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Author's Reply

Dear Editor,

I have read with great interest the letter to the editor entitled 'Increased Mean Platelet Volume in Patients with Familial Mediterranean Fever (FMF) may not be a Marker of Atherosclerosis Risk'(1). Our main aim in this study was not to search platelet functions in familial Mediterranean patients. Mean platelet volume (MPV) measurement was an additional parameter to evaluate the atherosclerosis risk of these patients. As have been demonstrated by many studies before increased MPV was found to be related to increased cardiovascular and cerebrovascular disease risk. In a wide spectrum from stable angina pectoris to the acute coronary syndromes and acute cerebrovascular attacks MPV increases in these pathological conditions and even in some disease states, it is closely related to the prognosis.

MPV measurement time was in the first hour of the sample collection. Blood was taken to the ethylenediaminetetraacetic acid containing tubes.

On the other hand, MPV increase in FMF patients can not solely be attributed to the C-reactive protein (CRP) increase. There was a difference at CRP level between control group and FMF patients, but this cannot explain the MPV difference between these groups. Because CRP level increased during attack period in FMF patients compared to attack free period. According to the CRP-MPV relationship theory, we should expect an increase in MPV during acute attack period. However, as seen in the manuscript there is no significant difference in MPV between attack and attack free period. In addition, MPV level was found to be decreased in ankylosing spondyloarthritis, ulcerative colitis and Crohn's disease, which are characterized with high CRP levels.

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References

1. Karakurt Artürk O, Üreten K, Sarı M, Yazıhan N, Ermiş E, Ergüder I. Relationship of paraoxonase-1, malondialdehyde and mean platelet volume with markers of atherosclerosis in familial Mediterranean fever: an observational study. Anadolu Kardiyol Derg 2013; 13: 357-62.

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Mean platelet volume in patients with idiopathic and ischemic cardiomyopathy

İdiyopatik ve iskemik kardiyomiopati hastalarda ortalama trombosit hacmi

We have read the article published in the Anatolian Journal of Cardiology by Açıkgöz et al. (1) with a great interest. They investigated mean platelet volume (MPV) in patients with idiopathic cardiomyopathy (CMP) and ischemic CMP and compared these values with those of the controls. They also investigated whether there is a relationship between MPV and echocardiographic parameters in patients with CMP.

They have shown that MPV values were significantly higher in patients with idiopathic and ischemic CMP than those of the controls. The MPV values were not different in patients with idiopathic CMP and patients with ischemic CMP. The MPV values positively correlated with left ventricular end-diastolic and end-systolic diameters and left atrial diameter, but inversely correlated with left ventricular ejection fraction. In conclusion, they speculated that regardless of the etiology, patients with idiopathic or ischemic CMP have higher MPV values indicating increased platelet activation when compared to controls and an enlarged dysfunctional left ventricle is also associated with higher MPV values. This is a very interesting study. On the other hand, we want to make minor criticism about this study from the methodological and pathophysiological aspect.

In generally method of MPV assessing is correct. They studied the blood samples within 2 hours to prevent EDTA induced swelling. On the other hand, there are significant associations of MPV with type 2 diabetes mellitus, prediabetes, acute coronary syndromes, smoking, hypertension, hypercholesterolemia, obesity, metabolic syndrome, atrial fibrillation and some cardiovascular drug use (2). Although there are no statistically difference between three groups in terms of diabetes mellitus, hypertension and smoking, they did not mention about the body mass index, cholesterol levels and cardiovascular drugs used in heart failure and coronary artery disease in patients with idiopathic or ischemic CMP and control subjects. These factors can greatly influence the MPV values. It has been shown that statins might decrease MPV values (3) and beta blockers might increase MPV values (4).

Platelet size is regulated at the level of the megakaryocyte. It has been reported that cytokines such as interleukin-3 and interleukin-6 (IL-6) influence megakaryocyte ploidy and can lead to the production of more reactive and larger platelets (4). On the other hand, serum IL-6 levels were shown to be elevated in patients with heart failure (5). So, IL-6, a major inflammatory cytokine which increased in patients with heart failure can cause an increase in MPV values by stimulating the megakaryocyte ploidy (6).

