Coronary high-intensity plaque on $T_1$-weighted magnetic resonance imaging and its association with myocardial injury after percutaneous coronary intervention

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Aims
Non-contrast $T_1$-weighted imaging (T1WI) has emerged as a novel non-invasive imaging for vulnerable coronary plaque showing a high-intensity plaque (HIP). However, the association between HIP and percutaneous coronary intervention (PCI) has not been evaluated. We investigated the association between the presence of HIP and the incidence of myocardial injury after PCI.

Methods and results
A total of 77 patients with stable angina were imaged with non-contrast T1WI by using a 1.5 T magnetic resonance system (HIP and non-HIP group, $N=31$ and 46 patients, respectively). We defined HIP as a coronary plaque to myocardium signal intensity ratio (PMR) of $\geq 1.4$. High-sensitive cardiac troponin-T (hs-cTnT) was measured at baseline and 24 h after PCI. Percutaneous coronary intervention-related myocardial injury (PMI) was defined as an elevation of hs-cTnT. High-intensity plaque was associated with the characteristics of ultrasound attenuation and positive remodelling on intravascular ultrasound. Although baseline hs-cTnT was similar between the groups, increase in hs-cTnT was significantly greater in the HIP vs. non-HIP group (0.065 [0.023–0.304] vs. 0.017 [0.005–0.026], $P<0.001$). Percutaneous coronary intervention-related myocardial injury occurred more frequently in the HIP than non-HIP group (58.1 vs. 10.9%, $P=0.001$), and the cut-off value of PMR found to be 1.44 for predicting PMI (sensitivity 78.3% and specificity 81.5%). In multivariate analysis, a PMR of $\geq 1.4$ was a significant predictor of PMI (odds ratio 5.63, 95% confidence interval 1.28–24.7, $P=0.022$).

Conclusion
High-intensity plaque on non-contrast T1WI was characterized as vulnerable coronary plaque on IVUS and was associated with higher incidence of PMI.

Keywords
Coronary artery disease • Magnetic resonance imaging • Plaque

Introduction
Post-procedural myocardial injury/infarction following percutaneous coronary intervention (PCI) is common in clinical practice and is manifested by the elevation of cardiac biomarkers such as creatine kinase or cardiac troponin. PCI-related myocardial injury (PMI) is a multifactorial phenomenon with diverse aetiologies that depend on patient-related, lesion-related, and procedure-related factors and is associated with worse short- and long-term clinical outcomes even in patients undergoing elective PCI. Intracoronary imaging modalities, such as gray-scale intravascular ultrasound (IVUS), radiofrequency IVUS, and optical coherence tomography (OCT), provide useful information for predicting the occurrence of PMI. Several multidetector computed tomography (MDCT) studies have also examined the relation between target lesion plaque composition and PMI in patients undergoing elective PCI.
Recently, non-contrast T1-weighted imaging (T1WI) has emerged as a novel non-invasive imaging modality when searching for vulnerable coronary plaque, although coronary plaque magnetic resonance (MR) imaging is still challenging due to the small diameter of the coronary arteries and cardiac and/or respiratory motion. High-intensity plaque (HIP) detected on non-contrast T1WI has been considered to be vulnerable coronary plaque. In comparison with MDCT and MR images of coronary arteries, HIP on non-contrast T1WI is associated with a high frequency of plaque with low CT density. A preliminary study by Kawasaki et al. also reported that coronary HIP is associated with ultrasound attenuation and vessel positive remodelling on IVUS. A small sample study has shown that HIP might be associated with coronary thrombus on OCT. Recently, Noguchi et al. reported that the presence of HIP and a coronary plaque to myocardium signal intensity ratio (PMR) of $\geq 1.4$ were risk factors for coronary events among patients with coronary artery disease. However, the clinical significance of coronary plaque MR imaging (MRI) for PCI has not been fully investigated. Therefore, we hypothesized that the presence of HIP on non-contrast T1WI has the potential to predict an elevation of cardiac troponin-T after PCI. The aim of this study was to investigate the coronary plaque characteristic of HIP on T1WI and the association between the presence of HIP and the incidence of PMI.

