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A Review on Patient Diet Management for Chronic Kidney Disease Based on Clinical Trials

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***Abstract*: The public health issue of chronic kidney disease (CKD) is especially acute in India. Only 10% of patients with incident end stage renal disease (ESRD) cases receive therapy in India due to limited availability and expensive costs. To best manage CKD, ensure a sufficient intake of protein and energy, prevent or correct protein-energy wasting, and optimise nutritional management of CKD, a balanced and personalised low protein diet (LPD) approach should be developed. The relationship between mineral intake and chronic renal disease has only been briefly studied (CKD). In a cross-sectional analysis, the Health Examinee (HEXA) cohort of the Korean Genome and Epidemiologic Study was used to examine the relationship between mineral intake (calcium, phosphorus, sodium, potassium, iron, and zinc) and CKD (KoGES). A food frequency questionnaire was used to determine the mineral consumption of 159,711 participants. An estimated glomerular filtration rate (eGFR) of less than 60 mL/min/1.73 m2 was used to characterise CKD. Each mineral's dietary intake was separated into quartiles, with the quartile containing the acceptable intake (AI) or recommended dietary allowance (RDA) of each mineral serving as the reference. As a result, every effort should be taken to stop the progression of CKD. The study has shown to require analyses of food variance, nutritional demand changes, and variation in dietary intake in order to strengthen the links between nutrient consumption and renal function. This review article examines the function of low protein diets (LPD) in the treatment of CKD patients and offers advice on how to use them in clinical settings. It also discusses the relationship between inadequate mineral consumption and the prevalence of CKD in the general population.**

***Keywords*: Calcium, Chronic kidney disease, Phosphate, Potassium, Protein, Sodium.**

1. Introduction

The impact of chronic kidney disease (CKD) on the world's public health has increased significantly. Nutritional therapy is essential for CKD patients' recovery, and changes in their nutritional status are important prognostic indications (Unwin et al 2011). The effects and combinations of prescription drugs or dietary supplements with meals are essential since the kidneys constitute a significant excretion pathway in CKD patients. As a result, depending on the patient's residual renal function, several drugs need to have their doses adjusted. Co-morbidities and advanced age are additional factors that may make it more challenging to balance food and medication interactions. Nephrologists and dietitians must work together to provide nutritional care and practise competent clinical medicine. Dietary treatments for CKD that decrease its progression and lessen its effects are crucial for controlling the condition. As a source of energy for ckd stages, patients in high tertile or both high and low tertile groups consumed more protein, energy, fat, carbohydrate, fibre, salt, and potassium. Partial correlation analysis showed that average salt intake was significantly correlated, dietary protein, energy, fat, carbohydrate, potassium, and fibre intake. High protein diets, which are commonly defined as ingesting more than 1.2 grammes of dietary protein per kilogramme of body weight per day (g/kg/day), have been related to significant alterations in renal function and kidney health (Kalantar et al. 2016). In contrast to dietary intake of fat and carbohydrates, higher protein intake modifies renal hemodynamic by raising intraglomerular pressure and renal blood flow, which results in a higher glomerular filtration rate (GFR) and more effective excretion of protein-derived nitrogenous waste products, though an increase in kidney volume and weight may also occur. However, while being the largest controlled trial of dietary protein management in CKD to date, the Modification of Diet in Renal Disease (MDRD) study was unable to show the LPD's obvious effectiveness in delaying the progression of CKD (Fouque et al. 2007). The prevalence of inadequate dietary intake among people with CKD and ESRD puts them at risk for protein malnutrition (Kalantar-Zadeh et al. 2016). It might be claimed, nonetheless, that consuming more than 25% less protein than is often advised over the long term could endanger metabolic balance and life through a steadily declining nutritional status in individuals who may also have hypercatabolic diseases such infections (Li et al. 2010). The term "protein energy waste" (PEW) is used to describe how renal disease patients simultaneously lose their protein and energy stores. It typically develops and gets worse as CKD gets

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worse (Fouque et al. 2008). When the energy supply is insufficient, patients with CKD and end-stage renal disease (ESRD) catabolize muscle to generate necessary energy, which causes a protein shortage (Ikizler et al. 2002). Limited physical activity in the context of increased resting energy demand would undoubtedly result in muscle atrophy and general deconditioning, even while it would temper overall body energy expenditure (Avesani et al. 2012).

