

Phosphorus, beverages, and chronic kidney disease

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Abstract: Phosphate is present in food in two forms, ie, organic and inorganic phosphate salts, which are naturally present in food and as phosphates added for industrial and commercial reasons. There is also a high content of added phosphate in beverages, and phosphates in this form are highly absorbable. The real content of phosphate contained in beverages is often unrecognized, and nephrologists do not always take into account the amount of phosphorus that patients ingest in this form.

Keywords: phosphorus, beverages, additive, diet

Introduction

Management of disorders of bone mineral metabolism in patients with chronic kidney disease is important, in particular because of the tight relationship between serum phosphorus levels and cardiovascular morbidity and mortality.^{1,2} The pathogenesis of bone mineral metabolism disorders is multifactorial, and is characterized by altered serum levels of phosphorus, calcium, and parathyroid hormone, as well as the phosphaturic hormone, FGF-23, a member of the fibroblast growth factor family; however, it is known that retention of phosphate due to dietary ingestion in excess of that appropriate for the level of renal function has a central role.³⁻⁷ Therefore, control of dietary phosphate ingestion and reduction of intestinal absorption of phosphate in the early stages of chronic kidney disease are needed.

About 60% of phosphorus contained in food is absorbed in the intestine as free inorganic phosphate, and in adults, the balance between phosphate excretion and intestinal absorption of phosphate is maintained via the kidney. Phosphorus levels remain within the normal range until renal function has decreased dramatically,⁸ but they can still increase, even within the normal range, in the early stages of chronic kidney disease (ie, glomerular filtration rate 50 mL per minute), and be accompanied by an increase in parathyroid hormone levels. Even earlier, there may be a rise in FGF-23; this hormone has a phosphaturic action and contributes to the balance of phosphate that would be otherwise positive from the earliest stages of chronic kidney disease, especially with an unrestricted diet.^{9,10}

Dietary phosphate comes from two sources, ie, either naturally contained in food as an organic phosphate or as inorganic salt bound to biological substances, or it can be an additive for industrial and commercial reasons. As an additive, phosphate is present as an inorganic and highly dissociable salt which is easily absorbable in the intestine; in fact, more than 90% of phosphate additives are absorbed in the

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intestinal tract, whereas only about 60% of the phosphate naturally present in food is absorbed, especially if ingested in animal product form as opposed to vegetable product form.¹¹ The content of phosphate in beverages is often unrecognized. More than 90% of this type of phosphate is absorbed, but nephrologists do not always take into account the exact amount of phosphorus that patients ingest through beverages.

Phosphorus content of common beverages

Savica et al¹² reported the phosphate content in common beverages to be as follows: red wine 303 mg/L; white wine 217 mg/L; cola beverages 171 mg/L; light cola 120 mg/L; beer 110 mg/L; coffee 51 mg/L; tea 30 mg/L; orange juice 16 mg/L; lemon juice 10 mg/L; and mineral water 6–10 mg/L.

Kalantar-Zadeh et al¹³ reports the following concentrations in mg (based on 12 oz servings): Coca-cola® classic 62 (172 mg/L); Diet cola 27 (75 mg/L); Fanta® 11 (30 mg/L); Gatorade® 36 (100 mg/L); Lipton® Brisk tea 97–189 (269–525 mg/L); and Diet Pepsi® 41 to 68 (114–189 mg/L). Cupisti and D'Alessandro¹¹ report that the concentration of phosphorus in beer is 280 mg/L, and Noori et al¹⁴ report that the concentration of phosphorus (based on a 12 oz serving) in cola is 40 (111 mg/L). Finally, the National Institute of Research for Food and Nutrition in Italy (http://www.inran.it/646/tabelle_di_composizione_degli_alimenti.html) reports the following concentrations of phosphorus in common beverages: white wine 740 mg/L; beer 280 mg/L; cola 180 mg/L; and orange juice 110 mg/L.

