<S5-3>

Update on Vulnerable Coronary Plaques - A Pathological Perspective -

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The majority of acute coronary thrombosis is caused by plaque rupture, while plaque erosion and calcified nodule are also responsible for coronary thrombosis. The term "vulnerable plaque" refers to any precursor lesion of coronary thrombosis in a broad sense. However, in a more limited sense, it refers to a precursor lesion of plaque rupture, i.e., thin-cap fibroatheroma (TCFA), whereas a precursor lesion of plaque erosion or calcified nodule remains unknown. TCFA is pathologically characterized by a relatively large necrotic core with an overlying thin fibrous cap measuring <65 μm typically containing numerous macrophages. This widely-used cut-off value of the fibrous cap thickness for TCFA is derived from a previous pathologic study in sudden coronary victims, in which the 95% of disrupted fibrous cap was <65 μm. Caution must be exercised when interpreting this pathologic evidence, since formalin fixation and paraffin embedding should result in tissue shrinkage in approximately 20-30%. Optical coherence tomography (OCT) enables us to measure fibrous cap thickness and detect macrophage infiltration in vivo, both of which are critical to identify vulnerable plaques. However, the presence of superficial foamy macrophages without underlying necrotic core and an artifact called tangential signal dropout can also create TCFA-like images. The presence of necrotic core can be detected by OCT as signal poor region with indiscriminant borders, while the similar morphology can be seen in lipid pool which is a hallmark of early plaques, i.e., pathologic intimal thickening. Evolution of imaging modalities may help us identify vulnerable plaques to evaluate the risk of future adverse cardiac events, where understanding the morphological features of atherosclerosis as well as the limitations of imaging technologies should be important.