**Methods**

**Study subjects and study protocol**

Between January 2012 and September 2013, 81 patients (104 coronary plaques) with stable angina pectoris, in whom significant coronary artery stenosis ($> 70\%$) was diagnosed on invasive coronary angiography, were prospectively enrolled in this study and then underwent coronary MRI with non-contrast T1WI. Among them, two patients who did not undergo PCI and two patients whose image quality of T1WI was poor (each with one coronary artery) were excluded. Therefore, 100 coronary plaques from 77 patients were analysed in this study (Figure 1).

When the patient had at least one HIP, we classified into the HIP group. Exclusion criteria included patients with a contraindication for MRI (pacemaker or implantable cardioverter defibrillator) and lesions with severe calcification by a visual estimate of coronary angiogram, chronic total occlusion, or involvement with the left main coronary artery.

The enrolled patients were imaged with non-contrast T1WI within 48 h before PCI. After acquisition of IVUS images, the PCI procedure was performed with the standard technique. Serial measurement of high-sensitive cardiac troponin-T (hs-cTnT) was performed at both baseline and 24 h after PCI with the aim of assessing PMI. This study was approved by the institutional review board of the University of Tsukuba (study no. H23-064), and written informed consent was obtained from all patients.

**Coronary magnetic resonance imaging and plaque analysis**

All subjects were imaged on a 1.5 T MR system (Achieva, Philips Healthcare, Best, The Netherlands) using a 32-element Torso/cardiac phased-array coil. After scout imaging to localize the heart and diaphragm, transaxial cine MR images were acquired to monitor the interval of minimal motion of the right coronary artery for the determination of the trigger delay of the following sequences. First, axial-based three-dimensional (3D) whole-heart coronary MR angiograms of T$_1$-prepared steady-state free-precession sequence were acquired with navigator-gated free-breathing and electrocardiogram-gated techniques, allowing visualization of the anatomy of the coronary lumens. Parameters of the sequence included repetition time, 2.5 ms; echo time, 1.3 ms; flip angle, 80°; sensitivity-encoding factor, 2.5; field of view, 300 $\times$ 300 $\times$ 120 mm; acquisition matrix, 192 $\times$ 192; acquired spatial resolution, 1.56 $\times$ 1.56 $\times$ 2.00 mm, and reconstructed voxel size, 0.59 $\times$ 0.59 $\times$ 1.00 mm. The following coronary plaque scan was a T$_1$-weighted, inversion-recovery, and fat-suppressed 3D black-blood gradient-echo sequence with navigator-gated free-breathing and electrocardiogram-gated techniques. Parameters of this sequence included repetition time, 4.9 ms; echo time, 2.3 ms; flip angle, 15°; sensitivity-encoding factor, 2.0; field of view, 300 $\times$ 300 $\times$ 120 mm; acquisition matrix, 208 $\times$ 208; acquired spatial resolution, 1.44 $\times$ 1.42 $\times$ 2.00 mm, and reconstructed voxel size, 0.59 $\times$ 0.59 $\times$ 1.00 mm. No contrast agent was used in any of these MRI sequences.

The images were stored on an optical disc in DICOM format. The data were analysed off-line with the DICOM Viewer R3.0 SP3 software (Philips Healthcare). Two experienced cardiologists (D.A. and S.S.), who were blinded to the patient’s clinical data and plaque information obtained by IVUS, measured the signal intensities of coronary plaque and cardiac muscle by placing a free-hand regions of interest (ROI) and calculating the value of PMR, which was defined as the signal intensity of the coronary plaque divided by the signal intensity of cardiac muscle. The signal intensity of the myocardium was measured at a site in the left ventricle near the coronary plaque. Coronary plaque with a PMR of $\geq 1.4$ was defined as HIP, and that with a PMR of $< 1.4$ was defined as non-HIP because coronary plaque with a PMR of $\geq 1.4$ had been shown to be associated with poor clinical prognosis in a previous study.

