PRACTICAL ASPECTS

Modified Nutritional Recommendations to Improve Dietary Patterns and Outcomes in Hemodialysis Patients

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The renal diet has traditionally been regarded as one of the most complex medical nutrition therapies to teach, understand, and implement. Specifically, patients are instructed to limit fruits, vegetables, nuts, legumes, dairy, and whole grains because of both phosphorus and potassium concerns. Furthermore, hemodialysis patients are often encouraged to decrease fluid intake to control interdialytic weight gain. These restrictions can result in frustration, lack of autonomy, and the perception that there is nothing left to eat. It is possible that the traditional renal diet may be liberalized, with a focus on whole foods low in sodium and phosphorus additives, to afford patients greater choices and ultimately improved outcomes. Therefore, the objective of this review is to concisely assess the evidence in support of a renal diet focused primarily on reducing the intake of sodium and inorganic phosphorus. Finally, the limited evidence for restrictions on dietary potassium intake is summarized.

Introduction

THE RENAL DIET has traditionally been one of the most complex medical nutrition therapies to teach, understand, and implement. End-stage renal disease (ESRD) medical nutrition therapy dictates that patients shift their nutrition goals from standard dietary recommendations to a pattern that manages levels of circulating waste products and minerals between dialysis treatments. This is especially true for patients undergoing chronic hemodialysis (HD) treatment due to the 48- to 72-hour span between treatments. Specifically, patients are instructed to limit fruits, vegetables, nuts, legumes, dairy, and whole grains because of both phosphorus and potassium (K^+) concerns.[1] These dietary restrictions are even more challenging due to elevated protein and energy needs. Furthermore, HD patients are often encouraged to decrease fluid intake to control interdialytic weight gain (IDWG).[2,3] These restrictions can result in frustration, lack of autonomy, and the perception that there is nothing left to eat.[1,4] In some cases, this can result in a disregard for the nutrition recommendations altogether and a reliance on processed, convenience, and restaurant foods.[5,6] These choices can further exacerbate complications and comorbidities associated with ESRD including cardiovascular (CV) disease, poor glycemic control, large IDWG, continued struggles with phosphorus and K^+ regulation, with a potentially confounding low intake of other vitamins, minerals, antioxidants, and dietary fiber.[7-9] Indeed, it appears that standard dietary prescription for HD patients may be doing more harm than good. There has been a slowly emerging discussion of "liberalizing" the diet prescription in an effort to decrease the total sodium and phosphorus additive load while inducing a corresponding increase in fiber, antioxidants, and phytochemicals, resulting in an overall improved dietary profile, particularly for CV health.

Considering the burden associated with ESRD and HD treatment, every effort must be made to support quality of life and patient dietary options. It is possible that the traditional ESRD medical nutrition therapy may be liberalized to afford patients greater choices and ultimately improved outcomes. The objective of this review is to assess the evidence in support of a renal diet focused on reducing the intake of sodium and inorganic phosphorus. In addition, the limited evidence for restricting dietary K^+ intake is summarized.

Sodium

Sodium is the main cation in the extracellular space and a key contributor to plasma osmolality.[10] In HD patients, increases in plasma osmolality may occur due to excessive dietary sodium intake or from treatment-related factors, such as the use of high-sodium dialysate solutions or

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hypertonic saline infusion used for the prevention of cramping and intradialytic hypotension. Each of these sodium sources may increase thirst and have been associated with higher IDWG, predialysis systolic blood pressure (BP) and chronic fluid overload,[11] which are in turn associated with acute and chronic CV complications and mortality.[12,13] To prevent the aforementioned complications, dietary sodium restriction is advised to HD patients.

