

## Is echocardiographic epicardial adipose tissue thickness measurement a reliable and reproducible method for risk stratification?

To the Editor,

With great interest, we read the article titled "Epicardial adipose tissue thickness is associated with myocardial infarction and impaired coronary perfusion" published by Tanindi et al. (1) in *Anatol J Cardiol* 2015; 15: 224-31. It is a good paper with well-conducted analysis. Tanindi et al. (1) investigated the association between epicardial adipose tissue thickness (EAT) and acute myocardial infarction (AMI) in their population. The measurement of EAT was performed manually at end-systole on the free wall of the right ventricle perpendicular to the aortic annulus in standard parasternal long-axis view. Tanindi et al. (1) found a positive correlation between EAT and AMI. They highlighted that the echocardiographic measurement of EAT is a useful method for risk stratification and for choosing patients who need more aggressive treatment in terms of risk reduction.

At present, the echocardiographic measurement of EAT, which reflects cardiac and visceral adiposity, has become one of the leading topics in cardiovascular imaging studies. EAT is suggested as a new cardiometabolic risk factor. Correlations between increased EAT and insulin resistance, metabolic syndrome, hypertension as well as cardiovascular diseases have been studied (2-4). The echocardiographic measurement of EAT is a widely available, simple, safe, non-invasive, cheap, and rapid method; however, it should be questioned whether EAT is a reliable and reproducible method. If it is not a reliable and reproducible method, then inaccurate measurements may affect our clinical decision and research results. In addition, EAT that was measured from the free wall of the right ventricle by echocardiography does not reflect all subepicardial adipose tissue volume.

Saura et al. (5) investigated the reproducibility of the echocardiographic measurement of EAT and compared the values with those obtained using multi-detector computed tomography (MDCT). Although the contrary was claimed, in a study by Saura et al. (5), they found a poor reproducibility of the echocardiographic measurements of EAT assessed by intraclass correlation coefficient. Moreover, measurements with echocardiography and MDCT showed low concordance. Saura et al. (5) found that echocardiography yielded larger values than those yielded by MDCT. In particular, there was a notable difference of up to 7 mm within two standard derivations of the mean values measured by these two different methods. The results of Saura et al. (5)'s study indicate that EAT measurements by echocardiography may lead to the misclassification of patients. Therefore, clinicians should be careful when this parameter is used as a diagnostic tool for risk stratification.

Furthermore, there are some other controversial issues regarding EAT. There are no normality values of EAT, and the discussion on how to measure EAT by echocardiography is still ongoing. EAT may be deformed through the cardiac cycle, and to ensure the maximal stability of true EAT, it should be measured in end-diastole (5). Further comprehensive studies are required to investigate the reproducibility of EAT and to answer the other questions.

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### Author's Reply

To the Editor,

We thank the authors for their comments and interest in our study titled "Epicardial adipose tissue thickness is associated with myocardial infarction and impaired coronary perfusion" (1) published in the *Anatol J Cardiol* 2015; 15: 224-31. In the last decade, many studies have highlighted an association between epicardial adipose tissue thickness (EAT) and coronary artery disease (CAD); thus, it has been proposed as a marker to detect cardiovascular risk. Of course, an ideal biomarker should reflect the degree of atherosclerosis, predict cardiovascular events, and indicate improvement after intervention. In addition, it would be further desirable if a biomarker could be easily and non-invasively measured, and if the measurement was reproducible and standardized (2).

As the authors mentioned, there has been a debate on the preferred method to measure EAT and on the range that should be considered as normal. Echocardiography is the method used to assess EAT by Iacobellis et al. (3) since 2003, and it is identified as the echo-free space between the outer wall of the myocardium and the visceral layer of the pericardium perpendicular to the free wall of the right ventricle at end-systole. Although some investigators suggested that the measurement should be made at the end-diastole to coincide with other imaging modalities such as computed tomography (CT) and magnetic resonance imaging (MRI), many authors including Iacobellis et al. (3), the investigator who first presented echocardiographic EAT thickness to the literature, prefer to measure EAT at end-systole because EAT may be compressed at end-diastole.

