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Nutrition Management of the Patient with Acute Renal Failure

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Abstract

Acute renal failure (ARF) is associated with a mortality rate up to 50% despite advances in clinical practice and

technology. ARF is an abrupt cessation or decline in normal renal function that is manifested by elevations of blood urea nitrogen (BUN) and serum creatinine that may be associated with decreased urine output. ARF is designated as prerenal, renal (intrarenal or intrinsic renal disease,) and postrenal, each of which has different causes. Systemic effects of ARF can be extensive, with associated metabolic alterations affecting carbohydrate, lipid, and protein metabolism. The initiation of renal replacement therapy (RRT) is an important consideration in patients who have ARF

and are receiving nutrition support. This article addresses nutritional requirements and considerations of patients with ARF.

The Kidneys

The primary functions of the kidneys are to excrete end products of metabolism, regulate electrolyte and mineral concentrations, and maintain fluid and electrolyte balance (1). Other functions include urine production, dilution, and concentration; maintenance of blood pressure; concentration of extracellular and intracellular fluids; gluconeogenesis; maintenance of Ca/PO₄ balance; and activation of vitamin and hormone synthesis (1). The kidney has approximately 1 million nephrons, each of which is composed of several functional segments, including the glomeruli, proximal tubules, distal tubules, loop of Henle, and collecting duct, which drains into the renal pelvis. The nephron clears plasma of the end products of metabolism (urea, creatinine, uric acid, inorganic and organic acids). Electrolytes (sodium, potassium, chloride, bicarbonate), minerals (calcium, phosphorus, magnesium), and micronutrients (zinc, selenium) are filtered through the glomeruli and are reabsorbed or excreted based on needs. Small nutrients such as glucose, small proteins, amino acids, and vitamins are filtered through the glomerulus and reabsorbed via active transport in the proximal tubule of the kidney (1).

Acute Renal Failure (ARF)

ARF is an abrupt cessation or decline in normal renal function manifested by

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Table 1. Systemic or Physiologic Effects of ARF (4, 5)

Kidney	Decreased urine production leading to fluid retention, edema. Accumulation of nitrogenous waste.
Cardiac	Pulmonary or peripheral edema, arrhythmias. Loss of muscle tone, weakness, neuromuscular irritability, tingling lips/fingertips, electrocardiographic changes with hyperkalemia. Potassium levels >6 mg/dL could trigger bradycardia, heart block, asystole or other arrhythmia, or heart failure.
Respiratory	Dyspnea at rest or exertion. Crackles in lungs.
Neurologic	Accumulation of metabolic waste can affect mental status. Changes in cognitive function and level of consciousness, sensory change, and weakness of extremities can be signs of uremic neuropathy. Paresthesias related to hypocalcemia. Headaches, syncope, and seizures.
Hematology	Impaired red blood cell production, hemolysis, bleeding, hemodilution, and reduced red blood cell survival (60 versus 120 d) contributing to anemia, fatigue, and malaise. Less production of erythropoietin. Low hemoglobin/hematocrit causing a decrease in oxygenation, leading to dyspnea and platelet dysfunction.
Gastrointestinal	Anorexia, nausea, vomiting, constipation, diarrhea, dysgeusia, and stomatitis affecting overall nutrition. Weight loss masked by fluid retention. Uremia can trigger colitis, gastric ulcer, and bleeding. Increased urea causes foul breath.
Visual Skin	Blurring related to electrolyte and fluid imbalance, hypertension. Dryness, pitting edema, anasarca. Pruritus, ecchymosis, petechiae, delayed healing, pallor, yellowness related to uremia.

From the Editor's Desk

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Articles about successful programs, research interventions, evaluations and treatment strategies, educational materials, meeting announcements and information about educational programs are welcome and should be sent to the Managing Editor by the next deadline.

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For dietitians who haven't fretted about the RD exam in several years, a credentialing exam is a great measure of clinical knowledge and competency. The Renal Practice Group (RPG) leadership is busy equipping members with valuable tools for practicing advanced renal nutrition. In particular, RPG is filling the lending libraries with recommended readings for the board certification as Specialist in Renal Nutrition (CSR) exam. These materials will help prepare CSR test takers for questions on chronic kidney disease (CKD), acute renal failure (ARF) and kidney transplant.

Because renal dietitians may lack regular exposure to patients with ARF, we have reprinted Marcia Kalista-Richards' recent article from Support Line, the Dietitians in Nutrition Support's newsletter. You may have attended Marcia's session on ARF at this year's National Kidney Foundation spring Clinical Meetings. The article is available for two continuing professional education units (CPEU) from the Commission on Dietetic Registration. Thank you goes to Cathy Folk, MA, RD, CSR, LDN, for writing our CPE questions. This Nashville-based renal dietitian also developed some of the CSR exam questions.

The nutritional benefit of fish oil supplements has recently become a hot topic in dietetics. Fish oil is the fastest growing dietary supplement in the U.S., and the American Heart Association recommends it for patients with documented coronary heart disease and hypertriglyceridemia (under physicians'

guidance). Philippa Norton Feiertag shares a timely article on the use of fish oil in patients with CKD.

The summer months are perfect for spending time outdoors. In her Rehab Corner article, Stephanie McIntyre reminds dietitians to encourage patients to get out and exercise. Also, Sharon Shatz offers a smorgasbord of ideas to enhance patients' calorie and protein intakes, which may slip during hot weather.

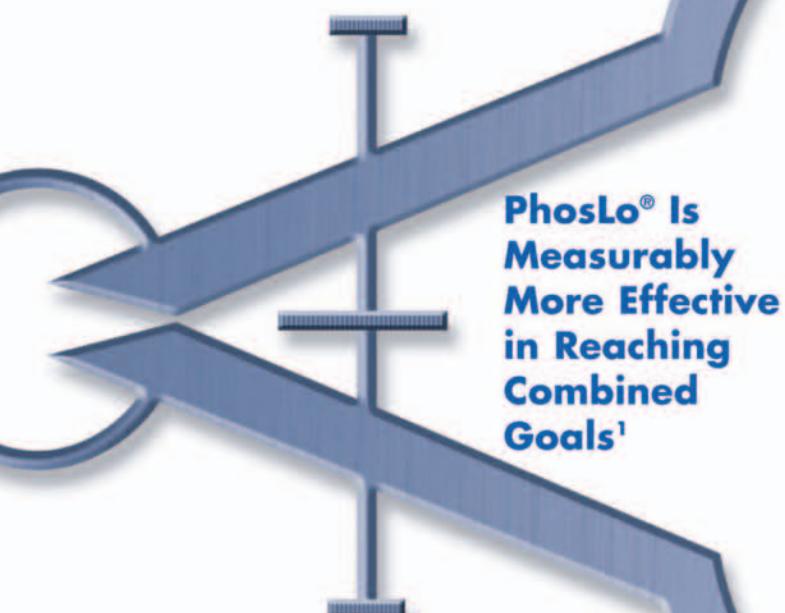
If you are taking a summer break from some of your professional activities, consider writing for the Renal Nutrition Forum. We need one-time feature article authors as well as regular column contributors. With a little creativity, you may be able to write an article while sitting on the beach during your vacation. If the thought of writing is overwhelming, consider serving as a peer reviewer. Several of our regular reviewers have reviewed articles while traveling all over the world. Being a peer reviewer is a highly portable volunteer position!

Summer marks the time of year when RPG officers start their new roles. I would like to thank Pat Weber for being such an amazing managing editor. She has mentored me through a position about which I had very little knowledge or experience. I am proud to work in her shadow and look forward to her leadership of RPG. Please welcome Sharon Griff as editor for the coming year. Sharon is a product manager for Abbott Laboratories, Renal Care Division. She brings strong business knowledge to our editorial board and an eagerness to learn. Catherine Goeddeke-Merickel will serve as assistant editor for an additional year while she nurtures the new addition to her family. Our fantastic advertising editor, Marianne Hutton, is frequently overlooked because she is so consistent. Her hard work and dedication allow us to focus on the publication, not the shipping costs. As always, the editorial team will strive to publish the information needed to help each member grow as a renal nutrition expert. Please offer your ideas for future articles to the editorial team at rnfeditor@yahoo.com. Have a great summer!



For the control of hyperphosphatemia in end stage renal failure,

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BRIEF SUMMARY OF PRESCRIBING INFORMATION

CONTRAINDICATIONS: Patients with hypercalcemia. **INDICATIONS AND USAGE:** For the control of hyperphosphatemia in end stage renal failure. **WARNINGS:** Patients with end stage renal failure may develop hypercalcemia when given calcium with meals. No other calcium supplements should be given concurrently with PhosLo. Progressive hypercalcemia due to overdose of PhosLo may be severe as to require emergency measures. Chronic hypercalcemia may lead to vascular calcification, and other soft-tissue calcification. The serum calcium level should be monitored twice weekly during the early dose adjustment period. **The serum calcium times phosphate (CaXP) product should not be allowed to exceed 66.** Radiographic evaluation of suspect anatomical region may be helpful in early detection of soft-tissue calcification. **PRECAUTIONS:** Excessive dosage induces hypercalcemia; therefore, early in the treatment during dosage adjustment serum calcium should be determined twice weekly. Should hypercalcemia develop, the dosage should be reduced or the treatment discontinued immediately depending on the severity of hypercalcemia. Do not give to patients on digitals, because hypercalcemia may precipitate cardiac arrhythmias. Always start PhosLo at low dose and do not increase without careful monitoring of serum calcium. An estimate of daily calcium intake should be made initially and the intake adjusted as needed. Serum phosphorus should also be determined periodically. **Information for the Patient:** Inform the patient about: 1) compliance with dosage, 2) adherence to diet instructions and avoidance of nonprescription antacids, and 3) symptoms of hypercalcemia. **Drug Interactions:** PhosLo may decrease the bioavailability of tetracyclines. **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Long term animal studies have not been performed. **Pregnancy:** Teratogenic Effects: Category C. Animal reproduction studies have not been conducted. It is not known whether PhosLo can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. Give to a pregnant woman only if clearly needed. **Pediatric Use:** Safety and effectiveness in pediatric patients have not been established. **Geriatric Use:** Of the total number of subjects in clinical studies of PhosLo (n = 91), 25 percent were 65 and over, while 7 percent were 75 and over. No overall differences in safety or effectiveness were observed between these subjects and younger subjects, and other reported clinical experience has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out. **ADVERSE REACTIONS:** In clinical studies, patients have occasionally experienced nausea during PhosLo therapy. Hypercalcemia may occur during treatment with PhosLo. Mild hypercalcemia (Ca>10.5 mg/dl) may be asymptomatic or manifest itself as constipation, anorexia, nausea and vomiting. More severe hypercalcemia (Ca>12 mg/dl) is associated with confusion, delirium, stupor and coma. Mild hypercalcemia is easily controlled by reducing the PhosLo dose or temporarily discontinuing therapy. Severe hypercalcemia can be treated by acute hemodialysis and discontinuing PhosLo therapy. Decreasing dialysate calcium concentration could reduce the incidence and severity of PhosLo induced hypercalcemia. The long-term effect of PhosLo on the progression of vascular or soft-tissue calcification has not been determined. Isolated cases of pruritus have been reported which may represent allergic reactions. **OVERDOSAGE:** Administration of PhosLo in excess of appropriate daily dosage can cause severe hypercalcemia (see ADVERSE REACTIONS).

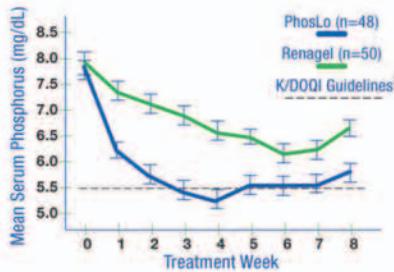
REFERENCE: 1. Qunibi WY, Hootkins RE, McDowell LL, et al. Treatment of hyperphosphatemia in hemodialysis patients: The Calcium Acetate Renagel Evaluation (CARE Study). In press.

2. K/DOQI guidelines (in press).

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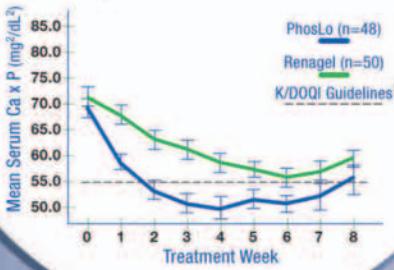
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elevations of blood urea nitrogen and serum creatinine that may be associated with decreased urine output (2,3). Urine flow or output rates used in the diagnosis of ARF include anuria ($<100\text{mL/d}$), oliguria (100 to 500mL/d), and polyuria nonoliguria ($>500\text{mL/d}$); normal urine output is 1 to 1.5L/d (3). ARF occurs when taxation of kidneys causes a sudden loss of function in at least 50% of nephrons (4). The problem may be specific to the kidney or part of multiple organ dysfunction syndrome (MODS). If the underlying cause is corrected, the nephrons may recover. In some cases, damage is permanent, and renal failure becomes chronic. ARF also may be superimposed on pre-existing renal insufficiency.

Although ARF is a disorder of the kidneys, it can have systemic effects (Table 1, page 1) and result in significant metabolic alterations (Table 2).

The prevalence of ARF is 1% in all hospitalized patients, 3% to 5% in general medical/surgical patients, 5% to 25% in intensive care unit (ICU) patients, 5% to 20% with open heart surgery, 20% to 60% in severe burns, 10% to 30% with aminoglycoside therapy, 20% to 30% in rhabdomyolysis, and 15% to 25% among those treated with the chemotherapeutic drugs cisplatin, bleomycin, and vinblastine (3,8).

Despite advances in clinical practice and technology, the 50% mortality rate associated with ARF remains constant (3, 4, 8). Death typically is due to severe infection or cardiopulmonary problems. Among predictive factors for a worse prognosis

are age, previous health status, pre-ICU hospitalization, delayed occurrence of ARF, sepsis, oliguria, and severity of illness (including need for ventilation and hypotension)(9). The predictive value of nearly each factor influencing outcome in acute renal failure is still debated and continues to require further studies (9).

Causes of ARF

ARF can be defined as prerenal, postrenal, and renal (intrarenal or intrinsic renal disease), and the causes vary by type.

