large ROH; type II, by a small ROH; and type III, by short leaflet lengths. STJ/annulus area ratio correlated well with ROH regardless of AR type. ROH was the highest discriminatory index for types I and II. This report is not a technical validation of the accuracy of this software for quantitative analysis of AVAp. However, the previous study using this software program to compute precise morphological quantification of AVAp from 3D TEE and computed tomography data demonstrated a strong intermodality and intersubject correlation (2). As a clinical implication, the present study by 3D TEE may potentially help the surgical team to recognize in-depth anatomic abnormalities or the classification with convincing quantitative information for surgical planning.

Kentaro Shibayama, MD,* Hiroyuki Watanabe, MD, Shunsuke Sasaki, MD, Keitaro Mahara, MD, Minoru Tabata, MD, Toshihiro Fukui, MD, Shuichiro Takanashi, MD, Tetsuya Sumiyoshi, MD, Hitonobu Tomoihe, MD, Takahiro Shiota, MD

*Cedars-Sinai Heart Institute, 127 South San Vicente Boulevard, Suite A3411 (Takahiro Shiota), Los Angeles, California 90048. E-mail: shibao_k@hotmail.com

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Coronary Plaque Composition and Post-PCI Complications in NSTEMI

The ability to identify those characteristics of coronary plaque morphology that are associated with a higher risk for microvascular impairment and myocardial injury after percutaneous intervention (PCI) may provide an opportunity to improve clinical outcomes in patients with non-ST-segment myocardial infarction (NSTEMI) treated with PCI (1,2). We investigated the impact of virtual histology intravascular ultrasound (IVUS)-veriﬁed pre-PCI coronary plaque composition on peri-procedural microvascular function as assessed using microvascular resistance (μVR) measurements in patients with NSTEMI undergoing PCI.

Thirty-four patients were enrolled (Fig. 1). Serum troponin T (TnT) levels were measured on admission, immediately prior to and 6 hours after PCI. The target coronary lesion was crossed by a dual-sensor-equipped guidewire (Doppler and Pressure Combo Wire, Volcano Corporation, San Diego, California). Prior to PCI and in the presence of epicardial stenosis, microvascular resistance (pre-PCI μVR) was calculated with coronary wedge pressure (Pw)-based correction, as follows: Pa(1/APV)[(Pd-Pw)/(Pa-Pw)], where Pa denotes aortic pressure, Pd is distal coronary pressure, and APV is an average peak velocity. After obtaining pre-PCI hemodynamic measurements, IVUS measurements were recorded as described elsewhere (3). Post-PCI hyperemic μVR was calculated as Pa/APV. The peri-procedural change in μVR (ΔμVR) was calculated as Post-PCI μVR – Pre-PCI μVR. Paired t test and linear regression analysis were used where appropriate. Normal distribution was achieved for TnT values by logarithmic transformation. Multivariate linear regression analysis was applied to identify the independent predictors of ΔμVR, including pre-PCI μVR, all IVUS parameters, total plaque volume, lesion and stent lengths, angiographic percent diameter stenosis, and the presence of thrombus on angiography.

There was a significant correlation between ΔμVR and ΔTnT (r = 0.454; p = 0.03). The necrotic core/dense calcium ratio was the only VH-IVUS characteristic correlated with pre-PCI μVR (r = 0.735; p < 0.001). Similarly, the percent of necrotic core volume (%NCV) was the only IVUS parameter correlated with ΔμVR (r = 0.601; p < 0.001) and post-PCI μVR (r = 0.55; p = 0.002). ΔTnT was correlated with %NCV (r = 0.473; p = 0.015). In the multivariate model, ΔμVR was predicted by %NCV (β = 0.472; p = 0.01) followed by pre-PCI μVR (β = 0.343; p = 0.045). The best cutoff value of %NCV for the prediction of an increase in post-PCI μVR was 18, with a sensitivity of 84% and a specificity of 66% (area under the curve = 0.826; 95% confidence interval: 0.67 to 0.96).

This study demonstrated that in patients undergoing PCI for NSTEMI, the ratio of necrotic core to dense calcium was associated with the extent of baseline pre-PCI microvascular injury, and that %NCV in the culprit lesion predicted the extent of post-PCI increase in μVR. In other words, the features suggestive of greater lesion instability were associated with more extensive microvascular obstruction at presentation, as well as PCI-related impairment of microvascular perfusion in NSTEMI. Although previous IVUS studies have demonstrated a relationship between necrotic core component and post-PCI embolization (4), in the current study, the extent of PCI-related microvascular impairment, which was assessed precisely by the difference between preand post-PCI MR values, was shown to have been predicted by the percentage (but not the absolute amount) of necrotic core volume across the entire lesion segment. Moreover, the status of microcirculation at baseline also emerged as one of the major determinants of peri-procedural microvascular injury, supporting the findings of a recently published study (5). Nonetheless, the results of this study should be interpreted with consideration of the limitation of relatively small patient numbers, and therefore additional predictors of baseline and post-PCI μVR may not have been detected.

In summary, in the NSTEMI setting, the proportion of necrotic core content in the target lesion was associated with the extent of both pre- and peri-procedural microvascular dysfunction and post-procedural myocardial. The pre-procedural identification of those coronary plaques most likely to induce microvascular injury during the PCI procedure may allow the strategies of protection of the microvasculature at the time of PCI to improve clinical outcomes in patients with NSTEMI.

Cansu Akdeniz, MD, Sabahattin Umman, MD, Abdullah Kaplan, MD, Yılmaz Nisanci, MD, Berrin Umman, MD, Zehra Bugra, MD, Emre Aslanger, MD, Derek J. Hausenloy, MD, PhD, Akar Yilmaz, MD, Nihat Polat, MD, Murat Sezer, MD*
Figure 1. Flow Chart
Patients and the relationships between microvascular resistance (MR) and coronary plaque composition. CFR = coronary flow reserve; FFR = fractional flow reserve; h-SR = hyperemic stenosis resistance.

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