Unexpected Therapeutic Response to Spironolactone: A Prospective Debate on Aldosterone and Potassium Ion in Hypertension
Letter to Editor:

A recent editorial from Goodfriend1 and the article by Chapman et al.2 published in Hypertension, addressed the noteworthy pharmacological effects of low doses of spironolactone in patients who failed to respond to 3 antihypertensive drugs. Although the mechanisms responsible for the blood pressure–lowering effects of spironolactone are still unclear, the simple fact that a 1-mmol/L increase in serum K was associated with greater systolic blood pressure reduction implies that a reassessment of the role of aldosterone and K ions in essential hypertension is warranted. In this context, 3 major points deserve consideration.

First, the equivocal assumption that serum K accurately reflects body K or cell K function, despite the clinical and experimental evidence, is questionable. In the article by Chapman, serum K was measured at 6-month intervals, and despite treatment with drugs affecting cell K uptake and renal K handling, there was no assessment of intracellular K in these subjects. Second, considering that the renin-angiotensin-aldosterone axis plays a critical role in cell K/Na homeostasis, why was the role of cell K transport3 in which an increased plasma aldosterone could be a nonclassical feedback for such cell K depletion, as recorded in normotensive offspring with lower red blood cell K content. Furthermore, studies documenting that aldosterone plays a physiological role in body K homeostasis and cell K distribution4 might be linked to the protective effects of K in hypertension and organ-target disease as described by Tobian et al.5

These foregoing considerations, thus, may apply to broader aspects in the evaluation and management of essential hypertension, in which a different approach is necessary for the understanding of body K physiology and disorders that occur in hypertensive subjects or that may follow the administration of antihypertensive drugs.6 In brief, aldosterone and cell K physiology in essential hypertension remain fascinating topics as they were decades ago, and this editorial and research article certainly confirmed it.

Disclosures

None.

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