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Should we consider serum potassium level as a mortality predictor in ST-elevation myocardial infarction?

To the Editor,

I read the article by Uluganyan et al. (1) entitled "Admission serum potassium level is associated with in-hospital and long-term mortality in ST-elevation myocardial infarction" with great interest, which was published online in your Anatol J Cardiol 2015 Febr 11. In their study, the authors reported that the admission serum potassium (sK) level of >4.5 mmol/L was associated with increased long-term mortality in patients with ST-elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention (pPCI). I would like to emphasize some confounding factors that can affect the results of the present study.

First, Uluganyan et al. (1) reported that patients were treated with drugs according to the European Society of Cardiology guidelines on myocardial revascularization. However, there are no data about the type of dual antiplatelet therapy (DAPT). It has been demonstrated that DAPT with ticagrelor reduced mortality than DAPT with clopidogrel in patients with STEMI who underwent pPCI (2). Additionally, in patients with STEMI undergoing pPCI, prasugrel is more effective than clopidogrel for the prevention of cardiovascular death and ischemic events (3). Hence, the higher incidence of treatment with ticagrelor and prasugrel in patients with sK levels of <4.5 may be a reason for lower mortality rates for these patients. Authors should state the incidence of DAPT with prasugrel, ticagrelor, and clopidogrel for each group, respectively.

Second, the authors did not report any data about the usage of aldosterone antagonists. The study by Uluganyan et al. (1) includes patients with impaired left ventricular systolic function. Aldosterone antagonists significantly reduce mortality in post-STEMI patients with left ventricular systolic dysfunction (ejection fraction<40%) (4). Hence, less treatment with aldosterone antagonists may be a reason for higher mortality rates for patients with sK levels of >4.5 mmol/L.

Finally, in the present study by Uluganyan et al. (1), there are no data about time to reperfusion and door-to-balloon time. It is known that delay in reperfusion and longer door-to-balloon time cause higher mortality rates (5, 6). Delay in time to reperfusion and longer door-to-balloon time may be another reason for higher mortality rates in patients with sK levels of >4.5 mmol/L when compared with patients with sK levels of <4.5 mmol/L. Therefore, the authors should state the time to reperfusion and door-to-balloon time for each group, respectively.

In conclusion, sK levels of >4.5 mmol/L may indicate worse outcomes in patients with STEMI undergoing pPCI. However, medical treatments, time to reperfusion, and door-to-balloon time may still affect the results of the study by Uluganyan et al. (1). To define the sK level of >4.5 mmol/L as a predictor of mortality, all factors associated with mortality should be considered.

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Author`s Reply

To the Editor,

We thank the authors for their comments about our article entitled "Admission serum potassium level is associated with in-hospital and

long-term mortality in ST-elevation myocardial infarction" published in Anatol J Cardiol 2015 Feb 11. (1). In the study, we conducted ST elevation myocardial infarction (STEMI) in patients undergoing primary percutaneous coronary intervention (PCI). An admission serum potassium (sK) level of >4.5 mmol/L was found to be associated with short- and long-term mortality (1).

Firstly, all patients in the study were treated with the dual antiplatelet therapy (clopidogrel 75 mg/day and acetylsalicylic acid 100 mg/day) for at least one year (1). Even though ticagrelor and prasugrel are associated with better results in patients with STEMI, during the period the study was conducted, neither prasugrel nor ticagrelor was administered in our center (2, 3). The effect of ticagrelor or prasugrel was not evaluated in our study.

The effect of aldosterone antagonists was not evaluated. The global left ventricular ejection fraction did not significantly differ between groups. Even though we did not evaluate the effect of aldosterone antagonists, in our opinion, this could not affect the outcome between the groups. However, cumulative end points will probably be affected. The effect of aldosterone antagonists could be a part of another study.

Thirdly, all patients with STEMI underwent primary PCI in our center regardless of the admission creatinine level. The patients' blood at the time of admission was drawn at the emergency department without procedure delay. No significant correlation was found between admission sK level and door-to-balloon time (p=0.19).

In conclusion, despite the presence of many confounding factors, we thought that an sK level of >4.5 mmol/L is associated with short- and long-term mortality. The effect of aldosterone antagonists, prasugrel, and ticagrelor could be evaluated in different studies.

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Mean platelet volume: When the size does matter

To the Editor,

Cardiovascular diseases are known to be associated with unstable atherosclerotic plaques matching with platelet reactions, which lead to thrombus formation and finally clinical events (1). Platelets are heterogeneous blood components, differing in size, density, and reactivity. It is recognized that several substances released from alpha-granules, dense granules, lysosomes, or the cytosol in larger platelets are either vasoactive and prothrombotic (thromboxane A2, coagulation factors), adhesion proteins (P-selectin), growth factors (TGF-beta), chemokines (platelet factor 4), or cytokine-like factors (CD40 ligand). These proteins act in a collaborative way to determine biological functions. In other words, activated platelets are larger, and the mean platelet volume (MPV), a measure of platelet size, could be an accurate and easily available marker of platelet activation. Several studies have reported an increasing MPV associated with the prognosis of either acute coronary syndromes (2) or cerebrovascular diseases (3). However, until this issue in which Kalkan et al. (4) entitled "Mean platelet volume is associated with aortic intima-media thickness in patients without clinical manifestation of atherosclerotic cardiovascular disease." published in Anatol J Cardiol 2015; 15: 753-8 report an association between MPV and the extent of subclinical thoracic aortic atherosclerosis in patients without a clinical manifestation of atherosclerotic cardiovascular disease, we did not know the role of this potential marker in the general population without cardiovascular events, namely, in people whose prothrombotic status is unknown or supposedly inactivated. The authors showed how the extent of thoracic aorta intima to media thickness, as a marker of diffuse atherosclerotic disease, is significantly related to an increasing MPV, supporting the role of systemic thrombocyte activation over the course of atherosclerosis, a relationship that has been previously reported, though in a different scenario such as coronary or carotid arteries (5) and again, in patients with atherosclerotic disease present.

The results reported by Kalkan et al. (4) are interesting, though some questions remain to be answered. MPV is uncomplicated and cheap to obtain, easy to elucidate, and is conventionally measured by automated cell counters. Its increase should suggest a careful assessment of cardiovascular risk; however, more studies are necessary in the general population to confirm the findings by Kalkan et al. (4), and new studies investigating the relationship of MPV with future cardiovascular events in healthy people beyond the wall of their arteries.

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