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+15878858911
editorial-office@sciformat.ca

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THE ROLE OF NUTRITIONAL INTERVENTIONS AND SUPPLEMENTATION IN CHRONIC KIDNEY DISEASE – A NARRATIVE REVIEW

Zofia Jędra *(Corresponding Author, Email: zjedra7@gmail.com)*
Samodzielny Publiczny Szpital Kliniczny im. prof. W. Orłowskiego CMKP, Czerniakowska 231, 00-416 Warsaw, Poland
ORCID ID: 0009-0000-5449-9546

Maciej Czapiuk
Medical University of Warsaw (WUM), 61 Żwirki i Wigury Street, 02-091 Warsaw, Poland
ORCID ID: 0009-0007-8675-2180

Maciej Jakub Kozicki
Medical University of Warsaw (WUM), 61 Żwirki i Wigury Street, 02-091 Warsaw, Poland
ORCID ID: 0009-0000-1966-0306

Damian Zienkiewicz
Szpital Praski pw. Przemienia Pańskiego, Solidarności 67, 03-401 Warsaw, Poland
ORCID ID: 0009-0008-7263-1545

Julia Maria Kostro
Stefan Żeromski Specialist Hospital, Osiedle na Skarpie 66, 31-913 Kraków, Poland
ORCID ID: 0009-0006-3455-2825

Gabriela Makulec
Wojskowy Instytut Medyczny, Szaserów 128, 04-141 Warsaw, Poland
ORCID ID: 0009-0009-1357-1340

Karolina Domosud
Wojskowy Instytut Medyczny, Szaserów 128, 04-141 Warsaw, Poland
ORCID ID: 0009-0007-4345-2188

Anna Libera
Wojewódzki Szpital Zespolony w Kielcach, Grunwaldzka 45, 25-736 Kielce, Poland
ORCID ID: 0009-0000-6806-3014

Karolina Bartkiewicz
Medical University of Warsaw (WUM), 61 Żwirki i Wigury Street, 02-091 Warsaw, Poland
ORCID ID: 0009-0009-3082-7272

Lizaveta Novik
Medical University of Warsaw (WUM), 61 Żwirki i Wigury Street, 02-091 Warsaw, Poland
ORCID ID: 0009-0000-2181-2284

ABSTRACT

Background: Chronic kidney disease (CKD) affects approximately 10% of the population and is associated with increased cardiovascular risk and mortality. In advanced stages, protein-energy wasting (PEW) and malnutrition-inflammation complex syndrome (MICS) are common and worsen clinical outcomes. Evidence suggests that nutritional interventions, including modification of protein, sodium, potassium, and phosphorus intake, and selected supplementation, may slow CKD progression and reduce metabolic complications.
Aim: This review summarizes current evidence on nutritional interventions and supplementation in CKD, considering disease stage and treatment modality, and identifies gaps requiring further research.
Materials and methods: A narrative review of international guidelines (KDIGO, KDOQI, ESPEN) and clinical trials and meta-analyses published between 2009-2026, identified through searches of PubMed, Cochrane Library, Scopus, and Google Scholar, focusing on CKD stages G3-G5.
Results: Individualized protein restriction, sodium limitation, and qualitative phosphorus control improve metabolic parameters, blood pressure and albuminuria. Low- and very low-protein diets, particularly with amino acid ketoanalogue supplementation, are associated with slower CKD progression and a delayed initiation of renal replacement therapy. Plant-based diets may additionally improve acid-base balance, phosphate metabolism, and inflammation. The strongest evidence for supplementation concerns vitamin D, iron, and amino acid ketoanalogues, while data on omega-3 fatty acids, probiotics, and B vitamins remain inconclusive.
Conclusions: Nutritional management is a key component of CKD care. Individualized dietary strategies and targeted supplementation may provide nephroprotective and metabolic benefits, although further high-quality randomized trials are needed to confirm effects on hard clinical endpoints.

KEYWORDS

Chronic Kidney Disease, Nutrition Therapy, Low-Protein Diet, Plant-Based Diet, Ketoanalogues

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Introduction and aim

Chronic kidney disease (CKD) is a global public health problem, affecting approximately 10% of the population. [1] The progressive deterioration of kidney function negatively affects the body's homeostasis, disrupting water and electrolyte, acid-base balance, phosphate and calcium metabolism, and nutritional balance. The most common cause of death in patients with CKD is cardiovascular complications, which are exacerbated by chronic inflammation, protein-energy malnutrition, and other metabolic disorders. [2]

A significant proportion of patients in the advanced stages of the disease develop protein-energy wasting (PEW) and malnutrition-inflammation complex syndrome (MICS), which are both independently associated with increased mortality. [3] Given this information, nutrition therapy and appropriate supplementation are key nephroprotective measures, along with pharmacotherapy and the control of modifiable risk factors such as hypertension, glycemia, albuminuria, and body weight.

In recent years, an increasing amount of data from clinical trials and systematic reviews have pointed to the beneficial effects of nutritional interventions in slowing the progression of CKD and reducing metabolic complications. These strategies include controlling the quantity and quality of protein, sodium, phosphorus, and potassium intake, as well as using plant-based diets. At the same time, there is growing interest in the role of supplementation with vitamin D, iron, omega-3 fatty acids, ketoanalogues, and probiotics in patients with CKD.

The aim of this publication is to provide a concise and evidence-based discussion of the principles of nutrition and supplementation in patients with chronic kidney disease, taking into account differences

depending on the stage of the disease and the method of treatment. Both the strengths of the recommendations and areas where the scientific data remain inconclusive are presented to help physicians make informed, individualized therapeutic decisions in their daily practice.

