

Patient education for phosphorus management in chronic kidney disease

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Objectives: This review explores the challenges and solutions in educating patients with chronic kidney disease (CKD) to lower serum phosphorus while avoiding protein insufficiency and hypercalcemia.

Methods: A literature search including terms “hyperphosphatemia,” “patient education,” “food fatigue,” “hypercalcemia,” and “phosphorus–protein ratio” was undertaken using PubMed.

Results: Hyperphosphatemia is a strong predictor of mortality in advanced CKD and is remediated via diet, phosphorus binders, and dialysis. Dietary counseling should encourage the consumption of foods with the least amount of inorganic or absorbable phosphorus, low phosphorus-to-protein ratios, and adequate protein content, and discourage excessive calcium intake in high-risk patients. Emerging educational initiatives include food labeling using a “traffic light” scheme, motivational interviewing techniques, and the Phosphate Education Program – whereby patients no longer have to memorize the phosphorus content of each individual food component, but only a “phosphorus unit” value for a limited number of food groups. Phosphorus binders are associated with a clear survival advantage in CKD patients, overcome the limitations associated with dietary phosphorus restriction, and permit a more flexible approach to achieving normalization of phosphorus levels.

Conclusion: Patient education on phosphorus and calcium management can improve concordance and adherence and empower patients to collaborate actively for optimal control of mineral metabolism.

Keywords: hyperphosphatemia, renal diet, phosphorus binders, educational programs, food fatigue, concordance

Introduction

Chronic kidney disease (CKD) is associated with an increased risk of cardiovascular disease (CVD) and mortality.¹ This increased mortality is related to traditional cardiovascular (CV) risk factors such as diabetes and hypertension, which are common comorbidities, but is also related to disorders of bone and mineral metabolism (renal osteodystrophy) and vascular calcification. This led to the concept of CKD-mineral bone disorder (CKD-MBD), which describes a systemic disorder characterized by laboratory abnormalities (calcium, phosphorus, parathyroid hormone [PTH], and vitamin D), bone abnormalities, and vascular calcification. A strong association exists between elevated serum phosphorus (>5.0 mg/dL) and calcium (>9.5 mg/dL) and mortality in dialysis patients, reflecting a preponderance of CVD-related deaths.^{2,3} Increased CV risk with hyperphosphatemia and hypercalcemia is also evident in the general population.⁴ Studies have also shown that hyperphosphatemia is associated with increased

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vascular stiffening⁵ and arterial⁶ and valvular calcification.⁷ This is postulated to be caused by elevated serum phosphorus promoting the transformation of vascular smooth muscle cells into an osteoblast phenotype that can mineralize.⁸

Of all the components of CKD-MBD, hyperphosphatemia conveys the highest risk of death (~12%) in hemodialysis patients, which is increased further once hypercalcemia and elevated PTH are considered (17.5%).² A higher dietary intake of phosphorus and higher phosphorus-to-protein ratio are both associated with increased risk of mortality in hemodialysis patients, even after adjustment for serum phosphorus and phosphorus binders, dietary protein, energy, and potassium intake (Figure 1).⁹ Efforts to reduce morbidity and mortality associated with CKD-MBD are therefore primarily directed at controlling hyperphosphatemia via diet, phosphorus binders, and dialysis. The goal in patients with end stage renal disease (ESRD) is to maintain a low intake of phosphorus (ideally 700 mg/day) while maintaining adequate protein intake.¹⁰

Phosphorus is found in almost every food and is derived primarily from proteins,¹⁰ phytates,¹¹ and additives.^{12,13} Two types exist – organic and inorganic – depending on the dietary source. Inorganic phosphorus is present in processed-food additives,¹¹ which are highly absorbable, and is highly abundant in the diet of postmodern industrialized regions.¹⁴ Organic phosphorus is found in animal- and plant-derived protein-rich foods; however, the phosphorus-to-protein ratio differs markedly depending on the food source.¹⁵

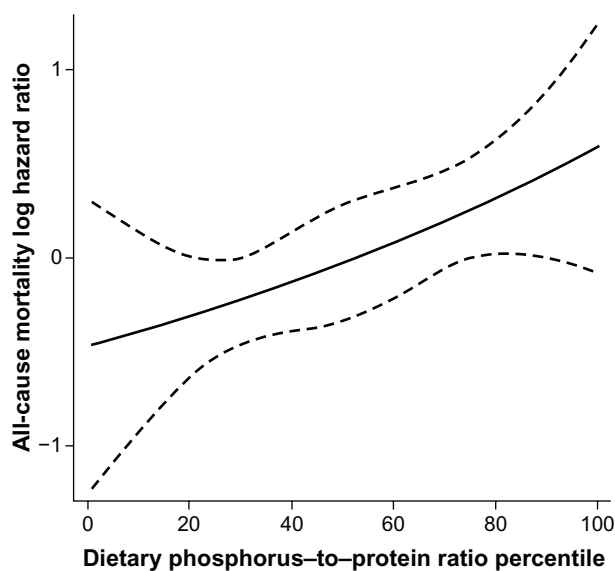


Figure 1 A high phosphorus/protein ratio increases mortality risk in CKD patients even after adjustment for binders, serum phosphorus, and diet. Noori N, Kalantar-Zadeh K, Kovesdy CP, Bross R, Benner D, Kopple JD. Association of dietary phosphorus intake and phosphorus to protein ratio with mortality in hemodialysis patients. *Clin J Am Soc Nephrol.* 2010;5(4):683–692.⁹ © American Society of Nephrology 2010.

