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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References

1. Demir M, Acet H, Kaya H, Taylan M, Yüksel M, Yılmaz S, et al. Relationship between metabolic syndrome and epicardial fat tissue thickness in patients with chronic obstructive pulmonary disease. Anatol J Cardiol 2016 Feb 10. Epub ahead of print.
2. Balta S, Demirkol S, Kurt Ö, Sarlak H, Akhan M. Epicardial adipose tissue measurement: inexpensive, easy accessible and rapid practical method. Anadolu Kardiyol Derg 2013; 13: 611.
3. Katsiki N, Mikhailidis DP, Wierzbicki AS. Epicardial fat and vascular risk: a narrative review. Curr Opin Cardiol 2013; 28: 458-63.
4. Şengül C, Özveren O. Epicardial adipose tissue: a review of physiology, pathophysiology, and clinical applications. Anadolu Kardiyol Derg 2013; 13: 261-5.
5. Demirkol S, Balta S, Öztürk C, Çelik T, Iyisoy A. Different imaging modalities in quantification of epicardial adipose tissue thickness. J Clin Hypertens (Greenwich) 2014; 16: 616.

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Author`s Reply

To the Editor,

We are grateful for the kind comments to our manuscript entitled "Relationship between metabolic syndrome and epicardial fat tissue thickness in patients with chronic obstruc-

tive pulmonary disease," published online in Anatolian Journal of Cardiology 2016 Feb 10 (1). In their Letter-to-Editor, the authors emphasize that epicardial fat is an endocrine organ secreting various pro-inflammatory cytokines and is associated with inflammatory processes, including subclinical atherosclerosis. Previous studies have shown a relation between epicardial fat thickness (EFT) and visceral adiposity, metabolic syndrome, cardiovascular (CV) disease, and pro-inflammatory activity. Because a vast majority of our COPD patients had emphysematous-type disease, there were few obese (BMI >30) patients.

As the authors stated, magnetic resonance imaging (MRI) or computed tomography (CT) provide best images to assess the amount of epicardial fat; however we used transthoracic echocardiography (TTE) because it is a cheap, easily available, reproducible, and radiation-free imaging technique that we used in concordance with the description of Iacobellis et al. (2). Poor echogenicity was the reason for exclusion from the study in eighty patients. Lack of MRI/CT data regarding EFT in our patient population should be mentioned as a limitation of our study. We were unable to calculate the intra- and interobserver variabilities for EFT measurement; this was included in the study limitations. We also excluded patients with hypothyroidism, either apparent or subclinical.

As the authors stated, epicardial fat thickness has recently been shown to be associated with subclinical atherosclerosis in patients with inflammatory processes, including psoriasis, hypothyroidism, etc. Chronic obstructive pulmonary disease (COPD) is one of those diseases in which inflammation plays a key role in the pathogenesis and disease progression. Thus, our study supports the assertion that CV risk may increase as the EFT values increase in COPD patients.

In conclusion, further studies on EFT in COPD patients should consider the abovementioned concerns and limitations.

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References

1. Demir M, Acet H, Kaya H, Taylan M, Yüksel M, Yılmaz S, et al. Relationship between metabolic syndrome and epicardial fat tissue thickness in patients with chronic obstructive pulmonary disease. Anatol J Cardiol 2016 Feb 10. Epub ahead of print.
2. Iacobellis G, Assael F, Ribaudo MC, Zappaterreno A, Alessi G, Di Mario U, et al. Epicardial fat from echocardiography: a new method for visceral adipose tissue prediction. Obes Res 2003; 11: 304-10.

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Erectile dysfunction and heart rate recovery. Is it autonomic nervous system?

To the Editor,

Ulucan et al. (1) observed in their valuable and interesting study that heart rate recovery (HRR) indices were attenuated in patients with erectile dysfunction when compared with healthy controls entitled " Deterioration of heart rate recovery index in patients with erectile dysfunction" published Anatol J Cardiol 2016; 16: 264-9. In addition, they observed in their analysis that HRR at 1 min (HRR1) and 3 min were independently associated with the presence of erectile dysfunction. Exercise testing can be abruptly terminated (cessation of exercise) with the patient in the standing or sitting positon (no "cool-down" period) or when the patient keeps walking in a predetermined speed and inclination (cool-down period), which can be a 2-min cool-down at 1.5 mph at 2.5 grade or 1-min cool-down at 1 mph at 0% inclination (2, 3). In protocols using cool-down, HRR1 is calculated by taking the difference between the heart rate at peak exercise and heart rate 1 min later, which is 1 min after the beginning of the cool-down period (2). Similarly, in exercise tests that stop abruptly, HRR1 is calculated by taking the difference between the heart rate at peak exercise and heart rate 1 min later at which time the patient is in complete rest in the supine or sitting positon. Abnormal HRR1 is usually defined as heart rate that declines to ≤12 beats/min in the first minute after exercise for protocols that use a post-exercise cool-down or ≤18 beats/min in the first minute post exercise for protocols that abruptly stop exercise (2, 4). Because the authors used post-exercise cool-down protocol, HRR1 ≤12 beats/min might be assumed to be abnormal in this case. HRR1 was 34.8± 1.2 in patients with erectile dysfunction in the authors' study. Thus, one should be very careful in interpreting their results. Approximately 95% of HRR1 was, statistically, between 12.4 and 57.2 beats/min in patients with erectile dysfunction. Therefore, we can assume that virtually all patients with erectile dysfunction had a normal HRR1. Hence, it might be misleading to suggest that patients with erectile dysfunction have impairment in autonomic nervous system. Looking carefully at the data, maximal heart rate was 158.2±18.7 beats/min in patients with erectile dysfunction and 167.2±16 beats/min in controls. Accordingly, most of the differences between patients with erectile dysfunction and controls with regard to HRR indices were due to lower heart rate attained at peak exercise in patients with erectile dysfunction. Namely, chronotropic incompetence, which might be due poor physical fitness, could be responsible for differences in HRR in this study population as demonstrated before (5). I believe that caregivers should be familiar with parameters gleaned from a standard exercise test and interpreting the results gained from it.

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