**Percutaneous coronary intervention procedures and measurement of high-sensitive cardiac troponin-T**

Percutaneous coronary intervention was performed through either the radial or femoral artery by using a 6 Fr catheter. After acquisition of IVUS imaging, balloon dilatation or stent implantation was performed with standard techniques. Transient slow flow was defined as an angiogram showing a deterioration of coronary flow of Thrombolysis In

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**Figure 1** Study flow chart. In all 100 coronary target lesions, HIP (PMR $\geq 1.4$) was detected in 36 lesions of the coronary plaques and non-HIP in 64 lesions. When the patient had at least one HIP, we classified into the HIP group. HIP group, $N = 31$ patients, and non-HIP group, $N = 46$ patients. HIP, high-intensity plaque; PCI, percutaneous coronary intervention; PMR, coronary plaque to myocardium signal intensity ratio; Pts, patients.
Myocardial Infarction (TIMI) grade 0, 1, or 2 seen during the procedure, regardless of the timing, and TIMI grade 3 restoring at the final angiogram.11

Serial measurement of hs-cTnT was performed at both baseline and 24 h after PCI. The hs-cTnT levels were measured by using an electrochemiluminescence immunoassay (Elecsys 2010 system; Roche Diagnostics GmbH, Mannheim, Germany). This method has a detection limit of 0.005 ng/mL, a 99th percentile upper reference limit (URL) of 0.014 ng/mL, and coefficient variation of <10% at 0.013 ng/mL.12 According to the third universal definition of myocardial infarction, PMI was defined as an elevation in hs-cTnT values of $5 \times 99$th percentile URL in patients with normal baseline values ($\leq 99$th percentile URL) or an increase in hs-cTnT values $>20$% if the baseline values are elevated and are stable.13

**Intravascular ultrasound image acquisition and analysis**

Intravascular ultrasound imaging was obtained after intracoronary administration of 2–5 mg of isosorbide dinitrate by using a 40 MHz IVUS catheter (View IT; Terumo Co., Tokyo, Japan) at an automatic pullback speed of 0.5 mm/s before PCI. Quantitative and qualitative analyses were performed in a blinded manner according to the American College of Cardiology Clinical Expert Consensus Document.14 The IVUS parameters were measured at the site of the minimum lumen cross-sectional area (CSA), and volumetric analysis was performed by each 1 mm slice within the ROIs for target lesions, which were matched using side branches as fiduciary landmarks.

The gray-scale IVUS analysis included the external elastic membrane (EEM) CSA, lumen CSA, and plaque plus media ($P + M$) CSA, which was calculated as EEM minus lumen CSA. Percent plaque burden was calculated as $([P + M] \text{ CSA}/\text{EEM CSA}) \times 100$. Ultrasound attenuation was defined as hypoechoic plaque with deep ultrasound attenuation without calcification or very dense fibrous plaque.14 The longitudinal attenuation length of $\geq 5$ mm was defined as the presence of ultrasound attenuation.15 The remodelling index was calculated as the lesion EEM CSA divided by the mean reference EEM CSA and positive remodelling was defined as a remodelling index of $>1.05$.

The integrated backscatter (IB) value for each tissue component is expressed in dB and was calculated using a fast-Fourier transform of the frequency component of the backscattered signal from a small volume of tissue. For tissue characterization, we applied the

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**Figure 2** Representative case of high-intensity plaque. (A) $T_1$-weighted magnetic resonance imaging of high-signal intensity of the right coronary artery with a plaque to myocardium signal intensity ratio of 3.09 (yellow arrow indicates lesion). (B) A coronary angiogram showing severe stenosis in the proximal portion of the right coronary artery. (C) Gray-scale intravascular ultrasound image showing ultrasound attenuation. (D) Integrated backscatter intravascular ultrasound showing lipid-rich plaque (blue area).
manufacture’s default setting on the basis of previous data to define a range of IB values, and coronary plaques were classified into three categories, lipid pool (blue), fibrosis (green/yellow), and calcification (red).

