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In This Issue

Binder Bracelet Activity to Help Improve Binder Medication Compliance and Serum Phosphorus in Hemodialysis Patients

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This article has been approved for 1.5 CPE units. The online CPEU quiz and certificate of completion can be accessed in the Members Only section of the RPG web site via the My CPEU link. This CPE offering is available to current RPG members only and the expiration date is October 15, 2013.

Abstract:

Objective: The purpose of this study was to evaluate the use of a bracelet to obtain positive serum phosphorus outcomes among patients requiring hemodialysis for end stage renal disease. **Design:** A twelve-week quasi-experimental study that assigned patients to intervention or control. **Setting/Subjects:** The study sample included patients from a rural, in-center hemodialysis clinic. Patients were invited to participate in an activity to improve serum phosphorus. **Intervention:** Patients were asked to wear a rubber bracelet with an inscription to help remind patients to take their binder medication. Per facility protocol, Mineral Bone Density (MBD) labs were collected and recorded monthly. **Main Outcome Measure:** Statistical analyses were run to determine if there was a significant difference between those that participated in the in-center activity and those that did not participate. Patient lab results were compared to the K/DOQI guidelines for serum phosphorus. **Results:** Although results were not found to be significant ($p<0.05$), there was positive trending in the activity group. At baseline, mean serum phosphorus in the activity group was abnormal; however, mean serum phosphorus met the K/DOQI guideline the last month of the activity. **Conclusion:** From this study one can conclude that the use of a bracelet reminder can serve as a positive reminder to increase compliance for patients struggling to remember binder medication and those newly prescribed a binder medication.

Key Words: Serum phosphorus; binder medication; compliance; bracelet reminder; learning tool

– Continued on page 3.

RPG

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From the Editor's Desk

Sara Erickson, RD, CSR, LDN, CNSC
Editor

For those members who were able to attend FNCE or the RPG hosted cruise I hope you had a productive and fun-filled experience! Please consider sharing any RPG event photos for inclusion in our next Forum issue by emailing them to RPGforumeditor@gmail.com. Also, if you attended a session that you feel would benefit your fellow RPG members please consider sharing your experience through a write up in the Forum. We are always in need of content!

In this issue, there are 3.0 CPEUs available. Our featured article, co-authored by Molly Ennis, RD, LD, Mildred Mattfeldt-Beman, PhD, RD, LD, Amy R. Moore, MPH, MS, RD, LD, and Ajlina Karamehic-Muratovic, PhD, features a study that evaluates a technique to improve patient motivation with binder compliance. As most renal dietitians know, this is an arduous task for which, unfortunately, there can seem to be no end in sight, particularly when considering the extensive use of phosphate additives in the food industry today. Their study reinforces renal dietitians' positive outcomes on patient care, which is both motivating and greatly appreciated! Our advances in practice article, co-authored by Cassandra Pogatshnik, RD, LD, CNSC and Cindy Hamilton, MS, RD, LD, CNSD, provides extensive information on how to perform a nutrition-focused physical exam. As patients with chronic kidney disease are at high risk for malnutrition, protein energy wasting, inflammation, and altered micronutrient levels, renal dietitians should consider including this practice as part of their patient nutrition evaluations. Improving clinical skills will not only benefit your patients but also solidify your contribution to the care team!

For our member spotlights we have three session reviews from RPG members who attended the National Kidney Foundation's Spring Clinical Meetings earlier this year. Thank you to Peggy Harum, RD, LD, Terri Tomak, RD, CSR, and Lynn Munson, MS, RD, LD for their in-depth reviews! Lynn also provides additional information on dietary sources of oxalate and their role in kidney stones, which can be found in the highlighted section on page twenty.

To our outgoing Managing Editor, Megan Sliwa, RD, LDN, MBA, I can't thank you enough for all your hard work, guidance, support, and mentoring you've given me over the past few years. I wish you the best of luck in all your future endeavors! To my fellow Editorial Team Members, Jackie Termont, RD and Amy Braglia-Tarpey, MS, RD, CSR, CNSC, a huge thanks for your dedication and hard work! As always, sincere thanks to the peer-reviewers who provide invaluable feedback, the authors for their contributions, and to Amy Hess-Fishl, MS, RD, LDN, BC-ADM, CDE, for providing the CPEU test questions. Without you the Forum could not be completed!

Best Regards,

Calling All Authors!

Renal Nutrition Forum Article Submissions Needed!

If you have ever considered submitting an article to the RNF now is the time! Please consider sharing your work with fellow RPG members or reaching out to colleagues to inquire about work they may be interested in submitting. We guarantee publication!

For more information, please contact RNF Editor, Sara Erickson at rpgforumeditor@gmail.com

Introduction:

Chronically high serum phosphorus levels can lead to a variety of complications. Hyperphosphatemia contributes to the pathogenesis of secondary hyperparathyroidism and its skeletal expression, namely osteitis fibrosa. Together with calcium (Ca), hyperphosphatemia also promotes the deposition of calcium phosphate crystals in soft tissues and vessel walls. In particular, calcification in the periarticular regions can lead to serious consequences (1). Cardiovascular events are responsible for almost 50% of the mortality of CKD patients receiving hemodialysis (HD). The presence and extent of vascular calcifications are strong predictors of cardiovascular mortality among these patients (2,3). Serum phosphorus above 6.5 mg/dL, corrected Ca above 9.5, and a Ca x Phosphorus product > 65 in HD patients increase risk of calcification and mortality rates. It is important to control serum phosphate in order to avoid or treat secondary hyperparathyroidism and soft-tissue calcifications (2).

Oral phosphate binder medication has become the basis of the therapeutic approach for dialysis patients diagnosed with hyperphosphatemia (4). Target levels of serum phosphate, however, are not consistently being met. The K/DOQI guideline recommends serum phosphorus levels be maintained below 5.5 mg/dL (4). Given the demonstrated efficacy of phosphate binders in clinical trials, it is unlikely that the failure to achieve targets is entirely due to a lack of availability of effective treatments (2,5-7). A number of other factors may contribute to the poor achievement of serum phosphate targets—including poor adherence to phosphate-binder treatment regimens (5,7). Poor adherence to medication is an increasing problem among patients with chronic kidney disease (CKD) Stage 5. These patients are subject to large daily tablet burdens and may be prescribed greater than 10 different medications (6). The cumulative effect of multiple dosing regimens can impose a confusing and overwhelming burden on a patient—often leading to noncompliance with medication guidelines (6,7). Phosphate-binder therapy contributes a considerable proportion of the daily tablet burden for patients with CKD stage 5 (6). Studies suggest that patients fail to take 18-20% or more of their prescribed phosphate binders (6).

Much has been done to try and improve patient compliance to binder medication. Several studies have investigated renal patients' self-efficacy and motivation regarding binder medication compliance (8). Research suggests that increased self-efficacy is associated with positive changes in health care behaviors (8). Patient self-management activities help increase self-efficacy and can lead to a range of positive health changes and outcomes. With regards to positive phosphorus outcomes, activities may include in-center group education sessions, various games and contests that help increase dietary knowledge of recommended foods and improve binder compliance (9-11). Many HD centers focus on nutrition intervention challenges to help control phosphorus intake. This includes, but is not limited to, educational posters displayed throughout centers, nutritional handouts, and small, inexpensive prizes as incentives to help increase motivation (9-11). However, studies have shown that despite increased knowledge, long-term behavior (dietary and medication compliance) often does not change (11).

The purpose of this study was to evaluate the use of a bracelet to obtain positive serum phosphorus outcomes among patients requiring hemodialysis for end stage renal disease.

Methods:

Study Design

This study was conducted using a quasi-experimental design to determine if patients who wore a reminder bracelet were more likely to comply with binder medication. Ethical clearance was obtained from the Institutional Review Board of Saint Louis University.

Prior to beginning the activity, patients were asked to complete a short questionnaire administered by the registered dietitian (RD). The questionnaire assessed binder medication compliance and gastrointestinal upset often associated with binder medications. After completion of the questionnaire, activity patients were asked to wear a bright green, rubber bracelet daily for three months. The bracelet was similar in design and appearance to the yellow, "Livestrong" bracelet. The bracelet was inscribed with the phrase: "Remember to take your binder". Participants were instructed and encouraged to wear the bracelet continuously.

Serum phosphorus levels were collected monthly over a total period of three months. October serum phosphorus provided baseline information to compare lab results with a data collection window of 90 days. Blood samples for serum phosphorus were drawn by facility registered nurses and were part of the monthly MBD lab collection. Labs were drawn the second week of each month on a Monday or Tuesday, per facility protocol. Bracelets were given to activity group participants after October labs were drawn. Serum phosphorus lab results were collected from an online medical record database specific to the dialysis center.

Sample

Total number of in-center patients was 27. To be eligible for the in-center activity, the patients had to meet the following criteria: 1) receiving hemodialysis > 90 days; 2) binder therapy medication >/ 3 months; 3) no previous complications or hospitalizations and were considered "stable" within 30 days prior to study; and 4) no history of latex or rubber allergy. Patients who did not meet inclusion criteria were not asked to participate.

Data Analysis

SPSS 20.0 software was used to analyze the study data. Independent sample t-tests were run to see if there was a significant difference in serum phosphorus between the activity and control groups. Paired t-tests were run to determine if there was a statistically significant difference in the serum phosphorus at baseline and at the end of the activity. An effect was considered significant when $P < 0.05$.

TABLE 1. Descriptive statistics of sample group in Jerseyville, IL						
Variable	**Frequency (n)**	**%**	**Minimum**	**Maximum**	**Mean**	**Standard Deviation**
Race						
White	24	100				
Age (years)	24		29	87	64.0833	17.71493
Gender						
Female	11	45.8				
Male	13	54.2				
Activity Participant	24					
Yes	11	45.8				
No	13	54.2				

TABLE 2. Results of in-center binder questionnaire completed by subjects		
Question:	**Frequency (n)***	**%**
1. Forgotten to take binder		
Yes	12	5
No	70.6	29.4
2a. Take binder before a meal/snack	13	76.5
2b. During a meal/snack	3	17.6
2c. After a meal/snack	1	5.9
3. Has binder made you feel sick		
Yes	0	17
No	0	100
4. Forgot to take binder during weekday		
Yes	10	7
No	58.8	41.2
5. Forgot to take binder during weekend		
Yes	10	7
No	58.8	41.2
6. Average times forgot to take binder		
0	7	41.2
1-2 times	4	23.5
3-5 times	3	17.6
6-10 times	1	5.9
> 10 times	2	11.8
*n=17 total		

Results

Of the 27 patients, 24 patients had been receiving hemodialysis > 90 days and were asked to participate in the in-center bracelet activity. Thirteen patients did not want to participate in the activity and were labeled the control group. Eleven patients agreed to participate in the activity. Table 1 summarizes these findings.

Of the 24 patients in the activity and control groups, 17 patients (70.83%) completed the questionnaire taken prior to beginning the activity. Table 2 summarizes the results from the questionnaire.

Independent samples t-tests results

When activity (M=5.8818, SD=1.17968) versus control (M=5.0083, SD=1.07995) serum phosphorus labs were compared at baseline in October, an independent samples t-test revealed that there was not a significant difference between the two groups (t=1.854, df=21, p=0.78). Likewise, when final serum phosphorus results were analyzed, an independent samples t-tests showed no significant difference (t= 0.92, df=22, p=0.927) between the activity (M=5.3636, SD=1.60641) and control (M=5.3077, SD=1.36349) groups. (Table 3 and 4).

