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Association of Inorganic Phosphates Intake with (Urinary P/UUN) Ratio in CKD Patients

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Abstract

Chronic Kidney Disease (CKD) is a major public health concern and managing the dietary intake of phosphorus is a key aspect of treatment. As Chronic Kidney Disease (CKD) progresses, the kidneys ability to filter and excrete waste products, including phosphate, decreases. This leads to a buildup of phosphate in the blood, known as hyperphosphatemia. However, In CKD patients, a diet high in inorganic phosphates, which are commonly added to processed foods and are highly bioavailable, can contribute to hyperphosphatemia in CKD patients associated with increased morbidity and mortality. The burden of CKD on public health is substantial as it reduces the quality of life for those affected and places a significant financial burden on healthcare systems. Assessing dietary phosphorus intake and monitoring the P/UUN ratio in addition to serum phosphate levels is crucial in fully assessing phosphorus balance in CKD patients. The P/UUN ratio has the potential to be used as a predictor of hyperphosphatemia and a target for intervention in CKD patients, the literature review aims to determine the relationship between the ratio of P/UUN in urine with a diet, high in inorganic phosphates in CKD patients to gather and analyze existing research on the topic to determine the current understanding of the relationship between the two. Dietitians can use this information to guide therapy and treatment decisions, such as adjusting dietary restrictions to improve a patient's nutritional status and slow down the progression of CKD.

Keywords: Phosphorus; Inorganic phosphate; PTH inhibitors; Serum phosphate; Hyperphosphatemia

Introduction

Chronic Kidney Disease (CKD) is a progressive loss of kidney function over time. It is a condition that can lead to kidney failure, also known as End-stage Renal Disease (ESRD) requiring dialysis or transplantation. Chronic Kidney Disease (CKD) is defined by abnormalities in kidney function and structure that persist over >3 months and is classified according to cause, GFR and albuminuria. The prevalence of CKD in stages 1 to 5 was 13.4% and 10.6% in stages 3 to 5, according to a recent systematic review and meta-analysis. Diabetes and high blood pressure are the more common causes of CKD in adults, other risk factors include heart disease, obesity, a family history of CKD, inherited kidney disorders, past damage to the kidneys and older age. According to the global burden of disease study, 1.2 million people died from CKD [1].

Literature Review

Role of endocrine factors in the management of phosphate homeostasis and risk factors in Chronic Kidney Disease (CKD)

Endocrine factors, including, vitamin D and Parathyroid Hormone (PTH), as well as newer factors such as Fibroblast Growth Factor (FGF)-23 and its coreceptor α-klotho, are closely involved in the control of Pi homeostasis. Therefore, vitamin D deficiency and elevated PTH can impact the absorption of phosphorus in the gut, contributing to hyperphosphatemia and other complications [2]. Elevated levels of FGF23 have been associated with an increased risk of Cardiovascular Disease (CVD) in patients with CKD. Additionally, both FGF23 and PTH are also associated with increased cardiovascular risk independent of their roles in mineral metabolism. Therefore, controlling phosphate retention and maintaining normal levels of FGF23 and PTH levels are important for reducing the risk of complications in patients with CKD [3].

Many studies have shown that hyperphosphatemia is highly associated with increased cardiovascular disease risk and mortality in CKD patients and is a serious condition that can lead to severe complications such as cardiovascular calcification, metabolic bone disease and secondary hyperparathyroidism. To manage hyperphosphatemia effectively in CKD patients, it is important to consider both pharmacological and non-pharmacological treatment options, as well as understand the underlying physiologic pathways involved in phosphorus homeostasis. Hence controlling phosphate load remains the primary goal in the treatment of CKD [4].

