in ACS lesions. Inaccurate detection of the borders shared by thrombus, plaque, and lumen may introduce large measurement errors of plaque composition. The site with the minimum lumen diameter may be misidentified. Thrombus may be misclassified as fibrous plaque, proportionally increasing this plaque component at the expense of the others. Thus, errors in border detection may lead to spurious conclusions about culprit plaque composition in ACS. Second, methods to define and measure coronary plaque vary widely, contributing to divergent results and conclusions. Surmely et al. do not define fixed margins for their region of interest. Moreover, they report relative plaque composition as calculated by the VH-IVUS software, a ratio of the absolute area of each plaque component to an estimated plaque area, one that excludes an estimated medial area. It is more appropriate to report relative plaque composition as the ratio of absolute area of each plaque component to the area bound by lumen and the external elastic membrane, as this utilizes direct measurements. Third, VH-IVUS-derived thin cap fibroatheroma (TCFA) is not yet a validated surrogate for plaque prone to thrombosis. VH-IVUS-derived TCFA utilizes direct measurements. Third, VH-IVUS-derived TCFA is not yet a validated surrogate for plaque prone to thrombosis. VH-IVUS-derived TCFA

is loosely based on the histopathological component to the area bound by lumen and as the ratio of absolute area of each plaque calcified by VH-IVUS. Complicating matters, Surmely et al. define TCFA differently from others. Larger prospective longitudinal studies, such as PROSPECT, may determine whether any VH-IVUS-derived definition of plaque phenotype has sufficient positive or negative predictive value for coronary thrombosis. Until then, it seems premature to conclude, as does the accompanying editorial, that a plaque with only fibrous content by VH can be considered as stable with minimal risk of causing thrombosis. To advance the invasive detection of vulnerable plaque, the research community should adopt standards to measure thrombus-associated plaque, calculate plaque composition, and define vulnerable plaque. We believe that with such standards investigators will be at lower risk of experimental error.

References

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Coronary plaque composition of culprit/target lesions according to the clinical presentation: a virtual histology intravascular ultrasound analysis

With great interest I read the article by Surmely et al.1 comparing plaque composition of patients with acute coronary syndrome (ACS) and stable angina pectoris (AP) using virtual histology intravascular ultrasound (VH-IVUS). The authors reported that ‘At the minimal lumen site, . . . necrotic core and dense calcium plaque area were smaller

in ACS lesions (Necrotic core: 6.8 ± 6.0 vs. 11.0 ± 8.3%, P = 0.02; Dense calcification: 2.6 ± 3.0 vs. 4.9 ± 5.8%, P = 0.03).’ However, the authors previously reported that ‘the frequency of necrotic core was significantly higher in the ACS group than in the stable AP group (in vitro histopathology: 22.6% vs. 12.6%, P = 0.02; in vivo virtual histology: 24.5% vs. 10.4%, P = 0.002)2 in another study.’ Given both studies conducted were of similar design and from the same institute, the patient/lesion characteristics seem similar. Potential explanations for the contradictions include the difference of the IVUS system used; a 30 MHz mechanically rotating catheter (Boston Scientific Scimed Inc., Maple Grove, MN, USA) in the previous study and a 20 MHz phased-array IVUS catheter (Eagle Eye, Volcano therapeutics, Rancho Cordova, CA, USA) in this study. Previous studies with grey-scale IVUS imaging have suggested that there is a significant difference in image representation among the IVUS systems studied in the diagnosis of tissue components of complex atherosclerotic plaque.2,4 Therefore, it would be of great help if the authors would provide VH-IVUS data comparing these two IVUS systems in same patient/lesion.

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