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 endpoints 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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Author`s Reply

To the Editor,

Firstly, my colleagues and I were very pleased to read the letter concerning an important issue in our article titled "Relationship between red cell distribution width and long-term mortality in patients with non-ST elevation acute coronary syndrome" after its publication in Anatol J Cardiol 2014 Jun 23 by Bekler et al. (1). Our study offers an easy and cost-effective approach to a significant issue in daily clinical practice. In our study, we showed that erythrocyte distribution width (RDW) predicts late mortality after the discharge of patients with non-ST elevation acute coronary syndrome (NSTE-ACS). In the critical comment, we were asked if patients had received optimal medical therapy after discharge and to what extent did this affect the results. First, as noted in the Methods section of our article, our study was a retrospective study, and as we mentioned during the evaluation process of the article, data on the optimal medical treatment of all patients could not be obtained on an objective basis; hence, this data was not included in the article. To clarify this issue, groups with high and low RDW values were compared; then, patient groups with and without cardiovascular events were compared. We showed that the RDW value at hospital admission could be a predictor of mortality similar to age and ejection fraction. Indeed, RDW has been shown to be an important predictor of heart failure and coronary artery disease in earlier studies (2-4), and we can easily see that there were no data regarding optimal medical treatment when these studies were analyzed. Of course, to know whether optimal medical treatment was received will contribute to our study, but we believe it will not change the fact that RDW is an independent predictor in light of the abovementioned studies.

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Arterial stiffness evaluation in patients with irritable bowel syndrome: Role of antihypertensive drugs and statins

To the Editor,

We are very pleased to read with great interest to the article by Durakoğlugil et al (1). They investigated heart rate variability, carotid intima-media thickness, and carotid-femoral pulse wave velocity (cf-PWV) as a measure of arterial stiffness in patients with irritable bowel syndrome in the recent study titled "The effect of irritable bowel syndrome on carotid intima-media thickness, pulse wave velocity and heart rate variability" and published in Anatol J Cardiol 2014; 14: 525-30 (1). They found that cf-PWV values were similar between patients with irritable bowel syndrome and controls. This is a well-written study. However, I want to pay attention to the antihypertensive drugs used by patients that can affect arterial stiffness.

Arterial stiffness is a complex process associated with confounding factors. Cecelja et al. (2) published a systematic review that showed that the contribution of cardiovascular risk factors other than age and blood pressure to aortic stiffness measured by cf-PWV is small or insignificant, and that age and blood pressure consistently showed an independent association with aortic stiffness. It has also been shown that some antihypertensive drugs such as angiotensin-converting enzyme inhibitors, calcium channel blockers, and spironolactone reduce arterial stiffness (3-5). In addition to angiotensin-converting enzyme inhibitors, β-blockers and aliskiren as direct renin inhibitors reduce arterial stiffness (5). Recent meta-analysis showed that angiotensin receptor blocker treatment also improves arterial stiffness (6).

In the study by Durakoğlugil et al. (1), there is no information regarding the antihypertensive drugs used. Similarly, statins also reduce arterial stiffness, but there is also no data regarding their use. From this aspect, antihypertensive drugs and statins should be considered in aortic stiffness evaluation. It would be helpful if the authors provided this information.