Background and rationale for studying the association between P/UUN ratio and inorganic phosphates intake in CKD patients

Reducing the intake of inorganic Phosphate (P) rather than limiting the intake of P contained in proteins is desirable because it helps to lower serum phosphate levels and reduce the risk of kidney disease, cardiovascular disease and bone mineralization disorders, while still allowing for adequate protein intake for overall health. Additionally, it's easier to reduce inorganic P intake than to limit P intake from proteins in the diet [5]. Urinary excretion of P should reflect intestinal absorption of P (inorganic plus protein-derived) and the total amount of P ingested did correlate with the P/UUN ratio. Therefore, the P/UUN ratio can be used to estimate the total P intake and monitor the balance between phosphorus and protein intake and excretion [6].

The background and rationale for studying the association between the P/UUN ratio and inorganic phosphates intake in CKD patients are that in CKD, the kidneys may not function effectively to excrete excess phosphate, leading to hyperphosphatemia, which can exacerbate kidney disease and in addition, high levels of inorganic phosphate intake can exacerbate this problem [7].

Understanding the relationship between inorganic phosphate intake and P/UUN ratio in CKD patients can help to guide dietary recommendations to prevent the progression of kidney disease and reduce the risk of cardiovascular disease. The P/UUN ratio is often used to evaluate patients with Chronic Kidney Disease (CKD) as it reflects the balance between dietary phosphate and protein intake and their excretion by the kidneys [8].

The association of urinary P/UUN ratio with hyperphosphatemia in Chronic Kidney Disease (CKD) patients is a key concept highlighted in recent literature reviews. The P/UUN ratio, which is the ratio of urinary phosphorus to urinary urea nitrogen, is used as a marker of phosphorus balance in the body. A high P/UUN ratio indicates an imbalance between phosphorus intake and excretion, which can lead to hyperphosphatemia. Another key concept is that assessing dietary phosphorus intake and monitoring the P/UUN ratio in addition to serum phosphate levels is crucial in fully assessing phosphorus balance in CKD patients. The P/UUN ratio has the potential to be used as a predictor of hyperphosphatemia and a target for intervention in CKD patients [9].

The impact of bioavailability and absorption on hyperphosphatemia risk

Patients with CKD have a decreased ability to excrete phosphorous, there is an increased risk of hyperphosphatemia and increased intestinal absorption of phosphate can exacerbate this problem by adding more phosphorus to the blood. Therefore, controlling phosphorus intake can help to mitigate the risk of hyperphosphatemia. Given the increased understanding of the bioavailability of different phosphorus sources, it is important to limit the consumption of easily absorbable inorganic phosphorus.

Phosphorus homeostasis is maintained through a complex interplay of factors, including absorption and secretion in the gastrointestinal tract, filtration and reabsorption in the kidneys, and shifts between extracellular and bone storage pools. In the gastrointestinal tract, the naturally existing organic phosphorus is only partially (~60%) absorbable; however, this absorption varies widely and is lower for plant-based phosphorus including phytate (<40%) and higher for foods enhanced with inorganic-phosphorus-containing preservatives (>80%) [10,11].

Another challenge in managing phosphorus levels is accurately estimating intestinal absorption. The net absorption is very variable, between 40% and 80%. This variability in absorption makes it challenging to predict the net absorption of phosphorus from the diet and makes it difficult to maintain appropriate phosphorus levels in patients with CKD and those on hemodialysis. Patients with Advanced CKD (ACKD) or on hemodialysis typically have a phosphorus intake of 800 mg/day-1600 mg/day, but the amount absorbed can vary widely (320 mg-1280 mg), which can make it difficult to maintain appropriate levels. This variability in absorption makes it difficult to accurately estimate the P balance and can lead to difficulties in managing phosphorus levels in patients with kidney disease and those on hemodialysis.

A combination of dietary restrictions, phosphorus binders and dialysis may be used to manage phosphorus levels, along with regular monitoring of blood phosphorus levels to adjust treatment as needed [12].