Among the plant foods that are high in fibre include vegetables, fruits, whole grains, nuts, legumes, and seeds (Slavin et al. 2012). A variety of health benefits of dietary fibre include enhanced gut bacteria, better blood sugar regulation, decreased body weight, lower blood pressure, and prevention of constipation (Ma et al. 2021). The American Dietetic Association recommended taking 25–30 g of dietary fibre per day for people in the general population (Gill et al. 2021). Similar to this, the 2015 Dietary Reference Intake for Koreans (KDRI) recommended that all Korean citizens, regardless of age, should consume 20 g of fibre for women and 25 g for men per day. To our knowledge, there are no specific recommendations for dietary fibre consumption in the population with CKD in the currently published nephrology guidelines (Slavin et al. 2008). High-fiber diets have a lower bioavailability of potassium and phosphorus than other sources of those nutrients, particularly processed foods. Additionally, a high-fiber diet increases faecal potassium excretion by increasing stool volume (DeSalvo et al 2016; Kalantar et al. 2016). In support of this, cohort studies failed to find a significant association between fiber-rich diets and blood potassium levels, and there were insufficient or nonexistent correlations between dietary potassium and serum potassium in advanced CKD (Palmer et al. 2017). These results led the most recent Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines to recommend "adjusting" potassium consumption "as needed" rather than consistently in order to maintain potassium levels in the normal range (Sabatino et al. 2016).

Dietary salt reduction can improve the antiproteinuric effects of ACE inhibitors (ACEIs) and angiotensin receptor blockers in addition to decreasing blood pressure (BP). In contrast to the lowest quartile of urinary sodium excretion (194.6mmol/24 hours), reducing dietary sodium intake to glomerular filtration rate (eGFR) 20-70mL/ min/1.73 m2 depending on age was linked to a 54% risk increase in end-stage renal disease (ESRD) or a 50% decrease in eGFR, as well as a 43% risk increase in all-cause mortality, according to the 2021 Kidney 3 guidelines. However, other cohort studies claim that salt consumption has little bearing on the progression of CKD into ESRD or the decline in eGFR. The outpatients rarely received dietary recommendations about their intake of potassium. Doctors would administer antihyperkalemic drugs like polystyrene sulfonate to treat hyperkalemia if serum potassium levels were found to be abnormal. Each patient's baseline was the day of the seventh 24-hour urine sample. Over the course of four years, each patient's kidney function was assessed on numerous occasions to monitor its development (Gill et al. 2021).

To keep the equilibrium of potassium in the body, dietary intake must be linked to concurrent excretory activities in the kidney and digestive system (Sharma et al. 2013). Patients with CKD are more prone to dyskalemia because sudden increases in K1 intake might result in the development of either hyperkalemia or hypokalemia. The Na1, K1-ATPase, and other predominantly muscle cell-based transporters and channels maintain an asymmetric distribution of ions in skeletal muscle cells. The transmembrane potential (90 mV) is a result of different ions not being distributed equally across the intracellular and external compartments. The pathophysiology of CKD-MBD is complicated and includes changes in calcium, calcitriol, parathyroid hormone (PTH), and fibroblast growth factor-23 (FGF23), in addition to hyperphosphatemia (Sharma et al. 2011). Phosphorus, which is present in nature as (po4) and has a high chemical reactivity, is an important component of numerous vital biological and enzymatic processes required to support life. The recently discovered FGF23 axis also affects calcium-phosphorus metabolism, leading to even more complex connections across the previously mentioned pathways. However, it is still believed that phosphorus retention is a substantial and early cause of CKDMBD (Gutierrez et al. 2010).

2. The Impact of a High-Protein Diet on Developing CKD

Consuming more protein may cause glomerular hyperfiltration and higher intraglomerular pressure. This may cause or worsen chronic kidney disease by damaging the glomerular structure. A low-protein diet of 0.6–0.8 g/kg/day is therefore usually advised for the management of CKD.

