Nutrition Management of a Chronic Kidney Disease Patient with Obesity and Diabetes: A Case Study

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Nutrition care for patients with chronic kidney disease (CKD) involves continuous assessment of numerous nutritional and medical factors. Appropriate nutrition care can help patients maintain good health, and possibly prolong the time before renal replacement therapy (RRT) is initiated. In addition, bone and mineral metabolism control can be enhanced, dangerous alterations in serum potassium levels can be avoided, and fluid retention can be minimized (1). Nutrition intervention can also help patients avoid the protein-energy malnutrition (PEM) that often accompanies late stages of CKD (2,3).

If a patient also presents with diabetes and obesity, the complexity of nutrition care increases and priority-setting becomes vital. Similar to healthy individuals, CKD patients who are overweight or obese may improve blood pressure and cholesterol with weight loss of 5-10% (1). Weight loss may reduce both cardiovascular risk and risk of progression of CKD (4,5). However, it can be challenging to implement weight loss strategies along with the dietary modifications needed to help manage the metabolic alterations that occur in CKD. The following case illustrates an approach to the dietary management of a patient with diabetes and class III obesity.

Case Presentation

CJ was a 77 year old female with obesity (BMI >40 kg/m^2), type 2 diabetes and CKD stage 5 (not on dialysis). Medical history included partial parathyroidectomy for secondary hyperparathyroidism (SHPT), hypertension, cardiovascular disease and hypothyroidism.

CJ appeared well-nourished, despite her late stage of renal failure. Her weight was 251 pounds, and at her height of 5'3", her BMI was 44.5 kg/m^2. This is considered class III, or extreme obesity (6). Physical assessment indicated mild wasting of quadriceps and calf muscles, with mild edema present at the ankles. Decreased skin turgor was noted, indicating some degree of dehydration. In view of her decrease in leg muscle mass, it was not surprising that CJ had poor balance and poor endurance. She relied on a walker for ambulation. CJ was short of breath and easily fatigued. This was in part attributed to both iron and red blood cell deficiencies, as a consequence of the anemia of chronic kidney disease. As a result of her fatigue and obesity, CJ's activity level was very low. Despite the partial parathyroidectomy to correct her SHPT, her serum calcium was still above desirable levels for CKD. Her albumin of 3.9 g/dL did not point to a state of inflammation, yet the CO_2 of 16 mEq/L indicated presence of metabolic acidosis, which can lead to protein catabolism. Her potassium (K) was slightly elevated. Her HbA1C of 5.3%, along with her reports of fasting glucose of 90-100 mg/dL indicated excellent glycemic control. Urinary albumin excretion was not available.

Prior to her visit to the clinic, CJ was on a commercial weight loss program and lost 10 pounds. This program used a macronutrient distribution of 45% CHO, 35-40% protein and 20% fat (7). Because the counselors had no experience with adjusting her diet for kidney failure, there were no modifications made for the metabolic alterations related to her failing kidneys. Although CJ was pleased with her weight loss, she was more interested in following diet recommendations that would be appropriate for her kidney condition.

Her usual dietary habits included a simple breakfast at home (toast and fruit or a bowl of cereal), frequent restaurant lunches, and take-out or purchased pre-prepared foods for supper. Her usual diet was very high in sodium, and she often consumed baked goods and candy. She had received diabetic diet education several years earlier, but did not count carbohydrates or follow a meal pattern with exchanges. On the weight loss program, she had reduced her portions by sharing her restaurant lunch entrees with her daughter (who spent her days with CJ), and she had replaced evening sweets with fruit. She limited some foods high in phosphorus (P) upon her nephrologist's advice. CJ experienced early satiety, and noted a decreased appetite for meat.

CJ was not taking any vitamin or mineral supplements with the exception of Oscal with vitamin D. She did not take any nutritional or herbal supplements, and did not drink alcohol or smoke.

Treatment of CJ's anemia, caused by deficient production of erythropoietin and by iron-deficiency, was a primary reason for her referral to the CKD clinic. She was started on darbepoetin alpha (Aranesp) and iron sucrose (Venofer) to correct her iron deficiency, which, as noted in Table 1, was evidenced by a serum ferritin of 46 ng/mL.

