# Relation between epicardial fat thickness and chronic obstructive pulmonary disease

To the Editor,

We read the article "Relationship between metabolic syndrome and epicardial fat tissue thickness in patients with chronic obstructive pulmonary disease" by Demir et al. (1) with great interest. The authors aimed to evaluate the usefulness of epicardial fat thickness (EFT) to predict metabolic syndrome (MS) in chronic obstructive pulmonary disease (COPD) patients. They concluded that EFT was a non-invasive and easily available parameter, which is valuable in the prediction of increased MS risk in COPD patients. Early diagnosis of patients at risk of MS might help prevent ischemic heart disease in these patients. We thank the authors for their good contribution of the present study, which is successfully designed and well-documented.

Cardiovascular diseases are the most important factors that are associated with higher morbidity and mortality rate in COPD patients. At present, epicardial tissue, which is one of the endocrine organ, plays an important role in releasing numerous markers that are related to inflammation, endothelial dysfunction, oxidative stress, and atherosclerosis (2, 3). Over the past years, various studies have investigated the potential importance of epicardial tissue in the risk of cardiovascular diseases (3). In this respect, a previous report showed that the amount of epicardial tissue is importantly correlated to abdominal visceral adiposity, metabolic syndrome, cardiovascular diseases, and proinflammatory activity (3, 4). In clinical practice, EFT is a widely used method that gives information about the amount of epicardial tissue. In addition, EFT has several advantages, including its inexpensiveness, easy accessibility, rapid applicability, and good reproducibility. However, some important conditions should be emphasized. First, EFT was measured using transthoracic echocardiography and was measured on the free wall of the right ventricle at end-diastole in the current study (1). The authors should exclude the mediastinal fat, presenting as an echolucent area above the parietal pericardium, because linear echodense parietal pericardium may be considered to be epicardial fat. Second, because EFT measurements are linearly assessed using transthoracic echocardiography, echocardiographic EFT may not accurately reflect the total epicardial fat volume. Therefore, because of three-dimensional distribution of EFT, the gold standard measurement of EFT is magnetic resonance imaging (MRI) or computed tomography (CT). Concordantly, the lack of MRI and CT use should have been one of the limitations of the present study (5). Third, two-dimensional echocardiography cannot give adequate window of all cardiac segments, especially in obese subjects, and is highly dependent on acoustic windows. With this point of view, inter- and intraobserver variabilities for EFT measurement should be addressed in future studies (4).

Moreover, hypothyroidism, overt or subclinical, has multiple effects on the cardiovascular system. EFT may be a useful marker of subclinical atherosclerosis in patients with hypothyroidism. Also, a recent report emphasized that EFT was increased in patients with psoriasis; EFT may be a possible marker of subclinical atherosclerosis and increased cardiovascular risk in patients with psoriasis.

As a conclusion, although EFT values give us important information about patients' inflammatory status, they may not provide information to clinicians about systemic inflammation without the abovementioned conditions. We believe that these findings will require further studies on EFT in COPD patients.

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