A. Low protein diet for stage 1-2 CKD patients

It has been demonstrated that a high-protein diet, which is commonly defined as more than 1.2 grammes of dietary protein per kilogramme of body weight per day, can significantly alter renal function and kidney health (Kalantar-Zadeh et al. 2016). Contrary to higher fat and carbohydrate intake, higher protein intake modifies renal hemodynamics by enhancing renal blood flow and elevating intraglomerular pressure, leading to an increase in glomerular filtration rate and a more effective excretion of nitrogenous waste products derived from protein while also increasing kidney volume and weight (Fouque et al. 2007). Both in animal models and in clinical studies including human participants, the so-called "glomerular hyperfiltration" brought on by a high-protein diet has been widely demonstrated (Tirosh et al. 2013).

B. Low protein diet for stage 3–4 CKD patients

120 CKD patients (stages 3 and 4) without comorbidities were enrolled in this 24-week randomised controlled non-blinded study from the Iranian outpatient clinics affiliated with Shiraz University of Medical Sciences. The procedure of finding patients started in July 2019 and was finished in August 2019. All 120 CKD patients, both male and female, were enrolled using this technique. The block randomization method, with a four-sequence block size and 30 possible block combination (Cases et al. 2019)s of group A or B, was employed to randomly assign participants to either group A or group B. Eventually, using the predetermined sequence, 60

people in each group were selected at random. Every four weeks the dietician would visit, and every eight weeks the nephrologist. The participants were instructed to continue taking their regular prescriptions, such as hypertension meds, as well as calcium and vitamin D supplements, during the trial. As part of the dietary counselling, a customised nutritional regimen was developed for each patient using the National Kidney Foundation Kidney Disease Outcomes Quality Initiative (NKF KDOQI) 2000 edition nutrition guidelines (Dis et al. 2000; Patani et al. 2020). As a result, the patients in this study were given a diet that included minimal sodium intake (less than 2000 mg per day), moderate protein intake (0.7 g per kilogramme of ideal body weight per day), and energy intake (up to 35 kcal/kg per day, depending on the patient's nutritional status). Clinical decisions for individualised modification of dietary potassium and phosphate were made based on a combination of current dietary intakes and biochemical data, and at each follow-up visit, a dietitian took into account the recommended daily intakes of potassium (2000-3000 mg) and phosphorus (10 mg/kg/day) (Cases et al. 2019).

Patients with CKD have a higher risk of morbidity, which increases their risk of developing ESRD (Krebs et al. 2012). Numerous clinical situations, such as the use of nephrotoxic drugs or the avoidance of nephrotoxic medications, unanticipated acute kidney injury (AKI), and nutritional therapies, may have an effect on the levels of eGFR (Li et al. 2010). The renal diet in this trial required 0.75 g/kg/day of protein, at least 50% of which was HBV. Despite the fact that the recently updated 2020 NKF KDOQI guideline recommends that people with CKD stages 3-5 who are not on dialysis and do not have diabetes ingest 0.55 to 0.6 g/kg of protein daily (Ikizler et al. 2020; Zuidersma et al. 2020).

C. Low protein diet for stage-specific CKD patients 4-5

Patients in Study received ketoanalog supplements added to low protein diets (0.58 g/kg/day) or very low protein diets (0.28 g/kg/day) for the duration of the study. Their GFR was between 13 and 24 ml/min (KA). The results of the investigation revealed that, while the rate of decline in study had not considerably changed, it had slowed slightly in study A's GFR reduction. (Patel et al. 2000). In 2002, Kher et al. discussed the large burden of ESRD in India as well as the restricted availability and high cost of RRT in a nephrology forum. This prompted us to evaluate the role of LPD in preventing and delaying the progression of CKD to ESRD. The possible benefits of dietary protein restriction over extended follow-up are shown in Figure 2. Even without this, the MDRD trial's secondary analysis showed that dietary protein restriction was beneficial (Levey et al. 1999).

