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**Moderate stepwise restriction of potassium intake to reduce risk of hyperkalemia in chronic kidney disease: A literature review**

AlSahow A. Diet for hyperkalemia in CKD

Ali AlSahow

**Ali AlSahow,** Department of Nephrology, Jahra Hospital, Jahra 00004, Kuwait

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**Corresponding author: Ali AlSahow, FASN, FRCP (C), MBChB, Consultant Nephrologist,** Department of Nephrology, Jahra Hospital, PO Box 2675, Jahra Central, 01028, Jahra 00004, Kuwait. alsahow@hotmail.com

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**Abstract**

A potassium-rich diet has several cardiovascular and renal health benefits; however, it is not recommended for patients with advanced chronic kidney disease or end-stage kidney disease because of the risk of life-threatening hyperkalemia. To assess the strength of evidence supporting potassium intake restriction in chronic kidney disease, the medical literature was searched looking for the current recommended approach and for evidence in support for such an approach. There is a lack of strong evidence supporting intense restriction of dietary potassium intake. There are several ways to reduce potassium intake without depriving the patient from fruits and vegetables, such as identifying hidden sources of potassium (processed food and preservatives) and soaking or boiling food to remove potassium. An individualized and gradual reduction of dietary potassium intake in people at risk of hyperkalemia is recommended. The current potassium dietary advice in chronic kidney disease needs to be reevaluated, individualized, and gradually introduced.

**Key Words:** Chronic kidney disease; Potassium intake; Plant-based diet; Hyperkalemia; Potassium removal

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**Core Tip:** A potassium-rich diet has several cardiovascular and renal health benefits; however, it is not recommended for patients with advanced chronic kidney disease or end-stage kidney disease because of the risk of life-threatening hyperkalemia. However, there is a lack of strong evidence supporting this restrictive approach. There are several ways to reduce potassium intake without depriving the patient of fruits and vegetables, such as identifying hidden sources of potassium (processed food and preservatives) and soaking or boiling food to remove potassium. An individualized and gradual reduction of dietary potassium intake in people at risk of hyperkalemia is recommended.

**INTRODUCTION**

A potassium-rich diet has several cardiovascular and renal health benefits[1-4]. However, it is not recommended in patients with advanced chronic kidney disease (CKD) or end-stage kidney disease (ESKD) because of the risk of hyperkalemia[5]. In fact, restriction of dietary potassium intake is the current standard of care. Due to the lack of strong evidence supporting this approach, there are calls to reevaluate and individualize the potassium dietary advice for patients with CKD[1,2]. In this review, the current recommendations, the argument against a restrictive approach, and an individualized, gradual reduction of dietary potassium intake were discussed.

***Potassium intake and health***

A potassium-rich diet can help decrease blood pressure and the risk of CKD and its progression, cardiovascular disease, stroke, and mortality[1-4]. Fruits and vegetables in most diets supply the majority of dietary potassium[5]. The alkaline content of such a potassium-rich, plant-based diet may correct CKD-associated metabolic acidosis. Fiber from potassium-rich fruits and vegetables improves the lipid profile, lowers body weight, increases stool volume and frequency, improves colonic epithelium integrity reducing toxin absorption, and improves the gut microbiome reducing systemic inflammation[1-4]. The Kidney Disease Outcomes Quality Initiative 2020 nutrition guideline suggested increasing fruit and vegetable intake to help reduce weight, blood pressure, and net acid production, all of which may help lower the rate of CKD progression[6]. A plant-based diet also helps decrease the risk of calcium oxalate stone formation by increasing urinary citrate levels and reducing the calcium excretion rate[3].

The minimum daily requirement for potassium in adults, estimated based on its unavoidable loss through sweat, stool, urine, and other sources, is about 1.6 g[7]. Table 1 Lists the recommended daily intake of potassium for the general population. It is important to know that the majority of people around the world consume less than what is recommended[8-10].