Methodology

This paper is a narrative review of the literature on nutrition and supplementation in CKD. It discusses current nephrology guidelines (KDIGO, KDOQI) and nutrition guidelines (ESPEN), as well as the latest clinical studies and meta-analyses from the period 2009-2026. The available literature was searched in the PubMed, Cochrane Library, Google Scholar, and Scopus databases. The studies included patients with CKD stages G3-G5 and addressed issues related to diet therapy, metabolism, pathophysiology, the role of clinical nutrition, and supplementation. The results were organized by topic and presented in four sections: (1) pathophysiology of nutritional disorders in CKD, (2) nutrition guidelines for CKD G3-G5ND, (3) nutrition guidelines for patients with end-stage renal disease (ESRD G5D) and (4) the role of supplementation in CKD.

Description of the state of knowledge

1. Pathophysiology of nutritional disorders in CKD

Progressive renal impairment causes a number of metabolic, inflammatory, and hormonal changes that underlie the development of PEW, mineral metabolism disorders, and skeletal muscle dysfunction. A key element of rational dietary intervention planning is understanding the pathophysiological mechanisms occurring in CKD. [2]

PEW is a condition characterized by the loss of the body's protein and energy reserves. It is commonly observed in people with CKD. According to a 2018 meta-analysis, the prevalence of PEW in patients with CKD stages G3-G5 ranges from 11% to 54%, while in the dialysis group, values corresponding to the 25th-75th percentile range from 28-54%. The heterogeneity of the study results is mainly due to differences in the clinical context and regional conditions. [4] The main mechanisms of PEW development include: uremic anorexia, associated, among others, with the accumulation of uremic toxins, resistance to anabolic hormones (e.g., insulin or IGF-1), chronic inflammation, and increased oxidative stress. [5] PEW is a strong, independent predictor of increased mortality and cardiovascular risk. [6] Combined with persistent inflammation, it can lead to the development of MICS, which is associated with a reduced quality of life and an increased risk of anemia. This syndrome is also considered a key mechanism underlying the phenomenon of reverse epidemiology, in which parameters usually interpreted as beneficial, such as low BMI, blood pressure, and cholesterol levels, paradoxically correlate with a worse prognosis due to malnutrition and inflammation. [7]

Low-grade inflammation is sustained by the accumulation of uremic toxins (including p-cresyl sulfate and indoxyl sulfate), intestinal microbiota dysbiosis, and chronic oxidative stress. [8] Disruption of the intestinal barrier integrity causes the translocation of lipopolysaccharides and uremic toxins of bacterial origin, which in turn leads to the overproduction of pro-inflammatory cytokines (IL-6, TNF-α). These cytokines directly intensify protein breakdown by activating the ubiquitin-proteasome pathway. This results in a negative nitrogen balance and the development of sarcopenia. [9, 10] An additional mechanism that self-perpetuates MICS is the disruption of the gut-kidney axis, whereby increased production of uremic toxins exacerbates inflammation. [11]

Chronic metabolic acidosis is another factor that exacerbates catabolic processes in patients with CKD. As a result of the loss of the kidneys' ability to excrete hydrogen ions, bicarbonate concentrations decrease, triggering systemic buffering processes that cause, among other things, the release of amino acids from muscle proteins. Even mild acidosis intensifies protein breakdown, promotes a negative nitrogen balance, and accelerates the decline in estimated glomerular filtration rate (eGFR). These mechanisms include, among others, the activation of cortisol-dependent proteolysis and the buffering of hydrogen ions through increased release of amino acids from muscles. [12]

Another condition closely related to diet is chronic kidney disease-mineral and bone disorder (CKD-MBD). Even in the early stages of CKD, there is an increase in the concentration of fibroblast growth factor 23 (FGF23), which stimulates phosphaturia and inhibits calcitriol synthesis. Increased production of this factor initially serves an adaptive function, allowing optimal serum phosphate levels to be maintained. In advanced stages, when the number of active nephrons is insufficient for effective excretion of phosphate in the urine, there is an extreme increase in FGF23. In addition, increased FGF23 concentration is associated with adverse effects on the cardiovascular system, such as left ventricular hypertrophy and increased vascular stiffness, as well as increased mortality in patients with CKD. Impaired

calcitriol synthesis and phosphate accumulation directly contribute to the development of secondary hyperparathyroidism, exacerbating bone disorders and contributing to vascular calcification. [13]

Nutrition is a targeted strategy aimed at reducing the metabolic burden on the kidneys, reducing inflammation, and preventing PEW/MICS, which directly translates into improved quality of life and prognosis for patients. Dietary interventions include controlling protein, phosphorus, sodium, and potassium intake, balancing metabolic acidosis, and optimizing protein quality (e.g., the role of plant-based diets and ketoanalogues).