Abbreviation: CKD, chronic kidney disease.

Although plant proteins also contain phosphorus, much plant phosphorus is in the form of less-absorbable phytates.¹³ While it is important to minimize the dietary phosphorus burden, a high dietary protein intake should be maintained in dialysis patients to avoid protein wasting and improve survival.¹⁶ Balancing dietary phosphorus and protein intake is best achieved using the phosphorus-to-protein ratio to select protein sources with the least serum phosphorus “cost.”¹⁵ In addition to dietary intervention, phosphorus binders lower serum phosphorus by reducing the absorption of dietary phosphorus from the gut. They are indicated in the US for hyperphosphatemic dialysis recipients; in certain other countries, they are indicated for hyperphosphatemic patients with CKD stages 3–5 or on dialysis.¹⁷

Achieving the recommended serum phosphorus levels using diet and phosphorus binders requires patient comprehension and active participation as well as adherence and persistence. In particular, practitioners need to communicate the consequences (especially mortality risk) of hyperphosphatemia in CKD-MBD. For example, clinicians should explain the consequences of patients’ behavior (eg, consuming a cola drink high in phosphorus needs to be accompanied by a binder). Given the complex pathologies and treatment regimen associated with CKD-MBD, close collaboration among the different members of the renal team and the patient is important in order to develop individualized treatment plans that fit patients’ lives and will improve adherence over time. Patient education and concordance can improve implementation of regimens for phosphorus control in CKD-MBD.

This review will examine challenges and solutions in educating patients with ESRD to lower serum phosphorus while avoiding protein insufficiency and hypercalcemia.

Phosphorus control methods – diet, binders, and dialysis

Dietary phosphorus

Three sources of dietary phosphorus exist: (1) Organic phosphorus present in plant foods (eg, phytates); (2) Organic phosphorus present in animal protein (eg, casein); and (3) Inorganic phosphorus (eg, additives in processed food). Because humans lack the degrading enzyme phytase, phosphorus in plant foods (seeds, nuts, legumes) has a low bioavailability (20%–40%). In contrast, the phosphorus in animal-derived food has a higher bioavailability (40%–60%) because it is easily hydrolyzed and absorbed. Inorganic phosphorus is the most readily absorbable with a bioavailability of approximately 100%.^{12,13} Overconsumption of bioavailable inorganic phosphorus in food additives

and preservatives contributes to hyperphosphatemia for patients with CKD. Thus, practitioners need to educate patients on the differences in phosphorus bioavailability. Phosphorus additives are abundant in cheap processed and fast foods,¹¹ which is reflected in the higher phosphorus intake observed in lower socioeconomic groups.¹⁸ Inorganic phosphorus additive intake in the USA rose dramatically between the 1970s and 1990s¹⁹ and may contribute as much as 1000 mg/day to the average American diet.¹⁴ Environmental differences can also affect the phosphorus content in certain food sources. For instance, the phosphorus content in farm-raised fish is higher than in wild fish due to exposure to higher dietary phosphorus and environmental factors that influence final mineral content.²⁰

The majority of dietary phosphorus is derived from protein. The recommended daily intake is 700 mg of phosphorus per day for CKD patients,²¹ though the usual intake ranges between 1000–2000 mg/day.¹⁰ However, given the difference in bioavailability of organic and inorganic phosphorus, predictions of phosphorus intake should not be based solely on protein intake. Furthermore, the restriction of poorly absorbed foodborne organic phosphorus is unnecessary; overzealous restriction of protein-associated phosphorus may even contribute to protein malnutrition with an adverse impact on survival.²²

Previous methods for estimating phosphorus content significantly underestimated phosphorus content by 15% to 25%,²³ which emphasizes the need for more information

via food frequency questionnaires, food tables, and nutritional databases on phosphorus content and bioavailability, phosphorus–protein ratios, and additives. The selection of appropriate foods low in phosphorus is hindered by the fact that food manufacturers are not required to list the phosphorus content on food labels.²⁴ Furthermore, inorganic phosphorus is often hidden by complex names in ingredients (eg, disodium monophosphate; Table 1).²⁵ Additionally, dialysis patients have a higher normalized protein catabolic rate than healthy persons, which results in endogenously increased inorganic phosphorus levels and higher binder requirements.²⁶ A strong association exists between dietary protein and phosphorus intake. The following regression equation was able to account for 83% of the variance in dietary phosphorus intake (Figure 2):¹²

$$\text{Dietary phosphorus (mg)} = 78 + 11.8 \times \text{protein intake (g)} \quad (1)$$

The phosphorus-to-protein ratio has several advantages as a dietary metric: it is (1) independent of size of food portion; (2) focused on dietary protein and phosphorus intake; (3) higher for foods with unusually high amounts of phosphorus; and (4) a way of identifying foods high in phosphorus.⁹ A limitation of absolute phosphorus content and phosphorus-to-protein ratio is

