to be an independent predictor of NSTE-ACS in young patients. Also, MPV of the young patients with NSTE-ACS was found to be significantly higher than the MPV of the subjects of control group.

As mentioned in the study, we aimed to provide additional information to the literature about acute coronary syndromes of young patients which is quite limited.

There are two criticism points in the aforementioned letter; one is about the control group which is said necessarily to be formed by the elderly NSTE-ACS patients, the other is the low number of patients.

Firstly, as mentioned before our hypothesis was; an increased MPV predicts development of NSTE-ACS in young patients. Namely, our target population is young patients. For this goal we formed the control group with subjects, younger than 45 years old with normal coronary arteries and without acute coronary syndromes. Thereby we could evaluate the predictive value of MPV on the NSTE-ACS in young patients. In other words, we evaluated the predictive value of a variable -MPV- about a risk factor -ACS- in a population of young patients-. If we had an aim of evaluating the relationship between age and MPV among NSTE-ACS patients, or, if our hypothesis was; MPV of young NSTE-ACS patients are higher than of the elderly NSTE-ACS patients, we could have an elderly NSTE-ACS control group. Because of the reasons we mentioned, we did not form the control group by the elderly patients and by the NSTE-ACS patients.

Secondly, we mentioned about the low number of patients as a major limitation in the text. New studies may be designed with high numbers and provide additional data about this issue.

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Increased mean platelet volume in patients with familial Mediterranean fever may not be a marker of atherosclerosis risk

Ailevi Akdeniz Ateşli olgularda ortalama trombosit hacmindeki artış ateroskleroz riski artışının bir belirteci olmayabilir

Dear Editor,

I have read with great interest the online published article of Karakurt Arıtürk et al. (1) about the early markers of atherosclerosis in patients with Familial Mediterranean Fever (FMF) by the measurements of serum paraoxonase-1 activity, mean platelet volume (MPV) and malondialdehyde level. Authors suggested that MPV is a marker of increased platelet activity. The current "gold standard" test of platelet function is turbidometric platelet aggregometry. Beyan et al. (2) did not observe any correlation between platelet indices measured including mean platelet volume and platelet aggregation responses obtained with turbidometric platelet aggregometry in healthy subjects. MPV shows only the rate of platelet production. There is an inverse relationship between the normal platelet count and the normal MPV, resulting in a roughly constant circulating platelet mass (3). A high MPV in a thrombocytopenic patient indicates active marrow production of platelets, whereas a low MPV is indicative of marrow suppression.

In addition, MPV measurement time was not defined in materials and method of this study. The MPV varies with time in ethylenediaminetetraacetic acid (EDTA)-anticoagulated samples (4). EDTA-induced platelet shape changes result in a progressive increase in MPV with impedance technology. The MPV increases up to 30 percent within five minutes of exposure with EDTA and increases further by 10 to 15 percent over the next two hours with impedance technology. However, some investigators have reported variable increases in MPV with EDTA storage up to 50 percent. Therefore, MPV should not be reported as a routine part of the complete blood count, because of the EDTA-induced changes over time (4).

On the other hand, results of this study may be telling the truth and the patients with FMF may have higher MPV values compared to control subjects. FMF is a chronic inflammatory disease and level of C-reactive protein (CRP), the prototypic human acute phase reactant increase during attacks. Potempa et al. (5) showed that a modified form of CRP had thrombopoietic activity in both in vitro and in vivo mouse models. Increased CRP during acute-phase response may be contribute increased platelet production and increased MPV.

As a result, increased MPV in patients with FMF may not be a marker of atherosclerosis risk.

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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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Mean platelet volume in patients with idiopathic and ischemic cardiomyopathy

İdiyopatik ve iskemik kardiyomiyopatili 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