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Nutrition in Renal Failure: Myths and Management



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Malnutrition occurs in up to 40% of patients with renal failure, and is associated with increased morbidity and mortality in this population. The cause of malnutrition in renal failure is multifactorial, but gastrointestinal symptoms frequently contribute to decreased food intake. Treatment of the GI manifestations of renal failure and co-existing conditions can improve nutrition status. Providing calories and protein that are appropriate for a patient's stage of kidney disease allows adequate nutrition and avoids unnecessary diet restrictions. This article reviews factors that frequently impair nutrition status in patients with renal failure, and provides suggestions for diet, supplements, and specialized nutrition support. Nutrition assessment, monitoring, and guidelines for vitamin and mineral supplements are discussed.

INTRODUCTION

A high prevalence of malnutrition exists in patients with renal failure. Several surveys have reported protein-calorie malnutrition in up to 40% of this patient population (1,2). Malnutrition in renal failure is multifactorial, but surveys consistently report inadequate oral intake as a major contributing factor (1,2). Indicators of nutrition status including reduced nutrient intake and muscle mass are each independently

associated with increased 12-month mortality (2). Gastrointestinal complaints are frequently seen in this patient population and likely contribute to decreased intake and malnutrition (3–5). Research suggests that addressing GI issues in patients with renal failure may improve nutritional status (5).

Although the traditional surrogate markers of malnutrition, such as decreased muscle mass or serum proteins have been associated with increased mortality, research is ongoing to determine if improving nutritional status will alter patient outcomes. Decreased muscle mass or serum proteins can also be attributed to acti-

(continued on page 42)

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(continued from page 40)

vation of the acute-phase response related to co-morbid conditions. In addition, it is possible that co-morbid conditions result in increased inflammatory cytokines and are the cause of both malnutrition and the increased mortality. However, there are several studies that demonstrate that the provision of increased nutrition to patients with malnutrition and renal failure may improve patient outcomes (1,6). This article will review some of the factors that affect nutrition status in patients with renal failure, discuss appropriate nutritional needs, and offer strategies for optimizing nutritional intake.

DECREASED NUTRIENT INTAKE

There are a number of factors that contribute to malnutrition in patients with renal failure (Table 1). Decreased intake of protein and calories is the most evident factor. Studies have demonstrated that even patients with a mild decline in glomerular filtration rate (GFR) (i.e. <50 mL/minute) have a decreased calorie and protein intake (1,2). Studies have also documented that dietary protein intake progressively declines with decreasing GFR (1,2).

Co-morbid conditions frequently contribute to decreased intake and malnutrition. Gastroparesis likely contributes to poor intake in those patients with renal failure who have diabetes mellitus. There is increasing evidence that *non-diabetic* patients with renal failure and poor intake should be evaluated for gastroparesis (4,5). Several studies have documented a high incidence of impaired gastric motility in maintenance dialysis patients (3–5). Those non-diabetic maintenance dialysis patients who had hypoalbuminemia and occult gastroparesis demonstrated improved nutrition status after treatment with erythromycin as a prokinetic agent (5).

Overzealous diet restrictions can also contribute to decreased intake. The provision of a “renal diet” that limits protein, salt, potassium, phosphorus and fluid may further limit intake in a patient with existing malnutrition and poor oral intake. Dietary intervention should not be instituted until nutritional status and eating habits have been investigated, and the patient demonstrates a clear need for dietary restriction. Furthermore, underlying causes for electrolyte abnormalities such as poor glucose control, use of potassium

Table 1
Factors contributing to malnutrition in renal failure

- Decreased Intake
 - Anorexia
 - Gastroparesis
 - Intraperitoneal instillation of dialysate in CAPD
 - Uremia
 - Increased Leptin
- Diet Restrictions
- Loss of nutrients in dialysate
- Concurrent illness and hospitalizations
- Increased inflammatory and catabolic cytokines
- Chronic blood loss
- Acidosis
- Accumulation of toxins such as aluminum
- Endocrine disorders
 - Insulin resistance
 - Hyperglucagonemia

containing salt-substitutes, or medications as a cause of hyperkalemia should be addressed before imposing diet restrictions.

Patients receiving maintenance dialysis have increased serum leptin and elevated serum acute phase mediators such as IL-6 and TNF (1,2). These mediators would be expected to exacerbate the anorexia and decreased oral intake in patients with renal failure. The presence of uremia is a more obvious factor that adds to the decreased appetite and nutrient intake.

