Letters to the Editor

Out of Sight, out of Mind; Subcutaneous, Visceral, and Epicardial Adipose Tissue

Ojos que no ven, corazón que no siente: el tejido adiposo subcutáneo, epicárdico y visceral

To the Editor,

I have read the article entitled “The Ratio Between Visceral and Subcutaneous Abdominal Fat Assessed by Computed Tomography Is an Independent Predictor of Mortality and Cardiac Events” by Ladeiras-Lopes et al.1 with great interest. The investigators report that the visceral adipose tissue/subcutaneous adipose tissue ratio was an independent predictor of death and cardiac events, irrespective of cardiovascular risk factors, coronary artery calcium, and the presence of coronary artery disease.1

Epicardial adipose tissue (EAT) is defined as the adipose tissue between the visceral pericardium and the outer margin of the myocardium.2 EAT is not only a passive lipid storage unit, but is also actively involved in lipid and energy homeostasis. The basic difference between EAT and visceral adipose tissue is its greater capacity for the release and uptake of free fatty acids and a lower rate of glucose utilization. Acetyl-CoA carboxylase and lipoprotein lipase activity are consistently lower in EAT than in subcutaneous adipose tissue. Free fatty acid oxidation is responsible for about 50% to 70% of the energy production of the heart.2 EAT is considered as an endoparacrine organ that secretes inflammatory adipokines, such as tumor necrosis factor alpha, monocyte chemoattractant protein-1, interleukin-6, interleukin-1β, plasminogen activator inhibitor-1, resistin, and many others.2 EAT volume is associated with coronary calcification, advanced atherosclerosis, cardiovascular risk factors, the incidence of myocardial infarction, and the severity of coronary artery disease in the general population.3 EAT volume has been reported to be significantly larger in patients with mixed or noncalcified plaques than in patients with calcified plaques or no plaques, which supports the hypothesis that EAT may be linked to early plaque components.4 A high epicardial fat volume index determined by computed tomography was an independent risk factor for the future development of noncalcified coronary plaque even after adjustment for traditional cardiovascular risk factors.4 Lu et al.5 reported that greater volumes of EAT are associated with high-risk plaque but that lower attenuation EAT was not an independent predictor for high-risk plaque features.

In the study by Ladeiras-Lopes et al.,1 the association between the visceral adipose tissue/subcutaneous adipose tissue ratio and all-cause mortality/cardiac events was evaluated and the correlation of the results with EAT (visceral adipose tissue/EAT, subcutaneous adipose tissue/EAT) might be beneficial due to the close relation between EAT and cardiac events.

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Response

Ojos que no ven, corazón que no siente: el tejido adiposo subcutáneo, epicárdico y visceral. Respuesta

To the Editor,

We have read with great interest the Letter to the Editor concerning our recently published paper on the association between an increased ratio of visceral to subcutaneous abdominal adipose tissue and higher risk of major adverse cardiovascular events, independently of traditional cardiovascular risk factors and coronary calcium.1

Epicardial adipose tissue (EAT) is an ectopic fat storage site in direct contact with adjacent coronary arteries and myocardium; therefore, it can have a paracrine effect on coronary atherosclerosis and myocardial function through the secretion of several adipokines that might regulate insulin resistance and inflammation.2

The putative association between EAT and visceral or subcutaneous abdominal fat is a promising research line that should continue to be addressed in future studies. Our group has already shown that EAT volume is positively correlated to coronary atherosclerotic burden, assessed by coronary artery calcium score, independently of abdominal visceral adipose tissue.3 Furthermore, in patients after a myocardial infarction, EAT volume was independently associated with decreased E’ velocity and increased E/E’ ratio, therefore suggesting impaired diastolic function.4