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3.21 Nutritional Management in Children with Chronic Kidney Disease

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Key Words

Chronic kidney disease · Short stature · Supplements · Protein · Enteral feeding

Key Messages

- Short stature is a common complication of chronic kidney disease. Inadequate intake to meet nutritional needs is a well-described cause of poor growth, and this worsens as the glomerular filtration rate declines
- Linear growth is particularly vulnerable during the infantile phase of growth; without early nutritional intervention, losses of as much as 2 height standard deviations may occur in the first 6 months of life
- Children with congenital abnormalities of the kidneys and urinary tract may lose salt (Na), water and bicarbonate and have chronic volume contraction and acidosis which impair growth; supplementation with sodium chloride and bicarbonate and free access to water is important in these circumstances
- Energy intakes should be maintained at a level equivalent to the estimated average requirement for the normal population of the same age, and protein intake at the recommended dietary intake for height age. Protein supplementation may be needed to compensate for dialysate losses
- Phosphate restriction is often necessary to prevent hyperparathyroidism. This may lead to calcium and vitamin D deficiency, since these nutrients are mainly found in phosphate-containing foods

Introduction

Children with chronic kidney disease (CKD) have a mortality that is 30 times higher than that of age-matched, healthy children. There is also a high incidence of malnutrition and short stature. These factors are interlinked: short stature at the start of dialysis is associated with a 2-fold increased mortality risk, decreased school attendance and increased hospitalisation. It is likely that malnutrition contributes to this as serum albumin correlates with morbidity and mortality [1]. Thus, strict attention to nutrition is essential to optimise linear growth, wellbeing and survival.

Epidemiology of Growth

National and international registries all show below-average height for children with CKD. In the USA, around one third have a height standard deviation score (Ht SDS) of less than the 3rd percentile, rising to half by the time they need dialysis, and with a continuing decline thereafter. There is, however, great variation, with a mean Ht SDS for children on dialysis ranging from –1.3 in the UK to –3.5 in Brazil amongst 21 countries. The mean BMI SDS does not parallel the Ht SDS,

being highest in the USA at 0.8 and lowest in India at –1.4. It is likely that some of these international differences reflect limited access to adequate resources in the developing world [2]. Infants are particularly vulnerable and more severely affected. Posttransplant catch-up growth depends on graft function and use of steroid therapy, and is most likely to occur in younger children. Secular trends in recent decades show that height growth is improving for all children with CKD [3].

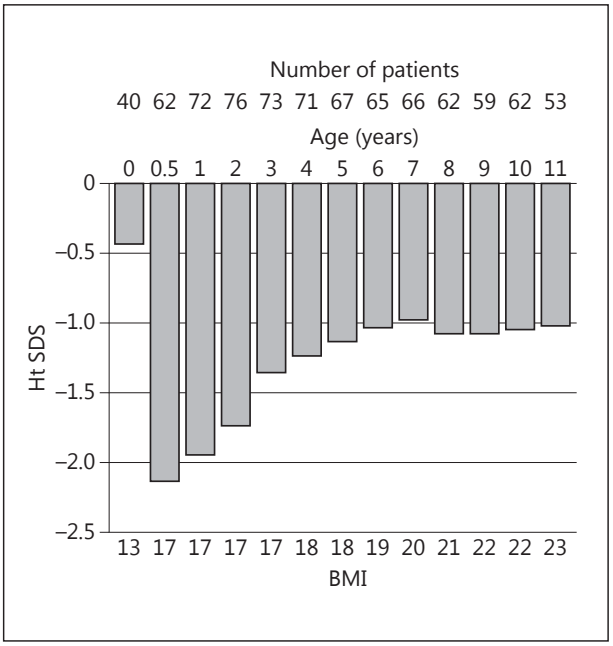

Fig. 1. Ht SDS of infants with CKD 5 showing the decline over the first 6 months of life. Adapted from Mekahli et al. [5].

Causes of Poor Growth

The best-described cause of poor growth is decreased nutritional intake. However, aetiologies are multiple and include: metabolic disturbances such as acidosis, chronic sodium depletion, and mineral and bone disorder; anaemia; and the growth hormone/insulin-like growth factor 1 axis and other hormonal derangements. Dialysis dose is another important factor affecting dietary intake, nutritional status and growth [1].