Paired samples t-tests

Paired samples t-tests revealed that there was not a significant difference (t=1.191, df=10, p=0.261) between the baseline serum phosphorus (M=5.8818, SD=1.17968) and final lab results (M=5.3636, SD=1.60641) (Table 5 and 6).

Similar to the activity group, paired samples t-tests showed that there was not a significant difference (t=-.343, df=11, p=0.738) between the baseline serum phosphorus (M=5.0083, SD=1.07995) and final lab results (M=5.1167, SD=1.22907) in the control group (Table 7 and 8).

TABLE 3. Serum phosphorus in activity and control groups at baseline and December

	Group:	*N*	*Mean Phosphorus*	*Std. Deviation*	*Std. Error Mean*
Baseline October 2010	Activity:	11	5.8818	1.17968	.35569
	Non-activity:	12	5.0083	1.07995	.31175
December Phosphorus:	Activity:	11	5.3636	1.60641	.48435
	Non-activity:	13	5.3077	1.36349	.37816

TABLE 4. Independent sample t-tests for equality of means between serum phosphorus in activity and control groups at baseline and December

	t	*df*	*p*	*Mean Difference*	*Std. Error Difference:*	*95% Confidence Interval of the Difference*	
						Lower	*Upper*
October Baseline:	1.854	21	.078+	.87348	.47108	-.10618	1.85315
December Phosphorus:	.092	22	.927+	.05594	.60585	-1.20051	1.31240

+ No significance

TABLE 5. Mean serum phosphorus in activity group at baseline and in December 2010

		Mean	*N*	*Std. Deviation*	*Std. Error Mean*
Month:	October:	5.8818	11	1.17968	.35569
	December:	5.3636	11	1.60641	.48435

TABLE 6. Paired samples t-test results in activity group of serum phosphorus at baseline and December

	Paired Differences					*t*	*df*	*p*
	Mean	*Std. Deviation*	*Std. Error Mean*	*95% Confidence Interval of the Difference*				
				Lower	*Upper*			
October – December:	.51818	1.44348	.43523	-.45156	1.48793	1.191	10	.261+

+ No significance

TABLE 7. Descriptive statistics of mean serum phosphorus in non-activity group at baseline and in December 2010

		Mean	*N*	*Std. Deviation*	*Std. Error Mean*
Month:	October:	5.0083	12	1.07995	.31175
	December:	5.1167	12	1.22907	.35480

TABLE 8. Paired samples t-test results in non-activity group of serum phosphorus at baseline and December

	Paired Differences					*t*	*df*	*p*
	Mean	*Std. Deviation*	*Std. Error Mean*	*95% Confidence Interval of the Difference*				
				Lower	*Upper*			
October – December:	-.10833	1.09333	.31562	-.80300	.58634	-.343	11	.738+

+ No significance

Discussion and Limitations

The pre-activity questionnaire revealed that over half of the in-center patients forgot to take their binder medication during the week and on weekends. These results further strengthen the need for in-center education, programs, and activities to help increase binder medication compliance. These activities and programs are in addition to the usual nutritional activities and programs that educate on nutritional phosphorus (8-11).

Although the statistical findings did not reveal significance between the activity and control groups, there were trends noted in the analyses that are considered positive with regards to phosphorus control in the activity group. Serum phosphorus, at baseline, was abnormal (M=5.8818). Table 3 and 5 reveal that the activity group final mean (M=5.363) reached the K/DOQI goal of 3.5-5.5 mg/dL (4). It is interesting to note that the final mean (M=5.1167) in the control group increased from baseline (M=5.0083). Although small and still within the K/DOQI guideline, the serum phosphorus mean did not improve in the control group, in fact it increased. Despite the fact that there was not statistical significance between baseline and final means in the activity group, there was a positive correlation between wearing the binder bracelet and reaching the K/DOQI goal. With the divergent trends in the activity and control groups, this further strengthens the recommendation to use the binder bracelets to help with compliance.

There are some limitations to this study that must be kept in mind when interpreting these findings. Due to the rural location of the dialysis center, sample size was small (n=24) and ethnicity was 100% Caucasian. The demographics and small sample size restrict the ability to randomize the participants and increased the risk of allocation bias. If this study were repeated, suggested sample size would include several in-center HD clinics in both urban and rural settings. To further strengthen results and analysis, randomization between the activity and control group is suggested. Another limitation noted was the inability to monitor if patients wore the bracelets continuously throughout the day—as instructed at the start of the activity. It was noted that the activity participants wore the bracelets during dialysis, but there was no way to track the participants upon leaving the dialysis center. A post-questionnaire could help assess activity participation.

Despite the small sample size and limited results, the binder bracelet activity did reveal early positive results in helping patients reach and maintain serum phosphorus within the K/DOQI guideline.

Practical Applications: The binder bracelet activity can be used as an inexpensive yet effective reminder tool for dialysis patients. It can be part of an in-center activity or included in the initial nutrition education dialysis patients receive in the first 90 days of beginning dialysis.

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Thank You...

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Nutrition-focused Physical Examination: Skin, Nails, Hair, Eyes, and Oral Cavity

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Abstract

Although often underused as part of nutrition assessment, a nutrition-focused physical examination (NFPE) can help dietitians create a more effective care plan and determine appropriate interventions for patients receiving nutrition support. Laboratory findings are often inconclusive, and physical assessment can help to identify nutritional abnormalities. Dietitians can increase their proficiency in clinical skills by learning physical assessment techniques. The purpose of this article is to describe the relevance of performing an NFPE, with focus on skin, nails, hair, eyes, and oral cavity; its impact on patient care; and the role of the dietitian.

Introduction

An NFPE is an integral part of nutrition care planning and should be incorporated into the nutrition assessment performed by the dietitian. No single parameter can determine nutrition status and the degree of malnutrition. Therefore, a nutrition care plan is derived from a comprehensive medical record review, a detailed nutritional history, a review of laboratory test results, and an NFPE. The depth and focus of an NFPE is guided by the information gathered in the other areas of assessment (1). Often, the NFPE reveals new findings or may confirm information gathered from the medical record or patient interview. Specifically, an NFPE may aid in determining vitamin, mineral, trace element, and protein-energy deficiencies or excesses (1).

The NFPE is especially useful when whole blood, plasma, and serum concentrations of micronutrients do not correlate with total body concentrations (2). For example, more than 85% of zinc is found in skeletal muscle, bone, and liver, while only 0.1% is present in the plasma (3), making the results of standard laboratory measurements inconclusive. Zinc deficiency can have several manifestations, but it frequently appears as an inflammatory, dry, scaly rash around the mouth and nasolabial folds (4,5). Not relying solely on laboratory measures for determining deficiencies or toxicities of micronutrients is especially important in patients receiving parenteral nutrition (PN) because blood or serum sample concentrations may reflect the therapeutic infusion rather than the actual concentration of bodily micronutrients (2). Another problem associated with laboratory measurements of micronutrients is the contamination of blood samples due to inconsistent laboratory techniques and practices. Seamless laboratory procedures are vital for accuracy, and the need for industry standards has been recognized (2).

Results of an NFPE may help solidify the diagnosis of a nutrient deficiency or toxicity when laboratory measures are not available or are unreliable and should enhance patient care, leading to a more definitive nutrition intervention. Performing an NFPE can bring the dietitian into greater cohesiveness with the multidisciplinary team and promote communication with team members who use similar skill sets. For example, communication between registered dietitians and speech-language pathologists can ensure safe food and liquid intake to meet the nutritional requirements of the patient who has dysphagia (6). An NFPE can be applied to many parts of the body (Table 1); this article focuses on the skin, nails, hair, eyes, and oral cavity.

Table 1. Components of the Nutrition-Focused Physical Examination
• General survey
• Vital signs
• Skin
• Hair
• Nails
• Head and neck: – Head – Eyes – Nose – Neck
• Oral cavity: – Lips – Tongue – Mouth
• Teeth
• Respiratory system
• Cardiovascular system
• Abdomen
• Neurologic system

Getting Started

The NFPE follows a thorough assessment of the patient's medical and social history, a review of laboratory results, and a nutrition history interview. The NFPE can be a head-to-toe review of each body system or focus on a specific system based on information gathered from the medical record and patient interview (1,5).

Four techniques may be used during the NFPE: inspection, palpation, percussion, and auscultation. Inspection involves visual observation of color, shape, texture, and size. Palpation is the use of touch to examine location, texture, size, temperature, tenderness, and mobility. The tips and pads of fingers are used to assess pulsations and tenderness, and the back of the hand is used to assess temperature. Percussion is the tapping of fingers against body surfaces and listening for sounds that reflect solids, fluids, or gas. Auscultation is listening to sounds that reflect the movement of fluid or air through organs and viscera with a stethoscope. Auscultation is used during examination of the lung, heart, stomach, and intestines (4,7). Physical examination techniques can be learned from a variety of resources and refined through working closely with other trained professionals such as nurses, physicians, and physician assistants. The examination techniques most commonly used for evaluation of skin, nails, hair, eyes, and the oral cavity are observation and palpation.

Skin

Findings of the skin examination frequently reflect vitamin and mineral deficiencies because rapidly proliferating tissues such as the skin are believed to change simultaneously with the development of a nutritional abnormality (4,5). The skin is the largest organ of the human body, accounting for 15% of total body weight (7). It is composed of the epidermis, the outermost layer of skin; the dermis, which contains richly vascular connective tissue; and the hypodermis, composed of subcutaneous tissue and fat (7). The skin has several important functions, including providing a protective and mechanical fluid loss barrier, regulating body temperature, generating heat, providing a reserve of calories, and allowing sensory perception. The skin is also involved in metabolism, producing vitamin D precursors from cholesterol with exposure to ultraviolet light and excreting waste products such as sweat, urea, and lactic acid (7).

The skin is assessed for temperature, turgor, color, moisture, edema, rashes, lesions, wounds, ulcers, bruises, and hygiene (4,7). Several factors can affect skin integrity. For example, it becomes less elastic with advancing age and is altered by exposure to the sun/ultraviolet light (7). Some medications can cause rashes or edema. Alterations in nutrition can cause pigment changes, rashes, and overhydration or dehydration of the skin (1,5).

Skin inspection includes identification of lesions that may be primary, resulting from a spontaneous pathologic process, or secondary, occurring as a later evolution of a primary lesion or due to trauma (7). A decubitus ulcer is an example of a secondary lesion that has nutritional implications. Proper nursing skin care and the provision of adequate nutrition, especially protein, are necessary to prevent or heal ulcers and wounds (1). Decubitus ulcers occur with skin breakdown, usually over a bony prominence due to compromised blood flow (4,8). Factors attributed to the development of decubitus ulcers include pressure, friction, shear, moisture, and ischemia (8). Patients at risk for the development of decubitus ulcers are those who are bedridden or immobilized (7,9), those who have neurologic disorders or trauma (specifically spinal cord injury), and those undergoing lengthy operations (8). Further examples of nutrition-related skin lesions are outlined in Table 2.

The skin regulates body temperature and normally should feel cool to slightly warm to touch, with minimal perspiration (7). The hypersensitive dorsal portion of the hand should be used to assess temperature (4,7). Excess perspiration may be related to anxiety during the examination. Skin that is warm or hot to touch and associated with shaking chills, weakness, or thirst can be a sign of illness or infection (10). The basal metabolic rate may be increased with fever and dehydration, indicating that nutrition care plans may need to include adjustments of energy and fluids (4).

An important part of the NFPE is the assessment of hydration status, especially in patients receiving nonvolitional feedings such as enteral nutrition (EN) and PN. Skin is dry and clammy and has poor turgor during episodes of dehydration. A positive test of decreased skin turgor

Figure 1. Pitting edema in the left ankle.