Prerenal ARF results from a decrease in renal perfusion that may be due to volume depletion (gastrointestinal fluid loss, excess diuresis, or salt-wasting nephropathy), volume redistribution (vasodilation from sepsis or antihypertensives), burns, peritonitis, pancreatitis, hypoalbuminemia (nephrotic syndrome, hepatic disease), reduced cardiac output (pericardial tamponade, myocardial infarction complications, acute/chronic valvular disease, cardiomyopathies, arrhythmias), or an embolus (thromboembolic or cholesterol) (3). Restoring renal blood flow and glomerular ultrafiltration can reverse prerenal ARF rapidly (3, 4).

Use of certain medications may contribute to the incidence of prerenal ARF. For example, nonsteroidal anti-inflammatory drugs (NSAIDs), cyclooxygenase inhibitors, or angiotensin-2 receptor blockers can impair renal autoregulatory responses by blocking the prostaglandin production that is needed to maintain renal perfusion. The risk for ARF associated with these medications is high in the elderly and those

who have renal insufficiency, heart failure, and advanced liver disease (4).

Postrenal ARF results from obstruction of urine flow. Prostatic hypertrophy, ureteral obstruction (usually bilateral), and bladder outlet obstruction are common causes. Malignancy, inflammatory processes, vascular diseases, papillary necrosis, intratubular crystals (from methotrexate, acyclovir, calcium phosphate, sulfonamide antibiotics) also can be causative (3). Postrenal ARF is found in 1% to 14% of hospitalized azotemic patients, most often among the elderly, and generally can be reversed with proper medical management (3).

Intrarenal ARF (intrinsic ARF) is associated with renal parenchyma damage. Prerenal ARF can trigger the problem, but a major cause of intrarenal ARF and ARF in general is acute tubular necrosis (ATN), which usually is associated with damage to renal tubules caused by ischemia or nephrotoxins such as radiocontrast or aminoglycoside (3). The terms ARF and ATN frequently are used interchangeably, although they are not the same condition.

Predisposing factors to ATN include renal ischemia from prolonged prerenal azotemia, nephrotoxins (radiocontrast agents, aminoglycoside antibiotics, amphotericin, vancomycin, NSAIDs, cyclosporine), chemotherapeutic agents (cisplatin, carboplatin, methotrexate), pigmenturia (rhabdomyolysis, hemolysis), interstitial nephritis (ciprofloxacin, thiazides, furosemide, phenytoin, tetracycline, penicillin), and infections (streptococcal, staphylococcal)(3). Some clinical conditions associated with ATN are abdominal aortic aneurysm repair, major trauma, postoperative states, and gram-negative bacterial sepsis (3).

Diagnosing ARF

A variety of tests are used to diagnose ARF, including a history and physical examination, determination of urine flow, laboratory investigations such as blood urea nitrogen or serum creatinine, urinalysis,

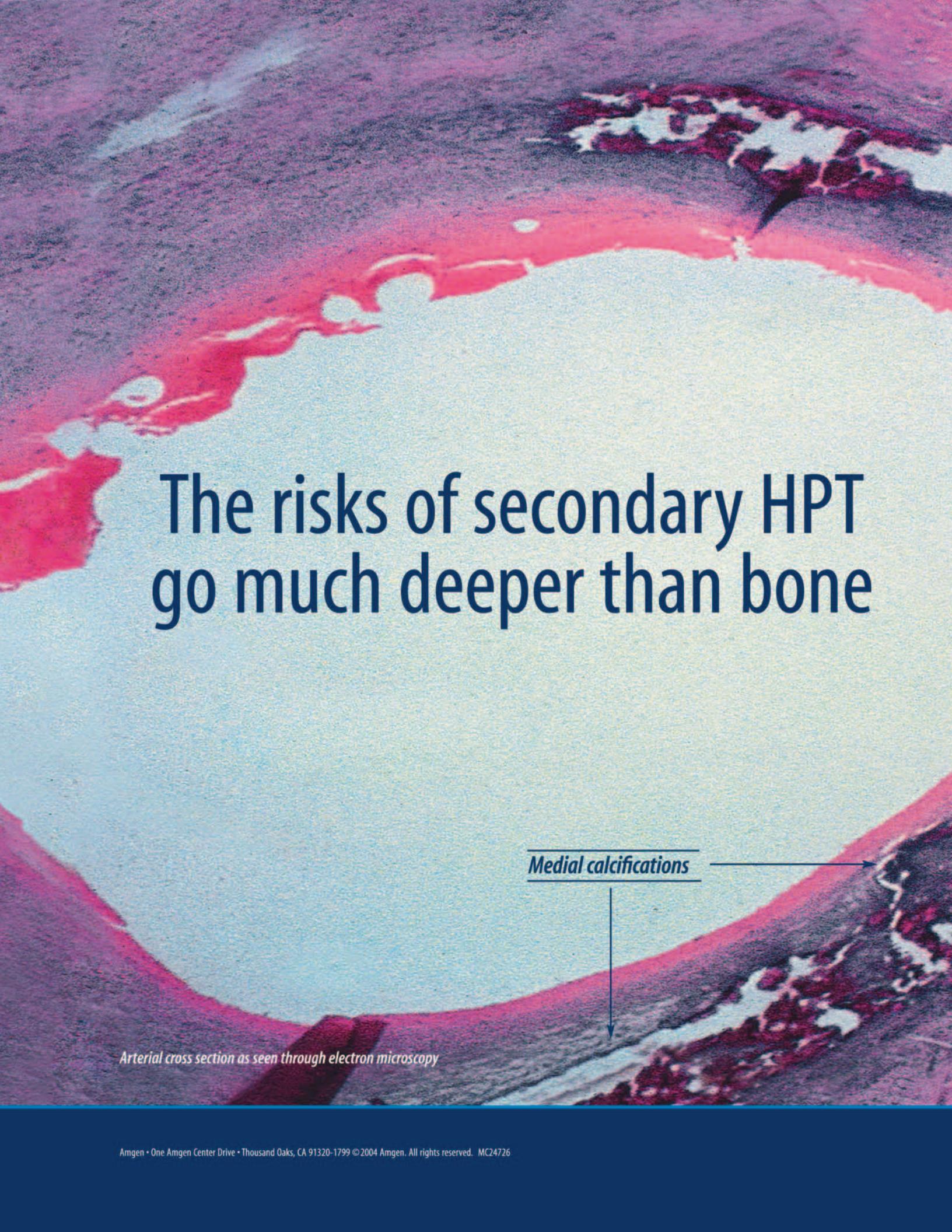
Table 2. Metabolic Alterations Associated with ARF (6, 7)

Carbohydrate	Hyperglycemia, insulin resistance, accelerated hepatic gluconeogenesis despite exogenous glucose infusions.
Lipid	Impaired lipolysis causing increased triglycerides and low-density lipoprotein and very low-density lipoprotein cholesterol; decreased total and high-density lipoprotein cholesterol.
Protein and Amino Acids	Acidosis and insulin resistance stimulating protein catabolism, skeletal muscle release of amino acids, and negative nitrogen balance; increased gluconeogenesis, ureagenesis, liver secretion of acute-phase proteins.

Continued on page 8

Table 3. Tests Used to Diagnose Acute Renal Failure (ARF) (3, 4, 5, 10, 11)

History and Physical Examination		
Chief complaint		
History		
General review of body systems, including pain, skin integrity, weight, hemodynamic parameters (including blood pressure, pulse)		
Determination of Urine Flow (Normal urine output: 1 to 1.5 L/d)		
Urine output		
Anuria	<100 mL/d	Associated with: Obstruction; bilateral renal, arterial, or venous occlusion; bilateral cortical necrosis; overwhelming acute tubular necrosis (ATN); severe acute glomerulonephritis
Oliguria	< 500 mL/d	Prerenal azotemia, intrinsic ARF, tubular necrosis, interstitial nephritis, glomerulonephritis, partial obstruction
Nonoliguria	>500 mL/d	An inability to excrete waste occurs with outputs of <500 mL/d
Polyuria excessive urine	3 L/d	Tubular necrosis, interstitial nephritis, partial obstruction Prerenal ARF if excessive urine output is the cause of prerenal state (rare), as in adrenal or mineralocorticoid deficiency states and excessive diuresis; more often indicative of polyuric acute tubular necrosis (2)
Laboratory Data		
<ul style="list-style-type: none"> - Blood urea nitrogen (BUN) - BUN/Creatinine ratio - Creatinine clearance - Bicarbonate - Hemoglobin - Calcium - Magnesium - Creatine kinase (CK-MM) (CK-MM is the dominant isoform in skeletal muscle, making it most sensitive to confirm rhabdomyolysis [10]). 		
Urinalysis		
Specific gravity (Normal, 1.010 to 1.025)		>1.020=prerenal failure 1.10 (isosthenuric)=intrinsic or postrenal failure
Proteinuria:		>3+ suggests intrinsic glomerular injury
Urine pH (Normal, 4.5 to 8.0)		More acidic in prerenal azotemia than other forms; acidic in rhabdomyolysis
Glycosuria without hyperglycemia:		Suggests proximal tubular injury
Blood:		Positive reaction on dipstick=acute glomerular or tubular injury, urinary tract infection, or nephrolithiasis. If blood is present on dipstick but not microscopically, a pigment nephropathy (hemoglobinuria or myoglobinuria) should be considered.
Urine sediment:		Unremarkable findings in pre- and postrenal azotemia except for occasional hyaline cysts Blood and crystals in postrenal due to stones Active sediment= intrinsic ARF Brown granular casts=rhabdomyolysis Microscopic evaluation can distinguish between glomerulonephritis, ATN, and tubulointerstitial nephritis
Urine-to-plasma creatinine ratio:		Eosinophils in large amounts in urine suggest drug-induced tubulointerstitial nephritis >40=prerenal; the urine concentration is higher than plasma concentration and kidney can concentrate urine 20 to 40=borderline <20=ARF
Urine Electrolytes		
Fractional excretion of sodium (less helpful in patients who are not oliguric or receiving diuretics):		<1% in oliguric patient suggests avid tubular sodium reclamation and prerenal azotemia with functional renal tubule >3% in oliguric patient suggests tubular injury and inability to absorb normal amounts of sodium >1% reflects tubular dysfunction and inability to absorb normal amounts of sodium Low value with diuretic use suggests volume depletion and prerenal ARF; elevated value may be result of ATN or diuresis
Urine sodium concentration (Normal range: varies widely)		<20 mEq/L associated with volume depletion, decreased blood flow to kidneys; suggests renal tubular mechanics intact, conserving sodium and able to concentrate urine >40 mEq/L suggests kidneys are losing the ability to concentrate as in some ARF, chronic renal failure, ATN. <10 mEq/L as an isolated measurement, often used as evidence of prerenal state; dependent on the state of water and sodium balance
Radiography		
Plain films: kidney size, stones, skeletal abnormalities of secondary hyperparathyroidism		
Intravenous pyelography: hazards make test of limited benefit		
Renal ultrasonography: sensitive and specific for hydronephrosis detection, obstruction		
Computed tomography can clarify cause of obstruction; not advantageous over ultrasonography		
Pulmonary Artery Catheterization		
Invasive hemodynamic monitoring that is an invaluable diagnostic adjunct in assessing and monitoring fluid balance (volume overload/hypovolemia)		
Renal Biopsy		
Recommended for ARF of unexplained cause or prolonged course. Used when other tests have not confirmed correct diagnosis and to decide on most appropriate therapy and prognosis.		

An electron micrograph showing a cross-section of an artery. The lumen is filled with a light blue, granular material. The tunica media is visible as a thick, pinkish-red layer containing many small, irregular white spots and larger, more organized structures labeled as 'Medial calcifications'. The tunica intima is the thin, pink layer at the top and bottom of the image.

The risks of secondary HPT
go much deeper than bone

Arterial cross section as seen through electron microscopy

Medial calcifications

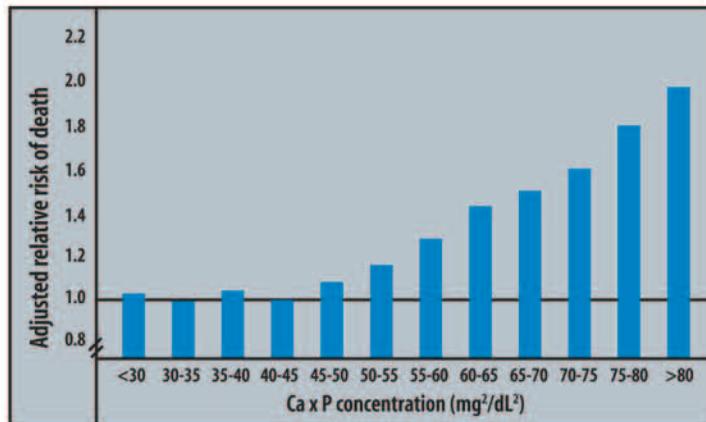
Failure to achieve NKF-K/DOQI™ bone metabolism goals* increases the risk of patient mortality¹⁻³

Uncontrolled secondary HPT can be harmful for your patients on dialysis. In addition to bone disease and parathyroid gland hyperplasia, adverse outcomes include soft-tissue and cardiovascular calcification, increased hospitalizations, cardiovascular events, and increased mortality risk.^{1,2,4} The majority of CKD patients on dialysis have metabolic parameters outside the K/DOQI™ goals despite use of traditional therapies.⁵

Only 17% of patients meet K/DOQI™ goals for both PTH and Ca x P, and only 8% meet all 4 goals^{5,6}

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New analyses show the adverse consequences of uncontrolled secondary HPT¹



Adapted from Block et al.¹

This significant increase in risk caused by secondary HPT can be controlled. Through optimal clinical management of bone metabolism parameters, more patients can achieve the 4 key K/DOQI™ goals and patient outcomes can potentially be improved.⁴

References: 1. Block GA, Klassen PS, Lazarus JM, Olshun N, Lowrie EG, Chertow GM. Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. *J Am Soc Nephrol.* 2004;15:2208-2218. 2. Block GA, Hulbert-Shearon TE, Levin NW, Port FK. Association of serum phosphorus and calcium x phosphate product with mortality risk in chronic hemodialysis patients: a national study. *Am J Kidney Dis.* 1998;31:607-617. 3. Ganesh SK, Stack AG, Levin NW, Hulbert-Shearon T, Port FK. Association of elevated serum PO₄, Ca x PO₄ product, and parathyroid hormone with cardiac mortality risk in chronic hemodialysis patients. *J Am Soc Nephrol.* 2001;12:2131-2138. 4. National Kidney Foundation. *K/DOQI Clinical Practice Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease.* *Am J Kidney Dis.* 2003;42(suppl 3):S1-S201. 5. Kim J, Pisani RL, Danese MD, Satayathum S, Klassen P, Young EW. Achievement of proposed NKF-K/DOQI bone metabolism and disease guidelines: results from the dialysis outcomes and practice patterns study (DOPPS). *J Am Soc Nephrol.* 2003;14:269A-270A. Abstract F-P0942. 6. Data on File. Amgen Inc, Thousand Oaks, Calif.

