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Review

Nutrition for the Prevention and Treatment of Chronic Kidney Disease in Diabetes

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ABSTRACT

The prevention and treatment of chronic kidney disease (CKD) in diabetes through diet and lifestyle have been a topic of much interest over the years. Consideration of the type and amount of carbohydrate, protein and fat is required for optimal blood glucose control, for clinical outcomes related to renal function and for consideration of risk reduction for cardiovascular disease. Controversy has existed regarding the clinical significance of a protein-controlled diet, not to mention the ideal recommended intake in view of the benefits and risks. Furthermore, the level of CKD with which to implement dietary changes should also be considered. This review seeks to provide guidance and clarity concerning the nutritional management of CKD in diabetes.

RÉSUMÉ

La prévention et le traitement de la néphropathie chronique (NC) à l'aide du régime alimentaire et du mode de vie lors d'un diabète ont suscité beaucoup d'intérêt au fil des années. La prise en considération du type et de la quantité de glucides, de protéines et de matière grasse est nécessaire à la régulation optimale de la glycémie, aux résultats cliniques liés au fonctionnement rénal ainsi qu'à la réduction du risque de maladie cardiovasculaire. Une controverse existait concernant l'importance clinique d'un régime pauvre en protéines, sans parler de l'apport idéal recommandé eu égard aux avantages et aux risques. De plus, le degré de NC pour la mise en pratique de changements au régime alimentaire devrait également être considéré. Cette revue cherche à fournir des conseils et des précisions sur la prise en charge nutritionnelle de la NC lors de diabète.

Introduction

Diet and lifestyle are key components of an interprofessional approach to diabetes care. A treatment plan that encourages a combination of self-management education, lifestyle and pharmacotherapy has been shown to be successful (1). Good nutrition can help to control blood glucose, blood pressure and lipids, thereby slowing the onset and progression of complications that include chronic kidney disease (CKD). Diets that reduce inflammation and oxidative stress may also delay the progression of CKD in diabetes (2). Controversy surrounds recommendations for low-protein diets in CKD in terms of the exact amount, when to implement any limitations and whether the benefits outweigh the potential risks. In more advanced CKD, the restrictions required as part of a renal diet may conflict with the dietary strategies that promote optimal glycemic control. This review examines the lifestyle factors that influence risk for and progression of CKD in diabetes, notes the clinical recommendations for nutrition therapy and provides guidance about how to achieve targets while managing both diabetes and CKD (Table).

Nutritionally Modifiable Risk Factors for Chronic Kidney Disease

Poor glycemic control, hypertension and smoking are well-known risk factors for CKD in persons with diabetes (3). There are several novel risk factors that are also amenable to dietary interventions, including obesity, hyperphosphatemia and advanced glycation endproducts (AGEs).

Obesity is associated with increased incidence of CKD (4,5), and some experts estimate that up to 25% of cases of kidney disease could be prevented through weight loss alone (6). Epidemiologic studies report that obesity is independently predictive of CKD (3) and that the link may occur through the physical compression of

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Table
Nutritional considerations for people with chronic kidney disease in Diabetes

	CKD Stages 1-2	CKD Stages 3-4	ESRD Stage 5 (on dialysis)
Glycemic control	Promote optimal glycemic control	May require medication adjustment Assess for hypoglycemia	Assess for hypoglycemia
Calories and weight	23-35 kcal/kg BW/d or use HBE Encourage 5%-10% weight loss if appropriate	23-35 kcal/kg/day for stage 3. 30-35 kcals/kg/day for stage 4. Encourage preservation of lean body mass and nutritional status	Hemo: 30-35 kcal/kg/day PD: 30-35 kcal/kg/day Encourage preservation of lean body mass and nutritional status
CHO and sugar	45%-60% of energy <10% of calories from sugar (as part of a mixed meal)	45%-60% of energy <10% of calories from sugar (as part of a mixed meal)	45%-60% of energy <10% of calories from sugar (as part of a mixed meal)
Protein	Encourage 0.8 g/kg/day	0.8g/kg/day with avoidance of intakes >1.3g/kg/day	Hemo: 1.2 g/kg/day PD: 1.2-1.3 g/kg/day
Fat saturated fat	<30%-35% of energy <7% of energy	<30%-35% of energy <7% of energy	<30%-35% of energy <7% of energy
Fibre	14-25 g/1000 kcal	14-25 g/1000 kcal	14-25 g/1000 kcal
Glycemic index	Promote low GI grains, fruit, dairy products	Encourage stepwise modifications in GI Watch overall carbohydrate content as GI of diet changes	Encourage lower GI, renally appropriate choices
Fluid	Usually unrestricted Watch for excessive thirst with hyperglycemia	Usually unrestricted Watch for excessive thirst with hyperglycemia	Hemo: 1L + urine output PD: Individualized Watch for thirst caused by hyperglycemia Educate on nonfluid sources of carbohydrate for treatment of hypoglycemia
Sodium	<2000 mg/day	<2000 mg/day	<2400 mg/day
Potassium	Individualized restriction if hyperkalemia present	<2400 mg/d if hyperkalemia present	Hemo: <2400 mg/day PD: Individualized
Phosphorus	Individualized restriction if elevated serum levels	800-1000 mg/day	Hemo: 800-1200 mg/day PD: 800-1000 mg/day
Dietary patterns	DASH/Mediterranean/Portfolio	Progressing dietary restrictions limit patterns based on protein content, potassium or phosphorus. Modifications as required	Progressing dietary restrictions limit patterns based on protein content, potassium or phosphorus. Modifications as required