Table 2. Physical Assessment Nutrient Chart

Body System	Physical Signs	Possible Nutrient Deficiency	Non-Nutritional Causes for Similar Findings
Skin/Nails			
Skin	Pallor: paleness	Iron (anemia)	Low-volume, low-perfusion states
	Poor, delayed wound healing	Protein, zinc, vitamin C, vitamin A	Peripheral vascular disease, arterial insufficiency
	Xerosis: abnormal dryness	Vitamin A, essential fatty acids	Environmental and hygiene factors, aging, hypothyroidism, uremia, ichthyosis (hypertrophy of outer skin layer that can be hereditary)
	Follicular hyperkeratosis: plaques around hair follicles	Vitamin A, essential fatty acids, vitamin C	Infection of hair follicle (*Staphylococcus*, fungal), Darier disease (hereditary), syphilis
	Perifolliculosis: pigmented plaques (thorax, abdomen, thighs, legs)	Vitamin C	Diabetic dermopathy (usually shins, toes, feet, ankles)
	Petechiae, ecchymosis: hemorrhagic spots on skin, mucous membranes	Vitamins K and C	Hematologic disorder, trauma, liver disease, anticoagulant overdose, Cushing disease
	Dermatitis, generalized	Zinc, essential fatty acids	Atopic dermatitis, contact dermatitis, allergic or medication rashes, psoriasis, connective tissue disease
	Pellagrous dermatitis: dermatitis with hyperpigmentation on areas exposed to sunlight, trauma	Niacin, tryptophan	Thermal, sun, or chemical burns; Addison disease; psoriasis
	Flaky paint dermatosis: hyperpigmented patches, usually backs of thighs, buttocks, that peel off to reveal hypopigmented skin	Protein	
	Poor turgor	Fluid (hydration)	
Nails	Koilonychia: thin, concave nails raised edges (spoon shape)	Iron, with or without anemia	Hereditary/congenital, infectious (fungal), endocrine (acromegaly, diabetes, hypothyroidism), hematologic (hemoglobinopathy, hemochromatosis) conditions, trauma, dermatitis (lichen planus, acanthosis nigricans, psoriasis), cardiopulmonary disease (clubbing of nails), carpel tunnel syndrome
	Lackluster, dull	Protein	Infection (lichen planus, *Candida albicans*), congenital (anonychia congenital), systemic lupus erythematosus
	Mottled, pale, poor blanching	Vitamins A and C	
	Splinter hemorrhages: distal ends of nails, multiple	Vitamin C	Septicemia (infective endocarditis), trauma, skin disorders (psoriasis), hemodialysis, hemochromatosis, vascular disease
	Ridging, transverse: more than one extremity	Protein	Beau lines/grooves caused by trauma, coronary occlusion, skin disease, hypercalcemia, transient illness
Head/Neck			
Skull	Craniotabes (<1 year of age): softening of occipital, parietal region	Vitamin D	
	Frontal or parietal bossing (infants): swelling or thickening of front and sides of head	Vitamin D	
	Open anterior fontanelle (persistent at >18 months of age)	Vitamin D	Hydrocephalus
Hair	Easily plucked, thinness, sparseness, lackluster	Protein, essential fatty acids	Male pattern baldness, hypopituitarism, hypothyroidism, cancer therapy, chemical alteration, infection, psoriasis, Cushing disease, medication (heparin, radiomimetics)
	Flag sign: alternating bands of depigmentation (in children, rare)	Protein; alternating periods	Chemical alteration
	"Corkscrew" hair	Copper (Menkes disease)	Chemical alteration
	Depigmentation: lightening of normal hair tint	Protein, copper	Chemical alteration
	Straightness in cultural groups with normally curly hair	Protein	Chemical alteration
Nose/Face	Diffuse depigmentation	Protein/energy	
	Nasolabial seborrhea: scaling around nostrils	Vitamin B-2, niacin, vitamin B-6	Tuberous sclerosis (epiloia)
	Moon face: rounding of the face	Protein/energy	Cushing's disease
	Temporal wasting (bilateral)	Protein/energy	Motor neuron diseases, congenital lipodystrophy
Eyes	Night blindness	Vitamin A	Tapetoretinal degeneration (e.g., retinitis pigmentosa), status postpanretinal photocoagulation (for diabetic retinopathy), photic exhaustion (i.e., all day in bright sun; short-term effects), advanced glaucoma
	Angular palpebritis (blepharitis): inflammation of lid margins/corners	Vitamin B-2, niacin, vitamin B-6	Infection (*Staphylococcus, Moraxella*), allergic reaction, seborrheic dermatitis
	Conjunctival xerosis: abnormal dryness	Vitamin A	Chemical or environmental irritation
	Pale conjunctivae (fornix area)	Iron, folate, or vitamin B-12 anemia	Low-output states
	Bitot spots: shiny gray spots on conjunctiva	Vitamin A	Pinguecula (elderly), Gaucher disease (hereditary), pterygium
	Corneal xerosis: abnormal dryness, progresses to keratomalacia	Vitamin A	
	Keratomalacia: hazy, dry, softened cornea; usually bilateral	Vitamin A	Band keratopathy (hyperthyroidism), interstitial keratitis, sclerosing keratitis
Lips/Mouth/ Tongue/Gums	Cheilosis: dry, swollen, or ulcerated lips	Vitamins B-6 and B-2, niacin, iron (severe deficiency)	Environmental exposure, herpes
	Glossitis: inflammation of the tongue, magenta (purplish-red) color	Vitamins B-2 and B-6, niacin, folate, vitamin B-12, iron (severe deficiency)	Crohn's disease, uremia, infectious disease (monilial), antibiotics, malignancy, anticancer therapy, mechanical trauma, irritants (excess tobacco, alcohol, spices), generalized skin disease

(Continued on next page)

Table 2. Physical Assessment Nutrient Chart – *continued*

Body System	Physical Signs	Possible Nutrient Deficiency	Non-Nutritional Causes for Similar Findings
	Bleeding, spongy gums	Vitamin C	Gingivitis (due to poor hygiene, malocclusion, dental calculus), amyloidosis, acute myeloid leukemia, drugs (e.g., phenytoin, nifedipine), periodontal disease
	Stomatitis: general inflammation of oral mucosa (palate, tongue, mouth floor, gingivae, buccal and labial mucosa)	Vitamin B complex, iron, vitamin C	Infection, mechanical trauma, dryness with aging, autoimmune disorders, cancer therapy, irritants (mouthwashes, toothpaste, hot and spicy foods, tobacco), drug therapy (e.g., phenytoin)
	Angular stomatitis: fissures/lesions in corners of mouth, bilateral	Vitamin B-2,niacin, iron, vitamin B-6	Irritation from poor denture fit, herpes, syphilis, chapping from harsh climate, infection (monilial)
	Edematous tongue	Niacin	Large, fleshy tongue due to acromegaly, myxoedema, primary amyloidosis
	Atrophic filiform papillae: tongue is smooth or slick	Niacin, folate, vitamin B-2, iron, vitamin B-12	Non-nutritional anemias, anticancer therapy
	Dysgeusia: distorted taste; Hypogeusia: diminished taste	Zinc	Cancer, anticancer therapies, aging, dryness of mouth due to heavy smoking, Sjögren syndrome, following acute influenza, radiation to head and neck, diabetes mellitus
Teeth	Mottled teeth: whitish opaque-to-severe brown discoloration	Fluoride excess	Tetracycline use during development, high fever during odontogenesis, rickets, cystic fibrosis, gingival calculus, smoking, coffee consumption, chlorhexidine mouthwashes, red wine
	Caries: tooth decay	Vitamin C	Eating disorders, xerostomia, radiation, poor dental hygiene
	Loose or missing teeth		Trauma, aging, malocclusion, periodontal disease
Neck	Enlarged thyroid	Iodine	Allergic or inflammatory process, Graves disease, malignancy, thyroglossal duct cyst, thyroiditis, bronchial cleft cysts and tumors
	Enlarged parotid (bilateral)	Protein, bulimia	Mumps, portal cirrhosis, Sjögren syndrome (usually females), allergic or inflammatory process, neoplasm, sarcoidosis, sialolithiasis (salivary duct stone)
Thorax	Thoracic deformity ("pigeon chest" or Harrison's sulcus"): horizontal depression along lower border of chest	Vitamin D	
	Rachitic rosary: costochondral beading	Vitamin D, calcium	Renal disease, diabetes mellitus, pseudohypoparathyroidism
Heart	Pitting edema: bilateral	Protein (albumin<2.5 to 3.0 g/dL), thiamine, vitamin C	Protein-losing enteropathy, varicose veins, venous obstruction, sodium and water retention states (e.g., heart failure, renal failure, pregnancy)
	Cardiac enlargement	Thiamine	Infection (bacterial, viral, fungal), granulomatous diseases, neuromuscular and neurologic disorders, heart disease, endocrine disorders (diabetes, hyper- or hypothyroidism, acromegaly), neoplasm, connective tissue disorders
	Tachycardia	Thiamine, fluid (dehydration)	Organic heart disease, severe and chronic lung disease, respiratory insufficiency, drug toxicity (digitalis, theophylline), excess alcohol ingestion
	S3 presence (with edema, dyspnea, loud crackles that do not disappear with cough)	Fluid excess	
Abdomen	Hepatomegaly: enlarged liver	Protein	Acute hepatitis, fatty liver, early biliary obstruction, passive congestion, malignancy
Musculoskeletal	Fat/muscle wasting: wasting in thorax, sacral and/or temporal regions; fine muscles of hand; prominent body skeleton	Energy/protein	Neuromuscular disorders
	Rickets/knock knees/bowleg	Vitamin D, calcium, phosphate	Congenital deformity, renal rickets
	Epiphyseal enlargement (ends of long bones)	Vitamin D (painless), vitamin C (painful)	Trauma, congenital deformity, renal disease
	Swollen, painful joints	Vitamin C	Arthritis
	Dwarfism/Hypogonadism	Zinc	Congenital hypogonadism, chronic renal failure, pituitary tumor, idiopathic hypopituitarism, growth hormone deficiency
Neuorologic	Motor weakness (lower extremity, bilateral)	Thiamine	Polyneuropathy of diabetes mellitus, weakness due to myogenic disorders
	Mental confusion, hyperirritability, apathy	Protein	
	Peripheral neuropathy: weakness, paresthesias, bilateral	Thiamine, vitamins B-12 and B-6	Lyme disease, polyneuropathy of diabetes mellitus, collagen vascular diseases
	Vibratory sense decrease, loss of position sense, loss of ankle or knee jerks (bilateral)	Thiamine, vitamin B-12	Hypothyroidism, sarcoidosis, amyloidosis, uremia, malignancy
	Tetany: lips, tongue, fingers, feet; generalized muscle aching; carpopedal, facial musculature spasm	Calcium, vitamin D	Respiratory alkalosis
	Calf tenderness: bilateral	Thiamine	Deep vein thrombosis, peripheral neuropathy of other causes
	Dementia	Niacin, vitamin B-12	Head trauma, cardiac arrest, cerebral hemorrhage, brain tumor, degenerative brain diseases (e.g., Huntington, Parkinson, Alzheimer disease, multiple sclerosis, amyotrophic lateral sclerosis.)