2. Nutrition guidelines for CKD G3-G5ND

Nutritional management in patients with CKD stages G3-G5 not undergoing dialysis therapy aims to slow disease progression by reducing the burden on the kidneys, maintaining adequate nutrition, and preventing metabolic complications such as metabolic acidosis, hyperkalemia, hyperphosphatemia, and the development of PEW. According to the current KDIGO (Kidney Disease: Improving Global Outcomes) and KDOQI (Kidney Disease Outcomes Quality Initiative) guidelines, a key element of intervention is the individualization of dietary recommendations, taking into account the stage of the disease, the rate of eGFR decline, nutritional status, level of physical activity, comorbidities, and the patient's biochemical profile. [2, 14]

2.1.1 Individualized protein intake

Controlling protein intake is a fundamental part of treatment with high nephroprotective potential in patients with CKD G3-G5ND. According to the KDIGO and KDOQI (2020) guidelines, a standard low-protein diet (LPD) with a protein intake of 0.8 g/kg body weight/day is recommended for most patients with an eGFR below 30 ml/min/1.73 m2 (G4-G5ND). [2, 14] Limiting protein intake to this level reduces glomerular hyperfiltration, reduces the production of uremic toxins, and slows the progression of CKD, while maintaining a positive or neutral nitrogen balance. [15] Increased protein intake (>1.3 g/kg body weight/day) should be avoided as it may accelerate the decline in eGFR by increasing hyperfiltration. [3]

In selected clinical situations, especially in rapidly progressive CKD or refractory metabolic acidosis, a very low-protein diet (VLPD) should be considered, in which protein intake is limited to 0.55-60 g/kg body weight/day, or its variant enriched with amino acid ketoanalogs (VLPD + KA), with a further reduction in protein intake to (0.28-0.43 g/kg body weight/day). Results from randomized clinical trials indicate that the VLPD+KA diet may contribute to delaying the initiation of renal replacement therapy, especially in patients with eGFR <20ml/min/1.73 m2, and allows for better correction of metabolic disorders (including metabolic acidosis and hyperphosphatemia). Due to the higher risk of PEW, this dietary approach requires constant supervision by an experienced multidisciplinary nephrology and dietetics team. [16]

2.1.2 Balancing electrolytes and metabolic complications

An important element of nephroprotective therapy is the reduction of sodium intake to 2 g/day, which corresponds to <5g/day of table salt. [3] Numerous meta-analyses confirm the nephroprotective effect of this restriction, resulting, among other things, from a reduction in blood pressure, albuminuria, and fluid retention. [17] In addition, it is believed that reducing sodium intake may improve the response to ACE inhibitors and sartans, enhancing their nephroprotective effect. [18] It is also important to limit the consumption of highly processed foods, which are a significant source of sodium in the diet.

Another key element in electrolyte balance is potassium, the intake of which should be individualized and adjusted to the patient's clinical condition. Routine potassium restriction in patients with normokalemia is unjustified, as it may unnecessarily eliminate foods such as vegetables and fruits, which are rich in fiber, alkali, and other nutrients, from the diet. It may be beneficial to educate patients about individual food groups, their potassium content, and ways of preparing food that can reduce the amount of potassium absorbed, such as cooking vegetables in large amounts of water. [19] Only in cases of persistent hyperkalemia (>5.5 mmol/L), after ruling out the effects of drugs that increase blood potassium levels, should potassium intake be limited to <2-3 g/day. [14]

A particularly important element of mineral metabolism control in patients with CKD is monitoring and limiting phosphorus intake. Excessive consumption accelerates the development of disorders characteristic of CKD-MBD (e.g., hyperparathyroidism, bone mineralization disorders, vascular calcification), which in turn affects prognosis and quality of life. [20] Of particular concern are inorganic phosphates with approximately 100% bioavailability, used as additives in processed foods. Organic phosphates, on the other hand, found in natural products, especially plant-based ones, have a much lower bioavailability and should therefore be the preferred source of phosphorus in the diet. [21] It is recommended that daily phosphorus intake be within the range of 800-1000mg, while minimizing inorganic phosphates. [14]

Progressive CKD is associated with a decrease in bicarbonate anions, which is linked to numerous systemic complications. For this reason, numerous studies have been conducted to assess the effect of oral sodium bicarbonate supplementation on the possible improvement of patients' clinical condition or slowing down the progression of the disease. A 2021 meta-analysis summarizing reports from 15 studies investigating the effect of oral NaHCO3 supplementation compared with placebo did not confirm a significant effect on disease progression. [22] At the same time, there are studies on nutritional interventions in which foods with a high acid load were restricted and the supply of products rich in alkaline loads was increased, resulting in an improvement in acid-base balance. The results of these interventions are comparable to oral NaHCO3 supplementation. [23] In clinical practice, it is worth considering a diet rich in plant products, modeled on the Mediterranean diet, in which the supply of alkaline components would be increased, which may be an effective strategy for preventing and alleviating metabolic acidosis in patients with CKD.

2.1.3 The role of a plant-based diet

A 2020 review published in Nature Reviews Nephrology discussed the impact of a plant-based diet on the course of CKD. The authors suggested that increasing the proportion of plant-based foods may reduce the risk of developing metabolic complications and selected symptoms in CKD. [24]

Dietary fiber was attributed important role, citing a meta-analysis of 14 studies, which showed that increased fiber intake reduces urea and creatinine levels, translating into improved eGFR. [24] A study by Stephen and Cummings in 1980 demonstrated that fiber increases the removal of nitrogen compounds, which are substrates for the production of uremic toxins, thereby reducing their production. [25] Fiber, which is supplied in plant products, is fermented by saccharolytic bacteria into short-chain fatty acids, which have anti-inflammatory and immunomodulatory properties and strengthen the intestinal barrier integrity. [26] These mechanisms are an important part of the gut-kidney axis and may contribute to the alleviation of chronic inflammation observed in the course of CKD.