***Dietary potassium intake recommendations in adult patients with CKD***

Patients with an estimated glomerular filtration rate (eGFR) ≥ 45 mL/min/1.73 m2 are generally at low risk of hyperkalemia when consuming the recommended amount of potassium for the general population, even during treatment with renin angiotensin aldosterone system (RAAS) blockers. Hyperkalemia may develop in patients receiving a high dose of RAAS blockers and/or who have a high potassium intake (> 5 g/d) in the presence of severe heart failure and/or diabetes[8]. Table 2 outlines the current potassium intake recommendations for patients with advanced CKD (stages 3b–5) and ESKD to avoid hyperkalemia. It reveals a lack of consensus regarding recommendations for a low potassium diet. A low potassium diet is defined as a dietary intake of 2-3g/d of potassium[5]. In CKD populations, potassium intake is estimated to average around 2.4 g/d[11].

***Uncertainties and challenges of dietary potassium restriction in CKD***

Recommendations to limit potassium intake in CKD and ESKD are standard practice for two reasons: to prevent acute hyperkalemia (when postprandial potassium absorption exceeds cellular uptake and body excretion); and to prevent chronic hyperkalemia (when potassium bioaccumulation or total body potassium exceeds cellular uptake and potassium excretion)[12]. However, this approach also restricts intake of fruits, vegetables, dairy, grains, nuts, and legumes because they are major sources of potassium[5,12-14]. This in turn deprives patients of the health benefits discussed earlier, and it may need to be reevaluated for several reasons.

There is a lack of direct evidence regarding the benefit of restricting potassium intake in CKD from randomized controlled trials[15,16]. The results from observational studies regarding the association between higher urinary potassium excretion and mortality and kidney outcomes are conflicting[4,6,15]. Dietary potassium restriction is difficult because it requires lifestyle changes that may lead to loss of enjoyment in food and impact social activities. It may result in extra financial costs for special diets[15,16]. Restriction may lower the intake of other beneficial nutrients resulting in a poor diet, which may contribute to malnutrition in advanced CKD[17] and may lead to cardiovascular disease[18].

The National Kidney Foundation defines potassium-rich food as food with more than 200 mg of potassium per serving[19]. This arbitrary threshold can be confusing. For example, tangerines are a low-potassium food while oranges are a high-potassium food, despite having relatively similar nutrient contents and densities[20]. In addition, evidence suggests that gut-kidney kaliuretic signaling initiates urinary potassium excretion before the rise in plasma potassium and independent of plasma potassium and plasma aldosterone[4], which may explain why little to no association exists between serum potassium levels and potassium intake in CKD patients[16,21-24].

An unprocessed plant-based diet rich in potassium may not always lead to hyperkalemia for several reasons. Not all fruits and vegetables are rich in potassium[1]. The 24-h urine potassium recovery from animal-based diets is about 80% and from unprocessed plant-based diets is about 50%-60%. This lower bioavailability of unprocessed plant potassium might enable CKD patients to benefit from plant-based diets without precipitating hyperkalemia[1,3,25]. Plants are the only natural source of fiber, a nondigestible, nonabsorbable carbohydrate polymer that increases stool quantity and frequency, facilitating colonic elimination of potassium and protecting against hyperkalemia[3]. Plants provide a natural alkali, which may facilitate the transfer of potassium to the intracellular compartment, especially in metabolic acidosis[1,3,5,16]. Complex carbohydrates found in fruits and vegetables increase the insulin-mediated cellular uptake of potassium, helping to lower the risk of hyperkalemia[1,3,16]. The mineral content of vegetables varies with vegetable size, weather conditions, and cultivation techniques[26]. Many dietary recommendations are based on the mineral content of raw whole foods and not chopped, cooked foods[23,27]. Restricting sodium intake and encouraging a potassium-rich, plant-based diet may help control hypertension, reducing the need for hyperkalemia-inducing antihypertensive medications, such as β-blockers. It may also allow the reduction of the dosage of RAAS blockers[2], although this approach is controversial due to the negative effects on kidney and patient survival[17,15].