This chart is not intended to be a complete list of nutritional or non-nutritional causes for presenting physical signs and symptoms. Adapted with permission from material from Jean Rindal, BS, RN, MA, ANP.
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Case #1 Dehydration

A 30-year-old woman who has a history of Crohn's disease that has involved multiple small bowel resections and an enterocutaneous fistula has been receiving home parenteral nutrition (HPN) for 1 year due to short bowel syndrome, with approximately 125 to 150 cm of small bowel to an end ileostomy. The HPN prescription is 150 g protein, 305 g dextrose, and 300 mL 20% (60 g) lipids two times per week in a 3-L volume. The home care nurse reports to the dietitian that the patient complains of headache, lightheadedness, weakness, and dry mouth. She also reports an elevated heart rate of 107 beats/min, decreased blood pressure of 92/56 mm Hg, and decreased weight of 105 lb (47.7 kg). Her baseline weight was 110 lb (50 kg). The patient reports that she ran out of her antidiarrheal medications 3 days ago. The dietitian asks the home care nurse to gather the patient's intake and output records and obtain laboratory work. Assessment of data indicates that the patient is dehydrated. Intravenous normal saline (NS) is sent to the patient's home by the home infusion pharmacy, and the patient is instructed to infuse 1 L/day of NS for 3 days for fluid resuscitation in addition to her HPN formula. Antidiarrheal medications are resumed and the patient returns to a normal hydration state within 4 days.

	Intake (mL)	Output (mL)	Net Balance# (mL)	Weight (lb) (kg)	BUN (mg/dL) normal: 8 to 25 mg/dL	Creatinine (mg/dL) normal: 0.7 to 1.4 mg/dL	Sodium (mmol/L) normal: 132 to 148 mmol/L	Hemoglobin (g/dL) normal: (%) 11.5 to 15.5 g/dL	Hematocrit normal: 36% to 46%
Current	Oral: 1,560	Urine: 670	-1,590	105 (47.7)	52	2.1	147	14.5	42
	HPN: 3,000	Stoma: 4,150							
Baseline	Oral: 1,480	Urine: 1,530	+950	110 (50.0)	24	0.9	140	14	38
	HPN: 3,000	Stoma: 1,705							
Corrected*	Oral: 1,680	Urine: 1,640	+620	109 (49.5)	22	1.1	142	14.1	40
	HPN: 3,000	Stoma: 2,060							

*3 L of NS infused in addition to HPN formula for three consecutive days.
#Values calculated based on the assumption of: 500 mL (insensible losses), 1,500 mL (adequate urine production), and stoma output.

is when the skin on the back of the hand, forearm, or the chest that is gently pinched to form a tent does not spring back rapidly into place when released (4,7). Note that the pinch test should not be attempted on elderly patients who have extremely thin skin that bruises easily (7). Other clinical symptoms of dehydration include decreased weight, low urine output, sunken and dry eyes, dry mucous membranes, and sticky saliva. Decreased blood pressure; increased heart rate (pulse); and elevated serum sodium, chloride, blood urea nitrogen, hemoglobin, and hematocrit values may also correlate with the diagnosis of dehydration (see Case 1- Dehydration). Nutrition care plans need to be adjusted to resolve the fluid deficits and provide maintenance fluid (4). Skin that is moist, edematous, and puffy around the eyes may be due to excess fluid accumulation in the tissues. Edema is most notable in dependent body parts such as the feet, ankles, and sacrum. Skin that is edematous retains a thumb or finger print indentation, referred to as pitting edema (4). Pitting edema or orthostatic edema is often associated with right-sided heart failure that leads to increased fluid volume and elevates the hydrostatic pressure in vascular space, causing accumulation of fluid in dependent body parts (Fig. 1). The severity of pitting edema may be assessed by using a grading scale of 1+ to 4+. 1+ suggests the indentation is about 2 mm and the indented space fills in rapidly; 4+ edema leaves an 8-mm indentation and refills in 2 to 5 minutes. In 4+ edema, the dependent parts of the body are grossly fluid-filled and taut. Non-pitting edema is so tense that the skin cannot be indented and can be an indication of circulatory disorder or may indicate lymphedema (7). Increased weight; high urine output; shortness of breath; increased blood pressure; and decreased serum sodium, chloride, blood urea nitrogen, hemoglobin, and hematocrit also can indicate overhydration (see Case 2- Overhydration). Edema may be caused by a number of conditions. Provision of excess fluids, systemic causes such as heart failure and renal insufficiency, and venous stasis during pregnancy or immobilization may cause edema (1). Hypoalbuminemia associated with critical illness and protein-losing enteropathy may cause a reduction in colloid-osmotic pressure with a corresponding shift of intravascular fluid into extravascular spaces, resulting in edema (4). It is important to discern the cause of edema to make appropriate alterations to the nutrition care plan.

Nutrition Support Access Devices

Dietitians caring for patients receiving nutrition support should evaluate nutrition support access devices (feeding tubes and venous catheters) as part of the NFPE. Malfunctioning, malpositioned, or infected devices affect the ability to deliver EN or PN safely and adequately. Inspection of the skin surrounding

devices should reveal no erythema, swelling, or drainage (11). Nutrition access devices that are torn or lacerated should be repaired or replaced as soon as possible. Broken feeding tubes or catheters or improperly cleaned nutrition access devices can lead to systemic infections (Figs. 2 and 3) that may require antibiotic treatment or hospitalization and can contribute to an increased risk of mortality (11).

Figure 2. Percutaneous endoscopic gastrostomy site infection.

Figure 3. Peripherally inserted central catheter exit site infection.

Nails

The nails should be inspected for color, length, configuration, symmetry, and cleanliness. Nail hygiene can reflect a patient's self-care and emotional order (7). Nails provide many clues to nutritional deficiencies as well as systemic diseases (Table 2). Findings that suggest nutritional abnormalities likely can be attributed to more than the nutrient deficiency (1,12).

The nail plate is composed of keratin, a fibrous protein, and should be firmly adherent to the nail bed, feel smooth, and appear uniformly thick and symmetric (12). It is normally translucent, with a pink hue derived from a complex capillary system located underneath the nail plate (12). To assess for circulation or capillary refill time, palpate the nail by gently squeezing between the thumb and forefinger. The nail blanches white and should return to its original pinkish color almost immediately. Normal capillary refill time is less than 3 seconds (4). Bleeding could indicate malnutrition, and nails that blanch poorly could indicate vitamin A or C deficiency (Table 2) (4,5). Any tenderness with palpation may indicate ischemia. Flaky nail plates may indicate deficiency in magnesium. Selenium is also believed to contribute to the health of the nail plate (12).

Changes in the shape or structure of the nail plate, which is normally flat or slightly convex, can be related to nutritional deficiencies. Concave or spoon-shaped nails (also called koilonychia) are associated with iron deficiency anemia (1,12). Beau lines or transverse grooves in the nails have been associated with protein deficiency and hypocalcemia but also can be caused by trauma, coronary occlusion, or transient illness. Small splinter hemorrhages can indicate scurvy or hemochromatosis (12).

The nail base angle is normally measured at 160 degrees. Clubbing, in which the nail fold and nail plate angle exceed 180 degrees, is associated with respiratory disorders (e.g., cystic fibrosis), cardiovascular diseases, cirrhosis, and colitis (7,12). These disorders are frequently associated with malnutrition (12).

Hair

The hair is inspected for color (consider chemically applied treatments), pigmentation, distribution pattern, shine, texture, and quantity (4). Healthy scalp hair is shiny, smooth, resilient, and not easily plucked (7). Poor hair qualities are associated with protein, zinc, essential fatty acid, and biotin deficiencies. Although rare, alternating hypo/hyperpigmented bands of hair are indicative of intermittent periods of protein deficiency and protein adequacy in children (Table 2) (1).

Alopecia (hair loss) is often linked with zinc deficiency, lack of protein, and lack of biotin (4,5). More diffuse alopecia, including hair loss from eyebrows, has been linked to essential fatty acid and selenium deficiency. Selenium deficiency also has been found to lighten hair color (13). In the elderly, "corkscrew" or "looped" hair on arms, legs, and trunk is related to follicular hyperkeratosis that occurs in scurvy (5,13). Hypertrichosis is defined as an increase in fine hairs, also known as lanugo. Such fine hair occurs on the back, abdomen, and forearms. It becomes more fragile and more easily plucked as energy deficiency prevails in the patient who has anorexia or bulimia (13). Iron deficiency has been shown to stimulate alopecia or result in stunted hair growth (13).

Case #2 Overhydration

A 56-year-old man who has esophageal cancer and underwent esophagectomy and percutaneous endoscopic gastrostomy (PEG) tube placement during hospital admission demonstrates gastric tolerance of a 1.2 kcal/mL of full-strength polymeric feeding. His PEG feeding is advanced to 65 mL/hr over 3 days, meeting his daily requirements of 1,800 kcal and 80 g of protein. Water flushes of 40 to 50 mL every 4 hours are given per nursing protocol. He is meeting his fluid requirements of 1,600 mL/day. The patient continues to receive intravenous fluids of normal saline at 100 mL/hour after surgery and during PEG feeding advancement. The dietitian assessing the patient at bedside notes sacral and bilateral lower extremity edema and swollen hands. She reviews the bedside flow sheets and notes the patient's elevated heart rate of 90 beats/min and elevated blood pressure of 146/94 mm Hg. The dietitian discusses the findings with the physician and maintenance fluids are discontinued.

	Intake (mL)	Net Balance# (mL)	Weight (lb) (kg)	BUN (mg/dL) normal: 8 to 25 mg/dL	Creatinine (mg/dL) normal: 0.7 to 1.4 mg/dL	Sodium (mmol/L) normal: 132 to 148 mmol/L	Hemoglobin (g/dL) normal: 11.5 to 15.5 g/dL	Hematocrit (%) normal: 36% to 46%
POD 3 (Current)	TF: 1,560 IVF: 2,700	+2,436	127 (57.7)	10	0.94	131	9.8	33
POD 2	TF: 1,320 IVF: 2,700	+2,232	121 (55.2)	24	1.1	137	11.5	37.5
POD 1	TF: 720 IVF: 2,700	+1,830	117 (53.1)	27	1.2	140	12.3	38
Corrected*	TF: 1,560 IVF: 300	+36	119 (54.1)	22	1.0	139	12.1	40

IVF=intravenous fluids, POD=postoperative day, TF=tube feeding
*Maintenance fluids discontinued #Values calculated based on the assumption that estimated fluid needs were based on 30 mL/kg of body weight.

Orofacial Examination

Examination of the eyes, face, lips, and oral cavity can reveal many nutritional deficiencies and correlate with findings of the skin, nails, and hair (Table 2). Structures of the face should appear symmetric. A "moon face" or rounding of the face may be due to Cushing's disease or administration of adrenal cortical hormones (5). Bilateral temporal wasting is seen in severely malnourished patients and, to a lesser degree, in aging patients. This should be carefully assessed and not be mistaken for malnutrition in the aging population (1). As skin ages, features change dramatically, including atrophy and thinning of epithelial and fatty skin layers, and results in loss of elasticity in the dermis (14). Many cosmetic treatments, such as botulinum toxin injections, microdermabrasion, filler injection, chemical peeling, and surgical procedures, are being performed to reduce the signs of aging (15). Other sites of facial wasting may appear in the forehead, periorbital region, buccal, and inner line of the nasolabial folds (15).

Nutritional deficiencies of the eyes can result from vitamin A deficiency and manifest as night blindness or may be observed as shiny gray spots on the eye conjunctiva (Bitot spots) (1,5). Vitamin A deficiency may also cause abnormal dryness of the cornea and progress to keratomalacia or hazy, dry, softened corneas (4).