Table 1 Common phosphorus additives used by the food industry

Phosphate salt	Purpose	Foodstuffs
Dicalcium phosphate	Calcium and phosphorus supplement, dough conditioner	Bakery mixes, cereals, flour, food bars, infant food, yogurt
Disodium phosphate	Sequestrant, emulsifier, buffering agent, absorbent, pH control agent, protein modifier, source of alkalinity, stabilizer	Breakfast cereal, cheese, condensed milk, flavored milk powders, ice cream, isotonic drinks, pasta, processed cheese, vitamin capsules
Monosodium phosphate	Acidulant, buffering agent, emulsifier, leavening agent, protein modifier and sequestrant, gelling agent	Cola beverages, dry powder beverages, egg yolks, gelatin, instant cheesecake, instant pudding, isotonic beverages
Phosphoric acid	Acidulant, pH control agent, buffering agent, flavor enhancer, flavoring agent, sequestrant, stabilizer	Cola beverages, carbonated and noncarbonated beverages
Sodium hexametaphosphate	Sequestrant, curing agent, dough strengthener, emulsifier, firming agent, flavor enhancer, humectant, stabilizer, thickener	Meat, seafood, poultry, vegetables, cream, ice cream, whey, processed cheese, eggs, table syrups, toppings
Sodium tripolyphosphate	Sequestrant, pH control agent, emulsifier, source of alkalinity, buffering agent, coagulant, dispersing agent, antioxidant, curing agent, flavor enhancer, humectant	Meat products, seafood, poultry, vegetable proteins, processed cheese, sour cream, dips, yogurt, eggs, table syrups
Tetrasodium pyrophosphate	Buffering agent, pH control agent, source of alkalinity, dispersing agent, coagulant, sequestrant, color stabilizer	Processed meat, poultry, seafood, processed cheese, potato products, ice cream, frozen desserts
Trisodium phosphate	Buffer, emulsifying agent, stabilizer, protein modifier, pH control, color stabilizer	Processed cheese, cheese products, isotonic beverages, cooked breakfast cereals

Reprinted with permission Kalantar-Zadeh K, Gutekunst L, Mehrotra R, et al. Understanding sources of dietary phosphorus in the treatment of patients with chronic kidney disease. *Clin J Am Soc Nephrol.* 2010;5(3):519–530.12 © American Society of Nephrology 2010.

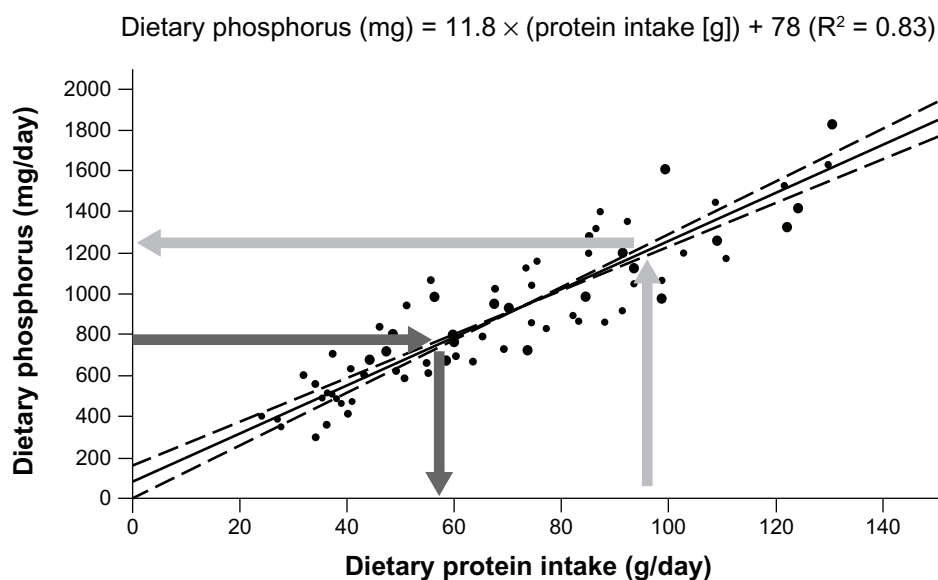


Figure 2 Dietary phosphorus can be estimated using a regression equation that accounts for 83% of the variance and demonstrates the strong association between dietary phosphorus and protein intake. Repinted with permission Kalantar-Zadeh K, Gutkunst L, Mehrotra R, et al. Understanding sources of dietary phosphorus in the treatment of patients with chronic kidney disease. *Clin J Am Soc Nephrol.* 2010;5(3):519–530.12 © American Society of Nephrology 2010.

their lack of information about bioavailability in different food types.⁹

In summary, dietary phosphorus is strongly related to protein content, but is unreliably estimated due to differences in bioavailability and the presence of inorganic phosphorus in additives. Despite these barriers, patient education and concordance can improve the implementation of an effective diet and binder regimen to control phosphorus in the CKD-MBD patient.²⁷