Platelet activation has a great role in pathophysiology of diseases prone to thrombosis and inflammation. It has been accepted that MPV is a link between thrombosis and inflammation (7). We can speculate that low grade chronic inflammation exists in patients with heart failure and this in turn causes increase in platelet reactivity as measured by MPV in these patients.

In addition, they didn't find a difference in MPV values between patients with idiopathic CMP and patients with ischemic CMP. This is

not surprising. It has been shown that MPV was elevated in acute coronary syndromes, not in chronic stable coronary artery disease (8). The prognostic studies are needed to determine the predictive value of MPV in occurrence of thromboembolic complications in patients with ischemic and idiopathic CMP.

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References

1. Açıkgöz N, Ermiş N, Yağmur J, Cansel M, Ataş H, Pekdemir H, et al. The evaluation of mean platelet volume levels in patients with idiopathic and ischemic cardiomyopathy: an observational study. *Anadolu Kardiyol Derg* 2011; 11: 595-9.
2. Vizioli L, Muscari S, Muscari A. The relationship of mean platelet volume with the risk and prognosis of cardiovascular diseases. *Int J Clin Pract* 2009; 63: 1509-15. [CrossRef]
3. Çoban E, Afacan B. The effect of rosuvastatin treatment on the mean platelet volume in patients with uncontrolled primary dyslipidemia with hypolipidemic diet treatment. *Platelets* 2008; 19: 111-4. [CrossRef]
4. Bakovic D, Pivac N, Eterovic D, Palada I, Valic Z, Paukovic-Sekulic B, et al. Changes in platelet size and spleen volume in response to selective and non-selective beta-adrenoceptor blockade in hypertensive patients. *Clin Exp Pharmacol Physiol* 2009; 36: 441-6. [CrossRef]
5. Debili N, Masse JM, Katz A, Guichard J, Breton-Gorius J, Vainchenker W. Effects of the recombinant hematopoietic growth factors interleukin-3, interleukin-6, stem cell factor, and leukemia inhibitory factor on the megakaryocytic differentiation of CD34+ cells. *Blood* 1993; 82: 84-95.
6. Yan AT, Yan RT, Cushman M, Redheuil A, Tracy RP, Arnett DK, et al. Relationship of interleukin-6 with regional and global left-ventricular function in asymptomatic individuals without clinical cardiovascular disease: insights from the Multi-Ethnic Study of Atherosclerosis. *Eur Heart J* 2010; 31: 875-82. [CrossRef]
7. Gasparyan AY, Ayvazyan L, Mikhailidis DP, Kitas GD. Mean platelet volume: a link between thrombosis and inflammation? *Curr Pharm Des* 2011; 17: 47-58. [CrossRef]
8. Chu SG, Becker RC, Berger PB, Bhatt DL, Eikelboom JW, Konkle B, et al. Mean platelet volume as a predictor of cardiovascular risk: a systematic review and meta-analysis. *J Thromb Haemost* 2010; 8: 148-56. [CrossRef]

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Author's Reply

Dear Editor,

I would like to thank you for your criticism to our study published in *The Anatolian Journal of Cardiology* (1).

Cardiomyopathies (CMP) are a group of disorders associated with an increased risk of thromboembolism due to low output state, blood

stasis by a dilated chamber and poorly contracting ventricle, platelet activation and altered coagulation status (2-4). Mean platelet volume (MPV) is an indicator of platelet activation (5). We have found that patients with idiopathic or ischemic CMP have higher MPV values indicating tendency to platelet activation regardless of the etiology, when compared to controls and an enlarged dysfunctional left ventricle may also be associated with higher MPV values (1).

