Thicker Epicardial Adipose Tissue in Nonobese Hypertensive Patients: An Innocent Bystander or Overlooked Villain?

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Elevated blood pressure is a key component of the metabolic syndrome. In reality, hypertension and the metabolic syndrome coexist in the same individual more often than would occur by chance. Several epidemiological studies showed that the prevalence of the metabolic syndrome in adult hypertensive patients ranged from 20% to as high as 50%, mostly depending on age and anthropometric features of the participants.1 The clinical significance regarding the coexistence of hypertension and the metabolic syndrome is twofold. First, it implies the presence of a relatively high-risk state. Hypertensive patients with the metabolic syndrome are associated with an ~30–50% higher risk of cardiovascular mortality than hypertensive patients without the metabolic syndrome.1 Second, it suggests that hypertension and the metabolic syndrome may share common underlying mechanisms. Visceral obesity has been recognized to be one of the underlying causes of the metabolic syndrome and hypertension.2 Indeed, visceral obesity commonly precedes dyslipidemia, with hypertension and diabetes developing later. Thus, assessment of visceral adiposity in hypertensive patients could be of particular significance both prognostically and therapeutically.

Previous studies exploring the associations between visceral obesity and the metabolic syndrome are mainly focused on intra-abdominal visceral fat depots. Epicardial adipose tissue (EAT), the adipose tissue confined within the pericardial sac, shares a common embryological origin with intra-abdominal visceral fat and is also a metabolically active visceral fat depot. Although EAT constitutes only 1% of total body fat mass and is often overlooked, some studies have demonstrated that the amount of EAT is quantitatively associated with individual and clustering of metabolic syndrome components, even after adjustment for intra-abdominal visceral adiposity.3 However, not all studies showed similar findings. There are at least two plausible explanations for the inconsistent associations between EAT and the metabolic syndrome observed in different studies. First, it has been shown that the dysmetabolic effect of EAT is more evident in individuals without general or abdominal obesity,3 which may obviously dilute the influence of EAT and make its contribution to the metabolic syndrome less significant. In the present issue, Pierdomenico and colleagues further reinforce this observation by showing an independent association between the thickness of EAT over the right ventricular free wall and the metabolic syndrome in 174 nonobese hypertensive patients after adjustment for body mass index and waist circumference.4 Second, the distribution of EAT around the heart is not symmetric and mostly accumulated in the perivascular grooves rather than in the nongrooved segments like the right ventricular free wall, where is the only place that EAT could be reliably assessed by echocardiography. Given its asymmetric distribution, the correlation between regional EAT thickness and total EAT volume is only moderate.3 There is still no consensus regarding whether global or focal EAT measurements will be more appropriate to assess the clinical significance of EAT. We have recently shown that, among all EAT measurements, EAT thickness in the left atrioventricular groove is most closely related to the metabolic syndrome and coronary atherosclerosis.5 However, our observations do not preclude the diagnostic value of echocardiographic measurement of EAT over the right ventricular free wall, particularly in nonobese individuals as shown by Pierdomenico et al.4

Currently, the major limitation of almost all published studies exploring the clinical significance of EAT is their cross-sectional design, which prevents inferring causality. Prospective studies are desperately needed to assess the potential roles of EAT in the development of hypertension, the metabolic syndrome, and cardiovascular morbidity and mortality. At the present time, it is still premature to announce whether EAT is just an innocent bystander or indeed an overlooked villain. In summary, thicker EAT could be viewed as the hallmark of “intrathoracic visceral obesity” and is often accompanied with the presence of various metabolic syndrome components. Despite its prognostic role is not yet determined, a cautionary note on the presence of thicker EAT in nonobese hypertensive patients seems prudent.
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