Dietary Sodium Intake and Clinical Outcomes in HD Patients

Guidelines from the Kidney Disease Outcomes Quality Initiative recommend to limit dietary sodium.[3] However, the expert panel did not recommend a specific dietary sodium prescription and called for the assessment of an ideal dietary sodium intake for HD patients, although previous guidelines recommended a dietary sodium intake below 2000 mg/day.[2] Surprisingly, there are little data assessing the relationship between dietary sodium intake and clinical outcomes in HD patients. Furthermore, most studies in the United States have been observational and based on self-reported intake, which may underestimate dietary intake due to underreport and variability of sodium content of foods.[14,15] In a post-hoc analysis of the Hemodialysis (HEMO) Study, McCausland et al.[12] reported a mean sodium intake of 2240 ± 1050 mg/day, which was higher in younger, male, and nonblack participants with longer dialysis vintage. Moreover, there was a positive association between dietary sodium intake and mortality risk. Interestingly, they did not find an association between dietary sodium intake and BP. In a cohort of 122 HD patients, Clark-Cutaia et al.[16] reported a mean dietary sodium intake of 2,346 ± 904 mg/day and found that younger participants had higher dietary sodium intakes, elevated IDWG, and lower adherence to dietary sodium restriction.

Evidence of Potential Benefits of Dietary Sodium Restriction in HD Patients

It has long been recognized that hypertension and related CV comorbidities can be controlled in many HD patients through nonpharmacological means. Dietary sodium restriction in conjunction with persistent ultrafiltration can limit extracellular volume expansion and hypertension in HD patients, without the need of antihypertensive medications.[17] The contributions of a sodium-restricted diet as a component of a comprehensive volume control strategy have been highlighted by data from the HD populations in Tassin, France, and Izmir, Turkey. For over 40 years, HD clinics in Tassin have used a protocol to control hypertension that includes the use of sodium-restricted diets in combination with longer dialysis sessions (up to 8 hours).[18] Interestingly, they have reported controlled BP in 90% of their patients without the use of antihypertensive medications and lower mortality rates.[19] HD clinics in Izmir have used a similar protocol, although with shorter HD sessions (~4 hours), which may be more applicable to the United States.[20] In a retrospective cross-sectional study, Kayikcioglu et al. compared CV parameters in patients at 2 HD centers that managed BP using different protocols. The first center controlled BP without the use of antihypertensive medications, primarily using dietary sodium restriction (<5 g of salt, or 1,950 mg of sodium/day) in conjunction with intensive ultrafiltration, whereas the second clinic controlled BP using antihypertensive medications. Patients relying on sodium restriction and enhanced ultrafiltration had lower IDWG, lower left-ventricular mass index, better systolic and diastolic function, and higher serum albumin. Furthermore, only 7% of patients were using antihypertensive medications versus 42% in the clinic using standard BP control practices.[13] In the Unites States, the application of Izmir's protocol was explored in a pilot study by Williams et al.[21] Dietary sodium restriction, as part of an intensive volume control strategy that involved slowly reducing post-dialysis weight by 200-300 g per treatment, resulted in a decrease in systolic BP in patients whose predialysis systolic BPs were above 160 mm Hg. In addition, IDWG was reduced in those whose predialysis systolic BP was above 140 mm Hg. These data suggest that dietary sodium restriction is a valuable component of a comprehensive volume reduction strategy aimed at improving CV function.

To date, only 2 randomized controlled trials (RCTs) have assessed the lone effects of dietary sodium restriction in HD patients. First, Rodriguez-Telini et al.[22] examined the effect of sodium restriction (prescribed reduction of 5 g of salt/1,950 mg of sodium per day) on BP, IDWG, and inflammatory markers. Although there was no change in BP or IDWG after 16 weeks, inflammatory markers (C-reactive protein, tumor necrosis factor alpha, and interleukin 6) were reduced by 54%, 64%, and 56%, respectively in the sodium-restricted group but no change in the control group. However, the small sample size in this study (n = 21 in sodium-restricted group) suggests that additional studies are needed before definitive conclusions can be drawn. Finally, in the BalanceWise study, which aimed to assess the efficacy of a technology-supported behavioral intervention for dietary sodium restriction, Sevick et al. reported baseline dietary sodium intakes between 2,298 ± 957 and 2,555 ± 2,090 mg of sodium per day. After 8 weeks, patients in the intervention group reduced dietary sodium an average of 371 mg/day, but these changes were not sustained at 16 weeks. Furthermore, there were no changes in IDWG at 8 or 16 weeks.[23] Despite the aforementioned potential benefits of dietary sodium restriction, more adequately powered RCTs are needed to guide a specific recommendation in HD patients.

