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EDITORIAL COMMENT

Predicting Periprocedural Myocardial Infarction

Target-Lesion Plaque Characterization With Coronary Computed Tomography Angiography*

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Periprocedural myocardial infarction (MI) is a well-recognized complication of percutaneous coronary intervention (PCI) occurring in approximately one-third of elective cases (1). Mild-to-moderate elevations in cardiac biomarkers after PCI have been shown to be associated with myocardial scar detectable on magnetic resonance imaging (2,3) and up to a 35% increase in mortality (1); yet whether post-procedural troponin elevation serves as an independent predictor of survival is debatable and has not been definitively established (4,5). Regardless, it seems the ability to identify lesion and vessel characteristics that place patients

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at risk for future procedural-related major adverse cardiac events would offer interventionalists valuable insight that might be helpful even in routine revascularization procedures. Prior identification of plaques responsible for these events would allow prognostic stratification and prompt initiation of targeted actions to prevent myocardial necrosis, namely through the use of distal embolic protection devices (6) and upstream loading with high-dose atorvastatin (7,8). Furthermore, as more PCI procedures are being done with same-day discharge, a "high-risk" plaque could be a candidate for overnight stay to evaluate for possibility of post-procedural troponin elevation.

Although many etiologies exist for periprocedural MI related to PCI, including but not limited to side-branch occlusion, dissection, and abrupt stent closure, it is distal embolization of plaque debris or thrombus at the time of balloon inflation or stent deployment that is generally felt to be the predominant mechanism. The significant association between periprocedural MI and adverse long-term clinical outcomes has led to the development of a variety of invasive and noninvasive imaging tools all aimed at safely and accurately identifying salient features of this pathological substrate predisposed to embolization. Several studies have examined the relationship between virtual histology-intravascular ultrasound (VH-IVUS)-derived measures of plaque volume and composition and rates of distal embolization (evidenced by ST-segment re-elevation and transient high-intensity signals on intracoronary Doppler) after PCI in patients with acute MI, unstable angina, or stable angina (9–12). Positive correlations were demonstrated between plaque necrotic core volume and these surrogate markers (9,10). Plaque necrotic core area emerged as an independent predictor of periprocedural infarction in patients with stable angina undergoing elective coronary stenting on the basis of the direct assessment of levels of cardiac biomarkers 18 to 24 h after the procedure (11,13). More recently, Uetani et al. (14) reported similar findings with coronary computed tomography angiography (CCTA)-based plaque quantification in 189 consecutive patients undergoing planned coronary intervention. The volume and fraction of low-attenuation plaque (<50 Hounsfield units [HU]) in target lesions was found to be independently associated with periprocedural infarction after adjustment for multiple confounders. Interestingly, the fraction of moderate attenuation (50 to 150 HU) or fibrous plaque inversely correlated with post-procedural levels of cardiac biomarkers.

As reported by Watabe et al. (15) in this issue of the *Journal*, CCTA-based plaque compositional analysis of culprit lesions in 107 patients with stable angina and normal cardiac troponin T levels was performed before PCI. Post-PCI troponin elevation was observed in 36 of 107 patients (33.6%). Positive remodeling (remodeling index >1.05) and spotty calcification were found to be statistically significant independent predictors of cardiac troponin T elevation ≥3× the upper limit of normal (0.010 ng/ml) 24 h after the procedure, whereas the presence of the 2 plus the finding of a computed tomography attenuation value <55 HU demonstrated a corresponding positive predictive value of 94% and a negative predictive value of 90% for said rise. Transient no-reflow occurred more frequently in patients with troponin release. Importantly, none of the conventional risk factors afforded differentiation between patients with and without post-PCI troponin elevation—observations also noted in a prior multidetector computed tomography (MDCT) study (16) and near-infrared spectroscopy data (17). The investigators conclude that "MDCT might play an important role in detecting lesions at high risk for myocardial necrosis after PCI." Of course, the ability to modify this result or intervene against this troponin rise must be prospectively established. Multidetector computed tomography, although resulting in a markedly lower radiation dose than either nuclear imaging or

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invasive angiography, still affords some cost, radiation, and contrast use for the patient undergoing the procedure.

Results in the current study echo prior findings (12,14) and suggest that target-lesion plaque composition might have a powerful impact on myocardial injury after PCI and that plaque evaluation on MDCT might play an important role in planning coronary interventions. It is interesting to note the high frequency of positive remodeling and spotty calcification in the plaques prone to embolization in these stable angina patients, given that these same features are often associated with so-called vulnerable plaques on VH-IVUS (18).

Furthermore, the small patient cohort is a notable limitation, as is the lack of concurrent VH-IVUS imaging of culprit lesions for comparison, at present the only imaging technique validated to identify patients and lesions at risk for future major adverse cardiac events. Coronary computed tomography angiography offers the possibility of identifying vulnerable atherosclerotic plaques noninvasively. Validation cohorts and outcome studies would certainly bolster the enthusiasm for this technique before routine use in this capacity. Recent rapid advancements in scanner technology have facilitated the development of manual and automated methods of plaque quantification and characterization on computed tomography. These techniques are not without their own inherent limitations, and future studies comparing CCTA with VH-IVUS and ultimately with histopathological features are necessary to validate the clinical utility of this technology.

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Key Words: coronary plaque ■ multidetector computed tomography ■ stents ■ troponin.