D. Patients on a very low protein diet who have stage 5 CKD

In CKD patients, we also examined the efficacy and safety of a very low-protein diet supplemented with KA. The study included 29 non-diabetic participants with stable stage V CKD. In contrast to 15 patients who declined therapy with the KA (treatment group), 14 patients agreed to it (control group). The patients in the two groups were comparable with respect to renal impairment, proteinuria levels, age, sex, eating habits, and other factors. Patients in the treatment group were instructed to ingest 0.3 g/kg/day of mixed protein supplemented with KA (ketosteril 1 tablet/5 kg body weight), whereas patients in the control group were permitted to continue eating normally. The evaluation of renal function and protein intake employed urinary creatinine clearance during a 24-hour period and urea nitrogen appearance (UNA), or 6.25. Each was checked every month for six months. The rate of deterioration in creatinine clearance in the treatment group was 0.09 ml/min/month, whereas it was 0.3 ml/min/month in the control group (Patel et al. 2000).

3. Effect of Sodium Intake on the Development of CKD

Cardiovascular disease (CVD) is the leading cause of death in dialysis patients each year, accounting for over 50% of deaths in patients with end-stage renal disease, including those receiving hemodialysis and peritoneal dialysis (PD) (Elsayed et al. 2007). Other studies have demonstrated that improved survival is predicted when sodium intake is positively correlated with energy intake, even though some intervention studies have demonstrated that sodium restriction improves left ventricular hypertrophy and cardiac function in patients receiving dialysis (Asci G et al 2006). At the beginning of PD treatment, it has been recommended that a daily intake of no more than 2.3 g of sodium, at least 0.8 g/kg of protein, and 25 kcal/kg of energy be followed. These guidelines come from the European Best Practice Guidelines and the International Society of Renal Nutrition & Metabolism (Fouque et al. 2008). Each patient was told to use a 1- or 2-g salt spoon to measure the soy sauce in a 5-ml small cup and the amount of additional

Table 1
Changes in renal function by changes in protein amount in different age humans

Sample size	Mean age (years)	Duration (weeks)	Protein amount (% of total energy amount or g/kg/day)	Renal function changes	References
100	49.3	52	HP-30%, 2.2g/kg/d NP/LP-15%, 1.1g/kg/d	Cr to increase in HP group, but not significant	Li et al. 2010
99	59.4	52	HP-30% NP/LP-15%	No significant differences of eGFR and mAlb.	Larsen et al. 2011
419	57.8	104	HP-30% NP/LP-15%	No significant differences of serum Cr and mAlb.	Krebs et al. 2012
307	45.5	104	HP-LC diet (unlimited protein intake) NP/LP 15%	eGFR and mAlb tended to increase in HP group, but not significant	Friedman et al. 2012
68	50.8	52	HP-1.24g/kg/d NP/LP-0.82g/kg/d	Ccr tended to increase in HP group, but not significant	Wycherley et al. 2012
318	51.1	104	HP-LC diet (unlimited protein intake) NP/LP 18.8–19.1%	eGFR increased in HP group especially among CKD stage III patients.	Tirosh et al. 2014

salt. Patients avoided packaged foods and eating out since it would be impossible to assess the amount of concealed salt (Foley et al. 2000). The amount of dialysis required was ascertained by tracking fluid and nutrient clearances with the help of dialysate and urine collection over the course of a day. The weekly total, peritoneal, and renal Kt/V urea, weekly total, peritoneal, and renal creatinine clearance, and residual renal function were calculated using standard methods. The square of height was also used to standardise lean body mass (LBM), which was determined using the creatinine kinetics method to indicate muscle protein reserves (Dong et al. 2008). Total sodium removal was calculated as the sum of sodium removal from the urine and dialysate (Cheng et al. 2006).

