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A Single-Center Study of Chronic Kidney Disease and Dietary Phosphate Restriction

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Abstract **Review Article**

Background: Phosphate Excretion Declines With Renal Failure. PTH And FGF23 Diminish Glomerular Phosphorus Filtration, Which Decreases Tubular Reabsorption. Protein And Phosphorus Are Linked. Proteinuria Who Eat A Low-Protein Diet Reduce The Progression Of Renal Disease And Improve Their Survival. Not All Animal Proteins And Plants Have The Same Phosphorus Content. Food Labels Must Indicate The Phosphorus-To-Protein Ratio For Accuracy. Low-Protein Diets May Increase Mortality And Morbidity In End-Stage CKD Patients, Sparking Debate. Using Phosphate-Binding Medications To Reduce FGF23 And Serum Phosphorus Had No Effect On Protein Intake. A Patient's Phosphate Binder Tolerance and Intestinal Dysbacteriosis May Hinder Dialysis.

Keywords: Chronic Kidney Disease Dietary Phosphate Restriction.

1. INTRODUCTION

Daily Phosphorus Intake Is 950 Mg. Bone Contains 29% Of The Body's Phosphorous, Whereas Blood Contains Just 1%. 70% Of Phosphorus Is In Cells; therefore, It May Be Used In Many Ways. The Stomach and Urinary Systems Excrete Phosphorus (150 And 800 Mg) [1, 2]. Normal Renal Function Patients With Acute Phosphaturia May Have Intestinal Phosphatonins To Blame. Phosphorus Excess Calls For Extra Phosphatonins. PTH And FGF23 Are The Body's Two Most Frequent Hormones (FGF23).

Phosphate Excretion Declines With Renal Failure. PTH And FGF 23 Activity Are Reduced To Compensate For Tubular Phosphorus Reabsorption. When Phosphorus Deficiency Is Restored, 24-Hour Excretion Normalises [3]. 24-Hour Peeing Isn't Enough.

We Can't Analyse Phosphorus Excretion Without Knowing How Much Was Consumed. Renal Dysfunction Causes A Positive Phosphorus Balance.

FGF23 Lowers Na/P Cotransporter Type II Activity And Inhibits Renal 1 Alpha Hydrox-Ylase In Proximal Tubules, Limiting Gut Phosphorus Absorption And Reabsorption. Reduced PTH Production And Reduced Vitamin D Levels Increase Renal Phosphorus Excretion. FGF23 Is Secreted By Bone To Maintain Phosphorus Balance [4].

2. Nutritional Effects of Consuming Protein and Phosphorus

Protein and phosphorus go together [7]. Most scientific organizations advise individuals with chronic renal failure to reduce protein intake early to minimize phosphorus intake. 30–70% of a gram of protein's phosphorus is absorbed.

Gut. By consuming 90 g of protein per day, you may absorb 600–700 mg of phosphorus. Hemodialysis leaves a 1200–1400 mg/day net positive phosphorus balance, removing 500–600 mg/session over 48 hours. Chronic renal disease has two major protein-restricting causes. Proteinuria should eat less protein [8]. A protein-restricted diet decreases phosphorus, which affects renal disease and patient lifespan. Low-protein diets have other advantages (Table 1). Meta-analyses reveal that 0.6 to 0.8 grams of protein per kilogram of body weight per day are best for people with severe chronic kidney disease (CKD) [9]. This constraint is sound nutritionally and metabolically [10].

After dialysis, eat more protein. Hemodialysis patients who ingest more protein had a better prognosis [11]. In a post-hoc study [12], HEMO patients who didn't follow a protein restriction diet performed better. A high-protein diet is connected to excessive phosphorus intake, which increases cardiovascular mortality risk. These factors, including protein and energy levels, are still linked to this finding [13]. Limit your phosphorus intake to acquire adequate protein.

Adults should have 700 milligrams of phosphorus per day, while children and pregnant women need 1250 [14, 15]. Renal patients should minimize intake. Limit phosphorus-containing food additives.

