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Effect of dietary protein source on calcium phosphorus product in patients with chronic kidney disease

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ABSTRACT

Introduction: Cardiovascular disease is highly prevalent in chronic kidney disease (CKD) and progresses rapidly. The calcium–phosphorus product (Ca×P) is a clinically relevant mineral metabolism parameter linked to cardiovascular risk in CKD. This study compared Ca×P and related clinical features according to predominant dietary protein source.

Methods: In this cross-sectional observational study, participants with CKD (n = 80) were classified into two groups using 3-day food records: a plant-protein group (VP; n = 40; plant protein >50% of total protein intake) and an animal-protein group (AP; n = 40; animal protein >50% of total protein intake).

Results: Anthropometric measurements did not differ between groups. Phosphate-binder use and pruritus were more frequent in the AP group. Estimated glomerular filtration rate (eGFR) was higher in the VP group, whereas serum calcium, phosphorus, potassium, and Ca×P were significantly higher in the AP group. Mean Ca×P was 47.87 ± 7.65 in the AP group and 33.75 ± 5.01 in the VP group (p = 0.001). Plant protein intake was strongly inversely correlated with serum phosphorus (r = −0.628, p = 0.014). No significant between-group differences were observed for iron, albumin, triglycerides, HDL, LDL, sodium, ferritin, C-reactive protein, or uric acid (p > 0.05).

Conclusion: Predominant plant protein intake was associated with a lower Ca×P product and more favorable phosphorus-related parameters, without higher serum potassium, and with fewer phosphate-binder prescriptions and less pruritus. Larger, well-designed randomized controlled trials—particularly in predialysis CKD—are warranted to confirm these associations and clarify causality.

Keywords: Cardiovascular risk, Chronic kidney disease, Dietary animal proteins, Dietary plant proteins

Introduction

Chronic kidney disease (CKD), affecting approximately 10% of adults worldwide, is a progressive condition characterized by structural and/or functional kidney abnormalities due to various causes (1). CKD is diagnosed when there is evidence of chronic kidney damage or a persistent reduction in renal function, typically defined as an estimated glomerular filtration rate (GFR) <60 mL/min/1.73 m² for more than 3 months. As a major global public health problem, CKD is associated with substantial morbidity, mortality, and healthcare costs (2). The most common aetiological causes of CKD in the community include diabetes mellitus (DM), hypertension (HT), glomerulonephritis, cystic kidney diseases, and other conditions (3). Because the disease may remain clinically silent until advanced stages, many patients progress to end-stage renal disease (ESRD), necessitating dialysis and/or kidney transplantation (4).

General management strategies for CKD include pharmacological treatment, renal replacement therapy, and medical nutrition therapy (5). Medical nutrition therapy is essential in CKD, as careful monitoring and regulation of protein, calcium (Ca), phosphorus (P), potassium (K), and sodium (Na) intake may contribute to slowing disease progression and optimizing clinical outcomes.[5] Nutritional therapy also plays a key role in alleviating CKD-related symptoms, reducing the risk of malnutrition, and supporting overall health status (6).

Emerging evidence suggests that the source of dietary protein may influence CKD-related outcomes (7-9). In a recent cohort study based on food frequency data, higher intakes of red and processed meat were associated with an increased risk of CKD, whereas dietary patterns rich in nuts, legumes, and low-fat dairy products were associated with a lower risk and appeared protective (10). Moreover, a low-protein diet (0.6-0.8 g/kg/day) in which at least 50% of protein is derived from plant sources has been proposed to improve CKD progression, potentially by reducing glomerular hyperfiltration (11). However, the extent to which dietary protein source relates to mineral metabolism and cardiovascular risk markers in CKD remains insufficiently characterized.

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Disorders of bone and mineral metabolism are common in CKD, and hyperphosphatemia is a frequent and clinically important problem, particularly due to reduced phosphate excretion with declining renal function (12). In parallel, disturbances in calcium homeostasis contribute to soft-tissue and vascular calcification, increasing the risk of cardiovascular events and related morbidity and mortality (12,13). Vascular calcification increases arterial stiffness, elevates pulse pressure, and may contribute to myocardial ischaemia, left ventricular hypertrophy, and heart failure (12,13). Notably, elevated calcium–phosphorus product (Ca×P) has been reported to correlate more strongly with cardiovascular events than elevated phosphorus or calcium alone (14). Therefore, the present study aimed to compare Ca×P according to dietary protein source in patients with CKD.