4. Effects of Potassium Consumption on the Development of CKD

The role of cell shifts in regulating potassium homeostasis, there are currently no randomised controlled clinical trials to guide nephrologists or renal dieticians in recommending the ideal daily potassium intake or even the ideal for CKD patients, regardless of stage, despite the fact that many patients in Western countries may have their dietary potassium intake excessively restricted. A daily intake of 4 g of potassium is commonly indicated for hypertensive patients with Stage 1 and 2 CKD. However, dietary potassium restriction is not advised until stage 3 or stage 4 renal impairment. Conversely, glucagon, hypertonicity, a- and b2-adrenergic agonists, and mineral metabolic acidosis drive K1 out of cells. Alkalosis, b2-adrenergic stimulation, and insulin all boost K1 absorption in cells. A significant divergence may be caused by acute or chronic hyperkalemia or hypokalemia (Dong et al. 2002).

The syndrome of hyperkalemic hypertension is seen in CKD. It has been demonstrated that the apical sodium chloride cotransporter (NCC) of the early distal convoluted tubule (DCT1) connects dietary potassium. through its effects on the aldosterone-sensitive late distal tubule (DCT2) and the primary cells of the collecting duct, h can generate more or decreased K1 secretion in accordance with food intake (Foley et al. 1998). The Cl2-sensing capacities of WNK 4 are linked to the regulation of NCC and manipulation of the supply of NaCl downstream to the aldosterone-sensitive distal tubule and collecting duct, which leads to these changes. The reaction to an increase in NaCl supply is particularly potent when aldosterone is present and simultaneously opens the epithelial Na1 channel. Familial hyperkalemic hypertension, also called pseudohyperaldosteronism Type II or Gordon's syndrome, is an uncommon condition but has a chance of passing down through the family. Treatment with calcineurin inhibitors may cause the acquisition of a comparable phenotype more commonly in patients who have had kidney transplants. It is well known that calcineurin inhibitors inhibit both the Na1, K1-ATPase and the K1-secretory channel on the apical membrane (ROMK) of the collecting duct main cell (Cohen et al. 2006). Patients with and without CKD have hyperkalemia for similar reasons. Even though pseudohyperkalemia is less common, the lab technician who checks the serum must check for it to rule out severe hemolysis. Since pseudohyperkalemia will result in elevated serum [K1] levels but normal plasma [K1] levels, the doctor is advised to compare the two measurements. Excessive food consumption seldom results in hyperkalemia in stages 2 or 3 of CKD, but it is much more common in ESRD and later stages of CKD (He et al. 1999).

Dietary potassium intake and the development of CKD in its early stages patients with stage 1 and stage 2 early-stage chronic renal disease participated in nine trials. Six of these studies revealed either a protective effect of high dietary potassium consumption on CKD advancement or a harmful effect of low potassium intake on CKD progression (Mun KH et al. 2019). The average daily intake of dietary potassium in the highest quartile/quintile was >2500 mg, while the average daily intake in the lowest quartile/quintile was 1500 mg (Deriaz et al. 2019). When comparing the highest with the lowest quartile of studies that suggested dietary potassium had a preventive effect on the progression of CKD, the HR ranged from 0.33 to 0.74. (Mun et al. 2019). According to Smyth et al. (2016), higher potassium intake was associated with a hyperkalemia; however, the association was insignificant after controlling for known risk factors, such as age, sex, eGFR, urine albumin-to-creatinine ratio, diabetes, RAASi, diuretic use, BMI, smoking, and urine sodium excretion (Tuomilehto et al. 2021).

5. Effects of Phosphate Intake on CKD Progression

Along with these endocrine and renal mechanisms, a gut-kidney axis may also play a role in the short-term regulation of phosphorus by detecting the load of intestinal phosphate, which causes phosphaturia even before a rise in circulating phosphorus does. It is abundantly found as phosphate in nature (PO4). Phosphorus should be consumed in amounts of 800–1,200 mg per day for adults, and 1,000–1,200 mg per day for children and women who are pregnant or nursing. Phosphorus in plasma should range from 2.5 to 4.5 mg/dL (Zoccali et al. 2011). The average phosphate valence in bodily fluids is 1.8, which is consistent with the fact that under typical physiologic settings, extracellular fluid pH is 7.4. Hydrogen ions are bonded to phosphate either as a monohydrogen structure (HPO4 2) or a dihydrogen structure (H2PO4) at a 4:1 ratio. Intestinal epithelial cells absorb phosphate by a co-transport mechanism involving at least three different kinds of active sodium/phosphate (Na/Pi) co-transporters. It's probable that the conventional strategy for treating CKD patients, which involves limiting their intake of legumes (beans, peas, lentils, and so forth), nuts, seeds, and chocolate, is actually of little significance (Ikizler et al. 1996).