Causes of Poor Nutritional Intake

CKD is characterised by a predisposition to anorexia and vomiting. Poor appetite may be due to abnormal taste sensation, the requirement for multiple medications, preference for water in the polyuric child, and a full abdomen in the child on peritoneal dialysis (PD). Vomiting is common, particularly with infants, and may result from gastro-oesophageal reflux and delayed gastric emptying in association with increased polypeptide hormones. The use of prokinetic, antireflux and antinausea drugs may be of benefit, although in infants with severe vomiting, a Nissen fundoplication may be necessary. Inadequate intake may occur during periods of sepsis and surgery, or as a result of fluid restriction in the child on dialysis. Loss of amino acids and protein occurs in dialysate. Acidosis and inflammation increase circulating cytokines such as leptin; levels can paradoxically be high in malnourished patients, since this hormone is excreted by the kidneys and not cleared by dialysis, thus contributing further to decreased food intake and increased energy needs [1].

Management of Poor Nutrition

Ensuring adequate nutrition in order to promote optimum growth is the most important aspect of care of a child with CKD. The aim is to control symptoms and prevent complications, particularly uraemia and renal bone disease. There is also some evidence that ensuring normal bicarbonate and phosphate levels may slow down the progression of CKD. In 2008 the Kidney Disease Outcomes Quality Initiative, a group of experts in the field of dietary management in children with

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Table 1. Energy and protein requirements according to age for CKD and for children on dialysis

	Energy,[1] kcal/kg	Protein RNI, g/kg/day	Protein for PD, g/kg/day	Protein for HD, g/kg/day
Preterm	120–180	2.5–3.0	3.0–4.0	3.0
0–3 months	115–150	2.1	≥2.4	≥2.2
4–6 months	95–150	1.6	≥1.9	≥1.7
7–12 months	95–150	1.5	≥1.8	≥1.6
1–3 years	95–125	1.1	≥1.4	≥1.2
4–6 years	90–110	1.1	≥1.3	≥1.1
7–10 years	1,740♀ – 1,970♂ kcal/day	28 g/day	≥1.2	≥1.0
11–14 years	1,845♀ – 2,220♂ kcal/day	42 g/day	3.0–4.0	3.0
15–18 years	2,110♀ – 2,755♂ kcal/day	55 g/day♂; 45 g/day♀	≥2.4	≥2.2

RNI + 0.3 g/kg/day to compensate for losses on PD; RNI + 0.1 g/kg/day to compensate for losses on HD. HD = Haemodialysis. [1] Estimated average requirement.

CKD, wrote guidelines covering all aspects of their nutritional care. These are used internationally [4].

The Role of the Dietician

Involvement of a paediatric renal dietician is essential for successful nutritional management. The aim is to preserve normal growth and body composition; this can be achieved by consuming appropriate amounts of calories, protein, fat, sodium, water, bicarbonate, iron, calcium, phosphate, vitamins and minerals. Nutritional assessment requires that height, weight and head circumference are plotted on centile charts at regular intervals. The most vulnerable time is infancy, and in particular the first 6 months of life, when loss of as much as 2 Ht SDS can occur (fig. 1) [5]. Frequent review is necessary for early detection of a decline in height gain velocity; prevention rather than treatment of malnutrition is the goal, so early intervention is crucial.

Energy

The diet should contain 100% of the estimated average requirement for energy for chronological age. Inadequate energy from non-protein sources will result in the use of dietary protein for energy rather than growth, and an increase in plasma urea and potassium levels. Children on PD having glucose-containing dialysate may absorbe up to 10–12 extra kilocalories per kilogram body weight per day [6].

Protein

Protein intake must provide at least 100% of the reference nutrient intake (RNI) to prevent growth failure. Serum albumin should be normal. To ensure adequate protein intake, the RNI for height age is used if the child is below the 3rd centile. On the other hand, excess protein will result in a high blood urea and toxic by-products of metabolism. The aim is for plasma urea levels to be <20 mmol/l in children under 10 years, and <30 mmol/l thereafter; nausea and anorexia increase when the urea exceeds 20 mmol/l. The blood urea level is a reflection of protein intake, unless there is a catabolic state, at which time it reflects tissue breakdown. A very low urea level suggests low protein intake and risk of protein malnutrition. Dietary protein intake is rarely inadequate in predialysis CKD, but on dialysis a

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supplement is needed to replace losses in dialysate (table 1). These losses are greatest in infants and after peritonitis [6].

