivo.

Disclosures

None

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