Sponsored content: This article was initiated and funded by Alliance Pharma, with medical writing and editorial support provided by HRS, London, UK. Alliance Pharma also had editorial input in the writing of this article. This article is intended for UK Healthcare professionals only. **Prescribing information and Adverse Event reporting can be found at the end of this article.** CN have had no input into the content or reviewing of this article.

Micronutrients in Kidney Care:

Challenges, considerations and clinical strategies

Helena Jackson, Specialist Kidney Dietitian, St George's Hospital, South London

About the author: Helena Jackson BSc (hons), PGDip, MSc, RD is a specialist kidney dietitian at St George's Hospital in South London, with a passion for all things renal! She has more than 25 years' experience in kidney care, including clinical practice, lecturing, writing and contributing to nutrition guidelines. She has recently commenced a National Institute of Health and Care Research funded PhD to codesign malnutrition screening strategies in kidney outpatient services.

The author has received honorarium from Alliance Pharma and has declared no conflicts of interest. The opinions expressed are not necessarily those of Alliance Pharma.

Micronutrient homeostasis is vital for maintaining healthy metabolic function.[1] Impaired kidney function in chronic kidney disease (CKD) impacts micronutrient intake, absorption and excretion.[1] Micronutrient deficiency is a common but under-recognised issue in CKD, often developing insidiously,[1] with a declining micronutrient intake linked to CKD progression.[2] This article explores vitamin and trace element management in CKD and highlights practical strategies for dietitians.

Challenges in kidney care

Many of the symptoms associated with CKD negatively impact micronutrient intake. Anorexia, taste changes, nausea and early satiety are common and contribute towards reduced food intake. Dietary restrictions, for example in potassium and phosphate, may further limit type and amount of food eaten.[1, 3-5]

Those undergoing kidney replacement therapy (KRT) may also experience depletion of water-soluble vitamins (WSV) and trace elements. Malabsorption, metabolic abnormalities and co-morbidities may exacerbate these issues. Polypharmacy risks medication non-adherence and alterations in micronutrient absorption and excretion. Potential drug-nutrient interactions include reduced absorption of micronutrients by phosphate binders.[4, 6, 7]

Conversely, reduced urinary excretion, abnormalities in transport proteins and absorption from dialysis fluid may cause the accumulation of vitamins and trace elements.[1] Therefore, caution is needed to avoid excessive intakes and subsequent toxicity.

Interpreting biochemical data in CKD is challenging due to factors such as altered nutrient distribution across body compartments, changes in protein binding, and altered protein clearance by the kidneys or KRT.[1] Biochemical data should be contextualised with a dietary, medication and clinical assessment.[4] A multidisciplinary team (MDT) approach is essential to consider anaemia management, bone health, dialysis effects, medication interactions, tissue viability, gastrointestinal function and co-morbidities such as diabetes.[4] For those with sustained inadequate diets, micronutrient deficiency should be considered, particularly with advanced CKD and KRT.[4] Micronutrient deficits are also a concern in the growing numbers of patients with CKD receiving surgical and medical obesity interventions.[8, 9]

Vitamins in kidney care

Whilst research demonstrates a prevalence of vitamin deficiencies amongst the CKD population, disordered protein metabolism and reduced renal excretion may mitigate the risk of deficiency in some patients.[6] Notably, a study of hospitalised patients with CKD found that serum levels of nine vitamins were generally within normal ranges, except for vitamin D, which was frequently low.[11] An individualised approach is required as there is limited evidence for routine supplementation and the clinical benefits for kidney, cardiovascular and patient-reported outcomes are uncertain.[6] There is also a risk of excessive intake.[6, 9] In the absence of specific indications, vitamin requirements in CKD are aligned with those of the general population, with individualised adjustments according to the clinical scenario.[4]

Water-soluble vitamins

WSV deficiencies are common in CKD, particularly with losses related to dialysis or haemofiltration, although urinary excretion may also decrease.[12] Anorexia, therapeutic dietary restrictions and other intake barriers risk depleting WSV status.[1, 6] A comprehensive, evidence-based approach to potassium and other restrictions is essential to maintain nutritional adequacy.[4]