Lips that are dry, swollen, or ulcerated can be associated with vitamin B-6 (pyridoxine), B-2 (riboflavin), or niacin deficiency. Environmental exposure can cause similar results and should be considered when assessing the lips. Bilateral fissures at the corners of the mouth (angular stomatitis) can also be caused by deficiencies of B vitamins such as riboflavin, pyridoxine, or niacin (1,5).

Examination of the gums, teeth, and tongue can reveal telltale signs of nutritional deficiencies. It is the position of the American Dietetic Association that nutrition is an integral component of oral health. The American Dietetic Association supports the integration of oral health with nutrition services, education, and research. Collaboration between dietetics and dental professionals is recommended for oral health promotion and disease prevention and intervention. Oral infectious diseases as well as acute, chronic, and terminal systemic diseases with oral manifestations impact the functional ability to eat as well as diet and nutritional status (16). Gums that are spongy, red, and inflamed can be associated with vitamin C deficiency (1,5) but also may be due to gingivitis or periodontal disease resulting from infection or poor oral hygiene (17). Similarly, dental caries and tooth decay may result from a diet

containing excessive sugars, eating disorders, and poor dental hygiene (1,17).

Anorexia and bulimia can cause erosion of the enamel of the anterior teeth from chronic acid exposure through vomiting (5). Skin lesions of the fingers used to induce vomiting may also be present. Bilateral or unilateral parotid gland enlargement reflects repeated episodes of purging. Parotid gland enlargement can be a useful indicator in diagnosing bulimia and may eliminate further testing (18).

As part of the orofacial examination, the tongue should be observed for inflammation or glossitis, which can be associated with one or more B vitamin deficiencies. A pale, smooth tongue may indicate iron or folate deficiency (1).

Conclusion

Dietitians need to include an NFPE as part of their nutrition assessment of patients. A nutritional deficiency or toxicity may go undiagnosed without the NFPE, making the nutrition care plan incomplete. Physical assessment techniques should become part of the dietitian's skill set, which can enhance the dietitian's value as a member of the clinical team. Examination of the rapidly proliferating tissues of the skin, hair, nails, eyes, and oral cavity may reveal nutritional deficiencies or toxicities. Rashes, skin color changes, and nail and hair abnormalities that correlate with findings from the medical history, nutrition interview, or laboratory measures may lead to a definitive nutritional diagnosis and care plan.

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A report from the NKF Spring Clinical Meeting 2012: Implications of Dietary Additives in Kidney Disease

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Recipient of an RPG educational stipend for the 2012 National Kidney Foundation Spring Clinical Meeting in Washington D.C.

Janeen Leon MS, RD, LD from the MetroHealth Medical Center in Cleveland, Ohio presented the phosphorus additives segment of a panel discussion, explaining how these are used to retain moisture, improve color and texture, enhance flavor, and extend shelf life of foods. Natural dietary phosphorus sources provide 800-1000 mg of phosphorus a day. An egg yolk has 66 mg phosphorus, while the egg white has only 5 mg phosphorus. Meat, poultry and fish have about 180-300 mg phosphorus per 3 ounce serving.

Ms. Leon showed the results of extensive research on fast food items and their phosphorus content from additives, referencing studies which she co-authored in the 2008 Journal of Renal Nutrition (1) and some from unpublished data. For example, she found that fast food popcorn chicken has 6 phosphorus additives, providing 380 mg phosphorus per serving. She also reported that the phosphorus content of a fast food meal of three chicken strips, a biscuit, and side of potato wedges would need 22 calcium acetate pills to bind the total amount, although she suggested trying to cover half the phosphorus amount with binders would be realistic because of dialysis phosphorus removal. Chicken strips or nuggets are generally higher in additives than a regular chicken sandwich – that is FAST FOOD. Fast food chicken has more additives than fast food burgers. Her soon-to-be-published study found that Yoplait Light is lower in additives than regular Yoplait (which is calcium fortified with tricalcium phosphate) and that one slice of Kraft Singles American cheese has 148 mg phosphorus. Phosphorus containing food additives are plentiful, cheap and widely consumed. The current food supply and labeling practices make phosphorus additive avoidance tricky. Binders can't control phosphorus with unlimited diet, therefore patients need our clinical guidance to avoid additives. The renal dietitians' job is ever more tedious.

She concluded her talk with some depressing information – that is if you are a dialysis patient. In studies she referenced, one Sevelamer 800 mg tablet binds approximately 27 mg of phosphorus, one calcium acetate tablet binds 30 mg of phosphorus, and one Fosrenol 1000 mg tablet binds 90 mg of phosphorus.

After hearing during this lecture that higher serum phosphorus, even within the normal range, is a potential danger to the general public, I was interested in finding more literature on the topic. Since humans do not have the enzyme phytase, the biological availability of plant-based phosphorus is less than 50% in contrast to an estimated 70% from animal protein (2). Animal-based foods are abundant in organic phosphorus. These include dairy products, meat, poultry and fish. Organic phosphorus is hydrolyzed in the intestinal tract and then absorbed into the circulation as inorganic phosphate. Usually only 40 to 60% of organic dietary phosphorus is absorbed. When instructing the dialysis patient, type of dietary phosphorus should be emphasized, as phosphate additives may contribute as much as 1,000 mg phosphorus a day to the average American diet (2). Plant-based phosphorus is not as readily absorbed as animal-based.

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CSR Exams have been updated!
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NKF Inflammation Workshop Review

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Recipient of an RPG educational stipend for the 2012 National Kidney Foundation Spring Clinical Meeting held in Washington D.C.

The NKF Spring Clinical meeting offered a day-long pre-conference session entitled "Advanced Practice In Renal Nutrition: Update 2012 (Strategies II)". The session was designed to integrate new research into clinical practice and stimulate thought on how to improve the nutritional status of patients with chronic kidney disease (CKD).

As we know, inflammation is an acute phase response that is important to health, a protective response to injury, which sustained over time is detrimental to patients with CKD. There is no consensus on which marker of inflammation to use or the threshold of the marker. C-reactive protein (C-RP) is easily measured and often used in studies. When C-RP is > 3, there is a 50% increased risk of death. Although there is no solid evidence warranting routine testing and only 1% of US centers measure it, C-RP can point to fluid problems, compromised access sites, and can be useful for clinical decision-making. The presenter recommended that practitioners should consider white blood cell count levels, stating it is not the greatest indicator but it is more readily available.

Approximately twenty five percent of all patients with CKD are severely inflamed, and at the highest risk for cachexia and wasting. Catabolism of both adipose and muscle tissue contributes to inflammation, which also has direct catabolic effects. The dialysis treatment causes net muscle loss. Catheters and thrombosis lead to increased inflammatory response; catheters impact C-RP levels even without infection. Metabolic acidosis and high parathyroid levels contribute to protein breakdown. Growth hormone metabolism, insulin resistance and testosterone deficiency are all impacted by inflammation.

Inflammation is a catalyst for other risk factors, further stimulates cytokine production, influences response to medical treatment (statins are ineffective in a patient with inflammation), increases susceptibility to atherosclerotic thickening by causing calcification, and promotes malnutrition. The triad of inflammation, cardiovascular disease and malnutrition leads to increased risk of death. Inflammation affects the satiety center and leads to decreased intake, which increases the chance of death in one year by 30%. Dental problems, oral thrush, problems with gastrointestinal absorption, decreased sense of smell, poor sleep quality and depression all lead to poor appetite, and inflammation is related to all of them. Treating depression can improve sleep quality and thereby improve nutritional status and inflammation.

According to the presenter, there is no stronger correlation to patient outcomes than that of serum albumin. There is an 88% higher risk of death with an albumin of 3.8 versus 4. Every 0.1 change is important and represents a 10-20% change in mortality. Higher albumin and higher creatinine levels are correlated with better quality of life. High cholesterol is better for dialysis patients than a level below 140 mg/dL. Low cholesterol causes an increased cytokine release due to a binding problem and therefore is linked with increased inflammation.

Persistent inflammation can be targeted by lifestyle/nutritional changes and medications. Dietitians need to embrace inflammation and ask "what do I need to do to improve this?". They should perform a nutrition-focused physical exam (SGA). The simple question "have you been bothered by lack of appetite?" can be telling. RDs can assess the potential for inflammation, seeking information on access site, temperature, co-morbid conditions, and dental problems. They can identify causes of inflammation including skin breakdown, wounds, rashes, fever, inflamed gums, and urinary tract infections. We can help to reduce or eliminate sources of inflammation by making or requesting referrals to a dentist, recommending consults for wound or access care, suggesting low dose antibiotics, recommending omega-3 fatty acids, and recommending physical therapy referrals. The protein-energy wasting that is related to dialysis is complex. Nutrition alone cannot reverse it, but as dietitians we can evaluate it and then enlist the help of our patient care team.

We can feed people more protein and calories, but muscle building cannot occur without a stimulus. That stimulus is exercise. Of the three types of exercise, resistance exercise is related to reduced inflammation. The question was asked "who gives the exercise advice?". The answer was basically, "no one". An RD can fill that gap by initiating the conversation, assessing physical activity habits, and giving general advice. Make exercise part of the assessment checklist and reassess annually. Practical tips for increasing exercise include: getting people outside of the gym, making exercise recording a part of the food diary, setting exercise goals with clients, suggesting exercise prior to a dialysis treatment, making exercise part of the meal plan and following up on exercise compliance during follow up visits.

All presenters agreed that what we can do is find the cause of inflammation, including potential dialysis-related causes, and

treat them. Nutritional interventions discussed by one or more presenters included high fiber, low fructose, low saturated fat, antioxidant rich diet, vegan diet (even for a short term), 2-3 servings of wild-caught fatty fish weekly, green tea extract and the use of oral nutritional supplements. The need for resistance exercise was stressed. Non-specific treatments with anti-inflammatory results included statins, sevelamer, losartin, ACE, nutritional vitamin D, and appetite stimulants. The panelists also mentioned that carnitine may also be anti-inflammatory and anti-cytokine therapy (barboxolone) is on the horizon.

We don't know if improving inflammation will improve outcomes, but we do know how to reduce inflammation. The treatment of protein energy wasting and inflammation needs to be done at multiple levels and the dietitian is one of them. The most promising treatment is most likely a combination of therapies that have failed on their own independently.

Dr. Alison Steiber noted that patients taking renal vitamins had 16% lower mortality risk than those not taking vitamins. Her recommendations:

- Vitamin C – most patients are deficient but intake should not exceed 500 milligrams.
- Vitamin E – should not supplement due to its association with heart problems.
- Vitamin K – give as vitamin K2.
- Vitamin A – there is insufficient information to recommend increased intake.
- Selenium – CKD patients are deficient and may be able to meet their needs with 1 Brazil nut daily.
- Zinc – plasma levels are generally low and patients on very low protein diets should be supplemented. However, study results regarding zinc supplementation have not been statistically significant.

NKF Kidney Stones 101 Workshop Review

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The Kidney Stones 101 Workshop was held during the National Kidney Foundation Spring Clinical Meeting in May 2012. The workshop was a three part presentation on basic diet and medication intervention for the treatment of kidney stones. Following is a summary of the first part, presented by R. Allen Jhagroo, M.D., which briefly addressed dietary factors in stone formation.

The diagnosis of kidney stones has increased over the past 30 years, both in the U.S. and internationally. Approximately 90% of kidney stones are calcium-based, with the majority of these (58.8%) being calcium oxalate stones. However, uric acid stones are increasing in prevalence, likely because insulin resistance is a risk factor. Insulin resistance leads to renal lipotoxicity and impaired NH3 synthesis, resulting in reduced NH4 excretion. With fewer NH3 molecules around to bind with, H+ ions increase, reducing urine pH and creating favorable circumstances for uric acid to precipitate out and form stones.

