9 Preventing Progression of Chronic Kidney Disease: Diet and Lifestyle

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Before You Start: Facts You Need to Know

- Patients with chronic kidney disease are often recommended to undergo a comprehensive assessment of their diet and lifestyle as part of their overall management strategy.
- Diet and lifestyle modifications are considered to be the cornerstone for the prevention and management of diabetes and hypertension.
- Most patients believe that changes in their diet and lifestyle are among the most important interventions for the management of their kidney disease.
- Most nephrologists are not trained in diet and lifestyle management and are unfamiliar with techniques to institute sustained and effective changes and the potential for adverse outcomes.

9.1 Diet and Lifestyle in the Management of Chronic Kidney Disease

All the major forms of chronic kidney disease (CKD) contain elements of diet and lifestyle in their pathogenesis and progression. Many of these actions are indirect, determined by the effects of diet and lifestyle on the common pathogenic mediators of CKD, including hyperglycemia, hypertension, hyperfiltration, oxidative stress, hyperphosphatemia, systemic inflammation, and activation of the renin angiotensin aldosterone system (RAAS), as well as modulation of the microbiome and the immune system. In addition, exposure to environmental toxins may also play a direct role in damaging the kidneys and accelerating the chronic progression of kidney disease in some patients. Equally, it is now widely recognized that most patients with CKD can benefit from changes in their diet and lifestyle, and current CKD management protocols are based on a foundation of dietary and lifestyle modifications. For the most part, these interventions are directed towards reducing the risk of comorbidities and complications of CKD, including bone demineralization, hyperkalemia, salt and water overload, cardiovascular disease, vascular calcification, and anemia. However, there is now evidence that diet and lifestyle can also significantly influence the progression of CKD and the decline of kidney function towards slowing the march towards end-stage kidney disease (ESKD). In the first instance, all patients with early CKD should be recommended to follow standard dietary recommendations for the general population. Collectively this means that most individuals with early CKD will be asked to moderate their energy, fat, and carbohydrate intake and an

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increase in their intake of fruit and vegetables. However, some patients with CKD will require additional dietary changes or more aggressive dietary restrictions to support failing kidney function and prolong their time before kidney failure. This chapter will review the dietary and lifestyle management of non-dialysis patients with established CKD, including its implementation, potential benefits, safety, and challenges for adherence. The specific dietary management of patients on dialysis and those with kidney stone disease is beyond the scope of this chapter.

9.2 Should Patients with CKD Restrict Their Intake of Protein?

Protein restriction is often the first thing that comes to mind, when considering implementing a dietary change in their non-dialysis patients with CKD. Most people with moderate to severe kidney impairment will already have spontaneously reduced their protein intake (to around 1.0–1.2 g/kg/day), due to the action of CKD on central appetite control centers. However, even this lower amount may still be more dietary protein than is probably optimal. It remains widely recommended in global guidelines that daily protein intake should be further restricted to <0.8 g/kg (i.e., a low protein diet) in most patients with an eGFR<60 mL/min/1.73 m^2, with the exception of patients with heavy proteinuria (>1 g/day) in whom protein losses must be compensated to avoid protein malnutrition [1]. A dietary protein intake of intake <0.8 g/kg is roughly half the amount of protein contained in a standard Western diet. Although this has become known as a "low protein diet," in fact, the globally recommended daily intake (RDI) of protein for the general population also targets this level of dietary intake, meaning that, in reality the nutritional goal is achieve a healthy protein intake, rather than continue a potentially unhealthy protein intake associated with over-nutrition that has become new baseline in most societies.

Dietary protein has a range of actions on healthy kidney function. In particular, a high protein intake induces pre-glomerular (afferent) arteriolar vasodilatation and hyperfiltration, possibly by activating tubulo-glomerular feedback as a result of increased proximal tubular sodium reabsorption. By restricting protein intake, it is hoped to increase afferent arteriolar tone and protect the remnant glomeruli from unnecessary hemodynamic stresses. Other benefits of a low protein diet may include modification of intestinal microbiota and a reduction in phosphate levels. Fifty years ago, when there was little or no effective RAAS blockade available and other antihypertensive therapies were suboptimal, dietary protein restriction was perceived as the best way to safety target kidney hemodynamics and their role in progressive glomerular damage, particularly in disease states where hyperfiltration was a pathogenetic important (e.g., diabetes, focal segmental glomerulosclerosis).

