Paediatric Chronic Kidney Disease

Pearl Pugh, PhD, MA, PG Dip Diet (SR), BSc (Hons)
Clinical Specialist Paediatric Renal Dietitian, Nottingham Children's Hospital, Nottingham University Hospitals NHS Trust, and Research Fellow, School of Medicine & Health Sciences University of Nottingham

Chronic kidney disease (CKD), classified as stages 1-5, may present at birth or later in life, progressing to established renal failure during childhood. Underlying diagnosis and classification of CKD stage will inform the treatment and dietary management. A nutritional assessment, essential to the management of children with CKD, is guided by an assessment of the child's growth and dietary intake. Early intervention by the dietitian, with regular reviews to respond to the changing and evolving needs of the child, ensures optimisation of nutrition and growth, and cardiac and bone health.

CKD & classification

CKD has been defined by the global body – Kidney Disease Quality Outcome Initiative (KDOQI) – as any kidney impairment causing a decrease in the glomerular filtration rate (GFR) for 3 months or more.[1] Early abnormal signs leading to a diagnosis of CKD may include blood (haematuria) and protein (proteinuria) in the urine or elevated blood pressure (hypertension).[2]

Kidney function is measured using the GFR by assessing blood flow through the glomeruli per minute.[3] It is expressed as a rate corrected for the body surface area of the average adult (1.73 m^2). Glomeruli are a mesh of capillaries in the kidneys that filter the blood as the first step in the nephrons' homeostatic elimination of waste. Healthy kidneys have a GFR of >90 ml/min/1.73 m^2;[4] therefore making it accessible to patients to consider GFR as a percentage of kidney function. In children and young people, the gold standard for measuring GFR involves the clearance of the complex sugar inulin from the kidneys by applying tests to timed urine samples.[5] In clinical practice other tracers are used, most commonly radioisotopes, such as 51Cr-EDTA and more recently DTPA (diethylenetriamine pentaacetate).[6] Such techniques involve repeated blood sampling after an intravenous injection of tracer and are not suitable for frequent repeated measurements. An easier, strong, surrogate marker is to estimate GFR (eGFR) using the serum creatinine level and height (as a measure of muscle mass). The Schwartz formula defines eGFR as a constant multiplied by height and divided by serum creatinine:[7]

$$\text{eGFR} = \frac{\text{k x Height (cm)}}{\text{Plasma creatinine µmol/L}}$$

K = 36.5 in males aged >13 years
K = 32.5 in others

CKD is classified as stages 1-5, from early kidney disease at stage 1 to established renal failure at stage 5d when dialysis is required. During the early stages (CKD 1-3b, see **Table 1**) there are no overt symptoms, or feelings of ill health, however underlying bone changes are taking place.[8] As CKD progresses, tiredness, biochemical changes, anaemia, fluid retention and hypertension may present. Established renal failure, marked by an eGFR <15ml/min/1.73 m^2, is accompanied by advancing symptoms and preparations for renal replacement therapy (RRT) – dialysis – or transplantation.[9]

Disease progression

Although the rate of progression varies, all children with CKD will experience deterioration in their renal function over time. The rate of decline in CKD function depends on the underlying disease. **Table 2** details some of the common aetiologies of CKD in children.

Prevalence of renal replacement therapy

Globally, the prevalence of RRT in 0-19-year-olds ranges from 18 to 100 per million age-related population (pmarp).[10] In the UK the prevalence of RRT in children and young people (<16 years) is 60.4 pmarp.[11] This may reflect early screening and improved access to treatment. By contrast, the prevalence in the United States is 85 pmarp, while much lower in Japan (34 pmarp) and Brazil (23 pmarp).[10] CKD, with its many underlying causes (see **Table 2**), is often identified antenatally and parents are educated about CKD when their child is a newborn.

Table 1: Stages of CKD[9]

CKD stage	Treatment required	Description	GFR ml/min/1.73 m^2
1	Kidney damage with normal or reduced GFR. Asymptomatic, but require annual blood pressure monitoring.	Do not usually feel unwell.	<90
2	Mildly reduced kidney function. Asymptomatic, but require routine blood pressure monitoring.		89-60
3a	Moderately reduced kidney function. Likely asymptomatic, with possible early signs of fatigue and fluid retention.		59-45
3b			45-30
4	Severely reduced kidney function. Biochemical changes, anaemia, fluid retention and hypertension may present. Preparing for established renal disease and dialysis.	May feel tired and symptomatic	30-15
5	Established renal failure. Advancing of the above symptoms. Preparing for established renal disease and dialysis.		<15
5d	Established renal failure. Receiving dialysis.		Dialysis

Source: Adapted from KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease[9]
Key: Green shaded area indicates the early stages of CKD

Table 2: Examples of the causes of CKD in CYP[10]

Aetiology	Description
Congenital anomalies of the kidney and urinary tract (CAKUT)	A group of abnormalities affecting the kidneys or other structures of the urinary tract.
Reflux nephropathy	Kidney damage due to urine flowing backward from the bladder towards the kidneys.
Obstructive uropathy	Structural or functional interference of normal urine flow.
Glomerulonephritis	Acute inflammation of the kidney due to an immune response.
Hereditary nephropathy	A genetically varied disorder characterised by blood or protein in the urine and hypertension.
Cystic kidney disease	A range of hereditary developmental and acquired kidney conditions, such as autosomal dominant polycystic kidney disease.

