Evaluation of Plaque Characteristics in Acute Coronary Syndrome: Non-invasive Assessment with Multi-slice Computed Tomography and Invasive Evaluation with Intravascular Ultrasound Radiofrequency Data Analysis

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Review

The most common cause of acute coronary syndromes (ACS) seems to be plaque rupture which can be simultaneously developed in the entire coronary arteries (1, 2). Therefore, early detection of vulnerable plaques in the culprit or non-culprit arteries is very important to prevent recurrent cardiovascular events (3). Intravascular ultrasound (IVUS) is an invasive tool widely used to assess atherosclerotic plaque through a transducer at the tip of an IVUS catheter. Nair et al. (4) reported that IVUS could discriminate atherosclerotic plaque into four distinct components: fibro-fatty plaque; fibrous plaque; calcified necrotic plaque; and calcified plaque without necrosis. Recently, atherosclerotic plaque characteristics have been studied by multi-slice computed tomography (MSCT) coronary angiography (3, 5). These studies showed that CT-low-density plaques were more frequently found in the culprit as well as non-culprit vessels of patients with ACS

"ASCI's Choice" of this month was about a comparison study between non-invasive MSCT and invasive virtual histology (VH) IVUS for the evaluation of plaque characteristics in patients with ACS reported by Pundziute G, et al. This study reported that non-calcified and mixed plaques were more frequently found in patients with ACS as compared with those with stable coronary artery disease, while the prevalence of completely calcified plaques was lower in patients with ACS on MSCT. Although previous studies have already reported similar observations on MSCT (3, 5), this study identified a larger amount of necrotic core and a higher prevalence of thin cap fibroatheroma (TCFA) with VH IVUS in the plaques of ACS patients. A large amount of necrotic core and the presence of TCFA were closely related to plaque vulnerability, which was already confirmed by previous pathological studies (6).

Distribution of non-calcified and mixed plaques on MSCT was similar in both culprit and non-culprit vessels of patients with ACS. VH IVUS also showed similar observations that no differences were found with the amount of necrotic core and the distribution of TCFA in both culprit and non-culprit vessels. These results may suggest the possibility of a pan-coronary

distribution of potentially vulnerable plaques in patients with ACS, which can lead to recurrent cardiovascular events during follow-up period after the initial presentation of coronary artery disease (7, 8). These findings of plaque vulnerabilities in culprit and non-culprit vessels on MSCT and VH IVUS may reflect the presence of generalized inflammation in patients with ACS, which enables clinical laboratory study to document the increase in C-reactive protein (CRP), fibrinogen, and von Willebrand factor in patients with ACS (9, 10).

Another interesting point in this study was that TCFA detected by VH IVUS was most frequently identified in mixed plaques on MSCT. As mentioned before, completely calcified plaques were rarely found in patients with ACS. However, we couldn't guess how much calcium deposit in the plaques was directly related to the occurrence of ACS in the patients. Motoyama et al. (11) recently reported a higher prevalence of spotty calcifications in ACS patients on MSCT. The culprit lesions even in a study using grey-scale IVUS presented more plaques with small calcium deposits in patients with ACS than in those with stable coronary artery disease (12).

MSCT could be a promising tool as a non-invasive modality for the evaluation of plaque characteristics although large prospective studies were lacking. More plaques with non-calcified tissue, as the authors emphasized, were observed on MSCT in ACS patients as compare with patients with stable coronary artery disease. These observations on MSCT were paralleled with a higher prevalence of TCFA in plaques of ACS patients on VH IVUS. Furthermore, similar findings were shown in culprit and non-culprit vessels of ACS patients, suggesting diffuse inflammation. Therefore, we needed to keep in mind the possibility of recurrent cardiovascular events when we identified the non-calcified or mixed plaques on MSCT regardless of culprit or non-culprit vessels.

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