Kidney stones form for a variety of reasons. People who are stone-formers have high variations of stone-promoting constituents in their urine, compared with non-formers. They may have higher calcium excretion, higher oxalate excretion and/or low urine volumes.

Dr. Jhagroo briefly addressed the following factors and how they relate to stone formation:

Fluid

An adequate intake of fluid may be more important than any other dietary modification. Active kidney stone-formers can become NON-active stone-formers when they achieve > 2 liters/day urine output. One exception to this may be uric acid stone formers. For them, alkalinizing the urine takes precedence over increasing fluid intake. Because fluid losses also occur via sweat, stool and respiration, actual intake of fluid must exceed 2 liters/day to achieve this output. Typically 3 liters intake/day is recommended, but both climate and work environments (e.g., if very hot and patient sweats more) need to be considered in determining individual recommendations for total daily fluid intake. Some general guidelines should be followed:

- Drink enough that you get up at night to urinate
- Don't wait until you are thirsty to drink
- Urine should be pale in color

Calcium

A long-held misconception is that because (most) kidney stones contain calcium, and because urinary excretion of calcium may be elevated in many stone-formers, dietary calcium should be restricted. However, a normal calcium intake can help prevent kidney stones and, in high-risk groups, a low calcium intake actually increases stone formation. In addition, low calcium intakes increase osteoporosis risk. Calcium intake should reach Dietary Reference Intake levels, but should not be excessive. Promoting the inclusion of milk/dairy at each meal can be beneficial in reducing stone formation.

Sodium

A high salt intake leads to higher urinary excretion of both sodium and calcium. Limiting sodium, when combined with a normal calcium intake and with animal protein intake that is not excessive, is associated with lower stone recurrence.

Oxalate

As noted previously, the majority of stones are calcium oxalate. Consequently, stone formers are often advised to restrict their oxalate intake. However, we absorb only ~10% of the oxalate ingested. The more oxalate we eat, the less we absorb, so dietary adjustments may not be all that effective. It is somewhat difficult to restrict oxalate, as it is so widespread in foods. However, it can be helpful to add calcium to the diet as it binds oxalate present in other foods.

Patients who have had gastric bypass surgery are at risk for forming calcium oxalate stones, as they may have some fat malabsorption, leaving a greater presence of fats in the bowel. These fats bind with calcium, leaving more oxalate unbound, and thus, free to be absorbed. The type of bariatric surgery should be noted, as those with restrictive procedures have little change in their urine oxalate excretion whereas roux en y gastric bypass patients tend to have a high incidence of stone formation. [See sidebar for further discussion of oxalate and dietary limitation of high oxalate foods.]

Lemonade

Lemonade has been recommended for stone-formers because of its citrate concentration. Citrate is filtered by the kidneys and then reabsorbed, based on pH. Urinary citrate excretion will increase when pH is low (acidic). It will decrease when pH is high (alkaline). To make a difference in systemic acid-base balance, the amount of citrate provided must be sufficient to result in a 600 mg change in citrate urine excretion. The citrate present in fruits and vegetables generally results in a change of only about 200 mg. Typical lemonade is not acidic enough to effect much more change than this either. To reach the 600 mg change, it is necessary to consume a lemonade mix of ½ cup lemon juice in 7 cups of water.

Protein/Meat

Cutting back on animal protein may be beneficial for calcium oxalate stone-formers, as well as for those who form uric acid stones. Not only red meats, but also poultry, fish and cheese produce high acid loads. In addition, high protein intakes increase calciuria and uricosuria and decrease citrate excretion. [The speaker did not specify recommended amounts of protein, but a review of diet in kidney stones by Borghi et al suggests limiting protein from animal flesh to 21 grams per day and protein from milk and dairy to 31 grams per day for a 70 kg male (1).]

Dietary Oxalates and Kidney Stones Facts

Lynn Munson, MS, RD, LD

In her 2007 review article, Massey points out that, despite the long-held belief that only 10% of ingested oxalates are absorbed, Holmes, et al observed that 24 to 53% of urinary oxalate originated from dietary oxalate when intake was 10 to 250 mg per day (2-3). Stone formers may absorb more oxalate than non-stone formers (4).

Determining exact oxalate intake is somewhat difficult. There is variability in food oxalate values, depending on the analytical methods used (2). In addition, boiling vegetables in water may decrease oxalate content 30 to 87% through loss of soluble oxalate, as long as the cooking liquid is not consumed (5). Finally, the different parts of a plant as well as genetic differences between cultivars and cultivation conditions affect oxalate content (2).

Other factors will affect the absorption and bioavailability of dietary oxalate. Generally, 80-90% of ingested oxalate is excreted in the urine within 8 to 11 hours (6). Consequently, a 24 hour urine collection will provide an estimate of the absorption of oxalate from the day's intake. The amount absorbed depends on the amount and form of oxalate in the food, the amount of calcium and magnesium in the food or meal (as they both bind oxalate and make it less available) and the presence or absence of oxalate-degrading bacteria in the gut (6).

Oxalate is widely distributed in foods. The recommendation of the American Academy of Nutrition and Dietetics is to limit dietary oxalate to no more than 40 to 50 mg per day. To accomplish this, foods highest in oxalate should be avoided. These include spinach, rhubarb, beets (roots and leaves), black tea, chocolate, nuts, legumes (peanuts and soybeans) and bran. In addition, consuming 150 mg calcium per meal is recommended to bind oxalate. A calcium supplement can be used if milk/dairy cannot be consumed.

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Brief Summary: Consult full package insert for complete Prescribing Information.

INDICATIONS AND USAGE: Phoslyra® (calcium acetate oral solution 667 mg per 5 mL) is a phosphate binder indicated to reduce serum phosphorus in patients with end stage renal disease (ESRD). Management of elevated serum phosphorus levels usually includes all of the following: reduction in dietary intake of phosphate, removal of phosphate by dialysis, and inhibition of intestinal phosphate absorption with phosphate binders.

DOSAGE AND ADMINISTRATION: The recommended initial dose of Phoslyra for the adult dialysis patient is 10 mL with each meal. Increase the dose gradually to lower serum phosphorus levels to the arget range, as long as hypercalcemia does not develop. Titrate the dose every 2 to 3 weeks until an acceptable serum phosphorus level is reached. Most patients require 15–20 mL with each meal.

CONTRAINDICATIONS: Patients with hypercalcemia.

WARNINGS AND PRECAUTIONS:

Hypercalcemia. Patients with end stage renal disease may develop hypercalcemia when treated with calcium, including calcium acetate (Phoslyra). Avoid the concurrent use of calcium supplements, including calcium-based nonprescription antacids, with Phoslyra. An overdose of Phoslyra may lead to progressive hypercalcemia, which may require emergency measures. Therefore, early in the treatment phase during the dosage adjustment period, monitor serum calcium levels twice weekly. Should hypercalcemia develop, reduce the Phoslyra dosage or discontinue the treatment, depending on the severity of hypercalcemia.
More severe hypercalcemia (Ca >12 mg/dL) is associated with confusion, delirium, stupor and coma. Severe hypercalcemia can be treated by acute hemodialysis and discontinuing Phoslyra therapy. Mild hypercalcemia (10.5 to 11.9 mg/dL) may be asymptomatic or manifest as constipation, anorexia, nausea, and vomiting. Mild hypercalcemia is usually controlled by reducing the Phoslyra dose or temporarily discontinuing therapy. Decreasing or discontinuing Vitamin D therapy is recommended as well.
Chronic hypercalcemia may lead to vascular calcification and other soft-tissue calcification. Radiographic evaluation of suspected anatomical regions may be helpful in early detection of soft tissue calcification. The long-term effect of Phoslyra on the progression of vascular or soft tissue calcification has not been determined.
Hypercalcemia (>11 mg/dL) was reported in 16% of patients in a 3-month study of a solid dose formulation of calcium acetate; all cases resolved upon lowering the dose or discontinuing treatment.
Maintain the serum calcium-phosphorus product (Ca X P) below 55 mg^2/dL^2.

Concomitant Use with Medications. Hypercalcemia may aggravate digitalis toxicity. Phoslyra contains maltitol (1 g per 5 mL) and may induce a laxative effect, especially if taken with other products containing maltitol.

ADVERSE REACTIONS: No clinical trials have been performed with Phoslyra in the intended population. Because the dose and active ingredients of Phoslyra are equivalent to that of the calcium acetate gelcaps or tablets, the scope of the adverse reactions is anticipated to be similar.
Hypercalcemia is discussed elsewhere *[see Warnings and Precautions]*.

Clinical Trial Experience. Because clinical trials are conducted under widely varying conditions, adverse reaction rates observed in the clinical trials of a drug cannot be directly compared to rates in the clinical trials of another drug and may not reflect the rates observed in clinical practice.
In clinical studies, calcium acetate has been generally well tolerated.
The solid dose formulation of calcium acetate was studied in a 3-month, open-label, non-randomized study of 98 enrolled ESRD hemodialysis patients and in a two week double-blind, placebo-controlled, cross-over study with 69 enrolled ESRD hemodialysis patients. Adverse reactions (>2% on treatment) from these trials are presented in Table 1.

Table 1: Adverse Reactions in Patients with End-Stage Renal Disease Undergoing Hemodialysis

	Total adverse reactions reported for calcium acetate n=167	3-mo, open-label study of calcium acetate n=98	Double-blind, placebo-controlled, cross-over study of calcium acetate n=69	
			Calcium acetate	Placebo
Preferred Term	n (%)	n (%)	n (%)	n (%)
Nausea	6 (3.6)	6 (6.1)	0 (0.0)	0 (0.0)
Vomiting	4 (2.4)	4 (4.1)	0 (0.0)	0 (0.0)
Hypercalcemia	21 (12.6)	16 (16.3)	5 (7.2)	0 (0.0)

Calcium acetate oral solution was studied in a randomized, controlled, 3-arm, open label, cross-over, single-dose study comparing calcium acetate oral solution to a solid formulation in healthy volunteers on a controlled diet. Of the observed drug-related adverse reactions, diarrhea (5/38, 13.2%) was more common with the oral solution.

Postmarketing Experience. The following additional adverse reactions have been identified during post-approval of calcium acetate: dizziness, edema, and weakness.

DRUG INTERACTIONS: The drug interaction profile of Phoslyra is characterized by the potential of calcium to bind to drugs with anionic functions (e.g., carboxyl, carbonyl, and hydroxyl groups). Phoslyra may decrease the bioavailability of tetracyclines or fluoroquinolones via this mechanism.
There are no empirical data on avoiding drug interactions between calcium acetate or Phoslyra and most concomitant drugs. When administering an oral medication with Phoslyra where a reduction in the bioavailability of that medication would have a clinically significant effect on its safety or efficacy, administer the drug one hour before or three hours after Phoslyra or calcium acetate. Monitor blood levels of the concomitant drugs that have a narrow therapeutic range. Patients taking anti-arrhythmic medications for the control of arrhythmias and anti-seizure medications for the control of seizure disorders were excluded from the clinical trials with all forms of calcium acetate.
Ciprofloxacin. In a study of 15 healthy subjects, a co-administered single dose of 4 calcium acetate tablets (approximately 2.7 g) decreased the bioavailability of ciprofloxacin by approximately 50%.

