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Original Research

Effect of dietary phosphorous load and risk factors associated with chronic kidney disease patients (stage 1 and 2)

Trisha Sachan[1], Anita Saxena[2], Amit Gupta[2]

[1]Research Scholar, Department of Nephrology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow (Uttar Pradesh), India-226014;
[2]Professor, Department of Nephrology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow (Uttar Pradesh), India-226014

ABSTRACT:
Objectives: Dietary phosphorus loading markedly affects fibroblast growth factor-23 (FGF-23), which is a key factor responsible for many of the cardiovascular disease (CVD) and death in early chronic kidney disease (CKD). This study aimed to examine the dietary phosphorus load in CKD patients according to their dietary phosphorous intake (DPhI) in CKD patients and to find the risk factors that might be associated with the progression of CKD (stage 1/2). **Design and setting:** This cross-sectional study was conducted in the nephrology and endocrinology departments of the institute. **Participants:** Seventy-nine CKD patients were included and based on their DPhI, all the patients were categorized into 2 groups: recommended phosphorous intake (RPhI) group (n=37) with <1000 mg/day and high phosphorous intake (HPhI) group (n=42) with >1000mg/day. **Results:** In HPhI group; low sKlotho and HDL levels and high serum phosphorous (SP), FGF-23 and cholesterol levels correlated significantly with high intake of dietary phosphorous. FGF-23, SP, dietary protein and total cholesterol levels were significantly higher and sKlotho and HDL levels were significantly lower in HPhI than RPhI group. Risk factors with a statistical bearing on the progression of CKD were animal-based diet, family history of CKD, hypertension, high cholesterol and low HDL levels. **Conclusions:** CKD patients should be examined and counselled on their first visit on the impact of dietary phosphorus intake responsible for the progression of CKD and the development of CVD. Restricting DPhI at early stages of CKD can be a probable therapeutic interventional strategy for preserving renal function in CKD patients.
Keywords: chronic kidney disease; fibroblast growth factor-23; phosphorous, dietary phosphorus intake, risk factors

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Corresponding author: Dr. Anita Saxena, Professor, Department of Nephrology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow (Uttar Pradesh), India-226014

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INTRODUCTION

Chronic kidney disease (CKD) has emerged as a public health problem requiring corrective measures at the early stages. (*1*) At the global level, an alarming rise in the burden of CKD is being experienced. A study by Coresh J et al showed the prevalence of CKD stages 1 to 4, increased from 10.0% to 13.1% in 10 years. (*1*)Whereas, in the US adult population it was 11% (19.2 million). (*2*)

In India, a study from two of the largest cities (Delhi and Chennai) showed 1 in 12 individuals with CKD, putting them at high risk for adverse outcomes. (*3*) The prevalence of CKD was observed 17.2% by Singh et al with approximately 6% having CKD stage 3 and above. (*4*)

This mandates identification of risk factors associated with progression of renal disease to irreversible loss of renal function. The traditional risk factors for the progression of renal disease include uncontrolled diabetes, hypertension, a family history of CKD, older age (*5*), smoking and obesity. (*6*)

Recently, evidences are supporting the deleterious health effect of excess dietary phosphorus intake in human and animal populations. High-phosphorus diets, independent of protein intake, have caused renal damage in animal models of CKD. (*7*)

The potential hazard of a high phosphorus intake in the healthy population has demonstrated that it may significantly disrupt the hormonal regulation of phosphorus leading to disordered mineral metabolism, vascular calcification, bone loss, and impaired kidney function. One of the hormonal factors that have been markedly affected by dietary phosphorus loading is fibroblast growth factor-23 (FGF-23), being a key factor responsible for many of the cardiovascular disease (CVD). The increase in phosphorus intake is due to the use of phosphorus-containing additives in processed food and the increased consumption of processed and fast foods is an important factor that needs to be emphasized. (*8*)

Experimental studies have shown that high phosphorous load increases serum FGF-23 levels (*9,10*) in the early stages of CKD which inhibits renal 1α-hydroxylase thus inhibiting calcitriol synthesis and reducing gut absorption of calcium which causes hypocalcaemia. Hypocalcaemia, in turn, augments increased production of parathyroid hormone (PTH), (*11,12*) thus leading to secondary hyperparathyroidism. (*13*) Increased FGF-23 has emerged as a new biomarker for death in early CKD when phosphate concentration is still normal. (*14*)

