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Nutritional management in children with chronic kidney disease

Abstract

Chronic kidney disease (CKD) in children disrupts the metabolic environment on which normal growth and development depend, making nutritional care central to management at every stage of the disease. This review synthesises current evidence and clinical practice guidance on nutritional assessment and dietary management in paediatric CKD, covering energy and protein prescription, mineral and vitamin D homeostasis, potassium and sodium/fluid management, and the use of enteral and parenteral nutrition support. Growth failure remains common, particularly in infants and young children, and is driven by a combination of inadequate intake, hormonal disturbance, unnecessary dietary restriction, and the metabolic consequences of uraemia itself. At the same time, obesity and over-nutrition are emerging as a parallel concern in this population. Regular, structured monitoring of dietary intake and growth parameters, individualised nutrient prescription against published paediatric CKD targets, and attention to palatability and the child's own priorities together form the basis of effective nutritional care. Consolidating this evidence into a single practical overview is intended to support clinicians in delivering nutrition that protects growth, limits uremic and metabolic complications, and improves quality of life for children with CKD and their families.

Keywords: chronic kidney disease, paediatric nutrition, growth failure, protein-energy wasting, enteral feeding, mineral bone disease, dietary management

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Introduction

Chronic kidney disease (CKD) has grown noticeably more common among children over the past several years, and the sharpest rise is being seen in communities of lower socioeconomic standing — a pattern that gives cause for concern on several counts.[1] Because childhood growth and development are so tightly bound to nutrition, dietary care sits at the very centre of managing paediatric CKD. A failing kidney unsettles the whole metabolic environment of the child, and it is this disturbed internal milieu that makes feeding these patients such a demanding undertaking.

Microbiome and uremic toxins

Studies in adults with CKD have tied gut-derived metabolites to adverse outcomes. Circulating trimethylamine N-oxide, a product of the choline pathway, climbs as renal function falls and has been linked with higher mortality.[2] A parallel signal has been described in children, in whom elevated serum indoxyl sulphate has been associated with progression of kidney disease.[3] Taken together, these findings raise the prospect that lowering the uremic toxin burden might yield better long-term outcomes in children with CKD, although this remains to be firmly proven.

Clinicians are tasked with safeguarding growth and development in a setting that works against both. No single diet suits every patient, the requirements shift with body weight, with the cause of the kidney disease, and with the stage of CKD. In everyday practice these children are too often subjected to long, poorly justified restrictions rooted in misinformation rather than evidence. A further and frequently underrated hurdle is palatability — a prescription helps only if the child will actually eat it — so making meals appealing is itself part of the therapeutic plan. The goal is not simply to add years to life but to add life to those years. It also bears emphasising that the problem is no longer one-sided: obesity, together with the downstream consequences of unbalanced eating and inactivity, is now a genuine and growing concern in the paediatric CKD population, and any nutritional scheme must reckon with over-nutrition as well as under-nutrition.[4]

Growth failure

Growth retardation is a familiar feature of paediatric CKD and is most striking when the disease declares itself in infancy, a window during which growth is overwhelmingly nutrition-driven. The reasons for growth failure are many and often overlapping: inadequate nutrient intake as a direct consequence of uraemia, hormonal disturbances, indiscriminate dietary restrictions, increasingly propagated through social media and by practitioners of alternative therapy, altered taste, psychological factors, loss of protein and amino acids across the dialysis membrane, recurrent infection and inflammation, the carrying-over of pre-dialysis restrictions into later stages, circulating growth inhibitors, and renal bone disease.

Protecting the nutritional status of these children is therefore of the first importance. As the glomerular filtration rate (GFR) falls, progressive uraemia brings anorexia, nausea and vomiting, and food intake dwindles. Left unchecked, this culminates in muscle breakdown, loss of body mass and fat, and weight loss — the clinical picture of combined malnutrition and inflammation.

Systematic, repeated assessment of nutritional status, paired with the delivery of adequate nutrition, forms the backbone of nutritional care in paediatric CKD. Growth velocity typically begins to slow once GFR drops below 25 mL/min/1.73 m^2.[5] The Kidney Disease: Improving Global Outcomes (KDIGO) initiative, along with the earlier KDOQI work, has issued dedicated guidance for nutrition in paediatric CKD.[6] The stakes are highest in the very young: in infants under two years, height standard deviation score (SDS) can fall by as much as 2 SD within the first six months of life when nutritional intake is not closely monitored and supported.[7]

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Much of what is known about growth in CKD comes from children on dialysis or under follow-up after transplantation. The traditional emphasis of nutritional management in children with reduced kidney function has been to head off protein-energy malnutrition (PEM) and to satisfy vitamin and mineral needs — needs that anorexia and poor appetite conspire to compound. A 2006 survey by the British Association for Paediatric Nephrology, covering 105 children on dialysis, found that 61% sat below the 10th centile and 44% below the 2nd centile for body mass index when set against healthy peers.[8] Comparable data from India are, at present, lacking.