INCREASED NUTRIENT LOSSES

Patients who receive maintenance dialysis experience a loss of nutrients as a direct result of the dialysis itself. Hemodialysis results in a loss of 6–12 grams of amino acids, 2–3 gms of peptides, and negligible amounts of protein per dialysis session (2). During peritoneal dialysis, patients lose only 2–4 grams of amino acids, but experience a total loss of 8–9 grams of protein per day (including 5–6 grams of albumin) (2). Patients on peritoneal dialysis can lose over 15 grams of protein each day during periods of peritonitis. This increased protein loss can continue for days after the peritonitis is treated (2).

Patients receiving maintenance dialysis also have protein losses due to frequent blood sampling for labs.

(continued on page 44)

(continued from page 42)

A patient with normal hemoglobin will lose approximately 16 grams of protein with each 100 mL of blood removed (1,2).

Malabsorption due to bacterial overgrowth is another route for nutrient loss in some patients. In a cohort of 22 patients with chronic renal failure, 36% had small bowel bacterial overgrowth (3). This is very likely an underappreciated route of fecal nutrient losses considering that over 25% of these patients did not have overt gastrointestinal symptoms.

INCREASED CATABOLISM

Patients with renal failure are frequently “anabolism challenged.” The increased acute-phase reactants observed with renal failure and dialysis inhibit hepatic production of albumin and increase catabolism of skeletal muscle tissue (1,2). Acidosis is an additional factor that precipitates catabolism in this population (7). Recent research documents the ability of acidosis to activate the ubiquitin-proteasome proteolytic system in muscle, one of the primary pathways of cell protein catabolism (8). Provision of bicarbonate to maintenance dialysis patients decreases the protein catabolic rate, and improves nutrition status (7,8). Acidosis also inhibits osteoblast and increases osteoclast activity, contributing to the osteodystrophy associated with renal failure (7).

NUTRITION NEEDS

Before any discussion of calorie and protein needs in patients with renal failure can proceed, one important point must be made. There are no large, prospective controlled trials that have randomized patients to receive graded levels of protein and calories to determine the intake that will lead to the best outcomes in nutritional status, morbidity, and mortality. Current recommendations have been based on surrogate markers of nutrition status, indirect calorimetry measurements, and studies of protein catabolic rate. Considering the revelations that medical outcomes research has yielded in the last 10 years, realize that the finest recommendations cannot reliably tell us how to feed this nutritionally vulnerable population in a way that will yield the best outcomes.

Several studies have investigated the calorie needs of maintenance hemodialysis patients under metabolic balance conditions. A review of these studies concluded that caloric expenditure of stable hemodialysis and CAPD patients were not significantly increased above normal (1). However, a review of surveys of food intake in the dialysis population suggests that calorie intake is frequently inadequate, and is compromised to a greater degree than protein intake (1,2). The National Kidney Foundation’s guidelines for stable dialysis patients are 30–35 calories/Kg (9) and can be found on Table 2. Patients who receive peritoneal dialysis with a dextrose containing dialysate solution can absorb up to 70% of the dextrose from the solution that is instilled (10). Many patients absorb 200–300 dextrose calories per day from chronic ambulatory peritoneal dialysis (CAPD), and this must be taken into account when estimating calorie intake and needs or when attempting to control hyperglycemia.

CALCULATING CALORIE NEEDS

Generally, caloric calculations should be done with actual edema-free body weight, determined post-dialysis for hemodialysis, and “post-drain” for peritoneal dialysis. The National Kidney Foundation recommends that when patients are <95% or >115% of the median standard weight (as determined from the NHANES II data), that an adjusted body weight be used (9). If actual body weight is used to calculate calorie requirements in patients that are obese, energy requirements may be overestimated. Adjusted body weight is calculated as follows:

$$\text{Adjusted weight} = \text{ideal weight} + [(\text{actual edema-free weight} - \text{ideal weight}) \times 0.25]$$

Estimation of calorie needs remains an issue of some controversy among nutrition experts. From a practical standpoint, any estimation of calorie requirements is only a starting point to avoid gross overfeeding or underfeeding. Monitoring a patients clinical status and trends in their edema-free weigh, with timely adjustment in the nutritional plan as needed, is far more important than absolute precision of the initial assessment of calorie needs.