Phosphorus binders

Dietary restriction and dialysis are usually insufficient to maintain phosphorus within the recommended range (2.7–5.5 mg/dL) in patients with CKD. Since their introduction over 30 years ago, phosphorus binders have been used to optimize the management of hyperphosphatemia and lower serum phosphorus to within target levels. Their importance is exemplified by a recent study showing a clear survival advantage in predialysis CKD patients using any binders versus no binder (Figure 3).²⁸ Aluminum-containing binders were the first to be introduced, but their use has been restricted due to the incidence of osteomalacia and encephalopathy;¹⁷ they are now generally reserved for rescue therapy where hyperphosphatemia is accompanied by highly elevated PTH and serum calcium levels. Calcium-based binders (calcium carbonate and calcium acetate) subsequently became widely used, but there are also growing safety concerns related to excessive total body calcium, which can induce adynamic bone disease and increase the risk of CV and soft tissue calcification.²⁹

Where there is evidence of hypercalcemia or calcification in predialysis and dialysis patients with CKD, Kidney Disease: Improving Global Outcomes (KDIGO) recommends restricting the dose of these binders.¹⁷ Vascular calcification is highly prevalent in predialysis (40%)³⁰ and dialysis patients (88%),³¹ and it is therefore possible that some patients would benefit from the use of calcium-free binders such as sevelamer carbonate/hydrochloride or lanthanum carbonate, which may reduce the risk of hypercalcemia and its consequences.^{32,33} Some studies suggest that the choice of binder in hemodialysis patients can affect the mortality rate;^{34,35} however, further studies are warranted to confirm any survival advantage for specific binders (Figure 4).³⁴ The Dialysis Clinical Outcomes Revisited (DCOR) study compared any-cause and cause-specific mortality between sevelamer and calcium binder recipients on prevalent dialysis. In the overall DCOR population, mortality endpoints did not differ between binder groups; however, sevelamer was associated with lower all-cause mortality compared with calcium binders in patients aged 65 years and older.³⁵

Dialysis

Optimal dialysis still requires combination with individualized dietary guidance and binder use to normalize serum phosphorus levels. In general, dialyzer clearance is dependent on an effective blood flow rate (eg, 250–300 mL/minute), which initially improves the clearance of phosphorus. However, serum phosphorus levels plateau after about 2 hours of dialysis; during the second half of treatment,

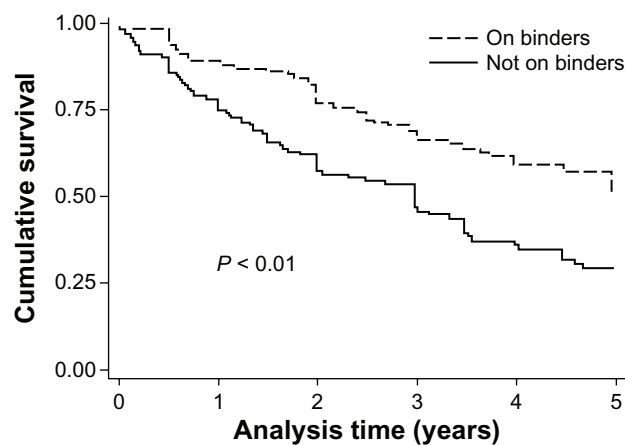


Figure 3 The use of phosphorus binders has a significant survival benefit in veterans ($n = 1188$) with moderate to advanced CKD. Reprinted with permission Kovesdy CP, Kuchmak O, Lu JL, Kalantar-Zadeh K. Outcomes associated with phosphorus binders in men with non-dialysis-dependent CKD. *Am J Kidney Dis.* 2010;56(5):842–851. ²⁸ © Elsevier 2010. **Abbreviation:** CKD, chronic kidney disease.

serum phosphorus does not decline any further and may actually increase again.³⁶ This rebound effect is due to the mobilization of intracellular phosphorus, which compensates for the increased clearance of phosphorus.³⁷ Optimal clearance of phosphorus is dependent on the timing and duration of dialysis. Long nocturnal hemodialysis is associated with increased phosphorus removal and decreased phosphorus binder requirements compared with conventional hemodialysis.³⁸ Limitations in phosphorus clearance are inherent in both hemodialysis and peritoneal dialysis, although the latter is less dependent on dialyzer clearance of urea and correlates better with creatinine clearance. Phosphorus clearance using peritoneal dialysis is also influenced by peritoneal membrane transport characteristics and modality (eg, continuous versus intermittent).³⁹ Dialysis therapy is therefore

limited in its ability to control phosphorus. Dialysis recipients need to closely control phosphorus intake rather than rely solely on dialytic phosphorus elimination. Patients with CKD prior to dialysis also need phosphorus management to reduce the cumulative biochemical impact of processes required to maintain phosphorus excretion as kidney function declines. Thus, efforts to improve patient education are directed at dietary intervention at all stages of CKD, and binder use at stages with locally approved indications.

