High-Intensity Plaques on Noncontrast T1-Weighted Imaging as a Predictor of Periprocedural Myocardial Injury

Although percutaneous coronary intervention (PCI) routinely achieves good angiographic success, 5% to 30% of patients experience periprocedural myocardial injury (pMI), which is associated with long-term adverse outcomes and immediate adverse events (1). Because coronary high-intensity plaques (HIPs) detected by noncontrast T1-weighted imaging (T1WI) represent plaque instability (2,3), we examined the relationship between cardiac magnetic resonance (CMR) characteristics of HIPs and pMI following elective PCI in patients with coronary artery disease (CAD).

Between October 2012 and March 2014, 57 patients with CAD (mean age 68 ± 11 years) underwent CMR within 3 months (median 2 days; interquartile range [IQR]: 1 to 24 days) of PCI. The plaque-to-myocardial signal intensity ratio (PMR) was calculated (2,3). pMI during PCI was defined as an increase in serum cardiac troponin T (cTnT) levels to more than 5 times the upper limit of normal (0.07 ng/ml) at 24 h after PCI. Intravascular ultrasound (IVUS) images were also obtained. All patients were scanned with a 3-T MR imager (MAGNETOM Verio, Siemens AG Healthcare, Erlangen, Germany) equipped with a 32-channel cardiac coil. Plaque imaging (Figure 1A) was performed using an inversion recovery prepared 3-dimensional T1W turbo FLASH (fast low angle shot) sequence with electrocardiogram-triggered, navigator-gated free breathing and fat suppression in transaxial sections covering the entire heart (inversion time 650 ms; field of view 280 × 228 mm; acquisition matrix 256 × 187; reconstruction matrix 512 × 374; acquisition slice thickness 1.0 mm; acquisition slice number 104 to 120; reconstruction spatial resolution 0.6 × 0.5 × 0.6 mm; fat suppression; effective repetition time/echo time 4.7 ms/2.13 ms; flip angle 12°; GRAPPA factor 2; navigator gating window ±1.5 to 2.5 mm; and data acquisition window duration time 84 to 120 ms). The trigger delay and acquisition window were set according to the phase with minimal motion of the right coronary artery as determined using cine MR imaging.

Clinical characteristics, cTnT levels at baseline, target vessels, or type B2/C lesions were comparable between the non-pMI (n = 42) and pMI groups (n = 15). All lesions were treated with stent implantation. Transient slow or no reflow was observed significantly more frequently in patients with pMI than those without (27% vs. 5%; p = 0.036). Importantly, the median (IQR) of the PMR of PCI lesions was higher in patients with pMI than those without (1.3 [1.1 to 2.0] vs. 1.0 [0.8 to 1.2]; p = 0.014) (Figure 1B). Receiver-operating characteristic (ROC) analysis revealed that the optimal PMR cutoff value for predicting pMI was 1.3 and the area under the ROC curve was 0.71 (Figure 1C).

At this value, the sensitivity and specificity for predicting pMI were 67% and 86%, respectively. The present cutoff value of PMR ≥1.3 for pMI is consistent with our previous finding that PMR ≥1.4 predicts future cardiac events (3). In the univariate logistic regression analysis, PMR ≥1.3 (odds ratio [OR]: 12.0; 95% confidence interval [CI]: 3.2 to 52.2; p < 0.001), ultrasound attenuation (attenuation arc $\leq 228$ mm; acquisition matrix 256 × 187; reconstruction matrix 512 × 374; acquisition slice thickness 1.0 mm; acquisition slice number 104 to 120; reconstruction spatial resolution 0.6 × 0.5 × 0.6 mm; fat suppression; effective repetition time/echo time 4.7 ms/2.13 ms; flip angle 12°; GRAPPA factor 2; navigator gating window ±1.5 to 2.5 mm; and data acquisition window duration time 84 to 120 ms). The trigger delay and acquisition window were set according to the phase with minimal motion of the right coronary artery as determined using cine MR imaging.

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FIGURE 1  High-Intensity Plaques on Noncontrast T1-Weighted Imaging Predicts Periprocedural Myocardial Injury During Percutaneous Coronary Intervention

(A) Representative cases of high-intensity plaques (HIPs) (a, b, c) and non-HIPs (d, e, f). A HIP (a, yellow arrow) and a non-HIP (d, yellow arrow) are seen in the proximal left anterior descending (LAD) artery on noncontrast T1-weighted imaging, with a plaque-to-myocardial signal intensity ratio (PMR) of 2.3 and 1.0, respectively. These lesions correspond to LAD plaques on computed tomography angiography (b and e, yellow arrowheads) and coronary arteriography (c and f, yellow arrowheads). (B) Comparison of PMR between patients with and without periprocedural myocardial injury (pMI). (C) Receiver-operating characteristic curve analysis for predicting pMI.

