

The authors conclude that despite the high prevalence of HF with PSF, the condition is not managed effectively and further specific studies are required.

Original article Fonarow GC *et al.* (2007) Characteristics, treatments, and outcomes of patients with preserved systolic function hospitalized for heart failure: a report from the OPTIMIZE-HF registry. *J Am Coll Cardiol* **50**: 768–777

Optical coherence tomography permits detailed characterization of vulnerable plaques

Intravascular optical coherence tomography (OCT) is an optical analog of intravascular ultrasonography (IVUS)—the current standard invasive method for diagnosing coronary artery disease. OCT has a markedly higher resolution than IVUS, and can resolve microstructures of atherosclerotic plaques that are thought to be associated with plaque vulnerability, such as thin fibrous caps, lipid cores and intracoronary thrombi. To determine whether OCT is superior to IVUS and coronary angiography for the evaluation of atherosclerotic plaques, Kubo *et al.* enrolled 30 patients with acute myocardial infarction and used each modality to analyze the culprit lesions.

OCT imaging enabled fibrous cap disruption to be identified in more patients than did either IVUS ($P=0.009$) or angiography ($P=0.035$). The detection of fibrous cap erosion was also higher with OCT than with IVUS or angiography—OCT detected this characteristic in 23% of cases, while angiography detected erosion in only 3% of cases and IVUS was unable to detect erosion in any of the cases. Notably, OCT was the only modality that enabled the estimation of fibrous cap thickness, which in this study was a mean of 49 μm . Both OCT and angiography were significantly better than IVUS for intracoronary thrombus detection; OCT and angiography enabled the identification of this microstructure in all cases, whereas IVUS detected it in only 33% of patients.

OCT, therefore, seems to be a feasible and safe imaging modality that facilitates the detailed characterization of vulnerable plaques in patients with acute myocardial infarction.

Original article Kubo T *et al.* (2007) Assessment of culprit lesion morphology in acute myocardial infarction: ability of optical coherence tomography compared with intravascular ultrasound and coronary angiography. *J Am Coll Cardiol* **50**: 933–939

Autopsy study demonstrates clustering of vulnerable coronary plaques

Acute coronary syndromes are usually caused by the rupture of vulnerable plaques, leading to thrombosis. It remains unclear, however, how these plaques are distributed throughout the coronary arterial tree.

Cheruvu *et al.* studied the prevalence and location of atheromatous plaques in 50 human hearts. The right coronary, left circumflex and left anterior descending arteries were resected and cut into 20 mm longitudinal sections. Further graphical analysis was performed using 3 mm segments, 3,639 of which were available in all.

The prevalence of thin-cap fibroatheroma (TCFA) and ruptured plaques was low. Overall, 23 TCFA and 19 ruptured plaques were identified from a total of 212 advanced coronary lesions, the majority of which were non-atheromatous. Among the 20 hearts that contained at least one TCFA or ruptured plaque, the mean ($\pm \text{SD}$) numbers of these lesions per heart, were 1.15 ± 1.23 and 0.95 ± 0.83 , respectively.

TCFA and ruptured plaques tended to cluster, particularly in the left anterior descending and left circumflex arteries, in which 90% of these lesions were found within the first 22 mm. In 16 of the 20 hearts that contained either of these lesions, all were located within two or fewer 20 mm sections. In 11 of these hearts, less than 10 mm of total coronary length was occupied by these lesions.

These findings suggest a focal distribution of vulnerable coronary plaques. Identifying these high-risk areas could facilitate the stabilization of plaques before rupture.

Original article Cheruvu PK *et al.* (2007) Frequency and distribution of thin-cap fibroatheroma and ruptured plaques in human coronary arteries: a pathologic study. *J Am Coll Cardiol* **50**: 940–949

$^{18}\text{FDG-PET}$ imaging of atherosclerotic plaque inflammation

^{18}FDG -uptake in the aorta and the carotid arteries has been used as a surrogate marker to monitor changes in atherosclerotic plaque inflammation during drug efficacy trials. Rudd *et al.* have established that FDG-PET imaging of atherosclerosis is highly