USE IN SPECIFIC POPULATIONS

Pregnancy: Category C. Phoslyra contains calcium acetate. Animal reproduction studies have not been conducted with Phoslyra, and there are no adequate and well controlled studies of Phoslyra use in pregnant women. Patients with end stage renal disease may develop hypercalcemia with calcium acetate treatment *[see Warnings and Precautions]*. Maintenance of normal serum calcium levels is important for maternal and fetal well being. Hypercalcemia during pregnancy may increase the risk for maternal and neonatal complications such as stillbirth, preterm delivery, and neonatal hypocalcemia and hypoparathyroidism. Phoslyra treatment, as recommended, is not expected to harm a fetus if maternal calcium levels are properly monitored during and following treatment.

Labor and Delivery. The effects of Phoslyra on labor and delivery are unknown.

Nursing Mothers. Phoslyra contains calcium acetate and is excreted in human milk. Human milk feeding by a mother receiving Phoslyra is not expected to harm an infant, provided maternal serum calcium levels are appropriately monitored.

Pediatric Use. Safety and effectiveness of Phoslyra in pediatric patients have not been established.

Geriatric Use. Clinical studies of calcium acetate did not include sufficient numbers of subjects aged 65 and over to determine whether they respond differently from younger subjects. Other reported clinical experience has not identified differences in responses between the elderly and younger patients. In general, dose selection for an elderly patient should be cautious, usually starting at the low end of the dosing range, reflecting the greater frequency of decreased hepatic, renal, or cardiac function, and of concomitant disease or other drug therapy.

OVERDOSAGE: Administration of Phoslyra in excess of the appropriate daily dosage may result in hypercalcemia *[see Warnings and Precautions]*.

HOW SUPPLIED/STORAGE AND HANDLING: Phoslyra for oral administration is a clear solution containing 667 mg calcium acetate per 5 mL. Phoslyra is supplied in a 473 mL (16 oz) amber-colored, multiple-dose bottle, packaged with a marked dosing cup. Store at 25°C (77°F); excursions permitted to 15–30°C (59–86°F) [see USP Controlled Room Temperature]. The shelf life is 24 months.

PATIENT COUNSELING INFORMATION: Inform patients to take Phoslyra with meals, adhere to their prescribed diets, and avoid the use of calcium supplements including nonprescription antacids. Inform patients about the symptoms of hypercalcemia *[see Warnings and Precautions and Adverse Reactions]*.
Advise patients who are taking an oral medication where a reduction in the bioavailability of that medication would have a clinically significant effect on its safety or efficacy to take the drug one hour before or three hours after Phoslyra.

Manufactured for:
Fresenius Medical Care North America
Waltham, MA 02451 1-800-323-5188

Manufactured by:
Lyne Laboratories
Brockton,MA 02301

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101493-01 Rev. B 08/2012

In patients with ESRD...

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First and only FDA-approved little LIQUID phosphate binder

- May lessen pill burden
- Potential to reduce optional fluid intake associated with administration of solid PBs
- No water required
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- No refrigeration, even after opening

Phoslyra®
calcium acetate oral solution
667mg per 5 mL
Clearly different.

INDICATION:

Phoslyra® (calcium acetate oral solution, 667 mg per 5 mL) is a phosphate binder (PB) indicated for the reduction of serum phosphorus in patients with end stage renal disease (ESRD). Phoslyra is administered orally with food.

IMPORTANT SAFETY INFORMATION:

- **Phoslyra is contraindicated in patients with hypercalcemia.**
- **Patients should have serum calcium levels closely monitored and their dose of Phoslyra adjusted or terminated to bring levels to normal.** No other calcium supplements should be given concurrently with Phoslyra.
- Phoslyra may decrease the bioavailability of tetracyclines or fluoroquinolones.
- There are no empirical data on avoiding drug interactions between calcium acetate or Phoslyra and most concomitant drugs. When administering an oral medication with Phoslyra where a reduction in the bioavailability of that medication would have a clinically significant effect on its safety or efficacy, administer the drug 1 hour before or 3 hours after Phoslyra or calcium acetate. Monitor blood levels of the concomitant drugs that have a narrow therapeutic range.
- The most common (>10%) adverse reactions experienced with Phoslyra are hypercalcemia, nausea, and diarrhea.
- Phoslyra may cause diarrhea with nutritional supplements that contain maltitol.

For additional important safety information, please see brief Prescribing Information on this page.

For more information on Phoslyra, please contact Fresenius Medical Care NA at 800-323-5188. Manufactured for and distributed by: Fresenius Medical Care NA, Waltham, MA 02451

101731-01 Rev. A 08/2012

Fresenius Medical Care
RENAL PHARMACEUTICALS

Calendar of Events

October 2012

ASN Kidney Week 2012
San Diego Convention Center, San Diego, CA
October 30–November 4, 2012
http://www.asn-online.org

December 2012

International Conference and Exhibition on Obesity & Weight Management
Philadelphia, PA
December 3-5, 2012
www.omicsonline.org/obesity2012

February 2013

Clinical Nutrition Week 2013
Phoenix Convention Center, Phoenix, AZ
February 9-12, 2013
http://www.nutritioncare.org/ClinicalNutritionWeek/index.aspx?id=502

CRRT 2013 Conference (Continuous Renal Replacement Therapies)
Hilton Bay Front, San Diego, CA
February 12-15, 2013
http://www.crrtonline.com/conference/

March 2013

Joint North West Renal Dietitians (NWRD) and Canadian Association of Renal Dietitians (CAND) Conference
Vancouver, BC
March 7-8, 2013
https://www.sites.google.com/site/nwrdonline/

33rd Annual Dialysis Conference
Seattle, WA
March 10-12, 2013
www.som.missouri.edu/Dialysis/

2013 Canadian Society of Transplantation Annual Scientific Conference
Lake Louise, AB; Canada
March 14-16, 2013
http://www.cst-transplant.ca/AnnualConference.cfm

April 2013

National Kidney Foundation Spring Clinical Meetings
Walt Disney World Swan and Dolphin, Orlando, FL
April 2-6, 2013
http://www.kidney.org/news/meetings/clinical/general/future_dates.cfm

May 2013

American Society of Pediatric Nephrology Annual Meeting
Washington D.C.
May 4-7, 2013
http://aspneph.com/educationmeetings.asp

June 2013

International Society of Nephrology (ISN) World Congress of Nephrology
Hong Kong
May 31-June 4, 2013
http://www.wcn2013.org/

RPG Outstanding Service Award Winner!

Congratulations to the 2012 Outstanding Service Award Winner, Jerrilynn D. Burrowes, PhD, RD, CDN!

Jerrilynn D. Burrowes is an internationally recognized clinical nutrition leader in the field of kidney disease. She is currently an Associate Professor of Nutrition and Chair of the Department of Nutrition, School of Health Professions and Nursing at Long Island University (LIU) Post in Brookville, NY. In September 2012, Dr. Burrowes was promoted to the rank of Full Professor in the Department of Nutrition. Prior to her appointment at LIU Post in 2003, Dr. Burrowes was the research coordinator for the Division of Nephrology and Hypertension at Beth Israel Medical Center in New York City, where she worked closely with one of the leaders in clinical and research nephrology, Dr. Nathan Levin. She has been actively involved as an investigator and collaborator on one of the landmark NIH sponsored clinical trials of morbidity and mortality in people receiving maintenance hemodialysis of the past decade, the Hemodialysis (HEMO) Study. Dr. Burrowes has authored and co-authored numerous research and review articles published in refereed journals relating to the HEMO Study and other topics on nutrition in kidney disease, and she has been an invited speaker at 70 professional meetings and conferences. In addition, Dr. Burrowes is the co-editor of a textbook published by Humana Press in 2008 entitled Nutrition in Kidney Disease. Springer will publish a second edition of this textbook in early 2013.

Dr. Burrowes has received numerous research grants over the past few decades. Her research currently centers on the nutrition assessment and management of adults with Stage 5 chronic kidney disease (CKD) who are receiving maintenance hemodialysis. She is particularly interested in: (1) the factors that influence appetite and their effect on health outcomes; (2) the methods used to improve nutritional status, especially in geriatric patients with kidney failure; (3) the influence of culture and ethnicity on dietary adherence in dialysis patients; (4) leaching potassium from tuberous root vegetables to prepare them for consumption by culturally-diverse patients with kidney failure whose dietary staples include these plant products; and (5) evidence-based studies of complementary and alternative therapies for people with kidney failure. Dr. Burrowes is currently examining whether sleep quality and sleep duration of dialysis patients have implications for the development of nutritional problems, or vice versa.

Dr. Burrowes has held many leadership and advisory roles in professional organizations and societies, and she has served on numerous association committees. She has served as a member of the advisory board for the National Kidney Foundation (NKF) Kidney Disease Outcomes Quality Initiative (KDOQI) and the Nutrition Work Group for the NKF Dialysis Outcomes Quality Initiative (DOQI), which developed clinical practice guidelines in nutrition for people with CKD. She was also a member of the NKF Kidney Disease: Improving Global Outcomes (KDIGO) Advisory Board. Dr. Burrowes has also served in various leadership capacities for the NKF Council on Renal Nutrition (CRN), the NKF/CRN of Greater New York, and the NKF of New York/New Jersey. She is a member of several professional organizations including the Academy of Nutrition and Dietetics, the Renal Practice Group of the Academy, the American Society for Nutrition, the NKF/CRN, and the International Society for Renal Nutrition and Metabolism (ISRNM). Dr. Burrowes is currently the Editor-in-Chief for the Journal of Renal Nutrition (JRN). She has also served as the Editor of the Nutrition Section for Advances in Chronic Kidney Disease and as the Contributing Editor of the Clinical Column for Nutrition Today. In 1998, Dr. Burrowes received the Recognized Renal Dietitian Award from the NKF/CRN; in 2005, she was the recipient of the Joel D. Kopple Award from the same organization for her outstanding contributions to the field of kidney disease.

RPG offers one OSA per year to a renal or nephrology dietitian who is a member of RPG and has demonstrated the qualities of leadership and initiative, promoted the dietitian's role with chronic kidney disease, and has also shown dedication to patient care. Dr. Burrowes' contributions have been valuable and beneficial. Her hard work and efforts are greatly appreciated!

RPG Chair Message

Sarah Kruger, MS, RD, CSR
RPG Chair

Welcome back from FNCE! Whew, I am exhausted. To me, FNCE is like an annual dietitian reunion. Every year I am able to attend FNCE, I am fortunate to be able to network with alumni and faculty from Michigan State University at the annual MSU/MDA breakfast. I love being able to reconnect with old friends, see the faculty from my college years, and hear about the progress the future dietitians of the world are making. I remember when I started in dietetics at MSU; I didn't know what path I wanted to follow exactly, I just knew I wanted to help teach people how to eat better and live healthier lives. Since graduating and becoming a dietitian, I have been fortunate in my career path. I have been able to progress upwards and I feel like my positions have been consistently evolving. However, I know that is not always the case for dietitians working in dialysis clinics. This year I was able to continue moving forward with my career progression, but I had to move out of the dietetics arena into a commercial/management type of position. My own personel growth has caused me to pause and wonder, what are the next steps for a renal dietitian? If a renal dietitian wants to continue working directly with patients, what is the next step in the career ladder? I don't know, but I hope with feedback from YOU, our members, we will be able to help define what that career ladder might look like and provide the resources you will need to approach management with it.