*NKF-K/DOQI™ Guidelines for Bone Metabolism and Disease in Chronic Kidney Disease.
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measurement of urine electrolytes, and radiography (Table 3). Invasive hemodynamic monitoring via a pulmonary artery catheter may aid in assessing and monitoring fluid balance of critically ill patients. A renal biopsy is recommended for ARF in which a cause cannot be determined or for ARF of prolonged duration.

Assessment of Nutritional Status

History

No single tool or measurement accurately reflects the nutritional status of a patient. Therefore, a comprehensive assessment should evaluate all available parameters, with ongoing monitoring of serial measurements. The use of a nutrition-focused physical assessment or Subjective Global Assessment is appropriate (12, 13).

The nutrition health history should examine nutrition-related problems, including intake, food intolerances, diet restrictions, allergies, the use of alternative therapies, and nutrient losses. This should be followed by a thorough physical examination and assessment of weight status that encompasses involuntary loss or gain and amount and percentage of weight change over an identified period of time. Depletion of muscle mass may be indicated by a prominent bony thoracic skeleton; hollow temples; clavicular prominence; or wasting of the scapula, quadriceps area, or interosseous muscle of the hand. Depletion of fat stores is evidenced by hollow eyes or decreased triceps skinfold. The gastrointestinal evaluation should include questions regarding the presence of nausea, vomiting, diarrhea, food intolerances, or malabsorption. Patients should be asked about their functional status, such as activities of daily living, and assessed for evidence of macro- and micronutrient deficiencies, such as skin lesions or hair loss, which can indicate zinc deficiency.

Biochemical Parameters

A thorough nutrition assessment should include evaluation of biochemical parameters and hepatic transport proteins, with careful consideration of factors that can alter results. It is generally accepted

that no single parameter can be used as an indication of nutritional status (14).

Albumin, which is easy and inexpensive to obtain and has a 21-day half-life, can serve as a valuable prognostic indicator for the risk of increased morbidity and mortality (14). However, shifting of albumin from the intravascular to extravascular space during stress, hydration, and intravenous albumin administration can limit the value of this measurement in the ICU. Prealbumin, which has a short half life (2 to 3 days), has been observed to have a more rapid response to therapy. Because prealbumin is degraded in the kidney, the values may be higher in ARF, but no specific guidelines have been set for a patient with ARF (2). Prealbumin values of less than 30mg/dL in chronic kidney disease patients on dialysis have been associated with increased mortality (8).

Both albumin and prealbumin are transport proteins. Metabolic insult and inflammation result in a release of inflammatory cytokines and interleukin-1, causing hepatic reprogramming of protein synthesis. This leads to increased production of acute-phase reactants (C-reactive protein, fibrinogen, ceruloplasmin, haptoglobin) and decreased transport protein (albumin, prealbumin) synthesis (14). C-reactive protein is a positive acute-phase protein involved in the immune response (2). Increases can occur within 4 to 6 hours of the onset of acute stress, and declines generally reflect resolution of the inflammatory response (15). The rapid decrease in serum protein levels and increase in C-reactive protein levels reflect severity of illness more so than nutritional status (2, 14).

Transferrin is responsible for iron absorption and transport, although it is strongly influenced by iron status (14). Low transferrin levels might indicate malnutrition, but a patient with iron deficiency anemia can have an elevated transferrin level, which can complicate interpretation of the transferrin concentration.

Nitrogen balance is a useful indicator of the degree of catabolism and guide to

nutrition support for critically ill patients with or without ARF. However, the validity of nitrogen balance is affected by severe nitrogen retention disorders (i.e., creatinine clearance <50 mL/min)(8). Nitrogen balance can be determined by calculating the urea nitrogen appearance (UNA) or the protein catabolic rate (PCR) in patients with ARF (8). UNA also can be used for patients receiving continuous renal replacement therapy (CRRT) including continuous ambulatory peritoneal dialysis (CAPD), continuous arteriovenous hemodialysis (CAVHD), and continuous arteriovenous hemofiltration (CAVH)(8).

Blood urea nitrogen (BUN) is the concentration of nitrogen (within urea) in the serum; such serum concentration depends on urea production (which occurs in the liver), tubular reabsorption, and glomerular filtration (11). Clinicians must consider factors other than filtration when interpreting changes in BUN (11). For example, BUN values may be elevated in the presence of internal hemorrhage, protein hypercatabolism, infection, excess protein provision, tetracycline, and steroids (11). On the other hand, BUN values can be low in patients who are malnourished, have profound liver damage due to the inability to synthesize urea, or when fluid overload initially dilutes BUN (11).

Creatinine, a byproduct of muscle catabolism, is a more sensitive indicator of kidney function that assesses urinary excretion. It is important to consider the baseline creatinine value and the patient's muscle mass. For example, a thin, debilitated person who has decreased muscle mass may not appear to have significantly elevated values in the presence of renal failure due to lower baseline values.

Creatinine concentrations may be increased by medications such as trimethoprim, cimetidine, and certain cephalosporins (11). Rhabdomyolysis can cause an elevated creatinine concentration related to muscle breakdown (10).

The normal range of BUN is 8 to 20mg/dL, and the normal value of creatinine is

Continued on page 9

0.5 to 1.5mg/dL (11). The BUN:creatinine ratio is usually 10:1 (5). In most cases of renal failure, both the BUN and serum creatinine increase; in stable ARF, the ratio is maintained at 10 to 15:1 (5). Certain renal abnormalities characterized by low tubular flow can cause the BUN to increase, and factors such as liver disease may lower BUN, which would alter the BUN:creatinine ratio (5).

Nutrition Support

Treatment goals for ARF are designed to address the systemic effects of the condition (Table 4). Nutrition support is an integral part of such treatment because the nutrition prescription can affect fluid, electrolyte, and mineral

status and the patient's nutritional status. Patients who have ARF and are at risk for nutrition disorders should be identified on admission to the hospital and their status and nutrition monitored throughout the hospital course. Patients who cannot or should not take oral foods for an extended time are candidates for nutrition support, with enteral nutrition preferred over parenteral nutrition (6).

Nutritional Requirements

Energy Needs

When available, the use of indirect calorimetry (IC) to measure oxygen consumption, carbon dioxide production, resting energy expenditure, and respiratory

quotient can help determine energy needs. Certain factors, such as mechanically ventilated patients requiring fractional inspired oxygen concentrations (FiO_2) greater than 60% and leaking chest or endotracheal tubes, may lead to inaccurate results (8). The energy needs of people who have ARF can be influenced by nutrition and metabolic status as well as by the degree of hypercatabolism (e.g., sepsis, infection, cause of renal failure, severity of underlying illness, comorbid conditions). When direct measurement is not available, calorie requirements can be met by providing 35 to 50kcal/kg ideal bodyweight, with the upper level reserved for patients who are severely catabolic and whose nitrogen balance does not improve at lower intakes (1,8). There is no appreciable increase for renal failure alone (1); the increase in energy expenditure is related more to the critical illness than to ARF (2). For patients who are receiving maintenance hemodialysis or chronic peritoneal dialysis, the target energy intakes are 35kcal/kg/d for those who are age 60 years and younger and 30 to 35kcal/kg/d for those older than age 60 years (15).

Protein

Factors to consider when determining protein allowances and restrictions include the loss of amino acids across the membrane with hemodialysis treatments (10 to 12g of amino acids are lost per treatment of high-efficiency hemodialysis (18), amino acid loss in CAVHD (up to 30g/d but usually <15g/day) (19), effectiveness and/or frequency of RRT (number of days per week and hours on treatment), and whether the patient completes fully scheduled dialysis treatments on RRT. Dialysis treatments may not achieve goals of fluid, electrolyte balance, and nitrogenous waste removal when treatments are shortened for reasons of hypotension, cardiac instability, or problems with patency of dialysis access.

Protein-controlled diets (0.6 to 0.8g/kg/d) traditionally have been used to reduce uremic complications, slow renal disease progression, and avoid or delay the need for

Table 4. Treatment Goals (16, 17)

<ul style="list-style-type: none"> • Eliminate cause of failure • Prevent further kidney damage • Achieve renal recovery • Support function of kidneys and other effected organ systems <ul style="list-style-type: none"> - Establish or maintain blood flow to kidneys for a prerenal condition - Treat intrinsic renal disease - Remove postrenal obstruction • Maintain fluid and electrolyte, acid-base, and mineral balance; regulate volume status <ul style="list-style-type: none"> - Observe physical signs of edema: extremities, sacral, eyelids, other; color and amount of urine - Monitor for signs of hyperkalemia, hypermagnesemia, hyponatremia, hyperphosphatemia - Avoid hypokalemia/magnesemia caused by aggressive diuretic therapy - Correct acidosis - Improve hemodynamic stability • Remove nitrogenous wastes <ul style="list-style-type: none"> - Monitor BUN, creatinine levels - Assess decision for start of renal replacement therapy based on: 1) presence of uremia, 2) significant renal failure with refractory hyperkalemia, 3) significant renal failure with refractory acidosis, 4) significant renal failure with refractory heart failure, 5) pericarditis • Manage anemia • Reassess use of nephrotoxic drugs • Maintain acceptable nutritional status through provision of adequate nutrition support <ul style="list-style-type: none"> - Prevent compromise to organ systems - Prevent wasting syndrome, loss of lean body mass, malnutrition - Provide substrates to meet needs of hypercatabolic state and comorbid conditions - Prevent deficiencies and/or toxicities - Facilitate use of gut as status permits • Protect and reserve vascular access for dialysis use; may place limits on intravenous access • Decrease ICU/hospital stay • Prevent mortality through aggressive treatment of conditions most related to mortality: sepsis, cardiac disease, or hemorrhage • Address the physical and emotional needs of patient/establish support systems
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dialysis (1). Protein restriction is indicated for patients who have advanced chronic renal insufficiency but are not undergoing dialysis if they are well monitored (18). If ARF is expected to resolve in a few days and dialysis will not be needed, a protein intake of 0.6 to 1g/kg/d is recommended (8). The patient's nutritional and metabolic status and renal diagnosis determine the exact dose (8). Patients with chronic renal failure (CRF) who are undergoing hemodialysis or peritoneal dialysis typically require 1.2 to 1.3g/kg/d (16, 17) or up to 1.5g/kg/d, respectively (15,18).

ARF patients who are severely malnourished and catabolic may require as much as 1.5 to 1.8g/kg/d protein; such levels of protein intake have been associated with increased protein catabolic rates, which is desirable (18).

Patients undergoing continuous hemofiltration therapy require a protein intake of at least 1.0g/kg/d; less than 1g/kg per day of protein has been associated with greater nitrogen deficits (18). Azotemia can be controlled while providing more than 1g/kg/d of protein (18). For patients on CRRT, the recommendation is 1.5 to 2.0g/d protein (8).

ARF patients should be given a balanced mixture of essential amino acids (EAA) and nonessential amino acids (NEAA) (18). Previous studies supported the use of small doses of EAA plus dextrose rather than dextrose alone (18). More recent investigations comparing EAA administration with administration of a balanced mix of EAA and NEAA showed no difference in mortality, nitrogen balance, or BUN (18). Additionally, when EAA formulations are used for longer than 2 to 3 weeks, hyperammonemia and metabolic encephalopathy can occur (7, 18). ARF patients require NEAA such as arginine, ornithine, and citrulline to enable detoxification of ammonia via the urea cycle (18).

Fat

The requirements for lipids are based on the goals of providing adequate nutrition while preventing essential fatty acid deficiency and overfeeding. Omega-6 fatty acid intake

must be monitored because high doses are associated with hypoxemia, bacteremia, and suppression of immune function (8). Serum triglyceride levels should be monitored before the initiation of parenteral nutrition and routinely through administration of intravenous lipids, as should serum glutamic oxaloacetic transaminase (SGOT), serum glutamic-pyruvic transaminase (SGPT), and alkaline phosphatase (8). Elevations in these measures suggest impaired hepatic clearance of the lipid load (8). Other lipid sources, such as propofol, which contributes a source of lipid calories, should be calculated in the formula. General recommendations are to provide 60% of total kcal as carbohydrate (not to exceed 5mg/kg/min) and 20% to 35% of total kcal as fat or 1g/kg/d in critical illness (8, 18).

Fluid

Each patient's fluid requirements depend on sodium concentration, fluid status, and renal function. Patients may present with anuria, oliguria, or polyuria. A general guideline for patients making urine is to replace losses (24-hr urine output + 500mL for insensible loss) (8). A weight loss of more than 1kg/d indicates volume loss or catabolism, which may alter the fluid allowance. It is important to assess weight gain for volume expansion because unintentional fluid gains also may place limits on the fluid allowance. When determining fluid needs, the clinician should consider use and success of RRT and comorbid fluid balance issues. In fluid overload states manifested by peripheral edema, hypoxemia due to pulmonary edema, or pleural effusion, intravenous fluid sources and parenteral nutrition substrates can be concentrated. Similarly, enteral products that provide up to 2kcal/mL are useful because they provide nutrient-dense substrates while limiting free water (8).

Micronutrients

Exact vitamin needs have not been established for patients who have ARF. In studies of ARF treated with dialysis and ARF associated with MODS, increased levels of ascorbate, carotene, and selenium were documented, with plasma levels of tocopherol being decreased only in MODS patients (2). The effects of vitamin

and mineral supplementation in amounts exceeding the dietary reference intakes during critical illness are not known (2). Additional research is needed before developing guidelines.

