Editorial / *Editöryal Yorum*

Epicardial adipose tissue, metabolic syndrome, inflammation, and cardiovascular risk

Epikart yağ dokusu, metabolik sendrom, yangı ve kardiyovasküler risk

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Better to starve free than be a fat slave.

Aesop

Fat accumulation is no longer regarded as a sign of prosperity. Besides being a major aesthetic concern for modern people, it is also associated with an unfavorable cardiovascular (CV) risk profile. Adipose tissue is an active and highly complex endocrine organ. Metabolic syndrome (MetS) is characterized by a constellation of multiple risk factors that arise from insulin resistance accompanying abnormal adipose tissue deposition and function.[1] Abundant evidence has emerged from studies of MetS that intra-abdominal fat accumulation is associated with inflammation and high concentrations of high sensitivity C-reactive protein (hs-CRP), which predict increased CV events.[2,3]

Although the impact of intra-abdominal fat on inflammation and CV risk has been studied extensively, the importance of epicardial and mediastinal fat deposits had been neglected until recent years. Increased waist circumference, reflecting abdominal obesity, is the widely accepted measure of visceral adiposity. Waist circumference, however, can be affected by large amounts of subcutaneous fat, especially in obese people.[4] In the last decade, emerging data have suggested that epicardial fat can be a more reliable measure of visceral adiposity.[5-7]

Epicardial fat, like intra-abdominal fat, evolves from brown adipose tissue during embryogenesis. Some adipose tissue extends from the epicardium into the myocardium following the adventitia of the coronary arteries.[6] Some authors have claimed that vascular wall inflammation and atherosclerosis originate from the adventitia (the outside-inside hypothesis).[8] The close anatomical relationship between epicardial fat and the adjacent coronary arteries can allow paracrine interactions between these tissues.[5] Epicardial adipose tissue has a significantly higher expression of chemokines and several inflammatory cytokines than subcutaneous fat.[9] Previous studies have suggested that the presence of inflammatory mediators surrounding the coronary arteries can lead to amplification of vascular inflammation, plaque instability, apoptosis, and neovascularization.[9]

Abbreviations:

CAD	*Coronary artery disease*
CV	*Cardiovascular*
EFT	*Epicardial fat thickness*
hs-CRP	*High sensitivity C-reactive protein*
MDCT	*Multi-detector computed tomography*
MetS	*Metabolic syndrome*

In a study published in the present issue of this journal, Tok et al.[10] have shown an independent association between echocardiographically measured epicardial fat thickness (EFT), hs-CRP and MetS. Other studies have shown an association between increased epicardial fat and increased atrial fibrillation persistence, independent of other risk factors.[11,12] Since inflammation has an important role in the pathogenesis

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of atrial fibrillation,[13] this finding also supports the co-existence of epicardial fat and inflammation.

Echocardiographic assessment of epicardial fat is easy and convenient. Epicardial fat is generally identified as the echo free space between the outer wall of the myocardium and the visceral layer of the pericardium. Epicardial fat is measured perpendicularly on the free wall of the right ventricle.[14] Although echocardiographic assessment samples a tomographic slice of the epicardial fat and may not reflect the total volume, echocardiographic assessments correlate highly with MRI fat measurements.[15] Multi-detector computed tomography (MDCT) is another suitable tool for volumetric quantification of epicardial fat.[16] In a recent study by Bachar et al.,[8] patients with coronary artery disease (CAD) detected by MDCT had significantly higher epicardial fat thicknesses than patients without CAD. Bachar et al. also found a significant correlation between MetS and EFT regardless of body mass index. On multivariate analysis, an EFT greater than 2.4 mm was the strongest independent predictor of significant CAD (>50% diameter).

There is an ongoing debate as to whether grouping several risk factors under the umbrella of MetS adds any additional diagnostic or prognostic value compared to traditional CV risk factors.[17] Since inflammation is a pivotal mechanism of atherosclerosis, research of EFT, abnormal fat deposition, and inflammation seems to support the view that MetS, as a marker of abnormal adiposity, has an undeniable role in atherosclerosis.

Epicardial fat measurement seems to be an important marker of CV risk. Since echocardiographic measurement is an easy, non-invasive and reproducible method for epicardial fat assessment, echocardiographic measurement has the potential to be used for risk stratification and diagnosis of CV diseases. Epicardial fat can also be used as a therapeutic target in the future.

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Key words: Adiposity; atrial fibrillation; C-reactive protein; coronary artery disease; inflammation; metabolic syndrome; risk factors.

Anahtar sözcükler: Yağlılık; atriyum fibrilasyonu; C-reaktif protein; koroner arter hastalığı; yangı; metabolik sendrom; risk faktörleri.