AJKD
In Practice

Balancing Nutrition and Serum Phosphorus in Maintenance Dialysis

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Elevated serum phosphorus levels are common in patients with chronic kidney disease and are associated with heart and vascular disease, conditions that in turn are associated with increased mortality. Accurately managing phosphorus intake by restricting dietary protein alone can prove challenging because protein from different sources can contain varying amounts of available phosphorus. Additives used in processed foods frequently are high in inorganic phosphorus, which is readily absorbed, compounding this difficulty. Recent evidence suggests that dietary protein restriction in some cases may do more harm than good in some patients treated with maintenance hemodialysis because protein restriction can lead to protein-energy wasting, which is associated with increased mortality. Accordingly, phosphorus binders are important for managing hyperphosphatemia in dialysis patients. Managing hyperphosphatemia in patients with late-stage chronic kidney disease requires an individualized approach, involving a combination of adequate dietary advice, phosphate-binder use, and adjustments to dialysis prescription. We speculate that increased use of phosphate binders could allow patients to eat more protein-rich foods and that communicating this to patients might increase their perception of their need for phosphate binders, providing an incentive to improve adherence. The aim of this review is to discuss the challenges involved in maintaining adequate nutrition while controlling phosphorus levels in patients on maintenance hemodialysis therapy.
Am J Kidney Dis. ■(■):■-■. © *2014 by the National Kidney Foundation, Inc.*

INDEX WORDS: Diet, protein-restricted; chronic kidney disease; phosphorus, dietary; phosphate binders.

CASE PRESENTATION

A 63-year-old white man with stage 5D chronic kidney disease (CKD) started on maintenance hemodialysis (HD) therapy 2.5 years ago. He presents with the following laboratory measurements (fasting midweek predialysis): serum phosphorus, 6.2 mg/dL (1.90 mmol/L); serum calcium, 10.4 mg/dL (2.60 mmol/L); serum parathyroid hormone (PTH), 280 pg/mL; serum 25-hydroxyvitamin D_3 (25[OH]D_3), 40 ng/mL (100 nmol/L); normalized protein catabolic rate, 1.25 g/kg/d; and serum albumin, 3.8 mg/dL. His medications include Calcijex (intravenous calcitriol, manufactured by Abbvie), 0.5 μg, each HD session; calcium carbonate, 1.5 g/d; and 100,000 units of cholecalciferol every 2 months. His dialysis dose is considered adequate, based on equilibrated Kt/V of 1.35. The patient's biannual food report shows sufficient protein intake of 1.1 g/kg/d and adequate energy intake of 31 kcal/kg/d, and the patient does not report excessive consumption of inorganic phosphate, so is advised not to modify his diet.

This patient has secondary hyperparathyroidism because of end-stage renal disease. He has elevated serum calcium and phosphorus levels, but a normal nutritional vitamin D (25[OH]D_3) level. As a first step toward reducing serum calcium levels, calcitriol treatment is stopped and calcium carbonate is replaced with a noncalcium phosphate binder.

After 3 months, the patient's laboratory values are as follows: serum phosphorus, 6.2 mg/dL (1.90 mmol/L); serum calcium, 8.8 mg/dL (2.20 mmol/L); serum PTH, 300 pg/mL; and serum 25(OH)D_3, 30 ng/mL (75 nmol/L). The patient's serum phosphorus level is still higher than the normal range, at a level that is associated with worse outcomes.[1] He has not adhered to treatment with the noncalcium phosphate binder. During his 6-monthly dietary interview, the patient reports frequently eating processed food and drinking soft drinks every day. The patient is counseled to improve adherence to phosphate binder treatment by discussing ways to fit his medications into his daily routine, as well as helping him understand the importance of taking them as prescribed. The phosphate contents of different foods and drinks also are discussed with the patient, and he is advised to eradicate processed food from his diet. The patient's serum phosphorus level subsequently decreases to 4.6 mg/dL (1.50 mmol/L) after 3 months.

INTRODUCTION

Serum phosphorus levels tend to be poorly controlled in patients with CKD.[1] Elevated serum phosphorus levels contribute to the disruption of bone metabolism and are associated with heart disease and increased mortality (Fig 1).[1] It therefore is important to control serum phosphorus levels in patients with CKD.

Target levels of serum phosphorus commonly are controlled using a combination of dietary restrictions

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Received May 6, 2013. Accepted in revised form January 14, 2014.