(A) Letters to the Editor
JACC: CARDIOVASCULAR IMAGING, VOL. 8, NO. 6, 2015
JUNE 2015:741–9

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http://dx.doi.org/10.1016/j.jcmg.2014.07.020

Please note: Mr. Komori is an employee of Siemens Japan KK. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.
Progression of coronary artery calcium (CAC) as measured by computed tomography independently predicts coronary heart disease (CHD) events (1). Several methods have been proposed to define and quantify CAC progression yet very few studies have compared different CAC progression definitions (2,3). We sought to determine the concordance between different CAC progression definitions and their association with traditional CHD risk factors.

The Cooper Center Longitudinal study is a cohort of >100,000 individuals referred for preventive medical examination in Dallas, Texas. This report includes 6,597 individuals (mean age 51.8 years, 22.8% female) who underwent serial cardiac computed tomography scanning (Imatron C-150XP or C-300, Siemens, Malvern, Pennsylvania) at least 9 months apart between the years of 1998 and 2006 (mean interval 2.9 years). Individuals with previous CHD or coronary intervention between scans were excluded. Participants provided informed consent and the study was approved by the institutional review board of the Cooper Institute. CAC incidence (development of CAC in individuals with baseline CAC = 0) and CAC progression (increasing CAC in individuals with baseline CAC > 0 in Agatston units [AU]) were analyzed separately. Various progression definitions were applied as originally described (2-4):

1. Continuous progression (annualized):
   a. Absolute change: CACfollow-up − CACbaseline
   b. Log transformed change:
   \[
   \ln(CAC_{\text{follow-up}} + 25) - \ln(CAC_{\text{baseline}} + 25)
   \]
   c. Square root transformed change:
   \[
   \sqrt{CAC_{\text{follow-up}}} - \sqrt{CAC_{\text{baseline}}}
   \]
   d. Percentage of change:
   \[
   \left(\frac{CAC_{\text{follow-up}} - CAC_{\text{baseline}}}{CAC_{\text{baseline}}}\right) \times 100
   \]

2. Categorical progression:
   a. Hokanson method:
   \[
   \sqrt{\frac{CAC_{\text{follow-up}}}{\text{CAC}_{\text{baseline}}}} > 2.5
   \]
   b. Raggi method:
   \[
   \frac{|(CAC_{\text{follow-up}} - CAC_{\text{baseline}})|}{CAC_{\text{baseline}}} > 15\% / \text{year}
   \]
   c. Berry method:
   \[
   \frac{(CAC_{\text{follow-up}} - CAC_{\text{baseline}})}{CAC_{\text{baseline}}} > 10 \%/ \text{year}
   \]

CAC progression was also analyzed according to commonly used risk categories (i.e., 0, >0 to <10, 10 to <100, 100 to <400, and ≥400 AU). At baseline, 3,336 participants (50.6%) had CAC scores of 0 and in this group, 520 (15.6%) had a positive follow-up score. Among those with baseline CAC >0 (n = 3,259), the median annualized change in CAC was 17.7 AU [interquartile range: –32.4 to 67.8 AU].

Applying categorical methods, the binary classification of CAC progression was as follows: 44.2% progressed by Hokanson (45.7% in men, 33.0% women; 35.0% in baseline CAC <100 AU, 55.0% CAC ≥100 AU); 57.1% by Raggi (57.1% in men, 57.4% women; 67.8% in baseline CAC <100 AU, 40.1% CAC ≥100 AU) and 52.5% by Berry (53.8% in men, 43.1% women; 44.0% in baseline CAC <100 AU, 62.3% CAC ≥100 AU). Cross-tabulation between the most commonly referenced methods, Hokanson and Raggi (2,3), revealed modest concordance (70.5%) with a kappa coefficient of 0.420. Concordance estimates stratified by age, sex, and baseline CAC score were: 70.1% for age <50 years, 70.4% age 50 to <100 years; 67.3% age ≥100 years; 66.7% for CAC <100 AU, 74.9% CAC ≥100 AU. When stratified by risk categories, 27.9% moved to a higher risk group, 0.7% moved to a lower risk group, and 71.4% remained in the same group.

Using logistic and linear regression models, the predictors of CAC progression varied across different definitions (Table 1). Notably, the annualized percentage of change method did not show consistent association with most well-established predictors of CHD.

The existing CAC progression definitions are based on a variety of principles including interscan variability, observed relative changes from case-control studies, and arbitrary yet logical constructs, so it is not surprising that they result in divergent classification. There are no formal comparisons between different definitions of CAC progression related to CHD events, just all-cause mortality (2). Though our study is unable to identify a superior method, it does highlight important limitations of definitions based on