Potassium restriction should also be reevaluated because guidelines do not make specific recommendations for potassium in processed food or food additives and do not discuss potassium bioavailability. Potassium additives provide almost three times the amount of potassium found in additive-free counterparts, and potassium additives in processed foods have a high bioavailability contributing to hyperkalemia[24]. Because of the pressure on the food industry to decrease sodium in processed food, but not the salty taste, potassium use has increased in low-sodium processed foods[24,28,29]. Advice to restrict sodium alone for patients who need dietary sodium and potassium restriction may inadvertently increase potassium intake if a patient unknowingly switches to low-sodium products with potassium additives[30]. Moreover, advising CKD patients to restrict potassium-rich food without proper counseling may lead them to choose sodium- and potassium-rich processed foods over healthier alternatives[30]. Potassium-based food additives are found in precooked or shelf-stable foods, powdered dressings, sauces, preserved meats, stuffed pasta, jellies, concentrated fruit juices, processed cheeses, and margarine[5]. A list of potassium-based food additives is available elsewhere[31].

There are pitfalls associated with the tools used to estimate dietary intake of potassium. Plasma potassium levels are usually measured before hemodialysis and in a fasting state for non-hemodialysis (not a postprandial state). This timing is better suited to evaluate chronic hyperkalemia risk but not to detect acute postprandial hyperkalemia[30]. The standard assessment method is a 24-h urine collection, which assumes that 24-h urinary potassium excretion always reflects 70% of ingested potassium. However, collection errors may lead to incorrect assumptions and advice[28]. In addition, a single collection cannot accurately estimate potassium intake due to day-to-day variability in intake, gastrointestinal excretion, which is higher at low eGFR, and cell distribution. Spot urine should not be used to estimate intake at an individual level[11,15].

Non-urinary-based dietary assessment tools (food frequency questionnaires, diet records, and 24-h diet recalls) are not affected by changes in potassium homeostasis in CKD but rely on food databases. Thus, database inaccuracies (*e.g.*, differences in nutrients between processed, restaurant-cooked, and home-cooked foods) can lead to measurement errors[11,15].

Food frequency questionnaires record the consumption frequency of a predefined list of foods and beverages over weeks or months. It is the most cost-effective and time-saving method with a low burden, but errors occur due to recall bias (*i.e.,* respondents not correctly memorizing intake) and when questionnaires are not relevant to local dietary habits or seasonal changes. Furthermore, they may not account for potassium additives and substitutes and cooking methods[11,15].

Diet recall documents all foods and beverages consumed in the previous 1-7 d with moderate precision, cost, time, and burden. However, the method is prone to recall bias and underreporting, especially in overweight individuals. Precision is acceptable because quantities of consumed foods are defined, including potassium additives and cooking methods. However, more detailed intake information increases data collection complexity[11,15].

Diet records are prospective documentation of intake with minimal recall bias, and it is the most accurate if foods are documented and weighed correctly. However, it is costly and time consuming. Respondents may simplify or underreport complicated meals or even alter eating behaviors choosing healthier foods (selective reporting). Keeping records for more than 4 d may lead to inaccurate results due to respondent fatigue. It is also less suitable for CKD patients, in whom limited health literacy and cognitive impairment are common[11,15].

***A gradual, individualized approach***

A stepwise reduction in potassium intake is advised to ensure a balanced intake of fresh fruits and vegetables and fiber by patients with advanced CKD who are not prone to hyperkalemia[5,16]. Table 3 summarizes several strategies that should be implemented to minimize the risk of hyperkalemia before restricting potassium intake. Table 4 describes the steps to gradually restrict potassium intake and to minimize the impact of this approach on the patient. Table 5 describes cooking methods that can help reduce potassium levels in fruits and vegetables to increase the number of permissible food items.

**CONCLUSION**

Although potassium intake restriction is the standard of care in advanced CKD and ESKD, there is a need for the reevaluation of this approach in CKD patients who do not have hyperkalemia. There are health benefits of a plant-based, potassium-rich diet, and it is difficult to implement a low-potassium diet. There is scarce and inconsistent data attributed to dietary potassium in patients with CKD, and the risk of hyperkalemia may have been overstated. Under the supervision of a skilled dietician, the list of permissible food items may be gradually expanded by introducing unprocessed and properly prepared plant-based foods, especially those with a low potassium to fiber ratio, to improve the quality of food intake. Further research on moderate gradual liberalization of potassium intake in patients with CKD is certainly warranted.