New approaches to dietary phosphorus control

Dietary phosphorus control is appropriate at any stage of CKD. Whereas 20 years ago dietitians emphasized controlling phosphorus by restricting protein, current recommendations focus on controlling phosphorus by reducing additives and seeking natural, unprocessed, low-phosphorus protein sources, as it is well-established that reducing protein intake may lead to protein-energy wasting and impaired survival.²² Paradoxically, increasing protein intake also worsens the survival rate in hemodialysis patients unless this is accompanied by reduced phosphorus intake.²² The lowest amount of phosphorus in proportion to protein comes from nondairy products and animal-derived foods, including egg whites and pork rinds (Table 2).¹³ Egg white is an unusually rich source of high biological value protein and has one of the lowest phosphorus-to-protein ratios, and is devoid of cholesterol, making it an ideal dietary choice for the CKD patient. This is supported by the results of a pilot study which showed that consuming egg whites as a primary protein source in maintenance hemodialysis patients effectively lowered serum

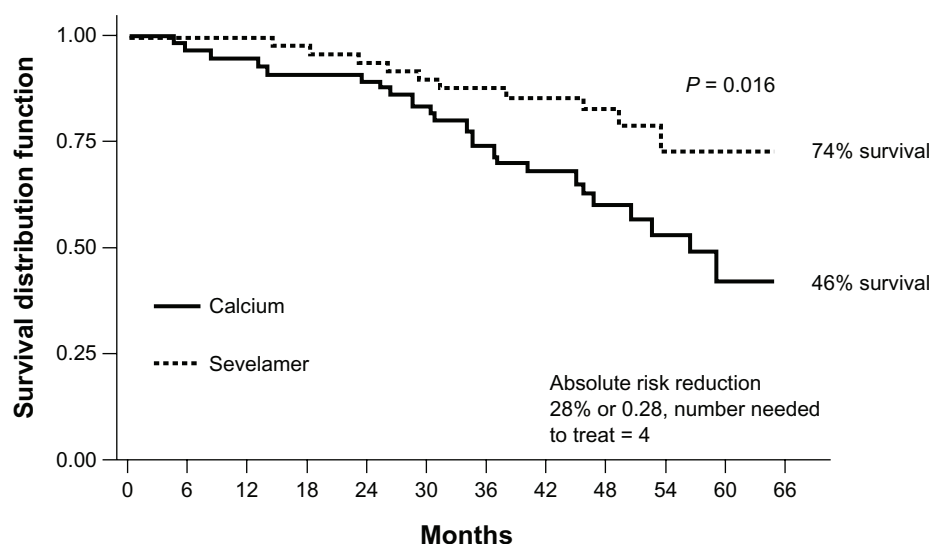


Figure 4 Calcium-free binders such as sevelamer are associated with reduced rates of mortality compared with calcium-based binders. Reprinted by permission from Macmillan publisher Ltd: *Kidney Int.*³⁴ Copyright 2007.

Table 2 Summary of phosphorus and protein content of selected foods according to US Department of Agriculture national nutrient database²¹

Food	Common measure	Phosphorus content (mg)	Protein content (g)	Phosphorus (mg)/protein (g) ratio
Veal (leg)	85 g	212	31	6.8
Chicken (broiled)	140 g	259	35	7.4
Lamb (leg)	85 g	162	22	7.4
Beef (roasted)	85 g	200	26.4	7.6
Turkey (roasted)	85 g	208	24	8.7
Fish (cod, canned)	85 g	221	19.4	11.4
Pork	85 g	224	18	12.4
Crab	85 g	238	16.5	14.4
Salmon	85 g	280	17	16.5
Bread (white)	1 slice	25	3.4	7.3
Bagel (plain)	3 1/2"	68	7.5	9.0
Bread (mixed grain)	1 slice	46	2.6	17.7
Almonds	28 g	134	6.0	22.3
Pistachio	28 g	137	6.0	22.8
Walnuts	28 g	98	4.3	22.8
Biscuits	2 1/2	98	4.2	23.3
Cereals (Kellogg's® Raisin Bran)	250 mL	259	5.2	49.8
Cereals (General Mills)	250 mL	232	4.4	52.7
Egg, white raw	1 large	5	3.64	1.4
Egg, whole, fried	1 large	96	6.27	15.3
Cheese (Muenster)	28 g	133	6.64	20.0
Egg, yolk, raw	1 large	65	2.63	24.7
Cheese (American)	28 g	124	4.65	26.6
Yogurt (plain, low-fat)	236 mL	327	11.9	27.5
Milk (whole)	250 mL	222	7.86	28.2
Milk shake	312 mL	378	9.15	41.3

phosphorus levels without risking malnutrition. In total, 13 patients with serum phosphorus > 4.0 mg/dL consumed one meal containing 8 ounces (225 g) of pasteurized liquid egg whites per day for 6 weeks.¹⁵ At the end of the study, twelve patients exhibited a significant reduction in mean serum phosphorus and a significant increase in mean serum albumin (Figure 5).¹⁵ Dietary phosphorus restriction for 3 months in chronic hemodialysis patients receiving a low-phosphorus and low-potassium whey protein concentrate to partially replace food protein also demonstrated a beneficial reduction in serum phosphorus and intact PTH without detrimental changes to nutritional status.⁴⁰

