Atherosclerosis is a chronic, progressive, inflammatory disease causing multiple lesions in the intima of large and medium-sized arteries. A minority of atherosclerotic lesions referred to as vulnerable plaques (VPs) may suddenly precipitate thrombosis, leading to life-threatening events such as acute myocardial infarction and ischemic stroke. The most common VP is the rupture-prone type, also known as thin-cap fibroatheromas, which are characterized by a large necrotic core with a thin and inflamed fibrous cap, outward remodeling mitigating luminal obstruction, neovascularization, plaque hemorrhage, adventitial inflammation, and a “spotty” pattern of calcifications. Among these features, inflammation and angiogenesis are believed to be critical by degrading the plaque matrix (proteolytic enzymes secreted by macrophages) and expanding the necrotic core (intraplaque hemorrhage from leaky microvessels). Both inflammation and angiogenesis are known to be associated with tissue edema, but because edema is difficult to detect in dehydrated, paraffin-embedded pathological specimens, it has escaped attention as a possible VP marker.

Cardiovascular magnetic resonance (CMR) with a T2-weighted short-tau inversion recovery sequence (T2-STIR) is an established technique for the visualization of myocardial edema as a measure of the area-at-risk in patients with acute myocardial infarction. Our own unpublished observations indicate that T2-STIR CMR often reveals localized coronary edema in the culprit artery of patients with acute myocardial infarction, indicating that VPs may be visualized by CMR.
This is a novel finding, and there are no prior references to the use of this technique for the identification of VPs.

A 37-year-old man was transferred to our institution for acute coronary intervention due to sudden onset of chest pain during bicycle spinning. The 12-lead ECG showed minor inferior ST-segment elevation suggestive of inferior myocardial infarction (Figure 1A). Laboratory tests showed a significant increase in cardiac troponin T to a maximum value of 682 ng/L (reference value <50 ng/L) and elevated creatine kinase-MB mass to 22.1 µg/L (reference value <7.0 µg/L). Left ventricular function was normal. Coronary angiography showed an intraluminal filling defect in the proximal left anterior descending (LAD) coronary artery with peripheral embolization to the distal LAD (Figure 1B through 1C). Thrombus aspiration was unsuccessfully attempted. Gray-scale intravascular ultrasound demonstrated a large eccentric plaque with outward remodeling throughout the proximal LAD (Figure 1B through 1C). No further coronary intervention was performed. The patient was treated with bivalirudin, clopidogrel, and aspirin. Further multimodality imaging was performed on day 3 after the myocardial infarction. Coronary angiography showed that the intraluminal filling defect was significantly reduced, and no stenosis was present. T2-STIR CMR demonstrated myocardial edema in the inferior apical wall of the left ventricle (Figure 1D), which corresponded to the distal LAD embolization. The parameters for the ECG-triggered, navigator-
Coronary magnetic resonance angiography demonstrated a large plaque in the proximal LAD positioned from the first to second diagonal branch (Figure 2A). Intravascular ultrasound virtual histology analysis was suggestive of fibroatheroma in the culprit lesion (Figure 3A). Multidetector computed tomography coronary angiography showed an eccentric, non-calcified, low-attenuation plaque at the same location (Figure 2B and Figure 3C). Frequency domain optical coherence tomography identified an eccentric, high-attenuation plaque with some residual luminal thrombosis proximal in the LAD and corresponding to the culprit lesion detected by coronary angiography, indicating the presence of a ruptured thin-cap fibroatheromas (online-only Data Supplement Movie 2 and Figure 3B). T1-weighted inversion recovery prepared coronary CMR showed an isointense plaque at the LAD culprit lesion, suggesting the absence of intraplaque hemorrhage or red thrombus (Figure 3D). T2-STIR CMR demonstrated a hyperintense plaque (defined as signal intensity above 2 SD of the mean signal intensity of the normal myocardium) suggestive of localized coronary edema at the matching location in the proximal LAD (Figure 3E). By T2-STIR CMR, coronary edema was present in the entire proximal LAD (Figure 4B through 4D), whereas the left main coronary artery was without edema (Figure 4A). The corresponding multidetector computed tomography images showed noncalcified low-attenuation plaques in those regions with coronary edema (Figure 4). The patient was discharged after 5 days with dual antiplatelet and statin therapy.

This case exemplifies a common mechanism of acute coronary syndrome with dynamic coronary thrombosis and peripheral embolization causing microembolic occlusion of the distal LAD. Inflammation and angiogenesis are considered critical components of VPs, and these data represent the first in vivo demonstration of the presence of a surrogate marker, plaque edema, in the culprit lesion of a patient with an acute coronary syndrome. Thus, the differentiation between “dry” and “wet” plaques with T2-STIR CMR holds promise as a noninvasive technique to differentiate between stable and high-risk plaques in patients with coronary atherosclerosis and to monitor disease activity and response to treatment.

Disclosures

None.

References


Key Words: acute coronary syndrome • atherosclerosis • magnetic resonance imaging • multidetector computed tomography • optical coherence tomography
SUPPLEMENTAL MATERIAL

Movie 1: Grayscale intravascular ultrasound demonstrated a large eccentric plaque with outward remodeling throughout the proximal left anterior descending coronary artery.

Movie 2: Frequency domain optical coherence tomography identified an eccentric, high-attenuation plaque with some residual luminal thrombosis proximal in the left anterior descending coronary artery indicating the presence of a ruptured plaque.