Uribarri et al. (*8*) and Ritz et al. (*15*) have reported that the increased use of phosphorus in food processing and the consequentially increased intake of phosphorus, exceeding the nutritional recommendation, is responsible for the association with cardiovascular diseases (CVD) and mortality, even when serum phosphate levels being in the normal range. (*14*)

Hence, it is important to investigate the consequences of high phosphorus intake and monitor the dietary phosphorus intake (DPhI) of patients with CKD. Therefore, considering the negative impact of high-phosphorus diets in CKD patients, we conducted a cross-sectional study to examine the dietary phosphorus load in CKD patients according to their dietary phosphorous intake on biochemical parameters and nutritional status in CKD patients and to find the risk factors that might be associated with the progression of CKD (stage 1/2).

MATERIALS AND METHODS

This is a cross-sectional study, approved by the Ethics Committee of the institute Sanjay Gandhi Post Graduate Institute of Medical Sciences with the research grant number, 2015-116-IMP-87. Patients were recruited from outpatient departments of nephrology and endocrinology. All of the patients studied gave their informed consent to the study. The inclusion criteria were a) patients $\geq$ 18 years of age, and b) eGFR with >90 (CKD stage 1) and 60-89 ml/min/1.73m^2 (CKD stage 2). (*16*) The exclusion criteria where the patient should not have taken dietary counselling for restricting protein or phosphorus intake in the past 6 months and was not taking calcium with meals.

Estimated GFR (eGFR) was calculated using the MDRD formula.(*17*)

eGFR: 186* (serum creatinine (mg/dl))$^{-1.154}$ * (age (years))$^{-0.203}$ *(0.742 (if female))

DATA COLLECTION

This study presents the data from 79 CKD patients. Demographic data of the patients such as age, gender, medical history of the patient; vitals (blood pressure), dietary habits i.e., vegetarian (plant-based diet) or non-vegetarian (meat-based diet preferably taking 4-5 times a week), biochemical parameters including glomerular filtration rate (GFR), serum creatinine, serum phosphorous, intact parathyroid hormone (iPTH), urinary phosphorous, serum calcium, vitamin D, lipid profile such as total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL) and dietary data including dietary energy, dietary protein and dietary phosphorous intake (DPhI) were collected of all the patients using questionnaires at a face-to-face interview. Phosphorous-to-protein ratio (PPR) of food items was calculated by dividing DPhI (mg/day) by dietary protein (gm/day).

Four ml of fasting blood sample was taken from all CKD patients for analysing FGF23 (Kainos Lab Inc., Tokyo, Japan) (*18*) and sKlotho (Immuno-Biological Laboratories Co., Ltd.)(*19*) levels which were assayed using an enzyme-linked immunosorbent assay (ELISA) technique as per the manufacturer's protocol.

FOOD INTAKE ASSESSMENT

Individual assessment of dietary habits was carried out by the renal dietitian for all of the patients studied. Nutrient intake assessment was obtained by a 3-day dietary recall in a structured interview with the help of a dietary questionnaire prepared and designed for this study. The questionnaire included the demographic section where the patient's age, weight, blood pressure, medications, diagnosis were taken and recorded. Next section included a table where the dietary intake of the patients was recorded. The table consisted of 6 six columns named as meals, food items with various names of food items mainly found in Indian households, the quantity of the ingested food, dietary energy, protein and phosphorous intake columns. The last section of the questionnaire included questions like total water intake, salt intake, any herbal supplements and any additional food items that are not mentioned in the list to provide extensive coverage. All the patients were shown food models and standard cups/utensils for estimation of portion size accuracy. This method was of value in assisting patients in estimating the amounts eaten. The energy intake, protein and phosphorous intake of the patients were calculated which was done using Standard Nutrition Tables of National Institute of Nutrition (NIN) published by *Indian Council of Medical Research* (*ICMR, 2017*). Average of the three days was taken as the final value which was further used

for the analysis of the dietary data. All of the values refer to the natural content of raw, unprocessed foods.
Based on DPhI, patients were divided into two groups: Recommended phosphorous intake (RPhI) and High phosphorous intake (HPhI).
The recommended phosphorous intake (RPhI) group comprised of patients consuming dietary phosphorus which was within the recommended allowance for CKD patients <1000 mg/day (*20*); without any previous counselling or dietary intervention.
High phosphorous intake (HPhI) group included patients with high DPhI i.e., >1000mg/day.

