Detection and Prediction of Plaque Rupture Using IVUS

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Spontaneous or PCI induced plaque rupture is associated with acute coronary events and peri-procedural myocardial infarction, so detection and prediction of rupture-prone plaque is very important. In pathology rupture-prone plaque with large lipid core and thin fibrous cap infiltrated by macrophages, erosion-prone plaque with proteoglycan matrix in a smooth muscle cell-rich plaque, eroded plaque with thrombus, intraplaque hemorrhage, calcific nodule protruding into the vessel lumen and eccentric lumen are typical findings of vulnerable plaque.

Unfortunately, it is impossible to gray scale IVUS determine whether this lesion has the histologic and mechanical substrates for a rupture-prone plaque, plaques with nearly similar morphology in terms of lipid core and fibrous cap may look similar with IVUS imaging aimed at morphology only. But many gray scale IVUS findings which can predict peri-procedural complications such as cardiac enzyme elevation and no-reflow phenomenon. Generally hypoechoic (soft) plaque is common in patients with acute coronary syndrome (ACS) and reduced echodensity may also be due to necrotic zone within plaque, intramural hemorrhage or thrombus. Lipid pool–like image defined as a pooling of low-echoic material or echolucent material covered with a high-echoic layer, large vessels with lipid pool–like image are at high risk for no reflow after primary intervention for AMI. Attenuated Plaque defined as a deep ultrasound attenuation without calcification, was frequently found in ACS patients and no-reflow was frequently developed. Also calcified nodules, especially close to the luminal surface of the plaque, can protrude through and rupture the fibrous cap, leading to thrombus formation and acute coronary syndromes. Positive remodeling was associated with no-reflow post-primary angioplasty, recurrent ischemia within one month after thrombolysis, in-hospital complications such as CKMB elevation, target lesion revascularization, major cardiac events, and intimal hyperplasia after drug eluting stent implantation.

VH-IVUS TCFA are defined by a focal, necrotic core-containing (≥10% of the total plaque area) in direct contact with the lumen, and in the presence of a percent atheroma volume ≥40%. PROSPECT study prospectively characterized 3,160 nonculprit lesions, demonstrating by multivariable analysis that the 3 independent baseline predictors of future nonculprit-lesion–related MACE during a median follow-up of 3.4 years were a large plaque burden, a small lumen area, and a VH-TCFA. VIVA study performed 3-vessel VH-IVUS in 170 patients (1,096 lesions) with stable or unstable ischemic heart disease. At a median follow-up of 1.7 years, the strongest univariate correlates of future nonculprit-lesion–related MACE were large plaque burden, small lumen area, remodeling index, and a VH-TCFA. Most recently large LCP (defined as a maxLCBI 4 mm >500) detected by NIRS are associated with a high risk of periprocedural MI.

In summary, although a complete understanding of the etiology and mechanisms of atherothrombosis remains an elusive goal, 3 IVUS systems have been a tool capable of providing incremental discrimination to identify patients and lesions at risk for future MACE..