Cardiovascular images

First In Vivo Demonstration of Coronary Edema in Culprit Lesion of Patient With Acute Coronary Syndrome by Cardiovascular Magnetic Resonance

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Atherosclerosis is a chronic, progressive, inflammatory disease causing multiple lesions in the intima of large and medium-sized arteries.1 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.2 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of myocardial edema as a measure of the area-at-risk in patients with acute myocardial infarction.3 Our own unpublished observations indicate that T2-STIR CMR often reveals localized coronary edema in the culprit artery of patients with acute myocardial infarction, indicating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.

Figure 1. A, Twelve-lead ECG demonstrates minor ST elevation in the inferior leads. B, X-ray coronary angiography shows a nonocclusive intraluminal filling defect in the proximal LAD coronary artery (arrow) with peripheral embolization to the distal LAD (C) (arrow). D, T2-STIR CMR imaging performed on day 3 after infarction shows hyperintense signal (edema) in the inferior apical segment of the left ventricle (arrow) indicative of myocardial ischemia in the segment supplied by the distal LAD. LV indicates left ventricle; RV, right ventricle.

Figure 2. A, Coronary magnetic resonance angiography shows a large nonstenotic and outward remodeled plaque positioned from the first and second diagonal branch in the proximal LAD (arrows). B, Multidetector computed tomography coronary angiography shows a noncalcified, low-attenuation plaque in the same location (arrows).

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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 g/L (reference value 7.0 g/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). The circumflex and right coronary arteries appeared normal on the angiography. Thrombus aspiration was unsuccessfully attempted. Gray-scale intravascular ultrasound demonstrated a large eccentric plaque with residual luminal thrombosis (arrows) opposite to the first diagonal branch (DB) in the proximal LAD, indicating the presence of a ruptured thin-cap fibroatheroma. Multidetector computed tomography coronary angiography shows a large, eccentric, noncalcified, low-attenuation and positively remodeled plaque (P), indicating the presence of a thin-cap fibroatheroma. The coronary lumen (L) is depicted with a bright signal caused by the contrast injection. T2-weighted inversion recovery CMR shows isointense signal in the eccentric coronary plaque proximal in LAD (P), indicating the absence of intraplaque hemorrhage or red thrombus at the culprit lesion. T2-STEIR CMR with a black blood spin-echo sequence shows hyperintense signal (edema) in the eccentric coronary plaque proximal in LAD (P), indicating the presence of inflammation and/or angiogenesis in the culprit lesion. The coronary lumen (L) appears black as the result of suppression of signal from the coronary blood pool.

Figure 3. A, Intravascular ultrasound with color-coded virtual histology shows an eccentric plaque in the proximal LAD, suggesting the presence of a fibroatheroma. Red color indicates necrotic core; dark green, fibrous tissue; light green, fibrofatty tissue; and white color, dense calcium. B, Optical coherence tomography demonstrating an eccentric, high-attenuation plaque with residual luminal thrombosis (arrows) opposite to the first diagonal branch (DB) in the proximal LAD, indicating the presence of a ruptured thin-cap fibroatheroma. C, Multidetector computed tomography coronary angiography shows a large, eccentric, noncalcified, low-attenuation and positively remodeled plaque (P), indicating the presence of a thin-cap fibroatheroma. The coronary lumen (L) is depicted with a bright signal caused by the contrast injection. D, T2-weighted inversion recovery CMR shows isointense signal in the eccentric coronary plaque proximal in LAD (P), indicating the absence of intraplaque hemorrhage or red thrombus at the culprit lesion. E, T2-STEIR CMR with a black blood spin-echo sequence shows hyperintense signal (edema) in the eccentric coronary plaque proximal in LAD (P), indicating the presence of inflammation and/or angiogenesis in the culprit lesion. The coronary lumen (L) appears black as the result of suppression of signal from the coronary blood pool.

Figure 4. Corresponding images of the proximal LAD showing coronary edema by T2-STEIR CMR predominantly in regions with noncalcified low-attenuation plaques by multidetector computed tomography (B through D, yellow arrows). The left main coronary artery shows no edema and no plaque (A).
gated, dark-blood, T2-STIR fast spin-echo sequence were as follows: TR, 2 RR intervals; TE, 100 ms; echo train length of 20; 0.68×0.68×8 mm³ voxels; and 2 signal averages.

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 non-calcified 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
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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.