Editorial

Epicardial Adipose Tissue as a Predictor for the Development of Non-Calcified Coronary Plaque

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Epicardial adipose tissue (EAT) has emerged as a novel target for stratification of cardiometabolic risk factors because of its distinctive location and multifaceted metabolic properties with systemic and local effects. Because EAT is not separated from adjacent myocardium or the coronary artery vascular wall by fascia, it is possible that EAT might belong to the endocrine active assemblage of visceral body fat with paracrine impact on the initiation and progression of coronary artery disease (CAD)[1-3]. Previous large cohort studies demonstrated that EAT volume is associated with atherogenic risk factors, presence of CAD, and plaque burden[2, 4-7]. Furthermore, some reports suggest that elevated EAT volume is independently associated with increased incidence of future myocardial infarction[8, 9]. However, a causal relationship between EAT and the development of non-calcified coronary plaque (NCP) has not yet been investigated.

In this issue of the journal, Hwang *et al.* investigated a causal relationship between EAT and the development of NCP in 122 asymptomatic individuals without prior history of CAD and without NCP or obstructive CAD according to baseline cardiac computed tomography (CT)[10]. The authors showed that baseline epicardial fat volume index (EFVi) was significantly higher in the NCP group than in the CP but not in the plaque groups. Multivariable logistic regression analysis demonstrated that the presence of diabetes and the 3rd tertile of EFVi were significant predictors for the development of NCP on follow-up CT. This is the first study of serial CT scans to dem-

onstrate the impact of EAT on the development of NCP that is thought to be a high-risk coronary plaque. Their findings expanded the current knowledge related to EAT and cardiovascular risk and revealed that excessive EAT promotes the development of high-risk coronary plaque, which may further contribute to a higher risk of cardiovascular events.

One of the important findings is that the presence of diabetes mellitus is a strong predictor for NCP development in the present study population. Although the authors described that increased EAT was an independent predictor of NCP development even after adjusting for the presence of diabetes mellitus, we should recognize that the presence of diabetes as well as increased EAT is strongly associated with the future development of NCP among participants without prior history of CAD and without metabolic syndrome.

The present findings suggest the clinical usefulness of EAT measurement for risk stratification and its application in preventive measures and support the additional prognostic value of EAT. Although as a screening tool, EAT measurement would provide simple but important clinical information on the future risk of NCP development especially among asymptomatic individuals, the present study lacks the data describing the association between EAT, NCP, and future cardiovascular events in this cohort. Further studies are required to validate the clinical usefulness of EAT and NCP measurement for prognosis in large study populations. We hope that EAT and NCP measurement helps to predict future cardiovascular events in asymptomatic individuals without prior history of CAD.

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Conflict of Interest

None.

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