There are studies indicating that choosing plant protein over animal protein may have a beneficial effect on the course of CKD, including by reducing the metabolic burden on the kidneys, reducing the acid load, and reducing the production of uremic toxins. [27] Cohort studies have observed a lower risk of death in patients with reduced eGFR who follow a diet high in plant-based protein, but the exact mechanism underlying this association is not fully understood. [28]

Plant-based products contain more organic than inorganic anions. The most important of these are citrates and malates, which are metabolized into bicarbonates after absorption, promoting alkalization of the body and relieving the kidneys of excess acids. Organic anions mainly occur in the form of potassium salts, so the more potassium-rich a product is, the greater its alkalizing potential. It should be remembered that not all organic acids in plants have such properties. For example, oxalic acid, present in spinach and rhubarb, releases hydrogen ions during metabolism, thus acidifying the body. In addition, oxalates can crystallize in the kidneys, which can cause kidney stones. Another example is benzoic and quinic acid, present in some fruits such as cranberries and plums. It is metabolized by the gut microbiota, resulting in increased acid secretion. [29]

In summary, a diet based mainly on plant products is a promising nutritional strategy that supports the treatment of CKD. The potential benefits of this diet result from its lower acid load, reduced phosphorus bioavailability, and higher fiber content. Although further research is needed, current reports indicate that a properly balanced plant-based diet may slow the progression of metabolic disorders and improve the overall health of patients with CKD.

2.1.4 Energy, carbohydrate, and fat requirements

The energy requirements of patients with CKD depend on their age, metabolic status, physical activity, and nutritional status. According to the KDOQI guidelines (2020), recommended energy intake is approximately 25-35 kcal/kg body weight/day. Calculations should be based on ideal body weight, especially in people with abnormal BMI or clinically significant edema. To avoid catabolism, the main sources of energy should be carbohydrates and fats. This allows restricted protein supply, thus maintaining an adquate nitrogen balance. [14]Due to the high cardiovascular risk in patients with CKD, according to KDOQI guidelines, it is recommended to follow dietary patterns such as the Mediterranean diet, which is rich in unsaturated fats. [14] Nephrology guidelines do not specify the exact proportions of macronutrients in the diet. In clinical practice, it is assumed that carbohydrates (especially complex ones) should provide approx. 50-60% of total energy, while fats should provide approx. 25-35% of daily energy requirements. [30]

2.1.5 Summary of nutritional recommendations for patients with CKD G3-G5ND

The table below summarizes the nutritional recommendations for patients with CKD in this chapter. (Table 1)

Table 1. Summary of nutritional recommendations for patients with CKD G3-G5ND

Parameter	Recommended range
Protein (LPD)	0.8 g/kg bw/day
Protein (VLPD)	0.28-0.43 g/kg bw/day + KA supplementation
Energy requirement	25-35 kcal/kg of body weight/day
Sodium	<2 g/day (<5 g NaCl/day)
Potassium	Individual supply; restriction in hyperkalemia
Phosphorus	800-1000mg/day (preference for sources with low bioavailability)
Bicarbonates	No significant effect from oral supplementation
Fats	25-35% of daily energy requirements (preferably unsaturated fats)
Carbohydrates	50-60% of daily energy requirements (preferably complex carbohydrates)

Source: own study based on KDOQI 2020, KDIGO 2024 guidelines and selected studies
Abbreviations: CKD - chronic kidney disease; LPD - low-protein diet; VLPD - very low-protein diet; KA - ketoanalogues

3. Nutrition guidelines for patients with end-stage renal disease (ESRD G5D)

Patients undergoing hemodialysis (HD) or peritoneal dialysis (PD) are at a significantly higher risk of PEW compared to patients receiving conservative treatment. This is due to increased catabolism, loss of amino acids and small peptides during dialysis, numerous metabolic disorders, and chronic inflammation. [31] Dialysis itself carries additional risks of complications such as hypertension, hypervolemia, and electrolyte disturbances. [32]

The goal of nutritional interventions in this population is to maintain proper nutrition, prevent metabolic complications, and reduce cardiovascular risk. [33] The KDQOI and ESPEN guidelines emphasize the importance of individualizing recommendations by taking into account the type of dialysis, the effectiveness of treatments, energy requirements, current nutritional status, and the presence of complications. Therefore, dietary recommendations for dialysis patients differ from those for patients receiving conservative treatment. [14, 34]

3.1 Individualized protein intake

Dialysis treatment is associated with an increase in catabolic processes in the body. During a single HD session, approximately 9.3-12 g of protein is lost, mainly in the form of amino acids or small peptides. These losses may accelerate the development of hypoproteinemia, which, in combination with chronic inflammation, is an important mechanism leading to the development of PEW. [35]

In response to the higher metabolic requirements of dialysis patients, the 2020 KDOQI guidelines recommend a higher protein intake compared to patients receiving conservative treatment. The recommended daily protein intake is 1.1-1.2 g/kg of body weight. This is sufficient to maintain a neutral nitrogen balance and proper nutrition. [14]

3.2 Energy requirements

According to the current KDOQI 2020 guidelines, the recommended energy intake for CKD patients treated with HD or PD is 25-35 kcal/kg of body weight per day. This level of energy intake ensures adequate nutrition and helps to prevent the development of PEW. [14] In patients treated with PD, special attention should be paid to the additional energy source from the dialysis fluid, which contains glucose, the absorption of which from the peritoneal cavity is approximately 100-300 g/day, corresponding to approximately 400-1200 kcal/day, and should be included in the total dietary energy balance. [36]

3.3 Balance in oral fluid and sodium intake

One of the key elements in the management of dialysis patients is the control of water and electrolyte balance. Loss of kidney function significantly reduces the ability to excrete sodium, promoting sodium retention and increasing plasma osmolality. This directly stimulates the thirst center, leading to polydipsia and, consequently, increased interdialytic weight gain (IDWG). [37]