The renoprotective effects of aggressive protein restriction are clearly observed in experiential models of kidney disease [2]. However, its benefits in real-world patients with CKD remain controversial. A recent meta-analysis of ten clinical trials concluded that dietary protein restriction is not beneficial in slowing progressive kidney disease or reducing mortality when compared to standard dietary protein intake [3]. However, most of these studies were small and short term. The best-known clinical trial to test the utility of protein restriction was the Modification of Diet in Renal Disease (MDRD) study, that followed 585 non-diabetic participants with an eGFR<55 mL/min/1.73 m^2 (average 39 mL/min/1.73 m^2). Participants were randomly assigned to a "normal diet" (targeting 1.3 g/kg/day but achieving 1.1 g/kg/day) or a low protein diet (0.58 g/kg/day but achieving 0.77 g/kg/day). Similar to the hemodynamic response with an SGLT2 inhibitor or RAAS inhibitor, there was an initial greater fall in eGFR in those receiving with a low protein diet, followed by a slower rate of decline in eGFR (2.8 vs. 3.9 mL/min/1.73 m^2; i.e., a slowing of 28%). Although ESKD was similar in both arms of the trial, a 6-year follow-up of participants also suggested that this slowing translated into lower rates of ESKD and mortality in those receiving a low protein diet [4]. Although

small, this delay could be advantageous in providing additional time for comprehensive preparation for ESKD management, and which is strongly associated with improved outcomes when commencing dialysis.

Hyperfiltration and increased intra-glomerular pressure is also an important mediator of progressive nephron loss in diabetes. In so far as reducing protein intake may also reduce intra-glomerular pressures, there may be particular benefits of reducing protein intake in people with diabetes. However, the utility of protein restriction in patients with diabetes and CKD also remains problematic [5]. Early studies in patients with type 1 diabetes and CKD have suggested a modest but significant effect of protein restriction on slowing of the rate of decline in kidney function [6], as well a reduction in all-cause mortality [7]. In contrast, studies in people with type 2 diabetes have not shown the same benefits. Moreover, whether these data are equally applicable to modern patients with CKD that are already optimally treated with RAASi and SGLT2 inhibitor is unclear, as the proposed mechanisms of action may be similar to those induced following initiation of a low protein diet.

It has also been argued that conventional protein restriction does not go far enough, and that very low protein diets (<0.3 g/kg/day) may be required to slow kidney function decline in patients with CKD. Consistent with this hypothesis, a recent meta-analysis of ten trials suggested that a very low protein diet (targeting 0.3–0.4 g/kg/day) likely reduces the number of participants reaching ESKD when compared to a low protein diet (targeting ~0.6 g/kg/day) or unrestricted protein intake [3]. However, the challenges of achieving and maintaining a very low protein diet are real. Keto supplements and essential amino acids may need to supplemented, to maintain adequate nutrition. Moreover, in the long-term follow-up of the MDRD study, mortality increased in participants randomized to a very low-protein diet (0.28 g/kg/day + supplements) when compared to a low-protein diet (0.58 g/kg/day) [8].

Overall, the long-term adherence to a low-protein diet can be difficult outside of an intensive trial setting, especially if fat content is also restricted (see below), meaning that such low-protein diets must therefore be high in carbohydrate (which has its own challenges especially in patients with diabetes). Alternatively, all dietary elements must be reduced to achieve these targets, which increases the risk of malnutrition, especially in catabolic patients with uremia. Ultimately, the intensive and restrictive nature of protein restriction means that, although recommended, it is seldom rigorously implemented outside of specialist centers.

9.3 Should Patients with CKD Become Vegetarian or Vegan?

There is a widely held belief that eating vegetable 'protein' may be better for patients with CKD than a regular intake animal 'protein'. Of course, vegetarianism or stricter veganism have a number of potential advantages, including dietary changes in the amount and composition of fat, fiber, minerals and vitamins which impact on health and well-being, and likely convey the benefits of vegetable protein. Recommended diets, such as the Mediterranean diet and the DASH diet, have a regular intake of vegetables as a key component, while minimizing intake on meat, butter, and cheese.