**Statistical analysis**

Continuous variables are reported as mean ± SD or median and interquartile ranges (IQR), as appropriate. The unpaired t-test and Mann–Whitney U-test were used to determine differences between mean values for parametrically and non-parametrically distributed variables, respectively. Categorical variables are reported as absolute values and percentages, and were analysed by Fisher’s exact test. Intraclass correlation coefficients with 95% confidence intervals (CI) were calculated to assess intra- and interobserver agreement of the measurement of PMR, and kappa statistics for agreement of the detection of HIP. The optimal cut-off points for detecting PMI and transient slow flow were determined using receiver-operating characteristic (ROC) curve and the maximum value of Youden’s index [(sensitivity + specificity) − 1]. Variable selection was guided by bootstrap selection, which would provide relatively unbiased estimates of predictive accuracy. A resampling analysis with 1000 iterations was performed to identify the variables that entered into 50% of the logistic regression models to determine the independent predictor of PMI with \( P < 0.05 \) for retention variables.\(^{17}\)

**Results**

**PMR and detection of high-intensity plaque**

Representative images of coronary plaque with HIP and non-HIP are shown in Figures 2 and 3, respectively. The intra- and interobserver intraclass correlation coefficients for PMR were 0.96 (95% CI 0.91–0.98, \( P < 0.001 \)) and 0.94 (95% CI 0.94–0.99, \( P < 0.001 \)), respectively, and the kappa statistics for intra- and interobserver agreement for the detection of HIP were 0.82 and 0.91, respectively, with all values indicating good intra- and interobserver agreement. In all 100 coronary target lesions, HIP on non-contrast T1WI was detected in 36 lesions of the coronary plaques and non-HIP in 64 lesions. When the patient had at least one HIP, we classified into the HIP group; HIP group, \( N = 31 \) patients, and non-HP group, \( N = 46 \) patients (Figure 1). The median and IQR of the PMR were

\( P \)-values of <0.05 were considered significant through this study. Statistical analysis was performed by using the SPSS software, version 22 (SPSS, Inc., Chicago, IL, USA).

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**Figure 3** Representative case of non-high-intensity plaque. (A) \( T_1 \)-weighted magnetic resonance imaging of the right coronary artery with a plaque to myocardium signal intensity ratio of 1.09 (yellow arrow indicates lesion). (B) A coronary angiogram showing severe stenosis in the distal portion of the right coronary artery. (C) Gray-scale intravascular ultrasound image showing a fibrous plaque. (D) Integrated backscatter intravascular ultrasound showing a fibrous plaque (green and yellow area).
1.77 [1.54–2.35] and 1.04 [0.95–1.12] for the HIP and non-HIP groups, respectively (P < 0.001).

Patients, procedures, and plaque characteristics
Baseline patient characteristics are summarized in Table 1. There were no significant differences in the characteristics (age, sex, and cardiovascular risk factors) between the two groups. Patients with the HIP group received statin treatment more frequently than did those in the non-HIP group. Angiographic and coronary plaque characteristics assessed by IVUS are presented in Table 2. In both cross-sectional and volumetric IVUS analyses, the HIP group showed significantly greater EEM CSA/volume, plaque CSA/volume, Cross-sectional IVUS analysis

<table>
<thead>
<tr>
<th>Cross-sectional IVUS analysis</th>
<th>HIP (+)</th>
<th>HIP (−)</th>
<th>P-value</th>
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<tbody>
<tr>
<td>EEM CSA, mm³</td>
<td>16.9 ± 5.6</td>
<td>12.3 ± 4.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Plaque burden, %</td>
<td>62.6 ± 13.1</td>
<td>45.9 ± 30.3</td>
<td>0.039</td>
</tr>
<tr>
<td>Ultrasonic attenuation³</td>
<td>22 (71.0%)</td>
<td>2 (4.3%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Attenuation length, mm</td>
<td>7.2 ± 4.5</td>
<td>0.5 ± 1.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Intracoronary thrombus</td>
<td>10 (34.3%)</td>
<td>1 (2.2%)</td>
<td>0.026</td>
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</table>

