Effect of mean serum potassium level on in-hospital and long-term outcomes in ST segment elevation myocardial infarction

To the Editor,

Serum potassium level plays a substantial role in cardiovascular disorders. Based on previous studies, current guidelines for serum potassium in cardiovascular disorders recommend maintaining the serum potassium level of 4–4.5 mEq/L in patients with AMI (1, 2). Recent studies have examined the recommendations of the guidelines, and the lowest mortality was observed with serum potassium level of 3.5–4 mEq/L in patients with AMI (3, 4). Because of important improvements in revascularization and drug therapies, it is important to re-evaluate the impact of serum potassium level with respect to mortality and ventricular arrhythmias in patients with AMI. To address this critical diversity, further investigations are needed for confirmation. We report a study evaluating the impact of mean serum potassium level on in-hospital and long-term outcomes in a large patient population with STEMI (5). In total, 3,760 consecutive patients diagnosed with STEMI were retrospectively analyzed. At least two serum potassium measurements were taken from each patient, and the mean serum potassium levels were categorized accordingly: <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 mEq/L. Hierarchical logistic regression and Cox-proportional regression analysis were used to establish the relationship between mean serum potassium levels and clinical outcomes. The lowest in-hospital and long-term mortality was determined in patients with serum potassium level of 4–<4.5 mEq/L, whereas mortality was higher in patients with serum potassium levels of ≥5.0 and <3.5 mEq/L. There was a U-shaped association between mean serum potassium level and mortality. In multivariable hierarchical logistic regression analysis, in-hospital mortality risks were higher for patients with serum potassium level of ≥5 mEq/L [odds ratio (OR), 2.60; 95% confidence interval (CI), 1.30–4.2 and OR, 3.22; 95% CI, 1.14–9.07 for patients with serum potassium levels of 5–<5.5 mEq/L and ≥5.5 mEq/L, respectively]. In a multivariable Cox-proportional regression analysis, the mortality risk was higher for patients with serum potassium levels of ≥5 mEq/L [hazard ratio (HR), 2.11; 95% CI, 1.23–4.74 and HR, 4.20; 95% CI, 1.08–8.23, for patients with potassium levels of 5–<5.5 mEq/L and ≥5.5 mEq/L, respectively]. In-hospital and long-term mortality risks were also higher for patients with serum potassium levels of ≤3.5 mEq/L. Conversely, ventricular arrhythmias were higher only for patients with serum potassium levels of ≤ 3.5 mEq/L. Therefore, serum potassium level plays a substantial role in patients with AMI in terms of mortality and ventricular arrhythmias; however, there is no current consensus on optimal serum potassium level in patients with AMI. The current study challenges the current guidelines in clinical practice, which recommend maintaining serum potassium level at 4.0–5.0 mEq/L in patients with AMI. In addition, our findings were in line with recent studies with respect to maintaining serum potassium level at 3.5–4.5 mEq/L. Although the lowest mortality and ventricular arrhythmia range in the current study was 4.0–4.5 mEq/L, it was 3.5–4.0 mEq/L in the recent studies.

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Author`s Reply

To the Editor,

First, we would like to thank the author(s) for their interest and valuable contribution to our research. Both studies were designed in a similar manner (1, 2). The studied populations were both ST-elevation myocardial infarction patients who had undergone primary percutaneous coronary intervention (1, 2). Both

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