Novel Approach for In Vivo Detection of Vulnerable Coronary Plaques Using Molecular 3-T CMR Imaging With an Albumin-Binding Probe


OBJECTIVES:
This study sought to investigate the potential of the noninvasive albumin-binding probe gadofosveset-enhanced cardiac magnetic resonance (GE-CMR) for detection of coronary plaques that can cause acute coronary syndromes (ACS).

BACKGROUND:
ACS are frequently caused by rupture or erosion of coronary plaques that initially do not cause hemodynamically significant stenosis and are therefore not detected by invasive x-ray coronary angiography (XCA).

METHODS:
A total of 25 patients with ACS or symptoms of stable coronary artery disease underwent GE-CMR, clinically indicated XCA, and optical coherence tomography (OCT) within 24 h. GE-CMR was performed approximately 24 h following a 1-time application of gadofosveset-trisodium. Contrast-to-noise ratio (CNR) was quantified within coronary segments in comparison with blood signal.

RESULTS:
A total of 207 coronary segments were analyzed on GE-CMR. Segments containing a culprit lesion in ACS patients (n = 11) showed significant higher signal enhancement (CNR) following gadofosveset-trisodium application than segments without culprit lesions (n = 196; 6.1 [3.9 to 16.5] vs. 2.1 [0.5 to 3.5]; p < 0.001). GE-CMR was able to correctly identify culprit coronary lesions in 9 of 11 segments (sensitivity 82%) and correctly excluded culprit coronary lesions in 162 of 195 segments (specificity 83%). Additionally, segmented areas of thin-cap fibroatheroma (n = 22) as seen on OCT demonstrated significantly higher CNR than segments without coronary plaque or segments containing early atherosclerotic lesions (n = 185; 9.2 [3.3 to 13.7] vs. 2.1 [0.5 to 3.4]; p = 0.001).

CONCLUSIONS:
In this study, we demonstrated for the first time the noninvasive detection of culprit coronary lesions and thin-cap fibroatheroma of the coronary arteries in vivo by using GE-CMR. This method may represent a novel approach for noninvasive cardiovascular risk prediction.
KEYWORDS:
CMR; atherosclerosis; endothelial dysfunction; molecular imaging; vulnerable plaque