3. Different Types of Protein and Phosphorus Absorption

Dietary phosphorus takes several forms. Protein-bound phosphorus is poorly absorbed. Inorganic phosphorus in additives and preservatives absorbs above 90%. Phosphate is a typical food and drink preservative, especially in cola. Plant protein has less organic phosphorus than animal protein. Mammals lack enzymes that break down phytates, plant-based phosphorus sources. Organic phosphate is quickly hydrolyzed and absorbed [16].

Rats with slowly developing renal failure had the same blood phosphorus levels on a casein-based or grain-based protein diet. Casein-fed rats had higher urinary phosphorus excretion and serum FGF23 levels [17].

Table-1: Restriction of protein intake in advanced chronic renal disease: consequences.

Reduces the amount of protein excreted in the urine. Enhances lipid regulation.
Remove toxic and acidic waste from the kidneys to improve insulin resistance and lowers levels of oxidative damage, lowering the phosphorus burden.

11 CKD stages 3- 4 patients received animal or vegetable protein for seven days. Animal protein intake affected serum phosphorus and FGF23 more than vegetable protein. Vegetable-heavy, low-meat, and convenience foods should be avoided. Reduce preservatives and additives.

Animal proteins and plants have different phosphorus levels. Tables and photos demonstrate phosphorus levels in other meals. Labels must show the phosphorus-to-protein ratio (in mg) (grams). The wide variance between 10 and 65 mg/g. High cheese-to-soft-drink ratio. KDOQI recommends this ratio [19].

a) It does not rely on the amount of food supplied.
b) It represents both phosphorus and protein in onemolecule.
c) Phosphorous-rich foods, such as soft drinks andfood additives, are highlighted.

This ratio doesn't indicate phosphorus bioavailability from different sources, but it's still informative. CKD patients should eat a low-phosphorus, low-inorganic-phosphorus, and low-phosphorus/protein diet with adequate protein to boost food appeal.

In Spain, the Mediterranean diet reduces homocysteine, serum phosphorus, microalbuminuria, and cardiovascular risk [20]. Additives and preservatives include phosphorus [21]. The average American eats 1000 mg of phosphorus each day. Hemodialysis patients should know this [22]. Soft drinks and cheese have a high phosphorus-protein ratio [23].

Low-quality food means increased phosphorus intake. Serum phosphorus levels were more significant in low-income participants, perhaps because they ate more packed and fast food [24].

4. Malnutrition As A Result Of A Low-Protein Diet

Diet in advanced CKD has been controversial throughout Nephrology's history. Protein-calorie deficiency causes CKD [25]. A low-protein diet may cause malnutrition, illness, and mortality [7]. Low-protein diets may slow the renal disease. High-protein diets may cause uremic symptoms and hyperphosphatemia. Seek equilibrium. A low-protein diet in CKD may reduce uremic symptoms [11], improve phosphorus control [11], delay dialysis [9], not increase protein malnutrition if accompanied by essential amino acid supplement [26], not increase mortality in patients with a low-protein diet after starting dialysis [27], and protect against oxidative stress [28].

Protein intake should not be lowered in dialysis despite a higher phosphorus intake because protein insufficiency and mortality outweigh hyperphosphorusemia [11]. Real protein consumption is lower than projected when dialysis patients are recommended a low-protein diet, perhaps due to the diet's difficulties. Thus, 0.3–0.6 g/kg/day of protein should result in 0.48–s0.84 g/kg/day [26, 29]. Low-protein diet implementation requires nurses, dietitians, and nephrologists. The net phosphorus balance on a normoproteic diet in hemodialysis patients is positive after removing dialysis phosphorus. Hemodialysis removes 800 mg phosphorus/session (2400/week). Thus, a 1 g/kg B.W./day protein intake would result in a 2000 mg weekly net phosphorus balance.

Savica *et al*. propose that patients undergoing periodic H.D. consume 1.2–1.4 g of protein per kg of body weight and 800 mg of phosphate per day. 1, 2–1,4 g protein equals.