Calcium and Phosphate

Dietary phosphate usually should be restricted in CKD to prevent secondary hyperparathyroidism. Phosphate is principally in protein-containing foods such as meats and dairy products. Processed foods contain phosphate in significant quantities as it is a component of moisture and flavour enhancers. Foods that are a good source of calcium and vitamin D are also high in phosphate, so phosphate restriction is commonly associated with 25-hydroxyvitamin D and calcium deficiency [6].

Sodium, Bicarbonate and Water

The requirements for salt, water and bicarbonate vary with the type of renal disease. Children with congenital abnormalities of the kidneys and urinary tract, which predominantly affect the renal tubule, are usually sodium, bicarbonate and water losers. Therefore, these children need salt and bicarbonate supplementation and free access to water. Many infants on PD lose excessive amounts of sodium in dialysate, and they too need supplementation. Children with CKD predominantly due to glomerular disease may retain salt and water and develop hypertension. Such children should be managed with a 'no-added-salt' diet [3].

Potassium

CKD can be associated with potassium retention, but hyperkalaemia does not usually occur until the glomerular filtration rate is <10% of normal. Adequate control of plasma potassium can usually be achieved by improving the energy intake to prevent protein catabolism and the avoidance of foods that are very high in potassium [6].

Vitamins and Minerals

Few data are available on the micronutrient requirements for children with CKD. Vitamin supplements should not be routinely prescribed as renal excretion of vitamin A metabolites is impaired in CKD. Nutritional vitamin D (25-hydroxyvitamin D) deficiency is common, and the activated form may also need replacement as the glomerular filtration rate falls to <40 ml/min/1.73 m^2 [6].

Enteral Feeding

Oral supplements and/or enteral feeding are necessary when spontaneous intake is inadequate to maintain growth, and should be considered as soon as the growth rate falls below normal. Percentage time with gastrostomy feeding has been shown to be an important positive predictor of longitudinal growth in under-2-year-olds on PD, being superior to provision of feed orally or by nasogastric tube [7]. Reports of whether enteral feeding can induce catch-up when started after the infantile phase of growth are conflicting, although it can improve nutritional status [3]. Supplements can be given as top-up bolus feeds, an overnight feed or an overnight continuous feed with daytime boluses, depending on the severity of the anorexia. A whey-based formula is used for children aged <2 years and a whole-protein enteral feed for those aged >2 years. These can be supplemented with fat or carbohydrate or both. Protein can be supplemented as a whey protein concentrate with amino acids [6].

Obesity

Obesity in CKD is increasing around the world, paralleling its incidence in the normal population. A high BMI increases the risk of cardiovascular disease, to which patients with CKD are already predisposed. Care must be taken not to augment energy intake above requirements, particularly in an enterally fed child [1].

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References

1 Rees L, Mak RH: Nutrition and growth in children with chronic kidney disease. Nat Rev Nephrol 2011;7:615–623.
2 International Pediatric Dialysis Network. http://www.pedpd.org/index.php?id=98.
3 Rees L, Jones H: Nutritional management and growth in children with chronic kidney disease. Pediatr Nephrol 2013;28:527–536.
4 KDOQI Work Group: KDOQI Clinical Practice Guideline for Nutrition in Children with CKD: 2008 Update. Am J Kidney Dis 2009;53(suppl 2):S11–S104.
5 Mekahli D, Shaw V, Ledermann SE, Rees L: Long-term outcome of infants with severe chronic kidney disease. Clin J Am Soc Nephrol 2010;5:10–17.
6 Rees L, Shaw V: Nutrition in children with CRF and on dialysis. Pediatr Nephrol 2007;22:1689–1702.
7 Rees L, Azocar M, Borzych D, Watson AR, Büscher A, Edefonti A, Bilge I, Askenazi D, Leozappa G, Gonzales C, van Hoeck K, Secker D, Zurowska A, Rönnholm K, Bouts AH, Stewart H, Ariceta G, Ranchin B, Warady BA, Schaefer F; International Pediatric Peritoneal Dialysis Network (IPPN) registry: Growth in very young children undergoing chronic peritoneal dialysis. J Am Soc Nephrol 2011;22:2303–2312.

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