Potential Concerns With Dietary Sodium Restriction

In a recent review by Kalantar-Zadeh et al.,[1] it was stated that sodium restriction may be beneficial as long as optimal

nutritional status and food intake are not compromised. Indeed, malnutrition and the risk of protein-energy wasting are primary concerns with a sodium-restricted diet due to the wide use of sodium in foods. Data supporting this concern were reported by Dong et al.,[24] who found an association between low-dietary sodium intake and higher mortality. However, these results should be interpreted with caution considering this was in a cohort of peritoneal dialysis patients. Furthermore, the average body mass index was ~23 kg/m^2, and serum albumin concentrations were low across all sodium quartiles (~3.5 g/dL), which suggests possible malnutrition among all participants in this study. By contrast, in the RCT by Rodrigues-Telini noted previously,[22] patient's sodium intake was reduced by 2 g/day (baseline dietary sodium intake of 9.25 g/day) for 16 weeks without a reduction in total caloric or protein intake. This demonstrates that a sodium-restricted diet with uncompromised energy and protein intakes may be achieved with adequate nutritional counseling.

Finally, an additional concern regarding dietary sodium restriction is the loss of residual renal function. Residual renal function has been associated with positive outcomes in HD including survival benefit.[25] In addition, the preservation of residual renal function has an important role in nutrition, in part due to a better control of K^+ and phosphorus.[26] In a retrospective study by Ozkahya et al.,[27] loss of residual renal function was observed in nearly all patients after 3 years of implementation of Izmir's volume reduction protocol. However, little information was provided regarding the percentage of patients that were anuric/oliguric at baseline, their average residual urine output, or how long it was before their residual urine output was lost. Because few patients maintain significant urine output long after initiating HD, it is impossible to determine whether this loss of residual function was related to the dietary sodium restriction, volume reduction, or dialysis vintage. Furthermore, some antihypertensive medications (e.g., loop diuretics and angiotensin-converting enzyme inhibitors) have been proposed to preserve residual renal function in HD patients,[28] which were discontinued in the aforementioned study. Despite this, it should be noted that several factors impacting CV disease risk in HD patients, including BP, cardiothoracic index, and IDWG, improved in this study. This suggests that even if a loss of residual renal function is a manifestation of sodium restriction, the CV-related benefits may outweigh this potential concern. Studies that aim to assess the effect of dietary sodium restriction on residual renal function and clinical outcomes are needed.

Limitations and Barriers With Dietary Sodium Restriction

Dietary sodium is found ubiquitously in the Western diet and is particularly excessive in processed foods. It has been estimated that 75% of dietary sodium in the US diet comes from processed foods, 15% is added in food preparation as salt, and 10% is endogenous to foods.[29] Due to the high proportion of sodium coming from processed foods, the concept of "foods consumed away from home," such as fast-food and full-service restaurants, being associated with unhealthy eating patterns has been reported in the general population.[30] This concept, however, assumes that foods prepared and consumed at home are a healthier option. To examine this, we recently conducted a pilot study examining food's point of purchase in 60 HD patients (56 ± 14 years old, 62% male, 71% African American). On dialysis days, 75% of the dietary sodium came from grocery/convenience stores, whereas 15% and 5% of sodium came from fast-food restaurants and full-service restaurants, respectively. By contrast, on nondialysis days, 49% of dietary sodium came from grocery stores, 31% from full-service restaurants, and 14% from fast-food restaurants. Surprisingly, in this study, the ratio of sodium to kcal was similar from foods purchased at each location. This challenges the concept that healthier foods, at least in terms of sodium content, are consumed at home. This information may be helpful for renal dietitians for implementing nutritional education and counseling techniques focused on improving shopping skills and reading labels to limit major sources of dietary sodium.

Salt-taste perception may represent an additional barrier to comply with sodium-restricted diets. Salt-taste perception has been reported to be altered in ESRD patients.[31,32] Changes in taste acuity may occur with increased age and nutrient deficiencies, such as zinc.[33] In addition, it may take up to 3 months to adjust to a low-sodium diet in healthy populations,[34] with no difference reported in HD patients.[35] Therefore, palate adjustment to low-sodium diets may represent a challenge to HD patients due to repeated exposure to foods high in dietary sodium, and an altered salt-taste acuity.

