

and written study. However, I want to make minor criticism for this study from the methodological aspect.

Arterial stiffness is a complex process associated with confounding factors. Cecelja et al. (2) published a systematic review which showed that the contribution of CV risk factors other than age and BP to aortic stiffness measured by carotid-femoral pulse wave velocity is small or insignificant and age and BP were consistently independently associated with aortic stiffness. It has also been shown that some antihypertensive drugs like angiotensin-converting enzyme inhibitors, calcium channel blockers and spironolactone reduce arterial stiffness (3-6). Recently, it has also been shown that in addition to angiotensin-converting enzyme inhibitors, beta blockers and aliskiren, direct renin inhibitor, also reduce arterial stiffness (6). Tok et al. (1) study included 63% hypertensive patients in metabolic syndrome group. However, there are no details regarding the antihypertensive drugs used. Similarly, statins also reduce arterial stiffness but there are no details regarding their use (3). Antihypertensive drugs and statins are used frequently in metabolic syndrome patients. From this aspect, antihypertensive drugs and statins should be considered in aortic stiffness evaluation in metabolic syndrome patients.

Arterial stiffness describes the reduced capability of an artery to expand and contract in response to pressure changes and it is an independent predictor of cardiovascular morbidity and all-cause mortality (3). It has been suggested that aortic stiffness occurs as a result of atherosclerosis along the aorta. However, it is closely associated with age, hypertension, antihypertensive drugs and statins. Antihypertensive drugs and statins can influence arterial stiffness. It would be helpful if the authors provided this information.

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Author's Reply

To the Editor,

Thank you very much for your very helpful comments on our article published in May issue of *The Anatolian Journal of Cardiology* 2013; 13: 227-34 (1). As you mentioned 63% of our study patients were on antihypertensive medication and 12% of patients (unpublished data) were on statins. In literature there is controversial results on effects of the different antihypertensive agents or statins on aortic stiffness (2, 3).

In our study, use of antihypertensive agents and statins could have some positive implications on aortic stiffness parameters. In fact, we were still be able to show the increased aortic stiffness in metabolic syndrome patients compared to healthy controls despite the fact that aortic stiffness might has been reduced by antihypertensive drugs and statins. Aortic stiffness should be evaluated in further studies performed in drug naive metabolic syndrome patients.

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Related factors should be considered in evaluation of mean platelet volume in patients with familial Mediterranean fever

To the Editor,

I read the article published by Karakurt et al. (1) published in June 2013 issue of *Anatolian Journal of Cardiology* with great interest. They assessed the early markers of atherosclerosis in patients with familial Mediterranean fever (FMF) by the measurements of serum paraoxonase-1 (PON-1) activity, mean platelet volume (MPV) and malondialdehyde (MDA) level. The mean PON-1 activity in FMF patients was significantly lower than in the healthy population. Serum MDA levels were the same between the groups. MPV was higher in FMF patients than in the control group. PON, MPV and MDA levels were the same in FMF patients with acute attack

and attack -free period. I congratulate the authors for this study. On the other hand, there are some problems in MPV measurement methodology and I want to make minor criticism about this study from this aspect.

Firstly, methods section is not clear. They did not mention about the tube that blood sample collected and the time interval between blood sampling and analysis. This is very important. It is clear that platelets exhibit a time dependent swelling when blood samples are anticoagulated with ethylenediaminetetraacetic acid (EDTA), while minimal swelling occur in the presence of citrate (2). With impedance counting, the MPV increases over time as platelets swell in EDTA, with increases of 7.9% within 30 min. The recommended an optimal measuring time of MPV is 120 min after venipuncture (2). For reliable MPV measurement, the potential influence of anticoagulant on the MPV must be carefully controlled, either using an alternative anticoagulant (such as citrate) or standardizing the time delay between sampling and analysis (less than 2 h). This situation is not clear in study.

Secondly, there are significant associations between MPV and type 2 diabetes mellitus, pre-diabetes, acute coronary syndromes, moderate to severe valvular heart disease, smoking, hypertension, hypercholesterolemia, obesity, the metabolic syndrome, stroke, drug use affecting MPV like statin and antihypertensive drugs (3). As a result, MPV is highly variable and dependent on multiple related factors. They excluded hypertension, diabetes mellitus, congestive heart failure, malignancies, renal, hepatic and thyroid diseases, and immunological diseases, patients with acute coronary syndromes, coronary heart disease and severe valvular heart diseases. On the other hand, they didn't mention about the body mass index, smoking and metabolic syndrome incidence in patients and controls. It has been shown that obesity, smoking and metabolic syndrome increase MPV values. It would have been useful if the authors had provided information about these factors.

Thirdly, 75% of the patients were on colchicine treatment. It has to be kept in mind that colchicine can increase MPV (4). As a result, colchicine might have a role in increased MPV in patients with FMF in some degree. It would be better if the authors mention about this effect of colchicine on MPV.

MPV might be a link between thrombosis and inflammation (5). It might be speculated that low grade chronic inflammation exists in patients with FMF and this in turn causes increased platelet reactivity as measured by MPV in these patients. However, all related factors including drugs should be taken into account and standardized methods must be used in MPV measurement.

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Author's Reply

To the Editor,

I have read with great interest the letter to the editor entitled 'Related factors should be considered in evaluation of mean platelet volume in patients with familial Mediterranean fever' published in this issue of *Anatolian Journal of Cardiology* (1). In our study, mean platelet volume (MPV) measurement time was the first hour of the sample collection (2). Blood was taken to the ethylenediaminetetraacetic acid (EDTA) containing tubes. As have been stated in the letter to the editor, MPV were measured in the optimal time range that means less than two hours (1). Body mass index values, metabolic syndrome and smoking prevalence were similar between the control subjects and familial mediterranean fever patients. MPV difference cannot be attributed to these parameters. In the manuscript by Laufer et al. (3) colchine effect on platelet size had been studied *in vitro*. But there is not any study searching colchine effect on MPV *in vivo*, at animals or humans. So that we cannot conclude that colchine increase MPV in humans. In our study, MPV was similar between patients who were on colchicine treatment and not on colchicine treatment. Even a slight decrease at MPV was observed with colchicine treatment but this did not reach statistical significance. Because of the small number of patients who were not on colchine treatment, only 7.5% of patients, we think that colchicine effect on MPV should be investigated in other studies with large patient numbers.

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