A. Changes of phosphorous intake by industrial processing

Food preservation in today's society, food and drinks are frequently consumed a long time after they are produced or in locations far from the manufacturing site. Despite these facts, the dietary supplement needs to adhere to safety standards and have a certain flavor. This is why the food business is using an increasing amount of food additives, often known as preservatives. Although the usage of chemicals dates back to ancient times, the use of food preservatives has increased during the past three decades. Pork and fish are salted, fruits and vegetables are given a lemon squeeze to keep them from

browning, canned vegetables are prepared with vinegar, and saltpeter is added to meat sausages. Phosphorus-containing additives are utilized in the European Community as emulsifiers and thickeners (E442, E450-E452, E544-E545), as well as acidity regulators (E338-E343). In other words, quantities more than 5 g are generally not used because they are not beneficial, while it is possible that some food products may contain larger concentrations (Uribarri et al. 2007; Patel et al. 2020).

B. Changes of phosphorous intake by homemade processing

However, boiling may be viewed as a beneficial strategy for CKD patients because it lowers the phosphorus content of the food along with lowering the content of sodium, potassium, calcium, and several other minerals in both plant-based and animal-based food. Boiling is generally the least preferred method for preserving bioavailability because this procedure removes many nutrients (primarily minerals) from the food. The amount of phosphorus in food is significantly reduced after prolonged boiling in water. According to the available data, it is possible to reduce the phosphorus content of foods by up to 51% for vegetables, 48% for legumes, 38% for meat, 70% for flour, and 19% for cheddar cheese (Friedman et al. 2012).

6. Effects of Calcium Intake on CKD Progression

Regulation of calcium and phosphorus is essential for maintaining bodily homeostasis. By promoting intestinal absorption, bone (de) mineralization, and renal excretion/reabsorption of both ions, a number of organs help to tightly regulate calcium and phosphorus balance. Several hormones (Quarles et al. 2008) are involved in the regulation of these processes. Patients with CKD are less likely to get natural vitamin D supplementation. Diabetes (6.9%), glomerulonephritis/vasculitis (37.5%), interstitial nephritis (2.8%), cystic/hereditary/congenital (11.1%), other (6.9%), and unknown or missing (34.7%) were the most common renal diseases. According to eGFR, CKD patients were split into three categories. The majority of people with advanced renal failure were men (Bouillon et al. 1984).

Table 2
Impact of protein, sodium, potassium, phosphorus and calcium on renal function

