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Address for Correspondence: Dr. Yavuzer Koza,
Atatürk Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı,
Yakutiye, Erzurum-Türkiye
Phone: +90 442 344 79 38
Fax: +90 442 344 13 01
E-mail: yavuzerkoza@hotmail.com



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Author's Reply

To the Editor,

We thank the authors for their interest in our study entitled "Epicardial adipose tissue thickness is associated with myocardial infarction and impaired coronary perfusion." published in *Anatolian Journal of Cardiology* reporting an association between acute myocardial infarction and increased epicardial adipose tissue (EAT) (1). As mentioned, the association between coronary atherosclerosis and EAT has been demonstrated in many studies using either echocardiography or computed tomography (CT)/magnetic resonance imaging (MRI). EAT is three dimensional and its evaluation using CT or MRI would provide more accurate information about its volume. However, studies using echocardiography and CT/MRI have shown that these techniques are in good compliance (2). We think that it is important to categorize a patient as having increased EAT rather than precisely providing the exact EAT volume to classify a patient as "at high cardiovascular risk". Echocardiography is adequate for that purpose and is advantageous because it is inexpensive, repeatedly and easily available in almost every cardiology clinic.

The authors mentioned that EAT may have cardioprotective properties because of the secretion of anti-inflammatory and anti-atherogenic adipokines such as adiponectin and adrenomedullin, and cited Iacobellis et al. (3) However, in that study, Iacobellis et al. (3) found that EAT partially contributed to the adiponectin levels in the coronary circulation. Instead, intracoronary adiponectin levels reflected the adiponectin levels in the peripheral circulation. In addition, Iacobellis et al. (4) also stated that adrenomedullin gene and protein expression in EAT were downregulated in the presence of CAD, and there was no direct evidence that EAT contributed to intracoronary adrenomedullin levels.

We agree with the authors that stable angina pectoris, unstable angina pectoris, and acute myocardial infarction (AMI) are different clinical entities with respect to pathophysiology, presentation, and clinical management; however, we think that there is some merit in showing that EAT thickness is different among different clinical entities and providing a cut-off value to predict AMI. We accept that EAT is associated with traditional risk factors such as diabetes, hypertension, age, waist circumference, and metabolic syndrome (5). However, EAT has an additional predictive value over these, as there are studies highlighting an independent association between CAD and well-known risk factors.

We provided an association between increased EAT thickness and AMI. Apparently, this was not a prospective study where patients were followed in terms of adverse cardiac events. However, there are recent follow-up studies in the literature reporting increased cardiovascular

death and myocardial infarction in patients with thicker EAT, that can be considered as supportive based on our hypothesis (6).

Finally, we think that patients with increased EAT thickness should receive more aggressive risk factor modification. What we mean by saying that is to maximize proved medical therapies based on cardiovascular protection in accordance with the current relevant guidelines. Although physicians agree on the idea of prescribing mortality reducing medications in maximum applicable doses, real life experience is not so promising. These high risk patients may be followed up closer than others to check for the status of the modifiable risk factors.

Aslı Tanındı, Aycan Fahri Erkan
Department of Cardiology, Faculty of Medicine, Ufuk University;
Ankara-Turkey

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Address for Correspondence: Dr. Aslı Tanındı,
Ufuk Üniversitesi Tıp Fakültesi, Kardiyoloji
Anabilim Dalı, Ankara-Türkiye
Phone: +90 312 204 40 82
Fax: +90 312 204 40 55
E-mail: aslitanindi@gmail.com

The detection of cardiac tamponade by hemodynamic transesophageal echocardiography after left ventricular assist device implantation

To the Editor,

Hemodynamic Transesophageal Echocardiography (hTEE) is a new technology in the follow-up of postoperative patients in the cardiovascular surgery intensive care units. It can provide bedside, continuously available, direct cardiac imaging by its disposable probe for up to 72 hours and guide treatment by the detection of complications and assessing ventricular filling and volume status (1-3). This is the reason that it is called as hemodynamic.

We present the case of a 61-year-old patient hospitalized with the diagnosis of decompensated heart failure and given diuretic treatment.

Left ventricular ejection fraction (LVEF) was 14%. INTERMACS score was 4 and left ventricular assist device was implanted. The patient was followed postoperatively in the intensive care unit by hTEE. There was no pericardial effusion at 1 hour and minimal effusion at 4 hour. At 10 hour, pericardial effusion showed progression but no constriction. Therefore, imaging was performed hourly. Finally, a progressive decline in arterial blood pressure and cardiac tamponade was detected at 16 hour postoperation and the patient was re-operated. This is the first practice of hTEE after cardiac surgery, and the first report of cardiac tamponade by hTEE in our country.

Postoperative cardiac surgery patients should be followed closely and continuously in terms of mechanical complications and volume status. Early diagnosis of complications is crucial to perform medical and surgical treatment. However, it is usually hard to detect these complications because of the limited echogenicity by transthoracic echocardiography and inability to perform transesophageal echocardiography continuously in the postoperative period (2, 3). hTEE does not have these limitations and can provide early diagnosis and treatment of complications, such as pericardial tamponade, by continuous bedside imaging postoperatively in intensive care units (1-3).

**Mustafa Kurşun, Sanem Nalbantgil, Serkan Ertugay*,
Cağatay Engin*, Tahir Yağdı*, Mustafa Özbaran***

**Departments of Cardiology, *Cardiovascular Surgery, Faculty of
Medicine, Ege University; İzmir-Turkey**

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Address for Correspondence: Dr. Mustafa Kurşun,

Ege Üniversitesi Tıp Fakültesi,
Kardiyoloji Anabilim Dalı, 35100, İzmir-Türkiye

Phone: +90 232 374 62 78

Fax: +90 232 390 54 60

E-mail: mustafakursun35@gmail.com

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