Materials and Methods

This cross-sectional observational study included 80 patients with CKD stages 3-4 who attended the XXX University Nephrology Outpatient Clinic between October 2022 and January 2023 and agreed to participate. The sample comprised 45 females (56%) and 35 males (44%). CKD stage was defined as an eGFR of 15-59 mL/min/1.73 m². Written informed consent was obtained from all participants prior to data collection. Ethics approval was granted by the XXX University Clinical Research Ethics Committee (07/12/2022; No: 22/208). The required sample size was calculated using G*Power (version 3.2) with an alpha of 0.05 and power of 0.95, indicating that at least 74 participants were needed. The inclusion and exclusion criteria for the study are as follows.

Inclusion criteria:

- Age 30–65 years
- Diagnosed CKD stages 3–4 (eGFR 15–59 mL/min/1.73 m²)
- Stable clinical status (no hospitalization for acute illness within the previous 4 weeks)
- Ability to communicate in Turkish or English
- Provided written informed consent

Exclusion criteria:

- Pregnancy or lactation
- End-stage kidney disease (eGFR <15 mL/min/1.73 m²), dialysis, or prior kidney transplantation
- Acute kidney injury or rapidly progressive renal deterioration
- Active malignancy or ongoing chemotherapy/radiotherapy
- Severe liver disease or other severe systemic disease likely to substantially affect diet/metabolism
- Autoimmune/inflammatory systemic diseases
- Current diagnosis of a severe psychiatric disorder or cognitive impairment
- Inability or unwillingness to complete dietary assessment procedures

A structured questionnaire was administered via face-to-face interview to collect demographic information, health status, and the presence of uremic symptoms. Anthropometric measurements included height, body weight, mid-upper arm circumference (MUAC), waist circumference, and triceps skinfold thickness (TSFT). Body mass index (BMI) was calculated and categorized according to the World Health Organization classification (15).

Dietary intake was assessed using 3-day food records (two weekdays and one weekend day). All food records were reviewed by a registered dietitian using a standardized procedure. Records were checked for completeness (meal occasions, ingredients, cooking methods, portion sizes), and participants were contacted as needed for clarification using probing questions and household measures/portion-size aids. Participants were instructed to record intake on typical days; days considered atypical (e.g., acute illness, fasting, celebrations/travel, or unusually high/low intake) were flagged during the review. Energy and nutrient intake were analyzed using the Computer Assisted Nutrition Program (BEBIS), full version 6.1 (16).

Plant-protein percentage was calculated from the 3-day food records as: (plant protein [g]/total protein [g]) × 100. We initially explored categorizing participants using the 1st and 3rd quartiles of the plant protein intake proportion. However, because prior studies commonly operationalize exposure using plant-to-total protein ratios and often compare “higher vs. lower” intake using pragmatic cut-offs, we defined our primary grouping based on the sample median (below vs. above the median) to ensure balanced group sizes and stable estimates. In addition, we performed a secondary analysis comparing participants with a plant protein proportion >50% versus those with ≤50%, as this threshold offers a clear and clinically interpretable classification of predominantly plant- versus animal-based protein intake and has been used in previous studies with ratio-based definitions (17-19).

Dietary adequacy and nutrient intake were evaluated in accordance with Kidney Disease: Improving Global Outcomes (KDIGO) clinical practice guidelines (20). Consistent with Chen et al. (21), plant protein sources included cereals, vegetables, legumes (e.g., peas, dried beans, chickpeas, lentils), and nuts/oilseeds (e.g., pumpkin seeds, almonds, walnuts, hazelnuts). Although soy protein is classified as a plant protein, it was not included because it is not commonly consumed in our population or among our participants. Animal protein sources included red meat, processed meat products (e.g., salami, sausage), eggs, milk, and dairy products.

Routine biochemical parameters recorded from hospital records included serum glucose, urea, creatinine, uric acid, albumin, triglycerides, HDL-cholesterol, LDL-cholesterol, sodium, potassium, calcium, iron, phosphorus, and C-reactive protein (CRP). eGFR was also recorded. All laboratory values were interpreted according to the hospital’s reference ranges.

Statistical analyses were conducted using SPSS Statistics (version 25; licensed). Assumptions for parametric testing were evaluated prior to hypothesis testing. Normality of continuous variables was assessed using the Shapiro–Wilk test and visual inspection of histograms and Q–Q plots, and

homogeneity of variances was evaluated using Levene's test. Between-group comparisons were performed using the independent-samples t-test for continuous variables and the chi-square test for categorical variables. Given the number of statistical comparisons performed, p-values should be interpreted as nominal. No formal adjustment for multiple testing was applied, and the analyses should be considered exploratory. Pearson correlation analysis was used to examine associations between continuous variables. A two-sided p-value of <0.05 was considered statistically significant.

