REVIEW ARTICLE

Nutrient Non-equivalence: Does Restricting High-Potassium Plant Foods Help to Prevent Hyperkalemia in Hemodialysis Patients?

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Hemodialysis patients are often advised to limit their intake of high-potassium foods to help manage hyperkalemia. However, the benefits of this practice are entirely theoretical and not supported by rigorous randomized controlled trials. The hypothesis that potassium restriction is useful is based on the assumption that different sources of dietary potassium are therapeutically equivalent. In fact, animal and plant sources of potassium may differ in their potential to contribute to hyperkalemia. In this commentary, we summarize the historical research basis for limiting high-potassium foods. Ultimately, we conclude that this approach is not evidence-based and may actually present harm to patients. However, given the uncertainty arising from the paucity of conclusive data, we agree that until the appropriate intervention studies are conducted, practitioners should continue to advise restriction of high-potassium foods.

Introduction

HYPERKALEMIA IS A life-threatening complication of end-stage renal disease (ESRD),[1] and accounts for about one-quarter of emergent dialysis treatments.[2] Serum potassium concentration is a key determinant of the resting cell membrane potential of neurons and muscle fibers. Consequently, hyperkalemia is associated with a variety of neuromuscular complications including abdominal cramping, weakness, paresthesia, and most concerningly, cardiac arrhythmias that can result in cardiac arrest. Under normal conditions, the kidneys excrete most of the excess dietary potassium (~80%-90%) to help maintain potassium balance; however, this process becomes compromised as glomerular filtration declines.

To prevent and manage hyperkalemia, ESRD patients treated with intermittent hemodialysis (HD) are advised to follow a low-potassium diet (2,000-3,000 mg/day), which involves avoiding high-potassium, plant-based foods (>200 mg/portion), including nuts, seeds, beans, peas, lentils, and many commonly consumed fruits and vegetables (e.g., tomatoes, potatoes, bananas).[3] Although this approach seems prudent, numerous factors may modify the effect of dietary potassium on serum potassium concentrations (S_K).

In this commentary, we evaluate the recommendation that HD patients should avoid high-potassium foods, considering: (1) observational studies of dietary potassium intake in relation to S_K; (2) experimental studies on potassium kinetics in ESRD; and (3) nutritional characteristics of plant-based potassium. We will not attempt to review all the many variables, including dialysis modalities and prescription, and medications, which have profound effects on S_K values.

Dietary Potassium and its Relation to Serum Potassium

The assumption that dietary potassium intake is an important determinant of S_K in HD patients is fundamental to the recommendation to avoid high-potassium foods. However, although potassium salts have been shown to result in postprandial S_K excursions in patients with chronic kidney disease (CKD, discussed in the following section), dietary potassium intake appears to be weakly (if at all) associated with predialysis S_K in HD patients. In a secondary analysis of 224 HD patients in the Nutritional and Inflammatory Evaluation in Dialysis study, Noori et al.[4] found that reported dietary potassium explained only about 2% of the variance in quarterly mean predialysis S_K ($r = 0.14$, $P < .05$; Fig. 1). The regression line describing this relationship indicates that, as reported dietary potassium intake went from a low of 500 mg/day to a high of 4,500 mg/day (a 9-fold difference), S_K was only about 0.4 mEq/L higher (Fig. 1).

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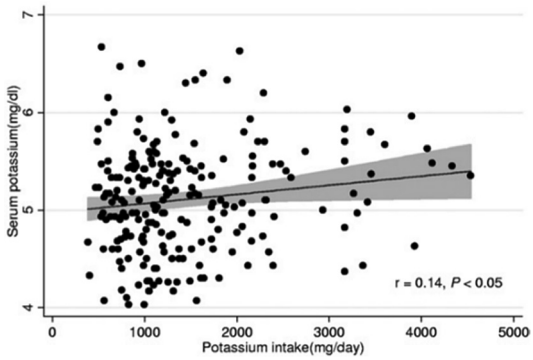

Figure 1. Associations of reported dietary potassium intake with serum potassium concentration in hemodialysis patients from the Nutritional and Inflammatory Evaluation in Dialysis study (n = 224). Regression line (solid line) and 95% confidence interval (shadedarea) are shown for the linear regression analysis. Reproduced with permission from Noori et al., Am J Kidney Dis 2010;56(2):338-347.