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**Footnotes**

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**Table 1** **Recommended adequate intake of potassium for adults in the general population**

|  |  |
| --- | --- |
| **Institution** | **K intake g/d** |
| WHO and EFSA[32,33] | 3.5 |
| United States and Canada[34,35] |  |
| Male | 3.4 |
| Female | 2.6 |
| Japan[7] | 2.7–3.0 |

EFSA: European Food Safety Authority; K: Potassium; WHO: World Health Organization.

**Table 2** **Recommended potassium intake for adult patients with chronic kidney disease**

|  |  |
| --- | --- |
| **Source** | **Advice** |
| KDOQI |  |
| 2000[36] | CKD 1-5 ND: Unrestricted intake unless serum potassium level is elevated; HD: Intake up to 2.7-3.1 g/d; PD: Intake close to 3-4 g/d |
| 2004[18] | CKD 1-2: intake > 4 g/d; CKD 3-4: intake 2-4 g/d |
| 2020[6]; with the Academy of Nutrition and Dietetics | Statement 3.3.2: in adults with CKD 1–4, we suggest that prescribing increased fruit and vegetable intake may decrease body weight, blood pressure, and net acid production (2C); Statement 6.1.1: in adults with CKD 1–4, we suggest reducing net acid production through increased dietary intake of fruits and vegetables (2C) to reduce the rate of decline of residual kidney function; Statement 6.4: Adjust dietary potassium intake to maintain serum potassium within the normal range for adults with CKD 3–5D (opinion) |
| 2019[19]; NKF educational website | A potassium restricted diet is typically about 2 g/d. A physician or dietitian will advise patient on the specific level of restriction needed based on individual health |
| EBPG |  |
| 2007[37] | Recommendation of 4.2 g/d; in patients with pre-dialysis serum potassium greater than 6 mmol/L, a daily intake of potassium of 1950–2730 mg or 1 mmol/kg IBW is recommended |
| Spanish Society of Nephrology |  |
| 2008[38] | A low-potassium diet (1.5–2.0 g/d) is recommended for GFR < 20 mL/min or GFR < 50 mL/min if potassium-increasing drugs are taken |
| Academy of Nutrition and Dietetics |  |
| 2010[39] | Daily potassium intake of less than 2.4 g for CKD stages 3-5 who exhibit hyperkalemia |
| 2014[40] | CKD stages 3-5 not on dialysis: no restriction until hyperkalemia is present, then individualized; HD: 2-4 g/d or 40 mg/kg of BW/d; PD: individualized to achieve normal serum levels |
| Australian guidelines |  |
| 2005[41] | Reduced potassium diet should commence when serum potassium in pre-dialysis patients is > 5.5 mmol/L (Opinion). A reduced potassium diet limits the 24-h intake to about 3.1 mg |
| 2013[42] | Early CKD patients with persistent hyperkalemia restrict their dietary potassium intake with the assistance of an appropriately qualified dietitian (2D) |
| Italian Society of Nephrology |  |
| 2019[43] | Statement 1.2: serum potassium ≥ 5 mmol/L must be considered pathologic in CKD; Statement 1.3: restriction of potassium intake for non-dialysis CKD of mild-to-moderate degree is not recommended unless potassium levels are above 5 mmol/L in the absence of any other apparent cause. Then, it is recommended to limit food with high potassium content, especially if low in fiber, and pretreat (soaking and boiling) before cooking to remove potassium; Statement 2.3: restrict potassium intake to 2-3 g/d for advanced CKD and HD patients |
| Renal Association |  |
| 2020[17] | A low potassium diet should be instituted for patients with advanced CKD and ESKD with persistent hyperkalemia with serum potassium > 5.5 mmol/L; a low potassium diet is defined by a dietary intake of 2-3 g/d |
| Cupisti *et al*[5] | Limit intake to 2.5-3.0 g/d or 1 mmol/kg/IBW for mild hyperkalemia of 5.0-5.5 mmol/L; limit intake to 2.0-2.5 g/d for moderate hyperkalemia of 5.5-6.0 mmol/L; 1.5-2.0 g/d for severe hyperkalemia of 6.0-6.5 mmol/L |
| Yamada *et al*[7] | Limit intake to ≤ 2 g/d for eGFR ≥ 30 and ≤ 1.5 g/d for eGFR < 30; limit intake to ≤ 2 g/d for HD and 2.0-2.5 g/d for PD |
| Kalantar-Zadeh *et al*[44] | Recommended potassium intake is the same as for the general population: (1) eGFR ≥ 60 mL/minute/1.73 m2 without substantial proteinuria (< 0.3 g of protein/d) but high risk for CKD because of DM, HTN, PCKD, or a solitary kidney, *etc*; or (2) eGFR of 30-59 mL/minute/1.73 m2 without substantial proteinuria (unless frequent or severe hyperkalemia episodes are likely); Limit potassium intake to < 3 g/d: (1) eGFR < 30 mL/minute/1.73 m2 if hyperkalemia occurs frequently during high-fiber intake; (2) Any eGFR if there is substantial proteinuria and frequent hyperkalemia during high-fiber intake; (3) Patients with good residual kidney function transitioning to dialysis if hyperkalemia occurs frequently during high-fiber intake; or (4) Dialysis patients or patients at any stage with existing or imminent protein-energy wasting defined according to the International Society of Renal Nutrition and Metabolism criteria |
| Clegg *et al*[13] | Limit potassium intake to 3 g/d |