(continued on page 46)

(continued from page 44)

Table 2
Selected nutritional parameters for varying levels of kidney failure^a

Nutritional parameter	Normal kidney function	Stages 1–4 Chronic kidney disease	Stage 5 Hemodialysis	Stage 5 Peritoneal dialysis	Transplant
Calories (kcal/kg/d)	30–37	35 < 60 yrs 30–35 ≥ 60 yrs	35 < 60 yrs 30–35 ≥ 60 yrs	35 < 60 yrs 30–35 ≥ 60 yrs include calories from dialysate	30–35 initial 25–30 for maintenance
Protein (gm/kg/d)	0.8	0.6–0.75 50% HBV ^b	1.2 50% HBV	1.2–1.3 50% HBV	1.3–1.5 initial 1.0 for maintenance
Fat (% total kcal)	30%–35%	Patients considered at highest risk for cardiovascular disease; emphasis on PUFA ^c , MUFA ^d , 250–300 mg cholesterol/day			<10% saturated fat
Sodium (mg/d)	Unrestricted	2,000	2,000	2,000	Unrestricted; monitor medication effect
Potassium (mg/d)	Unrestricted	Correlated to laboratory values	2,000–3,000 (8–17 mg/kg/d)	3,000–4,000 (8–17 mg/kg/d)	Unrestricted; monitor medication effect
Calcium (mg/d)	Unrestricted	1,200	≤ 2,000 from diet and medications	≤ 2,000 from diet and medications	1,200
Phosphorus (mg/d)	Unrestricted	Correlated to lab values	800–1,000	800–1,000	Unrestricted unless indicted
Fluid (ml/d)	Unrestricted	Unrestricted with normal urine output	1,000 + urine output	Monitored; 1,500–2,000	Unrestricted unless indicated

a Meant as guidelines only for initial assessment; individualization to patient's own metabolic status and co-existing metabolic conditions is essential for optimal care.

b HBV=high biological value.

c PUFA=polyunsaturated fatty acids.

d MUFA=monounsaturated fatty acids.

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PROTEIN REQUIREMENTS

Chronic Renal Failure

Protein requirements for patients with renal failure are dependent on the acute or chronic nature of the renal failure and the presence and type of dialysis. The nutritional status and adequacy of current intake of the patient should also be considered. Adults with chronic renal failure who are not receiving dialysis can usually maintain a neutral nitrogen balance consuming 0.6 g of protein per kilogram *if adequate calories are ingested* and most of the protein is of high biological value (11). A reduced protein intake may decrease uremic symp-

toms and delay the need for dialysis in a stable patient with chronic renal insufficiency. However, a reduced protein intake is not advisable in the setting of significant malnutrition, or inadequate calorie intake.

Acute Renal Failure

There is no data to suggest that a protein restriction is of any benefit in the setting of acute renal failure associated with severe illness or multi-organ dysfunction. In patients who are acutely ill with increasing uremia, there is a temptation to focus on the protein content of nutritional support as a major contributor to uremia. It is impor-

tant to remember that dietary protein comprises only 25% of the total nitrogen pool that is metabolized by the body each day, and that the difference between 1.0 gm protein/Kg and 1.3 gm protein/kg in a 70 Kg patient is only 21 gm of protein per day. Nitrogen load from GI bleeding, inadequately controlled serum glucose, or no nutritional intake in the setting of acute illness would lead to significantly more urea generation than 10–20 gm of additional protein over 24 hours. Patients with multi-system organ failure frequently require dialysis and specialized nutrition support that meets the calorie and protein requirements for critical illness.

The protein requirements for patients receiving dialysis are increased above the requirements for healthy adults. Hemodialysis and peritoneal dialysis increase nitrogen losses. In addition, there is information that hemodialysis itself is an inflammatory and catabolic process (12). The National Kidney Foundation's guideline is 1.2 g protein/Kg/day for stable maintenance hemodialysis patients, and 1.2–1.3 gm protein/day for stable peritoneal dialysis patients (9). Patients with malnutrition, acute catabolic illness, or with postoperative wounds should receive greater than 1.3 gm protein/Kg/day. There is data that increasing protein to 2.0–2.5 gm protein/Kg will result in improved nitrogen balance in hospitalized patients with acute renal failure (13–15). However, increasing protein intake beyond 1.5–1.6 gm/Kg may increase the rate of urea nitrogen appearance, and increase the need for frequent dialysis (16). There is no prospective study that adequately addresses the question of what protein provision will result in the best outcomes for acutely ill patients requiring dialysis. See Table 2 for protein recommendations in renal failure.