A vegetarian-based diet is safe for CKD patients and may be a practical way to achieve dietary protein restriction goals by avoiding dairy and meat (i.e., animal protein). In addition, some small studies have supported the hypothesis that a vegetarian diet may also slow the decline in kidney function in some individuals and therein delay the initiation of kidney replacement therapy in patients with advanced CKD. For example, one crossover study suggested that the addition of vegetable protein was not associated with eGFR decline, while animal protein intake was associated with progressive decline in kidney function [9]. Benefits on blood pressure, phosphate, and lipid control have also been reported. However, at the same time fruits and vegetables can be high in potassium, meaning every diet must be carefully individualized and

some patients at risk of hyperkalemia will need to be directed away from these foods.

One critical component of a healthy diet is the regular intake of dietary fiber. Increasing the intake of vegetables while total protein intake declines is one way to ensure adequate fiber intake of at least 30 g/day, which is the same as for the general population. Most people will get half of this amount from their diet. Observational studies in patients with CKD have suggested a positive association between fiber intake and survival in patients with CKD. Some small studies have also reported improvements in kidney function [10].

9.4 Should Patients with CKD Restrict Their Intake of Calcium and Phosphorus?

Disturbances of mineral metabolism are common-place in patients with CKD, including increased renal phosphorus retention and hyperphosphatemia, especially in advanced CKD as the GFR falls below 30 mL/min/1.73 m^2. That it seldom occurs before this, is due to the activation of compensatory pathways that promote phosphate loss, including secondary hyperparathyroidism and activation of fibroblast growth factor 23 (FGF23). Restriction of dietary phosphate in proportion to the reduction in eGFR in patients with CKD can prevent the development of excessive parathyroid hormone (PTH) levels. Phosphate restriction (to less than 0.8–1.0 g/day) is often recommended to patients with CKD when serum phosphate or PTH levels are found to be elevated (i.e., in individuals with hyperphosphatemia or hyperparathyroidism) [1]. Again, this target corresponds to the recommended dietary intake for phosphate for healthy adults, so should not be considered a 'low phosphate diet'. However, the amount of phosphate regularly taken each day by most Americans is almost twice the recommended dietary intake. The rationale for treating/preventing hyperphosphatemia or hyperparathyroidism related to its deleterious effects on vascular calcification/stiffness, calciphylaxis, and cardiovascular risk. Phosphate restriction is usually achieved by restriction of dairy products and animal protein intake (Box 9.1), which may already be being undertaken for their respective benefits. However, it is possible to restrict protein without fully restricting phosphorus, so careful selection of protein sources must also be undertaken. Processed foods may also contain higher amounts of processed phosphate with much higher bioavailability compared with organic phosphate from unprocessed sources.

Box 9.1 Foods Naturally High in Phosphate (Which Should Be Avoided or Eaten in Small Amounts in Patients with CKD When Serum Phosphate or PTH Levels Are Elevated)

- Drinks: beer, milk, cocoa, cola
- Dairy products: cheese, custard, yogurt, ice cream
- High-protein foods: meat, liver, shellfish, legumes (beans and peas), nuts and seeds, whole-grain products

To reduce the calcium-phosphate product, alongside dietary phosphate restriction, some kidney dieticians also recommend limiting total calcium intake to <1 g/day, consistent with healthy intake guidelines. Certainly, limiting intake to below the usual 2 g/day may reduce the risk a positive calcium balance and ectopic calcification. The major calcium source in most diets is dairy products, which are also restricted when attempting to reduce potassium intake, addressing both targets simultaneously.

9.5 Should Patients with CKD Restrict Their Intake of Potassium?

Hyperkalemia is a common finding in patients with CKD, especially in those with diabetes and those using beta blockers, RAAS blockers, mineralocorticoid receptor antagonists (MRA), alone

or in combination. Excessive levels of potassium may contribute to bradycardia, severe muscle weakness, paralysis or even sudden death in some patients. In patients with CKD, hyperkalemia is a common reason for hospitalization and (emergency) initiation of dialysis that may be associated with poor outcomes when compared to a timely staged introduction of kidney replacement therapy. The advent of effective oral potassium binders can substantially reduce the risk of hyperkalemia in some settings. However, these do not eliminate the need for dietary potassium restriction.