Results

The distribution and averages of sociodemographic and anthropometric variables are presented in Table 1. No difference was found between the Animal Protein (AP) and Plant Protein (VP) groups in terms of these variables (p > 0.05).

It was found that 40% of the AP group and 15% of the VP group used phosphorus binders (p = 0.04). The frequency of itching symptoms was statistically higher in the AP group than in the VP group (p = 0.026). There was no statistically significant difference between the two groups in other uremic symptoms (nausea, fatigue, anorexia, cramps, edema, hiccups) (p = 0.65) (data not shown).

Fasting glucose, creatinine, urea, potassium, calcium, phosphorus, and calcium x phosphorus product were lower while GFR was higher in the VP group (p = 0.037, p = 0.005, p = 0.005, p = 0.012, p = 0.001, p = 0.001, p = 0.001, respectively) (Table 2).

TABLE 1 - Distribution of sociodemographic characteristics and anthropometrics

		Animal Protein (n = 40)		Vegetable Protein (n = 40)		
		n	**%**	**n**	**%**	**p**
Gender	**Male**	21	52.50	14	35.00	0.115Ω
	Female	19	47.50	26	65.00	
Education	**Middle**	12	30.00	18	45.00	0.359 Ω
	High	20	50.00	13	32.50	
	Undergraduate	8	20.00	9	22.50	
		X	**SD**	**X**	**SD**	
Age (years)		57.20	11.25	55.65	12.62	0.564
BMI (kg/m²)		28.54	4.79	28.46	4.10	0.937
WC (cm)		105.53	12.68	104.43	12.29	0.695
UMAC (cm)		32.45	3.26	32.30	4.03	0.927
TSFT (mm)		15.48	3.57	15.10	3.45	0.634

*p < 0.05, independent sample t-test, Ω chi-square

TABLE 2 - Means of biochemical parameters according to the groups

		X	**SD**	**t**	**P**
Fasting glucose (mg/dL)	AP	117.85	26.06	2.12	**0.037***
	VP	106.85	19.95		
Iron (mg/dL)	AP	92.78	15.88	1.451	0,151
	VP	86.98	19.68		
Creatinine (mg/dL)	AP	2.08	0.92	2.955	**0.005***
	VP	1.60	0.44		

		X	SD	t	P
GFR (mL/min/1.73 m^2)	AP	32.85	11.24	-3.482	**0.001***
	VP	41.02	9.69		
Urea (mg/dL)	AP	54.13	31.68	2.946	**0.005***
	VP	37.32	17.30		
Albumin (mg/dL)	AP	4.03	0.28	0.17	0.866
	VP	4.02	0.32		
Triglyceride (mg/dL)	AP	171.48	53.05	1.234	0.221
	VP	156.83	53.13		
HDL-C (mg/dL)	AP	46.78	9.55	-1.786	0.078
	VP	50.88	10.94		
LDL-C (mg/dL)	AP	125.80	27.74	1.544	0.127
	VP	115.45	32.05		
Sodium (mmol/L)	AP	137.90	9.47	1.019	0.311
	VP	138.55	9.32		
Potassium (mmol/L)	AP	4.84	0.54	2.576	**0.012***
	VP	4.53	0.53		
Calcium (mg/dL)	AP	9.78	0.40	3.352	**0.001***
	VP	9.47	0.44		
Ferritin (mg/L)	AP	199.38	160.17	0.63	0.530
	VP	178.78	130.57		
Phosphorus (mg/dL)	AP	4.94	0.63	10.59	**0.001***
	VP	3.59	0.50		
PTH (pg/mL)	AP	77.45	38.52	3.56	**0.015***
	VP	65.64	29.98		
25-OH D (ng/mL)	AP	12.23	8.89	1.15	0.240
	VP	16.64	7.47		
FGF23 (RU/mL)	AP	340.20	215.34	2.589	**0.010***
	VP	304.82	155.78		
CRP (mg/dL)	AP	3.80	4.81	1.765	0.083
	VP	2.33	2.10		
Uric Acid (mg/dL)	AP	5.75	1.17	-0.742	0.460
	VP	6.01	1.87		
CalciumxPhosphorus	AP	47.87	7.65	8.17	**0.001***
	VP	33.75	5.01		

*p < 0.05. independent sample t-test

There were negative strong relationships between consumed VP percentage with phosphorus and calcium (p = 0.014 and p = 0.021). CaxP product was significantly lower in the VP group (p = 0.001) (Table 3). While energy and macronutrient intakes of both groups were similar, fiber and potassium intakes were higher in the VP group and cholesterol intakes were higher in the AP group (p = 0.001 for all) (Table 4).

