Emmanuelle et al. (3). The level of AT activity and the type of AT deficiency determine the clinical picture (4). The occurrence of multiple thrombi at the age of 62 made our case interesting.

The criticism about testing the AT level only once makes sense; however, we only had one chance to test the AT activity in our patient. Due to multiple mobile intracardiac thrombi, intravenous anticoagulation therapy was initiated as soon as possible. The patient did not recover, and was under medical therapy throughout the hospitalization period. Therefore, repeat testing for AT activity while under anticoagulation therapy would be misleading. It is known that AT level decreases as a result of anticoagulation therapy (5).

We agree with the opinion that when someone has inherited natural anticoagulant deficiencies, clinical problems often occur at an early age. On the other hand, as you mentioned, it was presented in a cross-sectional study that 3 patients who were demonstrated to have AT deficiency with repeated tests had no personal or family history of thrombosis (6). Precipitating factors play a major role in these circumstances. In our patient, apart from AT deficiency, atrial fibrillation concomitant with severe apical hypokinesia in the left ventricle due to myocardial infarction exacerbated the situation. It is impossible to link the multiple thrombi to only one of the underlying causes in this case report.

Mert İlker Hayıroğlu, Muhammed Keskin
Department of Cardiology, Haydarpaşa Sultan Abdülhamid Han Training and Research Hospital; İstanbul-*Turkey*

References

1. Hayıroğlu MI, Keskin M, Dönmez C, Günay MB, Dayı SU. Antithrombin III deficiency concomitant with atrial fibrillation causes thrombi in all chambers: 2D and 3D echocardiographic evaluation. Anatol J Cardiol 2016; 16: E21-2.
2. Wiles KS, Hastings L, Muthuppalaniappan VM, Hanif M, Abeygunasekara S. Bilateral renal artery thrombosis in inherited thrombophilia: a rare cause of acute kidney injury. Int J Nephrol Renovasc Dis 2014; 7: 35-8.
3. Emmanuelle T, Husein B, Iqbal J, Macheta M, Isaacs P. Concomitant homozygosity for the prothrombin gene variant with mild deficiency ofantithrombin III in a patient with multiple hepatic infarctions: a case report. J Med Case Rep 2010; 4: 122.
4. Patnaik MM, Moll S. Inherited antithrombin deficiency: a review. Haemophilia 2008; 14: 1229-39.
5. Holm HA, Kalvenes S, Abildgaard U. Changes in plasma antithrombin (heparin cofactor activity) during intravenous heparin therapy: observations in 198 patients with deep venous thrombosis. Scand J Haematol 1985; 35: 564-9.
6. Wells PS, Blajchman MA, Henderson P, Wells MJ, Demers C, Bourque R, et al. Prevalence of antithrombin deficiency in healthy blood donors: a cross-sectional study. Am J Hematol 1994; 45:321-4.

Address for Correspondence: Dr. Mert İlker Hayıroğlu
Dr. Siyami Ersek Göğüs Kalp ve Damar Cerrahisi Eğitim ve Araştırma Hastanesi Kardiyoloji Bölümü, İstanbul-*Türkiye*
E-mail: mertilkerh@yahoo.com

Inflammatory activity of adipose tissue

To the Editor,

There is growing interest in inflammation, adipose tissue, and the atherosclerotic process in vessels. As a result of recent studies, it is known that obesity and increased epicardial adipose tissue are important factors affecting the pathogenesis of atherosclerosis. Adipose tissue releases inflammatory mediators like an endocrine organ. It produces cytokines, such as adiponectin, leptin, resistin, and interleukins, and these mediators cause an increase in inflammatory activation in the arterial wall. Adipose tissue acts as a source of proinflammatory activity, and it is therefore called obesity-related inflammatory activity (1).

We read the article entitled "An increase in epicardial adipose tissue is strongly associated with carotid intima-media thickness and atherosclerotic plaque, but LDL only with the plaque" published in The Anatolian Journal of Cardiology 2017; 17: 56-63 by Kocaman et al. (2) with great interest. The authors sought to investigate whether epicardial adipose tissue (EAT) has proliferative effect on carotid intima-media thickness (CIMT) and carotid plaque. They concluded that EAT had a relationship with both CIMT and the presence of carotid plaque. The authors also said that this finding suggested that EAT thickness may be a risk factor and a biomarker, playing an important role beginning in early stages of atherosclerosis. We congratulate the authors for these valuable results, which are compatible with the literature. They also drew attention to an interesting topic related to the inflammatory capacity of adipose tissue. There are hypotheses related to interactions of the heart and epicardial fat. One suggests that lack of fascia between heart and epicardial fat allows inflammatory mediators to easily diffuse to the vessels and myocardium (1). Having read the authors' report, we want to contribute to a seemingly missing aspect. In the results of the study, it was reported that EAT correlated to BMI, waist circumference, and CRP, in addition to CIMT ($p<0.001$) (Table 2). CIMT, BMI, waist circumference, and presence of carotid plaque increased with increase of EAT thickness ($p<0.001$) (Table 3). These results show that CIMT and carotid plaque formation may also be related to obesity of the study patients, as EAP and BMI are directly proportional in the study. In the limitations section, the authors said that their study group had increased visceral adipose tissue. BMI is a widely used marker of obesity and there are many studies about obesity and inflammatory effect on progression of atherosclerosis (1). So there is a need to differentiate whether these results belong to visceral or epicardial adipose tissue. The authors were also interested in question of if CRP level increased as EAT thickness increased, and if there is a possible inflammatory link between EAT and CIMT. We think there is a need for more studies to investigate the inflammatory pathways of EAT, independent of other clinical variables like obesity, and that there is also a need for a patient group that isolates increase in EAT to obtain more significant results.