Volumetric IVUS analysis

<table>
<thead>
<tr>
<th>Volumetric IVUS analysis</th>
<th>HIP (+)</th>
<th>HIP (−)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEM volume, mm³</td>
<td>223.2 ± 131.8</td>
<td>145.3 ± 86.8</td>
<td>0.003</td>
</tr>
<tr>
<td>Plaque burden, %</td>
<td>11.5 ± 20.1</td>
<td>9.9 ± 16.2</td>
<td>0.001</td>
</tr>
<tr>
<td>Calculated volume, mm³</td>
<td>1.2 ± 1.1</td>
<td>1.1 ± 0.8</td>
<td>0.001</td>
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</table>

Values are mean ± SD or N (%).  
³Positive remodelling was defined as remodelling index of >1.05.  
⁴Ultrasound attenuation was defined as a longitudinal attenuation length of ≥5 mm.
and plaque burden than the non-HIP group. The lipid pool was also significantly larger in the HIP group than that in the non-HIP group. Positive remodelling, ultrasound attenuation, and intracoronary thrombus were more frequently detected in the HIP group than in the non-HIP group.

**Receiver-operating characteristic curve and multivariable analysis for prediction of percutaneous coronary intervention-related myocardial injury**

Although the baseline hs-cTnT levels were similar between the two groups, the increase in hs-cTnT was significantly greater in the HIP group than in the non-HIP group (0.065 [0.023–0.304] ng/mL, P < 0.001, respectively; Table 3 and Figure 4). Percutaneous coronary intervention-related myocardial injury and transient slow flow phenomenon were more frequently detected in the HIP group than in the non-HIP group (PMI: 58.1 vs. 10.9% and transient slow flow: 38.7 vs. 0%, P < 0.001 for both; Table 3 and Figure 5). Table 4 shows similar results when coronary plaques were classified according to the presence of IVUS-derived ultrasound attenuation.

The optimal cut-off point of PMR was found to be 1.44 for predicting PMI, with a sensitivity of 78.3% and specificity of 81.5% (area under the curve, 0.780; Figure 6A). The cut-off point of PMR for predicting transient slow flow was 1.42, with a sensitivity of 91.7% and a specificity of 72.3% (area under the curve, 0.895; Figure 6B).

**Discussion**

In the present study, we evaluated the coronary plaque characteristics of HIP identified by T1WI by using IVUS and investigated the

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**Table 3** Measurement of cardiac troponin-T and incidence of complication

<table>
<thead>
<tr>
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<th>HIP (+) (N = 31)</th>
<th>HIP (-) (N = 46)</th>
<th>P-value</th>
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</thead>
<tbody>
<tr>
<td>Level of hs-cTnT, ng/mL</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Baseline</td>
<td>0.011 [0.007–0.017]</td>
<td>0.010 [0.005–0.020]</td>
<td>0.390</td>
</tr>
<tr>
<td>After PCI</td>
<td>0.100 [0.048–0.314]</td>
<td>0.028 [0.014–0.048]</td>
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<tr>
<td>Increase in hs-cTnT</td>
<td>0.065 [0.023–0.304]</td>
<td>0.017 [0.005–0.026]</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PCI-related myocardial injury</td>
<td>18 (58.1%)</td>
<td>5 (10.9%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Slow flow</td>
<td>12 (38.7%)</td>
<td>0 (0%)</td>
<td>&lt;0.001</td>
</tr>
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</table>

Values are median [interquartile rage] or N (%).
HIP, high-intensity plaque; hs-cTnT, high-sensitive cardiac troponin T; PCI, percutaneous coronary intervention.