STATISTICAL ANALYSIS:
Statistical analyses were performed using IBM SPSS Statistics for Windows, version 20. Results were presented as mean ± SD and n (%). For comparisons of differences between the two groups, bifurcated by the dietary phosphorous intake of the patients, chi-square test for categorical variables and independent t-test for continuous variables were used. For correlation analysis, Pearson correlation was used and reported as a correlation coefficient, r. To identify significant associated risk factors for CKD, odds ratios (OR) with 95% confidence interval (CI) using binary logistic regression analysis was calculated. Two-tailed P-values <0.05 were considered statistically significant.

RESULTS
Clinical characteristics of CKD patients are shown in Table 1. The mean age of CKD patients was 38.16±12.12 years with the mean eGFR of 82.95±16.93 ml/min/1.73m^2. Table 2 shows the demographic, biochemical, dietary and lipid profile data of 79 CKD patients. Overall, CKD patients had high mean phosphorous intake (1104.64±248.53 mg/day). The urinary phosphorus and urinary protein were tested in 63 and 59 patients respectively because of non-availability of data from hospital records.
Further, we spilt all the patients according to their dietary phosphorus intake into two groups; RPhI (n=37) and HPhI (n=42) group to examine the effect of the dietary phosphorous load as shown in Table 1 and Table 2.

Table 1: Clinical characteristics of CKD patients (n=79) and according to their dietary phosphorous intake (mg/day)

Parameters	All CKD patients (n=79)	CKD patients based on dietary phosphorous intake [$]	
		RPhI	HPhI
Number of patients	79	37	42
Mean age, years Male Female	38.27±12.06 41.42±9.22 34.70±13.91	38.27±12.37 41.25±9.69 34.76±14.45	38.28±11.92 41.59±8.99 34.65±13.81
GFR, ml/min/1.73m^2 Male Female	83.69±17.37 85.10±17.95 82.08±16.78	85.00±18.64 88.25±20.42 81.17±16.05	82.53±16.30 82.23±15.27 82.85±17.76
Diabetes, number (%)	26 (32.9%)	11 (29.7%)	15 (35.7%)
Dyslipidaemia	15 (18.9%)	0 (00.0%)	15 (35.7%)
Hypertensive	23 (29.1%)	6 (16.2%)	17 (40.5%)
Family history with CKD	37 (46.8%)	11 (29.7%)	26 (61.9%)
Renal diagnosis			
Diabetic *kidney disease*	18 (22.7%)	6 (16.2%)	12 (28.6%)
Polycystic kidney disease	14 (17.7%)	4 (10.8%)	10 (23.8%)
Other renal diseases	16 (20.2 %)	11 (29.7%)	5 (11.9%)
Unknown	31 (39.2 %)	14 (37.8%)	17 (40.5%)
Medications			
Anti-hypertensive drug(s)	23 (29.1%)	6 (16.2%)	17 (40.5%)
Statins	16 (20.2 %)	1 (2.7%)	15 (35.7%)
Insulin or oral hypoglycaemic agent	26 (32.9%)	11 (29.7%)	15 (35.7%)

Values are presented in Mean±SD/n (%).
CKD: Chronic Kidney Disease; GFR: Glomerular Filtration Rate
[$] Statistical analysis performed between RPhI(n=37) and HPhI (n=42) groups.

Diet type (p=0.042) categorized as vegetarian and non-vegetarian was found to be statistically different between the two groups. In both RPhI and HPhI groups; the serum phosphorous levels (p<0.05) and PTH (p=0.04) were within the normal range through the mean values of patients in HPhI were higher when compared those in the RPhI group and the difference was statistically significant. FGF-23 (p<0.05) and urinary protein (p<0.05) were significantly higher in HPhI than RPhI. sKlotho levels were significantly low (p<0.05) in the HPhI group when compared to the RPHI group.

