Epicardial adipose tissue measurement: inexpensive, easy accessible and rapid practical method

Epikardiyal yağ dokusu ölçümü: Ucuz, kolay erişilebilir ve hızlı pratik yöntem

Dear Editor,

We have read the article "Epicardial adipose tissue (EAT) is independently associated with increased left ventricular mass in untreated hypertensive (UHT) patients: an observational study" written by Erdoğan et al. (1) with a great interest. The authors aimed to evaluate the relationship between EAT and left ventricular hypertrophy (LVH) in patients with UHT. They concluded that EAT was related to increased LVM independent of body mass index (BMI), waist circumference, weight, systolic and diastolic blood pressure and other risk parameters, in patients with UHT. Determination of increased EAT by echocardiography may have an additional value as an indicator of cardiovascular risk and total visceral adipose tissue. Thanks to the authors for their contribution of the present study, which is successfully designed and documented.

Cardiovascular diseases are the most important causes of mortality and morbidity in developed countries worldwide. It is widely recognized that accumulation of EAT is strongly related to the development of coronary artery disease (CAD). EAT amount may contribute to systemic inflammation beyond traditional cardiovascular risks and body fat composition. EAT measured by echocardiography has been known to be associated with metabolic syndrome (2). Additionally, echocardiography-based EAT measurement was related to several metabolic abnormalities and independently associated fatty liver disease (3). On the other hand, in kidney disease patients, EAT was positively correlated with atherosclerosis and the presence of coronary artery calcification (4). In addition, the duration of hypertension (HT) may be different in these patients. We think that the results of the study would be stronger, if the authors had mentioned these factors including the duration of HT, liver and kidney function tests.

EAT can also be affected by the atherosclerotic risk factors such as alcohol consumption, hypothyroidism, impaired glucose tolerance and higher inflammatory status (5) such as an inflammatory disease, cardiac syndrome X and infection (6). In this point of view, in the present study, the authors did not mention some of these possible contributing factors. It would be better, if the authors gave information about these factors.

EAT measurement with echocardiography has several advantages, including its inexpensive, easy accessibility, rapid applicability and good reproducibility. EAT has a 3-dimensional distribution and two-dimensional echocardiography cannot give adequate window of all cardiac segments especially in obese subjects and is highly dependent on acoustic windows. In this point of view, it would be better to give inter-observer and intra-observer variability for EAT measurement in the current study (7).

Finally, EAT itself without other inflammatory markers may not provide information to clinicians about the systemic inflammation. Therefore, we think that it should be evaluated together with other serum inflammatory markers. We believe that these findings will evaluate further studies about EAT on cardiovascular risk factors.

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Author`s Reply

We thank the authors for their supportive comments on our article published in the Anatolian Journal of Cardiology related to the relationship between epicardial adipose tissue (EAT) and left ventricular mass (1) in their letter entitled as 'Epicardial adipose tissue measurement: inexpensive, easy accessible and rapid practical method'. They also pointed that EAT amount may contribute to systemic inflammation beyond traditional cardiovascular risks and body fat composition. Several factors including the duration of hypertension (HT), liver and kidney function tests as well as alcohol consumption, hypothyroidism,

impaired glucose tolerance and higher inflammatory status such as an inflammatory disease, cardiac syndrome X and infection may affect EAT, therefore if provided they would be valuable. In addition, inter-observer and intra-observer variability for EAT measurement are asked, which had already been provided.

We accept that above-mentioned additional factors may have effects on EAT. We checked them and the existing data was provided herein. EAT was correlated to uric acid, glucose and C-reactive protein (CRP) but not creatinine, liver functions and duration of HT in our data. On the other hand, we had performed a multivariate analysis including these related parameters and we had determined that left ventricular mass (LVM) is independently related to uric acid and glucose as well as EAT, but not CRP.

In our opinion, the possible effects of increased epicardial adipose tissue on vasculature and heart an active local paracrine role and passive thermogenic effect or systemic endocrine effects are possible mechanisms for active participation of EAT in this process. We believe that further studies on LVM are needed to clarify more accurately the mechanisms and possible causative cells, cytokines and may be receptors and to confirm the importance of modulating real underlying mechanism to improve clinical outcome.

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LDL cholesterol measurement in terms of CHOLINDEX

LDL kolesterol ölçümünün CHOLINDEX açısından değerlendirilmesi

Dear Editor,

I have read the manuscript of Akpınar et al. (1), which was published in the Anatolian Cardiology Journal on March 2013 entitled 'A new index (CHOLINDEX) in detecting coronary artery risk' with great enthusiasm and interest. Authors have validated a new index, which can be applicable to our daily practice, while taking care of our patients.

As we understand from the manuscript that serum level of LDL cholesterol was measured by enzymatic method utilizing an auto analyzer. However in our daily practice, we all know that when we order 'Lipid profile' for our patients, whether in a university or a government or a special hospital, lipid profile results mostly reported with an indirect formulated measurement, which is Friedewald formula. This formula was validated in 1972 and still inside the market. Besides this formula there are new formulas under investigation and still validating for our daily practice. These new automated LDL measurement formulas are competing with the former Friedewald formula (2, 3).

The gold standard method for LDL-cholesterol measurement is ultra-centrifugation followed by beta-quantitation, which is expensive and inconvenient for routine clinical application (4). More recently, direct methods of LDL cholesterol measurement using specifically designed detergents have been developed, which outperform those based on inhibition with monoclonal antibodies (5, 6). However, these methods are still quite expensive for most laboratories, and thus direct determination of LDL cholesterol is uncommon in most laboratories worldwide.

I suggest to authors that, if they want to validate this new index for our daily practice, they have to adjust their new index according to Friedewald as well as the newly described validated indirect LDL cholesterol measurement formulas. Otherwise because of time and financial shortage of most of the hospitals, this new Cholindex may not find its value for the scientific and cardiologic assessment of coronary artery disease.

As I conclude, after adjustment of the Cholindex to indirect LDL cholesterol measurements (Friedewald, de Cordova CM) we all can be happy with this new coronary artery disease index.

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