Phosphate

Hyperphosphatemia and Its Clinical Management

Phosphate is the second most abundant mineral in the body, with 85% stored in bone and the other 15% in soft tissues, blood, and extracellular fluid.[36,37] Factors that influence phosphate metabolism have been reviewed elsewhere.[38] Hyperphosphatemia, or serum phosphorus ≥ 5.5 mg/dL, is one component of chronic kidney disease-mineral and bone disorder (CKD-MBD), and its prevalence in HD patients is as high as 50%.[39] Hyperphosphatemia has been associated with an increased risk of mortality,[40] specifically from CV causes, where it may increase the risk vascular calcification and left-ventricular hypertrophy.[41] CKD-MBD is also associated with decreased physical

function, increased risk of fractures, and reduced quality of life.[42] The current clinical management of hyperphosphatemia is focused on dialysis treatment, phosphate-binder therapy, and dietary phosphorus restriction.[43,44] The efficiency with which phosphate is removed by dialysis and control of phosphate with phosphate binders have been reviewed elsewhere.[45-47] Dietary phosphorus restriction represents a cornerstone of the renal diet and the treatment of CKD-MBD. The recommended dietary phosphorus intake is less than 800-1000 mg/day.[44] However, the efficacy of this recommendation has not been established. Furthermore, this restriction does not distinguish between types of dietary phosphorus, and in conjunction with dietary K^+ restriction may lead to a lower intake of other nutrients, such as dietary fiber.[8]

Types of Dietary Phosphate: Focus on Avoidance of Phosphate Additives

Dietary phosphorus can be divided into 2 kinds: organic and inorganic. Organic phosphate is found mostly as phosphoproteins and membrane phospholipids in animal sources (e.g., meat and dairy) and as phytate in vegetable sources (e.g., legumes, whole grains, and nuts). Animal sources have an absorption rate of 40%-80%, being higher when vitamin D is present.[48] Meanwhile, vegetable sources have an absorption rate of 20%-40%[48] because most of the phosphorus is found as part of phytate, which must be hydrolyzed by phytase to be released and absorbed in the small intestine. Phytase, however, is not expressed in the small intestine; therefore, absorption of phytate-containing foods is even more limited. Vegetable sources containing yeast may have increased phosphate availability due to yeast phytase activity.[49] Phosphate concentrations in both animal[50] and vegetable sources[51] also may be reduced with some cooking methods, such as boiling, slicing, and pressure-cooking.

By contrast, inorganic phosphate is not normally found in fresh/unprocessed foods. Instead, it is used as an additive in processed foods to increase palatability and shelf life, among other reasons.[48] Inorganic phosphate has an absorption rate of more than 90%, representing a major concern for HD patients.[52] Consumption of phosphate additives has been estimated to be as high as 1000 mg/day.[29] However, it is difficult to precisely determine intake because the food industry is not required to report the quantity used in their products. Instead, they only need to list the additives in the ingredient list.[52] This generally leads to an underestimation of dietary phosphorus intake and represents a challenge to nutrition professionals when assessing patients' intake. Recently, the Academy of Nutrition and Dietetics submitted a request to the Food and Drug Administration to add dietary phosphorus to the nutrition facts panel and make a distinction between naturally contained phosphate and phosphate additives.[53] Unless this is adopted and enforced, assessing patients' true phosphorus load and corresponding dietary prescriptions will remain a challenge.

Liberalized Organic Phosphate Restriction in HD Patients

Intensive nutritional counseling has been used by dietitians as a tool to reduce serum phosphate.[54] The focus of the counseling sessions has evolved, from a phosphate restriction without a differentiation between organic and inorganic sources of phosphate, to a more recent focus on phosphate additives. This shift occurred due to a concern of low-protein and energy intakes, which may lead to malnutrition and protein-energy wasting, and associated with lower physical function, reduced quality of life, and higher mortality.[55] Furthermore, foods with only organic phosphorus typically are more nutrient dense and have a higher nutritional value compared with processed foods containing phosphate additives, which tend to have a lower nutritional value, and are often paired with sodium and K^+ additives.[29] In a post-hoc analysis from the HEMO study, Lynch et al. found that phosphate restriction was not associated with improved survival and was associated with a higher risk of mortality. Conversely, a more liberal phosphorus prescription tended to improve survival.[56] Furthermore, Sullivan et al.[57] found that by educating patients on how to avoid foods with phosphate additives by reading food labels and providing renal-friendly options at local fast-food restaurants, serum phosphorus decreased by 0.6 mg/dL compared with controls with standard dietary recommendations. Recently, a meta-analysis by Karavetian et al.[58] suggested that nutritional counseling based on a structured behavioral change may be successful in HD patients. However, only half of the studies reviewed were RCTs, and most were short term with a follow-up ranging from 1 to 6 months. Although more RCTs are needed to assess the efficacy of dietary phosphate restriction on hyperphosphatemia, nutritional education focused on restricting processed foods and cooking methods to reduce the availability of phosphate may benefit HD patients.