Most patients with advanced CKD (eGFR <30 mL/min/1.73 m^2) and those with CKD at risk of hyperkalemia (e.g., those on high potassium levels, on RAAS blockers or MRA) are often recommended to reduce their dietary intake of foods that are rich in potassium (Box 9.2) and aim to eat between 2 and 4 g of potassium per day as a means to reduce the risk of dangerous hyperkalemia. This is usually achieved by choosing lower-potassium fruit and vegetables and their juices (Box 9.3) and limiting the intake of milk, legumes, nuts, tomatoes, and stone fruit. Many products now provide potassium content as part of their nutritional information, allowing patients to choose the lower-potassium alternatives.

At the same time, diets naturally rich in potassium (e.g., the Mediterranean diet) may be associated with improved outcomes, including lower blood pressure, and slower decline in eGFR. For example, in the MDRD cohort higher potassium consumption was associated with improved survival. In addition, some studies suggest that patients with a potassium in mild to moderate hyperkalemia (5–5.5 mol/L) may have a lower risk of dying than those with low or even low-normal potassium levels (<4 mmol/L) [11], partly due to actions on cardiac arrhythmogenicity. Outside of the setting of individuals at risk for hyperkalemia, most patients with CKD should not restrict their potassium intake, although potassium levels should be carefully monitored, especially when starting new agents or during intercurrent illness when potassium levels can risk due to an acute fall in eGFR.

Box 9.2 Foods Naturally High in Potassium (Which Should Be Avoided or Eaten in Small Amounts in Patients with CKD at Risk of Hyperkalemia?)

- Grains
  - Whole-grain breads, wheat bran, granola, and granola bars
- Dairy products
  - Milk and milk products
- Drinks
  - Sports drinks, energy drinks, vegetable juices, soy milk
- Snack foods/sweets
  - Peanut butter, nuts or seeds, chocolate, dried fruit
- Fruits
  - Stone fruit (e.g., apricots, avocado, dates, prunes, mango, papaya, cherries), bananas, kiwifruit, coconut, melon, nectarines, oranges, pears, pomegranate
- Vegetables
  - Tomatoes and tomato products, raw brassica (e.g., broccoli, Brussels sprouts, cabbage greens), carrots, olives, legumes (e.g., pinto beans, kidney beans, black beans, baked beans, peas) potatoes, pumpkins, parsnips
- Seafood
  - Shellfish, lobster, whitefish, salmon
- Beef
  - Ground beef, sirloin steak (and most other beef products)

Box 9.3 Foods Naturally Low in Potassium (Which Should Be Preferred in Patients with CKD at Risk of Hyperkalemia?)

- Foods prepared with white flour (e.g., pasta, bread)
- White rice
- Fruits: apples, watermelon, berries (e.g., blackberries, blueberries, cranberries, raspberries, strawberries)
- Vegetables: cauliflower, asparagus, zucchini, spinach, corn, onions
- Meat: chicken, turkey, tuna, eggs
- Dairy products: Cheddar, Swiss or cottage cheese

9.6 Should Obese Patients with CKD Lose Weight?

The majority of adults are now overweight or obese. This is also the case in most patients with CKD. The accumulation of fat, and subsequently deposition of ectopic fat in the development of diabetes, hypertension, and atherosclerotic vascular disease, the major causes of CKD. But even outside these obvious settings, more and more of our patients with glomerular diseases and other kidney pathology are overweight or obese. This may be considered part of a global trend for all adults to progressively gain weight over their lifetime, amplified by the reduced physical activity associated with chronic illness. Put together, obesity is now an everyday companion for the nephrologist. But should we be doing something about it?

Certainly, obesity in patients with CKD is associated with the increased incidence and severity of CVD, hypertension, dyslipidemia, diabetes, and reduced survival. Obesity itself may be associated with focal and segmental glomerulosclerosis, possibly due to changes in intraglomerular hemodynamics induced by obesity. In observational studies, weight gain is independently associated with incident CKD, even after adjusting for blood pressure and incident diabetes [12]. In addition, excess body fat is associated with faster rate of decline in kidney function and increased incidence of end-stage kidney disease (ESKD) in patients with CKD [13].