Many dialysis services routinely supplement WSV, due to an association with significantly lower mortality risk.[12, 13] Dialysis-specific multivitamins contain WSVs, namely vitamin C and the eight B vitamins (thiamine, riboflavin, niacin, pantothenic acid, pyridoxine, biotin, folic acid, cobalamin), based on expert consensus.[15] Special attention to vitamin C, folate and thiamine is recommended during critical illness or continuous KRT.[14] However, concerns about excess intake and polypharmacy highlight the importance of an individualised assessment and supplement prescription.[4]

Vitamin C

A vitamin C intake of at least 90 mg/d for men and 75 mg/d for women is recommended.[4] A correlation between elevated serum vitamin C levels and decreased CKD risk has been reported, however this may be related to other diet and lifestyle factors.[9] High doses of vitamin C (above 500 mg per day) risk increasing serum oxalate levels, leading to kidney stone formation and nephropathy.[16]

Vitamin B12 and folate

The management of anaemia in CKD includes addressing any B12 or folate deficiencies through individual supplementation or with dialysis multivitamins if applicable.[17] Serum folate levels reflect recent intake and can be used to indicate adherence, whilst red blood cell folate levels are more reflective of longer-term folate status.[18] Shorter red blood cell lifespans and pharmaceutical therapies may deplete stores more rapidly. Excessive folate intakes can reduce zinc absorption and may also mask signs of pernicious anaemia, so closely monitoring serum B12 levels is essential.[4]

Fat-soluble vitamins (FSV)

FSV status in CKD is complex due to altered metabolism, changes in protein binding and reduced excretion. While some FSV may accumulate due to reduced clearance (e.g. vitamin A), others, such as vitamin D, are frequently deficient. Careful assessment is essential, as both deficiency and excess carry clinical risks.

Vitamin D

Vitamin D deficiency is common in patients with CKD, and impaired activation of vitamin D contributes to the development of CKD-related mineral and bone disorders. Supplementation with native vitamin D – ergocalciferol (D2) or cholecalciferol (D3) should be used to correct deficiency.[19, 20] General population strategies are appropriate for many people with CKD, including loading doses where indicated. With advancing CKD and impaired conversion to the active form, higher maintenance doses of at least 1000 IU (25 mcg) daily may be required to achieve the minimum recommended serum 25(OH)D levels of 75 nmol/L.[4, 20] Active vitamin analogues (e.g. calcitriol or alfacalcidol) may be indicated for the management of progressive secondary hyperparathyroidism, provided that vitamin D deficiency and hyperphosphatemia have been addressed and patients are monitored for hypercalcaemia and other side effects.[19, 20]

Vitamin A

Kidney dysfunction is associated with increasing vitamin A levels due to the accumulation of plasma retinol and its binding proteins.[21] Vitamin A should not be routinely supplemented, and people with CKD should avoid doses above general population requirements, unless there is evidence of malabsorption and deficiency.[4, 6] High dose retinol supplements (including fish liver oils) are contraindicated in CKD due to toxicity risks including hypercalcaemia, bone catabolism and hepatotoxicity.[22] Beta-carotene, by contrast, is converted to retinol in the intestinal mucosa as required by the body and is under tight enzymatic regulation, reducing the risk of toxicity.[23]

Vitamins E and K

In a recent US study, higher vitamin E intake was associated with lower prevalence of CKD, possibly due to its protective role against oxidative tissue damage.[24] However, the prevalence of vitamin E deficiency remains unclear. Routine supplementation is not generally recommended, and high doses may inhibit vitamin K and increase bleeding risk.[25] Subclinical vitamin K deficiency is common in CKD, possibly due to dietary restrictions, and has been associated with vascular stiffness, calcification and mortality.[6] Despite this, supplementation does not appear to improve outcomes and should be avoided with vitamin K inhibitors such as warfarin.[4]