1.450–1600 mg/day of phosphorus equals 0.6 mg/kg b.w./day. Patients should limit their daily phosphate intake to 800 mg. Dialysis or phosphate-binding agents [15] don't remove phosphate. 74% of CKD patients drink; thus, we may estimate a weekly net positive phosphate balance of 2,800 mg. Consuming this much phosphate per week is detrimental for CKD patients [30].

New techniques are needed to minimize phosphorus consumption in dialysis patients on a low-protein diet. Two choices exist. High-calorie, high-protein, low-phosphorus supplements are one approach. Serum phosphorus is not affected in this diet, and greater phosphorus binders are not needed [31]. The Second, provide dietary education. The correct and early usage of phosphorus binders [11] is vital to adhering to a low phosphorus/protein ratio and using additions [12].

5. Binders for Phosphorus

Phosphorus-binding medicines increase the long-term survival rate of dialysis patients with high phosphate levels (3.7 mg/dL or higher) [32]. The first 90 days of dialysis and subsequent binders had the same results. According to the study's authors, FGF23 or a compensatory mechanism may explain these results [33]. Mortality did not reduce in incident dialysis patients treated with calcium-containing binders, calcium acetate, or calcium carbonate [34]. Phosphate binders decrease serum phosphorus and FGF23. Binders may lower FGF23 levels in early CKD despite serum phosphorus alterations (Figure 2).

Standard phosphate binders reduce phosphorous absorption or preserve normal phosphatemia. Typically, they bind and excrete phosphate. Dialysis patients have a high salivary phosphorous ratio [36, 37]. Chewing gum and salivary phosphate binders reduce serum phosphate levels [38].

6. Different Predators' Efficacy and Tolerance: Acid Binding

Clinical phosphorus-binding drugs vary in potency and side effects. Binding capacity and side effects may alter efficacy [39]. 15% to 20% of people experience intestinal tolerance concerns after therapy [40, 41]. In cases of intolerance, prescription binders may be changed to reduce phosphorus absorption. Lanthanum carbonate has helped patients with binders discrimination [42]. Uremic patients have particular digestive difficulties that necessitate investigation into their inefficiency and intolerance. CKD patients may develop intestinal dysbacteriosis for several reasons [43]. 2. Dysbacteriosis promotes the discharge of bacterial waste products, such as phenols and indoles, into the blood.

Table-2: There are several reasons why CKD patients suffer from intestinal dysbacteriosis.

Those on dialysis consume less fiber than the general population because of dietary restrictions that limit the amount of fruit and vegetables they eat.
Intestinal acidity occurs as a consequence of Uremia.
Bacteria in the intestines may be altered by medications such as antibiotics and Phosphate-binding agents.
Constipation or an increased transit time in the intestines may be caused by bowel dysfunction.
Nutritional deficiency is possible due to the altered metabolism and absorption of proteins.

Pollution may increase heart and bone disease risks. In uremic patients, protein, carbohydrates, and fats may be malabsorbed. Possible causes include bacterial overgrowth or pancreatic or biliary dysfunction. Uremia is associated with high plasma levels of secretin, pancreatic secretagogues, and gastrin, and abnormal pancreatic secretion, including low bicarbonate and amylase levels.

Phosphate binders bind bile salts. Bacterial overgrowth was connected to dyspepsia in 10 of 49 dialysis patients. Sevelamer aggravated dyspepsia, while pancreatic enzyme supplementation improved symptoms and phosphate binder efficacy. Intestinal dysbiosis, phosphate binder efficacy, and patient tolerance impact patient outcomes. Phosphorus binders that bind to bile salts may interfere with soluble chemical absorption. When attached to bile salts, Sevelamer reduces cholesterol absorption, LDL-cholesterol, and vitamin D absorption.

7. CONCLUSIONS

Hypophosphatemia causes vascular calcification, cardiovascular mortality, left ventricular hypertrophy, and chronic renal failure. A regular protein diet may exacerbate Uremia and hypophosphatemia. However, low protein intake and a higher mortality rate in dialysis patients suggest more strategies are needed to decrease phosphorus absorption. Preservatives and additives, protein sources' phosphorus concentration, and diets with a low phosphorus/protein ratio are vital for nutrition.

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