In clinical trials, weight loss clearly results in reduction in blood pressure, especially in patients already taking antihypertensive drug treatment like many of those with CKD. Moreover, amongst overweight patients with chronic kidney disease, weight loss interventions may be associated with a decrease in albuminuria. For example, in a cohort of Dutch patients from the Prevention of Renal and Vascular Endstage Disease (PREVEND) study, weight loss was associated with a reduction in urinary albumin excretion [14]. Significant weight loss associated with bariatric surgery and its effects of kidney function [15] further exemplifies the potential of benefits of weight loss that are seldom realized by diet alone, but never achieved without it. Moreover, the broad effects of obesity on cardiovascular health, sleep, cancer, mood, wound healing, self-image and a myriad of other areas means that most obese patients with CKD should be encouraged to lose weight, chiefly through dieting.

Fundamentally, weight loss diets aim to provide less food energy (measured as calories or kilojoules) than is required for metabolism and daily energy expenditure (known as a negative energy balance). The daily energy requirement can be roughly calculated (Box 9.3). To lose weight, the energy intake must be less than that of the daily energy requirement. Most weight loss diets start at an energy deficit of about 500 kcal/day. For example, if you calculate your patient's energy requirement as 8000 kJ a day, to slowly lose weight, they can target 7500 kJ/day. This will generally achieve a weight loss rate of approximately 1 lb (~0.5 kg) per week.

Reducing the amount of energy obtained from the diet can be achieved in any number of different ways. There is no 'one size fits all' approach, which means a comprehensive diet and lifestyle assessment by a trained dietician is an important first step. Sometimes only minor changes are required to reduce the energy content of a diet. For example, the energy in a can of Coke is around 500 kJ. So to lose weight, subtrating all the additional calories contained in soft drink and other calorie rich foods by omitting them from your diet, may be enough for may patients with CKD to acheive a negative calorie balance and lose weight.

The most common way to reduce energy intake is to go on a diet. This means regulating some or all of food intake according to a formula, recipe book or strategy. Whether the composition of a diet affects how well it produces weight loss remains highly contentious. Rigorous head-to-head studies of different diets have failed to show any superiority of one over another. On average they all achieve about the same amount of weight loss of 2–4 kg. It may be that what they are eating is probably not as important as the fact that they are adhering to some sort of plan for what they eat. It is likely that the mere process of embracing any dietary restrictions, thinking about and coordinating the foods they eat, makes them tend to eat less (energy) and eat better.

Diets that promote weight loss can be broadly divided into four categories, which chiefly restrict one element (for the sake of simplicity and compliance):

Low- fat diets—(e.g., STEP, Pritikin and Ornish diets) reduce energy from fat, without reducing meals. Reducing the fat in the diet can also improve lipid levels (see below). However, reducing fat often means increasing the content of carbohydrate and/or protein in the diet which may have drawbacks in insulin-resistant patients with CKD.

Low- carbohydrate diets (e.g., Atkins diet)—are popular for the management of type 2 diabetes, because of their beneficial effects on glucose control as well as caloric intake. There are a range of other diets that share roughly the same principles with respect to carbohydrate but vary in regard to other nutrients (e.g., fat or protein). For example, the Atkins diet does not restrict the (animal) fat you eat, while the CSIRO Total Wellbeing Diet and the 'Zone diet' reduce both fat and carbohydrate in your diet, so the relative proportion of energy from protein goes up. While this can have the added effect of suppressing hunger and promoting your sense of fullness earlier in the meal, it may also have adverse effects in the kidney and is therefore not generally recommended to patients with CKD.

Low- energy/calorie diets (e.g., DASH diet and Weight Watchers)—specifically target the problem of too much energy in the diet, by focusing on reducing the intake of processed 'energy-dense' foods exchanging them for low-calorie substitutes without focusing on diet composition. This strategy is generally preferred in obese patients with CKD and can be readily achieved by calorie counting, meal substitutes or following recipe plans.