BW: Body weight; CKD: Chronic kidney disease; DM: Diabetes mellitus; EBPG: European Best Practice Guidelines; eGFR: Estimated glomerular filtration rate; ESKD: End-stage kidney disease; GFR: Glomerular filtration rate; HD: Hemodialysis; HTN: Hypertension; IBW: Ideal body weight; KDOQI: Kidney Disease Outcomes Quality Initiative; ND: Not on dialysis; NKF: National Kidney Foundation; PCKD: Polycystic kidney disease; PD: Peritoneal dialysis.

**Table** **3 Strategies to reduce the risk of hyperkalemia in chronic kidney disease before employing potassium dietary restriction**

|  |
| --- |
| **Strategy** |
| Calculate the eGFR to assess the patient’s risk of hyperkalemia |
| Avoid hidden sources, especially potassium-based additives in processed food and salt substitutes in low-sodium processed food |
| Increase stool frequency to increase the proportion of potassium excreted by the gut |
| Identify and correct non-dietary factors that influence serum potassium levels to keep potassium within normal range: (1) Drugs that may elevate serum potassium that can and should be discontinued/avoided (NSAIDs, COX-2 inhibitors, PPIs, potassium supplements, herbal remedies); (2) Inorganic metabolic acidosis associated with advanced CKD when serum bicarbonate level < 22 mmol/L; (3) Uncontrolled DM. Insulin deficit and/or hyperglycemic hyperosmolality lower acute potassium load movement into cells; and (4) Other causes that may directly or indirectly increase potassium level include volume depletion, adrenal insufficiency, catabolic state (major cell damage, hemolysis) and GI problems (diarrhea, constipation, bleeding) |
| Reduce the dose of medications known to elevate serum potassium level if in use or switch to an alternative if possible: RAASi (DRA, ACEI, ARB, MRA, ARNI, ASI), β-Blockers, potassium-sparing diuretics (Amiloride, Triamterene), calcineurin inhibitors (cyclosporine, tacrolimus), digoxin, heparin, trimethoprim/co-trimoxazole |
| Use diuretics. The effect depends on eGFR. Dialysis patients with reasonable residual kidney function may respond to loop diuretics. Diuretics work best when diuresis is desired, or an additional antihypertensive agent is considered |
| Implement sick-day rules by advising patients on the risk of AKI and hyperkalemia during acute illness and on measures to avoid them. However, it can be difficult and counterproductive |
| Improve potassium removal in hemodialysis (thrice weekly, 4-h sessions), which is mainly by diffusion: (1) Increase session duration and frequency, increase blood and dialysate flow, increase filter surface area, correct vascular access status recirculation, and use lower dialysate potassium; (2) Higher dialysate glucose concentration triggers insulin release, which enhances cellular uptake of potassium; (3) Higher bicarbonate bath concentration increases blood pH, which enhances cellular uptake of potassium; (4) Minimize pre-dialysis exposure to drugs that increase cellular uptake of potassium such as insulin and β2-agonist inhalers because they reduce pre-dialysis potassium, which reduces dialytic removal and exacerbates potassium rebound post-dialysis |
| Use one of the newer generation potassium binders (patiromer or sodium zirconium cyclosilicate). However, they primarily target chronic hyperkalemia not acute postprandial hyperkalemia and may increase pill burden |