NUTRITION INTERVENTION

Oral

The frequent occurrence of malnutrition in patients with renal failure, and the consistent association between markers of malnutrition and poor outcome in this population emphasize the need for appropriate and timely nutrition intervention. Nutrition assessment and counseling with the patient and family is advisable; but it is the consistent follow-up, with modification of the

Table 3
Strategies to enhance oral intake

- Avoid diet restrictions in patients with poor intake
- Offer oral liquid supplements and snacks
- Treat gastroparesis and other GI maladies
- Achieve glycemic control
- Correct electrolyte abnormalities
- Provide foods appropriate for patient's dentition
- Evaluate for, and address depression

nutrition plan as clinical status changes, that is essential. Modifying the diet consistency to match dentition, adjusting traditional recipes and foods to fit the current plan, or providing oral liquid supplements can all improve nutrition intake. Non-essential diet restrictions should be avoided. Frequently a multi-disciplinary effort is required to address reversible conditions or to identify medications that may contribute to anorexia or inability to eat. Cohort studies of occult gastroparesis and bacterial overgrowth point to some of the treatable GI manifestations that may contribute to malnutrition (5). Table 3 provides suggestions to enhance oral intake.

Renal compensatory mechanisms maintain normal serum potassium levels until GFR drops below 15–20 mL/minute (17). Dietary potassium is generally restricted to 2000–3000 mg/day for patients requiring hemodialysis, and 3000–4000 mg/day for patients requiring peritoneal dialysis. There are a number of non-food factors that can cause or contribute to hyperkalemia (Table 4). Correcting underlying factors causing hyperkalemia, such as inadequate glucose control (18) will frequently allow patients a more liberal diet restriction that will encourage good oral intake.

Dietary sodium intake is frequently restricted to 2000–4000 mg per day for patients with chronic kidney disease in an effort to aid in the control of hypertension, and to avoid excessive thirst and fluid consumption in those patients with oliguria or anuria. Salt substitutes frequently contain potassium chloride, and patients should be instructed to avoid salt substitutes that are not approved by their dietitian or physician.

Patients with chronic kidney disease frequently experience hyperphosphatemia when their glomerular

(continued on page 51)

(continued from page 47)

Table 4
Dietary and Non-Dietary Causes of Hyperkalemia

Rule out dietary causes of hyperkalemia

- Misconceptions by patient concerning unlimited intake of no-potassium foods.
- Ingestion of new products and/or new varieties of fruits and vegetables.
- Continuation of previous emphasis on potassium if hypertensive.
- Ingestion of large amounts of licorice containing glycyrrhizic acid.
- Use of ethnic-cultural traditions influencing intake, i.e., fasting, home remedies.
- Document portion control is not out of control.
- Consumption of nutritional supplements outside of diet parameters.

Rule Out Non-dietary Causes of Hyperkalemia

- Decreased gut excretion with chronic constipation.
 - Consider increasing fiber in diet to promote gut motility.
 - Change phosphate-binding agents to reduce constipation effect.
- Check validity of laboratory values.
 - Verify prior treatment post-dialysis potassium.
 - Rule out hemolyzed blood error.
- Evaluate interaction of laboratory values.
 - Check acid-base status acidosis.
 - Each 0.1 change in arterial pH may increase serum potassium by 6-1.0 mEq/L.
 - Consider effect of serum glucose-insulin relationship.
 - Hyperkalemia secondary to insulin deficiency particularly in diabetics.
 - Elevated PTH (hyperparathyroid) may cause calcium-potassium intracellular shift.
 - Indicators of inadequate dialysis by presence of elevated BUN or other parameters.