Source: Adapted from Harambat J, *et al.* (2012)[10]

Optimising nutritional & growth status

A nutritional assessment, essential to the management of children with CKD, is guided by an assessment of the child's growth and dietary intake. The causes of a poor nutritional status and the impact on growth have been well-described.[12, 13] The assessment of a child's spontaneous dietary intake (without requiring repeated encouragement), and appetite, is the most important determinant of the nutritional status of children with CKD.[14] Frequency of dietary assessment will be guided by many factors, including the underlying disease, severity and stage of CKD, any nutritional concerns, serial measurements of growth – height, weight and head circumference, excessive or inadequate dietary intake, quality of the diet, gastrointestinal symptoms, and deranged biochemical indices.[15] When an older child is unable to stand, or is acutely unwell, the most useful surrogate method for measuring height is arm span. Although it may underestimate standing height, studies demonstrate that it is reproducible and consistent.[16, 17]

As there are no dietary assessment tools tailored specific to the needs of children with CKD, validated tools to assess dietary intake in the general paediatric population are applied to children with CKD.[18-22] The most accurate tool to determine dietary intake is a prospective 3+ day dietary record.[15] The accuracy of this method may be compromised in older children when eating/snacking with friends, when families eat outside of the home or with extended family/carers, literacy problems, or due to hectic lives with limited time and support. In the clinical setting, the 24-hour diet recall is frequently used as the next most reliable assessment tool;[15] with technological variants including pictorial food records, food tracking apps or emailed records, potentially improving accuracy.

In practice, serial measurements of weight and height on standard growth charts, allow growth trends to be monitored, and any deviation across the centiles can easily be identified. For children with oliguria or anuria, or unstable relapsing nephrotic syndrome, fluid retention may be an issue. Likewise, if receiving renal replacement therapy, fluid retention can mask a child's true weight. In these situations, the post haemodialysis, or the end of peritoneal dialysis (PD), continuous ambulatory PD or automated PD, weight should be used. This is known as a child's euvolemic or 'dry' weight – often it is a somewhat crude estimate of their true weight.

Nutritional prescription

If a child has the capacity for spontaneous intake by mouth, this should be encouraged. When a nutritional assessment demonstrates inadequate dietary intake to support appropriate growth, the diet should be optimised with the tailored use of nutritional supplements. These products provide either individual macronutrients, which can fortify individual foods/meals and deliver energy and/or protein, or complete nutritional products which supplement a child's overall daily intake. If faltering growth continues to be an issue, enteral tube feeding should be considered.[23] As described in the Paediatric Renal Nutrition Taskforce Clinical Practice Recommendation for delivery of nutritional prescription by enteral tube feeding, supplemental or exclusive enteral tube feeding should begin when a child is not meeting their nutritional requirements orally, often evidenced by a downward trend on the weight and height centile charts.[24] Proactive dietary intervention should be encouraged irrespective of age. However, there is clear evidence of improved growth following tube feeding in the infantile phase, when growth is predominantly driven by nutrition.[25, 26]

Types of tube feeding

As a short-term enteral feeding option, or bridge to a longer-term alternative, nasogastric (NG) feeding should be considered in practice. There are many pros and cons to NG feeding: insertion is simple and can easily be taught, and there is no risk of peritonitis in children receiving PD. However, displacement is a common feature with the need for repeated replacement.[22] It can inhibit the development of oral motor skills, resulting in subsequent speech and swallowing difficulties.[24] Parents report the presence of an NG tube to be a visible sign that a child is unwell.[22] Vomiting, a frequently reported problem in children with CKD,[12] is often associated with NG tube feeding, with the associated risk of aspiration.[27] The use of continuous, unsupervised, overnight NG feeding is contraindicated in the home environment due to the risk of displacement and aspiration.[24]

"Although the rate of progression varies, all children with CKD will experience deterioration in their renal function over time. The rate of decline in CKD function depends on the underlying disease."