Water-soluble vitamin supplementation is indicated with RRT because of increased clearance (1, 8, 18). The need for vitamin B6 and folic acid also is increased due to losses (1, 8, 18). Riboflavin, biotin, niacin, and pantothenic acid do not appear to be cleared by hemodialysis, thereby obviating the need for added supplementation above the dietary reference intake (1). For dialysis patients who have wounds, a standard renal multivitamin should be administered enterally or a multivitamin infusion and multitrace element infusion administered parenterally (20). Oral vitamin preparations have been formulated to meet the needs of patients requiring dialysis and include: vitamin C (40 to 100mg), vitamin B1 (1.5mg), vitamin B2 (1.5 to 1.7mg), niacin (20mg), vitamin B6 (10 to 50mg), vitamin B12 (6mcg/1mg), folic acid (800mcg, 1mg, 5mg), pantothenic acid (5 to 10mg), and biotin (150 to 300mcg). Some preparations also contain iron and/or zinc.

The specific needs of vitamin A in ARF are not known, but because vitamin A levels are elevated with CRF, supplementation in ARF is not recommended for at least 2 weeks (8) or when RRT is not used. Excessive amounts can lead to toxicity, which would require withholding of vitamin A (1).

Supplementation of vitamin K may be indicated in ARF, especially when long-term antibiotic therapy that suppresses intestinal growth of bacteria that usually synthesize the vitamin, is prescribed (8). The use of coagulation therapy should be factored into decisions about whether to administer vitamin K and in determining the appropriate amount. The newer multivitamin infusions and lipids include vitamin K.

Vitamin C intake recommendations are 60 to 100mg/d (8) and should not exceed 200mg/d to prevent increased oxalate production, which can result in deposition

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of oxalate in the heart, kidney, and blood vessels (1). A dose of 10mg/d of pyridoxine hydrochloride (8.2mg/d pyridoxine) is recommended to avoid deficiency in patients with ARF (8).

Micronutrient recommendations for wound healing for patients with renal failure on dialysis in whom zinc levels are believed to be depleted or who have an increased risk of deficiency include 50mg zinc for 10 days (220mg ZnSO₄) (oral/enteral) and 10mg for 10 days (parenteral) (20). Excess zinc supplementation (>150mg/d) interferes with copper absorption (20). Gastrointestinal loss or a catabolic state such as injury or burns can increase requirements.

Because of diminished urinary clearance, aluminum provision can be toxic in patients with renal failure if aluminum-containing antacids or phosphate binders are used over long periods of time (21). Excess aluminum can lead to osteodystrophy, anemia, and encephalopathy with dementia (1,21). Patients at highest risk for aluminum toxicity are those who have uremia and those receiving long-term parenteral nutrition (21).

The need for iron supplementation must be based on overall patient status, potential losses, sepsis, and the use of blood transfusions. Intake, dialysis-induced losses, frequent laboratory testing, impaired intestinal iron absorption, and occult gastrointestinal bleeding are likely to cause iron deficiency anemia (8). Added supplementation is needed when erythropoietin is administered (8). Certain laboratory parameters must be evaluated to determine if supplemental iron is needed and the amount required (8). Iron losses can be monitored by assessing serum iron and ferritin concentrations, percent transferrin saturation, and total iron binding capacity (8).

Carnitine

Carnitine is an amine compound required for the transport of long-chain fatty acids into the mitochondria for oxidation and ultimate production of ATP by oxidative phosphorylation (2). Carnitine is lost with dialysis, but the amount lost is unknown,

and supplementation of L-carnitine is controversial. Kidney Disease Outcomes Quality Initiative guidelines have concluded that evidence is insufficient for routine supplementation (15). In July 2002, the Centers for Medicare & Medicaid Services issued a national coverage determination for the use of L-carnitine for end-stage renal disease that likely will increase use (22, 23). Patients should be monitored for symptoms related to carnitine deficiency, which include malaise, asthenia, general weakness or fatigue, skeletal muscle cramps, and decreased exercise capacity or low peak oxygen consumption after dialysis (15). Carnitine levels have been noted to be normal with ARF (2).

Electrolytes

Electrolyte supplementation should be individualized. Suggested doses of sodium are 1.1 to 3.3g/d orally, 500mg (22mEq/kg) enterally, and 1 to 2mEq/kg parenterally. Suggested doses of potassium are 780 to 2,000mg/d orally, 2g (51mEq/kg) enterally, and 1 to 2mEq/kg parenterally. The enteral and parenteral doses of both electrolytes are standard and not specific to ARF (8, 24). CRRT causes significant losses of magnesium, calcium, phosphorus, and potassium that require careful monitoring and repletion (25).

The Nutrition Prescription

Enteral

Several tube feeding formula options are available for patients with ARF. Standard 2-kcal/mL formulas can be used for patients requiring fluid restriction. The risk for refeeding syndrome should be assessed, keeping in mind that renal formulas tend to be low in potassium, sodium, and phosphorus compared with standard nonrenal products. Dialysis patients with persistent hyperkalemia or hyperphosphatemia may benefit from renal enteral products.

Parenteral

Concentrated substrates that include 20% to 30% lipid emulsion, 50% to 70% dextrose, and 10% to 15% amino acids generally are administered via a central access to avoid

excessive fluid administration with fluid limits. Electrolytes (sodium and potassium) may be added as acetate salts or bicarbonate in the presence of metabolic acidosis. The dialysis bath formula (known as dialysate) has varying ion and mineral compositions that affect serum electrolyte values and, therefore, must be considered when developing the parenteral formula.

Conclusion

Patients with ARF are at nutritional risk and require thorough nutrition assessments to formulate appropriate nutrition prescriptions. The decision to initiate nutrition support should be based on the patient's overall condition, nutritional status, and laboratory results. ARF patients requiring nutrition support must be monitored closely to avoid potential complications.

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Nominations Wanted for American Dietetic Association Renal Practice Group

The Nominating Committee is accepting nominations for enthusiastic and dedicated individuals for the 2006 ADA/RPG Executive Committee. The positions open are for Chair-Elect, Secretary, and Nominating Committee.

Please refer to RPG's website at www.renalnutrition.org after mid-August for descriptions of these positions and the nominations form. Deadline for submitting nominations is October 1. Please contact Susan Knapp, Nominating Committee Chair, with questions regarding nominations at sknapp@davita.com or 918-392-0290 Ext. 19.

Advances in Practice

Therapeutic Benefits of Fish Oils for Patients with Chronic Kidney Disease

By Philippa Norton Feiertag, MEd, RD, LD. Philippa is a clinical analyst/renal nutrition specialist with Clinical Computing, Inc. in Cincinnati, Ohio. She can be reached at feier@fusenet.com.

Interest in the therapeutic effects of fish oils began when investigations into the health status of Greenland Eskimos revealed low rates of heart disease, despite high fat intake from fish, seals and whales. Subsequent clinical intervention studies confirmed the ability of fish oils to decrease heart disease by preventing cardiac arrhythmias, inhibiting atherosclerosis, and through their antithrombotic and hypolipidemic properties (1). Additional roles have been identified for fish oils in the management of autoimmune diseases and some types of cancer (2-4).

Recent studies indicate that fish oils might have unique benefits for patients with chronic kidney disease (CKD). Treatment with daily doses of fish oils over a 2-year period slowed the progression of renal disease in patients with Immunoglobulin A (IgA) nephropathy, the most commonly occurring form of primary glomerulonephritis (5,6). Fish may also be a beneficial protein source for patients with CKD undergoing maintenance dialysis therapy. Patients who reported eating fish at least once in a three-day period had a 50% lower mortality rate during a 3-year follow-up period than those who did not report eating fish (7).

The physiological effects of fish oils are attributed to the long-chain, polyunsaturated omega-3 fatty acids they contain. Omega-3 fatty acids are essential fatty acids and important structural components of phospholipid membranes in animal cells (1). The fish and marine mammals consumed by Eskimos contain the omega-3 fatty acids docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA). Green plants are a source of the omega-3 fatty acid alpha-linolenic acid (ALA), from which DHA and EPA may be synthesized.

Today, omega-3 fatty acids are available as fish oil supplements, including Coromega® (European Reference Botanical Laboratories, Carlsbad, CA) and Omacor® (Pronova Biocare, Oslo, Norway) (4,5). In addition, omega-3 enriched spreads and oils are marketed as part of the Smart Balance® product line (GFA Brands Inc., Cresskill, NJ) (8). Renal dietetics professionals may need to provide information on these products as their patients become aware of the potential health benefits of fish oils. This column will review the applications of fish oil therapy to improve health outcomes in patients with CKD.

1. Treatment of IgA nephropathy

IgA nephropathy is an immune complex-mediated glomerulonephritis characterized by deposition of IgA in the kidneys and leading to CKD in 20-40% of those affected (9). While there is no cure for this disease, corticosteroids, angiotensin-converting enzyme (ACE) inhibitors and omega-3 fatty acids have all been investigated as strategies for slowing progression of IgA nephropathy.

Clinical trials with omega-3 fatty acids have shown mixed results overall, but the largest trial provided clear evidence that supplementation with fish oil was accompanied by decreased proteinuria and improved glomerular filtration rate (GFR) in patients with severe IgA nephropathy (10). One hundred and six patients received daily doses of DHA and EPA in the form of four soft gel capsules for 2 years. Follow-up observations averaging 6.5 years indicated that disease progression was significantly delayed.

In an investigation into optimum omega-3 fatty acid dosing to retard IgA nephropathy progression, 73 patients were randomly assigned to receive either high- (2.94g DHA and 3.76g EPA) or low- (1.47g DHA and 1.88g EPA) dose treatments (11). Serum creatinine levels were used to track disease progression. Low- and high-dose omega-3

fatty acid therapies were equally effective in slowing the rate of renal function loss.

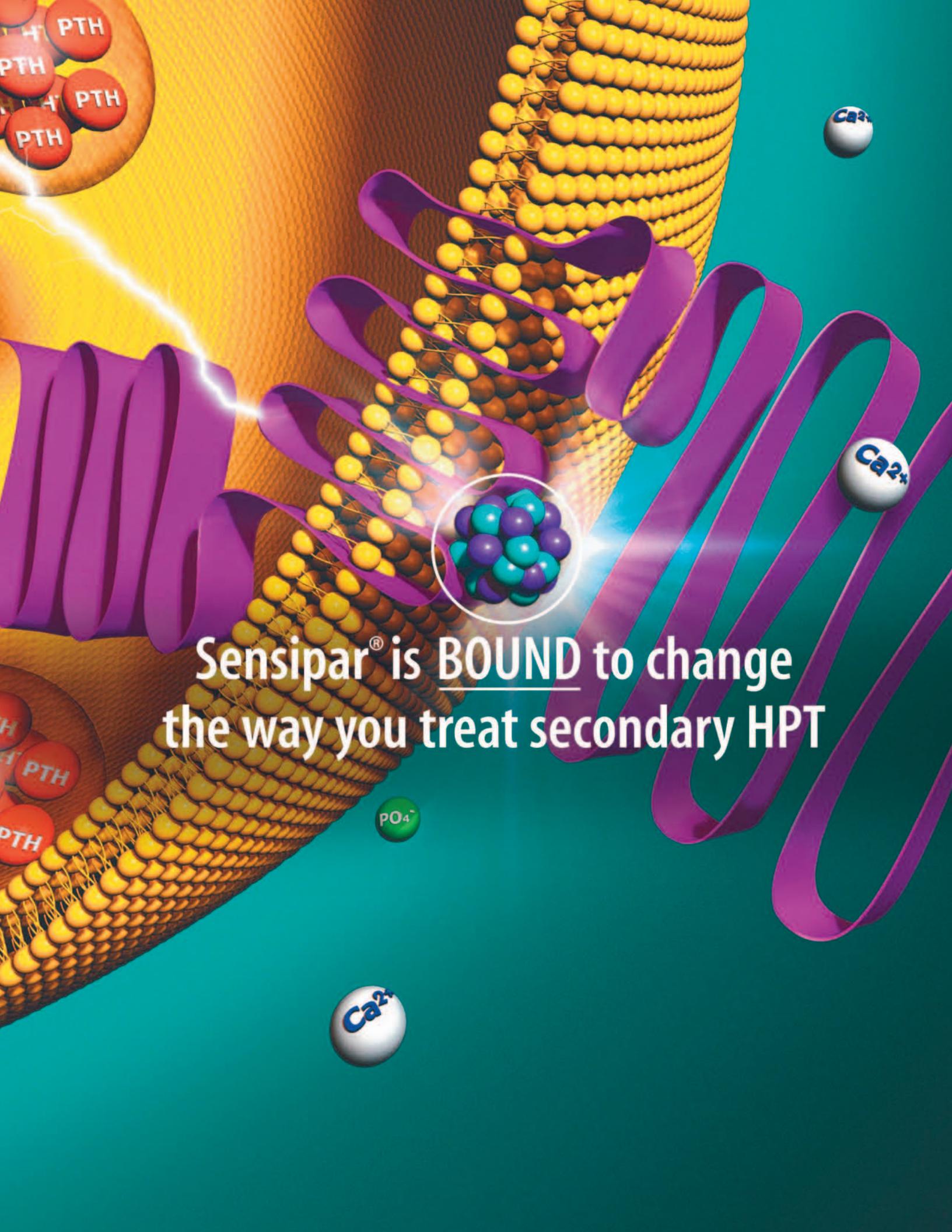
2. Lipid-lowering effects

Patients with IgA nephropathy show a significant reduction in serum triglycerides after 6 months of omega-3 fatty acid therapy (12). In addition, serum cholesterol decreases and high density lipoprotein (HDL)-cholesterol increases. Studies of patients undergoing maintenance hemodialysis (HD) also show an association between fish intake and decreased cardiac symptoms. This may occur because omega-3 fatty acids in fish oils decrease serum triglyceride levels as well as ratios of low density lipoprotein (LDL) to HDL-cholesterol, total cholesterol to HDL-cholesterol and triglycerides to HDL-cholesterol (13-15).

In a randomized, placebo-controlled trial, lipid-lowering effects of corn, fish and sesame oils were studied in 60 HD patients assigned to four different study groups (13). Patients in each group received daily doses of corn oil (4.5g), fish oil (1.5g), sesame oil (4.5g) or placebo. Serum triglycerides, total cholesterol, LDL-cholesterol and HDL-cholesterol were measured before and after 2 months of therapy. After supplementary corn and fish oil, HDL-cholesterol increased and LDL-cholesterol decreased significantly. Serum triglycerides also decreased after fish oil therapy. Significant decreases occurred in the ratios of triglyceride to HDL-cholesterol, total cholesterol to HDL-cholesterol and LDL-cholesterol to HDL-cholesterol after fish and corn oil therapy. Sesame oil had no significant effect on lipid profile.