TABLE 3 - Correlation between plant protein consumption and calcium phosphorus product

		Phosphorus	Calcium	CaxP
VP (%)	r	**–.628****	**–.258***	**–.804****
	p	0.014	0.021	0.001

**p < 0.05. Pearson correlation

TABLE 4 - Comparison of the means of energy and nutrient intakes by groups

		X	SD	t	p
Energy (kcal/d)	AP	1762.75	310.29	-0.578	0.565
	VP	1803.75	324.18		
Carbohydrate %TE	AP	58.30	2.43	0.222	0.825
	VP	58.18	2.60		
Protein %TE	AP	14.10	1.41	-0.672	0.503
	VP	14.30	1.24		
Fat %TE	AP	27.63	3.14	0.149	0.882
	VP	27.53	2.86		
Fiber (g/d)	AP	13.18	2.41	-10.47	**0.001***
	VP	19.31	2.81		
Protein (g/kg/d)	AP	0.80	0.12	-1.381	0.172
	VP	0.85	0.17		
Cholesterol (mg)	AP	220.90	18.06	6.795	**0.001***
	VP	198.00	11.32		
Potassium (mg)	AP	1574.40	188.78	-7.986	**0.001***
	VP	1887.08	160.27		
Phosphorus (mg)	AP	920.95	112.53	0.905	0.368
	VP	783.78	151.87		
Calcium (mg)	AP	739.43	64.08	0.728	0.469
	VP	728.85	65.88		

*p < 0.05. independent sample t-test

Discussion

Current guidelines and recent evidence underscore the need to manage multiple nutrients to slow CKD progression and reduce related complications; nevertheless, there is still no clear consensus on the optimal dietary protein source in predialysis CKD. Against this backdrop, the present study examined whether predominant dietary protein source (largely animal vs. largely plant) is associated with the calcium–phosphorus product (Ca×P), anthropometric indices, and selected uremic symptoms among patients with stage 3-4 CKD (22,23).

A key finding was that Ca×P was significantly higher in the predominantly animal-protein group than in the predominantly plant-protein group. This difference is clinically meaningful because elevated Ca×P has been linked to vascular and soft-tissue calcification and to higher cardiovascular morbidity and mortality in CKD. Biologically, higher Ca×P may facilitate calcium–phosphate deposition, thereby contributing to arterial stiffness, myocardial injury, and coronary pathology. In addition, observational evidence suggests that higher Ca×P may be associated with adverse kidney-related outcomes, including an increased risk of acute kidney injury at higher Ca×P levels. Accordingly, the observed divergence in Ca×P between dietary patterns may reflect not only a biochemical distinction but also a broader difference in cardiovascular and mineral-bone risk profiles in predialysis CKD (24).

The Ca×P differences between groups are biologically plausible. Phosphorus is abundant in protein-rich foods, and intake of animal protein is strongly correlated with dietary phosphate exposure (25). While some plant foods (e.g., nuts, seeds, and legumes) may contain substantial phosphorus, a considerable proportion of plant-derived phosphorus is phytate-bound. Because humans lack endogenous phytase, phytate-bound phosphorus is less efficiently hydrolyzed and absorbed, resulting in lower intestinal bioavailability relative to many animal sources. Moreover, phosphate-containing additives—common in processed foods, including some animal-based products—are highly absorbable and can markedly increase phosphorus burden (26,27). Together, these mechanisms provide a coherent explanation for why predominantly animal-based protein patterns may confer a higher absorbable phosphorus load and, consequently, higher Ca×P. Although total dietary phosphorus intake did not significantly differ between groups in our 3-day food records, serum phosphorus levels were higher in the predominantly animal-protein group. This apparent discrepancy is biologically plausible because total phosphorus intake does not necessarily reflect the absorbable phosphorus load. Phosphorus from plant foods is often stored as phytate, which is less efficiently hydrolyzed and absorbed in humans due to limited endogenous phytase activity, whereas phosphorus from many animal sources is generally more readily absorbed. In addition, inorganic phosphate from food additives—frequently present in processed meats, convenience foods, and cola-type beverages—has very high intestinal bioavailability and can disproportionately increase phosphorus burden even when estimated total phosphorus intake appears similar. Because our dietary assessment captured total phosphorus but did not quantify phosphate additives or directly measure phosphorus absorption/excretion (e.g., urinary phosphate), our nutrient estimates may have underestimated the true absorbable phosphorus exposure, particularly in participants consuming more processed foods (28,29). Therefore, the observed between-group differences in serum phosphorus likely reflect differences in phosphorus bioavailability and additive-derived inorganic phosphate, underscoring the importance of distinguishing between total phosphorus intake and absorbable phosphorus load when interpreting mineral metabolism outcomes in CKD (30).