An important element of nutritional management in dialysis patients individualized fluid intake control, taking into account residual diuresis, IDWG values, and cardiovascular load. According to the recommendations of the National Kidney Foundation, patients without residual diuresis should have fluid intake limited to approximately 700-950 ml per day, while patients with vpreserved residual diuresis may tolerate higher fluid intake. [38]

The IDWG value should be less than 4.0-4.5% of dry body weight. [39] Excessive IDWG leads to the development of hypervolemia, which is associated with volume overload of the cardiovascular system and an increased risk of complications such as left ventricular hypertrophy and congestive heart failure. [40] Therefore, a key element of therapy is education on sodium restriction, including awareness of hidden sources of sodium (e.g., in medications or food), cognitive-behavioral interventions, and awareness that the dialysis fluid itself is a factor affecting total sodium intake. [41, 42, 43]

According to KDIGO guidelines, based on World Health Organization (WHO) recommendations, sodium intake should be limited to 2 g/day. [3] Numerous studies indicate that a low-sodium diet lowers blood pressure and albuminuria. [44] At the same time, the KDQOI 2020 guidelines emphasize the role of sodium restriction in controlling fluid balance and achieving optimal IWG. [14]

3.4 Risk of hyperkalemia in dialysis patients

The limited ability of the kidneys to excrete potassium can potentially lead to potassium accumulation between dialysis sessions. The risk of developing hyperkalemia is particularly high in hemodialysis patients, therefore regular monitoring of potassium levels and adjustment of dietary recommendations is essential in this population. [45]

In patients with pre-dialysis or inter-dialysis hyperkalemia, measures to reduce blood potassium levels should be considered, including: limiting the consumption of foods high in potassium, increasing the frequency of dialysis treatment, or using oral potassium-binding resins as part of pharmacological therapy. [46, 47, 48]

3.5 Phosphate metabolism in hemodialysis patients

Hyperphosphatemia remains one of the most significant metabolic problems in hemodialysis patients due to impaired removal of phosphorus during standard dialysis sessions. [49] Phosphate metabolism disorders in this population are strongly associated with the progression of bone changes, vascular calcification, and increased mortality. In patients undergoing HD, it is often necessary to use phosphate-binding drugs to maintain target serum phosphate levels, even though limiting inorganic phosphate and favoring plant sources remains the basis of dietary management. [50] The 2020 KDOQI guidelines emphasize the role of optimizing dialysis parameters along with dietary control and pharmacological intervention in the effective management of hyperphosphatemia in hemodialysis patients. [14]

3.6 Acid-base balance in patients with CKD G5D

Acid-base balance remains an important issue in the dialysis patient population. However, routine oral bicarbonate supplementation is usually not necessary, as metabolic acidosis is typically effectively corrected during dialysis treatments. In cases of persistent metabolic acidosis, consideration should be given to increasing dialysis frequency. [47]

3.7 Summary of nutritional recommendations for patients with end-stage renal disease (ESRD G5D)

The table below (Table 2) summarizes the key nutritional recommendations for patients with ESRD G5, taking into account the differences between patients receiving conservative treatment and those undergoing dialysis.

Table 2. Nutritional recommendations for patients with ESRD: G3-5ND vs G5D

Nutritional parameter	CKD G3-G5ND	CKD G5D
Protein	LPD - 0.8 g/kg bw/d VLPD - 0.28-0.43 g/kg/d + KA supplementation	1.1-1.2 g/kg bw/d
Energy requirement	25-35 kcal/kg bw/d	25-35 kcal/kg/d; taking into account the clinical condition and glucose absorption from dialysis fluid in patients treated with PD
Potassium	Avoid hyperkalemia; routinely no restriction necessary as long as serum potassium concentration remains normal	In hemodialysis patients - strict control of potassium intake
Phosphorus	800-1000 mg/day	800-1000 mg/d; if necessary, consider phosphate-binding drugs
Sodium	<2 g/d	<2g/d and nutritional education on sodium intake control
Bicarbonates	No clear evidence of significant effect from oral supplementation; consider a plant-based diet	Routine correction of acidosis during dialysis

Source: own study based on KDOQI 2020, KDIGO 2024 guidelines and selected studies
Abbreviations: CKD - chronic kidney disease; LPD - low-protein diet; VLPD - very low-protein diet; KA - ketoanalogues

4. The role of the supplementation in CKD

Supplementation is an important part of the therapeutic management of patients with CKD, including both those receiving conservative treatment and those undergoing dialysis. Due to dietary restrictions, metabolic disorders, chronic inflammation, and nutrient losses during dialysis, this population is at an increased risk of deficiencies in certain vitamins and minerals. At the same time, scientific data indicate variable quality and strength of evidence for specific supplementation interventions. Decisions about supplementation should be made on an individual basis, based on clinical assessment, laboratory findings and current guideline-based recommendations. The following chapter discusses key areas of supplementation with potential and well-documented clinical significance in patients with CKD.