A more recent study showed similar favorable effects of omega-3 fatty acids on lipid profiles in patients with serum creatinine levels between 150-400 micromol/L (1.7-4.52 mg/dL)^a (16). The 64 patients in this study were randomly

^aTo convert micromol/L creatinine to mg/dL, multiply micromol/L by 0.0113. To convert mg/dL creatinine to micromol/L, multiply mg/dL by 88.4. Creatinine of 150 micromol/L = 1.70 mg/dL



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Sensipar® is indicated for the treatment of secondary hyperparathyroidism (HPT) in patients with chronic kidney disease on dialysis. Sensipar® lowers serum calcium. Significant reductions in calcium may lower the threshold for seizures. Secondary HPT patients, particularly those with a history of a seizure disorder, should be carefully monitored for the occurrence of low serum calcium or symptoms of hypocalcemia. The most commonly reported side effects were nausea and vomiting.

Please see adjacent brief summary of prescribing information.

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Brief Summary

See package insert for full prescribing information

SENSIPAR® (cinacalcet HCl) Tablets

INDICATIONS AND USAGE

Sensipar® is indicated for the treatment of secondary hyperparathyroidism in patients with Chronic Kidney Disease on dialysis.

CONTRAINDICATIONS

Sensipar® is contraindicated in patients with hypersensitivity to any component(s) of this product.

WARNINGS

Seizures: In three clinical studies of CKD patients on dialysis, 5% of the patients in both the Sensipar® and placebo groups reported a history of seizure disorder at baseline. During the trials, seizures (primarily generalized or tonic-clonic) were observed in 1.4% (9/656) of Sensipar®-treated patients and 0.4% (2/470) of placebo-treated patients. Five of the nine Sensipar®-treated patients had a history of a seizure disorder and two were receiving anti-seizure medication at the time of their seizure. Both placebo-treated patients had a history of seizure disorder and were receiving anti-seizure medication at the time of their seizure. While the basis for the reported difference in seizure rate is not clear, the threshold for seizures is lowered by significant reductions in serum calcium levels. Therefore, serum calcium levels should be closely monitored in patients receiving Sensipar®, particularly in patients with a history of a seizure disorder (see PRECAUTIONS, Hypocalcemia).

PRECAUTIONS

General

Hypocalcemia: Sensipar® lowers serum calcium, and therefore patients should be carefully monitored for the occurrence of hypocalcemia. Potential manifestations of hypocalcemia include paresthesias, myalgias, cramping, tetany, and convulsions. Sensipar® treatment should not be initiated if serum calcium is less than the lower limit of the normal range (8.4 mg/dL). Serum calcium should be measured within 1 week after initiation or dose adjustment of Sensipar®. Once the maintenance dose has been established, serum calcium should be measured approximately monthly (see DOSAGE AND ADMINISTRATION). If serum calcium falls below 8.4 mg/dL but remains above 7.5 mg/dL, or if symptoms of hypocalcemia occur, calcium-containing phosphate binders and/or vitamin D sterols can be used to raise serum calcium. If serum calcium falls below 7.5 mg/dL, or if symptoms of hypocalcemia persist and the dose of vitamin D cannot be increased, withhold administration of Sensipar® until serum calcium levels reach 8.0 mg/dL, and/or symptoms of hypocalcemia have resolved. Treatment should be re-initiated using the next lowest dose of Sensipar® (see DOSAGE AND ADMINISTRATION). In the 26-week studies of patients with CKD on dialysis, 66% of patients receiving Sensipar® compared with 25% of patients receiving placebo developed at least one serum calcium value < 8.4 mg/dL. Less than 1% of patients in each group permanently discontinued study drug due to hypocalcemia. In CKD patients with secondary HPT not on dialysis, the long-term safety and efficacy of Sensipar® have not been established. Exploratory investigation indicates that CKD patients not on dialysis have an increased risk for hypocalcemia compared to CKD patients on dialysis, which may be due to lower baseline calcium levels. In a small, short-term study in which the median dose of cinacalcet was 30 mg at the completion of the study, 74% of cinacalcet treated patients experienced at least one serum calcium value < 8.4 mg/dL. **Adynamic Bone Disease:** Adynamic bone disease may develop if iPTH levels are suppressed below 100 pg/mL when assessed using the standard Nichols IRMA. One clinical study evaluated bone histomorphometry in patients treated with Sensipar® for one year. Three patients with mild hyperparathyroid bone disease at the beginning of the study developed adynamic bone disease during treatment with Sensipar®. Two of these patients had iPTH levels below 100 pg/mL at multiple time points during the study. In the three 6-month, phase 3 studies conducted in CKD patients on dialysis, 11% of patients treated with Sensipar® had mean iPTH values below 100 pg/mL during the efficacy-assessment phase. If iPTH levels decrease below the NKF-K/DOQI recommended target range (150-300 pg/mL) in patients treated with Sensipar®, the dose of Sensipar® and/or vitamin D sterols should be reduced or therapy discontinued. **Hepatic Insufficiency:** Cinacalcet exposure as assessed by AUC(0-inf) in patients with moderate and severe hepatic impairment (as indicated by the Child-Pugh method) were 2.4 and 4.2 times higher, respectively, than that in normals. Patients with moderate and severe hepatic impairment should be monitored throughout treatment with Sensipar® (see CLINICAL PHARMACOLOGY, Pharmacokinetics and DOSAGE AND ADMINISTRATION). **Information for Patients:** It is recommended that Sensipar® be taken with food or shortly after a meal. Tablets should be taken whole and should not be divided. **Laboratory Tests:** **Patients with CKD on Dialysis with Secondary Hyperparathyroidism:** Serum calcium and serum phosphorus should be measured within 1 week and iPTH should be measured 1 to 4 weeks after initiation or dose adjustment of Sensipar®. Once the maintenance dose has been established, serum calcium and serum phosphorus should be measured approximately monthly, and PTH every 1 to 3 months (see DOSAGE AND ADMINISTRATION). All iPTH measurements during the Sensipar® trials were obtained using the Nichols IRMA. In patients with end-stage renal disease, testosterone levels are often below the normal range. In a placebo-controlled trial in patients with CKD on dialysis, there were reductions in total and free testosterone in male patients following six months of treatment with Sensipar®. Levels of total testosterone decreased by a median of 15.8% in the Sensipar®-treated patients and by 0.6% in the placebo-treated patients. Levels of free testosterone decreased by a median of 31.3% in the Sensipar®-treated patients and by 16.3% in the placebo-treated patients. The clinical significance of these reductions in serum testosterone is unknown. **Drug Interactions and/or Drug/Laboratory Test Interactions:** See CLINICAL PHARMACOLOGY, Pharmacokinetics and Drug Interactions. Effect of Sensipar® on other drugs: Drugs metabolized by cytochrome P450 2D6 (CYP2D6): Sensipar® is a strong in vitro inhibitor of CYP2D6. Therefore, dose adjustments of concomitant medications that are predominantly metabolized by CYP2D6 and have a narrow therapeutic index (e.g., flecaïnide, vinblastine, thioridazine and most tricyclic antidepressants) may be required. Amitriptyline: Concurrent administration of 25 mg or 100 mg cinacalcet with 50 mg amitriptyline increased amitriptyline exposure and nortriptyline (active metabolite) exposure by approximately 20% in CYP2D6 extensive metabolizers. Effect of other drugs on Sensipar®: Sensipar® is metabolized by multiple cytochrome P450 enzymes, primarily CYP3A4, CYP2D6, and CYP1A2. Ketoconazole: Sensipar® is metabolized in part by CYP3A4. Co-administration of ketoconazole, a strong inhibitor of CYP3A4, increased cinacalcet exposure following a single 90 mg dose of Sensipar® by 2.3 fold. Dose adjustment of Sensipar® may be required and PTH and serum calcium concentrations should be closely monitored if a patient initiates or discontinues therapy with a strong CYP3A4 inhibitor (e.g., ketoconazole, erythromycin, itraconazole; see DOSAGE AND ADMINISTRATION). **Carcinogenesis, Mutagenesis, and Impairment of Fertility** **Carcinogenicity:** Standard lifetime dietary carcinogenicity bioassays were conducted in mice and rats. Mice were given dietary doses of 15, 50, 125 mg/kg/day in males and 30, 70, 200 mg/kg/day in females (exposures up to 2 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). Rats were given dietary doses of 5, 15, 35 mg/kg/day in males and 5, 20, 35 mg/kg/day in females (exposures up to 2 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). No increased incidence of tumors was observed following treatment with cinacalcet. **Mutagenicity:** Cinacalcet was not genotoxic in the Ames bacterial mutagenicity assay or in the Chinese Hamster Ovary (CHO) cell HGPRT forward mutation assay and CHO cell chromosomal aberration assay, with and without metabolic activation or in the *in vivo* mouse micronucleus assay. **Impairment of fertility:** Female rats were given oral gavage doses of 5, 25, 75 mg/kg/day beginning 2 weeks before mating and continuing through gestation day 7. Male rats were given oral doses 4 weeks prior to mating, during mating (3 weeks) and 2 weeks post-mating. No effects were observed in male or female fertility at 5 and 25 mg/kg/day (exposures up to 3 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). At 75 mg/kg/day, there were slight adverse effects (slight decreases in body weight and food consumption) in males and females. **Pregnancy Category C:** In pregnant female rats given oral gavage doses of 2, 25, 50 mg/kg/day during gestation no teratogenicity was observed at doses up to 50 mg/kg/day (exposure 4 times those resulting with a human oral dose of 180 mg/day based on AUC comparison). Decreased fetal body weights were observed at all doses (less than 1 to 4 times a human oral dose of 180 mg/day based on AUC comparison) in conjunction with maternal toxicity (decreased food consumption and body weight gain). In pregnant female rabbits given oral

gavage doses of 2, 12, 25 mg/kg/day during gestation no adverse fetal effects were observed (exposures less than with a human oral dose of 180 mg/day based on AUC comparisons). Reductions in maternal food consumption and body weight gain were seen at doses of 12 and 25 mg/kg/day. In pregnant rats given oral gavage doses of 5, 15, 25 mg/kg/day during gestation through lactation no adverse fetal or pup (post-weaning) effects were observed at 5 mg/kg/day (exposures less than with a human therapeutic dose of 180 mg/day based on AUC comparisons). Higher doses of 15 and 25 mg/kg/day (exposures 2-3 times a human oral dose of 180 mg/day based on AUC comparisons) were accompanied by maternal signs of hypocalcemia (periparturient mortality and early postnatal pup loss), and reductions in postnatal maternal and pup body-weight gain. Sensipar® has been shown to cross the placental barrier in rabbits. There are no adequate and well-controlled studies in pregnant women. Sensipar® should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. **Lactating Women:** Studies in rats have shown that Sensipar® is excreted in the milk with a high milk-to-plasma ratio. It is not known whether this drug is excreted in human milk and because of the potential for clinically significant adverse reactions in infants from Sensipar®, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the lactating woman. **Pediatric Use:** The safety and efficacy of Sensipar® in pediatric patients have not been established. **Geriatric Use:** Of the 1136 patients enrolled in the Sensipar® phase 3 clinical program, 26% were ≥ 65 years old, and 9% were ≥ 75 years old. No differences in the safety and efficacy of Sensipar® were observed in patients greater or less than 65 years of age (see DOSAGE AND ADMINISTRATION, Geriatric Patients).

ADVERSE EVENTS

Secondary Hyperparathyroidism in Patients with Chronic Kidney Disease on Dialysis: In 3 double-blind placebo-controlled clinical trials, 1126 CKD patients on dialysis received study drug (656 Sensipar®, 470 placebo) for up to 6 months. The most frequently reported adverse events (incidence of at least 5% in the Sensipar® group and greater than placebo) are provided in Table 1. The most frequently reported events in the Sensipar® group were nausea and vomiting.

Table 1. Adverse Event Incidence (≥ 5%) in Patients On Dialysis

Event*	Placebo (n = 470) (%)	Sensipar® (n = 656) (%)
Nausea	19	31
Vomiting	15	27
Diarrhea	20	21
Myalgia	14	15
Dizziness	8	10
Hypertension	5	7
Asthenia	4	7
Anorexia	4	6
Pain Chest, Non-Cardiac	4	6
Access Infection	4	5

* Included events that were reported at a greater incidence in the Sensipar® group than in the placebo group.

The incidence of serious adverse events (29% vs. 31%) was similar in the Sensipar® and placebo groups, respectively. **12-Month Experience with Sensipar®:** Two hundred and sixty-six patients from 2 phase 3 studies continued to receive Sensipar® or placebo treatment in a 6-month double-blind extension study (12-month total treatment duration). The incidence and nature of adverse events in this study were similar in the two treatment groups, and comparable to those observed in the phase 3 studies. **Parathyroid Carcinoma:** The most frequent adverse events in this patient group were nausea and vomiting. **Laboratory Values:** Serum calcium levels should be closely monitored in patients receiving Sensipar® (see PRECAUTIONS and DOSAGE AND ADMINISTRATION).

OVERDOSAGE

Doses titrated up to 300 mg once daily have been safely administered to patients on dialysis. Overdosage of Sensipar® may lead to hypocalcemia. In the event of overdosage, patients should be monitored for signs and symptoms of hypocalcemia and appropriate measures taken to correct serum calcium levels (see PRECAUTIONS). Since Sensipar® is highly protein bound, hemodialysis is not an effective treatment for overdosage of Sensipar®.

