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Review Article

Dietary management of hyperphosphatemia in chronic kidney disease

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ABSTRACT

Dysregulation of phosphate homeostasis occurs in chronic kidney disease (CKD). Hyperphosphatemia is an ongoing challenge in treating CKD patients. Restriction of dietary proteins remains one of the cornerstones of nutritional management of CKD patients foods from animal sources are rich in organic phosphorus. Foods sources including certain beverages like colas, enhanced meats, frozen meals, snack bars, processed or spreadable cheeses, instant food products, and refrigerated bakery products are rich in inorganic phosphorus.

Phosphate additives added to foods further increases the phosphorus burden. It is estimated that the intestinal absorption of inorganic phosphorus is usually more than 90% compared to only 40%–60% from that of the organic phosphorus. Phosphates from animal food are more readily absorbed compared to that present in plant foods sources as majority of it is present in the form of phytate and hence not readily absorbed. Intensive nutritional counseling regarding phosphorus content of foods, their bioavailability with an emphasis on consumption of a mixed diet including foods from animal sources and plant sources high in phytate. While limiting or avoiding the intake from foods very high in phosphorus to protein ratio and foods rich in phosphorus additives but with an adequate protein content to avoid malnutrition, reinforcement on dietary compliance and judicious use of phosphorus binders are important for the better management of hyperphosphatemia in CKD. Methods like soaking foods in water and boiling them helps in reducing the dietary phosphorus content per gram of protein in foods.

1. Introduction

Chronic Kidney Disease (CKD) is one of the important causes of morbidity and mortality worldwide. The associated mineral and bone disorder (MBD) is one of the significant contributors to CKD.[1] The progressive renal failure causes accumulation of many substances including phosphorus (P). In normal conditions, phosphorus is excreted by the kidney. A variety of

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compensatory mechanisms keeps the serum P level within normal range until kidney functions starts deteriorating progressively to end stage renal disease (ESRD).[2] This reduced P filtration and excretion with the progressive deterioration of kidneys leads to hyperphosphatemia.[3] Serum phosphate above 5 mg/dl (1.6 mmol/l) is known as hyperphosphatemia.[4] Optimum management of hyperphosphatemia is often a major challenge for renal care team involving nephrologists, nutritionist, nurses and for the patients themselves.[5] The present article is aimed at reviewing the various aspects of dietary management of hyperphosphatemia in CKD patients Tables 1 and 2.

2. Causes of hyperphosphatemia

Hyperphosphatemia is multifactorial in nature. (a) Neither hemodialysis nor peritoneal dialysis is able to effectively remove the dietary phosphorus[6,7] (b) Non compliance with phosphate binder medications including pill burden and cost etc leading to insufficient phosphate removal[8] (c) over concern regarding the consequences of dietary protein also limits the success in reducing dietary phosphorus intake[9,10] (d) Lack of knowledge and awareness about the sources and content of phosphorus in various foodstuffs and non-compliance to dietary advice is also one of the important reasons for hyperphosphatemia.[11–14] (e) Phosphate additives or preservatives used in processed foods contribute to phosphate burden.[15]

3. Mechanism of hyperphosphatemia

It is now known that the serum phosphate concentration is controlled by two newly discovered factors called fibroblast growth factor 23 (FGF 23) and klotho[16,17] CKD patients are not able to excrete P properly. This diminish glomerular filtration of phosphorus is initially maintained by various compensatory mechanisms including decrease tubular reabsorption of P by PTH and FGF 23. This helps in maintenance of normal serum P.[18] Afterward the serum phosphate increases with advanced stages of CKD.

3.1. *Consequences of hyperphosphatemia*

Hyperphosphatemia in long run may results in hyperparathyroidism, metabolic bone disease, calcifying uremic arteriolopathy, and cardiovascular calcification. Higher risk of death is caused by progressive increase in arterial calcification.[19]

3.2. *Target for serum phosphorus*

The NKF KDOQI 2003 guidelines recommended aiming a normal phosphate level range of 2.7–4.6 mg/dl in patients with CKD stage 3–4 and a target of 3.5–5.5 mg/dl in CKD stage 5 and 5D.[20]As suggested by the Kidney Disease Improving Global Outcomes 2009 guidelines, the need is to regularize the levels of serum phosphates in stages 3–5 of CKD patients. It is also recommended that the levels of serum phosphorus should be within normal range for CKD patients in stage 5D; however these guidelines refrained from recommending specific targets[21]

3.3. *Treatment of hyperphosphatemia*

Hyperphosphatemia remains ongoing challenge in clinical management of CKD and ESRD patients on either long-term peritoneal dialysis (PD) or Maintenance Hemodialysis (MHD). Therapeutic strategies targeting phosphorus control include restriction of dietary phosphorus, reducing intestinal absorption of phosphorus with appropriate phosphate binders, and removing phosphorus with dialysis.[22] Dialysis patients on high protein diet are more prone to a higher dietary phosphorus load compared to the patients at early stages of CKD on medical management who are maintained on a low protein diet.[23] A diet low in phosphorus together without/with inclusion of phosphate binders for stage 3 and 4 CKD even in patients with normal serum phosphorus levels patients may prevent the hyperphosphatemia.[20,21,24]

3.4. *Dietary management*

Restriction of dietary proteins remains one of the cornerstones of nutritional management of CKD patients.[25] Comprehensive management of hyperphosphatemia includes (5.26):

(a) Judicious restriction of dietary phosphorus while maintaining adequate dietary protein intake
(b) Decreasing intestinal absorption of phosphorus through use of appropriate phosphate binders
(c) Removal of excess phosphate by intensifying dialysis therapy

Irrespective of the various options available, control of dietary phosphorus remains the forefront of management of hyperphosphatemia. Proper and adequate awareness and knowledge of various dietary sources of P is rudimentary and crucial in the dietary management of hyperphosphatemia in CKD patients.[22] However, restriction of dietary phosphorus is

Table 1 – Table recommending intake of dietary phosphorus for stage 5 CKD patients.

Guidelines	Hemodialysis	Hemodialysis
NKF-K/DOQI 2003	Restricted to 800–1000 mg/day if serum phosphorus level >5.5 mg/dL	Restricted to 800–1000 mg/day if serum phosphorus level >5.5 mg/dL
ESPEN	17 mg/kg IBW	17 mg kg IBW
EBPG	800-1000 mg/day	–
EDTNA/ERCA	1000–1400 mg/day	1000–1400 mg/day

Adopted from: NKF 2000 (DOQI)[30]: Toigo et al (2000) (ESPEN);[31] EDTNA/ERCA 2002;[32] Fouque et al (2007) (EPBG).[33]