**Table 4** Measurement of cardiac troponin-T and incidence of complication based on IVUS-derived ultrasound attenuation

<table>
<thead>
<tr>
<th></th>
<th>IVUS-UA (+) a (N = 24)</th>
<th>IVUS-UA (-) (N = 53)</th>
<th>P-value</th>
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</thead>
<tbody>
<tr>
<td>Magnetic resonance imaging</td>
<td></td>
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<tr>
<td>Presence of HIP</td>
<td>22 (91.7%)</td>
<td>9 (17.0%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PMR</td>
<td>1.77 [1.47–2.29]</td>
<td>1.06 [0.96–1.17]</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Measurement of hs-cTnT</td>
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<tr>
<td>Level of hs-cTnT, ng/mL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>0.011 [0.006–0.017]</td>
<td>0.010 [0.005–0.020]</td>
<td>0.559</td>
</tr>
<tr>
<td>After PCI</td>
<td>0.115 [0.034–0.321]</td>
<td>0.032 [0.018–0.052]</td>
<td></td>
</tr>
<tr>
<td>Increase in hs-cTnT</td>
<td>0.068 [0.022–0.309]</td>
<td>0.020 [0.005–0.034]</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PCI-related myocardial injury</td>
<td>15 (62.5%)</td>
<td>8 (15.1%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Slow flow</td>
<td>11 (45.8%)</td>
<td>1 (1.9%)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Values are median [interquartile rage] or N (%).
HIP, high-intensity plaque; hs-cTnT, high-sensitive cardiac troponin-T; IVUS-UA, intracoronary ultrasound derived ultrasound attenuation; PCI, percutaneous coronary intervention; PMR, coronary plaque to myocardium signal intensity ratio.
aUltrasound attenuation was defined as longitudinal attenuation length of ≥5 mm.
association between HIP and PMI. The major findings included (i) HIPs, defined as a PMR of $\geq 1.4$, were detected in 36% of the coronary plaques examined; (ii) HIPs showed characteristics of vulnerable plaque as assessed by IVUS measurements; and (iii) HIPs were significantly associated with PMI, with a PMR of 1.44 found to be the optimal cut-off value for predicting PMI. Our study is unique in detailed description of IVUS correlating with HIP on T1WI and consequent clinical significance.

Relation between high-intensity plaque and intravascular ultrasound parameters

A few clinical studies have compared coronary plaque MRI on non-contrast T1WI with MDCT, IVUS, and OCT. In comparison with MDCT and MR images of the coronary arteries, HIP on non-contrast T1WI was associated with a high frequency of plaque with low CT density. Kawasaki et al. reported an average minimal CT density of $-23.2 \pm 20.7$ Hounsfield units (HU) in HIP lesions, but $9.6 \pm 20.5$ HU in non-HIP lesions. They also showed that coronary HIP was associated with ultrasound attenuation and vessel positive remodelling by gray-scale IVUS. In contrast, Ehara et al. showed that HIP was associated with intracoronary thrombus on OCT. Referring to these previous studies, coronary plaque with a PMR of $>1.0$ was defined to be positive for HIP. Using this definition, as previously reported in these studies with small sample size (Kawasaki et al., $n=25$ lesions and Ehara et al., $n=26$ lesions), the prevalence of HIP was rather high (60–70%) even for patients with stable or unstable angina pectoris. Therefore, the optimal cut-off point of PMR for determining vulnerable plaque remains to be elucidated. Recently, Noguchi et al. showed that the presence of HIP (plaques with a PMR of $\geq 1.4$) on non-contrast T1WI is significantly associated with future coronary events. Therefore, we opted to use the definition of HIP as coronary plaques with a PMR of $\geq 1.4$ in this study. On this basis, we detected HIP in 36% of the coronary plaques in patients with stable angina pectoris. We showed with IVUS assessment that HIP was significantly associated with ultrasound attenuation and positive remodelling. Additionally,
we demonstrated for the first time, to our knowledge, that the lipid pool was significantly greater in the patients with HIP based on IB-IVUS assessment. These IVUS findings would generate that HIP is correlating with the characteristics of vulnerable plaque.