According to Ban-Hock Khor, et al., a cross-sectional study in Malaysia was conducted on 435 MHD patients based on 27 food groups shortlisted from 3-day dietary recalls. The patients dietary phosphate intakes were estimated using food composition databases. Three dietary patterns emerged: Home Foods (HFdp), Sugar Sweetened Beverages (SSBdp) and Eating Out Noodles (EO-Ndp). The SSBdp was associated with greater consumption of inorganic phosphate and higher serum phosphorus levels, possibly due to greater intakes of inorganic phosphate found in beverages with added condensed milk. However, the lack of an inorganic phosphate database and identification of processed food labels made it difficult to accurately distinguish between the two types of phosphate, so an assumption model was used. The inclusion of parathyroid hormone to examine phosphate resorption from bone and gut availability from dietary phosphate origin would have been ideal, but cost prevented its measurement in this population. Information on ultrafiltration volume was also not available [13].

According to Britta Dobenecker et al., a study was conducted on dogs to test the effects of increased mid-term phosphorus intake from different sources on postprandial serum levels of calcium, phosphorus and PTH. Eight healthy young beagles were fed a balanced diet and different sources of phosphorus were added to the diet. The study found that inorganic phosphates, but not organic phosphorus sources, caused significant changes in these regulatory factors compared to the control diet. The authors conclude that the use of inorganic phosphates in pet food is potentially harmful and should be restricted. However, the study had limitations, including a small sample size and a

short duration, which may not fully capture the long-term effects of consuming high levels of phosphates in pet food.

Discussion

High intake of inorganic phosphate can lead to an imbalance in the ratio of urinary Phosphorus (P) to Urinary Urea Nitrogen (UUN) in individuals with Chronic Kidney Disease (CKD). This can increase the risk of both cardiovascular disease and bone disease. In addition, high phosphorus levels can disrupt the balance of minerals in the bones, leading to bone weakness and an increased risk of fractures. The ratio of phosphorus to urea nitrogen (P/UUN) can be elevated in Chronic Kidney Disease (CKD) patients for several reasons. One of the main reasons is that as kidney function declines, the kidneys are not able to effectively remove excess phosphorus from the blood. This can lead to a build-up of phosphorus in the body, which can increase the P/UUN ratio [14].

Another reason for an elevated P/UUN ratio in CKD patients is that they may be consuming a diet that is high in inorganic phosphorus. However, as kidney function declines in CKD patients, the kidneys are not able to effectively remove excess phosphorus from the blood. In addition, modern food processing has led to an increase in the amount of dietary phosphate intake. This is because phosphates are commonly added to processed foods as preservatives, emulsifiers and leavening agents. As a result, many processed foods, such as processed meats, baked goods and processed cheeses, have high levels of phosphates. Additionally, fast food and restaurant meals also tend to have high levels of phosphates. The absorption rate of inorganic phosphates is 100% which may affect the ratio of P/UUN. High levels of dietary phosphate intake can have negative effects on kidney function, particularly in individuals with pre-existing renal disease. This is because the kidneys have to work harder to filter out the excess phosphate, which can lead to damage and dysfunction over time.

Avoiding foods high in phosphorus relative to protein and those with high amounts of phosphorus-based preservatives such as certain soft drinks and enhanced cheese and meat is an effective and patient-friendly approach to reduce phosphorus intake without depriving patients of adequate proteins, preparing protein-rich foods by boiling or other types of cooking induced demineralization and adjusting the dose of phosphorus-binding therapy separately for the amount and absorbability of phosphorus in each meal, with the help of dietician counseling is instrumental in achieving a reduction of phosphorus load [15]. Additionally, the use of certain medications, such as aluminum-containing antacids, can contribute to elevated P/UUN ratio in CKD patients by binding to phosphorus in the gut and preventing its absorption. Overall, an elevated P/UUN ratio in CKD patients can be caused by a combination of factors, including poor kidney function, a high phosphorus diet and certain medications.