- Review medical and treatment effects.
 - Inadequacy of dialysis contributing to intradialytic accumulation of potassium.
 - Type of dialysate being used, i.e., high-potassium bath.
 - Inefficiency or access site.
 - Evaluation of continuous ambulatory peritoneal dialysis compared with hemodialysis to maximize potassium removal.
 - Consider drug interactions.
 - Penicillin
 - Beta-blockers, i.e. propranolol
 - Angiotensin-converting enzyme inhibitors, i.e., captopril
 - Muscle relaxants, i.e. succinylchloride
 - Non-steroidal anti-inflammatory agents, indomethin
 - Steroids
 - Over-the-counter drugs
 - Presence of concomitant diseases, i.e., defects of aldosterone or renin (hyporeninism, hypoaldosterone), Addison's disease, sickle cell anemia.
 - Invasive therapy causing release of potassium through tissue destruction.
 - Infection, gangrene, chemotherapy, surgical stress, gastrointestinal hemorrhage.
 - Catabolism, starvation, malnutrition with change in dry weight.
 - Clotting, bruising, inflammation of access site or other area.
 - Increased activity and exercise due to use of erythropoietin to improve anemia status.

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filtration rate (GFR) drops to 20–30 mL/min (19). A dietary phosphorus restriction of 800–1000 mg per day should be implemented when serum phosphorus rises >4.6 mg/dL (19). There is recent evidence that phosphorus excretion is affected when GFR drops below 60 mL/min, contributing to secondary hyperparathyroidism. The increased serum parathyroid hormone normalizes serum phosphorus level until GFR drops below 20–30 (19). A dietary phosphorus restriction of 800–1000 mg/day decreases PTH levels and may reduce bone resorption in those patients with elevated PTH. Patients with hyperphosphatemia frequently receive calcium-containing phosphate binders, which

can contribute to hypercalcemia or elevation of the serum calcium-phosphorus product. The National Kidney Foundation recommends that serum calcium-phosphorus product be maintained at <55 mg/dL to prevent soft tissue calcification (19). Calcium from phosphorus binders should be maintained below 1500 mg/day, and total calcium intake (supplements and diet) should not exceed 2000 mg/day (19).

ENTERAL FEEDINGS

Enteral nutrition support should be used in those patients who are unable to meet their nutrition needs with appro-

**Table 5
Comparison of Standard vs Renal Products**

<i>Product</i>	<i>Kcal/kg</i>	<i>Protein gm / 1000 kcal</i>	<i>Potassium mEq/1000 kcal</i>	<i>Phosphorus mEq/1000 kcal</i>	<i>Sodium mEq/1000 kcal</i>	<i>Cost per 1000 kcal</i>
Standard Products						
Jevity 1.5 (1)	1.5	45.0	31.6	51.6	40.6	\$6.33
Nutren 1.5 (2)	1.5	40.0	32	43	34	\$3.22
TwoCalHN (1)	2.0	42.0	31	33.8	32	\$3.08
Deliver 2.0 (3)	2.0	37.5	21.5	555	33	\$3.41
Renal Products						
Nepro (1)	2.0	35.0	14	22.4	34	\$5.83
Suplena (1)	2.0	15.0	14	22.4	32	\$3.58
Magnacal (3)	2.0	37.5	16	32.2	43	\$3.33
RenalCal (2)	2.0	17.0	Negligible	Negligible	Negligible	\$14.96
NovaSource Renal (4)	2.0	37.0	14	41.9	20	\$6.07
1) Ross	1-800-986-8502	www.rosstore.com				
2) Nestle	1-800-776-5446	www.nestleclinicalnutrition.com				
3) Mead Johnson	1-800-247-7893	www.meadjohnson.com				
4) Novartis	1-800-828-9194	www.walgreens.com/store/novartis				

appropriate counseling and encouragement. Hospitalized patients may benefit from a period of nasogastric feedings until acute illness resolves. A patient's willingness to accept nasogastric tube (NGT) placement is frequently dependent on the method of presentation. If feeding tube placement is discussed as an important and beneficial part of their overall care, a patient is much more likely to agree to NGT placement. Experience shows that "threatening" a patient with NGT placement if they do not eat adequately, seldom changes the factors that limit intake, is frequently perceived as a "punishment" and only serves as a barrier to providing needed nutrition support.

Enteral feeding allows provision of complete nutrition in a minimal fluid volume, is associated with fewer infectious complications, and is significantly less expensive than parenteral nutrition. Parenteral nutrition should be reserved for those patients who are unable to receive or tolerate enteral feedings.