Anorexia and vomiting are well-recognised in infants with CKD, and it is in this age group that the case for enteral feeding is strongest. Clinical experience suggests that some infants with severe CKD never establish a normal appetite until they have received a transplant. Why certain children develop profound anorexia while others, at an identical level of renal impairment, do not, remains unexplained.[9]

Much of the published guidance on nutrition in paediatric CKD is drawn from adult data or from small, largely dialysis-based paediatric cohorts, and comparable data from India remain sparse. Against this background, the objective of this review is to consolidate current evidence and consensus recommendations on nutritional assessment and dietary management across the stages of paediatric CKD, spanning energy and protein intake, mineral and vitamin D homeostasis, potassium and sodium/fluid balance, and enteral and parenteral nutrition support, so as to provide clinicians with a practical, stage-specific framework for protecting growth and limiting uremic and metabolic complications in this population.

Goals of nutritional care

Across the whole spectrum of paediatric CKD, nutritional care should keep three objectives in view:

a) Maintaining an optimal nutritional state — that is, achieving a normal pattern of growth and body composition through appropriate amounts and types of nutrients.

b) Averting uremic toxicity, metabolic derangement and malnutrition.

c) Reducing the risk of chronic morbidity and premature mortality carried into adult life.

The NKF-KDOQI recommendations set out how the nutritional problems of children with CKD should be addressed.[5] In brief, the nutritional status and growth of every child with CKD stages 2 to 5 and 5D should be reviewed on a regular schedule, and the following parameters should be interpreted together when evaluating children across CKD stages 2 to 5 and 5D:

1) Dietary intake records (a 3-day diet record or three 24-hour dietary recalls).

2) Length- or height-for-age centile or SDS.

3) Length or height velocity-for-age percentile or SDS.

4) Estimated dry weight and weight-for-age percentile or SDS.

5) BMI-for-height-age percentile or SDS.

6) Head circumference-for-age percentile or SDS (in children under three years only).

7) Normalised protein catabolic rate (nPCR) in haemodialyzed adolescents with CKD stage 5D.[2][5]

Dietary management

Dietary management carries great weight in children with CKD, whose deranged renal function reshapes their entire metabolic environment and poses several challenges for the treating team as it works to secure normal growth and development. The starting point is an appraisal of intake, gathered through a 3-day diet diary, 24-hour recalls, or food frequency questionnaires (FFQ).[5,10] Using FFQ data, the CKiD cohort study found that children were in fact taking in more sodium, phosphorus, protein and calories than recommended. This gap between actual intake and guideline recommendations points to a pressing need for better dietary counselling and closer monitoring.[10,11] Equivalent data from India are not available.

Energy

Energy provision should at minimum reach the recommended dietary allowance (RDA) for healthy children of the same height-age (Table 1). Where PEM has already set in, both protein and energy targets must be pushed higher to recover weight gain and linear growth. Calorie intake has to be generous enough to spare dietary protein and to keep the child out of a catabolic state, poor intake is common in this setting, driven by anorexia, nausea and imposed restrictions.[12] When chronological age fails to reflect the child's growth disruption, height-age should serve as the basis for estimating energy needs.

Table 1 Approximate energy requirements by age. In children with CKD, energy is prescribed at 100% of the estimated energy requirement (EER) for chronological age and then adjusted for physical activity level and body size (BMI)

Age group	Energy requirement (kcal/kg/day)
0–6 months	100–110
7–12 months	95–100
1–3 years	100
4–6 years	90
7–10 years	70
Boys, 11–14 years	55
Girls, 11–14 years	47
Boys, 15–18 years	45
Girls, 15–18 years	40

Values are approximate age-based guides, individual requirements are set against the EER for chronological age and refined by response in weight and growth.

For children with CKD stages 2 to 5 and 5D, energy requirements are calculated as 100% of the estimated energy requirement (EER) for chronological age, adjusted for physical activity level and body size (i.e. BMI), and then fine-tuned according to how weight tracks over time. Nutritional supplements deserve consideration whenever a child's usual intake falls short of energy needs and expected weight gain or growth for age is not being met. Energy-dense oral nutritional supplements are the preferred first step, when oral supplementation still cannot meet requirements, tube feeding should be brought into play.

