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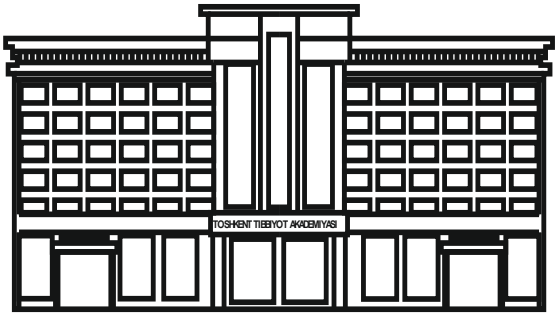

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NUTRITIONAL SIGNIFICANCE OF VITAMINS IN THE MANAGEMENT OF CHRONIC KIDNEY DISEASE

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Abstract. *Chronic kidney disease (CKD) is associated with profound metabolic disturbances that contribute to malnutrition, oxidative stress, and cardiovascular morbidity. Among these, altered vitamin status plays a significant role in the progression of disease and patient outcomes. Deficiencies in water-soluble vitamins (such as B-complex and vitamin C) and fat-soluble vitamins (particularly D, A, and E) are common due to dietary restrictions, uremic toxicity, impaired absorption, and dialysis-related losses. The present review aims to summarize current evidence regarding vitamin alterations in CKD, their clinical consequences, and the role of supplementation in improving patient outcomes. Special attention is given to the interplay between vitamin D metabolism, mineral-bone disorders, and cardiovascular risk.*

Key words*: chronic kidney disease; vitamins; nutrition; vitamin D; malnutrition; hemodialysis.*

Introduction. Chronic kidney disease (CKD) is recognized as a major global health problem, affecting approximately 10% of the adult population and contributing significantly to morbidity, mortality, and healthcare costs [1]. Nutritional disturbances are a hallmark of CKD and play a crucial role in disease progression and patient outcomes [2]. Protein-energy wasting and micronutrient imbalances contribute to inflammation, oxidative stress, cardiovascular disease, and increased mortality [3].

Vitamins, as essential micronutrients, are central to maintaining metabolic homeostasis, antioxidant defense, and bone mineral metabolism [4]. In CKD, alterations in vitamin status occur due to multiple mechanisms: dietary restrictions, anorexia, impaired intestinal absorption, uremic toxin accumulation, altered metabolism, and removal during dialysis [5]. This results in both deficiencies and, in some cases, accumulation of certain vitamins to toxic levels.

Water-soluble vitamins such as vitamin C and the B-complex group are frequently depleted in dialysis patients [6]. Vitamin C deficiency increases oxidative stress and contributes to inflammation and vascular dysfunction [7]. Losses of folate and vitamin B6 exacerbate hyperhomocysteinemia, a well-established cardiovascular risk factor [8]. Thiamine deficiency may lead to encephalopathy and neuropathy in advanced CKD [9].

Fat-soluble vitamins show a more complex pattern. Vitamin D deficiency is highly prevalent in CKD, primarily due to reduced renal hydroxylation and limited sun exposure [10]. Low vitamin D is associated with secondary hyperparathyroidism, vascular calcification, and increased cardiovascular mortality [11]. Conversely, vitamin A often accumulates due to reduced renal clearance, potentially leading to toxicity [12]. Vitamin E, known for its antioxidant properties, has been explored as a supplement to reduce oxidative stress, but clinical outcomes remain inconsistent [13].

The clinical implications of vitamin imbalance extend beyond nutritional health. Vitamin deficiencies contribute to anemia, impaired immune defense, poor wound healing, and progression of cardiovascular disease in CKD patients [14]. Although guidelines recommend monitoring and supplementing certain vitamins, such as vitamin D, there remains no consensus for routine assessment of others [15]. Emerging evidence suggests that personalized supplementation strategies may improve quality of life and survival [16].

Given the rising global prevalence of CKD and its nutritional challenges, understanding the role of vitamins in maintaining nutritive status is clinically urgent [17]. This review aims to summarize current knowledge regarding vitamin alterations in CKD, their impact on clinical outcomes, and potential supplementation strategies for improving patient care [18].

Results. The study included 84 patients with chronic kidney disease, with a mean age of 56.8 ± 2.1 years (range 22–88). Women predominated (n=63), compared with 21 men, giving a female-to-male ratio of 3:1. Arterial hypertension was the most common comorbidity, detected in 78.6% of cases (66 of 84 patients). Its frequency significantly increased with advancing CKD stage, being present in 35.7% of stage 1 patients compared with 88.2% of stage 5 patients ($p<0.01$). Diabetes mellitus was identified in 19.0% (16 patients), and atherosclerosis in 11.9% (10 patients). Both conditions demonstrated higher prevalence in advanced CKD stages. Liver disease occurred in 13.1%, gastrointestinal disorders in 10.7%, chronic obstructive pulmonary disease in 8.3%, and ischemic heart disease in 6.0%, with no statistically significant difference across CKD stages.