Potassium

Potassium (K^+) is the main intracellular cation, which is important to maintain the cell's membrane potential, heart function, nerve-impulse transmission, and skeletal muscle contraction.[59,60] Regulation of serum K^+ is an important concern for HD patients due to its effect on acute CV complications and mortality.[60,61] In HD patients, the prevalence of hyperkalemia, defined as $K^+ \geq 5.5$ mmol/L, has been reported to be 4.5%-6.3%.[60] Serum K^+ levels are influenced by many factors, including HD-related variables (blood and dialysate flow rate, dialysate K^+ and buffer concentrations, dialysis length, and efficiency), residual renal function, acid-base balance, fecal excretion, glucose metabolism, and shifts from intracellular compartments.[62-64]

Current Dietary Guidelines for K^+ in HD Patients

HD patients are recommended to restrict certain foods, such as fruits, vegetables, nuts, legumes, and dairy products that are high in K^+[60], primarily due to concerns with hyperkalemia. The Kidney Disease Outcomes Quality Initiative guidelines do not have a specific recommendation for dietary K^+ for HD patients. However, the Joint Standards Task Force of the Academy of Nutrition and Dietetics and the National Kidney Foundation Council on Renal Nutrition recommend an intake of 2-4 g/day.[65] However, there is little evidence to support these recommendations. In a cohort of 224 HD patients with 5-year follow-up, Noori et al.[66] found that the association between dietary K^+ (estimated by food frequency questionnaire) and serum K^+ was weak ($r = 0.14$, $P < .05$). Furthermore, St-Jules et al.[67] found no association between dietary K^+ intake and serum K^+ in the BalanceWise Study cohort of 140 HD patients. Dietary K^+ intake was positively associated with energy, protein, and phosphorus intake, which have been associated with better outcomes in other large HD cohorts, such as the HEMO study.[55,68] This suggests that dietary K^+ restriction may be potentially deleterious if not implemented with caution in the face of increased risk of malnutrition and protein-energy wasting.[66] Indeed, low K^+ levels have also been associated with increased mortality in dialysis patients.[61,69] Further studies are needed to assess whether the current restrictions on dietary K^+ in HD patients are warranted.

Alternative Approaches to Restricting Dietary Potassium

When restricting foods that naturally contain K^+, other important nutrients are also restricted, such as dietary fiber. Indeed, dietary fiber intake in HD patients has been reported below the adequate intake of 25 g/day for women and 38 g/day for men.[8,70] Fortunately, dietary K^+ exposure can be limited using alternative cooking methods, such as boiling and leaching, particularly in vegetables and legumes.[71-73] Depending on the cut and prepreparation steps, the amount of K^+ loss can be reduced by as much as 75%.[71] Moreover, K^+ salts may be added to foods, such as meats, fish, dairy, and legumes, to enhance flavor or as a preservative.[74] Sherman et al.[75] compared the K^+ content in a variety of enhanced and nonenhanced meat and poultry products and found that K^+ levels averaged 8.7% higher in the enhanced products. The highest K^+ concentration found in a nonenhanced product was 387 mg K^+/100 g, whereas the average K^+ concentration in the top 5 enhanced products analyzed was 692 mg/100 g (max of 930 mg/100 g). Moreover, some salt substitutes are K^+-based, containing an average of 600 mg of K^+ per ¼ teaspoon, potentially contributing to excessive dietary K^+ load.[65] Similar to phosphorus, dietary K^+ is not required to be reported on the nutrition facts label of packaged foods, and a recent study found that K^+ levels were included on less than 10% of the foods studied.[76] The addition of dietary K+ to nutrition fact labels has been recommended. If these changes are adopted, new labels should include total K^+.[74]

