Journal of Cardiology xxx (2017) xxx–xxx

Contents lists available at ScienceDirect

Journal of Cardiology

journal homepage: www.elsevier.com/locate/jjcc

Original article

Characterization of high-intensity plaques on noncontrast T1-weighted magnetic resonance imaging by coronary angioscopy

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ARTICLE INFO

Article history:
Received 11 November 2016
Received in revised form 8 April 2017
Accepted 26 April 2017
Available online xxx

Keywords:
High-intensity plaques
Angioscopy
Thrombus
Yellow plaque

ABSTRACT

Background: A recent study showed that coronary high-intensity plaques (HIPs) visualized by noncontrast T1-weighted imaging (T1WI) in cardiac magnetic resonance were associated with coronary events. We used coronary angioscopy to analyze HIP plaque morphology.
Methods and results: A total 17 lesions from 17 patients with stable or unstable angina pectoris were evaluated at the culprit lesion by noncontrast T1WI using 1.5-T magnetic resonance; of them, nine (53%) were HIPs and eight (47%) were non-HIPs, and all were analyzed by coronary angioscopy. We assessed the existence of thrombus and plaque yellow color grade (YG). YG was assessed visually according to a four-grade scale: 0, white; 1, light yellow; 2, yellow; 3, intense yellow. The frequency of thrombus was significantly higher in HIPs than in non-HIPs (89% vs. 25%, respectively; $p = 0.007$). YG was significantly more frequent in HIPs than in non-HIPs (2.2 ± 0.4 vs. 0.7 ± 0.7, respectively; $p = 0.01$).
Conclusions: These data indicated that HIPs on noncontrast T1WI were associated with the presence of high-grade yellow plaque with thrombus.

Introduction

The in-hospital mortality rate of acute myocardial infarction (AMI) has improved, whereas approximately half cases of out-of-hospital AMI are fatal [1]. Therefore, the detection of vulnerable plaques is important to preventing the onset of acute coronary syndrome (ACS). A recent study showed that coronary high-intensity plaques (HIPs) visualized by noncontrast T1-weighted imaging (T1WI) on cardiac magnetic resonance (CMR) were associated with coronary events [2].

Many investigators have speculated that HIPs on noncontrast T1WI indicates the presence of mural or intraplaque hemorrhage containing methemoglobin in the carotid arteries [3,4]. However, because of the small dimensions and continuous motion of coronary artery, the visualization of coronary plaques is challenging, so it is unclear what causes HIPs of the coronary artery on T1WI [5–8]. The aim of this study was to analyze HIP morphology using coronary angioscopy.

Methods

Study subjects

This study was a prospective, observational, single center study. Patients with stable angina pectoris (SAP) or unstable angina pectoris (UAP) were prospectively enrolled. In all of these patients, significant coronary stenosis (>50%) was detected on multislice computed tomography (MSCT) or invasive coronary angiography; all were scheduled for elective percutaneous coronary intervention (PCI) between October 2013 and August 2014. Patients were excluded from the study if they had prior PCI, coronary bypass grafting, an occluded coronary vessel, or contraindications to CMR and they needed to take early invasive treatment according to the American College of Cardiology Foundation/American Heart Association Guideline. Patients had been evaluated by noncontrast T1WI in CMR within 7 days before PCI. In all patients, the angioscopic analysis was performed on a native de novo atherosclerotic lesion considered to be the culprit lesion. SAP was defined as effort angina pectoris that showed no change in frequency, duration, or intensity of chest pain, evidence of ischemia in stress tests or nuclear cardiology imaging, and the finding of a stenosis >50% with subsequent coronary angiograms

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http://dx.doi.org/10.1016/j.jjcc.2017.04.009
0914-5087/

(CAG) [9]. UAP was defined as new onset of severe, progressive, or resting angina suggestive of ACS coinciding with appropriate objective evidence of myocardial ischemia in electrocardiograms (ECG) and nuclear cardiology images, invasive CAG demonstrating a stenosis of >50%, and no increase in the serum creatine kinase levels [10].

Informed consent was obtained from all patients prior to the study, and the study protocol conformed to the guidelines of our institution's ethics committee.

CMR coronary plaque imaging

Coronary plaque imaging was performed using a 1.5-T MR imager (Magnetom Aera; Siemens Healthcare, Berlin, Germany) with 18-channel (body) coils and 32-channel (spine) coils. When heart rates were ≥65 beats/min, the rates were adjusted by administration of 20–60 mg of metoprolol 90 min before imaging. The survey images were focused on the heart, and reference images were obtained to ensure the sensitivity of parallel imaging under free breathing. Transaxial cine MR images were then acquired with a steady-state sequence as the patient breathed freely [repetition time, 2.65 ms; echo time, 1.11 ms; flip angle, 80°; field of view, 340; voxel size, 1.77 mm × 1.77 mm × 8.00 mm; cardiac phases, 100; Generalized Autocalibrating Partially Acquisitions (GRAPPA) factor, 2] to determine the trigger delay time in the presence of minimal right coronary artery motion. Next, coronary plaque images were obtained when patients were breathing freely using a three-dimensional T1WI inversion-recovery gradient-echo sequence with a black-blood condition (inversion time, 400–500 ms), fat-suppressed (water excited) and linear phase-encoded (right to left) (repetition time, 6.35 ms; echo time, 2.95 ms; flip angle, variable; GRAPPA factor, 2; number of excitations, 1; navigator gating window, 3 mm with diaphragm drift correction; field of view, 360; voxel size, 0.80 × 0.80 × 1.25 mm). Next, coronary MR angiography was performed while patients underwent steady-state free precession sequence with T2 preparation and linear phase-encoded (anteroposterior) (repetition time, 3.50 ms; echo time, 1.48 ms; flip angle, 90°; GRAPPA factor, 2; number of excitations, 1; navigator gating window, ±2.5 mm with diaphragm drift correction; field of view, 360; voxel size, 0.66 mm × 0.66 mm × 1.00 mm).