Dietary counseling should encourage the consumption of foods with the least amount of inorganic phosphorus, low phosphorus-to-protein ratios, and adequate protein content, consistent with acceptable palatability.¹² Differences in the source of dietary protein and the bioavailability of phosphorus can have a significant impact on serum phosphorus control. In predialysis CKD patients who ate a metabolic-laboratory vegetarian diet for just 1 week, serum phosphorus levels and fibroblast growth factor-23 levels were lower compared with patients consuming a meat diet with the same protein,

calories, and phosphorus intake level.⁴¹ Other measures that should be encouraged include the use of selective over non-selective vitamin D activators to minimize intestinal phosphorus absorption,⁴² the diligent use of phosphorus binders to reduce the pill burden, and patient-friendly educational tools such as the concept of a dietary "phosphate unit" and its relationship with binder dose.⁴³ There is evidence that education on avoiding foods with phosphorus additives does contribute to better reduction in serum phosphorus levels versus control participants.⁴⁴ For 3 months, participants received education on avoiding foods with phosphorus additives when purchasing groceries or visiting fast food restaurants, or continued to receive usual care. After 3 months, the decline in serum phosphorus levels was significantly greater among intervention versus control participants. Intervention participants also had statistically significant increases in reading ingredient lists and nutrition facts labels.

In teaching patients to take control of dietary phosphorus and protein intake, educators need to provide patients tools and demonstrate how to recognize and avoid inorganic phosphorus additives; how to select protein sources and achieve protein adequacy; and how to estimate the phosphorus

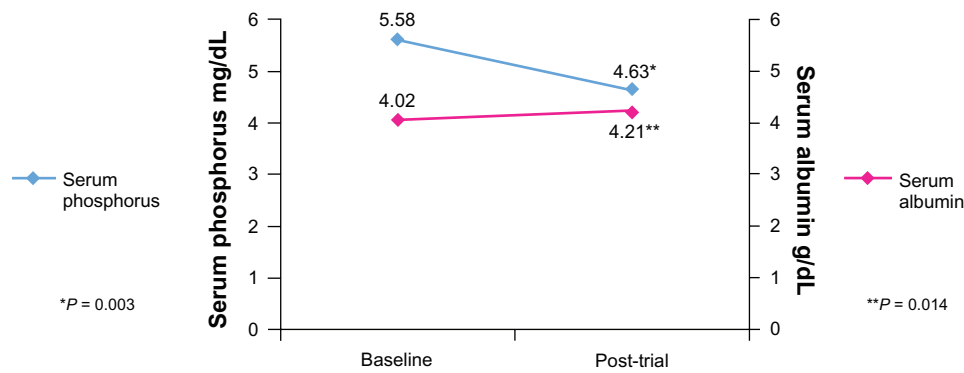


Figure 5 A pilot study in maintenance hemodialysis patients demonstrated the benefit of egg whites (1 meal a day for 6 weeks) as a major protein source to provide a high protein/low phosphorus intake. Mean serum phosphorus decreased and mean serum albumin increased. Data graphed de novo from Taylor et al.¹⁵

content of chosen foods. Glossaries of additives used in conjunction with label-reading (see Table 1 for an example) can help patients restrict consumption of phosphorus in processed and fast foods. Food charts and tables can help patients identify foods low in phosphorus. Reliable estimation of the phosphorus content of chosen foods is possible with the use of comprehensive labelling of phosphorus additives, and a “traffic light” scheme can help to simplify foods into “high,” “intermediate,” or “low” phosphorus content.⁴⁵ Protein adequacy with lesser phosphorus burden can be facilitated by teaching patients cooking methods that reduce the phosphorus content while preserving protein content, such as boiling chicken or beef.⁴⁶ In addition, motivational interview techniques that adopt a goal-oriented, patient-centered counseling approach are useful to encourage adherence to selected protein sources while emphasizing that dietary changes reduce the risk of mortality (Table 3).⁴⁷ Regular monitoring of dietary phosphorus and protein intake can be achieved with food frequency questionnaires, which allow patients’ difficulties with adherence to be easily identified.⁴⁸ However, it has been proposed that food frequency questionnaires should be used for population-level dietary comparisons of patients rather than for individual assessment.⁴⁹

Food fatigue is an emerging issue with specialized diets required in the management of chronic diseases, including CKD; food fatigue is a greater threat to dietary persistence than food allergies or intolerance. For instance, CKD patients required to consume specific foods, such as egg whites, may gradually become disinterested and less adherent. This can be overcome by diversifying the diet to include additional low-phosphorus, high-protein alternatives (eg, poultry). Variety is important, although choice should be limited where preference bias strongly affects nutritional content.⁵⁰ Phosphorus binder use can also help overcome food fatigue by allowing

patients’ preferred mainstream foods while moderating their impact on serum phosphorus levels.

Phosphorus binder use: education for concordance and adherence

Current KDIGO guidelines recommend using phosphorus binders to treat hyperphosphatemia in patients with CKD stages 3–5D.¹⁷ In contrast, the US Food and Drug Administration labelling of prescription phosphorus binders is specific to dialysis recipients with hyperphosphatemia. Incident dialysis patients receiving binders within the first 90 days experience a survival benefit.⁵¹ Similarly, in non-dialysis-dependent CKD, phosphorus binders have demonstrated a survival benefit in men with moderate and advanced disease (Figure 3).²⁸ Some studies suggest that sevelamer may improve surrogate markers of CKD progression (eg, serum phosphorus, bicarbonate) and CV risk (eg, calcification) in predialysis patients;^{52,53} however, larger and more rigorous studies are warranted to confirm these findings. Phosphorus binders also allow greater dietary variety and liberalized protein intake, which may address both protein needs and food fatigue. Furthermore, the availability of tablet or powder formulations improves patient choice and versatility.