Medications are listed in Table 2. CJ was on several medications commonly employed in patients with CKD to help control blood pressure, limit fluid retention and reduce proteinuria. These included furosemide (Lasix), valsartan (Diovan) and verapamil

Table 1
Laboratory Values

Year	Desired Values*	Date	Date	Date
Date		3/2/05	5/11/05	11/9/05
Weight	pounds	251	243	240
BMI	kg/m^2	44.5	43	42.5
Creatinine	mg/dL	3.9	3.5	3.0
BUN	mg/dL	97		
Estimated GFR	mL/min/1.73 m^2	12	14	16
CO_2	>22 mEq/L	**16**	**16**	24
Albumin	>/=4.0 g/dL	**3.9**	4.0	4.0
K	3.5-5.0 mEq/L	**5.1**	4.6	4.7
Calcium (corrected)	8.4-9.5 mg/dL	**10.5**	**9.8**	**9.9**
Phosphorus	3.5-5.5 mg/dL (stage 5)	5.2	4.0	4.8
Ca x P product	<55	54.6	39.2	47.5
PTH (bio-intact in 2005)	75-150 pg/mL for stage 5**	64	**192**	
Hgb	11-12 gm/dL	**9.2**	11.1	12.3
Transferrin Saturation (TSAT)	20-50%	21		21
Ferritin	100-800 ng/mL	**46**		390
HbA1C	<7%		5.3	
Blood Pressure	<130/80	132/60	128/64	
Cholesterol	mg/dL	159		
HDL Chol.	mg/dL	52		

*represent norms or desired values based on K/DOQI guidelines and/or institution guidelines. Patients with CKD do not achieve normal values for creatinine, BUN or GFR.
**Institution goals in 2005 were for bio-intact PTH: stage 4: 35-55 pg/mL; stage 5: 75-150 pg/mL

(Isoptin-SR). Her secondary hyperparathyroidism was managed with adjustments in the pro-active vitamin D doxercalciferol (Hectorol). Metabolic acidosis was managed with the use of sodium bicarbonate. This medication increases total sodium retention; thus dietary sodium restriction was warranted.

The nutrition strategy for this patient was to develop a meal plan that would help control blood pressure, reduce edema, and maintain appropriate serum K levels. Additionally, the plan's intention was also to normalize P and calcium balance so as to minimize exacerbation of the SHPT. CJ was instructed in a diet with 1800 calories, 48 gm protein, 2000 mg sodium, 800 mg P and 2730 mg K. Her calorie level was matched to that of the weight loss program on which she had been losing two pounds per week. Protein was restricted to 0.6 gm/kg desirable body weight, per K/DOQI guidelines for stage 5 CKD, but not on dialysis. This was to help minimize uremic waste products and potentially slow the progressive nature of her kidney failure (8). Sodium was restricted to help minimize fluid retention.

Although her serum P was not elevated, the dietary P restriction was continued as previously advised by her nephrologist. A level of 800 mg was prescribed, per K/DOQI guidelines (9). Calcium carbonate (OsCal), usually prescribed to bind P, was held due to the high serum calcium of 10.5 mg/dL. This presented another reason to limit dietary P intake, as no replacement phosphate-binder was prescribed. Finally, K was limited to approximately 70 mEq/day (2730 mg/day) to restore serum K levels to a normal range. Dietary measures to optimize lipid levels were also incorporated into the meal plan. To help maintain acceptable glycemic control, distribution of carbohydrate was discussed, along with food choices, portions and meal-timing. Continued weight loss was also encouraged, but not at the expense of impairing her nutritional status. Emphasis was placed on consuming adequate protein and the prescribed calories daily. A renal vitamin was prescribed, as patients on controlled-protein diets may need vitamin supplementation (9).

Selecting a weight to use for determining energy and protein needs is one of the most perplexing challenges in caring for the obese patient with CKD. As discussed further in this article, there are no validated methods for determining energy requirements in CKD patients. At this time, it is practical to make determinations based on the K/DOQI practice guidelines, also taking into consideration "the patient's values and preferences in the overall treatment plan (10)." Routine monitoring of a patient's progress and outcomes must serve as a guide for adjusting diet parameters (10). K/DOQI guidelines suggest using an "idealized body weight" for obese patients (11). When calculating needs for obese patients, adjusted body weight has historically been used, despite its lack of scientific foundation. Consistent with general practice at the CKD clinic at the time, both standard body weight (SBW) and adjusted body weight were considered in determining the body weight to use for calculations in obese patients. CJ's actual body wt was 114 kg. The SBW was