4.1 Vitamin D

Vitamin D deficiency or suboptimal levels are highly prevalent among patients with CKD. A prospective study conducted at Seoul National University Boramae Medical Center evaluated vitamin D levels in CKD patients receiving conservative treatment. The prevalence of vitamin D deficiency was 40.7% in stage G3, 61.5% in stage G4, and 85.7% in stage G5, demonstrating clear stage-dependent increase in deficiency rates. [51]

The increased risk of vitamin D deficiency or suboptimal levels in patients with CKD is multifactorial and related, among others, to advanced age, coexisting diabetes mellitus and obesity, loss of vitamin D-binding proteins during dialysis, impaired tubular reabsorption of 25-hydroxyvitamin D (25-[OH]D), and dietary restrictions. Vitamin D deficiency is defined as a serum 25(OH)D concentration <20ng/mL, suboptimal vitamin D concentration as 20-29 ng/mL, while concentrations >30 ng/mL are considered normal. [14]

Several studies suggest the potential superiority of cholecalciferol supplementation over ergocalciferol in the general population, however, the KDQOI 2020 guidelines recommend both forms of vitamin D supplementation in patients witch CKD. [52, 14] A review of nine randomized controlled trials evaluating vitamin D2 or D3 supplementation in patients with CKD stages G3-G4 demonstrated a significant increase in serum 25(OH)D concentrations. The supplementation regimens included, doses ranging from 2,000-4,000 IU/ day or 40,000-50,000 IU/week for a period of 1-12 months. [53]

For the treatment of vitamin D deficiency in the general population, the Endocrine Society recommends a dose of 50,000 IU/week or 6,000 IU/day for 8 weeks, followed by maintenance therapy of 1,500-2,000 IU/day. [54] No CKD-specifiic dosing guidelines for native vitamin D have been established. Therefore, it is generally recommended to use the same regimens as in the general population, with the possibility of a more intensive or prolonged supplementation in selected cases, based on clinical judgement and biochemical monitoring. [14]

Observational studies suggest an association between low 25(OH)D concentrations and a higher prevalence of albuminuria in patients with CKD; however the results of three small randomized clinical trials assessing the effect of vitamin D supplementation on proteinuria remain inconclusive. [14]

4.2 Iron and renal anemia in CKD

Anemia is a frequent and clinically relevant complication of CKD. A study of the U.S. adult population with stage G3-G5 CKD showed that approximately 25% of patients are affected by anemia. [55] The main mechanisms leading to the development of renal anemia include reduced erythropoietin production, chronic low-grade inflammation, impaired iron absorption from the gastrointestinal tract, and insufficient dietary iron intake. [56]

Patients undergoing hemodialysis are at particularly high risk of iron deficiency. In this population, increased iron loss is associated with, among other factors, frequent blood sampling for testing, gastrointestinal bleeding, and direct blood loss during the hemodialysis procedure, including blood loss in the dialyzer, from arteriovenous fistulas, and from vascular catheters. [56] Reduced appetite, malnutrition, and protein restriction are further contribute to the development of absolute iron deficiency in this group. [57]

In the setting of chronic inflammation in patients with CKD, there is an increased concentration of hepcidin, which impairs iron absorption from the intestines. [58] In addition, medications commonly used in patients, such as proton pump inhibitors and calcium-based phosphate binders, further impair iron absorption. As a result, even adequate dietary iron intake or oral supplementation may be insufficient to meet erythropoietic demands. [56]

The assessment of iron metabolism in patients with CKD is primarily based on measurements of serum ferritin and transferrin saturation (TSAT); however, both parameters have significant diagnostic limitations. Ferritin is an acute-phase reactant, while TSAT is highly variable and may accurately reflect iron availability for erythropoiesis, particularly in the presence of inflammation. [56] Due to chronic inflammation, the diagnostic thresholds for absolute iron deficiency in CKD are higher than in the general population. Absolute iron deficiency is usually defined as serum ferritin <100-200ug/l in non-dialysis and dialysis patients, with TSAT <20% considered an indicator of deficiency, although higher values do not exclude iron-restricted erythropoiesis. [58]

Despite apparently adequate iron stores and circulating iron concentrations, iron availability for erythropoiesis may remain insufficient. This phenomenon is referred to as functional iron deficiency and may result from intensive stimulation of erythropoiesis with erythropoiesis-stimulating agents (ESAs) and impaired iron release from macrophages mediated by inflammation and elevated hepcidin levels. [58]

Iron supplementation plays a key role in the treatment of renal anemia. In many patients, oral iron therapy is ineffective due to impaired absorption and ongoing inflammation. Therefore, in patients undergoing dialysis, intravenous supplementation is the preferred route, as it provides more effective and sustained improvement in hemoglobin levels and TSAT. [14]

At the same time, the potential risks associated with intravenous iron supplementation should not be carefully considered. Evidence from experimental and observational studies suggest possibile associations

with increased oxidative stress, progression of atherosclerosis, higher cardiovascular risk, increased susceptibility to infections, and hypersensitivity reactions. Consequently, decisions regarding iron supplementation, especially intravenous supplementation, should be individualized, balancing expected benefits against potential risks and supported by, regular monitoring of iron metabolism parameters. [58]

4.3 Amino acid ketoanalogues

Amino acid keto analog supplementation represents a specialized element of nutritional component of nutritional management in patients with CKD treated with a low-protein diet. KAs are nitrogen-free precursors od essential amino acids which, after ingestion, undergo transamination using endogenous nitrogen, allowing for amino acid synthesis without increasing the systemic nitrogen load. [59]

The use of KA allows for a significant reduction in dietary protein intake, even to 0.3-0.4 g/kg body weight/day, thereby reducing the generation of urea and other uremic toxins. [59] Evidence from a meta-analysis of randomized controlled trials indicates that KA supplementation combined with a protein-restricted diet may exert nepfroprotectiove effects. Specifically, this strategy has been shown to preserve eGFR, suggesting a potential role in slowing the CKD progression and delaying the initiation of renal replacement therapy. Concomitantly, significant improvements in mineral metabolism parameters have been reported, including a decrease in phosphorus concentration and an increase in serum calcium concentration. One proposed mechanism underlying likely these effects is the fact that KA are administered as calcium salts, which may exert phosphate-binding properties in gastrointestinal tract.