Our data

In a recent paper, we showed that a very low protein diet (0.3 g/kg bodyweight per day) significantly reduced serum and urinary phosphate levels in patients with chronic kidney disease,¹⁵ in particular, if the very low protein diet was obtained using low-protein products (pasta and bread), vegetables, essential amino acids, and keto analogs of nonessential amino acids. This diet allowed a reduction in serum phosphate from 4.0 ± 0.5 mg/dL to 3.5 ± 0.6 mg/dL ($P < 0.001$) and in urinary phosphate from 703 ± 276 mg/day to 464 ± 209 mg/day ($P < 0.008$), compared with a usual low protein diet,¹⁵ which is consistent with a recent report by Moe et al.¹⁶ However, if we compare the observed urinary phosphate with the expected measurement (from the dietary phosphate intake prescribed) for each patient, the urinary phosphate should be 629 ± 41 mg/day in a low protein

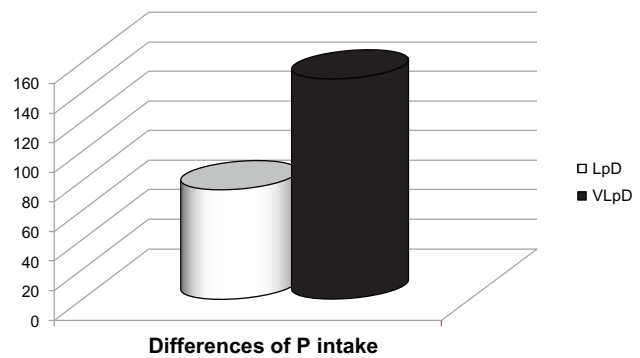


Figure 1 Differences in prescribed and observed dietary phosphorus intake (mg/day). **Abbreviations:** LpD, low protein diet; VLpD, very low protein diet.

diet, with a difference of 74 ± 35 mg/day compared with the observed urinary phosphate ($P < 0.001$); in a very low protein diet, the urinary phosphate should be 316 ± 27 mg/day, representing a difference of 149 ± 91 mg/day ($P < 0.001$, Figure 1). This significant difference between the observed and expected urinary phosphate in the two diets can only be explained with the phosphate contained in additives and beverages.¹⁷ In fact, in our study population, the amount of proteins ingested with the diet (evaluated from the urinary urea excretion) corresponded perfectly with the amount of dietary protein prescribed (0.73 ± 0.07 in a low protein diet and 0.34 ± 0.09 in a very low protein diet).¹⁵ The dietetic interview performed with our patients demonstrated that beverage intake was 245 ± 181 mL/day for red wine, 102 ± 89 mL/day for cola, and 65 ± 112 mL/day for fruit juice. Considering that the additives contained in the food ingested by our patients partially explained the differences in urinary phosphate levels, the beverages account for the additional phosphate intake of about 73 mg/day for red wine, 17 mg/day per cola, and 8 mg/day for fruit juice, for a total of about 90–100 mg/day.

Conclusion

Reduction of net intestinal absorption is the first goal during the earliest stages of chronic kidney disease, in order to prevent or to treat the onset of disordered bone mineral metabolism in patients with chronic kidney disease, such as an increase in FGF-23, inhibition of alfa-1-hydroxylase, reduced calcitriol, and an increase in synthesis and secretion of parathyroid hormone.

Therefore, the control and reduction in dietary intake of phosphorus are the first steps in the management of patients with chronic kidney disease. It is evident that there is variability in the phosphate content of beverages reported by the authors and by the Italian National Research Institute for Food and Feeding, and this problem needs to be taken

into account in the management of patients with chronic kidney disease.

If patients are drinking various beverages with a high phosphate content, these could contribute to their hyperphosphatemia and to poor results obtained by treatment to reduce hyperphosphatemia. High dietary phosphorus intake has deleterious consequences for renal patients and may be harmful also for the general population.¹⁸ To prevent hyperphosphatemia, patients with end-stage renal disease must limit their intake of foods that are naturally rich in phosphorus, and education programs for end-stage renal disease patients aimed at avoiding phosphorus-containing food and additives result in improved control of hyperphosphatemia.¹⁷

Disclosure

The authors report no conflicts of interest in this work.

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