Trace elements

Dietary and biochemical assessment of trace element intake is challenging and requirements are unclear.[7] Trace element accumulation and losses may occur with KRT, however this varies by element, modality and by blood and dialysate concentrations.[7] Serum levels are affected by hypoalbuminaemia, other plasma protein abnormalities and reduced renal excretion. For example, in a Canadian study of patients on haemodialysis, few had low zinc, selenium and manganese concentrations, whereas high plasma concentrations of manganese, zinc, selenium and chromium were common.[26] There is increased risk of toxicity with supplementation and current guidelines recommend an individualised approach.[4]

Zinc, selenium and copper

Zinc deficiency is common in CKD and has been linked to renal anaemia, impaired wound healing, altered taste, reduced appetite, oxidative stress, inflammation and cardiovascular disease.[27, 28] However, interpreting the research is challenging, as circulating zinc makes up only 0.1% of total body zinc[27] and can be influenced by CKD and albumin levels.[29] Caution should be exercised with zinc supplementation, as it can interfere with copper absorption in the small intestine.[27] A higher dietary intake of selenium has been associated with a lower risk of CKD in adults,[30] however elevated selenium levels have been correlated with an increased risk of kidney damage, highlighting the complexity of selenium metabolism and supplementation.[31] Losses of copper and zinc have been reported with intermittent and continuous KRT.[30] Hospitalised patients require monitoring of selenium, zinc and copper status due to increased needs and/or losses during acute illness.[27]

Iron

Iron is essential for erythropoiesis, with iron deficiency being common in CKD. Causes include poor gastrointestinal absorption, blood losses, chronic inflammation, use of erythropoietin-stimulating agents and poor clearance of hepcidin – a hormonal regulator of iron homeostasis.[33]

Iron supplementation is considered when serum ferritin is below 500 mcg/L or transferrin saturation is below 30%.[17] Dietetic intervention includes advice on dietary iron, supplementation, symptoms, malnutrition and other consequences of anaemia.[33] Intravenous iron is preferable for those on dialysis and with advanced CKD, as oral iron may have limited absorption and cause gastrointestinal side effects.[33]

Micronutrient management – top tips

1. **Individualised nutritional assessment:** Consider stage of CKD, KRT modality, comorbidities, gastrointestinal function, acute illness, malnutrition risk, medication profile and psychosocial barriers. Assess intake from diet and non-prescribed supplements. Consider micronutrient requirements with nutrition support.
2. **Food first approach:** Aim for a nutritionally adequate 'whole food' approach and regularly review any dietary restrictions (e.g. potassium) that may reduce intakes of other micronutrients (e.g. folic acid, vitamin C, etc.).

3. **Collaborative care:** Work closely with multidisciplinary teams to integrate micronutrient management within the overall treatment plan and to ensure consistent, evidence-based and patient-centred advice.
4. **Patient education:** Empower patients with practical advice on food choices, cooking methods, appropriate supplementation and reliable sources of information.
5. **Regular review:** Revisit micronutrient needs over time, particularly with changes in dialysis modality, biochemistry or dietary intake.
6. **Micronutrient supplementation:** Identify and, where appropriate, correct micronutrient deficiencies such as vitamin D, B vitamins and iron, according to guidelines. Do not routinely supplement with FSVs (A, E and K) and trace elements. Recognise the limitations of assessment methods, be aware of drug-nutrient interactions and toxicity risks. Consider polypharmacy. Recommend dialysis-specific multivitamins or general multivitamin/mineral supplements as clinically indicated.

Conclusion

Micronutrient management in CKD is complex, requiring careful balance to prevent both deficiency and toxicity. An individualised approach to optimal management of vitamins and trace elements is essential (see **Table 1**). Dietitians are a vital component of the MDT, responsible for optimising patients' nutritional status, encouraging a nutritionally adequate diet, and initiating supplementation where appropriate.