Dietary constituent	Study	Sample size	Diet (variables)	Baseline $eGFR_{cr}$, mL/min/1.73 m^2	Findings
Protein	Knight et al. 2003	1,624	Protein intake	Normal: > 80	A high protein diet was not associated with $eGFR_{cr}$ decline in normal kidney function. However, it was associated with accelerated $eGFR_{cr}$ decline in mild CKD.
	Jhee et al. 2020	9,226	Protein intake (divided into quartiles)	Mild CKD: 55–80	A high protein diet increased the risk of kidney hyperfiltration and a rapid decline of kidney function.
	Klahr et al. 1994 MDRD study 1	585	Usual protein diet (1.3 g/kg/day) vs. low protein diet (0.6 g/kg/day)	Mean 93.9 ± 14.1	Mean $eGFR_{cr}$ decline at 3 years did not differ between the diet group. Supplemented very low protein diet marginally slower $eGFR_{cr}$ decline.
	Klahr et al. 1994 MDRD study 2	255	Low protein diet (0.6 g/kg/day) vs. supplemented very low protein diet (0.3 g/kg/day with ketoacid)	25–55 (mean 38.6)	Supplemented very low protein diet decreased the risk of progression of CKD.
	Garneata et al. 2016	207	Low protein diet (0.6 g/kg/day) vs. supplemented very low protein diet (0.3 g/kg/day with ketoacid)	13–24 (mean 18.5) < 30 (mean 18.0)	
Sodium	Smyth et al. 2014	28,879	Urinary sodium	Mean 68.4 ± 17.6	Urinary sodium excretion was not associated with increased risk of CKD progression Higher urinary sodium excretion was associated with increased risk of CKD progression.
	He et al. 2016	3,939	Urinary sodium	41.5–48.5	
Potassium	He et al. 2016	3,757	Urinary potassium	41.5–48.5	Higher urinary potassium excretion was associated with increased risk of CKD progression.
	Leonberg-Yoo et al. 2017	812	Urinary potassium	32.6	Higher urine potassium excretion was associated with lower risk for all-cause mortality, but not kidney failure.
Phosphorus	Selamet et al. 2016	795	Urinary phosphate	33	Higher urinary phosphate excretion is not associated with the risk of KF and mortality.
	Lynch et al. 2011	1,751	Prescribed dietary phosphate	Hemodialysis	Prescribed dietary phosphate restriction is not associated with improved survival among prevalent hemodialysis patients.
Calcium	Spiegel et al. 2012	12	Dietary calcium	54.8	Total elemental calcium intake should be within 800–1,200 mg/day to prevent calcium deficiency and calcium loading.

Abbreviations: CKD-chronic kidney disease, eGFR-estimated glomerulo filtration rate

7. Role of Dietitian on CKD Clinical Practice

Dietitians play a crucial role in the management and prevention of CKD. The most crucial component of managing CKD is accurate and thorough nutrition planning for patients. A risk factor for the development of metabolic acidosis is the steady drop in GFR. This metabolic acidosis has been demonstrated to slow the progression of CKD to end-stage renal disease, hence the main goal of therapy is to avoid or rectify it (Kramer, H et al 2019). The consumption of a diet richer in animal proteins is the main cause of this acid pool (Joshi et al. 2020). Dietary management, such as cutting back on protein intake or increasing the intake of plant-based proteins, is the most straight forward treatment for this metabolic acidosis (Levey et al. 1996). A low-protein diet, for example, may slow the rate at which kidney function declines and minimize the risk of ESKD in CKD patients, according to primary and secondary studies that came out of the MDRD trial (Levey et al. 2007). It has been demonstrated that dietary treatments, such as a low-protein diet, can halt the course of CKD (Zeller et al. 1991). Protein and phosphorus intake restrictions have been seen to slow the deterioration in renal function in people with type 1 diabetes (Levey et al. 1999). Clinical experts agree that dietary changes can potentially lower the risk of end-stage renal disease in patients with diabetes and CKD by slowing the rate of kidney function decline.

8. Future Research and Clinical Practice

According to an analysis of the MDRD trial, patients who consumed less protein during the course of their follow-up experienced uremic symptoms at lower GFRs than those who consumed more protein. Improved uremic symptoms rather than postponed kidney deterioration may be to blame for the reported decreased risk of end-stage renal failure (Levey at al. 1999). The study also included 24% individuals with polycystic kidney disease (PKD), which may have contributed to findings demonstrating a delay in renal failure because PKD and CKD progress their diseases differently (Levey et al. 1999). In order to strengthen the connections between nutrient consumption and blood pressure, the study has been demonstrated to require assessments of food variance, nutritional requirement changes among different countries, and variation in dietary intake (Ikizler et al. 2020).

Despite the numerous clinical trials that are being conducted in the clinical and nutritional management of CKD, only a small number of these have been translated into clinical practices because there aren't many conclusive associations, the research design isn't very clear, or there aren't many study subjects. Future studies are needed to produce conclusive data that will help physicians and dietitians formulate the best recommendations for their patients. It is necessary to assess the effect of MNT on CKD progression in patients with comorbidities by analyzing associated risk variables (Ikizler et al. 2020). In the future, researchers should concentrate on increasing patient adherence to diet recommendations by creating strategies that will strengthen adherence and long-term adhere.

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