CKD, chronic kidney disease; *DASH*, Dietary Approaches to Stop Hypertension; *ESRD*, end stage renal disease; *GI*, glycemic index; *HBE*, Harris Benedict equation; *Hemo*, hemodialysis; *PD*, peritoneal dialysis.
Adapted from Whitham D, Sharma Parpia A. (37).

the kidneys, the activation of the renin-angiotensin aldosterone system (RAAS) or hyperfiltration (6). American guidelines for diabetes and CKD encourage attaining a normal body weight as a potential means of reducing risk (6).

Recent observational studies have shown an association between serum phosphorus levels and rate of progression of CKD (7,8). The hypothesis is that dietary phosphorus intake leads to calcification and vascular damage, although direct links have not been demonstrated in clinical trials (7).

AGEs have been associated with both vascular damage (2) and structural changes in the kidney, which may lead to the progression of renal disease and to a higher prevalence of cardiovascular disease in those with CKD (2). AGEs are a group of highly oxidant compounds also known as glycotoxins (9), which may be consumed in the diet or may be produced as a part of normal metabolism. AGEs accumulate in tissues, binding to and cross-linking proteins, thereby altering cell structure and function (9) as well as causing oxidative stress and inflammation (9). AGE formation is accelerated in states of high blood glucose and uremia (9).

Dietary AGEs (dAGEs) are naturally found in uncooked animal-derived foods. Cooking, particularly frying, broiling and grilling at high temperatures, can lead to very significant increases in dAGE levels (10). Simple alterations of cooking methods have been shown to decrease serum levels of AGEs, oxidative stress, inflammation and insulin resistance (9). In addition to the avoidance of very high temperatures, the addition of moisture through either water or marinades can reduce the formation of AGEs (9).

Prevention of oxidative stress may be important in many chronic diseases, including CKD. With conditions such as hyperglycemia and uremia, which lead to increases in oxidative stress, and a typical North American dietary pattern that promotes oxidative stress and yet has insufficient antioxidants, it could be speculated that increasing the intake of antioxidants would be of benefit to the progression of complications. Although not shown in humans, some animal-model studies have demonstrated a benefit of antioxidant supplementation on vascular disease (11).

In summary, management of hyperglycemia, hypertension and lipids and a more healthful dietary pattern that promotes an ideal body weight plus balanced intake may be of benefit to minimizing the progression of CKD in persons with diabetes.

Weight management

For those who are overweight, lifestyle therapy aimed at achieving and maintaining an ideal body weight may improve multiple risk factors for kidney disease, including blood glucose, blood pressure, lipids and proteinuria (12–16). One systematic review found that for each kg of weight loss, there was a reduction of 110 mg of proteinuria (17). Both body weight and waist circumference targets should be considered when setting goals. Waist circumference is strongly associated with visceral fat in patients with CKD (18) and carries a greater risk than does body mass index (1).

For overweight and obese people with stage 1 to 3 CKD and diabetes, moderate weight loss of 5% to 10% of body weight is recommended. Moderate reductions in energy intake (500 to 1000 kcal/day) combined with an increase in physical activity is the preferable weight-loss plan for this population (3,6). High-protein diets, such as Atkins, Protein Power, South Beach, Sugar Busters and Zone (defined as ≥20% of total daily calories from protein) are not recommended. Higher protein intakes have adverse effects on kidney hemodynamics and increase hyperfiltration, intraglomerular pressure and albuminuria. These deleterious effects are more pronounced in people with diabetes (6). Epidemiologic evidence suggests an association between high-protein diets and the development of proteinuria in people with diabetes and hypertension and in women, a loss in kidney function (6).