Many patients tolerate gastric feedings, although some patients may require the use of a calorie-dense formula and slower feeding rate during periods of delayed gastric emptying. A recent prospective trial suggested that hospitalized patients with renal failure receiving dialysis experienced increased gastric residuals, and had increased use of prokinetic drugs, com-

pared to similar patients not receiving dialysis (20). Some patients may require small bowel positioning of the feeding tube tip if they have a history of gastroparesis or if they do not tolerate gastric feedings. Patients with active pancreatitis should have the tip of the feeding tube placed beyond the ligament of Treitz.

There is no data to suggest an outcome advantage of enteral feeding formulas with essential amino acids. However, there may be patients who benefit from the calorie-dense renal formulas with reduced amounts of potassium and phosphorus. Patients without hyperkalemia or hyperphosphatemia, or those at risk for refeeding syndrome, frequently can be maintained on standard enteral formulas. Many calorie-dense enteral formulas have modest amounts of electrolytes, and some standard formulas fit within the electrolyte guidelines for a maintenance dialysis population. Patients with non-oliguric renal failure receiving large doses of loop diuretics, and those receiving continuous renal replacement therapies as seen in intensive care units, may actually require electrolyte replacement, and restriction of electrolytes is counterproductive. See Table 5 for a comparison of standard calorie dense vs. renal products.

(continued on page 54)

(continued from page 52)

PEG Placement

Patients who will require extended nutrition support may be appropriate for placement of long-term feeding access such as a percutaneous endoscopic gastrostomy (PEG). Peritoneal dialysis is generally considered a contraindication to PEG placement due to the risk of peritonitis. In two different patient series, maintaining hemodialysis for six weeks after PEG placement decreased, but did not eliminate, peritonitis when peritoneal dialysis was restarted (21,22). There is limited randomized data specific to maintenance hemodialysis patients receiving PEG feedings. Cohort data suggest that PEG feedings are effective and safe in patients receiving long-term hemodialysis (23).

Parenteral Nutrition

Total parenteral nutrition is associated with increased infectious complications and is significantly more expensive than enteral feedings. Parenteral nutrition also requires a greater fluid volume to meet calorie and protein needs than the equivalent enteral nutrition. Parenteral nutrition should be reserved only for those patients who are unable to receive enteral nutrients.

One method of delivering parenteral nutrition to hemodialysis patients is to provide intradialytic parenteral nutrition (IDPN). IDPN allows parenteral nutrition without concern for volume overload, additional time commitment of the patient, or additional vascular access. The two primary disadvantages of IDPN is the cost (~\$200–500 per “dose”), and the small amount of nutrition that is actually provided per “dose.” IDPN cannot meet a patient’s basic calorie or protein needs, in part because it is provided only three times per week during dialysis. Typically IDPN provides an average of only 400–500 calories and 30–40 gm protein per day. Finally, there is no prospective, randomized controlled evidence supporting improved clinical outcomes with IDPN.

NUTRITION MONITORING

Monitoring of the nutrition status of patients with renal failure is complicated by the fact that many traditional markers of nutrition are altered by the primary disease, and do not correlate with changes in nutrition intake.

Weight status is frequently altered by changes in volume status and dialysis. The large shifts in weight that occur with changes in fluid status may mask a slow persistent loss of lean muscle or fat for several weeks to months. Patients who receive hemodialysis are frequently dialyzed to a specific weight. It is not uncommon for slow weight loss to go unnoticed until a new “dry” or “target” weight has been established in order to achieve adequate clearance of uremia.

There is a persistent myth that serum proteins, such as albumin and prealbumin are markers of nutrition status in hospitalized patients. There may be some very specific instances of isolated protein malnutrition coupled with a surplus of carbohydrate (kwashiorkor) in which malnutrition may reduce albumin, such as seen in third world countries. However, there is a large body of evidence that documents that serum proteins correlate inversely with acute phase reactants, and do not correlate with nutrition intake in a physiologically stressed patient (24–29). Reduced serum albumin is associated with increased morbidity and mortality in renal failure, but this association reflects the presence of comorbid conditions. Albumin synthesis is reduced or inhibited in humans by acidemia or increased IL-1 respectively (2,7,25). Prealbumin levels are elevated in renal failure, but may be decreased immediately after hemodialysis. Prealbumin levels have a strong, inverse association with C-reactive protein and other acute-phase reactants (24–29).