DOSAGE AND ADMINISTRATION

Sensipar® tablets should be taken whole and should not be divided. Sensipar® should be taken with food or shortly after a meal. Dosage must be individualized. **Secondary Hyperparathyroidism in Patients with Chronic Kidney Disease on Dialysis:** The recommended starting oral dose of Sensipar® is 30 mg once daily. Serum calcium and serum phosphorus should be measured within 1 week and iPTH should be measured 1 to 4 weeks after initiation or dose adjustment of Sensipar®. Sensipar® should be titrated no more frequently than every 2 to 4 weeks through sequential doses of 60, 90, 120, and 180 mg once daily to target iPTH consistent with the NKF-K/DOQI recommendation for CKD patients on dialysis of 150-300 pg/mL. Sensipar® can be used alone or in combination with vitamin D sterols and/or phosphate binders. During dose titration, serum calcium levels should be monitored frequently and if levels decrease below the normal range, appropriate steps should be taken to increase serum calcium levels, such as by providing supplemental calcium, initiating or increasing the dose of calcium-based phosphate binder, initiating or increasing the dose of vitamin D sterols, or temporarily withholding treatment with Sensipar® (see PRECAUTIONS). **Special Populations: Geriatric patients:** Age does not alter the pharmacokinetics of Sensipar®; no dosage adjustment is required for geriatric patients. Patients with renal impairment: Renal impairment does not alter the pharmacokinetics of Sensipar®; no dosage adjustment is necessary for renal impairment. **Patients with hepatic impairment:** Cinacalcet exposures, as assessed by AUC(0-inf), in patients with moderate and severe hepatic impairment (as indicated by the Child-Pugh method) were 2.4 and 4.2 times higher, respectively, than in normals. In patients with moderate and severe hepatic impairment, PTH and serum calcium concentrations should be closely monitored throughout treatment with Sensipar® (see CLINICAL PHARMACOLOGY, Pharmacokinetics and PRECAUTIONS). **Drug Interactions:** Sensipar® is metabolized in part by the enzyme CYP3A4. Co-administration of ketoconazole, a strong inhibitor of CYP3A4, caused an approximate 2-fold increase in cinacalcet exposure. Dose adjustment of Sensipar® may be required and PTH and serum calcium concentrations should be closely monitored if a patient initiates or discontinues therapy with a strong CYP3A4 inhibitor (e.g., ketoconazole, erythromycin, itraconazole; see CLINICAL PHARMACOLOGY, Pharmacokinetics and PRECAUTIONS).

Storage: Store at 25°C (77°F); excursions permitted to 15-30°C (59-86°F). [See USP controlled room temperature]. **Rx Only:** This product, or its use, may be covered by one or more US Patents including US Patent Nos. 6313146, 6211244, 6031003 and 6011068, in addition to others, including patents pending.

References: 1. National Kidney Foundation: K/DOQI clinical practice guidelines: bone metabolism and disease in chronic kidney disease. American Journal of Kidney Disease 42:S1-S201, 2003



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assigned to daily treatment with 2.4g of omega-3 fatty acids or control treatment with olive oil for 8 weeks. Researchers measured fasting lipid and lipoprotein levels in the subjects before and after supplementation. In the group receiving omega-3 fatty acids, there was a significant 8% increase in HDL-cholesterol and 21% decrease in serum triglyceride levels. The hypolipidemic effects of omega-3 fatty acids may therefore help to decrease the incidence of atherosclerosis in patients with CKD.

3. Reduction in hemodialysis graft thrombosis

The synthetic polytetrafluoroethylene (PTFE) graft is the most commonly placed hemodialysis access in the United States (17). However, graft thrombosis is a serious problem and several drugs have been investigated to improve graft patency including heparin, aspirin, calcium channel blockers, ACE inhibitors and warfarin (18,19). Inconsistent clinical results from these drug trials and side effects, including gastrointestinal hemorrhage, have prompted a search for novel strategies to prevent graft thrombosis.

Recently, a double-blind, randomized study was conducted to test the hypothesis that fish oil supplements would decrease the incidence of thrombosis in newly constructed PTFE grafts (17). Twenty-four patients received a daily regimen of either four Ig capsules of fish oil concentrate or four Ig capsules of corn oil. All patients were recruited from the same outpatient dialysis program, and all grafts were placed by the same surgical team. Treatment was initiated within 2 weeks after graft placement and patients were monitored for 1 year or until thrombosis occurred. Only two patients developed graft thrombosis in the group treated with fish oil compared with nine in the control group, and the graft patency rate at the end of the study was 75.6% in patients receiving fish oil supplement versus 14.9% in controls. Thus, findings from this study support the use of fish oils to decrease the incidence of graft thrombosis.

4. Alleviation of pruritis

Up to 86% of patients undergoing maintenance HD therapy experience a poorly localized itching sensation called pruritis (20). Pruritis has been attributed to uremic toxins, calcium-phosphorus imbalance, hyperparathyroidism, changes in vitamin A and magnesium homeostasis, and accumulation of the inflammatory eicosanoid leukotriene B4 in the skin.

Studies in patients with psoriasis have shown significant reduction in skin inflammation and itching following supplementation with fish oil capsules for 8 weeks (21). It is thought that omega-3 fatty acids may exert their beneficial effects by competing for lipoxygenase enzymes, which metabolize arachidonic acid in the skin to inflammatory eicosanoids, including leukotriene B4. Consequently, the effects of essential fatty acids and their metabolites have been investigated in maintenance dialysis patients with pruritis (15,20).

In a double-blind study, 25 HD patients with a history of pruritis were randomly assigned to receive daily supplements of fish oil, olive oil or safflower oil (15). After 8 weeks of treatment, patients who received fish oil had higher levels of DHA and EPA, a greater decrease in arachidonic acid and improvement in symptoms of pruritis.

In a more recent study, 35 maintenance HD patients with pruritis symptoms were randomized to receive six capsules daily of either fish oil or safflower oil for 16 weeks (20). A 3-day food record obtained from each patient before supplementation was analyzed for nutrient content, and patients were instructed to maintain the same eating pattern throughout the study. In addition, symptoms of pruritis were evaluated at baseline and after supplementation by means of a subjective questionnaire. After supplementation, both groups of patients showed nonsignificant reduction in leukotriene B4. However, patients who received fish oil had significantly higher levels of omega-3 fatty acids and a greater, though nonsignificant, decrease in pruritis score compared with patients who received safflower oil.

Summary

Patients with CKD may benefit from fish oil supplementation to delay the progression of renal disease, improve their lipid profile, decrease the incidence of graft thrombosis and relieve pruritis. The ability of fish oils to reduce erythropoietin (EPO) requirements and produce modest increase in serum albumin levels has also been documented (7, 22).

Use of fish oil supplements by patients with CKD appears to carry a low risk of side-effects (22). Taking fish oil supplements with meals reduces the likelihood of gastrointestinal distress. Heparin doses given during HD treatments may also need to be adjusted to prevent prolonged bleeding when patients are using fish oil supplements.

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Renal Nutrition Forum - CPE Questions

Nutrition Management of the Patient with Acute Renal Failure

by Marcia Kalista-Richards, MPH, RD, CNSD, Robert Pursell, MD, and Robert Gayner, MD.

This activity is approved for 2 CPEU, Level 2, by the Commission on Dietetic Registration (CDR) for registered dietitians and dietetic technicians, registered who are members of the Renal Practice Group. Valid through May 31, 2006. After reading the continuing professional education article, please answer the following questions by indicating your responses on the CPE answer sheet (see insert). Please be sure to submit your registration number and write legibly. Do not submit this CPE Questions page. Upon mailing the answer sheet to the assistant editor, you may fill out the certificate of completion on page 21, retain it in your portfolio, and record the activity on your Step Activity Log. Members will not receive mailed certificates of completion. Answers to the continuing professional education questions can be found on page 21.

Multiple Choice

1. What is the approximate mortality rate in acute renal failure?
 - a. 25%
 - b. 10%
 - c. 33%
 - d. 50%
 - e. 65%
2. What is the usual situation regarding urine output in ARF?
 - a. polyuria
 - b. normal urine output
 - c. oliguria
 - d. anuria
 - e. any of the above
3. Which diagnosis/treatment carries the highest rate of ARF?
 - a. rhabdomyolysis
 - b. cancer with chemotherapy
 - c. open heart surgery
 - d. severe burns
 - e. aminoglycoside therapy
4. Which of the following laboratory values for blood is the most sensitive and generally reliable indicator of kidney function?
 - a. blood urea nitrogen
 - b. serum creatinine
 - c. serum sodium
 - d. C-reactive protein
 - e. serum transferrin
5. Serum albumin can be a good indicator of nutritional status except when the patient is also dealing with
 - a. inflammation
 - b. infection
 - c. acute stress
 - d. dehydration
 - e. any of the above
6. Which of the following is a dietary intake - related cause of an elevated BUN value?
 - a. dehydration
 - b. GI bleeding
 - c. liver failure
 - d. kidney failure
 - e. heart failure
7. Which of the following does NOT increase a patient's need for calories beyond his/her baseline requirement?
 - a. kidney failure
 - b. stress
 - c. infection
 - d. fever
 - e. sepsis
8. Which nutrients are generally recommended for supplementation in all patients undergoing renal replacement therapy?
 - a. vitamins A and K
 - b. zinc, selenium, and aluminum
 - c. B-complex and vitamin C
 - d. carnitine and essential amino acids
 - e. omega-6 fatty acids
9. What is the first nutrition support treatment of choice for patients who cannot or will not eat enough food to provide adequate calories and protein?
 - a. enteral supplementation
 - b. carnitine administration
 - c. parenteral nutrition
 - d. zinc administration
 - e. B-complex enhancement
10. The general recommendation regarding fluid allowance for patients with acute renal failure is....
 - a. 1000 cc per day plus amount of urine output in 24 hours
 - b. 500 cc per day plus amount of urine output in 24 hours
 - c. no limit as long as no edema or shortness of breath occurs
 - d. 2 cc per kilogram body weight
 - e. 1 cc per kilogram body weight

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Kidney Friendly Food Facts

Road Map to Improve Intake

By Sharon Schatz, MS, RD, CSR, CDE.

Sharon is a renal dietitian with Gambo Healthcare in Lumberton, N.J. She can be reached at Srsmsrd@aol.com or sharon.schatz@us.gambro.com

Barriers that impede adequate nutritional intake may include patients' resistance to trying new foods, economic constraints, lack of physical energy to prepare meals and poor appetites. The challenge for the renal population includes pushing energy-dense foods and protein while avoiding excesses of sodium, potassium, and phosphorus. Dietitians should individualize dietary goals to maximize glycemic control and decrease saturated fat intake for heart health while prioritizing protein and caloric balance. Post-prandial monitoring of glucose helps determine optimal carbohydrate tolerance of meals and snacks for patients with diabetes.

When dialyzers were less efficient, diets were more limited than what are prescribed today. Instead of recommending Polycose powder in applesauce, liquid Polycose in cranberry juice, Microlipid in hot cereal, or the intake of butterballs, dietitians now have more options to employ.

The presentation of ideas can be an influential factor, as whatever dietitians recommend should be appetizing and easy to prepare. Many candies and snacks are good sources of calories. Consider highlighting some of these candies:

- Hard candies such as sour balls, butterscotch, lemon drops, peppermint and spearmint stars, cinnamon balls, root beer barrels, Life Savers™
- Gummy candies such as gum drops, gummy bears, spearmint leaves
- Jellied candies such as jelly beans, Skittles™, jellied fruit slices
- Marshmallows, marshmallow type candies such as circus peanuts, harvest creme mix, candy corn, marshmallow chicks
- Chewy candies such as Tootsie Roll™, Tootsie Roll™ fruit flavored candies,

Tootsie Roll Pops™, Charleston Chews™, Starburst™ fruit chews

Patients should be encouraged to consume calorie-containing liquids instead of drinking water. They could try popsicles, fruit ice, frozen fruit juice bars, allowed carbonated beverages, Kool-aid™, lemonade, limeade or cranberry juice cocktails. Ice cubes can be made from lemonade or limeade to add flavor as well as calories to iced tea. Fruit flavored gelatin desserts and salads can be prepared with chilled ginger ale or lemon-lime soda in place of cold water.

Low potassium fruits are multifunctional. Fruits canned in heavy syrup will provide more calories than those in lite syrup or fruit juice. Fruit pies, crisps and cobblers pack a powerful concentrated caloric punch. Commercial fruit pie fillings can be served as-is for dessert or be warmed for use as a sauce served over plain or pound cake. Cranberry sauce, warmed applesauce and glazed apple slices are flavorful accompaniments to poultry and meats. Fruit leather is an easy-to-tote snack.

Baked goods and products provide extra sugar and fat to the diet. Depending on the type of product, they can be used for breakfast, snacks, or desserts. Binder therapy may need to be adjusted for items with more phosphorus. Some of these more caloric items could be attempted:

High Calorie Foods

- glazed donuts
- jelly donuts
- Danish pastry
- toaster pastry or Pop Tarts™

Table 1. Frosting & Fudge

Item	Nutrient Analysis per 1 ounce						
	Kcal	Carb (gm)	Fat (gm)	Ca (mg)	Phos (mg)	Na (mg)	K (mg)
Cream cheese frosting	118	19	5	1	1	54	10
Creamy vanilla frosting	119	19	5	1	5	52	10
Sour cream frosting	117	19	5	1	1	58	55
Creamy chocolate frosting	113	18	5	2	22	52	56
White chocolate fudge	160	21	8	21	24	50	52
Butterscotch fudge	143	21	7	5	29	45	29

- crumb type coffeecake
- fruit filled coffeecake
- biscuits
- pancakes and waffles
- French toast
- graham crackers
- shortbread
- butter cookies
- sugar cookies or wafers
- ladyfingers
- vanilla sandwich cookies
- pound cake
- sponge cake
- rice cakes
- low potassium fruit
- quick breads
- applesauce cake
- blonde brownies
- cupcakes
- Rice Krispies Treats™

Brands of packaged snack cakes vary by geographic location and offer an array of sweet treats. Dietitians should be aware of the ingredients for various brands, encouraging the high-calorie baked products with minimal saturated fats and trans fats. Tastykake™ dominates the snack food market in some areas of the United States, and the web site, www.tastykake.com features nutrition label information as well as mail order purchase. McKee Foods Corporation, www.mckeefoods.com, manufactures Little Debbie and Sunbelt brand products, and a detailed nutritional information handout for Little Debbie Family Packs is available.

Ready-to-eat frosting can be a versatile cupboard staple. Frosting can be spread on

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Kidney Friendly Food Facts

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plain cakes or cookies or as a filling inside ladyfingers. If thinned with non-dairy liquid creamer, it can be a dip for strawberries or other cut fruit. For quick and easy fudge: melt one bag (10 or 12 oz size) of white vanilla (white chocolate) or butterscotch chips and combine with one can (16 oz) creamy vanilla frosting, spread mixture in foil-lined 9" square pan, refrigerate for at least 1 hour, and cut into 24 pieces. See Table I for the nutrient analyses for ready-to-eat frostings and the above fudge recipe.