An important point is to set a clear and widely accepted reference range for a biomarker, as mentioned previously. Although EAT has been studied for more than a decade, there is no universally accepted cut-off point above which the EAT values can be definitely considered as abnormal. Many studies have provided cut-off values, but these studies have evaluated EAT from different points of view, such as its association with atherosclerosis, subclinical atherosclerosis, presence of CAD, extent of CAD, and plaque morphology. Thus, there are many proposed EAT cut-off values in the literature. However, in the light of the current literature, EAT thickness of >5 mm is safely considered as abnormal (4).

Another important point in the evaluation of EAT is the method of choice. Echocardiography is easily available, inexpensive, and reproducible, as mentioned previously. CT and MRI have also been increasingly used to assess the amount of EAT, and they have high spatial resolution; their most important advantage is the possibility of volumetric assessment. However, similar to echocardiography, there is no universally accepted cut-off value above which EAT is considered as abnormal (4).

The authors mentioned the study by Saura et al. (5) that reports a poor reproducibility and poor tomographic concordance for echocardiographic EAT measurement. However, it may not be appropriate to depend on a single study to conclude that echocardiography is not a reliable method. There are many other studies that report a good correlation of echocardiographic EAT determination with CT and MRI (6).

The aim of our study was to show the association between EAT thickness, myocardial infarction, and coronary perfusion. We agree that EAT still has a long way to go before universally accepted ranges are set, and other criteria that are needed to establish a marker as a routinely used one are fulfilled. Nevertheless, we think that the findings of our study add to the current literature as we provide a cut-off value to predict AMI and poor coronary perfusion among patients with a clinical diagnosis of CAD.

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## What is the real predictive value of red cell distribution width for the mortality in non-ST elevation acute coronary syndrome?

To the Editor,

I have read the recently published article by Bekler et al. (1) "Relationship between red cell distribution width and long-term mortality in patients with non-ST elevation acute coronary syndrome" entitled with great interest in *Anatol J Cardiol* 2014 Jun 23. In their study, authors reported that high red cell distribution width (RDW) level on admission is a predictor of long-term mortality in patients with non-ST elevation acute coronary syndrome (NST-ACS). In this paper, I would like to emphasize the possible effects of medical treatment of patient groups on the end-points of this study. In the present study by Bekler et al. (1), there are no data regarding patient groups' medications. It is well known that optimal medical therapy reduces the early and long-term mortality in patients with NST-ACS. Based on our previous knowledge and according to the current guideline, it is recommended to use oral beta-blockers, long-term treatment with aspirin, and dual antiplatelet therapy for at least 12 months as well as to use statins and angiotensin-converting enzyme inhibitors (ACEI)/angiotensin-receptor blockers (ARB) to reduce mortality and major adverse cardiovascular events (MACE) in NST-ACS patients (2). Also, it has been reported that dual antiplatelet therapy with ticagrelor significantly reduced the mortality and MACE in NST-ACS patients as opposed to the patients treated with aspirin and clopidogrel (3, 4). Hence, authors should comment on the incidence of patients treated with optimal medical therapy in both high RDW and low RDW groups and compare the groups regarding beta-blockers, ACEI/ARB, statins, dual antiplatelet usage rates, and the type of dual antiplatelet therapy. Because the results of the present study by Bekler et al. (1) may not be due to high RDW level, less medications rates with optimal medical therapy in high RDW level group may be the main reason for higher mortality.

In conclusion, the statistical data of the present study by Bekler et al. (1) may be improved. Authors should report the patients' medications in both groups. High RDW level may indicate poor prognosis in NST-ACS patients. However, to define its exact role on mortality, conventional medical treatments that are known to reduce the mortality should be considered.

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