Information compiled from references[1,3,5,13,15-17,45-51]. ACEI: Angiotensin converting enzyme inhibitor; AKI: Acute kidney injury ARB: Angiotensin receptor blocker; ARNI: Angiotensin receptor/neprilysin inhibitor; ASI: Aldosterone synthase inhibitor; CKD: Chronic kidney disease; COX-2: Cyclooxygenase 2; DM: Diabetes mellitus; DRA: Direct renin antagonist; eGFR: Estimated glomerular filtration rate; GI: Gastrointestinal; MRA: Mineralocorticoid receptor antagonist; NSAID: Nonsteroidal anti-inflammatory drugs; PPI: Proton pump inhibitor; RAASi: Renin angiotensin aldosterone system inhibitors.

**Table 4 Strategies to gradually introduce a mild potassium-restrictive dietary plan**

|  |  |
| --- | --- |
| **Strategy** | **Steps** |
| Ensure care is patient centered | (1) Involve dietitians, nurses, psychologists, pharmacists, and social workers if resources allow to ensure patient understanding. Clarify roles and responsibilities of each regarding dietary education to reduce conflicting information and deliver nutrition training to non-dietetic staff to promote consistency of message; (2) Individualize the plan according to patient’s lifestyle, and personal, religious, and sociocultural background. Train staff, particularly dietitians, to ensure expertise on culturally important foods and dietary patterns; (3) Explain the plan’s benefits and limitations and give the patient ample time to accept, to change dietary habits, and to adhere. Plant-based diets can be eco-friendly and economically advantageous. These arguments may promote adherence; (4) Adapt to all levels of education. Health literacy improves patient access and use of health information. Many patients have low health literacy, which may hinder communication, comprehension, and use of digital technology, prolonging the time to convey the message; (5) Identify vulnerable patients (young, socially isolated) who may need more nutritional education and support; and (6) Provide early and continuous access to the renal dietitian for collaboration on plan design and implementation |
| Instruct patients to identify potassium content of foods to avoid potassium-rich food. Food with > 200 mg of potassium/serving is defined as a potassium-rich food by the National Kidney Foundation | (1) Check serving size/weight. A large low-potassium food serving may have more potassium than a small high-potassium food serving; (2) Spread potassium-rich food items throughout the day to avoid acute postprandial hyperkalemia; (3) Increase the intake of low-potassium to fiber ratio fruits (apple, apricot, berries) and vegetables (green beans, peas, asparagus, lettuce, onions) and reduce the intake of high-potassium to fiber ratio food (processed juice and sauces); (4) Avoid food items that are very rich in potassium, such as edamame, molasses, and white and black beans; (5) Switch to soy-, rice-, and almond-based milk and yogurt because they may have less potassium and phosphorus than dairy; and (6) Avoid 93% lean ground beef since it has substantially more protein, phosphorus, and potassium than 70% lean ground beef |
| Describe food preparation methods and cooking procedures that may help reduce the potassium content of food | See Table 5 |
| Apply dietary plan to real life | (1) Invite and engage both patients and family members responsible for buying and preparing food (spouse, relative) in the appointments with the dietitian to translate dietary recommendations into accessible plans; (2) Adopt a stepwise approach allowing patients to adapt to dietary recommendations gradually. Patients do not eat calories, protein, or carbohydrates; they eat food, therefore, translate information about nutrients into food using food models, pictures, and recipes to make dietary counseling real and achievable; (3) Simplify nutrition education/advice particularly for those with multiple dietary needs. Avoid weighing or counting servings if possible so it is more practical for most patients. Use the hand to estimate serving size; (4) Explore unrestricted food. Discussion should focus less on what is not allowed and more about what is. Counseling based on food habits highlights foods that should be avoided with alternatives for replacement. For example, animal-based food (cow’s milk) may be replaced by plant-based food (vegetable milk such as soy, almond, and coconut); (5) Nothing should be “forever.” Social life is shared around a table; more restaurants have plant-based options, but the main courses in family meetings are often animal-based. Patients will not easily give up food they love. Allowing occasional freedom can improve long-term adherence; (6) Suggest cooking classes for recipes desired by patients to stimulate emotional involvement. Explore seasonal fruits and vegetables; (7) Offer positive feedback for the patient/caregiver during visits to motivate the patient to continue with the plan. Show how it affects laboratory values, intradialytic weight over time, or even number of pills patient is taking; (8) Meal delivery service may benefit patients who live alone or have difficulty shopping and preparing meals; and (9) Utilize the internet and social media: (a) Smartphones and tablets help interactive sessions (sharing pictures of dishes, describing sizes and preparation methods); (b) Telehealth/virtual nutrition counseling supports patients who need frequent reinforcement that outpatient clinic timing does not allow for or for those who have difficulty attending in-person clinical settings. They are cost effective in communities where long distances represent a barrier to face-to-face nutritional education; (c) Dietitian-facilitated and -supervised online peer support programs can promote healthful dietary behaviors. Age and cultural appropriateness of group participants should be considered; and (d) Online resources and technology-based interventions using smart phones (E-learning) can be used as platform for teaching and workshops reinforcing nutrition education and self-management plans |