Nutrition after dialysis initiation poses a protein dilemma. The intake of protein often decreases when a person is uremic before starting dialysis. There may be a psychological adverse association to eating meat. Some patients find it difficult to eat solid large pieces of meat and may need ideas for other forms. Sometimes, the person may only need preparation suggestions to relieve menu boredom. If a person experiences early satiety, encourage consumption of protein foods before other items at a meal. Optimize protein intake by including protein sources

in snacks. Sandwiches may seem easier to eat than a full plate of food.

Protein bars are plentiful in the marketplace. There are too many brands to do justice to the field, and availability is subject to change. Cost is a factor, but use of bars may seem less expensive when it is presented to the patient as an alternative to a sandwich that has two slices of bread and two ounces of meat. Nutrient profiles can vary greatly. The protein source is usually whey or soy, and whey products often have less potassium and phosphorus than soy. Calorie content is impacted by whether the product is a meal replacement, energy bar or "low carb" item. Vitamin and mineral additives can prohibit the use of some bars. Dietitians may analyze bars to find the ones with the least amount of phosphorus per gram of protein avoiding those high in calcium, vitamin A or vitamin C. Check out these web sites for information about nutritional bars. (Please note this is not a definitive list):

<http://www.vitaminshoppe.com>
<http://www.vitacost.com>
<http://www.allstarhealth.com>

<http://www.appletothecore.com/weightloss/proteinbars.htm>
<http://www.powerbar.com/Products/>
<http://atkins.com/atkins-products/morning-start-breakfast-bars.html>
<http://www.balance.com/products/default.asp>
<http://www.nutrinergy.com>
http://www.kashi.com/bars.aspx?SID=1&aspx?s=product&m=product/product_display&u3=*****4300015602***
(Post Carb Well high protein cereal bar)
http://kraftfoods.com/main.aspx?s=product&m=product/product_display&u3=*****4300015604***
(Post Carb Well high protein cereal bar)

Editor's note: All websites were accessed March 26, 2005.

CERTIFICATE OF COMPLETION

Nutrition Management of the Patient with Acute Renal Failure

Title of Program

Date of Completion

Renal Nutrition Forum

Commission on Dietetic Registration CPE Accredited Provider

AM 003

CPE Provider Accreditation Number



CPE Accredited Provider

Participant's Name

Has successfully completed 2 CPEUs CPE Level 2

Sarah Carter, RD CDE, Editor, Renal Nutrition Forum

Signature of CDR CPE Accredited Provider

6/1/05 to 5/31/06

Date Valid

Answers to CPE questions:

- | | | | | |
|------|------|------|------|-------|
| 1. D | 2. E | 3. D | 4. B | 5. E |
| 6. A | 7. A | 8. C | 9. A | 10. B |

RPG Chair Message

By Cathi J. Martin, RD, CSR, LDN

Cathi is a regional dietitian with Renal Care Group in Nashville, Tenn. She can be reached at cjmartin@renalcaregroup.com.

Greetings and welcome to all new members and returning members. It is an honor for me to serve as the Chair of the Renal Practice Group this year. I would like to congratulate our new officers and offer many thanks to all the RPG members who ran on the ballot. In addition, a number of members have been newly appointed to various positions. Please join me in welcoming Connie Cranford, MS, RD as Membership Chair; Chhaya Patel, MA, RD as Area 1 Coordinator and Chair of Quality and Outcomes; Patti Barba', MS, RD as Area 7 Coordinator and Historian; Patricia Williams, MS, RD as Area 3 Coordinator and Education Chair; Lisa Anderson, RD, CSR, LD as Area 4 Coordinator and East Lending Librarian; Karen Basinger, RD, "current" Area 6 Coordinator and Legislative Chair, and Mary Jo Dahms, RD, Area 2 Coordinator and Awards/Scholarship Chair who will remain on the executive committee for another term.

Many of the RPG board members were able to attend the NKF Spring Clinical Meeting in Washington, DC this year. They

participated in a joint networking breakfast with members of the Council on Renal Nutrition's Executive Committee and an all day legislative workshop that included visits to Capitol Hill and meeting members of the Kidney Caucus. This was especially timely with the new legislative proposals regarding MNT and the CMS Conditions of Coverage that define a renal dietitian's qualifications as well as many other important standards that affect our clinical practice. RPG issued formal statements regarding both of these legislative issues which can be found on our website.

RPG is working on many goals right now, including improving the lending libraries. By the end of the summer, each library will have a copy of the references recommended by CDR to study for the CSR exam. In addition, the Spanish version of our simplified renal diet (ESRD Diet – Eating Simply with Renal Disease) will be available soon. Please contact Pat Williams at pwilliams@renalcaregroup.com to order these materials.

Our featured speaker at FNCE this year will be Dr. Kevin Martin from St. Louis University. He will be discussing vitamin D needs and functions in Chronic Kidney Disease. Catherine M. Goeddeke – Merickel, MS, RD will be on the panel as

well to provide the dietitian's perspective. The session is being supported by a grant from Abbott Laboratories and is entitled "No Bones About It: The Role of Vitamin D in Chronic Kidney Disease". It will be held on Tuesday, Oct 25th from 10am-11 am. This topic stems from recent research being conducted with CKD patients to determine other organ systems that are being affected by vitamin D deficiency and if physiological treatment doses would be beneficial in these patients. Dr. Martin is involved in this ongoing research, and you will not want to miss this session! In addition, we will also be offering our Annual Networking Breakfast at FNCE and presenting the Outstanding Service Award to a distinguished renal dietitian. Make your plans now to attend the FNCE Meeting in St. Louis and take advantage of the educational stipends available through RPG!

Finally, I would like to add how much we appreciate YOU, as a member of this organization. I look forward to meeting many of you at upcoming meetings and personally encourage each of you to find a way to become more involved with RPG. Please consider participating in the new member survey that will be posted online in September and let us know how we can serve you better!

CRN Chair Message

By Deborah Brommage, MS, RD, CSR, CDN Deborah is chair of the Council on Renal Nutrition (CRN) of National Kidney Foundation (NKF) and is administrative dietitian at Winthrop-University Hospital Dialysis Center in Mineola, Long Island, N.Y. She can be reached at dbrommage@winthrop.org

Raising CKD Awareness

In the January/February 2005 issue of House Magazine an article titled "What is a CKD?" caught my attention. While the renal community will recognize this acronym as chronic kidney disease (CKD),

the readers of this decorating magazine were learning about certified kitchen designers. Seeing CKD presented in this manner reminded me of how important it is to work together to increase awareness the public's awareness of chronic kidney disease (CKD).

CKD is a growing public health problem. There are now 20 million Americans who have CKD and another 20 million who are at risk of developing this disorder. It is expected that the number of patients with kidney failure will increase to 650,000 by the year 2010. Surprisingly, most people

are unaware that they have kidney disease or that they are at risk. The National Kidney Foundation (NKF) has numerous initiatives to increase CKD awareness. As renal dietitians we have many opportunities to become involved in this effort.

Kidney Month

As nutrition professionals we know that March is National Nutrition Month, but were you aware that March is also Kidney Month? This year NKF's focus was on the fact that one in nine Americans has

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Proven ReZults

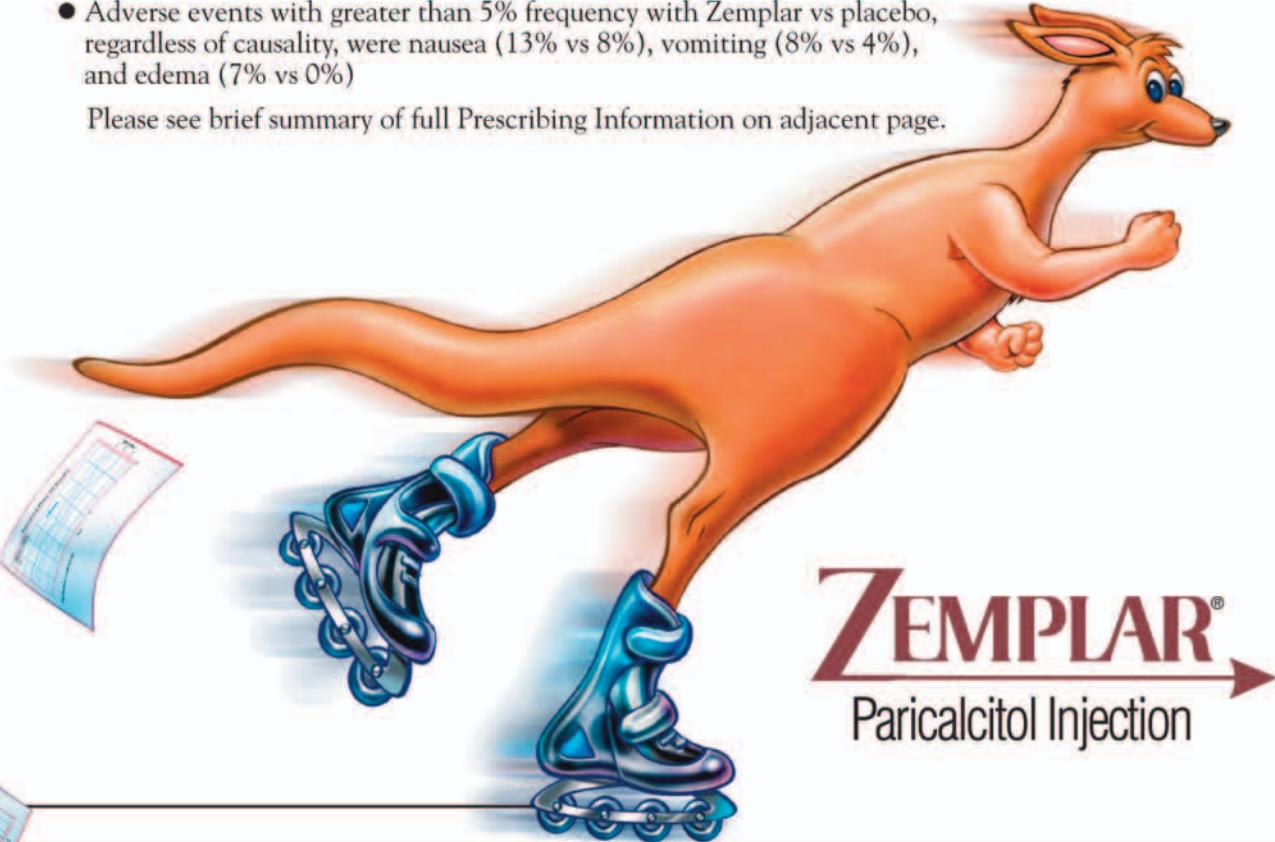
There is more to know about Zemplar...

- Clinically studied in over 700 patients¹⁻⁶
- PTH outcomes: proven to safely and rapidly reduce PTH levels with no significant difference in the incidence of hypercalcemia or hyperphosphatemia when compared to placebo²
- Zemplar is indicated for both the treatment and prevention of secondary hyperparathyroidism associated with chronic renal failure

Important Safety Considerations

- Zemplar is contraindicated in patients with vitamin D toxicity, hypercalcemia, or hypersensitivity to product ingredients
- Administration may place patients at risk for hypercalcemia, elevated Ca × P product, and metastatic calcification
- Adverse events with greater than 5% frequency with Zemplar vs placebo, regardless of causality, were nausea (13% vs 8%), vomiting (8% vs 4%), and edema (7% vs 0%)

Please see brief summary of full Prescribing Information on adjacent page.



ZEMPLAR®
Paricalcitol Injection

 Abbott Laboratories
Abbott Park, IL 60064

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REFERENCES: 1. Clinical data on file, 2003. 2. Martin KJ, et al. *J Am Soc Nephrol*. 1998;9:1427-1432. 3. Martin KJ, et al. *Am J Kidney Dis*. 1998;32:s61-s66. 4. Lindberg J, *Clin Nephrol*. 2001;56(4):315-23. 5. Martin KJ, et al. *Am J Kidney Dis*. 2001;38(5):s57-s63. 6. Sprague SM, et al. *Kidney Int*. 2003;63:1483-1490.

BRIEF SUMMARY
CONSULT PACKAGE INSERT FOR FULL PRESCRIBING INFORMATION

Zemplar®

(paricalcitol injection, USP)

Fliptop Vial

Rx only

INDICATIONS AND USAGE

Zemplar® is indicated for the prevention and treatment of secondary hyperparathyroidism associated with chronic renal failure. Studies in patients with chronic renal failure show that Zemplar® suppresses PTH levels with no significant difference in the incidence of hypercalcemia or hyperphosphatemia when compared to placebo. However, the serum phosphorus, calcium and calcium x phosphorus product (Ca x P) may increase when Zemplar® is administered.

CONTRAINDICATIONS

Zemplar® should not be given to patients with evidence of vitamin D toxicity, hypercalcemia, or hypersensitivity to any ingredient in this product (see **PRECAUTIONS, General**).

WARNINGS

Acute overdose of Zemplar® may cause hypercalcemia, and require emergency attention. During dose adjustment, serum calcium and phosphorus levels should be monitored closely (e.g., twice weekly). If clinically significant hypercalcemia develops, the dose should be reduced or interrupted. Chronic administration of Zemplar® may place patients at risk for hypercalcemia, elevated Ca x P product, and metastatic calcification. Signs and symptoms of vitamin D intoxication associated with hypercalcemia include:

Early

Weakness, headache, somnolence, nausea, vomiting, dry mouth, constipation, muscle pain, bone pain, and metallic taste.

Late

Anorexia, weight loss, conjunctivitis (calcific), pancreatitis, photophobia, rhinorrhea, pruritus, hyperthermia, decreased libido, elevated BUN, hypercholesterolemia, elevated AST and ALT, ectopic calcification, hypertension, cardiac arrhythmias, somnolence, death, and rarely, overt psychosis.

Treatment of patients with clinically significant hypercalcemia consists of immediate dose reduction or interruption of Zemplar® therapy and includes a low calcium diet, withdrawal of calcium supplements, patient mobilization, attention to fluid and electrolyte imbalances, assessment of electrocardiographic abnormalities (critical in patients receiving digitalis), hemodialysis or peritoneal dialysis against a calcium-free dialysate, as warranted. Serum calcium levels should be monitored frequently until normocalcemia ensues.

Phosphate or vitamin D-related compounds should not be taken concomitantly with Zemplar®.