Because of these concerns, we propose an alternative approach to broad restrictions on high K^+ foods for HD patients: (1) focus on limiting foods with added K^+ and (2) liberalize the restriction on foods that naturally contain K^+, while using food preparation and cooking methods such as boiling and leaching to limit K^+ intake, especially in vegetables and legumes. We recognize that some adjustments to this approach may be warranted for patients with overt hyperkalemia. However, because K^+ is an intracellular ion, other causes should also be suspected, such as inflammation, hemolysis, acidosis, and nutrition-related causes including poor appetite and low food intake that potentially promote muscle catabolism.[67,77] A liberalized dietary approach for controlling K^+ intake may have important health consequences for most HD patients. Prospective studies using this approach are needed in addition to studies that aim to explore the efficacy of current practices and the safety of this approach.

Conclusion and Future Directions

It is well accepted that the HD dietary recommendations, namely to reduce the intake of sodium, phosphorus, and K^+, while increasing protein and total energy, may be confusing, counterintuitive, and thus difficult to follow. As a result, patient compliance with the renal diet is comprehensibly low.[78] Based on the data presented herein, an alternative approach consisting of a simplified message focusing on dietary patterns to limit the intake of processed foods (from grocery/convenience stores, fast-food and full-service restaurants), enhancing patient's self-efficacy on how to shop and read nutrition fact labels and promoting a whole foods approach, may be warranted. We hypothesize that this approach will be associated with an overall decrease in IDWG and chronic volume overload, secondary to a decrease in overall dietary sodium, while supporting a dietary pattern consistent with lowered inorganic phosphorus and K^+ additives and increased fiber. This dietary pattern is likely to reduce risk factors for CV disease, the primary cause of death in HD patients.[79] A similar dietary strategy, along with persistent volume reduction, has consistently proven to significantly lower BP, nearly eliminate the need for antihypertensive medications, improve indices of cardiac structure and function, and reduce mortality rates in HD patients in Tassin[20] and Izmir.[27,80]

An added challenge to this approach is the need for patient and staff education. Intensive dietary counseling has been shown to improve nutritional markers such as serum albumin and phosphorus,[57] but renal dietitians lack the time to provide this level of patient interaction.[81,82]

Table 1. Current Dietary Recommendations for Hemodialysis Patients and Summary of the Proposal for a More Liberalized Diet Approach

Nutrient	Current Recommendations	Hypothesized Rationale	Evidence/Comments	Proposed Changes and Potential Benefits
Sodium	<2,000 mg/d	↓ Thirst stimulation: prevention of plasma expansion, chronic volume overload, and CV complications	Observational studies showing benefits of dietary Na^+ restriction along with intensive ultrafiltration on CV complications and reduced use of antihypertensive medications[19,20,27] ↓ Inflammatory biomarkers[22]	(1) ↓ Processed food intake (grocery store, fast-food restaurants, full-service restaurants, convenience stores), which are usually ↑ in Na^+ and P additives, as well as K^+ additives - If processed foods are consumed, educate on how to read food label to avoid ↑ Na+ and P additives (2) ↑ Intake of whole foods and meals prepared at home: - Ease restrictions on fruits, vegetables, whole grains, nuts, legumes, and dairy. Despite containing P and K^+, they also contain fiber and other heart-healthy nutrients - Educate on food preparation methods that can ↓ P and K^+, such as boiling and leaching - Introduce use of spices without Na^+ to ↑ palatability of foods - Use P-bioavailability as education tool on *good* food options (↑ organic P) vs *questionable* options (↑ inorganic P) - Educate on portion control of known foods with ↑ or moderate K^+; monitor blood K^+
Phosphorus	<800-1000 mg/d	Prevention of hyperphosphatemia and consequences: vascular calcification, CV mortality, and overall mortality	Overall P restriction has been associated with lower protein and energy intake[55,56] Recommendation does not differentiate between organic and inorganic P[47]	
Potassium	<2-4 g/d	Prevention of hyperkalemia and consequences: cardiac arrhythmia, cardiac arrest and mortality	Very weak association between dietary K^+ intake and serum K^+[67]	

CV, cardiovascular; K^+, potassium; Na^+, sodium; P, phosphorus.