Table 2
Medications

Medication	Indication	Comments
Aranesp (darbepoetin alpha) 100 mcg Q 2 weeks	Anemia	
Dialyvite 800 1/day	Multivitamin	Contains water-soluble vitamins only
Diovan (valsartan), 80 mg bid	Angiotensin receptor blocker, relaxes blood vessels.	
Glucotrol (glipizide) AM: 10 mg PM 10 mg	Oral hypoglycemic agent	May lead to hypoglycemia if meals are skipped; can promote weight gain; needs close monitoring in pts with CKD
Hectorol (doxercalciferol) 0.5 mcg (begun in late 2005)	Active Vitamin D analog	Increases calcium absorption, reduces PTH stimulation Started in 2005; held in Oct 2007 as calcium levels were in high 9's
Lasix (furosemide) 80 mg and 160 mg alternating nights	Diuretic	Enhances K losses. Need to monitor K levels and adjust diet or provide K supplement if serum K too low.
Lipitor (atorvastatin) 20 mg	HMG-CoA reductase inhibitor, used to help lower cholesterol and TG	Grapefruit juice can increase levels in the blood.
Oscal with D (March 2005 only)	Calcium supplement and which can also perform as P-binder	Can raise calcium levels. Vitamin D in this form unlikely to be converted to active form in failing kidneys.
Sodium bicarbonate 650 mg tab 2 bid	Prevent and treat acidosis caused by kidney disease.	Contributes to total sodium intake
Synthroid (levothyroxine) 88 mcg	Hypothyroidism	Should be taken separately from calcium supplements, iron supplements, and antacids.
Venofer (iron sucrose) 200 mg q 2 weeks I.V.	Fe deficiency	Oral iron usually not adequate to meet needs in depleted CKD pt; thus IV Fe used.
Isoptin-SR (verapamil SR) 240 mg 1.5 tabs/day	Calcium channel blocker. Lowers blood pressure and reduces workload on heart, allowing it to work with less oxygen and blood flow.	

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70 kg, based on a medium frame size. Her adjusted body weight, using the K/DOQI method was 103 kg. These numbers were very far apart. Using too high a weight can result in overestimation of protein needs (11). A value between these numbers, 80 kg, was chosen, in part because this was the weight CJ reported being at when she was in her 30's.

Using this weight, her protein requirements were calculated at 48 grams/day and calorie needs at 2400 kcal/day (30 kcal/kg) (12). However, a pattern for 1800 calories was created, to help the patient continue with her weight loss efforts.

Implementation

To support her dietary change efforts, over the course of several visits, CJ was provided with *Dining Out with Confidence: A Guide*

for Kidney Patients (13), sample menus (developed with her input), and lists of high biological value protein, sodium, P and K food sources. Portion sizes were discussed, as well as strategies for continued weight loss.

During the time CJ was a patient at the clinic, her creatinine decreased from 3.9 to 3.0. Her anemia was corrected (see Table 1), and she reported that her energy and sense of well-being increased. She began to moderately increase her activity upon staff recommendations, but was limited due to her reliance on a walker. Dietary recalls indicated good adherence with restrictions in protein, phosphorus and potassium. Her sodium intake continued to be above recommended, as she continued her dependence on restaurant meals. Her calorie intake was variable and often greater than prescribed. Nevertheless, CJ had some success with continued weight loss, and reached 240 pounds by the end of 2005. Her nutritional status remained stable and albumin was maintained at 4.0 g/dL for several months.

Discussion

Obesity and CKD

Obesity may be the number one risk factor today for developing CKD, primarily because it is a risk factor for diabetes and hypertension (14). Together, diabetes and hypertension contribute to approximately 60% of the patients who develop CKD (15). Obesity may also have a direct impact on impairing renal function (14). Obesity increases the metabolic demands on the kidney, resulting in increased glomerular filtration rate (GFR) and renal plasma flow (RPF) and renal hypertrophy (14,16). As GFR and RPF rise, so does intracapillary pressure (14). Hyperfiltration and glomerulomegaly may result in glomerular damage and albuminuria in the severely obese (17).

Kambhan *et al* termed this condition obesity-related glomerulopathy (ORG). The course of ORG is somewhat indolent, and may not always progress to CKD stage 5. Although proteinuria is significant in over half the patients, hypoalbuminemia is present in only about 14% of patients. Kambhan *et al* reviewed 121 renal biopsies, including 71 patients with ORG and 50 patients with idiopathic focal segmental glomerulosclerosis. They found that the patients with ORG had a lower incidence of nephrotic syndrome (5.6% vs. 54%; $p<0.001$), higher serum albumin (3.9 vs. 2.9 g/dL; $p<0.001$) and less edema (35% vs. 68%; $p=0.003$). The mean BMI in ORG was 41.7. Kambham *et al* noted that the incidence of (ORG) increased from 0.2% in 1986-1990 to 2.0% in 1996-2000 (16). This parallels the increase in rates of obesity seen in the United States.

Lifestyle modifications that include weight loss and physical activity are recommended for the general public for improving blood pressure, blood glucose and lipid levels (4). Chagnac *et al* found improvements in glomerular hemodynamics in obese patients without overt renal disease following weight loss. Their findings suggest that weight loss may be helpful in reducing the metabolic demands on the kidney and potentially delaying progression of renal disease (17).