Our findings align with prior work indicating that plant-forward dietary patterns may facilitate phosphorus control in predialysis CKD. Interventional and observational studies have reported lower serum phosphorus and reduced urinary phosphorus excretion with vegetarian or soy-based dietary patterns compared with animal-protein-based diets, suggesting that shifting protein intake toward plant sources may represent a pragmatic strategy to reduce bioavailable phosphorus exposure (29,30). Mechanistic evidence further supports the relevance of protein source: experimental data indicate that protein matrices with higher phosphorus bioavailability may influence phosphate handling and mineral metabolism signaling even when serum phosphorus appears similar. In real-world settings, plant-based patterns have also been associated with lower dietary protein and phosphorus intakes in dialysis populations, reinforcing the linkage between protein source and phosphorus load across CKD stages. In addition, food preparation practices may meaningfully alter mineral content; for example, boiling vegetables and legumes can reduce their phosphorus content, potentially amplifying the phosphorus-related advantages of plant-forward patterns (31).

Several biologically plausible confounders should be considered when interpreting the association between protein source and Ca×P (32,33). Phosphorus exposure is determined not only by total phosphorus content but also by bioavailability, which can differ substantially across plant- and animal-based patterns and is further modified by the presence of phosphate additives and by food processing. Beyond phosphorus, predominantly animal-based dietary patterns may be accompanied by higher dietary acid load and lower fibre intake, both of which may influence metabolic and inflammatory pathways relevant to CKD. Finally, because Ca×P is embedded within the broader context of CKD–mineral and bone disorder and vascular calcification risk, unmeasured factors—such as phosphate additive intake, objective markers of phosphorus absorption and excretion, and medication use (including phosphate binders) may contribute to residual confounding. Because kidney function directly influences serum phosphorus and the Ca×P product, this between-group difference in eGFR may have contributed to the observed mineral-metabolism differences independent of dietary protein source, and residual confounding cannot be excluded (34,35). These considerations highlight the need for prospective and randomized studies incorporating objective phosphorus handling metrics and comprehensive dietary pattern indices to better clarify causality.

A clinically tangible signal in our cohort was the distribution of phosphate-binder use: among participants prescribed phosphate binders, the majority were in the predominantly animal-protein group. Although binder prescription is influenced by multiple clinical factors, this pattern is consistent with the premise that animal-protein–dominant diets may carry a higher absorbable phosphorus burden, thereby increasing the likelihood of requiring pharmacologic phosphate control. This observation strengthens the real-world relevance of the biochemical findings and suggests that dietary protein source may translate into meaningful differences in clinical management.

With respect to symptoms, pruritus was more frequent and/or severe in the predominantly animal-protein group. CKD-associated pruritus is common and can substantially impair quality of life (36). While its pathophysiology is multifactorial, disturbances in mineral metabolism—particularly elevations in calcium, phosphorus, and related parameters—have been associated with pruritus in large cohorts. One plausible mechanism is that higher calcium–phosphorus levels may promote cutaneous deposition and mast-cell activation, with downstream histamine release contributing to itch. In this context, our symptom findings are congruent with the higher Ca×P observed in the animal-protein group and underscore that protein source may have clinical implications beyond laboratory markers (37).

A common barrier to recommending plant-forward diets in CKD is concern regarding hyperkalaemia. Notably,

despite higher dietary potassium intake in the predominantly plant-protein group, serum potassium was higher in the predominantly animal-protein group. This apparent dissociation may reflect food selection and preparation practices (e.g., soaking and boiling), which can substantially reduce the potassium content of plant foods (38). In addition, differences in macronutrient composition may be relevant: higher-carbohydrate meals may stimulate insulin secretion and promote intracellular potassium uptake, potentially attenuating postprandial rises in serum potassium compared with lower-carbohydrate meals (39). Collectively, these findings suggest that a plant-forward pattern does not necessarily exacerbate serum potassium when appropriate food choices and preparation techniques are implemented—an important practical point for dietary counselling.