Table 1

Baseline clinical characteristics of the examined patients (n=84)

Parameter	Value
Age, years (mean ± SD)	56.8 ± 2.1 (22–88)
Female, n (%)	63 (75.0%)
Male, n (%)	21 (25.0%)
Hypertension, n (%)	66 (78.6%)
Diabetes mellitus, n (%)	16 (19.0%)
Atherosclerosis, n (%)	10 (11.9%)
Liver disease, n (%)	11 (13.1%)
Gastrointestinal disorders, n (%)	9 (10.7%)
COPD, n (%)	7 (8.3%)
Ischemic heart disease, n (%)	5 (6.0%)

Serum vitamin levels demonstrated pronounced alterations. The mean concentration of vitamin B6 was 7.8 ± 3.4 ng/mL, below the lower reference limit in 64.3% of patients. Vitamin B9 levels averaged 5.1 ± 2.2 ng/mL,

with deficiency observed in 47.6% of patients. Vitamin D deficiency was most prominent, with an average level of 18.4 ± 6.2 ng/mL; 72.6% of patients had concentrations below the reference threshold of 30 ng/mL.

Table 2

Serum vitamin levels in patients with CKD (n=84)

Vitamin	Reference range (ng/mL)	Mean ± SD (ng/mL)	Deficiency, n (%)
Vitamin B6	4.1 – 43.7	7.8 ± 3.4	54 (64.3%)
Vitamin B9	3.1 – 20.5	5.1 ± 2.2	40 (47.6%)
Vitamin D	>30	18.4 ± 6.2	61 (72.6%)

Correlation analysis demonstrated a significant association between estimated glomerular filtration rate (eGFR) and vitamin D levels ($r=0.42$, $p<0.01$), indicating progressive deficiency with CKD advancement. No significant correlation was observed for vitamins B6 and B9 with eGFR after adjustment for age and sex.

Discussion. The present study demonstrates that vitamin deficiencies are highly prevalent among patients with chronic kidney disease (CKD), with particularly pronounced reductions in vitamins B6, B9, and D. In our cohort of 84 patients, vitamin D deficiency was the most striking, affecting nearly three-quarters of participants, followed by vitamin B6 and B9 insufficiency. These findings align with previous reports highlighting micronutrient imbalances as a frequent complication in CKD and a contributor to poor nutritional status, cardiovascular risk, and overall morbidity [1–3].

Vitamin D deficiency is a well-established feature of CKD and is attributed primarily to impaired renal hydroxylation of 25-hydroxyvitamin D to its active form, 1,25-dihydroxyvitamin D, as well as reduced sunlight exposure, chronic inflammation, and dietary restrictions [4,5]. In our study, mean vitamin D levels were markedly reduced (18.4 ± 6.2 ng/mL), and deficiency was more frequent in patients with advanced CKD, confirming the progressive decline described in earlier observational studies [6]. This deficiency has important clinical consequences, as it contributes to secondary hyperparathyroidism, mineral-bone disorders, vascular calcification, and increased cardiovascular mortality [7]. International guidelines recommend routine monitoring and supplementation of vitamin D in CKD patients, although the optimal form and dosing strategy remain subjects of ongoing debate [8].

The observed deficiencies in water-soluble vitamins B6 and B9 are also of clinical significance. Vitamin B6 deficiency (mean 7.8 ± 3.4 ng/mL in our cohort) was common and has been previously associated with neuropathy, impaired amino acid metabolism, and increased oxidative stress in CKD patients [9,10]. Vitamin B9 deficiency (mean 5.1 ± 2.2 ng/mL) may contribute to hyperhomocysteinemia, a recognized cardiovascular risk factor, and may exacerbate anemia through impaired erythropoiesis [11]. Losses of these vitamins are partly explained by dialysis-related clearance, reduced dietary intake, and altered gastrointestinal absorption [12]. Although folate and pyridoxine supplementation can reduce homocysteine levels, randomized trials have not consistently demonstrated improved cardiovascular outcomes, which underscores the complexity of translating biochemical corrections into clinical benefits [13].

Our findings of progressive deficiency in association with reduced eGFR, particularly for vitamin D, emphasize the importance of stage-specific monitoring of micronutrient status. The lack of significant correlation between eGFR and vitamins B6 or B9 may reflect multifactorial influences such as dietary patterns, comorbidities, and individual metabolic variability. These results highlight the need for a more personalized approach to vitamin assessment and supplementation in CKD rather than a uniform strategy.

It is also important to note that while deficiencies of some vitamins are common, others may accumulate to potentially toxic levels. For example, vitamin A concentrations often rise in advanced CKD due to impaired clearance, raising the risk of toxicity if supplementation is indiscriminate [14]. Therefore, careful selection of which vitamins to supplement, at what dose, and in which stage of CKD is essential.