Since phosphorus binders need to be taken properly and consistently with meals and snacks to be effective, patient education is important in their successful use. Patients should be aware that phosphorus binders have to be taken at mealtimes, either during or towards the end of each meal. Patient dosing errors observed by dialysis teams have included failure to coordinate each dose with a meal, consuming the entire daily dose first thing in the morning before a dialysis session, or taking a fixed dose irrespective of food intake or phosphorus content. The daily pill burden in ESRD is one of the highest among chronic

Table 3 The motivational interviewing tool kit⁴⁷

Motivational factor	Objective	Example	Follow-up
1. Express empathy	To establish rapport and avoid resistance by demonstrating understanding of the patient's situation	Patient expresses difficulty making all these changes	Remind the patient that current levels put them at risk for more serious diseases
2. Roll with resistance	Avoid magnifying resistance by allowing patient to explore their barriers in a nonjudgmental supportive manner	Patient is reluctant to continue medication since it is hard to remember to take and they no longer feel unwell	Ask the patient where they see themselves in 6 months if they stop taking the medication
3. Elicit/provide reminder/elicited	Find out what the patient already knows, fill in the gaps or correct misconceptions, and explore how the change you suggest will fit into the patient's life	Elicit: Ask patient what they know about managing their CKD	Reminder: For example, to take statins and closely monitor blood pressure Elicit: "What do you think the biggest barrier is for you right now in managing this condition?"
4. Support autonomy	To reduce resistance by assuring patients you know you can't make them do anything – it is their choice	Emphasize to patient that it is their choice but as their doctor you are concerned if they do not try medication	Reiterate that it is the patient's choice and they need to consider all the options. Reassure patient that if they do decide to try a particular medication they will be regularly monitored for side effects and dose adjusted accordingly
5. Explore ambivalence	Help patient consider pros and cons of change in a relaxed yet systematic manner	Encourage a discussion about the pros and cons, eg, eating egg whites as part of a low-phosphorus diet	Summarize current situation with the patient and explain that the benefits will outweigh the potential drawbacks associated with an egg-white diet
6. Elicit change talk	To evoke the patient's reasons, desire, ability, and need for change. This predicts increased commitment to the lifestyle change and good clinical outcome	"What makes it important to you to start an exercise program?" "What benefits would come from losing weight?" "Why do you want to quit smoking?"	Remind patient of the benefits of regular exercise and how well it made them feel previously. These measures will help patient become a good role model for their children and allow them to play sports together
7. Develop an action plan	To help the patient develop a plan that is realistic and suitable for their life	Enquire about the next step for the patient. Ask what they think they can do or are willing to do to improve health and make a difference	Motivate patient to follow plan and reiterate the steps agreed, ie, eat more vegetables, avoid fast foods, exercise more, etc

Abbreviation: CKD, chronic kidney disease.

diseases; phosphorus binders appear to be the largest single contributor.⁵⁴ The high pill burden may affect adherence and the ability to maintain optimal phosphorus levels,⁵⁵ which emphasizes the need to educate patients on the consequences of avoiding their medication, and to adapt binder use to the phosphorus content of meals. Binders are available in different dosage forms (eg, sevelamer carbonate tablets or powder for oral suspension; lanthanum carbonate chewable wafers); thus, patients who object to a particular dosing method should be given other options. In addition, motivational teaching methods can help patients overcome the pill burden, and reports from electronic medical records could identify the least adherent patients who require the most assistance. Medication therapy management programs

can provide patients with ongoing adherence follow-up and problem-solving.

Recent educational initiatives by German nephrologist Martin Kuhlmann teach patients to estimate the phosphorus content of food and adjust the number of binder tablets accordingly at each meal.^{43,56,57} The Phosphate Education Program (PEP) provides simple training tools to instruct patients to eye-estimate meal phosphorus content based on newly defined phosphorus units (1 phosphorus unit = 100 mg/serving), and to self-adjust phosphorus binder dosage accordingly.^{43,56} Phosphorus units can be assigned to whole food groups since similar food groups (eg, meat, seafood, vegetables) tend to have similar phosphorus content. Using this approach, patients do not have to memorize

the phosphorus content of each individual food component but only the phosphorus unit value for a limited number of food groups. A set of 180 photo cards illustrating servings of different foods with their phosphorus unit values has been used to teach this approach to juvenile patients.⁵⁷ After eye-estimating the phosphorus unit content of a meal, the patient self-adjusts the binder dose according to a binder/unit ratio prescribed by the nephrologist. The binder/unit ratio is titrated to the patient's individual needs by repeatedly measuring predialysis serum phosphorus levels and readjusting the binder/unit ratio until phosphorus targets are achieved. Experience with the PEP showed significantly improved control of hyperphosphatemia even without reducing dietary inorganic phosphorus intake.⁵⁷