Information compiled from references[1,2,5,12,15,16,19,20,31,45-50,52].

**Table 5 Food preparation methods to help lower food contents of potassium and ease restriction intensity**

|  |  |
| --- | --- |
| **Strategy** | **Tips** |
| Wash and peel the fruit and vegetable skin to lower potassium content then chop into small pieces. Peeling reduces fiber content |  |
| Place in cold water so they do not darken, then rinse in warm water for a few seconds |  |
| Soak for at least 4 h in warm water, then rinse, change the water, and soak for another 12 h (longer for legumes) | (1) Use ten times the amount of water to the amount of fruits or vegetables; (2) Water may need to be changed every 4 h for potassium-rich items; (3) Legumes may need to be soaked for 36 h at a ratio of 100 g per 1.5 L water with frequent changing of water; (4) Soaking lowers potassium more efficiently in water-rich soft foods like tomatoes and apples compared to potatoes or chocolate; (5) Soaking fresh potatoes, canned potatoes, and frozen fries alone without cooking afterward is ineffective in removing potassium; (6) Soaking alone is also ineffective for potassium removal from bananas, but boiling improves its removal; and (7) Soaking after normal cooking may increase potassium removal |
| Rinse afterwards under warm water for a few seconds before eating or cooking |  |
| Cook vegetables with five times as much water as vegetables | (1) Vegetables can be boiled in conventional cookware, a pressure cookware/autoclave, or microwave oven; (2) Potassium-rich items may need to be boiled twice. The double cooking technique; (3) Potassium removal by cooking shredded potatoes exceeds that of cooking cubed potatoes; (4) Adequate soaking followed be cooking may allow legume consumption twice weekly. Soaking dried legumes alone is ineffective for potassium removal; cooking after soaking significantly reduces potassium content. Cook in water at a ratio of 100 g per 1.5 L water (3 L water for dried chickpeas due to the long cooking duration they need). Canned legumes, drained and rinsed, contain less potassium and phosphorus than dried legumes, and the final contents after soaking and normal cooking make them a better alternative to the laborious preparation method for dried legumes. However, precooked legumes often contain salt and/or salt substitutes; (5) Freezing of green beans/chard (home frozen and industrial frozen) alone does not reduce potassium content. However, it leads to greater reduction of potassium contents when followed by soaking than soaking fresh ones. Cooking without soaking reduces potassium content less than cooking of soaked frozen ones. Soaking plus cooking is superior to either soaking alone or cooking without soaking alone for fresh green beans / chard; (6) Cooking methods that avoid contact with water (dry heat cooking, steam cooking, dehydration cooking) remove less potassium; and (7) Aromatic herbs can improve food taste and palatability reduced by peeling and boiling |

Information compiled from references[6,19,20,52-60].



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