The beneficial effects of KA supplementation also included improved nutritional status, manifested by an increase in serum albumin concentration and an increase in the thickness of the triceps skin fold, especially in patients with a baseline eGFR ≥20 ml/min/1.73 m2. [60]

Current KDOQI guidelines emphasize that KA can be incorporated into comprehensive nutritional therapy for patients with CKD, provided that dietary interventions are individualized and that metabolic, biochemical, and nutritional parameters are regularly monitored. [14]

4.4 Long-chain n-3 fatty acids (LC n-3 PUFA)

Patients with CKD tend to have lower concentrations of long-chain n-3 polyunsaturated fatty acids (LC n-3 PUFA) compared withe the general population, which has provided the rationale for investigating the potential role of supplementation. [61] LC n-3 PUFA have been evaluated as a potential adjunctive intervention for modifying cardiovascular risk, lipid abnormalities, CKD progression, vascular access patency, and inflammatory parameters. [14]

According to current KDOQI guidelines, routine supplementation with LC n-3 PUFA to reduce all-cause mortality or the risk of cardiovascular events is not recommended in adult dialysis patients (HD and PD) and after kidney transplantation. [14] Despite individual randomized controlled trials suggesting a possible reduction in cardiovascular events in hemodialysis patients receiving high doses of fish oil, this effect has not been replicated in meta-analyses and has not translated into a clear reduction in cardiovascular events. [62, 14]

Available data from six randomized controlled trials also indicate that LC n-3 PUFA supplementation does not significantly influence CKD progression, based on the absence of significant changes in eGFR or serum creatinine in non-dialysis, dialysis, and kidney transplant patients. Overall, the results of the studies remain heterogenous, and meta analyses do not confirm the nephroprotective effect of n-3 acids. [14]

The best-documented potential benefit of LC n-3 PUFA supplementation in patients with CKD relates to lipid metabolism. Meta-analyses of randomized clinical trials have demonstrated that supplementation at doses of approximately 1.3-4 g/day in dialysis patients and approximately 2g/day in patients with CKD stages G3-G5may result in a moderate reduction in serum triglyceride concentrations. The effects on total cholesterol and LDL cholesterol are small and of limited clinical relevance, whereas a modest increase in HDL concentration has been reported, albeit with substantial inter-study heterogeneity. [14]

However, no consistent effect of LC n-3 PUFA supplementation on reducing the circulatory inflammatory markers in the blood, such as C-reactive protein or interleukin-6, has been demonstrated. [14] Apart from isolated observations of limited clinical significance, supplementation has not been shown to exert a meaningful effect on blood pressure. [63, 14] Similarly, the routine use of fish oil to improve the patency of arteriovenous fistula or vascular graft potency is not recommended. [14]

The purpose of LC n-3 PUFA supplementation should be clearly defined. If the aim is to increase EPA and DHA concentrations, the most effective method is to use capsule preparations or consumption of fatty sea

fish. Plant preparations mainly increase the supply of alpha-linolenic acid. [14] The quality of available commercial preparations is difficult to assess, which makes precise dosing difficult. [14, 64]

In summary, LC n-3 PUFA supplementation in patients with CKD should not be used routinely, and its consideration may be justified mainly in the treatment of hypertriglyceridemia, with individual assessment of the benefits and risks and taking into account the patient's preferences and financial capabilities. [14]

4.5 Probiotics

The chapter on nutritional pathophysiology in CKD describes the role of chronic low-grade inflammation, sustained, among other mechanisms, by the accumulation of uremic toxins resulting from intestinal dysbiosis. Against this background, interest has emerged in the possibility of modulating the gut-kidney axis through probiotic supplementation as a supportive therapeutic strategy in CKD.

In 2024, West China Hospital of Sichuan University published a systematic review and meta-analysis evaluating the effectiveness of probiotic supplementation in patients with CKD. Analysis of studies assessing the effect of probiotics on gut-derived uremic toxins, including indoxyl sulfate and p-cresyl sulfate, showed no significant differences between the study group and the control group. [65]

With regard to renal parameters, a meta-analysis of 12 studies involving a total of 527 patients demonstrated a significant reduction in blood urea nitrogen (BUN) levels in individuals receiving probiotics compared to the control. The greatest effect was observed in the Asian population and in interventions lasting longer than 3 months. In contrast, an analysis of 11 studies evaluating serum creatinine levels showed no significant differences between the groups. [65]

Regarding inflammatory parameters, 10 studies including 356 patients evaluated the impact of probiotic supplementation on C-reactive protein (CRP) levels. A significant reduction in CRP was observed in patients receiving probiotics compared to the control group; however this effect was primarily limited to non-dialysed CKD patients. No significant changes in CRP levels were found in the dialysis subgroup. [65]

In summary, current evidence suggest that probiotic supplementation may have a beneficial effect on selected inflammatory and metabolic parameters in patients with CKD, but its effectiveness in reducing key uremic toxins or improving renal function has not been confirmed. Due to the limited quality of available data, the role of probiotics in the treatment of CKD remains unclear and requires further well-designed randomized trials

