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More About the Effect of Dynamic Potassium Change in STEMI

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We thank our colleagues for their comments[1] on our article entitled "Effect of Dynamic Potassium Change on In-Hospital Mortality, Ventricular Arrhythmias, and Long-Term Mortality in STEMI" published in the January 2018 issue of the *Angiology*.[2]

The aim of our paper[2] was to evaluate the effect of serum potassium (K) deviation (from normokalemia to hypokalemia and normokalemia to hyperkalemia) on in-hospital mortality, ventricular arrhythmias, and long-term mortality in ST segment elevation myocardial infarction (STEMI).

Uluganyan et al showed an increased risk of mortality and ventricular arrhythmias with admission K levels <3 and ≥5 mmol/L in STEMI.[3] Similarly, Keskin et al showed the association of mean serum K levels with in-hospital mortality, ventricular arrhythmias, and long-term mortality after STEMI.[4] The former study shows the importance of early intervention to restore serum K levels to normal limits and to perform more frequent serum K measurements according to admission serum K levels. The latter study draws attention to mean serum K levels derived from all K measurements during hospitalization. We showed[2] the importance of serum K measurements even in patients with normal K levels at admission. In any circumstance, in patients with STEMI, serum K level should be checked at least daily, according to these studies.

Magnesium, one of the essential minerals that serves as a cofactor in more than 300 enzymatic reactions (blood pressure control, lipid peroxidation, and glycemic control), has a critical role in the cardiovascular system.[5] Only 1% (1.5-2.0 mEq/L, 1.7-2.4 mg/dL) of the total body magnesium circulates (24 g) is in serum; the rest is stored either in bone or soft tissue.[5] The biochemical and cellular effects of magnesium could be summarized as follows: (1) activation of adenosine triphosphatase to produce energy from ATP and stabilizing cell membrane (by providing energy needs of the Na^+-K^+ pump), (2) appropriate cell membrane polarization (malignant arrhythmias caused by low serum magnesium levels), (3) to be a cofactor of the cardiac mitochondria, and (4) modulation of the K proton exchange (protective against K loss). Arterial vasospasm, increased catecholamine release, increased fatty acids and lipids, and intravascular hypercoagulability could be associated with low cellular levels of magnesium.[6,7] Besides these effects of magnesium on the cardiovascular system are usually overlooked. It was shown that in hospitalized patients only 7% of patients had their magnesium levels checked despite 42% of them having hypomagnesaemia.[8] Unfortunately, we had no routine measurements of admission serum magnesium levels in patients with STEMI in our center. The measurement of serum magnesium levels was performed only in selected patients such as those with malignant cardiac arrhythmias and severe serum electrolyte disturbances; magnesium replacement was administered if levels were low. This could be a limitation which we did not mention.

Another issue that was not discussed in the paper[2] was the use of proton pump inhibitors (PPIs). Treatment with PPIs is suggested in patients receiving dual antiplatelet therapy with a class I recommendation and a level of evidence B in the last update of the European Society of Cardiology guidelines.[9] However, PPI usage could be associated with lower magnesium levels.[10] Since most of the study population received PPI treatment during hospitalization, we did not consider reporting PPI usage in the results.

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