Coronary plaque image analysis was performed by one cardiologist and one radiologist. We calculated the signal intensity of the coronary plaque to muscle ratio (PMR; defined as the signal intensity of the coronary plaque divided by the signal intensity of the left ventricular muscle near the coronary plaque). Areas with a PMR ≥ 1.4 were defined as HIP, while those with a PMR < 1.4 were defined as non-HIP, as described in a previous report [2].

Coronary angioscopy

The coronary angioscopy system we used was supplied by Intertec Medicals (Osaka, Japan). Fiber imaging was performed by a microscope with reflex illumination (FT-203F; Fiber Tech Co. Ltd., Tokyo, Japan). The size of fiber was 0.75 mm. Before use, focus and white balance was adjusted for color correction. The light power was adjusted automatically to avoid refraction and determine plaque color. Coronary angioscopy was performed as follows. First, we cross the guide wire into the target coronary artery and introduce the 4F probing catheter into the distal lesion we want to visualize and then insert the fiber catheter instead of the guide wire. The angioscopic observation was performed while the blood was washed away from the view by the injection of low molecular weight dextran. The angioscopic images were recorded by a hard disk recorder.

Angioscopic analysis

The angioscopic images were evaluated by two coronary angioscopy specialists who were blinded to the patients' clinical data. The color of the culprit lesion was graded as 0 (white), 1 (light yellow), 2 (yellow), or 3 (intense yellow), according to the sample colors reported previously [11,12]. We also assessed the existence of thrombus according to the Japanese Society of Cardiovascular Angioscopy guidelines for red thrombus, mixed thrombus, and white thrombus.

Statistical analysis

Continuous data are summarized as means ± SD. Categorical variables were compared using the Fisher's exact test and the χ^2 test. Intergroup differences were assessed using analysis of variance, a χ^2 test, an unpaired t test, or Mann–Whitney U test as appropriate. Values of $p < 0.05$ were considered statistically significant.

Results

Forty two patients with SAP and UAP were initially enrolled. Fifteen patients were excluded, because of exclusion criteria: prior PCI (n = 11), coronary bypass grafting (n = 1), an occluded coronary vessel (n = 1), and contraindications to CMR (n = 2). Ten patients were excluded because angioscopy was not performed before PCI (n = 8; 3 HIPs and 5 non-HIPs), angioscopic images of sufficient quality were not demonstrated (n = 1), magnetic resonance images of sufficient quality were not demonstrated (n = 1). Thus, 17 lesions from 17 patients were examined in this study. Of the 17 lesions from 17 patients, 9 (53%) had HIPs and 8 (47%) were non-HIPs. The baseline characteristics of those 17 lesions are shown in Table 1. Mean age, diagnosis, cardiovascular risk factors, and angiographic findings were similar in the two groups. The correlation between HIP and non-HIP lesions on noncontrast T1WI and the plaque morphology obtained by angioscopy is shown in Figs. 1 and 2. The thrombus frequency was significantly higher in HIP lesions than in non-HIP lesions (89% vs. 25%, respectively; p = 0.007). Yellow color grade (YG) was significantly more common in HIPs than in non-HIPs (2.2 ± 0.4 vs. 0.7 ± 0.7, respectively; p = 0.01). A representative case of HIP lesion on T1WI assessed for plaque morphology on coronary angioscopy is shown in Fig. 3. Coronary MR angiography and CAG showed severe coronary

Table 1
Patient characteristics.

	HIP (n = 9)	Non-HIP (n = 8)	p
PMR	1.88 ± 0.61	0.84 ± 0.18	0.0006
Age (years)	68 ± 11	64 ± 18	0.601
Male	7 (78%)	6 (75%)	0.893
Diagnosis			0.064
SAP	5	7	
UAP	4	1	
Hypertension	6 (67%)	3 (38%)	0.230
Dyslipidemia	7 (78%)	6 (75%)	0.893
Diabetes mellitus	4 (44%)	3 (38%)	0.772
Smoking	1 (11%)	1 (13%)	0.930
Culprit vessel			0.094
LAD	1	3	
LCx	1	2	
RCA	7	2	
Percent diameter stenosis	72 ± 32	82 ± 17	0.767

Values are mean ± SD or n (percentage).
HIP, high-intensity plaque; PMR, signal intensity of coronary plaque to cardiac muscle ratio; SAP, stable angina pectoris; UAP, unstable angina pectoris; LAD, left anterior descending coronary artery; LCx, left circumflex coronary artery; RCA, right coronary artery.

Please cite this article in press as: Oshita A, et al. Characterization of high-intensity plaques on noncontrast T1-weighted magnetic resonance imaging by coronary angioscopy. J Cardiol (2017), http://dx.doi.org/10.1016/j.jjcc.2017.04.009