4.6 B vitamins

B vitamins play a central role in energy metabolism, redox processes, nervous system function, and erythropoiesis. [66] The risk of deficiency is increased in patients with CKD, particularly those undergoing hemodialysis. This results from losses associated with the dialysis procedure, as well as reduced dietary intake, intestinal malabsorption, and altered vitamin metabolism. [67]

Deficiencies in water-soluble vitamins often remain undiagnosed because, apart from vitamin B12 and folic acid, their concentrations are not routinely assessed in clinical practice. A two-center cross-sectional study involving 142 hemodialysis patients and 31 healthy controls demonstrated significantly lower concentrations of vitamins B1, B6, and B12 in the hemodialysis group compared to the control group. In addition, vitamin B1 and B2 concentrations showed a negative correlation with BUN, while vitamins B3, B12, and vitamin C correlated positively with albumin concentration, which may suggest a relationship between nutritional status and the levels of these micronutrients. Notably, significantly lower vitamin B1 concentrations were observed in patients with diabetes, which is of particular clinical relevance in light of reported cases of Wernicke's encephalopathy in hemodialysis patients with diabetic nephropathy described in the literature. [67]

In advanced stages of CKD, hyperhomocysteinemia is a highly prevalent metabolic abnormality, resulting from impaired renal clearance of homocysteine and vitamin B metabolism disorders. [68] Although supplementation with folic acid, vitamin B6, and vitamin B12 effectively reduces its concentration in the blood, randomized trials have not shown clear benefits in terms of reducing cardiovascular risk or all-cause mortality. Consequently, current clinical guidelines do not recommend routine B vitamins supplementation solely for the treatment of hyperhomocysteinemia. [14]

Supplementation with folic acid, vitamin B12, or combined B vitamin preparations remains justified only in cases of clinically or laboratory-confirmed deficiencies. However, special caution is required when administering high doses of folic acid, as it may mask the symptoms of vitamin B12 deficiency, potentially delaying the diagnosis of irreversible neurological disorders. This issue is particularly relevant in older adults, who represent a substantial proportion of the dialysis population. [14]

In summary, B vitamin supplementation in patients with CKD should be individualized and targeted toward documented deficiencies, especially in hemodialysis patients and individuals with diabetes. Despite the demonstrated effects on selected biochemical parameters, there is currently insufficient evidence to justify the routine use of these supplements to improve hard clinical endpoints. [14]

4.7 Summary of supplementation in CKD

The table below (Table 3) summarizes the current evidence-based recommendations for supplementation in patients with CKD.

Table 3. Current recommendations for supplementation in patients with CKD

Supplement	Current recommendations
Vitamin D	Recommended supplementation in case of deficiency, in accordance with current guidelines
Iron	Supplementation in accordance with laboratory criteria and nephrological guidelines
Amino acid ketoanalogs	May be used in selected patients with CKD on a VLPD diet, provided that nutritional status and metabolic parameters are closely monitored
Long-chain n-3 fatty acids	No indications for routine supplementation; to be considered for the treatment of hypertriglyceridemia
Probiotics	Insufficient evidence for routine use
B vitamins	No indications for routine supplementation; recommended only in cases of confirmed deficiencies

Source: own study based on KDOQI 2020 guidelines and selected studies Abbreviations: CKD - chronic kidney disease; VLPD - very low protein diet

Conclusions

Available studies indicate that appropriately planned nutritional interventions constitute a fundamental component of nephroprotective management in patients with CKD, both those treated conservatively and those undergoing dialysis. Adjustment of protein intake to the stage of CKD remains a key element of dietary management, with low-protein and very low-protein diets, particularly when combined with amino acid ketoanalogue supplementation, contributing to slower disease progression and delaying the initiation of renal replacement therapy in selected patients populations.

Restriction of sodium intake has a well-documented beneficial effect on blood pressure control, albuminuria, and fluid balance. However, routine potassium restriction is not justified in patients with normokalemia and may unnecessarily limit the intake of nutritionally valuable foods. In terms of phosphate metabolism, it is particularly important to limit highly bioavailable inorganic phosphates and favoring natural dietary sources of phosphorus, particularly plant-based ones.

A growing body of evidence suggests that dietary patterns based predominantly on plant-derived products, modeled on the Mediterranean diet, may exert beneficial effects on acid-base balance, phosphate metabolism, and the inflammatory status, while potentially improving cardiovascular prognosis. Choosing plant protein over animal protein may further reduce the metabolic burden on the kidneys, although the mechanisms underlying these associations require further elucidation.

In dialysis patients, the primary goal of nutritional management is to maintain adequate nutritional status and prevent metabolic complications, which requires highly individualized recommendations based on the type and effectiveness of dialysis, nutritional status, and the presence of residual diuresis. The control of hyperphosphatemia remains a major clinical challenge, requiring coordinated dietary, pharmacological, and dialysis management.

With regard to supplementation, the correction of vitamin D and iron deficiencies is of greatest clinical importance, with decisions regarding the route of iron administration requiring individualized risk-benefit assessment. Among the evaluated supplements, amino acid ketoanalogues demonstrate the most consistent evidence supporting a role in slowing the progression of CKD. In contrast, the routine use of omega-3 fatty acids, probiotics, and B vitamins is not currently supported by sufficient evidence demonstrating improvement in hard clinical endpoints and should therefore be considered on an individual basis.

In summary, nutrition and supplementation in CKD require an individualized, guideline-based approach, informedly te quality and strength of available scientific evidence. Despite the expanding body of research, many areas, particularly those related to supplementation, require further well-designed, adequately powered randomized controlled trials to clarify their impact on long-term patient outcomes.

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