The mean eGFR of patients in HPhI group was lower (82.53±16.30 ml/min/1.73m^2) than those of RPhI (85.00±18.64 ml/min/1.73m^2) but the difference was not statistically significant. There was no statistical difference between groups in serum creatinine, urinary phosphorous, serum calcium and Vitamin D levels. (Table 2)

Higher intake of dietary protein (p<0.05) and dietary phosphorous were significantly (p<0.05) observed in HPhI when compared with RPhI group. Because of the higher intake of protein and phosphorous by CKD patients of HPhI group; PPR (p<0.05) was significantly higher when compared with the RPHI group. Energy intake of CKD patients between both groups was not statistically different (p=0.166). (Table 2)

On comparing lipid profile between RPhI and HPhI group; it was observed that total cholesterol levels (p<0.05) of CKD patients were significantly different and higher in HPhI group when compared with RPhI group. Also, HDL levels (p=0.000) of CKD patients were significantly lower in the HPhI group when compared with the RPhI group. But, LDL levels (p=0.432) were not significant. (Table 2)

Table 2: Demographic, biochemical, dietary, lipid profile and anthropometric data of the CKD patients and according to their dietary phosphorous intake (mg/day)

Parameters (unit)	All CKD patients (n=79)	CKD patients based on dietary phosphorous intake [$]		
		RPhI(n=37)	HPhI (n=42)	p-value
Diet type				
Vegetarian	46	26	20	0.042†
Non-vegetarian	33	11	22	
Gender				
Male	42	20	22	0.882†
Female	37	17	20	
GFR (ml/min/1.73m^2)	83.69±17.37	85.00±18.64	82.53±16.30	0.531
Serum creatinine (mg/dL)	0.97±0.22	0.96±0.22	0.99±0.21	0.577
Serum phosphorous(mg/dL)	3.58±0.67	3.21±0.60	3.91±0.54	< **0.05**
iPTH (pg/mL)	51.55±10.75	48.91±9.65	53.87±11.24	0.040
FGF-23 (pg/mL)	57.66±7.52	54.45±4.43	60.50±8.53	< **0.05**
sKlotho(pg/mL)	724.06±105.26	797.51±79.43	659.36±79.88	< **0.05**
Urinary phosphorous(mg/day) *	640.12±219.88	648.42±192.30	633.04±243.61	0.785
Serum calcium(mg/dL)	8.79±0.77	8.89±0.60	8.70±0.89	0.267
Vitamin D (ng/mL)	26.30±8.37	25.72±8.23	26.81±8.56	0.566
Energy intake (kcal /kg/day)	27.47±2.51	27.05±2.06	27.84±2.82	0.166
Dietary Protein intake (gm/kg/ day)	0.63±0.11	0.54±0.06	0.71±0.09	< **0.05**
Dietary phosphorous (mg/day)	1104.64±248.53	868.96±69.99	1312.26±137.57	< **0.05**
Phosphorous-to-protein ratio	25.36±3.95	23.24±2.18	27.22±4.24	< **0.01**
Total Cholesterol (mg/dL)	244.30±49.26	201.07±29.16	282.38±26.43	< **0.05**
High-density lipoprotein (mg/dL)	44.08±12.89	55.15±8.34	34.34±6.90	< **0.05**
Low-density lipoprotein (mg/dL)	155.79±44.03	159.97±45.36	152.11±43.04	0.432

*n=29 (RPHI) and n=34 (HPHI)

FGF-23: Fibroblast Growth Factor-23; iPTH: Intact Parathyroid Hormone; GFR: Glomerular Filtration Rate

Values are presented in Mean±SD.

[$] Statistical analysis performed between RPhI(n=37) and HPhI (n=42) groups.

†p-value calculated by chi-square test.

Significant values are presented in **BOLD**.
Table 3 shows the binary logistic regression analysis result which was performed to find out the risk factors for progression CKD based on their dietary phosphorous intake of patients. Risk factors which had statistical bearing on progression of CKD who were on high phosphorous diet were diet type, family history of CKD, hypertension, high cholesterol levels and low HDL levels.

Table 3: Significant Risk Factors in Multivariable Logistic Regression Model for Chronic Kidney Disease Stages stratified by their dietary phosphorous intake

Parameters	Odds Ratio (OR)	95% Confidence Interval (CI)	P-value
Age	1.179	0.411-3.376	0.760
Gender	0.935	0.386-2.267	0.882
Diet type (veg/non-veg)	2.600	1.027-6.585	**0.044**
Family history of CKD	3.841	1.499-9.839	**0.005**
Diabetes mellitus	0.762	0.296-1.962	0.573
Hypertension	0.084	0.018-0.397	**0.002**
Total cholesterol	0.002	0.000-0.022	< **0.05**
High-density lipoprotein	0.010	0.001-0.081	< **0.05**
Low-density lipoprotein	2.316	0.777-6.901	0.132

p-value is calculated by binary logistic regression test.
Significant values and parameters are in **BOLD**.