This topic of career ladder aligns with one of the Academy's House of Delegates mega issues last year on the continuum of Professional Progression and Growth. The Academy developed a Dietetics Career Development Guide (Figure 1) to help define the different levels of advancement. The six stages in the guide are Novice, Beginner, Competent, Proficient, Advanced Practice, and Expert. I suggest you take a look at this guide. You can find it at www.eatright.org, ->members->practice->council on future practice->dietetics career development guide. I am interested to see if the guide is a useful tool to help you grow in your career and if there are any other tools that might be helpful in moving along the continuum.

"The only job security you have today is your commitment to continuous personal improvement"
~ Ken Blanchard, The Heart of a Leader

Does one have to aspire to move up the career ladder? I don't think so. I think a more positive suggestion is to focus on personal improvement. Even if you are extremely satisfied with your professional position, I believe it is through personal growth and stretching that we become fulfilled and satisfied. One of the things the Academy has available for personal improvement is an online series of leadership modules. After going through the five modules, you can take an exam and if you have an 80% pass rate, then you receive a certificate of training. This is one simple thing you can do to continue to work on personal improvement. Another idea would be to read a leadership or development book to help expand your thinking, improve processes, and increase efficiency. The most recent leadership book I read was "The Checklist Manifesto." Now I want to create a checklist for everything! The last idea I would recommend is to become a Board Certified Specialist in Renal Nutrition (CSR). RPG is committed to increasing our resources and one of those areas is the CSR exam. We are updating our website and will be providing a section committed to providing RDs with resources to become a CSR. It will include tips on taking the exam, top 10 reasons to become certified, top 10 reasons to hire a CSR dietitian, and much more. You can check it out at www.renalnutrition.org. The point is to continue striving for personal improvement. Never let yourself become satisfied with just surviving.

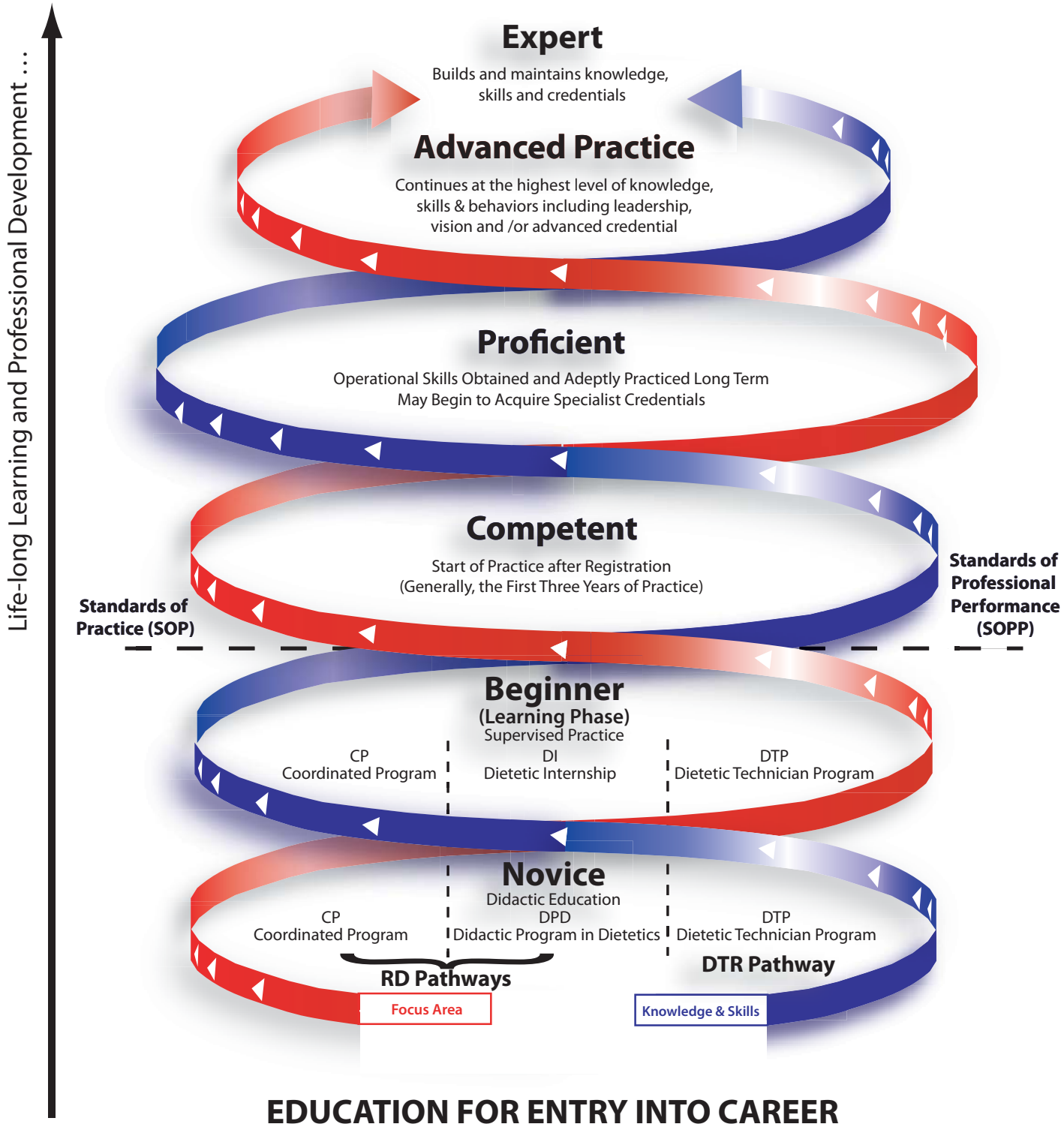
STAY TUNED FOR NEW & UPDATED CSR WEBINARS!!!

The CSR Webinar Series is in the process of being updated. The newly created webinar modules will include detailed reviews on disease state, lab values, interventions, and case studies. These modules can be used to study for the CSR exam as well as to 'brush up' on clinical practice skills. RPG remains committed to developing webinars with topics of interest to enhance learning and professional advancement!

New resources coming soon!

A collaborative project with DHCC is underway to provide a webinar on Renal Patients in the Long Term Care setting - stay tuned for more details! To purchase other RPG produced Webinars please visit *www.renalnutrition.org/materials/index.php*

Dietetics Career Development Guide



EDUCATION FOR ENTRY INTO CAREER

Associate, Baccalaureate or Advanced Degree

Definition of Dietetics: Dietetics is the integration, application and communication of principles derived from food, nutrition, social, business and basic sciences, to achieve and maintain optimal nutrition status of individuals through the development, provision and management of effective food and nutrition services in a variety of settings.

Approved 9/29/10; 10/11/10

CRN Chairperson Message

Lisa Gutenkunst, MSEd, RD, CSR, CDN
NKF-CRN Chair

The smell of fallen leaves is in the air. As the year begins to wind down, the Council on Renal Nutrition begins to ramp up. After the Clinical Meetings in the spring and the meeting of the International Society on Renal Nutrition and Metabolism (ISRNM) in the summer, the group is coming together on projects that came out of both meetings.

Statistical evaluation of the survey looking at current work habits since the implementation of the Conditions for Coverage (CfC) will be completed soon. The raw data showed that since the CfC started, we find ourselves sitting in front of a computer charting more than sitting with our patients educating. It is the sad reality of the field today as education is the key to improved outcomes. Improved outcomes not only improve patient lives, but also require less paperwork. As a result, many who participated in the survey voiced their comments regarding this change in work tasks.

The joint CRN-RPG group working on the Kidney-Friendly Foods Initiative is moving towards identifying the educational resources needed by dietitians working with the CKD population, as well as the needs of dietitians who practice in diabetes, food manufacturing, and food marketing. Once educational needs are identified, the group will move forward looking at the optimal ways to bring the needed information to the group. Forward thinking includes the use of apps, interactive media, and web education, in addition to the traditional routes of workshops, handouts, and seminars.

Sarah Kruger, RPG Chair, and and I had the opportunity to participate at an all day international consortium on phosphorus at the ISRNM meeting. The discussions focused on what we currently know about phosphorus in the development of cardiovascular disease in the renal population (including the pre-dialysis stage) and the general public. There were additional talks about how to combat the problems phosphorus salts (additives) create for our patients. I had the pleasure of speaking on the international regulations of phosphorus labeling across the world. At the end of the day, it was decided to produce two consortium papers geared for the primary care/general practitioner on the role phosphorus plays in the development of cardiovascular disease in the Chronic Kidney Disease patient. Limiting or avoiding foods containing phosphorus salts will be recommended as they offer no nutritive value to most foods.

As for the possibility of putting phosphorus back on the label, it's a long shot right now. Though we have correlation studies between serum phosphorus levels and cardiovascular disease, we don't have the definitive cause. Is it phosphorus? Is it phosphorus salts? Is it something else? More studies are required before we have the true link between what we see and what we THINK is happening.

It's an ever changing world right now. I'm truly excited, and sometimes frustrated, by it. Never the less, I know that our field and patients will benefit from our work and commitment.

May 2012 Board Certified Specialist in Renal Nutrition (CSR) Recipients

Congratulations to the following Registered Dietitians!

California
Sandy Lu
Wendy Raymond
Maria Tointon
Wandu Yu

Florida
Isabelle Faucher

Georgia
Anne Borders
Jane Roberts
Deborah Schick

Hawaii
Patricia Barba
Keanani Custodio

Idaho
Lindsey Daniels

Illinois
Jacqueline Hankins
Melissa Prest

Maryland
Wendy Caesar Gibbs
Linda Salamone
Susan Skelly-Miller

Michigan
Sarah Carpenter
Sara Zerba

Missouri
John McElwee

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Janet Garnett
Melanie Gustavson-Hobson

Nevada
Jessica Ondell

New York
Kwee Peng Hong
Erin Tolchin

North Carolina
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Maria Stasios

Ohio
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Jennifer Kernc
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Susan Vorherr

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LaRita Parkison
Jessica Stauffer

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Jean Park

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Jane Valley
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Linda Winslow

Wisconsin
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Vision: RPG members are a valued source of expertise in nephrology nutrition.

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RNF Guidelines for Authors

Article length:
Article length is determined by the Editor for each specific issue. The feature article (including abstract) is approximately 3000 words (not including tables/graphs). Other articles are usually 1000-1500 words; member highlights and reports are approximately 400-500 words.

Text format:
Times New Roman font, 12 point, double space.

Tables/Illustrations:
Tables should be self-explanatory. All diagrams, charts and figures should be camera-ready. Each should be accompanied by a title and brief caption that clearly explains the table, chart, diagram, figure, illustration, etc.

References:
References should be cited in the text in consecutive order parenthetically. At the end of the text, each reference should be listed in order of citation. The format should be the same as the Journal of the Academy of Nutrition and Dietetics.

Reference citation examples:
Article in periodical:
Knower WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Eng J Med.* 2002;346:393–403.

Book:
Institute of Medicine. *Dietary Reference Intakes: Applications for Dietary Assessment*. Washington, D.C.: National Academy Press; 2001.

Chapter in a book:
Walsh J. Which insulin to use and how to start. In: Using Insulin. San Diego, Calif.: Torry Pines Press; 2003.

Web site:
Medscape drug info. Available at www.medscape.com/druginfo. Accessed August 15, 2011.

Author information:
List author with first name, middle initial (if any), last name, professional suffix and affiliation below the title of the article. Also include the primary author's complete contact information including affiliation, phone, fax and email address.

All submissions for publication should be submitted to the editor as an email attachment (MS Word file). The feature articles from the Renal Nutrition Forum will be posted on the Members Only Section of the RPG website (password protected). Thus, please include a brief abstract and 2-3 key words along with feature article submissions.

Sara Erickson, RD, CSR, LDN, CNSC
Editor, *Renal Nutrition Forum*
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