Enteral feeding

Enteral feeding is warranted in children with CKD when calorie or protein intake remains insufficient to sustain growth despite dietary adjustment and medication. As noted, vulnerability to malnutrition peaks during the first six months of life, when growth is fastest and most dependent on nutrition, and responds best to interventions such as gastrostomy feeding. Across Europe and Latin America, nasogastric feeding remains the most commonly used route for this purpose.[7]

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For malnourished children on maintenance haemodialysis (BMI-for-height-age below the 5th centile) who cannot meet their needs through oral and tube feeding, a trial of intradialytic parenteral nutrition (IDPN) is a reasonable step to top up intake. When energy is supplemented orally, enterally or parenterally in children with CKD stages 2 to 5 and 5D, calories should be balanced between carbohydrate and unsaturated fat within the physiological ranges set out as the acceptable macronutrient distribution range (AMDR) of the DRI.

Obesity is surfacing as a fresh problem in paediatric CKD and appears to mirror trends in the wider population. The IPPN registry captures marked regional variation, with mean BMI SDS ranging from 0.8 in the United States to −1.4 in India across children of all ages.[13] In overweight or obese children with CKD stages 2 to 5 and 5D, dietary and lifestyle modification is advised to bring weight under control.

Protein

Protein is needed to hold a positive nitrogen balance for growth and to keep body protein turning over. Intake must be steered carefully between two hazards — protein malnutrition from an over-restricted diet on one side, and accumulation of nitrogenous waste from an over-generous one on the other. A reasonable prescription is 1.1–1.2 g/kg/day, with 60–70% drawn from high-biological-value sources (Table 2). Proteins of high biological value matter a great deal, since they favour muscle anabolism and blunt muscle wasting. Dietary protein intake (DPI) is best kept at 100–140% of the dietary reference intake (DRI) for ideal body weight in CKD stage 3, and at 100–120% of the DRI in stages 4 to 5.[14] In stage 5D, DPI should sit at 100% of the DRI for ideal body weight plus an added allowance for protein and amino acids lost during dialysis. Protein supplements should be considered whenever children with CKD stages 2 to 5 and 5D cannot meet requirements from food and fluids alone. Routine protein restriction has no place in children, as it has not been shown to slow the loss of renal function in paediatric CKD. The distinction between protein-energy wasting (PEW) and malnutrition is set out in Table 3.[15]

Table 2 Recommended dietary protein intake (g/kg/day) for children with CKD, by age and CKD stage (adapted from NKF-KDOQI). HD, haemodialysis, PD, peritoneal dialysis, DRI, dietary reference intake

Age	DRI	Stage 3	Stages 4–5	HD	PD
0–6 months	1.5	1.5–2.1	1.5–1.8	1.6	1.8
7–12 months	1.2	1.2–1.7	1.2–1.5	1.3	1.5
1–3 years	1.05	1.05–1.5	1.05–1.25	1.15	1.3
4–13 years	0.95	0.95–1.35	0.95–1.15	1.05	1.1
14–18 years	0.85	0.85–1.2	0.85–1.05	0.95	1.0

Stage 3 targets 100–140% and stages 4–5 target 100–120% of the DRI for ideal body weight, dialysis figures add an allowance for protein and amino-acid losses. At least 60–70% should be of high biological value.

Table 3 Distinguishing protein-energy wasting (PEW) from simple malnutrition in children with CKD

Feature	Malnutrition	Protein-energy wasting (PEW)
Primary driver	Inadequate nutrient intake	Inflammation and catabolism, compounded by poor intake
Inflammation	Usually absent	Present, often prominent
Serum albumin	May be normal	Typically low
Body compartments	Fat and muscle both reduced	Muscle wasting prominent, weight may be masked by fluid
Resting energy expenditure	Reduced	Normal or increased
Response to feeding	Reverses with adequate intake	Only partial, needs anti-inflammatory measures too
Reversibility	Reversible	Difficult to reverse with nutrition alone

Phosphorus and calcium

Childhood and adolescence are the years in which bone is laid down, with some 90% of peak bone mass accrued by age 18. Calcium supplementation therefore matters for growth and for guarding against fractures. The concern is not theoretical: one large prospective study in early CKD reported a two- to three-fold higher fracture rate than in healthy controls,[16] and in another series roughly 10% of children and young adults with CKD stages 4 to 5 or on dialysis carried a lifetime history of fracture.[17]

As GFR declines, phosphate excretion falls and serum phosphorus rises. Two measures counter this drift: dietary phosphorus restriction and the regular use of phosphate binders with meals. In children with CKD stages 3 to 5 and 5D, cutting dietary phosphorus to 100% of the age-appropriate DRI is advised when parathyroid hormone (PTH) is above the target range for the CKD stage but serum phosphorus is still within the age-specific reference range. When PTH is above target and serum phosphorus has also climbed above the reference range, the intake should be trimmed further, to 80% of the DRI. Once restriction is under way, serum phosphorus is best checked at least every three months in stages 3 to 4 and monthly in stages 5 and 5D. At every stage, the aim is to keep serum phosphorus from straying either above or below the age-specific reference range.[18]