Low- GI diets—(e.g., New Glucose Revolution, South Beach diet) have also become popular as a means to both slow the delivery of carbohydrate for meals and induce weight loss. High-GI (>70) foods such as white bread, potatoes or corn flakes break down their sugars quickly during digestion requiring insulin to surge in response to the extra demand. Over and above the extra energy they contain, a diet rich in high-GI foods is strongly associated with weight gain. By contrast, low-GI (<55) foods deliver their sugar load more slowly, so the demands on the pancreas are not so steep and fat accumulation is reduced. It is thought that low-GI diets may assist weight control by improving satiety and hunger between meals as slow sugars continue to be absorbed well after a meal.

9.7 Should All Patients with CKD Be on a Low-Fat Diet?

There is strong evidence that the presence and severity of dyslipidemia is associated with the risk of progressive kidney function decline in both diabetic and non-diabetic kidney diseases. Whether dyslipidemia is simply a marker of kidney dysfunction or a mediator of progressive damage remains to be firmly established. Certainly, a kidney phenotype is not seen in familial hypercholesterolemia or familial mixed dyslipidemia that would suggest its primary role in kidney injury. However, treatment with statins may reduce urinary albumin excretion and has been shown to modestly slow the rate of decline of GFR [16]. In each case, these kidney benefits were not correlated with improvements in lipid levels leading to the argument that any kidney actions are pleiotropic effects of statins rather than the result of lipid lowering. Yet, because of the high cardiovascular risk and clear benefits of

lipid lowering on cardiovascular outcomes in patients with CKD (not on dialysis), most patients will be recommended to reduce their lipid levels. This usually takes the form of statin therapy in combination with reduction in dietary fat intake, whether or not patients are overweight.

There is some observational data to suggest dietary fat is associated with progressive kidney disease. For example, in one study the nutritional pattern of patients with diabetes progressing from normo-albuminuria from micro-albuminuria was characterized by greater intake of saturated fat and a reduced intake of polyunsaturated and monounsaturated fat [17]. These lipid differences are also characteristic of diets associated with hypertension, weight gain and insulin resistance, all of which may contribute to progressive kidney disease.

Limited intake of intake of saturated fat (to <10% of total energy) and total fat (to <30% of daily energy intake is recommended for all healthy adults, and is also recommended for all patients with CKD. The broader utility of this strategy is exemplified by the Mediterranean diet and the DASH diet) that are associated with a lower risk for CKD progression and all-cause mortality among people with CKD [18].

9.8 Should Patients with CKD Restrict Their Intake of Salt?

Urinary sodium retention is a major contributor to hypertension and volume overload in patients with chronic kidney disease. Consequently, limiting the dietary intake of sodium appears a logical and appealing intervention for the prevention and management of hypertension in patients with CKD. Most guidelines suggest patients with CKD should target an intake of <60 mmol/day, equivalent to about one-third of the salt consumed by the general public. However, this target remains controversial. The dietary intake of sodium represents only a small fraction of the filtered sodium load (<1%), so its effects on kidney load are minimal. Any reduction in sodium intake is also associated with activation of sodium retention pathways including the RAAS and sympathetic nervous system, which may be counterproductive in the setting of CKD. The anticipated reduction in blood pressure from sodium restriction (1–3 mmHg in a trial setting) is also much lower and more variable than that achieved by antihypertensive therapy, and if blood pressure control is desired, it may be more effectively achieved by medications. Finally, the long-term benefits of sodium restriction in patients with CKD remain unclear. One study in patients with type 1 diabetes and macroalbuminuria suggested that a low sodium intake was associated with an increased risk of progression to ESKD [19]. By contrast, short-term studies have suggested additive benefits on both blood pressure and albuminuria when sodium restriction is added to patients with CKD already on RAAS blockers [20]. This may be because the RAAS is the chief counter-regulatory response to sodium restriction, and blocking it prevents escape. Consequently, it is reasonable to consider that RAAS blockade should be given to any patients adhering to a low-salt diet and a low-salt diet be considered for any patient on RAAS blockade, because of this synergism. As the majority of patients with CKD struggle to control their blood pressure and prevent volume overload, in practice this means a low-sodium diet is appropriate for most patients with CKD.

