Is increased epicardial fat thickness a marker of the presence of severe coronary artery disease?

Epikardiyal yağ kalınlığının artışı şiddetli koroner arter hastalığı varlığının bir belirteci midir?

Fat is mainly deposited in subcutaneous tissue, but it also accumulates in the abdominal or thoracal region (1). Other major sites of fat accumulation are visceral and cardiac areas; Cardiac fat deposition is now recognized as a new cardiometabolic risk marker, as it is associated with increased insulin resistance, cardiovascular risk factors, as their measurement is practical (2). Fat accumulation in the heart appears in three different types: intracellular, epicardial and pericardial. Intracellular fat is the microscopic lipid accumulation within the cytoplasm of cardiac muscle and can be the result of myocardial ischemia, cell damage or cell death. Epicardial fat is located between the outer wall of the myocardium and the visceral layer of pericardium (3). Pericardial fat exists anterior to the epicardial fat layer and therefore located between visceral and parietal pericardium. Due to the close anatomic relation between myocardium and the epicardial fat, the two tissues share the same microcirculation (4). In previous studies have been reported that epicardial fat is metabolically active and is the source for several adipokines. Potential interactions through paracrine or vasocrine mechanisms between epicardial fat and myocardium are strongly suggested (4).

The study by Shemirani et al. (5) in this issue of The Anatolian Journal of Cardiology highlights the correlation between the echocardiographic epicardial fat thickness (EFT) and the severity of coronary artery disease (CAD). Echocardiographic EFT measurement was performed in a total of 292 subjects who were referred for coronary angiography. All subjects underwent coronary angiography and then they were classified into the 2 groups: normal and CAD. EFT was significantly increased in CAD group (5.4±1.9 mm vs. 4.4±1.8 mm, p=0.0001). EFT was also significantly correlated with the severity of CAD (Califf scoring) when the (partial correlation) confounding variables were controlled (Spearman r=0.213, p=0.002). EFT showed significant positive correlation with low-density lipoprotein, body mass index, serum triglyceride levels and waist circumference. The authors propose that transthoracic echocardiographically measured EFT is significantly correlated with the severe multiple coronary artery stenosis in patients with known CAD and EFT could be used for in the risk stratification of those patients.

The finding regarding the significant correlation between the CAD severity and EFT in a relatively large population (n=292) who underwent invasive angiography is important. In agreement with this study, previous studies also reported a close relationship between the EFT and severity of CAD (6). However, these studies did not include a normal coronary artery group, which was documented by invasive angiography. Present study provides an additional information which allows to compare the EFT measurement between angiographically documented CAD group (n=171) and the subjects who had normal coronary angiography group (n=121).

Epicardial fat is thought to promote the development and progression of coronary atherosclerosis. Elevated inflammatory infiltrate has been described in epicardial fat of subjects with CAD (7). The paracrine or vasocrine secretion of epicardial inflammatory molecules, contributes to the metabolic and inflammatory milieu that also promotes atherogenesis (7). In vitro studies have shown that paracrine dialogs between human adipocytes and inflammatory cells present in adipose tissue (i.e., macrophage, lymphocytes, and others) promote an increased synthesis of numerous biomolecules, leading to a low-grade inflammatory microenvironment (8). These conditions most likely promote plaque formation and coronary stenosis. In accordance of these pathogenetic mechanisms, present study confirms the strong relationship between the epicardial adipose tissue deposition and the severity of coronary artery disease. But, large scale studies evaluating the relationship between EFT, inflammation and coronary atherosclerosis in patients with angiographically documented CAD may provide a more definite conclusion in this issue.

Califf scoring system, which was used for defining CAD severity by the authors of the present study. Authors cited an article related Califf scoring system, but in this article, Califf et al. (9) validated Duke Jeopardy Score which was developed by Dash et al. (10). Therefore, referring the Duke Jeopardy Score would be more accurate.

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Epicardial fat can be measured with several imaging techniques. Multi-detector computed tomography (MDCT) or cardiac magnetic resonance imaging (MRI) clearly allows a precise, but more expensive and cumbersome measurement (11). However, echocardiographic assessment of EFT would certainly be less expensive than MDCT and MRI. Additionally, echocardiography is simple, accurate and routinely performed in high-risk cardiac patients. In addition to previous studies, present study supports that echocardiographic epicardial fat measurement may play a role in the prediction of severity of coronary involvement in patients with CAD.

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