Anthropometric indices—including mid-upper arm circumference, triceps skinfold thickness, and waist circumference—did not differ significantly between groups, suggesting that a higher proportion of plant protein was not associated with anthropometric evidence of nutritional compromise in this cohort. This is clinically relevant because concerns about protein–energy wasting and insufficient protein quality are often raised when recommending plant-forward dietary patterns in CKD (40). Our findings support the feasibility of plant-forward protein distribution without apparent detriment to selected anthropometric indicators, at least within the dietary context observed.

Finally, participants in the predominantly animal-protein group exhibited a less favourable metabolic and kidney-function profile, characterized by higher plasma glucose, creatinine, urea, potassium, calcium, and phosphorus levels and lower eGFR. Prior evidence has associated higher animal protein intake with faster declines in kidney function, whereas higher plant protein intake has been linked to slower declines after adjustment for relevant covariates. Potential mechanisms include differences in dietary acid load and renal hemodynamic responses; animal-protein–rich diets tend to generate higher net endogenous acid production, potentially increasing ammoniagenesis and contributing to maladaptive hyperfiltration over time (41). Moreover, dietary fibre intake was higher in the predominantly plant-protein group, offering an additional pathway of benefit. Higher fibre intake has been associated with more favourable gut microbial metabolism, reductions in gut-derived uremic toxins, lower inflammation, and improved insulin sensitivity in CKD populations. This broader metabolic milieu may be consistent with the lower serum glucose observed in the plant-protein group (42-45).

From an overall perspective, the present findings suggest that, among patients with stage 3-4 predialysis CKD, a predominantly plant-based protein pattern may be associated with a more favourable Ca×P profile and potentially relevant differences in symptom burden and metabolic parameters, without evident anthropometric compromise. Nonetheless, given the potential for residual confounding—particularly related to phosphate additives, objective phosphorus handling, and medication use—future well-designed prospective and randomized studies are warranted to confirm these associations and delineate causal mechanisms.

Clinical implications

Overall, our findings suggest that a dietary pattern in which protein intake is predominantly plant-derived may facilitate phosphorus control, lower Ca×P, reduce reliance on phosphate binders, and potentially alleviate pruritus—while not necessarily increasing serum potassium when appropriate dietary practices are followed. These results support considering protein source, not only total protein amount, as a meaningful component of medical nutrition therapy in predialysis CKD.

Limitations

This study has several limitations. First, the cross-sectional design precludes causal inference. Second, the sample size was modest, which may limit generalisability. Third, dietary intake was assessed using self-reported 3-day food records, which are subject to reporting error despite dietitian review. Although PTH, 25(OH) vitamin D, and FGF23 data were available, we did not assess urinary phosphate excretion, phosphate additive intake or absorption, blood gas analysis, proteinuria, or bone turnover markers; therefore, mechanistic interpretation remains limited. Residual confounding—including medication use (e.g., phosphate binders) and other clinical factors—may also have influenced the observed associations. Because eGFR differed between groups and renal function is a major determinant of serum phosphorus and Ca×P, residual confounding is possible; thus, the observed associations should be interpreted cautiously. Future prospective studies, ideally randomized controlled trials with larger samples and more detailed measures of phosphorus bioavailability, are warranted to confirm these findings and clarify potential dose–response relationships. An additional limitation is the risk of type I error due to multiple statistical comparisons. Because numerous outcomes and subgroup comparisons were examined, the probability of observing statistically significant findings by chance may be increased. As we did not apply a formal correction for multiple testing (e.g., family-wise error or false discovery rate control), the reported p-values should be interpreted cautiously and the results viewed as hypothesis-generating. Future studies with larger samples and pre-specified primary outcomes should confirm these associations using appropriate multiplicity control.

Conclusion

Plant-based protein sources may help improve phosphorus control in patients with CKD without exacerbating hyperkalemia, potentially lowering cardiovascular risk by reducing the calcium–phosphorus product (Ca×P), alleviating hyperphosphatemia-associated pruritus, and possibly slowing disease progression. However, these potential benefits should be confirmed in well-designed prospective studies. Further large-scale randomized controlled trials—particularly in predialysis CKD populations—are warranted to clarify the clinical impact of higher plant-protein intake and its role in medical nutrition therapy.

Disclosures

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