The major sources of dietary sodium are processed foods and condiments, rather than salt that is added onto meals by patients. Switching to low-salt version of products and using fresh ingredients where possible are the simplest ways to reduce sodium intake for most patients with CKD.

9.9 Should Patients with CKD Be Undertaking Regular Physical Activity?

Inactive people have an increased risk of developing kidney disease compared with very active people. Most patients with CKD are sedentary, undertaking little physical activity on a regular basis [21]. Although physical activity can improve blood pressure, lipid, glucose, and weight control and alleviate their mood status, it

is seldom stressed as an intervention in patients with CKD. This is mostly because of reduced exercise tolerance and comorbidity, such as hypoglycemia, anemia, postural dizziness, foot disease, and cardiovascular disease. Indeed so many patients with CKD have established CVD or risk factors for it that vigorous activity is usually contraindicated. However, this does not mean that moderate activity is inappropriate or unhelpful. Indeed, even in patients with established CVD, a program of regular moderate physical activity is associated with improved clinical outcomes.

There is a robust association between kidney function decline in patients with established CKD and physical activity [18]. Only limited research has been undertaken on the effects of exercise in the management of patients with CKD. Some trials have reported improvement in albuminuria following initiation of exercise programs [22], implying kidney benefits, although this could reflect better hemodynamic control. However, taken together, physical activity and exercise interventions have not been associated with slower kidney function decline in patients with established CKD [23].

9.10 Should Patients with CKD Give Up Drinking Alcohol?

Many patients believe that excessive alcohol intake is a common cause of chronic kidney disease (because of its obvious polyuric effects). Indeed, many patients believe that moderating or giving up their drinking is the most important way to protect their kidney function. Certainly, a high intake of alcohol (>5 units per day in men) is associated with an increased risk of cardiovascular disease, hypertension, cancer, and other health problems including chronic kidney disease. Whether this association is confounded by the adverse lifestyle of heavy drinkers remains to be fully established. Overall, a J-shaped association between alcohol intake and adverse health outcomes (such that abstainers have an increased risk of some health problems compared to those who regularly drink 1–3 units every day) appears to exist in patients with CKD [18]. This means that abstinence need not be recommended to most patients with CKD. Where patients can maintain control of their drinking, a healthy habit should not be discouraged. However, binge drinking may be potentially more dangerous in patients with CKD [24] and abstinence may be appropriate in heavy drinkers with CKD.

9.11 All Smokers with CKD Should Be Encouraged to Stop Smoking

There is clear evidence that smoking is a risk factor for progressive kidney disease. Inhaled toxins and generated reactive oxygen species pass to the kidney as well as to other parts of the body where they are both directly injurious and amplify injurious processes including inflammation and fibrosis in the kidney. Smoking also results in neurohormonal surges that may be particularly injuries to stiff vascular architecture that characterizes patients with CKD. There is some data to suggest that smoking cessation reduces the rate of loss of kidney function amongst patients with progressive kidney disease [18]. At the same time, some studies have reported acute increases in urinary albumin excretion 6 months after quitting [25]. This may be similar to the increase in diabetes and weight gain also observed with smoking cessation, which abates and ultimately leads to reduction in the long term. The long-term effects of smoking cessation on kidney function remain to be established but appear to be positive [26]. By contrast, smoking cessation should be reiterated for cardio-protection and cancer risk as these remain the major causes of death in patients with CKD [1].

9.12 Does Diet and Lifestyle Really Matter in Patients with CKD?

More evidence is needed regarding the best approach to diet and lifestyle in non-dialysis patients with established CKD. This cannot be

simply extrapolated from patients without CKD, as the complex effects of comorbid illness, polypharmacy, and the uremic milieu itself each present their own challenges. Overall there is limited data that initiating comprehensive changes in diet and lifestyle is able to protect kidney function (Box 9.4). At the same time, dietary change exposes patients to significant culinary restrictions. Many of these patients can anticipate very poor clinical outcomes, so quality of life is also often an important consideration. A 'healthy' diet can be safely recommended in most patients with CKD as a baseline, consistent with general population recommendations, with additional restrictions only added on an as required basis, as appropriate in patients with or high risk of specific complications, such as hypertension, volumeoverload,hyperkalemia,hyperparathyroidism (Boxes 9.5 and 9.6).

