JACC: CARDIOVASCULAR IMAGING
VOL. 8, NO. 10, 2015

ISSN 1936-878X/$36.00
PUBLISHED BY ELSEVIER INC.
http://dx.doi.org/10.1016/j.jcmg.2015.06.013

# Localization of Coronary High-Intensity Signals on T1-Weighted MR Imaging

## Relation to Plaque Morphology and Clinical Severity of Angina Pectoris

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## ABSTRACT

**OBJECTIVES** This study sought to investigate the relationship between localization of high-intensity signals (HISs) on T1-weighted imaging (T1WI) with the noncontrast magnetic resonance technique and plaque morphology detected on optical coherence tomography, and the clinical severity of angina pectoris.

**BACKGROUND** Since the introduction of the T1WI noncontrast magnetic resonance technique for plaque imaging, some groups have reported that HISs in the coronary artery on T1WI are associated with a vulnerable morphology and future cardiac events. However, the association between the localization of HISs, such as coronary intrawall or intraluminal, and plaque morphology has not been investigated.

**METHODS** One hundred lesions with either stable or unstable angina were included and divided into 3 groups according to the following criteria using T1WI. First, the plaques with the ratio between the signal intensities of coronary plaque and cardiac muscle ≤1.0 were classified as non-HISs (n = 39). Then, HISs with the ratio between the signal intensities of coronary plaque and cardiac muscle >1.0 were classified into 2 types by using cross-sectional T1WI. Those localized within the coronary wall when the lumen was identified were defined as intrawall HISs (n = 37), whereas those occupying the lumen when the lumen was not, or even if only partly, identified, were defined as intraluminal HISs (n = 24).

**RESULTS** Multivariate analysis revealed that intrawall HISs were associated with macrophage accumulation and the absence of calcification assessed by using optical coherence tomography. In contrast, thrombus and intimal vasculature were independent factors associated with intraluminal HISs. Furthermore, 50% of patients with intraluminal HISs experienced rest angina, such as Braunwald class II or III.

**CONCLUSIONS** This study shows that intrawall and intraluminal HISs on T1WI in patients with angina are related to the different types of vulnerable plaque morphology and the clinical severity. (J Am Coll Cardiol Img 2015;8:1143–52)


Plaque rupture or erosion of the endothelial surface with subsequent thrombus formation is recognized as the most important mechanism in acute coronary syndromes (1). Moreover, histopathologic reports have demonstrated that coronary intraplaque hemorrhage could lead to an increase in plaque burden, in addition to being a reflection of the biological activity of the lesion (2–4). However, the association between intraplaque hemorrhage and the onset of clinical symptoms resulting from coronary artery disease is still a controversial issue.

Since the introduction of the T1-weighted imaging (T1WI) noncontrast magnetic resonance (MR) technique for plaque imaging, many researchers have shown that high-intensity signals (HISs) in the carotid arterial wall on T1WI indicate the presence of intraplaque hemorrhage containing methemoglobin (5–7). Furthermore, some groups, including ours, have

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Manuscript received March 10, 2015; revised manuscript received June 3, 2015, accepted June 26, 2015.

**ABBREVIATIONS AND ACRONYMS**

**CAG** = coronary angiography

**HIS** = high-intensity signal

**MR** = magnetic resonance

**OCT** = optical coherence tomography

**PMR** = the ratio between the signal intensities of coronary plaque and cardiac muscle

**TCFA** = thin-cap fibroatheroma

**T1WI** = T1-weighted imaging

**UAP** = unstable angina pectoris

reported that coronary artery HISs on T1WI are associated with a vulnerable morphology and future cardiac events (8–11). However, the association between the localization of HISs, such as coronary intrawall or intraluminal, and plaque morphology was not investigated in previous studies.