Diabetes and CKD

Diabetes is a primary cause of CKD with the first clue of diabetic nephropathy often being the presence of microalbuminuria (18). Glycemic control has long been emphasized as a goal in managing diabetes and preventing complications such as nephropathy. However, the level of protein in the diet may also play a significant role. High protein intakes promote glomerular hyperfiltration and increase pressure in the glomeruli. In contrast to the general population, higher protein intakes in diabetics may have more deleterious effects on kidney hemodynamics (11), and dietary protein limitation may help slow the loss of kidney function. "Competing needs for nutritional management of hyperglycemia, hypertension, and dyslipidemia can make determination of appropriate protein intake challenging," but even a modest decrease in protein intake to 0.89g/kg/d may be of benefit in reducing the progression to stage 5 CKD (11). A level of 0.6 to 0.8 gm protein/kg/day for CKD stages 3 and 4 with diabetes is recommended (11). Of this protein, 50-75% should be of high biological value (HBV), ideally from lean poultry, fish, and soy-based and vegetable-based proteins (11). "Protein of high biological value has an amino acid composition that is similar to human protein, is likely to be an animal protein, and tends to be utilized more efficiently by humans to conserve body proteins. The increased efficiency of utilization of high biological value protein is particularly likely to be observed in individuals with low protein intakes" (19).

With low protein levels, calories from protein may add up to only about 10% of total calories. To achieve adequate energy intake, non-protein calories should be derived from fat (~30% of calories) and carbohydrates (up to 60% of calories) (11). As patients reduce their protein intake, they may be increasing their baseline carbohydrate intake, leading to an increased need for insulin. Medications for glycemic control may need adjustment. Whenever possible, the patient should be encouraged to increase activity to improve insulin utilization and promote weight loss/prevent weight gain.

Carbohydrates that are considered low-glycemic should be emphasized to help decrease postprandial hyperglycemia and improve overall glycemic control (11) . Incorporating both soluble and insoluble fiber into the diet may help lower CVD risk (4); however, consideration needs to be given to the phosphorus and

potassium content of high fiber foods that are implemented in the diet.

Estimating Requirements for the Obese CKD Patient

The Metropolitan Life weight tables, which were developed to present weights associated with lowest mortality, were used in this population in the past, but no longer represent many Americans, as the data has tended to under represent those who do not purchase life insurance (20). The Hamwi optimum weight formula is familiar to most dietitians (20), but has no scientific data supporting its use. The NHANES II (National Health and Nutrition Evaluation Survey) SBW tables, recommended by K/DOQI, are not related to health outcomes. They also require measurement of frame size, which may not be feasible in some clinic settings, and they do not include values for adults over the age of 74 (20). The use of a formula for adjusted body weight in the obese also lacks research support (20).

A definitive approach is not currently available to estimate calorie requirements in the CKD population. At this time, the practitioner must rely on predictive equations. A survey of renal dietitians' practices conducted in 2005 illustrates the fact that there are no standardized methods employed across the country for assessing weight or determining calorie requirements in patients with CKD (21), though a large percentage of renal dietitians use the kcal/kg equation published by K/DOQI (21). This K/DOQI guideline is 35 kcal/kg/day for patients younger than age 60, and 30-35 kcal/kg/d for patients age 60 and older (12). Obese pts should be managed with lower calorie levels (1). In the end, the "practitioner must adopt a 'reasonable' approach," and let monitoring of patient's progress and outcomes provide a guide for adjusting medical nutrition therapy (10).

Conclusion

The number of patients with CKD and obesity is rising, in parallel with the increased incidence of obesity in the general public. Questions regarding the optimum treatment approaches in these patients will need to be addressed. For now, it appears that in early CKD (stages 1-3), weight loss should be aggressively pursued, as it may improve the hyperperfusion/hyperfiltration that accompanies obesity (17). However, the overweight and even obese patient with late stage CKD may have survival advantages once RRT has begun (22). It is unclear whether a higher BMI provides a survival benefit to the patient with a later stage of CKD, and not on RRT (23). Thus, weight loss in later stages of CKD must be addressed on an individual basis, taking into consideration the patient's personal goals, co-morbidities, their ability to make changes in activity level and food intake, and their future medical goals for RRT. Presently, no K/DOQI guidelines specifically address the treatment of obesity in CKD. Thus, recommendations from existing K/DOQI guidelines should be employed. Regardless of weight, every patient with CKD should receive close follow-up, re-education and adjustment of nutrition therapy as their clinical condition changes. ◆

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