Şahin İşcan, İsmail Yürekli, Habib Çakır, Orhan Gökalp
Department of Cardiovascular Surgery, Katip Çelebi University İzmir Atatürk Training and Education Hospital; İzmir-*Turkey*

References

1. Scridon A, Dobreanu D, Chevalier P, Serban RC. Inflammation, a link between obesity and atrial fibrillation. Inflamm Res 2015; 64: 383-93. [CrossRef]
2. Kocaman SA, Baysan O, Çetin M, Altuner TK, Ocaklı EP, Durakoğlugil ME, et al. An increase in epicardial adipose tissue is strongly associated with carotid intima-media thickness and atherosclerotic plaque, but LDL only with the plaque. Anatol J Cardiol 2017; 17: 56-63.

Address for Correspondence: Dr. Şahin İşcan
Katip Çelebi Üniversitesi İzmir Atatürk,
Eğitim ve Araştırma Hastanesi
Kalp Damar Cerrahisi Bölümü Karabağlar, İzmir-*Türkiye*
E-mail: sahiniscan@hotmail.com

DOI:10.14744/AnatolJCardiol.2017.7729

Author`s Reply

To the Editor,

We would like to thank the authors for their comments on our article entitled "An increase in epicardial adipose tissue is strongly associated with carotid intima-media thickness and atherosclerotic plaque, but LDL only with the plaque." published in Anatol J Cardiol 2017; 17: 56-63(1) in their letter entitled "Inflammatory activity of adipose tissue." Visceral obesity is strongly associated with atherosclerosis. Even though waist circumference and body mass index (BMI) are the most common assessment methods of total visceral adipose tissue and cardiometabolic risk, these methods lack direct measurement of adipose tissue and seem to have better correlation to subcutaneous fat, rather than visceral fat. This may explain why BMI was related to carotid intima-media thickness (CIMT) in univariate analysis, but not an independent variable in multivariate analyses in our study.

The metabolically healthy obese phenotype and the metabolically unhealthy non-obese phenotype may possibly blunt the predictive power of BMI for CIMT. Perivascular adiposity is primarily related to visceral adipose tissue, which is not necessarily related to increased BMI.

In our personal opinion, the liver may have a central role in determining visceral or subcutaneous adiposity. Genetic determinants, diet, and physical activity may have some role in some specific liver functions, which determine lipid influx from the bloodstream, lipid synthesis in liver, and efflux to subcutaneous tissue or visceral organs. Healthy and unhealthy obese and non-obese phenotypes that have isolated increase in EAT may help us to understand precise roles of EAT in vascular disease.

Additional data would be required in order to clarify the diagnostic role of EAT in managing obese and non-obese patients, and to decrease cardiometabolic risk.

Sinan Altan Kocaman
Department of Cardiology, Ankara Güven Hospital; Ankara-*Turkey*

Reference

1. Kocaman SA, Baysan O, Çetin M, Altuner TK, Ocaklı EP, Durakoğlugil ME, et al. An increase in epicardial adipose tissue is strongly associated with carotid intima-media thickness and atherosclerotic plaque, but LDL only with the plaque. Anatol J Cardiol 2017; 17: 56-63.

Address for Correspondence: Dr. Sinan Altan Kocaman
Ankara Güven Hastanesi, Kardiyoloji Bölümü
Ankara-*Türkiye*
E-mail: sinanaltan@gmail.com

Predictors of postoperative atrial fibrillation after coronary artery bypass grafting surgery

To the Editor,

We read the article written by Geçmen et al. (1) titled "SYNTAX score predicts postoperative atrial fibrillation in patients undergoing on-pump isolated coronary artery bypass grafting surgery" published in Anatol J Cardiol 2016;16:655-61 with great interest. In their study, the authors reported that there was an independent association between age, chronic obstructive pulmonary disease, and SYNTAX score in predicting postoperative atrial fibrillation. We would like to emphasize some important points about this well-written study.

It has been demonstrated that volume overload could increase postoperative atrial fibrillation incidence by elevating intraatrial pressure (2). It has also been reported that increased cross-clamp and cardiopulmonary bypass time could increase risk for postoperative atrial fibrillation (3). We think that intraoperative factors should be taken into consideration when evaluating these patients.

Another important point is that body mass index, presence of metabolic syndrome, and waist-to-hip ratio are important markers for coronary artery disease, and moreover, obesity is associated with higher levels of inflammatory cytokines in circulation (4). As inflammation has been shown to cause deterioration in atrial conduction and predispose patients to develop atrial fibrillation postoperatively, authors should state these factors for each group (5).

In our opinion, to verify whether SYNTAX score is an important predictor of postoperative atrial fibrillation development, the