To confirm these findings, we investigated the associations of mean reported potassium intake (mg/day) and potassium density (mg/1,000 kcal) with predialysis S_K among 140 HD patients in the BalanceWise Study who completed 3, 24-hour dietary recalls (1 dialysis weekday, 1 nondialysis weekday, 1 nondialysis weekend day).[5] The scatterplots of these variables are shown in Fig. 2A and B. No significant correlations were found between S_K and either absolute reported potassium intake ($r = 0.06$, $P = .50$) or potassium density ($r = -0.003$, $P = .97$; unpublished data). These associations remained nonsignificant after adjusting for age, gender, race, and body mass ($P > .05$, data not shown).

Although high predialysis S_K is used clinically to assess hyperkalemia risk and is associated with worse survival in HD patients,[6] lack of a correlation between reported dietary potassium intake and predialysis S_K is not, in itself, evidence that high-potassium foods do not affect hyperkalemia risk in HD patients. Dietary potassium intake is measured with error, and S_K reflects a complex interaction of numerous intrinsic factors, including nervous/endocrine signals (e.g., epinephrine, aldosterone, insulin), intracellular/extracellular chemical concentrations (e.g., osmolality, H^+), circadian rhythms, and organ system functionality, which are influenced by environmental exposures such as diet and medications. It is possible that the association of dietary potassium intake with predialysis S_K is too weak to overcome these sources of measurement error, or that dietary potassium intake is correlated with S_K when measured in other metabolic states (e.g., postprandial, fasting). However, the lack of a discernable relationship between these variables in the BalanceWise study (Fig. 2A and B) contradicts the belief that the amount of potassium consumed influences predialysis S_K in HD patients.

Distribution and Excretion of Potassium in Kidney Disease

Kidney disease has been recognized as a condition of impaired potassium tolerance for 100 years. In 1915, Smillie published findings from a series of functional tests performed in 5 patients with chronic nephritis. Patients ingested either 5 or 10 g of potassium chloride (provides 2.6-g or 5.2-g potassium), and 1 of the patients given the 10-g dose later exhibited symptoms of weakness, collapse, abdominal distress, chest pain, vomiting, and cyanosis, which were attributed to potassium poisoning.[7] Despite some concerns, potassium salts continued to be regularly used as diuretics in patients with renal insufficiency to manage edema (standard dose of potassium nitrate provided approximately 4.8 g of potassium per day).[8]

In the 1940s, potassium balance studies by Winkler et al. and Keith and Osterberg demonstrated impaired renal clearance of potassium and higher S_K in patients with renal insufficiency after ingesting 2 to 5 g of potassium.[9,10] It was then concluded that caution should be exercised when using potassium-based diuretics in patients who were anuric[9] or uremic (blood urea ≥ 100 mg/dL).[10] In both studies, the increases in S_K were highly variable and less

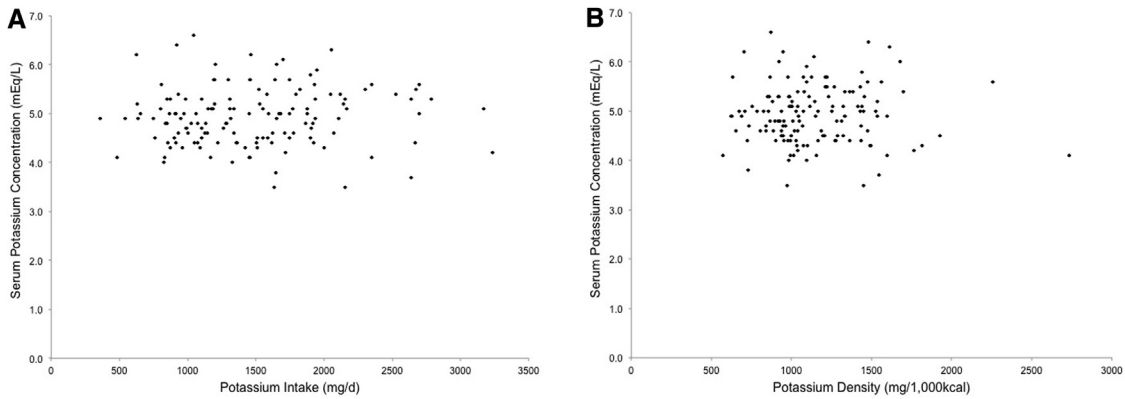

Figure 2. Associations of reported dietary potassium intake with predialysis serum potassium concentrations in hemodialysis patients from the BalanceWise Study (n = 140). (A) Potassium intake; $r = 0.06$, $P = .50$. (B) Potassium density; $r = -0.003$, $P = .97$.