Frequently, the most valuable nutrition assessment must rely on an evaluation of intake, compared to an estimation of the patient’s energy and protein needs (30). Monitoring edema-free body weight over time, in the outpatient setting is a practical, albeit insensitive, monitoring tool. Subjective Global Assessment (SGA) combines assessment of intake, physical findings and functional status. SGA is a practical approach to nutrition assessment, but is affected by the subjective nature of the assessments, and may not be sensitive to acute changes in oral intake (31). In practice we find that it is exceedingly rare that a patient’s nutritional status is an enigma that requires sophisticated labs to decipher. Interacting with a patient, their family and caregivers, and a review of intake will frequently identify the patient with nutrition compromise. In the clinical setting, time and energy are more effectively spent ensuring that the patient has a

Table 6
Renal Vitamin Supplements

Product Information	Diatx	Nephrovite+	Nephro Caps++	Nephlex RX+++	Nephron FAA++++
Folic acid	5 mg	1 mg	1 mg	1 mg	1 mg
Cobalamin (B ₁₂)	1 mg	6 mcg	6 mcg	6 mcg	6 mcg
Pyridoxine (B ₆)	50 mg	10 mg	10 mg	10 mg	10 mg
Thiamine (B ₁)	1.5 mg	1.5 mg	1.5 mg	1.5 mg	1.5 mg
Riboflavin (B ₂)	1.5 mg	1.7 mg	1.7 mg	1.7 mg	1.7 mg
Niacinamide or Niacin	20 mg	20 mg	20 mg	20 mg	20 mg
Pantothenic acid (B ₅)	10 mg	10 mg	5 mg	10 mg	10 mg
Biotin	300 mcg	300 mcg	150 mcg	300 mcg	300 mcg
Vitamin C	60 mg	60 mg	60 mg	60 mg	40 mg
Prescription required	YES	YES	YES	YES	YES

Note: Replavite AND Hillvite – Identical to Nephlex except has 100 mg vitamin C

Information accessed from: <http://www.diatx.com/comparison.php> (8/1/04)

+Nephrovite RX is a registered trademark of R&D Laboratories

++Nephrocaps is a registered trademark of Fleming

+++Nephlex RX is a registered trademark of Nephro-Tech

++++Nephron FA RX is a registered trademark of Nephro-Tech

reasonable and consistent nutrition intake, either by oral, supplemental, or enteral tube feeding, than in trying to precisely define nutritional status.

Protein Equivalent of Total Nitrogen Appearance (PNA)

The protein equivalent of total nitrogen appearance (PNA) is used to estimate protein intake in a dialysis patient in the stable, “steady-state.” It is calculated from the change in BUN and body water (normalized to body weight) between dialysis sessions. In patients with urine output, urinary nitrogen must be measured as well (31–33). PNA is altered by any anabolic and catabolic factors such as corticosteroids, sepsis, or anabolic hormones. PNA is closely correlated with dietary protein intake *only* in the steady state; i.e., when protein and energy intake are constant, when there is a constant level of stressors, when there is no recent change in catabolic or anabolic hormones, and the dose of dialysis is constant. In the individual patient who is in a stable steady-state and who has none of the previously mentioned conditions that would interfere with the normal-

ized PNA (nPNA), it may be reasonable to assume that nPNA reflects the dietary protein intake (33,34). Due to the multiple factors stated here that alter the reliability of nPNA, nPNA is of limited utility in the hospitalized patient as a measure of protein requirement or intake.

Vitamins and Minerals

Patients with renal failure are at risk for several micronutrient deficiencies. Patients who require dialysis are known to lose certain water-soluble vitamins, and deficiencies of thiamine, folate, pyridoxine and vitamin C have been reported (35). However, patients with renal failure have decreased excretion of vitamin A and cases of hypervitaminosis A have been described in patients who receive standard multivitamin supplements (35). In light of newer information that links increased intake of vitamin A with increased fracture incidence, this is especially concerning. Vitamin E toxicity has not been reported, and doses of 300 to 800 IU of vitamin E are associated with reduced oxidative byproducts, but outcome information is equivocal, and vitamin E supplementation remains controversial (35). Patients who require dialysis should receive a multivitamin supplement designed for patients with renal failure, that avoids excessive vitamin C and limits vitamin A (Table 6).

