It has been well established that coronary computed tomography angiography (CCTA) is useful for evaluating significant obstructive coronary artery disease (CAD), associated with sensitivity, specificity, positive and negative predictive values of 88, 96, 79, and 98\%, respectively.\(^1\) CCTA can precisely visualize coronary plaques and subsequently evaluate the risk of cardiac events in addition to invasive coronary angiography.\(^2\) Although the CT characteristics of plaques associated with acute coronary syndrome (ACS) include positive vascular remodeling, low plaque density and spotty calcification,\(^3\) patients demonstrating positively remodeled coronary segments with low-attenuation plaques on CCTA are at a higher risk of ACS developing over time in comparison with patients having lesions without these characteristics\(^4\) (Figure 1). Furthermore, meta-analyses of 18 studies comprising 9,592 patients also confirm that the risk of adverse cardiac events is significantly associated with the extent and severity of underlying CAD.\(^5\)

In contrast to the patients with significant coronary atherosclerosis, the current guidelines do not recommend using CCTA for asymptomatic subjects to evaluate their degree of CAD. Choi and colleagues evaluated 1,000 asymptomatic subjects who underwent CCTA as a general health evaluation and in 215 (22\%) patients, atherosclerotic plaques were identified and of them only 52 (5\%) subjects had stenosis greater than 50\%\(^6\). Although CCTA is able to detect coronary artery stenosis noninvasively, one of its major limitations as a screening method is the extremely low prevalence of CAD in asymptomatic patients. As we also have to consider contrast media, radiation exposure, and cost effectiveness, CCTA is not recommended as a screening tool.

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findings suggest that asymptomatic subjects with CAC score 0 have a low possibility of CAD and cardiac events, but risk stratification is a prerequisite for primary prevention of cardiac events.

In this issue of the Journal, Tsushima and his colleagues evaluate EAT and VAT by plain CT, and noncalcified plaque (NCP) by CCTA in 352 patients with suspected CAD and CAC score 0. Although they found an association between EAT, VAT, and the presence of NCPs, they suggest that both high VAT area and high EAT volume are implicated in the development of NCP, possibly the lipid core. Notably, an increased EAT volume identified patients with NCPs even when the VAT area was low, implying that accumulation of EAT contributes to coronary plaque formation even in patients without abdominal obesity. This study suggests that lipid tissues, including EAT, VAT and coronary plaque could be moving in a similar direction as well as indicating the importance of primary prevention of CAD using plain CT examination with low X-ray exposure, low price and no contrast media.

Two strategies can be theoretically useful for patients with or without significant chest pain (Figures 1, 2). Firstly, in patients with anginal chest pain, CCTA has significant ability to detect significant fixed stenosis as well as to predict future cardiac events using plaque analysis, including positively remodeled coronary segments with low-attenuation plaques (Figure 1). Secondly, in patients without significant chest pain, obvious ST segment changes or significant elevation of troponin I, a plain CT examination is recommended including EAT and VAT estimations (Figure 2).

Based on these findings, alternative imaging techniques such as the coronary artery calcium (CAC) score or the degree of epicardial adipose tissue (EAT) have been offered as predictors of coronary atherosclerosis in patients without cardiac symptoms. The CAC score can be obtained with extremely low X-ray exposure. Blaha et al reported that for 44,052 consecutive asymptomatic patients, the annual all-cause mortality rate increased from 0.87 through 1.92 to 7.48 deaths/1,000 person-years for CAC score zero, CAC score between 1 and 10, and CAC score >10, respectively. Yang and coworkers revealed that the number of Japanese patients in their study with a CAC score of 0 was 64, that of 1–100 was 58, that of 101–400 was 76, that of 401–1,000 was 70, and that of >1,000 was 49, respectively, and the frequency of CAD was 5%, 36%, 76%, 80%, and 94% respectively (P<0.001). Mahabadi et al reported that EAT was associated with coronary events in the general population independent of traditional cardiovascular risk factors. Therefore, current guidelines recommend CAC scoring for asymptomatic patients especially those with an intermediate risk of CAD (ie, class 2b, level B). Furthermore, Oka, Kihara and their colleagues presented that a high EAT volume was associated with the presence of vulnerable plaque components, independent of both obesity measurements (BMI and VAT) and CAC score. Some interesting reports have also examined serial EAT and CAC changes; baseline as well as the increase in EAT were associated with CAC progression.

More recent reports focused on the relationship between CAC score 0 and the prevalence of CAD, and these authors concluded that CAC score 0 did not exclude CAD. A sub-study of CORE 64 (Coronary Evaluation Using Multi-Detector Spiral Computed Tomography Angiography Using 64 Detectors), including 291 suspected CAD patients, found that 72 patients had CAC score 0, and 14 (19%) had at least 1 stenosis ≥50%. Another interesting sub-study of the ROMICAT II Trial reported that CAC score 0 did not exclude ACS in emergency department patients with acute chest pain. These findings suggest that asymptomatic subjects with CAC score 0 have a low possibility of CAD and cardiac events, but risk stratification is a prerequisite for primary prevention of cardiac events.

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