For children with CKD stages 2 to 5 and 5D, total calcium intake from food and phosphate binders together is best held between 100% and 200% of the DRI. Recommended elemental calcium runs to 500–600 mg/day for ages 1–10 years and 800–1000 mg/day for ages 11–18 years, corresponding to roughly 80–100 mg/kg/day. Sustained high phosphorus loads impair growth and, over time, invite renal osteodystrophy, while prolonged elevation of both calcium and phosphorus drives vascular calcification.

Vitamin D

In children with CKD stages 2 to 5 and 5D, serum 25-hydroxyvitamin D is best measured once a year, a level below 30 ng/mL (75 nmol/L) calls for supplementation with either ergocalciferol (vitamin D2) or cholecalciferol (vitamin D3). During repletion, corrected total

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calcium and phosphorus should be checked one month after starting or changing the dose and at least every three months thereafter. Ergocalciferol and all other forms of vitamin D should be stopped if serum calcium exceeds 10.2 mg/dL, if serum phosphorus rises above 4.6 mg/dL, the phosphate binder dose should be added to or increased, and persistent hyperphosphatemia is itself grounds to withhold vitamin D.[19] Once a child is replete, continued vitamin D supplementation with yearly monitoring of 25-hydroxyvitamin D is appropriate.

Potassium

Potassium should be prescribed individually against measured serum levels, with roughly 1600–2400 mg/day a usual working range.[20] Levels warrant close and continuing monitoring, with intake adjusted as they change. Hyperkalaemia can follow excessive intake of potassium-rich foods, catabolism, or other causes, where it appears, leaching of pulses and vegetables before cooking is a useful measure. Anuric children need particular care. Because the gut handles as much as 30% of potassium excretion in chronic renal failure, regular daily bowel movements are therefore an important, easily overlooked part of potassium management.

Sodium and fluid

A no-added-salt approach, together with avoidance of salty snacks, is the sensible default. How far salt should be limited depends on the presence of oedema and hypertension and on any sodium-containing medications, a ceiling below 2400 mg/day is a reasonable target. In renal patients even a modest reduction in salt brings lower blood pressure, less proteinuria and better outcomes.[21]

When a child is both hypertensive and oedematous, salt and fluid should be curtailed more strictly — the notable exception being salt-losing nephropathies, in which sodium is lost excessively in the urine and restriction would be harmful. Dietary needs shift again once a child moves on to dialysis or transplantation, and a dietitian should be re-engaged at that point.

Vitamins and trace elements

The published literature does not yet support blanket recommendations on routine supplementation or on measuring vitamin and trace-element status in children on dialysis. Supplemental dosing is therefore best individualised to each child's needs, risk profile and anticipated dialysis losses. Even so, an intake supplying at least 100% of the DRI for thiamine (B1), riboflavin (B2), niacin (B3), pantothenic acid (B5), pyridoxine (B6), biotin (B8), cobalamin (B12), ascorbic acid (C), retinol (A), α-tocopherol (E), vitamin K, folic acid, copper and zinc should be considered for children with CKD stages 2 to 5 and 5D.[5,22]

Supplementation of vitamins and trace elements is advised for children with CKD stages 2 to 5 when diet alone falls short of 100% of the DRI, or when there is clinical evidence of deficiency — ideally corroborated by low blood levels of the nutrient in question. Children with CKD stage 5D should, as a matter of course, receive a water-soluble vitamin supplement.

Conclusion

Taken together, the evidence reviewed here confirms that nutritional care is an indispensable strand of management in paediatric CKD, directly shaping growth, uremic and metabolic control, and long-term morbidity. Regular monitoring of intake and growth, individualised prescription of energy, protein, minerals and vitamin D against stage-specific targets, and timely use of enteral or parenteral support where oral intake is insufficient together form the practical framework set out in this review. At the same time, the evidence base remains skewed toward adult and dialysis populations, and data from India and other under-represented settings are still limited, underscoring the need for further paediatric-specific research. Restrictions imposed on diet and fluid are disorienting and impose a heavy burden on affected children and their families, and this burden must not be understated. Approaches that put the child's own priorities first, that draw on their motivation to stay well enough for transplantation or to stave off dialysis, and that frame adaptation to restriction as a shared journey rather than a series of prohibitions, offer the most promising way to lighten that load while keeping growth and quality of life at the centre of care.

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Conflicts of interest

The authors declare no conflicts of interest.

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