Correlations between dietary phosphorous intake of patients and various parameters are shown in Table 4. In the HPhI group, high dietary phosphorous intake was negatively associated with lower levels of eGFR, sKlotho and HDL-C whereas; dietary phosphorous intake correlated positively with serum phosphorous, FGF-23, cholesterol levels and LDL levels. Protein intake showed a significant positive correlation with dietary phosphorous intake in both the groups. PPR correlated with dietary phosphorous intake in both groups.

Table 4: Correlation of biochemical parameters with dietary phosphorous intake in CKD patients

Parameters(unit)	Dietary Phosphorous Intake (mg/day)			
	RPhI (n=37)		HPhI (n=40)	
	r-value	p- value	r-value	p- value
GFR (ml/min/1.73m^2)	0.311	0.061	-0.604	< **0.05**
Serum phosphorous(mg/dL)	-0.302	0.070	0.807	< **0.05**
FGF-23 (pg/mL)	-0.185	0.273	0.642	< **0.05**
sKlotho(pg/mL)	0.201	0.233	-0.552	< **0.05**
Urinary phosphorous(mg/day) *	0.081	0.675	0.143	0.421
Serum calcium(mg/dL)	-0.082	0.630	-0.011	0.944
Vitamin D (ng/mL)	0.060	0.723	0.261	0.095
Dietary Protein intake (gm/kg/ day)	0.506	**0.001**	0.390	**0.011**
Phosphorous to protein ratio	**0.357**	**0.030**	**0.421**	**0.006**
Total Cholesterol (mg/dL)	-0.199	0.237	0.573	< **0.05**
High-density lipoprotein (mg/dL)	0.269	0.107	-0.494	**0.001**
Low-density lipoprotein (mg/dL)	-0.198	0.240	0.339	**0.028**

*n=29 (RPhI) and n=34 (HPhI)
r: Pearson Correlation; FGF-23: Fibroblast Growth Factor-23; iPTH: Intact Parathyroid Hormone; GFR: Glomerular Filtration Rate
p-value calculated by Pearson correlation test.
Significant values are presented in **BOLD**.

DISCUSSION

In the present study, CKD patients were divided into two groups based on their DPhI to see the effect of phosphorous load on the biochemical parameters associated with the risk of CKD progression.

We observed that FGF-23 levels were high in CKD patients with high DPhI. Yoshikawa et al found higher levels of FGF-23 in the mice fed on high phosphate diet group than in the mice on low phosphate diet group whereas; renal *Klotho* mRNA expression levels were found to be lower in the mice fed on high phosphate diet group than in the mice on low phosphate diet group. (*21*) Our study also found sKlotho levels being significantly low in HPhI group than in RPhI group.

In the early stage of CKD, we found that FGF-23 positively associated with high DPhI which was reflected in eGFR of 82.53±16.30 ml/min/1.73m^2. This shows that phosphorus load causes a decline in renal function, especially with reduced nephron mass and to maintain phosphorus homeostasis, the FGF-23 levels increase. (*22*) Few studies from the past have shown that increased levels of FGF-23 are associated with mortality, CKD progression, and cardiovascular events in CKD patients (*23*, *24*, *25*) and sKlotho with signs of vascular dysfunction (*26*) and soft-tissue calcification in CKD (*27*). As phosphate is a major regulator of FGF-23 expression (*28*) and dietary phosphate loading increases serum FGF-23 concentration, experimental studies have shown that a decrease in the dietary phosphate intake induces a substantial decrease in the concentration of FGF-23 of over a 7-fold range in a linear, dose-dependent fashion in normal mice,(*28*) so it seems obvious that dietary control of phosphorous intake, with due consideration of cultural habits, may be useful to prevent CVD events thereby reducing FGF-23 levels. (*29*)

We also found that a higher intake of dietary phosphorous correlated positively with higher normal levels of serum phosphorous. However, not only the amount of phosphorus intake was responsible but also the type and source of phosphorus intake were responsible for significantly increasing serum phosphorus concentration. Moore et al performed a clinical trial and their analysis showed that cereals and dairy products with inorganic phosphate additives significantly increased serum phosphorus concentration, despite being consumed less frequently than foods without phosphate additives. (*30*)