Box 9.4 Estimating Energy Intake in Adults

Women:

[655.1 + (9.56 × weight in kg) + (1.85 × height in cm) − (4.68 × age in years)] × 4.2 × activity factor

Men:

[664.7 + (13.75 × weight in kg) + (5 × height in cm) − (6.76 × age in years)] × 4.2 × activity factor

The activity factor in each equation (which adjusts for how active you are) is:

- For those who do little or no exercise each day, multiply by 1.2
- For those who do light exercise on 1–3 days a week, multiply by 1.375
- For those who do moderate exercise on 3–5 days a week, multiply by 1.55
- For those who do hard exercise on 6–7 days a week, multiply by 1.725
- For those who do daily exercise, a physical job or hard training, multiply by 1.9

Box 9.5 What the KDIGO Guidelines Say You Should Do [1]

We recommend that individuals with CKD receive expert dietary advice and information in the context of an education program, tailored to severity of CKD and the need to intervene on salt, phosphate, potassium, and protein intake where indicated

Restriction of Dietary Salt Intake in Patients with CKD

We recommend lowering salt intake to <90 mmol (<2 g) per day of sodium (corresponding to 5 g of sodium chloride) in adults, unless contraindicated

Restriction of Dietary Protein Intake in Patients with CKD

We suggest lowering protein intake to 0.8 g/kg/day in adults with diabetes or without diabetes and GFR <30 mL/min/1.73 m^2 with appropriate education. We suggest avoiding high protein intake (41.3 g/kg/day) in adults with CKD at risk of progression

Lifestyle in Patients with CKD

We recommend that people with CKD be encouraged to undertake physical activity compatible with cardiovascular health and tolerance (aiming for at least 30 min 5 times per week), achieve a healthy weight (BMI 20–25, according to country-specific demographics) and stop smoking

Box 9.6 Relevant Guidelines

1. KDOQI Guideline 2020

Ikizler TA, Burrowes JD, Byham-Gray LD, et al.; KDOQI Nutrition in CKD Guideline Work Group. KDOQI clinical practice guideline for nutrition in CKD: 2020 update. Am J Kidney Dis. 2020;76(3)(suppl 1):S1–S107. http://www.kdigo.org/clinical_practice_guidelines/pdf/CKD/

2. National Institute for Diabetes, Digestive and Kidney Diseases
Nutrition for Advanced Chronic Kidney Disease in Adults. https://www.niddk.nih.gov/health-information/kidney-disease/chronic-kidney-disease-ckd/eating-nutrition/nutrition-advanced-chronic-kidney-disease-adults

3. National Institute for Health and Clinical Excellence (NICE) Guideline [NG203] 2021
Chronic kidney disease: assessment and management. https://www.nice.org.uk/guidance/ng203/chapter/Recommendations

4. SIN-ANDID-ANED: Italian Society of Nephrology-Association of Dieticians-Italian Association of Hemodialysis, Dialysis and Transplantation 2018
Nutritional treatment of advanced CKD: twenty consensus statements https://link.springer.com/article/10.1007/s40620-018-0497-z

5. Renal Association (UK)
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Before You Finish: Practice Pearls for the Clinician

- All patients with CKD should be encouraged to adopt a healthy diet, consistent with nutritional guidelines for all adults.
- There is limited evidence that additional dietary restrictions or lifestyle modifications significantly improves kidney outcomes in patients with chronic kidney disease.
- A more liberal approach to diet and lifestyle should be considered in patients with advanced CKD in keeping with their poor prognosis and comorbidity and the overall goal of palliation.
- Targeted interventions can be highly appropriate for some patients, such as those with bone-mineral disorder, poorly controlled hypertension or hyperkalemia.
- In severely obese patients, significant weight loss may improve cardiovascular health, mood, healing, sleep and a myriad of other outcomes and should be encouraged.
- Nephrologists should engage in their patients' diet plans to ensure that their safety is not compromised and its potential for success is reinforced.

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