Vitamin D deficiency (defined as 25-OH vitamin D <67 to 80 nmol/L) is associated with elevated PTH levels, and increased incidence of hip fractures (19). Patients with chronic renal insufficiency (GFR 20–60 mL/min) who have elevated PTH should have serum 25-OH vitamin D checked (not 1,25-OH vitamin D). Those patients with 25-OH vitamin D <75 nmol/L should receive standard vitamin D supplements (19). Patients who have a GFR <20mL/min, or who receive dialysis may no longer benefit from standard vitamin D due to their inability to produce 1,25-OH vitamin D. Patients who require dialysis generally receive a form

(continued on page 58)

(continued from page 55)

Table 7
Recommended vitamin intake for patients receiving maintenance hemodialysis

Thiamine (B ₁)	1.1–1.2 mg/day
Riboflavin (B ₂)	1.1–1.3 mg/day
Niacin	14–16 mg/day
Pantothenic Acid	5 mg/day
Vitamin B ₆	10 mg/day
Vitamin B ₁₂	2.4 mg/day
Biotin	30 mcg/day
Vitamin C	75–90 mg/day
Folic acid	1 mg/day
Zinc	15 mg/day

of “active” vitamin D (1,25 dihydroxycholecalciferol). A full discussion of vitamin D forms, doses, and monitoring is beyond the scope of this article. See the National Kidney Foundation’s K-DOQI clinical practice guidelines for bone metabolism and disease (19). A complete review and recommendations of vitamin D in kidney disease is also available online at: http://www.kidney.org/professionals/kdoqi/guidelines_bone/index.htm.

Iron supplementation is often necessary in patients who receive erythropoietin. Those patients who do not tolerate oral iron supplementation, or are unable maintain adequate iron stores with oral supplementation receive parenteral iron. There is some data that patients who receive hemodialysis may be at increased risk of zinc deficiency. Several studies have found that patients with decreased serum zinc who receive zinc supplements have improvement in taste alterations and sensitivity (35).

Patients with renal failure have elevated levels of homocysteine, which has been associated with increased risk of cardiovascular disease in population studies of healthy adults. Supplemental folic acid and pyridoxine reduce, but do not normalize, homocysteine in dialysis patients (36). Paradoxically, increased homocysteine was associated with improved survival in patients with renal failure, which has been called “reverse epidemiology” (37). The significance of homocysteine, and the role of vitamin supplementation to reduce homocysteine remains an area of substantial debate and research.

Carnitine is present in the diet, and also synthesized in the liver from the amino acid lysine. Carnitine is

abundant in muscle, and is an essential co-factor for the oxidation of long-chain fatty acids by mitochondria. Patients undergoing maintenance dialysis frequently have decreased serum levels of free carnitine. Decreased muscle carnitine has also been demonstrated in some patients (9). L-carnitine has been proposed as a treatment for several metabolic disorders in renal failure, including hypertriglyceridemia, hypercholesterolemia, and anemia. It has also been proposed as a treatment for several symptoms of dialysis, including intradialytic arrhythmias and hypotension, low cardiac output, dialysis-related malaise, general weakness or fatigue, skeletal muscle cramps, and decreased exercise capacity (9). Randomized trials of L-carnitine in renal failure have produced inconclusive results, and current guidelines do not include routine supplementation of L-carnitine in chronic kidney disease. A trial L-carnitine supplementation may be considered in selected cases of erythropoietin-resistant anemia, fatigue, hypertriglyceridemia, or hypotension where standard therapy has produced inadequate response, and other contributors to these conditions have been ruled out or treated (9). Recommendations for vitamin/mineral supplementation in patients with renal disease can be found in Table 7.

CONCLUSION

Dietary management is an essential component for patients with chronic kidney disease. Current trends demonstrate a significant increase in this segment of our healthcare population, and gastroenterologists can anticipate increased interaction with patients who have kidney disease. Treating underlying GI issues that prevent adequate intake can improve nutrition status, and improved nutrition has the potential to reduce morbidity for those with renal failure.

Malnutrition occurs commonly in patients with chronic kidney disease, but traditional markers of nutrition status, such as albumin, may reflect inflammatory status, or co-morbid conditions. Monitoring trends in target weight, and evaluation of recent intake may be the best tools to alert the clinician to changes in nutrition status. Avoiding unnecessary diet restrictions, addressing underlying conditions that impair oral intake, and providing nutrition support when oral intake is inadequate can prevent the addition of iatro-

genic malnutrition to the list of complications that beset the patient with chronic kidney disease. ■

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