Our study reported hypertension as a significant risk factor for developing CKD, in our study with 62% of CKD patients being hypertensive. Yousri M Barri stated that CKD has been steadily rising with the incidence of risk factors, including elevated blood pressure being the most significant risk factor for developing CKD. (*31*) High blood pressure can be a cause and/or a consequence of CKD that may develop early in the course of CKD which is associated with adverse outcomes such as worsening renal function and development of cardiovascular disease (CVD). (*32*) Thus, optimal blood pressure control should be a major goal in preventing adverse outcomes among CKD patients. (*33*)

Lipid profile is also an important aspect which should be screened in CKD patients on their visit to hospitals or coming for follow-ups. As lipid abnormalities, dyslipidaemia has been a common cardiovascular risk factor in CKD patients which increases with worsening kidney function. Our study also reported lipid abnormalities as a risk factor for early CKD, such as reduced HDL-C and elevated cholesterol as eGFR decreased (*34*) in CKD patients. This pattern of dyslipidaemia is similar to reports from previous studies from a case-control study performed by Adejumo et al. (*35*) This calls for early detection and management of dyslipidaemia in CKD patients which will help in reducing the cardiovascular burden and retard progression of CKD with the help of both lifestyle modification and therapeutic intervention. (*35*)

In our study, the patients were consuming a mixed diet which included both animal and plant products. Most of our CKD patients were non-vegetarian (meat-eaters); there were 11 non-vegetarians in RPhI group and 22 non-vegetarians in HPhI group. As protein-rich foods are historically and naturally the main source of dietary phosphorus, (*11*) and phosphorus in animal protein is in the form of organic phosphate, which is readily hydrolyzed and gets absorbed (*36*) up to 60% to 80% (*11*) explaining the reason behind the high number of non-vegetarians in HPhI group being responsible for their high phosphorus intake. The findings of our study show the impact of "animal-based-diet" on phosphorus homeostasis and renal function which emerged as a risk factor for CKD progression. This study brings to the forefront the importance of the right dietary counselling of the plant-based diet. The National Kidney Foundation recommends a vegetarian diet, or part-time vegetarian diet being favourable to CKD patients. (*37*) A cross-sectional study by Liu et al showed a strong negative association between vegetarian diets and prevalence of CKD suggesting vegetarian diets could help reduce the occurrence of CKD. (*38*)

Some studies suggest the quality and type of diet independently affect CKD risk. (*39*)

Dietary interventions with low-protein and phosphorous content ensuring plant-based protein intake may be a candidate therapeutic approach for attenuating kidney injury (*40*) and ameliorating renal outcome and reduce cardiovascular risk factors. (*41*)

Bringing drastic changes in food habits of patients is a challenging task, however, if these changes are implemented at an early stage the acceptance may be better, although, the benefits are subtle and not very obvious to the patient. However, this early acceptance will make the patient more at ease with restricted food choices and will feel content with restricted food

choices during the entire natural course of disease progression.

Here, the role of the renal dietitian appears mandatory in educating the CKD patients through the management of nutrition counselling (*42*) and developing a nutritional intervention that is appropriate for patients. A renal dietician should educate patients on the type of diet CKD patient should follow and elaborate on the sources of high phosphorus foods. Rather than a structured dietary plan, a list of basic recommendations to improve compliance with a low-phosphorous diet in CKD may allow patients to reach the desired phosphorous target in the daily eating that best suits their preferences and clinical needs. (*43*) According to KDIGO CKD-MBD guideline Update (*44*), dietary control of phosphorous intake is certainly the first therapeutic approach and the most physiologic tool, with a stronger effect on dietary phosphate intake reduction when compared with phosphate binders. Hence, strict adherence to low dietary phosphorous intake can benefit patients and this should be initiated in the early stages of CKD (stage 1 and 2).

Conclusion

To conclude, CKD patients should be cautioned and counselled on their first visit on the impact of dietary phosphorus intake on the progression of CKD and development of CVD. The dietary intake should be monitored regularly, and the effectiveness of compliance should reflect in the clinical condition. The results of the present study emphasize the importance of controlling dietary phosphorous intake as the first therapeutic strategy right from the early stages (Stage 1). Also, there is a need to develop innovative approaches for dietary counselling and intervention for assuring compliance to prescribed diets to CKD patients. Timely dietary counselling can help stabilize biochemical parameters which may prevent accelerated deterioration kidney function.

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