Table 1: Micronutrient challenges and dietetic strategies

KEY CHALLENGES	DIETETIC STRATEGY
Dietary restrictions	• Regularly review and avoid unnecessary restrictions • Optimise diet variety and quality • Encourage an appropriate multidisciplinary team approach • Counter nutrition misinformation
Assessment and interpretation of micronutrient levels	• Confirm the reliability and relevance of biochemical data and seek expert advice if required • Align with dietetic and clinical history • Adjust for albumin level if appropriate (e.g. zinc)
CKD-related malnutrition	• Regularly screen for malnutrition and appropriate intervention • Promote micronutrient-dense foods • Consider micronutrients in nutrition support (including contribution from enteral and oral products) • Consider a dialysis-specific WSV or general multivitamin/mineral supplement if sustained inadequate food intake
Dialysis-related losses of water-soluble vitamins (WSV) and trace elements	• Encourage a balanced, wholefood approach with adequate fruit, vegetables and other micronutrient-rich sources • Supplement with dialysis-specific multivitamins if indicated • Consider a general multivitamin and mineral if other micronutrients are indicated
Polypharmacy and potential drug-nutrient interactions	• Check medication history, including non-prescription supplements and remedies • Consider drug-nutrient interactions (e.g. oral iron, phosphate binders, diuretics, Sodium-Glucose Cotransporter-2 inhibitors (SGLT2) inhibitors) • Consider multivitamins/minerals rather than individual or high-dose supplements
Altered metabolism: Vitamin D	• Ensure vitamin D3 repletion before starting vitamin D analogues • Aim for vitamin D3 serum levels of 75 nmol/L
Altered metabolism: Anaemia	• Ensure iron replete (intravenous iron may be required) • Monitor and supplement vitamins B12 and folate as indicated (consider a multivitamin supplement to reduce polypharmacy)
Altered metabolism: Risk of toxicity	• Assess for potentially toxic prescription and non-prescription supplements and educate staff and patients • Caution with trace elements, retinol, vitamins E and K
Malabsorption	• Individualised approach to supplementation, including vitamin D3, other fat-soluble vitamins and trace elements (not contained in renal multivitamins)
Co-morbidities	• Consider implications of other conditions and interventions, including hospitalisation, diabetes, cancer, surgery, wound healing, and obesity interventions

