The Reply

Mirrakhimov emphasizes the importance of determining the etiology of hyperkalemia, and especially whether or not the patient in question has underlying chronic kidney disease or end-stage renal disease. The presence or absence of normal kidney function indeed may affect how hyperkalemia is managed, in several ways. First of all, the presence of normal kidney function allows the use of certain interventions that may not be available to patients with little or no kidney function; such as the use of diuretics or forced diuresis.1 Second, with normal kidney function there is indeed the possibility of rapid resolution of hyperkalemia, provided that the kidney’s homeostatic responses are not blunted or abrogated by concomitant conditions (eg, medications such as renin-angiotensin system inhibitors or ongoing rapid release from endogenous sources of potassium such as tissue injury).2 I would, however, caution against developing a false sense of security regarding hyperkalemia in patients with normal kidney function, as its presence has been associated with substantially higher mortality compared with patients with chronic kidney disease or end-stage renal disease with similar severities of elevated potassium.3 The reason for this difference is unclear, but may be related to the fact that hyperkalemia is much rarer in patients with normal kidney function vs those with kidney disease,3 and hence the latter group may be more accustomed to hyperkalemia. Thus, I would agree with Mirrakhimov that an electrocardiogram (ECG) is always necessary to assess the electrophysiologic relevance of hyperkalemia. Third, patients with end-stage renal disease lend themselves to dialytic therapy, as dialysis vascular access is typically readily available and dialytic removal of potassium leads to definitive resolution of hyperkalemia (as opposed to therapies facilitating membrane stabilization or potassium redistribution). Nevertheless, here too I would advise caution. Hyperkalemia being extremely common and easy to treat in dialysis patients, it may be tempting to limit its management to dialysis. It is worth remembering that end-stage renal disease patients are at high risk for the development of arrhythmias of any kind, due to the high prevalence of pathological left ventricular hypertrophy in this group,4 and also due to the frequent concomitant presence of other electrolyte abnormalities predisposing to arrhythmias, such as hypomagnesemia5 or hypocalcemia.6 Furthermore, correction of hyperkalemia with hemodialysis may also pose additional hazards by virtue of the sudden decrease in serum potassium and the rapid development of hypokalemia, in itself arrhythmogenic. It is thus important that hyperkalemia is addressed the same way in this group as it would be in patients with normal kidney function; namely by performing an ECG and implementing immediate medical measures in case significant electrophysiologic abnormalities are detected.

In summary, while I agree with the tenets espoused by Mirrakhimov regarding the need for physiology-based thinking, especially when treating hyperkalemia chronically, I would also suggest that the presence of hyperkalemia should trigger an emergent assessment of its electrophysiologic effects (by performing an ECG) under most, or all, circumstances.

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References


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