It is known that there are significant associations of MPV with type 2 diabetes mellitus, pre-diabetes, acute coronary syndromes, smoking, hypertension, hypercholesterolemia, obesity, metabolic syndrome, atrial fibrillation and some cardiovascular drug use (6). Although there was no statistically significant difference between three groups in terms of diabetes mellitus, hypertension and smoking, the body mass index and cholesterol levels, we didn't mention about the body mass index and cholesterol levels in our study. On the other hand, regarding cardiovascular drug treatment, our study groups were also consisted of healthy control subjects as well as the patients with ischemic and dilated CMP. Cardiovascular drug usage is also a variable which is very difficult to make it similar in all groups. For this reason, cardiovascular drug usage may be a potential confounding factor in our study. As a result, the important limitation of our study is that the cardiovascular drug treatment has not been standardized in all groups. Moreover, this situation have already been mentioned as a limitation of our study.

Your speculation about the link between MPV values and low grade chronic inflammation in heart failure patients is of great interest. This issue, the relationship between thrombosis and inflammation, may also be a subject of another study. Because of that, we did not measure these inflammatory marker levels in our study.

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References

1. Açıkgöz N, Ermiş N, Yağmur J, Cansel M, Ataş H, Pekdemir H, et al. The evaluation of mean platelet volume levels in patients with idiopathic and ischemic cardiomyopathy: an observational study. *Anadolu Kardiyol Derg* 2011; 11: 595-9.
2. Koniaris LS, Goldhaber SZ. Anticoagulation in dilated cardiomyopathy. *J Am Coll Cardiol* 1998; 31: 745-8. [CrossRef]
3. Lip GY, Gibbs CR. Does heart failure confer a hypercoagulable state? Virchow's triad revisited. *J Am Coll Cardiol* 1999; 33: 1424-6.
4. Dotsenko O, Kakkar VV. Antithrombotic therapy in patients with chronic heart failure: rationale, clinical evidence and practical implications. *J Thromb Haemost* 2007; 5: 224-31. [CrossRef]
5. Park Y, Schoene N, Harris W. Mean platelet volume as an indicator of platelet activation: methodological issues. *Platelets* 2002; 13: 301-6. [CrossRef]
6. Vizioli L, Muscari S, Muscari A. The relationship of mean platelet volume with the risk and prognosis of cardiovascular diseases. *Int J Clin Pract* 2009; 63: 1509-15. [CrossRef]

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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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References

1. Erdoğan T, Çetin M, Kocaman SA, Dorukoğlu ME, Ergül E, Uğurlu Y, et al. Epicardial adipose tissue is independently associated with increased left ventricular mass in untreated hypertensive patients: an observational study. *Anadolu Kardiyol Derg* 2013; 13: 320-7.
2. Shemirani H, Khoshavi M. Correlation of echocardiographic epicardial fat thickness with severity of coronary artery disease-an observational study. *Anadolu Kardiyol Derg* 2012; 12: 200-5.
3. Lai YH, Yun CH, Yang FS, Liu CC, Wu YJ, Kuo JY, et al. Epicardial adipose tissue relating to anthropometrics, metabolic derangements and fatty liver disease independently contributes to serum high-sensitivity C-reactive protein beyond body fat composition: a study validated with computed tomography. *J Am Soc Echocardiogr* 2012; 25: 234-41. [CrossRef]
4. Turan MN, Güngör O, Aşçı G, Kircelli F, Acar T, Yaprak M, et al. Epicardial adipose tissue volume and cardiovascular disease in hemodialysis patients. *Atherosclerosis* 2013; 226: 129-33. [CrossRef]
5. Şengül C, Çevik C, Özveren O, Oduncu V, Sünbül A, Akgün T, et al. Echocardiographic epicardial fat thickness is associated with carotid intima-media thickness in patients with metabolic syndrome. *Echocardiography* 2011; 28: 853-8. [CrossRef]
6. Demirkol S, Balta S, Ünlü M, Yüksel UC, Çelik T, Arslan Z, et al. Evaluation of the mean platelet volume in patients with cardiac syndrome X. *Clinics* 2012; 67: 1019-22. [CrossRef]
7. Şengül C, Özveren O. Epicardial adipose tissue: a review of physiology, pathophysiology, and clinical applications. *Anadolu Kardiyol Derg* 2013; 13: 261-5.

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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,