PRECAUTIONS

General

Digitalis toxicity is potentiated by hypercalcemia of any cause, so caution should be applied when digitalis compounds are prescribed concomitantly with Zemplar®. Adynamic bone lesions may develop if PTH levels are suppressed to abnormal levels.

Information for the Patient

The patient should be instructed that, to ensure effectiveness of Zemplar® therapy, it is important to adhere to a dietary regimen of calcium supplementation and phosphorus restriction. Appropriate types of phosphate-binding compounds may be needed to control serum phosphorus levels in patients with chronic renal failure (CRF), but excessive use of aluminum containing compounds should be avoided. Patients should also be carefully informed about the symptoms of elevated calcium.

Essential Laboratory Tests

During the initial phase of medication, serum calcium and phosphorus should be determined frequently (e.g., twice weekly). Once dosage has been established, serum calcium and phosphorus should be measured at least monthly. Measurements of serum or plasma PTH are recommended every 3 months. An intact PTH (iPTH) assay is recommended for reliable detection of biologically active PTH in patients with CRF. During dose adjustment of Zemplar®, laboratory tests may be required more frequently.

Drug Interactions

Specific interaction studies were not performed. Digitalis toxicity is potentiated by hypercalcemia of any cause, so caution should be applied when digitalis compounds are prescribed concomitantly with Zemplar®.

Carcinogenesis, Mutagenesis, Impairment of Fertility

In a 104-week carcinogenicity study in CD-1 mice, an increased incidence of uterine leiomyoma and leiomyosarcoma was observed at subcutaneous doses of 1 to 10 mcg/kg (< 1 to 3 times the maximum recommended human weekly dose of 0.72 mcg/kg, based on body surface area, mg/m²). The incidence rate of uterine leiomyoma was significantly different than the control group at the highest dose of 10 mcg/kg.

In a 104-week carcinogenicity study of rats, there was an increased incidence of benign adrenal pheochromocytoma at subcutaneous doses of 0.15 to 1.5 mcg/kg (≤ 1 times the maximum recommended human weekly dose of 0.72 mcg/kg, based on body surface area, mg/m²). The increased incidence of pheochromocytomas in rats may be related to the alteration of calcium homeostasis by paricalcitol. In carcinogenicity studies in rats and mice, paricalcitol did not affect the incidences of tumors apart from benign rodent-specific lesions related to the effects of chronic hypercalcemia.

Paricalcitol did not exhibit genetic toxicity *in vitro* with or without metabolic activation in the microbial mutagenesis assay (Ames Assay), mouse lymphoma mutagenesis assay (L5178Y), or a human lymphocyte cell chromosomal aberration assay. There was also no evidence of genetic toxicity in an *in vivo* mouse micronucleus assay. Zemplar® had no effect on fertility (male or female) in rats at intravenous doses up to 20 mcg/kg/dose [equivalent to 13 times the highest recommended human dose (0.24 mcg/kg) based on surface area, mg/m²].

Pregnancy

Pregnancy Category C.

Paricalcitol has been shown to cause minimal decreases in fetal viability (5%) when administered daily to rabbits at a dose 0.5 times the 0.24 mcg/kg human dose (based on surface area, mg/m²) and when administered to rats at a dose 2 times the 0.24 mcg/kg human dose (based on plasma levels of exposure). At the highest dose tested (20 mcg/kg 3 times per week in rats, 13 times the 0.24 mcg/kg human dose based on surface area), there was a significant increase of the mortality of newborn rats

at doses that were maternally toxic (hypercalcemia). No other effects on offspring development were observed. Paricalcitol was not teratogenic at the doses tested.

There are no adequate and well-controlled studies in pregnant women. Zemplar® should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers

It is not known whether paricalcitol is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Zemplar® is administered to a nursing woman.

Pediatric Use

The safety and effectiveness of Zemplar® were examined in a 12-week randomized, double-blind, placebo-controlled study of 29 pediatric patients, aged 15-19 years, with end-stage renal disease on hemodialysis and nearly all had received some form of vitamin D prior to the study. Seventy-six percent of the patients were male, 52% were Caucasian and 45% were African-American. The initial dose of Zemplar® was 0.04 mcg/kg 3 times per week, based on baseline iPTH level of less than 500 pg/mL, or 0.08 mcg/kg 3 times a week based on baseline iPTH level of ≥ 500 pg/mL, respectively. The dose of Zemplar® was adjusted in 0.04 mcg/kg increments based on the levels of serum iPTH, calcium, and Ca x P. The mean baseline levels of iPTH were 841 pg/mL for the 15 Zemplar®-treated patients and 740 pg/mL for the 14 placebo-treated subjects. The mean dose of Zemplar® administered was 4.6 mcg (range: 0.8 mcg – 9.5 mcg). Ten of the 15 (67%) Zemplar®-treated patients and 2 of the 14 (14%) placebo-treated patients completed the trial. Ten of the placebo patients (71%) were discontinued due to excessive elevations in iPTH levels as defined by 2 consecutive iPTH levels > 700 pg/mL and greater than baseline after 4 weeks of treatment.

In the primary efficacy analysis, 9 of 15 (60%) subjects in the Zemplar® group had 2 consecutive 30% decreases from baseline iPTH compared with 3 of 14 (21%) patients in the placebo group (95% CI for the difference between groups -1%, 63%). Twenty-three percent of Zemplar® vs. 31% of placebo patients had at least one serum calcium level > 10.3 mg/dL, and 40/5 vs. 14% of Zemplar® vs. placebo subjects had at least one Ca x P ion product > 72 (mg/dL)². The overall percentage of serum calcium measurements > 10.3 mg/dL was 7% in the Zemplar® group and 75 in the placebo group; the overall percentage of patients with Ca x P product > 72 (mg/dL)² was 8% in the Zemplar® group and 7% in the placebo group. No subjects in either the Zemplar® group or placebo group developed hypercalcemia (defined as at least one calcium value > 11.2 mg/dL) during the study.

Geriatric Use

Of the 40 patients receiving Zemplar® in the three phase 3 placebo-controlled CRF studies, 10 patients were 65 years or over. In these studies, no overall differences in efficacy or safety were observed between patients 65 years or older and younger patients.

ADVERSE REACTIONS

Zemplar® has been evaluated for safety in clinical studies in 454 CRF patients. In four, placebo-controlled, double-blind, multicenter studies, discontinuation of therapy due to any adverse event occurred in 6.5% of 62 patients treated with Zemplar® (dosage titrated as tolerated) and 2.0% of 51 patients treated with placebo for 1 to 3 months. Adverse events occurring with greater frequency in the Zemplar® group at a frequency of 2% or greater, regardless of causality, are presented in the following table:

Adverse Event Incidence Rates For All Treated Patients In All Placebo-Controlled Studies

Adverse Event	Zemplar® (n=62) %	Placebo (n=51) %
Overall	71	78
Body as a Whole		
Chills	5	0
Feeling unwell	3	0
Fever	5	2
Flu	5	4
Sepsis	5	2
Cardiovascular		
Palpitation	3	0
Digestive System		
Dry mouth	3	2
Gastrointestinal bleeding	5	2
Nausea	13	8
Vomiting	8	4
Metabolic and Nutritional Disorders		
Edema	7	0
Nervous System		
Light-headedness	5	2
Respiratory System		
Pneumonia	5	0

A patient who reported the same medical term more than once was counted only once for that medical term.

Safety parameters (changes in mean Ca, P, Ca x P) in an open-label safety study up to 13 months in duration support the long-term safety of Zemplar® in this patient population.

Adverse events during post-marketing experience: Taste perversion, such as metallic taste, and allergic reactions, such as rash, urticaria and pruritis rarely have been reported.

OVERDOSAGE

Overdosage of Zemplar® may lead to hypercalcemia (see **WARNINGS**).

Revised: October, 2004

Ref: EN-0423 (10/04)

04L-130-F901-1 MASTER

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Rehab Corner

Exercise: Creating Positive and Fun Expectations for Dialysis Patients

By Stephanie McIntyre, RD. Stephanie is patient rehab director for Renal Care Group, Inc. and a renal dietitian at Renal Care Group-Phoenix. She may be contacted at smcintyre@renalcaregroup.com.

Just saying the word "exercise" to patients may create many different feelings and reactions – some negative and some positive. One of the roles of the dietitian is to encourage and educate patients about exercise. Exercise is an integral part of maintaining good health. It is a useful tool used to decrease cardiovascular disease risk factors. Good nutrition and daily exercise complement each other and provide faster and longer lasting results. An example would be the battle of the bulge – weight loss. Dietitians need to take advantage of any tools that complement nutritional efforts in order to improve patients' health, well-being and quality of life.

Benefits in Brief

Many articles have been published on the benefits of exercise, a few of which specifically pertain to dialysis patients (1-6). Here is an abbreviated list of the benefits of exercise:

- Increased energy
- Stronger muscles
- Improved balance
- Improved blood glucose
- Improved blood pressure control
- Improved bone health and strength
- Decreased incidence of depression
- Weight loss if needed
- Increased endurance

The Dietitian's Role in Exercise – Getting More Specific

Exercise encouragement and education is the responsibility of all healthcare team members. The dietitian is the ideal healthcare team member to lead these efforts. In a perfect world, all dialysis facilities would have exercise programs. Unfortunately, the percentage of facilities that have an in-center exercise program is

still small. Regardless of the availability of an in-center exercise program, dietitians should encourage and educate patients on the importance of daily exercise and physical activity.

Assessing Physical Activity Initially

Dietitians can include exercise discussions in their formal nutritional assessments. Patients should hear about exercise within their first month on dialysis. Determine if patients have been told that they cannot exercise or if they falsely believe that they must cease most pre-dialysis activities. The primary goal of rehabilitation is to assist patients in resuming their usual, or healthier, routines as soon as possible.

To promote daily physical activity one needs to talk about it routinely so that it becomes an expectation for the patient – a fun and positive expectation. Here are some examples of questions that may be used during nutritional assessments:

- What physical activities do you enjoy?
- How often do you participate in these activities?
- How long (duration) do you exercise or how far (distance) do you go?
- Do you believe that you can be physically active now that you are on dialysis?
- Would you like more information on exercise?
- Do you walk or use a motorized cart while grocery shopping?
- Do you use walker, cane, wheel chair or motorized scooter?
- Do you have physical limitations or previous injuries?
- Were you referred to physical or occupational therapy for these injuries?
- What was your experience with therapy?

Ongoing Exercise Discussion:

Depending on the uremic or anemic state during initial assessment, the patient may need activity questions and information repeated. Follow up on the activities that the patient reported during the initial

assessment and ask questions such as, "Are you back to (walking) again? How far do you (walk)?" If the patient has not resumed pre-dialysis activity, try to identify what barriers are hindering exercise. Inquire whether the patient has exercise support partners, such as a spouse, pets, children or grandchildren. Ask patients to rank their energy level on a scale of one to ten each month. The range of relevant questions will vary with each patient's experience and activities.

Dietitians play an active role in educating and encouraging exercise. Whether leading the rehabilitation team or participating as active team members, dietitians may utilize exercise to improve patients' quality of life and complement nutritional approaches.

Resources for Exercise Information

A great professional resource and patient education tool is the Life Option Rehabilitation Advisory Council's booklet, *Exercise: A Guide for People on Dialysis* by Patricia Painter, PhD. This booklet contains an easy to follow discussion of the three types of exercise and guidelines to help patients get started. This booklet can be downloaded from the website www.lifeoptions.org (accessed March 26, 2005).

Two exercise associations that offer scientifically sound information for health professionals and patients are the American Council on Exercise (www.acefitness.org) and the American College of Sports Medicine at www.acsm.org (both websites accessed March 26, 2005). These organizations are not the only resources for exercise information but are good places to start. Please feel free to contact me if you have other exercise-related sites and associations that you would recommend to dietitians.

REFERENCES

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Continued on page 26

CRN Chair Message

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CKD. There were educational efforts at the local level as well as ads placed in national publications such as *Family Circle* and *Ebony*. This is a good opportunity for renal dietitians to combine the nutrition and kidney themes for patient and public education in the month of March.

Kidney Early Evaluation Program

The momentum of the Kidney Early Evaluation Program (KEEP), sponsored by the local NKF Affiliates, is quite impressive. This free health screening program has served 40,000 individuals, and physicians have received 10,000 KEEP patient referrals. The goals of the program are to increase CKD awareness and to provide free testing for people at risk for kidney disease. Educational information and physician referral are also provided. Renal dietitians who want to volunteer for a KEEP program in their area can contact NKF at 800-622-9010 for the number of the local NKF Affiliate or visit the KEEP Health Screening Schedule Page at www.kidney.org/keep/keepevents.cfm (accessed March 25, 2005).

Public Health Kidney Disease Action Plan

In 2005 a priority of the NKF public policy program is the development of a CKD program at the U.S. Centers for Disease Control and Prevention (CDC). Creation of a national public health program of this kind would increase CKD awareness through surveillance activities that identify populations at risk and improve the timeliness of CKD detection in a greater number of people. Improved prevention, detection and early treatment, to include medical nutrition therapy, can reduce morbidity and mortality associated with CKD.

Congressional Kidney Caucus

In an effort to increase CKD awareness in Congress, the Congressional Kidney Caucus was established. The goals of the kidney caucus are to support legislation that would impact kidney patients and to influence pertinent federal agencies in the fight against CKD. Over the past 3 years, 40 U.S. Representatives have been recruited, but there is more work to be done. As

renal dietitians, we can participate in this recruitment effort by faxing letters to our members of Congress, asking them to join the Congressional Kidney Caucus. A sample letter can be found on the NKF website at www.kidney.org/general/pubpol/SAMPLEcaucusletter_GR.pdf. You can also find your members of Congress by visiting www.capwiz.com/c-span/dbq/ officials (both websites accessed March 25, 2005).

On May 4, 2005, CRN members along with other NKF professional council members participated in a trip to Capitol Hill to encourage support from members of Congress for programs that have an impact on people with CKD.

Take Action

There are several action items listed above that everyone can take part in. Take a moment to fit one of these opportunities into your busy schedule and contribute to the effort to raise CKD awareness. We can all make a difference in this endeavor.

Rehab Corner

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RPG Mission: The RPG is the advocate of the nutrition profession serving the public through the promotion of optimal renal nutrition, health and well-being.

RPG Vision: RPG members will be leaders in providing scientifically sound renal nutrition care and education for patients, the profession and the public.

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