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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

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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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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