Other initiatives to improve concordance and adherence include increasing the frequency of consultations and dietitian-to-patient ratio in dialysis clinics; monthly consultations for a period of 6 months and a ratio not exceeding 60 patients per dietitian have been shown to improve short-term phosphorus control.⁵⁸ Furthermore, nutritional counseling should involve the entire dialysis team – not only the nephrologist and dietitian but also the nurse and social worker. Educational materials including pamphlets, videos, websites, and posters are useful tools to reinforce instruction for both patients and members of the renal team on how to optimize serum phosphorus levels. Another educational initiative includes dietitian-led education programs that test patients' general knowledge of phosphorus and phosphorus binders, and its impact on serum phosphorus concentrations. Small teaching sessions delivered by a single dietitian have been shown to significantly reduce serum phosphorus levels after 1 month.⁵⁹

In summary, a variety of educational tools are available to improve the control of dietary phosphorus and adherence with phosphorus binders. Optimal phosphorus control requires an individualized approach with regular follow-up. This strategy should also be advocated in compliant patients achieving target phosphorus levels in order to provide encouragement and reinforce positive behavior.

Calcium risk-awareness and appropriate intake

In CKD patients, a positive calcium balance arises because intestinal absorption is greater than the kidney's capacity to excrete. Not all calcium sources are the same. Dietary calcium (especially dairy) coexists with phosphorus, protein, and fat; is absorbed more slowly than inorganic calcium supplements; and thus may be associated with less hypercalcemia and CV risk.^{60,61}

Calcium intake within the Institute of Medicine (IOM) recommendations is important for health. The IOM's revised calcium guidelines recommend keeping total calcium intake below 2000 mg/day for the general population; 1200 mg/day is the recommended daily allowance for seniors (women > 50 years and men > 70 years) and 1000 mg/day for all other adults.⁶² There is evidence, however, that total elemental calcium intake should be within 800–1200 mg/day to prevent calcium deficiency or calcium loading. Excessive calcium intake, especially from inorganic supplements, is associated with CV risk in the general public and patients with CKD.⁶³ A recent meta-analysis in 29,000 postmenopausal women suggested that calcium supplementation increases risk of myocardial infarction and possibly stroke.⁶⁴ The high media profile of osteoporosis has encouraged many Americans, particularly menopausal women, to consume over-the-counter calcium supplements on their own initiative to minimize the risk of fractures; some users erroneously assume that more calcium is better. Patients with CKD/ESRD should be encouraged to communicate openly to renal team members about any and all "self-prescribed" over-the-counter supplements, medications, or herbal products to avoid renal risks. Conversely, the renal team should educate patients about the risks associated with excessive calcium and teach patients how to obtain adequate and moderate calcium intake from foods rather than supplements.

Importantly, serum calcium concentration is regulated within narrow limits and thus is a poor guide to evaluate intake or calcium balance. Increased calcium intake significantly decreases active vitamin D and intact PTH levels but has no effect on serum calcium concentration.⁶⁵ It is important for nephrologists to account for total calcium intake not only from diet or supplements but also from calcium-based binders, as binder calcium can also be systemically absorbed.⁶⁶ In patients with CKD stages 3–5 and hyperphosphatemia, KDIGO advises restricting the dose of calcium-based phosphorus binders in the presence of hypercalcemia (persistent or recurrent), arterial calcification, adynamic bone disease, or persistently low PTH.¹⁷ High serum calcium promotes vascular calcification by mechanisms that are not completely resolved. At any serum phosphorus level, higher serum calcium concentrations are associated with increased risk of death in dialysis patients.⁶⁷ Studies have shown that calcium-free binders such as sevelamer reduce the progression of vascular calcification compared with calcium-based binders in dialysis^{32,68–73} and predialysis CKD patients,^{53,74,75} and thus merit consideration in patients with high CV risk.

Conclusion

Recalcitrant hyperphosphatemia in patients with ESRD is related to dietary nonadherence (including overexposure to phosphorus additives and excessive reliance on high phosphorus-to-protein ratio animal sources), binder-related issues (pill burden and medication nonadherence, dosing errors, and adverse effects), and dialysis inadequacy (missed or shortened sessions). Balancing the risk versus benefit of dietary phosphorus for the management of hyperphosphatemia in CKD-MBD requires more attention to the restriction of bioavailable inorganic dietary phosphorus (eg, fewer processed and fast foods); a liberal protein intake balanced by choice of low-phosphate entrees and appropriate binder use; more attention to phosphorus-to-protein ratio; and reinforcement of non-dietary phosphorus control by appropriate binder use and adherence, improved dialysis adequacy, and possibly longer or more frequent dialysis. While dietary control in the CKD patient is largely focused on achieving phosphorus and protein targets, it is also important to consider the impact of a positive calcium load, which can contribute to hypercalcemia, vascular and soft tissue calcification, and increased CV risk.

Patient education and open patient/team dialogue on phosphorus and calcium management can improve concordance and adherence and empower patients to collaborate actively for optimal control of mineral metabolism throughout CKD/ESRD. Emerging initiatives such as the PEP can provide a simple and effective means of identifying foods high in phosphorus and tailoring appropriate phosphorus binder use.

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