However, other members of HD clinic staff (nurses, social workers, technicians, and physicians) represent an underutilized opportunity for consistently reinforcing basic nutrition messages. Enlisting the support from the dialysis team may represent the best approach to achieve these food intake patterns without compromising energy and protein intake. The World Health Organization's Ottawa Charter states the need to create a supportive environment to empower patients and promote health.[83] In addition, family and/or caregivers should be included because they are often responsible for handling food purchasing and preparation. Evidence that this approach can work in HD patients has been demonstrated by the data from Tassin[20] and Izmir,[27] as well as the studies by Krautzig et al.[84] and Sullivan et al.,[57] where the dietary message was based on simple recommendations, such as encourage home cooking, read food labels, avoid salt in food preparation, and obvious high-salt foods. Furthermore, these protocols have been structured to involve the entire dialysis clinic staff supporting the dietitians in helping counsel patients on how to lower their dietary sodium intake while maintaining an adequate nutritional status.[20,80]

In contrast, in the United States, the responsibility for nutritional counseling is usually guided by the renal dietitian with little support from the clinic staff.[85] Dialysis providers in the United States have taken preliminary steps to remedy this with development of "Tech Talks," which serve as talking points for dialysis technicians to use with patients. These "Tech Talks" consist of simple nutrition-focused messages aimed at decreasing phosphorus and sodium intake while increasing albumin; however, they are rarely used. Therefore, a simplified message focused on dietary patterns, led by renal dietitians, with support of the whole clinic staff, may help improve patient's overall diet in a manner that can help reduce chronic volume overload and CKD-MBD and enhance overall nutritional status and patient's quality of life.

Recommendations for Implementation

Based on the concerns described previously, we propose the following modifications to the dietary recommendations for HD patients (Table 1):

1) Dietary restrictions on K^+ and phosphorus from nonprocessed/whole foods should be largely

eliminated. Specifically, few restrictions should be placed on fresh fruit, vegetables, nuts, legumes, and dairy products, as the health benefits associated with these foods likely outweigh the unsubstantiated risks attributed to them. This strategy will result in a shift from highly processed food products with added sodium, K^+, and efficiently absorbed inorganic phosphorus additives, to low-sodium foods with primarily poorly absorbed organic sources of phosphorus that have the added benefits of additional antioxidants, vitamins, and dietary fiber. This recommendation should be taken within the context of clinical judgment and patient-specific considerations, particularly in terms of K^+ intake. However, it is critically important to remember that little evidence exists in support of dietary K^+ intake and hyperkalemia.[67] Patients should also be instructed in food preparation methods known to reduce phosphorus and K^+ quantities.

2) Instead, the primary focus of dietary restrictions should be on the reduction/elimination of processed, restaurant, and convenience foods that are almost universally high in sodium, inorganic phosphorus, and added K^+. Apart from these obvious benefits is the advantage of a far simplified message compared with current HD diet recommendations. Patients adhering to these recommendations may also realize cost savings through increased food consumption within the home.

Implementing this approach will take a concerted effort focused on several factors. First, patient education must shift from providing lists of foods to *not* eat to instruction on how to identify and shop for unprocessed whole foods and label reading with an emphasis on sodium, K^+, and phosphorus additives. One method that we have implemented in our counseling experience is to encourage patients to look for packaged products that have a sodium (mg) to kcal ratio of 1:1 or less. Products that contain fewer milligrams of sodium than kilocalories are generally lower sodium options, and this concept is easily understood by patients. In addition, we firmly believe that HD clinic staff and caregiver support is essential to overall patient success. The clinic staff have the unique opportunity of extended face time with patients, allowing for consistent reinforcement of general nutrition principles. Caregiver support also is essential to ensure consistent messages and eliminate a reversion to habits at home. This can be accomplished through continued staff and caregiver education alongside, and independent of, patient education. Development of staff- and caregiver-specific tools and resources may be necessary. Overall, improved patient health and quality of life may be within reach through a simplification and corresponding liberalization of the HD diet prescription. Further work must be conducted to establish protocols that are appropriate for implementation in the United States, taking into consideration HD treatment standards, the Western diet, and clinic staff education.

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