Percutaneous coronary intervention (PCI) is an established treatment strategy for acute coronary syndrome. However, periprocedural myocardial infarction (PMI), which occurs in 5–50% of PCI procedures, has prognostic implications for long-term clinical outcomes. Coronary imaging modalities such as intravascular ultrasound (IVUS) and optical coherence tomography (OCT) are expected to predict PMI. IVUS is widely used to safely and reliably guide PCI. Several studies suggest that IVUS-derived coronary plaque characteristics, such as a large plaque burden (positive remodeling), echo-attenuated plaque (EA), or plaque with necrotic core, are associated with the risk of PMI.\(^1\)\(^,\)\(^2\) However, IVUS has an axial resolution of 100–200 μm and a lateral resolution of 250 μm,\(^3\) whereas OCT is a high resolution imaging modality for precise assessment of plaque structures and tissue characteristics with 10-fold the axial and lateral resolution of IVUS.\(^4\) Recent studies showed the presence of thin-cap fibroatheroma (TCFA) at pre-PCI OCT and of intrastent thrombus and dissection at post-PCI OCT as independent predictors of PMI.\(^5\)\(^,\)\(^6\) Although IVUS and OCT can provide useful tomographic information of coronary arteries in vivo, both modalities have disadvantages. IVUS has limited plaque characterization and resolution, and OCT has poor tissue penetration. Under these conditions, assessment of positive remodeling, which is one of the main findings by IVUS for plaque vulnerability, is difficult by OCT. Combined use of both modalities may be complementary.\(^7\)

The opinions expressed in this article are not necessarily those of the editors or of the Japanese Circulation Society.

Received July 20, 2015; accepted July 20, 2015; released online August 7, 2015

Second Department of Internal Medicine, School of Medicine, University of Occupational and Environmental Health, Kitakyushu, Japan

Mailing address: Shinjo Sonoda, MD, PhD, Second Department of Internal Medicine, School of Medicine, University of Occupational and Environmental Health, 1-1 Iseigaoka, Yahatanishi-ku, Kitakyushu 807-8555, Japan. E-mail: s-sonoda@med.uoeh-u.ac.jp


All rights are reserved to the Japanese Circulation Society. For permissions, please e-mail: cj@j-circ.or.jp
The occurrence of PMI can be detected by measuring cardiac biomarkers before the procedure, repeated 3–6 h later and, optionally, further re-measurement 12 h thereafter. In earlier studies, increased values of post-procedural cardiac biomarkers, especially creatine kinase MB isoenzyme (CKMB), were associated with impaired outcome. Based on the third universal definition of MI, elevations of cTn $>5\times99$th percentile upper reference limit (URL) occurring within 48 h of the PCI procedure, in addition to evidence of prolonged ischemia (>20 min) as demonstrated by prolonged chest pain, or ischemic ST changes, or new pathological Q waves, or angiographic evidence of a flow limiting complication, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality, is defined as PCI-related MI (type 4a) in patients with normal baseline cardiac troponin (cTn) concentrations. This threshold of high-sensitivity cTn values $>5\times99$th percentile URL is arbitrarily chosen, based on clinical judgement and societal implications of the label of PMI.6 Cardiac biomarker elevations are suggested to be linked with increased mortality. However, small and asymptomatic release of high-sensitivity cTn is sometimes defined as PMI after successful coronary intervention. Whether such a small PMI based on elevation of high-sensitivity cTn influences long-term mortality remains to be determined.7

In the study by Kimura et al in this issue of the Journal, the combined use of IVUS and OCT was useful to predict occurrence of PMIs in patients with stable angina pectoris (SAP) undergoing elective PCI.8 The OCT data showed that lesions with EA had larger lipid-rich plaques, thinner fibrous cap, and less and thinner calcification than those without EA. However, some lesions with EAs had stable plaque without TCFA. Some lesions with TCFA did not have unstable plaque without EA. Both IVUS-derived EA and OCT-derived TCFA findings showed higher predictive value of PMI, which was defined by elevation of high-sensitivity cTn T values than each single variable (Figure). The results of this study are consistent with those of a recent histopathological validation study in which neither IVUS nor OCT was optimal for detecting TCFA.11 The authors’ conclusion is that the combined use of IVUS and OCT may improve TCFA detection accuracy in the clinical setting. A recent study demonstrated a hybrid intravascular imaging catheter with dual IVUS and OCT capabilities.12 Furthermore, the present study showed that the frequency of EAs accompanied by lipid-rich plaques and TCFAs was lower, but not uncommon, in SAP patients than in ACS patients. The patients with PMI showed worse outcomes during the follow-up period than those without among the SAP patients. A previous study also demonstrated the relationship between PMI and poor outcome during a 1-year post-PCI follow-up in patients with SAP.5 PMI based on high-sensitivity cTn T elevations may correlate with the total coronary plaque burden and may reflect the instability of atherosclerotic plaque. This study has several important limitations. First, it did not demonstrate direct relationships between IVUS and OCT plaque morphologies and adverse cardiac events. Moreover, patients with PMI had higher frequencies of major adverse cardiac events, exclusively congestive heart failure (CHF), during follow-up than those without PMI. There was no significant difference in the incidence of unstable angina, nonfatal MI, ischemic target vessel revascularization, and cardiac death between the 2 groups. The majority of patients with CHF after PMI had left ventricular diastolic dysfunction and/or renal dysfunction at baseline. If CHF is included as a major adverse cardiac event after PMI, it may be difficult to predict long-term outcomes after PMI by only assessing pre-procedural plaque morphology, because CHF after PMI is multifactorial in origin. Second, the authors excluded patients treated with distal protection devices, those with final angiographic evidence of side branch occlusions, coronary dissection, or slow-reflow phenomenon after PCI. Therefore, they may have only analyzed low-risk patients. Third, the study included SAP patients with elevated baseline levels of high-sensitivity cTn T who had more vulnerable plaque and possibly should have been excluded from the analysis.

In conclusion, Kimura et al have made a great contribution to identifying the efficacy of “two eyes (combined IVUS and OCT assessments) more than one” for predicting PMIs in SAP patients undergoing elective PCI. The next crucial questions are when and how to prevent or reduce PMI. Many studies can be designed to solve them.

References