Gastrostomy tube feeding is considered the standard for long-term enteral feeding by the European Society for Paediatric Gastroenterology Hepatology and Nutrition (ESPGHAN) and the American Society for Parenteral and Enteral Nutrition (ASPEN).[28, 29] It can also help with the administration of daily fluids and medication. However, gastrostomy devices are not without associated problems, including malfunctioning tubes, and leakage of gastric juices causing skin breakdown. More serious, but less frequent, problems include intra-abdominal leakage and peritonitis.[27, 28]

The decision to tube feed is not one that is taken lightly, and must involve time for discussion with the parents and caregivers, providing clear rationale for why it is necessary, what it will involve, and the potential complications. Tube feeding allows for supplementary or exclusive enteral feeding at a rate and volume best suited to the child's needs and the family's routine. It can allow for the routine of family meals during the day, with the addition of a supplemental feed overnight, with the exception of continuous NG feeding. Vomiting may be reduced by continuous gastrostomy feeding.[22]

Monitoring

All children with CKD require a nutritional and growth assessment by a paediatric renal dietitian, and on-going dietary support to monitor and respond to their changing and evolving needs as the CKD progresses. The frequency of monitoring reflects the individual child's status (as described in the above section 'Optimising nutritional & growth status'), cardiac and bone health; with a proactive plan to start monitoring bone biochemistry from CKD stage 2. Serial monitoring of growth, phosphate, potassium, calcium, vitamin D and parathyroid hormone will inform dietary change. Early dietary advice to moderate phosphate intake may delay or halt CKD progression.[30, 31, 32] The advice should focus on the reduction of processed foods with their rich inorganic phosphate content, and the promotion of more home-cooked, plant-based options in the family diet.

As the child progresses to established kidney disease at CKD stages 4 and 5, the frequently fluctuating clinical and biochemical changes require more regular dietetic reviews. Practical resources, including written or online information, recipes, swap lists, a credit card size pocket guide, educational games, and online cookery workshops can provide education, support and upskilling to motivate adherence to the diet across the CKD journey.[33, 34]

In practice, regular review of phosphate binders will ensure optimal timing, and tailored dosing with meals and snacks. When administering an enteral feed via a nasogastric or gastrostomy device, the phosphate binders may be mixed into the feed or given as a bolus dose down the tube, at the beginning and/or end of the feeding period. Calcium-based phosphate binders will contribute to the overall calcium load consumed, this needs to be accounted for in the child's bespoke nutritional assessment.

Conclusion

Children with CKD require specialist paediatric renal dietetic assessment and ongoing frequent monitoring to optimise nutrition and growth, while minimising cardiac and bone health complications. The proactive use of oral nutritional supplements can enhance the delivery of an adequate nutritional intake. When serial measurements of growth and spontaneous oral intake are suboptimal, enteral tube feeding should be discussed with the parents and caregivers. The progression of CKD requires frequent reviews and adaptation of dietary advice, with practical resources to enable adherence and adaptation of the family diet.