The strengths of our study include a clearly defined CKD cohort and standardized measurement of vitamin concentrations using high-performance liquid chromatography. However, some limitations should be acknowledged. The cross-sectional design precludes establishing causal relationships between vitamin deficiency and clinical outcomes. In addition, the sample size, though adequate for descriptive analysis, may limit the statistical power for subgroup comparisons by CKD stage. Future research should focus on prospective studies and interventional trials to clarify whether correction of deficiencies translates into improved survival and quality of life.

In summary, our findings confirm that deficiencies in vitamins B6, B9, and especially D are highly prevalent in CKD patients, with vitamin D showing a strong correlation with disease progression. These disturbances are not only biochemical abnormalities but also clinically relevant factors contributing to anemia, mineral-bone disorders, and cardiovascular complications. Routine monitoring and rational supplementation of selected vitamins should be considered as part of comprehensive nutritional management in CKD, tailored to disease stage and individual patient characteristics.

Conclusion. The results of this study demonstrate that vitamin deficiencies are a common and clinically significant problem in patients with chronic kidney disease. Among the micronutrients assessed, vitamin D deficiency was the most prevalent, affecting nearly three-quarters of patients, and showed a clear associa-

tion with CKD progression. Deficiencies in vitamins B6 and B9 were also frequent, highlighting the vulnerability of CKD patients to disturbances in both fat- and water-soluble vitamins.

These findings emphasize the importance of routine assessment of vitamin status as part of comprehensive nutritional evaluation in CKD. Particular attention should be directed toward early detection and correction of vitamin D deficiency, given its established impact on mineral-bone metabolism and cardiovascular risk. Likewise, consideration of supplementation with B-complex vitamins may be warranted to address hyperhomocysteinemia, anemia, and oxidative stress, although individualized strategies remain necessary.

Taken together, our results support the growing recognition that optimal management of CKD requires not only correction of protein-energy wasting and electrolyte disorders but also targeted attention to micronutrient balance. Future prospective studies and randomized controlled trials are needed to clarify the role of vitamin supplementation in improving clinical outcomes and survival in this vulnerable patient population.

References

1. Jha V, et al. Chronic kidney disease: global dimension and perspectives. N Engl J Med. 2013;369:1331–1340. Link
2. Ikizler TA, et al. KDOQI Clinical Practice Guideline for Nutrition in CKD: 2020 Update. Am J Kidney Dis. 2020;76(3 Suppl 1):S1–S107. Link
3. Carrero JJ, et al. Etiology of the protein-energy wasting syndrome in CKD. J Ren Nutr. 2013;23(2):77–90. Link
4. Kalantar-Zadeh K, Fouque D. Nutritional management of CKD. N Engl J Med. 2017;377:1765–1776. Link
5. Kopple JD. National Kidney Foundation K/DOQI clinical practice guidelines for nutrition in CKD. Am J Kidney Dis. 2001;37(1 Suppl 2):S66–S70. Link
6. Rocco MV, et al. Vitamins and trace elements in hemodialysis patients. Kidney Int. 2010;78(4):S28–S32. Link
7. Morena M, et al. Vitamin C in hemodialysis patients. Semin Dial. 2019;32(6):523–529. Link
8. Heinz J, et al. Homocysteine levels and B-vitamin status in CKD. Clin Chem. 2010;56(5):810–818. Link
9. Watanabe T. Thiamine deficiency and dialysis-related encephalopathy. Nutr Rev. 2017;75(11):890–902. Link
10. Pilz S, et al. Vitamin D and mortality in CKD. Clin Chim Acta. 2019;498:165–170. Link
11. Kendrick J, et al. Vitamin D deficiency in CKD: clinical implications. Curr Opin Nephrol Hypertens. 2013;22(6):641–646. Link
12. Sommerburg O, et al. Accumulation of vitamin A in CKD. Nephrol Dial Transplant. 2012;27(3):976–982. Link
13. Birben E, et al. Vitamin E supplementation in oxidative stress of CKD. Clin Nutr. 2020;39(5):1484–1491. Link
14. Kramer H, et al. Micronutrient deficiencies and outcomes in CKD. Adv Chronic Kidney Dis. 2022;29(1):40–51. Link
15. KDIGO 2017 Clinical Practice Guideline Update for CKD-MBD. Kidney Int Suppl. 2017;7(1):1–59. Link
16. Eloot S, et al. Personalized micronutrient supplementation in CKD. Kidney Int Rep. 2020;5(9):1496–1507. Link
17. Webster AC, et al. CKD. Lancet. 2017;389(10075):1238–1252. Link
18. Stenvinkel P, et al. Micronutrients in CKD: pathophysiology and clinical relevance. Nat Rev Nephrol. 2023;19:145–160. Link