




Reply to G. Betts's letter referring to "Serum potassium dynamics during acute heart failure hospitalization"

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Sirs,

We would like to thank Dr. Betts for her comments on our article entitled "Serum potassium dynamics during acute heart failure hospitalization [1]." As Dr. Betts points out, there is strong evidence about the high frequency and prognostic implications of serum potassium disorders and their dynamic changes in heart failure (HF) patients. However, we want to highlight some aspects we consider relevant:

1. The prevalence of dyskalemia in HF varies between different studies due to different cut-off points employed for its diagnosis. The most accepted cut-off points to define hypokalemia and hyperkalemia are $K^+ < 3.5$ mEq/L

and $K^+ > 5$ mEq/L, respectively [2]. Dyskalemia is more frequent among chronic HF [3] patients (25%) than in the setting of acute HF (19.5%) [1]. These differences in dyskalemia frequency may be mediated by the longer follow-up times in chronic HF. Furthermore, the dyskalemic pattern observed in these two groups of patients is different, with a tendency towards hypokalemia in acute heart failure and hyperkalemia among chronic heart failure patients. This may be explained by the effect of diuretic treatment on serum potassium during acute HF hospitalization and renin–angiotensin–aldosterone system inhibitors in chronic HF patients.

2. The most common risk factors for hyperkalemia are advanced age, previous treatment with renin–angiotensin–aldosterone system inhibitors, and a personal history of chronic kidney disease or diabetes mellitus.

3. Dyskalemia is strongly associated with a worse prognosis, drawing a U-shaped curve, with a higher risk of mortality for those levels far from the central values. This suggests the existence of a narrow safety window ranging from 4 to 5 mEq/L. It is still unknown whether this association results from a direct effect of dyskalemia (arrhythmias); or whether the appearance of abnormal serum potassium values is only identifying a more fragile patient, with more significant comorbidity and with a lower rate of prescription of HF neurohormonal treatment. In our population, we observed that as the burden of comorbidities (evaluated by the Charlson index) increases, the risk of the appearance of dyskalemia does. These results are coincident with those observed in previously published works [4]. Due to the observational nature of these works, it is not possible to infer causality. Therefore, randomized studies are necessary to prove whether treatments aiming to correct dyskalemia may improve HF patients' prognosis. Ongoing studies with new potassium binders will help to solve this dilemma.

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To summarize, our work highlights the importance of closely monitoring potassium levels during acute HF hospitalizations. It may help identify patients at a higher risk of adverse events during follow-up.

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Compliance with ethical standards

Conflict of interest The authors have no conflict of interest to declare.

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