References: **1.** Levey AS, *et al.* (2005) Definition and classification of chronic kidney disease: A position statement from Kidney Disease: Improving Global Outcomes (KDIGO). Kidney Int.; 67(6): 2089-2100. **2.** Byrne C, *et al.* (2018) 20th Annual Report of the Renal Association UK Renal Registry, Bristol, UK. Nephron.; 139(1). **3.** Soveri I, *et al.* (2014) Measuring GFR: A systematic review. Am J Kidney Dis.; 64(3): 411-424. **4.** Denic A, Glassock RJ, Rule AD. (2016) Structural and Functional Changes with the Aging Kidney. Adv Chronic Kidney Dis.; 23(1): 19-28. **5.** Schwartz GJ, Work DF. (2009) Measurement and Estimation of GFR in Children and Adolescents. Clin J Am Soc Nephrol.; 4(11): 1832-1843. **6.** Soveri I, *et al.* (2014) Measuring GFR: A systematic review. Am J Kidney Dis.; 64(3): 411-424. **7.** Schwartz GJ, *et al.* (1976) A simple estimate of glomerular filtration rate in children derived from body length and plasma creatinine. Pediatrics.; 58(2): 259–263. **8.** Wesseling-Perry K, Salusky IB. (2013) Chronic kidney disease: mineral and bone disorder in children. Semin Nephrol.; 33(2): 169-179. 9. KDIGO (2013). KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Accessed online: https://kdigo.org/wp-content/uploads/2017/02/KDIGO_2012_CKD_GL.pdf (May 2023). **10.** Harambat J, *et al.* (2012) Epidemiology of chronic kidney disease in children. Pediatr Nephrol.; 27(3): 363–373. **11.** Hamilton AJ, *et al.* (2016) UK Renal Registry 18th Annual Report: Demography of patients receiving renal replacement therapy in paediatric centres in the UK in 2014. Nephron.; 132(Suppl 1): 99-110. **12.** Rees L, Jones H. (2013) Nutritional management and growth in children with chronic kidney disease. Pediatr Nephrol.; 28(4): 527–536 **13.** Silverstein AM. (2018) Growth and nutrition in pediatric chronic kidney disease. Front in Pediatr.; 6: 205. **14.** Ponton-Vazquez C, *et al.* (2017) Dietary intake, nutritional status, and body composition in children with endstage kidney disease on hemodialysis or peritoneal dialysis. J Ren Nutr.; 27(3): 207-215. **15.** Nelms CL, *et al.* Assessment of nutritional status in children with kidney diseases—clinical practice recommendations from the Pediatric Renal Nutrition Taskforce. Pediatr Nephrol.; 36(4): 995-1010. **16.** Forman MR, *et al.* (2014) Arm span and ulnar length are reliable and accurate estimates of recumbent length and height in a multi-ethnic population of infants and children under 6 years of age. J Nutr.; 144(9): 1480-1487. **17.** Monyeki KD, Sekhotha MM. (2015) The relationship between height and arm span, mid-upper arm and waist circumferences and sum of four skinfolds in Ellisras rural children aged 8-18 years. Public Health Nutr.; 19(7): 1195-1199. **18.** Bandini LG, *et al.* (1997) Validity of reported energy intake in preadolescent girls. Am J Clin Nutr.; 65(4 suppl): 1138S–1141S. **19.** Champagne CM, *et al.* (1998) Assessment of energy intake underreporting by doubly labeled water and observations on reported nutrient intakes in children. J Am Diet Assoc.; 98(4): 426-433. **20.** Yang J, *et al.* TEDDY Study Group (2016) Factors associated with longitudinal food record compliance in a paediatric cohort study. Public Health Nutr.; 19(5): 804-813. **21.** Gondolf UH, *et al.* (2012) Validation of a pre-coded food record for infants and young children. Eur J Clin Nutr.; 66(1): 91-96. **22.** Kobayashi T, *et al.* (2011) Reproducibility and validity of the food frequency questionnaire for estimating habitual dietary intake in children and adolescents. Nutr J.; 10: 27 **23.** Shaw V, *et al.* (2020) Energy and protein requirements for children with CKD stages 2-5 and on dialysis–clinical practice recommendations from the Pediatric Renal Nutrition Taskforce. Pediatric Nephrol.; 35(3): 519-531. **24.** Rees L, *et al.* (2021). Delivery of a nutritional prescription by enteral tube feeding in children with chronic kidney disease stages 2–5 and on dialysis – clinical practice recommendations from the Pediatric Renal Nutrition Taskforce. Pediatr Neprol.; 36(1): 187-204. **25.** Sienna JL, *et al.* (2010) Body size in children with chronic kidney disease after gastrostomy tube feeding. Pediatr Nephrol; 25(10): 2115-2121. **26.** Rees L, *et al.* International Pediatric Peritoneal Dialysis Network (IPPN) registry (2011) Growth in very young children undergoing chronic peritoneal dialysis. J Am Soc Nephrol.; 22(12): 2303-2312. **27.** Samaan S, Secker D. (2014). Oral feeding challenges in infants with chronic kidney disease. Infant Child Adoles Nutr.; 6(3): 164-171. **28.** Heuschkel RB, *et al.* (2015) ESPGHAN Position Paper on Management of Percutaneous Endoscopic Gastrostomy in Children and Adolescents. J Pediatr Gastroenterol Nutr.; 60(1): 131-141. **29.** ASPEN Enteral Nutrition Practice Recommendations (2009). J Parenter Enter Nutr.; https://onlinelibrary.wiley.com/doi/epdf/10.1177/0148607108330314. **30.** McAlister L, *et al.* (2020). The dietary management of calcium and phosphate in children with CKD stages 2-5 and on dialysis – clinical practice recommendation from the Pediatric Renal Nutrition Taskforce. Pediat Nephrol.; 35(3): 501-518. **31.** Portale AA, *et al.* (2016) Fibroblast growth factor 23 and risk of CKD progression in children. Clin J Am Soc Nephrol.; 11(11): 1989-1998. **32.** Komaba H, Fukagawa M. (2016) Phosphate – a poison for humans? Kidney Int.; 90(4): 753-763. **33.** Vitaflo International Ltd (2022). Paediatric Renal Nutrition Taskforce| Clinical Practice Recommendations. Accessed online: www.vitaflo-via.com/disorder-resources/paediatric-kidney-disease/paediatric-renal-nutrition-taskforce-clinical-practice (April 2023). **34.** Pugh P. (2022). Early dietary intervention. A tool to delay disease progression in children with early-stage CKD. Complete Nutrition.; 22(3): 19-22.