studies were retrospective in nature (1, 2). Apart from our study, Keskin et al. (2) conducted a study on a larger population and evaluated the mean serum potassium (sK) level rather than the admission sK level. Moreover, they differently categorized patients in terms of mean sK level (<3.0, 3.0–<3.5, 3.5–<4.0, 4.0–<4.5, 4.5–<5.0, 5.0–<5.5, and ≥5.5 mmol/L) (2). In our study, we categorized patients based on the admission sK level as <3.5, 3.5–<4, 4–<4.5, 4.5–<5, and ≥5 mmol/L (1).

The main finding of our study was the relation between admission sK level of >4.5 mmol/L and increased long-term mortality (1). The current guidelines recommend sK level of 4.0–5.0 mmol/L in patients with acute myocardial infarction (3). The results of recently undertaken studies and those of Keskin et al.'s study (2) were in accordance with our study (4). Moreover, we showed that the lowest mortality was associated with sK levels of 3.5–<4 mmol/L, which is similar to the findings by Choi et al.'s study (4). Keskin et al. (2) showed that the optimal sK level was 3.5–4.5 mmol/L, with the lowest mortality being associated with sK levels of 4.0–4.5 mmol/L. Another similar finding was the association between ventricular arrhythmias and sK level. Both studies showed that ventricular arrhythmias were associated with sK level of <3 mmol/L (1, 2). In addition, in our study, we also found that admission sK level of ≥5 mmol/L is associated with ventricular arrhythmias (1).

The recommended level of sK was done in rather an early time (3). Over time, following the release of the guidelines, various drugs and revascularization techniques and strategies have been developed. The combined findings from retrospective studies have pointed out that the most favorable clinical outcomes occurred with sK level between 3.5–4.5 mmol/L in acute myocardial infarction (1, 2, 4). In order to prevent ventricular arrhythmias, the same sK level should be maintained. Even though various retrospective studies demonstrated similar clinical end points, prospective studies are needed for strong advisement.

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LA thrombus formation in mitral valve disease

To the Editor,

Nonvalvular atrial fibrillation (AF) and mitral valve disease with AF are important causes of left atrial thrombus (1). Abnormal left atrial volume, changed left atrial anatomy, abnormal atrial contraction, abnormal blood flow into the atrium, impaired endothelial function in left atrium, and low mitral valve area are major predisposing factors consisting of thrombus in left atrium and also arrhythmia (2). These factors cause thrombogenic and arrhythmogenic tendencies in the atrium.

We have read with great interest the article by Belen entitled "Relationship between the presence of left atrial thrombus in patients with mitral stenosis and platelet-to-lymphocyte ratio" published in Anatol J Cardiol 2016; 16: 673-7 (3). The authors concluded that the presence of AF is an important risk factor for thrombus development in patients with mitral stenosis. A higher incidence of AF was determined in the left atrial thrombus group in their study [LA thrombus (+) group: 38 (41.3%), n=92 and LA thrombus (–) group: 31 (12%), n=259]. Presence of AF was found to be independently associated with the presence of LA thrombus. We agree with these statements. However, we observed that the patient population between the groups was not equal. Therefore, we cannot associate platelet-to-lymphocyte ratio with the presence of left atrial thrombus in these non-homogenous groups. If we want to investigate the platelet-to-lymphocyte ratio and thrombus, we have to equalize thrombogenic variables, such as AF, mitral valve area, mean gradient, and left atrial volume, between the two groups for obtaining more significant comments.

They concluded that there was a relationship between the presence of left atrial thrombus and platelet-to-lymphocyte ratio, which was independent of other important factors, including AF, in multivariate analysis. However, Table 2 shows that there is no significant difference between AF and platelet-to-lymphocyte ratio in univariate and multivariate analyses (AF-multivariate, p=0.014; platelet-to-lymphocyte ratio: univariate, p=0.02 and multivariate, p=0.016). We think that abnormal surface formation as in left atrial enlargement, inflammatory activation and stasis as in rheumatic valve disease and atrial fibrillation, and inflammatory capacity are not independent of each other (1, 2). Biological

markers, such as platelet-to-lymphocyte ratio and red cell distribution width, are affected by inflammatory reactions and abnormal blood flow dynamics in the vessels. It is difficult to determine these markers as independent markers, and we believe that all these anatomic and biochemical variables affect each other. These variables are indicators of diseases with low specificity.

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Author`s Reply

To the Editor,

We thank the authors for their valuable evaluation of our article entitled "Relationship between the presence of left atrial thrombus in patients with mitral stenosis and platelet-to-lymphocyte ratio" published in Anatol J Cardiol 2016; 16: 673-7 (1).

The aim of our study was to examine the relationship between the presence of left atrial (LA) thrombus and platelet-to-lymphocyte ratio (PLR), a marker of inflammatory process, in patients with rheumatic mitral valve stenosis (RMVS). This prospective cross-sectional study included patients with a mitral valve area less than 2 cm^2. Patients were divided into two groups: those with and without LA thrombus. Because of the pathophysiological properties of the disease, it is normal for variables, such as atrial fibrillation (AF), mitral valve area, mean gradient, and left atrial volume, to be different between the groups.

The relationship between AF and LA thrombus formation is also strong in patients with RMVS. It is known that rheumatic heart diseases are autoimmune inflammatory pathologies and that inflammation subclinically continues (2, 3). One of the primary aims of the study was to examine the relationship between thrombus formation and PLR, which is a marker of inflammatory activity. While the presence of AF was associated with LA thrombus formation, this was independent of PLR levels. While its role of inflammation in the pathophysiology of AF is known, the presence of paroxysmal AF, ambulatory ECG recordings, or time of AF were not recorded as they were not the primary aim of the study. Even though the relationship between inflammation and thrombus formation does not mean that there should be a relationship between PLR and AF, the presence of a link between AF and PLR may have been affected by the lack of these prelated parameters. At the same time, regression analysis results demonstrated that the relationship between PLR levels and AF continued to be independent of variables, such as AF, mitral valve area, mean gradient, and left atrial volume.

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