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© 2014 by the National Kidney Foundation, Inc.
0272-6386/$36.00
http://dx.doi.org/10.1053/j.ajkd.2014.01.429

and phosphate-binding drugs, in addition to adjusting dialysis session duration, filter surface, and convection during maintenance HD sessions.[2] One of the main dietary recommendations for patients with CKD who are not on dialysis therapy is restriction of protein consumption, which helps maintain serum phosphorus at controlled levels.[3] The National Kidney Foundation–Kidney Disease Outcomes Quality Initiative (NKF-KDOQI 2000) guidelines recommend protein intake of 0.6 g/kg of body weight per day for patients with glomerular filtration rates < 30 mL/min and 1.2 g/kg of body weight per day for patients on maintenance HD therapy.[4] In patients on maintenance HD therapy, the European Best Practice Guidelines recommend slightly lower dietary protein intake of 1.1 g/kg of ideal body weight per day.[3] Recent evidence has shown protein restriction to correlate with increased mortality in patients undergoing maintenance HD, suggesting that reduction of protein in the diet may be detrimental to these patients.[5] Thus, before maintenance HD therapy, serum phosphorus levels may be controlled by an optimal protein intake of 0.6 g/kg/d. However, after starting dialysis therapy, higher protein intake of 1.1-1.2 g/kg/d is recommended, and other strategies should be introduced to help control serum phosphorus levels. Depending on the extent of hyperphosphatemia and the patient's lifestyle requirements, these measures could include a combination of dietary counseling, phosphate-binder use, and dialysis prescription. Dietary counseling should be offered in the first instance; however, dietary support may be lacking in some renal wards.

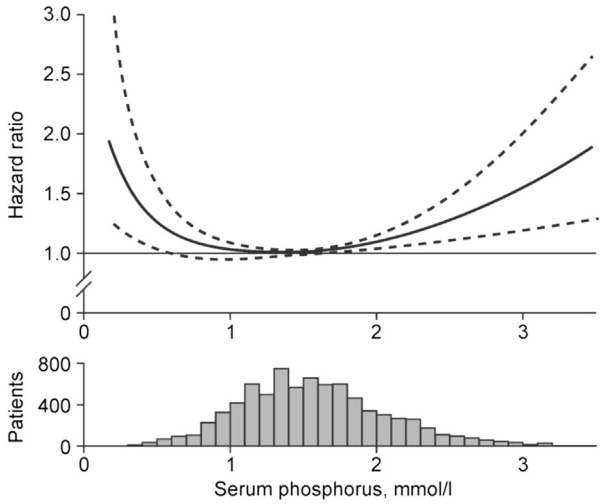

Figure 1. Mortality hazard ratio and serum phosphorus level. The hazard ratio for mortality (95% confidence interval noted by dashed lines) at 30 months after entry to the study, which is derived from Cox regression models adjusted for covariate values at baseline (age, sex, history of cardiovascular disease, diabetes, dialysis vintage, body mass index, serum albumin level, and hemoglobin level) and using fractional polynomials for (top) serum phosphorus level and (bottom) number of patients. Conversion factor for units: phosphorus in mmol/L to mg/dL, $\times 3.097$. Reproduced from Fouque et al,[1] with permission of Oxford University Press.

This review discusses the evidence surrounding protein restriction and phosphate control and highlights the challenges of maintaining appropriate nutrition at the same time as controlling serum phosphorus levels in patients undergoing maintenance HD. The review also explores the role of phosphate binders in managing serum phosphorus levels and reviews the potential additional benefits associated with phosphate-binder use.

PROTEIN INTAKE AND SERUM PHOSPHORUS

A common recommendation for the management of hyperphosphatemia is to reduce the amount of phosphorus in the diet.[2] The NKF-KDOQI guidelines and European Best Practice Guidelines recommend daily phosphorus intake of 800-1,000 mg/d for patients on maintenance HD therapy.[3,6,7] Foods with high protein content tend to have high phosphorus content, and an increase in dietary protein has been shown to correlate with an increase in serum phosphorus levels (Fig 2).[8] Limiting dietary phosphorus intake therefore generally involves restricting cheese and dairy products (Box 1).

Spontaneous reduction in food intake and progressive protein-energy wasting may occur in some patients.[9] Anorexia is the first result of this, followed by the possibility of overhydration, particularly during the long interdialytic interval. Whether limiting phosphorus intake per se will induce protein-energy wasting has not been addressed and deserves further study. However, dietary counseling should caution against excessive restriction. Phosphorus restriction in hyperphosphatemic patients may inadvertently result in a reduction in protein intake, which should be avoided.

Several trials have shown that controlled reduction of protein intake can help ameliorate the onset of the hyperparathyroidism that often is a consequence of CKD.[10] However, the ratio of phosphorus to protein in food is not constant, which can make it difficult to control dietary phosphorus intake accurately purely by reducing the amount of protein in the diet. For example, egg yolk has 15-20 times more phosphorus per gram of protein than egg white.[8]

Bioavailability should be considered when analyzing the relationship between phosphorus level and dietary protein. Animal proteins contain phosphorus primarily as organic phosphoesters, which are readily hydrolyzed and absorbed by the human digestive system.[8] However, plant foods that are high in protein, such as legumes, nuts, and chocolate, contain phosphorus mostly in the form of phytate or phytic acid, which is not readily broken down in the gut. The bioavailability of plant phosphorus therefore can be as low as 50%.

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