The ratio of Phosphorus to Urea Nitrogen (P/UUN) is used as a marker for assessing protein intake and excretion in Chronic Kidney Disease (CKD) patients because it provides important information about the balance between phosphorus and nitrogen in the body. Urea nitrogen, on the other hand, is a waste product that is produced by the breakdown of protein in the body. It is normally excreted by the kidneys, but as kidney function declines in CKD patients, the kidneys may not be able to effectively remove urea nitrogen from the blood. The review found that controlling phosphorus intake and monitoring the P/UUN ratio can be effective in managing hyperphosphatemia. Patients with CKD have a decreased ability to excrete phosphorous and controlling the intake of phosphorous can help reduce the risk of hyperphosphatemia. The P/UUN ratio is a useful marker for monitoring the balance between phosphorous intake and excretion in CKD patients.

It's important to note that the P/UUN ratio is not a definitive marker of inorganic phosphate intake and other factors such as renal function, medical conditions and medications can affect the ratio. Additionally, in CKD patients, the P/UUN ratio can be affected by factors such as muscle wasting and protein catabolism, which can lead to an increase in urea nitrogen production, making the P/UUN ratio less accurate. The P/UUN ratio provides important information about the balance between phosphorus and nitrogen in the body. A high ratio can indicate an imbalance between the two and may be an indicator of poor kidney function. By monitoring the P/UUN ratio, healthcare providers can better understand a patient's protein intake and excretion and make appropriate adjustments to their diet and treatment plan.

Research has shown that a high intake of inorganic phosphates is associated with an increase in the P/UUN ratio in CKD patients and this has been linked to an increased risk of cardiovascular disease, bone disorders and mortality. However, the current understanding of the relationship between high inorganic phosphate intake and the P/UUN ratio in CKD patients is not fully understood. Many studies have been conducted on small samples of CKD patients and most have relied on self-reported measures of inorganic phosphates intake such as food frequency questionnaires, which can be subject to recall bias.

The foregoing considerations strongly suggest that in patients with CKD, a mixed composition of dietary animal and plant foods rich in phytic acid should be encouraged, while the intake of processed foods should be restricted. Dietary prescriptions for patients with CKD should take into consideration both the absolute dietary phosphorus content and the phosphorus-protein ratio of foods and meals. Because a diet that is high in phosphorus but low in protein can be particularly harmful to patients with CKD, as it can lead to an imbalance in the body's phosphorus levels, which can contribute to the progression of the disease. Hence the importance of dietary management in patients with CKD and the need to consider both the phosphorus content with restriction of Inorganic phosphates and considering the phosphorus-protein ratio of foods could help to slow the progression of the disease.

Based on the current understanding of the relationship between the ratio of P/UUN in urine and a high intake of inorganic phosphates in CKD patients, some recommendations for future research include long-term studies that can track the P/UUN ratio and inorganic phosphate intake over a prolonged period and conducting longitudinal studies to investigate the relationship between the Ph/P ratio in the blood, inorganic

phosphates intake and the P/UUN ratio in urine over time in CKD patient.

Conclusion

It's important to note that muscle wasting and protein catabolism can have a complex impact on the P/UUN ratio. Therefore, healthcare providers need to consider these factors when interpreting the P/UUN ratio in CKD patients and make adjustments to the diet and treatment plan accordingly. The P/UUN ratio appears to reflect the intake of inorganic P. A high P/UUN ratio is typically indicative of a high intake of inorganic phosphates and a low excretion of urinary phosphate, which can occur when there is an overconsumption of a diet with high inorganic phosphates.

The association of inorganic phosphate intake with the urinary P/UUN ratio in CKD patients is an area of ongoing research. However, based on current studies, it is clear that in CKD patients, high levels of inorganic phosphate intake can lead to hyperphosphatemia and the P/UUN ratio is an indicator of the balance between dietary phosphorus and protein intake and their excretion by the kidneys. The P/UUN ratio can evaluate patients with CKD as it reflects the balance between dietary phosphate and protein intake and their excretion by the kidneys. It is important to note that this association is not conclusive and more studies are needed to understand the relationship between inorganic phosphates intake and the P/UUN ratio in CKD patients.

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