Optical coherence tomography (OCT) was recently developed as a high-resolution imaging device for plaque characterization. Several studies have already shown that OCT allows the identification not only of plaque rupture, fibrous cap thickness, and intraluminal thrombus, but also of macrophages and intimal vasculature within atherosclerotic plaques in vivo (12–14). Therefore, we hypothesized that OCT might allow us to assess in detail the characteristics of coronary intrawall and intraluminal HISs on noncontrast T1WI. In this study, the term "localization" was used as a differentiation between intrawall and intraluminal, not suggesting the longitudinal distribution of HISs along the course of the coronary artery. The aim of this study was to investigate the relationship between localization of coronary HISs on noncontrast T1WI and plaque morphology detected by OCT, and the clinical severity of angina pectoris.

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## METHODS

**PATIENTS.** One hundred twenty-six consecutive patients with angina were prospectively enrolled in this study between September 2010 and January 2014. Patients with prior percutaneous coronary intervention, coronary artery bypass grafting, an occluded coronary vessel, or contraindications for MR were excluded from the study. Patients eligible for an early invasive strategy according to the American College of Cardiology Foundation/American Heart Association Guideline (elevated levels of cardiac biomarkers and signs or symptoms of heart failure) were also excluded. All patients underwent MR within 24 h before the day on which invasive coronary angiography (CAG) and OCT were performed. Of the 126 patients initially enrolled, 26 were excluded from the analysis for technical reasons as follows: 23 patients did not undergo OCT examination before percutaneous coronary intervention (11 had failure of OCT crossing, 11 were eligible for coronary artery bypass grafting, and 1 had an angiographically significant left main coronary stenosis), and 2 had MR images and 1 had OCT images that were of poor image quality and thus could not be analyzed.

Taking into account intrapatient correlations when evaluating the data, 1 culprit lesion was used in the analysis for any patient with more than 2 lesions. Thus, 100 lesions from 100 patients who had angiographically documented narrowing of at least 50% of the luminal diameter of a major coronary artery on CAG were examined in this study. Unstable angina pectoris (UAP) was diagnosed in 58 patients according to Braunwald criteria. Class I indicates new-onset severe or accelerated exertional angina within 2 months (n = 26); classes II (n = 20) and III (n = 12) indicate angina at rest during the previous month. The remaining stable angina pectoris group was made up of 42 patients with chest pain typical of cardiac ischemia on exertion that was clinically unchanged for >2 months. The culprit vessel was identified based on clinical, scintigram stress test and angiographic data. Oral aspirin (100 mg) and clopidogrel (75 mg) were administered on admission. Patients at high risk were also treated with intravenous heparin, but no patient was given thrombolytic agents.

The study was approved by the hospital ethics committee, and informed consent was obtained from all patients before the study.

**MR CORONARY PLAQUE IMAGE ACQUISITION.** Coronary plaque imaging was performed using a 1.5-T MR imager (Achieva, Philips Medical Systems, Best, the Netherlands) with a 5-element cardiac coil. Nitroglycerin (0.3 mg) was administered sublingually immediately before image acquisition to obtain high-quality MR images. Initial survey images were focused around the heart, following which the reference images were obtained for the sensitivity of parallel imaging. Transaxial cine MR images were then acquired using a steady-state free-precession sequence with breath holding, to determine the trigger delay time when the motion of the right coronary artery was minimal.

First, to obtain detailed information on the location of the target lesion, free-breathing, steady-state, free-precession, whole-heart coronary MR angiographic images were obtained (repetition time, 3.7 ms; echo time, 1.8 ms; flip angle, 80°; SENSE factor, 2.0; number of excitations, 1; navigator gating window of ± 2.0 mm with diaphragm drift correction; field of view, 300 × 255 × 120 mm [rectangular field of view, 85%]; acquisition matrix, 240 × 240; reconstruction matrix, 512 × 512 × 160, resulting in an acquired spatial resolution of 1.25 × 1.25 × 1.5 mm reconstructed to 0.6 × 0.6 × 0.75 mm) (10).

Next, coronary plaque images were obtained while the patients were breathing freely, by using a 3-dimensional T1WI, inversion-recovery, gradient-echo technique with fat-suppressed and radial k-space sampling in the Y-Z plane (repetition time,