References: **1.** Jankowska M, Rutkowski B, Dębska-Ślizień A (2017). Vitamins and microelement bioavailability in different stages of chronic kidney disease. Nutrients; 9(3): 282. **2.** Lin HI, *et al.* (2022). Associations between dietary patterns and stages of chronic kidney disease. BMC Nephrol.; 23(1): 115. **3.** Elias MA, *et al.* (2025). Lived experiences of people with chronic kidney disease on maintenance dialysis: a systematic review and thematic synthesis of qualitative studies. BMC Nephrol.; 26(1): 22. **4.** Ikizler TA, *et al.* (2020). KDOQI Clinical Practice Guideline for Nutrition in CKD: 2020 Update. Am J Kidney Dis. 2020;76(3 Suppl 1): S1–S107. Erratum in: (2021) Am J Kidney Dis.; 77(2): 308. **5.** Sabatino A, *et al.* (2021). Sarcopenia in chronic kidney disease: what have we learned so far? J Nephrol.; 34(4): 1347–1372. **6.** Chazot C, Steiber A, Kopple JD (2023). Vitamin needs and treatment for chronic kidney disease patients. J Ren Nutr.; 33(6 Suppl): S21–S29. **7.** Filler G, *et al.* (2021). Principles responsible for trace element concentrations in chronic kidney disease. Clin Nephrol; 96(1): 1–16. **8.** Almandoz JP, *et al.* (2024). Nutritional considerations with antiobesity medications. Obesity (Silver Spring).; 32(9): 1613–1631. **9.** Wang C, *et al.* (2024). Serum vitamin C levels and their correlation with chronic kidney disease in adults: a nationwide study. Ren Fail.; 46(1): 2298079. **10.** Ben-Porat T, *et al.* (2019). Nutritional Management for Chronic Kidney Disease Patients who Undergo Bariatric Surgery: A Narrative Review. Adv Nutr.; 10(1): 122-132. **11.** Wang Y, *et al.* (2021). The weak correlation between serum vitamin levels and chronic kidney disease in hospitalized patients: a cross-sectional study. BMC Nephrol.; 22(1): 292. **12.** Fissell RB, *et al.* (2004). International variation in vitamin prescription and association with mortality in the Dialysis Outcomes and Practice Patterns Study (DOPPS). Am J Kidney Dis.; 44(2): 293–299. **13.** Wright M, *et al.* (2019). Clinical practice guideline on undernutrition in chronic kidney disease. BMC Nephrol.; 20(1): 370. **14.** Fah M, *et al.* (2022). Micronutrient deficiencies in critically ill patients receiving continuous renal replacement therapy. Clin Nutr ESPEN.; 50: 247–254. **15.** Swafford C. (2011). A current look at renal multivitamins. J Ren Nutr.; 21(6): e33–e42. **16.** Rathi S, Kern W, Lau K (2007). Vitamin C-induced hyperoxaluria causing reversible tubulointerstitial nephritis and chronic renal failure: a case report. J Med Case Rep.; 1: 155. **17.** Bhandari S, *et al.* (2025). UK Kidney Association clinical practice guideline: update of anaemia of chronic kidney disease. BMC Nephrol.; 26(1): 193. **18.** Green R (2011). Indicators for assessing folate and vitamin B-12 status and for monitoring the efficacy of intervention strategies. Am J Clin Nutr.; 94(2): 666S–672S. **19.** Ketteler M, *et al.* (2017). Executive summary of the 2017 KDIGO Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD) Guideline Update: what's changed and why it matters. Kidney Int.; 92(1): 26–36. **20.** Christodoulou M, Aspray TJ, Schoenmakers I (2021). Vitamin D supplementation for patients with chronic kidney disease: a systematic review and meta-analyses of trials investigating the response to supplementation and an overview of guidelines. Calcif Tissue Int.; 109(2): 157–178. **21.** Jing J, *et al.* (2016). Chronic kidney disease alters vitamin A homeostasis via effects on hepatic RBP4 protein expression and metabolic enzymes. Clin Transl Sci.; 9(4): 207–215. **22.** Borgan SM, Khan LZ, Makin V (2022). Hypercalcemia and vitamin A: a vitamin to keep in mind. Cleveland Clin J Med.; 89(2): 99–105. **23.** Harrison EH (2012). Mechanisms involved in the intestinal absorption of dietary vitamin A and provitamin A carotenoids. Biochim Biophys Acta Mol Cell Biol Lipids.; 1821(1): 70–77. **24.** Li J, *et al.* (2023). Association between dietary vitamin E intake and chronic kidney disease events in US adults: a cross-sectional study from NHANES 2009–2016. Clin Kidney J.; 16(12): 2559–2566. **25.** Booth SL, *et al.* (2004). Effect of vitamin E supplementation on vitamin K status in adults with normal coagulation status. Am J Clin Nutr.; 80(1): 143–148. **26.** Tonelli M, *et al.* (2017). Concentrations of trace elements in hemodialysis patients: a prospective cohort study. Am J Kidney Dis.; 70(5): 696-704. **27.** Fukasawa H, *et al.* (2023). Clinical significance of trace element zinc in patients with chronic kidney disease. J Clin Med.; 12(4): 1667. **28.** Elgenidy A, *et al.* (2023). Serum zinc levels in chronic kidney disease patients, hemodialysis patients, and healthy controls: systematic review and meta-analysis. J Ren Nutr.; 33(1): 103–115. **29.** Tsutsumi R, *et al.* (2014). Albumin-normalized serum zinc: a clinically useful parameter for detecting taste impairment in patients undergoing dialysis. Nutr Res.; 34(1): 11–16. **30.** Weng CO, *et al.* (2019). Micronutrient and amino acid losses during renal replacement therapy for acute kidney injury. Kidney Int Rep.; 4(8): 1094–1108. **31.** Pi Y, *et al.* (2024). Association between dietary intake of selenium and chronic kidney disease in US adults: a cross-sectional study of NHANES 2015–2018. Front Nutr.; 11: 1396470. **32.** Fu S, *et al.* (2022). Effects of selenium on chronic kidney disease: a Mendelian randomization study. Nutrients; 14(21): 4458. **33.** Hain D, *et al.* (2023). Iron-deficiency anemia in CKD: a narrative review for the kidney care team. Kidney Med.; 5(8): 100677.