**Tissue characteristics of high-intensity plaque**

Carotid endarterectomy specimens provide histological validation of carotid atherosclerotic lesions as the gold standard for assessing MR images. High-signal intensity of carotid plaque is reported to correlate with cerebral ischaemic events and is recognized as a vulnerable feature. On the basis of carotid MR studies, the presence of high-signal intensity lesions is considered to indicate methaemoglobin in intraplaque haemorrhage. Methaemoglobin, a product form the maturation of thrombus, leads to a significant shortening in $T_1$-relaxation time. Lipid-rich necrotic carotid plaque also shows a high-intensity signal on T1WI sequences, because intraplaque haemorrhage often occurs within lipid-rich necrotic cores. Recent case report described that coronary aspirated specimen with HIP on T1WI shows a large amount of the necrotic core with overlying platelet- and fibrin-rich thrombus, supporting our speculation. Accordingly, we speculate that coronary plaque with HIP on non-contrast T1WI may represent vulnerability, comprising components of an intraplaque haemorrhage, a lipid-rich necrotic core, a thrombus formation, or a mixture of these. However, the actual cause of high-signal intensity in atherosclerotic coronary plaque on T1WI remains under discussion. Further studies are needed to clarify the coronary plaque characteristics representing HIP on non-contrast T1WI.

**Predictive value of high-intensity plaque for detection of high-sensitive cardiac troponin-T elevation**

A cardiac biomarker such as hs-cTnT is sensitive and specific for the detection of myocardial damage. Several previous reports have...
PCI, minor elevations in cardiac enzymes, troponin T or I, occur in disease in a prospective observational study. Additionally, Asaumi coronary events among patients with suspected coronary artery of identifying vulnerable plaque because coronary plaque with a PMR predicting. We believe that this cut-off point may be suitable for and a PMR of 1.44 was found to be the optimal cut-off point for presence of HIP on T1WI was significantly associated with PMI, PCI, while resulting in relatively modest hs-cTnT elevation. The patients with stable angina pectoris who underwent non-emergent PCI, MDCT, MRI does not involve exposure to ionizing radiation. The associated with cardiac imaging have raised concerns. Unlike the potential risks of exposure to the ionizing radiation and simultaneous assessment of coronary plaque composition. Coronary MDCT has been a promising and widely expanding modality in the diagnosis of coronary artery disease and simultaneous assessment of coronary plaque composition. However, the potential risks of exposure to the ionizing radiation associated with cardiac imaging have raised concerns. Unlike MDCT, MRI does not involve exposure to ionizing radiation. The MR assessment of coronary plaques can be easily combined with radiation-free MR of cardiac function, perfusion, scar imaging, and more, as the comprehensive approach represents another major advantage of MR over MDCT. We believe that coronary plaque imaging with T1WI is a valuable non-invasive modality to assess the risk of myocardial damage without exposure to radiation or nephro-toxic contrast agents.

**Limitations**

This study has several limitations. First, the main limitation was the lack of standardization and quantitative nature of diagnosing HIP by MRI, which is based on relatively ‘eye balling’ nature of this diagnosis and manual ROI tracings. Secondly, it was conducted at a single centre, and the study population was relatively small. Thus, the results need to be confirmed with a larger multicentre study. Thirdly, although we analysed coronary plaque characterization by using IB-IVUS parameters, histological validation of coronary plaque with HIP is lacking because it is difficult to obtain coronary plaque specimens from human coronary arteries in vivo. Precise characterization of coronary HIP remains unknown. In addition to T1WI, simultaneous multiple MRI sequences (such as T2-weighted, proton density-weighted, or time-of-flight sequence) would be potentially useful for interpreting more precise coronary plaque characterization with a comprehensive/holistic approach. Fourthly, in this study, the MR assessment was performed only before PCI. When T2-weighted cardiac MRI was preformed post-PCI, it might have detected PCI-related myocardial oedema due to microvascular injury. Fifthly, in this study, statin therapy was more frequently in the HIP group than in the non-HIP group (97 vs. 76%). The results may have more striking if statin therapy had been balanced between both groups. Because the use of high-dose statin during or before the procedure might prevent PMI. Finally, we excluded patients with severely calcified lesions because of the difficulty in evaluating plaque composition behind calcification artefacts by IVUS. Therefore, our results cannot be extrapolated to all patients with coronary artery disease.

**Conclusions**

The present study demonstrated that coronary HIP on non-contrast T1WI may reflect the potential for plaque vulnerability on IVUS assessment, and that HIP may play a pivotal role in predicting the incidence of PMI.

**Acknowledgements**

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**Conflict of interest:** None declared.

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